Silent Armor Around the Heart: Calcific Pericarditis in a Young Man: A Case Report

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SOUHRN

Na oddělení urgentního příjmu byl přivezen 48letý muž pro bolesti žaludku a únavu. Netrpěl žádnou známou komorbiditou, jednalo se o nekuřáka a v jeho rodinné anamnéze nebylo žádné srdeční, ani jiné významnější onemocnění. Výsledky EKG a rentgenového vyšetření byly normální, zato vyšetření hrudníku výpočetní tomografií odhalilo malý perikardiální výpotek, ztluštění a kalcifikaci na bázi srdce. Transtorakální echokardiografie prokázala malý výpotek kolem perikardu, jeho ztluštění a zvýšenou echogenitu. Vyšetření srdce magnetickou rezonancí potvrdilo výpotek a ztluštění perikardu; nález odpovídal konstriktivní perikarditidě. Popisujeme tento zajímavý případ rozsáhlé kalcifikující perikarditidy nijak neupozorňující na závažnost stavu a bez přítomnosti dyspnoe, otoků nebo bolesti na hrudi.

Keywords:
Cardiac magnetic resonance
Chronic constrictive pericarditis
Computerized tomography

ABSTRACT

A 48-year-old male patient was admitted to the emergency room complaining of stomach pain and fatigue. He had no known comorbid diseases, was a non-smoker, and had no family history of heart disease or any other disease. Electrocardiography and X-ray were normal, but a thoracic computerized tomography scan revealed mild pericardial effusion, thickening, and calcification at the base of the heart. Transthoracic echocardiography showed mild pericardial effusion around the pericardium, thickening, and increased echogenicity. Cardiac magnetic resonance imaging confirmed pericardial effusion and thickening. The findings were consistent with constrictive pericarditis. We present the story of this interesting case due to the grand calcific pericarditis with silent complaint and no dyspnea, edema, or chest pain.

Introduction

Constrictive pericarditis involves the formation of granulation in the pericardial tissue and a loss of pericardial elasticity due to calcium deposition. In this condition, the pericardium envelops the heart like armor, and myocardial relaxation is disrupted during diastole. Patients typically present with vague complaints, with diastolic heart failure and indications of right heart failure being most prominent.

A 48-year-old male patient was admitted to the emergency room complaining of stomach pain and fatigue. He had no known comorbid diseases, was a non-smoker, and had no family history of heart disease or any other disease.

The head and neck examinations were normal, and the heart sounds were deep. Lung sounds are normal, and there are no pathological findings on abdominal examination. There was no pretibial edema on her legs.

Laboratory: hemoglobin was 14.1 g/dL, white blood count was 7 × 10^3, C-reactive protein was 4 mg/dl, troponin I was 4 nanogram/l (within normal limits), creatine level was 0.8 mg/dL. Liver tests were normal.

Electrocardiogram: The rhythm was normal sinus rhythm, and the heart rate was 75/min beat. The T wave was negative in D2,3 and aVF derivations, and all derivations had low voltage findings (**Fig. 1A**).

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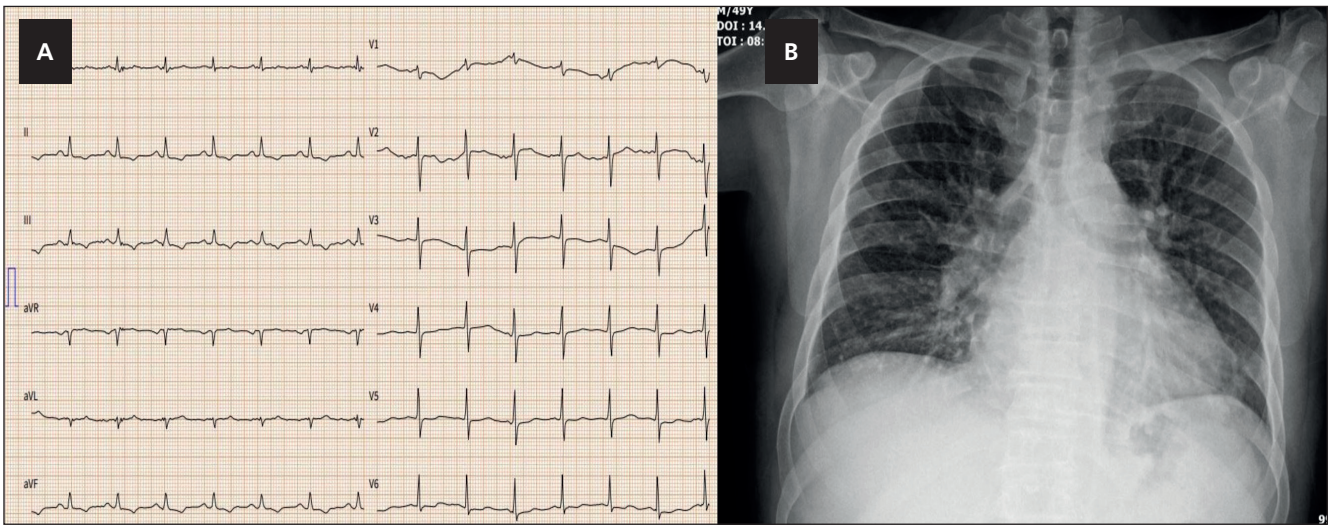

