A rare case of improved diastolic function following subxiphoid pericardiostomy for cardiac tamponade in a hypertensive patient with dressler's syndrome and non-tuberculous hemorrhagic effusive pericarditis

Un caso raro de mejoría de la función diastólica tras una pericardiostomía subxifoidea por taponamiento cardíaco en un paciente hipertenso con síndrome de Dressler y pericarditis hemorrágica efusiva no tuberculosa

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Abstract

This case report describes a 54-year-old hypertensive man with minor coronary artery disease (CAD; 30% stenosis in mid-LAD and RCA, managed medically with clopidogrel) and a recent history of non-tuberculous (Staphylococcus aureus) pneumonia who developed massive hemorrhagic effusive pericarditis (HEP) leading to cardiac tamponade. Hypertension, a major global cardiovascular risk factor, likely contributed to the underlying vascular and inflammatory milieu that predisposed the patient to this rare complication. Transthoracic echocardiography demonstrated a massive circumferential pericardial effusion (>20 mm diastolic separation) with tamponade physiology, including severely reduced left ventricular end-diastolic volume (LVEDV 52 mL, below the normal male reference range of 53–156 mL). Emergency subxiphoid pericardiostomy evacuated approximately 2000 mL of grossly hemorrhagic fluid, resulting in immediate hemodynamic stabilization. Post-procedure echocardiography confirmed complete effusion resolution and a significant 28.8% increase in LVEDV to 67 mL, documenting restoration of diastolic filling and normalization of previously impaired (restrictive-pattern) diastolic function. Pericardial fluid analysis revealed an exudative process (positive Rivalta test, protein 6.35 g/dL, LDH 1426 U/L, borderline ADA 26 U/L) with no bacterial growth on culture. Cytology and histopathology showed chronic inflammatory changes (dense lymphoplasmacytic infiltration) without malignancy. The patient improved clinically over an 11-day hospitalization and was discharged in stable condition. This rare presentation of cardiac tamponade secondary to HEP likely reflects overlapping mechanisms: autoimmune response consistent with Dressler's syndrome (post-injury pericarditis following minor CAD, occurring ~4 weeks post-ischemic insult) and pericardial involvement from recent non-tuberculous pneumonia, exacerbated by antiplatelet therapy and the hypertensive state. The case underscores the reversibility of tamponade-induced diastolic dysfunction with prompt surgical drainage and highlights the need for advanced imaging (cardiac CT and MRI) to clarify etiology, monitor for recurrence (including constrictive pericarditis), and prevent future morbidity.

Keywords: Hypertension, Cardiac Tamponade, Hemorrhagic Effusive Pericarditis, Dressler's Syndrome, Non-Tuberculous Pneumonia, Subxiphoid Pericardiostomy, Diastolic Dysfunction, Left Ventricular End-Diastolic Volume

Resumen

Este caso clínico describe a un hombre hipertenso de 54 años con enfermedad coronaria menor (EAC; estenosis del 30% en la arteria coronaria izquierda media y la arteria coronaria derecha, tratada médicamente con clopidogrel) y antecedentes recientes de neumonía no tuberculosa (Staphylococcus aureus), quien desarrolló una pericarditis hemorrágica efusiva masiva (PEH) que resultó en taponamiento cardíaco. La hipertensión, un importante factor de riesgo cardiovascular global, probablemente contribuyó al entorno vascular e inflamatorio subyacente que predispuso al paciente a esta rara complicación. La ecocardiografía transtorácica demostró un derrame pericárdico circunferencial masivo (separación diastólica >20 mm) con fisiología de taponamiento, incluyendo un volumen telediastólico del ventrículo izquierdo severamente reducido (VTDVI 52 mL, por debajo del rango de referencia normal masculino de 53-156 mL). La pericardiostomía subxifoidea de emergencia evacuó aproximadamente 2000 mL de líquido macrohemorrágico, resultando en una estabilización hemodinámica inmediata. La ecocardiografía posprocedimiento confirmó la resolución completa del derrame y un aumento significativo del 28,8% en el VTDVI a 67 mL, documentando la restauración del llenado diastólico y la normalización de la función diastólica previamente deteriorada (patrón restrictivo). El análisis del líquido pericárdico reveló un proceso exudativo (prueba de Rivalta positiva, proteína 6,35 g/dL, LDH 1426 U/L, ADA limítrofe 26 U/L) sin crecimiento bacteriano en el cultivo. La citología y la histopatología mostraron cambios inflamatorios crónicos (densa infiltración linfoplasmocitaria) sin malignidad. El paciente mejoró clínicamente tras 11 días de hospitalización y fue dado de alta en condición estable. Esta rara presentación de taponamiento cardíaco secundario a una EH probablemente refleja mecanismos superpuestos: una respuesta autoinmune compatible con el síndrome de Dressler (pericarditis postraumática tras una EAC leve, que se presentó aproximadamente 4 semanas después de la lesión isquémica) y afectación pericárdica por una neumonía no tuberculosa reciente, exacerbada por el tratamiento antiplaquetario y el estado hipertensivo. El caso subraya la reversibilidad de la disfunción diastólica inducida por taponamiento con drenaje quirúrgico inmediato y destaca la necesidad de imágenes avanzadas (TC cardíaca y RMN) para esclarecer la etiología, monitorizar la recurrencia (incluida la pericarditis constrictiva) y prevenir la morbilidad futura.

