CLINICAL IMAGE

Primary pericardial mesothelioma in a 48-year-old patient

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Primary pericardial mesothelioma (PPM) arises from the mesothelial cells of the pericardium. It represents less than 1% of all malignant mesotheliomas and accounts for nearly 50% of all primary pericardial tumors.[1] It is the third most common tumor after angiosarcoma (33%) and rhabdomyosarcoma (20%).[2] Its diagnosis is usually difficult and late, and is made at autopsy in most cases.[3] The optimal treatment for PPM has not been established.

A 48-year-old patient, a nonsmoker who denied prior exposure to asbestos, was hospitalized in our cardiac department in March 2017, with a 1-month history of malaise, dyspnea on exertion, and a weight loss of 5 kg. Physical examination revealed bilateral pitting edema of the lower legs and muffled heart sounds on cardiac auscultation, with fluid in the pleural cavities clearly visible on a chest radiograph. Transthoracic echocardiography revealed a circumferential pericardial effusion with a heterogeneous hyperechoic mass (23 mm in diameter) around the heart wall, without signs of cardiac tamponade, and with a 45% reduction in contractility of both ventricles. To determine the extent of the disease, chest magnetic resonance imaging was performed, which demonstrated a nearly isoattenuating nodular mass of about 3.8 × 2.3 cm in size (FIGURE 1A–C).

Pericardiocentesis was performed under fluoroscopic and ultrasound guidance, but the material was not aspirated. Laboratory studies revealed elevated C-reactive protein levels (14.38 g/l) and normal procalcitonin levels (0.107 ng/ml; reference range <0.5). The suspicion of autoimmune

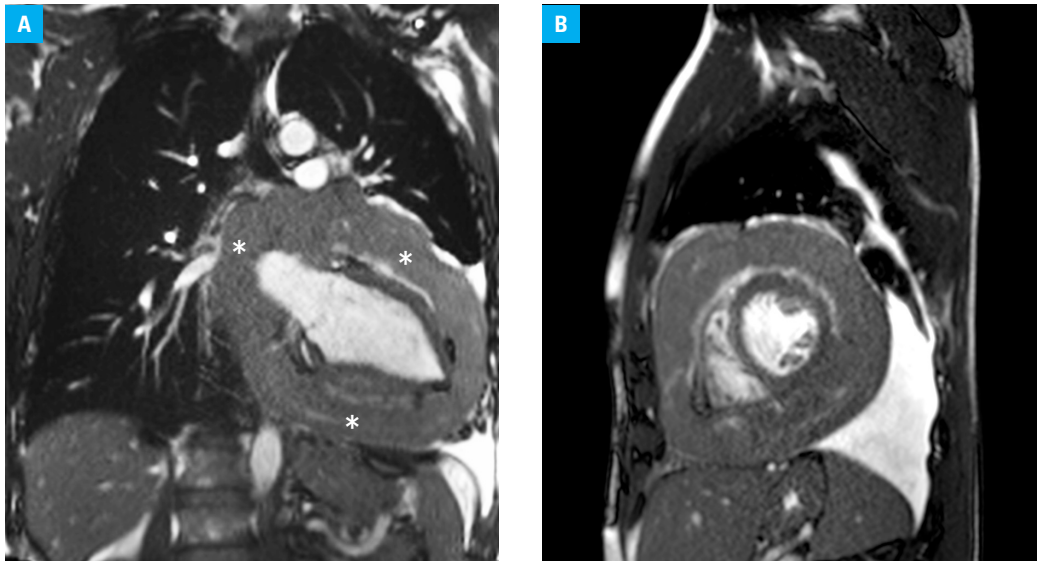

FIGURE 1 Magnetic resonance imaging; **A** – diffuse mass in the pericardial space surrounding the whole heart (asterisks); **B** – short-axis image of the mass obtained on expiration

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Received: July 12, 2017.
Revision accepted:
September 6, 2017.
Published online:
November 24, 2017.
Conflict of interests: none declared.
Pol Arch Intern Med
doi:10.20452/pamw.4155

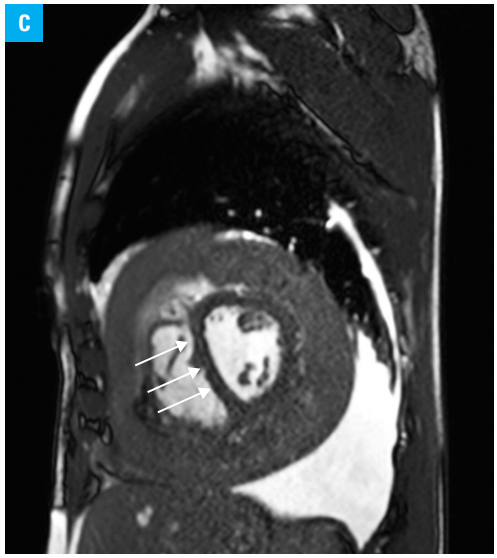

FIGURE 1 Magnetic resonance imaging; **C** – leftward ventricular septal bowing during the early diastolic phase shown on inspiration (arrows): a distinctive feature of pericardial constriction

background was not confirmed, as we did not detect positive antinuclear, antineutrophil cytoplasmic, or tuberculin antibody titers. Immunoglobulin G test results for Epstein–Barr virus, cytomegalovirus, and *Toxoplasmosa gondii* were negative. A sternotomy was performed, which revealed solid masses with small cartilaginous nodules in the mediastinum above the heart and aorta. After a careful crossover of the mass at the midpoint of the right ventricle, the moving heart wall was reached and part of the tumor from the pulmonary artery was removed.

The excised mass was directed towards the diaphragm, exposing a layer of about 30 mm between the heart and esophagus. The entire tumor layer was then removed from the right ventricle and the right atrium, resulting in a marked improvement in cardiac hemodynamic parameters. Due to the difficult conditions of heart release, the removal of further pathological lesions was abandoned. The cut surface of the tumor was fleshy and grey-white to yellowish in color. A pathological evaluation of the postpericardiotomy specimen confirmed the diagnosis of epithelioid mesothelioma in the pericardium. The epithelioid cells showed eosinophilic cytoplasm and open nuclei with prominent nucleoli. The nucleoli exhibited tubulopapillary patterns of growth, with connective tissue cores accompanied by a lymphoplasmacytic infiltration. Mitoses were frequent. On immunohistochemistry, neoplastic cells were strongly and diffusely positive for vimentin, calretinin, cytokeratin CK AE1/AE3 and CK7, with focal patchy immunoreactivity for human bone marrow endothelial cell marker 1 (HBME-1) and cytokeratin CK5/6. After the surgery, the patient was discharged in a good general condition, with the recommendation to continue oncological treatment.

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