Peritoneal involvement in acute pericarditis with polyserositis may mimic acute cholecystitis: a case report

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ABSTRACT

Acute pericarditis is an inflammatory disease of the pericardium that can exclusively affect the pericardium or extend and affect other serosae, including the pleura and peritoneum. The involvement and dysregulation of the inflammasome, a protein complex responsible for the innate immune response, appear to be central in these forms of idiopathic pericarditis. This multidistrict interest leads to considering recurrent pericarditis also as a possible systemic disease. Here, we report the case of a 56-year-old male with a negative past medical history who presented with dyspnea, chest and abdominal pain, and a low-grade fever. Routine investigations and echocardiography were consistent with acute pericarditis; a chest X-ray revealed pleural effusion, and an abdominal ultrasound detected modest peritoneal effusion and cholecystitis. The symptoms completely regressed within 24 hours of initiating therapy with non-steroidal anti-inflammatory drugs and colchicine. The pericardial, pleural, and peritoneal effusions, along with cholecystitis, regressed in the next 4 weeks. This is the first reported case in which pericarditis with polyserositis also involved the gallbladder in the inflammatory process. It appears that standard therapy for pericarditis was effective in inducing remission of the extracardiac inflammatory processes, further supporting the hypothesis of an autoinflammatory etiology for the cholecystic inflammatory process as well. Early identification of pericarditis with systemic involvement implies shorter hospitalization times and improved therapeutic classification of the patient, thereby reducing the likelihood of corticosteroid-dependent pericarditis and significantly lowering the risk of relapse.

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Key words: pericarditis, polyserositis, autoinflammatory pathology, cholecystitis.

Contributions: all the authors made a substantial intellectual contribution, read and approved the final version of the manuscript, and agreed to be accountable for all aspects of the work.

Conflict of interest: ALB, received funding from Kiniksa Pharmaceuticals, Ltd. as an investigative site; unrestricted research grant from SOBI, KINIKSA, and ACARPIA; travel and accommodation for advisory committee from SOBI, Kiniksa, and Monterosa. The other authors declare no conflict of interest.

Ethics approval and consent to participate: not applicable.

Patient consent for publication: written informed consent from the patient to secure permission to publish his clinical history was obtained.

Availability of data and materials: all data underlying the findings are fully available

Funding: the authors declare that this research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Received: 26 January 2025.
Accepted: 28 January 2025.
Early view: 31 March 2025.

Licensee PAGEPress, Italy
Italian Journal of Medicine 2025; 19:1931
doi:10.4081/itjm.2025.1931

Introduction

Acute pericarditis is an inflammatory disease of the pericardium with various underlying etiologies.[1-3] Important progress has recently been made in understanding this pathology through the identification of the underlying pathogenetic mechanisms in idiopathic forms. The involvement and dysregulation of the inflammasome, a protein complex critical to the innate immune response, appears to play a central role in many cases of idiopathic pericarditis. This hypothesis is further supported by the efficacy of anti-interleukin 1 (IL-1) drugs in treating this condition.[4-6] Dysregulation of the inflammasome leads to the excessive production of IL-1, contributing to the inflammatory process.

In addition to great progress in understanding the pathogenetic mechanisms underlying pericarditis, recent studies have demonstrated that this condition can either be isolated, exclusively affecting the pericardium, or extend to involve other serosal membranes in the body, such as the

pleura and peritoneum.[7] This multi-systemic involvement raises several questions and has led to the consideration of recurrent pericarditis not merely as an autoinflammatory pathology of the heart but also as a potential systemic disease.[8,9] Recent reports indicate that the inflammatory, clinical, and laboratory characteristics of pericarditis with systemic involvement are more pronounced than those seen in cases with isolated pericardial involvement, closely resembling systemic autoinflammatory diseases.[7]

The systemic involvement of serosal membranes during pericarditis presents numerous diagnostic challenges and often contributes to delays in diagnosis due to its complex presentation.[7,10]

