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COMPUTED TOMOGRAPHY OF BRAIN IN ECLAMPTIC MOTHER: AN OBSERVATIONAL STUDY IN A TERTIARY CENTRE OF SOUTH BENGAL

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ABSTRACT **Introduction:** Neurological complications are one of the commonest causes of maternal mortality and morbidity in eclampsia. 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 the involvement of brain in eclampsia by CT scan.

Methods: The prospective, randomized observational study was carried out in the Departments of Obstetrics & Gynaecology and Radiodiagnosis, Midnapore Medical College over a period of three years. Total 104 eclampsia patients either admitted through emergency or indoor patients who developed eclampsia after admission were chosen randomly. If the patient had not delivered, delivery of fetus was done either by induction of labour or caesarean section. CT scan was performed after delivery of fetus and after stabilising the mother. Maternal and foetal outcomes were recorded in all cases.

Results: Among the 104 patients recruited in the study, edema was the most common type of brain lesion as it was reported in 53 cases (51%), followed by ischemia (14.4%) hemorrhage (10.6%) and granuloma (9.6%) respectively. Normal study was found in 15 women (14.4%). The parietal lobe of brain was most commonly involved (49%).

Conclusion: It is evident from study that 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 sequels and reduce maternal mortality and morbidity

KEYWORDS : Eclampsia, computed tomography, brain imaging

INTRODUCTION:

Eclampsia is defined as the sudden occurrence of generalised tonic clonic seizures and/ or coma, not due to other neurological disorder (e.g. epilepsy) in a woman after 20 weeks pregnancy and during puerperium. Clinically multi-organ disorders like oliguria, pulmonary oedema, disseminated intravascular coagulation (DIC) with haemorrhagic manifestations and hepatic enzyme derangements are common. The neurological manifestations of eclampsia include headache, vomiting, confusion, hyperreflexia, visual hallucinations and blindness, in association with convulsions and coma. Recent studies using computed tomography (CT) and magnetic resonance imaging (MRI) helped in better understanding of brain lesions that may occur during eclampsia.[1]

It remains a significant life-threatening complication of pregnancy, yet there is no reliable test or symptoms for predicting the development of seizure. In the Western world, the incidence of eclampsia is near 1 in 2000 to 1 in 3000 pregnancies, but the incidence is 10-fold higher than that in tertiary referral centres of developing countries where antenatal care is poor.[2]

The different characteristics of cerebral lesions causing neurological features, including convulsions, occur as a result of disruption of cerebral circulatory autoregulation. Two hypotheses have been postulated, which focused on function and changes of the cerebral vasculature and autoregulation of cerebral blood flow due to elevated blood pressure in eclampsia patients.

First, the concept that the cerebral circulation is in a state of over autoregulation in response to elevated cerebral perfusion pressure during preeclampsia that causes ischemia. This is based on brain imaging that shows areas of vasospasm in both cytotoxic and vasogenic oedema in eclampsia and thus ischemic brain injury probably causes seizures and it is reversible in nature because vasogenic edema is predominant.[2]

The second hypothesis regarding the underlying mechanism for the neurological symptoms and oedema formation during eclampsia is that it represents a form of hypertensive encephalopathy in which a rapid rise in blood pressure overcomes the myogenic vasoconstriction of cerebral arteries and arterioles causing loss of autoregulatory capacity and blood-brain barrier (BBB) disruption that promotes vasogenic oedema. Hypertensive encephalopathy has been more recently termed posterior reversible encephalopathy syndrome (PRES) in order to highlight oedema formation in the posterior cerebral cortex but the exact cause is not known. The term PRES is misleading because at least a third of cases also involve other areas of brain as well.[2,3,4]

For minimising the maternal death in eclampsia, we are applying various neuroimaging modalities to evaluate the extent and severity of brain lesions, and early interventions are required according to the image characteristics of neurological changes. The objective of this study is to evaluate characteristics of different neurological findings and different changes observed through brain CT scan images in eclamptic mothers.

METHODS:

A prospective, randomized, observational study was carried out in the department of Obstetrics and Gynaecology, the department of Radiodiagnosis and Critical Care Unit, Midnapore Medical College over a period of three years. The study population were chosen by random sampling of eclampsia patients (at least one episode of seizure in women with more than 20 weeks gestation or less than 6 weeks postpartum with systolic blood pressure (SBP)> 140 mmHg and diastolic > 90 mmHg with urine albumin of more than 0.3gm/L) admitted through emergency and indoor patients who developed eclampsia after admission. Patients with history of epilepsy, metabolic disturbances, space occupying lesions, intracerebral infections, poisoning or trauma were excluded from the study.

