Short Communication   **Open Access**

Pathophysiology of Eclampsia

Williams KP[1]* and **Galerneau F[2]**

[1]Department of Obstetrics and Gynecology, Rowan School of Osteopathic Medicine, Stratford, NJ, USA

[2]Department of Obstetrics, Gynecology and Reproductive Sciences, Yale University School of Medicine, New Haven, Connecticut, USA

*Corresponding author: Williams KP, Department of Obstetrics and Gynecology, Rowan School of Osteopathic Medicine, Stratford, NJ, USA, Tel: 8565667099; E-mail: williakp@rowan.edu

Introduction

Hypertension in pregnancy complicates approximately 7-10% of pregnancies and remains a major cause of maternal and perinatal morbidity and mortality [1,2]. Severe pre-eclampsia/eclampsia (generalized convulsions with pre-eclampsia) which occurs in approximately 1-3 in 1000 women is thought to be a terminal progression of pre-eclampsia1 [1]. The maternal mortality rate for eclampsia is 2-3 cases per 10,000 births in Europe and North America and 16-69 cases per 10,000 births in developing countries. 10-15% of direct maternal deaths are associated with preeclampsia annually [2].

Of all organ systems affected, it is important to note that the brain can only be assessed by signs and symptoms. No simple laboratory blood or urine investigation can provide any mechanism of assessing cerebral function. Other techniques such as computed tomography (CT) scan and magnetic resonance imaging (MRI) are non-invasive, but not easily repeatable [3-5].

Radiological Imaging in Eclampsia

The radiological techniques used to assess the cerebral function in patients with pre-eclampsia/eclampsia include: (1) CT scanning and (2) MRI. With CT scanning, the incidence of patients with a cerebral abnormality is 50% [3,6]. The most frequent abnormalities include transient white matter hypodensities [4], in addition to cerebral, cortical and basal hypodensities and diffuse edema. Infarction and hemorrhage are uncommon. With MRI, a transiently high T2 intensity is especially common in the cerebral cortex and subcortical white matter which correlates with cerebral edema [6-9] Appropriate identification of a significant difference between MRI in severe pre-eclampsia vs. eclampsia is difficult to achieve. Digre [10] evaluated 16 women with severe pre-eclampsia and ten women with eclampsia and found that 50% of the pre-eclamptic women had abnormal scans with non-specific foci of increased signal in the deep cerebral white matter on T2-weighted images. Women with eclampsia had either a multifocal area of increased signal in the gray–white matter junction on T2-weighted images or edema and hemorrhage. However, review of the clinical findings of this eclamptic group revealed that the majority had significant persistent neurological dysfunction including two with coma.

Transcranial assessment in eclampsia

We previously reported on the use of trans cranial doppler to assess maternal middle cerebral blood flow velocity. By applying the modified formula of Belfort et al. [11] however, we were able to convert these velocity changes with simultaneous measurements of arterial pressure into estimated cerebral perfusion pressure. We were also able to determine an index of cerebral vascular resistance and thereby appropriately calculate an index of cerebral blood flow.

At the initial assessment, the estimated cerebral perfusion pressure was higher in women with eclampsia than in those with severe pre-eclampsia. In addition, cerebrovascular resistance was significantly decreased in the eclampsia group, and it remained decreased as long as 4 days in one patient with eclampsia [12].

We found these increases in cerebral perfusion pressure in patients with severe pre-eclampsia to be counterbalanced by increases in cerebrovascular resistance; consequently, cerebral blood flow did not change.

Our report documented a significant fall in cerebral vascular resistance in all patients with eclampsia with compared to values of patients with severe pre-eclampsia. This decrease in cerebral vascular resistance persisted for ≥ 24 h in one case, ≥ 48 hours in another case, and ≥ 4 days in a third case.

Naidu et al. [13] reviewed the cases of 65 women with eclampsia and assessed cerebral pathophysiological characteristics with the following techniques: cerebral CT, single-photon emission CT, and Doppler analysis of MCBFV (middle cerebral blood flow velocity). Single-photon emission CT revealed defects in 100% of women in the watershed areas of the brain, CT revealed hypodensities in 59% of women, and TCD (trans-cranial Doppler) values were recorded as elevated in 86% of women. Our study findings, however, supported by Zunker et al. [14], indicate that eclampsia occurs in a setting similar to that of hypertensive encephalopathy.

