

## Neuroimaging in Eclampsia: A Correlation between CT Imaging and Neurological Presentation

Dr. R. K. Talukdar<sup>1</sup>, Dr. D.J. Gharphalia<sup>2</sup>, Dr. Marami Das<sup>2</sup>, Dr. Manali Jaishwal<sup>3\*</sup>

<sup>1</sup>Professor & HOD, Department of Obstetrics and Gynaecology, Gauhati Medical College & Hospital, Guwahati, Assam, India

<sup>2</sup>Assistant Professor, Department of Obstetrics and Gynaecology, Gauhati Medical College & Hospital, Guwahati, Assam, India

<sup>2</sup>Associate Professor, Department of Neurology, Gauhati Medical College & Hospital, Guwahati, Assam, India

<sup>3</sup>Post Graduate Trainee, Department of Obstetrics and Gynaecology, Gauhati Medical College & Hospital, Guwahati, Assam, India

### Original Research Article

#### \*Corresponding author

Dr. Manali Jaishwal

#### Article History

Received: 13.11.2018

Accepted: 25.11.2018

Published: 30.11.2018



**Abstract:** The objective of the study was to compare computed tomography (CT) imaging of patients with respect to neurological signs. This is a hospital based prospective observational study. 50 patients of eclampsia (both antepartum and postpartum) were studied. CT scan was performed within 48 hours of admission. Statistical analysis was done chi square test and unpaired student's t test. In this study, of the 42 cases with CT scan abnormality, 39 women had neurological signs and symptoms and 3 cases did not have neurological signs and symptoms ( $p=0.000005$ ). The sensitivity, specificity, positive predictive value and negative predictive value were found to be 92.85%, 75%, 95.12% and 66.66 % and the diagnostic accuracy was found to be 90%. 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. Further studies including diagnosis of specific neurological manifestations would emphasize the precise clinical presentations in women with eclampsia. Neuroimaging therefore, will help in modifying management protocol in eclampsia.

**Keywords:** neurological, eclampsia, CT scan, symptoms.

### INTRODUCTION

Eclampsia is associated with considerable morbidity and mortality of the pregnant women. It is defined as occurrence of generalised seizures, not caused by any co-incidental neurological disorder (e.g. epilepsy) in a woman whose condition also meets the criteria for preeclampsia.

According to WHO estimation, eclampsia is the cause of 12 % of all maternal deaths globally [1]. Eclampsia probably accounts for 50,000 maternal deaths a year worldwide [2]. In India, reported incidence of eclampsia varies from 0.179 to 3.7 % and maternal mortality varies from 2.2 to 23 % among all eclamptic women [3-5].

It is observed that during the last 40-50 years, i.e., from 1976 to 2015 the incidence of eclampsia in India has not changed [6].

Data from National Eclampsia Registry published in 2014 showed an incidence of 1.09 % [7].

Though eclampsia is a multi-system complex hypertensive disorder, central nervous system involvement is common in these women and is evident

when specifically evaluated. A common cause of death is CNS pathology like intracerebral hemorrhage and massive cerebral edema. However, the neurological events of eclampsia are usually acute and transient, and long-term deficit is rare in properly managed patients.

Eclampsia and other neurological manifestations like headache, hyperreflexia, visual symptom, somnolence occur due to cerebral circulatory dysregulation [8]. Because preeclampsia and eclampsia are common, they are often the default diagnoses in pregnant and postpartum women who present with acute neurological symptoms. Many other conditions that overlap with eclampsia and with each other in terms of their presentations, including acute ischaemic stroke (AIS), intra cerebral and subarachnoid haemorrhage (ICH and SAH), and cerebral venous sinus thrombosis (CVT) [9].

Neuroimaging has revolutionized visualisation of lesions in eclampsia and other organic conditions and CT is a visualisation tool preferred for initial rapid assessment.

The goal of cerebral imaging is to define abnormalities that may be treated to help decrease the morbidity and mortality associated with the condition.

