CT scan findings in patients of eclampsia

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ABSTRACT

Background: Eclampsia is defined as the occurrence of one or more convulsions in association with raised blood pressure and proteinuria in a pregnant or puerperal woman, usually between 20 weeks’ gestation and the first 48 hours postpartum. Maternal mortality in India due to eclampsia is 2 to 30% and perinatal mortality is 30 to 50%. Abnormal findings on neuroimaging have been noted in as many as 80-90% of women with eclampsia. Most common lesions are seen in parieto-occipital lobes in the distribution of posterior cerebral arteries. This lesion occurs as a result of vasogenic oedema induced by endothelial damage and other changes contributing to pathophysiology of eclampsia. Objectives of present work were to study involvement of different areas of brain in eclampsia and to study maternal and fetal outcomes.

Methods: This is a prospective study done over a period of 6 months the study population were chosen by random sampling who were eclampsia patients admitted through emergency and also indoor patients who develop eclampsia after admission. Total 12 patients were analyzed. CT scan of brain performed after confinement of fetus and after stabilising the mother. Maternal and fetal outcomes were observed in these cases.

Results: 58.33% (7 cases) eclamptic mothers were primigravida and 41.66% (5 cases) were multigravida. Among them 16.66% (2 cases) had postpartum eclampsia while 83.33% (10 cases) had antepartum eclampsia. 75% mothers delivered by vaginal route and 25% mothers undergone LSCS. There were 14 births including 2 twin deliveries, out of which 12 were live births and 2 Intra uterine deaths. On CT Scan Brain stem edema was seen in 9 cases, infarction in 1 case, hemorrhage in 1 case and in 1 case CT was normal. Most common lobe involved is parietal (10 cases) followed by occipital (7 cases), frontal (4 cases), temporal (3 cases).

Conclusions: Early recognition of the disorder and prompt management by control of blood pressure, removal of the offending medications or treatment of associated diseases is essential to prevent irreversible brain damage. CT scan of brain in eclampsia can provide useful intra cerebral information and should be done in cases with severe neurologic manifestations, if possible for every eclamptic mother.

Keywords: CT Scan, Eclampsia, Eclamptic mother, Postpartum eclampsia

INTRODUCTION

Eclampsia is defined as the occurrence of one or more convulsions in association with raised blood pressure and proteinuria in a pregnant or puerperal woman, usually between 20 weeks’ gestation and the first 48 hours postpartum. In contrast, late postpartum encephalopathy (LPE) occurs between 48 hours and 1 month postpartum, frequently in women who have had a normal pregnancy and delivery and have no signs of a preeclamptic syndrome. Hospital incidence in India is between 1 in 500 to 1 in 30. It occurs more commonly in...
primigravidae, nearly 75% of all eclampsia. 50% of eclampsia are seen from 36 weeks of gestation to term. Convulsions in eclampsia occur 50% in antenatal period, 30% intrapartum, 20% in postpartum period. Maternal mortality in India due to eclampsia is 2 to 30% and perinatal mortality is 30 to 50%.  

Cerebral complications are the major cause of deaths in eclampsia. Cerebral complications have no permanent neurologic deficits, and there is evidence which suggests that the neuropathophysiology of eclampsia may be related to changes in cerebral blood flow. Proposed mechanisms include hypertensive encephalopathy, cerebral vasospasm, cerebral edema and petechial hemorrhage. Abnormal findings on neuroimaging have been noted in as many as 80-90% of women with eclampsia.

Most common lesions are seen in parieto-occipital lobes in the distribution of posterior cerebral arteries. This lesion occurs as a result of vasogenic oedema induced by endothelial damage and other changes contributing to pathophysiology of eclampsia.

Imaging is interesting for a better understanding of the pathophysiology of eclampsia, but in the clinical practice, the decision to perform CT or MR imaging should be more restrictive. Patients presenting with history of uncomplicated eclampsia, without focal neurological deficit should not be investigated by these techniques.

Patients with focal neurologic deficit, or signs of mass effect, or decrease in the level of consciousness should undergo CT scan as a first choice, in order to exclude haemorrhagic lesions or other major complications. Cerebral CT may be normal or may reveal transient white matter hypodensities.

Occasionally, haemorrhagic lesions can be found in more severe forms. Goal of CT scan was to define abnormalities that may be treated and decreases the morbidity and mortality associated with the condition.

METHODS

Patients admitted at our institution with fits were observed in order to study involvement of different areas of brain in CT scan over a period of 6 months. The study population were chosen by random sampling who were eclampsia patients admitted through emergency and also indoor patients who develop eclampsia after admission. Maternal and fetal outcomes were observed in these cases.

Inclusion criteria

Patients with eclampsia (atleast one episode of seizure in women with more than 20 weeks gestation or less than 06 weeks postpartum with blood pressure more than 140mm of Hg systolic and 90mm of Hg diastolic with urine albumin of more than 0.3gm/L).

