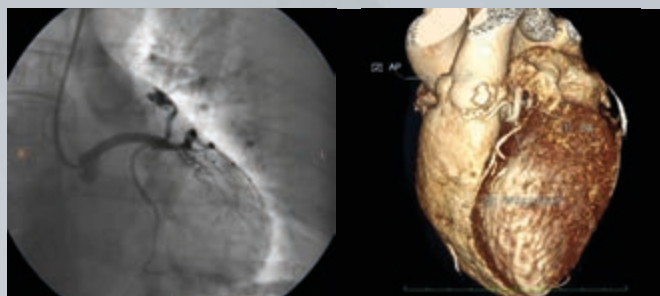




ARGENTINIAN JOURNAL OF INTERVENTIONAL CARDIOLOGY

October - December 2020 | Year 11 | Issue 4



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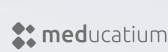
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UNIVERSIDAD DE BUENOS AIRES - FACULTAD DE MEDICINA

IX PROGRAMA DE ACTUALIZACIÓN UBA-CACI 2020/2022

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Manejo de la patología aórtica

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Enfermedad Vascular Esplácnica y Periférica

(marzo a junio 2022)

En agosto 2020 comienza el IX Programa de Actualización en Cardioangiología Intervencionista UBA-CACI dirigido a Cardioangiólogos Intervencionistas formados. Debido a las circunstancias actuales las charlas teóricas serán virtuales, teniendo en cuenta "en un futuro cercano" la posibilidad de sesiones con simuladores en el CACI y rotaciones para presenciar casos "en vivo".

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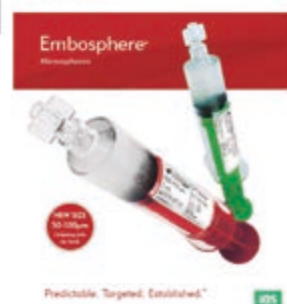
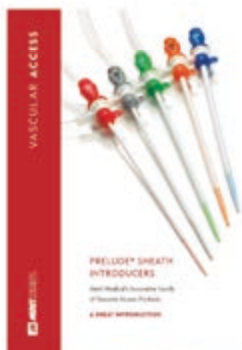
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EDITORIAL / EDITORIAL

PERCUTANEOUS IMPLANTATION OF AORTIC VALVE AT LOW OR INTERMEDIATE RISK: WHEN EFFECTIVENESS TRUMPS COST

Rodríguez AE

Very few things in internal medicine and/or interventional cardiology have been more interesting to me when doing clinical research than analyzing the cost-effectiveness ratio of revascularization techniques or strategies and/or therapy. Since the times of the randomized clinical trials between angioplasty vs coronary surgery to the rational use of stents until the option of using immunosuppressants to prevent restenosis, the cost-effectiveness ratio has always been studied from 2 different therapeutic positions. We used to base these different positions and we still do today on a similar effectiveness hypothesis of the hard cardiovascular adverse events we wish to compare between both techniques like rate of death, infarction or stroke. In these circumstances both the costs and the cost-effectiveness of each strategy can be analyzed. To this day, transcatheter aortic valve implantation (TAVI) was and still is the most important therapeutic achievement in our specialty. First designed for patients contraindicated for surgical aortic valve replacement (SAVR) or with high risk of SAVR related death (6-7), believe it or not, these are the only indications accepted to this day by our country's social security system. Over the last few years, the greater knowledge acquired in the implantation technique and the improvements made in the devices used that facilitate the use of percutaneous implants in over 90% of the cases via transfemoral access has brought TAVI indications to study in low and intermediate risk patients.

REVIEW ARTICLES / ARTÍCULOS DE REVISIÓN

TRANSCATHETER AORTIC VALVE REPLACEMENT IN LOW-RISK PATIENTS

Kolte D, Palacios IF

Approximately 12.4% of patients >75 years of age have aortic stenosis (AS) and 3.4% have severe AS. The prevalence of AS and its impact on public health and health care resources is expected to increase with the aging population. Since the first human percutaneous balloon-expandable transcatheter aortic valve implantation by Dr. Alain Cribier on April 16, 2002 in Rouen, France, this disruptive technology has evolved rapidly over the past two decades. Approximately 400,000 transcatheter aortic valve replacement (TAVR) procedures have been performed worldwide with an estimated growth of 40% per year, and the annual number of TAVRs have now surpassed the number of surgical aortic valve replacement (SAVR) procedures in some countries.

CONSENSO INTERSOCIETARIO ARGENTINO DE SÍNDROME DE CONGESTIÓN PELVIANA. PARTE 1

Eisele G et al., authors of the Intersocietary Argentine Pelvic Congestion Syndrome Consensus.

Chronic pelvic pain (CPP), which mainly affects women in reproductive age, shows a significant incidence rate in the general population, where pelvic congestion syndrome (PCS) is the second leading cause of CPP. Several medical societies have drafted documents to unify concepts seeking international consensus for the diagnosis and management of PCS. Based on the existing evidence and experience managing PCS, a group of Spanish-speaking experts on Vascular Surgery, Phlebology, Gynecology and Obstetrics, Diagnostic Imaging Modalities, Radiology, and Interventional Cardiology has drafted the first edition of the Argentine Pelvic Congestion Consensus. Using the Levels of Evidence and Recommendation established by the American College of Cardiology (ACC) and the American Heart Association (AHA) and accepted by the European Society of Cardiology (ESC), the validity of the diagnostic and treatment methodologies is established with proven utility in the lasting resolution of PCS cases.

ORIGINAL ARTICLE / ARTÍCULO ORIGINAL

SMOKING AS AN INDEPENDENT PREDICTOR OF RADIAL ARTERY SPASM

Mercado N et al.

Objective. To determine the independent influence of smoking on the appearance of radial artery spasm in cardiac catheterization studies and study the association of different conditions with the development of radial artery spasm. **Material and method.** Prospective, cross-sectional study. A 3-month follow-up of patients undergoing cardiac catheterization in 2 centers of the province of Córdoba, Argentina analyzed the influence of the procedural variables associated and clinical variables such as sex, age, and risk factors in the appearance of radial artery spasm (RAS). **Results.** Patients undergoing cardiac catheterizations via radial artery access between September and December 2019 in 2 centers in the city of Córdoba, Argentina were analyzed. A population of 347 patients was finally studied. A total of 131 were women (37.8%) and 216 were men (62.2%). The incidence rate of radial artery spasm (RAS) in the study sample was 14.7% (n=51). A significant association between smoking and this event was finally confirmed. In all cases, the incidence rate of RAS was significantly higher in smokers (17.6%) compared to non-smokers (6.5%). Smoking was a variable significantly associated with radial artery spasm (P=.020). Sex presented an OR associated with spasm (OR=8.2); the probability of wo-

men to present RAS would be 8 times greater compared to men. RAS occurred in almost a third of the women studied (29.0%) and in 6.0% of the males studied and was more commonly seen in smokers (33.3% of them suffered radial artery spasm). However, in non-smokers the incidence of spasm was also more common in women compared to men. Regarding procedural factors, it was observed that both the size of the catheter and the spasm kept a significant correlation ($P=.021$). Procedural time was significant ($P=.022$) with a higher risk of spasm associated with patients with procedural times > 20 min. ($OR=2.6$) and the type of study and occurrence of RAS ($P=.014$). The highest risk category was seen in the therapeutic study with an $OR=3.1$ (95%CI, 1.25-7.67).

Conclusions. The incidence rate of radial artery spasm was close to 15% in the study sample, which kept a positive correlation with female sex and smoking. Smoking was confirmed as an independent risk factor to predict the appearance of RAS in patients undergoing cardiac catheterization studies.

CASE REPORTS / CASOS CLÍNICOS

CORONARY PULMONARY FISTULA: PRESENTATION OF A RARE CASE WITH SUCCESSFUL ENDOVASCULAR RESOLUTION

Amicone S et al.

Introduction. 58-year-old female patient with dyspnea NY functional class III of 2 months of evolution. Echocardiogram showed no pathological findings. Echo stress with exercise showed inferior ischemia. Stratification was decided with coronary angiography in which a coronary pulmonary fistula was observed. The fistula was embolized with coils. After two months of follow-up, the patient presented clinical improvement and was asymptomatic.

Conclusion. Coronary pulmonary fistulas are a rare entity, present in 0.1% of coronary angiograms according to different series. A case with endovascular resolution is presented.

SUBCLAVIAN-CORONARY STEAL SYNDROME. CASE REPORT AND RESOLUTION BY ANGIOPLASTY

Cúneo T et al.

Introducción. El síndrome del robo subclavio-coronario es un cuadro poco frecuente. Se debe a una oclusión o estenosis severa de la arteria subclavia, anterior y proximal al origen de la arteria vertebral. Presenta buena respuesta al tratamiento percutáneo, ya sea con balón o implante de prótesis endovascular. El objetivo de este trabajo fue presentar un reporte de caso de síndrome de robo subclavio-coronario, su resolución percutánea, y una revisión bibliográfica.

Método. Reporte de caso. Conclusión. El síndrome de robo subclavio-coronario es una situación poco frecuente. Puede generar isquemia miocárdica y síntomas neurológicos. Se requiere de estudios preoperatorios completos para su prevención, y de una alta sospecha clínica para su diagnóstico. El tratamiento percutáneo precoz con stent lo permite resolver en forma satisfactoria.

SIX-YEAR ANGIOGRAPHIC FOLLOW-UP OF A CHRONIC TOTAL CORONARY OCCLUSION SUCCESSFULLY TREATED WITH BMS IMPLANTATION FOLLOWED BY ORAL RAPAMYCIN

Mieres J et al.

This is the case of a 76-month angiographic follow-up of a male patient with CTO (total chronic coronary occlusion) treated with a combined strategy of percutaneous coronary angioplasty (PCI) with multiple conventional stents (BMS) plus oral rapamycin (OR) for 13 days after the PCI. The patient had several risk factors for in-stent restenosis (ISR) including metabolic syndrome, a long segment of the CTO, small-diameter blood vessels, and BMS overlapping. The patient was treated with 3 BMSs making a total stent length of 64 mm with a minimal lumen diameter post-implantation of 1.82 mm and a reference diameter of 2.71 mm. The patient remained asymptomatic for 75 months. After this time, he showed CF dyspnea type III, which could be attributed to overweight. After several functional studies an angiographic study is decided that shows the patency of the stents in all their segments without ISR and a minimal lumen diameter of 1.61 mm, which is indicative of a late loss of 0.21mm. The vasoreactivity testing performed with adenosine looked normal showing a minimal lumen diameter of 2.24 mm after infusion. This is a brief review of this PCI strategy.

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CLOSURE OF A DIFFERENT AND VERY SPECIAL YEAR

Grinfeld D

Dear colleagues and members of the Argentine College of Interventional Cardioangiologists (CACI), I wish to take this opportunity, say "hi" to all of you, and brief you on everything that has been going on in our college during this very special year we've had. Despite the pandemic, we were able to hold our Congress remotely. After asking for different budgets we chose the company LANZILOTA to launch our Congress, which was very successful thanks to all your efforts. Regarding fees, CACI Fees Commission periodically updated the costs of our medical practice and the new Professional Commission was created. Due to the COVID-19 pandemic and the work and income decrease sustained by our members we decided not to make inflation-based adjustments in the fees and quotas of the college. In 2020 we held the V CACI Meeting for Medical Auditors and Sponsors that was coordinated by Dr. Jorge Leguizamón with the collaboration of medical auditors from different provinces of our country and discussed topics on myocardial infarction, calcified coronary artery disease, aortic and mitral valvular heart disease, and intracardiac imaging. We held the first CACI workshop with industry specialists on the reality of valvular implants in Argentina. The response from the audience was just great.

Percutaneous implantation of aortic valve at low or intermediate risk: when effectiveness trumps cost

Implante percutáneo de válvula aórtica en riesgo bajo o intermedio: cuando la efectividad se impone al costo

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Very few things in internal medicine and/or interventional cardiology have been more interesting to me when doing clinical research than analyzing the cost-effectiveness ratio of revascularization techniques or strategies and/or therapy.

Since the times of the randomized clinical trials between angioplasty vs coronary surgery (1) to the rational use of stents (2-3) until the option of using immunosuppressants to prevent restenosis (4-5), the cost-effectiveness ratio has always been studied from 2 different therapeutic positions.

We used to base these different positions (1-4) and we still do today (5) on a similar effectiveness hypothesis of the hard cardiovascular adverse events we wish to compare between both techniques like rate of death, infarction or stroke. In these circumstances both the costs and the cost-effectiveness of each strategy can be analyzed.

To this day, transcatheter aortic valve implantation (TAVI) was and still is the most important therapeutic achievement in our specialty.

First designed for patients contraindicated for surgical aortic valve replacement (SAVR) or with high risk of SAVR related death (6-7), believe it or not, these are the only indications accepted to this day by our country's social security system.

Over the last few years, the greater knowledge acquired in the implantation technique and the improvements made in the devices used that facilitate the use of percutaneous implants in over 90% of the cases via transfemoral access has brought TAVI indications to study in low and intermediate risk patients.

Therefore, 3 randomized clinical trials have been published to this day between TAVI and SAVR in these clinical circumstances (8-10) together with prospective registries with incredible results like the rates of mortality and incapacitating stroke at the 30-day follow-up in 0 out of 200 patients (11).

Two of these randomized clinical trials were conducted using self-expanding valves (8-9). The remaining one with balloon-expandable valve implantation (10).

The results from these 3 clinical trials have been exquisitely documented in this issue of RACI by the groups of experts from the Massachusetts General Hospital directed by Dr. Palacios, MD. There is no doubt that the reader will enjoy these results.

The results of the Partner 3, Evolut Low Risk, and NOTION show the advances made with this technique in low or intermediate risk patients.

It is obvious that, as it happens with all randomized clinical trials, these 3 trials had exclusion criteria that should be taken into consideration when indicating SAVR. These exclusion criteria included bicuspid aortic valves, large aortic annulus, impossibility of using femoral access, and young patients with high baseline risk for permanent pacemaker implantation, among other.

Still, when there is a patient eligible for both techniques who meets the inclusion criteria of these studies (9-10) we should not forget that the comparison between SAVR and TAVI brought the following results to light:

Fewer deaths with TAVI compared to SAVR.

Fewer or same incapacitating strokes with TAVI compared to SAVR.

More paravalvular regurgitation and leaks with TAVI compared to SAVR.

Higher rate of permanent pacemaker implantation in some TAVIs when the implants were placed at deep level and not on the aortic annulus.

Similar or less endocarditis compared to SAVR.

Fewer major bleeding or acute kidney injury in TAVI compared to SAVR.

Finally, the cost of TAVI was higher compared to SAVR.

The cost of the device used in TAVI is obviously a preoccupation in Argentina as well as in other regions. The COVID-19 pandemic has exposed the serious problems of healthcare systems even in the most developed countries today. The rate of permanent pacemaker implantation after TAVI is a complication we should seriously be looking into in patients < 70 years and low or intermediate risk. As far as I am concerned, the implantation technique depends largely on the type of valve used whether a balloon-expandable or a self-expanding heart valve. As a matter of fact, in our own experience with the Portico self-expanding valve (Abbott/Saint Jude) performing more supra-valvular implantations, the rate of permanent pacemaker implantation in 37 patients was 10.8%.

We have to accept the fact that our country's social security system has been experiencing serious difficulties making certain devices available due to their disproportionate costs. Also, with weird clinical indications, sometimes. However, this should not worry the patients or the interventional cardiology community in Argentina. It is a matter of discussion for the public and private healthcare systems under the control of the State. They need to solve these problems and make this technique available for all the patients who may need it.

When mortality rate is at stake, the cost-effectiveness ratio becomes insignificant. As we have been seeing in the recent analyses (9-10), the rates of overall mortality, cardiovascular mortality, incapacitating strokes, and acute kidney injury were all higher with SAVR even in the presence of a surgical mortality rate at the 30-day follow-up <2%.

Public and private healthcare systems together with the Argentinian Ministry of Public Health now have the ball in their court. They need to guarantee that low and intermediate risk patients with aortic valve stenosis will have access to the most suitable therapy in each particular case easily and with lower morbidity and mortality rates.

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REFERENCES

1. Rodríguez A, Bernardi V, Navia J, et al. Argentine Randomized Study: Coronary Angioplasty with Stenting versus Coronary Bypass Surgery in patients with Multiple-Vessel Disease (ERACI II): 30-day and one-year follow-up results. ERACI II Investigators. *J Am Coll Cardiol* 2001;37:51-8.
2. Rodríguez AE, Santaera O, Larribau M, et al. Coronary stenting decreases restenosis in lesions with early loss in luminal diameter 24 hours after successful PTCA. *Circulation* 1995;91:1397-402.
3. Rodríguez A, Ambrose J. Do we require a cure for "Stentmania." *J Am Coll Cardiol* 1996;28:4.
4. Rodríguez AE, Palacios I, Rodríguez-Granillo AM, et al. Comparison of Cost-Effectiveness of Oral Rapamycin Plus Bare-Metal Stents Versus First Generation of Drug-Eluting Stents (from the Randomized Oral Rapamycin in Argentina [ORAR] 3 Trial). *Am J Cardiol* 2014;113:815e-21.
5. Correa-Sadouet C, Rodríguez-Granillo AM, Gallardo C, et al. Randomized comparison between bare-metal stent plus colchicine versus drug-eluting stent alone in prevention of clinical adverse events after percutaneous coronary intervention. Trial Protocol, *Future Cardiol* 2020 Nov 11. doi: 10.2217/fca-2020-0161. Online ahead of print.
6. Smith CR, Leon MB, Mack MJ, et al. PARTNER Trial Investigators. Transcatheter versus surgical aortic-valve replacement in high-risk patients. *N Engl J Med* 2011; 364:2187-98.
7. Adams DH, Popma JJ, Reardon MJ, et al. U.S. CoreValve Clinical Investigators. Transcatheter aortic-valve replacement with a self-expanding prosthesis. *N Engl J Med* 2014;370:1790-8.
8. Thyregod HGH, Steinbrüchel DA, Ihlemann N, et al. Transcatheter Versus Surgical Aortic Valve Replacement in Patients With Severe Aortic Valve Stenosis 1-Year Results From the All-Comers NOTION Randomized Clinical Trial. *J Am Coll Cardiol* 2015;65:2184-94.
9. Popma JJ, Deeb M, Yakubo S, et al. Transcatheter Aortic-Valve Replacement with a Self-Expanding Valve in Low-Risk Patients. *N Engl J Med* 2019;380(18):1706-15.
10. Mack MJ, Leon MB, Thourani VH, et al. Transcatheter Aortic-Valve Replacement with a Balloon-Expandable Valve in Low-Risk Patients. *N Engl J Med* 2019;380(18):1695-705.
11. Waksman R, Corso PJ, Torguson R, et al. TAVR in Low-Risk Patients: 1-Year Results From the LRT Trial. *JACC Cardiovasc Interv* 2019;12(10):901-7.

Transcatheter aortic valve replacement in low-risk patients

Reemplazo percutáneo de válvula aórtica en pacientes de bajo riesgo

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Palabras clave: implante percutáneo de válvula aórtica, TAVI, estenosis valvular aórtica, remplazo quirúrgico de válvula aórtica.

Keywords: TAVI, aortic valve replacement, aortic valve stenosis, surgical aortic valve replacement.

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Approximately 12.4% of patients >75 years of age have aortic stenosis (AS) and 3.4% have severe AS.¹ The prevalence of AS and its impact on public health and health care resources is expected to increase with the aging population.² Since the first human percutaneous balloon-expandable transcatheter aortic valve implantation by Dr. Alain Cribier on April 16, 2002 in Rouen, France, this disruptive technology has evolved rapidly over the past two decades.³ Approximately 400,000 transcatheter aortic valve replacement (TAVR) procedures have been performed worldwide with an estimated growth of 40% per year, and the annual number of TAVRs have now surpassed the number of surgical aortic valve replacement (SAVR) procedures in some countries.^{4,5} The role of TAVR as a safe and effective treatment option in patients with symptomatic severe aortic stenosis who are at prohibitive, high, or intermediate risk for surgery is well established. Recently, based on results of the PARTNER 3 and Evolut Low-Risk trials, the United States Food and Drug Administration expanded indication for TAVR to patients at low risk for SAVR.^{6,7} This article will summarize the data on TAVR in low-risk patients, discuss considerations when choosing between TAVR vs. SAVR for low-risk patients, and highlight areas for future research.

TAVR VS. SAVR IN LOW-RISK PATIENTS

Prospective studies of TAVR in low-risk patients are summarized in **Table 1**.

Nordic Aortic Valve Intervention Trial (NOTION)

NOTION was an investigator-initiated, multi-center, non-blinded, superiority trial which randomized all-comer patients ≥70 years with isolated severe aortic valve stenosis to SAVR or TAVR in Denmark and Sweden.⁸ The trial included

280 patients, 81.1% of whom were low-risk (Society of Thoracic Surgeons Predicted Risk Of Mortality [STS-PROM] <4%). The primary outcome was the composite rate of death from any cause, stroke, or myocardial infarction (MI) at 1 year. There was no significant difference in the rate of the primary endpoint between TAVR vs. SAVR at 1 year (13.1% vs. 16.3%, $p=0.43$) and 5 years (38.0% vs. 36.3%, $p=0.86$).⁸ Compared with patients who underwent SAVR, those who underwent TAVR had significantly higher rates of permanent pacemaker (PPM) implantation and ≥ moderate total aortic regurgitation, and lower rates of major or life-threatening bleeding, acute kidney injury (AKI) stage 2 or 3, and new-onset or worsening atrial fibrillation (AF) at 30 days.⁸

Low Risk TAVR (LRT) Study

The LRT was an investigator-initiated, prospective, multicenter feasibility trial to test the safety of transfemoral TAVR in low-risk patients with symptomatic severe AS.¹⁰ The study enrolled 200 low-risk (STS-PROM ≤3%) patients at 11 centers who underwent transfemoral TAVR and were compared to a historical cohort of 719 patients who underwent isolated SAVR at the same institutions. At 30 days, there was zero all-cause mortality in the TAVR group vs. 1.7% in the SAVR group ($p=0.59$).¹⁰ PPM implantation rates were similar between TAVR and SAVR (5.0% vs. 4.5%, $p=0.74$). At 1-year follow-up, mortality was 3.0%, stroke rate was 2.1%, and PPM implantation rate was 7.3% in the TAVR group.¹¹

Placement of Aortic Transcatheter Valves (PARTNER) 3 Trial

The PARTNER 3 trial was a multicenter, randomized trial comparing transfemoral TAVR using the third-generation balloon-expandable SAPIEN 3 (Edwards Lifesciences, Irvine, CA) valve system with SAVR in low-risk patients (STS-PROM <4%).⁶ The primary endpoint was a composite of death, stroke, or rehospitalization at 1 year. Both noninferiority testing (with a prespecified margin of 6%) and superiority testing were performed in the as-treated population ($n=950$). At 1 year, the rate of the primary endpoint was significantly lower in the TAVR group than in the SAVR group (8.5% vs. 15.1%; absolute difference, -6.6%; 95% confidence interval [CI]: -10.8 to -2.5; $p<0.001$ for noninferiority; hazard ratio [HR], 0.54; 95%CI: 0.37 to 0.79; $p=0.001$ for superiority).⁸ Results

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TABLE 1. Prospective studies of TAVR in low-risk patients.

	NOTION ^{8,9}	Low-Risk TAVR Study ^{10,11}	PARTNER 3 ⁶	Evolut Low Risk ⁷
Year	2015	2018	2019	2019
Study Design	RCT, superiority	Prospective, single-arm	RCT, non-inferiority and superiority	RCT, non-inferiority
N	280	200	950	1,403
Key inclusion criteria	≥70 years of age; severe AS; heart team evaluation; symptomatic; asymptomatic with LVPWT ≥17 mm, decreasing LVEF, or new onset atrial fibrillation; >1 year survival.	Severe AS, symptomatic (NYHA functional class ≥2, angina pectoris, or syncope); STS ≤3%; eligible for transfemoral access; candidate for SAVR if offered; elective procedure; estimated life-expectancy >24 months.	Severe calcific AS and NYHA functional class ≥2, exercise tolerance test demonstrating a limited exercise capacity, abnormal BP response, or arrhythmia, or asymptomatic with LVEF <50%; STS <4% and low risk of operative mortality per heart team; eligible for transfemoral access.	Severe AS; symptomatic or asymptomatic with very severe AS, exercise tolerance test demonstrating a limited exercise capacity, abnormal BP response, or arrhythmia, or LVEF <50%; STS <3% and low risk of operative mortality per heart team.
Key exclusion criteria	Concomitant severe valve disease; CAD requiring intervention; prior cardiac surgery; MI or stroke within 30 days; ESRD on dialysis; pulmonary failure with FEV1 or diffusion capacity <40% of expected.	Bicuspid aortic valve; concomitant disease of another heart valve or aorta that requires intervention; ESRD on dialysis or CrCl <20 cc/min; LVEF <20%; recent (<6 months) stroke/TIA; recent (<30 days) AMI; symptomatic carotid/vertebral artery disease; severe unrevascularized CAD; recent (<30 days) or ongoing bleeding; uncontrolled atrial fibrillation; severe COPD (FEV1 <750 cc); liver failure with Child's class C or D; ongoing sepsis or infective endocarditis; preprocedural shock, inotropes, mechanical assist device, or cardiac arrest.	Unicuspid, bicuspid, or non-calcified aortic valve; severe AR/MR (>3+), ≥moderate MS; pre-existing bioprosthetic or mechanical valve in any position; complex CAD; MI within 30 days before randomization; stroke/TIA within 90 days of randomization; active bacterial endocarditis within 180 days of randomization; LVEF <30%; eGFR <30 or dialysis; severe lung disease (FEV1 <50% predicted) or home oxygen; severe pulmonary hypertension; cirrhosis or active liver disease; clinical frailty; estimated life-expectancy <24 months.	Bicuspid aortic valve; severe MR/TR; moderate or severe MS; pre-existing prosthetic heart valve in any position; multivessel CAD with SYNTAX score >22 and/or UPLM; MI ≤30 days prior to trial procedure; percutaneous coronary/peripheral intervention with BSM within 30 days or DES within 180 days prior to randomization; recent (<2 months) stroke/TIA; severe dementia; estimated life-expectancy <24 months.
TAVR Valve Type	CoreValve (Medtronic Inc., Minneapolis, MN)	Sapien 3 (Edwards Lifesciences, Irvine, CA) or CoreValve, Evolut R, or Evolut PRO (Medtronic Inc., Minneapolis, MN)	Sapien 3 (Edwards Lifesciences, Irvine, CA)	CoreValve, Evolut R, or Evolut PRO (Medtronic Inc., Minneapolis, MN)
Primary Endpoint	Composite of all-cause death, stroke, or MI at 1 year.	All-cause death at 30 days.	Composite of all-cause death, stroke, or rehospitalization at 1 year.	Composite of all-cause death or disabling stroke at 24 months.

Adapted and modified from Kolte et al. *J Am Coll Cardiol*.2019;74:1532-1540 (reference 13).

were consistent at 2-year follow-up (11.5% vs. 17.4%; absolute difference, −5.9%; HR, 0.63; 95%CI: 0.45 to 0.88; $p=0.007$).¹² TAVR resulted in a lower rate of stroke than SAVR at 30 days (0.6% vs. 2.4%, $p=0.02$) and 1 year (1.2% vs. 3.3%; $p=0.03$); however, this difference narrowed and was no longer statistically significant at 2 years (2.4% vs. 3.6%; $p=0.28$).^{6,12} There were no significant differences in PPM implantation rates between TAVR vs. SAVR at 1- and 2-year follow-up. At 2 years, Valve Academic Research Consortium (VARC)-2 defined valve thrombosis rates were higher in the TAVR groups compared with the SAVR group (2.6% vs. 0.7%, $p=0.02$).¹²

Evolut Low Risk Trial

The Evolut Low Risk Trial was a multinational, randomized, noninferiority trial comparing the safety and efficacy of TAVR with one of the three self-expanding, supraannular bioprostheses (CoreValve, Evolut R, or Evolut PRO; Medtronic, Minneapolis, MN) with that of SAVR in low-risk patients (STS-PROM ≤3%).⁷ The primary endpoint was a composite of death from any cause or disabling stroke at 24 months. The trial used Bayesian adaptive statistical methods with non informative prior distributions to assess the primary endpoint when 850 patients had reached 12-month follow-up. The prespecified noninferiority margin for the primary endpoint was 6%. The 24-month

estimated incidence of the primary endpoint was 5.3% in the TAVR group and 6.7% in the SAVR group (difference, −1.4%; 95% Bayesian credible interval for difference, −4.9 to 2.1; posterior probability of noninferiority >0.999).⁷ At 30 days, patients who had undergone TAVR, as compared with SAVR, had lower rates of disabling stroke (0.5% vs. 1.7%), bleeding complications (2.4% vs. 7.5%), AKI stage 2 or 3 (0.9% vs. 2.8%), and AF (7.7% vs. 35.4%), and higher rates of ≥ moderate aortic regurgitation (3.5% vs. 0.5%) and PPM implantation (17.4% vs. 6.1%).⁷

Meta-Analysis of TAVR vs. SAVR in Low-Risk Patients

In a meta-analysis that included 3 randomized controlled trials (NOTION, PARTNER 3, and Evolut Low Risk) and 1 *post hoc* analysis of the Surgical Replacement and Transcatheter Aortic Valve Implantation (SURTAVI) trial, we found that TAVR was associated with significantly lower risk of all-cause death (2.1% vs. 3.5%; risk ratio [RR], 0.61; 95% CI, 0.39 to 0.96; $p=0.03$; $I^2=0\%$) and cardiovascular death (1.6% vs. 2.9%; RR, 0.55; 95% CI, 0.33 to 0.90; $p=0.02$; $I^2=0\%$) at 1 year (**Figure 1**).¹³ Rates of new or worsening AF, life-threatening or disabling bleeding, and AKI stage 2 or 3 were lower, whereas those of PPM implantation and ≥moderate paravalvular leak were higher after TAVR vs. SAVR.¹³

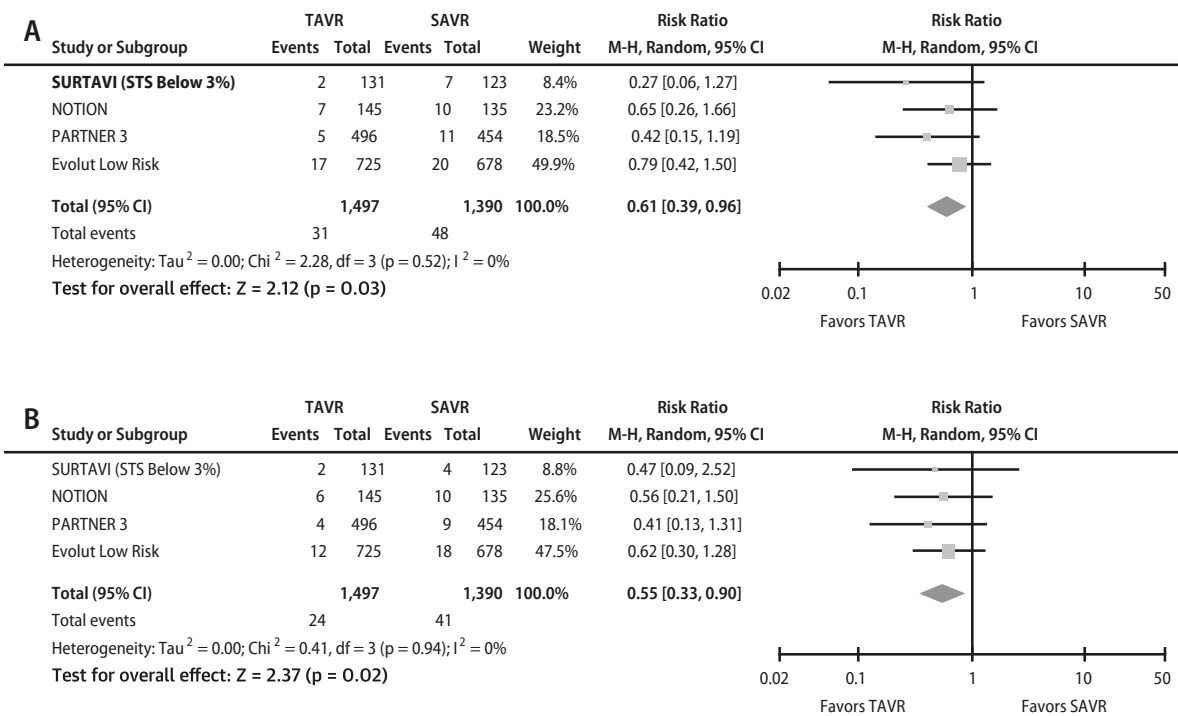


Figure 1. All-Cause and Cardiovascular Death at 1 Year After TAVR vs. SAVR in Low-Risk Patients. All-cause death (A) and cardiovascular death (B) at 1 year after TAVR versus SAVR in low-risk patients are shown. In low-risk patients with severe aortic stenosis, TAVR was associated with significantly lower risk of all-cause death (2.1% vs. 3.5%; RR, 0.61; 95% CI, 0.39 to 0.96; p=0.03; I²=0%) and cardiovascular death (1.6% vs. 2.9%; RR, 0.55; 95% CI, 0.33 to 0.90; p=0.02; I²=0%) at 1 year as compared with SAVR. Adapted from Kolte et al. J Am Coll Cardiol. 2019;74:1532-1540 (reference 13). CI = confidence interval; M-H = Mantel-Haenszel; NOTION = Nordic Aortic Valve Intervention Trial; PARTNER = Placement of Aortic Transcatheter Valves; RR = risk ratio; SAVR = surgical aortic valve replacement; STS = Society of Thoracic Surgeons; SURTAVI = Surgical Replacement and Transcatheter Aortic Valve Implantation; TAVR = transcatheter aortic valve replacement.

CONSIDERATIONS WHEN CHOOSING BETWEEN TAVR VS. SAVR IN LOW-RISK PATIENTS

The choice between TAVR vs. SAVR for patients with symptomatic severe AS, particularly low-risk patients, should involve a Heart Team and a shared-decision making approach to ensure incorporation of patient goals and preferences into the final decision making.¹⁴ It is important to note that the average age of patients in the pivotal low-risk trials was ~74 years, and patients not suitable for transfemoral access, with bicuspid aortic valves, prior bioprosthetic or mechanical valves in any position, severe aortic or mitral regurgitation, ≥ moderate mitral stenosis, low coronary height, severe aortic valve calcification, left ventricular outflow tract (LVOT) calcification were excluded from these trials (Table 1).¹³ Similarly, patients with multivessel coronary artery disease with SYNTAX score >22 were also excluded. Patients who do not fulfill the strict inclusion and exclusion criteria for the trials may potentially be better served with SAVR.¹⁵ Another important consideration is valve durability and the possible need for a second AVR in the future. Although studies have shown that >90% of patients remain free of structural valve degeneration between 5 and 10 years post-TAVR, longer-term data are not yet available.¹⁶ Similarly, while outcomes of valve-in-valve TAVR in patients with failed SAVR are comparable to native valve TAVR, data on TAVR-in-TAVR (or redo TAVR) are limited.^{17,18} These aspects should be discussed with patients/families as part of the shared-decision making process when choosing between TAVR vs. SAVR in low-risk patients.¹⁴ Severe AS due to bicuspid anatomy is now encountered more

frequently with the expansion of TAVR to younger low-risk patients. The Evolut Low Risk Bicuspid Study was a multicenter, prospective, single-arm study to assess the safety and efficacy of TAVR with one of the two self-expanding, supraannular bioprostheses (Evolut R or Evolut PRO; Medtronic, Minneapolis, MN) in low-risk patients with bicuspid aortic stenosis.¹⁹ Patients <60 years, SYNTAX score >22, ascending aortic diameter >4.5 cm, aortopathy requiring surgical intervention, prohibitive LVOT calcification, and anatomic dimensions outside the recommended range were excluded. The primary safety endpoint of all-cause death or disabling stroke at 30-days occurred in 1.3% of patients.¹⁹ The primary efficacy endpoint of device success (defined as absence of procedural mortality, correct position of 1 valve in the proper anatomical location, and absence of >mild aortic regurgitation) occurred in 95.3% of patients.¹⁹ Although these short-term results are promising, longer-term data including randomized trials of TAVR vs. SAVR in low-risk patients with bicuspid aortic stenosis are needed.

CONCLUSION

TAVR has rapidly evolved as a safe and effective treatment option for patients with symptomatic severe AS across the entire spectrum of surgical risk. The choice between TAVR vs. SAVR for patients with symptomatic severe AS, particularly low-risk patients, should involve a Heart Team and a shared-decision making approach to ensure incorporation of patient goals and preferences into the final decision making. Patients who do not fulfill the strict inclusion and exclusion criteria for the pivotal low-risk trials may potentially be better served with SAVR. Further data on long-term durability of TAVR bioprostheses, redo TAVR, and TAVR in bicuspid anatomy are needed.

REFERENCES

- Osnabrugge RL, Mylotte D, Head SJ, Van Mieghem NM, Nkomo VT, LeRou CM, Bogers AJ, Piazza N and Kappetein AP. Aortic stenosis in the elderly: disease prevalence and number of candidates for transcatheter aortic valve replacement: a meta-analysis and modeling study. *J Am Coll Cardiol.* 2013;62:1002-12. doi: 10.1016/j.jacc.2013.05.015.
- Moore M, Chen J, Mallow PJ and Rizzo JA. The direct health-care burden of valvular heart disease: evidence from US national survey data. *Clinicoecon Outcomes Res.* 2016;8:613-627. doi: 10.2147/CEOR.S112691.
- Cribier AG. The Odyssey of TAVR from concept to clinical reality. *Tex Heart Inst J.* 2014;41:125-30. doi: 10.14503/THIJ-14-4137.
- Kourkovelis P, Spargias K and Hahalis G. TAVR in 2017-What we know? What to expect? *J Geriatr Cardiol.* 2018;15:55-60. doi: 10.11909/j.issn.1671-5411.2018.01.005.
- Kundi H, Strom JB, Valsdottir LR, Elmariah S, Popma JJ, Shen C and Yeh RW. Trends in Isolated Surgical Aortic Valve Replacement According to Hospital-Based Transcatheter Aortic Valve Replacement Volumes. *JACC Cardiovasc Interv.* 2018;11:2148-2156. doi: 10.1016/j.jcin.2018.07.002.
- Mack MJ, Leon MB, Thourani VH, Makkar R, Kodali SK, Russo M, Kapadia SR, Malaisrie SC, Cohen DJ, Pibarot P, et al. Transcatheter Aortic-Valve Replacement with a Balloon-Expandable Valve in Low-Risk Patients. *N Engl J Med.* 2019;380:1695-1705. doi: 10.1056/NEJMoa1814052.
- Popma JJ, Deeb GM, Yakubov SJ, Mumtaz M, Gada H, O'Hair D, Bajwa T, Heiser JC, Merhi W, Kleiman NS, et al. Transcatheter Aortic-Valve Replacement with a Self-Expanding Valve in Low-Risk Patients. *N Engl J Med.* 2019;380:1706-1715. doi: 10.1056/NEJMoa1816885.
- Thyregod HG, Steinbruchel DA, Ihlemann N, Nissen H, Kjeldsen BJ, Petersen P, Chang Y, Franzen OW, Engstrom T, Clemmensen P, et al. Transcatheter Versus Surgical Aortic Valve Replacement in Patients With Severe Aortic Valve Stenosis: 1-Year Results From the All-Comers NOTION Randomized Clinical Trial. *J Am Coll Cardiol.* 2015;65:2184-94. doi: 10.1016/j.jacc.2015.03.014.
- Thyregod HGH, Ihlemann N, Jorgensen TH, Nissen H, Kjeldsen BJ, Petersen P, Chang Y, Franzen OW, Engstrom T, Clemmensen P, et al. Five-Year Clinical and Echocardiographic Outcomes from the Nordic Aortic Valve Intervention (NOTION) Randomized Clinical Trial in Lower Surgical Risk Patients. *Circulation.* 2019. doi: 10.1161/CIRCULATIONAHA.118.036606.
- Waksman R, Rogers T, Torguson R, Gordon P, Ehsan A, Wilson SR, Goncalves J, Levitt R, Hahn C, Parikh P, et al. Transcatheter Aortic Valve Replacement in Low-Risk Patients With Symptomatic Severe Aortic Stenosis. *J Am Coll Cardiol.* 2018;72:2095-2105. doi: 10.1016/j.jacc.2018.08.1033.
- Waksman R, Corso PJ, Torguson R, Gordon P, Ehsan A, Wilson SR, Goncalves J, Levitt R, Hahn C, Parikh P, et al. TAVR in Low-Risk Patients: 1-Year Results From the LRT Trial. *JACC Cardiovasc Interv.* 2019;12:901-907. doi: 10.1016/j.jcin.2019.03.002.
- Mack M and Leon MB. Two-Year Clinical and Echocardiographic Outcomes From the PARTNER 3 Low-Risk Randomized Trial. *ACC* 2020. 2020.
- Kolte D, Vlahakes GJ, Palacios IF, Sakhuja R, Passeri JJ, Inglessis I and Elmariah S. Transcatheter Versus Surgical Aortic Valve Replacement in Low-Risk Patients. *J Am Coll Cardiol.* 2019;74:1532-1540. doi: 10.1016/j.jacc.2019.06.076.
- Coylewright M, Forrest JK, McCabe JM and Nazif TM. TAVR in Low-Risk Patients: FDA Approval, the New NCD, and Shared Decision-Making. *J Am Coll Cardiol.* 2020;75:1208-1211. doi: 10.1016/j.jacc.2019.12.057.
- Sondergaard L and Costa G. Transcatheter Aortic Valve Replacement in Patients With Aortic Stenosis and Low Surgical Risk. *J Am Coll Cardiol.* 2019;74:1541-1542. doi: 10.1016/j.jacc.2019.07.067.
- Blackman DJ, Saraf S, MacCarthy PA, Myat A, Anderson SG, Malkin CJ, Cunningham MS, Somers K, Brennan P, Manoharan G, et al. Long-Term Durability of Transcatheter Aortic Valve Prostheses. *J Am Coll Cardiol.* 2019;73:537-545. doi: 10.1016/j.jacc.2018.10.078.
- Tuzcu EM, Kapadia SR, Vemulapalli S, Carroll JD, Holmes DR, Jr., Mack MJ, Thourani VH, Grover FL, Brennan JM, Suri RM, et al. Transcatheter Aortic Valve Replacement of Failed Surgically Implanted Bioprostheses: The STS/ACC Registry. *J Am Coll Cardiol.* 2018;72:370-382. doi: 10.1016/j.jacc.2018.04.074.
- Landes U, Webb JG, De Backer O, Sondergaard L, Abdel-Wahab M, Crusius L, Kim WK, Hamm C, Buzzatti N, Montorfano M, et al. Repeat Transcatheter Aortic Valve Replacement for Transcatheter Prosthesis Dysfunction. *J Am Coll Cardiol.* 2020;75:1882-1893. doi: 10.1016/j.jacc.2020.02.051.
- Ramlawi B. Primary Results From the Evolut Low Risk Bicuspid Study. *ACC* 2020. 2020.