The aim of this study is

- To evaluate the different neurological changes in brain by neuroimaging (CT) in eclampsia and their relation with different neurological symptoms.
- And show the relevance of these findings in modifying the management protocol to reduce maternal mortality and morbidity and to prevent long term neurological sequelae.

## METHODS

The present study was conducted in the Department of Obstetrics and Gynaecology, Gauhati Medical College and Hospital, Guwahati, India from June 2017 to May 2018. This was a prospective observational study. Women admitted with eclampsia (antepartum or postpartum) and who underwent neuroimaging (CT Scan) within 48 hours were included in the study. A total of 50 women with eclampsia were studied.

### Inclusion criteria

Patients with eclampsia and neurological presentation (at least 1 episode of seizure in a woman at more than 20 weeks gestation or within 6 weeks postpartum with a blood pressure more than 140 mm Hg systolic and/ or 90 mm diastolic with urine albumin more than 0.3 g/dL with neurological symptoms like headache, visual symptoms, somnolence, hyperreflexia, etc).

### Exclusion criteria

- Women who are a known case of chronic hypertension or epilepsy.
- Seizure due to metabolic disorders, space occupying lesions or intracerebral infection.

Eclamptic women were first stabilized with magnesium sulfate and antihypertensive. A written informed consent was obtained from the attendants/ legal guardians of the patients before enrolment. Data such as age, obstetric history, and period of gestation were obtained. Women were evaluated for the clinical presentation including neurological symptoms and signs. These women were subjected to computed tomography scan (plain). Antepartum patients underwent CT scanning with an abdominal shield. CT scan of all patients was carried out using Seimens dual slice CT machine, available at the emergency department of Gauhati medical college and Hospital,

under the Department of Radiology. The neurological presentation and neuroimaging findings were correlated in all antepartum and postpartum eclampsia patients who met the study criteria.

The categorical data was expressed as rates, ratios and proportions, and continuous data was expressed as mean  $\pm$  standard deviation (SD). The comparison was done using chi-square test and unpaired student 't' test. Sensitivity, specificity, positive predictive value and negative predictive value were calculated to find the accuracy of neurological presentation in determining the diagnosis and kappa agreement. A probability value (p value) of less than 0.050 was considered as statistically significant.

## RESULTS

The data was analysed and the final results and observations were as follows. Half of the study population (50%) was aged  $\leq$  20yrs and 38% of the women were aged between 21-25 years. The mean age of the study population was  $21.7 \pm 3.49$  years. 60% of the women presented with antepartum eclampsia while 40% presented with postpartum eclampsia. The youngest age at presentation in our study was 17 years and the oldest patient presented with eclampsia at 35 years.

In the study 80% patients belonged to lower socioeconomic class and 20% belonged to middle class. A total of 64% patients were unbooked.

The mean gestational age in women with antepartum eclampsia was  $34.82 \pm 17.6$  weeks. The shortest period of gestation at presentation was 24 weeks and longest was 40 weeks 5 days. In those with postpartum eclampsia the mean was found to be  $5.3 \pm 8.24$  days postpartum. The earliest presentation was at day 1 puerperium and latest was at day 30 post delivery.

Most of the women in the study (78%) were primiparous. Among the women with antepartum eclampsia, 80% were primiparous and 20% were multiparous. In those with postpartum eclampsia 75% were primiparous while 25% were multiparous.

50% of the women had  $\leq$  3 episodes of seizures while 50% of the women had 4 or more episodes. All the women (100%) had generalized tonic clonic seizures.

As seen in Table-1 the commonest clinical presentation was altered sensorium (46%). In those with antepartum eclampsia, 40% of the women presented with altered sensorium compared to 55% in women who had postpartum eclampsia. The other presentations included unconsciousness (34%), frothing (4%) and

incontinence (2%). 20% patients were conscious at admission.

