

Case Report

ISOLATED SPLENIC VEIN THROMBOSIS IN A POSTPARTUM PATIENT WITH ANTI-PHOSPHOLIPID ANTIBODY SYNDROME

SWARNAPRIYA.K^{1*}, P.SATHYADHARAN^{2*}, VASANTHA N SUBBIAH³, ANOOP SREEVALSAN⁴

1. ASSISTANT PROFESSOR (OBG), CHETTINAD MEDICAL COLLEGE AND HOSPITAL

2. ASSISTANT PROFESSOR (ORTHO), CHETTINAD MEDICAL COLLEGE AND HOSPITAL

3. PROFESSOR AND HEAD OF THE DEPARTMENT, CHETTINAD MEDICAL COLLEGE AND HOSPITAL,

4. ASSOCIATE PROFESSOR (OBG), CHETTINAD MEDICAL COLLEGE AND HOSPITAL

Corresponding Author: Dr. K.Swarnapriya, Tower 38, Flat no.021, DLF Garden City, Thalambur village, Chemmenchery, near PSBB Millennium School, CHENNAI, TAMILNADU - 603103

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ABSTRACT

Antiphospholipid antibody syndrome (APLAS) accounts for 1-5%¹ of young healthy individual. Among these only 4-14% of them develop venous thrombotic events². Isolated splenic vein thrombosis is a rare entity in postpartum patients with APLAS. We describe here a case of a 27-year old postpartum lady with newly diagnosed APLA which was complicated by isolated splenic vein thrombosis. Although isolated splenic vein thrombosis is a rare entity in APLAS, it should be included in the differential diagnosis of a patient with insidious onset of abdominal pain, especially when risk factors such as pregnancy or the puerperium co-exist. Since delay in the diagnosis can result in significant maternal morbidity, early diagnosis and prompt initiation of anti-coagulant therapy for adequate period is of major importance.

INTRODUCTION

Antiphospholipid antibody syndrome (APLAS) is an acquired thrombophilic entity. Pregnancy and puerperium are itself risk factors for increased thrombo-embolic events. A pregnant patient with APLAS has increased risk of miscarriages, pre-eclampsia and utero-placental insufficiency. Isolated splenic vein thrombosis in such patients has not yet been reported till date. Diagnosing thrombosis of mesenteric or portal system needs high degree of clinical suspicion, since clinical presentation might be confusing. Recent advances in radiological methods like computed tomography (CT) contrast enhanced have revolutionized the diagnosis of such rare entities. Hereby we present one such case of isolated splenic vein thrombosis in a postpartum patient with APLAS.

CASE REPORT:

27 years old, a case of G₃P₂L₀ came for antenatal booking at 10 weeks of gestation. She is married for 3 years, non-consanguineous marriage. Her first pregnancy was spontaneous conception. She had severe IUGR at 32 weeks. She was induced and delivered a

still born baby of 1 Kg vaginally with no anomaly. She also had Gestational Diabetes Mellitus.

She again conceived 3 months after the first delivery. She had pre-eclampsia and was started on medications at 18 weeks of pregnancy. She went in for intra-uterine fetal demise at 27 weeks. She was induced and delivered vaginally. Again fetus didn't have any anomaly. She was then found to have hypothyroidism and was started on T.Thyroxine 100µg OD. Since then she became chronic systemic hypertensive and was started on regular anti-hypertensive medications. Her mother was hypertensive, hypothyroid and diabetic. Father was hypertensive.

She again conceived spontaneously at 1 year after previous delivery. Since she had 2 third trimester fetal demises, she was evaluated for bad obstetric history. Basic blood investigations showed thrombocytopenia (0.65 lakhs/mm³). Activated Partial Thromboplastin Time (APTT) was 34.3 (control 28.7) & Prothrombin Time (PT) was 13.6 (control 12.4). She was later found to have strong positivity for anti-cardiolipin antibodies (IgG = 93.01 U/ml, IgM >300 U/ml). Lupus anticoagulant was negative. Since she was APLA positive, she was started on prophylactic dose of Inj. Low Molecular Weight Heparin (LMWH) 0.6ml s.c OD & T.Aspirin 75mg OD. Her dating and nuchal translucency scans were normal. Anomaly scan, fetal echocardiography and triple screening in 2nd were within normal limits. She developed gestational diabetes mellitus at 28 weeks of pregnancy and was started on Inj.Insulin accordingly. She developed oligohydramnios and had a breech presentation at 34 weeks, hence decided to terminate the pregnancy after administering steroids. She underwent elective lower segment casearian section at 34 weeks and delivered a live pre-term baby of 2 kg with Apgar 9/10 at 5 minutes. Post-operatively she was started on prophylactic dose of Inj.LMWH 0.6ml s.c OD as per RCOG 2004 green top guidelines, after 24 hours and was continued for 5 days. Post-operative period was uneventful. Baby and mother were discharged at 9th post-operative day.