Although less common, cases where pericarditis extends beyond the pericardium and pleura to involve the peritoneum have been observed. These cases may present with diffuse abdominal pain that is difficult to interpret. In such patients, peritoneal effusion is frequently detected, while the abdominal symptoms remain nonspecific, ranging from mild discomfort to more pronounced pain.[6,11]

Case Report

We present the case of a 56-year-old male admitted to the emergency department with complaints of dyspnea, bilateral positional chest pain (worsening with inspiration and when lying down), and diffuse mild abdominal discomfort, predominantly in the right upper quadrant, accompanied by a low-grade fever. The symptoms had developed and progressively worsened over the preceding 3 days. The patient had a silent past medical history, was not on medication for any pathological condition, and had no family history of autoimmune or autoinflammatory diseases.

Upon initial examination, the patient was hemodynamically stable with blood pressure of 100/60 mmHg, oxygen saturation of 94% in room air, heart rate of 90 beats per minute, and body temperature of 37.3°C. Physical examination was notable for decreased vesicular breath sounds at the right lung base. An electrocardiogram was performed, revealing sinus tachycardia with normal atrioventricular and intraventricular conduction, as well as normal repolarization. Complete blood count revealed leukocytosis with neutrophilia (white blood count of 15,150/μL; neutrophils 11,500/μL); C-reactive protein (CRP) level was 321.5 mg/L, fibrinogen >7 g/L, and D-dimer 1320 mcg/L; transaminases were normal. Procalcitonin levels were within normal limits. A chest X-ray (Figure 1) demonstrated a right-sided pleural effusion with associated basal atelectasis.

A contrast-enhanced chest computed tomography scan confirmed the presence of a right pleural effusion but ruled out pulmonary embolism and aortic dissection.

On clinical suspicion of pleurisy, the patient was started on empirical therapy based on ceftriaxone 2 g intravenously every 24 hours, along with paracetamol as needed, but without any improvement in chest or abdominal pain. Subjective dyspnea also remained unchanged. For further investigation, he was admitted to the internal medicine department.

An abdominal ultrasound revealed typical signs of cholecystitis, including thickened gallbladder walls and intramural fluid infiltration, with a gallbladder wall thickness of 11 mm (Figure 2). No gallstones were detected, and the intrahepatic biliary ducts, common bile duct, and portal vein were of normal caliber. The liver appeared of normal size, with regular margins, homogeneous parenchyma, and regular echogenicity. A moderate abdominal effusion was also present. Echocardiography was performed, revealing a moderate circumferential pericardial effusion layer, not hemodynamically significant, with a maximum size of 13 mm (Figures 3 and 4).

The presence of pericardial effusion, positional chest pain together with elevated CRP levels led to the diagnosis of pericarditis with systemic involvement, manifesting as pleural and peritoneal effusion. Autoimmune screening and tuberculosis serology were performed, and both yielded negative results. The patient was promptly initiated on treatment based on indomethacin 100 mg daily in continuous intravenous infusion over 24 hours and oral colchicine 1 mg daily. Remarkably, symptoms resolved within 24 hours, with

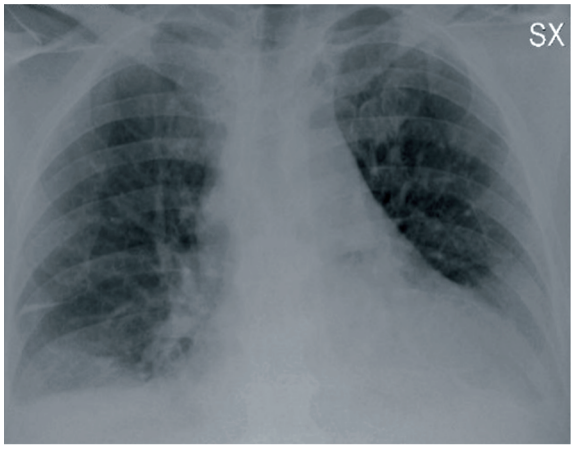

Figure 1. Chest X-ray shows a pattern of right unilateral pleural effusion together with a consensual area of right basal pulmonary atelectasis.