**Table 1: Clinical Presentation**

Presentation	Antepartum (n=30)		Postpartum (n=20)		Total (n=50)	
	No.	%	No.	%	No.	%
Conscious	7	23.33	3	15	10	20
Unconscious	11	36.66	6	30	17	34
Altered sensorium	12	40	11	55	23	46
Frothing	2	6.66	0	0	2	4
Incontinence	0	0	1	5	1	2

As shown in Table-2, headache was the commonest symptom reported by 46% of the women. Headache was present more common in women with postpartum eclampsia (50%) compared to women with

antepartum eclampsia (43.33%). The other symptoms were blurring of vision (42%) and vomiting (12%). None of the cases had twitching, tingling and numbness, and speech changes.

**Table-2: Symptoms**

Symptoms	Antepartum (n=30)		Postpartum (n=20)		Total (n=50)	
	No.	%	No.	%	No.	%
Headache	13	43.33	10	50	23	46
Blurring of vision	14	46.66	7	35	21	42
Vomiting	3	10	3	15	6	12

In the present study, 36% patients had a GCS score between 9-12 indicating moderate parenchymal involvement. 36.66% antepartum patients had a score  $\leq$  8, compared to 30% postpartum patients. In this study a GCS score  $\leq$  8 was noted in 6 antepartum eclampsia and 4 postpartum eclampsia patients with Hypertensive encephalopathy/PRES.

In the present study 55% of the women with postpartum eclampsia were drowsy compared to 40% antepartum eclampsia patients and 36.67% antepartum patients were unconscious compare to 35% postpartum eclamptics.

80% of the women with antepartum eclampsia had problems with orientation compared to 85% of women with postpartum eclampsia.

**Table-3: Consciousness**

Findings	Antepartum (n=30)		Postpartum (n=20)		Total (n=50)	
	No.	%	No.	%	No.	%
Conscious	7	23.33	3	15	10	20
Unconscious	12	40	11	55	23	46
Drowsy	11	36.67	6	35	17	34

**Table-4: Orientation**

Orientation	Antepartum (n=30)		Postpartum (n=20)		Total (n=50)	
	No.	%	No.	%	No.	%
Disoriented/ not oriented	24	80	17	85	41	82
Well oriented	6	20	3	15	9	18

Increased reflexes (both upper and lower limb reflexes, hyperreflexia) were seen in 30 % cases of postpartum eclampsia and 23.33% cases of antepartum eclampsia. Hyporeflexia was seen in 16.66% antepartum and 15% postpartum eclamptics. Motor paralysis was noted in 3 cases of antepartum and 1 case of postpartum eclampsia. Paraparesis was seen in 1

patient of antepartum eclampsia. Hemiparesis was seen in 1 patient of antepartum eclampsia. One patient of postpartum eclampsia had photophobia.

CT scan revealed no abnormal findings in 16% of the women. In those with abnormal CT scan findings, the commonest diagnosis was hypertensive

encephalopathy/ PRES (70%) followed by intracranial hemorrhage (8%) and multiple infarcts (6%).

**Table-4: Computed Tomography Findings**

Findings	Antepartum (n=30)		Postpartum (n=20)		Total (n=50)	
	No.	%	No.	%	No.	%
Hypertensive encephalopathy/pres	20	66.66	15	75	35	70
No abnormality	4	13.33	4	20	8	16
Hemorrhage	3	10	1	5	4	8
Multiple infarcts	3	10	0	0	3	6

In patients with hypertensive encephalopathy/ PRES the commonest neurological presentation was altered sensorium (58.33%), followed by headache (50%), blurring of vision (38.9%), unconsciousness (27.7%), vomiting (8.3%) and incontinence (2.7%).

In patients with no abnormality on CT, 50% presented with headache, 25% with blurring of vision, 12.5% with vomiting. 1 patient presented with altered sensorium and another with unconsciousness.

Out of the 3 patients with multiple infarcts on CT scan, 2 patients had symptoms of blurring of vision and 1 patient had headache. At admission 2 patients were unconscious and 1 had altered sensorium.

