

# Management of bilateral type II endoleaks following endovascular repair of abdominal aortic aneurysm: a case report and literature review

## Manejo de endoleaks tipo II bilaterales tras implante de endoprótesis aortobiilíaca: reporte de un caso y revisión bibliográfica

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### ABSTRACT

A patient underwent endovascular repair of an abdominal aortic aneurysm with aortobiliac endoprosthesis implantation. During follow-up, bilateral type II endoleaks originating from lumbar branches led to aneurysm sac growth, prompting embolization of those branches with coils. Subsequent angiographic follow-up showed no further growth of the aneurysm sac.

We present this case due to the current controversy regarding the management of type II endoleaks, adding by a brief literature review on the topic.

**Keywords** (MESH): aortic aneurysm, endovascular repair, endoleak.

### RESUMEN

Se trata de un paciente que fue sometido a reparación endovascular de aneurisma de aorta abdominal con implante de endoprótesis aortobiilíaca. Durante el seguimiento presentó endoleak tipo II bilateral, originado a partir de ramos lumbares, con crecimiento de saco aneurismático, por lo que se procedió a embolizar estos ramos con coils. En el seguimiento angiográfico no se volvió a evidenciar crecimiento del saco aneurismático.

Compartimos este caso debido a la controversia que existe actualmente en relación al manejo de endoleaks tipo II, y agregamos una revisión bibliográfica concisa del tema.

**Palabras clave** (MESH): aneurisma aórtico, reparación endovascular, endoleak.

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### CLINICAL CASE

The patient was a 74-year-old man with hypertension and type-2 diabetes, under medical treatment for more than 10 years. An ultrasound showed that he had a 52 × 48 × 43-mm asymptomatic infrarenal abdominal aortic aneurysm, which was treated without complications by implanting an Endurant II endoprosthesis (Medtronic, Santa Rosa, United States) in October 2015, with no immediate complications.

At the 6-month clinical follow-up, the patient remained asymptomatic. Follow-up CT angiography revealed a type II endoleak from lumbar branches of the hypogastric arteries, with aneurysm sac growth from 52 mm to 59 mm (**Figure 1**).

An angiography revealed well-developed lumbar branches originating from both hypogastric arteries (**Figure 2**) and emptying into the aneurysm sac.

Given the 7-cm sac growth over the previous 6 months, the chosen strategy was invasive treatment by means of embolization of both branches.

The *right lumbar branch* was the first to be embolized (**Figure 3A**). Using a 6-Fr right transfemoral arterial access and supported by a 6-Fr mammary guidewire catheter, operators advanced a 0.014" hydrophilic-coated guidewire (a 0.018" guidewire was unsuccessful due to severe vessel tortuosity). The guidewire was progressed distally, confirming the connection with the aneurysm sac, and an EchelonTM10 (Covidien/ev3, Medtronic, Irvine, USA) microcatheter was advanced over it. Angiography showed contrast passage into the aneurysm sac, leading to the release of four 5 × 10-mm and 5 × 20-mm AXIUM PRIME-3DTM (Covidien/ev3, Medtronic, Irvine, USA) detachable coils. The final angiography confirmed proper vessel occlusion with no contrast passage into the aneurysm sac. The patient had a good recovery. Embolization of the *left lumbar branch* was scheduled (**Figure 3B**) to be conducted three weeks later. The strategy used was similar, using a 6-Fr left transfemoral arterial access. After selectively catheterizing the lumbar branch with a mammary catheter, a 0.014" hydrophilic-coated guidewire was advanced to confirm connection with the aneurysm sac. A microcatheter was then advanced, and upon confirming contrast solution going into the aneurysm sac, four 5 × 10-mm and 5 × 20-mm detachable coils were released, demonstrating proper vessel occlusion with no contrast solution passage into the aneurysm sac.

On a follow-up CT angiography at 2 months, the coils were visualized in the lumbar branches (**Figure 4A**), with persistent type II endoleak that was smaller in size compared with the previous CT angiography (**Figure 4B**), and without aneurysm sac growth.