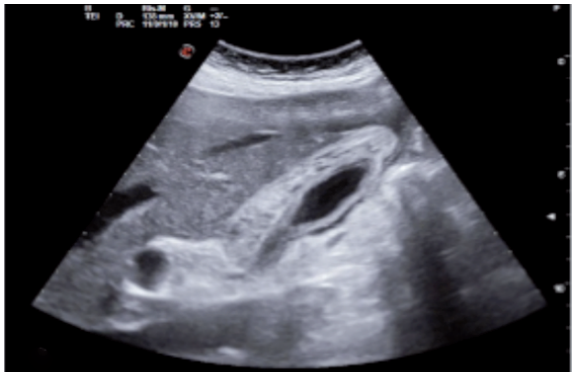

Figure 2. Abdomen ultrasound highlights a condition of cholecystitis with walls of increased thickness and intraparietal fluid on the gallbladder with a diameter of 11 mm, no stones, normal intrahepatic biliary ducts, common bile duct of normal caliber and regular course, and portal vein of normal caliber.

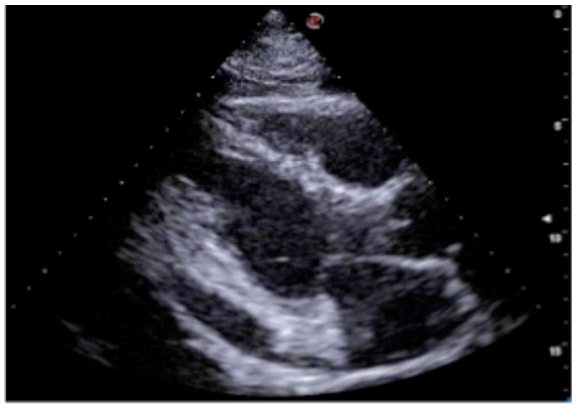

Figure 3. Parasternal long axis echocardiographic view showing pericardial effusion, predominantly localized along the inferolateral left ventricular wall and extending to the region surrounding the right ventricle.

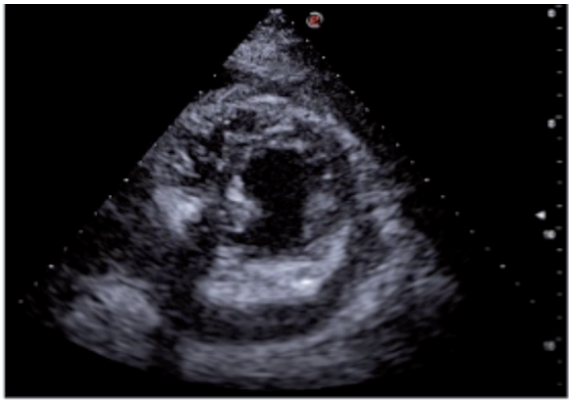

Figure 4. Parasternal short axis echocardiographic view showing circumferential pericardial effusion; maximal telediastolic size 13 mm.

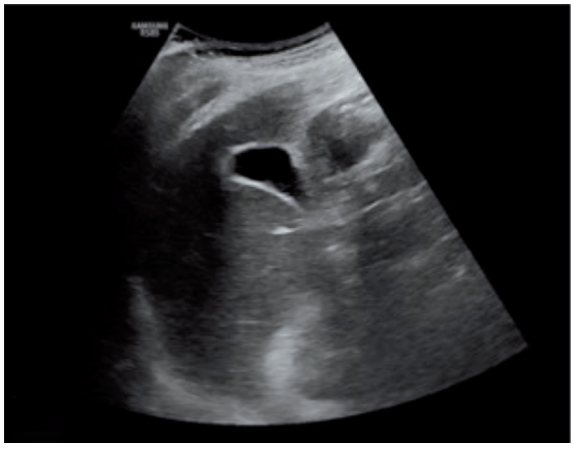

Figure 5. The second abdomen ultrasound examination showed that the peritoneal effusion layer had disappeared.

