Eggshell-Shaped Calcified Pericardium and Constrictive Pericarditis

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This 88-year-old man presented with progressive shortness of breath, poor appetite, and general malaise in the preceding 3 years. Bilateral lower legs edema became severe gradually. He had a medical history of diabetes mellitus and old ischemic stroke. He had an old pulmonary tuberculosis infection with complete treatment several years ago. Physical examination revealed paradoxical distention of the internal jugular vein, known as Kussmaul’s sign, abdominal distention, and grade 3 pitting edema of bilateral legs. Chest radiography (posterior-anterior view and lateral view) revealed eggshell-shaped calcification of the pericardium (Fig. 1A, B), and bilateral pleural effusion. Chest computed tomography (CT) revealed diffusely calcific pericardium (Fig. 1C-F), small pericardial effusion, and fibrothorax. Echocardiography showed adequate left ventricular systolic function, thickened calcific pericardium, abrupt flattening of posterior wall of the left ventricle, and preserved mitral septal annulus velocity. Coronary angiography revealed no significant stenosis in the overall coronary artery trees, but severe calcification of the pericardium presented under fluoroscopy (Fig. 1G). The hemodynamic study showed prominent x and y descents of the blood pressure curve of the right atrium, and equalization of the diastolic pressure in all four chambers with a “square root” sign (Fig. 1H) and diagnosed as constrictive pericarditis. Constrictive pericarditis (CP) caused by tuberculosis presented.

**Fig. 1.** (A, B) Posterior-anterior view and lateral view of chest radiography: Eggshell-shaped calcification of the pericardium was noted (black arrows), and bilateral pleural effusion. (C-F) Computed tomography: Diffusely calcific pericardium was like eggshell-shaped. (white arrows). (G) Fluoroscopy: Severe calcification with gray to black enhancement of the pericardium presented. (H) The hemodynamic study: A prominent x and y descents of the blood pressure curve of the right atrium, and equalization of the diastolic pressure in all four chambers with a “square root” sign presented.

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Infection was highly suspected. The patient refused surgery due to old age and procedural risks of pericardiectomy, and his symptoms were partially relieved after adjustment of medical treatment.

CP is caused by fibrosis and calcification of the pericardium, which inhibits the diastolic filling of the heart. Therefore, the clinical manifestations of CP include features of left heart failure (pulmonary edema, bilateral pleural effusion, and dyspnea) and right heart failure (ascites, peripheral pitting edema, and abdominal distension). Kussmaul's sign means paradoxical rise in jugular venous pressure or failure to fall on inspiration. Possible causative factors of CP include Coxsackie B virus, radiation therapy, trauma, cardiac surgery, tuberculosis, malignancy, inflammatory, and connective tissue diseases. The presence of calcification is related to a chronic course where any causative factors induce a chronically pericardial inflammation and subsequent healing with granulation tissue formation leading to development of adhesion and calcification. For such patients, the management would be a surgical pericardiectomy, but there is a higher surgical risk with chances of incomplete pericardial resection and an increased possibility of poor hemodynamic outcomes following surgery in cases with heavily calcified pericardium. In patients with CP, pericardial calcifications are a frequent finding on CT. The eggshell-shaped calcified pericardium is a rare manifestation and almost the whole heart is encased in plaque-like pericardial calcification. In our case, the patient refused surgical intervention, and symptoms were relieved by adjustment of medical treatment.

CONFLICT OF INTEREST STATEMENT

None declared.

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