CORTICAL VENOUS THROMBOSIS IN A CASE OF PREECLAMPSIA

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ABSTRACT

A 23 Year old primigravida presented to the Obgyn out patient department with severe early onset fetal growth restriction and oligohydramnios with persistent diastolic notch in the uterine artery. Patient subsequently went on to develop full blown early onset severe preeclampsia which further manifested itself in the post partum period as generalized clonic tonic convulsions due to cerebral hemorrhage & infarction with cortical venous thrombosis. This is a rare but dreaded complication of preeclampsia. This late onset eclampsia was then managed conservatively with anticonvulsants and thrombolytic agents.

Key words : *pre-eclampsia, postpartum period, venous thrombosis, intrauterine growth retardation, oligohydramnios, case reports.*

INTRODUCTION

Preeclampsia a pregnancy specific syndrome clinically manifesting as elevated blood pressure and proteinuria is one of the major contributors of maternal morbidity and mortality of pregnant mothers in the developing countries.(1,2)

World wide, 50,000 women die each year from preeclampsia and it still remains the most significant and intriguing unsolved problems in obstetrics. (3) Cerebral lesions have rarely been demonstrated with preeclampsia but more commonly associated with eclampsia,range from cerebral edema, hyperemia, ischemia, thrombosis to hemorrhage (ranging from petechiae to gross bleed). These lesions may be wide spread, focal and seldom fatal. Cortical cerebral venous thrombosis is a rare but dreaded complication of preeclampsia and this case is reported for its rarity and dramatic response to therapy.

CASE REPORT

A 23 years old primigravida presented to the Department of OBGYN in October 2006 with the anomaly scan at 23 weeks showing features suggestive of intrauterine growth restriction (symmetrical) with all fetal parameters below 5th percentile with severe oligohydroamnios. The Doppler study showed features suggestive of utero placental dysfunction and the uterine artery had persistant diastolic notch and the umbilical artery had absent diastolic flow. There were no obvious identifiable congenital anomalies in the fetus.

On clinical examination, the patient was moderately built with normal blood pressure 120/80mmHg Systemic examination was within normal limits. On abdominal examination there was a lag of > 4 wks with grossly decreased liquor with fetal heart rate of 156/min by Doppler.

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In view of severe early onset IUGR with oligohydroamnios and uterine artery showing persistent diastolic notch (which is a very sensitive marker for prediction of preeclampsia -78% sensitivity),the patient was explained regarding the poor fetal prognosis and possible maternal complications of continuing the pregnancy and counselled for termination. However the patient decided to continue the pregnancy and was lost in follow up. Subsequently, in the next 3 weeks she developed severe preeclampsia with uncontrolled blood pressure, for which termination of pregnancy was done. She expelled a dead fetus(weighing 300 gm with no obvious external congenital anomalies in the foetus and placenta showed no obvious external areas of infarction or calcification). After delivery and discharge patient did not go for postnatal check up and presented to the Emergency department of SRMC & RI with an episode of generalized tonic clonic seizures on the 10th post natal day. The seizures were preceeded by a headache (temporoparietal) for 3 days. The patient was immediately treated with parenteral anticonvulsants and antiedema measures. Investigations for preeclampsia were within normal limits. The Fundus showed Grade I hypertensive changes.

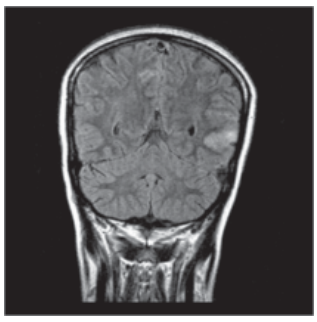

Fig. 1

Flare Coronal image Showing Haemorrhagic infarct in left Temporoparietal Region

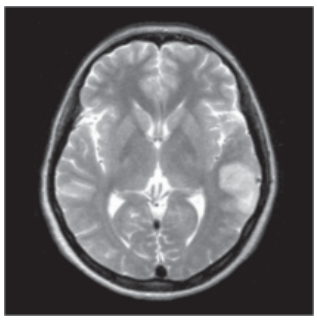

Fig. 2

T2 Weighted Axial image showing Infarct

MRI brain was done which showed haemorrhagic infarcts in the left temporoparietal region (fig 1 and fig 2) and in view of patients history MR venogram was performed in the same sitting which revealed left transverse

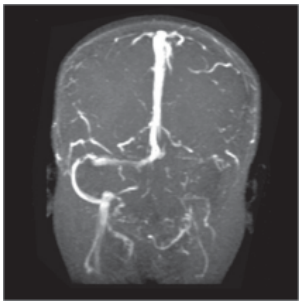

Fig. 3
MR Venogram showing Thrombosis
of Left Sigmoid and Transverse Sinuses

and sigmoid sinus thrombosis(fig 3). Therefore patient started on therapeutic low molecular weight heparin. Meanwhile patient was evaluated for thrombophilic disorder, all of which showed negative results. Patient showed drastic improvement symptomatically. After 5 days of overlap, anticoagulation was changed to Warfarin and the patient was discharged and is presently doing fine. Repeat MRI has been planned after 6 months for evidence of complete cure and to do antiphospholipid workup after anticoagulant is stopped.

DISCUSSION

Previously the controversial existence of delayed postpartum eclampsia is now acknowledged by most experts. Evidence suggests the increasing incidence of late onset eclampsia(6). By definition, convulsions with initial presentation more than 48 hours but less than 4 weeks after delivery are commonly referred to as late postpartum eclampsia. The presentation of late postpartum preeclampsia or eclampsia may differ from that occurring during pregnancy. This contributes to the difficulty in the diagnosis (4).

Lubrasky and Charms reported 44% and 79% patients respectively with late onset postpartum eclampsia who had not been identified as having preeclampsia before the onset of seizures (5, 6). Late postpartum eclampsia is most commonly preceeded by a prodrome of visual and cerebral symptoms (headache/ diplopia) which should not be ignored in any post natal patient.

Our patient had convulsions on the 10th postnatal day. The response to anticonvulsants with MRI showing haemorrhagic infarct with venous thrombosis with Fundus showing grade I hypertensive changes, with exclusion of metabolic, thrombophilic and infectious causes, strongly support the diagnosis of ECLAMPSIA.(Spontaneous cortical venous thrombosis which is a rare occurrence in pregnancy is a possible differential diagnosis for this case)

Lesions in MRI cannot predict whether damage is permanent or likely to be reversible. To reduce the rate of postpartum eclampsia, efforts should be directed to educate the patient and her health care providers regarding prompt reporting and evaluation of symptoms of preeclampsia during the postpartum period.

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