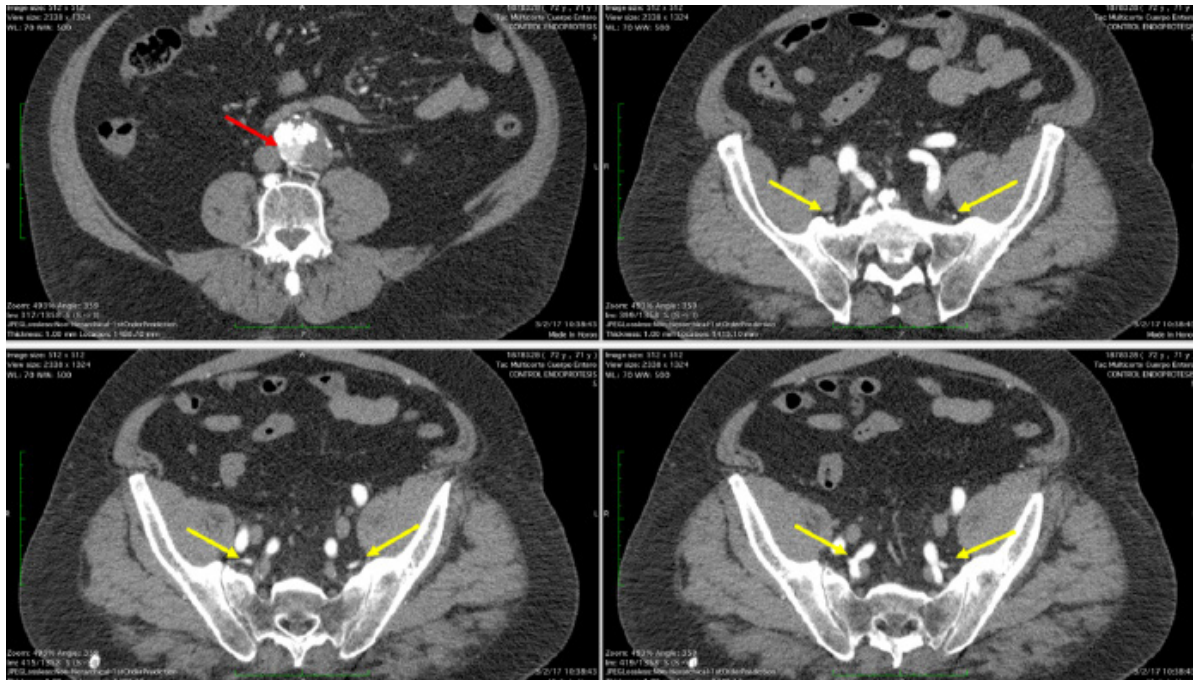
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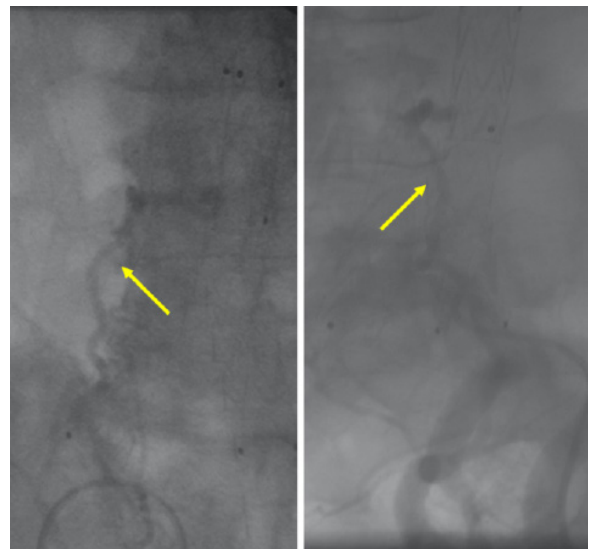
**Figure 1.** The lumbar branches (yellow arrows), from the hypogastric arteries to the aneurysm sac (red arrow).

## REVIEW

Half of all patients who suffer from abdominal aortic aneurysm rupture die before reaching the hospital and, of those who do, up to 50% die during hospitalization<sup>1,2</sup>. Given the minimal symptoms of a growing abdominal aortic aneurysm and the high mortality rate resulting from its rupture, we must be thorough during screening and follow-up of patients with this condition.

It is a well-known fact that the risk of aneurysm rupture significantly increases when it exceeds 5.5 cm in diameter (5.0 cm in women)<sup>3</sup>, or when its growth rate is greater than 1 cm/year<sup>4</sup> (compared always using the same imaging method). Thus, either of these factors constitutes a formal indication for invasive treatment<sup>3</sup>.

