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Presentation of Pericardial Effusion in an 18-Year- Old Female with Systemic Lupus Erythematosus

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ABSTRACT

Background: Pericardial effusion is a common manifestation of Systemic Lupus Erythematosus (SLE), a chronic autoimmune disease affecting young women. This rare symptom requires high suspicion and prompt diagnosis for appropriate treatment, emphasizing the importance of early detection and management. **Case Description:** A patient with pericardial effusion presented to the emergency department with symptoms such as weakness, mouth ulcers, and nausea. Despite no known medical conditions, a chest X-ray revealed cardiomegaly, suggesting an underlying cardiovascular condition. An echocardiographic evaluation revealed normal diastolic function and right ventricular contractility, but a moderate to severe pericardial effusion was identified. The patient was diagnosed with massive complex pericardial effusion, hypoalbuminemia, inflammatory anemia, and SLE. A multidisciplinary approach was implemented, including intravenous fluid therapy, corticosteroid therapy, gastroprotective therapy, anti-inflammatory therapy, and Video-assisted thoracoscopic surgery (VATS) pericardiectomy. **Discussion:** Pericardial effusion, a cardiac manifestation in SLE patients, is often caused by autoimmune-mediated inflammation of the pericardium. This inflammatory process, driven by autoantibodies and immune complexes, increases vascular permeability and facilitates fluid exudation, potentially leading to cardiac tamponade, requiring a multidisciplinary approach. **Conclusion:** Pericardial effusion in young SLE patients requires early recognition and multidisciplinary management, including VATS pericardiectomy. Treatment includes corticosteroids, immunosuppressive agents, and targeted antibiotics. Serial echocardiographic assessments and ongoing immunosuppressive therapy are crucial for long-term disease management.

Keyword: pericardial effusion, SLE, VATS, autoimmune

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by multisystem inflammation, predominantly affecting young women.¹ Cardiac involvement in SLE is common, with pericardial effusion being one of the frequent manifestations.² While pericardial effusion occurs in a significant number of SLE patients, it is often mild and asymptomatic; however, in rare instances, it can progress to a massive effusion leading to cardiac tamponade, a life-threatening condition requiring immediate intervention.^{3,4}

The clinical presentation of pericardial effusion in SLE varies, ranging from incidental findings to severe cases presenting with hemodynamic compromise.⁵ Notably, pericardial effusion as the initial manifestation of SLE is uncommon, posing diagnostic challenges.⁶ In young female patients, such presentations necessitate a high index of suspicion for SLE, especially when accompanied by other systemic symptoms. Early recognition and diagnosis are crucial for initiating appropriate immunosuppressive therapy to prevent further complications.^{7,8}

This case report discusses an 18-year-old female who presented with significant pericardial effusion as the initial manifestation of SLE. Her clinical course underscores the importance of considering autoimmune etiologies in young patients presenting with unexplained pericardial effusion. Through comprehensive evaluation, including imaging and serological testing, a prompt diagnosis was established, leading to effective management. This case highlights the need for awareness among clinicians regarding such presentations to facilitate timely diagnosis and treatment.

CASE REPORT

On October 18, 2024, an 18-year-old female patient, identified by the initials AN, presented to the emergency department. The initial primary survey revealed no abnormalities, and the secondary survey findings were within normal limits. Her hemodynamic status was stable, with a blood pressure of 120/80 mmHg, a heart rate of 78 beats per minute, a respiratory rate of 20 breaths per minute, and an oxygen saturation of 99%. Additionally, her body temperature was recorded at 36°C, indicating no signs of fever or hypothermia.

During the initial anamnesis, the patient reported experiencing generalized weakness that had persisted for three days. She also complained of painful mouth ulcers, which made eating difficult and led to a reduced appetite over the same period. Additional symptoms included dry lips and nausea, while she denied experiencing vomiting. The patient also reported mild abdominal pain, but her bowel movements and urination remained within normal limits.

