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Case Report

Internal Medicine Section

Antiphospholipid Syndrome Complicated by Unilateral Pleural Effusion and Deep Vein Thrombosis: A Case Report

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ABSTRACT

Antiphospholipid Antibodies (APLA) are autoantibodies directed against phospholipid-binding proteins. The development of thromboses in veins/arteries or the incidence of maternal morbidity, particularly miscarriages, in the presence of APLA constitutes Antiphospholipid Syndrome (APS). These Antiphospholipids (APLs) include antibodies against two glycoproteins, anticardiolipin antibodies, and Lupus Anticoagulant (LA). APLs are a diverse group of autoantibodies that have been linked to thrombus development, elevated maternal morbidity and mortality, and other symptoms that collectively constitute APS. Approximately half of APS cases are secondary disorders, while the remaining cases are autoimmune co-morbidities such as Systemic Lupus Erythematosus (SLE). This multisystem disorder mainly causes venous and arterial thrombosis and can also manifest as cutaneous, pulmonary, renal, and haematological involvement. APLA syndrome with pleural effusion is a rare complication. Patients with antiphospholipid syndrome can develop various types of pulmonary disease. The authors present the case of a 38-year-old male patient with a history of Deep Vein Thrombosis (DVT) and psoriasis, a known case of APLA syndrome, who presented with neck pain and breathlessness. Pulmonary embolism was ruled out, and the patient was discharged on oral anticoagulants. The present case highlights APLA as the culprit for the myriad presentations in the present case.

Keywords: Corticosteroid, Cardiovascular problem, Immune system, Lungs, Psoriasis

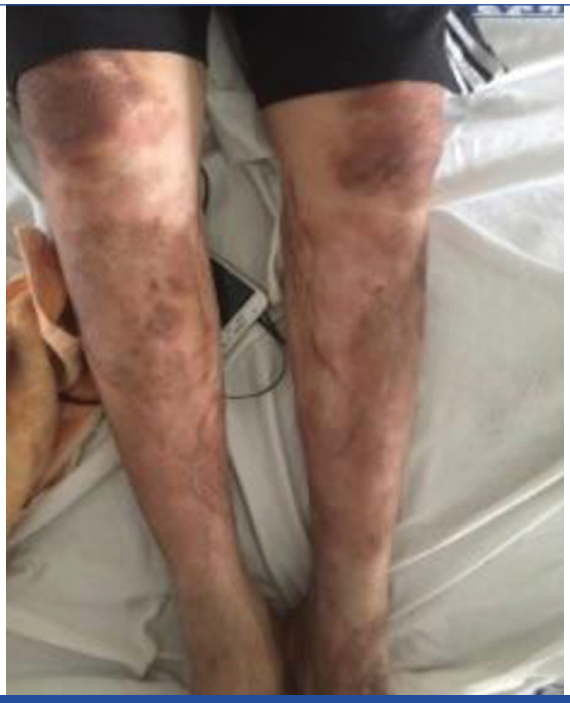
CASE REPORT

A 38-year-old male, a truck driver by occupation, presented to the Emergency Department with complaints of neck pain radiating to the back, breathlessness for two days, and throbbing pain in the right calf region. The pain in the calf was acute in onset, progressive, and accompanied by swelling in the right leg for six days. The patient had no history of hypertension, diabetes, bronchial asthma, or tuberculosis.