Palabras clave: Hipertensión, Taponamiento cardíaco, Pericarditis hemorrágica efusiva, Síndrome de Dressler, Neumonía no tuberculosa, Pericardiostomía subxifoidea, Disfunción diastólica, Volumen telediastólico del ventrículo izquierdo

Introduction

Hypertension is a significant global health burden and a major risk factor for cardiovascular diseases, including coronary artery disease (CAD) and its complications[1]. Among these complications, pericardial diseases, though less common, represent a critical intersection of inflammatory, ischemic, and hemodynamic pathologies[2]. Hemorrhagic Effusive Pericarditis (HEP) is an inflammatory disorder of the pericardium characterized by an effusion containing blood components[3,4]. While hemorrhagic pericardial effusion is most commonly associated with tuberculosis or malignancy, less frequent etiologies include non-tuberculous infections and autoimmune-mediated reactions, with reported prevalence rates of approximately 15–30% and 10–25%, respectively, in various series[5-7]. In the context of CAD, a specific form of HEP can occur known as Dressler's Syndrome (DS), a post-cardiac injury syndrome[8]. Furthermore, Non-Tuberculous Pneumonia (NTP) may also serve as a precursor to HEP, although this progression is rarely observed in immunocompetent individuals[5].

The pathophysiological mechanisms underlying HEP, particularly when secondary to DS and NTP, involve the extension of inflammatory processes to the pericardial space. In DS, myocardial injury during an ischemic event triggers an autoimmune-mediated inflammatory response that affects the pericardium[9]. This process may be significantly aggravated by concomitant pericardial inflammation associated with NTP, leading to increased pericardial fragility and susceptibility to bleeding. This risk is especially pronounced in patients receiving antiplatelet or anticoagulant therapy, commonly prescribed for CAD[10,11]. The temporal progression of HEP related to DS typically occurs within 2–6 weeks following the cardiac event, whereas the progression from NTP to HEP can vary widely, ranging from days to weeks or months[12,13].

The accumulation of pericardial fluid and the consequent increase in intrapericardial pressure impair cardiac expansion and filling during diastole. This leads to reduced ventricular preload, decreased stroke volume, and diminished cardiac output, culminating in the life-threatening condition of Cardiac Tamponade (CT)[5,14]. Another potential long-term sequela of HEP is Effusive-Constrictive Pericarditis (ECP), a condition characterized by both fluid accumulation and pericardial constriction[14].

Massive pericardial effusion, echocardiographically defined by an echo-free space exceeding 20 mm, poses a high risk for tamponade[15]. Clinical signs such as tachycardia, hypotension, pulsus paradoxus, and muffled heart sounds are key indicators necessitating immediate intervention[16-18]. While pericardiocentesis or surgical drainage, such as subxiphoid pericardiostomy, can be life-saving by relieving tamponade and improving diastolic filling, it is not a definitive treatment for the underlying etiology[19-20]. Comprehensive etiological evaluation is crucial, often requiring advanced imaging modalities like cardiac computed tomography (CT) or cardiac magnetic resonance imaging (MRI) to identify specific causes such as malignancy, aortic pathology, infection, or constrictive physiology, and to guide long-term management to prevent recurrence[21-23].