Exclusion criteria

- Women who were known case of epilepsy.
- Seizures due to metabolic disturbances, space occupying lesions, intracerebral infections, poisoning or trauma.

Total 12 patients were chosen according to inclusion criteria. Basic information including age, parity, gestational age, previous medical or obstetric history was taken. Detailed history of convulsion like duration, time, number of convulsion and premonitory symptoms were sought.

Basic investigations like urine albumin (by dipstick) are measured, complete hemogram, platelet count, serum uric acid, serum creatinine, liver enzymes were sent. Standard MgSO4 protocol was given. If patient was undelivered, assessment of cervix and delivery of fetus done accordingly either by induction of labour or caesarian section. CT scan perfomed after confinement of fetus and after stabilising the mother.

RESULTS

Total 12 patients were included in this study. 58.33% (7cases) eclamptic mothers were primigravida and 41.66% (5 cases) were multigravida. Among them 16.66% (2 cases) had postpartum eclampsia while 83.33% (10 cases) had antepartum eclampsia. 75% mothers delivered by vaginal route and 25% mothers undergone LSCS. There were 14 births including 2 twin deliveries, out which 12 were live births and 2 Intra uterine deaths.

On CT Scan- Brain stem edema was seen in 9 cases, infarction in 1case, hemorrhage in 1case and in 1 case CT was normal. Most common lobe involved is parietal (in 10 cases) followed by occipital (7 cases), frontal (4 cases), temporal (3 cases).

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypodense</td>
<td>9 (75%)</td>
</tr>
<tr>
<td>Infarction</td>
<td>1 (8.33%)</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>1 (8.33%)</td>
</tr>
<tr>
<td>Normal</td>
<td>1 (8.33%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lobs involved</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parietal</td>
<td>10 (83.33%)</td>
</tr>
<tr>
<td>Occipital</td>
<td>7 (58.33%)</td>
</tr>
<tr>
<td>Frontal</td>
<td>4 (33.33%)</td>
</tr>
<tr>
<td>Temporal</td>
<td>3 (25%)</td>
</tr>
</tbody>
</table>

Table 1: Distribution of lesions as seen on CT scan.

Table 2: Involvement of lobes in eclampsia.
DISCUSSION

Preeclampsia/eclampsia is considered to be primarily a placental disorder. Both poor placentation as well as hyperplacentosis is associated with this condition. Vasospasm which follows vasoconstriction as a result of severe hypertension is thought to cause local ischemia, arteriolar necrosis and disruptions of blood brain barrier which leads to cerebral oedema. It is possible that both vasoconstriction as well as forced vasodilatation causes cerebral edema. These are due to irregularities in the auto regulation of cerebral circulation.

Disruption of the blood-brain barrier occurs due to both the hypertension-induced capillary damage and the immune-mediated endothelial dysfunction. This leads to extravasations of red cells and plasma proteins into perivascular space causing cerebral edema. Cerebral vasospasm, produced by a combination of reaction to hypertension, prostaglandin deficiency, defects in the NOS gene (coding for nitric oxide synthase) and endothelial damage, play an important role, producing ischemia and infarction in the brain tissue.

The impaired blood coagulation system and the abnormalities and deficiency of platelets predispose to intra-cranial bleeds. Thus, a varied picture of cerebral pathology showing evidences of cerebral oedema, microinfarcts, cortical petechiae and pericapillary haemorrhages is observed in the brains of patients with pre-eclampsia or eclampsia, which clinically manifest as headache, visual disturbances, confusion and seizures. Characteristic lesion locations are parietal and occipital lobes, followed by the frontal lobes, the inferior temporaloccipital junction, and the cerebellum.

A study of 76 patients by McKinney AM et al, showed that the incidence of regions involvement was parieto-occipital 98.7%, temporal 68.4%, thalamus 30.3%, cerebellum 34.2%, brainstem 18.4%, and basal ganglia 11.8%. The incidence of less common manifestations was enhancement 37.7%, restricted diffusion 17.3%, haemorrhage 17.1% and a newly described unilateral variant 2.6%.

Bartynski WS et al, described vasogenic oedema in parietal or occipital regions 98%, frontal lobes 68%, inferior temporal lobes 40%, cerebellar hemispheres 30%, basal ganglia 14%, brainstem 13%, deep white matter 18% and splenium 10%.

CONCLUSION

It is evident from study that brain stem edema was most common cerebral lesion followed by infarction and hemorrhage and parietal lobe was most common affected area. CT scan of brain can provide useful information to detect different brain lesions in eclampsia which may need specific modification in management protocol to prevent long term neurologic sequel and reduce maternal mortality and morbidity; although these parameters are not included in this study.

Hira B and Moodley J have shown that CT scan does change management in 27% of eclamptic mothers which is statistically significant. Early recognition of the disorder and prompt management by control of blood pressure, removal of the offending medications or treatment of associated diseases is essential to prevent irreversible brain damage.

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REFERENCES