Regarding her past medical history, there was no known history of diabetes mellitus or hypertension. Furthermore, the patient had no known allergies or previous significant medical conditions.

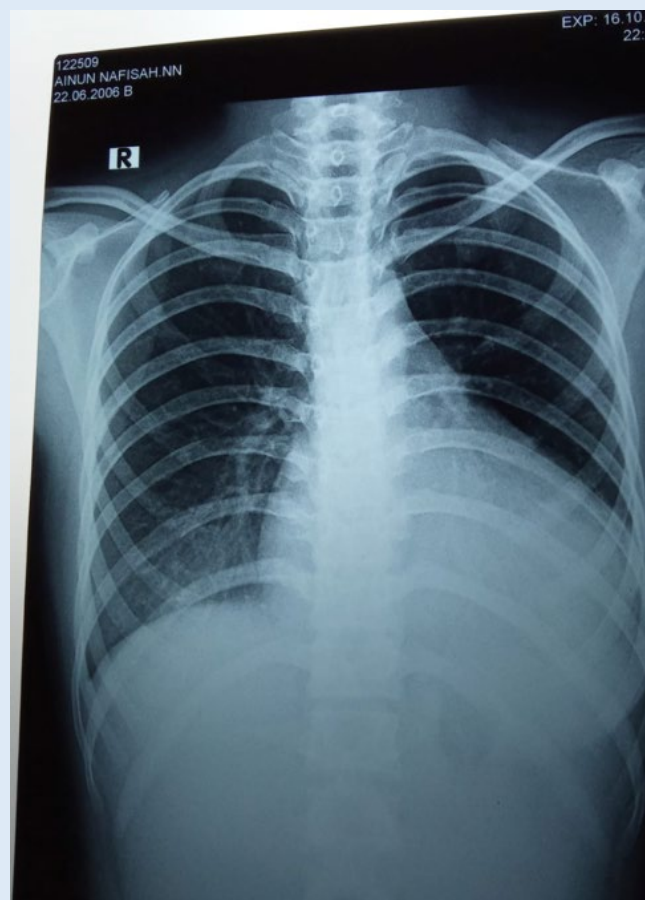


Figure 1. Chest X-Ray

A posteroanterior (PA) chest X-ray performed on October 16, 2024, showed no radiographic evidence of pneumonia or any ongoing infectious process. However, the imaging revealed cardiomegaly, indicating an abnormal enlargement of the heart, which may suggest an underlying cardiovascular condition requiring further evaluation.

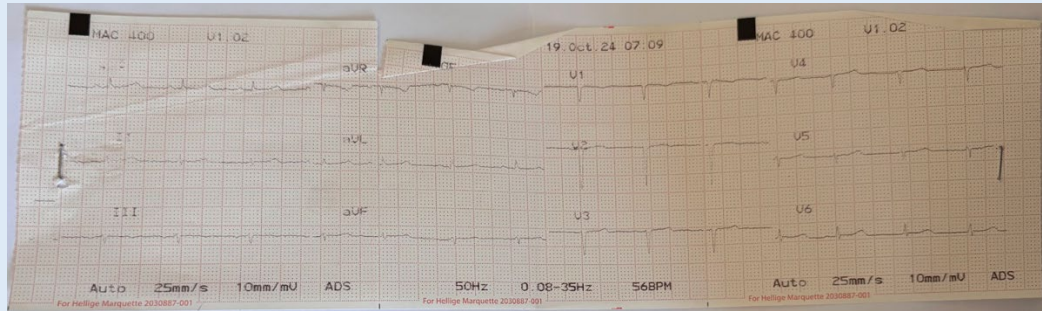


Figure 2. Image of electrocardiogram (ECG)

An electrocardiogram (ECG) performed on October 19, 2024, revealed a normal sinus rhythm. However, the findings also indicated low voltage, which may suggest the presence of pericardial effusion, myocardial disease, or other underlying conditions that warrant further clinical correlation and evaluation.

