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TRANSIENT ISCHEMIC ATTACK IN PREGNANCY DIAGNOSED AS PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

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Abstract : We report the case of young primiparous women with Transient ischemic attack diagnosed as Primary antiphospholipid antibody syndrome. Lupus anticoagulant and anticardiolipin antibodies were elevated. There was no clinical or laboratory evidence for other autoimmune or systemic illnesses. We are presenting the case due to the rarity of the same.

Keyword :APLA, TIA, Lupus anticoagulant, Anticardiolipin antibodies

INTRODUCTION:

Antiphospholipid antibodies are a heterogenous group of autoantibodies directed against negatively charged phopholipids or phospholipid binding proteins. Primary antiphospholipid syndrome occurs in patients without clinical evidence of another autoimmune disease. Whereas secondary antiphospholipid syndrome ocurrs in association with other autoimmune disease.

CASE REPORT:

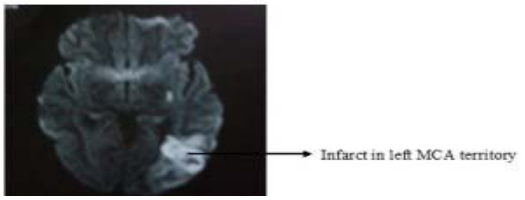
25years pregnant primiparous women with 33 weeks of gestation presented to our OPD with history of giddiness, slurred speech and weakness of her right upper and lower limbs for 10 minutes duration. She had similar complaints one week back, lasted for 15 minutes without loss of consciousness. She did not have dysphagia, diplopia or bladder and bowel disturbances. Past medical history was unremarkable, and there was no significant family history suggestive of hereditary neurological condition. On examination she was conscious, oriented, grade 4 power of Right upper and lower limbs and dysarthria present. As the patient presented as Transient ischemic attack we considered the possibility of a hypercoagulable state.

INVESTIGATIONS:

Investigation revealed the following findings: Blood routine, urine routine, serum electrolytes, renal, liver and thyroid function, ECG were normal. ECHO was normal study. Platelet count was 2.5 lakhs. Lipid profile was: Total cholesterol 288, Triglycerides 527, HDL 59, VLDL 105. Prothrombin time 14 seconds, Activated partial thromboplastin time 28 seconds, INR 1.3.

MRI Brain and MR Angiogram:

1] Early subacute infarcts involving left MCA territory and left MCA-PCA transformation zone. No haemorrhagic transformation / mass effect seen. 2] M1 segment of left MCA narrowed in caliber with poorly visualized M2 and M3 segment ? Thrombosis. 3] Normal flow void is seen in the major dural venous sinuses. 4] No evidence of any intracranial hemorrhage. Doppler study of 4 neck vessels normal. Foetal doppler study was normal.



ANA, Anti ds DNA, ENA profile and VDRL tests were negative.

Antiphospholipid antibody titres were as follows:

1] Lupus anticoagulant test were done which was prolonged by using a PTT and diluted Russel Viper Venom Time

2] Anti cardiolipin antibodies are measured by ELISA method which was (45 MPL units > 99th percentile)

3] Anti beta 2 glycoprotein 1 antibodies - Negative Procoagulant workup including protein s, protein c, antithrombin, urine homocysteine assays were normal. Factor V leiden mutation was not done

MANAGEMENT AND OUTCOME:

Based on the clinical and laboratory findings we made a definite diagnosis of primary APLA syndrome. We started her on low molecular weight heparin ENOXAPARIN 0.6mg s.c bd and T.ASPIRIN 300mg stat followed by 150mg once a day. Biweekly non-stress test and AFI measured. On 37 weeks 6 days she had persistent decreased foetal movements heparin was stopped for 24 hrs then emergency LSCS was done . She delivered on alive term boy baby of wt 3.1kg with good Apgar. 1unit of packed red blood cell and 4 unit of FFP transfused intraoperatively. Heparin restarted after 12 hrs. Heparin was switched over to oral anticoagulant on 5th post operative day INR maintained between 2-3. Post operative period was uneventful. Oral anticoagulant continued till 6 weeks postpartum. IgM anticardiolipin antibody was repeated after 12 weeks and it came out to be positive (42MPL units > 99th percentile), Lupus anticoagulant test was negative after 12 weeks.

