Regarding “Comparison of treatment strategies for thoracic endograft infection”

Moulakakis et al recently reported an interesting meta-analysis of infection after thoracic endovascular aortic repair treated with preservation of the endograft or surgical excision of the stent graft. They consider that compared with antibiotic therapy alone, antibiotic therapy, followed by drainage and repair of the fistula, may control the sepsis, providing a temporary benefit. Endograft explantation remains the gold standard of treatment.

We fully agree that conservative, nonsurgical therapy results invariably in a fatal outcome owing to massive hematemesis or hemoptysis or chronic mediastinitis. A radical surgical approach combining excision all infected tissues, in situ replacement of the thoracic aorta, and concomitant resection of involved pulmonary segments or the esophagus provides the most adequate approach from a microsurgical point of view. However, despite significant refinement in surgical techniques, the 1-year mortality of these complications after thoracic endovascular aortic repair reported by Moulakakis et al remains considerably high: 36.6%.

First, we believe that outcomes of this devastating pathology should be analyzed separately according to the presenting pathology. Outcomes of thoracic stent graft infections are radically different in the case of an aortobronchial or an aortoesophageal fistula. Secondary, in case of an aortobronchial fistula, we believe that a third way could be considered to decrease the postoperative mortality in these high-risk patients. We believe that the stent graft should be left in place and that the bronchial stump should be addressed. Direct contact between the stent graft and the pulmonary tissue should be avoided to prevent further erosive damage. The concomitant repair may entail aortic debridement, primary repair, or resection of the lung with coverage of the stent graft using muscle or pleural flaps. This minimally invasive approach was performed in five of our patients, and all of them are alive after a mean follow-up of 23.2 months. Clearly, any strategy in this patient population needs to be highly individualized given the risks of major thoracic aortic surgery, often in a hostile operative field.

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Reply

We appreciate Canaud et al for their valuable comments on our article.1 Our study concluded that treatment of infection after thoracic endovascular aortic repair (TEVAR) with endograft preservation seems not a durable and perspective option.3 Although our results failed to reach statistical significance, we observed a trend toward a dismal outcome in overall mortality of fistula patients compared with nonfistula patients.

With regard to differences in outcome among patients with aortoesophageal fistula (AEF) and aortobronchial fistula (ABF), we provided separate outcomes for AEF and ABF patients.1 In particular, five of the six patients with AEF, 21 of the 25 patients with AEF, and all four of the patients with concomitant AEF and ABF treated with endograft preservation died. Our study detected a trend of a better outcome for patients treated with antibiotic therapy, followed by additional procedures, compared with patients treated with antibiotic therapy alone. However, no further comparison between ABF and AEF patients could be performed because of the small patient sample.

Several authors have suggested TEVAR for rapid control of bleeding in the setting of AEF or ABF, followed by repair of the esophageal or bronchial defect.2,5 We congratulate Canaud et al4 for their excellent results with this less invasive approach. However, we must underline that their study refers to three patients with primary fistulas and two with secondary. Actually, one patient treated for an ABF after TEVAR was readmitted 4 months later due to severe mediastinitis associated with endograft infection. Endograft explantation and silver-coated graft replacement of the descending thoracic aorta were performed.

Primary fistulas pose the risk of acute exsanguination. Secondary fistulas may have a more devastating outcome because the infected endograft additionally promotes the continuous contamination of blood circulation with microorganisms.3 Therefore, we believe that the pathophysiology of the ABF should be considered when dealing with these patients.

Similar to the strategy suggested by Canaud et al,5 Kahlberg et al6 in a recent study, reported a single female patient with primary ABF who underwent endovascular exclusion of the aortic rupture, followed by lung repair and interposition of a pleural muscle flap between the aorta and the bronchus. She remains alive after 13 months. Lyons et al3 described a man with a secondary ABF after TEVAR who was treated with endograft preservation and lung repair. He underwent left lower lobectomy and bovine patch interposition between the graft and the left upper lobe 40 months after the index procedure, but eventually died of pleural sepsis and recurrence of hemoptysis.

In summary, it seems that a minimally invasive approach in case of an ABF, as suggested by Canaud et al, consisting of aortic debridement, primary repair, or resection of the lung with coverage of the endograft using muscle or pleural flaps, is a promising option in patients unfit for endograft explantation. However, this option also carries a risk for late fistula recurrence,5,6 and further evidence is needed to confirm its long-term efficacy. Finally, we agree with the suggestion that any strategy in this patient population should be highly individualized.
Regarding “The influence of contralateral occlusion on results of carotid interventions from the Society for Vascular Surgery Vascular Registry”

Drs Ricotta et al demonstrated in their review of the Society for Vascular Surgery Vascular Registry that there is essentially no difference in outcome for carotid endarterectomy (CEA) vs carotid artery stenting in the treatment of patients with a contralateral carotid occlusion (CCO). However, much discussion was made about the use of shunts in these patients. We draw attention to a previously published article in the Journal of Vascular Surgery that was not included in the references to the Ricotta paper. In that manuscript, our group specifically described a routine nonshunting approach to CEA even in the face of a CCO. In our experience with 147 CEAs contralateral to a CCO, we experienced three neurologic events, of which only one could potentially be ascribed to a nonshunting technique.

We believe the take-home message is similar to the Ricotta manuscript, and that is that CCO is not a contraindication to CEA nor an indication for carotid artery stenting. Although we favor routine nonshunting, shunting or not should remain an individual preference of the operating surgeon and in competent hands will not affect the outcome of the procedure.

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