ORIGINAL ARTICLE

Neurological Complications of Eclampsia

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ABSTRACT

Objective: *Neurological complications contribute significantly to maternal mortality in eclampsia. We studied neurological and obstetric outcome of such patients.*

Material and Methods: *A descriptive study was done at Gynaecology, Neurosurgery and Neurology Units of PGMI Lady Reading Hospital Peshawar, from January 2008 to December 2008. A total of 132 cases of eclampsia were included. Twenty seven cases of eclampsia with neurological complications were studied. We excluded cases with primary neurological abnormalities.*

Results: *During this period one hundred and thirty two patients were admitted with eclampsia. Among 132 cases 27 had neurological abnormalities. One hundred and eight cases were antenatal and twenty four were postnatal. None of them had any antenatal care. Management included initial stabilization followed by early delivery in antenatal cases. Primary anticonvulsant was magnesium sulphate. Phenytoin controlled convulsions in all cases with recurrent seizures. Neurological complications were seen in twenty seven patients. Minor complications like transient impairment of consciousness (97%), blurred vision (22.22%), and Bell's palsy (07.40%) were observed. Serious neurological complications like CVA (11.11%), recurrent seizures (37.03%) and deep coma occurred in (22.02%) of cases. The cesarean section rate was 18%. Fifty four perinatal deaths and five maternal deaths were recorded. Neurological recovery was complete in all survivors.*

Conclusion: *Critical care backup is essential at tertiary referral centers for a large proportion of neurological abnormalities in eclampsia. High mean arterial pressure and accompanying coagulation defects may be key factors in cerebral pathology. CT scan is a simple and effective investigation in these cases. Phenytoin is an effective second line anticonvulsant. Early delivery prevents worsening of systemic status.*

Key Words: *Eclampsia, Neurological complications, CT Scan, Phenytoin, CVA (cerebrovascular accident).*

INTRODUCTION

Preeclampsia is a complex disorder characterized by pregnancy – induced hypertension, proteinurea and oedema occurring after 20 weeks of pregnancy. The clinical presentation varies in severity and multi – organ involvement is common. The manifestations may include pulmonary oedema, oliguria, disseminated intravascular coagulopathy and hepatic hemorrhages. Neurological manifestations of preeclampsia include headache, confusion, hyper-reflexia, visual hallucinations and blindness. Eclampsia has been defined as occurrence of convulsions, not caused by any coincidental neurologic disease, (e.g epilepsy) in women whose condition also meets the criteria for preeclampsia.[1] Preeclampsia becomes eclampsia with the advent of seizures or coma. It occurs after 20 weeks of pregnancy, during labor or within 7 days of delivery. The cerebral lesions causing neurologic features, including seizures presumably occur as a result of cerebral circulatory deregulation. It has variously been proposed that eclamptic convulsions result from intracerebral hemorrhage, hypertensive encephalopathy and cerebral vasospasm.[2,3] Different mechanisms may be operating in different patients, with compounding effects of cerebral hypoxia, intravenous fluid and drug administration, and varying degrees of

hypertension. Typical postmortem cerebral findings are fibrinoid necrosis of vessels, thrombosed precapillaries, perivascular ring hemorrhages, subarachnoid, intraventricular and intracerebral hemorrhages, hypoxic ischemic damage and perivascular microinfarcts.[4,5] Studies with CT scan have shown that eclamptic patients with evidence of cerebral hemorrhage are likely to die as a consequence of their condition such patients may have a profound coagulopathy.[6] Surviving patients are more likely to have either normal scan or patchy, low density areas.

Generalized cerebral oedema has been documented in eclampsia, found on CT scan in unconscious eclamptic patients and has been suggested in patients in whom intracranial pressure was directly monitored and found to be raised.[7]

Brain MRI may identify abnormalities where CT scan has failed to do so. The characteristic neuroradiological features of severe preeclampsia and eclampsia are hypodense lesions on CT scan which show increased T_2 signals on MRI.[8,9] Abnormalities are more common in patients with eclampsia.[10]

MATERIAL AND METHODS

During the period of study 132 cases of eclampsia were admitted, out of these 24 cases had neurological manifestations. Total obstetric admissions were 6398 and total deliveries were 4890. Of these 24 cases were referred to neurosurgery unit, principally because of recurrent seizures, prolonged alteration in sensorium, severe headache and visual loss in varying combination. The patients were evaluated several times till full recovery and were later on followed up in OPD. All patients with neurological manifestations except those who were very serious had CT scan of brain. Imaging studies were repeated between three to six months of discharge.

RESULTS

General

The age range of the subjects varied from 14 – 40 years. Out of 132 cases 108 were antenatal and 24 were postnatal. One hundred and eight were primigravida, sixteen were second gravida and eight were multigravida. None had prior history of renal diseases, diabetes or seizure disorder. Eight patients were known hypertensive. None of them had proper antenatal care. Blood pressure on admission was over 140 mm Hg systolic and 100 mm Hg diastolic in all, with pedal edema and proteinurea.