Laboratory Result

18 th October 2024	Result	Normal Value
Hemoglobin	8.7 g/dl	13.5 – 17.5
Leukocyte	2,200 /mm ³	4,000 – 11,000
Hematocrit	25%	41 - 53
Thrombocyte	86,000/mm ³	150,000 – 440,000
Basophil	0%	0 - 2
Eosinophil	1%	0 - 5
Neutrophil	66%	47 - 80
Lymphocyte	21%	13 - 40
Monocyte	12%	2 - 11
Na	135.2	135-145 mmol/L
K	4.82	3.3 – 4.6 mmol/L
Cl	109	98-106 mmol/L
Glucose ad random	99	80 – 135 mg/dL
Albumin	3.70	3.40 – 4.80 g/L

An echocardiographic evaluation conducted on October 21, 2024, revealed normal diastolic function and right ventricular (RV) contractility, along with trivial aortic regurgitation. However, the imaging identified a moderate to

severe pericardial effusion, predominantly localized at the apical and lateral left ventricular (LV) regions. Additionally, a "swinging heart" motion was observed, a finding often associated with large pericardial effusions. Despite the significant effusion, pericardiocentesis was not performed due to the minimal amount of fluid present in the subcostal area, making the procedure unfeasible at that time.

Further diagnostic testing on the pericardial fluid was conducted on October 24, 2024, using the GeneXpert method to assess for *Mycobacterium tuberculosis*. The results were negative, indicating no evidence of tuberculosis-related pericardial involvement.

A pericardial fluid culture subsequently identified the presence of *Staphylococcus epidermidis*, a common skin commensal that may act as an opportunistic pathogen in immunocompromised individuals. An antibiotic susceptibility test was performed, revealing resistance to erythromycin and clindamycin, which necessitated targeted antimicrobial therapy to effectively manage the infection.

Based on the comprehensive evaluation, including anamnesis, physical examination, and supporting diagnostic tests, the patient was diagnosed with massive complex pericardial effusion, hypoalbuminemia, inflammatory anemia, and systemic lupus erythematosus (SLE). Given the severity of the pericardial effusion, a multidisciplinary approach was implemented to ensure optimal management.

The patient received both supportive and definitive treatment. Supportive management included a combination of oral and intravenous medications, while definitive intervention was performed in the form of video-assisted thoracoscopic surgery (VATS) pericardiotomy to facilitate pericardial fluid drainage and prevent recurrence. A collaborative treatment plan was established through consultations with specialists in internal medicine, cardiology, and dermatology to address the patient's systemic condition comprehensively.

The internal medicine specialist recommended intravenous fluid therapy with IVFD Futrolit administered over 12 hours. Corticosteroid therapy was initiated with an intravenous methylprednisolone injection at a dose of 125 mg once daily for two days, followed by a tapered dose of 62.5 mg on the third day.

Additionally, gastroprotective therapy was prescribed, including lansoprazole injection (30 mg once daily) and sucralfate syrup (10 cc three times daily) to prevent gastrointestinal complications associated with steroid use.

The cardiologist focused on managing the pericardial effusion and its associated complications, advising albumin correction to address hypoalbuminemia. Anti-inflammatory therapy was prescribed, consisting of ibuprofen (600 mg three times daily) and colchicine (0.5 mg once daily) to reduce pericardial inflammation. Serial echocardiography was scheduled to monitor the progression of the effusion and assess the response to treatment.

The dermatologist provided targeted therapy for the patient's cutaneous and mucosal manifestations of SLE. A topical corticosteroid, mometasone cream, was prescribed for application in the morning and at night to manage skin lesions. For mucosal involvement, nystatin drops were administered four times daily to address oral lesions and prevent secondary fungal infections.

