Postpartum eclampsia. Impossible to eradicate?

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Summary: Two cases of post partum eclampsia in previously healthy women are presented. Prior to the onset of eclamptic seizures, which occurred, one respectively eight days after delivery, the patients had no symptoms and were without signs of preeclampsia.

Prevention of eclampsia is further discussed.

Key words: Post partum; Eclampsia.

INTRODUCTION

Eclampsia is a serious obstetric complication with varying incidence depending partly on prenatal care. However, this cannot explain the differences found in developed countries. In Sweden and Denmark a low incidence of eclampsia was found, 0.25-0.36/1000 deliveries ([1, 2]), whereas in Great Britain an incidence of 1/1000 deliveries ([3]), and in some parts of the U.S.A. an incidence as high as as 1/310 deliveries ([4]) were found. 14-25% of eclamptic seizures occur post partum, 30-50% of these more than 48 hours after delivery ([1, 4-8]).

Two cases of eclampsia in the puerperium are presented, one within 48 hours of delivery, the other more than eight days after delivery.

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CASE REPORTS

Case 1.

A 27-year-old primigravida went through a normal pregnancy without signs of preeclampsia. Her weight gain was nine kg. The onset of labor in the 42nd gestational week was spontaneous, but due to foetopelvine disproportion a caesarean section was performed and she was delivered of a healthy boy, weight 3300 g. Apgar scores were 7/1 min. and 10/5 min.

Two days after delivery a fall in blood-haemoglobin from 6.7 mmol/l to 2.9 mmol/l was observed and a celiotomy was performed. No intraperitoneal source of bleeding was found, but the patient had a large subcutaneous haematoma. Prior to the celiotomy the patient received transfusion of 2.5 l blood. During the next three days the patient had a weight gain of four kg, but from the fourth to the eighth day six kg were lost. The blood-pressure was normal during this period.

On the eighth day post partum the patient had convulsions simulating grand-mal seizures. Intravenous treatment with Diazepam (10 mg/seizure) and Phenytoin (15 mg/kg) was initiated. During the next 24 hours another ten seizures were observed.

Due to intermittent rise in blood-pressure (up to 160/90 mmHg), Labetalol-treatment was commenced. Blood pressure instability lasted for four days. A transient proteinuria was present.

The patient was seen by a neurologist and was found normal at physical examination. The EEG

and CAT-scan of the brain as well as the spinal fluid were normal.

Blood-glucose, serum-calcium, serum-potassium, serum-sodium, serum-creatinine and blood-gas analysis were all within normal range.

Phenytoin was discontinued after two months and the patient has had no seizures in the eight month follow-up period.

Case 2.

A 16-year old primigravida with no history of convulsive disease. In the second trimester triplets were diagnosed by ultrasound. The pregnancy was uneventful until the 35th week of gestation. She was then admitted with premature rupture of membranes and went rapidly into labor. She was delivered vaginally of a live girl in breech presentation, a dead (macerated) boy in cephalic presentation and finally another live girl in cephalic presentation. The infants' weight was 1970 g, 1300 g, and 1740 g respectively.

Nine hours later she had convulsions simulating grand mal seizures. During pregnancy the blood-pressure had been normal and no proteinuria had been found. After the seizure the patient developed massive oedema, hypertension and proteinuria. She was treated with Apresolin for two days, after which her condition was stabilized.

The neurologist found nothing abnormal at physical examination and the EEG was also normal. The patient was discharged eight days after delivery.

DISCUSSION

Theoretically, in order to prevent eclampsia it is relevant to be able to predict preeclampsia. Women at risk can be found by evaluating factors like family history, parity, and the roll over test ([9]).

New laboratory tests like the plasma fibronectin concentration and the urinary calcium/creatinine ratio have given promising results ([10]).

Other causes of convulsions, such as epilepsy, central vein thrombosis and Sturge Weber syndrome should be ruled out, but even if all predisposing factors are taken into consideration to prevent the development of eclamptic seizures, a group of patients still remains where no sign of preeclampsia has been demonstrated before the eclampsia occurs. Sibai *et al.* has named these the « unavoidable » cases and estimate that as many as 30% of eclamptic cases belong to this group ([6, 7]), whereas Møller *et al.* ([1]) found that predictors of eclampsia (rise in blood pressure or proteinuria) were either not present or were present in less than four days in 60-80% of the patients, thus making it difficult to prevent seizures.

Both our patients belong to this « unavoidable » group of patients, as neither of them had had signs of preeclampsia during pregnancy.

Adamsons and Wallach ([10]) suggest as a hypothesis that a single cause exists for initiation of both labor and preeclampsia-eclampsia and state that the decline in oxygen tension in intervillous space causes both. Adamsons and Wallach further claim that postpartum toxaemia is produced by the retained (extraplacental) trophoblast, perhaps facilitated by the rapid clearance of progesterone, which is a central nervous system and smooth muscle stabilizer. In a recent work, Richer and co-workers discuss the possibility of a vascular spasm in the brain as the origin of the eclamptic crisis ([11]).

Pearce ([3]) also reports that in 41% of eclamptic cases proteinuria was not present. A predisposing fatcor is the HELLP syndrome, which was present in 30% of eclamptic patients ([7]). One of our patients initially had abnormal EEG but has been followed for more than eighth months and is neurologically normal. This is in accordance with the findings of Sibai *et al.* ([12]) who evaluated a group of patients with regard to neurological abnormalities after eclampsia with EEG (and CAT-scanning as well as cerebral arteriograms in some cases) and found that after six months the initial abnormalities had subsided. Treatment of eclamptic seizures with Magnesium-sulphate is widely used, but, however was not always able to prevent seizures ([6, 7]). In Denmark, Magnesium-sulphate is not the first drug of choice. Our patient with late post-partum eclampsia needed Phenytoin of 15 mg/kg.

CONCLUSION

In spite of extensive antenatal care directed towards early detection of pre-eclampsia, a group of patients exists in whom either no symptoms or only discrete symptoms and signs arise. Obviously, these cases cannot be totally avoided. Therefore, it is essential to recognize the condition and treat it accordingly.

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