Early postpartum eclampsia complicated by subarachnoid haemorrhage, cerebral oedema and acute hydrocephalus

V Mokoka
Department of Anaesthesiology, Medunsa, South Africa

Eclampsia is one of the dreaded complications of pregnancy as it carries high morbidity and mortality to the mother and baby. Eclampsia is defined as the occurrence of seizures and/or coma on a background of preeclampsia. Eclampsia has also been described without prior development of pre-eclampsia. The incidence of eclampsia depends on a variety of factors and varies widely from region to region. In the United States (USA) the incidence ranges from 0,5-2%,whereas in the United Kingdom (UK) it is less than 0,1%.

Eclampsia accounts for approximately 50,000 maternal deaths worldwide annually. In the United States, the maternal mortality from eclampsia has been reduced with early diagnosis and aggressive management and currently is less than 1%.The incidence in developing countries is 14.4%,with the maternal mortality of approximately 33%. In South Africa 9 out of 1000 deliveries have eclampsia, and the mortality rate is (5-10%). The reported case fatality rates for the condition range from 1% to 20%. Data from developing countries is likely to be underestimated due to deficiencies in health infrastructure and under reporting. The fetal mortality rate has decreased, but still remains at approximately 12%.

Neurological complications due to hypertensive disorders of pregnancy account for 28-50% of maternal deaths. However, the pathogenesis of eclamptic seizure, coma, visual disturbances and localizing neurological defects and cerebral oedema remains poorly understood. Intracranial haemorrhage occurs in 28-73% of these patients and its almost always fatal. The management of eclampsia calls for an emergent multidisciplinary approach.

Case report

A 17 year old primigravida booked at 28 weeks presented to the obstetricians with headache, blurred vision and pain in the right hypochondrium.

She also complained of vomiting, rigors and burning on micturition. On clinical examination she was 34 weeks pregnant and not in labour. Her Glasgow Coma Scale (GCS) was 15/15, and her blood pressure was 160/100mmHg with a pulse rate of 92/min and 3+ proteinuria. A diagnosis of imminent eclampsia was made and treatment was commenced with magnesium sulphate. She received 5g intramuscularly (imi) in each buttock, 4g in 200ml of normal saline infused intravenously over 20 minutes, and a maintenance dose of 5g imi every four hours. Toxicity was excluded by patellar tendon reflex testing.

Blood tests were ordered for urea and electrolytes, uric acid, full blood count, liver function and coagulation profile. The results were normal. The patient was admitted to the obstetric high care unit for observation and stabilization.

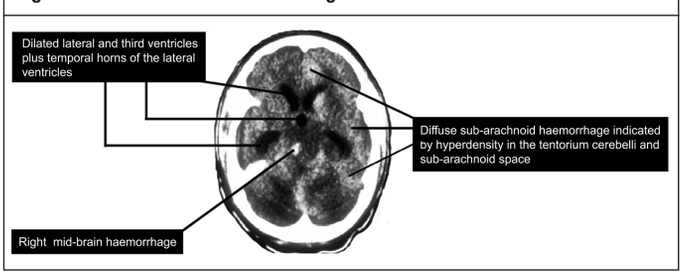
Labour was induced with prostaglandin E2. Eight hours later a 2,1kg infant was delivered vaginally with an APGAR score of 10. The patient's blood pressure had increased to 190/120mmHg after four hours of labour. She received a single dose of 10mg nifedipine sublingually.

Two hours post delivery she complained of dizziness, and her blood pressure was 160/90mmHg. Thirty minutes later she lost consciousness and her blood pressure was recorded at 200/140mmHg. At this time the GCS decreased to 4/15, the right pupil was dilated and both pupils remained reactive to light. The airway was secured with an endotracheal tube. She was sent for an urgent CT scan of the brain. The scan showed a massive subarachnoid haemorrhage (SAH), cerebral oedema and acute hydrocephalus. She was admitted to the intensive care unit (ICU) for controlled ventilation and preparation for an urgent ventriculoperitoneal (VP) shunt. She received 8mg of dexamethasone intravenously in ICU.

