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# Concomitant Pericardial Effusion and Pulmonary Embolism Secondary to a Systemic Lupus Erythematosus with Antiphospholipid Antibody Syndrome: A Rare Case Report

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***Authors' contributions***

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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***Case Report***

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## ABSTRACT

**Background:** Association of pulmonary embolism and pericardial effusion is rarely described in the literature, it is associated in the majority of cases with a neoplastic origin followed by a tuberculous origin. Within the limits of our research, no cases of lupus associated with antiphospholipid syndrome have been reported.
**Case Report:** We illustrate a case of a 25-year-old patient, presenting with respiratory discomfort and palpitations and in whom echocardiography shows a pericardial tamponade. The realization of athoracic CT angiography shows a concomitant pulmonary embolism. The etiological assessment poses the diagnosis of systemic lupus erythematosus (SLE) associated to antiphospholipid antibody syndrome.

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**Conclusions:** Pulmonary embolism and pericardial effusion can be life-threatening for the patient. This association highlights a difficult clinical dilemma with regard to anticoagulation in the presence of pericardial effusion, which remains a relative contraindication to anticoagulation.

*Keywords: Pericardial effusion; pericardial tamponade; pulmonary embolism; systemic lupus erythematosus; antiphospholipid antibody syndrome.*

## ABBREVIATIONS

*SLE : Systemic Lupus Erythematosus*
*PE : Pulmonary Embolism*
*APS : Anti-phospholipid Syndrome*
*AVK : Anti Vitamin K*

## 1. INTRODUCTION

Cardiac tamponade is a diagnostic and therapeutic emergency secondary to rapid accumulation or great abundance of pericardial effusion causing compression of the right heart chambers with left hypoflow [1]. Pulmonary embolism is also an emergency [2]. The association of the two pathologies is rare and often associated with a neoplastic origin, [3,4] and in our context , lupus with anti phospholipid syndrome, a cause very rarely reported in the literature.

The association of pulmonary embolism with pericardial effusion poses a diagnostic, etiological and therapeutic dilemma.

## 2. CASE PRESENTATION

A 25-year-old woman, was admitted to the emergency for respiratory discomfort evolving for 1 month, exacerbated 2 days ago, associated with palpitations. She didn't have obstetrical history, had been treated for 2 years for arthralgia of the knees and ankles with analgesics and non-steroidal anti-inflammatories without etiological assessment.

Medical examination revealed glasgow at 15/15, blood pressure 87/56 mmHg, dyspnea, oxygen saturation 92% , tachycardia at 227 beats per minute. The cardiovascular and pulmonary examination showed a decrease in heartsounds with jugular distension and edema of the lower limbs.

EKG revealed supra ventricular tachycardia at 227 beats per minute and repolarization disorders (Fig. 1).

An echocardiographic showed : an abundant pericardial effusion next to the right cavities: 38mm next to the right ventricule, 30mm next to the right atrium with significant variations in the respiratory flows, a non-compliant inferior vena cava dilated to 23 mm, right cavities collapsed by the effusion, with fibrin deposit (Fig. 2).

Patient was admitted to our intensive care unit, received oxygen therapy, infusion of 500 ml of crystalloids and pericardiocentesis of 900 ml of sero haematic fluid. A clear hemodynamic and respiratory improvement was obtained after pericardiocentesis.

Chest CT angiography was performed after pericardial drainage noted appearance of a saddle pulmonary embolism associated with bilateral pneumopathy suggestive of pulmonary infarction (Fig. 3). The venous doppler echo of the lower limbs showed no thrombosis.

On the biological assessment, there was a normochromic normocytic anemia (hemoglobin 9 g/dl), lymphopenia at 370/mm3, slight thrombocytopenia at 100,000/mm3, D-Dimers at 1450ug/L.

The pericardial fluid was exudative without the presence of tumorcells. A covid-19 serology came back negative. A search for tuberculosis was inconclusive. C-reactive protein was elevated at 136 mg/dL, erythrocyte sedimentation rate was 87 mm. Procalcitonin was negative.

Rheumatoid factor was positive, natives anti-DNA antibodies were high and anti-cardiolipin were positive.

A diagnosis of lupus with antiphospholipid antibody syndrome was concluded in the presence of several positive criteria.

There was no recurrence of pericardial effusion after percidardiocentesis. An anticoagulant treatment based on vitamin K antagonists was introduced for the treatment of pulmonary embolism with close clinical and echocardiographic monitoring. Treatment with oral corticosteroid therapy and hydroxycloroquine was started.

