Localized constrictive pericarditis resulting in biventricular aneurysm-like apical outpouching

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Introduction

Constrictive pericarditis (CP) is a disease of the pericardium resulting from chronic inflammation or scarring. Adhesion of the pericardium to the myocardium limits diastolic expansion of the left and right ventricles and leads to ventricular failure (1). Although the entire pericardium is commonly involved, focal sparing rarely occurs which can result in unique morphological changes that may pose diagnostic challenges (1). We present a rare case of CP with apical sparing that caused an unusual protrusion of the biventricular apical wall. In addition, we highlight the importance of multimodality imaging.

Case Report

A 45-year-old man was admitted to our outpatient clinic with progressive dyspnea and weight loss. He had no history of chronic disease or previous surgery. He had a history of tuberculosis 15 years earlier for which he was treated medically. Findings of examination consisted of paleness of the skin. Physical examination showed increased distention in jugular veins and paradoxical inspirational venous response. Chest x-ray showed calcified cardiac silhouette, which raised suspicion of CP (Fig. 1). Echocardiography revealed increased echogenicity and a thickened pericardium, respiratory variations in transmitral flow, dilated inferior vena cava, and apical left ventricle (LV) ballooning with septal bounce as a sign of ventricular interdependence phenomenon (Fig. 2, Supplemental Videos 1-3). For better evaluation of the pericardium and to exclude coronary artery disease, computed tomography angiography was performed and showed no significant coronary artery disease. Diffuse pericardial calcification up to 17 mm in the thickest part of the pericardium, increased liver dimensions, bilateral pleural effusion, increased calibration of vena cavae, and biventricular apical outpouching where the pericardium was free of calcification and within normal thickness were revealed (Fig. 3a and 3b). Before catheterization, we ordered cardiac magnetic resonance (CMR) to further evaluate the findings on echocardiog-

