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Cardiac Tamponade and Pulmonary Embolism as a Dual Initial Presentation of Systemic Lupus Erythematosus: A Case Report

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Abstract | **Case Report**

Introduction: Systemic lupus erythematosus (SLE) associated with antiphospholipid syndrome (APS) significantly increases cardiovascular risk. While pericarditis is common in SLE, progression to inaugural cardiac tamponade is exceptional. The simultaneous occurrence of tamponade and pulmonary embolism (PE) as the primary manifestation is extremely rare. ***Case Presentation:*** A 41-year-old female with a history of spontaneous abortions was admitted for obstructive shock and respiratory distress. Echocardiography revealed a large pericardial effusion with marked right chamber dilation. Following life-saving pericardiocentesis (900 ml), persistent hypoxemia led to the diagnosis of proximal PE via CT angiography. Laboratory tests confirmed an SLE-APS overlap syndrome. The patient was successfully managed through therapeutic sequencing: pericardial drainage, followed by cautious anticoagulation and high-dose corticosteroid therapy. ***Conclusion:*** This case highlights an "inverted echocardiographic phenotype" where pulmonary hypertension masks the typical right chamber collapse of cardiac tamponade. A sequential and multidisciplinary approach is essential to address this dual mechanical and thrombotic obstruction.

Keywords: Systemic lupus erythematosus; antiphospholipid syndrome; Cardiac tamponade; Pulmonary embolism; Obstructive shock.

1. INTRODUCTION

Systemic lupus erythematosus (SLE) is frequently associated with antiphospholipid syndrome (APS), a coexistence that significantly increases the risk of complex cardiovascular and valvular involvement [1]. Although pericardial involvement is the most common cardiac manifestation of SLE, its progression to inaugural cardiac tamponade remains an exceptional clinical event [2]. Similarly, while thromboembolic events such as pulmonary embolism (PE) or myocardial infarction are formidable and well-documented complications of APS [3], the simultaneous occurrence of tamponade and PE as the very first manifestation of an SLE-APS overlap syndrome is extremely rare [4]. This 'double obstructive shock,' arising from distinct but pathophysiologically intertwined etiologies, constitutes a major diagnostic challenge and a therapeutic paradox. We report the case of a 41-year-old female patient illustrating this exceptional inaugural presentation, highlighting the importance of multidisciplinary management in the face of these concurrent hemodynamic emergencies.

2. OBSERVATION

A 41-year-old multiparous female (mother of two) with a history of two early spontaneous abortions was admitted to the emergency department for acute respiratory distress associated with sudden-onset, intense chest pain exacerbated by respiratory movements. She had no cardiovascular risk factors or significant prior medical history. Further history taking revealed progressive dyspnea over the preceding two months, occurring alongside constitutional symptoms (fatigue, anorexia, and a 2 kg weight loss), intermittent polyarthralgia, and Raynaud's phenomenon.

Upon admission, clinical examination revealed an obstructive shock state: blood pressure of 89/50 mmHg, heart rate of 120 bpm, tachypnea, and oxygen saturation of 87% on room air. Physical examination showed muffled heart sounds, signs of bilateral pleural

effusion, and evidence of right heart failure (pitting lower limb edema reaching the knees). The electrocardiogram (ECG) demonstrated sinus tachycardia and electrical alternans (Figure 1).