Invasive treatment can be surgical, although there is compelling evidence in favor of percutaneous treatment as the technique of choice because these patients have shorter hospital stays and lower short-term mortality (3-5% vs. 0.5-2%), even though long-term mortality remains similar<sup>5-12</sup>. The DREAM-Trial (Dutch Randomized Endovascular Aneurysm Management trial) randomized 351 patients to open or percutaneous treatment, demonstrating lower mortality perioperatively<sup>7</sup> (4.6% vs. 1.2%) and at two years<sup>8</sup> (5.7% vs. 2.1%). Additionally, there was a lower rate of major complications in favor of percutaneous treatment<sup>7,8</sup> (26.4% vs. 11.7%). The EVAR1-Trial, after randomizing 1252 patients, showed lower 30-day mortality in favor of percutaneous treatment (1.8% vs. 4.3%)<sup>9</sup>; however, in the long-term follow-up, this benefit diminishes due to late rupture of the aneurysm sac (possibly related to endoleak), with similar mortality rates observed at 12.7 years (9.3% vs. 8.9%)<sup>10</sup>. Similarly, the OVER-Trial, which randomized 881 patients, also demonstrated initial benefits in mortality favoring percutaneous treatment (0.5% vs. 2% at 4 years)<sup>11</sup>, but with similar figures in terms of survival at 14 years<sup>12</sup>.



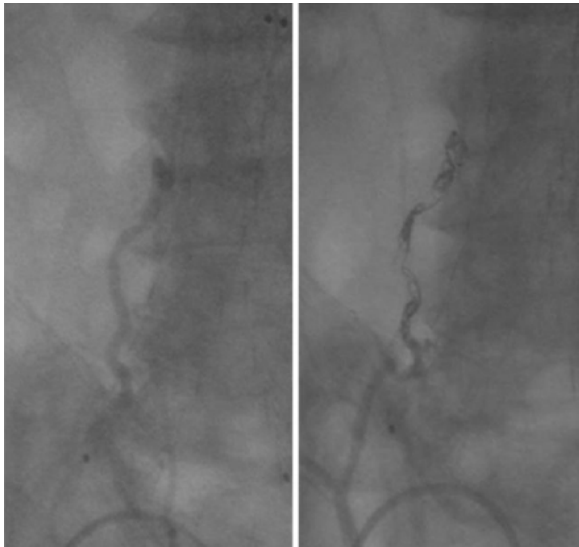
**Figure 2.** Angiography of the lumbar branches connecting the hypogastric arteries to the aneurysm sac.

Regarding follow-up of patients who have undergone percutaneous treatment, recommendations are CT angiography at one month and twelve months in search for displacement of prosthetic bodies, endoleak, and signs of aneurysm sac growth. If any anomaly is found, the imaging study should be repeated at the sixth month<sup>15</sup>.

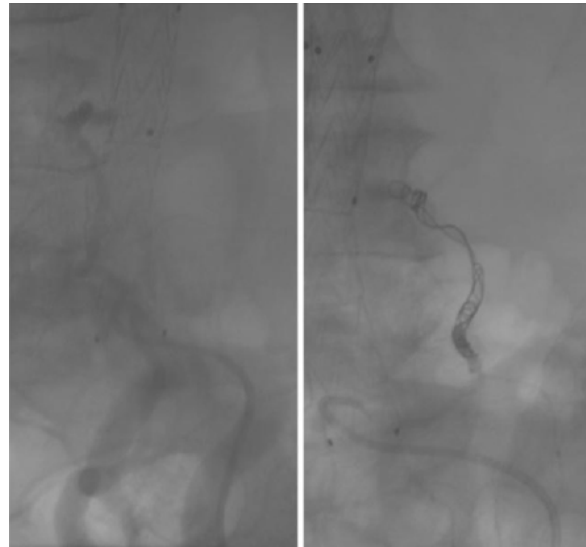
The presence (immediate or delayed) of periprostatic flow continuing to feed the aneurysm sac is called an endoleak, one of the main medium- to long-term complications, reported in 20-50% of patients<sup>13-15</sup>. Depending on the source of the flow, endoleaks are classified into 5 variants<sup>(14)</sup>:

*Type I*, with the leak occurring at the level of the proximal (1A) or distal (1B) anchoring site.