Fig. 1 – Electrocardiography and chest X-ray of the patient on admission to the emergency department.

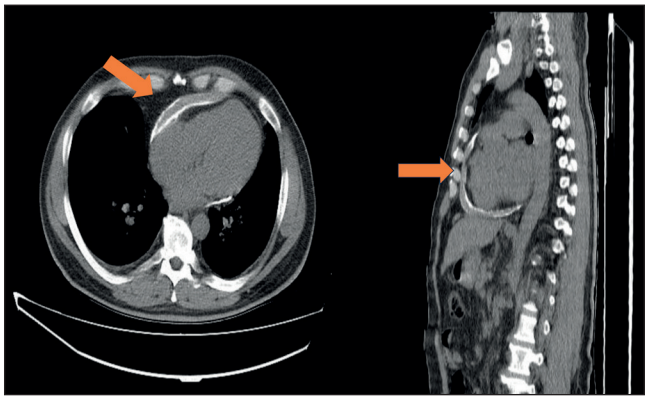

Fig. 2 – Calcification around the pericardium in the computerized tomography taken due to the patient's complaints.

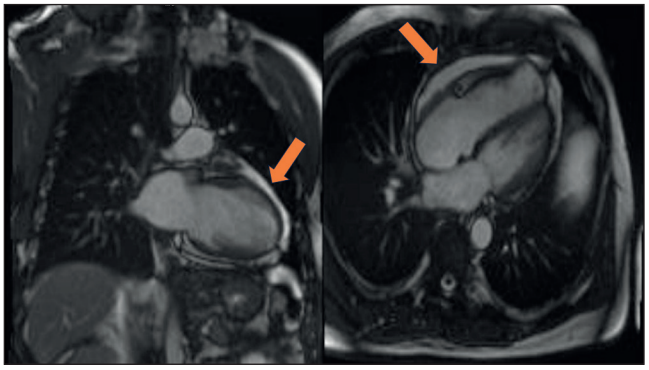

Fig. 3 – Cardiac magnetic resonance images showed pericardial thickening and calcification.

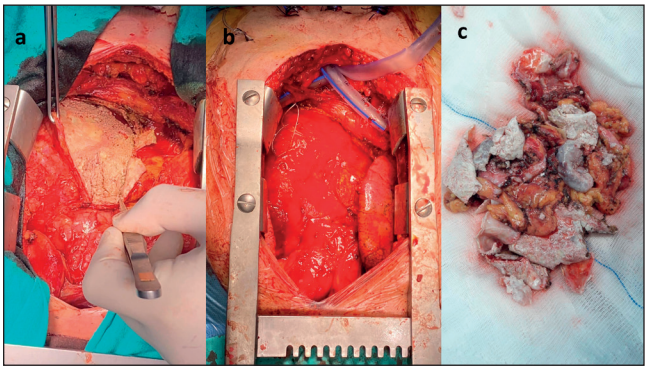

Fig. 4 – Surgical demonstration of the patient's calcified pericardium (A), the postoperative image of the heart with the calcification removed (B), and the image of the removed calcified pericardium (C).

Chest X-ray showed the heart size was normal width, and no obvious calcification was observed around the heart (**Fig. 1B**).

Thoracic computerized tomography scan revealed mild pericardial effusion, pericardial thickening, and pericardial calcification at the base of the heart (**Fig. 2**).

Transthoracic echocardiography was performed in an emergency room. Ejection fraction was normal, mild pericardial effusion around the pericardium, pericardial thickening, and increased echogenicity were observed.

Cardiac magnetic resonance ımaging showed pericardial effusion (14 mm) and pericardial thickening, as well as a decrease in left ventricular EF (LVEF 55%) and an increase in left atrial volume. The findings were consistent with constrictive pericarditis (**Fig. 3**).

