Aortic aneurysm surgery has traditionally been associated with significant perioperative mortality and morbidity, resulting, as one would expect, in a significant volume of research in the field focusing on perioperative outcomes and short-term survival. The evolution of newer technology in the form of endovascular aneurysm repair (EVAR) has again resulted in a focus on short-term outcomes in the last two decades.

EVAR is now well established, with perioperative and short-term survival after elective abdominal aortic aneurysm (AAA) surgery having improved considerably compared with open repair; however, long-term survival as a whole remains poor for this group of patients (1,2). Given that patients undergoing elective EVAR for uncomplicated aortic aneurysms can now expect to be quoted a 30-day mortality figure in the region of 1%–2% (2,3), we are turning our attention to improving longer-term outcomes in this cohort of high-risk patients who typically suffer from multiple comorbidities (4).

Initial EVAR devices were basic tube grafts with unacceptably high rates of distal endoleak (5,6). The introduction of bifurcated grafts was the most important development for EVAR (7,8), with newer modular designs opening up the potential for a larger proportion of patients to be treated in this minimally invasive manner (9). Early endografts showed poor durability with the absence of proximal fixation implicated in late migration of stents, recurrent sac reperfusion, and subsequent late aneurysm rupture (10).

Over the course of the last two decades, more modern devices have proven far more reliable and robust; changes in stent graft design and lower-profile improved delivery systems have increased the range of AAs that is now amenable to durable endovascular treatment. More recently, endovascular aneurysm sealing has emerged as an alternative mode of EVAR (11). However, perhaps one of the most significant developments in securing stent graft durability and longer-term efficacy of aneurysm treatment has been the acceptance that proximal fixation was the cure for stent migration, typically with suprarenal fixation with an uncovered segment of the stent graft at the juxta-renal portion of the aorta or infrarenal proximal fixation.

AKI in the context of emergency ruptured AAA repair is well described and expected given the nature of the typical in extremis presentation (12). Again, open surgical repair, especially if suprarenal clamping is required intraoperatively, inevitably results in some renal ischemia (13). However, after elective endovascular AAA repair, the reasons for a deterioration in renal function, especially in the longer term, are more multifactorial (14).

A recent systematic review and meta-analysis of renal dysfunction after EVAR estimated that the average proportion of patients with a clinically relevant deterioration in renal function ≥1 year after EVAR was of the order of 18% (95% confidence interval [95% CI], 14% to 23%; $P=82.5\%$) (14).

A large retrospective study from the United States analyzed a cohort of 10,184 patients aged ≥66 years old who did not have an AAA and found little or no deterioration in renal function in the majority of patients over the 2-year period. However, predictably, those patients with diabetes mellitus and those with poorer renal function at the outset showed greater levels of deterioration (15). This perhaps highlights that we should not accept a decline in renal function in patients with EVAR as a natural progression of disease.

Serum creatinine has been shown to be strongly associated with both 3- and 5-year mortality rates (16); studies have reported that larger creatinine elevations after hospitalization predict the highest risks of death, and even minor changes in renal function are associated with adverse outcomes (17).

Saratzis et al. (18) report a study in this issue of the Clinical Journal of the American Society of Nephrology on their findings of long-term renal function in patients undergoing elective aneurysm repair compared with matched patients with atherosclerotic arterial disease in the absence of aneurysmal aortic pathology. The main findings of this study were that, at 1 and 5 years postsurgical intervention, patients with EVAR showed a significantly greater decline in renal function compared with case-matched patients undergoing arterial surgery for carotid stenosis (carotid endarterectomy).

Of note, Saratzis et al. (18) also report that patients undergoing open aneurysm repair experienced less of a decline in their renal function at 5 years, with an average loss of 7.4 ml/min per 1.73 m² at 5 years (95% CI, 4.8 to 10.6) compared with 8.2 ml/min per 1.73 m² (95% CI, 6.5 to 10.8; $P<0.001$) for infrarenal fixation of EVAR, 16.9 ml/min per 1.73 m² (95% CI, 13.0 to 21.9;
de Bruin AAA repair (19,20). Of the DREAM Elective AAA Trial, Cylindrical Aneurysm Management (DREAM Elective AAA) pair (EVAR-1/EVAR-2) and Dutch Randomised Endovascular Aneurysm Management (DREAM) Elective AAA pair requires no contrast or typically, any follow-up surveillance imaging with computed tomography could all be cumulative factors. Some of these factors could explain the disparity in renal function decline between open repair and EVAR—open repair requires no contrast or typically, any follow-up surveillance imaging; however, this does not explain the significantly greater decline seen in suprarenal fixation EVAR.

Both the United Kingdom Endovascular Aneurysm Repair (EVAR-1/EVAR-2) and Dutch Randomised Endovascular Aneurysm Management (DREAM) Elective AAA Trials have reported on longer–term renal dysfunction after AAA repair (19,20). Of the DREAM Elective AAA Trial, de Bruin et al. (19) reported that, at 5 years, there was a mean deterioration in eGFR of 3.9 ml/min per 1.73 m² (95% CI, 1.3 to 6.5) in the EVAR group, whereas the results from the EVAR-1/EVAR-2 Trial showed a mean drop in eGFR of 1.13 ml/min per year (20). However, neither of these studies drew a distinction between infrarenal and suprarenal fixation for EVAR, and these studies used older stent graft technology (19,20).

Microembolization as a consequence of suprarenal fixation might be a potential factor; however, the scientific literature is notoriously poor at reporting prescribing and compliance to medication, such as antiplatelets, that may mitigate this both acutely and in the longer term (1). A systematic review aimed at identifying a correlation between stent graft technology with robust, prospective, randomized trials. The evidence for stent graft placement with proximal fixation increased the risk of renal dysfunction; however, the effect disappeared when statistical modeling was performed to account for study heterogeneity (21). A myriad of other factors, such as aortic morphology, thrombus, and plaque load, as well as technical skills of the operator could all have a significant role to play.

The evidence for stent graft placement with proximal fixation in terms of long-term efficacy of aneurysm repair is well established, and there is only weak evidence to support the assertion that this might contribute to the decline reported in renal dysfunction at long–term follow–up. Perhaps our focus should be on mitigating the decline in renal function seen in this patient group rather than trying to re-invent the stent graft.

A host of strategies has been proposed to ameliorate the effect of AAA repair on renal function; antioxidant therapies have yet to be shown to be effective (22), and ischemic preconditioning has shown potential evidence of renoprotection in animal studies (23) in the context of AKI. However, the heterogeneity of this patient group makes retrospective analyses difficult. The only way to fully address this issue is with robust, prospective, randomized trials.

Disclosures
None.

References


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