This report highlights a rare case of massive HEP leading to cardiac tamponade in a hypertensive patient with a background of minor CAD and non-tuberculous pneumonia, discussing the management and the resultant improvement in diastolic function following surgical intervention.

Materials and methods

1. Clinical and Laboratory Evaluation

A comprehensive diagnostic workup was initiated upon the patient's presentation. This included a detailed medical history, with specific focus on cardiovascular risk factors, recent infections, and medication use. A complete physical examination was performed, emphasizing vital signs, cardiopulmonary auscultation, and systematic evaluation for signs of cardiac tamponade, such as pulsus paradoxus, elevated jugular venous pressure, and muffled heart sounds. Standard laboratory investigations comprised a complete blood count, renal and liver function tests, serum electrolyte and albumin levels, and arterial blood gas analysis during episodes of acute respiratory distress.

2. Imaging and Diagnostic Procedures

The primary imaging modality was transthoracic echocardiography (TTE), performed using a standard ultrasound system. The examination followed established protocols to obtain parasternal, apical, and subcostal views. Key measurements included the quantification of pericardial effusion size, defined as the maximum diastolic echo-free space between the epicardium and pericardium. Left ventricular systolic function was assessed by calculating the ejection fraction (EF) using the biplane Simpson's method. Diastolic function and filling pressures were evaluated through Doppler measurements, including mitral inflow and tissue Doppler imaging. The left ventricular end-diastolic volume (LVEDV) was specifically calculated from the apical four-chamber and two-chamber views using the method of discs, as recommended by current guidelines for chamber quantification.

Serial posteroanterior chest radiographs were obtained throughout the hospitalization to monitor cardiac silhouette size and pulmonary parenchymal changes. Coronary angiography, performed prior to this admission, was reviewed to confirm the extent of underlying coronary artery disease.

3. Intervention: Surgical Technique

The definitive therapeutic procedure was an emergency subxiphoid pericardiostomy. Under general anesthesia, a vertical midline incision was made over the xiphoid process. The xiphoid was dissected and often partially excised to access the substernal plane. The pericardium was identified, incised under direct vision, and a sample was excised for histopathological analysis. Approximately 2000 mL of hemorrhagic fluid was evacuated. A 28-French chest tube was then inserted into the pericardial space and connected to an underwater seal drainage system to prevent reaccumulation and allow for continued output monitoring.

4. Pericardial Fluid and Tissue Analysis

The evacuated pericardial fluid was subjected to a standard analytical panel. This included gross description, biochemical analysis (protein concentration, lactate dehydrogenase [LDH], and adenosine deaminase [ADA] levels), and cytological examination for malignant cells. Microbiological studies included Gram stain and aerobic/anaerobic cultures. The pericardial tissue specimen was fixed in formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin for histopathological evaluation by a certified pathologist.

5. Data Collection and Analysis

Patient data, including demographic information, clinical findings, laboratory results, imaging parameters, surgical details, and outcomes, were collected prospectively during the hospitalization. Echocardiographic parameters pre- and post-intervention were compared descriptively to assess the hemodynamic impact of the procedure.

The patient was a 54-year-old male with a history of hypertension and minor coronary artery disease (30% stenosis in the mid-LAD and RCA), medically managed with clopidogrel. One month prior, he was treated for *Staphylococcus* pneumonia. He presented with a three-day history of dyspnea, nausea, and vomiting. Initial chest radiography confirmed cardiomegaly (Figure 1b). Upon acute deterioration, repeat radiography showed persistent cardiomegaly with a new, minimal left pleural effusion (Figure 1c). Preoperative transthoracic echocardiography was critical for diagnosis, revealing a massive circumferential pericardial effusion (>20 mm diastolic separation) with signs of tamponade physiology, including a severely reduced left ventricular end-diastolic volume (LVEDV).