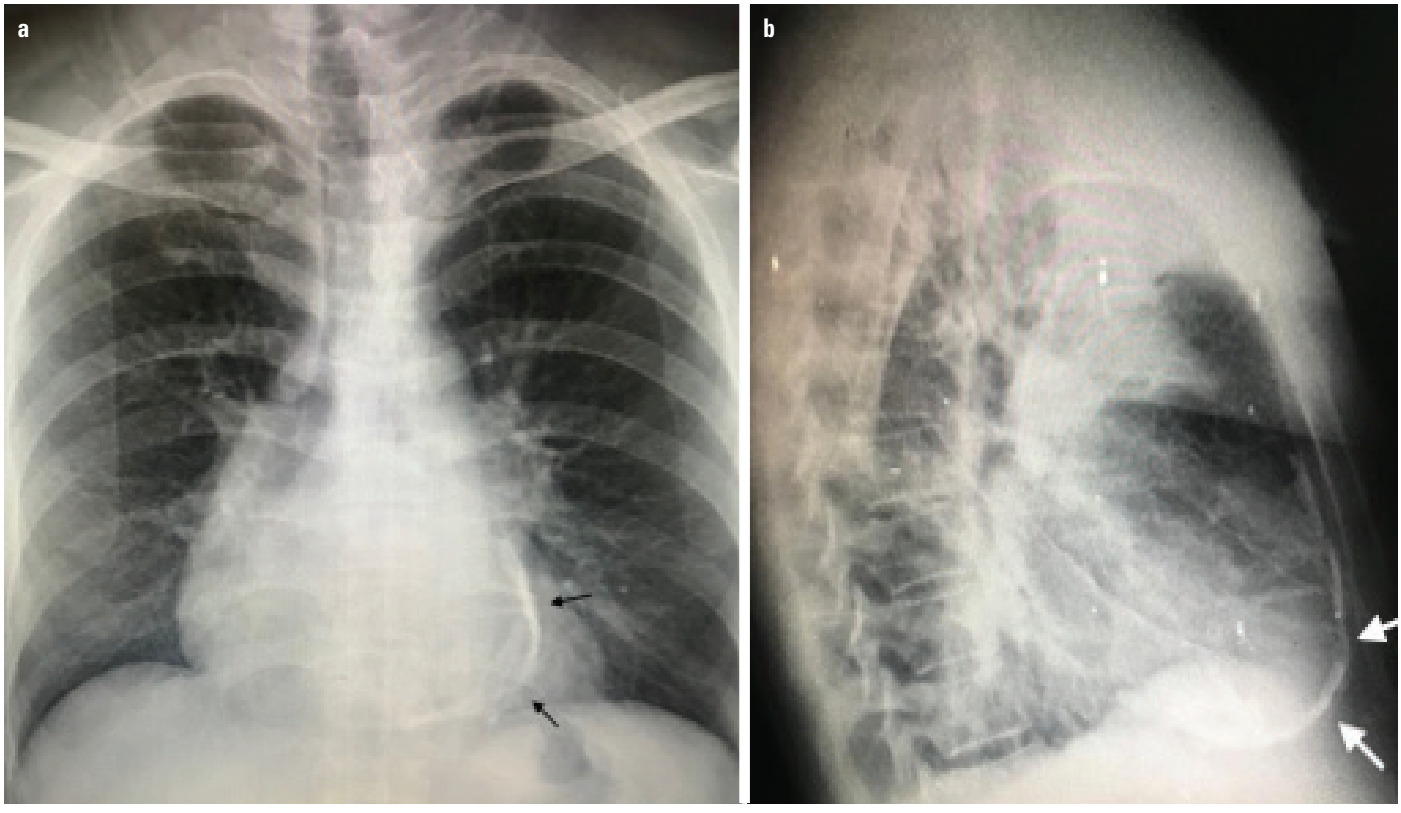

Figure 1. (a) Chest x-ray image showing a calcified pericardial thickening (black arrows) surrounding the ventricular wall. (b) Lateral x-ray of the chest showing the calcified pericardium (white arrows)

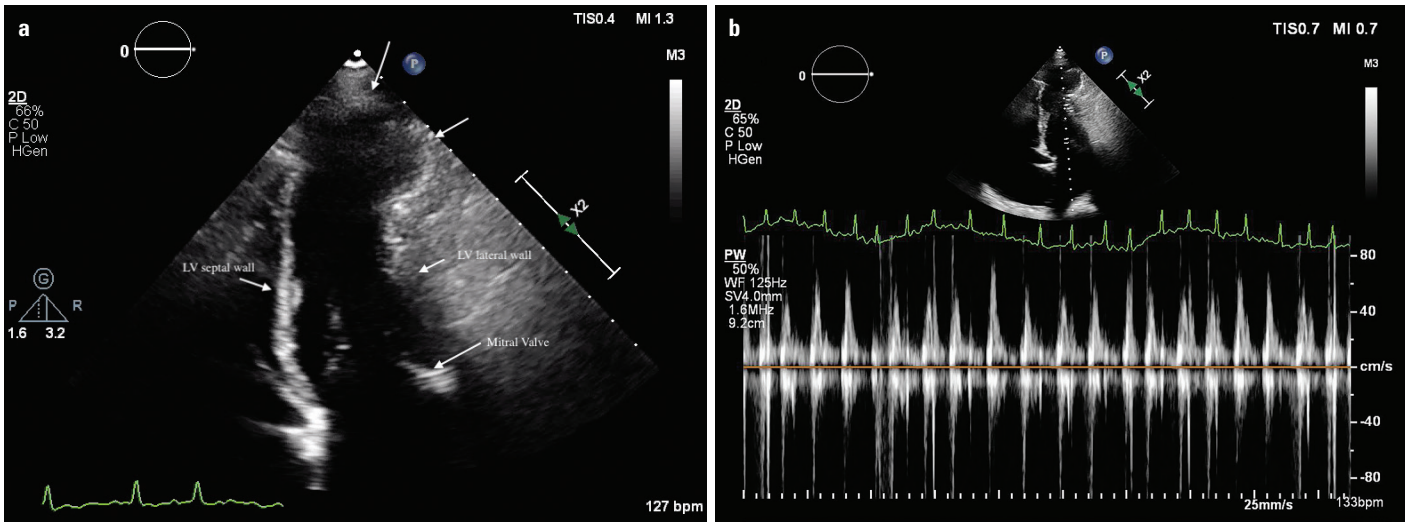

Figure 2. (a) Echocardiography image showing the LV apical outpouching (white arrows). (b) Pulse wave Doppler images of the mitral inflow, which showed respiratory variations

