

Effusive-constrictive pericarditis in a patient with end-stage renal disease (RCD code: VI-4D.1)

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Abstract

Renal disease, especially end-stage renal disease, is associated with possible pericardial involvement. We report the case of a 51-year-old patient with a history of end-stage-renal disease, currently receiving chronic haemodialysis. The patient was admitted to the ward due decreased exercise tolerance and elevation of inflammatory markers. Features of active exudative-constrictive pericarditis were detected upon imaging studies (echocardiography, computed tomography). The haemodialysis regimen was intensified and anti-inflammatory treatment was administered. In the follow-up examination, the patient presented with improved clinical status and echocardiography revealed a decreased amount of pericardial fluid with no pattern of constriction. The elevation of CRP and evidence of active pericardial inflammation upon contrast enhancement seen in CT may help to identify patients with potentially reversible forms of pericardial constriction. In this group of patients, in addition to intensification of dialysis, anti-inflammatory therapy should be considered to prevent the need for pericardiectomy. JRC D 2019; 4 (2): xx-xx

Key words: rare disease, echocardiography, end-stage renal disease, pericarditis, transient constrictive pericarditis

Introduction

Renal disease, especially end-stage renal disease, is associated with possible pericardial involvement [1]. Pericardial involvement in ESRD is manifested most commonly by acute pericarditis and chronic pericardial effusion and infrequently as chronic constrictive pericarditis (CP) [1, 2]. In addition to the classic form of chronic permanent CP, we identify a form of transient CP which resolves with anti-inflammatory therapy [3]. Multimodality imaging with echocardiography, computed tomography (CT), or cardiac magnetic resonance (CMR) may be helpful in the evaluation of pericardial inflammation [4, 5].

Case report

A 51-year-old patient with end-stage renal disease (ESRD) secondary to glomerulonephritis was admitted to the ward due to decreased exercise tolerance (New York Heart Association [NYHA]

functional class III) and elevated inflammatory markers. The patient was receiving chronic dialysis, initially with peritoneal dialysis which was transitioned to haemodialysis. Furthermore, the patient had undergone two kidney transplants and graftectomy due to adenocarcinoma in the transplanted kidney. In laboratory tests, the initial c-reactive protein (CRP) level was 137 mg/dl, procalcitonin level was normal, and blood cultures (aerobic and anaerobic) were negative. Transthoracic echocardiography (TTE) revealed pericardial effusion, mitral inflow respiratory variation > 25%, abnormal motion of the interventricular septum (septal shudder), along with dilatation of the inferior vena cava (IVC) (2.3 cm) and cessation of its respiratory motion. Sizes of cardiac chambers were normal, left ventricular end-diastolic dimension (LVEEd): 4.6 cm, right ventricular outflow track (RVOT): 3.0 cm₂, left atrial area (LAAr): 19 cm², right atrial area (RAAr): 18 cm². Additionally, wall thickness was in the normal range, interventricular septum (IVS): 1.1 cm, posterior wall (PW): 0.8 cm. The lack of atrial enlargement indicated a non-chronic process of constriction in the pericardium and possibly a reversible form of

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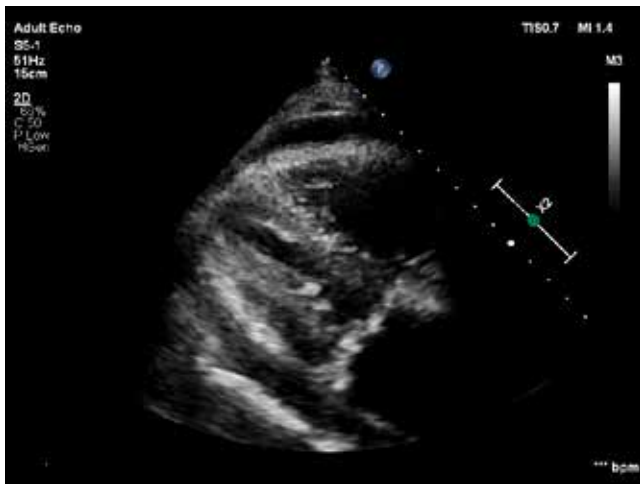
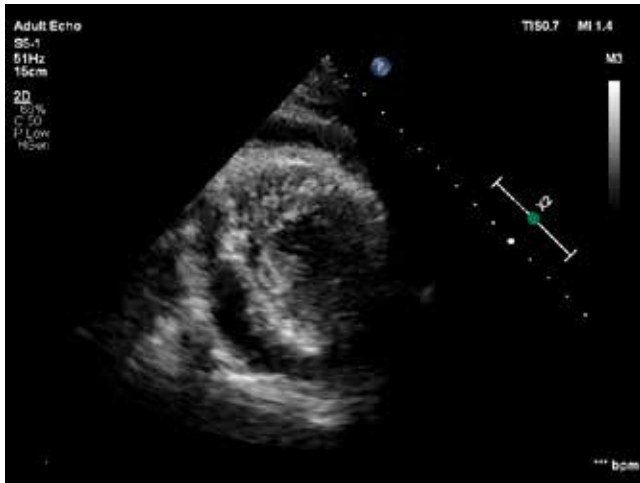


