

Unveiling Tuberculous Constrictive Pericarditis: A Case of Ascites as the Telltale Sign

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Abstract

Introduction: Ascites, often associated with cirrhosis decompensation, can have diverse origins, including extra-hepatic causes. Constrictive Pericarditis (CP) represents a persistent inflammatory condition marked by enduring scarring, fibrosis, and calcification of the pericardium, resulting in impaired diastolic function and, ultimately, decreased cardiac output leading to heart failure. Despite idiopathic pericarditis being the primary cause of CP in the Western world, tuberculous pericarditis remains predominant in developing nations. Here, we present a case where ascites revealed tuberculous constrictive pericarditis.

Case presentation: A 40-year-old male presented with severe abdominal distension and lower limb edema. Diagnostic workup revealed moderate ascites and complete calcification of the pericardium. Imaging and laboratory findings supported the diagnosis of chronic constrictive pericarditis, likely of tuberculous origin. The patient showed clinical improvement with medical management and was scheduled for surgical intervention.

Conclusion: This case underscores the importance of considering constrictive pericarditis as a potential etiology for ascites, even in the absence of typical liver-related symptoms. Accurate diagnosis requires a comprehensive evaluation involving clinical, imaging, and laboratory assessments. Surgical intervention remains the mainstay of treatment, supplemented by medical management in select cases. Early recognition of cardiac involvement in ascites patients is vital for optimizing clinical outcomes.

Keywords: Constrictive pericarditis; Ascites; Cardiology; Morocco.

Introduction

Ascites, often associated with cirrhosis decompensation, can have diverse origins, including extra-hepatic causes. Constrictive Pericarditis (CP) represents a persistent inflammatory condition marked by enduring scarring, fibrosis, and calcification of the pericardium, resulting in impaired diastolic function and, ultimately, decreased cardiac output leading to heart failure. In the Western world, idiopathic pericarditis stands as the primary instigator of CP, trailed by CP induced by surgical interventions and radiotherapy. Conversely, tuberculous pericarditis continues to be the foremost cause of CP in developing nations and among immunocompromised individuals. We present a case where ascites unmasked tuberculous constrictive pericarditis [1].

Case presentation

A 40-year-old male patient presented to the emergency department with a six-month history of severe abdominal distension and edema of the lower limbs. He had a smoking history and a previous episode of lung abscess in 2022. On admission, he was afebrile with moderate ascites and abdominal collateral venous circulation. Lower limb edema was noted. Cardiac examination revealed no murmurs, and his blood pressure was symmetric at 120/83 mmHg. Pulmonary examination revealed decreased vesicular breath sounds. Laboratory findings were as follows: Hemoglobin: 13.1 g/dL; white blood cell count: 5370/mm³, lymphocyte count: 290/mm³, platelet count: 142000/mm³, Blood ionogram, plasma urea, creatinine, and hepatic function tests were normal. Abdominal ultrasound revealed moderate

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ascites. Ascitic fluid analysis showed an exudative, predominantly lymphocytic appearance. Chest x-ray and cine-radiography revealed complete calcification of pericardium (Figure 1).

Thoracic echocardiography demonstrated preserved left ventricular function, restrictive mitral profile (Figure 2), with annulus paradoxus and annulus reversus. Additionally, the right ventricle was non-dilated with longitudinal systolic dysfunction, pulmonary artery pressure was 58 mmHg, and the inferior vena cava was dilated and poorly compliant. The pericardium appeared thickened and calcified without effusion (Figure 3), with respiratory flow variations. Chest CT scan revealed interstitial damage sequelae along with bilateral calcified pachypleuritis and chronic calcified pericarditis, suggestive of previous tuberculosis infection. The diagnosis was chronic constrictive pericarditis, likely of tuberculous origin despite lacking bacteriological evidence. The patient was managed with a low-sodium diet and furosemide, resulting in clinical improvement. Surgical intervention was planned.

Discussion

Herein, we present a case of ascites revealing constrictive pericarditis. Portal hypertension due to liver cirrhosis represents the most common cause of ascites (85%) [2]. Additionally, 7% of ascites cases are attributable to peritoneal tumor origin, 3% to cardiac decompensation, and 5% to multiple causes [3].

Linking ascites to its cardiac origin can sometimes be challenging, underscoring the importance of comprehensive explorations. The lack of specificity in symptoms of constrictive pericarditis can lead to delayed diagnosis. Studies report ascites occurrence in 28% to 85% of cases [3].

Systematic biological analysis of ascitic fluid may unveil cardiac-related conditions. Protein concentration in ascites exceeds 25 g/L in the vast majority of cardiac ascites, compared to only 10% to 20% of cirrhotic ascites [4]. The serum-ascites albumin gradient reflects portal pressure well; a gradient exceeding 11 g/L favors cardiac ascites.

Abdominal ultrasound coupled with pulsed Doppler can reveal dilated suprahepatic veins or loss of triphasic Doppler signal, indicative of cardiac origin. Electrocardiography often reveals repolarization abnormalities [5].

Chest X-ray alone does not confirm diagnosis despite possible calcifications associated with constriction. Echocardiography combines supportive elements for diagnosing constriction. Computed tomography and magnetic resonance imaging have become invaluable for diagnosing constrictive pericarditis, allowing measurement of pericardial thickness. Dynamic catheterization exploration may show equalization of end-diastolic pressures between right and left sides and a dip-and-plateau pattern. While not pathognomonic, these findings can also occur in restrictive cardiomyopathy [6].

