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Case Report

Massive Inflammatory Pericardial Effusion in Spondyloarthritis: Noninvasive Management Success

Mochamad Yusuf Alsagaff[1*], MD; Ken Christian Kawilarang[1], MD; Hendri Susilo[1], MD; Nadya Luthfah[1], MD; Cahyo Wibisono Nugroho[2], MD

ABSTRACT

Spondyloarthritis is a chronic systemic inflammatory disease that primarily affects the spine, sacroiliac joints, and tendons. Cardiac involvement is uncommon but may manifest as pericardial effusion, posing diagnostic and therapeutic challenges. This case underscores the significance of recognizing systemic inflammatory diseases as potential causes of pericardial effusion and the role of conservative management in avoiding unnecessary invasive interventions.

A 24-year-old man presented with acute dyspnea, orthopnea, and pleuritic chest pain. Clinical evaluation and imaging revealed a massive pericardial effusion without echocardiographic signs of cardiac tamponade. Laboratory workup demonstrated elevated inflammatory markers, leading to a differential diagnosis that included systemic inflammatory diseases. The patient was initially managed with nonsteroidal anti-inflammatory drugs (NSAIDs) and colchicine. Nonetheless, persistent pericardial effusion prompted further evaluation, ultimately leading to a diagnosis of spondyloarthritis based on radiographic evidence of sacroiliitis and positive rheumatoid factor. A regimen of corticosteroids and sulfasalazine resulted in complete resolution of pericardial effusion and normalization of inflammatory markers.

Inflammatory pericardial effusion can be the initial manifestation of spondyloarthritis, and targeted medical therapy may obviate the need for invasive intervention. Clinicians should consider systemic inflammatory diseases in unexplained pericardial effusion and prioritize individualized treatment approaches. ***(Iranian Heart Journal 2026; 27(1): 93-100)***

KEYWORDS: pericardial effusion; spondyloarthritis; pericardiocentesis; systemic inflammatory disease; conservative management

[1] Department of Cardiology and Vascular Medicine, Dr Soetomo General Academic Hospital, Surabaya, East Java, Indonesia.
[2] Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, East Java, Indonesia.

*** Corresponding Author:** Mochamad Yusuf Alsagaff, MD; Department of Cardiology and Vascular Medicine, Dr Soetomo General Academic Hospital, Surabaya, East Java, Indonesia.
Email: yusuf_505@fk.unair.ac.id **Tel:** +62 8123543388

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Spondyloarthritis encompasses systemic inflammatory diseases that primarily affect the spine and sacroiliac joints and can also involve extra-articular sites, including the heart. The prevalence of cardiac manifestations among patients with spondyloarthritis has been documented at 13.6%.[1] This finding has prompted clinical consideration in managing massive pericardial effusion due to systemic inflammation.

The present case report describes a young patient with massive pericardial effusion secondary to spondyloarthritis.

Case Presentation

A 24-year-old man presented to the emergency department with progressively worsening shortness of breath and orthopnea over the past several days. He reported a sharp, pleuritic chest pain localized to the left side of the chest 10 days before admission. The pain worsened with deep inspiration but gradually subsided. He denied fever, cough, weight loss, or any significant medical history, and he was not taking any medications.

On physical examination, the patient's temperature was 37°C, blood pressure was stable, and he was tachycardic. Jugular venous distension was noted, and his heart sounds were muffled. There were no murmurs, rubs, or adventitious lung sounds on auscultation. The rest of the physical examination, including abdominal and extremity examinations, was unremarkable.

Initial laboratory investigations revealed leukocytosis with a white blood cell count of 15.3×10^9/L, thrombocytosis with a platelet count of 689×10^9/L, and an elevated erythrocyte sedimentation rate of 120 mm/h. Electrocardiography (ECG) showed low-voltage QRS complexes without electrical alternans (Figure 1). Chest radiography demonstrated cardiomegaly with a cardiothoracic ratio of 0.65 (Figure 2). Transthoracic echocardiography revealed a large pericardial effusion, measuring up to 4 mm, with fibrinous deposits within the pericardial sac (Figure 3). Nevertheless, there were no echocardiographic signs of cardiac tamponade, such as right atrial or right ventricular collapse, nor was there any significant respiratory variation in transvalvular flow.

