

Multimodality imaging evaluation of a primary cardiac lymphoma

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Primary cardiac lymphoma is a rare form of non-Hodgkin lymphoma that involves the heart with extension to pericardium and great vessels. Prognosis is poor in the absence of a prompt diagnosis and adequate therapy. Differential diagnosis includes malignant neoplasms such as angiosarcoma or metastatic carcinoma and melanoma. Clinical manifestations may be heterogeneous. Multimodality imaging work-up represents the best method for tumor detection and evaluation of its size and extension: echocardiography, computed tomography, magnetic resonance imaging, and nuclear imaging are the best imaging tools. Definitive diagnosis is achieved with cytological and histological evaluation. We report the case of a 76-year-old woman admitted to our emergency department with symptoms of congestive heart failure. Multimodality imaging work-up showed a mediastinal bulky tumor involving heart and pericardium. Pathology revealed a large B-cell primary cardiac lymphoma.

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Case report

A 76-year-old woman with no history of cardiac disease was admitted to our emergency department because of symptoms of dyspnea at rest, and episodes of chest pain occurred in recent weeks. The patient had no complaints of fever, sweat, or weight loss (B symptoms). She was tachycardic and tachypneic with blood pressure

of 90/60 mmHg. Blood count, liver, and renal functions and coagulation profile test were all within normal limits. Written consent was obtained from the patient for each of the subsequent examinations.

Chest X-ray showed an enlarged heart with loss of normal right cardiac silhouette in absence of signs of pulmonary edema (Fig. 1). Transthoracic echocardiography revealed pericardial effusion

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Figure 1. (A) Posterior–anterior and (B) latero–lateral chest X-ray projections showing the enlargement of the heart with loss of right cardiac silhouette and no signs of pulmonary edema.

with an ill-defined hypoechoic mass infiltrating the free wall of the right heart chambers.

Contrast-enhanced chest computed tomography (CT) excluded other causes of acute chest pain such as aortic dissection and pulmonary embolism. The scan revealed a large infiltrating mass localized in the pericardial space, contiguous to the anterior myocardial walls of right atrium and ventricle. The bulk showed extension to the atriocaval junction, superior vena cava, and through the atrioventricular groove involving the base of the heart. Furthermore, there was evidence of pericardial effusion that reached the upper recesses. Relative homogeneous enhancement of the mass was observed after intravenous contrast material administration (Fig. 2).

Pericardiocentesis with drainage was performed, with a substantial improvement of patient clinical and hemodynamic conditions. A large amount of mixed serous–hemorrhagic fluid was collected and used for cytologic examinations: analysis of pericardial fluid did not allow a definitive diagnosis. A subsequent second contrast-enhanced CT scan was made; unfortunately electrocardiography (ECG) synchronization was not available for this examination (Fig. 3).

During hospitalization, contrast-enhanced cardiac magnetic resonance imaging (MRI) was performed to evaluate motion relationship between the mass and the right heart chambers, the right atrioventricular wall involvement, the functional impairment of the right ventricle, the left ventricular function, and for a better tissue characterization of the mass. In order to study the possible alterations of cardiac wall kinetics, myocardium thickness, and parietal contractility caused by the neoplasia, retrospective ECG-triggered bal-

anced turbo field echo (steady state free precession) sequences were performed. The sequences were oriented on short and long axis (atrium–ventricular and 4-chamber view), with the following parameters: repetition time 3.8 ms; echo time 1.8 ms; flip angle 70°; matrix scan 256 × 256; thickness 8 mm; gap 0 mm; 20 cardiac phases/cycle; and retrospective synchronization.

The examination showed impaired motion of lateral and inferior right ventricular wall, right atrium compression, and basal invasion of right atrium (Figs. 4 and 5, Cine 1–3).

Precontrast black blood turbo spin echo T1, T2, and fat saturation T2 (STIR) on the axial plane were performed (Fig. 6); triggered short axis and four-chamber view T2 black blood sequences were performed to avoid heart motion artifacts (technical parameters were: repetition time 1333 ms; echo time 100 ms; TI 290 ms; echo planar imaging factor 1; turbo factor 33; matrix scan 512 × 512; slice thickness 8 mm; gap 0.8 mm; prospective diastolic synchronization; and 1 slice per breath-hold) and four-chamber T1 high-resolution isotropic volume excitation and turbo spin echo T1 sequence postinjection of 17 mL gadolinium–diethylenetriamine pentacetate (0.5 mmol) were performed on the axial plane. The postgadolinium sequences showed hyperintensity of the signal within the mass after 140 seconds (Fig. 7).

Whole body fludeoxyglucose–positron emission tomography/CT scan was used for the staging procedure: the mass showed elevated fludeoxyglucose accumulation with high Standardized Uptake Values suggesting a malignant nature of the neoplasm with high glucose metabolism (Fig. 8). The examination showed no other localizations of disease (Fig. 9).

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