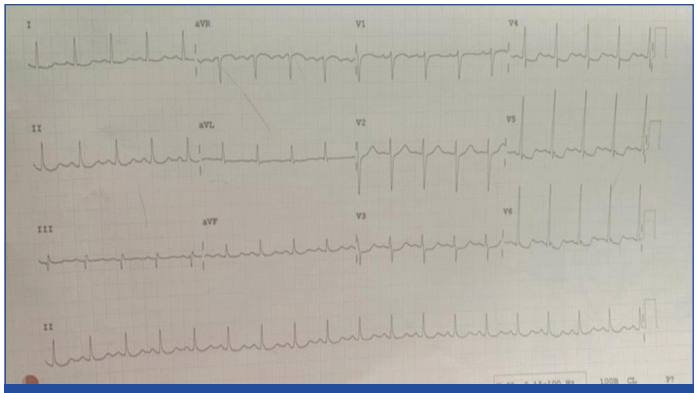
The patient had a known case of Antiphospholipid Syndrome (APLA) for the past eight years and psoriasis for the past five years. He also had a history of Deep Vein Thrombosis (DVT) for the past one year and was taking a daily dose of 5 mg of warfarin for nine months. On examination, the patient appeared anxious, with a pulse rate of 110/min and blood pressure of 110/70 mmHg. There were no signs of pallor or icterus, and the Jugular Venous Pressure (JVP) was normal. However, there was unilateral oedema in the right leg, [Table/Fig-1,2]. and cardiovascular examination revealed normal heart sounds without murmurs. Respiratory examination revealed dull percussion note on the left side of the chest and absent air entry on the left side of the lung. The patient also complained of chest pain, leading to an Electrocardiogram (ECG) [Table/Fig-3] that showed ST segment depression (1 mm in V1-V3) and ST segment depression of 1 mm in V1 to V6. D-dimer test was negative, but Creatine Phosphokinase-Myocardial Band (CPK-MB) and Troponin Y were positive, suggesting a probable diagnosis of unstable angina. The patient was given a loading dose of aspirin, clopidogrel, and atorvastatin and was then shifted to the cardiac Intensive Care Unit (ICU). A 2D-echo was performed, revealing a Left Ventricular Ejection Fraction (LVEF) of 60% and Grade 1 Diastolic Dysfunction [1]. Coronary angiography was advised, but the patient declined.

Laboratory findings	Results
Haemoglobin	13.1 gm%
Prothrombin time	30.3 seconds
International normalised ratio	2.34 ratio
Creatine phosphokinase- Myocardial band (CPK-MB)	19.0 IU/L
Troponin T	32.10 ng/L
Lupus Anticoagulant (LA)	Present
Perinuclear antineutrophil cytoplasmic antibodies	Negative
Cytoplasmic antineutrophil cytoplasmic antibodies	Negative
Antinuclear antibody	Negative
Anti-ds-DNA antibody	Negative
Beta-2 glycoprotein 1-Immunoglobulin G (IgG)	Negative
Beta-2 glycoprotein 1-Immunoglobulin M (IgM)	Negative

[Table/Fig-1]: Laboratory findings.
DNA: Deoxyribonucleic acid

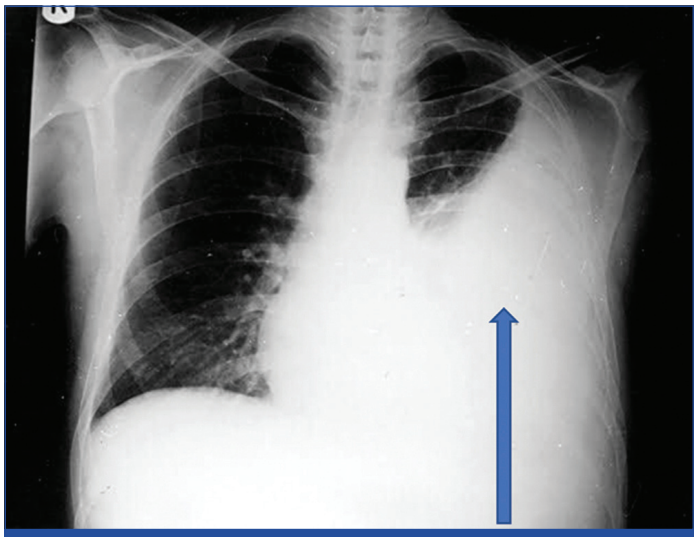


[Table/Fig-2]: Showing oedema in right leg.



[Table/Fig-3]: Showing ECG: ST segment depression.

A chest X-ray showed pleural effusion on the left side of the lung. [Table/Fig-4]. Other potential causes, such as cardiac or pulmonary thrombosis, malignancy, and collagen vascular disease, were ruled out. A right lower limb doppler revealed a long segment complete lumen occluding echogenic thrombus extending from the mid-femoral vein into the saphenofemoral junction, popliteal vein, tibia-peroneal trunk, peroneal vein, and short saphenous vein up to 10 cm distal to the knee joint.



[Table/Fig-4]: Chest X-ray showing left-side pleural effusion.

As a result, aspirin, clopidogrel, and low molecular weight heparin were discontinued, and warfarin was initiated to maintain an International Normalised Ratio (INR) of 2.5. The patient was discharged without any follow-up.