A total of 4 patients showed intracranial haemorrhage on CT scan, 2 patients had complaints of blurring of vision, one had headache and another vomiting. All 4 patients were unconscious at admission.

In this study, of the 42 cases with CT scan abnormality, 39 women had neurological signs and symptoms and 3 cases did not have neurological signs and symptoms ( $p=0.000005$ ). The sensitivity, specificity, positive predictive value and negative predictive value were found to be 92.85%, 75%, 95.12% and 66.66 % and the diagnostic accuracy was found to be 90%.

The kappa value showed good strength of agreement (Kappa = 0.646; SE of kappa = 0.145; 95% confidence interval: From 0.361 to 0.931) suggesting good correlation between the neurological signs and symptoms and CT diagnosis.

**DISCUSSION**

Despite the availability of intensive care units and improved antenatal care such as RCH, NRHM programs, etc., some women still die from eclampsia. Cerebral complications are the major cause of deaths in eclampsia patients, but the neuropathophysiology of eclamptic seizure still remain undiscovered. It is obvious that improving our understanding of the neuropathophysiology of eclamptic seizures is imperative to appropriate management and reduction of morbidity and mortality.

A total of 50 women with eclampsia (antepartum and postpartum) were studied. Half of the study population (50%) was aged  $\leq 20$  yrs. The youngest age at presentation in our study was 17 years and the oldest patient presented with eclampsia at 35 years. The mean age of the study population was  $21.7 \pm 3.49$  years.

Jindal *et al.*, in their study correlating CT and MRI findings with neurological presentation in eclampsia reported that majority of patients belonged to age group between 20 to 25 years (60%) [10]. Another similar study from Hubli, Karnataka, reported mean age of  $22.61 \pm 2.72$  years [11].

Kokila *et al.*, Tumkur, Karnataka, in their study on clinical and neuroimaging findings affecting management in postpartum eclampsia, reported mean maternal age at presentation as 23.89 years (range 18-30 years) [12].

Age has an important influence on the incidence of hypertensive disorders of pregnancy. Development of pre-eclampsia and eclampsia before 20 years of age, have been attributed to due to initial trophoblastic invasion and maternal immune response to it. The failure of the normal invasion of trophoblastic cells leads to mal-adaptation of the spiral arterioles, which are related to the causation of pre-eclampsia/eclampsia. The major risk factor in occurrence of preeclampsia/ eclampsia in women greater than 30 years seems to be the increased villous reaction.

Eclampsia is a common pregnancy associated disorder in this part of the country especially in primigravida and teenagers. The disorder is common in low socioeconomic class. In the present study, 80% patients belonged to lower socioeconomic class (40% lower and 40% lower middle class) and 20% belonged to middle class.

Dahiya *et al.*, reported 12% patients were of lower middle class, 24% patients were of upper lower class and 32 64% patients were belonging to lower class [13].

Halimi S, also noted that the prevalence was high (82.40%) in poor socioeconomic class patients [14].

This value is consistent with our study.

Globally, however, only 64% of women receive antenatal care four or more times throughout their pregnancy [15]. In our study, a total of 64% patients were unbooked. 70% antepartum and 55% postpartum eclampsia cases were unbooked.

Dahiya *et al.*, reported that 48 women (96%) were unbooked cases and only 2 (4%) cases were booked in their study [13]. All 3 patients in this study who expired were unbooked cases.

In a retrospective study conducted at Malda Medical College and Hospital, maternal mortality in eclampsia, it was observed that 90.09% cases were unbooked [16]. Majority of patients in the present study belong to low socio-economic group and were illiterate. Most of them were from rural areas, had no antenatal visit and presented late with complication of eclampsia.

Worldwide studies claim that postpartum eclampsia is more common nowadays [10]. However, studies have shown contradictory results.

In their study on the neurology of eclampsia, Chakravarty *et al.*, reported that seizure onset was more in antepartum than postpartum in their study [17].

In contrast, a study conducted at PGIMS Rohtak reported, that, 66% of the women presented with postpartum eclampsia while 34% had antepartum eclampsia [13].

