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Risk Factors for Pulmonary Embolism

We read with great interest the extremely informative and well performed paper recently published by Grobben and colleagues,¹ demonstrating that silent pulmonary embolism (PE) after endovascular aortic repair occurs in approximately one in 10 patients, despite routine thrombo-embolism prophylaxis. Although various risk factors for PE have been considered, the influence of systemic immuno-inflammatory disease, such as psoriasis, has not. However, it is well known that clinical data have convincingly demonstrated that systemic immuno-inflammatory disease patients have an increased risk of developing cardiovascular disease and an increased risk of PE.^{2,3} A recent study by Chung and colleagues reported that patients with psoriasis presented a 2.02 fold adjusted hazard ratio of venous thrombo-embolism compared with the general population.⁴ Therefore, systemic immuno-inflammatory disease should be investigated for studies in which the risk factors for PE are evaluated. Data should be collected in the baseline clinical characteristics. We are surprised that this potentially important factor has not been considered, despite the known increase in PE risk with a prior medical history of systemic immuno-inflammatory disease.

If systemic immuno-inflammatory disease had been investigated in the study presented by Grobben and colleagues, it would have been of utmost interest to consider this risk factor and to evaluate its potential contribution to PE. Such knowledge would not only reinforce the clinical relevance of systemic immuno-inflammatory disease effects on PE but also enable preventive recommendations.

CONFLICT OF INTEREST

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Response to Letter to the Editor: Risk Factors of Pulmonary Embolism

We thank Dr Kang and Dr Zhang for their interest in our study and their note regarding the potential relevance of systemic inflammatory disease (e.g. psoriasis) as a risk

¹ GuanYang Kang and Hui Qing Zhang equally contributed to this work.

factor for pulmonary embolism (PE) after endovascular aortic repair (EVAR).

We acknowledge the clinical relevance of psoriasis as a risk factor for venous thromboembolism (VTE) in the general population, with hazard ratios ranging from 1.35 to 2.02.^{1,2} Notably, associations for PE have been reported to be lower: 0.97 (0.89–1.06) for mild psoriasis and 1.34 (1.01–1.80) for severe psoriasis.² Although such an association has not been convincingly shown in the surgical population, it could be postulated that the systemic inflammation after EVAR may be more pronounced in patients with psoriasis (i.e. because systemic inflammation is inherently attenuated in these patients) resulting in a higher risk of PE.

We chose not to evaluate risk factors other than those included in the Wells score because the study was neither designed nor powered to assess such an association. Still, based on a psoriasis prevalence of 2% (of which < 10% severe) and a relative risk of ≤ 2 for PE in the general population, it is unlikely that psoriasis will have influenced the results significantly.^{2,3} We believe that prolonged immobilisation and intensive care admission should be regarded as the most important risk factors for VTE in the surgical population, and consequently deserves consideration for adequate prophylaxis.³

Additional studies that include systemic inflammation should be performed to assess the most relevant risk factors for VTE within this population, and the requirement for prophylaxis.

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Cardiac Effects of Aortic Endografts

We have read with great interest both the paper by Rong *et al.*,¹ and the accompanying Editorial by Lejay *et al.*,² regarding the effects of aortic graft implantation on the heart and downstream vessels. The authors of the paper and editorial, and the journal editors, deserve congratulations for bringing a fundamental issue of great clinical importance to the readership.

We have been working on the subject for several years and have published the results of clinical studies and presented on the subject at multiple international meetings.³

We fully agree with the authors' conclusions about altered left ventricular function and impaired ventricular arterial coupling due to insertion of the rigid graft, although we believe that increased inflammation also plays a major role.

In previous studies we found variations in the inflammatory response related to graft type,⁴ and also that haemodynamics and biological markers like N terminal pro-brain natriuretic peptide can be affected.⁵

We wonder whether the authors of this paper have seen similar variations in their study population.

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