Table 1: Echocardiographic Parameters Before and After Subxiphoid Pericardiostomy

Parameter	Pre-Procedure	Post-Procedure (Day 1)	Reference Range
LV Ejection Fraction (%)	73%	72%	52-72%
LV End-Diastolic Volume (mL)	52	67	53-156
Pericardial Effusion Size	Massive (>20 mm)	Resolved	None
Diastolic Function	Impaired (Restrictive pattern)	Normalized	Normal

The drained fluid was grossly hemorrhagic. Biochemical analysis confirmed an exudative process, as detailed in Table 2. Cytology showed chronic inflammatory cells with no malignancy. Pericardial tissue histology demonstrated fibrous tissue with dense lymphoplasmacytic infiltration, consistent with chronic pericarditis (Figure 2b).

Table 2: Pericardial Fluid Analysis

Analyte	Result	Interpretation
Appearance	Hemorrhagic	-
Rivalta Test	Positive	Exudative
Protein (g/dL)	6.35	High (Exudate)
LDH (U/L)	1426	Markedly Elevated
ADA (U/L)	26	Borderline Elevated
Culture	No growth	-
Cytology	Chronic inflammation; No malignant cells	Reactive process

The patient's hemodynamic status improved immediately following pericardiostomy. Follow-up chest radiography on the first postoperative day showed a significant reduction in cardiac silhouette and resolution of the pleural effusion (Figure 1d). Repeat echocardiography confirmed complete evacuation of the pericardial effusion (Figure 3) and a corresponding 28.8% increase in LVEDV from 52 mL to 67 mL, indicating restored diastolic filling.

Figure 1

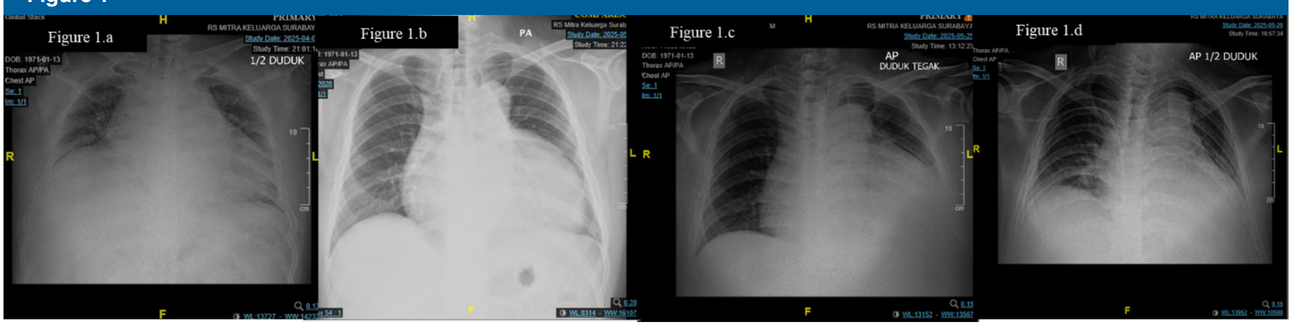
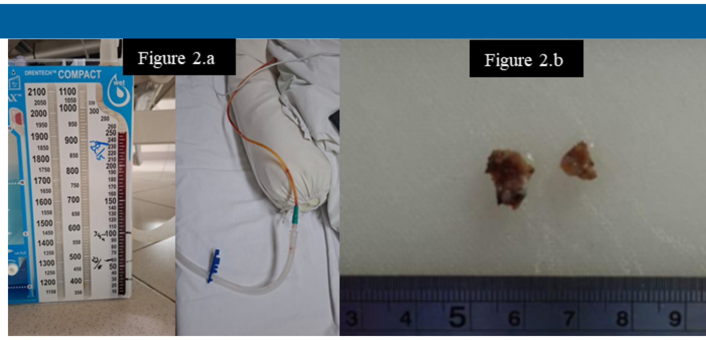


Figure 1. (a) Chest radiograph (CXR) obtained one month prior to hospitalization showing cardiomegaly and pneumonia.
(b) CXR on the first day of hospitalization demonstrating cardiomegaly.
(c) CXR on the second day of hospitalization during an episode of significant dyspnea.
(d) CXR after subxiphoid pericardiostomy and evacuation of pericardial fluid.