A clinical study conducted at GGS Hospital, Faridkot, Punjab, it was reported that antepartum eclampsia was that commonest variety (68%) [18].

On the other hand, a study conducted at Hubli, correlating neuroimaging and neurological presentation, showed that 66% of the women presented with postpartum eclampsia while 34% had antepartum eclampsia [11].

In our study, 60% of the women presented with antepartum eclampsia while 40% had postpartum eclampsia. The higher number of patients in the antepartum group again confirms the role of placenta in the etiopathogenesis of eclampsia which in most cases subsides with termination of pregnancy.

Patients with hypertensive encephalopathy/PRES had as many as 7 eclamptic seizures.

Among the patients with postpartum eclampsia in our study, 60% patients presented within 48 hours of delivery and 40% after 48 hours, of which 2 patients presented as late as at day 26 and day 30 puerperium.

Between 14% and 33% of cases of eclampsia occur after delivery [19]. Late postpartum eclampsia has been shown to affect between 4% and 26% of patients with eclampsia and between 28% and 79% of patients with postpartum eclampsia [19-22].

Jens Minnerup *et al.*, reported a case of postpartum eclampsia occurring at 8 weeks postpartum [23]. Half of the women in this study had  $\leq 3$  episodes of seizures while the other 50% of the women had 4 or more episodes. All the women (100%) had generalized tonic clonic seizures.

Ugran *et al.*, found that in their study, 84% of the women had  $\leq 3$  episodes of seizures while 16% of the women had 4 or more episodes. All the women (100%) had generalized tonic clonic seizures [11].

This need not necessarily represent the general pattern as only selected cases were included. In our study, the relationship between number of seizures and neuroimaging findings varied. Patients with hypertensive encephalopathy/PRES has as many as 7 eclamptic seizures. Out of the 4 patients with intracerebral haemorrhage, 3 had more than 4 seizure episodes. In the 3 patients with multiple infarcts on CT scan, 2 patients had more than 4 seizures. These observations show that the number of seizure need not reflect in the neuroimaging findings.

In the present study, most of the women (78%) were primiparous. Among the women with antepartum eclampsia, 80% were primiparous and 20% were multigravidas. In those with postpartum eclampsia 75% were primiparous while 25% were multiparous.

In a similar study conducted at Hubli, most of the women 59% were primiparous. Among the women with postpartum eclampsia 56.06% were primiparous while 43.94% were multiparous. In those with antepartum eclampsia, 64.71% were primiparous and 35.29% were multiparous [11].

The association between primiparity and pre-eclampsia and eclampsia is so widely accepted that it is at the core of several pathophysiological theories. It is proposed that pre-eclampsia and eclampsia are the consequence of a maternal immune reaction against paternal antigens expressed in the placenta and that this reaction might result in defective trophoblast invasion and subsequent placental dysfunction. The lower risk of pre-eclampsia among multiparous women has been attributed to desensitisation after exposure to paternal



antigens in the placenta during previous pregnancies. The lower risk has also been attributed to smoother trophoblastic invasion after modification of maternal spiral arteries during the first pregnancy.

Impaired cerebral blood flow autoregulation is thought to be a major influence in the development of eclampsia due to decreased vascular resistance and increased pressure on the microcirculation that promotes vasogenic edema. Most studies have found that preeclampsia is associated with elevated cerebral perfusion pressure, a result that is not surprising given the appearance of hypertension in those patients [24, 25].

In our study, the mean systolic BP in antepartum group was 159.7 mmHg and diastolic being 108.2 mm Hg. The mean systolic BP in postpartum group being 164.8 mmHg and diastolic being 110.2 mm Hg.

The neurological manifestations of eclampsia consist of seizures and alteration of sensorium or coma on a background of pre-eclampsia. Occasionally there can be focal neurological deficits too.

In our study, the commonest clinical presentation was altered sensorium (46%). In those with antepartum eclampsia, 40% of the women presented with altered sensorium compared to 55% in women who had postpartum eclampsia.

