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CASE REPORT ON A SUCCESSFUL PREGNANCY OUTCOME OF ANTIPHOSPHOLIPID ANTIBODY SYNDROME

Gynaecology

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ABSTRACT

Antiphospholipid syndrome is an auto immune disorder resulting in a hypercoagulable state due to involvement of anticardiolipin , lupus anticoagulant & anti-β2 glycoprotien autoantibodies.

APLA is defined by presence of thromboembolic complication & pregnancy morbidity in presence of increased titre of antiphospholipid antibodies.

Antiphospholipid syndrome is classified as primary or secondary depending on its association with other auto immune disorders

Primary Antiphospholipid syndrome is diagnosed in patients demonstrating the clinical & lab. criteria for disease without recognizing auto immune disease.

Secondary Antiphospholipid syndrome is diagnosed in patient with other auto immune disease like SLE

Management: such patients are managed with low molecular weight heparin it is started in early gestation & continue till 34 weeks

KEYWORDS

fetal death, lupus antibody, thrombosis, vasocclusive event

INTRODUCTION

Antiphospholipid syndrome is an auto immune disorder resulting in a hypercoagulable state due to involvement of anticardiolipin , lupus anticoagulant & anti-β2 glycoprotien autoantibodies.

APLA is defined by presence of thromboembolic complication & pregnancy morbidity in presence of increased titre of antiphospholipid antibodies.

Primary antiphospholipid syndrome is diagnosed in patients demonstrating the clinical & lab. criteria for disease without recognizing auto immune disease.

Such patients are managed with low molecular weight heparin it is started in early gestation & continue till 34 weeks.

In this case report we present a case of primary antiphospholipid syndrome in young woman who had previous one full term unexplained intrauterine death & one spontaneous abortion of gestation age 10 week.

CASE REPORT

We here present a case of primary antiphospholipid syndrome in young woman who had previous one full term unexplained intrauterine death & one spontaneous abortion of gestation age 10 week.

A 27 year old female with 9 months amenorrhoea presented with pain abdomen & decreased fetal movement. She had a spontaneous abortion of 10 week ,1 year back.She had also intra uterine death at term pregnancy 2 year back.

Before conception she visited a gynaecologist. Her routine investigation were done including complete blood count, liver function test, glucose tolerance test, which were also normal.

In view of previous spontaneous abortion & unexplained intra uterine death, her complete anti nuclear antibody profile was done, she was strongly positive for β2 glyco protein IgM antibody with values 25.35(control<25). She was diagnosed as primary antiphospholipid antibody syndrome & put on aspirin 75 mg once a day & inj. enoxaparin 40IU sc once a day, during complete pregnancy. She was counselled about the maternal and fetal risks. She conceived in next month & than all routine blood investigations & ultra sonography were done. Patient was very regular regarding her antenatal visits timely. After completing 35 weeks of pregnancy aspirin & enoxaparin were withdrawn. At 37 weeks patients routine investigations including coagulation profile PT, APTT & INR. All investigation were normal & she was taken for elective cesarean section. She delivered a healthy baby boy of 2.7 kg. Her blood picture was normal during antenatal check up & post natal period.

DISCUSSION

The occurrence of antiphospholipid antibody syndrome associated with vaso occlusive events without any underlying disease process is termed the primary antiphospholipid antibody syndrome. The clinical criteria for its diagnosis include evidence of thrombosis like peripheral gangrene secondary to venous, arterial or small vessel thrombosis .Repeated fetal loss before 10 week or unexplained after 10 week.

Lab. Criteria include presence of anticardiolipin antibodies (IgG or IgM isotype in medium to high titre) lupus antibody identified twice, at least 6 weeks apart prolong APTT.

Obstetric features of antiphospholipid antibody syndrome:-

- *Unexplained fetal death or still birth*
- *Recurrent pregnancy loss-3 or more spontaneous abortion with no more than one live birth*
- *Unexplained 2nd or 3rd trimester fetal death*
- *Severe pre ecclampsia at less than 34 week gestation*
- *Unexplained severe IUGR*
- *Chorea gravidarum*

Despite the name lupus anticoagulant antibodies are associated with thromboembolic events rather than clinical bleeding. Antiphospholipid antibodies can interfere with both pro & anticoagulant pathways. In vitro phospholipid surfaces inhibit pro coagulant pathway so prolong clotting .In vivo cell membrane promotes greater inhibition of anticoagulation pathways so resulting in thrombosis. Activation of endothelial cells by intraction with β2

GLYCOPROTIEN-I triggering coagulation pathways coupled with inhibition of antithrombin-III , activated protein-C, inhibition of fibrinolysis & interference with tissue factor& thrombin promotes thrombosis

Annexin A5 binds to phospholipid membrane to prevent cell membrane from initiation of activation of coagulation. Annexin V is present abundantly in intervillous space of placenta. If autoantibodies displace Annexin A5, procoagulant endothelial cell surfaces may be exposed & provide arterial & venous thrombosis & thrombosis in intervillous spaces.

The most commonly detected subgroups of antiphospholipid antibodies are β2 glycoprotien-I , lupus anticoagulant & anticardiolipin antibodies .Deep vein thrombosis of legs is most common manifestation. Arterial thrombosis are less common& most frequently manifest with features consistent with ischemia or infarction. The brain is most common site accounting for 50% of arterial occlusion .Rarely can involve peripheral arteries causing gangrene.

Other common manifestation of antiphospholipid antibody syndrome include thrombocytopenia & hemolytic anemia. Pregnancy in APLA patients presents with increased risk of fetal loss. Multiple infarction of the placenta due to microthrombi is a frequent finding, if it is extensive may cause severe growth retardation of fetus leading to repeated pregnancy losses. Toxaemia of pregnancy is also commonly seen in such patients since pregnancy is hypercoagulable state. Presence of APLA can precipitate thromboembolic events at anytime of pregnancy or during post partum period. With availability of safer & efficient anticoagulation successful pregnancy can be planned in patient with APLA. Low molecular weight heparin is anticoagulant of choice in the treatment of pregnant women with APLA syndrome.

CONCLUSION

Clinician need to be more vigilant & should have high index of suspicion of APLA in patient with recurrent abortions, preterm delivery& in patients with term IUD with unexplained cause. Clinician should investigate for the presence of antiphospholipid antibodies as early diagnosis & treatment will definitely improve the fetal outcome & thus will improve maternal physical and mental status. Furthermore , resources for detection of antiphospholipid antibodies should be made readily available in resource limited settings so as to test patients with history of thrombosis or pregnancy complication.

Management of antiphospholipid syndrome involves improving maternal and fetal outcome, prevention of thrombosis with close monitoring of patient on an anticoagulant could be quite challenging. Patient education on importance of drug compliance, periodic monitoring& prevention of thrombosis is indispensible , especially as mortality could be associated with effects of vascular thrombosis &effects of bleeding due to anticoagulant.

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