LETTER TO THE EDITOR

Aim Before You Shoot: Within Graft Thrombus After EVAR is Generally Benign and Escalation of Antithrombotic Therapy is Not

Dear Editor,

We read with interest the study published by Russell et al., which recommends antithrombotic therapy escalation whenever intraluminal prosthetic graft thrombus (IPT) is identified.[1] This recommendation was deduced from the finding that among 26 patients who developed IPT (10.1% of the reported 258 patients), six (23.1% of the IPT group) developed symptoms potentially attributable to IPT and two patients required re-interventions.

Albeit IPT may explain the symptoms for the two patients intervened upon (the first presenting with occlusion and undergoing a femorofemoral crossover bypass, the second requiring an endovascular intervention), the presented data do not clarify the potential underlying causes of such events which, as previously reported, are often related to technical aspects (inadequately assessed obstructive arterial disease in the iliac outflow, endograft limb kinking or ending in the apex of a tortuous iliac segment).[2] Furthermore, the presented data do not exclude other potential causes of the ischaemic symptoms for the remainder of symptomatic patients, such as underlying presence of peripheral arterial disease (PAD). Nor was the severity of the identified mural thrombus in the respective iliac limbs in these symptomatic patients quantified or its haemodynamic repercussion characterised, thus creating doubt over the causal association presented for every case.

Also, the authors report that IPT progressed in 46% (n = 12) of the 26 cases. Morphological data stating luminal narrowing or longitudinal development of IPT could had been produced, as grade of stenosis was recorded according to methodology. Possibly, evaluation of all post-operative imaging rather than relying upon IPT referral in the CT angiography reports for case selection could have assisted in addressing this issue and reducing the risk of bias rightfully identified by the authors. Additionally, while thrombus progression may have been reduced by additional or more aggressive antithrombotics, it is unclear whether any imaging resolution was obtained by such strategy since no clear clinical benefits can be attributed to such strategy in this small group of patients, and there was no comparator.

Another caveat refers to characterising risk factors for IPT occurrence. While clinically relevant limb occlusions have been associated with outflow impairment, as aforementioned, other factors play a significant role in the development of IPT, as previously reported by our group.[3] Noteworthy, haemodynamic factors such as aorto-uni-iliac endograft and associated plug flow, as well as barrel configured main body sections of the endograft may favour mural thrombus accumulation, and these are most frequently benign. Endograft fabric has repeatedly also been identified as a risk factor. While Russell et al., refer that none of the assessed intervention related or morphologic factors were significantly associated with IPT, these analyses could have been described in the manuscript.

Perhaps more relevant, it is not clear if the rate of occlusion increases in patients with IPT compared with those without. In our study, with a mean follow up of 3.5 years, no increase in graft occlusion was observed for patients with pre-existing mural graft thrombus, compared with those without (about 4% for each group).[3] A later meta-analysis corroborated these findings.[4] This benign course seems especially true when considering the thrombus inside the main body segments of the graft, which is most often the case.

Unlike IPT, long term dual antiplatelet therapy or a switch to full anticoagulation is not so benign and may have serious consequences for patients. In a recent meta-analysis studying antiplatelet therapy in patients with PAD, dual antiplatelet therapy increased the risk of major bleeding by 37 more events per 1000, compared with monotherapy.[5] Full dose anticoagulation at least duplicates the risk of major bleeding compared with single antiplatelet agents. If we consider the increased bleeding risk observed in many AAA patients, one could argue for a less aggressive antithrombotic strategy unless the risk benefit ratio is well demonstrated.

In conclusion, before escalating antithrombotic therapy for patients with IPT, one should first aim at the individual patient, considering the expected risk of developing occlusion and the potential harm caused by excessive bleeding. Surely there will be patients where occlusions can be prevented while accepting a small increase in bleeding risk, but it may be the exception rather than the rule.

CONFLICTS OF INTEREST

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