Follow-Up

- **19th October 2025**

On the second day of follow-up, the patient continued to experience generalized weakness, persistent mouth ulcers, pain while swallowing, and nausea, which affected her ability to eat and drink comfortably. Her vital signs remained stable, with a blood pressure of 126/99 mmHg, a pulse rate of 65 beats per minute, a temperature of 36.7°C, a respiratory rate of 20 breaths per minute, and an oxygen saturation of 99%. To further assess her condition, a blood albumin test was planned to evaluate potential hypoalbuminemia. Meanwhile, the initial therapeutic regimen was continued to manage her symptoms and underlying conditions.

- **20th October 2025**

On the third day of follow-up, the patient continued to report persistent symptoms, including weakness, mouth ulcers, and discomfort while eating and drinking. However, her vital signs remained within normal limits, indicating stable hemodynamic status. Given the ongoing

symptoms, the current therapeutic regimen was maintained to ensure continued management and symptom relief.

- **21st October 2025**

On the fourth day of follow-up, the patient continued to experience persistent symptoms without significant improvement. Despite these ongoing complaints, her vital signs remained within normal limits. Echocardiographic evaluation confirmed a diagnosis of systemic lupus erythematosus (SLE) with moderate-to-severe pericardial effusion, raising concerns for impending cardiac tamponade. In response, the cardiologist prescribed ibuprofen (600 mg three times daily), colchicine (0.5 mg once daily), and gomino (two tablets three times daily) to manage inflammation and support symptom relief. Given the severity of the pericardial effusion, the patient was referred to a thoracic and cardiovascular surgeon for evaluation and planning of video-assisted thoracoscopic surgery (VATS) pericardiotomy as a definitive intervention.

- **23rd October 2025**

The patient was scheduled for an urgent VATS pericardiotomy by the thoracic and cardiovascular surgery team. In preparation for the procedure, one unit of packed red cells (PRC) was arranged, and an intensive care unit (ICU) ventilator was prepared. A preoperative consultation with the anesthesia team was conducted, and the anesthesiologist approved the emergency surgery. One hour before the procedure, the patient received a preoperative dose of methylprednisolone (1 ampoule) and subcutaneous heparin (5,000 IU) to reduce inflammation and prevent thrombotic complications.

Following the VATS pericardiotomy, the patient was scheduled for a postoperative chest X-ray to assess lung expansion and any potential complications. Additionally, the postoperative management plan included intravenous antibiotic therapy with levofloxacin (750 mg once daily), pain management with dexketoprofen (three times daily), and gastric protection with intravenous ranitidine (twice daily). A

pericardial fluid specimen was also sent for culture and sensitivity testing to guide further antimicrobial therapy if necessary.

- **29th October 2025**

On October 29, 2024, the patient reported mild shortness of breath along with discomfort at the postoperative site. However, her vital signs remained stable and within the normal range. After careful evaluation, she was deemed fit for discharge with a prescribed outpatient medication regimen. This included ibuprofen (600 mg three times daily) and colchicine (0.5 mg once daily) to manage inflammation, an albumin sachet (once daily) to support protein levels, methylprednisolone (8 mg twice daily) for continued immunosuppression, and sucralfate (10 cc three times daily) for gastrointestinal protection. The patient was advised to follow up regularly for monitoring and further evaluation.

DISCUSSION

Pericardial effusion refers to the pathological accumulation of fluid within the pericardial cavity, exceeding the normal physiological range of 15–50 mL.⁵ In young female patients with systemic lupus erythematosus (SLE), pericardial effusion is a well-documented cardiac manifestation, frequently arising due to autoimmune-mediated inflammation of the pericardium.⁹ SLE, a chronic autoimmune disorder, is characterized by widespread immune complex deposition and complement activation, leading to systemic inflammation that can affect multiple organs, including the cardiovascular system.³ The pathogenesis of pericardial effusion in SLE involves both direct immune-mediated injury to the pericardial tissue and secondary insults such as infections, renal dysfunction, and adverse drug reactions, all of which can contribute to increased pericardial fluid accumulation.¹⁰ While mild effusions may be clinically silent, larger or rapidly accumulating effusions pose significant hemodynamic risks, necessitating timely diagnosis and intervention.