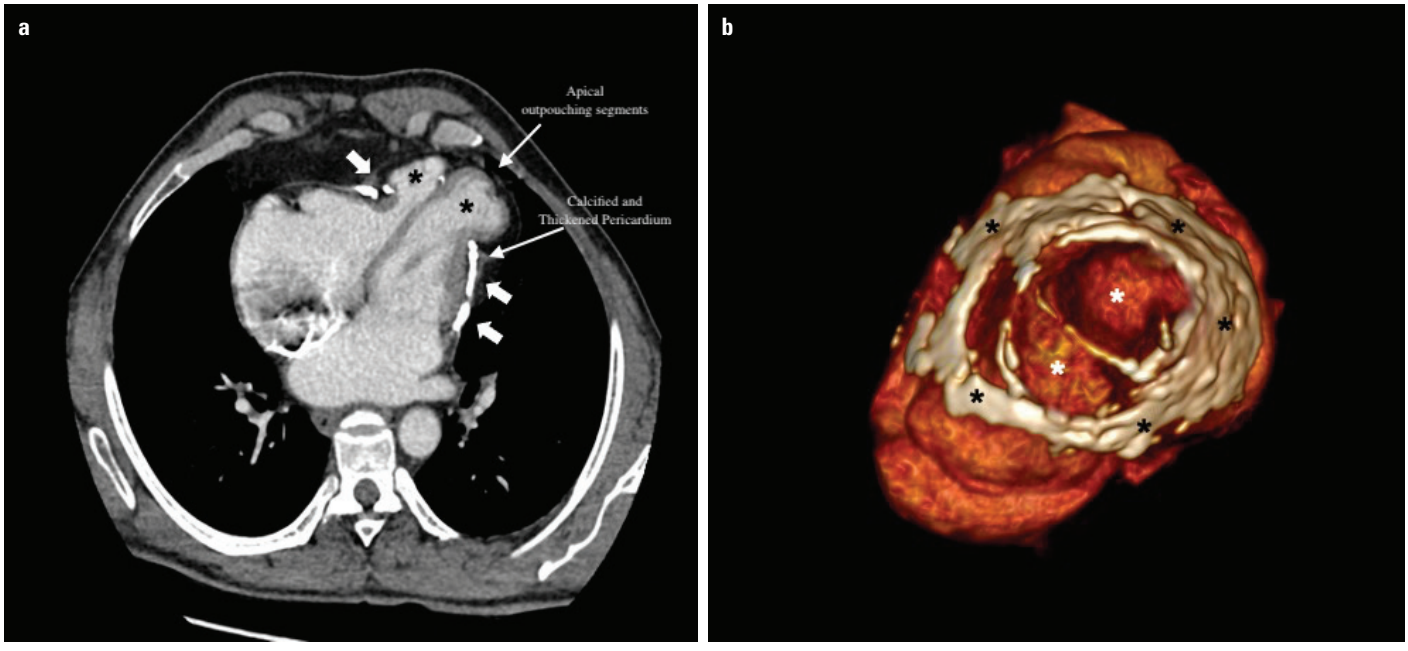

Figure 3. (a) Transverse contrast enhanced CT image depicting the calcified pericardial thickening of the basal and midventricular segments (thick white arrows), whereas the apical pericardium is relatively spared. Biventricular apical outpouching is noted (asterisk). (b) 3-dimensional volume rendering image of the heart clearly showing the calcified pericardial plaques (black asterisk) encircling both ventricles. Both apices are ballooning (white asterisk) through the neck of the pericardial calcified plaque

raphy. CMR revealed thickening of the basal and midventricular portion of the pericardium with a relatively spared apical segment, and biventricular apical outpouching through the spared pericardium. The apical segment outpouching had a narrow neck without wall motion abnormality or fibrosis on late gadolinium images (Fig. 4a and 4b). Septal bounce was also noted in CMR cine images (Supplemental Video 4). These findings suggested that ventricular expansion was possible only in the weakest part of the pericardium, which resulted in apical outpouching. Right and left heart catheterization was performed and demonstrated diastolic equalization of pressures with square root sign. All multimodality imaging suggested a constrictive physiology. Because the patient showed symptoms of increased diastolic filling pressures, the patient was evaluated by the heart team and surgery was scheduled as the treatment of choice.