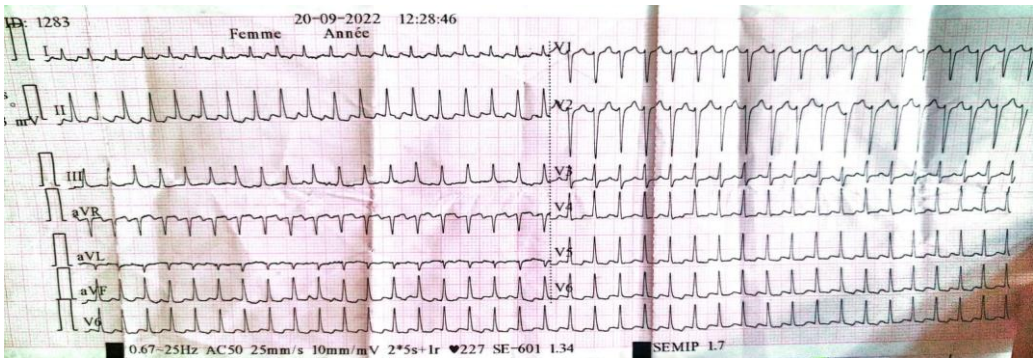


**Fig. 1. 12-lead ECG notifying supraventricular tachycardia at 227 beats per minute**

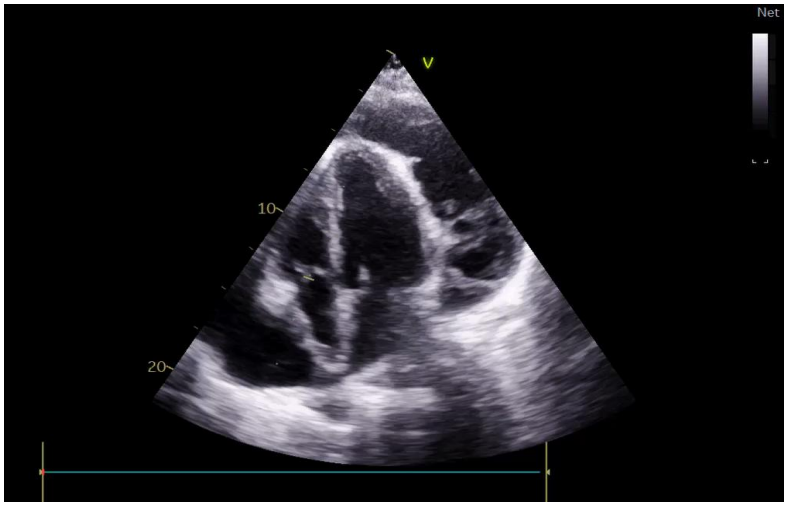


**Fig. 2. Apical 4-chamber echocardiographic image showing significant pericardial effusion with fibrin deposition**

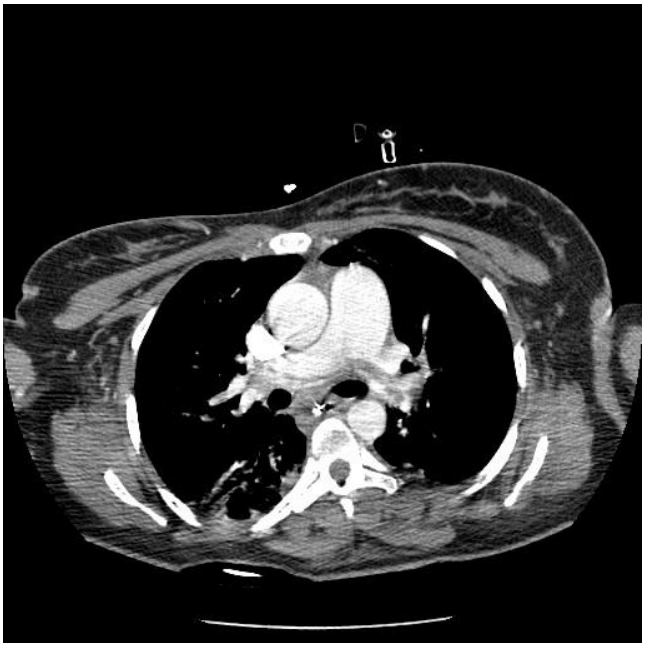

**Fig. 3. Chest CT angiography noted appearance of a saddle pulmonary embolism**

The patient is still currently being followed up by cardiology and internal medicine units with no recurrence of effusion, no bleeding under anticoagulant detected and stabilization of her lupus.

