Calcified constrictive pericarditis can hide other cardiac diseases!

Une péricardite constrictive calcifiée peut cacher d’autres maladies cardiaques !

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A 76-year-old woman presented with alteration of general status associated with cough, anorexia and dramatic weight loss of 15 kg. Clinical presentation consisted of decompensated heart failure with dyspnoea, tachycardia, peripheral oedema and mild ascite. Chest X-ray showed important pericardial calcifications (Fig. 1). A diagnosis of constrictive pericarditis was stated despite the lack of echocardiographic data due to a very poor acoustic window. Tuberculosis was considered to be the underlying cause, mainly based on the criterion of pericardial calcifications; however, as is usual, no bacterial evidence could be obtained. Owing to the persistence of symptoms after several therapeutic treatments, and despite the age of the patient, an indication for surgery (pericardectomy) was decided, leading to complementary investigations. Computed tomography confirmed the presence of circumferential pericardial calcifications (Fig. 2A), and magnetic resonance imaging (MRI) showed a signal void due to calcifications accompanied by a thickened pericardium from 4 to 10 mm (Figs. 2B and 3). Cine loop MRI demonstrated the sudden inverted motion of the septum towards the left ventricle in diastole, which is a frequent sign of constriction (cine loop imaging 1 and 2). Moreover, MRI revealed the existence of an asymmetric left ventricular hypertrophy (Figs. 2B and 3) localized on the inferolateral and septal walls with systolic apical exclusion (cine loop imaging 1 and 2). Invasive
Figure 1. Chest X-ray, showing calcifications throughout the pericardium.

Figure 2. A. Circumferential calcifications of the pericardium with a large calcified mass on the mitral valve in horizontal long-axis view on computed tomography (dense calcified pericardium shown by the white arrow). B. Magnetic resonance imaging (balanced steady state free precession sequence in the long-axis view) (signal void due to thickened pericardium shown by the white arrow).

Figure 3. Magnetic resonance imaging: left ventricular hypertrophy and thickened pericardium (4 to 10 mm thick) grasping both ventricles. The pericardium appears as a thickened black line (white arrows) (black blood T1-weighted spin echo sequence in the short-axis view).

Figure 4. Coronary artery angiography: existence of triple-vessel coronary artery disease (multiple severe coronary artery stenoses on [A] left and [B] right coronary arteries indicated by black arrows).
Calcified constrictive pericarditis can hide other cardiac diseases. Investigations were performed before planned surgery; since echocardiography was not suitable, right heart catheterization was then performed, and revealed normal pulmonary artery pressures (systolic 32 mmHg, mean 22 mmHg, mean capillary wedge 17 mmHg) and a relatively low cardiac output (3 L/min). Analysis of pressure curves showed a certain degree of constriction, expressed by a levelling of right-sided pressures despite the absence of a typical aspect of dip-plateau on right ventricular pressure curves. Left ventricular angiography showed hypertrophic cardiomyopathy with a normal ejection fraction of 68%, and coronary angiography revealed the presence of extensive triple-vessel coronary artery disease (Fig. 4).

Owing to the patient’s age, her alteration in general status, and the presence of multiple cardiac diseases (multivessel coronary artery disease, hypertrophic cardiomyopathy and calcified pericarditis with a degree of constriction), the final decision was medical therapy, in agreement with the patient and her family. A beta-blocker at a low dose was initiated. However, no specific antibiotic therapy against the suspected infection was given due to the lack of proof concerning tuberculosis, hepatic alterations and the weak efficacy generally associated with tuberculosis antibiotherapy at this stage of the disease.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.acvd.2008.11.005.