

APLA Syndrome in Pregnancy and its Outcome: A Case Report

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Case Report

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Abstract

Antiphospholipid antibody syndrome is a heterogenous autoantibody mediated acquired thrombophilia, which is associated with severe life threatening complications during pregnancy and most important treatable cause of recurrent pregnancy loss. It was described in 1980s by E Nigel Harris and Aziz Gharavi. It is also called as Hughes syndrome. Clinician should have high index of suspicion of APLA in patient with history of recurrent abortions, Intra uterine death with unexplained cause. We are presenting a case of 27yr old G2A1 with 38 wks. of POG with history of pulmonary thromboembolism and pulmonary hypertension and deep vein thrombosis 2 years ago, she was diagnosed with APLA syndrome. Patient was followed up to term and she was asymptomatic throughout the pregnancy. She had full term normal delivery. Both mother and baby were stable and healthy. She was asymptomatic in her postpartum period.

Keywords: APLA Syndrome; Pulmonary Thromboembolism; Recurrent Abortions; Low Molecular Weight Heparin; Full Term Normal Delivery

Introduction

Antiphospholipid Syndrome (APS) is an autoimmune disorder of hypercoagulable state that is marked by the presence of antibodies that attack phospholipid-binding proteins, characterized by vascular thrombosis and pregnancy complications especially recurrent spontaneous miscarriages and, less frequently, maternal thrombosis (Table 1). A modified Sapporo criterion is used to diagnose APLA syndrome [1-4].

Clinical criteria	Lab criteria	
1. ≥1unexplained deaths of morphologically normal foetus ≥ 10wks	1. Lupus Anticoagulant	
2. Severe preeclampsia& delivery ≤34wks	2. Medium/High IgG/IgM anticardiolipin anti- bodies	
3. ≥3 unexplained consecutive spontaneous abortions before 10 wks	3.Anti β 2 gylcoprotein antibodies	
$4. \ge 1$ episodes of arterial, venous/small vessel thrombosis in any tissue / organ		

Case Report

A 27 year old, G2A1 with 38weeks of POG gives H/o polycystic ovarian syndrome treated with OCPs for 3 months, which triggered pulmonary embolism. She had syncopal attacks 2 years ago, 2D ECHO showed pulmonary hypertension and CT pulmonary angiogram done and diagnosed to have pulmonary thromboembolism, started on Tab. Acitrom for 1 year. Next year she developed right lower limb DVT On further evaluation she was found to be having APLA syndrome, Hyperhomocysteinemia, Protein C and S deficiency and continued anticoagulant therapy. Patient had spontaneous abortion at 2MOA 1year ago. Pre conception counselling done. Patient presented with pregnancy test positive in first trimester and followed up till term. Patient was asymptomatic throughout the present pregnancy. She was on Tab. Ecosprin 75mcg and Inj Dalteparin 5000IU OD daily from first trimester. 2DECHO, Lower limb Doppler were normal. PT, APTT, INR were monitored. Patient was admitted at 38 weeks with h/o decreased fetal movements and oligohydramnios. After taking cardiology opinion and Preanesthetic evaluation, LMH was changed into unfractionated heparin 5000IU IV tid and it was stopped 12 hours before planning for delivery. Induction of labour done with dienoprostone gel, patient had an uneventful full term vaginal delivery of female baby of 2.9kg. Both mother and baby were healthy. Inj Heparin 5000IU IV and Tab Acitrom

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2mg OD were restarted after 24hours of delivery for 5days, serial coagulation profile monitored. After five days Inj Heparin stopped and Patient discharged with Tab Acitrom 2mg OD. With PT 11, APTT 26, INR 0.9.

Discussion

APLA is defined by presence of thromboembolic complications and pregnancy morbidity in the presence of increased titer of antiphospholipid antibodies. APLA syndrome without any underlying disease is termed as primary APLA syndrome and secondary antiphospholipid antibody syndrome is associated with SLE. Antiphospholipid reduce hCG release and inhibit trophoblast invasion which may explain miscarriages and fetal loss in the second trimester is associated with severe growth restriction, oligohydramnios and early onset preeclampsia possibly due to abnormal placentation related to thrombosis (Figure 1). Abnormalities in decidual spiral arteries like narrowing, intimal thickening, acute arthrosis and fibrinoid necrosis may be the immediate cause of fetal loss. Some authors state that antiphospholipid activates endothelial cells and complement system and hence cause pregnancy loss. Glycoprotien-1 triggers coagulation and inhibits ant thrombin 3 and fibrinolysis leading to thrombosis [5,6].



Figure 1: Presence of thromboembolic complications and pregnancy.

Clinical Features

- Venous thromboembolism (DVT or PE)
- Arterial thrombosis, infarcts & Stroke
- Recurrent miscarriage

- Severe pre-eclampsia & IUGR
- Placental abruption & Intrauterine demise
- Livedo reticularis (esp with lupus)
- Rare –Thrombocytopenia Haemolytic anaemia
- Catastrophic APS: rapid onset thrombosis, multiorgan

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dysfunction with a systematic inflammatory response. Death occurs in 50% of patients.

Management of APS during pregnancy should be aimed to avoid early pregnancy loss, normalize placental and

fetal circulations to prevent early birth from preeclampsia and growth restriction and to prevent maternal vascular thrombosis in pregnancy and postpartum. Using appropriate treatment strategies, the likelihood of successful pregnancy in APS is about 70% (Table 2).

	Management of APS during Pregnancy		
	Counsel regarding risks of APS	Counsel regarding risks of APS	
1.Pre pregnancy	Discuss anticoagulation prophylaxis		
	Transition from warfarin to low molecular- weight heparin in patients with thrombosis history		
	Provide multidisciplinary care		
	Prenatal visits every 2-4weeks until 20-24 weeks and every 1-2weeks thereafter		
	Initiate fetal surveillance at 32weeks		
	Anticoagulation prophylaxis ⁵		
	APS without prior thrombosis	APS with thrombosis	
2. Antenatal	Recurrent pregnancy loss/ fetal death or early delivery <34weeks due to severe preeclampsia :		
	Aspirin (75- 150mg) acts by preventing thrombosis & damage to trophoblast.	Low-molecular-weight heparin Eg: enoxaparin 1mg/ kg 12hourly with monitoring of anti-Xa activity	
	Unfractionated heparin (5000IU BD) subcutaneous		
	Low-molecular-weight heparin :Enoxaparin 40 mg sc OD		
	Only antibodies + no clinical features :	-	
	Aspirin only		
3.Labour and delivery	Avoid postdates		
	Continuous FHR monitoring		
	Stop anticoagulation 12-24 hours before planned induction/ caesarean section		
	Arrange blood and blood products		
	Active management of third stage of labour and follow PPH protocols		
	ICU availability		
	Compression stockings		
4. Postnatal	Resume anticoagulation 6hours after vaginal delivery and 12 hours after caesarean section		
	Continue anticoagulation for 6weeks for women with no prior thrombosis and for lifelong in women with prior thrombosis		
	Warfarin is substituted for heparin		

Table 2: Management of APS during pregnancy.

Conclusion

Clinician need to be more vigilant and should have high suspicion of APLA in patient with recurrent abortions. Resources for detection of APLA should be made readily available in resource limited settings. Management of APLA involves improving maternal and fetal outcomes, prevention of thrombosis with close monitoring of patient on anticoagulant could be challenging.

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