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Case Report

Persistent Constriction Despite Repeated Pericardial Drainage. A Case of Effusive-Constrictive Pericarditis Treated with Visceral Pericardiectomy

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Abstract

Effusive-Constrictive Pericarditis (ECP) is an uncommon clinical syndrome characterized by the coexistence of pericardial effusion and constriction involving the visceral pericardium. We present a case involving an obese man in his 30s who presented with a pericardial effusion alongside other comorbidities. Despite undergoing multiple rounds of pericardiocentesis and right heart catheterization without clinical improvement, the patient experienced significant relief following a pericardiectomy involving the visceral pericardium.

Keywords: Effusive-constrictive pericarditis; Pericardiocentesis; Pericardiectomy

Introduction

Effusive-Constrictive Pericarditis (ECP) is a rare clinical syndrome characterized by the simultaneous presence of pericardial effusion and constriction involving the visceral pericardium. This differs from typical constrictive pericarditis, marked by thickening and contraction of the parietal pericardium without effusion [1].

Case Presentation

A 32-year-old man with morbid obesity (BMI 53 kg/m^2), intellectual disability, and a history of hypertension presented with flu-like symptoms, anasarca, and acute renal failure. At an external medical facility, his renal failure was suspected to be due to hemolytic uremic syndrome and managed with intermittent acute dialysis. A transthoracic echocardiogram revealed a pericardial effusion, leading to subxiphoid tube pericardiostomy. The patient received pulse steroids, and a pathological examination confirmed fibrinous pericarditis, prompting transfer to our hospital. Renal stabilization occurred without further dialysis, but recurrent pericardial effusion necessitated repeated interventions.

Pericardial fluid cultures revealed *Enterococcus faecium*, followed by Bacteroides a week later. The patient exhibited deteriorating renal function, cool extremities, and elevated lactate levels, raising concerns about cardiogenic shock attributed to effusive-constrictive pericarditis. Aggressive diuresis and Dobutamine treatment were initiated. Right Heart Catheterization (RHC) and Left Heart Catheterization (LHC) revealed significantly reduced filling pressures, consistent with constrictive physiology but without ventricular interdependence (Table 1 and 2).

The patient's suitability for cardiac MRI was hindered by morbid obesity and intellectual disability. Faced with clinical deterioration and a strong suspicion of effusive-constrictive pericarditis, a sternotomy was performed. The parietal pericardium, approximately ½ cm thick, exhibited a leathery texture and was separated from the heart surface by ample space. Excision of the parietal pericardium, extending from phrenic nerve to phrenic nerve, was carried out. The visceral pericardium displayed a white, smooth sheen, incised with a 15-blade knife, revealing bulging epicardial heart fat. An anterior plane was meticulously developed, liberating the anterior part of the left ventricle, followed by the right ventricle, right atrium, and some left atrium, thereby freeing the heart from the constrictive layer.

Intraoperatively, the Central Venous Pressure (CVP) dropped from 22 to 15, and the left ventricular cavity size in diastole, measured by Transesophageal Echocardiography (TEE), increased from 42 cc to 62 cc post-pericardiectomy. Systolic function remained preserved. Mediastinal and bilateral pleural drains were strategically placed,

Table 1: Post pericardiocentesis RHC findings.

RA	27/24 mmHg (24)
PW	28/28 mmHg (27)
PA	30/23 mmHg (27)
CO(Fick)	5.48 L/min

Table 2: RHC findings following aggressive diuresis and Dobutamine.

RA	16 mmHg
RV	27/10 (13) mmHg
PA	29/12 (21) mmHg
PCWP	18 mmHg
LHC: LVEDP	94/5 (23) mmHg
CO (Fick)	7/57 L/min

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and the sternotomy was routinely closed. In the Intensive Care Unit (ICU), the patient's cardiac index reached 3.3 liters/min/m^2 BSA, accompanied by normal pulmonary artery pressures and a CVP of 8. Recovery proved gradual due to persistent renal disease necessitating intermittent dialysis and a compromised functional baseline, leading to tracheostomy for ventilator weaning. Nevertheless, the patient was ultimately discharged home, free from dialysis. The Institutional Review Board (IRB) waived the need for written consent on 03Apr, 2023.