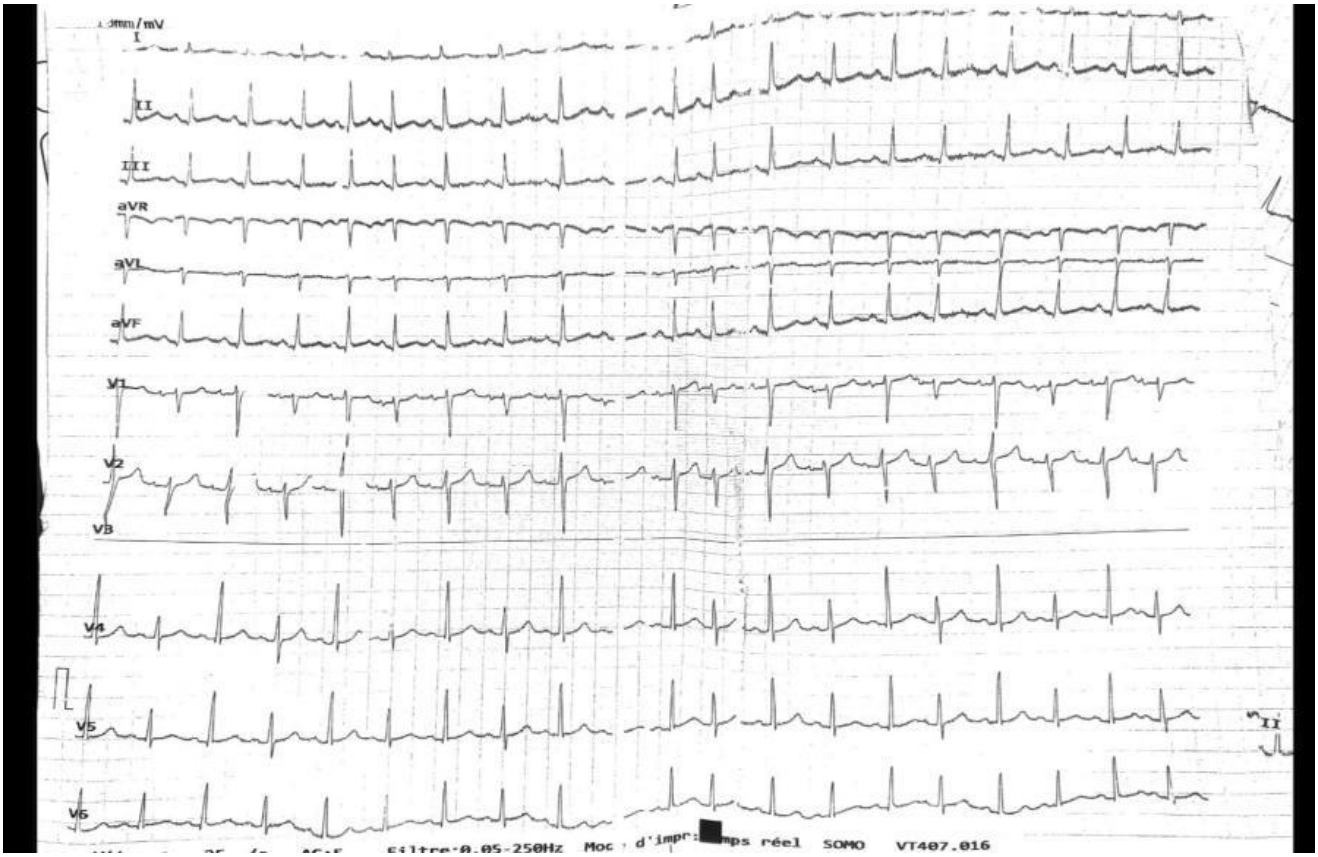

Figure 1: Twelve-lead ECG on admission: sinus tachycardia with electrical alternans

Emergency transthoracic echocardiography (TTE) confirmed cardiac tamponade, showing a large-volume pericardial effusion and marked dilation of the right heart chambers (Figure 2). A life-saving pericardiocentesis was performed, draining 900 ml of serosanguinous fluid. Biochemical analysis identified the fluid as an exudate, rich in protein with inflammatory cellularity, suggesting a systemic etiology.

Despite the resolution of the tamponade and initial hemodynamic improvement, persistent hypoxemia necessitated a chest CT angiography. This revealed a proximal right pulmonary embolism (PE) associated with bilateral pleural effusions (Figure 3). Anticoagulant therapy with low-molecular-weight heparin (LMWH) was initiated.

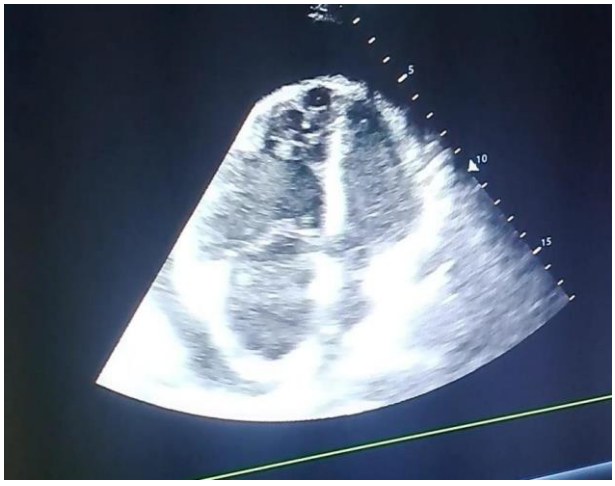

Figure 2: Transthoracic echocardiography (apical four-chamber view): large circumferential pericardial effusion (*) withright heart dilatation