Cerebral pathophysiology in eclampsia

Two hypotheses exist regarding the vascular changes associated with eclampsia. Vasospasm causing local ischemia will result in arterial necrosis and disruption of the blood-brain barrier, which leads to cerebral edema [1,15]. Angiographic studies have demonstrated both cerebral edema and vasospasm [16]. Conversely, however, vasoconstriction may be a protective rather than a pathological response to extremes of arterial pressure. It may prevent an uncontrolled increase in arterial perfusion and damage to the distal microcirculation. At high arterial pressures, the vascular smooth muscle may reach the limits of its strength and then dilate. Short segments vasodilate first, but they extend until the length of the vessel is compromised [17-19]. This sequence can be shown during experimental hypertension. The dilation is associated with damage to the vessel wall, focal edema, and a passive increase in cerebral blood flow. Which of these mechanisms is responsible for the changes in eclampsia is unknown [1,15,20,21]. Both vasogenic and cytotoxic edema have been shown in eclampsia. Studies using diffusion weighted MR images found out that these hyperintense areas on MR had an

apparent high diffusion coefficient value, indicative of vasogenic and not cytotoxic edema.

To date, most studies have shown that in established pre-eclampsia/eclampsia, significant changes occur in cerebral hemodynamics related to vasospasm and hyperperfusion. In spite of this, the exact mechanism by which women undergo seizure during preeclampsia is unknown. Longitudinal studies need to be designed to assess the prediction of eclampsia to improve maternal and neonatal morbidity and mortality.

References

1. Redman CW (1988) Eclampsia still kills. Br Med J (Clin Res Ed) 296: 1209-1210.

2. Sibai BM (1990) Eclampsia. VI. Maternal-perinatal outcome in 254 consecutive cases. Am J Obstet Gynecol 163: 1049-1054.

3. Brown CE, Purdy P, Cunningham FG (1988) Head computed tomographic scans in women with eclampsia. Am J Obstet Gynecol 159: 915-920.

4. Fredriksson K, Lindvall O, Ingemarsson I, Astedt B, Cronqvist S, et al. (1989) Repeated cranial computed tomographic and magnetic resonance imaging scans in two cases of eclampsia. Stroke 20: 547-553.

5. Milliez J, Dahoun A, Boudraa M (1990) Computed tomography of the brain in eclampsia. Obstet Gynecol 75: 975-980.

6. Royburt M, Seidman DS, Serr DM, Mashiach S (1991) Neurologic involvement in hypertensive disease of pregnancy. Obstet Gynecol Surv 46: 656-664.

7. DeWitt LD, Wechsler LR (1987) Transcranial Doppler. Stroke 22: 31-36.

8. Dierckx I, Appel B (1989) MR findings in eclampsia. AJNR Am J Neuroradiol 10: 445.

9. Crawford S, Varner MW, Digre KB, Servais G, Corbett JJ (1987) Cranial magnetic resonance imaging in eclampsia. Obstet Gynecol 70: 474-477.

10. Digre KB, Varner MW, Osborn AG, Crawford S (1993) Cranial magnetic resonance imaging in severe preeclampsia vs eclampsia. Arch Neurol 50: 399-406.

11. Aaslid R, Lundar T, Lindegaard KF, Nornes H (1989) Estimation of cerebral perfusion pressure from arterial blood pressure and transcranial Doppler recordings. In: Miller JD, Taesdale GM, Rowan JO, Galbraith SL, Mendelow AD (eds.) Intracranial pressure VI. Proceedings of the Sixth International Symposium on Intracranial Pressure, Glasgow, Scotland, pp. 26-29.

12. Williams KP, Wilson S (1999) Persistence of cerebral hemodynamic changes in patients with eclampsia: A report of three cases. Am J Obstet Gynecol 181: 1162-1165.

13. Naidu K, Moodley J, Corr P, Hoffmann M (1997) Single photon emission and cerebral computerised tomographic scan and transcranial Doppler sonographic findings in eclampsia. Br J Obstet Gynaecol 104: 1165-1172.

14. Zunker P, Happe S, Georgiadis AL, Louwen F, Georgiadis D, et al. (2000) Maternal cerebral hemodynamics in pregnancy related hypertension. A prospective transcranial Doppler study. Ultrasound Obstet Gynecol 16: 179-187.

15. Aaslid R, Markwalder TM, Nornes H (1982) Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 57: 769-774.

16. Trommer BL, Homer D, Mikhael MA (1988) Cerebral vasospasm and eclampsia. Stroke 19: 326-329.

17. Hauser RA, Lacey DM, Knight MR (1988) Hypertensive encephalopathy. Magnetic resonance imaging demonstration of reversible cortical and white matter lesions. Arch Neurol 45: 1078-1083.

18. Chester EM, Agamanolis DP, Banker BQ, Victor M (1978) Hypertensive encephalopathy: a clinicopathologic study of 20 cases. Neurology 28: 928-939.

19. Strandgaard S, Paulson OB (1984) Cerebral autoregulation. Stroke 15: 413-416.

20. Will AD, Lewis KL, Hinshaw DB Jr, Jordan K, Cousins LM, et al. (1987) Cerebral vasoconstriction in toxemia. Neurology 37: 1555-1557.