Discussion

The uncommon disease process of effusive-constrictive pericarditis involves both constriction of the visceral pericardium and effusion causing a tamponade-like effect on the heart [1]. Conventional constriction manifests with physical examination findings such as Jugular Venous Distention (JVD), Beck's triad, Kussmaul sign, pericardial knock, and edema [2]. ECG tracings in constrictive pericarditis exhibit reduced voltages. Due to ventricular interdependence within a rigid pericardium, there is an observed increase in right ventricular volume relative to the left ventricle on echocardiography, leading to septal bounce [2]. Echocardiogram findings may reveal a restrictive left ventricular filling pattern [2]. Cardiac catheterization in constrictive pericarditis exhibits increased atrial pressure with prominent x and y descents, equalization of end-diastolic pressures, and a dip-and-plateau or square-root sign of ventricular diastolic pressure [2].

Unlike the conventional approach of treating typical pericardial constriction with pericardiectomy once the diagnosis is confirmed, ECP often responds favorably to as-needed (prn) pericardial drainage and anti-inflammatory medications. In a study at the Mayo Clinic examining ECP after pericardiocentesis, only 33 out of 205 patients undergoing pericardiocentesis were diagnosed with ECP, and only 2 patients required pericardiectomy due to persistent constrictive features [3].

Effusive-constrictive pericarditis initially presents as a pericardial effusion, addressed through pericardiocentesis [3]. However, in a subset of patients, sustained symptoms and altered hemodynamics persist post-pericardiocentesis, attributed to visceral constrictive pericarditis, leading to the designation of this condition as effusive-constrictive pericarditis.

Accurate diagnosis of effusive-constrictive pericarditis is achieved through simultaneous measurements of intrapericardial and right atrial pressures during pericardiocentesis [4]. It is defined by persistent elevation in right atrial, end-diastolic right ventricular, and left ventricular diastolic pressures even after intrapericardial pressure reduction through pericardiocentesis [4]. The initial hemodynamic presentation mimics cardiac tamponade, but despite fluid removal, hemodynamic abnormalities manifest as constriction, impacting cardiac function during diastole.

While both cardiac tamponade and constrictive pericarditis restrict heart filling, raising systemic and pulmonary venous pressures, the venous pressure waveforms differ between these conditions [5]. In effusive-constrictive pericarditis, the constricting visceral pericardial layer and the overlying pericardial effusion simultaneously contribute to reduced myocardial transmural pressure and filling [5]. This syndrome is considered an end-stage manifestation of persistent pericardial inflammation, characterized by the loss of pericardial elasticity through fibrosis and dystrophic calcification [4].

Diagnostic criteria for ECP include the failure of right atrial pressure to decrease by 50% or reach a new level below 10 mmHg after normalization of intrapericardial pressure [4]. Patients with ECP may exhibit symptoms resembling right-sided heart failure and volume overload due to limited intracardiac end-diastolic volume [6]. The initial therapeutic approach emphasizes pericardial fluid removal through pericardiocentesis, but in a subset of cases, fluid reaccumulation may necessitate repeated procedures. Treatment options range from medication management to pericardiectomy, with the definitive resolution achieved through the latter, involving the release of the visceral pericardium (Figure 1) [6].

Our patient exhibited remarkable hemodynamic improvement following sequential parietal and visceral pericardiectomy. It is crucial to emphasize that in any pericardiectomy procedure for constriction, the left ventricle should be liberated first, followed by the right ventricle. This sequential approach prevents the liberated right ventricle from potentially overloading the left ventricle, mitigating the risk of intraoperative pulmonary edema. The enhancement in diastolic capacitance was demonstrated through intraoperative Transesophageal Echocardiography (TEE), revealing a substantial increase in left ventricular diastolic volumes from 42 cc before to 62 cc after pericardiectomy. The heart dynamically emerged from the visceral pericardium; this impactful transformation is visually depicted in Figure 2 and 3.

