eComment: Cardiac mesothelial/monocytic incidental excrescence and antiphospholipid syndrome

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We read with great interest the article on mesothelial/monocytic incidental cardiac excrescence (MICE) [1] in a young female with antiphospholipid syndrome (APLS). Patients with APLS present a significant involvement of the cardiovascular system, as coronary artery disease and valvular abnormalities constitute the most frequent manifestations, representing more than two-thirds of cases [2]. Notably, hypercoagulability in APLS patients predisposes to high rates of thromboembolic events as well as a high rate of restenosis after percutaneous interventions (PCI) or CABG respectively, causing significant morbidity and mortality [2]. In particular, angioplasty-induced arterial injury leads to platelet aggregation, adhesion, and thrombosis. Several factors involved in the thrombogenesis influence the restenosis rate after PCI, as thrombosis is one of the possible mechanisms of restenosis [2]. Moreover, restenosis may occur more frequently in antithrombopin-positive patients, as in the case described in this article [1].

MICE is a rare intracardiac lesion, whose importance stems from the symptoms it may cause and the errors in diagnosis it can lead to, either in clinical and image assessment or during surgery. MICE appears to be equally distributed between males and females in the age range 5–80 years [3], with most of the cases involving individuals above 60 years, and lesion size ranging from microscopic to 3.5 cm [3]. Lesions have been found more frequently in the left cardiac chambers and on valve surfaces, especially during aortic and mitral valve surgery, or in endomyocardial biopsy specimens. The only importance commonly attributed to this lesion is that it may potentially be mistaken for a primary or metastatic malignancy. Differential diagnosis is necessary with metastatic adenocarcinoma, particularly in clear cell tumors or a mesothelial tumor, because both lesions are frequently strongly positive for cytokeratine AE1/AE3, as in MICE disease [4].

The role of mechanical irritation, inflammation, or neoplasm as possible triggers for a reactive process, or a form of mesothelial hyperplasia, has been emphasized in the reactive theory. Specifically, the atrial wall is focally very thin and could easily be perforated during cardiac catheterization, leading to the displacement of mesothelial cells into a cardiac chamber. Once exposed to the bloodstream, the mesothelial cells could aggregate with histiocytes and fibrin through an unknown mechanism, forming a loose tissue mass [4]. Nodular histiocytic/mesothelial hyperplasia (NHMH), a similar entity to MICE, is also predominantly composed of histiocytes with scattered mesothelial cells that can occur in the pericardium, pleura, peritoneum, and pelvis [3]. NHMH may also be a reactive lesion which could result from inflammation, mechanical irritation, or tumor. Based on their similar morphologic features and the fact that the term MICE does not exactly reflect the essence of this entity, Hu et al. propose to unify MICE and NHMH and suggest that NHMH might be a better choice [3]. Interestingly, the hypothesis that active cell-to-cell interaction may be involved in the formation of NHMH, due to expression of CD34 strongly positive mesothelial cells, has been reported, a factor that might also be considered for MICE [5].

References