RESEARCH LETTER: FOR DEBATE

Long Term Benefits of EVAR in the Modern Era: The Importance of Looking at Stent Graft Durability without Forgetting the Right Pathophysiology

Endovascular repair (EVAR) for abdominal aortic aneurysms (AAA) treatment has gained progressive acceptance owing to better peri-operative outcomes than open aortic repair (OAR). However, concerns exist regarding stent graft durability and loss of benefits over the long run. The meta-analysis by Antoniou *et al.* confirmed that although the hazard of all cause and aneurysm related death within six months of surgery was significantly lower after EVAR, with further follow up the pooled hazard estimate moved in favour of OAR.[1] Interpreted in the context of an ever increasing life expectancy, the findings reinforce the European Society for Vascular Surgery guidelines, which recommend OAR for patients with reasonable prospects of long term survival.[2]

However, EVAR still represents the solution of choice in older patients and in those with increased comorbidity for whom durability of repair has to be measured within a shorter time frame. In the meta-analysis by Li *et al.*, the authors conducted a subgroup analysis based on the included studies' last year of patient recruitment (before or after 2010).[3] Interestingly, when more recent studies were analysed, there was no longer a mortality difference between EVAR and OAR, suggesting that EVAR outcomes have improved over time. Re-intervention rates, however, remained higher for EVAR in both older and newer studies.

The inferior long term EVAR outcomes could be due to lack of durability; endografts are at higher risk of failure in the long term and it seems reasonable to assume that the cumulative effects of endograft specific complications, need for re-intervention, secondary rupture, and poorer baseline status put EVAR patients at a higher risk of late death. This is especially important given that EVAR patients are surviving longer with advances in health care; therefore, better strategies are needed to maintain consistent long term endograft durability which should be prioritised by the designers of new stent grafts

Aneurysms are treatable but not curable diseases; exclusion of the sac from systemic pulsatile flow remains crucial to prevent aneurysm related death. Although from a biomechanical standpoint AAA rupture occurs as a consequence of the acting load exceeding the AAA wall load bearing capacity, the mechanism of rupture is a complex combination of physiological, biomechanical, histopathological, and genetic factors. Using a computational fluid dynamic approach from computed tomography angiography images of ruptured AAA, Boyd *et al.* showed that, regardless of AAA size or configuration, rupture did not occur at sites of high pressure and wall shear stress (WSS), but instead at sites of predicted flow recirculation where low WSS and thrombus deposition predominated.[4] These findings raise the possibility that this flow pattern may lead to thrombus deposition, which may elaborate adventitial degeneration and eventual AAA rupture.

Aneurysm sac regression is correlated with improved survival and a reduced rate of secondary interventions and EVAR related complications.[5] Nevertheless, aneurysm shrinkage is a complex three dimensional phenomenon that implies remodelling of the entire sac and diameter measurements are surrogate (although adequate) markers of treatment success. Therefore, further research regarding the biology involved in AAA remodelling after EVAR and its relationships with external factors (such as anticoagulation) may shed further light on the mechanism beyond vessel response to stent graft treatment. However, it is well known that placement of aortic endografts in hostile necks and/or outside their instructions for use represents the strongest risk factor for late stent graft failure. The pathophysiological mechanisms underlying the intraluminal thrombus represent an intriguing new field of research. However, stating that AAA treatment should primarily address intraluminal thrombus in order to increase EVAR durability is entirely speculative and not supported by either strong evidence or pathophysiological rationale at this time. Securing a safe sealing zone must remain the primary aim of EVAR and surveillance strategies should be tailored on well known risk features including quality of proximal seal, presence of endoleaks, and early sac shrinkage.[2]

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