Late Onset Postpartum Seizure and Magnetic Resonance Image Findings

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Two young women were brought to the Emergency room with generalized tonic and clonic seizures. Seizure developed seven and ten days after delivery respectively without the clinical signs of pre-eclampsia throughout the pregnancies. Magnetic resonance(MR) image of the brain showed characteristically symmetrical abnormal signals in the parietal and occipital regions. After several days of medical treatment, they were discharged without neurologic sequelae and follow-up MR images taken three months after discharge showed complete disappearance of the previous abnormal signals.

KEY WORDS: Pre-eclampsia · Eclampsia · Seizure · MRI.

Introduction

Pre-eclampsia is a well-known complication of pregnancy, characterized by hypertension, proteinuria, and edema. When pre-eclampsia is associated with seizure, it is defined as eclampsia that accounts for high maternal mortality. Seizure usually develops in the pre-eclamptic woman sometime between the 20th week of gestation and 48 hours after delivery. But some of the patients develop seizure after that period without experiencing the clinical triad of pre-eclampsia during the pregnancy²⁾. In one report, seizure occurred as late as 23 days after delivery⁷⁾. The previously controversial existence of this late onset eclampsia is now generally accepted.

High signals in the parieto-occipital white matter or basal ganglia are reported to be characteristic T2 wighted MRI findings of these patients. Authors report two cases of late onset postpartum seizure.

Case Report

Case 1

A 29-year-old woman was hospitalized due to the development of generalized tonic and clonic seizure. She had a history of cesarean section delivery of her first baby 7days before the seizure. She had no hypertension, proteinuria and edema

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during the pregnancy and the operation was successful without any complications. She recovered well from the operation without any problems. At admission, she was hypertensive (160/100mmHg) but routine laboratory findings were all reported normal. The brain CT scan at ER was normal. The FLAIR images of MRI taken after admission showed multiple symmetric patch high signals in the posterior frontal and parietal areas (Fig. 1). The CT angiography showed no vascular abnormalities. She was given anticonvulsants and antihypertensives. She recovered well and was discharged without any neurologic abnormalities. Follow-up MRI taken 3months after discharge showed complete disappearance of previous abnormal signals (Fig. 2). Her blood pressure became normal without antihypertensive medications. Anticonvulsants were no longer prescribed.

Case 2

A 31-year-old woman was hospitalized due to the development of generalized tonic and clonic seizure which lasted approximately 3minutes. She had a history of normal full-term vaginal delivery of her second baby 10 days before the seizure. She had a

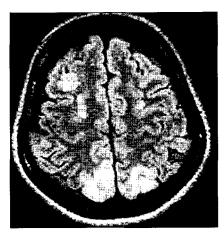


Fig. 1. Multiple symmetric high signals in the posterior frontal and parietal areas on FLAIR image of magnetic resonance image.

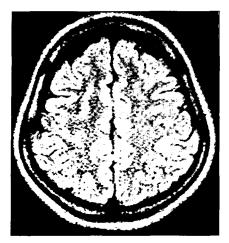


Fig. 2. No abnormal signals on follow-up magnetic resonance image.

mild generalized densities on CT or edema for two mohigh signals on T2 nths before delivery weighted or FLAIR MRI⁷⁾. Although but other signs of the MRI lesions pre-eclampsia weobserved in our care not reported. At admission, she shses were not solowed borderline ely confined to the hypertension (140/ posterior circula-90mmHg) The brtion territories, the ain CT scan at ER symmetric patch revealed a small distribution of the left sylvian aracreversible lesions hnoid cyst but she in the fronto-pardenied any seizure



Fig. 3. Similar signals as in Fig. 1.

in her past medical history. The FLAIR images of the MRI showed multiple symmetric high signals in the posterior frontal and parietal areas (Fig. 3). She recovered well and was discharged without any neurological abnormalities. Follow-up MRI taken 3months after discharge showed complete disappearance of abnormal signals (Fig. 4). Her blood pressure became normal without antihypertensive medications. Anticonvulsants were also discontinued.