Anaesthetic management

On arrival in theatre the patient had a generalised seizure, which was controlled with intravenous midazolam. Anaesthesia was induced with fentanyl and thiopentone, and vecuronium was given for neuromuscular blockade. The depth of anaesthesia was main-

Fig.1 SAH and Brainstem haemorrhage



Correspondence:
Dr V Mokoka
email: dikonelo@samedical.co.za

Fig. 2 Hydrocephalus

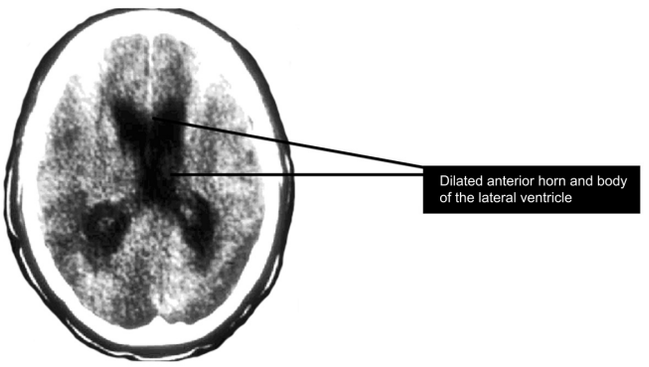


Fig. 3 Pontine Hemorrhage

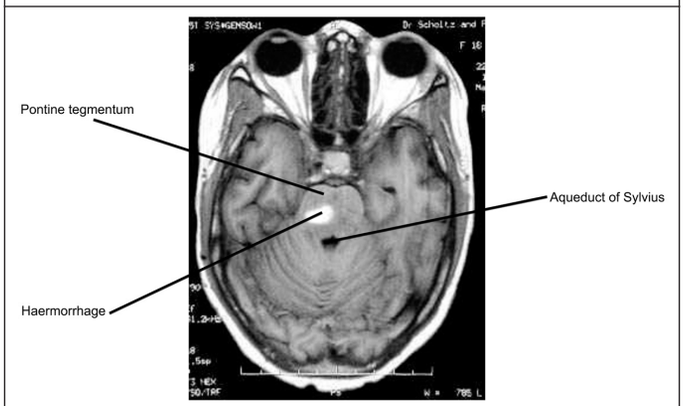
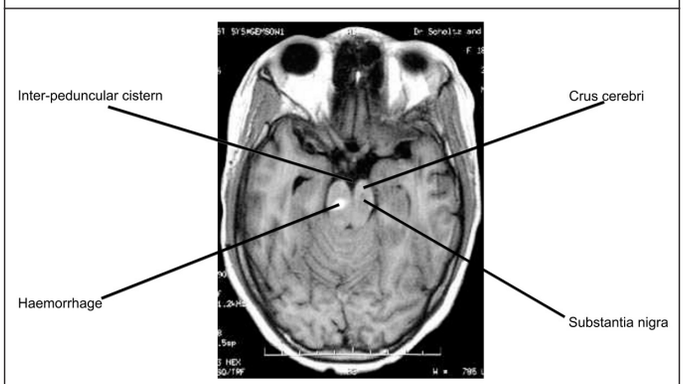


Fig. 4 Midbrain Haemorrhage



tained with 50% N_2O in O_2 and 1.0MAC of isoflurane. Ventilation was controlled with an end-tidal CO_2 maintained within the range 3,5 to 4,5Kpa. Adjunctive therapy included dexamethasone 4mg intravenously and cefuroxime 1,5g intravenously. Normal saline was used as the maintenance fluid. Her blood pressure was maintained within 20% of baseline. In response to intraoperative oliguria she received an intravenous fluid challenge of 500ml normal saline. The central venous pressure (CVP) and urinary output did not improve. She then received another 500ml of normal saline. The CVP remained at 9cmH_2O and the urinary output improved from 50ml to 150ml at end of the operation. The duration of the operation was two hours and she received a total of three litres of normal saline including a litre used for the fluid challenge. She was transferred back to ICU for controlled ventilation and blood pressure control. Combination therapy of amlodipine, α-methyldopa and sodium nitroprusside infusion was instituted to control the blood pressure. Satisfactory blood pressure control was achieved four days post VP shunt and GCS improved to 15/15 with residual right - sided hemiparesis.

Postoperative findings of the magnetic resonance imaging scan (MRI) were pontine haemorrhage and surrounding oedema of the brainstem. The oedema also involved the left tempero-occipital region and basal ganglia, with patchy changes also present in the brainstem and right basal ganglia. Signal changes were also noted in the cerebellar hemispheres. These findings are consistent with the sequelae of ischaemia.

Discussion

The normative ranges for middle cerebral artery (MCA) velocity, resistance index(RI) and the cerebral perfusion pressure(CPP) have been defined in normal human pregnancy using longitudinally collected data. The results of the study concluded that MCA systolic velocity decreased (24%) as did the mean velocity (17%). The diastolic velocity did not change significantly. The MCA RI decreased by 19% and the pulsatility index (PI) decreased by 25%. The MCA CPP increased by 52% between 12 and 40 weeks of gestation.

Having defined normal ranges, identification of abnormalities in cerebral haemodynamics during pregnancy is now possible. This may help researchers and clinicians to elucidate etiologies and treatments for pregnancy related pathophysiologic states such as pre-eclampsia. It was previously reported that patients with established pre-eclampsia had a lower PI and RI and an elevated baseline CPP compared with normotensive women.

