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A Case Report On Mild Symptomology -Antiphospholipid Antibody (Apla)

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ABSTRACT

Antiphospholipid Antibody (APLA) Syndrome is an autoimmune condition marked by a hypercoagulable state, which predisposes individuals to thrombosis in both arterial and venous vessels throughout the body. This disorder is commonly associated with complications such as thromboembolic events and pregnancy issues. According to population-based studies, the estimated incidence ranges from 1 to 2 cases per 100,000 individuals, while the prevalence is reported to be between 40 and 50 cases per 100,000. APS can present as a primary or as a secondary condition in association with Systemic Lupus Erythematosus (SLE) or other autoimmune diseases. APLA is a diverse and can vary from clinical presentation. Therefore here, a case of 21year old female patient presented with complaints of numbness in left UL for 6 months, neck pain in (R) side. At the age of 5, the patients had medical history of jaundice and the patient had persistent headache and was worsen and Brain MRI showed (L) Thalamic infarct hypoplasia transverse and sigmoid colon. The patient was treated with initial stage of first line medication such as blood transfusion, multivitamin and analgesic medication. Here, I have presented with antiphospholipid antibody syndrome patient with observed minor complication such as splenomegaly, constipation, calcified granuloma which was detected in CT whole abdomen. In MRI with lumbar spine screening showed mild disc bulging with small profusion in the lumbar region.

Keywords: *APLA (Antiphospholipid Antibody), Systemic Lupus Erythematosus (SLE), transverse sigmoid colon.*

1. INTRODUCTION

Antiphospholipid Syndrome (APS) is an autoimmune thrombo-inflammatory disorder characterized by recurrent vascular thrombosis and pregnancy related complications associated with antibodies targeting phospholipid-binding proteins[1]. The diagnosis of APS requires the presence of at least one clinical event such as thrombosis or pregnancy morbidity along with persistent positivity for antiphospholipid antibodies, which include anticardiolipin antibodies, anti-β2 glycoprotein I antibodies or lupus anticoagulant.[1]

Epidemiology:

According to population-based studies, the estimated annual incidence of antiphospholipid syndrome (APS) in the general population ranges between 1 and 2 cases per 100,000 individuals, while the prevalence is approximately 40 to 50 cases per 100,000 individuals [11]. The presence of antiphospholipid antibodies (aPLs) varies depending on the associated clinical condition. Based on a review of literature, aPLs have been identified in approximately:

- 10% of patients with deep vein thrombosis (DVT),
- 14% of individuals with stroke, and
- 6% to 9% of patients with obstetric morbidity, including complications such as recurrent pregnancy loss and preeclampsia [12].

2. MANAGEMENT

In patients with AntiphospholipidSyndrome (APS) who have elevated levels of antiphospholipid antibodies (aPL), non-pharmacological interventions form the first line of management by focusing on the reduction of additional thrombophilic risk factors[1,2]. Lifestyle modifications such as engaging in regular physical activity, achieving and maintaining a healthy weight, lowering cholesterol levels, avoiding smoking and alcohol should be strongly encouraged. The use of oral contraceptives and hormone replacement therapy should be limited to women who are already receiving anticoagulation therapy[2].

3. CASE HISTORY

A 21 years old Female was admitted in Internal Medicine Unit on October 3 rd 2024 for 14 days with complaints of numbness in left UL for 6 months, neck pain ® side and with the history of headache (3 years) and later the patient was normal for (3 years) back and then presented with the chief complaints of neck pain (R) side and noticed hemolytic transfusion.

His personal medical history showed jaundice at the age of 5 year with recurrent visit for jaundice on OPD bases. The patient had persistent headache and was worsen. Brain MRI showed (L) Thalamic infarct hypoplasia transverse and sigmoid colon, unfortunately the medical reports could not retrieve. The patient had no history of bronchial asthma / CAD / Tuberculosis. Her sleep pattern was apparently normal.

