Coronary microvasculopathy and intracardiac thrombosis in antiphospholipid syndrome

Coronaropathie microvasculaire et thrombose intracardiaque dans le syndrome des antiphospholipides

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A 57-year-old woman, with a history of recurrent deep venous thromboses, was admitted for progressive dyspnoea rapidly improved by diuretic treatment; her electrocardiogram showed deeply inverted T-waves in precordial leads. Pulmonary embolism was dismissed by a normal lung scan. Early coronary angiography was completely normal. However, echocardiography revealed a bilobulated mass attached to the apex of an apparently normally contracting LV, compatible with mural thrombus (Fig. 1A; Video 1), prompting heparin therapy.

CMR examination performed 2 weeks later demonstrated a small apical area of subendocardial delayed contrast enhancement, characteristic of ischaemic injury, with resolution of the previously described mass (Fig. 1B) and no obvious LV wall motion abnormalities (Videos 2 and 3).

Laboratory tests were remarkable for positive immunoglobulin G and M anticardiolipin antibodies. Primary APS was diagnosed as no other underlying disease could be identified, the anticardiolipin antibodies remaining elevated 13 weeks later.

Our findings suggest that myocardial ischaemia in this patient was caused by thrombotic cardiac microvasculopathy. Eventually, the ischaemically injured overlying endocardium would act as a trigger for clot formation. However, mural thrombosis into a normally contracting LV remains most unusual. We therefore believe that systemic APS-related coagulopathy played a substantial role in both events.

Abbreviations: APS, Antiphospholipid syndrome; CMR, Cardiac magnetic resonance; LV, Left ventricle.

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Figure 1. A. Transthoracic echocardiography shows a bi-lobulated mass attached to the apex of the left ventricle. B. Cardiovascular magnetic resonance: inversion recovery delayed-enhanced four-chamber left ventricle view acquisition with phase-sensitive detection demonstrates the resolution of the intracardiac mass after anticoagulation and subendocardial delayed enhancement pattern of ischaemia in the related left ventricle apex.

This case points toward a possible pathogenic mechanism for intracardiac thrombosis in APS, illustrating the subtle interplay of microvascular and intracardiac thrombotic phenomena in this setting. It also emphasises the prominent role of CMR imaging in the diagnostic work-up of these difficult cases.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.acvd.2011.05.014.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.