Incidental Mesothelial/Monocytic Cardiac Excrescence and Literature Review

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Abstract

We present a case of the so-called mesothelial monocytic incidental cardiac excrescences (MICE) of the heart and a brief review of related literature.
The patient was a 74- year- old woman. She had no history of previous cardiac instrumentation and underwent pericardial drainage and biopsy due to cardiac tamponade. A tissue sample was submitted as thrombus attached to the pericardium. Histologically, these lesions were composed of a mixture of plump histiocytoid cells, a papillary and strip arrangement of cuboidal cells, and various sized vacuoles and fibrin. The nests of cuboidal cells resembled cancer cells but showed features of mesothelial cells. Immunohistochemically, histiocytic cells were positive for CD68 but negative for cytokeratin. The cuboidal cells were positive for cytokeratin but negative for CD 68. These lesions were probably reactive because of their heterogeneous components. The significance of MICE is its possibility of being misdiagnosed as metastatic carcinoma. **(*Iranian Heart Journal*. 2002, 2003; 3(2&3): 61-65)**

Key Words: pericardial < mass < mesothelial < tamponade < MICE

Case Report

A 74- year- old woman was admitted due to aggravated dyspnea and atypical chest pain. The patient had been hospitalized previously at a lung center, and was subsequently referred to our center due to increased shortness of breath, tachycardia and probable impending cardiac tamponade. She complained of dyspnea for the previous 7-8 months, with exacerbation during the recent few weeks. Lately, she had had weight loss, episodic fever and anorexia. She did not have a history of productive coughs, febrile illness or tuberculosis. She had not taken any drugs.
Physical examination at admission revealed BP: 100/70mmHg, PR: 70/min and regular, RR: 20/min and T: 37° C. Peripheral pulses were symmetric without paradoxical pulse. The jugular veins were distended. Heart sounds were muffled with rales at the base of both lungs. No lymphadenopathy or ascites were present; lower extremities showed 1+ to 2+ edema.
On lab tests, PPD (purified protein derivative) was reported to be 5mm. The liver and thyroid function tests were normal. The erythrocyte sedimentation rate was 30. C-reactive protein and rheumatoid factor were negative. Other routine laboratory tests such as BUN, creatinine, etc. were within normal limits.
Electrocardiography showed normal sinus rhythm, inverted T-wave in V_1 to V_6, and Q wave in V_1 to V_3. Chest x - ray showed an increased cardiothoracic ratio with small calcified nodules in both hila and blunted costophrenic angles due to pleural effusion (Figure 1).

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Fig. 1. CXR shows huge cardiomegaly with blunted costophrenic angles.

Spiral chest CT scan revealed massive pericardial effusion with no lymphadenopathy (Figure 2).

Fig. 2. Spiral chest CT scan with contrast shows massive pericardial effusion with sequential collapse of lung.

Echocardiography showed right ventricular collapse as well as massive pericardial effusion. The swinging heart pericardial fluid was drained with a pigtail catheter and 2200 ml of straw - yellow fluid was obtained. A pericardial fluid diagnosis reported suspected tuberculosis.

The patient's general condition again deteriorated after three days with severe dyspnea and hypotension. Repeated echo showed massive pericardial effusion with suspected homogenous density. Transesophageal echocardiography also showed massive pericardial effusion with an early diastolic collapse of the right atrium. A large homogenous mass was seen in the pericardial cavity which was attached to the epicardial surface of the right atrium and right ventricle without a compressive effect on the cavities. The borders of the mass were about 3^{cm} x 5.5^{cm}. The systolic function of both ventricles was normal. There was moderate LV diastolic dysfunction. All cardiac valves were normal. We evaluated cardiac chambers, great arteries and the venous system for other masses, but we could find none (Figure 3).

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Fig. 3. Tranesophageal echocardiographic view to show intrapericardial mass and pericardial effusion.

Based on the patient's symptoms and transesophageal echocardiography report, surgery was done for both drainage and pericardial biopsy at the same time. Further evaluations, such as abdominal and pelvic sonography, were normal.