Discussion

CP is usually suspected either because of symptoms of right heart failure or pericardial thickening noted during chest imaging. Right ventricular outpouching or right ventricular aneurysm in pa-

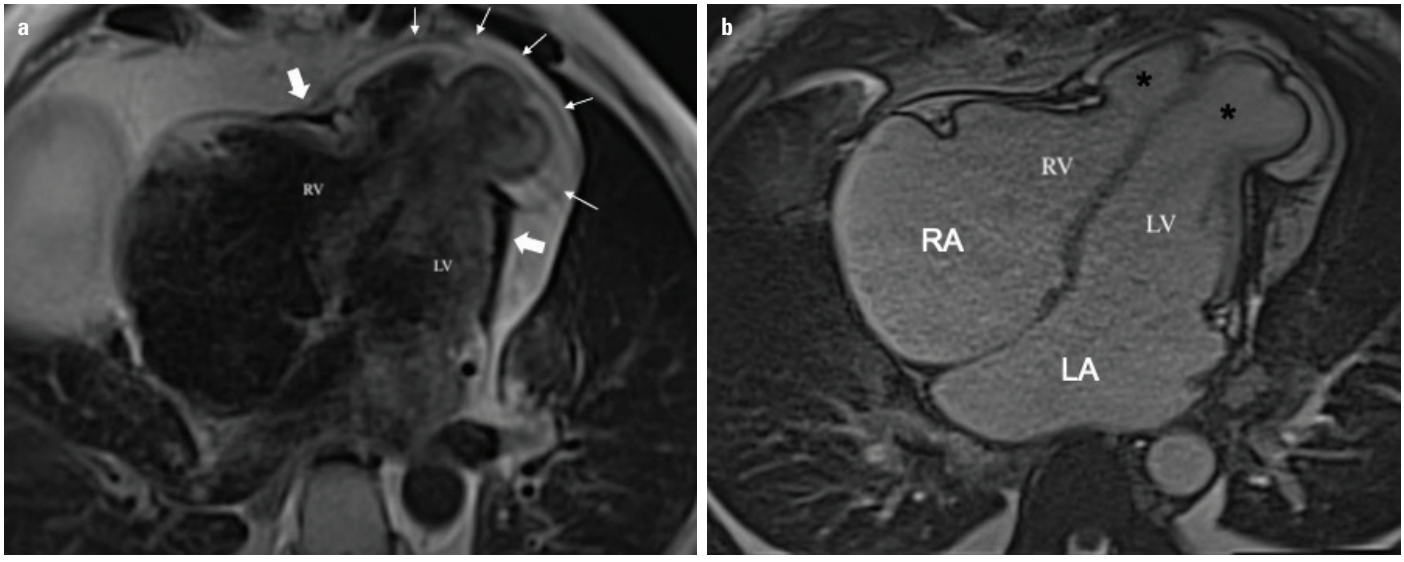

Figure 4. (a) Transverse T1-weighted CMR image correlating with the CT findings and better delineating the spared apical pericardium (small arrows). LV - left ventricle; RV - right ventricle. (b) Cine image intersecting of the 4-chamber plane demonstrating biventricular apical ballooning and biatrial dilatation. No wall motion abnormality was noted, and no fibrosis was detected on late gadolinium enhanced images (not shown)