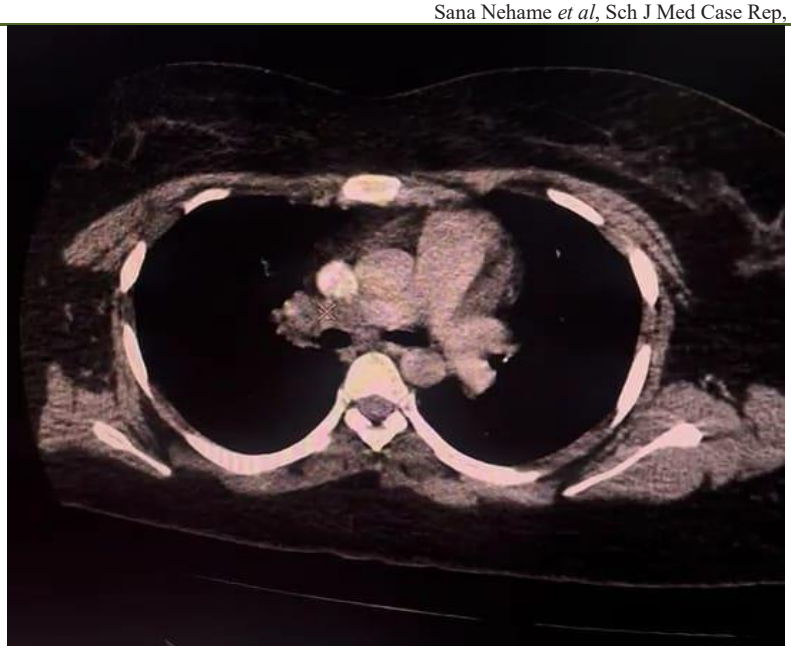

Figure 3: CT pulmonary angiography: proximal right pulmonary embolism

The etiological workup revealed leukopenia, hypocomplementemia, and positivity for antinuclear antibodies (ANA), anti-dsDNA, and anti-Sm antibodies. Initial thrombophilia screening showed the presence of anticardiolipin antibodies. The diagnosis of Systemic Lupus Erythematosus (SLE), meeting the 2019 EULAR/ACR criteria, associated with antiphospholipid syndrome (APS), was established.

The patient received pulse corticosteroid therapy with methylprednisolone (1 g/day for 3 days), followed by oral prednisone (1 mg/kg/day) and hydroxychloroquine. The clinical course was favorable, with complete hemodynamic stabilization and resolution of respiratory symptoms. A transition to vitamin K antagonists (VKA) was initiated for the PE.

Follow-up at 12 weeks confirmed the APS diagnosis according to the Sydney criteria, with persistent IgG anticardiolipin antibodies and the presence of lupus anticoagulant (LAC). Long-term clinical and echocardiographic follow-up has remained satisfactory.

3. DISCUSSION

3.1 Cardiac Tamponade: An Atypical Inaugural Manifestation of SLE

Although pericardial involvement is a classic manifestation of systemic lupus erythematosus (SLE), reported in 10% to over 50% of patients [1], its progression to cardiac tamponade remains a rare complication, occurring in less than 3% to 5.9% of cases overall [4,5,6]. Its occurrence as the inaugural manifestation revealing the autoimmune disease is even more exceptional, representing only 1% to 2.8% of cases according to large retrospective series. The literature has identified several clinical and biological predictive factors for this severe hemodynamic evolution, including female sex, concomitant pleurisy, renal involvement, cytopenias (notably anemia), and, most discriminantly, severe hypocomplementemia with a marked decrease in the C4 fraction [7]. In summary, our patient's clinical presentation—marked by cardiac tamponade as the first sign of her disease, accompanied by pleural effusion, leukopenia, and hypocomplementemia—perfectly aligns with the high-risk phenotypic profile identified in cohort studies.

3.2 APS and Thromboembolic Risk: Pulmonary Embolism

Pulmonary embolism (PE) represents the most frequent and feared pulmonary complication of antiphospholipid syndrome (APS), occurring in approximately 14% to 15% of patients. This thromboembolic risk is considerably amplified when APS occurs in the context of underlying SLE. Indeed, the deleterious synergy between the endothelial dysfunction induced by chronic lupus inflammation and the prothrombotic state inherent to antiphospholipid antibodies creates a highly thrombogenic environment [8]. Consequently, lupus patients have a risk of PE three times higher than the general population, with an incidence of around 7.3%. The coexistence of APS drastically worsens the prognosis, with thromboembolic complications responsible for more than a quarter (26.7%) of deaths in this specific subpopulation [4].