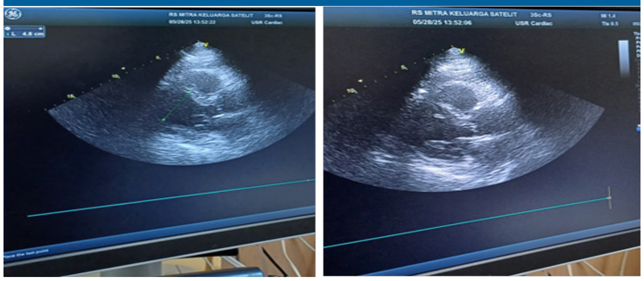
Figure 2

(a) Drainage catheter demonstrating hemorrhagic pericardial effusion



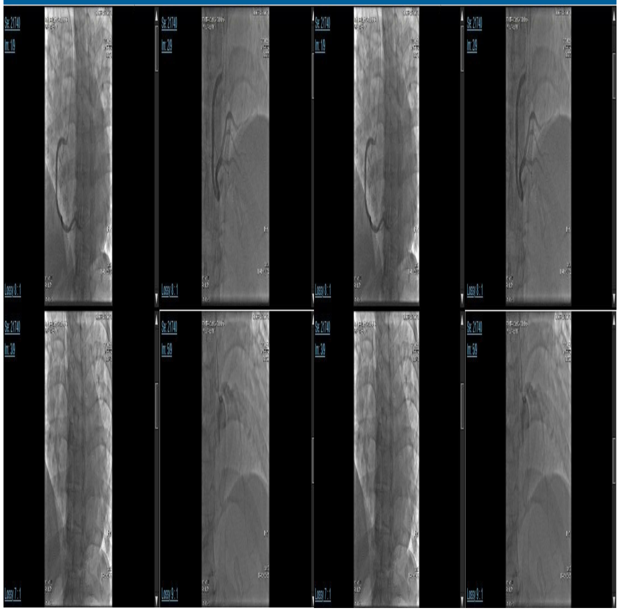
(b) Pericardial tissue specimen obtained during subxiphoid pericardiostomy

Figure 3. Echocardiographic evaluation one day after subxiphoid pericardiostomy, demonstrating complete resolution of pericardial effusion



The pericardial drain was removed on postoperative day three after output became negligible. The patient was discharged on day 11 in stable condition. Final histopathology and cytology results, received post-discharge, corroborated the diagnosis of chronic hemorrhagic effusive pericarditis.

Figure 4. Coronary angiography demonstrating minor Coronary Artery Disease (CAD), with 30% stenosis in the mid left anterior descending (LAD) artery and 30% stenosis in the right coronary artery (RCA)



Discussion

Hemorrhagic effusive pericarditis (HEP) is a relatively uncommon yet potentially life-threatening form of pericardial inflammation characterized by bloody effusion. It can arise from multiple etiologies, including infectious, neoplastic, autoimmune, and post-injury mechanisms. Although precise epidemiologic data for the hemorrhagic variant are limited, effusive pericarditis in general accounts for a significant proportion of pericardial disease cases in published series. Physiologically, normal pericardial fluid volume is typically small, with intrapericardial pressure maintained in a narrow range. Effusions are conventionally classified by onset, extent, volume, echocardiographic appearance, and fluid nature.

The causes of effusive pericarditis include both infectious and non-infectious factors—autoimmune diseases, malignancy, iatrogenic complications, trauma, uremia, and others—although a considerable proportion are labeled idiopathic. Larger effusions are significantly more likely to have an identifiable specific etiology. Among rarer triggers, Dressler's syndrome (post-myocardial infarction or post-cardiac injury pericarditis) classically develops weeks after myocardial damage, even when the underlying coronary artery disease is only mild. In the contemporary era of widespread reperfusion therapy, its incidence has declined sharply. Importantly, the degree of myocardial necrosis shows poor correlation with the development of Dressler's syndrome, allowing it to occur in patients with minimal CAD. The underlying process is thought to be autoimmune, driven by exposure of myocardial antigens following injury. Another infrequent but recognized etiology is bacterial infection of non-tuberculous origin, which may involve contiguous spread from adjacent pneumonia or hematogenous seeding.

