Moreover, late complications of TEVAR, including postprocedural AEFs and ABFs, have become more evident with time. The exact pathophysiologic mechanisms of AEF formation after TEVAR are unknown. Hypotheses include (1) direct erosion of the aortic wall, (2) pressure necrosis of the esophageal or bronchial wall from the self-expansion of the endoprosthesis, (3) ischemic necrosis caused by stent-graft coverage of aortic side branches feeding the esophagus or bronchus, and (4) esophageal or bronchial wall erosion caused by infection of stent-graft prosthesis.

For these reasons, we agree with Mosquera and colleagues¹ that TEVAR in the treatment of ABFs and AEFs is a bridge solution and that close follow-up is mandatory. Our question for them is as follows: what diagnostic methods, in your opinion, should be more sensitive and specific to monitor a possible reinfection during follow-up?

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THE KEY FOR SUCCESSFULLY MANAGING AORTOBRONCHIAL AND AORTOESOPHAGEAL FISTULAE MAY LAY IN THEIR ETIOLOGY

Reply to the Editor:

Aortoesophageal fistulas (AEFs) and aortobronchial fistulas (ABFs) are uncommon but life-threatening conditions whose outcomes are ultimately determined by the etiology of the fistulas. Both open and endovascular management of aortic fistulas can prevent death by exsanguination, but patients remain at high risk for infectious complications.

We have recently published a study¹ of a consistent series of 26 consecutive patients with thoracic aorta fistulas (18 ABFs and 8 AEFs). The aim of this study was to identify potential differences in the early and long-term results, stratified by the etiology, type, and management of the fistulas, and also to determine the predictors of mortality in this critical subset of patients.

All patients in our series who sustained an aortic fistula with an infectious cause died during hospitalization, and those who sustained an aortic fistula with an underlying malignancy as the primary cause presented a poor midterm prognosis.¹ In the literature, in-hospital mortalities associated with aortic fistulas secondary to mycotic aneurysms or graft infections range between 25% to 60%,²⁻⁴ whereas those caused by esophageal or lung malignancies are associated with a mortality close to 30%.^{3,5}

On the contrary, patients in our series with posttraumatic fistulas showed a great long-term survival because in those cases aortic surgery alone was curative. We concluded, as have other authors, that patients with posttraumatic fistulas, as well as fistulas caused by ingestion of a foreign body or atherosclerotic aortic pathology, are the ideal candidates for endovascular treatment.^{2,4,5}

Midterm and long-term outcomes are ultimately conditioned by the fistula etiology. Failure to treat the underlying cause therefore entails a poor midterm outcome.¹

Treatment modality was identified as a predictor of in-hospital mortality among patients with aortic fistulas in our study, as were sepsis and hemodynamic instability on admission.¹ Mortality was 100% among patients selected to undergo a conservative management.¹ In-hospital mortality associated with open surgery ranges between 15% and 25% for ABF^{6,7} but tends to be even higher for AEF.⁸ In contrast, 30-day mortality after ABF and AEF treated with thoracic endovascular aortic repair (TEVAR) has been reported to run between 28% and 45%.^{2,5} In our series, in-hospital mortality among the patients undergoing open repair was slightly lower than that among the patients undergoing TEVAR (14.3% vs 37.5%). The group of patients undergoing TEVAR not only presented an older mean age and with a higher logistic euroSCORE but also included patients with fistulas caused by infection or malignancy who were not considered candidates for open surgery. The advent of TEVAR has expanded the spectrum of patients amenable to surgical repair despite their critical condition, advanced age, and severe comorbidities, including malignancies.^{1,2,5}

It must be highlighted that almost 36% of the patients enrolled in our study had sepsis on arrival at the hospital or had sepsis develop shortly after admission. In fact, septic shock was the most common cause of in-hospital death.¹ Even when successful open or endovascular treatment has been accomplished, most patients remain at high risk for infectious complications.⁴ Long-term or lifelong treatment with oral suppressive antibiotics should be considered when an endovascular approach is performed or when it has not been possible to remove all the infected tissue or graft

in open surgery. Antibiotic treatment should be only discontinued if it is demonstrated that there is no clinical, bacteriologic, or radiologic evidence of ongoing sepsis. Our institutional antibiotic protocol is highly individualized, but in general terms our policy for antibiotic treatment involves at least 4 weeks of periprocedural intravenous antibiotics followed by case-specific administration of oral suppressive antibiotics according to clinical and laboratory parameters of infection.¹

Early diagnosis of aortic fistulas is of utmost importance to improve these patients' probability of survival. Contrast-enhanced computed tomography, especially multidetector computed tomography, has been demonstrated to be an invaluable tool for early diagnosis of either primary or secondary aortic fistulas.^{9,10} Although hemorrhage of the aortic wall and pseudoaneurysm bulge were the most common radiologic findings in our series,¹ the identification of active contrast extravasation and the presence of periaortic or ectopic gas were the most ominous radiologic signs of prognosis for in-hospital mortality.

In addition, in those patients who have undergone either open or endovascular repairs and for whom there remains a suspicion of infection persistence or relapse, other advanced imaging techniques can be useful. There is a growing body of evidence that (18)F-fluorodeoxyglucose positron emission tomography with computed tomography can be

used to confirm the presence of thoracic aortic graft infection and to exclude other causes of inflammation.^{11,12} Moreover, the combination of data from multidetector computed tomography and data from (18)F-fluorodeoxyglucose positron emission tomography with computed tomography seems to be the key for early detection of aortic fistulas or infectious complications after TEVAR.

The cornerstones of aortic fistula management are early diagnosis and a surgical approach adjusted to the fistula's etiology. TEVAR seems an attractive alternative for high-risk patients, especially as a bailout or bridging procedure. Unfortunately, the Achilles heel of aortic fistula management remains the difficulty of controlling the associated infection, and lifelong imaging surveillance is mandatory.

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