DISCUSSION:

Antiphospholipid antibody syndrome (APLA, APS, Hughes syndrome) is characterized by thrombosis or fetal loss in association with presence of lupus anticoagulant [LA] or anti cardiolipin antibodies. Important obstetric criteria include recurrent pregnancy loss, fetal death, severe preeclampsia or placental insufficiency requiring delivery prior to 34 weeks gestation. Pregnancy complications due to APS are due to abnormal placental function. This results in narrowing of the spiral arterioles, intimal thickening, acute atherosis and fibrinoid necrosis. Extensive necrosis, infarction and thrombosis are seen in the placenta. Venous thrombosis, especially deep venous thrombosis of the legs, is the most common manifestation of the antiphospholipid syndrome occurring in 29 to 55 percent of patients with the syndrome during an average follow-up of less than six years.1 Arterial thromboses are less common than venous thromboses and most frequently manifest with features consistent with ischemia or infarction.1 The brain is the most common site, with strokes and transient ischemic attacks accounting for almost 50 percent of arterial occlusions.2 Other prominent manifestations of the antiphospholipid syndrome include thrombocytopenia (in 40 to 50 percent of patients), haemolytic anaemia (in 14 to 23 percent), and livedo reticularis (in 11 to 22 percent).1 Since pregnancy itself is a hypercoagulable state presence of aPL antibodies can precipitate any of the above mentioned complications at anytime, either during pregnancy or during postpartum state.3 So in short, suspect the presence of aPL antibodies when there is clinical symptoms such as DVT, arterial occlusive events, recurrent fetal loss, TIA etc. Gestational hypertension/preeclampsia occurs in about 30% of APS pregnancies. Preeclampsia is usually more severe in APS pregnancies.4 aPLs frequently cause IUGR in almost one in three pregnancies with APS. Preterm birth rates are high, ranging from 30-60% in APS pregnancies. APS related pregnancy loss could be an early recurrent pregnancy loss. Usually it is an embryonic fetal loss. International consensus statement on preliminary criteria for the classification of antiphospholipid antibody syndrome is as follows.

Revised Sapporo Criteria for the Diagnosis of Definite Antiphospholipid Syndrome

APS is present if at least one of the clinical and one of the laboratory criteria that follow are met

CLINICAL:

· A documented episode of arterial, venous, or small vessel thrombosis — other than superficial venous thrombosis — in any tissue or organ by objective validated criteria with no significant evidence of inflammation in the vessel wall, and/or

· 1 or more unexplained deaths of a morphologically normal fetus (documented by ultrasound or direct examination of the fetus) at or beyond the 10th week of gestation and/or 3 or more unexplained consecutive spontaneous abortions before the 10th week of gestation, with maternal anatomic or hormonal abnormalities and paternal and maternal chromosomal causes excluded or at least 1 premature birth of a morphologically normal neonate before the 34th week of gestation due to eclampsia or severe pre-eclampsia according to standard definitions, or recognized features of placental insufficiency *plus*

LABORATORY:

· Anti-cardiolipin IgG and/or IgM measured by standardized, non-cofactor dependent ELISA on 2 or more occasions, not less than 12 weeks apart; medium or high titre (i.e., > 40 GPL or MPL, or > the 99th percentile) and/or

· Anti-2 glycoprotein 1 IgG and/or IgM measured by standardized ELISA on 2 or more occasions, not less than 12 weeks apart; medium or high titre (> the 99th percentile) and/or

