

Protein S Deficiency Causing Poor Outcome of Pregnancy – A Prospective Follow up

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Abstract

It has become evident in the past few years, that a large number of obstetric pathologies are actually associated with congenital thrombophilias. Many authors relate this factor with preeclampsia, intrauterine fetal growth retardation, spontaneous abortion, unexplained cases of still birth, placental abruption, and thromboembolic complications during and after parturition.

We report one case of fetal loss due to severe preeclampsia with uncontrolled post partum hypertension was diagnosed with protein s deficiency in the post partum period.

CONCLUSION: Women with hypertensive disorder in pregnancy are to be thoroughly evaluated to find the etiology so as to improve the management and also prognosis for the woman and the fetus.

KEYWORDS: protein s deficiency, pregnancy, preeclampsia, IUGR , LSCS, Heparin, inherited thrombophilia

Introduction:

Protein S is a non-enzymatic and vitamin K-dependent cofactor of activated protein C. Without protein S, the anticoagulant function of protein C is almost depleted and thrombotic events occur.¹ In women from symptomatic families these defects may be associated with an increased risk of venous thrombosis in pregnancy and recurrent fetal loss. Assuming that fetal loss is due to placental thrombosis, anticoagulant treatment might improve pregnancy outcome. Inherited thrombophilia is common and appears to be a multigene disorder. The thrombotic risk would seem to be greatest in women with antithrombin deficiency and more than one thrombophilia defect. In pregnancy, heparin is the anticoagulant of choice.² In women from symptomatic families with antithrombin deficiency, adjusted dose heparin throughout pregnancy is recommended and warfarin for at least 3 months post partum.

We report one case of fetal loss due to severe preeclampsia with uncontrolled post partum hypertension ,was diagnosed with protein s deficiency in the post partum period.

Case report:

A 28 year primigravida with 30 weeks period of gestation presented to us with severe preeclampsia,with severe oligohydramnios,abnormal fetal doppler indices was taken up for caesarean section and 820g of baby was delivered ,baby expired on day 2. As she had uncontrolled post partum hypertension, she was evaluated and diagnosed to have decreased protein s levels of 18% , normal renal doppler, auto antibody profile ,

normal protein c activity of 125%, and all other secondary hypertension causes were found to be normal. Her hypertension was further controlled with antihypertensives and warfarin.

DISCUSSION:

It has become evident in the past few years, that a large number of obstetric pathologies are actually associated with congenital thrombophilias. Many authors relate this factor with preeclampsia, intrauterine fetal growth retardation, spontaneous abortion, unexplained cases of still birth, placental abruption, and thromboembolic complications during and after parturition.³ Protein S is a cofactor of protein C which lowers the activated factors VIII and V. Pregnancy reduces the level of protein S to 40-50% of normal levels but it is not clear whether the lowered protein S levels increase the risk of developing thrombo-embolism during pregnancy.⁴ In assessing thrombotic risk in pregnancy, acquired risk factors as well as genetic predisposition should be considered.² Increasing age, obesity, immobility, and delivery by cesarean section are major risk factors. Pregnancy should be planned, and each patient should be managed on an individual basis. In pregnancy, heparin is the anticoagulant of choice, and as far as possible, treatment with warfarin should be avoided because of the risks to the fetus.² When patients are on long-term treatment with warfarin, pregnancy should be avoided, and warfarin should be discontinued prior to embarking on a pregnancy or as soon as pregnancy is suspected and before 6 weeks' gestation. Women diagnosed with hypertensive disorder in pregnancy are to be not only evaluated for the severity and progression of the disease but also for the etiology of the disease. When the cause for the disease is identified, the management will be more accurate and also the prognosis for fetus.

CONCLUSION:

Women with hypertensive disorder in pregnancy are to be thoroughly evaluated to find the etiology so as to improve the management and also prognosis for the woman and the fetus.

Preconceptional counselling and evaluation should be advised for women with history of thromboembolism, and inherited thrombophilias. Management of these women with anticoagulants and constant follow up can improve the pregnancy outcome.

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