At the time of admission to our clinical unit, physical examination revealed an abnormal heart sound was detected during contraction phase (systolic murmur), presented with splenomegaly and pallor.

Laboratory findings were as follows:

Parameter	Value	Normal Range
Hb	5.7	12-15 g/dl
HCT	20.1	40-54%
MCV	58.4	83-101 fL
MCH	16.6	27-32 Pg
MCHC	28.4	31.5-34.5 g/dl
Platelets	1.45 lakhs/mm3	2-4 x lakhs/mm3
Alkaline Phosphatase	178	40-125 U/L
Protein C	63.8	70-140 %
Free Protein	56.8	60.1-113.6 %
Prothrombin Time (PT) - INR	1.20	0.94-1.15

Total Iron Binding Capacity (TIBC)	489.4	250-450 ug/dL
Lactate Dehydrogenase	353	135-214 U/L
Iron	22	50-170 ug/dL
Reticulocyte count	2.5 %	
Reticulocyte Production Index (RPI)	0.52	

If the RPI value shows less than 2 it indicates the failure of bone marrow of RBC production or hypo proliferative anaemia. Protein Electrophoresis pattern shows increase in total protein and increase in Gamma Globulin which reveals chronic inflammation.

Activated Partial Thromboplastin Time (aPTT) ratio 2.54 (Normal Value - < 1.20)

Red Blood cell, White blood cell (WBC), Monocytes, Basophils, Eosinophils mixed (MXD) neutrophils, C-reactive protein, serum urea, creatinine, bilirubin levels, Rheumatoid factor, D- dimer, Thyroid profile test were all within normal range. Microbiological tests (i.e. HIV I &HIV II, VDRL, HbsAg) were all negative. Peripheral blood smear showed moderate degree of microcytic (smaller than the normal size) and hypochromic (reduced haemoglobin content).

Workup for autoimmune disorder the Lupus Anticoagulant (LAC) was performed with Diluted Russell Viper Venom Time (dRVV) test in two steps (screening and confirmatory); the screening ratio for dRVV 1.44 (0.89-1.10) and the confirmatory test for dRVV 1.12 (0.9-1.07). The lupus anticoagulant was normalised with screening and confirmatory ratio with 1.29 (1.2-1.5) which indicates weakly present, whereas Antinuclear antibody (ANA), Anti cyclic citrullinated peptide (Anti CCP) was negative, indicates no serological evidence of rheumatoid arthritis.

The abdominal ultrasound was normal, except for **splenomegaly**, with the spleen measuring 13.6 cm. A contrast-enhanced CT scan of the whole abdomen confirmed the splenomegaly and revealed a few ill-defined peripheral hypodense areas in the spleen, likely representing **chronic infarcts**. The pelvic region and other abdominal organs appeared normal.

Based on MRI, lumbar spine with whole screening impression showed the mild disc bulge worth small left foraminal profusion causing mild left lateral abscess narrowing and mild abundant of left transversing nerves in the 4 th and 5 th region of lumbar.

Based on multiple previous courses of therapy, the patient was treated with normal blood transfusions, iron, folic acid, and vitamin B12 supplements (in syrup form), vitamin C, aspirin, and Nicoumalone tablets (2 g). For further treatment, the patient was referred to a Rheumatologist and subsequently transferred to another hospital."

Based on medical history, clinical and laboratory features, and confirmed with past clinical criteria such as thalamic infarct and hypoplastic left transverse colon and sigmoid colon which was associated with autoimmune diseases, later diagnosed with primary antiphospholipid antibody syndrome (APLAs) was made.

Based on the patient's medical history, clinical presentation and supporting laboratory findings a diagnosis of primary antiphospholipid syndrome (APS) was established. This diagnosis was made despite the absence of fully established clinical criteria, only after excluding other possible causes and including associated autoimmune diseases.