The cytology of pericardial fluid was negative for malignancy, but some isolated and small clusters of reactive mesothelial cells were seen. A pericardial fluid analysis showed an exudative pattern with no reported acid-fast bacilli. The adenosine desaminase of pericardial fluid was 36.

Pathologic Findings

A tissue sample was submitted as thrombus; it was attached to the pericardium and measured 2 x 1 x 0.6cm. A microscopic examination showed a biphasic pattern composed of diffused, compact arrangements of polygonal to ovoid cells with grooved, reniform, oval nuclei, indistinct nucleoli and abundant cytoplasm. The nuclei showed a mild variation in size. In close proximity to the cellular sheets were folded-to-coiled–up ribbons of low cuboidal cells with regular nuclei compatible with stripped mesothelial cells. Mitotic figures were not seen. Blood vessels or stromal tissue were not evident among the cells. Scattered red blood cells and inflammatory cells were present, as were the typical large round spaces. (Figure 4)

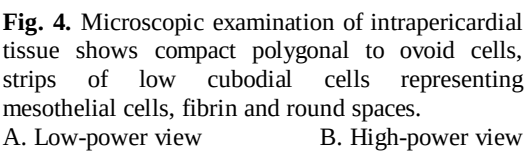

Fig. 4. Microscopic examination of intrapericardial tissue shows compact polygonal to ovoid cells, strips of low cubodial cells representing mesothelial cells, fibrin and round spaces.
A. Low-power view B. High-power view

Immunohistochemically, histiocytic cells were positive for CD68 but negative for cytokeratin. The cuboidal cells were

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positive for cytokeratin but negative for CD68. (Figure 5)

Fig. 5. Immunohistochemical staining.
A) The majority of cells are stained by CD68 in a granular pattern. The strip of mesothelial cells are negative.
B) The strip of mesothelial cells are positive for cytokeratin whereas the others cells are negative.

Discussion

MICE is a benign lesion comprising a mixture of histiocytes, mesothelial cells, inflammatory cells and fibrin. Rosai et al. published the first and largest series of 14 MICEs.[1] Ten lesions were endocardial, one was within an ascending aortic aneurysm and three were found in the pericardial sac. They explained that the introduction of mesothelial cells into the endocardium by cardiac perforation during perioperative catheterization could incite mesothelial cell hyperplasia and mesothelial cell in-growth from pericardium to endocardium. Veinot et al.[2] published four additional cases, all endocardial. One patient had no prior history of cardiac catheterization. They performed electron microscopy in two cases to confirm mesothelial participation and found a mixture of histiocytes and cells with microvilli and desmosome-like junctions, consistent with mesothelial cells. Courtice et al.[3] found fragments of tissue identical to the reported cases of MICE in 18 of 20 extracorporeal bypass pump filters and adherent to two of 15 postoperative chest tubes. They postulated that MICEs are artifactual, formed by the manipulation of the cardiac surgeon, compact by suction vacuum at the time of the procedure in which they are identified. Argani et al.[4] reported a case of MICE that was seeded by clusters of metastatic adenocarcinoma. The patient had no history of previous cardiac instrumentation, and was found to have an adenocarcinoma of the right lung involving the hilum, but apparently not invading the pericardium. Chan's group[5] reported transbronchial biopsies from two patients showing similar histological changes that were described in MICE.

No prior catheterization occurred in our case. In addition, this tissue fragment was found by the surgeon immediately upon entering the pericardiac sac and before significant suctioning had occurred. Therefore, the exact mechanism of this case cannot be identified, but it may be due to chronic injury. Malignancy should be considered in differential diagnoses of lesions composed of clusters of epithelioid cells in the pericardium or endocardium, but the recognition of the mixture of cell types in typical MICE, including a distinct population of plump histiocytes and the mesothelial cells as well as immunohistochemical findings, usually helps the pathologist to avoid the pitfall of misdiagnosing carcinoma.[6,7] More attention should be considered by the pathologist because occasionally

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carcinoma may be beneath this characteristic architecture.

In summary, these lesions are reactive and benign but their pathogenesis remains unclear. Their main practical importance resides in the fact that a pathologist unaware of their existence may mistake them for metastases or some other neoplasm.

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