The patient opted against hospitalization for further treatment and chose to be discharged upon his own request. He was prescribed a regimen consisting of ibuprofen (800 mg/3 times daily), colchicine (0.5 mg twice daily), and bisoprolol (2.5 mg/d) for rate control. He was advised to return for follow-up in 2 weeks to reassess the pericardial effusion and repeat inflammatory markers.

Two weeks later, the patient returned for follow-up. He reported that his symptoms had substantially improved, with no recurrence of dyspnea or orthopnea. He did experience occasional, brief episodes of chest discomfort lasting seconds. Given the patient's geographic location in Indonesia, where tuberculosis is endemic, tuberculous pericarditis was considered in the differential diagnosis. An interferon-γ release assay was ordered to rule out tuberculosis as the cause of the pericardial effusion. Repeat laboratory testing showed improvement in inflammatory markers, with the erythrocyte sedimentation rate decreasing to 63 mm/h and C-reactive protein measured at 3.67 mg/L. Thyroid function tests were within normal limits, and the interferon-γ release assay was negative. Despite these improvements, repeat echocardiography revealed no significant reduction in the size of the pericardial effusion, and the fibrinous deposits within the effusion had increased.

The patient also mentioned that over the past year, he had been experiencing brief episodes of facial muscle weakness, which he described as consistent with Bell palsy. Additionally, he reported adult-onset asthma symptoms. Given these new findings, further workup was warranted to explore possible systemic inflammatory etiologies. The rheumatology team was consulted due to the incomplete response to therapy and suspicion of an underlying systemic inflammatory condition. Rheumatoid factor testing returned positive at 128 IU/mL, and chest radiography of the sacroiliac joints showed bilateral sacroiliitis, with early syndesmophytes and bone erosions, leading to a diagnosis of spondyloarthritis (Figure 4).

The patient's treatment was escalated to include prednisone, 30 (mg/d), in addition to

ongoing colchicine and ibuprofen. Two weeks later, sulfasalazine was added at 500 mg twice daily, which was subsequently increased to 1000 mg twice daily as his condition stabilized. One month after starting prednisone and 2 weeks after initiating sulfasalazine, the patient returned for follow-up. He reported no symptoms, and repeat echocardiography showed complete resolution of the pericardial effusion (Figure 5). The patient's inflammatory markers had normalized, with erythrocyte sedimentation rate of 25 mm/h and C-reactive protein concentration less than 0.6 mg/L. Because of suspicion of a systemic inflammatory disorder, additional testing was performed. The human leukocyte antigen B27 test was negative, which suggested a more favorable prognosis. The patient was advised to continue follow-up with the rheumatology team to monitor for recurrence of symptoms or inflammation and for management of the underlying spondyloarthritis.

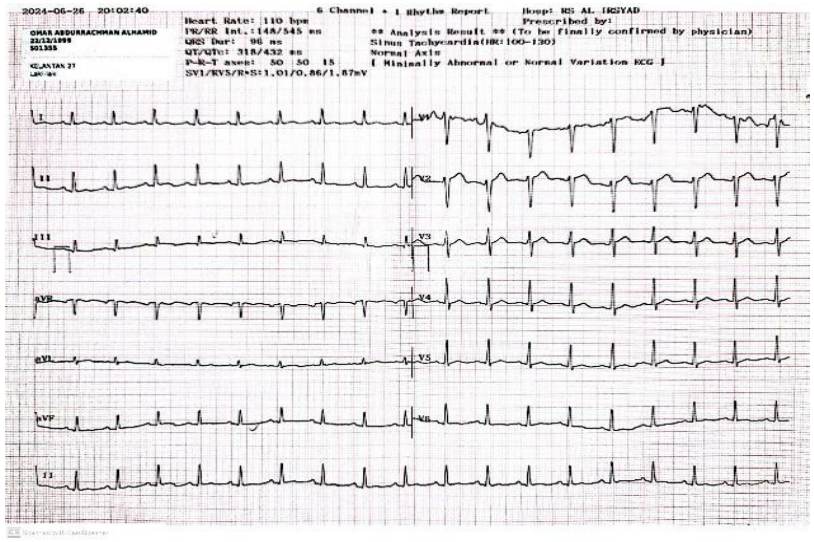

Figure 1. The electrocardiogram revealing low-voltage QRS complexes with no electrical alternans