Intersocietary Argentine Pelvic Congestion Syndrome Consensus. Part 1

Consenso Intersocietario Argentino de Síndrome de Congestión Pelviana. Parte 1

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1. METHODOLOGY USED IN THE CONSENSUS DOCUMENT

1.1 Introduction

Chronic pelvic pain (CPP), which mainly affects women in reproductive age, shows a significant incidence rate in the general population, where pelvic congestion syndrome (PCS) is the second leading cause of CPP.

Several medical societies have drafted documents to unify concepts seeking international consensus for the diagnosis and management of PCS.

Based on the existing evidence and experience managing PCS, a group of Spanish-speaking experts on Vascular Surgery, Phlebology, Gynecology and Obstetrics, Diagnostic Imaging Modalities, Radiology, and Interventional Cardiology has drafted the first edition of the Argentine Pelvic Congestion Consensus.

Using the Levels of Evidence and Recommendation established by the American College of Cardiology (ACC)

TABLE 1. Levels of Evidence and Classes of Recommendation

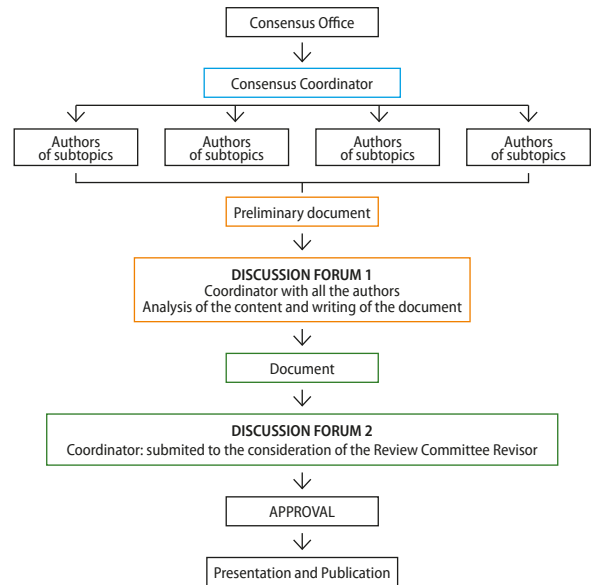
• Level of Evidence A (high): when data come from multiple randomized clinical trials or meta-analyses.
• Level of Evidence B (intermediate): when data come from a single randomized clinical trial or non-randomized studies.
• Level of Evidence C (low): where there is consensus only in the experts' opinion, case data or method of standard care.
• Class I Recommendation: disorders in which there is evidence and/or general consensus on the fact that the procedure/treatment is beneficial, useful, and effective. IT IS RECOMMENDED or IT IS INDICATED.
• Class II Recommendation: disorders where there is contradictory evidence and/or triggers controversial opinions on the utility/effectiveness of a procedure/treatment.
o Class II a: the burden of the evidence/opinion is favorable to the utility and effectiveness of a treatment or procedure. IT SHOULD BE TAKEN INTO CONSIDERATION.
o Class II b: utility/effectiveness is not well established by the evidence/opinion. SE PUEDE RECOMENDAR.
• Class III Recommendation: disorders where there is evidence and/or general agreement on the fact that the procedure/treatment is not useful or effective. As a matter of fact, it can even be harmful in some cases. IT IS NOT RECOMMENDED.

and the American Heart Association (AHA) and accepted by the European Society of Cardiology (ESC), the validity of the diagnostic and treatment methodologies is established with proven utility in the lasting resolution of PCS cases (**Table 1**).

According to this classification, the strength of the evidence and the degree of recommendation associated with a specific diagnosis or treatment option are based on the data available (https://www.wikidoc.org/index.php/ACC_AHA_guidelines_classification_scheme).

1.2 Methodology

The process of elaborating this consensus document was carried out following the algorithm of the Consensus area of the Argentine College of Interventional Cardioangiologist (CACI). Once the topic of discussion is determined, the consensus coordinators are summoned; they are responsible for the supervision, distribution of the project, and coordination of the discussion during the stages of analysis of the document at stake. The consensus office together with the coordinators invite experts that will participate as authors, who will be carrying out a thorough, systematic search of the literature available, conduct the critical analysis, and write the draft or preliminary document. Once the preliminary document is completed, it is posted on the CACI website long enough for it to be analyzed by all the authors and coordinators involved. To this end, a tool called FORO has been created by CACI that grants access limited only to participant members. It allows the document to be viewed and triggers participation in the discussion via comments that will appear chronologically and will be supervised by the coordinators. This tool increases author participation and saves time. Once this stage is completed, the coordinators collect all the opinions and suggestions to draft the final document with the same writing style. Afterwards, the final document should be submitted for external review and to this end, it is once again posted on the FORO (replacing the previous version) and access is granted to those who will act as judges for final approval purposes; these people will make up the Review Committee. We should mention that at this stage it is also possible to make corrections

**Diagram 1.** Algorithm used to write the consensus document.

as long as an agreement is reached between the authors and the review committee. Its members (coordinators, authors, and reviewers) are responsible for ensuring the rigor and quality of the document generated. Once the document has been approved, the consensus is presented in scientific sessions at different CACI societies and is published in scientific journals.

2. ABBREVIATIONS

- AMC: aortomesenteric clamp
- BMI: body mass index
- CAT scan: computerized axial tomography scan
- CDE color-coded Doppler echocardiography
- CHIVA: outpatient conservative haemodynamic correction of vein incompetence
- CPP: chronic pelvic pain
- DVT: deep venous thrombosis
- GIDP: gonadal and iliac dynamic phlebography
- IVC: inferior vena cava
- IVUS: intravascular ultrasound
- LL: lower limbs
- LRV: left renal vein
- MPA: medroxyprogesterone acetate
- MRI: magnetic resonance imaging
- MTS: May-Thurner syndrome
- NCS: Nutcracker syndrome
- PCS: pelvic congestion syndrome
- PFL pelvic floor leak
- PTA: percutaneous transluminal coronary angioplasty
- PTE: pulmonary thromboembolism
- PV: peak velocity
- PVI: pelvic vein incompetence
- QOL: quality of life
- SMA: superior mesenteric artery
- SPJ: saphenofemoral junction
- TE: transcatheter embolization
- TV: transvaginal
- TVCDE: Transvaginal color-coded Doppler echocardiography
- VAS: visual analogue scale

3. SUMMARY OF CONSENSUS

3.1 Generalities

Overview

The first description of ovarian varicose veins was made by Richet back in 1857. In 1949, Taylor, who associated anatomical changes with the symptoms of ovarian vein incompetence, proposed the concept of PCS suggesting ovarian vein ligation as the treatment of choice.

At the beginning, the etiology of pelvic venous incompetence (PVI) as the main pathophysiology of PCS was unknown and led to the psychological-psychiatric theory as an explanation of the symptoms of patients with pelvic pain disorders without an apparent cause.

Back in the 1980s, Lechter defined female gonadal vein incompetence as the causative of lower limb (LL), pelvic, and vulvar varicose veins. At that time, Beard proposed hysterectomy and bilateral oophorectomy as the treatment of PCS. In 1993, Edwards treated utero-ovarian varicose veins through transcatheter embolization (TE) for the first time ever, and in 2003, Chung proved the significant benefits of this therapy when compared to surgery and hormone therapy.

Epidemiology

PCS is, after endometriosis, the second leading cause of CPP in 30% to 40% of the cases. PVI is the etiological element that characterizes PCS. Costs derived from the management of CPP are high (over 880 million dollars each year in medical appointments and 2 billion dollars each year in overall costs in the United States). Although numerous medical journals find a correlation among PVI, PCS, and LL vein incompetence, no articles have been published of good methodological quality confirming it.

Definition

PCS is defined by the presence of 2 or more of the following conditions: 1) utero-ovarian varicose veins with or without gonadal vein incompetence, 2) congestive pelvic pain, 3) hypogastric vein incompetence, 4) pelvic floor reflux, typical or atypical LL varicose veins of pelvic origin. Although the term PCS cannot cover all forms of presentation and causes, it has been agreed to be used because it appears in the International Classification of Diseases and the VEIN TERM Transatlantic Interdisciplinary Consensus Document.

3.2 Pathophysiology

The origin of PCS is multifactorial with valvular insufficiency, venous obstruction, and hormonal changes as the most common causes. The cause of pain in PCS is distension due to venous stasis, which stimulates the release of local inflammatory and pain mediators.

Pathophysiologically speaking, the causes of PCS can be categorized into:

Primary, due to ovarian venous valve agenesis or incompetence; these are the ones most commonly associated, or not, with LL vein incompetence and the ones that best respond to TE procedures.

Secondary, due to the occlusion of iliac veins (May-Thurner syndrome [MTS]), inferior vena cava (IVC) or left renal vein (LVR) known as the *nutcracker* syndrome (NCS). The typical symptoms of PCS are associated with the venous obstruction

area, and the ideal endovascular resolution combines percutaneous transluminal coronary angioplasty (PTA) and TE. CPP-related *nonvenous* PCS are mainly due to non-vascular pelvic causes, basically correspond to endometriosis and other pelvic causes. PVI, though present, is not the main cause of the clinical signs. In these cases, therapies target the causative lesions detected.

3.3 Clinical signs

PCS has 4 clinical presentations:

1. Typical pelvic clinical signs compromising the uterus, ovaries, bladder and/or rectum.
2. Typical LL, vulvar, and gluteal (saphenous) or atypical (non-saphenous) varicose veins.
3. Edema and LL pain due to venous obstruction.
4. Renal clinical signs with pain and hematuria.

Pelvic damage often occurs with pelvic pain and LL varicose veins.

The dull, deep, prolonged, non-cyclical pain is exacerbated by movements and postures that increase abdominal pressure. It is a unilateral or bilateral, asymmetric and, chronic pain that lasts over 6 months. Associated with dyspareunia, dysmenorrhea and post-coital pain, it can cause nausea, distension or abdominal cramps, rectal discomfort, dysuria, and greater urinary frequency. It is often associated with neurological and psychosomatic symptoms like reactive depression.

The clinical signs of PVI are tenderness to palpation at ovarian, uterine, and cervical sites, as well as utero-ovarian, vulvar, gluteal, atypical of thigh and leg varicose veins associated with pelvic floor leak (PFL). It is often associated with symptomatic hemorrhoids. Varicose veins are predominant in the left LL for 2 reasons: gonadal and utero-ovarian PVIs are more significant on the left side of the body as well as PFL sites; also, in MTS cases, the left side is the common side where this condition develops. Clinical detection of the pelvic origin of LL varicose veins is not always evident and it requires performing a color-coded Doppler echocardiography (CDE); it should be suspected in the presence of post-saphenectomy relapses.

The predisposing factors of PCS are multiple pregnancies, sedentary lifestyle, increased estrogenic activity and low body mass index.

The clinical signs associated with venous obstructions (MTS, NCS, IVC occlusion, etc.) only occur when they are the causes of PCS.

3.4 Differential diagnosis

Both questioning and physical examination should cover the different possible causes of PCS. Suspected conditions will justify performing early clinical evaluation that will eventually lead to ordering additional laboratory tests, invasive imaging modalities or not selected based on their effectiveness, invasiveness, and availability.

In this diagnostic stage, 2 etiological groups should be defined:

1. Patients with typical (primary or obstructive) PCS who will benefit from TE and/or PTA therapies. Medical (and rarely surgical therapies) can complement these therapies. This consensus document focuses on the management of these patients.
2. Non-venous PCS with clinical signs and CPP and PCS combined additional studies should be especially con-

sidered by the heart team. It is mandatory to confirm differential diagnosis where the main cause of CPP does not correspond to PCS. Also, specific treatment should be offered for each condition, especially if gynecological or extra-gynecological

3.5 Diagnostic imaging modalities

Color-coded Doppler echocardiography

It is the first-line diagnostic assessment tool thanks to its non-invasive, non-radiation capabilities. It performs real-time studies of vein hemodynamics and allows performing maneuvers and position changes that improve its sensitivity and specificity (> 90% in PCS).

The assessment protocol should include: 1) evaluation of compression syndromes such as NCS and MTS covering IVC, left renal vein (LRV), and primitive iliac veins; 2) gonadal veins; 3) internal iliac veins, and 4) PFL points at the pelvic PVI and LL junction.

Access routes are transabdominal, transvaginal (TV) and PFL. The latter is used to study inguinal, gluteal, obturator and perineal points.

There are proven diagnostic criteria in the CDE of the different PCS components that should be confirmed for diagnostic precision purposes. The CDE of **Vein Compression Syndromes** facilitate the detection of *Direct Signs* of stenosis that have a high diagnostic value and are associated with a reduction of vein diameters and speed changes; and *Indirect Signs* that originate in the circulatory changes occurred on both sides of the stenosis and the development of collateral circulation.

The diagnostic criteria of **Pelvic Varicocele** include findings of gonadal veins (proximal with diameters >6 mm, Valsalva reflux, and distal with increased number and caliber with tortuous, ecstatic morphology, and ecstatic flow) and hypogastric veins.

Also, the CDE is key in the postoperative management of PTA (to treat compression syndromes) and TE (to treat insufficient pelvic varicose veins, PFL points, and LL).

Computed axial tomography scan and magnetic resonance imaging

Both imaging modalities should cover from the renal veins to the pelvis and the roots of the thighs to characterize incompetent pelvic veins, different drainage routes, and possible vein compression causes.

The MRV has a 88% sensitivity and a 67% specificity for the identification of gonadal vein PVI compared to conventional phlebography in the diagnosis of PCS. Also, it can characterize other causes of CPP (endometriosis, adenomyosis, myomas). It requires dynamic evaluation with IV contrast with images in the arterial phase (to view venous reflux and arterial vascular causes) and in the venous phase (venography, for the measurement of vascular diameters, for the assessment of compromised veins, to identify venous obstruction).

Both the computed axial tomography scan (CAT scan) and the MRI are used for monitorization purposes after the endovascular treatment. Also, it is necessary to first assess the residual clinical signs and identify the possible redistribution of venous drainage to another (hypogastric) territory. There can be artifacts in the MRI due to TE material and venous PTA that should be taken into account.

Gonadal and iliac dynamic phlebography

It is the diagnostic study with the highest sensitivity and specificity (80% to 100%) and it is considered the gold standard in the diagnosis of PCS. It provides a high-precision venous dynamic assessment with confirmation of incompetent veins, varicose veins, and their collateral leaks and even obstructive lesions. It is the imaging modality used to guide endovascular treatments.

Performed via brachial, internal jugular or femoral venous catheterization, it requires minor sedation (or not) and short hospital stays. It should include the assessment of IVC and LRV, gonadal, primitive iliac, hypogastric veins and their pelvic floor branches. It allows us to measure venous pressures from renal and primitive left iliac stenoses. The details of these findings are essential to confirm the diagnosis of PCS.

Gonadal and iliac dynamic phlebography (GIDP) is indicated in 3 situations mainly: 1) in the presence of inconclusive findings of non-invasive studies; 2) when it is necessary to confirm the findings of non-invasive studies; and 3) at the proposal and planning of endovascular treatment for the PCS.

3.6 Medical therapy

To start medical therapy for the management of PCS, first it is necessary to rule out other causes of CPP. Once the PCS has been confirmed as the causative agent and since it is often multifactorial, medical therapy will be targeted at controlling the symptoms derived from PVI and dilatation (pain killers, venotonic drugs, and elastic compression bandages) and the hormone condition involved in the development of the syndrome (hormone medication).

Non-steroidal anti-inflammatory drugs

Used as a first-line therapy, they provide temporary relief, are limited by adverse events, and are often used temporarily until specific therapies are implemented.

Ergotamine

Vasoconstrictor that, when administered IV, improves the clinical signs of PCS pain in 30% of the patients. Limited use due to its short effect and multiple contraindications.

Hormone medication

Progestogens. Used to treat CPP due to endometriosis; to treat the PCS, medroxyprogesterone acetate (MPA) of antiestrogenic and gonadotropic effect is used. They partially improve the symptoms of PCS.

GnRh agonists. Using synthetic analogs, menopause-like hypoestrogenism is achieved. Although they can improve the clinical signs of PCS, their use is limited due to the associated rate of climacteric symptoms and osteoporosis.

Venotonic drugs

Diosmin, hesperidin. They improve vein and lymphatic circulation by reducing venous stasis and symptoms in PCS and LL, with optimal tolerance.

Compression treatment of pelvis and LL

The use of compression pants and stockings has shown clinical improvement in over 80% of the cases of patients with PCS and LL varicose veins.

3.7 Endovascular treatment

The main purpose of this therapy is to eliminate PVI components (varicose veins, pelvic refluxes, and collateral circulation), which happen to be the major contributors of the PCS clinical signs. Endovascular treatments of venous catheterization that are performed at the cath lab are outpatient procedures or have a very short hospital stays and are technically successful in over 95% of the cases with clinical improvement in 68% to 100% of the cases and evidence level 1B. The endovascular treatment techniques used are the TE of choice for the management of gonadal or hypogastric vein incompetency, while the PTA and venous recanalization often solve venous obstructive phenomena.

Transcatheter embolization

Based on the information provided by imaging modalities, but mainly the GIDP, the TE should occlude previously found incompetent gonadal, utero-ovarian varicose veins and hypogastric branches with the use of sclerosing liquids and metal devices (*coils, plugs*). TE is usually performed outpatiently with 24-hour hospital stays.

TE related complications often occur in less than 3% of the cases and are associated with postembolization syndrome; coil migration can be treated via an endovascular approach. Postoperative management is clinical and based on scales like the visual analog scale (VAS) and the quality of life (QOL) scale. CAT scans or MRI, but especially the DCE, are important to assess the post-TE results at PFL points and LL varicose veins level.

Vein compression syndrome angioplasty

The vein obstruction-compression causes of PCS are often underestimated; however, the MTS has been reported in up to 80% of all PVI cases with PCS.

When PCS is associated with IVC obstruction or MTS, the PTA will initially be performed with *astent*, and in the presence of persistent PCS clinical signs, a TE will be performed at a second stage on the pelvic varicose veins. However, when PCS is associated with the NCS, a TE on the veins with reflux will be performed first, and the PTA of the LRV will be spared for cases of hematuria, severe lumbar pain or varicose veins persistent after the TE. The ilio-caval PTA shows primary, assisted primary and secondary patency of 79%, 100%, and 100%, respectively, in non-thrombotic disease, and 57%, 80%, and 86% respectively, in DVT at the 7-year follow-up.

3.8 Surgical therapy

Conservative

It includes the **ligation of ovarian veins by laparotomy or laparoscopy** where the main benefit is diagnostic confirmation, especially for the management of gynecological conditions. The PCS of around 70% of patients is controlled using both techniques, but these are short-term experiences and with frequent relapses. These therapies have a 2B level of evidence recommendation and they are indicated only in cases of unavailability or therapeutic failure of less invasive techniques because they are associated with common major complications and high costs.

Non-conservative

The resection of uterus and/or annexes (uni- or bilateral) contributes a significant benefit in the control of PCS, in ad-

dition to the patients' psychological and social recovery. However, the high dispersion of results, common relapses due to lack of VPI control, but above all, the higher morbidity and mortality rates and recovery time reported have turned these therapies into last-line alternatives, only after other simpler, less bloody, and effective treatments have failed.

Vascular and endovascular surgical treatment of compression syndromes as cause of PCS

Basically, it includes the management of the MTS and the NCS.

The May-Thurner Syndrome. Before 2000, 75% of the procedures were performed by open surgery (25% via endovascular approach). Bypass surgical techniques were used that would create arteriovenous fistulas with unsatisfactory results due to high rates of DVT and restenosis. After 2000, this ratio reversed and 4.1% of the patients are operated on through open surgery and 95.9% via endovascular approach with balloon PTA and stenting and, sometimes, coadjuvant thrombolytic drugs (33.2%) or not (53.2%). As medical therapy (7%), anticoagulation and elastic compression are used.

The Nutcracker Syndrome. *Conservative treatment* is based on a 24-month wait-and-see approach in patients under 18 and a 6-month wait-and-see approach in adult patients, in whom weight gain and the development of collateral circulation improves clinical signs in 70% of the cases.

Intervention is decided once this period of time has elapsed or if there is clinical worsening with hematuria and severe recurring pain, an impaired kidney function, and significant varicocele.

Surgical therapy includes complex and different techniques such as renal vein transposition, renal autotransplantation, gonado-caval or splenorenal *bypass* and LRV ligation with bypass to iliac vein. Although a suitable resolution of the symptoms is initially achieved initially in over 80% of the patients, recurrence can occur at the end of follow-up due to venous restenosis. The complications reported require hospitalization and long controls, which is why this therapeutic decision is made by expert surgical teams in the management of these cases.

Endovascular treatment is performed using PTA with LRV *stenting*. A PCS comprehensive treatment is recommended, first performing TE on gonadal and hypogastric varicose veins, and then renal PTA. With clinical improvement >95%, it requires a careful PTA technique and *stent* selection.

3.9 Additional extrapelvic management of PCS

The rates close to 80% reported in the association among pelvic, vulvar, perineal, and LL varicose veins are in correlation with the PCS, which represents nearly 35% of the cases of vulvovaginal varicose veins, and 90% of the cases of LL varicose veins. After the endovascular treatment of PCS with PFL related-LL vein incompetence, at times, relapses or varicose vulvoperineal remnants can occur and are responsible for the persistence of postoperative LL vein incompetence.

The study protocol of LL vein incompetence due to PCS is similar to the one used in LL varicose vein relapse after TE. It includes detailed clinical assessment of persistent LL varicose veins associated with the CDE study (enhanced with the use of augmented reality) of the PFL points and LL varicose veins involved. TV-CDE, CAT scans, MRI, GIDP, and varicography

are used based on the need for more detailed anatomical and/or functional information on refluxes or their possible causes. Special attention should be paid to the coexistence of superficial and/or deep LL vein incompetence, either essential or post-thrombotic associated with PCS, which should be properly detected and treated.

The management of these relapses if defects or omissions of already-embolized hypogastric or gonadal refluxes are reported, starts by correcting these errors with a second TE that should be as complete as possible, especially on the tributaries that irrigate the relapsed venous territories.

Several ultrasound-guided sclerosis techniques (with or without augmented reality) used on perineal varicose veins and PFL points are described with promising early results. However, studies long enough follow-up periods are still needed on this regard.

Finally, the procedure will be performed on the LL vein incompetence (saphenous trunks or accessories, leak perforator vessels) using conventional techniques (conventional major or minor saphenectomy, staged removal of varicose segments, perforator vessel surgery).

4. INTERSOCIETAL ARGENTINE PELVIC CONGESTION SÍNDROME CONSENSUS

4.1 History and epidemiology

Authors: Juan Esteban Paolini, Néstor Omar Giráldez.

4.1.1 History

Back in 1857, Albert Richet (1) came up with the very first anatomical description of ovarian varicose veins. Almost a century later, in 1949, Taylor (2) associated the anatomical variables with the physiological disturbances generated by ovarian vein incompetence. Also, he was the first to propose the concept of pelvic congestion syndrome (PCS) and suggested the ligation of ovarian veins as a therapy. He also considered that the origin of that vein incompetence was somehow connected with changes of the autonomous gonadal nervous system, suggesting a possible psychosomatic association with the symptoms. Since the true pathophysiology of gonadal vein incompetence was not known, the disease was treated as a psychological/psychiatric disorder in patients who presented with pelvic pain without an apparent cause (3).

In 1954, Dixon and Michell performed the first radiological studies of circulatory anomalies with phlebographic findings. In 1964, Clark described the right ovarian vein syndrome (4). In the 1980s, Colombian gynecologist Abraham Lechter coined the term female gonadal system incompetence or pelvic vein incompetence (PVI) as the causative agent of lower limb (LL), pelvic, and vulvar varicosity. Afterwards, he conducted studies in cadavers and was able to determine the anatomical variants and proposed the open bilateral ligation of ovarian veins (5,6). British gynecologist Beard redefined PCS (7-9) by proposing hysterectomy with bilateral oophorectomy as the best way to treat this condition (10).

Taking into account the complexity of this procedure and the hormonal changes it causes, it was considered mutilating surgery, one that is hard to accept by young women with reproductive potential, a presentation characteristic of this disease (3).

In 1993, Edwards (11) proposed for the first time, the endovascular treatment option with embolization of ovarian veins, thus changing the paradigm of the female pelvic varicocele therapy. Leal Monedero et al. (12,13) determined the pathophysiological basis of sub-diaphragmatic refluxes and embolizing treatment. This highly effective, low morbidity and minimally invasive therapy replaced almost all the remaining conventional open surgical procedures considered mutilating treatments to this date (3).

In some Latin-American countries the laparoscopic treatment of pelvic varicocele is still an elective procedure (14-16) but thanks to the development of diagnostic means and technological advances, compared to the remaining therapies, endo-

vascular treatment has been issued a Class II b Recommendation by the American Venous Forum and the Society of Vascular Surgery (17,18).

Chung et al. (19) conducted a comparative study about PCS therapies available today and confirmed the statistically significant benefits of endovascular treatment.

4.1.2 Epidemiology

According to Candance (20), PCS is associated with chronic pelvic pain (CPP) in 30% to 40% of the cases where PVI is the characteristic etiological component of PCS.

CPP is a complex nosological entity consisting of pelvic pain of over 6 months of clinical evolution often affecting one or several systems (gynecological, urinary, osteo-artro-muscular, digestive, nervous) in different order and intensity, and followed by often emotional repercussions. Mainly, it affects women (but not exclusively), and it is highly prevalent among the general adult population. Still, according to the reports available its incidence rate is variable: 15% in the USA and 43% in other countries (21,22).

Zondervan et al. (23) reviewed a UK primary healthcare database that included the medical histories of 162 women from 12 to 70 years old. They found an incidence rate of 38/1000 per patients-year going to doctors's offices due to CPP.

CPP amounts to 20% of all outpatient gynecological appointments. An annual US\$ 881,5 million are spent in outpatient care and the overall annual cost in the United States is US\$ 2 billion and £158 million in the UK (21,24). Several epidemiological studies, both prospective and retrospective, despite their different scientific quality, have proved that PVI is responsible for between 11.2% and 28% of the cases of CPP (25-27). Similarly, other PVI assessment studies in women with recurrent LL varicose veins have found prevalence rates of 20% to 76% (28).

4.1.3 References

1. Richet NA. *Traité pratique d'anatomie medicochirurgicale*. Paris: E. Chamerot, Libraire Editeur; 1857.
2. Taylor HC. Vascular congestion and hyperemia; their effects on structure and function in the female reproductive system. *Am J Obstet Gynecol* 1949;57:637-53.
3. Paolini JE. *Síndrome Congestivo Pélvico, Tto Quirúrgico, Guías latinoamericanas de terapéutica para la patología venosa*: Roberto Simkin, et al. 1a ed. Edit. Narayit, 2016. Pp. 299-303. ISBN 978-987-28471.
4. Kornberg Alejandro. Diagnóstico y tratamiento de venopatías pélvicas. *Síndrome de congestión pélvica. Várices vulvares*. *Rev Forum de Flebología y Linfología* Vol 8, Número 1, Mayo 2006.

5. Lechter To. Pelvic varicose veins treatment. *J Cardiovasc Surg* 1985;26:111.
6. Lechter A, Alvarez A, Lopez G. Pelvic Varicose and Gonadal Veins, *Phlebologie*, September 1987; vol. 2,3: pp. 181-188.
7. Beard RW, Highman JH, Pearce S, Reginald PW. Diagnosis of pelvic varicosities in women with chronic pelvic pain. *Lancet* 1984;2:946.
8. Beard RW, Reginald PW, Wadsworth J. Clinical features of women with chronic lower abdominal pain and pelvic congestion. *Br J Obstet Gynaecol* 1988;95:153.
9. Beard RW, Reginald PW, Pearce S. Psychological and somatic factors in woman with pain due to pelvic congestion. *Adv Ext Biol Med* 1988;245:413.
10. Beard RW, Kennedy RG, Gangar KF. Bilateral oophorectomy and hysterectomy in the treatment of intractable pelvic pain associated with pelvic congestion. *Br J Obstet Gynaecol* 1991;98:988-92.
11. Edwards RD, Robertson IR, Maclean AB, Hemmingway AP. Case report: pelvic pain syndrome – successful treatment of a case by ovarian vein embolization. *Clin Radiol* 1993;47:429-31.
12. Leal Monedero J, Zubicoa S, Grimmer M. Subdiaphragmatic Venous Insufficiency, Embolization Treatment using mixed technique (Coils and Foam). *Phlebology* 2004;45:269-75.
13. Leal Monedero J. Indicaciones y tratamiento del síndrome de congestión pélvica. *Rev de Flebología y Linf. Lecturas vasculares*. 2010;14:841-7.
14. Grabham JA, Barrie WW. Laparoscopic approach to pelvic congestion syndrome. *Br J Surg* 1997;89:1264-6.
15. Gómez G. Varicoceleotomía por laparoscopia: Descripción de una nueva técnica y su acción sobre el dolor pélvico. *Med Reprod* 1998;1:14-7.
16. Navarro H, Vinuesa B, Martínez D. Síndrome de congestión pélvica. Evaluación del dolor después del tratamiento laparoscópico con Ligasure. *R Colomb Med* 2005;36:153-7.
17. Głowiczki P, Comerota AJ, Dalsing MC. The care of patients with varicose vein and associated chronic venous disease. *Clinical practice Guidelines of the Society of Vascular Surgery and the American Venous Forum*, *J Vasc Surg* 2011;53:25-485.
18. Głowiczki P, Dalsing M, Eklöf B, Moneta G, Wakefield T, et al. *Handbook of venous disorders* (4th. ed.). Guidelines of the American Venous Forum 2009;56:617-25.
19. Chung MH, Huh CY. Comparison of treatments for pelvic congestion syndrome. *Tohoku J Exp Med* 2003;201:131-8.
20. Pelvic Congestion Syndrome: Systematic Review of Treatment Success. Candace L. Brown. *Semin Intervent Radiol* 2018;35:35-40.
21. Hangari A. Prevalence of chronic pelvic pain among women: an updated review. *Pain Physician* 2014;17(2):E141-E147.
22. Riding DM, Hansrani V, McCollum C. Pelvic vein incompetence: clinical perspectives. *Vasc Health Risk Manag* 2017 Nov 27;13:439-47.
23. Zondervan KT, Yudkin PL, Vessey MP, Dawes MG, Barlow DH, Kennedy SH. Prevalence and incidence of chronic pelvic pain in primary care: evidence from a national general practice database. *Br J Obstet Gynaecol* 1999;106(11):1149-55.
24. Matias SD, Kuppermann, Chronic Pelvic Pain: prevalence, health-related quality of life, and economic correlates. *Obst Gynecol* 1996;87:321-7.
25. Almeida EC, Nogueira AA, Candido dos Reis FJ, Rosa e Silva JC. Cesarean section as a cause of chronic pelvic pain. *Int J Gynaecol Obstet* 2002;79(2):101-4.
26. Hebbar S, Chawla C. Role of laparoscopy in evaluation of chronic pelvic pain. *J Minim Access Surg* 2005;1(3):116-2.
27. Howard FM. The role of laparoscopy in chronic pelvic pain: promise and pitfalls. *Obstet Gynecol Surv* 1993;48(6):357-87.
28. Marsh P, Holdstock J, Harrison C, Smith C, Price BA, Whiteley MS. Pelvic vein reflux in female patients with varicose veins: comparison of incidence between a specialist private vein clinic and the vascular department of a National Health Service District General Hospital. *Phlebology* 2009;24(3):108-13.

4.2 Pathophysiology and clinical signs

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4.2.1 Pathophysiological mechanisms

4.2.1.1 Introduction

The characteristics of the pelvic congestion syndrome (PCS) were initially described and published by Richet back in 1857 and then associated with chronic pelvic pain (CPP) in 1947 by Taylor (1,2). In recent years, it has been observed that there is not a unanimous consensus on the denomination of PCS

to designate this entity; however, it is included in the International Classification of Diseases and VEIN-TERM of the Transatlantic Interdisciplinary Consensus (3). Although it is considered that there can be a multiple origin of PCS (hormonal, anatomical, post-partum, etc.), gonadal, hypogastric pelvic vein incompetence (PVI) and its spread towards the lower limbs (LL) is without a doubt the determinant pathophysiological factor. The coexistence of other participant mechanisms in PCS make the causal relation of PVI, though proven, not categorically established as the main causative agent of PCS (4-8). In this well-known underestimation of PCS (9,10), 2 elements of conceptual confusion are involved, both highly frequent among the female population of reproductive age. On one hand, utero-ovarian varicose veins can be found in up to 10% of the women. In 40% of these women, they are not associated with PCS, and it is an anatomical finding without any associated clinical signs. This means that no specific therapy or follow-up is required. On the other hand, 30% of the women's CPP is due to PVI only, while another 15% of the patients with PCS have other additional non-venous related pelvic etiology. This makes the isolate identification of main and secondary problem ambiguous when it comes to administering evidence-based therapies (11).

To properly distinguish when the PCS is mainly associated with PVI or in what patients PCS is only a minor component is often ideally approached by multidisciplinary working groups, both at the diagnostic stage and when having to decide the most suitable approach or therapy (12).

4.2.1.2 Pathophysiology

It is commonly accepted that the origin of PCS is multifactorial, and vein incompetence, venous obstruction, and hormonal changes are the most usual suspects.

The cause of pain in PCS is associated with the progressive venous distension that together with vascular stasis stimulate the release of local inflammatory and pain mediators (13). In fact, the histopathological examination of these pelvic varicose veins shows fibrosis of tunica intima and tunica media with muscular hypertrophy and capillary endothelium proliferation (14). It is likely that the pain mechanism of PCS in its chronification process also acquires characteristics of growing complexity as it happens with CPP (15).

PVI, the leading cause of PCS, is anatomically characterized by the abnormal and insufficient dilatation of veins in the ovarian, uterine, hypogastric territories and very commonly in the LL too. The interconnection of ovarian and hypogastric venous systems mainly occurs in the broad ligament of the uterus and its fixation elements. From there, PVI can transmit venous reflux towards the hemorrhoidal, sacro and vesical circuits. More frequently, PVI of internal pudenda veins, round-ligament venous plexuses and parametrium lead to PCS *per se*; the predominant compromise of obturator and femoral circumflex veins often occurs as vulvar varicose veins and LL vein incompetence; and ovarian vein incompetence often occurs with both clinical signs (16). Back in 1997, Leal Monedero introduced the accurate concept of sub-diaphragmatic vein incompetence (a denomination that unified PCS and LL chronic vein incompetence), which is in turn pathophysiological divided into "centrifugal reflux" (the predominant anti-physiological pelvic venous flow or in the direction of pelvic and LL varicose veins) and "centripe-

tal reflux" (the utero-ovarian flow has a physiological direction, but with increased volume, and it is part of the collaterality of central venous obstruction, post-thrombotic syndrome (PTS) with ilio-caval compromise, May-Thurner syndrome (MTS).

The hormonal involvement of PCS can be seen in the different clinical instances. The regularity of PCS pain is associated with menstrual cycle in women whose symptoms decrease after the menopause, and with the response to treatment with anovulant drugs and human chorionic gonadotropin agonists where estrogen depletion, among other mechanisms, would be decreasing the response of the smooth musculature of the venous wall (9).

The pathophysiological mechanisms of PCS show a close correlation between its origin, frequency and response to the different modalities of treatment. They have been schematically categorized by different authors (9,17-19): *Primary PCSs* are due to ovarian venous valvular incompetence, venous anatomical variants, venous kinking following uterine malappositions, and structural and hormonal changes associated with pregnancy and labor. *Primary PCSs* occur most often with typical clinical signs of PCS associated, or not, with LL vein incompetence and they are the group that best responds to transcatheter embolization of endovascular treatment (ET).

Secondary PCSs are associated with severe stenosis or occlusion of drainage veins at the pelvic or suprapelvic level. Often chronic post-thrombotic, it compromises the iliac veins (primitive or internal) or the inferior vena cava (IVC) and they are often called, respectively, iliac compression syndrome or MTS and IVC syndrome. Some authors include *congenital* causes in this group (while other authors place them separately) like pelvic vascular malformations, IVC agenesis and aplasia, iliac veins and changes to the left renal vein (LRV) like compression due to aorto-mesenteric clamp (AMC) (or anterior nutcracker syndrome) or its retro-aortic location (posterior NCS) (9,20-22). Although they show clinical signs of PCS, in this group, patients often show symptoms associated with the territory of the venous obstruction that originate it. Endovascular resolution, properly associating percutaneous transluminal coronary angioplasty (PTA) with stent and TE is often the optimal therapeutics.

Finally, there is a group of *non-venous pelvic* causes that can come from different origins, mostly endometriosis. Other etiologies can correspond to myomas, benign uterine and ovarian tumors, adenomyosis, post-partum uterine retroversion, postoperative and post-infectious adhesions and staples, Master-Allen syndrome (lesion of uterine fixation elements), and non-obstetric trauma. In these cases, the presence of varicose veins and PVI often responds to a coincidental epiphenomenon rather than a direct causal relation. Therefore, once the differential diagnosis has been confirmed, if the cause of CPP is not associated with PCS, the specific treatment of each particular condition should be provided. In these cases, there is no indication for a TE procedure on the utero-ovarian varicose veins since they are rarely involved in the mechanism of pelvic pain.

In conclusion, the pathophysiology of PCS is due to several venous circulatory and functional changes mainly determined by PVI. Understanding the correlation between clinical signs and changes of pelvic vein dynamics is essential

regarding the endovascular and sclerosing therapies. Similarly, cases of primary and secondary PCS of strictly non-venous conditions where multi-disciplinary action would be required should be differentiated.

4.2.2 Clinical signs

CPP is a common cause of consultation in women between 18 and 50 years old. Actually, 39% of women have reported it at one time or another during their lifetime (12). Pain is by definition, a sensory and emotional experience associated with a real or potential damage of some tissue. CPP is an incapacitating situation that not only has physical repercussions, but also psychological and emotional. Several studies have discussed this issue and it is estimated that it causes the loss of jobs in 15% of the population and a 45% productivity reduction (8). In the United Kingdom, 18% of women report CPP and between 2% to 10% of all gynecological appointments are due to this cause (9,23).