The development of pericardial effusion in SLE is primarily attributed to pericarditis, an inflammatory process driven by autoantibodies and immune complexes targeting pericardial tissues.^{11,12} This immune response induces a cascade of inflammatory mediators, increasing vascular permeability and

facilitating the exudation of fluid into the pericardial space. If inflammation persists, impaired lymphatic drainage may exacerbate fluid accumulation, resulting in persistent or progressive pericardial effusion. The clinical impact of the effusion largely depends on the rate of fluid accumulation rather than the absolute volume. A slow, progressive effusion allows the pericardium to stretch and accommodate the fluid, whereas a rapidly accumulating effusion can abruptly elevate intrapericardial pressure, impairing ventricular filling and potentially leading to cardiac tamponade—a life-threatening emergency characterized by hemodynamic instability and circulatory collapse.^{13,14}

The diagnosis of pericardial effusion requires a comprehensive evaluation, incorporating clinical assessment and multimodal imaging. Echocardiography remains the gold standard for detecting and characterizing pericardial effusions, offering real-time assessment of fluid volume, distribution, and its hemodynamic consequences.¹⁵ Chest radiography, although less sensitive, may reveal cardiomegaly or an enlarged cardiac silhouette in cases of significant effusion. Electrocardiography (ECG) findings, while nonspecific, may show low voltage QRS complexes or electrical alternans in large effusions, suggestive of a swinging heart within the fluid-filled pericardial sac.¹⁶ In complex or equivocal cases, cardiac computed tomography (CT) and magnetic resonance imaging (MRI) provide high-resolution anatomical detail, aiding in the differentiation of transudative versus exudative effusions and detecting associated pericardial thickening, inflammation, or fibrosis.

The management of pericardial effusion in SLE patients is determined by its severity, underlying cause, and clinical impact, necessitating a multidisciplinary approach involving rheumatologists and cardiologists. For mild, asymptomatic effusions, conservative management with close monitoring and pharmacologic therapy—primarily nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, and corticosteroids—is often sufficient to control inflammation and prevent progression.⁴ In cases where effusion volume increases or symptoms such as dyspnea or chest pain develop, more aggressive intervention may be required. Hemodynamically significant effusions or cardiac tamponade necessitate urgent pericardiocentesis to rapidly drain the fluid and relieve cardiac compression.^{17,18} In

patients with recurrent effusions, particularly those unresponsive to medical therapy, pericardial window surgery or pericardiectomy may be performed to prevent reaccumulation and minimize long-term complications.⁵

Complications of pericardial effusion vary in severity, ranging from asymptomatic, self-limiting cases to life-threatening conditions. The most critical complication is cardiac tamponade, in which increased intrapericardial pressure impairs ventricular filling, leading to hypotension, pulsus paradoxus, and eventual cardiovascular collapse if untreated.^{18,19} In chronic cases, prolonged pericardial inflammation can result in pericardial fibrosis, ultimately leading to constrictive pericarditis—a condition where the rigid, thickened pericardium restricts diastolic filling and causes heart failure-like symptoms.^{20,21} Given these potential complications, early recognition, vigilant monitoring, and timely intervention are paramount in ensuring favorable outcomes for young female patients with SLE and pericardial involvement.

CONCLUSION

A patient with systemic lupus erythematosus (SLE) developed a severe pericardial effusion, causing potential cardiac tamponade. The effusion was attributed to lupus-associated pericarditis and a secondary bacterial infection. The patient was treated with a combination of corticosteroids, immunosuppressive agents, and targeted antibiotic therapy. The effusion was severe enough to necessitate VATS pericardiotomy, ensuring adequate drainage and preventing recurrence. This case underscores the importance of early recognition and multidisciplinary management of pericardial effusion in young SLE patients, requiring close monitoring and individualized treatment. Serial echocardiographic assessments and ongoing immunosuppressive therapy are crucial in long-term disease management.

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