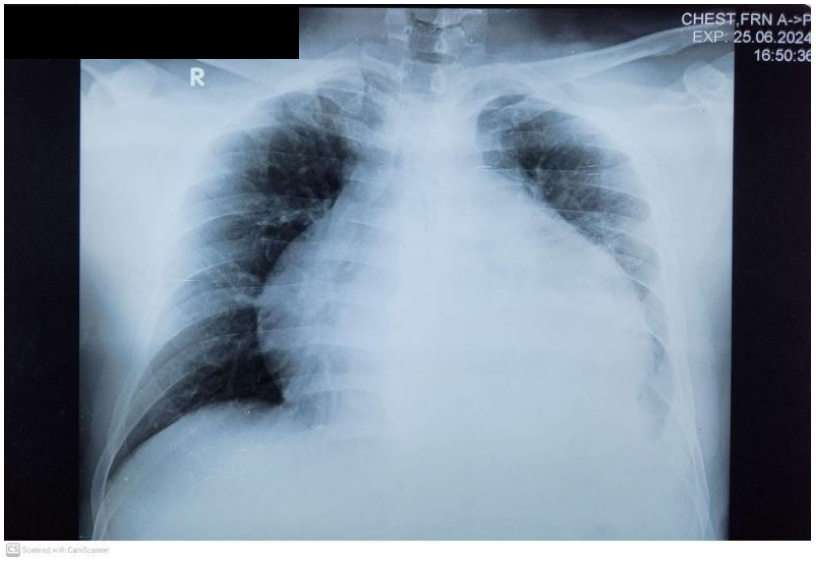

Figure 2. Chest radiogram demonstrating cardiomegaly with a cardiothoracic ratio (CTR) of 65%

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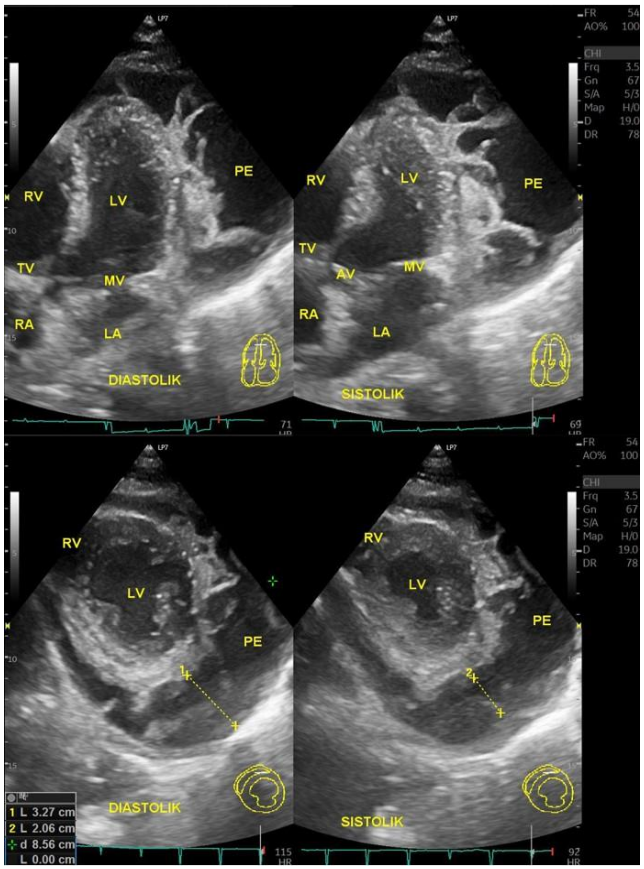

Figure 3. Transthoracic echocardiography revealing a large pericardial effusion, measuring up to 4 mm, with fibrinous deposits within the pericardial sac