Coronary angiography demonstrated coronary arteries were normal, left ventricular and diastolic pressures were increased, and in right heart catheterization, there was an increase in right ventricular end-diastolic pressure. Pulmonary artery wedge pressure was increased.

When all the patient's findings were combined, it was concluded that he had calcific constrictive pericarditis. A consensus decision was made considering all the results and the relevant latest guideline recommendations, and pericardiectomy was considered appropriate.

The decision for surgery was shared with the patient, and verbal and written consent was obtained.

The patient underwent pericardiectomy due to constrictive pericarditis (**Fig. 4 A, B**). Thickened pericardial tissue containing intense calcification was observed in the surgical materials (**Fig. 4C**).

The pericardial biopsy result was interpreted as compatible with the findings of inflamed fibrinous material.

In the materials taken for the differential diagnosis of tuberculosis, no bacilli were detected, and no tuberculosis-specific pathological necrosis was observed.

The patient was not symptomatic during the postoperative check-up, follow-up echocardiograms were normal, and he was discharged with recommendations. There have been no problems with follow-up visits for regular check-ups for 3 years.

Discussion

Constrictive pericarditis is characterized by the formation of granulation in the pericardial tissue. There is a loss of pericardial elasticity, and this leads to restriction in ventricular filling.[1] The leading cause of constrictive pericarditis worldwide is tuberculosis, and it can occur even in patients receiving antituberculous therapy. In developed countries, idiopathic or post-viral infections are the most common causes. Other important causes may occur as complications after heart surgery and after the radiation therapy to the mediastinum. It may also be associated with connective tissue diseases. Often, as in our patient, it is very difficult to determine a definitive etiological cause. It is often thought to be an asymptomatic attack of viral pericarditis.[2,3]

Constructive calcific pericarditis is a clinical condition that is rare in young patients and is particularly characterized by right heart failure findings. According to the latest guideline, the first method recommended for the diagnosis of constrictive pericarditis is echocardiography.[4] At the time of the first admission to our hospital, a chest X-ray was requested due to shortness of breath, and then a thorax CT was requested. Upon observation of pericardial calcification on thorax CT, the patient underwent echocardiography under emergency conditions. Afterward, cardiac magnetic resonance was performed to confirm the diagnosis and elucidate the etiology. All guideline recommendations were completed in the patient, who also underwent cardiac catheterization.

Since our patient did not develop signs of right heart failure, there were no findings suspicious for constrictive pericarditis in the physical examination. Patients with constrictive pericarditis often have vague complaints such as fatigue, weakness, and shortness of breath. They may also present with high fever, palpitations, paroxysmal nocturnal dyspnea, sweating, and swelling in the feet.

Low voltage, T wave changes, and even atrial fibrillation can often be seen in the ECGs of these patients. Our patient also had low voltage and T-wave changes. In echocardiography, minimal effusion and thickening were observed only in the pericardium. The patient was diagnosed with constrictive pericarditis after imaging examinations and a surgical method was recommended for the treatment of the disease and clarification of the etiology.

A pericardiectomy is the accepted standard treatment for patients with chronic constrictive pericarditis with persistent and significant symptoms.[4]

Surgical removal of the pericardium has a mortality rate ranging from 6–12%. End-stage patients benefit little from pericardiectomy, and operative mortality is increased. Recently, cachexia, atrial fibrillation, low flow rate findings, hypoalbuminemia due to protein loss enteropathy, cardiac cirrhosis, and impaired liver tests have been observed.[5]

If the underlying cause is known, medical treatment can be initiated (e.g., tuberculosis or other infectious diseases, connective tissue diseases). Although no etiological cause can be found in our hospital, the patient comes for regular check-ups after surgery without needing medical treatment.

Conflict of interest
The authors declare no conflict of interest.

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Ethical statement
This case report includes both verbal and written patient consent. No experiments on humans are performed. It complies with the ethical standards of our institution's committee or the Declaration of Helsinki and its revisions.

Informed consent
Written informed consent was obtained from the patient for the publication of this article.

AI disclosure
This case report did not use artificial intelligence (AI)-powered technologies (such as Large Language Models [LLMs], chatbots, or image generators).

Author contribution
Concept/design: SU, SSP. Data analysis/interpretation: Drafting article: SU, SKO. Critical revision of the article: SU, EK. Approval of article: SU, EAD. All authors discussed the results and contributed to the final manuscript. All authors provided critical feedback and helped shape the research, analysis, and manuscript. All authors read and approved the final version of the paper.

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