A patient was treated by an open procedure. Left postero-lateral thoracotomy in the fourth and sixth intercostal spaces was performed, allowing the control of the descending aorta, the middle part of which was totally adhering to the pulmonary parenchyma. Femoral vessels were then cannulated. Further thoracic dissection demonstrated a narrow zone where the pulmonary parenchyma was condensed against the depressurized, non-pulsatile aneurysm wall. Femoro-femoral extracorporeal circulation was started in order to insure lower body perfusion. The aortic wall was opened, showing, on the anterior border of the aorta, a clear communication with the pulmonary parenchyma. No patent infectious signs were noted. The fistula was removed, and the pulmonary tissular abrasion covered with biological glue. The stent graft was removed, showing on close examination, one small perforation measuring 2 mm located against a more caudal and posterior region of the vessel wall (Fig. 2). No retrograde flow through thoracic collateral arteries was found. The thoracic aorta was then replaced by an aortic homograft. The postoperative course was free from complications. Bacterial analysis of periaortic tissue was negative, allowing the interruption of antibiotics. After an 8-month follow-up, there were no signs of recurrence. CT scan showed distal anastomotic interruption of antibiotics. After an 8-month follow-up, there were no signs of recurrence. CT scan showed distal anastomotic interruption of antibiotics. After an 8-month follow-up, there were no signs of recurrence.
consequence of a large atherosclerotic aneurysm. The two recent episodes of pneumonia could indicate a secondary infection. Two scenarios are possible:

(i) Recurrence of infectious pre-existing fistula (primary or secondary infection) in spite of the absence of fever and leucocytosis, negative blood culture and 4 years intercourse free from events with no antibiotics.

(ii) Recurrence of mechanically induced pre-existing fistula, facilitated by a type III endoleak.

We believe that this fistula existed before TEVAR. However, Chiesa et al. [1] report an incidence of 0.53% of ABF after TEVAR. This can be explained by endoleaks, bronchial wall necrosis through exclusion of nourishing arteries and erosion of the aortic wall by the stent.

An early recurrence of haemoptysis up to 15 days after TEVAR seems to resolve spontaneously, matching the expulsion of residual clots. However, there has been a case where early recurrence revealed an endoleak treated by restenting [2]. Late recurrence was found in 9% of cases of ABF treated by TEVAR, presenting at a mean interval of 13.2 months. It might reflect primary or secondary aortic infection or the progression of the aortic pathology [2]. The mortality rate among ABF recurrence situations reached 50%. Left untreated, any ABF is permanently lethal [3, 4].

The overall mortality of TEVAR for ABF nears 6% [3]. Bailey and AI report interesting results with no 30-day mortality, and no late recurrence in ABF, with a mean follow-up of 8.8 months [4]. In contrast, Chiesa et al. reported a 30-day mortality rate of 40% concerning TEVAR for aorto-oesophageal and ABF without bronchial or oesophageal procedures. Most series represent a single-centre experience with a heterogeneous frame of comorbidities and the lack of stratification.

Open aggressive debridement and in situ replacement or extra anatomic bypass [2] are associated with a reported mortality rate of 15-41% [3]. ABF recurrence after open repair is poorly documented, unlike mortality rates: 1 of 4 patients in Topel’s series [5] had an ABF recurrence. No other similar cases are explicit.

We believe that endovascular therapy in a contaminated environment is useful as a bridge-therapy or palliative solution, especially in situations of graft infection or mycotic aneurysm.

In our patient, the small round perforation in the stent wall is a potential source of type IIIb endoleak, defined as fabric disruption with midgraft holes that could explain the recurrence of mild bleeding. This perforation can also be explained by the chronic mechanical erosion of the stent wall because of the bending of the graft as shown in Fig. 1B, matching with the posterior and caudal location of the hole.

**CONCLUSION**

We believe that the patient should have undergone open repair at his first presentation, having been in younger shape and with no emergency criteria. Actually, there is not enough evidence to consider TEVAR a safe and durable therapy for ABF. This is an arguable position that we would have questioned if the patient was older or had more serious comorbidities.

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REFERENCES