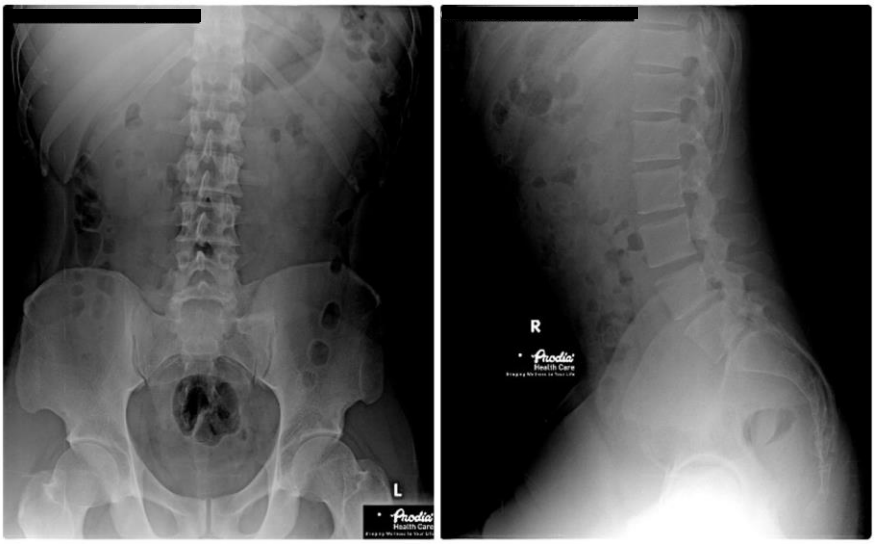

Figure 4. Chest radiograms of the sacroiliac joints showing bilateral sacroiliitis, with early syndesmophytes and bone erosions, leading to a diagnosis of spondylosis arthritis

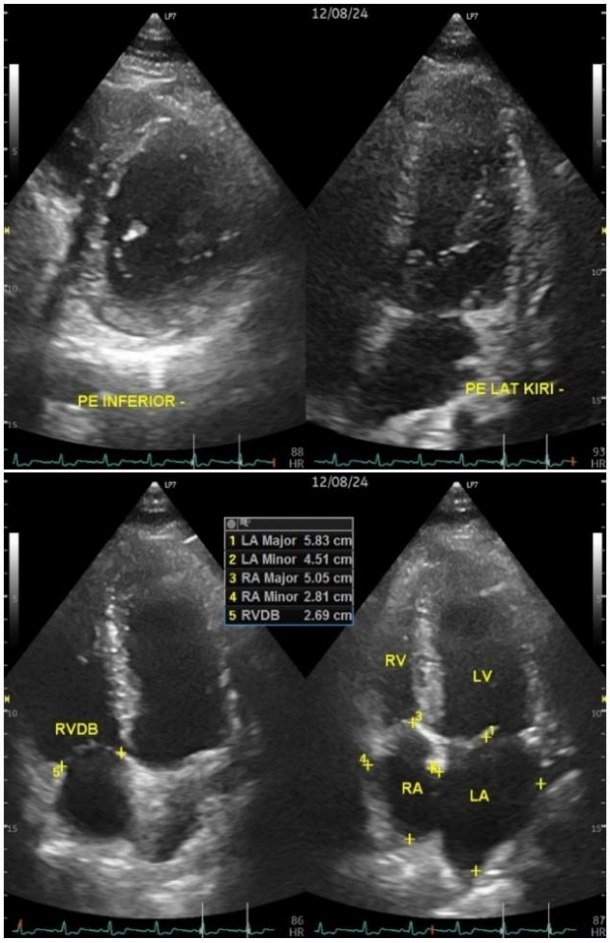

Figure 5. Evaluation of echocardiography showing complete resolution of the pericardial effusion following the administration of prednisone and sulfasalazine

DISCUSSION

Large pericardial effusions often present clinicians with a critical decision: whether to pursue an invasive approach with pericardiocentesis or to opt for conservative management. Pericardiocentesis is typically indicated when patients exhibit signs of cardiac tamponade or hemodynamic compromise.[2] Still, when these signs are absent, as in this case, a more measured approach is not only reasonable but potentially preferable.

The decision to defer pericardiocentesis in this case represents a deliberate departure from the conventional approach often employed in the management of fibrinous pericardial effusion. Traditionally, pericardiocentesis is performed preemptively due to the concern that fibrin deposition may progress to organized effusion, potentially culminating in constrictive pericarditis. However, emerging evidence suggests that conservative medical management may be effective in halting this progression by addressing the underlying inflammatory process.[2]

In this case, we chose a noninvasive approach supported by the absence of clinical signs of tamponade (hypotension, muffled heart sounds, or jugular venous distension), favoring targeted anti-inflammatory therapy over immediate intervention. This strategy allowed resolution of inflammation and effusion without exposing the patient to the risks of pericardiocentesis, including infection, pneumothorax, and iatrogenic cardiac injury.[4]