Total 104 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) were measured, complete hemogram, platelet count, serum uric acid, serum creatinine, liver enzymes were sent. Standard $MgSO_4$ protocol was given. If the patient had not delivered the baby, assessment of cervix and delivery of fetus was done accordingly either by induction of labour or caesarean section. CT scan was performed after delivery of foetus and after stabilising the mother. Maternal and foetal outcome were observed in all cases.

Association between different CT observations with eclampsia was tested with chi square test with null hypothesis that no association between eclampsia and CT brain changes. P value of <0.05 was considered statistically significant.

RESULTS:

A total of 104 mothers were enrolled in the study. Among them 43 (41.3%) mothers were primigravida and 61 (58.7%) mothers were multigravida. Forty-seven mothers (45.2%) suffered from antepartum eclampsia, 37 (35.2%) and 20 (19.2%) mothers had intrapartum and postpartum eclampsia respectively.

LSCS was the most common mode of delivery as a total of 83 (79.8%) mothers underwent caesarean section. Nineteen mothers (18.3%) delivered vaginally and 2 (1.9%) were delivered by operative vaginal delivery (Obstetric forceps).

One hundred and two women delivered a liveborn baby (98.1%), while 2 (1.9%) women delivered a stillborn child.

Headache prior to the onset of convulsion was reported by 59 women (56.7%). Epigastric pain was complained by 30 (28.8%) women. Diminished vision was reported in 33 (31.7%) mothers. Among the mothers reporting diminished vision, 15 had intrapartum eclampsia and 12 had postpartum eclampsia. On urine Dipstik test, 3+ proteinuria was seen in 51 mothers (49%), 2+ proteinuria in 41 (39.4%) and 1+ proteinuria in 12 (11.5%) cases.

As far as the type of brain lesion was concerned, oedema was the most common type of brain lesion as it was reported in 53 cases (51%). Ischemia was next commonest lesion with 14.4% incidence. In 11 (10.6%) patients intracerebral hemorrhage and in 10 (9.6%) patients granuloma was reported on CT scan. Normal study was found in 15 patients (14.4%). (Table 1). The parietal lobe of brain was most commonly involved with 49% (51 patients) incidence. (Table 2)

Table1: Distribution of lesion as seen on CT scan

BRAIN LESION	FREQUENCY	PERCENTAGE
OEDEMA	53	51
GRANULOMA	10	9.6
HAEMORRHAGE	11	10.6
ISCHEMIA	15	14.4
NORMAL	15	14.4
TOTAL	104	100

Table 2: Distribution of patients as per involvement of different lobes of brain

BRAIN LOBE	FREQUENCY	PERCENTAGE
ALL LOBES	24	23.08
FRONTAL	2	1.92
PARIETAL	51	49.04
TEMPORAL.	6	5.77
OCCIPITO-PARIETAL	6	5.77
NORMAL	15	14.42
TOTAL	104	100

As far as the type of brain lesion and the type of eclampsia was concerned, oedema was most commonly associated with intrapartum eclampsia. Among the 53 cases of oedema, 22 (41.5%) were found in cases of intrapartum eclampsia.

Haemorrhage was also most commonly associated with intrapartum eclampsia. Nine out the 11 cases of intracerebral haemorrhage (81.8%) were seen to be suffering from intrapartum eclampsia. Antepartum eclampsia was most commonly associated with ischemia with 73.3% incidence.

As far as the need for extra injection Magnesium Sulphate was concerned, 5 patients needed extra injection magnesium sulphate. All of those patients had intrapartum 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.[5] Disruption in the auto regulation of cerebral circulation is mainly responsible for cerebral edema.[6]

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.[7] 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.[8]

The impaired blood coagulation system and the abnormalities and deficiency of platelets predispose to intra-cranial bleeds.[9] Thus, a varied picture of cerebral pathology showing evidences of cerebral oedema, micro-infarcts, 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.[10]

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%.[11]

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%.[12]

Thus previous studies have also observed parietal lobe as the primary area of involvement and cerebral edema as most common pathology, similar to our study.[11,12]

Hira B et al. 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.[13]

Thus we can conclude from the present study that 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 sequels and reduce maternal mortality and morbidity.

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