tients with localized CP have been demonstrated in a few case reports in the literature (3, 4). Rankawa and Joshi (5) reported a case of CP with focal sparing that behaved like a defect in a thickened pericardium, resulting in protrusion and outpouching of a normal right ventricular myocardium like an aneurysm between the margins of thickened pericardium. There are 2 case reports in the literature illustrating localized CP with LV outpouching. Blaha et al. (6) reported a patient with localized CP in the LV compressing to the mid and apical segments of the LV and masquerading as a basal aneurysm. In another case report, Zacharaki et al. (7) reported a case with localized CP in the LV that caused cardiac apical diverticulum. In this case, CT images showed that the LV apex was outpouched from the spared part of the pericardium without calcification or thickening. To the extent of our knowledge, this is the first case that demonstrates biventricular apical outpouching owing to localized CP. The etiology of CP includes postinfectious, postradiation, postischemic, posttraumatic, and idiopathic causes. In developing countries, infectious etiology, especially tuberculosis, is the most prevalent cause. In our case, there was pericardial constriction with focal sparing of the apical pericardium, likely related to the patient's history of tuberculosis. The basal and midventricular calcified and thickened portions of the pericardium restricted the expansion of the effected ventricular segments and, as a consequence, apical segmental outpouching developed through the relatively spared apical pericardium.

A multimodality imaging approach plays a pivotal role in diagnosing CP and related morphological changes. In our case, echocardiography was used as the first-line imaging modality to gather fast and relevant data about the patient. CT was used to successfully delineate pericardial involvement and the extension of the disease. CMR provided further information about the affected outpouching ventricular wall, its signal, and its motion characteristics, which were used to exclude other potential differentials such as ischemic aneurysm and pseudoaneurysm, mural thrombus, and right ventricular pathologies. This was of great importance for patient management.

Conclusion

In conclusion, we present the first case of localized CP causing biventricular apical outpouching and highlight the importance of multimodality imaging. Any ventricular wall changes not relevant to the clinical context should warrant further examination to establish the correct diagnosis.

Informed consent: The patient has given informed consent for publication of this case report including the radiologic imaging methods.

Supplemental Video 1. Echocardiographic video showing apical left ventricle (LV) ballooning with septal bounce as a sign of ventricular interdependence phenomenon.

Supplemental Video 2. Apical view of echocardiographic evaluation shows left ventricular apical ballooning together with septal bounce.

Supplemental Video 3. Echocardiographic video showing the dilated inferior vena cava.

Supplemental Video 4. Cine MRI image showing biventricular apical outpouching and septal bounce as a sign of ventricular interdependence

References

1. Maisch B, Seferović PM, Ristić AD, Erbel R, Rienmüller R, Adler Y, et al.; Task Force on the Diagnosis and Management of Pricardial

Diseases of the European Society of Cardiology. Guidelines on the diagnosis and management of pericardial diseases executive summary; The Task force on the diagnosis and management of pericardial diseases of the European Society of Cardiology. Eur Heart J 2004; 25: 587-610.
2. Hasuda T, Satoh T, Yamada N, Sakamaki F, Kyotani S, Nakanishi N, et al. A case of constrictive pericarditis with local thickening of the pericardium without manifest ventricular interdependence. Cardiology 1999; 92: 214-6.
3. Ojha V, Vadher A, Chandrashekhara SH, Kumar S. Constrictive Pericarditis With a Right Ventricular Apical Outpouching Masquerading as a Diverticulum. Ann Thorac Surg 2019; 108: e203.
4. Ocak I, Turkbey B, Lacomis JM. Constrictive pericarditis presenting with an outpouching of the right ventricle free wall simulating an aneurysmal dilatation. Br J Radiol 2011; 84: e58-61.
5. Ranwaka A, Joshi MV. Aneurysm-like outpouching of ventricular wall through a gap caused by constrictive pericarditis with focal sparing. Int J Cardiovasc Imaging 2015; 31: 1071-3.
6. Blaha MJ, Panjrath G, Chacko M, Schulman SP. Localized calcific constrictive pericarditis masquerading as a basal aneurysm. J Am Coll Cardiol 2011; 57: e65.
7. Zacharaki A, Patrianakos AP, Pagonidis K, Parthenakis FI, Vardas PE. Localized constrictive pericarditis mimicking a cardiac apex diverticulum. Int J Cardiol 2015; 184: 366-8.

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