Case report - Cardiac general

Cardiac mesothelial/monocytic-incidental-excrescence: more than an artifactual lesion?

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Abstract

Mesothelial/monocytic incidental cardiac excrescence (MICE) is a benign finding made up of a mixture of cuboidal mesothelial cells, histiocytes and fibrine and is mainly found incidentally during open heart surgery, commonly after cardiac catheterization. Clinical importance of this lesion has been emphasized because of its potential confusion with malignancies, especially with metastatic carcinoma. We report a case of an asymptomatic 72-year-old man with incidental finding of a pericardial effusion and a small mass attached to the left appendage.

Keywords: Mesothelial/monocytic incidental cardiac excrescence; Cardiac surgery; Pericardium; Cardiac neoplasm

1. Introduction

Mesothelial/macrophage incidental cardiac excrescences (MICE) are considered a benign finding made up of a mixture of mesothelial cells, macrophages, scattered inflammatory cells and fibrin, without a vascular network or supporting stroma. Clinical importance of this lesion has been emphasized because of its potential confusion with malignancies, especially with metastatic carcinoma. Pathogenesis of MICE is still controversial.

To our knowledge, this is the first case of MICE detected in the pericardial sac of an asymptomatic patient without a history of prior cardiac instrumentation.

2. Case report

A 72-year-old man was admitted to our institution for the detection of a circumferential pericardial effusion and a small mass next to the left appendage. The patient’s anamnesis was negative. A mild pericardial effusion was occasionally diagnosed one year before. Diagnostic blood tests were performed and referred as negative. Although the patient was asymptomatic a medical therapy was started but effusion had not resolved.

Transesophageal echocardiography showed a free-floating mass (21x15 mm) at the apex of the left appendage. The cardiac RMN was performed (Fig. 1).

To achieve a definitive diagnosis an anterior pericardectomy was performed by a mini-thoracotomy approach. At surgery a small soft tissue mass, loosely adherent to the apex of the left appendage, was identified and removed.

The pericardial fluid and biopsy specimens were sent for cytology, histopathology examination and blood cultures. Cultures failed to show any growth of microorganism and cytology of effusion detected a mixture of histiocytes and hyperplastic mesothelial cells.

Macroscopically the lesion was fragmented and showed a tissue-like tumor, hemorrhagic, the largest one measuring 1.2 cm. Specimens, fixed in 10% buffered formalin and embedded in paraffin block, were sectioned and stained with hematoxylin and eosin. The microscopic features are shown in Fig. 2.

3. Discussion

To date 35 cases have been reported in the literature [1-3], even if the true incidence of cardiac MICE is unknown.

They have more frequently been found in the left cardiac chambers and on valve surfaces, especially during aortic and mitral valve surgery or in endomyocardial biopsy specimens [4, 5]. The only importance commonly attributed to this lesion is that it may be potentially mistaken for a primary or metastatic malignancy. Differential diagnosis is necessary with metastatic adenocarcinoma, particularly in clear cell tumors or a mesothelial tumor, especially because both lesions frequently are strongly positive for cytokeratine AE1/AE3 as in MICE disease [6].

This case is interesting for its peculiar clinical context; the MICE was found in the pericardial sac of an asymptomatic patient without a history of prior cardiac instrumentation and prior to any surgical or invasive manipulation.
The histopathogenesis of MICE is still unclear and represents the most intriguing aspect of this entity. Two hypotheses have been proposed to explain how the mesothelial cells migrate into the intravascular space.

A 'reactive' theory emphasizes the role of mechanical irritation, inflammation, or neoplasm as possible triggers for a reactive process, a form of mesothelial hyperplasia. Luthringer et al. noticed that 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 [3]. They pointed out that 'in all likelihood all of our patients had a cardiac catheterization preceding the surgical procedure that led to the identification of the lesion'. Once exposed to the bloodstream, the mesothelial cells could aggregate with histiocytes and fibrin through an unknown mechanism, forming a loose tissue mass. An alternative and complementary hypothesis suggests an 'artificial' origin. Courtice et al. examined the contents of extracorporeal bypass pump filters and mediastinal drains, demonstrating that material recovered from 82% of the pump filters and 13% of the drains had histologic features similar to MICE [7]. Therefore the authors suggested that MICE are an amalgam of mesothelial strips, tissue and debris artificially compacted in the operating field. The fragments that are not drawn into the bypass pump are transferred by the suction catheter tips to the intravascular space during open heart surgery. Histiocytes, fibrin, mesothelial cells, fibers and squames are aggregated and found free-floating in the heart or attached to the specimen by the surgeon.

This hypothesis is considered a convincing explanation for the majority of the cases, especially for the reported cases in which no prior catheterization was performed, but cannot explain cases detected by endomyocardial biopsy without prior cardiac surgery [3, 4].

Not all the MICE can be considered incidental findings without clinical relevance. An autopsy case of arterial embolization was reported after mitral valvuloplasty [5]. Celiac artery was occluded by thrombotic material containing mesothelial debris, causing multi-organ abdominal infarcts and patient's death. Most of the cases of chronic pericardial effusion are considered 'idiopathic' and patient's medical history rarely reports a previous acute pericarditis. This case challenges the 'artificial' hypothesis on the pathogenesis of cardiac MICE and further sustains the discussion about its etiology. Although there are sound evidences for both theories of how MICE is formed, neither the 'reactive' nor 'artificial' theory is sufficient to explain all the reported cases.

Recently a case of nodular histiocytic/mesothelial hyperplasia, a lesion with morphologic and immunohistochemical features similar to MICE, has been reported. In this case most of mesothelial cells were strongly positive for CD34. The authors hypothesized that the development of aggregates of mesothelial cells and histiocytes may be related to aberrant expression of cell-to-cell adhesion molecules [8]. However, in our case no evidence of positivity for CD34 was documented. The hypothesis that active cell-to-cell interaction may be involved in the formation of mesothelial/mönocytic incidental cardiac excrescence is attractive.
but available data are not univocal and further investigation is required.

References