Of the many causes of CPP, in this consensus document we have been focusing on PCS. In patients without any other apparent cause for CPP, it was confirmed that 30% had PVI and 10% of dilatation of one or both ovarian veins. Back in 2001, Soysal et al. studied women with CPP by conducting pelvic assessments, laparoscopies, ultrasounds and venographies. Thirty-one per cent of them had PCS as the only abnormality while 12% showed PVI associated with another condition like endometriosis, pelvic inflammatory disease, postoperative adhesions, and uterine disease (myoma or adenomyosis) (23-25). PCS is the second leading cause of CPP after endometriosis (26).

Although CPP is a common problem in premenopausal multiparous women, there are several etiologies that lead to these clinical signs and diagnosis can be complex. Fortunately, the evermore precise diagnostic tools available provide accurate information. However, in certain cases the differential diagnosis is complex since multiple conditions can coexist during a woman's reproductive age.

PCS symptoms and signs are varied and some of them are unspecific of PVI. Patients with PCS report a dull, deep, non-cyclical *pain* that is exacerbated by movements, postures, and activities that increase abdominal pressure. Pain can be unilateral or bilateral and it is often asymmetrical and chronic of > 6-month clinical evolution (27,28). It does not have an obvious source and it can be associated with *dyspareunia* (71% of the cases), *dysmenorrhea* (66%), and *post-coital pain* (65%). Less often, it can cause *nausea*, *distension or abdominal cramps*, *rectal discomfort*, *dysuria* and *an increased urinary frequency*. Symptoms of the neurological and psychosomatic spectrum such as *reactive depression* are also prevalent (24,28).

Physical findings suggestive of PCS include *sensitivity at ovarian, uterine and cervical points* that are exacerbated during bimanual uterine palpation. The presence of *utero-ovarian and pelvic varicose veins in vulva, gluteus and legs* are characteristic signs associated with pelvic leak points, which will be dealing with below (9). Association with symptomatic hemorrhoids is common.

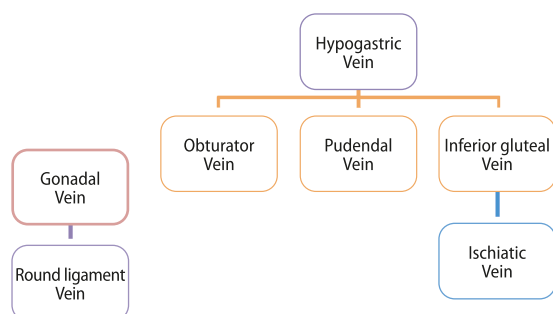
As we have already mentioned among the causes of CPP, PCS ranks second as the entity causing it. PCS can be due to different causes that determine PVI and, in turn, develop more or less signs and symptoms depending on the degree of dilatation and personal factors such as phys-

ical build, activity, number of pregnancies or hormonal status that can have a negative or positive impact.

The factors that favor the development of PCS include *normal or slim body builds* with a low body mass index (BMI). A study conducted by Labropoulos et al. categorizes patients into normal weight (BMI <25), overweight (BMI, 25-29.9), and obese (BMI > 30). It was confirmed that most of the patients with PCS have a low BMI ($24.1 \text{ kg/m}^2 \pm 5.6 \text{ kg/m}^2$) (27). Regarding activity, those with a *sedentary lifestyle or an increased intra-abdominal pressure* are considered harmful (8). The number of pregnancies also has a negative impact and PCS is more common and severe among multiparous women. However, nulliparous women can also develop PCS (29). There is also a correlation between vein dilatation and *estrogen* level since symptoms decrease with the arrival of the menopause (28).

Venous circulation patterns in PVI are closely associated with the different causes of PCS (30). In *primary PCSs* due to venous wall *insufficiency*, centrifugal flow is established (which is inversely proportional to the usual flow). When the situation is perpetuated due to reflux and varicose veins, venous hypogastric and pelvic floor collaterality that do not stabilize and increase over time are established. In *secondary obstructive PCSs* due to MTS, NCS or IVC occlusion, centripetal flow is established and, in this case, venous collaterality tries to compensate the venous hypertension established by the stenosis or the obstruction. If pressures are equalized, venous collaterality finally stabilizes. *Concomitant PCSs* due to local causes (endometriosis, tumor mass, postoperative of gynecological surgeries) can coexist with utero-ovarian varicose veins; in these cases, we should see whether the PVI, the gynecological condition or both are determinant factors of the painful clinical signs described.

This venous collaterality, contained in the pelvis as pelvic varicose veins, will seek **pelvic floor leak points (PFL)**. These are preexisting anatomical connections between pelvis and LL through extrapelvic branches of the hypogastric vein that end up developing hemorrhoids, vulvar varicose veins, testicular varicocele in men, and LL varicose veins. Leak points are well-known and established by Dr. Leal Monedero and Dr. Zubicoa who spare the following veins with extrapelvic communication (see diagram below).



Vulvar varicose veins are more common during the fifth month of second pregnancies. However, they often disappear during the first month after delivery (31). They are more prevalent on the left side, but they can occur on both sides and in some cases the obstetrician prefers

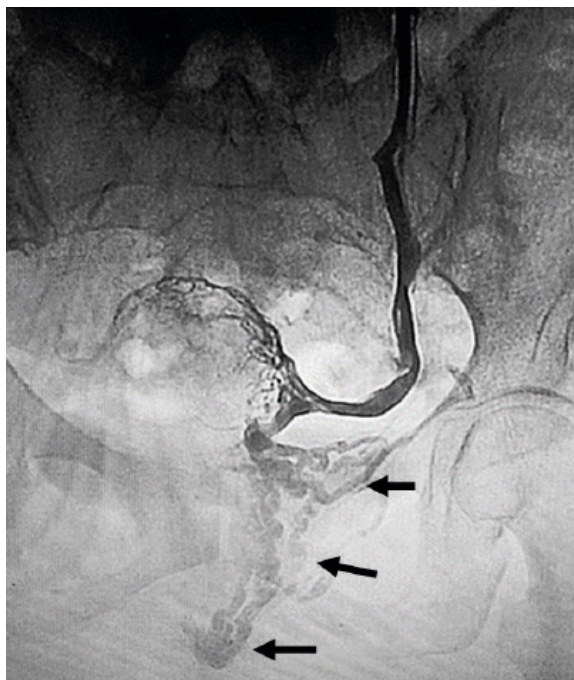


Figure 1. Left gonadal phlebography with the Valsalva maneuver showing reflux with leak (arrows) toward left round ligament vein and vulvar veins.

to perform a C-section incision since episiotomy, if required, could bleed heavily.

LL varicose veins of pelvic origin should be assessed with special care since they do not respect the saphenous axes and their appearance and distribution in the LL responds to the above-mentioned leak points. When they appear semiologically, we should complete the diagnostic tests while suspecting the presence of a possible PCS in case this was not the reason for the patient's consultation. Otherwise, relapses and failed phlebological therapies can be a common thing here. Another indirect cause for patient's consultation regarding PCS can precisely be *LL varicose relapse*. Perrin et al. showed PVI in 17% of 170 patients with relapse (32). Another study of 100 women with PVI (as seen on the venography and confirmed in the clinical examination in 71% of the cases) revealed the presence of leak points on the LL (in 58% of the cases). Actually, two thirds of these women showed varicose relapse after previous *stripping* (8,32).

Finally, obstructive PCS associate **typical symptoms of venous compressions**. Regarding CNS, *pain on the left side and macro- or micro-hematuria* can occur, sometimes intermittently. For several authors, when it is present it is the ultimate indication for treatment and to abandon a conservative treatment stand. The cases of MTS can present with *edema* associated with *atypical left LL varicose veins*. However, let us remember that compressions on the right iliac veins are also present.

4.2.2.1 Differential diagnosis

The diagnosis of PCS is achieved after discarding other causes, mainly *gynecological ones of CPP* since these are common in the age group afflicting patients with PCS. Similarly, PCS as cause of CPP is very common and it represents the second etiology of CPP after endometriosis. PCS is a misdiagnosed condition, one on which we should use all diagnostic tools available since, in some cases, the PCS can be associated with other gynecological

TABLE 2. PCS-CSS: The Clinical Severity Score is focused on assessing the severity of 7 symptoms (dysmenorrhea, dyspareunia, lower back pain, hypogastric heaviness, LL stasia, dysuria, and constipation) and 3 signs of PCS (genital varicose veins, atypical varicose veins, and hemorrhoids), and their response to the therapy administered

Symptoms	Absent = 0	Mild = 1	Moderate = 2	Severe = 3
1. Dysmenorrhea	No	Painful menstruation, but it rarely inhibits normal activity, pain medication is rarely required.	Daily activity is affected. Pain medication is required. It allows everyday activities.	Daily activity clearly inhibited, poor response to pain medication and frequently associated with vegetative symptoms (headache, fatigue, nausea, vomiting, diarrhea).
2. Dyspareunia (at the end of sexual intercourse and/or post-coital).	No	1-3/10 (VAS) Tolerable (regular sex life).	4-6/10 (VAS) Tolerable (keeps sporadic sexual relations).	7-10/10 (VAS) Intolerable (avoids sexual intercourse).
3. Lower back pain (trauma origin discarded; it gets worse with menstruation).	No	1-3/10 (VAS) Ocasional, no restringe la actividad y no requiere analgésicos.	4-6/10 (VAS) Diariamente, limitación moderada de actividad, analgésicos ocasionales.	7-10/10 (VAS) Diariamente, limitación severa de las actividades y requiere el uso regular de analgésicos
4. Lower abdomen heaviness (gynecologic origin discarded; it gets worse with menstruation).	No	Occasional, does not restrict activity and does not require pain medication.	Daily, moderate limitation of activity, occasional pain medication and it improves with phlebotonic drugs	Daily, severe limitation of activities and requires regular use of pain medication and phlebotonic drugs.
5. Stasis (pain and/or heaviness associated with the LL; it gets worse with menstruation).	No	Occasional, does not restrict activity and does not require pain medication.	Daily, moderate limitation of activity, occasional pain medication and it improves with phlebotonic drugs	Daily, severe limitation of activities and requires regular use of pain medication and phlebotonic drugs.
6. Dysuria with burning sensation and/or pollakiuria and/or vesical tenesmus (urological origin discarded; it gets worse with menstruation).	No	Occasionally	Frequently	Always
7. Chronic constipation (for at least 3 consecutive months, or not, during the previous 6 months).	No	Bowel movement every other day.	At least three bowel movements a week.	Less than three bowel movements a week.
Signs				
8. Genital varicose veins.	No	C1 (CEAP)	C2 (CEAP)	C2 (CEAP) with stasis discomfort and/or dyspareunia.
9. Atypical LL varicose veins.	No	C1 (CEAP)	C2 (CEAP)	C2-C3 (CEAP) and/or stasis +
10. Hemorrhoids (internal and/or external with or without bleeding).	No	Sensation of a foreign anal body. (Requires anoscopy).	Prolapsed (they can be reduced manually).	Prolapsed (they CANNOT be reduced manually).

conditions and make clinical signs confusing (24,26). That is why the *interdisciplinary differential diagnosis* of this condition should be the rule of thumb. The assessment of patients should include initially a thorough *clinical examination* with questions and physical examination oriented to PVI and other gynecological and non gynecological causes (33-35). This early assessment will lead to the selection of *laboratory and invasive and non-invasive imaging* tests, which will eventually guide the ultimate diagnosis and therapeutic decision-making process.

In this diagnostic stage, it should be possible to differentiate 2 groups of patients:

1. Patients with typical PCS (primary or secondary) who often benefit from TE and occasionally from PTA; it is possible to treat them with medication and they rarely require surgery. The management of these patients is done by consensus.
2. Patients with CPP and isolated signs of PCS should be considered with special care. It is mandatory to confirm whether the main cause of CPP is not due to PCS and specific treatment should be provided for this gynecological or extra-gynecological condition.

In order to be able to achieve the differential diagnosis of PCS, the *Royal College of Obstetricians and Gynaecologists* (RCOG) (23) classification of the Gynecological and Extra-gynecological **causes of CPP** should be cited:

Gynecological

- Endometriosis/adomyosis.
- PCS.

- Uterine fibromas.
- Ovarian tumors.
- Pelvic inflammatory disease.
- Postoperative or post-inflammatory adhesions.

Extra-gynecological

- Surgeries: appendectomy, adhesions.
- Urological: interstitial cystitis, chronic urinary inflammation, urolithiasis, urethral syndrome.
- Intestinal: irritable bowel syndrome, inflammatory bowel disease.
- Ortho-neuro-muscular: degenerative changes, neuropathies, prolapse of nucleus pulposus, nerve pinching (pudendum).
- Psychosomatic: depression, sleep disorders, anxiety, migraine with abdominal symptoms, history of sexual abuse.
- Neurological: scar induced nerve incarceration, chronic pain in the region provided by the affected nerve, in 3.7% of the cases caused by Pfannenstiel incision (23).

4.2.2.2 Clinical evaluation of the results of the therapies applied

Clinical Severity Score

In the clinical assessment, it is important to know the *scores* that quantify the symptoms and signs of PCS and group the patients in comparable diseased groups. This way it is feasible to obtain greater accuracy regarding the estimation of the results obtained with the therapies applied. Regarding clinical assessment, several authors have used indices such

as quality of life (QOL) or the visual analogue score (VAS) validated to be used to measure PCS (7,36). The classification of patients based on the clinical signs and pathophysiology has been proposed recently (37-39), seeking to differentiate subgroups of patients with PCS and observe the response to the therapies prescribed. To this date, a clinical severity score PCS (PCS-CSS) has been presented to assess these patients' pain with a score and also be able to unify criteria (40) (**Table 1**).

Regarding this clinical severity scale (PCS-CSS), different scores are assigned to the study patient:

- Minimum score: 0 points.
- Maximum score: 30 points.
 - *Absent or unlikely*: 0-3 points
 - *Mild*: 4-7 points
 - *Moderate*: 8-15 points
 - *Severe*: >15 points.

Several medical societies proposed years ago standard forms to report findings and results (7), and despite the important worldwide experience in the diagnosis and treatment of PCS, studies of high scientific quality are still scarce. To this end, important efforts are being made, worldwide, by different medical societies involved in the management of PCS. The International Union of Phlebology (IUP) has recently presented a very comprehensive consensus document on this regard (37) and other societies are evaluating unified ways to study and classify patients with PCS (38,39), among them the American Vein and Lymphatic Society (AVLS) (37-40).

4.2.2.3 AVLS Classification (40)

This anatomo-clinico-pathophysiological classification considers a gradation of symptoms (S), venous signs (V), and damaged venous territories (P), where the latter concept is in turn subclassified into anatomy (A), type of hemodynamic alteration (H), and etiology (E).

Symptoms: "S"

- S0: No clinical signs of venous disease.
- S1: Renal symptoms (pain on the side, hematuria).
- S2: Chronic pelvic pain due to vein incompetence (pain, dyspareunia, dysuria).
- S3: Transitional symptoms (leak points: gluteal, pudendal, vulvar).
- S4: LL symptoms:
 - a. Unspecified symptoms (pain, edema).
 - b. Venous claudication.

Signs – varicose reservoirs involved: "V"

- V0: No varicose veins in the imaging clinical assessment.
- V1: Renal hilar varicose veins.
- V2: Pelvic varicose veins.
- V3: Transitional varicose veins (leak points) (included in CEAP).
- V4: LL varicose veins (included in CEAP).

Pathophysiology: "P"

- Anatomy: (A)
 - LRV (left renal vein).
 - OV (ovarian veins).
 - IV (common iliac veins; (external iliac veins;) (hypogastric veins;).
 - P (perforator pelvic veins).

Hemodynamic Anomalies: (H)

- R (reflux) – decompensated.
- RC (reflux) – compensated.
- O (occlusion) – decompensated.
- OC (occlusion) – compensated.

Etiology: (E)

- Thrombotic: due to DVT (reflux/obstruction).
- Non-thrombotic: primary reflux or compression syndromes (reflux/obstruction).
- Congenital: vascular malformations; venous or mixed.

Definitions

1. *CPP*: intermittent or constant pelvic pain of, at least, 6-month evolution unrelated to menstruation or pregnancy.
2. *Pelvic varicose veins*: dilatations and tortuosities of periuterine veins > 5 mm in diameter.
3. *Transitional varicose veins*: subcutaneous dilated veins >3 mm in diameter due to reflux of pelvic veins and their leak points.
4. *Renal veins related symptoms*: symptoms due to high blood pressure of renal veins including micro-/macro-hematuria, left side pain or abdominal pain.
5. *Venous claudication*: exertional pain in the limbs often described as "painful tension" commonly on the thighs, but not in a single muscle group only.

In conclusion, PCS poses a diagnostic and therapeutic challenge. Therefore, integrating the knowledge of several medical specialties is required.

4.2.3 Recommendations

1	Multidisciplinary clinical assessment of patients with suspected PCS to rule out other causes of CPP and together with laboratory tests and imaging modalities differentiate the populations of primary or obstructive PCS due to PVI (Level of Evidence C, Class II a Recommendation).
2	Use clinical assessment indices (QOL, VAS) to measure symptom improvement before and after the therapies prescribed.

4.2.4 References

1. Richet MA. *Traite pratique d'anatomie medico-chirurgicale*. Paris, 1857.

2. Taylor HC. Vascular congestion and hyperemia; their effects on structure and function in the female reproductive system. *Am J ObstetGynecol* 1949; 57:637-53.

3. Eklof B, Perrin M, Delis KT, Rutherford RB, Gloviczki P. American Venous Forum; European Venous Forum; International Union of Phlebology; American College of Phlebology; International Union of Angiology. Updated terminology of chronic venous disorders: the VEIN-TERM transatlantic interdisciplinary consensus document. *J Vasc Surg* 2009;49:498-501.

4. Venbrux AC, Lambert DL. Embolization of the ovarian veins as a treatment for patients with chronic pelvic pain caused by pelvic venous incompetence (pelvic congestion syndrome). *CurrOpinObstetGynecol* 1999;11:395-9.

5. Zehra N, Kurt A, Ipek A, et al. The relation between pelvic varicose veins, chronic pelvic pain and lower extremity venous insufficiency in women. *Diagn IntervRadiol* 2006;12(1):34-8.

6. Liddle AD, Davies AH. Pelvic congestion syndrome: chronic pelvic pain caused by ovarian and internal iliac varicose veins. *Phlebology* 2007; 22(3):100-4.

7. Black CM, Thorpe K, Venbrux AC. Research reporting standards for endovascular treatment of pelvic venous insufficiency. *J VascIntervRadiol* 2010;21:796-803.

8. Koo S, Fan CM. Pelvic congestion syndrome and pelvic varicosities. *Tech VascIntervRadiol* 2014;17(2):90-5.
9. Kuligowska E, Deeds L 3rd, Lu K 3rd. Pelvic pain: overlooked and underdiagnosed gynecologic conditions. *Radiographics* 2005;25:320.
10. Wozniak S. Chronic pelvic pain. *Annals of Agricultural and Environmental Medicine* 2016, Vol 23, No 2, 223-6.
11. Hansrani V, Abbas A, et al. Trans-venous occlusion of incompetent pelvic veins for chronic pelvic pain in women: a systematic review. *Eur J Obstet-GynecolReprod Biol* 2015;185:156-63.
12. Black CM, Thorpe K, Venbrux AC. Research reporting standards for endovascular treatment of pelvic venous insufficiency. *J VascIntervRadiol* 2010;21:796-803.
13. Soysal ME, Soysal S, Vicdan K, Ozer S A randomized controlled trial of goserelin and medroxyprogesterone acetate in the treatment of pelvic congestion. *Hum Reprod* 2001;16(5):931-9.
14. Durham JD, Machan L. Pelvic congestion syndrome. *Semin InterventRadiol* 2013;30(4):372-80.
15. Farrell JF, Vilos GA, et al. N° 164 Consensus Guideline for the management of Chronic Pelvic Pain. *J ObstetGynaecol Can* 2018;40(11):e747-e787.
16. Lopez AJ. Female pelvic vein embolization: indications, techniques, and outcomes. *Cardiovasc InterventRadiol* 2015;38(4):806-20.
17. Phillips D, Deipolyi AR, Hesketh RL, Midia M, Oklu R. Pelvic congestion syndrome: etiology of pain, diagnosis, and clinical management. *J VascIntervRadiol* 2014;25(05):725-33.
18. Durham JD, Machan L. Pelvic congestion syndrome. *Semin InterventRadiol* 2013;30(04):372-80.
19. Greiner M, Dadon M, et al. How does the patho-physiology influence the treatment of pelvic congestion syndrome and is the result long-lasting? *Phlebology* 2012;27 Suppl 1:58-64.
20. Leal Monedero J, ZubicoaEzpeleta S, et al. Treatment options for pelvic congestion syndrome. *Phlebology* 2016;23:135-41.
21. Menezes T, Haide E, et al. Pelvic congestion síndrome due to agenesis of the infrarenal inferior vena cava. *Radiology case reports* 2019;14:36-40.
22. Niclot J, Stansal A, et al. A post-thrombotic pelvic congestion syndrome. *JDMV* 2018;43:310-15.
23. Slawomir Wozniak. Chronic Pelvic Pain. *Annals of Agricultural and Environmental Medicine* 2016, Vol 23, No 2, 223-6.
24. Lazarashvili Z, Antignani PL, Leal Monedero J. Pelvic congestion syndrome: prevalence and quality of life. *Phlebology* 2016;23:135-41.
25. Metzger DA. Mechanism of pain in pelvic congestion syndrome. Presented at the 9th Scientific Meeting of the IPPS, San Diego, CA, 2002.
26. Soysal ME, Soysal S, Vicdan K, et al. A randomized controlled trial of goserelin and medroxyprogesterone acetate in the treatment of pelvic congestion. *Hum Reprod* 2001;16:931-9.
27. RuhaniNanavati, Patrick Jasinski, Demetri Adrahtas, AntoniosGasparis, NicosLabropoulos, Stony Brook, NY. Correlation between pelvic congestion syndrome and body mass index. *Journal of Vascular Surgery*. 2018. Article in press.
28. Leal Monedero J, ZubicoaEzpeleta S, Perrin M. Pelvic congestion syndrome can be treated operatively with good long-term results. Review article phleb. 2011.012503. *Phlebology* 2012;27 Suppl 1:65-73.
29. Kies DD, Kim HS. Pelvic congestion syndrome: a review of current diagnostic and minimally invasive treatment modalities. Review article. *Phlebology* 2012;27 Suppl 1:52-7.
30. Hartung O. Embolization is essential in the treatment of leg varicosities due to pelvic vein incompetence. Review article. *Phlebology* 2015, Vol. 30(15):81-5.
31. Antignani PL, Geroulakos G, Bokuchava M. Clinical aspects of pelvic congestion syndrome. *Phlebology*. 2016;23(3):127-9.
32. Perrin M, Labropoulos N, Leon L: Presentation of the patient with recurrent varicose after surgery (REVAS). *J Vasc Surg* 2006;43:327-34.
33. Asciutto G, Asciutto K, Mumme A, et al: Pelvic venous incompetence: Reflux patterns and treatment results. *Eur J Endovasc Surg* 2009;38:381-6.
34. Belenky A, Bartal G, Atar E, Cohen M, Bachar GN. Ovarian varicose veins in healthy female kidney donors: incidence, morbidity, and clinical outcome. *AJR Am J Roentgenol* 2002;179:625-7.
35. Monedero J. Insuficienciavenosacronica de la pelvis y de los miembros inferiores. Madrid, Spain: Mosby/DoymaLibros SA; 1997.
36. Laborda A, Medrano J, de Blas I, et al. Endovascular Treatment of Pelvic Congestion Syndrome: Visual Analog Scale (VAS) Long-Term Follow-up Clinical Evaluation in 202 Patients. *Cardiovasc InterventRadiol* (2013) 36:1006-14.
37. Antignani PL, Lazarashvili Z, Monedero JL, et al. Diagnosis and treatment of pelvic congestion syndrome: UIP consensus document. *International Angiology* 2019 August;38(4):265-83.
38. Mark Meissner. Villavicencio Symposium: Advances in Pelvic Venous Disease (PEVD). Classification of Pelvic Venous Disorders. American Venous Forum 2020; 32° annual meeting; Omni Amelia Island Plantation Resort; Amelia Island, Florida, USA.
39. Khilnani MN, Meissner MH, Learman LA, et al. Research Priorities in Pelvic Venous Disorders in Women: Recommendations from a Multidisciplinary Research Consensus Panel. Standards of Practice. *J VascIntervRadiol* 2019;30(6):781-9. <https://doi.org/10.1016/j.jvir.2018.10.008>. Article in Press
40. Leal Monedero J, Sánchez Galán A. Score de severidadclínica para Síndrome de CongestiónPélvica. <https://vimeo.com/422926430>, 2020. Simposio Virtual.

4.3 Diagnostic imaging modalities

4.3.1 Color-coded Doppler echocardiography

Authors: Luis Miguel Catalina, Carlos D'Alotto

4.3.1.1 Introduction

Inside the diagnostic algorithm of patients with CPP and/or suspected PCS, the color-coded Doppler echocardiography (CDE) is in the first diagnostic line. This is mainly due to the fact that it is a *non-invasive*, low-cost imaging modality that provides *real-time hemodynamic information* and allows the *assessment of the patient in different positions* (decubitus, standing) using provocative maneuvers (Valsalva), which increase the sensitivity of this imaging modality regarding the detection of pelvic varicocele (1-3)

The main objective of this segment in this consensus document is to establish the study protocol and diagnostic ultrasound and Doppler criteria to identify pelvic varicocele and be able to select patients who will be examined using an abdominopelvic phlebography to plan endovascular therapeutics.

4.3.1.2 Study protocol

We believe that the study protocol should be *comprehensive* and include all the possible sectors where abdominal and pelvic veins can be affected and develop pelvic varicocele including pelvic leaks and development of LL varicosities.4 For this reason, the study protocol has been divided into 4 different steps:

1. Assessment of compression syndromes: NCS, MTS.
2. Assessment of gonadal axes.
3. Assessment of internal iliac axes.
4. Pelvis-LL connection: Assessment of pelvic leak points.

4.3.1.3 Access routes and patient preparation:

Currently, there are 3 *ultrasound-guided access routes* used to assess PCS:

1. **Transabdominal** (3-5 Mhz convex transducer). This access route is used to study the abdomen and the pelvis in the search for high (NCS) and low (MTS) compression syndromes, view the proximal and medial sectors of gonadal axes, and the iliac confluence (external and internal iliac veins). Also, it is useful at pelvic level to obtain a panoramic general view of the periuterine and periaxial venous structures prior to transvaginal (TV) assessment.

It allows us to examine the patient in different positions (decubitus and standing) by increasing the sensitivity of the Doppler study to detect vascular venous

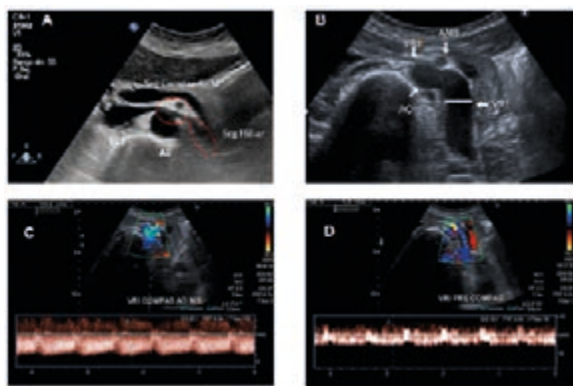


Figure 2. A) Cross-sectional view at normal epigastrium level showing the 2 segments (red circles), the trajectory of the LRV where peak velocity and diameter are measured. AO, abdominal aorta; IVC, inferior vena cava. B) NCS, LRV dilatation in the hilar LRV segment and significant caliber reduction at AMC level. SMA, superior mesenteric artery. C and D) Measurements of velocity with spectral Doppler in the compressed (AMC) and hypoflow regions (hilar segment).

compressions. If possible, the operating table should be in an inclined plane (about 30°) to increase the study sensitivity.

2. **Transvaginal (TV)** (intracavitary transducer). It is the access route of choice to assess periuterine plexuses and the cervicovaginal plexus. It provides a focalized visualization of them and a correct performance of the Valsalva maneuver looking for refluxes. If it is possible to incline the operating table, we suggest doing so at approximately 30° to increase the sensitivity of the study.
3. **Pelvic leaks** (7-12 Mhz linear transducer). We use this access route to assess the 4 main pelvic floor leak (PFL) points where the pelvic reflux connects to the LL (inguinal point: I; perineal point: P; obturator point: O; gluteal point: G). This study should be performed whenever there is clinical evidence of subdermal varicosities at inguinal or vulvo-vaginal level, on the thigh inner and posterior side, which is known as *atypical varicosities*.

There are two ways of study leak points: with the patient *standing* in a position similar to that used during the venous assessment of LL or else with the patient lying in *decubitus in the lithotomy position*; the latter is the one the authors prefer because it exposes the pelvic floor much better for the evaluation of the 4 leak points and it is often a more comfortable position for the patient. The drawback here is that venous refill is usually better in the standing position so when in doubt, the patient should be examined while standing keeping in mind that this often increases detection sensitivity since all the venous structures increase their caliber while standing.

Regarding the patient's preparation for transabdominal examination, we recommend an 8-hour fasting preferably with an empty bladder, so it does not compress the vascular structures and alters the findings. The anthropometric profiles of most patients with PCS are often favorable for ultrasound examination since they usually have a low body mass index, which in 80% of the cases is < 25.5. The TV access route and pelvic floor assessment not used to look for leaks do not require any special preparation.

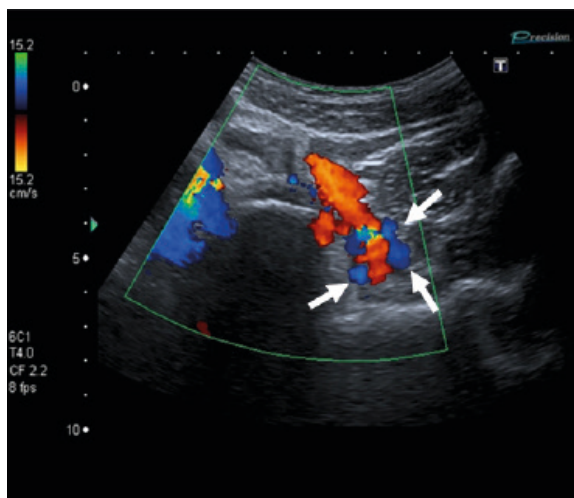


Figure 3. Cross-sectional view at iliac artery bifurcation level showing multiple left paravertebral venous structures surrounding the left primitive iliac artery, a finding consistent with collateral circulation (white arrows). Patient with NCS and MTS with secondary left iliac thrombosis.

4.3.1.4 Diagnostic criteria of vascular compression syndromes

In every stenosis or vascular compression, whether arterial or venous, vascular flow changes occur at the compression site together with secondary changes that are distal to of compression site. These vascular flow changes are findings we look for on the Doppler echocardiography to achieve the diagnosis.

The changes that occur at the compression site are called *direct signs* and those that are distal to the compression site are called *indirect signs*. *Direct signs* are often more sensitive and specific compared to the indirect ones since they involve direct ultrasound imaging of the compression site and flow assessment at this level.

The nutcracker syndrome (6-11)

Direct signs (Figure 2):

- Aortomesenteric clamp (AMC) < 3 mm (normal: 4 mm to 5 mm).
- Absence of flow in the left renal vein (LRV) in the AMC.
- Diameter ratio; caliber of the LRV hilar segment/caliber of the AMC segment: ≥ 5 .
- Velocity ratio; peak velocity at AMC/peak velocity of hilar segment: ≥ 5 .
- AMC angle < 30° to 35°.
- Dynamic assessment while the patient remains standing or seated: in the true compression there may not be changes with respect to decubitus or an even greater closure of the AMC can occur with increased dilatation of the LRV hilar segment. In pseudo-NCS there is often decreased compression and, therefore, a reduction of the LRV caliber in the hilar segment.

Indirect signs (Figure 3):

- Collaterality through renal hilum and paravertebral veins.
- Collaterality through left gonadal vein (derivative flow type).

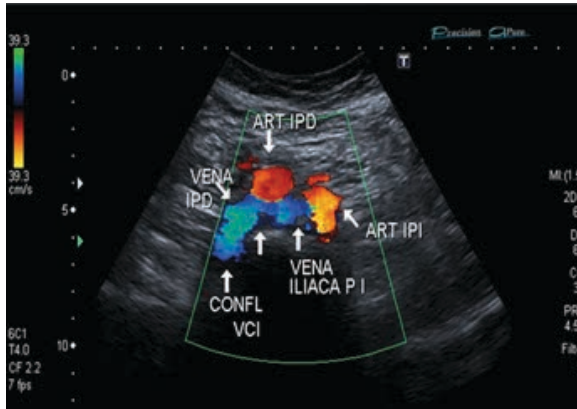


Figure 4. Cross-sectional view at normal umbilical level showing the iliac venous confluence and the junction between the right primitive iliac artery (RPIA) and the left primitive iliac vein (LPIV) one of the sites where vascular compression occurs most often in the MTS.



Figure 6. A) Cross-sectional view at left side level showing the ostium of the dilated right gonadal vein (white arrow) at the level of the IVC; see its relation to the psoas muscle (P) and the paravertebral location (VB: vertebral body). B) Cross-sectional view at left side level showing the dilated left gonadal vein (white arrow). (ao: abdominal aorta).

The May-Thurner syndrome

Direct signs (Figure 4):

- Total collapse of left primitive iliac vein in the junction with the right primitive artery with absence of flow in the Doppler examination.
- Diameter ratio: Caliber of segment prior to crossing/caliber of iliac crossing: ≥ 5 .
- Velocity ratio: peak velocity at iliac crossing/peak velocity at segment prior to crossing: ≥ 5 .

Indirect signs (Figure 5):

- Flow asymmetry in external iliac veins (the left external iliac vein loses its respiratory phasicity and spectral flattening occurs).
- Flow reversal in left internal iliac vein.
- Increased flow in right internal iliac vein.
- Presence of left ascending lumbar collaterals and uterine and cervicovaginal plexuses.

4.3.1.5 Diagnostic criteria of pelvic varicocele

There are 2 venous axes that can be assessed on the CDE that can lead to developing pelvic varicocele. They are the gonadal veins (left and right) and the internal iliac or hypogastric veins (right and left).

Gonadal veins

The assessment of gonadal veins begins from their proximal sector using a transabdominal transducer while trying to locate the ostial sector that will be found on the right side, in the inferior vena cava (usually between hours 9 and 11), and on the left side at the middle third of the LRV level. Then,

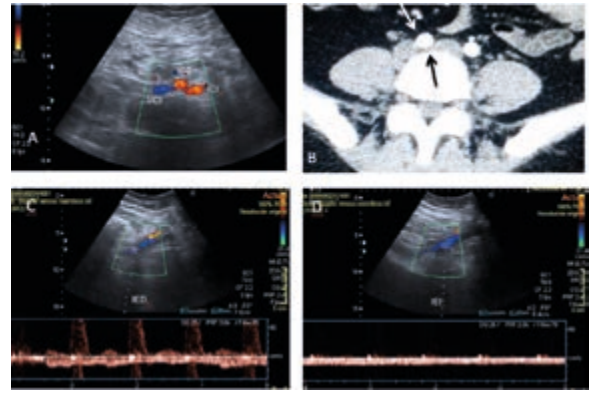


Figure 5. A) Cross-sectional view at umbilical level showing the right primitive iliac artery is observed (RPIA) and the absence of flow in the left primitive iliac vein, a direct sign of MTS. B) Correlation with the CAT scan: the RPIA compresses the left primitive iliac vein against the vertebral body (arrows). C) Comparative spectral Doppler between the right external iliac vein (preserved venous phasicity) and D) left external iliac veins with a flattened spectral pattern with loss of phasicity, an indirect sign of MTS.

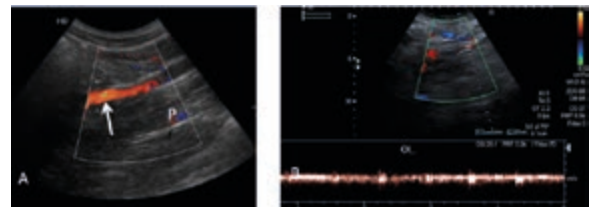


Figure 7. A) Longitudinal view at the left side level showing the dilated left gonadal vein (white arrow) with reflux after Valsalva maneuver; see its correlation with the psoas muscle (P). B) The spectral Doppler assessment performed on the longitudinal view of the left gonadal vein confirms the presence of venous reflux.

the gonadal veins run anteriorly and in close correlation with the psoas-iliac muscle, which should be our main anatomical reference since it can be easily identified on the ultrasound exam (Figure 6).

We use the following reference measurements in the cross-sectional view of gonadal veins:

- 4 mm: normal.
- 4 mm to 6 mm: borderline.
- >6 mm: dilated.

Regardless of venous caliber, it is always important to *functionally* assess the vein using the Valsalva maneuver to rule out venous reflux; once we see it dilated on the cross-sectional tracking, we perform a longitudinal cut of the vein and assess the presence of venous reflux using a color-coded spectral Doppler echocardiography. We are interested in the proximal (ostial) and medial sectors of the gonadal veins via transabdominal access (Figure 7).

The distal sector at pelvic level is often more difficult to assess via transabdominal access; gonadal veins at this level usually look plexiform with multiple connected venous structures, which is why we prefer to use the TV access to perform this assessment (Figure 8).

Venous reflux, whether from the internal gonadal or iliac axes, will unload at plexoperiuterine and cervicovaginal vein level (Figure 8). The definition of PCS consists of an increase in both *number* and *caliber* of intrapelvic venous structures. These are veins with a varicose, tortuous, and ectatic morphology and with an ectatic slow flow.

From this definition the *diagnostic criteria for PCS via TV* access can be established:^{13,14} (Figure 9):

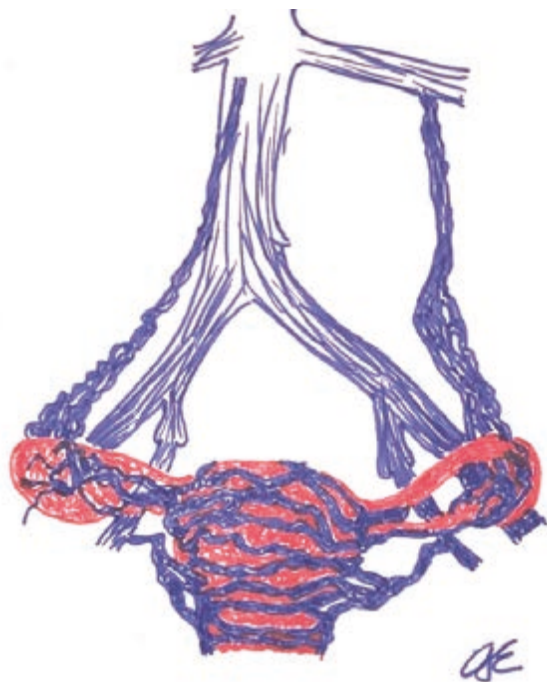


Figure 8. Diagram showing the most common anatomical configuration of gonadal veins (12). See the plexiform feature acquired by the distal third of the veins keeping a close correlation with the annexes; this makes the transabdominal assessment of this segment more difficult. At this level the TV access site is the preferred one. In this diagram we can also note how the 2 main venous axes (internal gonadal and iliac) contribute to the development of pelvic varicocele by dilating venous structures of the periuterine plexus. (Modified from Intermountain Vein Center 2014).

- Tortuous periuterine plexus veins of between 5 mm and 6 mm caliber.
- Dilated veins of the intrauterine arcuate plexus crossing the body of the uterus towards the contralateral side (pelvic collaterality).
- Confirmation of > 5 dilated venous structures.
- Slow ectatic flow (sometimes it is identified as venous smoke or *rouleaux* flow).
- Presence of reflux after the Valsalva maneuver.

The visualization of dilated periuterine venous structures bilaterally can occur as a *sign of collaterality* of an incompetent gonadal axis, generally the left one, where the reflux seeks a return pathway towards the IVC through the contralateral gonadal axis. Or else, it can be a sign of *bilateral gonadal incompetence*, where both axes reflow contributing to pelvic varicocele. This situation is often differentiated using the Valsalva maneuver where the incompetent axes will show reflux, while the axis that acts as physiological return with hyperflow of centripetal direction will not show reflux after the same maneuver. We should mention that Dr. Leal Monedero's group describes 2 possible situations that can occur in the flow of venous structures of the periuterine plexus and the gonadal axis in the presence of pelvic varicocele (1).

