

INVITED COMMENTARY

Infectious Complications of EVAR are Deadlier than Those of Conventional Surgery

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The group from the University of Münster have written an interesting paper in which they compare mortality and complications of endoprosthesis and abdominal aortic prostheses explantation for infection.¹

It is a single centre report with a significant number of patients ($n = 26$). It is the only published work to compare explant of endovascular aneurysm repair (EVAR) with explant of open aortic repair (OAR) in a consecutive series by the same surgical team.

Three recent works on EVAR infection have been published. The first is an Asian meta-analysis with 401 very disparate patients.² The second is a French multicentre study with 33 patients.³ The third, an older one, was an American multicentre study from 2016, which included 180 patients.⁴

These three studies looked at fever, abdominal pain, and leucocytosis. This profile is not present in all patients. We wonder how prosthetic infection could be suspected without fever, pain, or leucocytosis. The Münster group uses positron emission tomography in 60% of its patients, obtaining a high diagnostic yield only in EVAR.

If we apply Koch's postulate (the microorganism must be isolated from a diseased organism and grown in culture), a quarter of the patients had a negative culture, which is in accordance with previous publications.²⁻⁴ One reason could be incorrect sample harvesting. Microbiology laboratories should study these tissues and fluids. To obtain a better performance it has been proposed to analyze bacterial DNA and make samples sonication.⁵

A further diagnostic algorithm would have been expected from Torsello's team, to support their aggressive surgical approach.

Mortality is a key point in this work, being higher for EVAR than for OAR (37% vs. 0%). The overall mortality results are also within the published range. Analysis of the life threatening complications reveals higher rates in EVAR (50% vs. 0% OAR).

The pathogenic explanation of these results, as discussed by Schaefer et al., is that additional thoracotomies (50% of cases) need to be added to laparotomy in EVAR patients, together with supravisceral clamping in 90%.¹ No mention is made of the difficulty of extracting the iliac legs of the EVAR and its subsequent repair. This increases the complexity.

Another remarkable aspect of this work is the method of repair used: *in situ* rifampicin soaked polyester graft. The authors

avoided extra-anatomical bypass, homograft, or heroic reconstructions with pericardium, vein, or fascia lata. Surprisingly, they did not have reinfections with this technique, although there are publications of up to 5% of reinfections per year.³

All series show a long latency period between the first operation and infection, 60 months in the Münster series (OAR 8 years vs. 2 years for EVAR). Degeneration of the prosthetic material may have been the reason for OAR, although the rate of aorto-enteric fistulae is similar.

There is no reference to possible precipitating factors of aortic device infection either in this study or any other. They did not identify which situations could have resulted in EVAR infection after 2 years of implantation. Cardiologists and cardiac surgeons are well aware of the serious problem of prosthetic valve endocarditis. In fact, they continue to recommend antibiotic prophylaxis in certain dental and surgical procedures.⁶

Two unresolved dilemmas are present in this study: (i) Should we inform our patients that infections following EVAR are deadlier than those after OAR? We cannot report all the complications and their consequences, as survival depends on the experience of the team treating the complication. (ii) Should we design studies to identify potential situations of aortic prosthesis infection? Given the low incidence rate and the controversial nature of prophylaxis, it is not worth it.

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