The CPP changes are most likely secondary changes and may indicate the development of abnormal cerebral autoregulation or simply a resetting of auto regulatory thresholds as a result of sustained hypertension. Cerebrovascular autoregulatory considerations may help to explain the pathogenesis and distribution of abnormalities in patients with hypertensive encephalopathy. Brain perfusion is maintained by an autoregulatory system of the small arteries and arterioles that has myogenic and neurogenic components.

Endothelial damage or vasculopathy may attenuate or abolish the myogenic response and therefore cause breakdown in autoregulation of cerebral blood flow. The perivascular sympathetic nerves which serve to protect the brain if the myogenic response is blunted or overwhelmed as is the case in severe pre-eclampsia, travel in the adventitial layer of the cerebral vessels and are relatively protected from agents that cause endothelial damage.

Since the vertebrobasilar system and posterior cerebral are sparsely innervated by sympathetic nerves, the occipital lobes and other posterior brain regions may be particularly susceptible to breakthrough of autoregulation with elevated systemic pressure. The loss of autoregulatory capacity causes acute hypertensive vasculopathy which in turn leads to seizures.

Intracranial haemorrhage could possibly be due to rupture of blood vessels, caused by the rapid increase in the hydrostatic forces that overwhelm autoregulatory mechanisms. The aetiology of oedema in eclampsia remains unclear. One study concluded that brain oedema in patients with pre-eclampsia-eclampsia syndrome is primarily associated with laboratory based evidence of endothelial damage.

This is the first report of an eclamptic patient with pontine haemorrhage due to eclampsia. The low GCS of this patient and prompt improvement following a VP shunt can only be explained, at least in part by raised intracranial pressure secondary to acute hydrocephalus. The management of an unconscious eclamptic

patient has been described extensively in the literature. The standard management consists of ventilatory support, seizure prophalaxis, pain and blood pressure control. In the management of this patient, specific attention was paid to the central nervous system and securing the airway to improve cerebral oxygenation. Two similar cases were reported in 1986 and 1999.

Surgical intervention in carefully selected patients based on clinical and CT scan findings should lead to clinical improvement if timeously performed as is evidenced by this patient and the one reported in 1986. However, one should appreciate the fact that not all neurological complications of eclampsia can be treated surgically. But the importance of an urgent CT scan of the brain in a comatose eclamptic patient can never be over emphasized to exclude intracranial haemorrhage.

Early use of steroids in these patients may be beneficial. Prompt recovery of this patient supports previous recommendations that close attention to the neurological management of eclamptic patients with low GCS may be beneficial.

Conclusion

This case report emphasizes the importance of early clinical and radiological assessment of the central nervous system in eclampsia, so that surgically remediable causes of a sudden decrease in the level of consciousness may be detected and timeously remedied.