The development of adult-onset asthma in this patient raised suspicion of an underlying systemic inflammatory condition, as systemic inflammation is a well-established contributor to both asthma and autoimmune diseases. Proinflammatory cytokines, particularly interleukin 6, play a central role in these conditions, and elevated interleukin 6 levels have been associated with worse lung function in patients with comorbid systemic rheumatic diseases, highlighting the interplay between inflammation and asthma onset.[5] This connection guided the investigation toward identifying a systemic inflammatory etiology for the patient's pericardial effusion. Common causes such as tuberculosis and systemic lupus erythematosus were systematically ruled out with negative interferon-γ release assay and antinuclear antibody tests, while normal thyroid function tests excluded thyroid-related inflammation. Although an elevated rheumatoid factor was detected, its nonspecific nature and potential for false positives necessitated further diagnostic evaluation. The imaging finding of sacroiliitis, combined with the elevated rheumatoid factor, pointed to spondyloarthritis as the underlying diagnosis, unifying the patient's asthma, systemic inflammation, and pericardial effusion under a single autoimmune process.

Spondyloarthritis is primarily known for its musculoskeletal involvement, but its extra-articular manifestations, particularly cardiac complications, are equally important to recognize.[7,8] The spectrum of spondyloarthritis encompasses conditions such as ankylosing spondylitis, psoriatic arthritis, and reactive arthritis and is characterized by inflammatory joint and spine involvement, with axial spondyloarthritis being a key subtype.[7] In axial spondyloarthritis, inflammation in the axial skeleton typically presents as sacroiliitis or spondylitis and can progress to osteodestructive changes and new bone formation such as syndesmophytes.[9] These processes contribute to spinal rigidity and, in severe cases, complete ankylosis. Nonetheless, the systemic nature of spondyloarthritis means that other organ systems, including the eyes, lungs, and heart, may also be affected. This case demonstrates that pericarditis and pericardial effusion may occur as cardiac complications of spondyloarthritis. Pericardial involvement in inflammatory conditions is less commonly recognized but may result in clinically important consequences if untreated. Early recognition permits the timely initiation of anti-inflammatory therapy and may reduce the need for invasive interventions. Once spondyloarthritis was identified as the underlying cause of the pericardial effusion, the treatment plan was adjusted to include sulfasalazine and prednisone, which are commonly used to manage systemic inflammation in inflammatory arthritis. Sulfasalazine was selected for its efficacy in controlling chronic inflammation, particularly in axial spondyloarthritis, in which it has been shown to reduce symptoms in early-stage disease or in younger patients.[10,11] Prednisone was introduced to provide immediate relief by rapidly reducing inflammatory markers, thereby addressing both the pericardial effusion and the systemic disease.[12] This therapeutic approach reflects the importance of targeting the root cause of the effusion rather than focusing solely on symptomatic relief. By addressing the underlying inflammation, the medical team managed the cardiac complications while avoiding unnecessary invasive interventions such as pericardiocentesis, which could have introduced additional risks.

The choice of sulfasalazine (tumor necrosis factor α inhibitors) is particularly notable, as it offers a relatively favorable adverse effect profile compared with other disease-modifying antirheumatic drugs (DMARDs), such as methotrexate. Recent guidelines from the Assessment of Spondyloarthritis International Society and the European League Against Rheumatism now

recommend the use of biologic agents, particularly tumor necrosis factor α inhibitors, as first-line therapies in axial spondyloarthritis.[11] The patient's dosage of sulfasalazine was initially set at 500 mg twice daily, with gradual titration to 1000 mg twice daily, and the potential for further increases depending on therapeutic response over a 3- to 6-month period.

CONCLUSIONS

This case provides several key lessons for clinical practice, particularly in managing pericardial effusions in stable patients. First, it reinforces the importance of individualized treatment approaches-tailoring the management plan based on the patient's overall clinical picture rather than rushing into invasive procedures. Second, it highlights the value of a thorough diagnostic workup, including a focus on systemic causes when common etiologies are ruled out. Third, the case emphasizes the value of collaborative, multidisciplinary care in complex cases involving systemic inflammatory diseases. Finally, it serves as a reminder that conservative management, when appropriate, can lead to excellent outcomes, sparing patients the risks associated with invasive interventions. The case encourages clinicians to adopt a cautious, patient-centered approach when managing similar cases.

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Patient Consent
Written informed consent was obtained from the patient for the publication of this case report.

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