dyspnea and abdominal pain subsiding completely. CRP levels normalized in 1 week, with a gradual reduction observed. The patient was discharged after 8 days on a regimen of indomethacin 50 mg every 8 hours and colchicine 1 mg daily.

At the follow-up visit 7 days post-discharge, the patient reported complete resolution of the symptoms. During this visit, the patient underwent an echocardiographic examination, which revealed the permanence of the pericardial effusion (11 mm maximum) and reduced pleural effusion, while the abdomen ultrasound examination showed that peritoneal effusion had disappeared (Figure 5). The parietal thickness of the gallbladder also appeared to decrease. A gradual tapering of indomethacin was planned, contingent on normal monthly CRP levels. Pleural and pericardial effusions were solved in the next 4 weeks. At the 8th month of follow-up, the patient remained free of pericarditis recurrences, and both the pericardial, pleural, and abdominal effusions, as well as the gallbladder wall thickening, had resolved. CRP levels remained within normal ranges throughout, even with the progressive tapering of indomethacin therapy.

Discussion

Acute and recurrent pericarditis often presents with extracardiac involvement, particularly pleural effusion (44-52%) and also peritoneal effusion (13%) (polyserositis).[6,7,10-12]

Polyserositis, in general, can arise from various etiologies, including neoplastic, infectious, autoimmune, or autoinflammatory conditions; however, in many cases, it remains idiopathic.[13]

The symptomatology, radiological findings, and laboratory results in patients with polyserositis pose significant challenges in differential diagnosis, frequently leading to diagnostic delays and suboptimal initial therapy.[13] In cases where patients present with chest pain and pleural effusion prior to the manifestation of pericardial effusion, the diagnosis of an autoinflammatory process is rarely considered, even when CRP is elevated, and neutrophilia is present alongside negative procalcitonin levels. Diagnostic uncertainty increases further when peritoneal involvement is detected. It is only upon the manifestation of pericardial effusion that a diagnosis can be definitively made according to the European Society of Cardiology criteria.

The recognition of idiopathic pericarditis as a disease often caused by autoinflammatory mechanisms leads to the consideration of the polyserositis found during its course as an epiphenomenon of systemic autoinflammatory processes. This perspective supports the notion that pericarditis may represent not merely an organ-specific disease but a manifestation of a broader autoinflammatory pathology affecting the pericardium and other serosal membranes.[7,14]

Conclusions

In the presented case, the autoinflammatory process appeared to involve not only the pericardium but also extended to the pleura and peritoneum, resulting in a complex symptomatology characterized by typical chest pain, dyspnea, and abdominal pain, mimicking acute

cholecystitis. Accordingly, the patient demonstrated the involvement of the gallbladder wall, which responded to the same non-steroidal anti-inflammatory drug and colchicine therapy that effectively managed the pericardial, pleuritic, and peritoneal manifestations.

This case represents the first documented instance of pericarditis with polyserositis also involving the gallbladder wall as part of the inflammatory process. We report it because we previously observed other similar cases in our pericarditis clinic, and we think that the medical community may take advantage of this atypical presentation. The successful resolution of both cardiac and extracardiac inflammation with standard pericarditis therapy supports the hypothesis that the cholecystic inflammation in this patient may also have an autoinflammatory origin.

Elevated CRP values accompanied by neutrophilia and normal procalcitonin values should lead to the consideration of an underlying autoinflammatory process. Furthermore, elevated D-dimer, as recently demonstrated, could also reflect the inflammatory process itself.[6,15]

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