Bibliography

1. *Thomas ST. Neurological aspects of eclampsia. J of Neurological Sciences 1998;155:37-43*
2. *Richards A, Graham D, Bullock R. Clinicopathological study of neurological complications due to hypertensive disorders of pregnancy. J of Neurology Neurosurgery and Psychiatry 1988;51:416-421*
3. *Belfort MA,Tooke-Miller C,Allen JC, Saade GR,Dildy GA, Grunewald C,et al. Changes in flow velocity resistance indices and cerebral perfusion pressure in the maternal middle cerebral artery distribution during normal pregnancy. Acta Obstet Gynecol Scand 2001;80:104-112*
4. *Schwartz RB, Feske SK, Polak JF, DeGirolami U, Iaia A, Becner KM,et al. Preeclampsia- Eclampsia Clinical and Neuroradiographic Correlates and Insights into the pathogenesis of Hypertensive Encephalopathy. Radiology 2000; 217: 371-376*
5. *Belfort MA, Grunewald C,Saade GR,Varner M,Nissel H.Preeclampsia may cause both overperfusion and underperfusion of the brain. Acta Obstet Gynecol;1999;78:586-591*
6. *Belfort MA,Varner MW, Dizon-townson S, Grunewald C, Nissel H. Cerebral perfusion pressure and not cerebral blood flow may be the critical determinant of intracranial injury in preeclampsia A new hypothesis.A J Obstet Gynecol 2002; 187:626-34*
7. *Mwinyoglee J, Amoko DHA, Simelela N, Marivate M. Eclampsia at Ga-Rankuwa Hospital. S Afr Med J 1996; 86:1536-1539*
8. *Moore PJ;Munoz WP.Eclampsia in the black population of the midlands.SAMJ 1985 April; 67:597-599*
9. *Bhagwanjee S, Paruk F, Moodley J, Muckart DJJ. Intensive care unit morbidity and mortality from eclampsia: An evaluation of the Acute Physiology and Chronic Health Evaluation II score and the Glasgow Coma Scale score. Crit Care Med 2000;28:120-124*
10. *Moodley J, Daya P. Eclampsia: a continuing problem in developing countries. Int J Gynecol Obstet 1993;44:9-14*
11. *Obed SA, Wlson JB, Elkins TE. Eclampsia: 134 consecutive cases. Int J Gynecol Obstet 1994;45:97-103*
12. *Konje JC, Obisesan KA, Odukoya, Ladipo OA. Presentation and management of eclampsia. Int J Gynecol Obstet 1992;38:31-35*
13. *Felz MW, Barnes DB,Fgueroa RE. Late Postpartum Eclampsia 16 Days after Delivery: Case Report With Clinical Radiologic and Pathophysiologic correlations. J Am Board Fam Pract 2000;13:39-46*
14. *Bogod DG. A long and dangerous journey maternal mortality in Africa. Anaesthesia 1999;54:1025-1027*
15. *Devitt JH, Noseworthy TW, Shustack A, Hoskins FC, Petruk CK. Acute hydrocephalus and eclampsia. Can Med Assoc J 1986 Feb; 134:371-371*
16. *Levy DM, Jaspan T. Anaesthesia for Caesarean section in a patient with recent subarachnoid haemorrhage and severe preeclampsia. Anaesthesia 1999;54:987-998*
17. *Douglas KA, Redman CWG. Eclampsia in the United Kingdom. BMJ 1994 Nov;309:1395-1399*
18. *Giannina G, Belfort MA, Cruz LA, RVT, Herd JA. Persistent cerebrovascular changes in postpartum preeclamptic women: A Doppler evaluation. Am J Obstet Gynecol 1997; 177:1213-8*
19. *Riskin-Mashia S, Belfort MA, Saade GR, Herd JA. Transcranial Doppler measurement of cerebral velocity indeces as a predictor of preeclampsia. Am J Obstet Gynecol 2002; 187:1667-72*
20. *Bhorat IE, Naidoo DP, Rout CC,Moodley J. Malignant ventricular arrhythmias in eclampsia: A comparison of labetalol with dihydralazine. Am J.Obstet Gynecol 1993;168:1292-6*
21. *Sibai BM, Spinnato JA, Watson DL, Hill GA, Anderson DA. Pregnancy Outcome in 303 Cases With Severe Preeclampsia. Obstet Gynecol 1984;64:319-325*
22. *Samuels B. Postpartum Eclampsia. Obstet and Gynecol 1960 June 15;748-752*
23. *Fugate SR, Chow GE. Eclampsia. eMedicine 2003 May;1-21*
24. *Shah AK. Preeclampsia and Eclampsia. eMedicine Journal 2002 January;3:1-17*
25. *Begum MR, Akhter S, Begum A, Khatun M,Quadir E, Choudhury SB. Conservative Management of eclampsia and severe Preeclampsia - A Bangladesh Experience. Medscape Womens Health eJournal 2002;7:1-7*
26. *Redman CWG. Eclampsia still kills. British Medical Journal 1988;296:1209-1210*
27. *Evans S, Frigoletto FD, Jewett JF. Mortality of Eclampsia. The New England Journal Of Medicine 183 Dec;29:1644-1647*
28. *Richards AM, Moodley J, Graham DI, Bullock MRR. Active management of the unconscious eclamptic patient. British Journal of Obstetrics and Gynaecology1996 June;93:554-562*
29. *Mcacci F, Carignani L, Cioni R, Parreti E, Mignosa M, Piccioli A, et al. Time Course of Recovery and Complications of HELLP Syndrome with Two Different Treatments: Heparin or Dexamethasone. Thrombosis Research 2001;102:99-105*
30. *Vigil-De Gracia P, Garcia-caceres. Thrombocytopenia and mortality by eclampsia. International Journal of Gynecology and Obstetrics 1997;56:61-62*
31. *Onrust S, Santema JG, Aarnoudse JG. Preeclampsia and the HELLP syndrome still cause maternal mortality in the Netherlands and other developed countries; can we reduce it? European Journal of Obstetrics and Gynecology and Reproductive Biology 1999; 82:41-46*
32. *Dziewas R, Stögbauer F, Freund M, Lüdemann P, Imai T, Holzapfel C, et al. Late onset postpartum eclampsia a rare and difficult diagnosis. J Neurol 2002;249:1287-1291*
33. *Lazebnik N, Pazmino R, Dierker L, Takaoka Y, Warf BC. Maternal Intracranial Hemorrhage Complicating Severe Superimposed Preeclampsia. The Journal of Reproductive medicine1989 Oct;34:857-860*
34. *Cheng AY, Kwan A. Perioperative .Management of Intrapartum Seizure. Anaesthesia and Intensive care 1997 Oct;25:535-538*