Figure 1. TTE- pericardial effusion

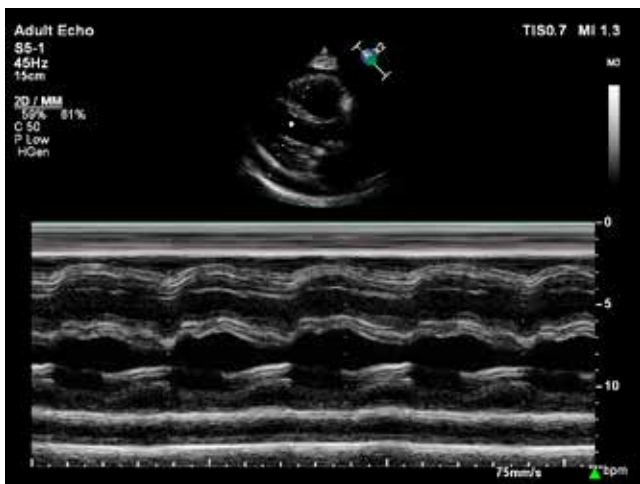


Figure 2. TTE- M-mode presentation- abnormal motion of the interventricular septum (septal shudder)

constrictive pericarditis (CP). Computed tomography (CT) scans of the thorax, abdomen, and pelvis minor did not reveal any malignancies. Imaging of the pericardial sac revealed a fluid layer with thickness of 18 mm and density of 10–20 HU, thickening of pericardial layers (2.8–3 mm), contrast enhancement of peri-

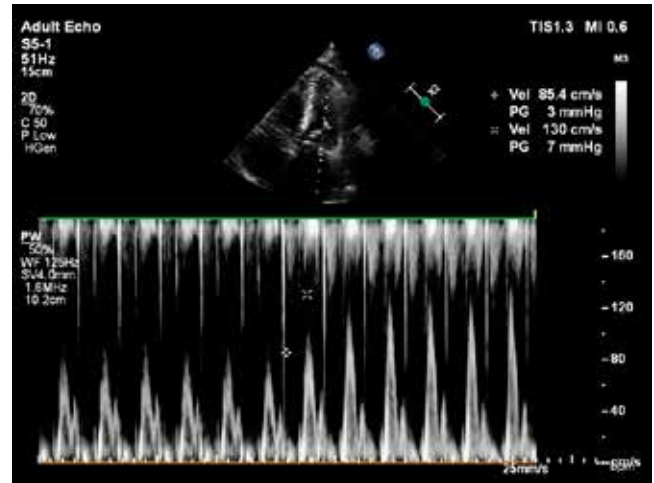


Figure 3. PW doppler- mitral inflow respiratory variation > 25%

cardial layers, indistinct adjacent adipose tissue with bands of increased density, as well as contrast reflux into the IVC and hepatic veins. Moreover, a slight amount of fluid was seen in the pleural cavities and the peritoneal cavity. Due to the observed enlargement of mediastinal lymph nodes, bronchoscopy was performed, which did not reveal any pathology within the respiratory tract. Laboratory tests revealed the following: cardiac markers were within normal range (troponin, CK, CK-MB), tumour markers (AFP, CEA, CA 15–3, CA 19–9) were negative, virological tests of the IgM variety (CMV, EBV, Enterovirus, Coxsackievirus) were negative, and QuantiFERON-TB test was negative. Serology examination did not detect signs of autoimmune disease.