- Continuous venous flow *interrupted* after the Valsalva maneuver. According to the author, this finding is highly indicative of a *derivative type of flow* as it occurs in vascular compression syndromes (NCS and MTS) (**Figure 10**).
- Slow or absence of flow in pelvic varicosities showing *reflux* after the Valsalva maneuver. This is indicative of primary gonadal or iliac internal incompetence.

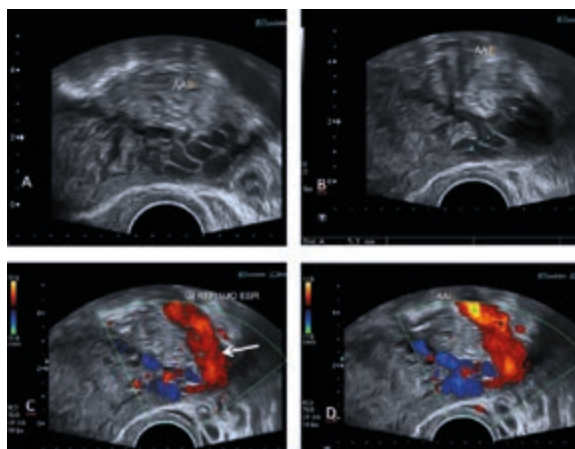


Figure 9. A) and B). TV ultrasound of the left adnexal area (LAA) showing multiple dilated venous structures of the periuterine plexus. C) and D) CDE assessment showing spontaneous reflux from an incompetent left gonadal vein (white arrow) that increases after the Valsalva maneuver, confirmed through the intense color red seen and greater refill of the venous structures.

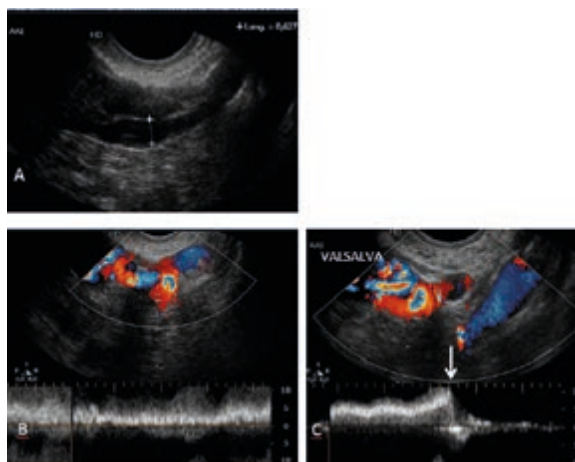


Figure 10. A) TV ultrasound showing the dilated left gonadal vein distal sector. B) Spectral Doppler assessment showing a continuous increased flow at rest. C) After the Valsalva maneuver (white arrow) the flow decreases significantly until it becomes interrupted, a finding that is highly indicative of derivative flow due to NCS-type proximal vascular compression.

Internal iliac or hypogastric veins

According to the PCS study protocol, internal iliac veins are the most difficult vascular structures to assess through CDE. This is due to the fact that they are short veins that emit multiple segmental branches, they are located in the depth of the pelvis, they are hard to assess functionally with dynamic maneuvers whether transabdominal or transvaginally. They are often valveless veins, unlike their segmental visceral or parietal branches (15). Studying the iliac confluence via transabdominal access is advised. For all this and contrary to what occurs with diagnostic signs in gonadal axes and compression syndromes, the *indirect* signs seen in the iliac axes are often more important than the *direct* signs when studying these veins.

Internal iliac veins have visceral (uterine, vaginal, vesical, rectal) branches, and parietal tributaries (gluteal, perineal, obturator); the latter have an *extrapelvic* trajectory towards the LL.

The assessment protocol of internal iliac axes would be next.16 (**Figure 11**):

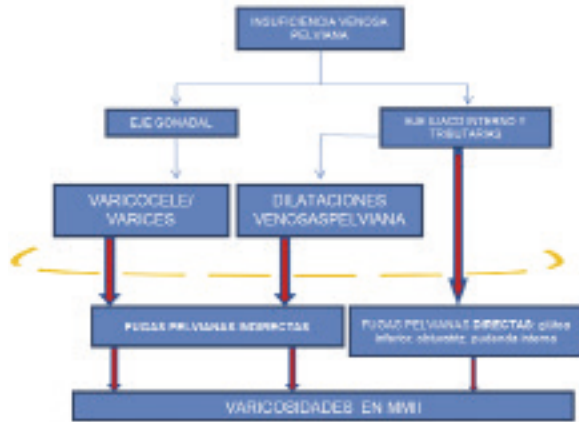


Figure 11. Algorithm whose relevance is seeing how there are times when there may not be any pelvic varicose dilatation but still presence of vein incompetence from the pelvis leaking into the LL through direct leaks that are segmental extrapelvic branches of internal iliac veins (internal gluteal, obturator and pudendal) (16).

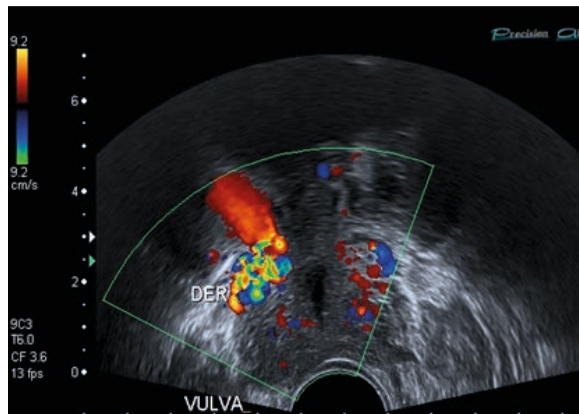


Figure 12. B) TV ultrasound study of the cervicovaginal plexus; the tip of the transducer is at vulvar level in the vaginal introitus. Right reflux from the internal pudendal vein can be seen.

- After *gonadal axes* have been assessed and they appear *normal*, and there are no periuterine or cervicovaginal intrapelvic venous dilations but atypical varicosities in the LL, it is assumed that they are fed by venous pressure from the internal iliac and/or parietal segmental branches.
- After *gonadal axes* have been assessed and they appear *normal*, and there are no intrapelvic venous dilations but atypical varicosities in the LL, it is assumed that they are fed by venous hypertension from the internal iliac and/or parietal segmental branches.
- In both cases, whenever there is clinical evidence of atypical varicosities in the vulvo-vaginal, perineal, gluteal regions the PFL points should be assessed.

From what has been explained we should mention that, in the TV study, the periuterine-periadnexal plexus (high plexus) and the cervicovaginal plexus (low plexus) should be routinely evaluated (**Figure 12 A and B**). The latter is assessed by removing the TV transducer almost up to the vaginal introitus by performing the Valsalva maneuver. In the absence of significant periuterine plexus dilatation, but in the presence of low cervicovaginal plexus dilatation, it is highly indicative of segmental incompetence of the internal iliac branches, mainly the internal pudendal and obturator veins.

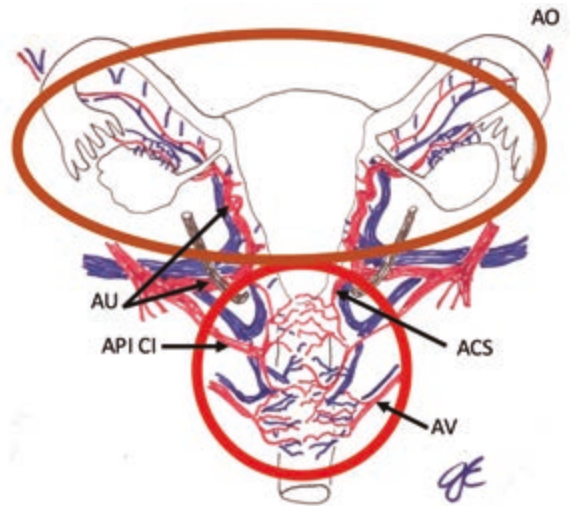


Figure 12. A) Diagram showing the periuterine-gonadal plexus or high plexus (brown oval) and the cervicovaginal or low plexus (red circle). IPICA, internal pudendal and inferior cervical arteries; OA, ovarian artery; SCA, superior cervical artery; UA, uterine artery; VA, vaginal artery.

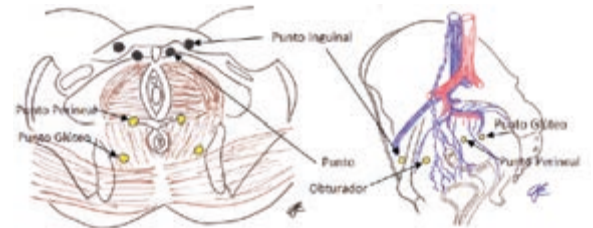


Figura 13. A) Inferior view of pelvic floor in supine position showing the topographic location of the 4 PFL points in each hemipelvis. B) Lateral view of the right hemipelvis showing the trajectory of the pelvic veins and the location in the antero-posterior direction of the 4 leak points. Diagrams changed and taken from NicosLabropoulos, educational material Venous Symposium New York. venous-symposium.com.

4.3.1.6 Pelvic leak points

Pelvic leak points are the connections between the intrapelvic venous system and LL veins. From the comparative point of view, they would be similar to LL perforator veins that connect the superficial to the deep venous system. These are the points through which the venous hypertension originated at the pelvis seeks to unload towards the LL superficial venous system.

Traditionally, 4 *leak points* are examined and described (**Figure 13**) (16,17):

- Inguinal point (Point I)
- Perineal point (Point P)
- Obturator point (Point O)
- Gluteal point (Point G)

We should mention that there are no *ultrasound anatomic landmarks that can be used to look for them*. Therefore, they are searched for based on the clinical examination of the subdermal varicose trajectories on the perineal and LL regions. They are followed in ascending direction until the area where they enter the pelvis can be seen. This is how it is suspected that a leak point may be involved in the origin of the varicose trajectory.

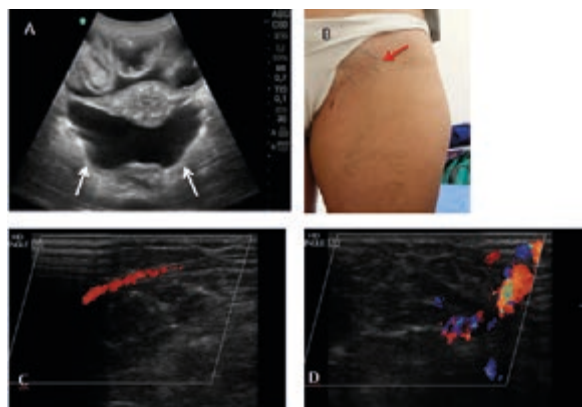


Figure 14. A) Cross-sectional view of a patient with ascites showing the uterine and round ligaments (white arrows) that, under normal conditions, do not show patent vascular structures. B) Patient with leak through inguinal point and subdermal varicosity descending on the thigh antero-external side; the origin is highlighted with a red arrow above the inguinal fold, a very characteristic finding of leak through this point. C) and D: Study using a 7.5 MHz linear transducer at groin level where there is reflux coming from the pelvis and draining into the inguinal point. In this case, it generates epifascial subdermal varicosities in the thigh (B) without compromising the sapheno-femoral junction (SFJ).

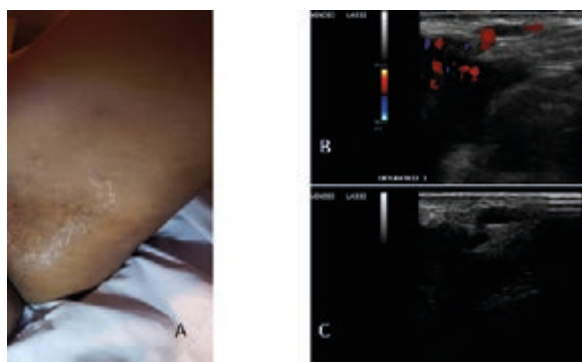


Figure 16. A) Left inner thigh varicosity in ascending direction coming from a leak through the obturator point. B and C) Study with a 7.5 MHz linear transducer following the varicose trajectory and seeing how it connects to the pelvis. See in C the characteristic shape of a "hook-like" image showed by the obturator vein leaking through this point.

Inguinal point (Figure 14)

It is the re-patency of the round ligament vein; varicose trajectories are seen close to the pubis and groin that bringing their reflux into the aortic arch of the *great saphenous vein* or its *tributaries* (superficial inguinal veins). The varicosities come from above the inguinal fold and drain into the LL.

Perineal point (Figure 15):

It is located on the pelvic floor and is responsible for vulvo-perineal varicose trajectories towards the inner thigh; it is searched by moving the transducer parallel to the vulva. It is usually one of the most common leaks, it is associated with internal pudendal vein incompetence, a segmental branch of the internal iliac vein.

Obturator point (Figure 16):

The obturator point can be found underneath the inguinal fold, close to the SFJ. It holds an anterior location to the perineal point. Reflux comes from the obturator vein, which is a segmental intrapelvic branch of the internal iliac vein. Due to its intermediate location between the in-

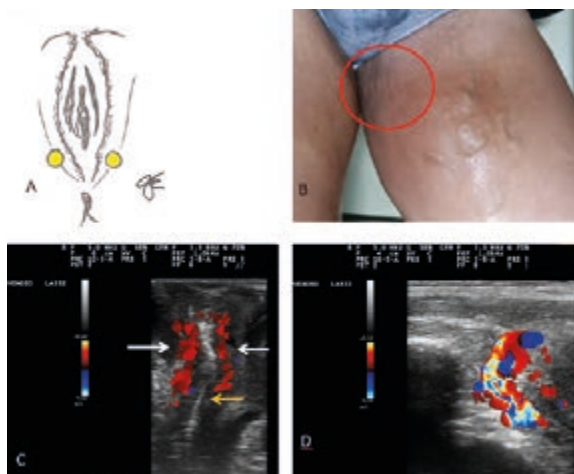


Figure 15. A) Diagram showing the topographic location of the perineal point located on the pelvic floor at the junction of the vulva superior two thirds and inferior one third. B) Clinical examination showing atypical varicosities on the inner thigh (red circle), the usual region of influence of the leak through the perineal point. C) Study with a 7.5 MHz linear transducer parallel to the vulva showing vaginal air as a medial echo-refracting white line (orange arrow) and towards both reflux sides through venous structures of vaginal plexus (white arrows). D) Pelvic leak through perineal point.

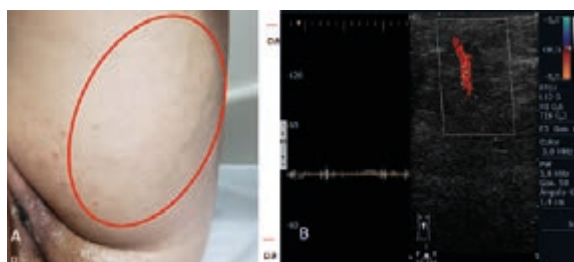


Figure 17. A) Left thigh posterior side varicosity in ascending direction coming from a leak through the superior gluteal point. B) Study with a 7.5 MHz linear transducer following the varicose trajectory showing the leak that, at this level, is found between the muscular masses of the gluteal muscles.

guinal and the perineal points, it is responsible for varicosities both at vulvo-vaginal and inner thigh level.

Gluteal point (Figure 17)

The reflux leaks through the superior and inferior gluteal veins, both extrapelvic branches of the internal iliac vein. Two points are described: leaks through the superior gluteal vein are topographically located at the middle third of the gluteus and leaks through the inferior gluteal vein are topographically located through the inferior gluteal fold. The compromised territory is the thigh posterior side that follows the trajectory of the sciatic nerve.

4.3.1.7 Color-coded Doppler echocardiography during postoperative management

Afer completing the diagnostic part (study protocol and diagnostic criteria for PCS), in the last section of this consensus document we will be describing the utility of CDE during the postoperative management of patients with PCS. Among the endovascular therapeutic possibilities used today, there are 2 excellent choices available (18-20): percutaneous transluminal coronary angioplasty (PTA) with venous stent for the management of compression syndromes; and transcatheter embolization (TE) for the management of gonadal and internal iliac axis incompetence. In both cas-

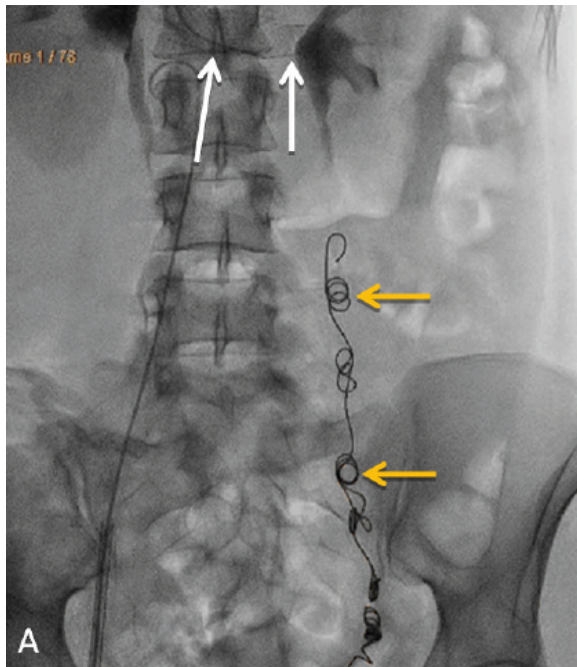


Figure 18. A) Phlebography and PTA with stenting in LRV (white arrows) in a patient with CNS and left gonadal TE with coils (orange arrows)..

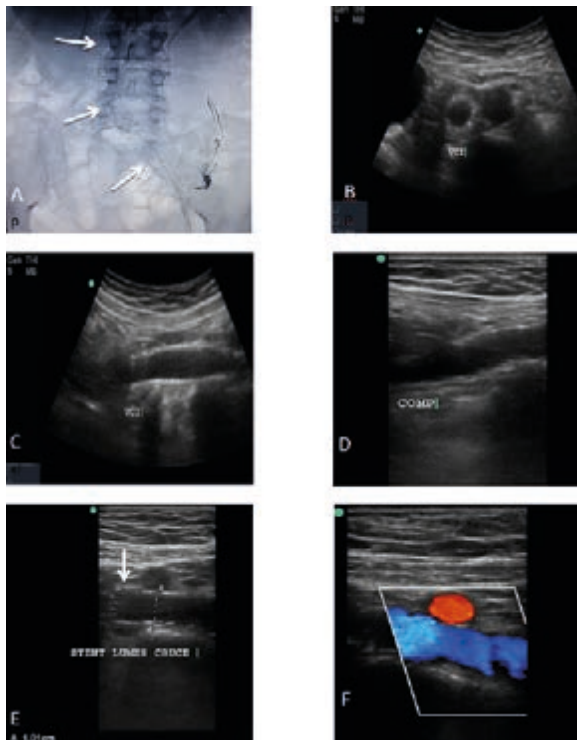


Figure 20. A) Post-PTA phlebography of a patient with MTS and ilio-caval stenting (white arrows). B) and C) Postoperative echo-Doppler, cross-sectional and longitudinal view of the stent proximal border at IVC level. D) Compression (COMP) is performed confirming partial collapse of the stentless IVC segment without changes in the stented segment, characteristic of the venous stent, where its radial strength prevents collapse thanks to the exertion of extrinsic compression. E) and F) Study with a 7.5 MHz linear transducer at iliac vascular crossing level showing the right primitive iliac artery (white arrow) on the left primitive iliac vein with the stent in its lumen, patent, and with a suitable caliber without any signs of compression.

es, the CDE becomes an excellent diagnostic tool for postoperative management purposes both to detect complications and to assess therapeutic success. The same study sys-

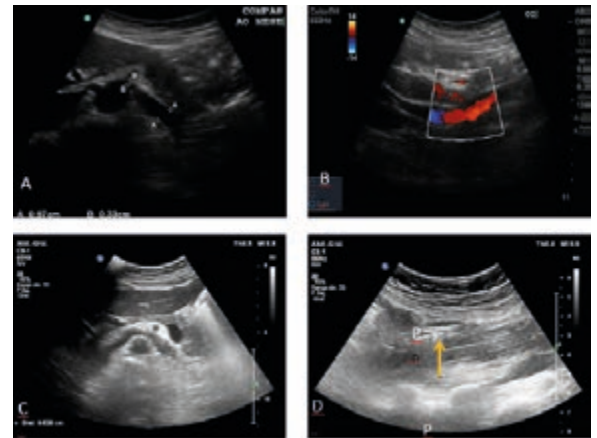


Figure 19. A and B) Preoperative CDE showing the dilated LRV and spontaneous reflux through the left gonadal vein of the derivative type (longitudinal view). C) Postoperative CDE showing the stented LRV; note how the vein recovers its lumen at AMC segment level where it was compressed. D) Longitudinal view showing the occluded left gonadal vein over the psoas muscle (P); the coil can be seen in its lumen as a hyperechoic line with refringence (orange arrow)

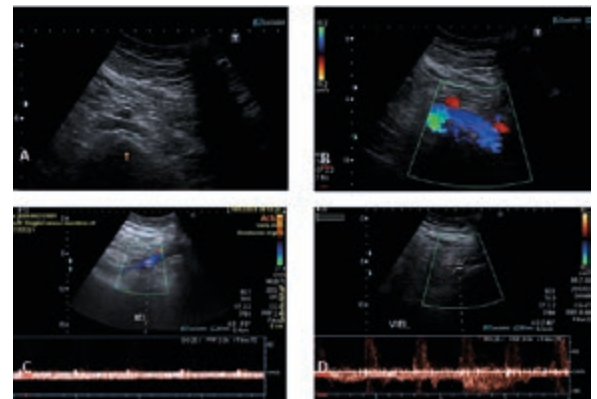


Figure 21. A and B) Postoperative management of a patient with MTS (Figure 4). Note the stent inside the left primitive iliac vein (patent). C) Previous spectral Doppler of left external iliac vein with flattened spectral pattern with loss of phasicity, an indirect sign of MTS. D) Spectral Doppler of left external iliac vein post-PTA that recovers phasicity when proximal compression is resolved.

tematics and approaches described should be used during the postoperative and diagnostic stage alike to assess the results of the different endovascular and/or surgical therapies implemented.

The control CDE should be performed every 6 months for follow-up purposes and assessment of the PTA with stenting and TE (18,19).

During stent control we will be looking for (Figures 18, 19, 20, 21, and 22):

- Patency.
- Input, in-stent, and output velocities.
- Correct positioning (migration).

Using the mixed TE technique (coils and foam) we should become familiar with post-embolization findings. These are often the findings we are talking about (Figure 23):

- Complete occlusion of the venous axis treated, whether gonadal, internal iliac or its segmental branches.
- Findings of small refringences (hyperechoic comet-tail artifact images) along the vascular axis treated correspond to the coils and the foam artifact often present at adnexal area level.

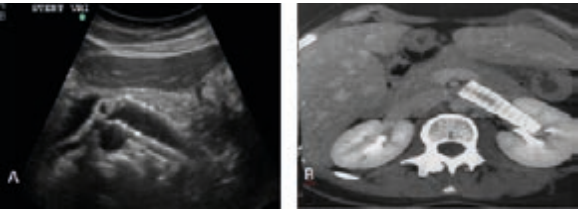


Figure 22. A) and B). Postoperative management of a patient with NCS and PTA with stenting on the LRV. Note its migration towards the renal hilum showing the LRV compression at AMC level.

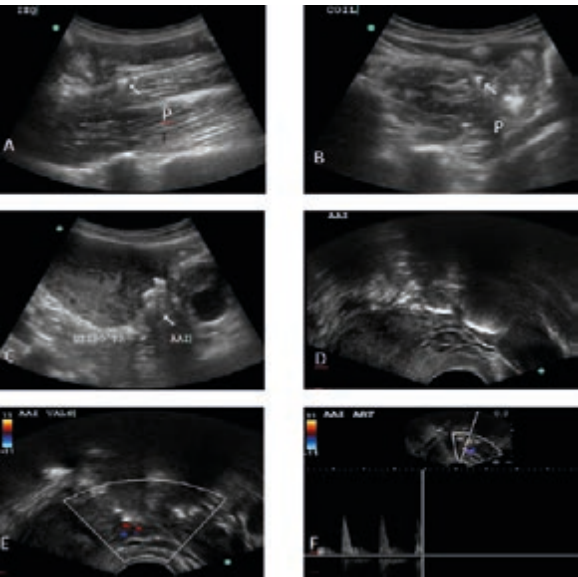


Figure 23. A) Post-TE longitudinal view of left gonadal vein with refrigent hyperechoic line (white arrow) on the psoas muscle (P) indicative of coils in the occluded vein. B) Cross-sectional view with comet-tail artifact generated by the coil (white arrow). C) Transabdominal cross-sectional view with snowstorm artifact image on the left adnexal area (LAA) due to embolizing foam in distal left peritubal and gonadal varicose veins. D) Same TV imaging. E) and F) Control CDE showing arterial circulation only due to occlusion of the venous axes treated.

4.3.1.8 Conclusion

Within the diagnostic algorithm of patients with CPP and clinical suspicion of PCS, ultrasound studies and CDE are among the first diagnostic imaging modalities of choice. Using the diagnostic criteria described, this imaging modality has acceptable percentages of sensitivity and specificity that are often above 90%. There is no doubt that this study has limitations since among the PCS diagnostic studies it is the most *operator-dependent one*. Therefore, to a greater extent, its diagnostic performance will depend on the operator’s skill and training. Other limitations can be the significant presence of abdominal and pelvic gas that will be limiting the visualization of vascular structures and the scarce penetration of ultrasound waves senn in some obese patients, but these are less common. We consider that the *comprehensive approach* is very important in the study of PCS. It should include a *trans-abdominal, transvaginal, and pelvic floor approach to rule out leaks* towards the LL. This is how all the possible causes that trigger pelvic varicocele and the atypical varicosities it can cause on the LL can be studied comprehensively.

Authors’ note: the ultrasound images contained in this chapter were obtained from patients actually. examined by the authors. The diagrams shown are modifications made by Dr. Guillermo Eisele from the references cited in each case.

4.3.1.9 Recommendations (21)

1	We hereby recommend the use of CDE as the imaging modality of choice for the assessment of patients with suspected PCS and in the endovascular postoperative follow-up (Evidence level B-C, Class I Recommendation).
2	We hereby recommend comprehensive studies including transabdominal and TV CDE, and pelvic leak points to study all the possible causes of PCS and its spread towards the LL.
3	We hereby recommend experienced, trained operators to optimize diagnostic sensitivity.

4.3.1.10 References

1. Monedero JL, Ezpeleta SZ, Perrin M. Pelvic congestion síndrome can be treated operatively with good long term results. *Phlebology* 2012;27:65-73.

2. Ignacio EA, Dua R, Sarin S, et al. Pelvic congestion síndrome: diagnosis and treatment. *Semin InterventRadiol*2008;25:361-8.

3. Kies DD, Kim SH. Pelvic congestion syndrome: a review of current diagnostic and minimally invasive treatment modalities. *Phlebology* 2012;27 Suppl 1:52-7.

4. Labropoulos N, Jasinski PT, Adrathas D, et al. A standardized ultrasound approach to pelvic congestion syndrome. *Phlebology* 2017;32(9):608-19.

5. Nanavati R, Jasinsky P. Labropoulos M, et al. Correlation between pelvic congestion syndrome and body mass index. *J Vasc Surg* 2018;67:536-41.

6. Kim SH, Cho SW, Kim HD, et al. Nutcracker Syndrome: Diagnosis with Doppler US. *Radiology* 1996;198:93-7.

7. Takebayashi S, Ueki T, Ikeda N, et al. Diagnosis of the Nutcracker syndrome with Color Doppler Sonography: Correlation with flow patterns on retrograde Left Renal Venography. *AJR* 1999;172:39-43.

8. Ananthan K, Onida S, Davies AH. Nutcracker Syndrome: An update on current diagnostic criteria and management guidelines. *Eur J VascEndovasc Surg* 2017;1-9.

9. Ahmed K, Sampath R, Khan MS. Current trends in the Diagnosis and management of renal nutcracker syndrome: A review. *Eur J VascEndovasc Surg* 2006;31:410-6.

10. Zhang H, Zhang N, Li M, et al. Treatment of six cases of left renal nutcracker phenomenon: Surgery and endografting. *Chin Med J* 2003;116(11):1782-4.

11. Hartung O, Grisoli D, Boufi M, et al. Endovascular stenting in the treatment of pelvic vein congestion caused by nutcracker syndrome: Lessons learned from the first five cases. *Journal Vasc Surg* 2005;42(2):275-80.

12. Lechter A, Lopez G, Martinez C, et al. Anatomy of the gonadal veins: a re-appraisal. *Surgery* 1991;109:735-9.

13. Park SJ, Lim JW, Ko YT, et al. Diagnosis of pelvic congestion syndrome using transabdominal and transvaginal sonography. *AJR* 2004;182:683-8.

14. Steenbeek M, Van der Vleuten C, Schultze Kool L. Noninvasive diagnostic tools for pelvic congestion syndrome: a systematic review. *Acta ObstetGynecolScand*2018;97:776-86.

15. Amore MA, Casal F, Tapia L, et al. Bases anatómicas y fisiopatológicas del síndromecongestivopélvico. *Flebología y Linfología. LecturasVasculares* 2013 Año 8 N 19: 1184-96.

16. Lemasle P, Greiner M. Duplex US investigation in PCS. *Phlebolympology* 2017;24(2):79-87.

17. Franceschi C, Bahnini A. Treatment of lower extremity venous insufficiency due to pelvic leak points in women. *Ann Vasc Surg* 2005;19:284-8.

18. Leal Monedero J. Indicaciones y tratamiento del síndrome de congestiónpélvica. *Rev de Flebología y Linfol. LecturasVasculares*2010;14:841-7.

19. Dándolo M. Tratamiento Endovascular del Síndrome de Congestiónpélvica y SíndromesCompresivos. *Relatooficial 2da parteRevista Argentina de Cirugía Cardiovascular*. 2017. Vol XV; 2: 122-30.

20. Whiteley M, Lewis-Shiell C, Bishop S. Pelvic vein embolization of gonadal and internal iliac veins can be performed safely and with good technical results in an ambulatory vein clinic, under local anaesthetic alone. Results from two year experience. *Phlebology* 2018;33(8):575-9.

21. White JV, Schwartz LB, Ryjewski C. Management of pelvic congestion syndrome and perineal varicosities. 685-696 in *Guidelines of the American Venous Forum 2017, Handbook of Venous and Lymphatic Disorders*. 4th Edition. CRC Press Taylor & Francis Group

4.3.2 Computed axial tomography scan and magnetic resonance imaging

Authors: *Thiago Vasconcellos, Carolina Chacon, Pamela Inés Causa Andrieu, Noelia Napoli*

4.3.2.1 Indications

Imaging studies have diagnostic value in patients with suspected of. Not all patients with venous dilatation are symptomatic,¹ the history of pregnancy and vaginal delivery is often an example of this situation, that does not cause any symptoms or require treatment. Computed axial tomography scan (CAT scan) and magnetic resonance imaging (MRI) are non-invasive imaging modalities that are used to identify venous dilatations, look for signs of venous reflux and, eventually, determine the compromised territory and presence of venous compression/obstruction, diagnose vascular or non-vascular conditions that cause the syndrome, and rule out other causes of chronic pelvic pain or establish differential diagnoses.

4.3.2.2 Technique

Both imaging modalities obtain a number of images that can be reconstructed in both 2D and 3D. The CAT scan uses radiation (the amount of radiation used can be controlled using *radiation modulation techniques* provided by state-of-the-art scanners) and IV iodinated contrast with potential nephrotoxic risk. Therefore, it is important to assess the kidney function before performing this examination. The MRI uses radiofrequency pulses in a given magnetic field plus IV gadolinium as contrast agent without the risk of allergic reaction to iodine. Eventually, the MRI can reveal pelvic venous dilatation with better resolution compared to the CAT scan, without IV contrast, and characterize other causes of chronic pelvic pain (endometriosis, adenomyosis, myomas). In both imaging modalities the patient remains in the supine position during the examination and the varicose veins can be less prominent.

The study covers the abdomen (from the renal veins) and the pelvis in order to characterize different drainage paths and possible compression causes.

Dynamic assessments with IV contrast are required to obtain images in the arterial (to see venous reflux and assess eventual vascular arterial causes) and the venous phase (venography, to measure vascular diameters, assess the venous territory compromised, and identify the venous vascular obstruction). Acquisitions in apnea without inspiration are advised here (to avoid changing the aortomesenteric angle, which could eventually alter the correct interpretation).

4.3.2.3 How to request the study

The request these studies we should specify the type of examination, *abdominal and pelvic venous angiography with IV contrast*, the imaging modality required, whether *tomography or magnetic resonance imaging* and the presumed diagnosis so that the radiologist can select the study protocol correctly, for example: chronic pelvic pain, suspected pelvic venous congestion. The study of the lower limbs is associated with the clinical context and it is necessary to specify it in the medical request as an additional region to be assessed.

4.3.2.4 Results

The magnetic resonance venography (MRV) seems to have good diagnostic precision compared to convention-

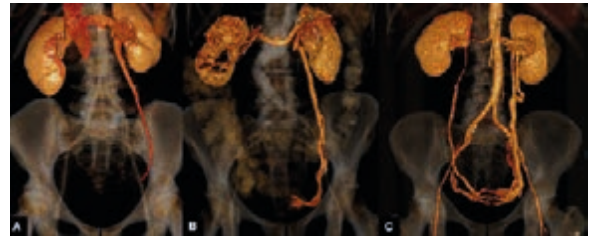


Figure 24. Grades of gonadal vein reflux. A) Grade I: left ovarian vein opacification. B) Grade II: retrograde flow advanced towards the left parauterine veins. C) Grade III: retrograde flow crosses the midline through the uterus towards the right parauterine plexus.

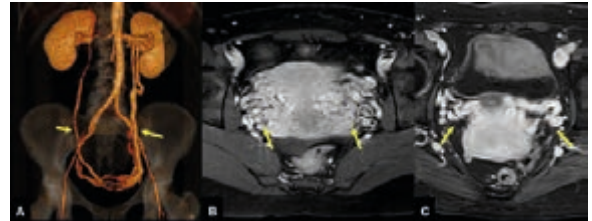


Figure 25. Pelvic congestion syndrome. A) 3D reconstruction, abdominal and pelvic coronary computed tomography angiography (CCTA), reflux of contrast material towards the left gonadal vein crossing the midline through the periuterine venous plexus until reaching the right gonadal vein. B) and C) Two examples of periuterine venous plexus dilatation.

al selective venography, but the evidence available on this regard is limited as there are very few good quality studies (Evidence level II-1: controlled non-randomized clinical trials), which is indicative of an 88% sensitivity rate and a 67% specificity rate for the identification of gonadal vein incompetence (2,3).

CAT scan venography does not have proper correlation studies with conventional venography (Evidence level II-3 / III: multiples series compared in time, descriptive studies) (3).

The main pathophysiological mechanism of PCS is the valvular insufficiency of gonadal veins, and it basically affects adult, multiparous women. The findings suggestive of a diagnosis of PCS in the CAT scan and the MRI are:

- **Gonadal venous reflux:** visualization in the arterial phase of the gonadal vein.
 - **Grade I:** left ovarian vein opacification (it does not reach the parauterine veins).
 - **Grade II:** retrograde flow advanced towards the left parauterine veins.
 - **Grade III:** retrograde flow crosses the midline through the uterus towards the right parauterine plexus (2-5) (**Figure 24**).
- **Gonadal veins:** >8 mm in diameter (6-9). They are measured using IV contrast in the venous phase, where the vascular diameter is more visible. We suggest taking the transversal diameter on the coronal plane (**Figure 25**).
- **Parauterine veins:** 4 or more tortuous homolateral veins with one of them > 5 mm (6-9) (**Figure 25**).

We suggest considering and assessing the presence of perivaginal, periurethral, presacral, venous plexus dilatation, drainage towards the hypogastric territory, and venous leak point assessment.

Rare pathophysiological mechanisms associated with extrinsic venous compressions affect a generally younger, nulliparous population, and they can be suitably assessed

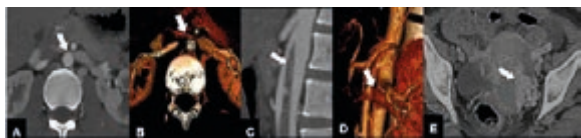


Figure 26. Nutcracker Syndrome. Left renal vein (arrows in images A) to D) compressed between the aorta and the superior mesenteric artery as seen on the tomography with IV contrast in abdomen and pelvis. A) and C) MIP reconstruction. B) and D) 3D reconstruction. E) Dilatated periuterine venous plexus (arrows).



Figure 28. Pelvic congestion syndrome. Pre-embolization: para-uterine veins dilatation as seen on the MRI. A) Dilatated left gonadal vein as seen on the angio-MRI (B) and CTCA (C). Post-embolization: reduction in the number and caliber of para-uterine veins (D) and absent left gonadal as seen on the CTCA (E), embolization material as seen on the CAT scan (F).

using these imaging modalities, such as the nutcracker syndrome (**Figure 26**), where there is left renal vein compression between the aorta and the superior mesenteric artery; we should see whether there is a reduced aortomesenteric angle (normal $>25^\circ$) and aortomesenteric distance (normal >10 mm).

In the May-Thurner syndrome (**Figure 27**) we find the left primitive iliac vein compression exerted by the right primitive iliac artery; other compression causes can be associated with a large uterus (due to the presence of myomas or adenomyosis).

The following is a list of the aspects that we should study in the images of patients with suspected PCS (8)

In the abdomen:

- Left renal vein (LRV): the nutcracker syndrome.
- Left primitive iliac vein: the May-Thurner syndrome.
- Gonadal veins: normal anatomy (right gonadal vein draining into the IVC and left gonadal vein draining into the LRV). Diameter >8 mm. Retrograde flow.

In the pelvis:

- Pelvic varicose veins: tortuosity/dilatation (diameter >5 mm)/amount.
- Involvement of internal iliac veins.
- Varicose veins of atypical location (examine the leak points: inguinal, gluteal, obturator, etc., and their association with pelvic varicose veins and PCS).

During imaging monitorization that follows the endovascular treatment (**Figure 28**) it is particularly advisable to assess the clinical signs and identify the possible redistribution of venous drainage into other territories (eg, the hypogastric region). Both imaging modalities are useful for monitoring purposes, there may be an artifact associated with the embolization material when using MRI images.

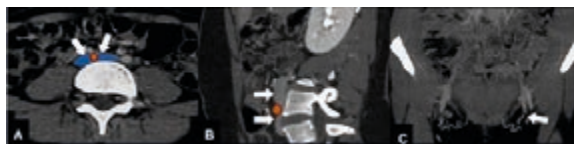


Figure 27. May Thurner syndrome. Tomography with IV contrast of abdomen and pelvis. A) and B) Left primitive iliac vein compression between the right primitive iliac artery and the spine. C) Bilateral inguinal point of pelvic venous leak.

4.3.2.5 Summary

Imaging studies have diagnostic value in patients *with suspected PCS*. Both imaging modalities, the CAT scan and the MRI, complement the Doppler echocardiography (the modality of choice) and have the proper diagnostic precision to be able to diagnose PCS; the latter recommendation has a II-1 level of evidence for the MRI (controlled non-randomized clinical trials in association with the conventional selective venography) and a II-3/III level of evidence for the CAT scan (multiple series compared across time, descriptive studies). Both imaging modalities are adequate for monitoring purposes of the therapy administered; also, the MRI is a useful imaging modality to diagnose other causes of non-chronic pelvic pain (eg, endometriosis, adenomyosis, myomas). Finally, we always recommend examining the patient's clinical signs and working together with the physician who requested the study for a correct interpretation of the imaging modality at stake.