It is extremely rare to observe antiphospholipid syndrome with unilateral pleural effusion. In this case, the patient was discharged on oral anticoagulant warfarin with a daily dose of 10 mg.

DISCUSSION

Males and females are almost equally affected in terms of thrombosis developed in arteries/veins in the clinical presentation of APS, an autoimmune illness that affects more women than men on average. APS can be diagnosed based on clinical and test results. Symptoms of the illness include recurrent venous and arterial thrombosis, as well as females experiencing pregnancy loss. The patient tested positive for LA, anti-beta2 glycoprotein IgG, and borderline antiphospholipid antibodies (IgG). It is well recognised that LA increases the risk of thrombotic events, and several publications have shown the relation of antiphospholipid syndrome with thrombosis at various sites [2-4].

The clinical symptoms of APS can affect numerous organs and systems, leading to a wide range of manifestations. In the current case, the emergence of arterial/venous thrombosis was observed in a patient who had a history of the same problem and presented with DVT in the left leg. Additional thromboembolic events at various sites have been documented, such as pulmonary embolism and cerebral venous thrombosis. While hypercoagulability may be the first symptom in many cases, joint pain and swelling have also been described. APS can affect various systems, including cardiovascular, genitourinary, endocrinological, and central neurological systems [5-8].

In a 75-year-old patient with erythema, purpura, and dyspnoea who had a history of APS and was taking aspirin and warfarin for the same, a similar case of unilateral pleural effusion was observed. The patient also had dermal and subcutaneous small vessel thrombosis. A patient's Computed Tomography (CT) and chest X-ray revealed a left pleural effusion, which was repeatedly drained but recurred. To treat this, oral prednisolone was given, which helped the patient with both a skin lesion and a pleural effusion. Pleural effusion associated with APS is relatively uncommon [9].

Pulmonary thromboembolic illness, which is a condition in a patient who tests negative for PL and is clinically identical to typical emboli, was the first serious pulmonary consequence to be noted. Pulmonary hypertension may develop as a result of recurrent pulmonary emboli. There are numerous potential reasons for pulmonary hypertension in the APLA syndrome, and between 10% and 20% of individuals with chronic thromboembolic pulmonary hypertension also have APL. A statistically significant link between pulmonary hypertension and the presence of IgA anticardiolipin above 2SD has been found in a prospective examination of 500 SLE patients. Chronic anticoagulation is required in every case to treat pulmonary hypertension and avoid the development of fresh thrombotic events. Along with these symptoms, patients with pulmonary capillaritis, alveolar haemorrhage, and microvascular pulmonary thrombosis have also been documented [10].

In addition to thrombosis, APS can cause serious cardiovascular problems. A collection of symptoms known as Acute Coronary Syndrome (ACS) is brought on by the rupture of coronary atherosclerotic plaques with thrombi. ST-segment elevation, non ST elevation, and unstable angina are all components of ACS. Recent research has shown a connection between APS and ACS. For instance, Djokovic A et al., observed that out of 374 patients with APS, including 36 patients with unstable angina, 101 had cardiovascular problems. Acute myocardial infarction and APS are frequently treated with Percutaneous Coronary Intervention (PCI), as documented in several examples [11-16].

Warfarin and heparin are two of the most common treatments for acute thrombotic events. Given the significant risk of recurrence in the first six months after therapy withdrawal, it is imperative that patients who experience a thrombotic episode take steps to prevent recurrent thrombosis [17].

It is still unknown what caused the pleural effusion in the present case. Malignancies, viral illnesses, pulmonary emboli, collagen vascular disease, and heart failure are among the common causes of pleural effusion. Few cases of APS-related pleural effusion have been documented, and those that have appeared to be side-effects of concomitant SLE, pulmonary embolism, or catastrophic APS. The most typical pleuropulmonary sign of SLE is pleuritis, which can lead to pleural effusion [18-21].

CONCLUSION(S)

Antiphospholipid syndrome (APS) with DVT, pleural effusion, psoriasis, and unstable angina is a rare presentation. DVT is common in patients with a history of APS, and the mainstay of treatment is long-term anticoagulation. Since pleural effusion can be a life-threatening condition, timely diagnosis and treatment are essential, along with proper follow-up. Corticosteroids and immunosuppressants continue to be the preferred choices for patients with APS and pleural effusion.

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