Because features of effusive-constrictive pericarditis were seen on imaging studies, along with elevated inflammatory markers, the patient's dialysis regimen was intensified. Additionally, anti-inflammatory therapy was initiated using ibuprofen with an initial dosage of 3×600 mg/day. Moreover, colchicine was administered at a dosage of 1×0.5 mg/day throughout the period of hospitalization. Considering the presence of ESRD and chronic haemodialysis, the decision was made to decrease the dosage of colchicine. Total duration of anti-inflammatory therapy, which was gradually decreased after the patient's condition had become stable, was 3 months. At the 1-month follow-up examination, the patient reported that he was feeling better and his exercise tolerance had increased. TTE revealed decreased pericardial effusion, normal size and respiratory motion of the IVC, as well as decreased mitral inflow respiratory variation.

Discussion

CP may be a complication of bacterial (especially purulent and tuberculous), neoplastic, or autoimmune pericarditis, but may also be the result of cardiac surgeries and radiotherapy. CP is rarely caused by viral or idiopathic pericarditis [6]. The pathological condition is probably the result of the evolution of subacute/chronic pericarditis into CP and there may be a long period between an episode of acute pericarditis and the development of constrictive lesions [7]. The clinical picture of CP consists of signs of right

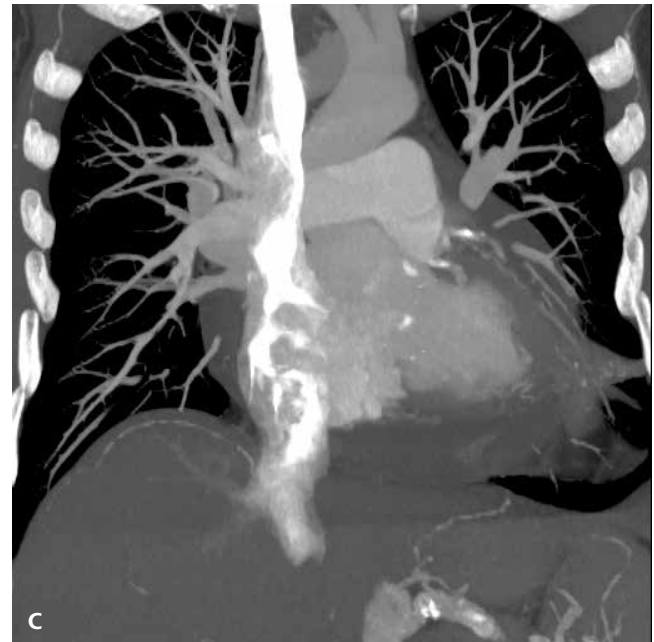


Figure 4. CT with iodinated contrast- pericardial fluid, thickened pericardial layers, contrast enhancement of pericardium layers (A, B), contrast reflux into IVC and hepatic veins (C)

heart failure (peripheral oedemas, hepatomegaly, ascites, pleural effusions), as well as signs of low cardiac output (with normal left ventricular ejection fraction) [8]. Renal diseases, especially ESRD, are connected with an increased risk of pericarditis. There are three possible causes of pericarditis in patients with uraemia: lesions connected with the renal failure itself (before or during

the initial 8 weeks of renal replacement therapy), lesions connected with dialysis (once the patient's condition has stabilized due to renal replacement therapy), and very rarely, constrictive lesions. Following initiation of dialysis therapy, the incidence of pericarditis in patients with ESRD decreases to about 5%. It must also be remembered that ESRD patients are more predisposed to chronic fluid accumulation in the pericardial cavity due to constant volume overload of the circulatory system [1].

In CP, gradual fibrosis is observed, followed by calcification and stiffening of the pericardium, which leads to elevation of diastolic pressures in the ventricles. The key aspect in the pathophysiology of CP is the interdependence of ventricles, which is limited by the stiffened pericardial sac. During inhalation, the right ventricle becomes filled at the expense of the left ventricle, which is observed in echocardiography as a movement of the interventricular septum towards the left ventricle. During exhalation, the opposite occurs, which is why a movement of the interventricular septum towards the right ventricle is observed, together with flow reversal in hepatic veins during cardiac diastole. Other echocardiographic signs of CP include: respiratory variation of mitral inflow E-wave > 25%, tricuspid inflow E-wave > 40%, pulmonary flow D-wave > 20%; mitral inflow profile with E/A ratio > 0.8, reduced deceleration and cessation/reduction of the respiratory motion of IVC, as well as its dilatation [9]. Assessment of mitral annular velocity with the use of tissue doppler imaging is vital for differentiating CP from restrictive cardiomyopathy (normal values of myocardial velocities and the presence of annulus paradoxus and annulus reversus in CP) [10]. In its classical form and in the case of advanced lesions, thickened pericardial layers and the presence of calcifications are present, although in as many as 20% of patients, constrictive lesions may occur even with regular thickness of the pericardium [11].