4.3.2.6 Recommendations (10)

1	To complement the early CDE study, we recommend using the MRI and the CAT scan for the diagnosis of suspected PCS, monitor the PCS therapy administered, and for the differential diagnosis of CPP. Level of Evidence B. Class I recommendation.
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4.3.2.7 References

1. Nascimento AB, Mitchell DG, Holland G. Ovarian veins: magnetic resonance imaging findings in an asymptomatic population. *J Magn Reson Imaging* 2002; 15: 551-556.
2. Asciutto G, Mumme A, Marpey J, et al. MR venography in the detection of pelvic venous congestion. *Eur J Vasc Endovasc Surg* 2008;36:491-6.
3. Champaneria R, Shah L, Moss J, Gupta JK, Birch J, Middleton LJ, et al. The relationship between pelvic vein incompetence and chronic pelvic pain in women: systematic reviews of diagnosis and treatment effectiveness. *Health Technol Assess* 2016;20(5).
4. Kim CY, Miller MJ Jr, Merkle EM. Time-resolved MR angiography as a useful sequence for assessment of ovarian vein reflux: *Am J Roentgenol* 2009; 193: W- 458-W463.
5. Yang DM, Kim HC, Nam DH, et al. Time-resolved MR angiography for detecting and grading ovarian venous reflux: comparison with conventional venography. *Br J Radiol* 2012; 85: e117-e122.
6. Dick EA, Burnett C, Anstee A, et al. Time-resolved imaging of contrast kinetics three-dimensional(3D)magnetic resonance venography in patients with pelvic congestion syndrome. *Br J Radiol* 2010; 83: 882-887.
7. Coakley FV, Varghese SL, Hricak H. CT and MRI of pelvic varicose veins in women. *J Comput Assist Tomogr* 1999; 23: 429-434.
8. Arnoldussen CWKP, de Wolf MAF, WittensCHA. Diagnostic imaging of pelvic congestive syndrome. *Phlebology* 2015, Vol. 30(15) 67-72.
9. Antignani P-L, Lazarashvili Z, Monedero JL, Ezpeleta SZ, Whiteley MS, Khilnani NM, et al. Diagnosis and treatment of pelvic congestion syndrome: UIP consensus document. *Int Angiol*. 2019 Aug;38(4):265-83.
10. White JV, Schwartz LB, Ryjewski C. Management of pelvic congestion syndrome and perineal varicosities.: 685-696 in *Guidelines of the American Venous Forum 2017, Handbook of Venous and Lymphatic Disorders*. 4th Edition. CRC Press Taylor & Francis Group

4.3.3 Dynamic gonadal and iliac phlebography

Authors: *Damián Simonelli, and Guillermo Eisele*

4.3.3.1 Introduction

The pelvic congestion syndrome (PCS) is part of an even greater syndrome called chronic pelvic pain (CPP) described by Richet back for the first time back in 1857 (1).

Although the progress made in non-invasive diagnostic imaging modalities (CDE, CAT scan, and MRI) facilitates the diagnosis of PCS with satisfactory sensitivity and specificity rates, the gonadal and iliac dynamic phlebography (GIDP) is still considered essential for the diagnostic stage in the presence of inconclusive findings of the non-invasive diagnosis of PCS and in the assessment prior to endovascular treatment.

Technical aspects will be detailed in this chapter as well as the importance of GIDP in the venous catheterization of PCS.

4.3.3.2 Anatomy

Pelvic organs drain into their venous flow both to right and left through the large internal left and right iliac veins.

These veins meet in the pelvic wall with the external iliac vein, continuation of the femoral vein, making up the primitive (right and left) iliac vein; the convergence of these primitive iliac veins at the level of the right anterolateral side of the body of the fifth lumbar vertebra make up the beginning of the inferior vena cava (IVC).

Another drainage path of venous flow is both gonadal veins which, by deriving the blood flow coming from the ovaries and the homolateral Fallopian tube, ascend parallel to the spine to drain directly into the IVC from the right gonadal vein and into the left renal vein (LRV) in the case of the homolateral gonadal vein.

Didactically speaking, if we divide pelvic veins into 2 groups, on the one hand we will find veins running through the pelvic wall. On the other hand, we will find veins coming from the pelvic organs, called parietal and visceral, respectively. Among the *parietal* veins we can identify the gluteal, sciatic, pudendal, obturator, and lateral sacral veins. The first 4 branches mentioned have a high capacity of showing anastomosis with LL veins. Therefore, they are indicative points of probable flow reversal for the outflow of venous content from the pelvis called pelvic leaks.

In the case of *gluteal veins*, anastomoses will occur on the gluteus medius and gluteus maximus inferior lines and edges, and they will be noticeable on the thigh posterior side. *Obturator veins* can anastomize with the inner thigh branches. *Internal pudendal veins* can anastomize with the external pudendal veins through perivulvar and sciatic venous branches with deep femoral vein perforator branches in the LL thighs (2,3).

On the other hand, *lateral sacral veins* can often show an important number of anastomoses with contralateral lateral sacral veins, which is why this is a very effective path to derive flow from left to right or vice versa. Congenitally speaking, unlike gonadal and iliac veins, sacral veins are valveless.

Perivesical plexus is among the *visceral* veins, which in its lateral portion drains into the vesico-prostatic plexus and



Figure 29. Left iliac vein anatomical variants. Double left iliac vein. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

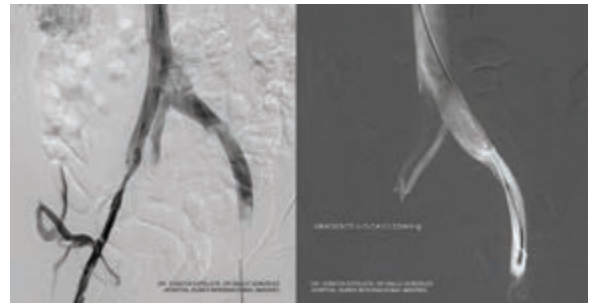


Figure 30. Left iliac vein anatomical variants. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

it can also drain through numerous branches of the internal iliac vein (4).

The medial *hemorrhoidal veins* run along the sides of the rectum making up a plexus that ends in 1 or 2 bundles that eventually drain into the internal iliac vein. They run across the sacro-lateral ligaments, contrary to the superior hemorrhoidal veins (branches of the portal system).

Vaginal and uterine veins drain into the internal iliac vein through bundles located at the wide ligament (5) (**Graphic 1**).

We can have a single (in 50% of the patients) or a duplicated internal iliac vein or it can have a venous plexus presentation (6). According to Champaneria, the incidence rate of internal iliac vein duplication or its plexus presentation is 30% (7). In a study of 42 cadavers, Le Page determined that the internal iliac vein was present as a single trunk in 73% of the cases, and as 2 trunks in 27%. Another interesting fact revealed by this study is the presence of valves in the external iliac vein in 26% of the cases, and the presence of valves in this vein was 3 times more common on the right compared to the left side (4).

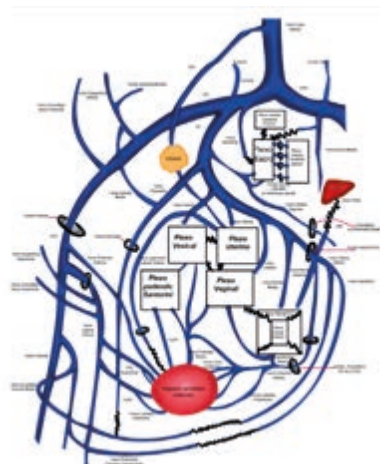
The main venous trunks and their ostia are subject to multiple anatomical presentations that vary regarding their location, duplication, etc. (**Figures 29 and 30**).

All of these are valved veins, except for the diseased ones that generate leak points and presacral plexus.

4.3.3.3 Gonadal and iliac dynamic phlebography

Overview

GIDP is the most sensitive and specific diagnostic imaging modality (80% to 100%) and it is considered the *gold standard* in the diagnostic tests of PCS. 8-10 This minimally invasive study provides a highly precise dynamic venous assess-



Graphic 1. Venous anatomy of the pelvis. Adapted from: David Kachlik et al. The Venous System of the pelvis: New Nomenclature. *Phlebology* 2010;25:162- 73.

ment, confirms the presence of obstructive lesions, and facilitates the endovascular therapeutics guidance. During the same procedure it is possible to perform both the diagnosis and the therapeutics. With a sensitivity similar to transvaginal (TV) CDE, it provides a graphic representation of the affluent and effluent varicose trajectories. To a large extent pelvic phlebography is based on performing the technique correctly: it is necessary to attempt every distal catheterizations in the internal iliac veins tributary branches and inject the contrast agent while using the Valsalva maneuver to properly visualize the pelvic leaks.

Both the CCTA (coronary computed tomography angiography) and the angio-MRI are highly sensitive and specific imaging modalities to study PCS and compression and/or occlusive syndromes. Still, they need accurate measurements of venous times in the contrasted series and avoid artifacts that can degrade the diagnostic quality significantly. However, it does not quite work like this in leak point or reflux assessments where the selective and distal GIDP is obviously more effective.

The invasive character of GIDP as a diagnostic imaging modality is justified by the fact that within the same procedure it is possible to continue with the endovascular treatment.

Technique

Before performing a GIDP, the operator should already be familiar with the patient's clinical signs and findings obtained from non-invasive studies.

This study can be performed outpatiently under local anesthesia or, if required, adjuvant neuroleptoanalgesia. We should remember that, for a proper dynamic phlebography assessment, the patient should cooperate and remain lucid. In addition to the informed consent, we teach patients to perform the maneuvers of deep inspiration accurately (Valsalva and apnea). General anesthesia is exceptional and spared for certain clinical or mood conditions of the patient.

The GIDP prior assessment includes lab tests with coagulogram and kidney function tests since the average iodinated contrast volume injected is 120 ml.

With the patient in the dorsal decubitus position and after the administration of proper antiseptics, venous access is often obtained through an ultrasound-guid-



Figure 31. Vascular radiology ward. Access through the arm basilic vein facilitates the use of radiation protection screen. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

ed puncture or with anatomical landmarks by inserting a 5-Fr-or-6-Fr vascular guidewire using the Seldinger technique.

Vascular access can be obtained via femoral or internal jugular access route, although jugular and/or brachial accesses are more comfortable and easier to perform regarding the study of both sides of the pelvis (**Figure 31**).

Access via jugular vein makes it difficult to use the radiation protection screen (**Figure 31**) and, statistically speaking, it is associated with more complications (pneumothorax, inadvertent puncture of the carotid artery).

In most cases some authors often use the arm basilic vein. Also, we always shave and administer antiseptics on the right groin in case right femoral vein access is required when the retroaortic access has gone unnoticed on previous tests or when the right gonadal vein catheterization is impossible from the superior vena cava. Occasionally, it is more accessible via femoral vein for catheterization purposes with the Simmons 1 catheter using a distal microcatheter to administer microcoils therapy or see the right gonadal vein ostium. Also, a multipurpose catheter from the brachial vein is often used for catheterization purposes.

After vascular access is obtained, the purpose of the study is to obtain dynamic images of the 4 main venous axes of pelvic drainage.

In the early studies of hypogastric veins and their eventual leaks it would not be necessary to use a vesical probe; on the contrary, if the diagnostic study starts with gonadal and LRV assessment or diagnosis and endovascular treatment anticipated within the same procedure, vesical probing should come before the GIDP (11,12).

The **GIDP protocol** of some authors consists of studying the left renal vein and the left iliac vein to rule out any possible compression syndromes. Then, move on to study the right gonadal vein because its access is more complicated. Afterwards, the left gonadal vein should be studied since the LRV has already given us an idea of the morphology that we will eventually find. Lastly, we should study the internal iliac veins to see any possible refluxes (13). Vesical probing is recommended since diagnostic procedures are always complemented by a TE therapeutic procedure.

If the catheterization of the right gonadal vein with the multipurpose catheter is not possible, we should move on



Figure 32. 5-Fr 125 cm long multipurpose catheter. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

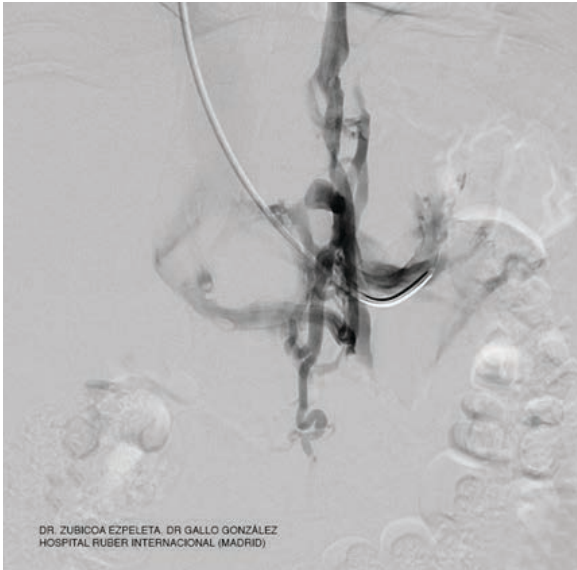


Figure 34. LRV phlebography showing NCS with paravertebral and paralumbar collaterality and possible double LRV. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

with the next step in the work protocol, thus studying the remaining veins. Once the study has been completed, the catheter should be changed to access the right gonadal vein.

Using a 5-Fr 100 cm or 125 cm-long vascular preformed multipurpose catheter with distal orifice (**Figure 32**) and under the protection of a Terumo hydrophilic guidewire (curved tip, 0.035 in, 150 cm-to-180 cm long) (**Figure 33**), the goal was to catheterize and draw a sketch of both renal veins, the gonadal veins, the primitive and hypogastric iliac veins, the femoral veins, and their most significant bundles. With the current material, it is not usually necessary to use long introducer sheaths.

Contrast injections are performed manually with 10 mL-syringes using pure idonated contrast or at 20% dilution.

Ideally, the best thing to do is to use low radiosopic doses and radiosopic registers to reduce secondary radiation. A full study should not exceed 50 mGy.

For the registration of series in digital subtraction, cadence does not require more than 2 images per second. Ideally, the panoramic fields also minimize the need for multiple focal registrations of small fields. To improve the quality of the images, compression syndromes are studied in apnea to obtain more precise assessments of the compression notches and the collateral circulation.

Left renal phlebography has 2 different purposes: to assess renal vein stenosis and the presence of venous reflux towards the homolateral gonadal vein. Its other objective

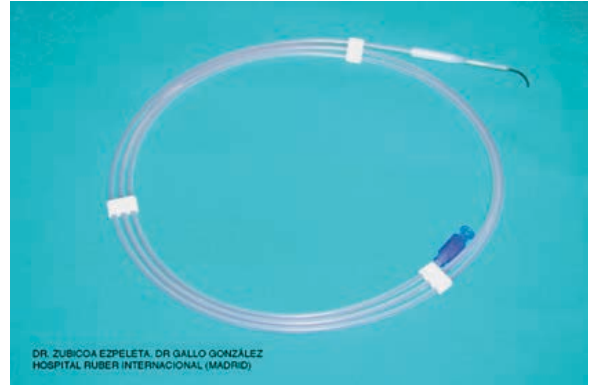


Figure 33. 0.035 in 150-180 cm-long Terumo hydrophilic guidewire with curved tip. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

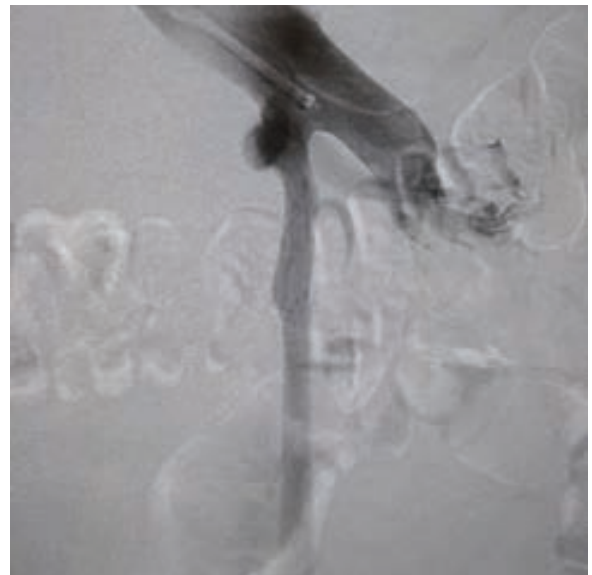


Figure 35. Left renal phlebography with spontaneous reflux towards left gonadal vein.

is to assess the possible presence of other collateral branches that may develop in compression syndromes (perirenal, paralumbar, paravertebral, and pararenal) (**Figure 34**).

We shall begin by positioning the catheter in the LRV trunk and performing a renal phlebography with and without the Valsalva maneuver (6) (**Figure 35**).

In the cases of NCS, stenosis of the LRV trunk can be confirmed (6). Afterwards, the hemodynamic study will be completed with the measurement of venous pressures through the stenosis, first looking into proving the existence of a pressure gradient >3 mmHg according to Greiner (6) or else ≥ 5 mmHg according to Monedero (13) between the proximal and distal portions to the stenosis. Pressure should always be measured (**Figure 36**), not only in case of NCS.

Secondly, we will try to catheterize the compression of the LRV. The most common one is to *reduce the aorto-mesenteric clamp (AMC) angle*. However, the *retroaortic renal vein* runs behind the aorta in its way towards the IVC, which also generates anterior compression (much less common); the third option is to have a *circumaortic renal vein* to reduce the caliber in the anterior and posterior passage towards the aorta (even less common).



Figure 36. Pressure monitor. Courtesy of Dr. ZubicoaEzpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).



Figure 38. Left renal phlebography showing a renal vein anatomical variant (atypical renal vein). Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

These anatomical variants open the possibility of compressing the LRV in the aortomesenteric space or between the spine and the abdominal aorta, thus causing syndromes (NCS) with a lower back pain component, possible hematuria, and syndromes derived from venous collateral circulation while trying to decompress renal venous drainage, which can originate PCS or left testicle varicocele when flow is diverted by the gonadal vein through reflux with flow reversal.

The gonadal vein develops PCS usually in thin women who could even be nulliparous. As a matter of fact, in these cases, we can see how the right gonadal vein has a normal morphology as opposed to the left one that appears clearly dilated and valveless.

The right renal vein often has a shorter trajectory and is also more angled with compared to the vena cava. Its possible variants do not have so much repercussion regarding pelvic venous conditions. Although, at times, we can say that the right gonadal vein drains directly into the renal one, some other times the gonadal branch drains into the renal vein, often accessory, of smaller caliber, thus commu-

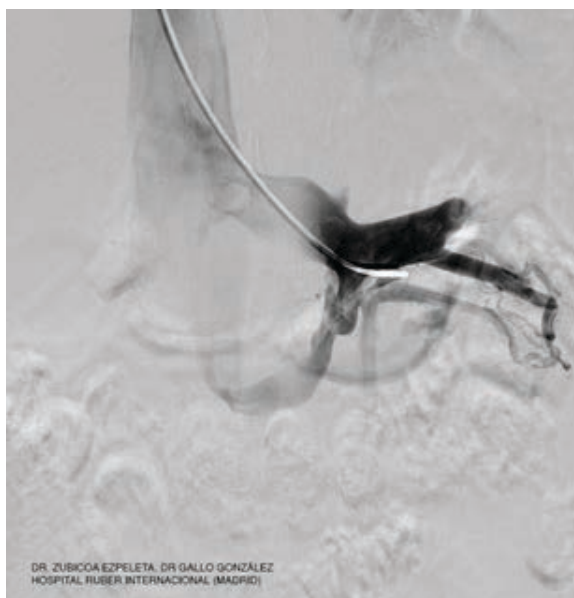


Figure 37. Phlebography showing a LRV anatomical variant (double renal vein). Courtesy of Dr. Zubicoa Ezpeleta Dr. Gallo González. Ruber International Hospital (Madrid, Spain).



Figure 39. Right gonadal vein phlebography. Anatomical variant, entrance in right renal vein. Courtesy of Dr. Zubicoa Ezpeleta Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

nicating directly with the gonadal vein. Also, there are times that the right gonadal vein drains into the angle formed between the right renal vein and the inferior vena cava (**Figures 37, 38, and 39**).

Another aspect that should be characterized during renal phlebography is the presence of stenosis-related thrombosis and/or myofibrotic bands due to the myointimal aggression generated in the venous wall by the superior mesenteric artery arterial beat.

Performing a LRV phlebography with the Valsalva maneuver also gives us the possibility to diagnose left gonadal vein reflux. Although with this maneuver its presence is a conclusive sign of gonadal vein incompetence, its absence does not rule out the diagnosis. The explanation is that the GIDP is usually performed in the decubitus position since very few

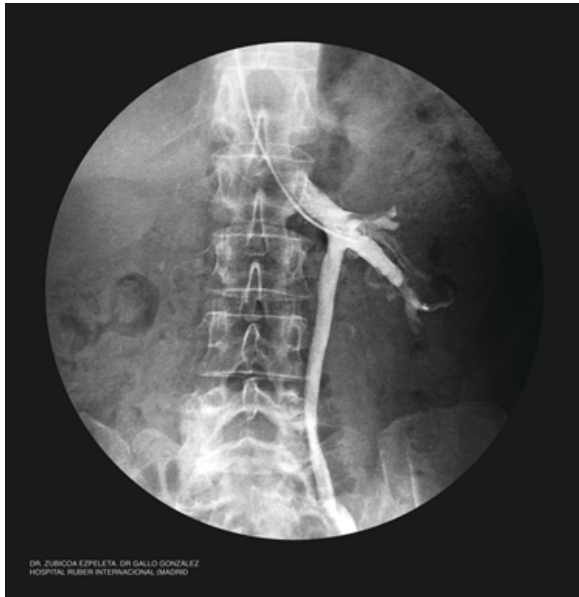


Figure 40. LRV phlebography performed in a digital remote control with possibility of maneuvering while in the standing position. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).



Figura 41. Development of collateral perivertebral veins of left gonadal vein.

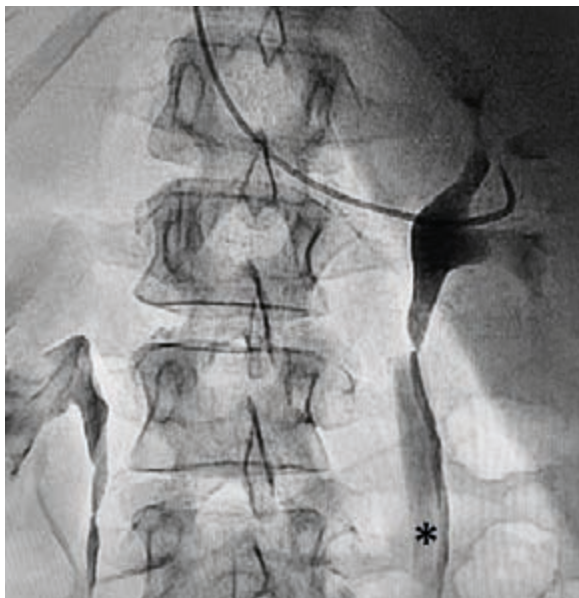


Figure 42. 30-second contrast retention in left gonadal vein after the injection of contrast (*).



Figure 43. Pelvic flow diversion towards the contralateral side.

radiology machines at the cath lab have remote control tables capable of achieving the supine position. We do not know of any publications that show better sensitivity or specificity rates compared to phlebography in the supine position (Figure 40).

Left gonadal phlebography should be completed with its selective catheterization and opacification in order to specifically look for the following data:

- **Diameter of the gonadal vein.** Most authors estimate that a diameter of up to 5 mm is normal: mild incompetence (between 5 mm and 8 mm); major incompetence (between 8 mm and 10 mm); and severe incompetence with diameters >10 mm. Still, we should mention that the association between the diameter of the gonadal vein and the presence of reflux is not always so linear, as Dos Santos confirmed in his study conducted back in 2014 (14).

- **Development of high paravertebral collaterals diverting renal flow towards the paravertebral plexus.** This can be considered an indirect sign of misdiagnosed LRV stenosis or thrombosis. Still, the LRV thrombosis associated with the NCS is rare.
- **Number and height of bifurcations.** The gonadal vein commonly undergoes divisions at different heights that should be known on the GIDP since this can lead to relapses in all of its branches if not properly embolized (6).
- **Presence of contrast retained for 30 seconds after the injection of contrast,** which is strongly indicative of the presence of gonadal reflux and stasis.
- **Flow reversal:** the GIDP allows us to confirm incompetent gonadal drainage both spontaneously and thanks to the Valsalva maneuver. This incompetence can be so significant that it determines left gonadal flow diversion

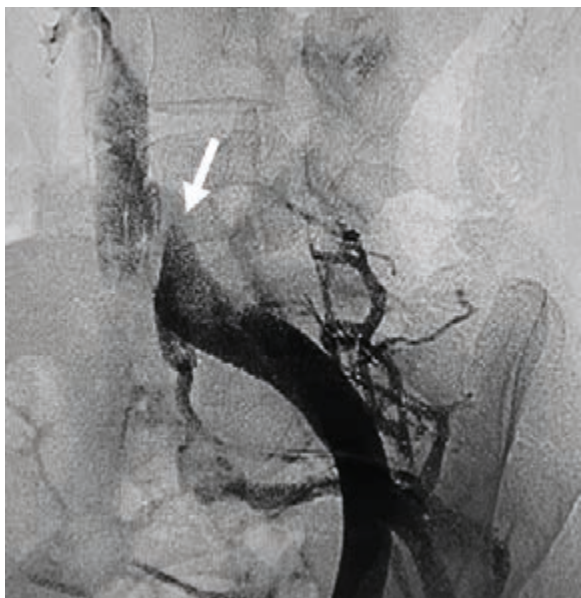


Figure 44. May-Thurner Syndrome: compression of the arterial segment on the left primitive iliac vein (white arrow).

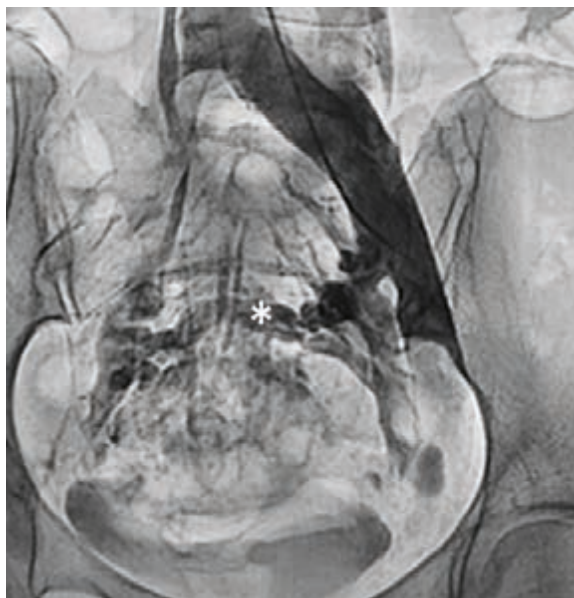


Figure 45. Left hypogastric vein incompetence (*).

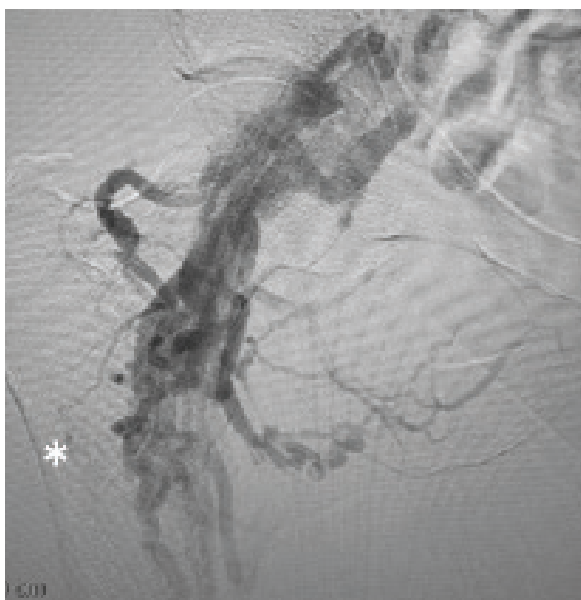


Figure 46. Right inferior gluteal vein incompetence, sciatic varicose veins (*), and superficial venous system varicose veins.

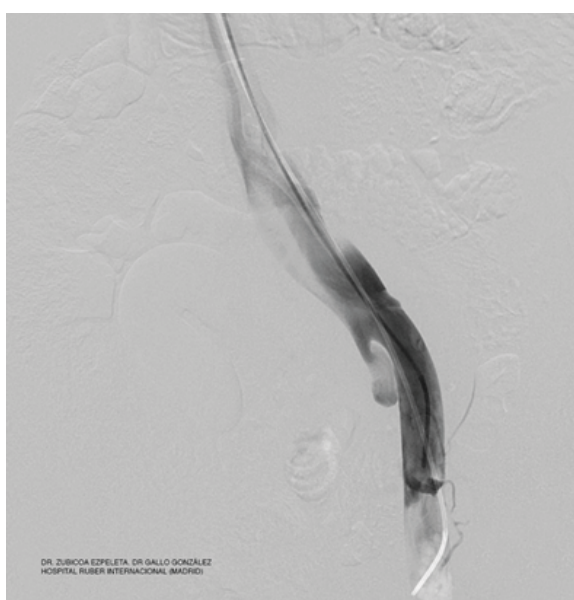


Figure 47. Left iliac vein phlebography showing the typical valveless condition of the left internal iliac vein. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

towards the contralateral side through the pelvic veins (lateral sacral) or towards the internal contra- or homolateral iliac vein. (Figures 41, 42, and 43).

Right gonadal phlebography looks for the same findings as it happens with the left side. However, we should mention that, if not dilated, its selective catheterization can be more difficult compared to the left side.

Iliac phlebography consists of catheterizing both the iliac and femoral veins by performing bilateral ascending phlebographies with and without the Valsalva maneuver (Figures 44, 45, and 46).

The study of the left iliac vein is often performed by injecting contrast into the external iliac vein proximal to the common femoral vein to obtain an ascending phlebography while in apnea and analyze the possibilities of compression syndro-

mes, May-Thurner Syndrome (MTS) or other abnormalities. Afterwards, a selective study of both internal iliac veins and their anatomical variants is conducted with the C-arm in counterclockwise rotation (25°) and using the Valsalva maneuver to assess the possibilities of reflux or leak through the tributary veins.

In their origin the internal iliac veins are valveless and, as we have already said, so are the presacral veins. We do find valves in the internal iliac vein tributary branches except for those with incompetence consistent with pelvic leaks. In our work methodology we always perform a proximal injection that works as a general map for the selective distal catheterization of incompetent veins (Figure 47).

It is advisable to start on the left side since the disease is more prevalent here. In these GIDPs we will try to rule out:

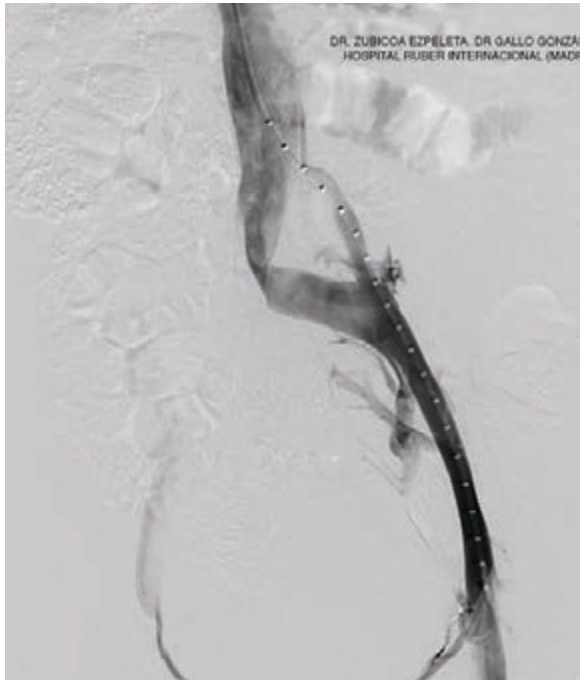


Figure 48. Left iliac vein phlebography showing a central synechia associated with MTS. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).



Figure 49. In-stent left iliac vein dilatation with balloon showing a somewhat hourglass vein morphology created by central synechia, and venous walls fibrous adhesions. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

- A.** Significant stenosis of the primitive iliac vein. In case of MTS, the disease is significantly more prevalent on the left side and the stenosis corresponds to the primitive iliac artery crossing on the left primitive iliac vein trunk anterior side. Iliac venous thrombosis can be associated. The development of collaterals is also feasible (in general they are ilio-lumbar, ascending lumbar, and perivertebral). According to Neglen, there is not such a thing as a standard pattern to determine the significance of a MTS venous obstruction, and although small transtenotic pressure gradient differences (from 2 mmHg to 5 mmHg) can be hemodynamically significant, normal gradient measurements do not rule out the presence of critical venous obstruction. Therefore, the measurement of transtenotic pressures should be taken into consideration together with phlebographic, CDE, CAT scan, MRI images, and clinical signs. Arbitrarily, the intravascular ultrasound (IVUS) is capable of showing superior iliac vein stenoses > 50%. It may very well be the most precise imaging modality to define significant stenosis eligible for treatment with angioplasty and *stent* therapy (15). We were able to confirm that thrombosis associated with the iliac stenosis of the MTS amplifies the existing obstructive symptoms. In cases of right MTS, the right primitive iliac vein is the compressed vein, and stenosis is often more longitudinal (not so cross-sectional) and it can expand quite a few centimeters from the proximity of the iliac confluent to the caudal. When the arteries are more elongated, we may find compressions on the right side too. We believe that there is MTS when the right iliac artery compresses the left iliac vein.
- B.** The GIDP of the MTS can also show membranes of myointimal hyperplasia caused by the vascular damage due to the arterial beat on the venous wall in the form of longitudinal negative images (without flow) of different

lengths and without a precise distribution pattern. They are also called central synechiae, fibrous adhesions between the venous walls. When treatment is performed, the vein hourglass morphology can be seen directly when balloon dilatation is performed. In the presence of thrombosis, manipulation can be risky (**Figures 48 and 49**).

These synechiae are often associated with occlusive thromboses already diagnosed on the CDE, CAT scan or MRI (**Figure 50**).

There are times that the primitive iliac vein catheterization via jugulo-brachial access may become difficult. If so, this should inform us of the presence of severe stenosis that should eventually be studied from the corresponding LL.

- C.** Also, we should not rule out the possibility that a stenosis, thrombosis or anomaly reported on the IVC complicates the iliac veins catheterization. In general, in these cases, non-invasive diagnostic studies usually inform us on this situation, meaning that the patient should be studied from the LL.
- D.** Stenoses following extrinsic, asymmetrical and non-circumferential compressions with smooth edges and obtuse angles can be due to adenopathies. They should be distinguished from tissue invasion/infiltration of the venous wall as stenoses show irregular surfaces, acute or variable angles, and heterogeneous images. As in the previous point made, the patients' clinical data and non-invasive studies should inform us on this diagnostic possibility.
- E.** It is important to confirm the presence of venous reflux towards the pelvis through the internal iliac veins, which can be the case because 80% of them are valveless. Unlike their tributary branches, internal iliac (and presacral) vein *ostia* are valveless.



Figure 50. Difficulty trying to catheterize the left iliac vein due to occlusive thrombosis. Use of Simmons 1 catheter from the right femoral vein. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. International Ruber Hospital (Madrid, Spain).

F. Also, we should confirm whether there is flow diversion towards the contralateral side through different pelvic plexuses and whether after contrast injection some contrast remain for over 30 seconds in the pelvic veins (unequivocal sign of PCS). If so, this would be associated with postural mechanisms.

The last stage of the GIDP consists of the **phlebography of both internal iliac veins and their branches**. This part of the study is usually time-consuming, requires training, and the proper anatomical knowledge. In order to optimize the time that it takes to perform the study, the use of iodinated contrast and radiation exposure should be minimized, and the targets of this study should be crystal clear. Dr Zubicoa Ezpeleta's study protocol includes the assessment of the possible compression syndromes first. Afterwards, the study of gonadal veins, and finally the internal iliac veins selective catheterization. Clinical assessments and non-invasive studies should have already informed us on the presence of pelvic, vulvar, gluteal and/or LL varicose veins and suspect the presence of pelvic venous leak points (see section on anatomy). At this point, we should diagnose suprapelvic obstructive causes (NCS, MTS, thrombosis, hypoplasia, extrinsic compression of both the iliac vessels and the IVC), venous incompetence and reflux at both gonadal and iliac levels. Uterine veins and internal iliac vein branches can also be involved in the development of periuterine varicose veins of the PCS. Therefore, in the study of internal iliac veins, added to the possible existence of leaks, we should rule out the presence of uterine branch incompetence and perform a TE procedure on this branch, when applicable (**Figure 23**). In conclusion, the GIDP performed on internal iliac veins and their branches should inform on the following findings:

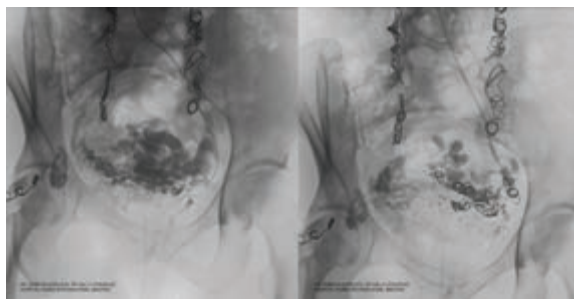


Figure 51. Uterine vein catheterization, a branch of the internal iliac vein. Embolization treatment. Courtesy of Dr. Zubicoa Ezpeleta and Dr. Gallo González. Ruber International Hospital (Madrid, Spain).

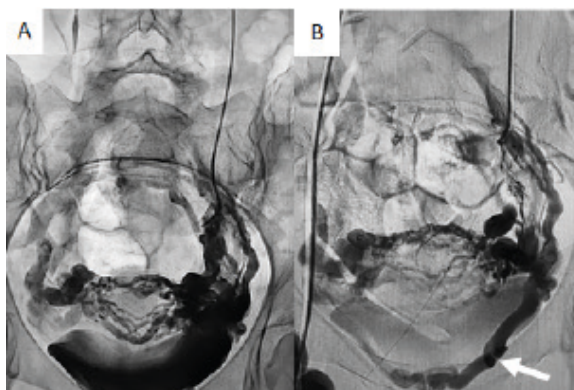


Figure 52. A) Left gonadal venous flow diversion towards the contralateral side. B) Presence of significant left suprapubic vein (white arrow).

- Presence of incompetent pelvic varicose veins in uterine veins and internal iliac vein branches (internal pudendal, obturator, etc.).
- Diagnose hypogastric vein anomalies (duplications, and/or anomalous drainages or trajectories).
- Map and quantify the significance of major venous leaks into the homo- or contralateral LL and establish which will eventually require endovascular treatment (**Figure 24**).

Although the multipurpose catheter facilitates the acquisition of highly precise images of the leaks without having to use balloon catheters, it can be useful to use high-flow catheters or perform injections with balloon catheters that will partially occlude the internal iliac vein lumen facilitating the detection of venous leaks.

The main PFL points that should be studied are the superior and inferior pudendal, gluteal, obturator, and round ligament veins. The round ligament vein is a gonadal vein branch, unlike the others, that are internal iliac vein branches.

The systematic study of these pelvic veins and their annexes will be crucial to confirm or rule out diagnoses and make the corresponding therapeutic decisions. It is important to take into account that the information provided by the GIDP cannot be isolated from the patient's clinical signs. Otherwise, it will just be an image and the results will not be the expected ones.

Results

Several studies have established the phlebographic findings that define patients with PCS and PVI based on morphological and hemodynamic parameters. Still, their scientific

quality is not good enough to confirm the sensitivity and specificity rates of GIDP. These findings are:

- Gonadal vein/s incompetence and dilatation.
- Utero-ovarian plexus varicose veins.
- Prolonged retention of contrast in the gonadal vein/s and the utero-ovarian plexus.
- Reflux from the utero-ovarian varicose veins towards the hypogastric and/or gonadal contralateral drainage.
- Reflux towards vulvo-vaginal varicose veins and/or LL varicose veins (16-18).

Back in 1984, Beard was the first one to describe a diagnostic score using pelvic phlebography via transuterine puncture by comparing patients with PCS symptoms to case-control studies whose findings had been extrapolated to the GIDP (9,12). This imaging modality has been totally abandoned today because it does not allow us to perform TE procedures when incompetence has been confirmed.

The 3 phlebographic criteria with which this score is used are:

- Larger diameter of the ovarian vein (<5 mm; 5 mm to 8 mm; or >8 mm).
- Delayed contrast washout times in the ovarian vein (to 0 s, 20 s, and 40 s).
- Significance of utero-ovarian varices (normal, moderate or severe).

Each criterion measures a severity level that goes from 1 to 3, and the sum of 5 or more points in the score is indicative of the presence of PCS with a 91% and 89% sensitivity and specificity rates, respectively (10-12).

In his systematic review, Champaneria assigns scores ≥5, and odds ratio = 31 to the GIDP (7).

Therefore, it seems clear that the GIDP plays a key role in 3 different situations to achieve a definitive diagnosis of PCS:

- When findings from non-invasive studies are inconclusive.
- When it is necessary to confirm the findings from non-invasive studies.
- When endovascular treatment has been proposed as a possible alternative (1).

For these reasons, and despite these limitations, the GIDP is still considered the gold standard (17) imaging modality for the diagnosis of PCS.

Note: both the images and phlebographies showed in this chapter are actual cases managed by the authors and reviewers of this consensus document.

