LETTER TO THE EDITOR

Intragraft Thrombosis After TEVAR for BTAIs: Mounting Evidence and Open Questions

We read with great interest the report by Beijer et al.[1] presenting an example of graft thrombosis in a young patient previously treated by thoracic endovascular aortic repair (TEVAR) for a blunt traumatic aortic injury (BTAI). As demonstrated by the concomitant literature review, partial or complete graft thrombosis after TEVAR for BTAI remains a rare long term complication, occurring most frequently at the level of the descending thoracic aorta. Post-operative complications such as graft infolding and/or migration are more commonly reported and consistently linked to incorrect evaluation of pre-operative aortic diameters that may occur during emergency interventions.

Likewise, the most accepted hypothesis behind the phenomenon of late intragraft thrombosis relates to excessive oversizing of the prosthesis with respect to the non-dilated aortic segments of the mainly young population involved in BTAIs.[2]

Our Vascular Surgery Unit has been involved in the care of a 19 year old female patient, diagnosed with an asymptomatic intragraft partial thrombosis on 12 month follow up imaging after an emergency TEVAR performed for rupture of the thoracic aorta due to polytrauma after free fall from a height (Fig. 1). In consideration of the haemodynamic instability and the availability of suitable endovascular material at the time of treatment, a Medtronic Endurant 16-16-93 straight abdominal graft was implanted, with 13% oversizing at the proximal and 25% at the distal landing zone compared with the pre-operative aortic diameters (respectively of 14 mm and 13 mm).

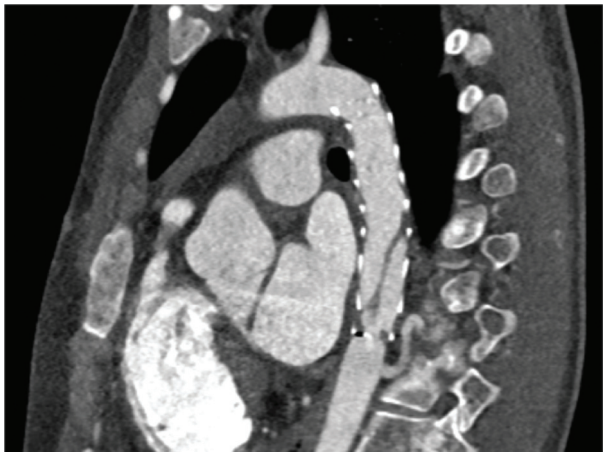

Figure 1. Asymptomatic intragraft mural thrombus on 12 month follow-up CT scan.

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Although the patient underwent further orthopaedic and general surgery interventions (pelvic and femoral fracture stabilisation, partial colectomy, and secondary intestinal recanalisation) the post-operative course was uncomplicated and the patient was discharged after 135 days. The presence of an asymptomatic mural thrombus was first detected 53 weeks after the event by magnetic resonance imaging and subsequently confirmed with computed tomography angiography, where the aortic diameters at the proximal and distal landing zones were measured as 18 mm and 16 mm respectively. The patient also underwent transoesophageal echocardiography, excluding the presence of any mobile thrombus. Unfortunately, the possible cause of this lesion has not been clarified and the patient has been managed with single antiplatelet therapy. No further endovascular or surgical interventions have been deemed necessary.

As mentioned by some authors,[3,4] intragraft thrombosis was discovered as an incidental and asymptomatic finding. Furthermore, mural thrombus was observed in our patient as well as in several of the other reported cases, suggesting that such morphology may represent the first stage of development of the thrombotic apposition preceding the onset of a related clinical presentation.

We hypothesise that marked pre-operative hypotension may lead to underestimated measures of aortic diameters, therefore negatively affecting the degree of desired oversizing. As a result, intragraft thrombosis could be a consequence of mechanical stress on the aortic wall and damage to the aortic intima. Despite the scarcity of documented cases in the literature, we advocate the investigation of the pathophysiological mechanism behind such phenomena following endovascular treatment of BTAIs, possibly with the aid of haemodynamic simulation studies. We believe that adequate pre-operative planning will effectively prevent such complication.

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Gianluca Buongiovanni
Postgraduate School of Vascular Surgery, Università degli Studi di Milano, Milan, Italy
Department of Vascular Surgery, Fondazione IRCCS Cà Granda Ospedale Maggiore Policlinico, Milan, Italy

Alberto Settembrini*, Daniele Bissacco, Silvia Romagnoli
Department of Vascular Surgery, Fondazione IRCCS Cà Granda Ospedale Maggiore Policlinico, Milan, Italy

Santi Trimarchi
Department of Clinical and Community Sciences, Università degli Studi di Milano, Milan, Italy
Department of Vascular Surgery, Fondazione IRCCS Cà Granda Ospedale Maggiore Policlinico, Milan, Italy

*Corresponding author. Fondazione IRCCS Ca' Granda Policlinico Milano, Milan, Italy.
Email-address: amsettembrini@gmail.com (Alberto Settembrini)

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