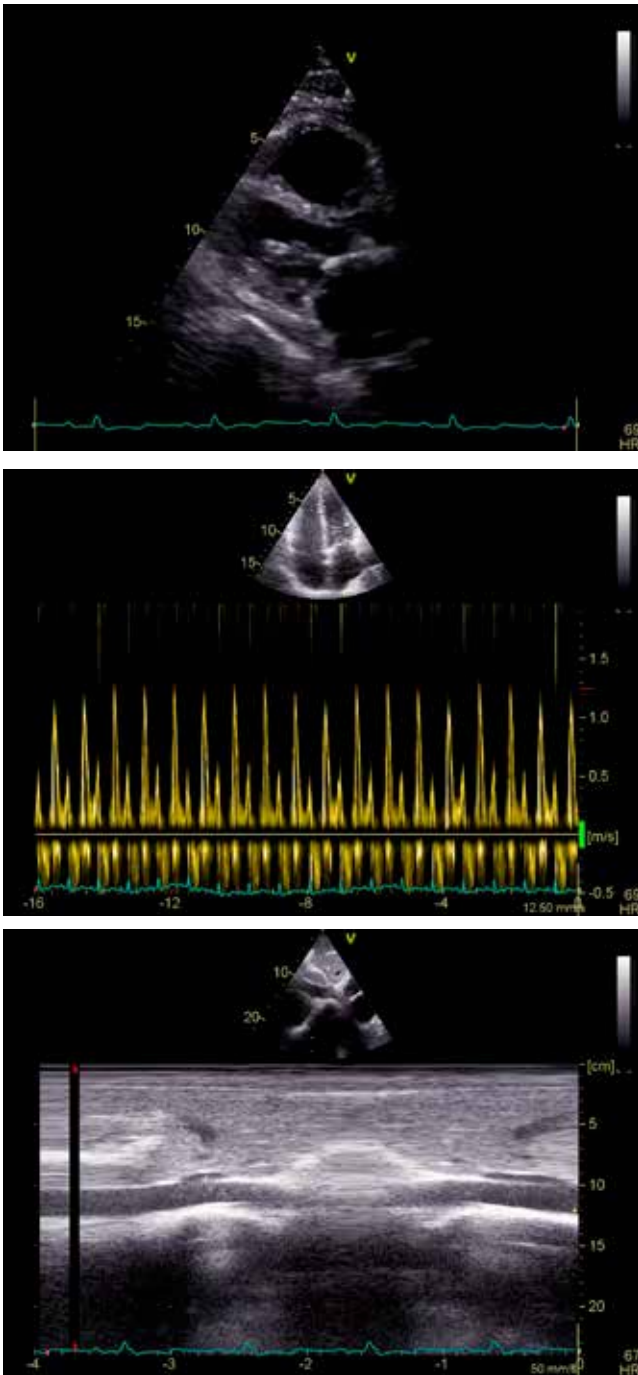


Figure 5. TTE after treatment

Apart from the chronic form of CP, there is also a transient form of CP, which resolves after anti-inflammatory treatment [3]. Therefore, if there are no indicators of the chronic form of the condition (e.g. atrial fibrillation, cachexia, hepatic impairment or the presence of calcifications in the pericardium), patients which are newly diagnosed with CP and are haemodynamically stable are advised to continue anti-inflammatory treatment for 2 – 3 months before a decision is made to perform pericardiectomy [12]. Inflammation of pericardium is visible in CT and/or CMR as signal enhancement after contrast has been administered, therefore, these imaging mo-

dalities may facilitate detection of an active inflammatory process in the pericardial sac [4, 13].

Conclusion

Although ESRD is rarely the cause of CP, when features of the condition are present in addition to signs of active inflammation (elevated CRP, contrast enhancement of pericardial layers in a CT scan), CP should be considered in the differential diagnosis. Intensification of dialysis along with initiation of anti-inflammatory therapy may result in regression of CP.

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