4. DISCUSSION

APLAs (Antiphospholipid autoimmune syndrome) disease is defined as the immune system is mistakenly attacks the normal components such as phospholipid called proteins and leads to the formation of blood clot in the arteries and veins. This case report discuss about the mechanisms associated with splenomegaly, constipation, hemangioma, mild disc bulging in the lumbar spine for phospholipid molecules.

Mechanisms linking splenomegaly to Antiphospholipid Antibody Syndrome:

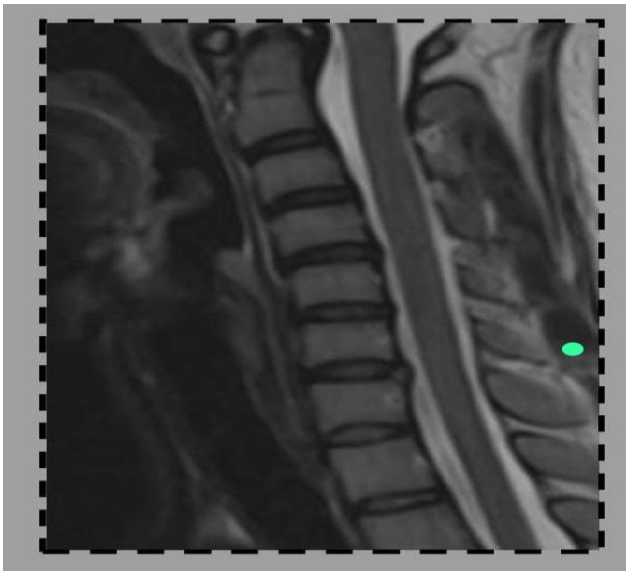
The Antiphospholipid antibody syndrome APS, the levels of substances such as fibrin degradation, microparticles and procoagulants are elevated due to increased coagulation activity, especially after the blood clot. Coagulation activation markers such as fibrin polymer, D-dimer, and thrombin-antithrombin complexes are higher in APLAs patients. Repeated thrombotic events increase the quantity of coagulation activation markers and MPs, intensifying the spleen's workload in clearing these substances and Studies suggest the spleen has intrinsic anticoagulant and fibrinolytic activities, which may be upregulated in response to the prothrombotic state in APS. Regular intake of anticoagulant decreases the level of Microparticles /activation markers and decrease the spleen workload[4].

In essence, splenomegaly in APS is likely due to increase the spleen's activity in clearing prothrombotic substances such as Microparticles and fibrin degradation products and its role in blood clotting process. Effective anticoagulant therapy reduces

the burden and risk of causing splenomegaly[4].

Hemangioma: Patients with antiphospholipid antibody syndrome (APLS) do not exhibit a documented association with the development of hemangioma. However, few case report have observed a low incidence of developing the hemangioma in APLAs patients undergoing with lumbar puncture[2]

Lumbar spine: The exact pathophysiological relationship between epidural venous thrombosis and fatty deposition in the vertebral body is not clear. But we suggest that the prolonged gradual disturbance of the microvascular circulation possibly results in partial epidural venous thrombosis associated with antiphospholipid antibody, together with long-term use of steroid, may have contributed to fatty infiltrates in the vertebral body. The mechanisms look different pattern which is caused by sudden vascular insufficiency.[2]



5. NEOPLASM

B lymphocytes are proteins that play a crucial role in the production of antibodies, which help to modulate and activate the immune system. These antibodies recognize antigens from foreign microorganisms and can potentially trigger the inflammatory cascade [6].

Research has been conducted on patients with cancer regarding the occurrence of positive tests and elevated levels of antiphospholipid antibodies (aPLs), which include anticardiolipin (aCL), lupus anticoagulant (AL) and anti-β2 glycoprotein I (anti-β2 GP I). It has been established that the probability of an increase in these antibodies is particularly predominant in individuals with solid tumours [7].