Patient returned to casualty on post-operative day 20th with high grade fever, vomiting and pain in left hypochondrium for 2 days. On examination she was febrile, had tachycardia, tenderness and guarding in left hypochondrium without abdominal distension. Tender splenomegaly was present. Bowel sounds were normal. Breast was soft. Bimanual pelvic examination showed uterus 6 weeks and a healthy lochia. She was admitted and evaluated for the same. Her platelet count was 1.12 lakhs/mm³, APTT was 28.3 (control = 27.2), PT was 17.8 (control = 13.4), INR was 1.0. ESR was 103 mm/1 hr, CRP was 19.2 (positive). Blood culture was negative. She was started on T.Chloroquine suspecting clinical malaria. Later on ultrasound abdomen revealed an altered area of echogenicity in interpolar region of spleen. Soon patient was started on therapeutic dose of Inj.LMWH 0.6ml s.c BD and T.Warfarin 1mg OD with suspicion of portal or splenic vein thrombosis on post-operative day 21st. CECT abdomen confirmed splenic infarct with partially thrombosed pseudoaneurysm in splenic hilum. In the meantime patient's condition also improved dramatically. Patient was then discharged with only T.Warfarin 2mg OD to maintain INR at 2.0 on post-operative day 28th. During regular follow-up after 2 weeks, her INR was 2.0. She was asked to continue T.Warfarin 2mg OD for 6 more weeks and then stop. After 11 months post-delivery, patient was asymptomatic with no specific complaints.

Discussion:

Pregnancy and puerperium are well recognized as a hypercoagulable states. However venous thromboembolism remains a rare entity during pregnancy. But patients with APLAS are more prone for venous thromboembolism (VTE) during pregnancy and puerperium like this case. Hence clinical presentations of VTE should be kept in mind when a patient with APLAS presents with fever and localizing signs.

Isolated splenic vein thrombosis is most frequently considered as a complication secondary to pancreatic disease³. Usually splenic vein thrombosis is suspected only in 1) patients with history of pancreatitis and gastrointestinal haemorrhage 2) patients with splenomegaly without any liver diseases 3) in isolated gastric varices⁴. Splenic vein thrombosis is mostly asymptomatic. Variceal bleeding often is the first clinical manifestation of isolated splenic vein thrombosis. This patient neither had any history of pancreatitis, nor any liver diseases nor presented with gastrointestinal haemorrhage. Hence splenic vein thrombosis was not kept as first diagnosis. So, she was started on anti-malarials and investigated for other infectious causes.

Symptomatic splenic vein thrombosis can also present with abdominal pain, nausea and fever. Symptoms depend upon the extent of thrombosis involving mesenteric veins or when infarct ensues in spleen. This patient presented with such complaints. Altered echogenicity in the spleen revealed by ultrasound with associated APLAS lead to suspicion of venous thrombosis of portal system. Negative blood culture, negative smear study for MP/MF, along with borderline changes in APTT, PT further pointed towards non-infectious cause. CECT confirmed that it was isolated splenic vein thrombosis with infarct. Further, clinically patient's general condition improved immediately after starting on Inj.LMWH and T.Warfarin.

Similar case has also been reported in which mesenteric vein thrombosis has occurred in a low risk postpartum patient without APLAS, even after 5 days of prophylactic low molecular weight heparin (RCOG 2004 guidelines)⁵.

Conclusion:

Diagnosing venous thrombo-embolism in splenic, portal or mesenteric system is a challenge. High degree of clinical suspicion is needed to diagnose such complications. Even though isolated splenic vein thrombosis is very rare, it should always be kept as a part of differential diagnosis in all post-partum patients with left hypochondrial pain and fever, especially when risk factors like APLAS co-exist. Ultrasound abdomen is helpful in suspecting such conditions. But computed tomography (CT) with or without contrast appears to be the best non-invasive investigation in diagnosing such cases. Low molecular weight heparin remains treatment of choice in such cases, later on switching to oral anti-coagulants. Our patient was on oral anti-coagulant for nearly 8 weeks.

This show that even in APLA patients with no previous history of any thrombo-embolic events, like our case, 5 days prophylactic LMWH may not be sufficient.

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