## 3. DISCUSSION

Cardiac tamponade is a life-threatening emergency that occurs when bulky fluid accumulates in the pericardial sac compressing the heart and causing hypotension and cardiogenic shock [1].

Pulmonary embolism (PE) occurs when there is a disturbance of blood flow in the pulmonary artery or its branches by a thrombus originating elsewhere: the veins of the lower limbs in general [2].

The association of pulmonary embolism and pericardial effusion is rare, the main etiology reported in the literature is neoplastic origin , and mainly pulmonary neoplasia as reported in this recently published case [5], or adenocarcinoma of colon [6], followed by tuberculosis [3,4,7,8]. Another case has been reported in the literature concerning a patient initially admitted for a pulmonary embolism, put on anticoagulant treatment based on apixaban, the evolution was marked by the appearance of a cardiac tanponnade, the possible explanation was a reaction to anticoagulation, a cause rarely found, a second explanation which was more plausible but also rare, is a pericardial reaction secondary to pulmonary embolism and which responds to anti-inflammatories [9] .

In our case, after ruling out the causes mentioned above, a diagnosis of SLE was made according to the American College of Rheumatology Classification Criteria for Systemic Lupus Erythematosus [10].

Systemic lupus erythematous is a chronic, multisystemic, inflammatory, autoimmune disease, occurring mainly in young women. The best-known manifestations include arthralgia, Raynaud's syndrome, malar rash, pleurisy or pericarditis, renal or central nervous system involvement, and autoimmune cytopenias. Diagnosis requires clinical and serological criteria including the presence of antinuclear antibodies. Treatment of severe assets requires corticosteroids and immunosuppressants [11].

Anti-phospholipid syndrome (APS) is a rare autoimmune disease comprising clinical manifestations: venous or arterial thrombosis, or obstetrical complications (fetal death in utero) in women, associated with biological manifestations: presence of auto- various antibodies directed against one or more proteins associated with phospholipids, and/or anti-cardiolipin antibodies, and/or an elongated TCA with the presence of a circulating anticoagulant. In the absence of symptoms related to APS, we speak of isolated anti-phospholipid biology (and not of a syndrome). In some cases, APS is associated with other autoimmune diseases (such as systemic lupus). 20 to 30% of people followed for systemic lupus have associated APS, and 40% have associated isolated anti-phospholipid biology. As with lupus, women are 4 to 5 times more often affected. The treatment is essentially based on anticoagulant and/or anti-platelet aggregation treatments [12].

The association of pulmonary embolism with pericardial effusion poses a diagnostic, etiological and therapeutic dilemma.

Clinically, the symptoms and signs of cardiac tamponade and pulmonary embolism are nonspecific. The association of tamponade and pulmonary hypertension in the same patient is responsible for a compensatory mechanism, improving the prognosis. Indeed, the pulmonary hypertension exerted by the pulmonary embolism, if it is extensive, makes it possible to increase the pressures at the level of the right ventricle and to attenuate the collapse and the dysfunction of the right ventricle caused by the tamponade, thus finding a echocardiography a moderate collapse of the free wall of the RV contrasting with the abundance of the effusion. And this improves the prognosis of the patient in the acute phase [3].

The therapeutic dilemma concerns the anticoagulation of pulmonary embolism in the face of a hemorrhagic risk linked to the pericardial effusion, especially if a neoplastic origin is found. There are no clear recommendations on this subject, management is then done on a case-by-case basis, starting with a careful assessment of the risks and benefits of anticoagulation, then anticoagulation and etiological treatment, with close monitoring as was the case with our patient. In addition, the placement of a vena cava filter in certain situations of venous thrombosis has been proposed.

Regarding anticoagulation in the context of anti phospholipid syndrome and in the current state of knowledge, the recommendations issued at the International APS Congress in 2020 were as follows: the rule remains to favor anti vitamin K (AVK) in the treatment of APS, certainly in patients who are triple positive and/or with arterial thromboembolic manifestations and/or Liebman-Sacks endocarditis. On the other hand, one could, on a case-by-case basis, consider a direct oral anticoagulants in single or double positive patients who have only suffered from a single episode of venous thrombosis, in particular if the monitoring of AVK treatment is likely to be suboptimal [13].

## 4. CONCLUSIONS

Pulmonary embolism and pericardial effusion are two serious pathologies requiring urgent treatment. The association of the two is rare, the main causes found in the literature, of this association, are neoplasia, tuberculosis and in our case a systemic disease. This association represents a therapeutic emergency with a greater hemorrhagic risk of anticoagulation and a need for etiological treatment to avoid a recurrence.

## CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

## ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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