I read with great interest the recent editorial by Nardone Soscia and Romain [1] on the role of antiphospholipid antibodies in cardiac valvular disease and its thromboembolic complications. That antiphospholipid antibodies (APLA) can occur in patients without systemic lupus erythematosus or other connective tissue diseases has been reported previously [2-4]. Their role in causing thromboembolic complications in valvular disease of the heart has only recently been implicated [1-4].

Thromboembolism is a dreaded, though rare, complication of mitral valve prolapse (MVP), the commonest valvular disease in the world [5]. The most likely cause of cerebral ischemic attacks in MVP is embolism of a noninfected contact thrombus originating either from the atrial surface of the prolapsing mitral leaflet, or in the angle formed by the junction of the left atrial endocardium and the atrial surface of the prolapsing posterior leaflet (‘angle lesion’) [6-8]. But it has never been satisfactorily explained why some patients with MVP and normal sinus rhythm develop strokes and most other patients with MVP do not.

Perhaps either a retrospective or a prospective study of APLA in patients with MVP with/without an embolic history will shed some light on this enigma. If an association between APLA and stroke could be established in patients with MVP, irrespective of arrhythmias, the need for prophylactic anticoagulant therapy in this subgroup of patients must be considered. On the other hand, a more conservative approach of anti-platelet therapy (aspirin and dipyridamole) seems to be the prudent measure in these patients.

References

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