Discussion

clampsia is defined as seizure before, or after delivery, which usually develops in a pre-eclamptic pregnant women. Classic autopsy studies of eclamptic patients reveal widespread cerebral edema, but multiple petechial hemorrhages or localized edema in the occipital lobe are more common. In some patients, fatal intracerebral hemorrhage develops as a result of hypertension or rupture of pre-existing cerebral vascular diseases such as aneurysms or arteriovenous malformations^{1,6)}. MRI of the eclampsia shows high signals in subcortical white matter or basal ganglia on T2-wighted or FLAIR images⁷⁾. These findings are usually reversible and caused by transient impairment of the blood-brain barrier in the edematous cortex⁸. The pathophysiology of the cerebral lesion is postulated as increased permeability of small vessels due to endothelial injury and transient breakdown of cerebral vascular autoregulation by hypertension^{5,9)}. Another hypothesis is that vasospam of cerebral arteries, in response to paradoxical increase in thromboxane, results in cerebral ischemia and cytotoxic edema⁷⁾. Extensive edema is postulated to be caused by cytotoxic edema intensified by vasogenic edema4).

Up to half of the patients with eclampsia are known to have transient changes in the parieto-occipital areas verified by low ietal area implies these disorders to be in the category of posterior reversible encephalopathy syndrome (PRES).

PRES is a recently discovered disorder characterized by reversible vasogenic edema mainly in the posterior circulatory territories. Eclampsia is one of the main causes of PRES, but hypertensive encephalopathy caused by other diseases can also result in PRES. The predilection for the involvement of posterior circulation is explained by the relatively sparse sympathetic innervation of the vertebrobasilar system. Cerebral autoregulation is normally maintained by both myogenic and neurogenic components, but in patients with PRES, the myogenic response is blunted by either passive overdistention of the vessels due to hypertension or direct toxic effect on the endothelium. Because autoregulatory mechanisms are more dependent on the neurogenic component, the more poorly innervated areas in the posterior circulation are more vulnerable³).

Though late postpartum eclampsia is known to have low prevalence rate, in recent report, the incidence of antenatal eclampsia is decreasing because of early evaluation and appropriate treatment of preeclampsia but postnatal eclampsia is increasing²⁾.

Late postpartum eclampsia is difficult to predict because of frequent lack of classic triad of hypertension, proteinuria, and edema.

The treatment of eclampsia is mostly supportive measures, seizure control, and blood

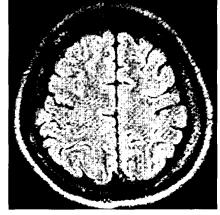


Fig. 4. No abnormal signals on follow-up magnetic resonance image.

pressure management. When the diagnosis is made by exclusion of any other medical or neurologic disorders causing seizure, first line treatment for eclampsia is giving MgSO4 with or without other anticonvulsants. Mg SO4 acts primarily to stabilize membrances and stops seizure in most of the patients⁷⁾. Hypertension often resolves with ce-ssation of seizure and usually does not need continued medication but when systolic blood pressure exceeds 160mmHg or diastolic blood pressure is over 105mmHg, antihypertensive medication is recommended.

Conclusion

hen a woman develops seizure even a couple of weeks after delivery, late onset eclampsia should be ruled out for rapid and appropriate treatment.

References

 Ahn KU, Whang K, Pyen JS, Hu C, Hong SK, Kim HJ, et al: Cerebrovascular disease during pregnancy. J Korean Neurosurg Soc 28: 658-662, 1999

- Chames MC, Livingston JC, Ivester TS, Barton JR, Sibai BM: Late postpartum eclampsia: a preventable disease? Am J Obstet Gynecol 186: 1174-1177, 2002
- Covarrubias DJ, Luetmer PH, Campeau NG: Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion weighted MR images. Am J Neuroradiol 23: 1038-1048, 2002
- Cunningham FG, Twickler D: Cerebral edema complicating eclampsia. Am J Obstet Gynecol 182: 94-100. 2000
- Koch S, Rabinstein A, Falcone S, Forteza A: Diffusion-weighted image shows cytotoxic and vasogenic edema in eclampsia. Am J Neuroradiol 22: 1068-1070, 2001
- 6. Lee CW, Kim YS, Park MS, Ha HG, Lee JS, Jung H, et al: Intracranial hemorrhage developed from patients who had been preeclampsia at five days postpartum. J Korean Neurosurg Soc 30: 371-375, 2001
- Martin J, Sidman R: Late postpartum eclampsia: a common presentation of an uncommon diagnosis. J Emerg Med 25: 387-390, 2003
- Veltkamp R, Kupsch A, Polasek J, Yousry TA, Pfister HW: Late onset postpartum eclampsia without pre-eclamptic prodromi: clinical and neuroradiological presentation in two patients. J Neurol Neurosurg Psychiatry 69: 824-827, 2000
- Watanabe Y, Mitomo M, Tokuda Y, Yoshida K, Choi S, Hosoki T, et al: Eclamptic encephalopathy: MRI, including diffusion weighted images. Neuroradiology 44: 981-985, 2002