The surgical treatment for constrictive pericarditis is subtotal pericardiectomy, with medical treatment playing a limited role. Corticosteroids may be prescribed in cases of postoperative constrictive pericarditis or connective tissue disorders. Antitubercular treatment is prescribed only with strong epidemiological, clinical, or morphological suspicion [7].

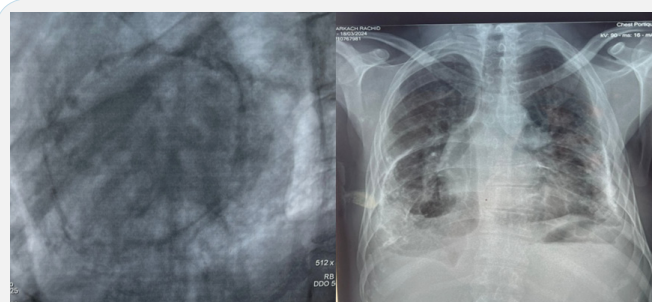


Figure 1: Chest x-ray and fluoroscopy showing significant pericardial calcification.

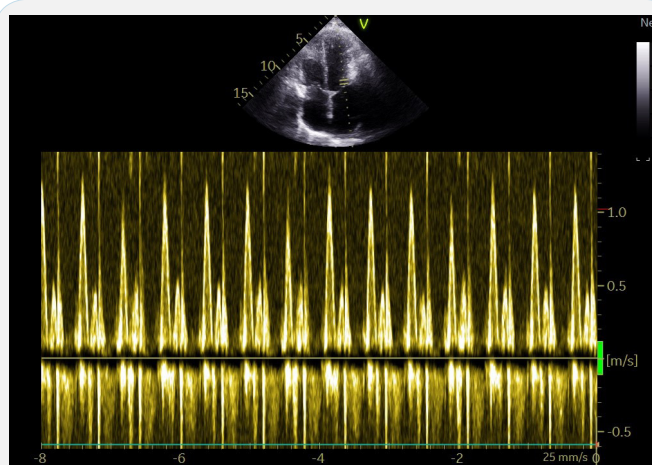


Figure 2: Restrictive profile with respiratory variation of the mitral flow.

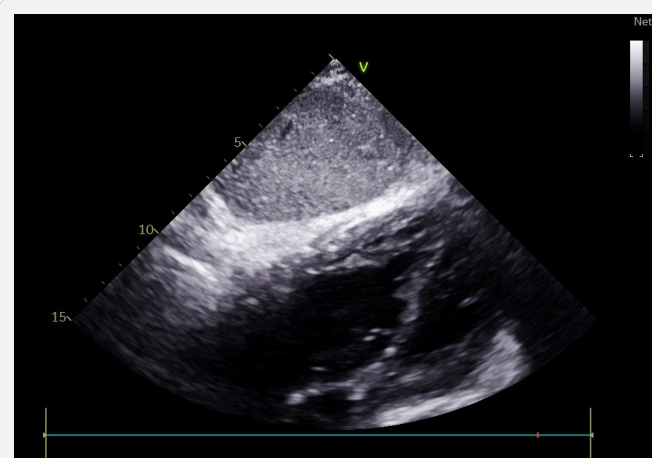


Figure 3: Thickening and calcification of the pericardium.

Conclusion

In summary, this case highlights the importance of recognizing constrictive pericarditis as a potential cause of ascites, even in the absence of typical liver-related symptoms. Accurate diagnosis relies on a thorough evaluation involving clinical, imaging, and laboratory assessments. Surgical intervention, particularly subtotal pericardiectomy, remains the mainstay of treatment, complemented by medical management in certain cases. Early identification of cardiac etiology in ascites patients is crucial for initiating appropriate interventions and improving clinical outcomes.

Declarations

Ethical approval: Written informed consent was obtained from the patient described in this article.

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References

1. Imazio M, Brucato A, Trincheri R, Adler Y. Diagnosis and management of pericardial diseases. *Nat Rev Cardiol.* 2009; 6: 743-51.
2. Farias AQ, Silvestre OM, Garcia-Tsao G, da Costa Seguro LFB, de Campos Mazo DF, Bacal F, et al. Serum B-type natriuretic peptide in the initial workup of patients with new onset ascites: a diagnostic accuracy study. *Hepatology.* 2014; 59) :1043-1051.
3. Patel YA, Muir AJ. Evaluation of new-onset ascites. *JAMA.* 2016; 316: 340-341.
4. Runyon BA. Care of patients with ascites. *N Engl J Med.* 1994; 330: 337-41.
5. Cacoub P, Wechsler B, Chapelon C, Gangjakhch I, Bletry O, Pilette JC, et al. Péricardites chroniques constrictives. 27 observations. *Presse Med.* 1991; 20: 2185-90.
6. Nataf P, Cacoub P, Dorent R, Jault F, Fontanel M, Regau M, et al. Péricardites chroniques constrictives. Etude rétrospective d'une série de 84 malades. *Arch Mal Cœur.* 1994; 87: 241-5.
7. Matshela MR. Constrictive pericarditis: prevention and treatment. *European Society of Cardiology.* 2017; 15: 24.