Cardiac involvement in lymphoma mimicking hypertrophic cardiomyopathy

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Cardiac involvement in lymphoma represents a rare type of myocardial pathology. The right side of the heart is most frequently involved, especially the right atrium [1]. Epicardial and pericardial infiltration with pericardial effusion are regarded as typical [2]. We describe an unusual case of biventricular myocardial thickening caused by infiltration due to non-Hodgkin lymphoma and mimicking the phenotype of hypertrophic cardiomyopathy (HCM). A 38-year-old man with a month-long history of abdominal lymphadenopathy was referred to our department for evaluation of pericardial effusion and a suspicion of HCM. Transthoracic echocardiography showed non-dilated left and right ventricles with significantly reduced systolic function, impaired left ventricular relaxation, and diffuse thickening of ventricular walls (interventricular septum 24 mm, right ventricle free wall 18 mm). Moreover, dilatation of both atria and thickening of the interatrial septum with large pericardial effusion was present (Fig. 1, Suppl. Video 1 — see journal website). In order to better characterise cardiac involvement, cardiac magnetic resonance (CMR) was performed (Suppl. Video 2 — see journal website). CMR depicted not only previously described heart pathologies but also showed diffuse inhomogeneous late gadolinium enhancement in the myocardium of both ventricles and interatrial septum (IAS), highly suspicious of myocardial involvement associated with probable lymphoproliferative disease (Fig. 2). To clarify the cause of lymphadenopathy, positron emission tomography-computed tomography was performed, and generalised lymphadenopathy typical of lymphoproliferative haematological disorder was found. Regarding the cardiac involvement, high inhomogeneous uptake of 18-fluoro-deoxyglucose in the myocardium of both ventricles and IAS was detected (Fig. 3). The patient underwent surgical extirpation of the enlarged lymph node from the left groin, and non-Hodgkin lymphoma was diagnosed. After four cycles of chemotherapy including corticosteroids complete remission was achieved. This treatment led to normalisation of ventricular wall thickness and systolic function of both ventricles as well as disappearance of pericardial effusion (Fig. 4, Suppl. Video 3 — see journal website), which definitively confirmed our suspicion of cardiac involvement associated with lymphoma infiltration. In differential diagnosis of biventricular myocardial thickening accompanied by pericardial effusion many entities might be considered, including a broad spectrum of sarcomeric HCM phenocopies and infiltrative diseases such as cardiac amyloidosis. The latter can result in a significant increase in the thickness of the left and right ventricular myocardium, IAS, and pericardial effusion, as was shown in our case. However, the presence of only mild diastolic dysfunction in an advanced form of cardiac amyloidosis would be highly unlikely.

References