ECLAMPSIA AT 16 WEEKS GESTATION ASSOCIATED WITH PARTIAL MOLAR PREGNANCY

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Abstract

Eclampsia is a complication of severe preeclampsia. It’s commonly defined as new onset of grand mal seizure activity and/or unexplained coma during pregnancy or postpartum in a woman with signs or symptoms of preeclampsia. It typically occurs during or after the 20th week of gestation or in the postpartum period. [1, 2]. Otherwise, hydatidiform mole can be associated with very early-onset preeclampsia. In both pathologies various maternal symptoms arise from placental abnormalities. We present a very early case of eclampsia complicating a partial molar pregnancy associated with an exceptional Presssyndrom. Keyword: pre-eclampsia, hydatiform mole, placental dysfunction.

Introduction:

Eclampsia is a complication of severe preeclampsia. It’s commonly defined as new onset of grand mal seizure activity and/or unexplained coma during pregnancy or postpartum in a woman with signs or symptoms of preeclampsia. It typically occurs during or after the 20th week of gestation or in the postpartum period. [1, 2]. Otherwise, hydatidiform mole can be associated with very early-onset preeclampsia. In both pathologies, various maternal symptoms arise from placental abnormalities.

We present a very early case of eclampsia complicating a partial molar pregnancy associated with an exceptional Presssyndrom.

Case:

A 23-year-old white woman, gravida 1, para 0, with no medical history and no family history of genetic abnormalities, was admitted to the hospital after a generalized tonic-clonic seizure following an eclamptic fit at home at 16 weeks of pregnancy. Medical examination showed an unconscious patient with grand mal seizure. Blood pressure was 160/10 mm Hg. Positive proteinuria was found on urine testing. There was no peripheral edema. The eclampsia seizure stopped under treatment with magnesium sulphate and nicardipine, the blood pressure dropped progressively to 130/90 mmHg. The ultrasound examination showed a viable fetus with no detectable abnormalities. The following day the patient regained fully consciousness. Biological investigations showed normal levels of complete blood profile, electrolytes, urea, creatinine, ASAT/ALAT, coagulation studies. Cerebral computed tomographic scan showed no abnormalities. The b-HCG level was 130 000 IU/L. 24H Proteinuria was at 2.40 g. The MRI showed a white matter signal abnormality under the right parieto-occipital cortex associated with a
signal abnormality of the right parietal white matter in T1 iso-signal, T2 hypersignal and FLAIR without translation on the diffusion sequence suggesting a PRES atypical unilateral syndrome.

Therapeutic termination of pregnancy has been suggested to the patient and the induction was performed after patient consent.

Fetal description: a female fetus with no visible malformation. weight: 200g.

The pathological study of the placenta revealed a partial molar pregnancy with defective remodeling of the spiral arteries.
The chorionic villi are bordered by a regular trophoblastic coating. The site of inconstant polar hyperplasia with some aspects of Bulbous dystrophy

A remodeling defect of some spiral arteries with a type of partial retention of the vascular musculature

The patient recovered well post-delivery and was discharged home on oral antihypertensive and LMWH for a few more days. The blood pressure settled over the next few days to 140/90 mm Hg without treatment. She was monitored carefully with bHCG levels which have steadily reduced to normal levels after three weeks.

**Discussion:**

The incidence of hydatidiform mole with coexistent fetus is 0.005% to 0.01% of all pregnancies. It should be suspected when cystic placental changes are found in association with fetal malformations on ultrasonography. [7] Very few live pregnancies have been reported except in twin pregnancies with one surviving fetus and co-existing molar changes in the other sac (Marcorelles et al. 2005). Early neonatal deaths due to fetal anaemia or severe growth restriction have been reported. Although 90% of cases of partial mole are associated with triploidy.[8]

In the case presented molar vesicles were not seen on ultrasonography, and the fetus singleton seemed to have an appropriate growth at 16 weeks. The fetal karyotype in our case was not explored.
Hydatidiform mole and preeclampsia are two disorders unique to pregnancy. Placenta dysfunction is a common disorder in both pathologies. There have been very few studies on the molecular mechanisms that link hydatidiform moles with preeclampsia (PE), much regarding these mechanisms remains unknown. Many recent studies have demonstrated that placent al dysfunction underlies the development of PE due to hypoxia elicited by defective invasion of the spiral arteries (9). Anti-angiogenic factors produced by trophoblast cells enter the maternal blood and induce PE symptoms. High levels of soluble fms-like tyrosine kinase 1 (sFlt-1), an antagonist of vascular endothelial growth factor and placental growth factor, have been found in women with PE.[10][11][12]. Enhanced expression of sFlt-1 has also been reported in the blood and placenta of patients with hydatidiform moles. These findings suggested that sFlt-1 may be involved in the underlying pathophysiological mechanism of PE subsequent to hydatidiform mole. The placental dysfunction plays a central role in the development of early-onset PE. Therefore, investigation of the pathological mechanisms associated with the development of maternal PE symptoms in hydatidiform mole may lead to the further clarification of the pathophysiology of placental abnormalities related to PE[13,14].

Although early preeclampsia in the second trimester is a frequent association with both partial and complete molar pregnancy, eclampsia is rare. We found only 58 cases of eclampsia in association with molar pregnancy reported in the literature since 1866. In only 10 of those was a fetus discovered concurrently. They noted that neurologic manifestations were present in a large majority of patients before the first seizure and that most patients experienced multiple seizures.[10]

Conclusion:
Development of preeclampsia/eclampsia prior to 20 weeks of gestation should prompt a clinical evaluation to exclude the possibility of an underlying hydatidiform molar pregnancy.

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