4.3.3.4 Recommendations (20)

1	Use the GIDP: a. In inconclusive findings of non-invasive PCS studies. b. To confirm the findings of PCS in non-invasive studies. c. When proposing and planning the endovascular management of PCS it should be performed in a single-staged procedure, when applicable. (Level of Evidence B, Recommendation I).
2	To study patency, diameters, and IVC, common renal, gonadal, primitive iliac, external, hypogastric, femoral vein incompetence with and without the Valsalva maneuver. Venous collateral circuits should be assessed too.
3	Assign significance levels to the abnormal pressure gradient measurements of the left primitive renal and iliac veins with suspected CNS and MTS, respectively. In the presence of non-significant gradient measurement mismatch and suspected PCS, an IVUS should be performed.

4.3.3.5 References

1. Richet A. *Traité pratique d'anatomie médico-chirurgicale*. 1. -5. ed. Paris; Lauwereyns et al; 1857.

2. Beckett D, Dos Santos SJ, Dabbs EB, Shiangoli I, Price BA, Whiteley MS. Anatomical abnormalities of the pelvic venous system and their implications for endovascular management of pelvic venous reflux. *Phlebology* 2018;33(8):567-74.

3. Amore M, Casal F, Tapia L, Irolart JA. Bases anatómicas y fisiopatológicas del Síndrome Congestivo Pélvico. *Flebología y linfología, lecturas vasculares*. 2013;8(19):1184-96.

4. LePage PA, Villavicencio JL, Gomez ER, Sheridan MN, Rich NM. The valvular anatomy of the iliac venous system and its clinical implications. *J Vasc Surg* 1991;14(5):678-83.

5. Kachlik D, Pechacek V, Musil V, Baca V. The venous system of the pelvis: new nomenclature. *Phlebology* 2010;25(4):162-73.

6. Greiner M, Dadon M, Lemasle P, Cluzel P. How does the patho-physiology influence the treatment of pelvic congestion syndrome and is the result long-lasting? *Phlebology* 2012;27 Suppl 1:58-64.

7. Champaneria R, Shah L, Moss J, et al. The relationship between pelvic vein incompetence and chronic pelvic pain in women: systematic reviews of diagnosis and treatment effectiveness. *Health Technol Assess* 2016;20(5):1-108.

8. Rane N, Leyon JJ, Littlehales T, Ganeshan A, Crowe P, Uberoi R. Pelvic congestion syndrome. *Curr Probl Diagn Radiol* 2013;42(4):135-40.

9. Kies DD, Kim HS. Pelvic congestion syndrome: a review of current diagnostic and minimally invasive treatment modalities. *Phlebology* 2012;27 Suppl 1:52-7.

10. Herrera-Betancourt AL, Villegas-Echeverri JD, Lopez-Jaramillo JD, Lopez-Isaño JD, Estrada-Alvarez JM. Sensitivity and specificity of clinical findings for the diagnosis of pelvic congestion syndrome in women with chronic pelvic pain. *Phlebology* 2018;33(5):303-8.

11. Beard RW, Highman JH, Pearce S, Reginald PW. Diagnosis of pelvic varicosities in women with chronic pelvic pain. *Lancet* 1984;2(8409):946-9.

12. Chung MH, Huh CY. Comparison of treatments for pelvic congestion syndrome. *The Tohoku journal of experimental medicine*. 2003;201(3):131-8.

13. Monedero J. Indicaciones y tratamiento del síndrome de congestión pélvica. *Flebología y linfología- lecturas vasculares*. 2010;5(14):841-7.

14. Dos Santos SJ, Holdstock JM, Harrison CC, Lopez AJ, Whiteley MS. Ovarian Vein Diameter Cannot Be Used as an Indicator of Ovarian Venous Reflux. *Eur J Vasc Endovasc Surg* 2015;49(1):90-4.

15. Neglen P. Endovascular reconstruction for primary iliac vein obstruction. pp:523-32. In: *Handbook of venous and lymphatic disorders*. 4th edition. 2017, CRC Press. Taylor & Francis Group.

16. Ganeshan A, Upponi S, Hon L-Q, Uthappa MC, Warakaulle DR, Uberoi R. Chronic pain due to pelvic congestion syndrome: The role of diagnostic and interventional radiology. *Cardiovasc Interv Radiol* 2007;30:1105-11.

17. Ezpeleta Zubicoa S. Instrumental diagnosis of pelvic congestion syndrome. *Phlebolympology* 2016;23:121-64.

18. Tu FF, Hahn D, Steege JF. Pelvic congestion syndrome-associated pelvic pain: a systematic review of diagnosis and management. *Obstetrical & gynecological survey*. 2010;65(5):332-40.

19. Knuttinen M-G, Xie K, Jani A, Palumbo A, Carrillo T, Mar W. Pelvic Venous Insufficiency: Imaging Diagnosis, Treatment Approaches, and Therapeutic Issues. *AJR* 2015; 204:448-58.

20. White JV, Schwartz LB, Ryjewski C. Management of pelvic congestion syndrome and perineal varicosities. pp:685-96. In: *Guidelines of the American Venous Forum 2017, Handbook of Venous and Lymphatic Disorders*. 4th Edition. CRC Press Taylor & Francis Group.

Smoking as an independent predictor of radial spasm

Tabaquismo como predictor independiente de espasmo radial

Natalia Mercado¹, Mariano Rubio¹, Martín Cisneros¹, Santiago Trejo¹, Maximiliano Giraudo¹

ABSTRACT

Objective. To determine the independent influence of smoking on the appearance of radial artery spasm in cardiac catheterization studies and study the association of different conditions with the development of radial artery spasm.

Material and method. Prospective, cross-sectional study. A 3-month follow-up of patients undergoing cardiac catheterization in 2 centers of the province of Córdoba, Argentina analyzed the influence of the procedural variables associated and clinical variables such as sex, age, and risk factors in the appearance of radial artery spasm (RAS).

Results. Patients undergoing cardiac catheterizations via radial artery access between September and December 2019 in 2 centers in the city of Córdoba, Argentina were analyzed. A population of 347 patients was finally studied. A total of 131 were women (37.8%) and 216 were men (62.2%). The incidence rate of radial artery spasm (RAS) in the study sample was 14.7% (n=51). A significant association between smoking and this event was finally confirmed. In all cases, the incidence rate of RAS was significantly higher in smokers (17.6%) compared to non-smokers (6.5%). Smoking was a variable significantly associated with radial artery spasm ($P=0.020$).

Sex presented an OR associated with spasm ($OR=8.2$); the probability of women to present RAS would be 8 times greater compared to men. RAS occurred in almost a third of the women studied (29.0%) and in 6.0% of the males studied and was more commonly seen in smokers (33.3% of them suffered radial artery spasm). However, in non-smokers the incidence of spasm was also more common in women compared to men. Regarding procedural factors, it was observed that both the size of the catheter and the spasm kept a significant correlation ($P=.021$). Procedural time was significant ($P=.022$) with a higher risk of spasm associated with patients with procedural times > 20 min. ($OR=2.6$) and the type of study and occurrence of RAS ($P=.014$). The highest risk category was seen in the therapeutic study with an $OR=3.1$ (95%CI, 1.25-7.67).

Conclusions. The incidence rate of radial artery spasm was close to 15% in the study sample, which kept a positive correlation with female sex and smoking. Smoking was confirmed as an independent risk factor to predict the appearance of RAS in patients undergoing cardiac catheterization studies.

Key words: radial spasm, smoking, cardiac catheterization. Resumen

RESUMEN

Objetivo. Determinar la influencia independiente del tabaquismo en la aparición de espasmo de la arteria radial en estudios de cateterismo cardíaco y el análisis de la asociación de diferentes condiciones para el desarrollo del espasmo arterial radial.

Material y método. Estudio prospectivo, transversal. Seguimiento durante 3 meses de pacientes sometidos a cateterismo cardíaco en 2 centros de la provincia de Córdoba, analizando la influencia de variables asociadas al procedimiento y variables clínicas como sexo, edad y factores de riesgo en la aparición de espasmo arterial radial (EAR). **Resultado.** Se analizó a los pacientes sometidos a cateterismo cardíaco por acceso arterial radial entre septiembre y diciembre de 2019 en 2 centros de la ciudad de Córdoba. Se obtuvo una población de 347 pacientes; 131 eran mujeres (37,8%) y 216, varones (62,2%). La ocurrencia de espasmo de arteria radial (EAR) en la muestra fue del 14,7% (n=51). Se verificó una asociación significativa entre tabaquismo y este evento. Todos los casos la proporción de EAR fue significativamente más elevada en fumadores (17,6%) en contraste con los no fumadores (6,5%). El tabaquismo fue una variable asociada significativamente al espasmo radial ($p=0,020$).

El sexo presentó un odds ratio (OR) asociado a espasmo de 8,2, la probabilidad de que las mujeres presenten EAR sería 8 veces mayor que la asociada a varones. El EAR ocurrió casi en un tercio de las mujeres estudiadas (29,0%) y en el 6,0% de los varones, y fue más observado en mujeres fumadoras (33,3% de ellas presentó espasmo), aunque también en no fumadores la incidencia de espasmo fue más frecuente en mujeres que en varones. En cuanto a los factores relacionados con el procedimiento, se observó que el tamaño del catéter y el espasmo se asociaba significativamente ($p=0,021$). El tiempo de procedimiento resultó significativo ($p=0,022$), con un mayor riesgo de espasmo asociado a pacientes con tiempos procedimentales superiores a 20 minutos ($OR=2,6$) y el tipo de estudio y ocurrencia de EAR ($p=0,014$), siendo la categoría de mayor riesgo el estudio terapéutico, con una $OR=3,1$ (intervalo de confianza del 95% [IC95%]: 1,25-7,67).

Conclusiones. La incidencia de EAR se ubicó cerca del 15% de la muestra estudiada, positivamente relacionada con el sexo femenino y el tabaquismo. Se observó que el tabaquismo es un factor de riesgo independiente para predecir la aparición de EAR en pacientes sometidos a estudios de cateterismo cardíaco.

Palabras claves: espasmo radial, tabaquismo, cateterismo cardíaco.

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INTRODUCTION

Radial access has been gaining traction over the last few years regarding percutaneous coronary interventions. To this date, it is one of the most commonly chosen access routes by interventional cardiologists worldwide when performing endovascular procedures (1-3). Evidence shows benefits for both patients and health centers with fewer vascular complications, more comfort and early mobilization, better results in high-risk patients, shorter hospital stays, and a significant reduction of hospital expenses (3,4,6).

Multiple randomized clinical trials such as the RIVAL (6,7) and the RIFLE-STEACS (8-10) confirm that the use of the radial access is associated with fewer adverse cardiovascular events in patients with ST-segment elevation myocardial infarction (STEMI). Based on the growing body of evidence, the most recent iteration of the clinical practice guideli-

nes published by the European Society of Cardiology (ESC) (2017) regarding percutaneous coronary interventions (PCI) for the management of patients with STEMI changed the recommendation to a class I recommendation (level of evidence A) regarding the use of the radial artery as the access route (19).

Radial access is associated with lower rates of procedural success and a greater need for crossing to the femoral access when performing percutaneous coronary interventions (8,9), which is commonly associated with radial artery spasm (RAS) (6,7). The appearance of severe RAS requires changing the access site because RAS, a common complication, does not facilitate the proper advancement and manipulation of devices through this access route (11-13).

There are different concepts that can be used to define RAS. Maybe the most wide spread definition is the sudden, abrupt, and transient reduction of the arterial caliber clinically and/or angiographically diagnosed during the procedure (16), the presence of pain at the puncture site while advancing the guidewire, pain in the forearm when moving the catheters and/or pain and incapacity to manipulate the catheters (9-12); at times, pain is due to device displacement in the presence of factors like tortuous or spiral radial, brachial and/or subclavian arteries.

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TABLE 1. Model summary. From left to right: factor studied, category with higher chances of RAS; statistical significance (P); OR and 95%CI.

Explicative factor (predictor)	Category of highest risk of spasm	P	OR	95%IC for the OR	
				Lower limit	Upper limit
Sex	Women	<0,001	8,26	3,79	18,02
Ischemic heart disease	Yes	0,006	2,77	1,34	5,72
Smoking	Smokers	0,020	3,47	1,22	9,89
Catheter diameter (French)	6-Fr	0,021	2,91	1,18	7,19
Procedural time	≥ 20 min	0,022	2,63	1,15	6,02
MRS	Yes	0,046	3,53	1,02	12,22
Peripheral vascular disease	No	0,067	2,47	0,94	6,47
Treatment	Without treatment	0,122	1,96	0,83	4,59
Hypothyroidism	No	0,200	2,66	0,60	11,87
DLP	No	0,216	1,65	0,75	3,67
BMI	> 30	0,435	1,35	0,63	2,89
Interventional cardiologist	Experienced	0,455	1,33	0,63	2,79
Radial access (side)	-	0,535	1,39	0,49	3,88
Contrast agent	> 70 cc	0,618	1,25	0,52	2,97
AHT	-	0,638	1,13	0,42	3,02
DM	-	0,676	1,10	0,53	2,31
Age group	-	0,792	1,07	0,51	2,24
Absorbed dose rate (mGy/min)	-	0,804	1,03	0,93	1,13
Radioscopy time	-	0,854	1,00	1,00	1,00

RAS can have different degrees of severity and occur at any stage during the procedure resulting in a mismatch among the introducer sheath, the catheter, and the size of the artery; it is the most common cause of procedural failure when performing cardiac catheterizations (11,12,16).

The incidence rate of RAS during a percutaneous coronary intervention is between 4% and 18% (8,9,12,14-16). Currently, it is well-known that the mechanism that causes RAS is associated with the existence of different factors that predispose to its appearance: clinical, anatomical, and procedural factors alike (9-11).

Female sex, age group between 20 and 40 years, low body mass index, and a past medical history of diabetes mellitus, smoking, ischemic heart disease are among the clinical factors that have been reported. The diameter of the radial artery, the presence of atherosclerotic lesions, arterial tortuosity, and radial artery anomalies and/or variants are some of the anatomical factors that have been reported. Successive failed attempts when trying to catheterize the radial artery, large-caliber devices, long procedural times, and the number of catheters used are some of the procedural factors reported too.

Today in Argentina there are very few studies correlating the patients' clinical predictors with the appearance of RAS. Instead, they are mostly focused on preventing this event by using drug therapy.

It is well-known that chronic smoking changes the endogenous mechanisms of pain and affects how pain is perceived and felt due to the chronic exposure to nicotine (15,16,18,20,24). Some epidemiological studies

claim that smoking is a risk factor for chronic and acute pain (17,18) which is why smoking has been established as a predictor of intense pain, which may be associated with the state of radial artery hyperreactivity and induce spasm while performing percutaneous coronary interventions.

Given the scarce evidence available and the case reports presented by different cardiology societies since pharmacological prevention (11,12,14,15), and taking into consideration that we are dealing with complex, multifactorial events that lead to the occurrence of RAS, the objective of this study is to identify the presence of smoking as an independent predictor variable of RAS for a better understanding and prevention of radial artery spasm risk in patients with such a risk factor.



































































Objectives

To determine the impact of smoking on the appearance of RAS in cardiac catheterization studies and analyze the association of different conditions for the development of RAS.

MATERIALS AND METHODS

Analytical, prospective, and cross-sectional study. The target population included a total of 347 patients between 18 and 90 years-old studied over a 3-month follow-up period (from September through December 2019) in the *Clínica Privada Vélez Sarsfield* Interventional Cardiology Unit and the *Sanatorio Francés* Interventional Cardiology Unit, both in the City of Córdoba, Argentina.

TABLE 2. Relative and absolute frequencies of RAS occurrence based on smoking and category of different factors (the color scale is associated with percentages).

Factor	Category	Spasm in non-smokers		Spasm in smokers	
Sex	Feminine	 11,5%	(3/26)	 33,3%	(35/105)
	Masculine	 4,5%	(3/66)	 6,7%	(10/150)
Catheter diameter (French)	5-Fr	 4,3%	(3/69)	 14,0%	(25/178)
	6-Fr	 13,0%	(3/23)	 26,0%	(20/77)
Ischemic heart disease	Ischemic heart disease(no)	 2,0%	(1/51)	 13,3%	(20/150)
	Ischemic heart disease(yes)	 12,2%	(5/41)	 23,8%	(25/105)
Smoking	Non-smokers	 6,5%	(6/92)		
	Smokers			 17,6%	(45/255)
Procedural time	< 20	 4,4%	(2/45)	 11,2%	(13/116)
	≥ 20	 8,5%	(4/47)	 23,0%	(32/139)
MRS	No	 6,6%	(6/91)	 16,4%	(39/238)
	Yes	 0,0%	(0/1)	 35,3%	(6/17)
Study	Coronary computed tomography angiography	 13,0%	(3/23)	 21,6%	(16/74)
	Cine coronary angiography and others	 4,3%	(3/69)	 16,0%	(29/181)
Peripheral vascular disease	Peripheral vascular disease (no)	 8,2%	(6/73)	 21,5%	(37/172)
	Peripheral vascular disease (yes)	 0,0%	(0/19)	 9,6%	(8/83)
DLP (dyslipidemia)	DLP (no)	 8,8%	(6/68)	 21,6%	(27/125)
	DLP (yes)	 0,0%	(0/24)	 13,8%	(18/130)
Hypothyroidism	Hypothyroidism (no)	 7,1%	(6/85)	 17,9%	(42/235)
	Hypothyroidism (yes)	 0,0%	(0/7)	 15,0%	(3/20)
Treatment	Without treatment	 0,0%	(0/4)	 50,0%	(2/4)
	With treatment	 6,8%	(6/88)	 17,1%	(43/251)
BMI	BMI < 30	 3,3%	(2/61)	 18,1%	(30/166)
	BMI ≥ 30	 12,9%	(4/31)	 16,9%	(15/89)
Contrast agent	< 70 cc	 4,5%	(2/44)	 16,1%	(19/118)
	≥ 70 cc	 8,3%	(4/48)	 19,0%	(26/137)
AHT	AHT (no)	 0,0%	(0/32)	 14,8%	(8/54)
	AHT (yes)	 10,0%	(6/60)	 18,4%	(37/201)
DM	NO	 9,8%	(5/51)	 16,2%	(18/111)
	Yes	 2,4%	(1/41)	 18,8%	(27/144)
Interventional cardiologist	Not experienced	 8,0%	(4/50)	 15,8%	(24/152)
	Experienced	 4,8%	(2/42)	 20,4%	(21/103)
Age group	Up to 60 years	 2,9%	(1/34)	 17,0%	(17/100)
	Over 60 years	 8,6%	(5/58)	 18,1%	(28/155)

Patients eligible for diagnostic and/or therapeutic left cardiac catheterization via radial artery access were included. Patients who met any of the following criteria were excluded:

- Allergy to drugs used as prophylactic therapy to prevent radial artery spasm.
- Hemodynamically unstable patients: severe hypotension requiring vasoactive drugs.
- Negative Allen's test: it is used to measure the state of circulation of the palmar arch cubital collateral artery to avoid any possible ischemic complications associated with palmar capillary refill. It consists of asking the patient to close his hand—which compresses the cubital and radial arteries—which turns the palm of the hand into white color due to blood flow interruption.

Then, the cubital artery pressure is released and the time it takes for the skin to go back to normal color is measured.

Different variables were analyzed in each patient and then categorized into clinical and procedural variables. Some of the clinical variables reported were the age of the patients measured in years, feminine/masculine sex, and the presence of previous comorbidities like diabetes mellitus (DM), arterial hypertension (AHT), dyslipidemia (DLP), chronic kidney disease (CKD), body mass index (BMI) < or > 30, ischemic heart disease, previous myocardial revascularization surgery (MRS), and smoking.

Procedural variables reported were the type of interventional cardiologist who performed the procedure (experienced

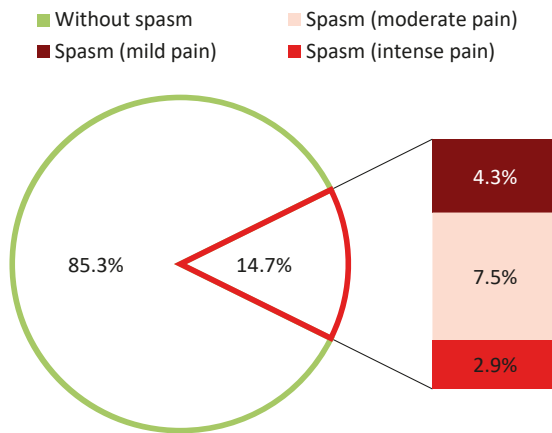


Figure 1. Percentage of cases based on the incidence rate of spasm and pain intensity perceived associated with the spasm.

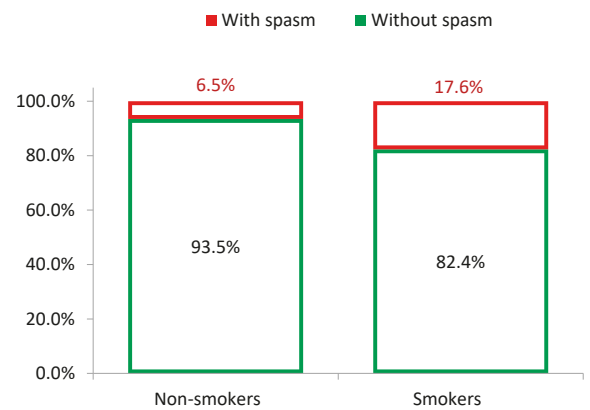


Figure 2. Relative frequencies of spasm based on smoking.

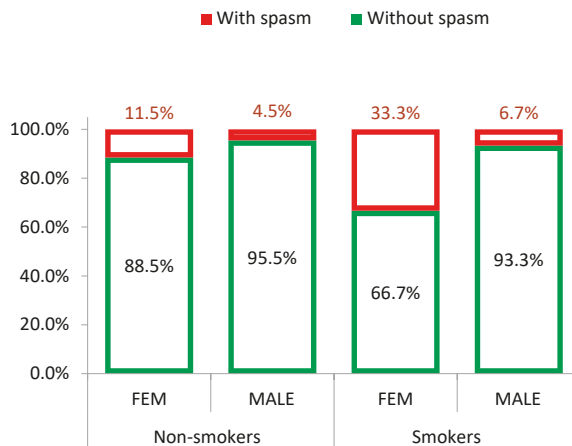


Figure 3. Relative frequencies of radial spasm based on sex and smoking.

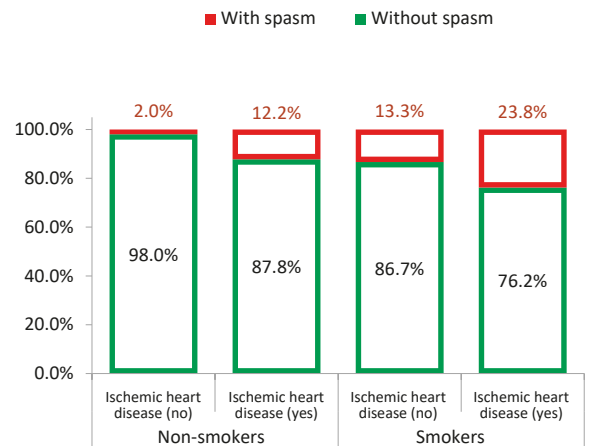


Figure 4. Relative frequencies of radial spasm based on ischemic heart disease and smoking.

or non-experienced). Experienced interventional cardiologists were those who had performed over 150 percutaneous coronary interventions as lead operator and 450+ coronary diagnostic catheterizations; procedural variables were also the number of radial access punctures (more or less than 1); the catheter French size (5-Fr, 6-Fr), the number of catheters used, and the procedural time measured in minutes counting from the puncture until the introducer sheath was retrieved via radial access (> 20 min or < 20 min).

The presence of RAS was defined as the difficulty and/or tamponade and/or severe pain when trying to move the catheter or introducer sheath that literally stops the procedure in its tracks. The degree of pain was assessed using an intensity index (score from 1 to 10) that was subjectively measured by the patient. Scores of up to 3 were indicative of mild pain, between 4 and 6 were indicative of moderate pain, and scores > 7 were indicative of intense pain and/or incapacity to move the catheter, and/or need for a difference access route.

First, a descriptive analysis was conducted of all the variables based on the measuring scales used: percentage of occurrence was used to express the qualitative variables, while central tendency and variability measures were used to express the

continuous variables. The inter-groups differences were established regarding sex and age. The association between risk factors and dependent variables was estimated too as categorized data were used to measure the odds ratio (OR) with a 95% confidence interval (IC95%).

To assess the degree of association of the different factors involved with radial artery spasms a binary logistics regression model was used. This regression analysis was completed with a stratified descriptive analysis that associated the 2 most relevant study variables (spasm and smoking) with different study factors. The variable of the type of procedure performed, whether diagnostic and/or therapeutic, was also used to study its possible association with RAS.

Ethical considerations

All data were gathered from the patients' clinical histories upon admission to the cath lab following their authorization to use these data with statistical purposes only. If they would not give their authorization, they were removed from the study. All data were analyzed in full compliance with ethical, confidential, privacy, and anonymity requirements making the patients' identification virtually impossible.

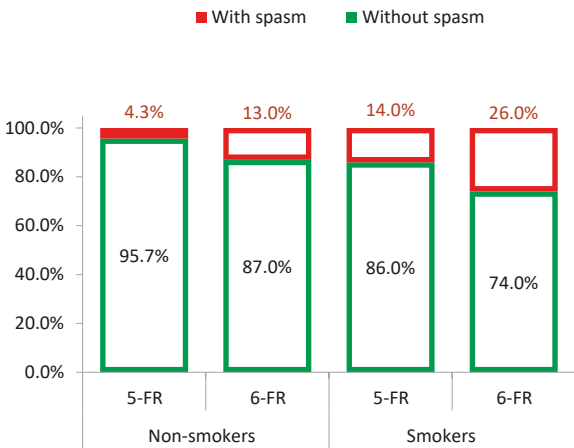


Figure 5. Relative frequencies of radial spasm based on Fr and smoking.

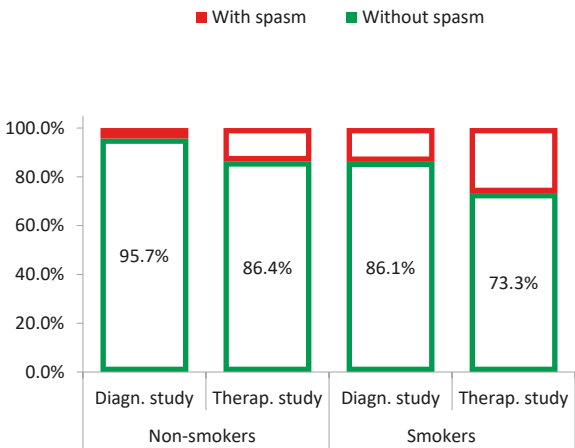


Figure 6. Relative frequencies of radial spasm based on type of study and smoking.

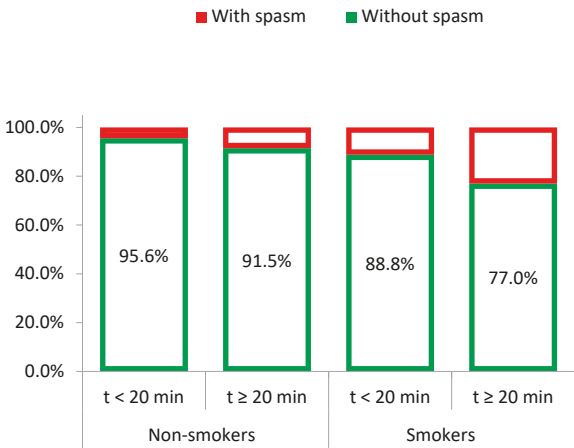


Figure 7. Incidence rate of radial spasm based on smoking and procedural time.

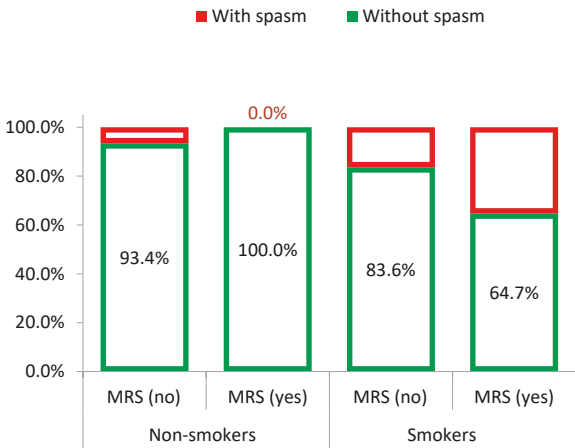


Figure 8. Relative frequencies of radial spasm based on MRS and smoking.

RESULTS

This analysis was generated from a sample of 347 patients of an age group between 37 and 93 years old with a mean age of 64.1 years; 131 were women (37.8%) and 216 were men (62.2%). Procedural complications were reported in zero patients. The occurrence of RAS in the study sample was 14.7% (n = 51). **Figure 1** shows the information reported. **Table 1** shows the factors introduced in the logistics regression model and arranged in descending order based on their statistical significance indicating in each case, the strength of the association with the RAS through its OR and 95%CI. **Table 2** shows the relative frequencies of the RAS event based on the categories of each factor. This descriptive analysis included both the factors considered significant in the logistics regression analysis and those considered significant enough for this study. Within the non-smokers group, the rate of spasm exceeds the values obtained for the overall rate [that was 14.7% (n = 59)]. In all the cases, the rate of spasms reported was higher among smokers.

Regarding smoking, the rate of RAS was significantly higher in smokers (17.6%) compared to non-smokers (6.5%). As **Figure 1** shows, smoking was the variable most significantly associated with the occurrence of radial spasms ($P = .020$) as smokers had a higher risk of having spasms compared to non-smokers. On the other hand, sex was the most explicative factor of the RAS event in the logistics regression model used ($P < .001$), the OR associated with the RAS was 8.2, and the chances that women had RAS were 8 times higher compared to men. RAS occurred in almost a third of the women studied (29.0%) compared to 6.0% of the men. These results are shown on **Figure 3** and adjusted to smoking. The RAS event was most commonly seen among female smokers (33.3% of them had spasms), although among non-smokers, the rate of spasm was more common in women compared to men. Regarding pre-existing conditions, as shown on **Figure 4**, patients with previous ischemic heart disease had more chances of developing radial artery spasm compared to patients with index episodes. The degree of association between this factor and the spasm was significant ($P = .006$).

Regarding procedural factors, the size of the catheter used, measured in French units, and the spasm were significantly associated ($P = .021$). The largest caliber catheter (6-Fr) was more commonly associated with the appearance of RAS compared to the smallest caliber catheter (5-Fr) as shown on **Figure 5**.

We should mention that in 100% of the therapeutic studies conducted ($n = 97$) 6-Fr catheters were used while in the diagnostic studies conducted only 5-Fr catheters were used (98.8%). Therefore, the results obtained for the size of the catheters are also valid for the type of study conducted. As a matter of fact, when the catheter size was changed by type of study conducted in the logistics regression analysis, very similar values to those reported on **Table 2** were obtained. Therefore, a significant strength of association was seen between the type of study conducted and the occurrence of RAS ($P = .014$) being the therapeutic study the category of the highest risk [OR = 3.1 (95%CI, 1.257.67)] as already seen (**Figure 6**).

Procedural time was confirmed as a significant factor ($P = .022$) with a higher risk of spasm associated with patients with procedural times > 20 min (OR = 2.6). In both smoking categories, the occurrence of radial artery spasm was more common in the group of patients with procedural times > 20 min, and a significantly higher risk of RAS was confirmed when both factors were present (**Figure 7**).

Regarding the clinical variable of previous MRS, a significant association was seen among smokers with a past medical history of MRS compared to non-smokers with a past medical history of MRS ($P = .046$), indicative of a higher risk of RAS associated with patients with MRS, particularly in current or former smokers (**Figure 8**).

Regarding DM, age, body mass index, and PVD, no significant differences were seen with the occurrence of RAS. There was a higher, though statistically insignificant, occurrence of this event in patients over 60. The same thing happened with radioscopy time inside procedural variables.

DISCUSSION

Despite the widespread use of this technique, to this day, very few studies have focused on the association of risk factors with the occurrence of RAS in diagnostic and/or therapeutic cardiac catheterizations. On this regard we found no scientific articles on smoking while conducting our bibliographic search. However, the results show that this habit clearly predisposes to the occurrence of the study primary endpoint.

In the scientific literature, the incidence rate of RAS varies based on the definition used and type of study conducted (10-13). In our study, the overall incidence rate of radial artery spasm was 14.7% and kept a positive correlation with feminine sex, previous ischemic heart disease, previous MRS, and smoking. Consistent with multiple studies, Ruiz-Salmerón RJ et al. studied a population of 637 patients. They found that in 127 patients (20.2%), radial artery spasm occurred during the procedure. In 25 patients (3.9%) another access route had to be used (in 13 cases due to radial artery spasm, 2% of the total) (31-33).

Experienced centers on this approach reported that radial artery spasm occurred in 15% to 30% of all the procedures. The SPASM and the SPASM-3 clinical trials (15,16) showed a significant increase in the incidence rate of RAS (from 3% to

73%) depending on the criterion used to define radial spasm, use of vasodilators prior to the procedure, and also associated with the study population (1219 patients). In a Latin American bibliographic review, more journals like the *Cuban Journal of Cardiology and Cardiovascular Surgery* (1,3,20,27) reported fewer cases with an incidence rate of radial spasms starting at around 10%. The same incidence rate was reported in the study conducted by Goldsmit et al. and published on *RACI* (16): access spasms occur in approximately 10% of the patients, which makes the procedure tougher and longer to perform, sometimes even having to cross to the femoral access route. Goldberg et al. (30,31) studied an early series of patients on whom the radial artery was used by interventional cardiologists not experienced in the use of the arm as an access site to perform percutaneous coronary interventions. Clinical success was achieved through the radial artery in 87% of 32 lesions treated and in 84% of 27 patients. The main characteristic that limited success through the arm was radial/brachial artery spasm, which occurred in 30% of the cases (clinical success: 50% with spasm vs 95% without it, $P < .05$). Spasm was more common in patients with peripheral vascular disease and in hypertensive patients too. In their study, they showed that prevention is probably more effective than the treatment of spasms, which is why it is so important to know the predictive factors of this event (33,34).

In this study, feminine sex was the most significant variable for the occurrence of RAS [$(P < .01)$, OR=8.24], and the rate of RAS was 8 times higher in women compared to men. Consistent with the scientific literature available, the RIVAL clinical trial confirmed feminine sex as one of the 4 predictive factors independently associated with the appearance of RAS: feminine sex, first failed attempt trying to access the radial artery, emergency procedure, and use of diltiazem. In their study of patients operated via radial access, Dehghani et al. (29,36), confirmed that 17% of the patients were women, which proves that sex is associated with higher rates of procedural failure.

Recently, Gorgulu et al. studied 1722 patients and reported that feminine sex was the only independent predictor of RAS at the beginning of the procedure. However, our study identified additional factors such as smoking (45,46).

In this study, smoking was an independent risk factor to explain the increasing occurrence of RAS, that is, the presence of this factor increased the probabilities of RAS occurrence [OR=3.46 (95%CI: 1.22-9.89)]. Therefore, current smokers have almost 3.5 times more chances of suffering from RAS as a procedural complication.

Women who also happen to be smokers have a 33.3% incidence rate of RAS. Therefore, these two variables combined can help predict, before performing any diagnostic and therapeutic cardiac catheterizations, the occurrence of RAS and any possible associated complications. These results were not found anywhere else.

In their comparative study of 200 patients in whom 6-Fr introducer sheaths were used, Gul et al. revealed a risk sub-analysis that suggests that patients with a history of smoking probably have lower chances of procedural success regardless of the type of introducer sheath used. In this RAS registry, dyslipidemia—added to feminine sex—was a strong predictor of moderate-to-severe radial artery spasm (47,48,50).

However, Gorgulu et al. reported a RAS incidence rate of 10.3%, and feminine sex as the only independent predic-

tor of risk; surprisingly, smoking was less common in patients with spasms (17% vs 29%, $P < .001$).^{45,46} Giannopoulos et al. presented 3 different cohorts of patients who underwent elective PCIs via transradial access where 5 weighed factors could be used to build up a risk score for developing spasm: body mass index, height, current smoking status, hypertension, and PVD. Feminine sex was considered the main predictor in all the groups, while the occurrence of RAS in smokers was also relevant in the different subgroups ($P < .01$), yet not statistically significant.^{20,22} Although the effect of smoking on coronary artery disease is well-known as a preventable factor, its role in the development of the pathological process triggered by this condition or in procedural complications is still unknown. According to this logistics regression model, the probability of experiencing radial artery spasms based on the contribution of different risk factors can be estimated. For example, patients with the following clinical parameters: women over 60 years old, current smoker, previous ischemic heart disease, hypertension, cardiac catheterization with a 6-Fr catheter, and a long procedural time would have, on average, 76% chances of having a spasm based on the predictive model proposed. For this reason, the analysis of the factors associated with RAS was presented independently from smoking to predict the occurrence of RAS.

REFERENCES

- Amador García Hernández R, Hernández Navas M, Aroche Aportela R, Aldama Pérez LI, Obregón Santos AG. Espasmo en la arteria radial en procedimientos intervencionistas. *Revista Cubana de Cardiología y Cirugía Cardiovascular* 2016;22(3).
- Sanmartín M, Cuevas D, Goicolea J, Ruiz-Salmerón R, Gómez M, Argibay V. Complicaciones vasculares asociadas al acceso transradial por cateterismo cardíaco. *Rev Esp Cardiol* 2004;57(6):581-4.
- Salgado FJ, Calviño SR, Vázquez Rodríguez JM, et al. Coronariografía y angioplastia coronaria por vía radial: experiencia inicial y curva de aprendizaje. *Rev Esp Cardiol* 2003;56:152-9.
- Kiemeneij F, Vajifdar BU, Eccleshall SC, Laarman G, Slagboom T, Van der WR. Measurement of radial artery spasm using an automatic pullback device. *Catheter Cardiovasc Interv* 2001;54:437-41.
- Waggoner T, White R, Wassmer PV, et al. Radial Artery Spasm Prevention (RASP Study): A Retrospective Analysis of Oral Pharmacologic Prophylaxis for Prevention of Radial Artery Spasm During Transradial Access for Cardiac Catheterization. *Cath Lab Digest* July 2014;22(7).
- Kiemeneij F, Vajifdar BU, Eccleshall SC, Laarman G, Slagboom T, Van der WR. Evaluation of a spasmolytic cocktail to prevent radial artery spasm during coronary procedures. *Catheter Cardiovasc Interv* 2003;58:281-4.
- Sanjit SJ, Salim Y, John C, et al. Radial versus femoral access for coronary angiography and intervention in patients with acute coronary syndromes (RIVAL): a randomised, parallel group, multicentre trial
- Romagnoli E, Biondi-Zoccai G, Sciahbasi A, et al. Radial versus femoral randomized investigation in ST-segment elevation acute coronary syndrome: the RIFLE-STEACS (Radial Versus Femoral Randomized Investigation in ST-Elevation Acute Coronary Syndrome) study. *J Am Coll Cardiol* 2012;60(24):2481-9.
- Budaj A, Eikelboom JW, Mehta SR, et al. Improving clinical outcomes by reducing bleeding in patients with non-ST-elevation acute coronary syndromes. *Eur Heart J* 2009;30:655-61
- Rodríguez Blanco S, Leyva Quert A, Valdés Recarey M, et al. Transradial Access way. Technique and involvement. *Revista Cubana de Cardiología y Cirugía Cardiovascular* 2014;20(4).
- Rathore S, Stables R, Pauriah M, et al. Impact of Length and Hydrophilic Coating of the Introducer Sheath on Radial Artery Spasm During Transradial Coronary Intervention. A Randomized Study. *JACC: Cardiovascular Interventions*. 2010;3(5):475-83
- Rodríguez Blanco S, Leyva Quert A, Mendoza Ortiz JL, et al. Midazolam en la prevención del espasmo de la arteria radial. Ensayo clínico aleatorizado a doble ciego. *Rev Argent Cardioangiol Interv* 2018;9(1):39-46
- Coppola J, Patel T, Kwan T, et al. Nitroglycerin, nitroprusside, or both, in preventing radial artery spasm during transradial artery catheterization. *J Invasive Cardiol* 2006;18(4):155-8.
- Varenne O, Jegou A, Cohen R, et al. Prevention of arterial spasm during percutaneous coronary interventions through radial artery: the SPASM study. *Catheter Cardiovasc Interv* 2006;68(2):231-5.
- Rosencher J, Chaib A, Barbou F, et al. How to limit radial artery spasm during percutaneous coronary interventions: The spasmolytic agents to avoid spasm during transradial percutaneous coronary intervention (SPASM-3) study. *Catheter Cardiovasc Interv* 2014.doi:10.1002/ccd.25163.
- Goldsmith A, Baucero G, Szejfman C, Bettinotti M. Utilidad del clorhidrato de nalbupina en la prevención del espasmo de la arteria radial. *Rev Argent Cardioangiol Interv* 2010;1:19-23.
- Giannattasio C, Mangoni AA, Stella ML, Carugo S, Grassi G, Mancia G. Acute effects of smoking on radial artery compliance in humans. *J Hypertens* 1994 Jun;12(6):691-6.
- Shing Kwok C, Rashid M, Fraser D, Nolan J, Mamas M. Intra-arterial vasodilators to prevent radial artery spasm: a systematic review and pooled analysis of clinical studies. *Cardiovasc Revasc Med* 2015;16(8):484-90.
- Sandoval Y, Bell MR, MD; Gulati R. Transradial Artery Access Complications. *Circ Cardiovasc Interv* 2019;12(11):e007386.
- Giannopoulos G, Raisakis K, Syntetos A, et al. Una puntuación predictiva de espasmo de la arteria radial en pacientes sometidos a intervención coronaria percutánea transradial. *Int J Cardiol* 2015;188:76-80.
- Brueck M, Bandorski D, Kramer W, Wiecek M, Höltinger R, Tillmanns H. A randomized comparison of transradial versus transfemoral approach for coronary angiography and angioplasty. *JACC Cardiovasc Interv* 2009 Nov;2(11):1047-54.
- Singh M, Rihal CH, Lennon R, Garrat K, Holmes DR. Comparison of Mayo Clinic Risk Score and American College of Cardiology/American Heart Association Lesion Classification in the Prediction of Adverse Cardiovascular Outcome Following Percutaneous Coronary Interventions. *J Am Coll Cardiol* 2004;44:357-61.