Table 1: *Demographic Data of Eclamptic Patients.*

Demographics	No 132	Percentage
Antenatal	87	65.90%
Intranatal	21	15.90%
Postnatal	24	18.18%

Table 2: *Age Range.*

14 – 20 years	43	32.57%
21 – 30 years	72	54.54%
31 – 40 years	17	12.87%

All antenatal patients except with breech were managed by induction of labor. Ninety – two patients had vaginal delivery, and sixteen had cesarean section. Indication for cesarean section were failed induction in eight cases, breech presentation in three, fetal distress in three and severe abruption with massive antepartum hemorrhage in two patients. All postnatal patients delivered outside the hospital had normal vaginal delivery.

Table 3: *Mode of Delivery.*

Mode of Delivery	No	Percentage
Normal vaginal delivery	61	56.48%
Instrumental delivery	31	28.70%
Cesarean section	16	14.81%

Perinatal Mortality

Perinatal mortality was almost 40.09%. Thirty – eight babies were stillborn and sixteen had early neonatal death. Eighty percent of those born alive had an apgar score of 7/10 or below.

Maternal Mortality

There were six maternal deaths. Two patients died of cardiopulmonary arrest, three had cerebral hemorrhage and one patient died of severe abruption leading to disseminated intracellular coagulation and HELLP

syndrome. Total maternal deaths during this one year study period were fifty six and eclampsia contributed to 10.70% maternal deaths.

Table 4: *Perinatal Statistics.*

Perinatal Mortality	No 132	Percentage
Babies born alive	78	59.09%
Still births	38	28.78%
Neonatal deaths	16	12.12%

Neurological Evaluation

Table 5: *Neurological Complications.*

Complications	No 27	Percentage
Recurrent seizures	10	37.03%
Deep Coma	6	22.22%
Blurred Vision	6	22.22%
Bell's palsy	2	07.40
Cerebrovascular Accident	3	11.11%

All patients had generalized tonic clonic recurrent seizures (3 or more over 24 hours). These seizures started antepartum in one hundred and eight and postpartum in twenty four cases. At initial neurologic evaluation, all were at varying stages of altered sensorium (GCS 8 – 10) without localizing sign but with hyper – reflexia and bilateral extensor planters. Almost 50 – 60% patients complained of severe headache at some stage of their illness and five patients complained of blurred vision either prior to losing consciousness or after improvement in level of consciousness. The visual problem in all appeared to be cortical in nature. All of them had diffuse retinal edema but no hemorrhages on fundoscopy. None had any meningeal sign. Improvement in sensorium started 36 – 48 hours after seizure control and all were fully conscious after 72 hours. Blurred vision persisted for – 7 days after admission in five patients who had this symptom.

All patients were given magnesium sulphate (Mg SO_4) for seizure control. Seizure control in 13 patients with recurrent fits was achieved with Phenytoin, using a similar protocol as practiced in status epilepticus but at a slower rate.

Blood pressure control was achieved with Nifedipine and Methyldopa and three patients with uncontrolled Blood Pressure required infusion of Glyceryl Trinitrate.

Neurological Evaluation

All the patients with neurological complications had CT scan within 48 hours of admission. The patients who died of suspected cerebral hemorrhage were so serious that they were not sent for CT scan. Three patients died of suspected cerebral hemorrhage and cerebral edema was seen on CT scan of those patients with recurrent seizures or deep coma. Those with temporary blindness had normal CT scan.

DISCUSSION

Transient neurological dysfunction in the form of paraesthesia and motor weakness of less than 72 hrs duration after labour and delivery has been reported to be associated with 18.9 in 10,000 deliveries.[11] Our study showed minor neurological deficit in the form of transient impairment of consciousness, blurring of vision and Bell's palsy. These results are same as studied by ChakravartyA and Chakrabarti SD.[12]

They described that the minor defects are due to cerebral oedema and resolve in 48 – 72 hrs or within few days.

In a study by D Shmorgan WS it was observed that Bell's palsy was associated with preeclampsia and eclampsia, usually occurs in 3rd trimester of pregnancy (22%) rate in preeclampsia. He mentioned cerebral oedema and increased extra cellular volume to be the cause and quoted that this condition resolves in 2 – 4 weeks time.[13]

The results of above mentioned studies regarding minor complications of eclampsia are in consistent with our study.

Major complications including deep coma, recurrent seizures show the severity of cerebral oedema and presence of minor infarcts which can be detected on CT scan and MRI.[14] He also has the same opinion that initial CT and MRI findings resolve after 4 – 6 weeks after initial episode of eclampsia.

CONCLUSION

It is concluded that management of eclampsia and severe preeclampsia must be made through close liaison between obstetrician and the neurologist.[45]

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