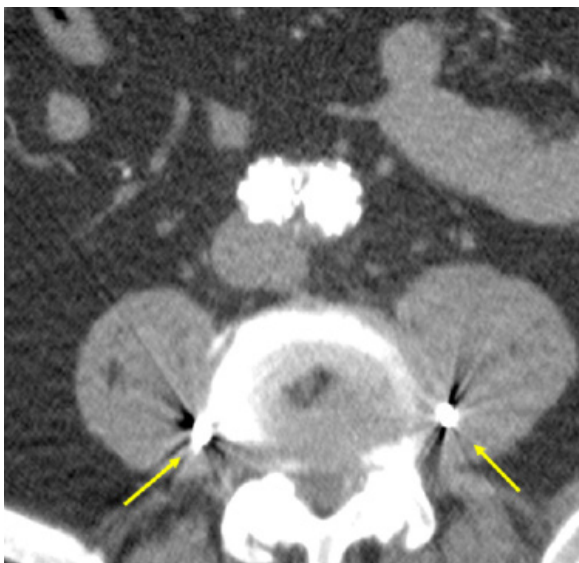
*Type II*, which is due to retrograde flow from an artery branch excluded by the endoprosthesis (most commonly, the inferior mesenteric, lumbar, or accessory renal arteries).



**Figure 3.** Embolization of the right lumbar branch.



**Figure 4.** Embolization of the left lumbar branch.



**Figure 5.** Metallic coils (yellow arrows) implanted in the lumbar branches, without contrast solution passage.

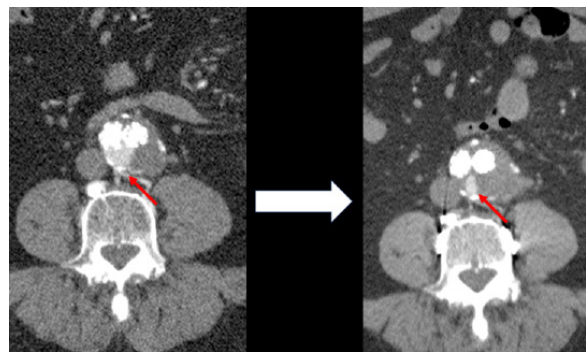
*Type III*, which entails direct leak through endoprosthesis rupture or defects.

*Type IV*, which is due to prosthesis porosity related to excessive anticoagulation.

*Type V*, with no apparent leak; it is considered to be due to blood pressure transmitted to the aneurysm sac through the prosthesis.

In the presence of type I or type III endoleaks, the usual consensus is that they should be treated percutaneously with a stent-graft or prosthetic extension<sup>16,17</sup>.

Type II endoleak is the most common, accounting for up to 76% of all endoleaks<sup>14</sup>; it affects up to 9% of patients undergoing percutaneous repair<sup>18,19</sup>. This is the type of endoleak in the case we have discussed. It is commonly accepted that most type II endoleaks resolve spontaneously<sup>20</sup>, so they should simply be monitored with a new CT angiography at 12 months, and intervention should only be considered in cases where sac growth exceeds 5 mm/12 months<sup>21</sup>.



**Figure 6.** Red arrows marking the decrease in contrast solution leaking into the aneurysm sac, before and after percutaneous closure of the leaks. Of note, no evidence of aneurysm sac growth.

For percutaneous treatment, there are two techniques: *Translumbar embolization*, which is reserved for cases where transarterial embolization is not possible (occlusion of iliac arteries) or when it has failed. It involves embolizing the aneurysm sac by direct puncture over the paraspinous lumbar region. This procedure is not widely practiced due to limited experience in most sites.

*Transarterial embolization*, which is the most used and widely known technique. Access to the branches causing the endoleak is achieved, usually through selective catheterization of internal iliac arteries, and they are embolized, typically with microcoils.

Regardless of the technique used, the recurrence rate is high, reaching 37.5% for transarterial approaches and 19% for translumbar approaches<sup>22</sup>. Ligating afferent branches through open surgery is associated with high rates of morbidity and mortality<sup>23,24</sup>.

## CONCLUSIONS

This case highlights the importance of postoperative follow-up to detect late complications, thereby improving patient survival.

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