23. Salgado Fernández J, Calviño Santos R, Vázquez Rodríguez JM, et al. Coronariografía y angioplastia coronaria por vía radial: experiencia inicial y curva de aprendizaje. *Rev Esp Cardiol* 2003;56(2):152-9.
24. Elizaga Corrales J. Acceso por vía radial: ¿debería ser más utilizado? *Rev Esp Cardiol* 2003;56(2):124-7.
25. Rao SV, Cohen MG, Kandzari DE, Bertrand OF, Gilchrist IC. The transradial approach to percutaneous coronary intervention. *J Am Coll Cardiol* 2010;55:2187-95.
26. Moscoso-Soriano N, Sepúlveda-Sepúlveda J, Salas-Sánchez O, Cruz-Wellmann R, Moxica del Ángel J. Técnicas de acceso transradial en intervenciones percutáneas coronarias. *Medicina Universitaria* 2013;15:33-9.
27. Abizaid A, Ribamar J. *Manual de Cardiología Intervencionista do Instituto Dante Pazzanese*. 1ra. Ed. Río de Janeiro: Elsevier; 2013.
28. Hamon M, Pristipino C, Di Mario C, et al. Consensus document on the radial approach in percutaneous cardiovascular intervention position paper by the European Association of Percutaneous Cardiovascular Interventions and Working Groups on Acute Coronary Care and Thrombosis of the European Society of Cardiology 2013;8:1242-51.
29. Obregón AG, Conde Cerdeira H, Aroche Aportela R. Acceso vascular para intervencionismo coronario percutáneo. En: *Manual de Cardiología Intervencionista*. 1ra Ed. Cuba. 2010. pp 46-55.
30. Goldberg SL, Renslo R, Sinow R, French WJ. Learning Curve in the Use of the Radial Artery as Vascular Access in the Performance of Percutaneous Transluminal Coronary Angioplasty. *Cathet Cardiovasc Diagn* 1998;44(2):147-52.
31. Abe S, Meguro T, Endoh N, et al. Response of the radial artery to three vasodilatory agents. *Catheter Cardiovasc Interv* 2000;49:253-6.
32. Ruiz-Salmerón R, Mora R, Vélez Gimón M, et al. Espasmo radial en el cateterismo cardíaco transradial. Análisis de los factores asociados con su aparición y de sus consecuencias tras el procedimiento. *Rev Esp Cardiol* 2005;58(5):504-11.
33. Mamas M, D'Souza S, Hendry C, et al. Use of the sheathless guide catheter during routine transradial percutaneous coronary intervention: A feasibility study. *Catheter Cardiovasc Interv* 2010;75(1):599-602.
34. Kiemeneij F, Vajifdar BU, Eccleshall SC, Laarman G, Slagboom T, van der Wieken R. Evaluation of a spasmolytic cocktail to prevent radial artery spasm during coronary procedures. *Catheter Cardiovasc Interv* 2003;58:281-4.
35. Chen CW, Lin TK, Lin CD. A simple and effective regimen for prevention of radial artery spasm during coronary catheterization. *Cardiology* 2006;105:43-7.
36. Dehghani P, Mohammad A, Bajaj R, et al. Mechanism and predictors of failed transradial approach for percutaneous coronary interventions. *JACC Cardio Vasc Interv* 2009;2:1057-64. doi: 10.1016/j.jcin.2009.07.014
37. Coppola J, Patel T, Kwan T, et al. Nitroglycerin, nitroprusside or both in preventing radial artery spasm during transradial artery catheterization. *J Invasive Cardiol* 2006;18:155-8.
38. Kim SH, Kim EJ, Cheon WS, et al. Comparative study of nicorandil and a spasmolytic cocktail in preventing radial artery spasm during transradial coronary angiography. *Int J Cardiol* 2007;120:325-30.
39. 33. Byrne J, Spencer M, Haegeli L, et al. Magnesium sulphate during transradial cardiac catheterization: a new use for an old drug? *J Invasive Cardiol* 2008;20:539-42.
40. Ouadhour A, Sideris G, Smida W, Logeart D, Stratiev V, Henry P. Usefulness of subcutaneous nitrate for radial access. *Catheter Cardiovasc Interv* 2008;1:343-6.
41. Colussi GL, Di Fabio A, Catena C, Chiuch A, Sechi LA. Involvement of endothelium-dependent and independent mechanisms in midazolam-induced vasodilation. *Hypertens Res* 2011;34:929-34.
42. Deftereos S, Giannopoulos G, Raisakis K, et al. Moderate Procedural Sedation and Opioid Analgesia During Transradial Coronary Interventions to prevent spasm. *J Am Coll Cardiol Intv* 2013;6:267-73.
43. Martínez Gabaldá S, Colominas Trilla M, Bellido Niebla M, et al. Protocolo de sedo-analgésia para prevención del espasmo radial en hemodinámica cardíaca. *Enferm Cardiol* 2008;45:26-9.
44. Martín Reyes R, Franco Példez JA, Piñero Lozano A, et al. Prevención del espasmo radial mediante la utilización de sedo-analgésia. Estudio prospectivo aleatorizado de 2 estrategias de tratamiento. *Rev Esp Cardiol* 2011;64 Supl 3:41-2.
45. Gorgulu S, Norgaz T, Karaahmet T, Dagdelen S. Incidence and predictors of radial artery spasm at the beginning of a transradial coronary procedure. *J Interv Cardiol* 2012.
46. Valgimigli M, Campo G, Penzo C, et al. Cateterismo coronario transradial e intervención en todo el espectro de resultados de pruebas de Allen. *J Am Coll Cardiol* 2014;63:1833-41.
47. Deftereos S, Giannopoulos S, Raisakis K, et al. Sedación procesal moderada y analgesia con opioides durante las intervenciones coronarias transradiales para prevenir el espasmo: un estudio prospectivo aleatorizado. *J Am Coll Cardiol Intv* 2013;6:1215-1215.
48. Abe S, Meguro T, Endoh N, et al. Respuesta de la arteria radial a tres agentes vasodilatadores. *Catheter Cardiovasc Interv* 2000;49:253-6.
49. Ruiz-Salmerón RJ, Mora R, Vélez-Gimón M, et al. Espasmo radial en el cateterismo cardíaco transradial. Análisis de los factores asociados con su aparición y de sus consecuencias tras el procedimiento. *Rev Esp Cardiol* 2005;58(5):504-11.
50. Gul B, Stolar M, Stair B, et al. Comparison of procedural success between two radial sheaths Comparison of the 6-Fr Glidesheath Slender to 6-Fr standard sheath. *Herz* 2020 Feb;45(1):79-85.

Coronary pulmonary fistula: presentation of a rare case with successful endovascular resolution

Fístula coronario pulmonar: presentación de un caso poco frecuente con resolución endovascular exitosa

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ABSTRACT

Introduction. 58-year-old female patient with dyspnea NY functional class III of 2 months of evolution. Echocardiogram showed no pathological findings. Echo stress with exercise showed inferior ischemia. Stratification was decided with coronary angiography in which a coronary pulmonary fistula was observed. The fistula was embolized with coils. After two months of follow-up, the patient presented clinical improvement and was asymptomatic.

Conclusion. Coronary pulmonary fistulas are a rare entity, present in 0.1% of coronary angiograms according to different series. A case with endovascular resolution is presented.

Key words: coronary pulmonary fistula, embolization, congenital anomalies of coronary arteries, coils.

RESUMEN

Introducción. Paciente de sexo femenino de 58 años que consulta por disnea clase funcional III de 2 meses de evolución. El ecocardiograma no mostró hallazgos patológicos. El ecoestrés con ejercicio evidenció isquemia inferior. Se decide estratificación con coronariografía en la que se observa fístula coronario pulmonar. Se realizó embolización de la fístula con coils. Luego de un mes de seguimiento la paciente presenta mejoría clínica y se encuentra asintomática.

Conclusión. La fístulas coronario pulmonares son una entidad poco frecuente, presente en el 0,1% de las coronariografías según distintas series. Se presenta un caso con resolución endovascular.

Palabras claves: fístula coronario pulmonar, embolización, anomalías congénitas de arterias coronarias, coils.

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INTRODUCTION

Coronary fistulas are rare anomalies that amount to 0.3% of congenital heart diseases (1-3). To this day, the incidence rate of coronary-pulmonary fistulas (CPF) has increased due to the widespread use of cardiac computed tomography and represents 15% to 30% of all coronary fistulas reported (4). In the last clinical practice guidelines published by the American College of Cardiology on congenital heart diseases (2018) no consensus has been achieved on the management of this condition (5). For some authors the therapeutic approach should be based on the size of the fistula, presence of symptoms, anatomy of the fistula, age of the patient, and presence of associated heart diseases (6).

CLINICAL CASE

We present the case of a 58-year-old woman (former smoker) with cardiovascular risk factors and no significant past medical history and NYHA functional class III dyspnea of 2-month clinical evolution.

Previous studies:

ECG: sinus rhythm, 80 bpm, no pathological findings.

Echocardiogram: no pathological findings.

Exercise echocardiography: positive for myocardial ischemia due to hypermotility of inferomedial and apical segments on maximum workload (900 kgm/min). During the exercise the patient showed signs of dyspnea.

It was decided to perform a coronary angiography:

Coronary angiography (**Figure 1**). Left main coronary artery: left main coronary artery of good caliber without angiographically significant stenosis. Left anterior descending coronary artery: of good caliber, reaches the apex, and there are not traces of angiographically significant stenosis. The coronary-pulmonary fistula (CPF)—of significant blood flow—originates in its proximal segment. Its first segment has a linear course of approximately 3 mm in diameter and 5 cm in length. Also, it shows another sinuous segment that runs into the pulmonary artery trunk. Circumflex artery: non-dominant, of good caliber with no signs of significant stenosis on the angiography performed. Right coronary artery: dominant, of good caliber and without angiographically significant stenosis.

After diagnosing CPF a cardiac computed tomography is performed to complete anatomical assessment.

Tomography (**Figure 2**)

The case was studied by the hospital heart team. Since it was a high-flow large-caliber CPF, it was decided to proceed with its endovascular closure.

The right radial access was used. A total of 7000 IU of sodium heparin were administered. The left main coronary artery was selectively catheterized using a 6-Fr extra-backup guide catheter. The Progreat microcatheter (Terumo) was placed at distal level with respect to the linear trajectory of the CPF. Embolization was attempted through the controlled release of two 3 mm x 5 cm detachable coils (AZUR detachable coil system, Terumo, United States). Fifteen minutes later, the coronary angiography performed reveals the

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TABLE 1.

Surgical ligation
Large sized high-flow fistulas.
Multiple communication and drainage sites.
Tortuous aneurysmatic fistulas.
Need for bypass surgery.
Branches that can be accidentally embolized..
Cierre percutáneo
Proximal origin of the fistula.
Single drainage site.
Non-tortuous fistula with an accessible distal area.
End of the fistula far from the coronary arteries.
Older patients of high surgical risk.

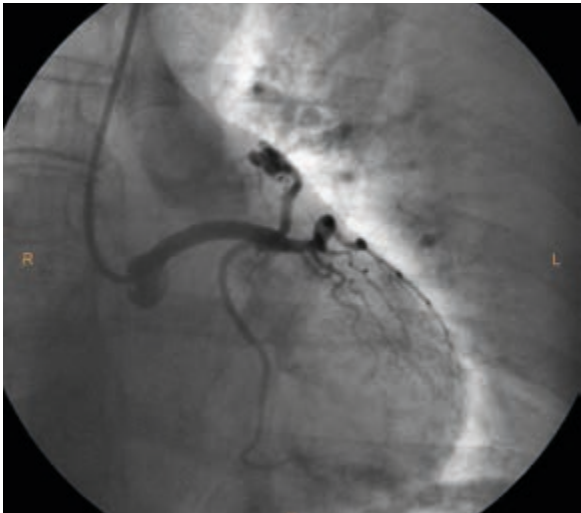


Figure 1.

partial closure of the CPF that remains patent with blood flow through the afferent branch not seen on the diagnostic study and that originates proximal to the coils recently implanted. It was decided to complete the embolization with a 3 mm x 2 cm fiber coil (AZUR, Terumo, United States). Ten minutes later the angiography performed reveals the total closure of the fistula (**Figure 3**). After completing the procedure, the patient remained in the coronary unit for 24 hours. At the 1-month follow-up the patient's symptoms improved with no signs of dyspnea while performing activities of daily living.

DISCUSSION

Coronary fistulas are rare anomalies that amount to 0.3% of all congenital heart disease (1-3). To this date, the incidence rate of CPF has increased thanks to the widespread use of cardiac computed tomography and represents 15% to 30% of all coronary fistulas (4). Although former studies have reported that the most common origin of CPFs is the right coronary artery, a recent systematic review confirmed that the most common origin of CPFs is the left main coronary artery (84% of the cases), most of them draining into the pulmonary artery trunk (89%) (7). Two different types of CPF have been reported, one of them is a large sized single fistula; the other a small sized fistula multiple connections (7). It is more likely that large sized single fistulas are associated with the generation of hemodynamic changes

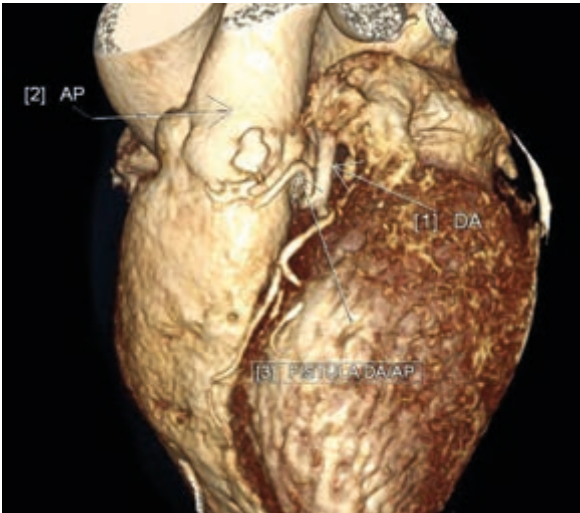


Figure 2. AP: pulmonary artery. DA: Left anterior descending coronary artery

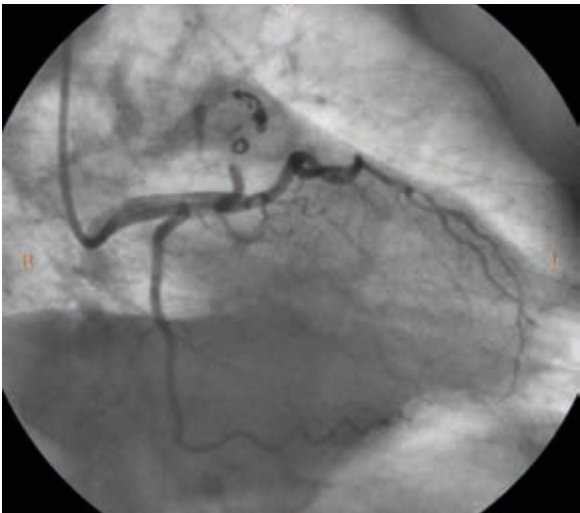


Figure 3.

and symptom onset. The therapeutic approach should be based on the size of the fistula, the presence of symptoms, the anatomy of the fistula, age of the patient, and presence of associated heart diseases (6). Asymptomatic patients with small sized fistulas are often treated with antiplatelet therapy and antibiotic prophylaxis. and disease progression monitoring. According to the clinical practice guidelines published by the American College of Cardiology—regardless of symptoms—the corrective treatment of large sized CPFs and small and medium sized fistulas in symptomatic patients (including myocardial ischemia, arrhythmias, ventricular dysfunction, and endarteritis) has a class IC indication.5 Treatment options are surgical ligation and endovascular treatment. The most adequate therapeutic option will depend on the characteristics of each particular case (**Table 1**) (7).

CONCLUSION

The scientific literature available on coronary-pulmonary fistulas is scarce. We presented the case of an asymptomatic APF successfully treated with endovascular treatment.

REFERENCES

1. McNamara JJ, Gross RE. Congenital coronary artery fistula. *Surgery* 1969;65(1):59-69.
2. Vavuranakis M, Bush CA, Boudoulas H. Coronary artery fistulas in adults: incidence, angiographic characteristics, natural history. *Cathet Cardiovasc Diagn* 1995;35(2):116-20.
3. Yamanaka O, Hobbs RE. Coronary artery anomalies in 126,595 patients undergoing coronary arteriography. *Cathet Cardiovasc Diagn* 1990;21(1):28-40.
4. Kim MS, Jung JJ, Chun HJ. Coronary to pulmonary artery fistula: morphologic features at multidetector CT. *Int J Cardiovasc Imaging* 2010;26(suppl 2):273-80.
5. Stout KK, Daniels CJ, Aboulhosn JA, et al. 2018 AHA/ACC guideline for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation* 2019;139:e698-800.
6. Latson LA. Coronary artery fistulas: how to manage them. *Catheter Cardiovasc Interv* 2007;70(1):110-6.
7. Gabin Yun, MD Tae Hyun Nam, MS Eun Ju Chun, MD, PhD. Coronary Artery Fistulas: Pathophysiology, Imaging Findings, and Management. *RadioGraphics* 2018;38:688-703.
8. Armsby LR, Keane JF, Sherwood MC, Forbess JM, Perry SB, Lock JE. Management of coronary artery fistulae: patient selection and results of transcatheter closure. *J Am Coll Cardiol* 2002;39(6):1026-32.
9. Jama A, Barsoum M, Bjarnason H, Holmes DR Jr, Rihal CS. Percutaneous closure of congenital coronary artery fistulae: results and angiographic follow-up. *JACC Cardiovasc Interv* 2011;4(7):814-21.
10. Gowda ST, Forbes TJ, Singh H, et al. Remodeling and thrombosis following closure of coronary artery fistula with review of management: large distal coronary artery fistula—to close or not to close? *Catheter Cardiovasc Interv* 2013;82(1):132-42.

Subclavian-coronary steal syndrome. Case report and resolution by angioplasty

Síndrome de robo subclavio-coronario. Reporte de caso y resolución por angioplastia

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ABSTRACT

Introduction. Subclavian-coronary steal syndrome is a rare condition. It is due to occlusion or severe stenosis of the subclavian artery, anterior and proximal to the origin of the vertebral artery. It responds well to percutaneous treatment, either with a balloon or an endovascular prosthesis implant. The objective of this work was to present a case report of subclavian-coronary steal syndrome, its percutaneous resolution, and a literature review. **Method.** Case report.

Conclusion. The coronary subclavian steal syndrome is a rare entity. It can cause myocardial ischemia and neurological symptoms. Complete preoperative studies are required to prevent it added to high clinical suspicion to diagnose it. Early percutaneous stent treatment resolves it successfully.

Key words: coronary disease, braquicephalic disease, percutaneous angioplasty, coronary subclavian steal syndrome, interventional radiology, subclavian stenosis.

RESUMEN

Introducción. El síndrome del robo subclavio-coronario es un cuadro poco frecuente. Se debe a una oclusión o estenosis severa de la arteria subclavia, anterior y proximal al origen de la arteria vertebral. Presenta buena respuesta al tratamiento percutáneo, ya sea con balón o implante de prótesis endovascular. El objetivo de este trabajo fue presentar un reporte de caso de síndrome de robo subclavio-coronario, su resolución percutánea, y una revisión bibliográfica. **Método.** Reporte de caso.

Conclusión. El síndrome de robo subclavio-coronario es una situación poco frecuente. Puede generar isquemia miocárdica y síntomas neurológicos. Se requieren estudios preoperatorios completos para su prevención, y de una alta sospecha clínica para su diagnóstico. El tratamiento percutáneo precoz con stent permite resolverlo en forma satisfactoria.

Palabras claves: enfermedad coronaria, enfermedad braquicefálica, angioplastia percutánea, síndrome de robo subclavio-coronario, cardiología intervencionista, estenosis subclavia.

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INTRODUCTION

Coronary artery disease is one of the most common entities across the world. At times, it can be associated with carotid artery and other territories disease (1). Although the management of coronary artery disease can be percutaneous [percutaneous transluminal angioplasty (PTA)] or surgical [myocardial revascularization surgery (MRS)] the remaining vascular territories should be taken into consideration too for a comprehensive approach of patients with concomitant peripheral and central vascular disease. If not, different complications can occur, being one of them the coronary subclavian steal syndrome (2,3).

Coronary subclavian steal syndrome (CSSS) is a rare condition. It is due to the occlusion or severe stenosis of the subclavian artery, anterior and proximal to the origin of the vertebral artery (4,5). It affects the left subclavian artery in 90% of the cases and the right subclavian artery in 10% of the cases. The prevalence reported is between 0.6% and 6.4% (6). It responds well to percutaneous treatment, either with a balloon or endovascular implantation (stent).

In this article we present the case of a coronary subclavian steal syndrome followed by its percutaneous resolution.

OBJECTIVES

The objective of this study was to present a case report and a bibliographic review of the coronary subclavian steal syndrome

CLINICAL CASE

This is the case of 64-year-old woman with the following cardiovascular risk factors: arterial hypertension (AHT), dyslipidemia (DLP), and smoking; her cardiovascular clinical history revealed coronary artery disease due to severe lesions in the left main coronary artery (LMCA) and left anterior descending coronary artery (LAD) at ostial and mid-1/3rd level, and right coronary artery (RCA), accompanied by asymptomatic carotid artery disease. Due to the patient's clinical history, she was treated with MRS with 2 bypasses (left internal mammary artery [LIMA] bridge to the LAD and saphenous vein graft to the RCA). Six months later she was admitted to the coronary unit with signs of unstable angina pectoris associated with heart failure. The patient had a good clinical response to the early anti-ischemic and diuretic treatment administered. The physical examination revealed asymmetrical brachial pulses, and a 30 mmHg difference of blood pressure measured between both arms. The cine coronary arteriography (CCA) performed 48 hours later revealed the presence of retrograde flow from the LIMA towards the left subclavian artery (**Figures 1 A and B**). The patient's study was completed with an angiography of the neck blood vessels that confirmed the left subclavian artery occlusion (**Figure 2**) and the presence of heavily calcified lesions of the common carotid artery (CCA) and left internal carotid artery (**Figure 3**). It was decided to administer percutaneous treatment through a 2-staged angioplasty of the lesions described. A 0.035 in hydrophilic guidewire was advanced via left humeral access (Radiofocus Guide Wire M, Terumo, Japan). Two balloons of 4 mm x 30 mm and 6 mm x 30 mm in length were used for predilatation purposes (PTA Balloons, Cordis, United States). The angioplasty was performed from the origin of the left subclavian artery using

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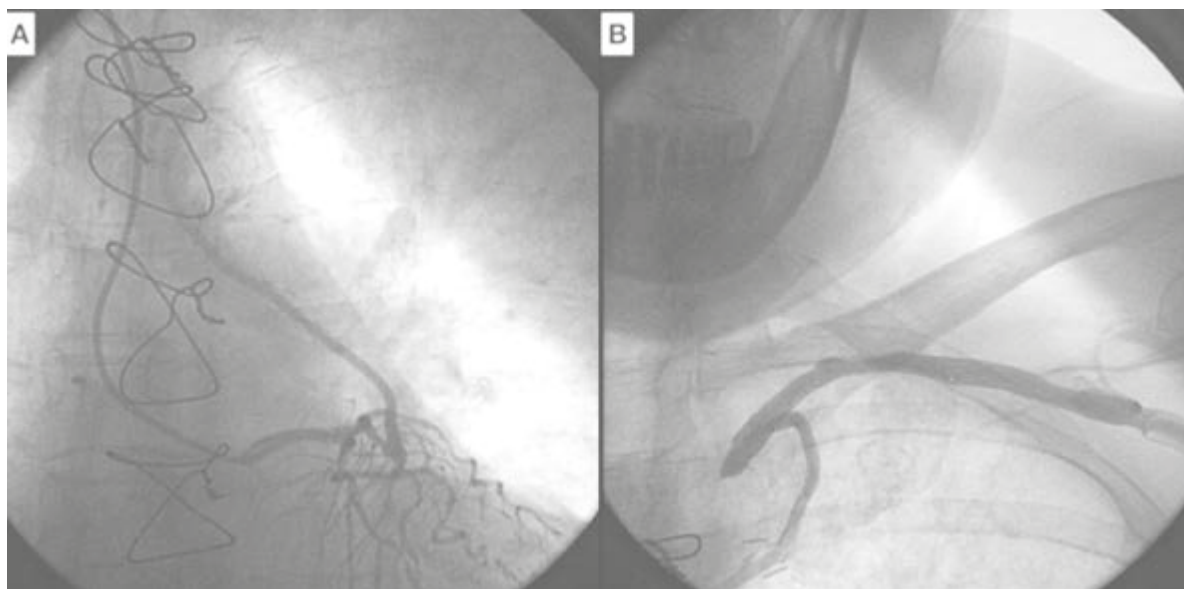


Figure 1. A) and B). Cine coronary arteriography showing reverse flow from the LIMA towards the left subclavian artery

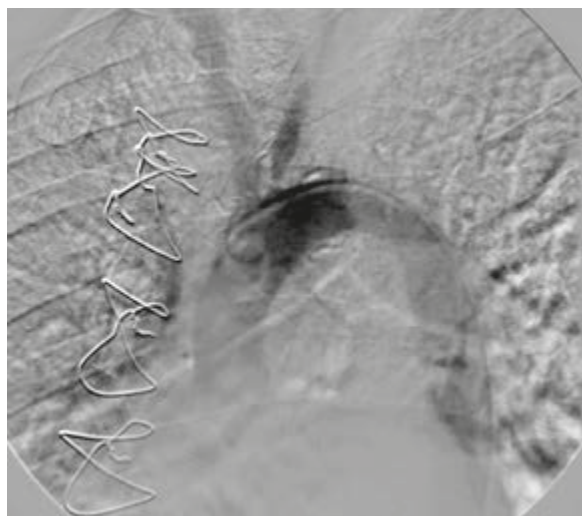


Figure 2. Thoracic digital subtraction angiography showing a left common carotid artery with a heavily calcified occlusion and left subclavian artery occlusion.

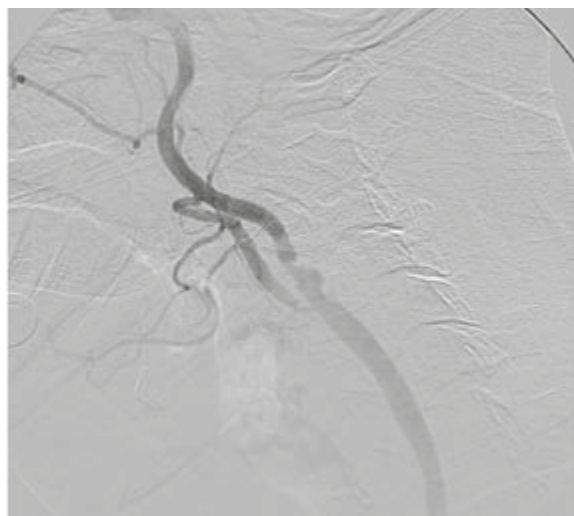


Figure 3. Digital subtraction angiography of neck vessels showing a left internal carotid artery with a heavily calcified occlusion.

a 9 mm x 38 mm balloon-expandable PTFE vascular peripheral covered stent (Advanta V12 Balloon Expandable Covered Stent, Atrium Medical Corporation, United States) (**Figures 4 A and B**). After the initial procedure, the patient's progression was good, and she was discharged 24 hours later on dual antiplatelet therapy (aspirin and clopidogrel) plus antihypertensive and lipid-lowering drugs. At a second stage, and since the patient showed signs of homolateral facial paralysis, a left common carotid artery angioplasty was scheduled using a 8 mm x 38 mm balloon-expandable PTFE vascular peripheral covered stent for the CCA (Advanta V12 Balloon Expandable Covered Stent, Atrium Medical Corporation, United States) and a left internal carotid artery angioplasty using a 7 mm x 30 mm self-expanding stent (Carotid Wallstent, Boston Scientific, United States) (**Figures 5 A and B**). A cerebral protection device was used to perform this procedure. The decision to choose the balloon-expandable PTFE covered stent to treat the left subclavian artery occlusion at a 1st stage and the left common carotid artery lesion at a 2nd stage was due to the fact that both arteries were heavily calcified, in aor-

to-ostial location, and with risk of dissection. A stent with enough radial strength was used for the proper expansion of the device and higher precision during implantation. Also, making sure that the origin of major blood vessels was not close by so they could be occluded by the stent (the left vertebral artery was already occluded and there was enough distance at the LIMA origin—subclavian PTA—and left carotid bifurcation—left common carotid artery PTA—to anchor the stents without compromising these vessels (7-10). The control cine coronary arteriography performed confirmed the correct antegrade flow of coronary arteries (**Figure 6**). The patient remained asymptomatic for angina pectoris, dyspnea or similar symptoms at the 6-month follow-up. The Doppler echocardiography of the neck vessels revealed normofunctioning stents with proper implant position and in-stent preserved velocity.

DISCUSSION

Patients with coronary artery disease often have lesions in other vascular territories. Preoperative studies are essential

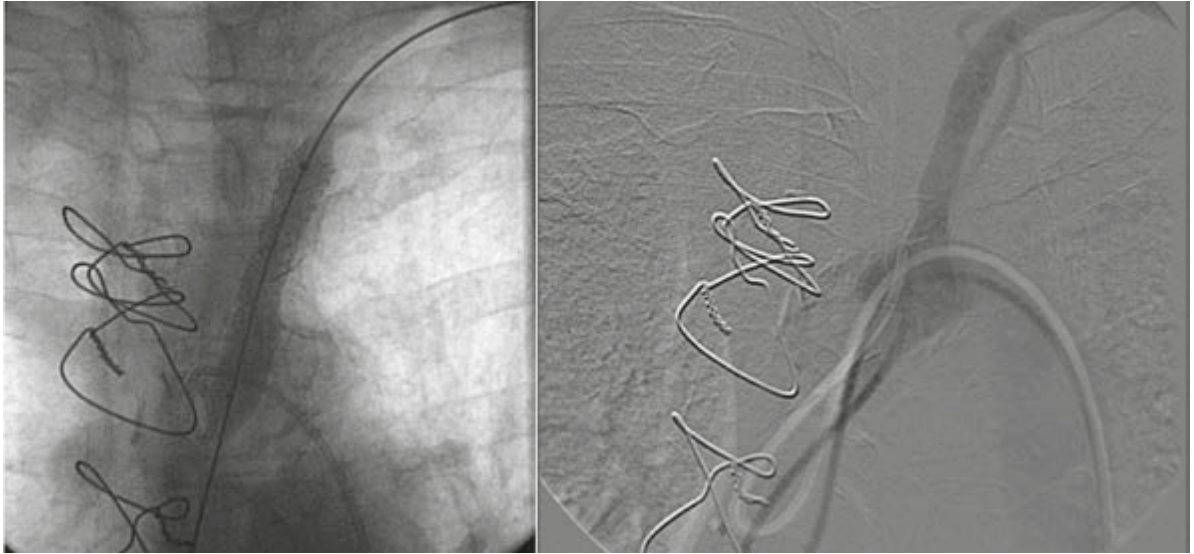


Figure 4. A) Left subclavian artery angioplasty with balloon-expandable endovascular stent-graft. B) The angiographic control confirms the final result of the angioplasty: correct stent positioning, no dissection images, and preserved flow.

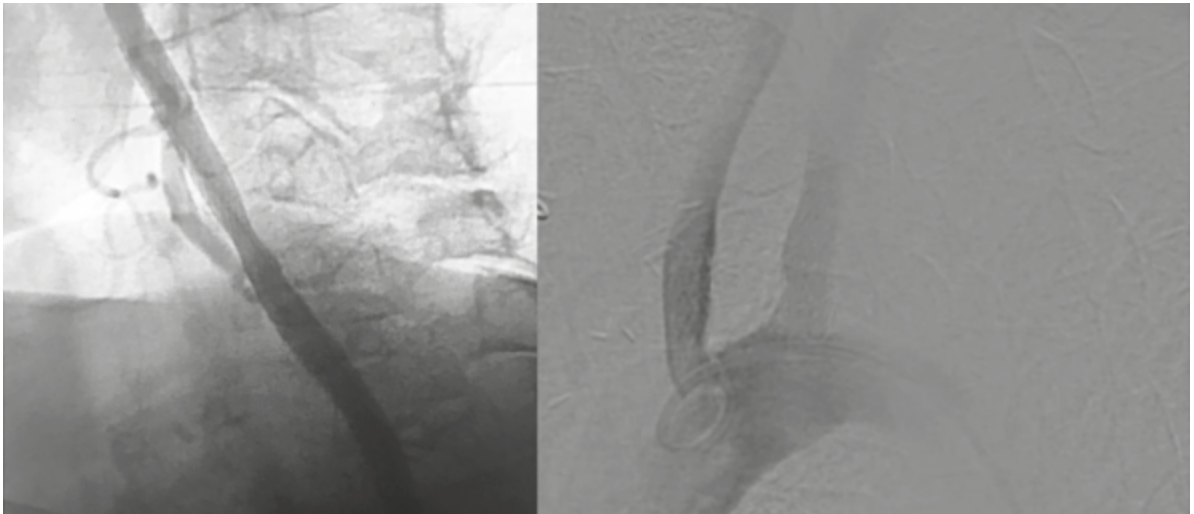


Figure 5. A) Left internal carotid artery angioplasty with self-expanding stent. B) Common carotid and left subclavian arteries angioplasty.

for a correct diagnosis, to omit concomitant conditions, and avoid complications (11). Cua et al. propose an algorithm that can be used in this group of patients (12).

The coronary subclavian steal syndrome was first described by Hargola and Valle more than 30 years ago (1974) (13). It is due to the presence of subclavian artery stenosis before the origin of the vertebral artery, which generates the retrograde flow from the ipsilateral vertebral artery (14-16). The presence of reverse flow in the left mammary artery when the contrast agent is injected into the left coronary artery is a definitive angiographic sign of coronary subclavian steal syndrome (17). The etiology of this rare entity (0.6% to 6.4%) (6) is atherosclerosis, vasculitis, temporal arteritis, and embryonic changes of the aortic arch (18,19). The clinical signs are cardiovascular symptoms (asymmetric pulses and AHT between both upper limbs, myocardial ischemia), neurological symptoms (in 5%, such as vertigo, ataxia, dysarthria, syncope, and binocular vision dysfunction) (20) and claudication of the diseased limb. A large number of these patients remain asymptomatic (21). There are different kinds of subclavian steal: type 1 (subclavian artery or brachiocephalic trunk proximal stenosis); type 2 (subclavian artery severe

proximal stenosis); type 3 (vertebral artery antegrade flow), and type 4 (subclavian artery antegrade flow). Regarding their severity, they can be categorized into 3 groups: group I (pre-subclavian steal): reduction of vertebral antegrade flow; group II (intermittent/partial/latent): alternate flow – antegrade flow in diastole and retrograde in systole; group III (permanent/advanced): permanent retrograde flow (6).

Diagnosis requires high clinical suspicion. As a matter of fact, the coronary subclavian steal syndrome has been reported in noncardiac surgery patients with previous bypass surgery using the internal mammary artery (22). It should be performed through vascular Doppler echocardiography (the vertebral artery flow reversal is confirmed) (23) cine coronary arteriography, CCTA or angio-MRI guidance (24). Treatment consists of balloon angioplasty and endovascular stent-graft into the occlusion site (25,26).

Traditionally, the surgical approach with prostheses or autologous grafts has been the treatment of choice for this type of patients. It consists of a carotid-subclavian or subclavian-subclavian bypass technique. Today, the development and progression of endoluminal procedures and the advances made with new materials (catheters, prostheses, etc.) fa-

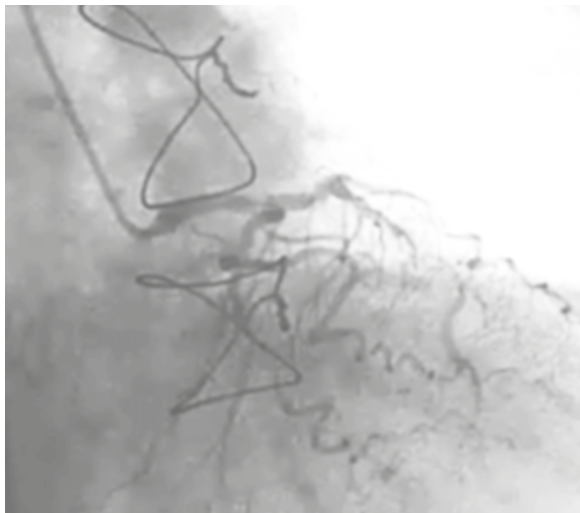


Figure 6. Cine coronary arteriography image at the end of the procedure confirming the presence of proper antegrade flow in coronary arteries, no signs of flow "steal" from the mammary bridge to the left anterior descending coronary artery.

cilitate the use of minimally invasive therapeutic alternatives. Also, they allow us to treat concomitant lesions simultaneously. Angle et al. reported a series of 21 patients with a 9.5% rate of major complications associated with this type of procedures.²⁷ On the other hand, in a comparative study,

Abu Rahma et al. concluded that the effects derived from surgical treatment remain in the long-term; however, the percutaneous approach is still the best option for patients at high surgical risk (28).

The case presented is a clear example of the complexity of patients with concomitant vascular lesions. We should mention that a complete preoperative study is essential to avoid overlooking the presence of stenosis in other arterial territories. Although this condition is rare, its high suspicion and early treatment avoid further complications.

CONCLUSION

The coronary subclavian steal syndrome is a rare entity that can cause myocardial ischemia in patients previously treated for this condition. Neurological symptoms can be associated too. Complete preoperative studies are required to prevent it and high clinical suspicion to diagnose it. Early percutaneous treatment with endovascular stent-grafts resolves it safely and effectively.

Conflicts of interest: None reported.

Funding: None reported.

Informed consent: All patients signed their informed consent forms before participating in the study giving us authorization to use and publish their data.