· Lupus anticoagulant detected on 2 occasions not less than 12 weeks apart according to the guidelines of the International Society of Thrombosis and Hemostasis. At present the therapeutic prevention of obstetric accidents related to APAS saves 70% of survival births compared to 10% without treatment [5]. Several treatments were studied, in particular aspirin, heparin, prednisone, immunoglobulin and plasmapheresis. However, most authors indicate the association of aspirin and heparin at low molecular weight [5-12]. Indeed, this association therapy allows reducing the foetal loss by 54%. Actually, most studies have shown that the best therapy of APAS is offered by aspirin (60 to 100 mg) and heparin of LMW (an injection a day in preventive dose). Prednisone are suggested only if there is an extra-obstetrical [5] or inflammatory system disease, this without exceeding 20 mg/day. Immunoglobulins generate a solubilisation of the immune complexes. However, a randomized study didn't show any profit of using IgG compared a placebo in patients treated by aspirin and heparin, particularly on the rate of children survival and the prematurity [13]. Plasmapheresis wasn't studied in the treatment of APAS during the pregnancy. Besides these treatments, an obstetric care based on the close observation is essential.

REFERENCES

[1] Jerrold SL, Branch DW, Rauch. The antiphospholipid syndrome. N Engl J Med 2002; 346:752-62.

[2] Ameri A, Bousser M. Cerebral venous thrombosis. Neurol Clin 1992; 10:87-111.

[3] Barry L Myons, Deborah Mccurdy. APLA syndrome, eMedicine update, Sept. 4, 2002.

[4] Moodiey J, Bhoola V, Duursma J, et al. The association of antiphospholipid antibodies with severe early onset preeclampsia. S Afr Med J. 1995; 85:105-107.

[5] Meyer, O. (2003) Lupus et autres connectivites et vie hormonale. Gynécologie Obstétrique & Fertilité, 31, 746- 756. Doi: 10.1016/S1297-9589(03)00203-0

[6] Carbonne, B. and Lejeune, V. (2003) Modalités thérapeutiques d'une maladie abortive d'origine dysimmunitaire. Gynécologie Obstétrique & Fertilité, 31, 789-793. Doi: 10.1016/S1297- 9589(03)00210-8

[7] Arvieux, J. and Hachulla, E. (2002) Le syndrome des antiphospholipides. Annales de Cardiologie et d Angé- iologie, 51, 146-151. doi:10.1016/S0003-3928(02) 00087-2

[8] Firkin, B.G., Howard, M.A. and Radford, N. (1980) possible relationship between lupus inhibitor and recurrent abortion in young women. Lancet, 2, 366. Doi: 10.1016/S0140-6736(80)90361

[9] Lejeune, V. (2006) Fausses couches spontanées précoces répétées: Quelleprise encharge proposer en 2006? Gyné- cologie Obstétrique & Fertilité, 34, 927-993. doi:10.1016/j.gyobfe.2006.08.008

[10] Nielsen, H.S. and Christiansen, O.B. (2005) Prognostic impact of anticardiolipin antibodies in women

with recurrent miscarriage negative for the lupus anticoagulant. Human Reproduction, 20, 1720-1728. doi:10.1093/humrep/deh79.
[11] Wilson, W.A., Gharavi, A.E. and Koike, T. (1999) International consensus statement on preliminary classification criteria for definite antiphospholipid syndrome. Arthritis & Rheumatism, 42, 1309-1311. Doi: 10.1002/1529-0131(199907)42:73.0.CO; 2-F
[12] De Maistre, E. (2003) Anticorps dits antiphospholipides et troubles de la reproduction: Les données biologiques. Gynécologie Obstétrique & Fertilité, 31, 794-796. Doi: 10.1016/S1297- 9589(03) 00207-8
[13] Branch, D.W., Peaceman, A.M., Druzin, M., et al. (2000) Multicenter, placebo-controlled pilot study of intravenous immune globulin treatment of antiphospholipid syndrome during pregnancy. The Pregnancy Loss Study Group. American Journal of Obstetrics & Gynecology, 122-182. doi:10.1016/S0002-9378(00)70500-X

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