



*Case Report*

# Eclampsia at 17 Weeks and 3 Days Gestation: A Diagnostic Dilemma

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## Abstract

Eclampsia is generally preceded by preeclampsia or proteinuric hypertension after the twentieth week of gestation. Only ten cases of eclampsia were reported in the presence of a foetus before 20 weeks of gestation over a 120 year epoch. It is usually believed that preeclampsia occurring before the twentieth week of gestation tends to occur in the presence of a molar pregnancy or the presence of lupus anticoagulant in the pregnant patient.

The occurrence of eclampsia in a multiparous patient all of whose previous pregnancies were by the same consort and who presented at 17 weeks and 3 days gestation was an obvious diagnostic dilemma. Not only was it difficult to make the initial clinical diagnosis, it also proved quite a challenge to the histologists. After much consultation, the histologist agreed that the histology of the placenta was suggestive of a condition called placental mesenchymal dysplasia.

**Keywords:** Placental mesenchymal dysplasia, eclampsia, molar pregnancy.

## Introduction

Eclampsia is the occurrence of generalized seizure activity in a patient with pre-eclampsia. Davey and MacGillivray (1988) defined pre-eclampsia as hypertension in combination with proteinuria, developing after twenty weeks gestation in a previously normotensive, non-proteinuric patient. Preeclampsia can develop before the twentieth week of pregnancy in two situations: extensive hydatidiform changes in the chorionic villi and/or the presence of lupus anticoagulant (ACOG, 1986). Our case

presenting with eclampsia at 17 weeks and 3 days was lupus anticoagulant negative. Eclampsia before the twentieth week of gestation is rare. In a 120-year review, Newman and Eddy (1998) found only ten cases of eclampsia occurred in the presence of a foetus before twenty weeks gestation.

## Case

A 30-year-old Caucasian housewife presented at 17 weeks and 3 days gestation in her fifth pregnancy by the same consort. Her four previous pregnancies resulted in

the delivery of three sons (the first at 35 weeks gestation and the other two at term) and a spontaneous miscarriage at 9 weeks gestation. She had no significant medical history and she did not abuse drugs or smoke.

At ten weeks gestation in the index pregnancy, she had an episode of vaginal bleeding. An ultrasound scan revealed a single intrauterine gestational sac with a 28mm embryo with cardiac pulsations, which was comparable to a foetus 9 weeks and five days gestation. There was a small retro placental blood clot measuring 1.7 X 0.7cm.

On presentation, she was confused. Her consort reported that she had been complaining of severe frontal headaches throughout the night and that she was noted to have had a generalised seizure at about 0500 hours, one hour prior to presentation.

Her blood pressure on admission was 180/120. She was assessed as being in the post-ictal phase and a metabolic cause for her seizure was considered likely. A full blood count, urea and electrolytes, glucose, magnesium, calcium, phosphate levels and liver function test were done. The results of which were all normal except for a serum  $\beta$ -hcg value  $> 10^5$  miu/ml, a platelet count of 128,000/mm<sup>3</sup> and an aspartate value of 120 iu/l. Her random serum glucose level was 100mg/dl.

Phenytoin was commenced with appropriate monitoring by the on call physicians. Within an hour of presentation, she had a second tonic-clonic generalised seizure. Diazepam was used to stop the seizure. A continuous infusion of labetalol was commenced at a rate of 2mgs/min with close surveillance in an effort to reduce her blood pressure.

The Obstetricians Opinion was sought.

### **Obstetricians Review**

She was drowsy and muttered unintelligently. Her blood pressure ranged from 180/120 to 150/95. She had 3pluses

of pitting oedema up to the level of both knees, her deep tendon reflexes were brisk and she had three beats of clonus. A catheterized urine specimen revealed four pluses of protein.

The possibility of eclampsia, though unusual at this gestation was considered; possibly secondary to a molar pregnancy. Ultrasonography revealed a dead foetus with an unusually large placenta containing multiple anechoic areas.

Within four hours of presentation, the patient developed status epilepticus, she was transferred to the intensive care unit with a magnesium sulphate infusion, and she was anaesthetized and intubated. The parenteral labetalol was continued and her blood pressure was gradually lowered to a safer range of 140-160/74-85. A head CT scan was normal with no detectable evidence of posterior reversible leukoencephalopathy.

Her uterus was evacuated the following day with an ovum forceps and suction curettage all of the recovered tissue was sent for histological analysis.

### **Histology Report**

Our local histologist found difficulty in diagnosing the underlying condition and a second opinion was sought from our local experts, Charring Cross Hospital.

Abundant chorionic villi, the majority of which are immature, the specimen also contained intermediate villi of varying sizes. Some villi show moderate to marked hydrops and there are occasional stem villi, which are markedly enlarged with cistern formation. In addition, there are prominent foetal blood vessels and numerous nucleated foetal red cells are seen. There is however, no significant trophoblastic hyperplasia.

The features are most suggestive of a non-molar pregnancy and some features suggest the possibility of placental mesenchymal dysplasia (a condition which is thought to be benign but can clinically

and sonographically mimic a molar pregnancy).

The patient's post evacuation period was uneventful, her platelet counts and aspartate value returned to normal on day 3 and she was discharged home on day 5. She was registered with the Charing Cross hospital where her serum  $\beta$ -hcg levels were monitored for a month. The local consultant saw her at six weeks post evacuation for counselling.

### Discussion

Preeclampsia is defined by Davey & MacGillivray (1988) as proteinuric hypertension, developing after twenty weeks gestation in a previously normotensive, non-proteinuric patient. Hitherto, preeclampsia was thought to develop before the twentieth week of pregnancy in two scenarios: extensive hydatidiform changes in the chorionic villi or in the presence of lupus anticoagulant (ACOG 1986). This case presenting at 17 weeks and 3 days gestation with eclampsia was lupus anticoagulant negative.

