IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 14, Issue 6 Ver. IV (Jun. 2015), PP 17-19
www.iosrjournals.org

Eclampsia Precedes Pre-Eclampsia

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Abstract: *Eclampsia is an extremely severe form of preeclampsia characterized by the sudden onset of generalized tonic clonic seizures. It may occur quite abruptly, without any warning manifestations. This is a case report where Mrs.X was normotensive throughout her pregnancy who underwent emergency cesarean section for cephalo pelvic disproportion and threw generalized tonic clonic seizures on the first post-operative day with a normal BP recording. Later she started developing hypertension with proteinuria with normal liver enzymes and platelet counts. This signifies that eclampsia may not always be preceded by preeclampsia and can also develop in a normotensive woman without derangement of the biochemical parameters.*
Keywords: *Eclampsia, hypertension, preeclampsia, proteinuria, seizures.*

I. Introduction

Hypertensive disorders complicating pregnancy are common and form one of the deadly triad in maternal mortality, along with hemorrhage and infection. Hypertensive disorders in pregnancy constitute a perplexing and clinically challenging group of pregnancy complications that are responsible for a substantial burden of illness in developed as well as underdeveloped countries of the world.[1] In most of the cases preeclampsia is preceded by eclampsia, rarely eclampsia precedes preeclampsia or eclampsia can occur without preeclampsia.[2]

II. Case Report

A 24 year old primigravida was hospitalized at 39 weeks gestation with regular uterine contractions. She had her regular antenatal visits, which revealed no abnormality. She was normotensive throughout her antenatal follow-up. On admission her BP was 116/80 mmHg. She was taken up for emergency LSCS for Cephalo Pelvic Disproportion (CPD). She delivered a healthy male baby. At 5 hrs after operation, she developed a generalized convulsion, lasting 7 min, despite being normotensive both in the antenatal and post operative period, and soon after this she had a blood pressure recording of 170/110 mmHg with proteinuria of 2+. She was treated with $MgSO_4$ (loading dose of 4 g over 15-20 min, followed by a maintenance dose of 1 g/h as a continuous intravenous infusion). Laboratory findings were unremarkable (hemoglobin 12.3 mg/dL, hematocrit: 37.2%, thrombocyte: 262000/mm^3, urea : 19 mg/dl, creatinine: 0.6 mg/dl, uric acid:3.2 mg/dl, ALT: 34 U/L, AST: 35 U/L, LDH: 519 U/L. Computed tomography (CT) was completely normal. The next day, urine sample revealed 1+ proteinuria; and blood pressure ranged between 160/110 to 140/100 mmHg, so she was started on Tab Labetelol 100mg bd following which her BP came down to 130/90 mmHg. She was discharged on 5th post operative day with a BP recording of 130/80 mmHg with trace proteinuria. She was advised alternate day BP charting and to continue antihypertensives and to review after 15 days. On follow up after 15 days her BP recording was 126/70 mmHg and she was advised to stop antihypertensives by tapering the dose.

III. Discussion

Preeclampsia is defined as the new onset of hypertension and proteinuria after 20 weeks of gestation in a previously normotensive woman. The occurrence of generalized convulsions in a woman with preeclampsia without an alternative identifiable cause is referred to eclampsia.[3] Eclampsia, a dramatic and often unpredictable complication of pregnancy-induced hypertensive disorders, is characterized by sudden hypertension, proteinuria, edema, and seizures.[4] Globally hypertensive disorders of pregnancy complicate approximately 5–10 % of pregnancies. Approximately 72,000 pregnant women die every year because of eclampsia and severe preeclampsia. That amounts to nearly 200 women every day. Preeclampsia– eclampsia ranks second only to hemorrhage as a specific, direct cause of maternal death. The risk that a woman in a developing country will die of preeclampsia or eclampsia is about 300 times that of a woman in a developed country. Incidence of hypertensive disorders in India is found to be 10.08 % as observed through the data collected by the National Eclampsia Registry (NER) (11,266 out of 1,11,725 deliveries) over the past 3 years with 2,554 patients out of this presenting with eclampsia [1]. Approximately one-half of all cases of eclampsia occur postpartum.[3, 4]

Eclampsia is an enigmatic syndrome, both in its pathogenesis and in its temporal relation to gestation. This pregnancy-related hypertensive encephalopathy and has been attributed to intense cerebral vasospasm with breakthrough of autoregulation of intracranial arterial vasculature. Subsequent cerebral edema results from microischemic damage to the blood-brain barrier. Proposed triggers include endothelial damage [5, 6], imbalance between vaso-constrictive and vasodilatory prostaglandins, sympathetic overactivity[7], and abnormal placentation[8]. Theoretically two derangements have been proposed to underlie the development of focal cerebral edema: vasospasm and forced dilation. The vasospastic theory suggests that cerebral overregulation leads to vasoconstrictive responses of arterial vasculature after explosive increases in blood pressure. Vasospasm, in turn, produces local ischemia, resulting in protein extravasation, arteriolar necrosis, disruption of the blood-brain barrier, and microischemic CNS lesions. In contrast, the forced dilation theory suggests that during sustained and extreme elevation of blood pressure, autoregulatory vasoconstrictive forces are overwhelmed, forced vasodilation ensues, tight junctions are opened, and the blood-brain barrier is disrupted through endothelial damage. Deregulated vascular injury to blood-brain barrier endothelium leads to edema, protein extravasation, and fibrinoid necrosis. Either of these proposed vasogenic events could provide the anatomic substrate for widespread CNS insults with resultant premonitory symptoms and convulsions that characterize eclampsia.[4]

