

COMMENTARY

Calcified Pseudocoarctation After Thoracic Endovascular Aortic Repair: A Hard Case to Crack

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Kuntz and colleagues present a unique case involving a pseudocoarctation resulting from an intraluminal calcified stenosis within an endograft, which occurred 11 years after thoracic endovascular aortic repair (TEVAR) for a blunt traumatic aortic injury (BTAI).¹ The patient presented with therapy resistant hypertension, without signs of intermittent claudication or renal dysfunction. Computed tomography angiography (CTA) showed a calcified stenosis at the distal end of the endograft. The patient was treated with explantation and open repair and subsequent macro and microscopic analyses of the explanted endograft verified the calcified nature of the stenosis.

Presently, TEVAR is the preferred treatment modality for BTAI, due to lower peri-operative morbidity and mortality compared with open repair, as was conducted in this specific case. A rare complication of TEVAR after BTAI is the occurrence of a symptomatic stenosis or occlusion, typically due to in stent thrombus formation.^{2–4} Suggested causes are excessive endograft oversizing, leading to infolding of the fabric, and hypercoagulable states. The formation of in stent thrombus may serve as an initial step in the development of calcified in stent thrombus, similar to that observed in this case.

While the occurrence of a highly calcified pseudo-coarctation within an endograft is unprecedented, similar clinical presentations have been documented in the native descending thoracic aorta and the aortic arch. Acquired obstructive pathology of the descending aorta is rare and aetiology diverse, e.g., traumatic, infective, inflammatory, and neoplastic.⁵ Truly calcified obstructive diseases in the aortic arch and descending aorta that mirror the presentation seen in this case are extremely rare, but have been reported previously.^{6,7} In these cases, similar calcified intraluminal thrombi were observed, suggesting that aetiologically, intraluminal thrombus gradually converts

into a highly calcified stenotic lesion without clear involvement of the surrounding aortic wall. It is likely that such a process also occurred in this case as was discussed by the authors.

Interestingly, in patients who receive a central venous catheter (CVC) a strikingly similar phenomenon can be observed. The inserted CVC acts as a foreign body that induces thrombus formation and development of a fibrin sheath that may become calcified over time.⁸ This process of calcification depends on a balance between factors that activate or inhibit calcification, which can be influenced by external processes such as inflammation, which also occurs after endograft implantation.⁹ The observations in CVC patients underscore the potential thrombotic origin of the calcified stenosis in this case.

The very low incidence of the observed complication makes it hard to gather sufficient similar cases to draw definitive conclusions on the potential cause and the exact underlying process of this phenomenon. However, it underlines that even in the endovascular era we might be confronted with traditional pathological processes that are not always optimally solved by merely endovascular means. Taking into account the ever decreasing open vascular and cardiothoracic surgical exposure, we must prepare for future hard cases to crack.

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