Eclampsia before the twentieth week of pregnancy is rare. In a 120-year review, by Newman & Eddy (1998), only ten cases of eclampsia occurred in the presence of a foetus before twenty weeks gestation.

The microscopic appearance of the placenta was suggestive of placental mesenchymal dysplasia. This condition was first described in 1991 by Moscoso and colleagues. It is of unknown incidence and hitherto was not associated with early onset preeclampsia/eclampsia.

Flow cytometric DNA analysis performed on formalin fixed, paraffin -embedded tissue blocks of placental and foetal tissue showed both the placenta and foetus were triploid. Placental mesenchymal dysplasia as the sole cause of this early onset eclampsia was therefore excluded; instead a triploid foetus coexisting with a triploid placenta containing features of placental mesenchymal dysplasia was responsible.

A literature search revealed only one other case of a non-trophoblastic tumour coexisting with a triploid foetus that presented with severe preeclampsia at 18 weeks and 3 days (Pietrantonio, 1995).

Our case is unique, in that, it is the first reported case in which features of placental mesenchymal dysplasia is noted to coexist with a triploid foetus and it is also among the earliest cases recorded in which eclampsia has occurred.

Could this have been a molar pregnancy, of the partial mole variety? Histological findings tell us that since it lacked trophoblastic hyperplasia and stromal trophoblastic inclusions, pathognomonic features of a partial mole, Paradinas (2001), this was histologically, not a partial mole.

Additionally, this unique combination differed in presentation and placental histology suggesting that it represents a more aggressive disease than the partial mole.

Moscoso (1991), first described placental mesenchymal dysplasia. It is characterised by placentomegaly and abnormal chorionic villi often clinically mistaken with a partial mole.

Unlike molar pregnancies, placental mesenchymal dysplasia usually features a normal foetus; however there is a strong association with intrauterine growth restriction and intrauterine foetal demise. Around 20% of cases also show features of Beckwith-Weidemann Syndrome (BWS) that include macrosomia, visceromegaly and macroglossia.

Cohen (2005), report an incidence of 0.02% with a definite female preponderance of 3.6:1. The pathogenesis is unknown and maternal complications associated with Placental Mesenchymal Dysplasia are rare.

The definitive diagnosis of placental mesenchymal dysplasia can only be made by histological examination of the placenta. There is good evidence to suggest that placentalomegaly is a contributor to the features of preeclampsia according to Jauniaux, (1997). However, McCowen, (1994), believes that the proteinuric hypertension reported in some cases of placental mesenchymal dysplasia is likely to be a coincidental finding.

The clinical presentation of placental mesenchymal dysplasia is diverse. The sonographic appearance of placental mesenchymal dysplasia is a thickened placenta containing hypoechoic areas. Jauniaux (1997), using serial ultrasound and colour Doppler examination in pregnancy with placental mesenchymal dysplasia did not identify blood flow within the placental lesion during the first five months of gestation. However, in the third trimester large vascular areas with turbulent blood flow was observed.

### Conclusion

We believe that this case report is unique since it describes a clinical condition which hitherto was unknown to be associated with eclampsia. The uniqueness of this case is further exemplified by (a) the early onset of eclampsia at 17 weeks and 3 days, (b) the age of the patient, (30years), (c) She was multiparous at gravidity 5, (d) All five pregnancies were by the same consort, and (e) The histology of the placenta. This is the first reported case of eclampsia associated with placental mesenchymal dysplasia in a case in which both the foetus and the placenta were triploid. This tacitly may suggest that this combination is associated with a more aggressive form of pregnancy induced hypertension.

### References

American College of Obstetrics and Gynaecology (1986). ACOG Technical bulletin no.91. Washington, DC.

Cohen, M. C., Roper, E. C., Sebire, N. J., et al. (2005). "Placental Mesenchymal Dysplasia Associated with Foetal Aneuploidy," *Prenatal diagnosis* 25 187-192

Davey, D. A. & MacGillivray (1988). 'The Classification of Hypertensive Disorders of Pregnancy,' *Am J Obstet and Gynecol*, 158 175-215.

Jauniaux, E., Nicolaides, K. H. & Hustin, J. (1997). "Perinatal Features Associated with Placental Mesenchymal Dysplasia," *Placenta*, 18 701 - 706.

McCowan, L. M. E. & Becroft, D. M. O. (1994). "Beckwith-Wiedemann Syndrome, Placental Abnormalities and Gestational Proteinuric Hypertension," *Obstet Gynecol*, 83 813-817

Moscoso, G., Jauniaux, E. & Hustin, J. (1991). "Placental Vascular Anomaly with Diffuse Mesenchymal Stem Villus Hyperplasia. A New Clinic-Pathological Entity?," *Path Res Pract*, 187 324 - 328.

Newman, R. B. & Eddy, G. L. (1998). "Association of Eclampsia and Hydatidiform Mole: Case Report and Review of the Literature," *Obstet and Gynaecol Survey*, 43 (4) 185-190.

Paradinas, F. J., Sebire, N. J., Fisher, R. A., Rees, H. C., Foskett, M., Secki, M. J. & Newlands, E. S. (2001). "Pseudo-partial Moles: Placental Stem Vessel Hydrops and the Association with Beckwith-Wiedemann Syndrome and Complete Moles," *Histopathology* 39 447 - 454.

Pietrantonio, M., Brees, C. K., Gerassimides, A., Cook, V., Youkilis, B. & Hersh, J. H. (1995). "A Non-Trophoblastic Tumour Coexisting with a Triploid Fetus," *Early pregnancy: Biology and Medicine* 1 67 - 71.