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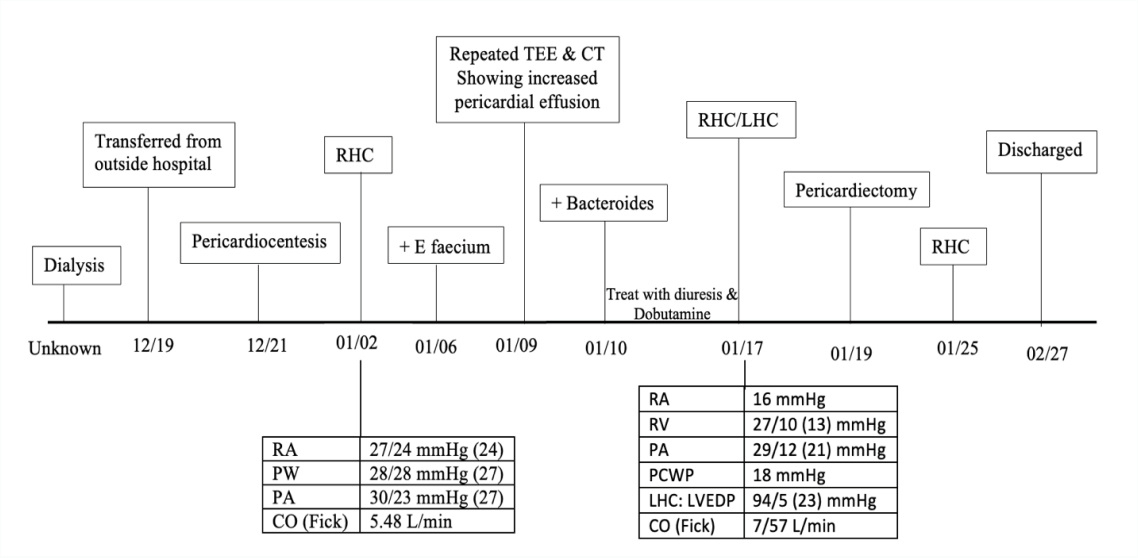

Figure 1: Chronological timeline of disease progression and hospitalization course.

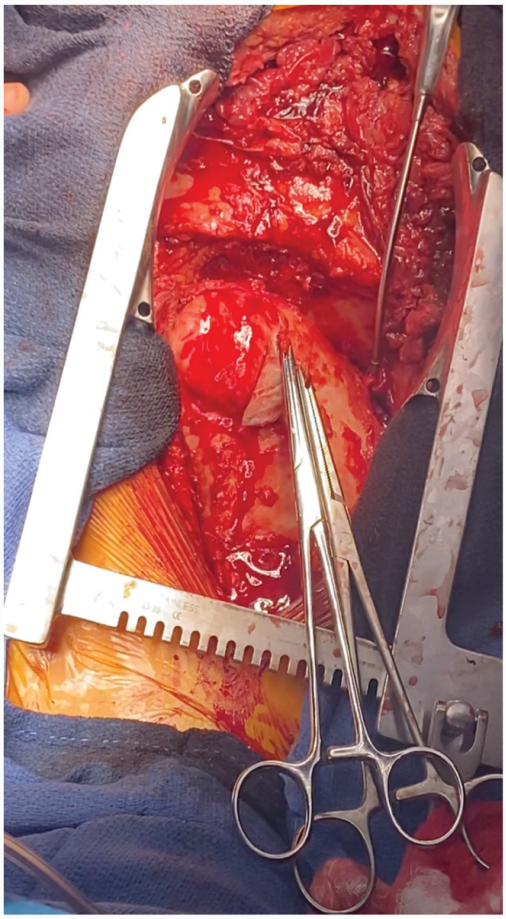

Figure 2: Partial resection of the visceral pericardium.

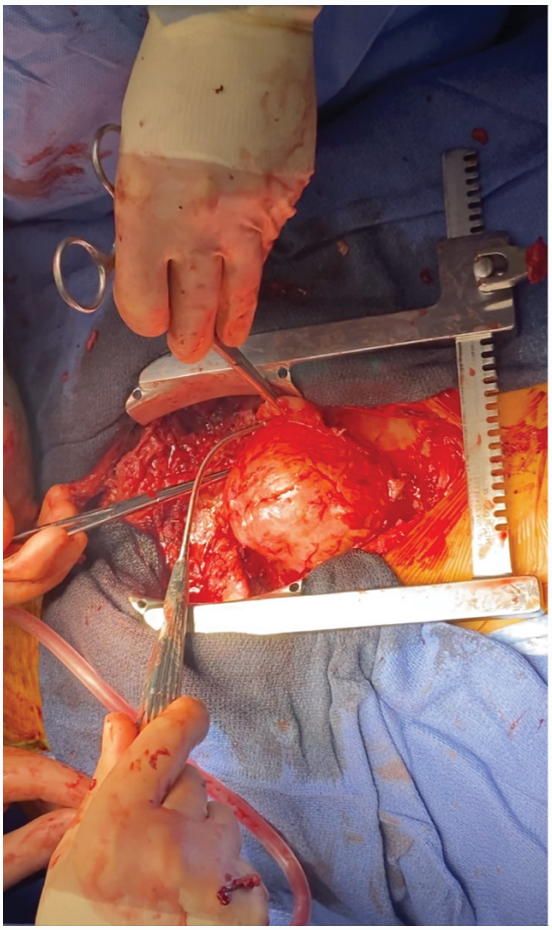

Figure 3: Complete resection of visceral pericardium.