6. ANTI-CYTOPLASMICNEUTROPHILANTIBODIES

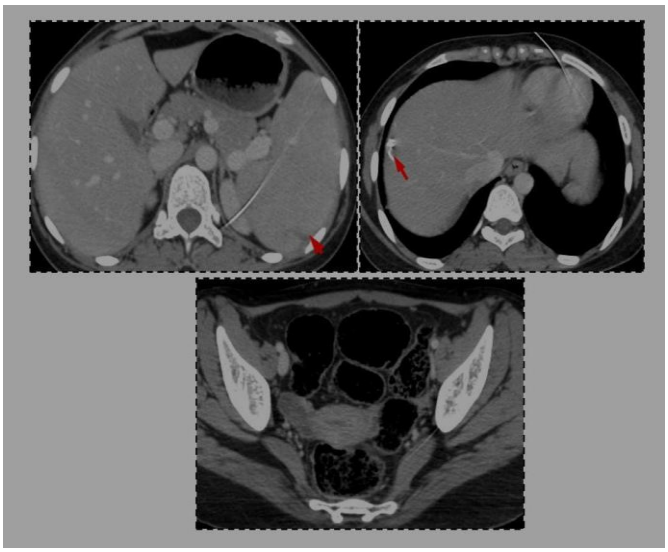
The expression of these antibodies may be associated with both systemic and localized inflammatory responses in cancer. These antibodies target proteins found in neutrophil granules and inhibit the proliferation of microorganisms. Neutrophils form an extracellular structure composed of proteins, DNA, and other elements, known as extracellular neutrophil traps (NETs). Any defects in these structures can trigger immune diseases and contribute to pathogenesis. Furthermore, NETs have been identified as inducers of anti-neutrophil cytoplasmic antibodies (ANCA), leading to mechanisms of chronic inflammation that facilitate cancer progression [3]. These antibodies have been observed in hematological neoplasms, regardless of the presence of autoimmune diseases, as well as in solid tumours such as lung and breast cancer [8]. However, it remains unclear whether cancer alone can induce ANCA production [8].

7. ANA (ANTINUCLEAR ANTIBODIES)

These antibodies have been identified in various neoplasms, which include hematological cancers, as well as pulmonary, colorectal, and breast cancers [9,10]. They are most frequently found in individuals with chronic infectious diseases, healthy individuals, and in connective tissues such as bone, cartilage, and joints [9,10]. In patients with non-Hodgkin's lymphoma and Chronic Lymphocytic Leukaemia (CLL), these antibodies can act as an independent characteristic factor, aiding in the early detection of cancer and the identification of precancerous lesions [3,9].

Furthermore, the necessary use of MRI examination of the lumbar spine region can help in the detection and prevention of

further malignant (tumour) cell development [3].



8. LIMITATION / DRAWBACK

The patient was observed only for first line treatment or therapy and was not treated for anticoagulation therapy. the patient was not aware of the disease and could not able to collect the evidence of past. The treatment facilities for this specific condition were not available at the current hospital. So the patient was recommended to consult a Rheumatologist. Consequently, the patient was referred and transferred to another hospital for specialized care.

9. CONCLUSION

Antiphospholipid antibody syndrome (APLA) is a rare autoimmune condition with limited treatment options and should be suspected when the patient had the symptoms of Splenomegaly, Constipation, Calcified granuloma and the patient who was observed with mild disc bulging in the lumbar region. At initial stage, continues monitoring of patient helps to identify further complication. The complete eradication of the autoimmune disease not yet identified but the complication of this APLA earlier helps for proper treatment and prevent further complication in APLA.

10. PATIENT PERSPECTIVE

I was deeply worried when, just a day before my scheduled check-up, I was unexpectedly diagnosed with **Antiphospholipid Antibody Syndrome**. My father and mother stood by me with continues strength and constantly motivated me to stay positive and fight through this challenging phase of being diagnosed with this autoimmune disorder. Their support gave me the courage to overcome worries and fear.

By sharing my experience, I hope to raise awareness about this lesser-known condition and encourage others to seek timely medical concern if they notice any unusual symptoms.

11. CONFLICT OF INTEREST

There is no conflict of interest.

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