REFERENCES

1. Takach TJ, Reul GJ, Duncan JM, et al. Concomitant brachio-cephalic and coronary artery disease: outcome and decision analysis. *Ann Thorac Surg* 2005;80:564-569.
2. Olsen KG, Lund C. Subclavian steal syndrome. *Tidsskr Nor Lægeforen*. 2006;126(24):3259-3262.
3. Lak HM, Shah R, Verma BR, et al. Coronary Subclavian Steal Syndrome: A Contemporary Review. *Cardiology*. 2020;145(9):601-607.
4. Kliever MA, Hertzberg BS, Kim DH, et al. Vertebral artery Doppler waveform changes indicating subclavian steal physiology. *Am J Roentgenol*. 2000;174:815-819.
5. Tahmasebpour HR, Buckley AR, Cooperberg PL, Fix CH. Sonographic examination of the carotid arteries. *Radiographics*. 2005;25(6):1561-1575.
6. Osiro S, Zurada A, Gielecki J, et al. A review of subclavian steal syndrome with clinical correlation. *Med Sci Monit*. 2012;18(5):RA57-63.
7. Alaraj A, Wallace A, Amin-Hanjani S, et al. Endovascular implantation of covered stents in the extracranial carotid and vertebral arteries: Case series and review of the literature. *Surg Neurol Int*. 2011;2:67.
8. Hoppe H, Barnwell SL, Nesbit GM, et al. Stent-grafts in the treatment of emergent or urgent carotid artery disease: review of 25 cases. *J Vasc Interv Radiol*. 2008;19:31-41.
9. Layton KF, Kim YW, Hise JH. Use of covered stent grafts in the extracranial carotid artery: Report of three patients with follow-up between 8 and 42 months. *AJNR Am J Neuroradiol*. 2004;25:1760-1763.
10. Saket RR, Razavi MK, Sze DY, Frisoli JK, Kee ST, Dake MD. Stent-graft treatment of extracranial carotid and vertebral arterial lesions. *J Vasc Interv Radiol*. 2004;15:1151-1156.
11. Waduud MA, Giannoudi M, Drozd M, et al. Coronary subclavian steal syndrome-is there a need for routine assessment for subclavian artery stenosis following coronary bypass surgery? *Oxf Med Case Reports*. 2018;2018(12):omy102.
12. Cua B, Mamdani N, Halpin D, et al. Review of coronary subclavian steal syndrome. *J Cardiol*. 2017;70(5):432-437.
13. Hargola PT, Valle M. The importance of aortic arch or subclavian angiography before coronary reconstruction. *Chest* 1974;66:436-438.
14. Potter BJ, Pinto DS. Subclavian steal syndrome. *Circulation*. 2014;129(22):2320-2323.
15. Moccetti F, Brinkert M, Wolfrum M, et al. Coronary subclavian steal syndrome. *Eur Heart J*. 2020;41(13):1345.
16. Fonseka N, Dunn J, Andrikopoulou E, et al. Coronary subclavian steal syndrome. *Am J Med*. 2014;127(8):e11-12.
17. Álvarez J, Urda T, Mancha I, et al. Angina caused by coronary-subclavian steal in patients with coronary artery bypass with internal mammary artery. *Rev Esp Cardiol* 1998;51:772-775.
18. Antón Vázquez V, Armario García P, García Sánchez SM, et al. Subclavian steal syndrome: A forgotten aetiology of acute cerebral ischaemia. *Neurologia*. 2020;35(1):65-67.
19. Kargiotis O, Siahos S, Safouris A, et al. Subclavian Steal Syndrome with or without Arterial Stenosis: A Review. *J Neuroimaging*. 2016;26(5):473-480.
20. Shadman R, Criqui MH, Bundens WP, et al. Subclavian artery stenosis: prevalence, risk factors, and association with cardiovascular diseases. *J Am Coll Cardiol*. 2004;44(3):618-623.
21. Labropoulos N, Nandivada P, Bekelis K. Prevalence and impact of the subclavian steal syndrome. *Ann Surg*. 2010;252(1):166-170.
22. Lobato EB, Kern KB, Bauder-Heit J, et al. Incidence of coronary-subclavian steal syndrome in patients undergoing noncardiac surgery. *J Cardiothorac Vasc Anesth*. 2001;15(6):689-92.
23. Harper C, Cardullo PA, Weyman AK, et al. Transcranial Doppler ultrasonography of the basilar artery in patients with retrograde vertebral artery flow. *J Vasc Surg*. 2008;48(4):859-864.
24. Toprak C, Yesin M, Mustafa Tabakci M, et al. Coronary Subclavian Steal Syndrome Evaluated with Multimodality Imaging. *Intern Med*. 2015;54(21):2717-2720.
25. Usmanji EA, Senden PJ, Meiss L, et al. Myocardial ischaemia due to subclavian stenosis after coronary artery bypass graft: a case report. *Eur Heart J Case Rep*. 2018;2(2):yry069. Che W, Dong H, Jiang X, et al. Subclavian artery stenting for coronary-subclavian steal syndrome. *Catheter Cardiovasc Interv*. 2017;89(S1):601-608.
26. Angle JF, Matsumoto AH, McGraw JK, et al. Percutaneous angioplasty and stenting of left subclavian artery stenosis in patients with left internal mammary-coronary bypass grafts: clinical experience and long-term follow-up. *Vasc Endovascular Surg*. 2003;37(2):89-97.
27. AbuRahma AF, Bates MC, Stone PA, et al. Angioplasty and stenting versus carotid-subclavian bypass for the treatment of isolated subclavian artery disease. *J Endovasc Ther*. 2007;14(5):698-704.

Six-year angiographic follow-up of a chronic total coronary occlusion successfully treated with BMS implantation followed by oral rapamycin

Seguimiento angiográfico a 6 años de un paciente con oclusión total crónica tratada con múltiples stents convencionales más rapamicina oral por 13 días

Juan Mieres¹, Hernán Pavlowsky¹, Diego Ascarrunz¹, Alfredo Matías Rodríguez-Granillo¹, Carlos Fernández-Pereira¹

ABSTRACT

This is the case of a 76-month angiographic follow-up of a male patient with CTO (total chronic coronary occlusion) treated with a combined strategy of percutaneous coronary angioplasty (PCI) with multiple conventional stents (BMS) plus oral rapamycin (OR) for 13 days after the PCI. The patient had several risk factors for in-stent restenosis (ISR) including metabolic syndrome, a long segment of the CTO, small-diameter blood vessels, and BMS overlapping. The patient was treated with 3 BMSs making a total stent length of 64 mm with a minimal lumen diameter post-implantation of 1.82 mm and a reference diameter of 2.71 mm. The patient remained asymptomatic for 75 months. After this time, he showed CF dyspnea type III, which could be attributed to overweight. After several functional studies an angiographic study is decided that shows the patency of the stents in all their segments without ISR and a minimal lumen diameter of 1.61 mm, which is indicative of a late loss of 0.21 mm. The vasoreactivity testing performed with adenosine looked normal showing a minimal lumen diameter of 2.24 mm after infusion. This is a brief review of this PCI strategy.

Keywords: oral rapamycin, bare metal stents, in-stent restenosis, drug eluting stents, chronic total coronary occlusion.

RESUMEN

Presentamos un seguimiento angiográfico de 76 meses en un paciente con oclusión total crónica (CTO), tratado con una estrategia combinada de angioplastia coronaria percutánea (PCI) con múltiples stents convencionales (BMS) más rapamicina por vía oral (RO) durante 13 días post-PCI. El paciente presentaba varios factores de riesgo de restenosis intrastent (ISR), incluyendo síndrome metabólico, un largo segmento de CTO, vasos de fino diámetro y BMS en overlapping. Este fue tratado con tres BMS que dan una longitud del stent de 64 mm, un diámetro luminal mínimo posterior a la implantación de 1,82 mm con un diámetro de referencia de 2,71 mm. El paciente estuvo asintomático por 75 meses tras lo cual presenta una disnea CF III, que podría ser atribuida a sobrepeso. Después de estudios funcionales se decide su estudio angiográfico observándose la permeabilidad de los stents en todos sus segmentos sin ISR, con diámetro mínimo luminal de 1,61 mm, lo que significa una pérdida tardía de 0,21 mm. Se realizó el análisis de vaso-reactividad con adenosina, que fue normal y un diámetro luminal mínimo de 2,24 mm posinfusión. Presentamos una breve revisión de esta estrategia de PCI.

Palabras claves: rapamicina oral, stents convencionales, restenosis, stents liberadores de fármacos, oclusión total crónica.

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HISTORY

Yet despite the introduction of the modern technology of drug-eluting stents (DES), over the last decade we have seen a reduction of revascularization procedures including the most common one in the United States (1), the percutaneous coronary intervention (PCI).

Also, in landmark randomized clinical trials (RCT) that compared DES and myocardial revascularization surgery (MRS) with long-term follow-up periods, a higher rate of spontaneous acute myocardial infarction (AMI) after 30 days was consistently observed with the PCI compared to the MRS (2-5). As a matter of fact, and yet despite the fewer target vessel revascularizations compared to BMS implantations within the first year following the PCI, this difference did not grow any shorter. Actually, there was a higher rate of adverse events with DES, suggestive of late loss of efficacy in

DES compared to BMS and MRS (6-8). The reasons for these findings are still under discussion.

Also, in the largest RCT of second-generation DES vs BMS, no statistically significant differences were seen between both revascularization strategies (8).

Back in 2014, we disclosed the clinical findings at the 5-year follow-up of a RCT with BMS implantation followed by oral rapamycin (OR). This trial confirmed a reduction of late cardiovascular adverse events. Also, it was cost-effective when compared to first-generation DES (9). These results are consistent with other RCTs (10-11); however, no definitive conclusions were drawn from these small sample studies and this strategy was not included in the clinical practice guidelines.

The objective of this case presentation is to show the findings of a patient with long-term angiographic follow-up. This patient had multiple factors for ISR and was treated with a combination of BMS and OR for 2 weeks.

CASE PRESENTATION

This is the case of a 67-year-old male patient who presented with progressive angina, arterial hypertension, dyslipidemia, obesity with metabolic syndrome, maximum values of blood sugar levels, and former heavy smoker. The patient was admitted to our center on October 4, 2013. He presented

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ted with progressive angina of 1-month duration. The functional test performed confirmed the presence of anterior and lower extensive ischemia that suggested that territories from the left anterior descending coronary artery (LAD) and right coronary artery (RCA) had been compromised. Therefore, a cine coronary arteriography was performed (**Figure 1**).

The cine coronary arteriography revealed the presence of a large segment of the CTO from the LAD proximal third. The left circumflex artery (LCx) showed an intermediate injury (50% through visual estimation) in the middle third. The RCA also showed the presence of a CTO in the proximal third (**Figure 1 A, B, C, and D**). The SYNTAX score was 33 while the ERACI score, 22 (2,12). Combined therapy with immunosuppressants and BMS was decided and agreed with the patient and his general physician. Based on our own experience (9,10) the patient received a loading dose of 10 mg of oral rapamycin that was administered when the PCI was performed. Afterwards, the antegrade recanalization of the LAD with a Runthrough NS intermediate guidewire (Terumo, Japan) was attempted using 1.5 mm and 2.0 mm Ryujin support balloons (Terumo, Japan). Both the reference diameter (RD) and the minimal lumen diameter (MLD) measured with the quantitative coronary angiography (QCA) after the plain balloon angioplasty (PBA) were 2.43 mm, and 0.28 mm respectively (**Figure 2 A, B, and C**). Then, three 3.0 mm x 20 mm, 2.5 mm x 20 mm, and 2.25 mm x 24 mm BMSs (Liberté Boston Scientific, Massachusetts United States) were implanted with an overall stent length of 64 mm. The RD and DLM after stent implantation were 2.19 mm and 1.61 mm, and the DLM increased with nitroglycerin to 1.82 mm (**Figure 3 A, B, C, and D**). The procedure ended with a PBA performed in a diagonal branch. After the PCI performed on the LAD, we saw that the RCA filled through collateral circulation. Both the moderate injury of the left circumflex artery and the CTO of the RCA were not operated. Instead, they were treated with medical therapy (**Figure 1**).

After the PCI, the patient received aspirin, atorvastatin, and bisoprolol indefinitely. Three mg per day of OR were indicated for a total of 13 days after the PCI. The patient also received a 75 mg dose of clopidogrel for 3 months. Lifestyle changes and a strict control of the sugar levels were recommended. The patient remained asymptomatic for a long period of time with myocardial perfusion controls without ischemia.

Six years after the procedure, in February 2020, the patient showed variable dyspnea of 1-month duration, lower limb swelling, and overeating. At that time the sugar blood levels were 119 mg/dL. The patient's myocardial perfusion study revealed the presence of moderate lower limb and apical ischemia without changes on the electrocardiogram. Therefore, a cine coronary arteriography is performed (**Figure 4 A and B**) that shows no significant injuries on the LAD. The QCA showed a 2.19 mm and 1.61 mm RD and DLM, respectively, with a total loss at the 76-month follow-up of 0.21 mm. The filling of the RCA occurred through collateral circulation without changes to the LCx injury (**Figure 4 A and B**).

The vascular vasoreactivity testing performed with adenosine (dose of 140 µg/kg/min) through a central venous catheter confirmed the improvement of the DLM and the RD that measured 2.34 mm and 2.56 mm post-adenosine, respectively, indicative of normal vasoreactivity (figure 4 C and D).

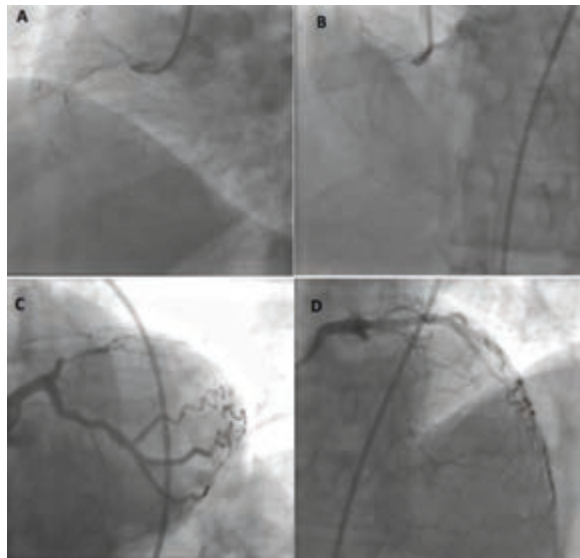


Figure 1. A) and B): LAO projection with cranial angulation showing a CTO from the RCA proximal third. C) and D): RAO and LAO caudal projections showing the long CTO segment of a LAD. LAD: left, anterior descending coronary artery. LAO: left anterior oblique. RAO, right anterior oblique. RCA: right coronary artery.

The patient's risk factors were strictly controlled and followed by salt restriction, and physical exercise recommendations. The patient was monitored 1 month later and remained asymptomatic until the last control was performed 6 months later.

DISCUSSION

This was the case of a patient who was recanalized due an entire LAD CTO from its proximal to its distal segments with 3 BMSs plus a 2-week course of OR. The patient also showed a CTO on the RCA, moderate injuries to the LCx, and received conservative therapy. Six years later a new cine coronary arteriography is performed that reveals the patency of the LAD without ISR in any of the stents implanted.

This patient had multiple clinical and angiographic risk factors for ISR like metabolic syndrome, presence of a CTO, extensive occluded segment, fine reference diameter, and multiple BMSs. Yet despite all this, the patient remained asymptomatic and ISR-free for 76 months after the initial angioplasty with normal vasomotility after the injection of adenosine.

The findings of this case should not be surprising since numerous small randomized clinical trials that compared immunosuppressant therapy to anti-inflammatory therapy administered orally after the implantation of a BMS have consistently proved the benefits of reducing ISR (9-11).

Also, in a randomized clinical trial that compared OR and BMS vs first-generation DES, the ORAR II trial, we proved that, at the 5-year follow-up (9), this strategy was cost-effective compared to first-generation DES, at all time, in 200 patients including diabetics (28%), small vessels (36%), and multiple vessel disease (48%). The composite of death, spontaneous AMI, and stroke at the 5-year follow-up showed a RR = 2.08, 1.10-3.91 P = .01 favorable to the OR/BMS strategy. Still, this difference in the composite endpoint was similar at the 1-year (RR = 0.60, 0.27-1.30 P = .34) and 3-year follow-up (RR = 0.49, 0.22-1.09 P = .08) suggestive that DES have a greater efficacy

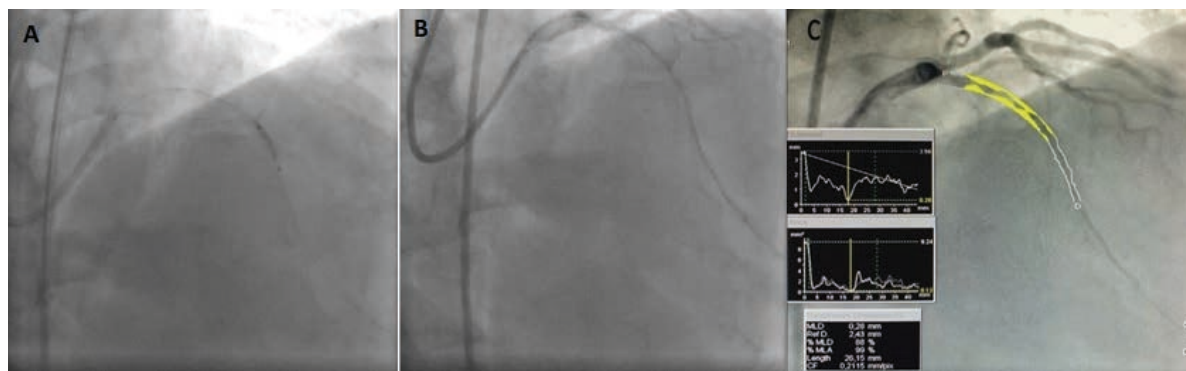


Figure 2. A) and B): recanalization and plain balloon angioplasty (PBA). C): QCA after PBA. QCA: quantitative coronary angiography.

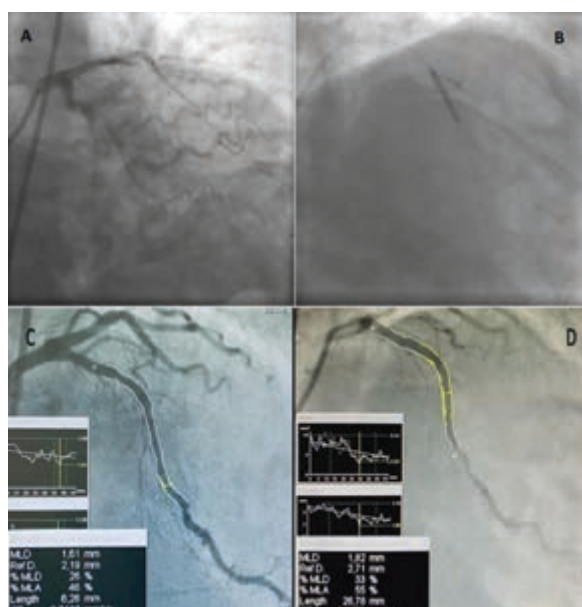


Figure 3. A) and B): Stent implantation in the LAD 3 thirds. C) and D): QCA of LAD segments before and after the infusion of nitroglycerin.

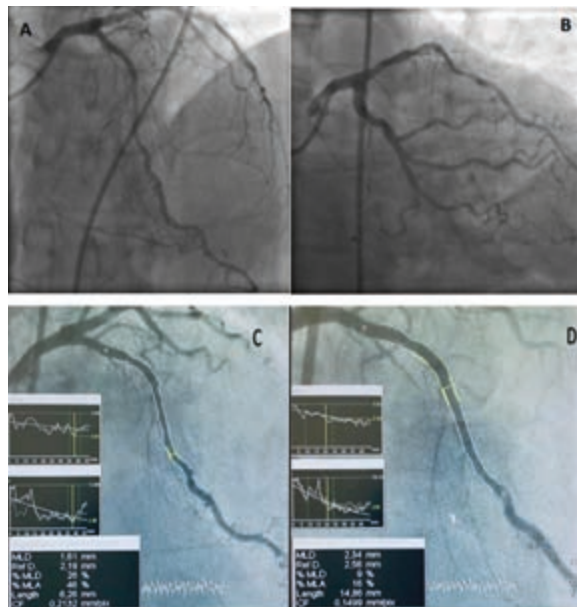


Figure 4. A) and B): RAO projection with cranial angulation and RAO projection of the LAD showing the patency of the stents and with collateral circulation to the RCA. C) and D): QCA of the LAD before and after the infusion of adenosine.

loss over time. By the time the patient was being treated, the 5-year follow-up findings of this clinical trial had already been submitted for publication (9). An interesting finding of this last study was that the stent overlapping rate of every patient was 31%.

We should mention that despite the significant safety increase of the latest DES designs, the presence of early neoatherosclerosis and endothelial dysfunction are still important limiting factors for the long-term evolution of these devices. This may be due to the polymer used, but mostly to the local action of the immunosuppressant drug, and the fact that they are responsible for the cardiovascular and noncardiac adverse events reported in the long-term (2-5,13,14). A recent and extensive meta-analysis of 23 RCTs reported, for the first time, an increase of cardiovascular and noncardiac mortality associated with the angioplasty when DES and

MRS studies were compared. However, no differences between BMS and MRS (15) were found, which may be indicative of a specific correlation between noncardiac mortality and the use of DES.

As long as these 2 problems with the technology of DES remained unsolved, early neoatherosclerosis, and endothelial dysfunction, strategies like the one described here should not be ruled out.

CONCLUSIONS

In this case of an extremely long follow-up of a patient with multiple angiographic and clinical risk factors for ISR, we saw favorable disease progression with complete patency of the blood vessel treated including normal vessel reactivity.

REFERENCES

1. Alkhouli M, Alqahtani F, Kalra A, et al. Trends in Characteristics and Outcomes of Patients Undergoing Coronary Revascularization in the United States, 2003-2016. *JAMA Netw Open*. 2020;3(2): e1921326. Published 2020 Feb 5.
2. Mohr FW, Morice MC, Kappetein AP, et al. Coronary artery bypass graft surgery versus percutaneous coronary intervention in patients with three-vessel disease and left main coronary disease: 5-year follow-up of the randomized, clinical SYNTAX trial. *Lancet* 2013;381(9867):629-38.

3. Chang M, Ahn JM, Lee CW, et al. Long-Term Mortality After Coronary Revascularization in Nondiabetic Patients with Multivessel Disease. *J Am Coll Cardiol* 2016;68(1):29-36.
4. Holm NR, Mäki-Kallio T, Lindsay MM, et al. NOBLE investigators. Percutaneous coronary angioplasty versus coronary artery bypass grafting in the treatment of unprotected left main stenosis: updated 5-year outcomes from the randomised, non-inferiority NOBLE trial. *Lancet* 2020;395(10219):191-9.
5. Stone GW, Kappetein AP, Sabik JF, et al. EXCEL Trial Investigators. Five-Year Outcomes after PCI or CABG for Left Main Coronary Disease. *N Engl J Med*. 2019;381(19):1820-30.
6. Piccolo R, Bona KH, Efthimiou O, et al. Coronary Stent Trialists' Collaboration. Drug-eluting or bare-metal stents for percutaneous coronary intervention: a systematic review and individual patient data meta-analysis of randomised clinical trials. *Lancet* 2019 Jun 22;393(10190):2503-10.
7. Park DW, Ahn JM, Yun SC, et al. Ten-Year Outcomes of Stents versus Coronary-Artery Bypass Grafting for Left Main Coronary Artery Disease. *J Am Coll Cardiol* 2018;72(23 Pt A):2813-22.
8. Bønaa KH, Mannsverk J, Wiseth R, et al. NORSTENT Investigators. Drug-Eluting or Bare-Metal Stents for Coronary Artery Disease. *N Engl J Med* 2016;375(13):1242-52.
9. Rodríguez AE, Palacios I, Rodríguez-Granillo AM, et al. ORAR-3 investigators. Comparison of cost-effectiveness of oral rapamycin plus bare-metal stents versus first generation of drug-eluting stents (from the Randomized Oral Rapamycin in Argentina [ORAR] 3 trial). *Am J Cardiol* 2014;113(5):815-21.
10. Cassese S, De Luca G, Ribichini F, et al. ORAI iMmunosuppressive therapy to prevent in-Stent rEstenosis (RAMSES) cooperation: a patient-level meta-analysis of randomized trials. *Atherosclerosis* 2014;237(2):410-7.
11. Deftereos S, Giannopoulos G, Raisakis K, et al. Colchicine treatment for the prevention of bare-metal stent restenosis in diabetic patients. *J Am Coll Cardiol* 2013;61(16):1679-85.
12. Rodríguez AE, Fernández-Pereira C, Mieres J, Santaera O, Antoniucci D; ERACI IV investigators. Modifying angiographic syntax score according to PCI strategy: lessons learnt from ERACI IV Study. *Cardiovasc Revasc Med* 2015;16(7):418-20.
13. Park SJ, Kang SJ, Virmani R, Nakano M, Ueda Y. In-stent neoatherosclerosis: a final common pathway of late stent failure. *J Am Coll Cardiol* 2012;59(23):2051-7.
14. Toya T, Sara JD, Corban MT, et al. Assessment of peripheral endothelial function predicts future risk of solid-tumor cancer. *Eur J Prev Cardiol* 2020;27(6):608-18.
15. Gaudino M, Hameed I, Farkouh ME, et al. Overall and cause-specific mortality in randomized clinical trials comparing percutaneous interventions with coronary bypass surgery. A Meta-analysis. *JAMA Intern Med* 2020 Oct 12;e204748.

Closure of a different and very special year

Cierre de un año distinto y muy especial

Revista Argentina de Cardioangiología Intervencionista 2020;11(4):213-214. <https://doi.org/10.30567/RACI/202004/0213-0214>

Dear colleagues and members of the Argentine College of Interventional Cardioangiologists (CACI), I wish to take this opportunity, say “hi” to all of you, and brief you on everything that has been going on in our college during this very special year we’ve had.

Despite the pandemic, we were able to hold our Congress remotely. After asking for different budgets we chose the company LANZILOTA to launch our Congress, which was very successful thanks to all your efforts.

Regarding fees, CACI Fees Commission periodically updated the costs of our medical practice and the new Professional Commission was created.

Due to the COVID-19 pandemic and the work and income decrease sustained by our members we decided not to make inflation-based adjustments in the fees and quotas of the college.

In 2020 we held the V CACI Meeting for Medical Auditors and Sponsors that was coordinated by Dr. Jorge Leguizamón with the collaboration of medical auditors from different provinces of our country and discussed topics on myocardial infarction, calcified coronary artery disease, aortic and mitral valvular heart disease, and intracardiac imaging. We held the first CACI workshop with industry specialists on the reality of valvular implants in Argentina. The response from the audience was just great.

We also held the first CACI imaging workshop with Philips, Siemens, and General Electric. The response from the audience was also great.

The Multisocietal Consensus of the Pelvic Congestion Syndrome was finally achieved.

CACI Consensus document and recommendations were achieved for the management of COVID-19.

The SAC, FAC, CONAREC, the Argentine Health Ministry, and other medical societies published the National Guidelines on the Management of Acute Myocardial Infarction.

CACI has kept its presence and agreements signed with different societies (SOLACI, SAC, FAC, TCT, PCR, India Live, C3, CIT) and added a new Latin American society, HENDOLAT. We have shared live case webinars and participated in their annual virtual congress with all of them.

The SOLACI-CACI Congress that should have been held in Buenos Aires, Argentina, in August 2020, was postponed until August 2021 in full compliance with SOLACI and SOCIME.

Regarding our scientific journal, *RACI*, we have supported it and kept its regular publication both digital (through our website) and in print.

The translation of the annual issues of the journal is conducted by Prof. Jorge C. Berriatúa from Spain. There is no doubt that this will improve the possibilities of indexation and increase the visualization of *RACI* and CACI thanks to the great work of *RACI* Director, Dr. Alfredo Rodríguez.

Despite the limitations and restrictions imposed by the pandemic, the Education Department kept offering classes to become a qualified specialist (period 2017-2020).

The specialist degree 2020-2023 was started. The Professional Update Course was started and remotely welcomed by our audience. The I Course of CACI Technicians and Nurses was held. I wish to thank all the efforts made by the Education Department to achieve these goals.

The fourth volume of the *Libro de Hemodinamia y Cardioangiología Intervencionista* was completed. It represents the huge effort made by all the authors and editors led by Dr. Marcelo Ruda Vega who perfects it on an ongoing basis. We have worked side by side with the recently created Professional Commission.

Back in December 2019 shortly after taking over the CACI Board of Directors, we had a serious problem with the PAMI regarding the mandatory referral of patients from the interventional cardiology unit to César Milstein Hospital. Eventually, the problem was solved in a short span of time and the PAMI regulation that caused the problem was revoked.

Due to the pandemic, several members who work for the CABA network of public hospitals and for some inland cities had to remain on call and assist COVID-19 patients in closed hospital areas. Their work at the cath lab was, therefore, seriously jeopardized. However, a letter sent from the CACI Board of Directors to the Argentine Ministry of Health reversed this situation and our members were able to go back to their jobs at the cath labs without having to assist these COVID areas anymore.

Other labor issues sustained by our members and different health entities were also worked out by our association.

The CACI Board of Directors ratified the Agreement of collaboration and inter-consultation signed by both entities. The II CACI-ANMAT Conference coordinated by Dr. Marcelo Halac was held remotely with the participation of ANMAT executive directors and the presence of Dr. Pablo Yedlin, national congressman and president of the Na-

tion Congressional Health Commission, members of the CACI-ANMAT commission, CACI members, and industry participants.

During 2019 the R+D Area directed by Dr. David Vetcher came up with an initiative that several members had suggested—the project of creating CACI R+D Area—that opened up the possibility of keeping relations with the ICI. Following in the early footsteps of this initiative I hereby proudly announce the first edition of Innovation in Cardiovascular Interventions in our country organized by ICI and CACI (2020). It will eventually take place one day in advance prior to the SOLACI CACI Congress of 2021. Through ICI and CACI R+D Area the very first course on innovation will be held in 2021 with support from ICI and in close collaboration with CACI Education Department. In 2020 we also created new commissions: The Professional Commission was created after hearing the voices of many members who wished to reinforce the professional side of CACI. This Professional Commission includes CACI Professional Secretary and CACI Fees and Social Works Commissions in a combined effort to improve quality and compensation in our clinical practice. This new commission is quarterbacked by its coordinator, Dr. Javier Woscoboinik. This Professional Commission signed a reciprocal agreement with the Argentine Association of Anesthesiology (also to be signed by other societies) to increase the professional competence of the commission and have more negotiation power with the insurance companies.

An agreement with CADIME was also signed to join this organization and improve our members' union rights and working schedules. Also, to be able to bill the contrast agents used separate from the imaging modalities *per se*.

The Women Interventional Cardiologists Commission was created as well. It will be working together with LATAM Women Interventional Cardiologists and Women as One with Dr. Carla Agatiello as coordinator.

Together with SOLACI we held the first Argentine women interventional cardiologist workshop, supported by Women as One and LATAM Women Interventional Cardiologists, including a webinar where top international female interventional cardiologists participated.

I wish to thank Dr. Carla Agatiello for her hard work on this regard.

Although our college offices were closed since March 2020 following the social, preventive and mandatory isolation decree issued by the nation's government, we never stopped working remotely. Also, we bought notebooks for all of our employees so they could work from the safety of their homes.

Despite all the limitations and restrictions imposed by the COVID-19 pandemic, we have been able to conduct all activities and fulfill all the promises made when this Commission started working back in the month of December. Also, we have been able to keep our funding as it was. Although we had to let some sponsors go, we welcomed new companies under a new and special COVID-19 category. Also, CACI paid all paychecks, invoices, and debts in timely manner.

We also conducted webinars with partner companies that, unexpectedly, ended up funding our activities right from the start.

Our college is now more visible than ever through mass and social media and we managed to sign several agreements with different international scientific societies.

COVID-19 took the lives of two of our colleagues, **Dr. Vicente Vargas** and **Dr. Eduardo Chort**, and several members contracted this disease.

I wish to thank each and every one of you and wish that 2021 will find us stronger and give us the opportunity to meet again, in person this time.

Diego Grinfeld
President of CACI 2020 – 2021

Publication Guidelines of the *Revista Argentina de Cardioangiología Intervencionista*

Reglamento de Publicaciones de la *Revista Argentina de Cardioangiología Intervencionista*

The *Revista Argentina de Cardioangiología Intervencionista (RACI)* is a quarterly journal published by the Argentinian College of Interventional Cardiologists (CACI). Its goal is to spread scientific and educational material on this medical specialty. Distribution is nation wide and open-access and is targeted at interventional cardiologists, clinical and pediatric cardiologists, radiologists, neurologists, operators, and other specialists. The publication is both digital (www.caci.org.ar) and in print.

The editorial principles of the journal are based on the Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly Work in Medical Journals and have been written by the International Committee of Medical Journal Editors - ICMJE in its most recent iteration available online at www.icmje.org.

For editorial reasons starting with issue #2, volume 9, year 2018 the graphic elements of the journal (figures, tables, and pictures) will be published in two colors only (blue and black). Readers who wish the full-color edition will need to pay an additional US\$200.

The articles submitted to the journal shall be originals. The Editorial Committee will study the papers submitted and confirm whether they follow the Publication Guidelines established by the journal. The Director, and/or Associate Directors will be responsible for submitting these papers for the external blind peer review process. This means that the authors do not know the reviewers' name and the reviewers do not know the name of other reviewers. This policy established by RACI follows the same criteria implemented by the Review and Editorial Committee of the *Journal of the American College of Cardiology (JACC)*, the highest impact factor cardiology journal. The Editorial Committee will make the final publication decision in accordance with the conclusions drawn by blind peer reviewers. Similarly, the Editorial Committee can introduce grammar related editorial changes according to the publication needs of the journal always after obtaining prior consent from the authors. Review articles and editorials will be subject to the same review process. Editorials are often required by the Editorial Committee as well. After the first review, the articles can be accepted in the same form they were initially submitted; minor reviews are those pertaining to articles with significant contributions that still have some minor limitations that need to be corrected or proof read before its eventual publication; major reviews are those pertaining to articles that are unfit for publication as originally submitted to the journal. In any case, the Editorial Committee can consider new submissions called *de novo* submissions as long as the article is modified substantially; the rejection of the article occurs when both the reviewers and the Editorial Committee deem the article unfit for publication in the RACI journal.

In special cases of diagnostic and/or treatment consensus achieved by CACI and related scientific societies combined, such consensus will be supervised by the latter and being the Editorial Committee fully aware. Only then this consensus can be published exceptionally by the official journals of both societies simultaneously.

INSTRUCTIONS TO AUTHORS AND GUIDELINES FOR MANUSCRIPT SUBMISSION

All authors and members from the Editorial Committee shall declare any conflicts of interest associated with the publications

Each article shall be presented with a first page that should include: (a) title (both informative and precise); (b) the complete names of the authors and centers involved in the writing of the manuscript; (c) a short version of the title for the runner head; (d) the total amount of words contained in the paper excluding the references; (e) the name and full address, fax, and e-mail address of the corresponding author. The second page will include the abstract in Spanish and English with 3-6 keywords at the end of both abstracts with terms from the Index Medicus term list (Medical Subject Headings - MeSH). The third page will carry the content of the manuscript (see Preparation of the manuscript) including a new page per section. All pages will be numbered from the title page.

The paper (text, tables, and figures) will be submitted electronically to the following e-mail address revista@caci.org.ar with a note signed by all authors (see model in website) with the name of the section the manuscript belongs to, and a clear statement that the contents of the manuscript have never been published before.

Those appearing as authors of the article need to have contributed to the study or writing of the manuscript and will be liable for the content published.

A maximum of eight (8) authors shall be allowed in each paper and they must follow the authorship standards established by the IMCJE. Each manuscript received is examined by the Editorial Committee and one or two external reviewers. Afterwards, the lead author will be notified on the acceptance (with or without corrections and changes) or rejection of the manuscript. After the article has been approved for publication, RACI has the copyright for its total or partial reproduction.

SECTIONS (See Preparation of the manuscript)

Original articles

These are scientific or educational papers of original basic or clinical studies. Requisites: a) general text, up to 5000 words

including references; b) abstract, up to 250 words; c) tables + figures, up to 8; d) authors, up to 10.

Brief communications

The studies published under this section follow the same criteria established for original articles, but do not have enough patients to be considered as such.

Review articles

These are articles on relevant issues on the specialty requested by the Editorial Committee to renowned authors (whether foreign or domestic). They can be written by different types of doctors (no more than 3 different authors). Requisites: the same ones established for the publication of original articles.

Continuing medical education

These are articles on the rational and protocolized management of the different circumstances that can occur in the routine clinical practice. They are reviewed and agreed previously with subject matter experts and include a flow chart on the diagnostic and therapeutic management of the disease. The following requisites have been established by the Editorial Committee. Requisites: a) general text, up to 2500 words excluding the references; b) abstract, up to 150 words; c) tables + figures, up to 6; d) references, up to 20; e) authors, up to 4.

Clinical case

This is the description of a clinical case of unusual characteristics with its diagnostic and therapeutic management, and final resolution. It needs to include a brief reference search. Requisites: a) general text, up to 1200 words; b) abstract, up to 100 words; c) tables + figures, up to 4; d) references, up to 10; e) authors, up to 5.

How did I approach it?

Under the title "How did I approach it?" the authors will be presenting a challenging case and a description of their management. The title needs to be included at the beginning of the text, for instance, "How did I treat an aneurysm in the left anterior descending coronary artery?" Then the authors' names, last names, specialties, and working centers should be included as well. Corresponding author, address, and e-mail will be included as well. All authors need to declare their conflicts of interest. If they do not have any they need to say so. Text, figures, and references will follow the same criteria established for the clinical case.

Interventional cardiology images

The publication of images describing exceptional cases that the Editorial Committee and external reviewers consider significant for the journal will be accepted for publication. They will need to be followed by an explanatory text and a brief summary of the clinical history. Requisites: a) general text, up to 300 words; b) 2 original figures only; c) references, up to 3; d) authors, up to 5.

Research protocols

The publication of research protocols—preferably multicenter—will be accepted and published by the journal as special articles as long as these protocols do not include the study partial or total results.

Editorials

They are analyses and/or comments on relevant issues on the specialty or general cardiology field in relation with our specialty and always upon request by the Editorial Committee to a subject matter expert. Similarly, comments on issues unrelated to an article in particular can be requested by the Editorial Committee. Requisites: a) general text, up to 2000 words; b) references, up to 40.

Letters to the editor

This is an opinion on an article published in the last issue of

the journal that requires the arbitrage of the members of the Editorial Committee. Requisites: a) text, up to 250 words; b) one table and/or figure can be published; c) references, up to 5. Only letters submitted within a month following the print edition of the issue of the journal where the original article was published will be accepted.

PREPARATION OF THE MANUSCRIPT

The article will be written in Spanish language using a Microsoft® Word text processor and saved under the *.doc file extension. The size of the page will be A4 or letter with double-spacing, 25 mm margins, fully justified text, and 12-point Times New Roman or Arial font. Pages will be numbered consecutively starting with the cover. The manuscript (original article) needs to follow the so-called IMRAD structure: Introduction, Material and method, Results, and Discussion (see the ICMJE Publication Guidelines). Also, it will include Title, Abstract, Conflicts of Interest, and References. In some cases, it will be necessary to add a Conclusion, Acknowledgements, and an Appendix. The metric system will be the standard system of measurement used with commas to write the decimals. All clinical, hematologic, and chemical parameters will be expressed in units of measure from the metric system and/or IU. Only common abbreviations will be used except for the title and the abstract. The first time these abbreviations are used they will be preceded by the whole term except for the use of standard units of measure.

Tables must be presented in individual sheets and they need to be numbered consecutively with Arabic numbers (0, 1, 2, etc.) according to the order in which they were quoted in the text with a short title for each and every one of them. All of the non-standardized abbreviations of the table need to be explained and developed. Explanatory notes will be placed at the foot of the table using the following symbols in this sequence: *, †, ‡, §, ¶, **, ††, ‡‡, etc.

Figures need to be submitted in TIFF, PSD or JPEG format and each figure will be submitted in a separate file with a resolution of 300 dpi in its final format. Each of them will be numbered consecutively together with the explanatory legend in a separate file. The normal size of the photographs will be 127 mm x 173 mm. Titles and detailed explanations will be included in the text of the legend, not the illustration.

References will be numbered consecutively with Arabic numbers between brackets. All of the authors will be included if they are six or fewer; if there are more authors involved, the third one will be followed by the expression «, et al.». The titles of the journals will be shortened based on the style used in Index Medicus. These are a few examples:

1. Registro de Procedimientos Diagnósticos y Terapéuticos efectuados durante el período 2006-2007. Colegio Argentino de Cardioangiólogos Intervencionistas (CACI). Disponible en <http://www.caci.org.ar/addons/3/158.pdf>. consultado el 01/01/2009. (Página Web.)
2. Magid DJ, Wang Y, McNamara RL, et al. Relationship between time of day, day of week, timeliness of reperfusion, and in-hospital mortality for patients with acute ST-segment elevation myocardial infarction. *JAMA* 2005;294:803-812. (Revistas en inglés.)
3. Aros F, Cuñat J, Marrugat J, et al. Tratamiento del infarto agudo de miocardio en España en el año 2000. El estudio PRLAMHO II. *Rev Esp Cardiol* 2003;62:1165-1173. (Revistas en español).

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