‘Keep in Mind an Endograft is a Spring!’: Re. ‘Aorto-enteric Fistula After Endovascular Repair for Behcet’s Disease Patient: a Case Report’

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In their paper, Arworn et al. report the case of a 42 year old male with Behcet’s disease presenting with aorto-enteric fistula (AEF) following endovascular treatment of a symptomatic infrarenal abdominal aortic aneurysm (AAA).[1] An endovascular approach to inflammatory AAA has become the first line therapy as it is associated with lower mortality compared with open surgery.[2] Moreover, placing an endograft eliminates the suture line of open surgery that has been considered as a potential risk factor for secondary AEF when in contact with the duodenum. However, as development of an AEF has been described in endovascularly managed inflammatory AAA, even their definite aetiology is not entirely understood. The most incriminated cause is infection, which may be spread to the endograft either directly from a nearby source of infection, or haematologically from a distant source.

However, other possible causes such as endograft migration and kinking might be involved. In the case described by Arworn et al., excessive oversizing was responsible for endograft migration.[1] Initially, computed tomography angiography (CTA) showed that the endograft filled the aneurysm sac while upstream and downstream diameters corresponded to initial aortic diameters. However, 13 months later, CTA demonstrated that the whole endograft had expanded, leading to a large discrepancy between the unstented and stented parts of the aorta. This discrepancy led to caudal migration of the endograft, resulting in kinking of the device in the aneurysm sac. Therefore, by enhancing frictional forces at the aortic wall, this could promote parietal damage, causing erosion of the aortic wall, further leading to enteric ulceration. Previous analyses of explanted endografts has shown how such kinking could create high stresses with major endograft damage.[3] Consequently, this excessive oversizing resulted in a more acute drop in compliance, which led to endograft migration through fluid-endograft interactions.[4] Unfortunately, the explanted endograft did not undergo specific observational, mechanical, and chemical evaluations. This would have been interesting to draw out correlations, and could be useful for future material improvements.[5]

In conclusion, two lessons should be remembered for clinical practice. First, an endograft acts as a “spring” at the level of the stent rings. Placing an endograft creates mechanical coupling between the endograft and the arterial wall. Excessive oversizing leads to increased forces acting permanently on the arterial wall with a creeping effect analogous to tree roots damaging pavement, causing the endograft to return to its equilibrium state, particularly in a diseased artery as in Behcet’s disease. Second, despite an initial “satisfactory” CTA image, it is necessary to ensure a close follow up to detect early shape modification and to convert to open repair before any complications arise.

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