The incidence of eclampsia is higher in preeclamptic or twin pregnancies, women of low socioeconomic status or in developing countries, and nulliparous patients younger than 20 years or multiparous patients older than 35 years of age. Whether preceded during prenatal visits by prodromal evidence of preeclampsia, or occurring without antecedent warning symptoms, the great majority of eclamptic seizures occur in the antepartum setting between 20 and 40 weeks of gestation or within a few hours to 2 days postpartum. Most authorities report that 50%, 25%, and 25% of seizures occur in the antepartum, intrapartum, and postpartum periods, respectively. Controversy surrounds the occurrence of eclampsia developing longer than 48 hours after delivery. Some authors are skeptical that a relation exists between pregnancy and any seizure occurring more than 2 days postpartum.[4]

Late postpartum eclampsia is a rare variant of eclampsia and has been recognized as a definite entity only recently. Late postpartum eclampsia occurring without preceding pre-eclampsia is even rarer. In the largest series published so far (54 patients over a period of 15 years), Lubarsky et al found that late postpartum eclampsia constitutes 56% of total postpartum eclampsia and 16% of all cases of eclampsia. Only 56% of patients had been identified as pre-eclamptic prior to the occurrence of seizures.[9, 10] Complications in eclampsia are common and include acute renal failure, acute liver failure, and respiratory complications, such as aspiration pneumonia and acute pulmonary edema. Mortality in eclampsia is mostly related to intra cranial haemorrhage. Because of the severe complications, the appropriate therapy of eclampsia should be initiated as early as possible. For preventing and treating seizures in eclamptic patients magnesium sulphate is the drug of choice, as it is associated with a significant reduction of recurrent seizures, the risk for pneumonia, and admission to an intensive care unit. Mortality rates were also found to be substantially lowered compared to diazepam and tended to be reduced compared to phenytoin treatment. This is quite remarkable, since neurologists have a tendency toward using these classic anticonvulsants in patients with seizures that are due to other causes than eclampsia.[3]

IV. Conclusion

Although etiologic features of eclampsia might be poorly understood, recognition is clinically obvious and highly alarming once seizures occur. Most common in pregnancy near term, but with onset generally between 20 weeks of gestation and the first 48 hours postpartum, eclampsia is characterized by volatile hypertension, proteinuria, edema, and seizures. Convulsions commonly occur after the onset of one or more isolated prodromal symptoms (headache, visual change, epigastric pain, facial or hand edema, or sudden weight gain), or they can accompany the emergence of overt preeclampsia in women receiving regular obstetric care. Yet there can be absolutely no premonitory warning symptomatology or documented blood pressure increase until just before convulsions, as was the instance in this case. So eclampsia should be the diagnosis until proven otherwise.

References

[1]. Gupte Sanjay, Wagh Girija. Preeclampsia-Eclampsia. The Journal of Obstetrics and Gynaecology of India 2014; 64(1):4-13.
[2]. Veltkamp R, Kupsch A, Polasek J, et al. Late onset postpartum eclampsia without pre-eclamptic prodromi: clinical and Neuroradiological presentation in two patients. J Neurol Neurosurg Psychiatry 2000;69:824-7
[3]. Jens Minnerup, Ilka Kleffner, Heike Wersching, et al., "Late Onset Postpartum Eclampsia: It is Really Never Too Late—A Case of Eclampsia 8 Weeks after Delivery," Stroke Research and Treatment, vol. 2010, Article ID 798616, 4 pages, 2010. doi:10.4061/2010/798616
[4]. Michael W Felz, MD, Daniel B. Barnes, MD, MSE, and Ramon E. Figueroa, MD. Late Postpartum Eclampsia 16 Days After Delivery: Case Report With Clinical, Radiologic, and Pathophysiologic Correlations. J Am Board Fam Pract 2000; 13:39-46.
[5]. Roberts JM, Redman CW. Pre-eclampsia: more than pregnancy-induced hypertension. Lancet 1993; 341:1447-51.

[6]. Ness RB, Roberts JM. Heterogeneous causes constituting the single syndrome of preeclampsia: a hypothesis and its implications. Am J Obstet Gynecol 1996; 175:1365- 70.
[7]. Schobel HP, Fischer T, Heuszer K, Geiger H, Schmieder RE. Preeclampsia - a state of sympathetic overactivity. N Engl J Med 1996; 335:1480 -5.
[8]. Malow BA, Sandson TA, Schwartz RB. Reversible MRI lesions in eclampsia with hydatidiform mole. Neurology 1990; 40:1471-2.
[9]. Mathew R, Raj R S, Sudha P. Late postpartum eclampsia without prodroma. Neurol India 2003;51:539-40
[10]. Lubarsky SL, Barton JR, Friedman SA, et al. Late postpartum eclampsia revisited. Obstet Gynecol 1994;83:502-5