3.3 An Exceptionally Rare Concomitant Association

While pulmonary embolism and cardiac tamponade can occur in isolation during the course of lupus, their inaugural and strictly simultaneous presentation remains exceptionally rare. The literature emphasizes that the coexistence of severe pericardial effusion and thromboembolic disease typically stems from neoplastic etiologies or severe infections.

Attributing this presentation to an SLE-APS overlap syndrome gives our observation a remarkable educational dimension, illustrating the potential severity of the revealing forms of this connective tissue disease [4,9-10].

3.4 Hemodynamic Interaction and the Echocardiographic Trap

The concomitant occurrence of cardiac tamponade and massive pulmonary embolism creates a particularly complex hemodynamic state, making clinical diagnosis and echocardiographic interpretation difficult. Each of these conditions independently causes obstructive shock with decreased cardiac output, elevation of right-sided heart pressures, and systemic hypoperfusion. Their simultaneous occurrence can lead to a convergence of their respective hemodynamic manifestations, which may mask or confuse each other, leading to diagnostic errors.

Pathophysiologically, this association generates a major pressure conflict: while isolated tamponade causes collapse of the right chambers, pulmonary embolism (PE) induces acute pulmonary arterial hypertension (aPAH) leading to their dilation [11]. Their coexistence creates a "hemodynamic stent" effect, where the intracavitary hyper-pressure from the PE mechanically opposes the pericardial compression. This results in a misleading "inverted echocardiographic phenotype," characterized by marked dilation of the right ventricle (RV) without classic compressive signs. This diagnostic trap risks underestimating the severity of the effusion and delaying life-saving drainage [12]. As highlighted in the literature, such RV dilation in a compressive context should immediately raise suspicion of associated pulmonary vascular pathology. In clinical practice, persistent severe hypoxemia despite pericardiocentesis constitutes the decisive clinical clue mandating chest CT angiography.

3.5 The Therapeutic Paradox: Drainage vs. Anticoagulation

The association of tamponade and pulmonary embolism (PE) creates a major therapeutic dilemma. These two emergencies require diametrically opposed treatments. On one hand, PE requires immediate curative anticoagulation. On the other hand, cardiac tamponade formally contraindicates anticoagulation. Anticoagulating a patient with an inflamed and freshly punctured pericardium carries the risk of fatal hemorrhage (hemopericardium) [13].

To resolve this issue, it is imperative to apply a three-step management sequencing [14-17]:

1. **Emergency Drainage (Pericardiocentesis):** The primary priority is to save the patient's life by relieving the obstructive shock and restoring cardiac filling. Pericardial fluid drainage must be performed immediately, before any other treatment [14].
2. **Cautious Introduction of Anticoagulation:** Anticoagulation is only started after pericardial drainage, once the risk of hemorrhage is controlled. Experts often recommend intravenous unfractionated heparin (UFH) due to its short half-life and the availability of an immediate antidote (protamine sulfate), alongside strict clinical and echocardiographic monitoring.
3. **Treatment of the Autoimmune Cause:** Finally, treating the cause of inflammation is essential to prevent recurrence. High-dose corticosteroids (pulse methylprednisolone) rapidly stop fluid secretion by the pericardium. This "drying" effect reduces the risk of further bleeding and allows for the safe continuation of anticoagulation.

However, the theoretical risk of immunosuppression induced by these corticosteroids, potentially favoring infectious complications following pericardiocentesis, further complicates the clinical picture.

4. CONCLUSION

This case illustrates an exceptional clinical presentation where cardiac tamponade and massive pulmonary embolism act as the dual revealers of an SLE-APS overlap syndrome. This case highlights that the absence of right chamber collapse on echocardiography should not rule out tamponade if shock persists, as associated pulmonary hypertension can mask this classic sign.

Management success relies on rigorous therapeutic sequencing: immediate mechanical relief via pericardiocentesis, followed by cautious anticoagulation and aggressive immunosuppression to control the immunological storm. A multidisciplinary approach is indispensable to navigate this therapeutic paradox and improve the vital prognosis of these young patients.

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Conflict of Interest Statement:

The authors declare that they have no conflicts of interest related to this article.

Patient Consent:

Written informed consent was obtained from the patient for the publication of this case report and the associated clinical and iconographic data, in accordance with current ethical standards.

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