In the present patient, an estimated large volume of hemorrhagic fluid accumulated, resulting in cardiac tamponade—a threshold complication that becomes highly probable above a certain volume and almost always necessitates urgent drainage when volumes become substantial. The development of such massive HEP here is best explained by the combined effects of recent non-tuberculous pneumonia, which likely increased pericardial tissue fragility, and concurrent antiplatelet therapy started earlier for minor CAD, substantially elevating the risk of hemorrhagic transformation within the inflamed pericardium.

Classic symptoms of HEP include fever and pleuritic-type retrosternal chest pain. When accumulation is gradual, the pericardium may initially accommodate the increasing volume with minimal clinical impact; symptoms often remain subtle or even absent until diastolic filling becomes severely restricted, at which point tamponade physiology rapidly supervenes. Our patient presented with episodic dyspnea without prominent pleuritic pain, culminating in an acute episode of severe dyspnea—clinical features fully consistent with tamponade, including tachycardia, pulsus paradoxus, distant heart sounds, and marked cardiomegaly on chest radiography.

Echocardiography is the gold-standard modality for diagnosing tamponade physiology. Key signs include diastolic collapse of the right atrium and/or right ventricle, abnormal septal “bounce,” swinging heart motion, and reduced ventricular end-diastolic volumes. The pre-procedure measurement in this case clearly demonstrated critical impairment of diastolic filling, reduced stroke volume, and consequent decrease in cardiac output.

Although ultrasound-guided pericardiocentesis is frequently the first-line approach for tamponade, surgical drainage—most commonly via subxiphoid pericardiostomy or creation of a pericardial window—is often preferred in cases of very large effusions, hemodynamic instability requiring rapid and complete decompression, or when simultaneous pericardial tissue biopsy is desired. Pericardiectomy is generally reserved for recurrent effusion or documented constrictive physiology. In non-tuberculous infectious effusions and certain malignant cases, subxiphoid pericardiostomy remains a widely accepted technique; comparative data suggest shorter operative and anesthesia times relative to video-assisted thoracoscopic approaches, with similar short-term morbidity and recurrence rates in many series—although the surgically created window carries a higher long-term risk of closure and reaccumulation compared with complete pericardiectomy.

In view of the patient’s acute life-threatening instability, extremely large effusion volume, need for both therapeutic evacuation and diagnostic sampling, and lack of preoperative evidence of constriction, emergency subxiphoid pericardiostomy was deemed the most appropriate intervention. The procedure produced immediate hemodynamic stabilization, complete resolution of the pericardial effusion on follow-up imaging, and a clinically significant increase in a key echocardiographic parameter, providing direct evidence of restored diastolic filling and reversal of the prior restrictive diastolic dysfunction pattern.

To comprehensively exclude alternative or coexisting causes of HEP and to evaluate the risk of delayed complications—particularly progression to effusive-constrictive or constrictive pericarditis—follow-up advanced imaging with cardiac computed tomography (CT) and cardiac magnetic resonance imaging (MRI) is strongly indicated.

Conclusions

This case highlights a rare and complex presentation of massive hemorrhagic effusive pericarditis leading to cardiac tamponade in a hypertensive patient with minor coronary artery disease and a recent episode of non-tuberculous (staphylococcal) pneumonia—most likely reflecting overlapping contributions from Dressler’s syndrome and infectious pericardial inflammation. The tamponade physiology, characterized by severely reduced left ventricular end-diastolic volume (LVEDV) and reversible diastolic dysfunction, was promptly and effectively reversed through emergency subxiphoid pericardiostomy, as demonstrated by complete effusion clearance and substantial LVEDV recovery on serial echocardiography. Thorough etiological investigation, including advanced cardiac imaging modalities such as cardiac CT and cardiac MRI, is essential during follow-up to identify any residual or undetected underlying pathology, to anticipate the possibility of late recurrence (including constrictive pericarditis), and ultimately to reduce the risk of readmission, long-term morbidity, and mortality in these unusual and high-risk clinical scenarios.

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Ethics Statement

Written informed consent was obtained from the patient and the patient’s family in accordance with the principles of medical ethics.

Conflict of Interest

The authors declare that there is no conflict of interest.

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