The other presentations in our study included unconsciousness (34%), frothing (4%) and incontinence (2%). 20% patients were conscious at admission. Unconscious state was more common in patients with antepartum eclampsia (36.66%).

Dahiya *et al.*, reported that the commonest clinical presentation was unconsciousness (34%) [13]. Mental changes varying from confusion to coma may develop as a manifestation of cerebral edema. It is usually the main reason behind altered state of sensorium which gradually improves with reduction of the same with time.

In our study, the commonest symptom was headache reported by 46% of the women. Headache was present more commonly in women with postpartum eclampsia (50%) compared to women with antepartum eclampsia (43.33%). The other common symptoms were blurring of vision (42%) and vomiting (12%). Also, 46.66% cases of antepartum eclampsia had blurring of vision compared to 35% cases of postpartum eclampsia.

Other studies by Lubarsky, and Chames reported 83% and 87% of patients with headache [21,

26]. Raised intracranial tension secondary to cerebral edema, is the central cause giving rise to the symptoms of headache and vomiting.

Several clinical symptoms are indicative of eclampsia diagnosis: persistent occipital or frontal headaches, blurred vision, photophobia, epigastric or right upper quadrant pain, and altered mental status. At least one of these symptoms can be seen in 59-75% of the cases [27].

The pathophysiology of eclampsia is explained by two polarized theories [28]. The first called 'overregulation', intense cerebral vasospasm due to explosive increase in blood pressure results in microischemic damage to blood-brain barrier leading to cytotoxic edema. The second called 'breakthrough', loss of autoregulation with resultant dilation leading to subsequent vasogenic edema. Both theories may be operative in eclamptics. Endothelial damage, abnormal placentation, imbalance between vasodilatory and vasoconstrictive prostaglandins are some proposed triggers for vasospasm [29]. Predilection of symptoms and neuroimaging findings in posterior circulation is explained by the lesser number of resistance vessels and a more anterior sympathetic innervation.

In our study, on neurological examination, a GCS score of less than or equal to 8 was seen in 11 antepartum and 6 postpartum patients. All but 1 of these had positive findings on neuroimaging. A total of 18 and 15 patients had GCS scores between 9-12 and 13-15 respectively.

A significantly higher number of women in our study, with postpartum eclampsia were drowsy (55%) compared to 40% antepartum patients. 36.67% antepartum eclampsia patients were unconscious comparable with 35% postpartum patients. With regard to orientation, 80% women with antepartum eclampsia had problems in orientation comparable to 85% women with postpartum eclampsia. Upper and lower limb reflexes were increased in 30 % cases of postpartum eclampsia and 23.33% cases of antepartum eclampsia. Decreased reflexes were seen in 16.66% antepartum and 15% postpartum eclamptics. Motor paralysis was noted in 3 cases of antepartum and 1 case of postpartum eclampsia. Paraparesis was seen in 1 antepartum eclampsia patient. Hemiparesis was also seen in 1 patient of antepartum eclampsia. 1 patient of postpartum eclampsia had photophobia.

The neurological deficits resolved with time in patients who were discharged.

These neurologic abnormalities are probably due to a transient insult, such as hypoxia, ischemia, or edema [30]. Hyperreflexia and clonus which are

warning signs of increased cerebral irritation. Decreased reflexes may be due to electrolyte imbalance such as magnesium toxicity. Ichaemia caused by vasospasm of the posterior cerebral arteries or cerebral edema in the occipital lobes may be causes of the visual symptoms in eclampsia.

Modalities such as CT enable early non-invasive diagnosis. Furthermore, even when the imaging changes are less specific, knowledge of likely possibilities will lead to more appropriate earlier use of imaging.

CT scan in eclampsia patients has revealed a wide spectrum of findings ranging from normal to focal occipital or more widespread lesions which were hypodense and non-enhancing and were ascribed to localised edema [31].

In this study CT scan revealed no abnormal findings in 16% of the women. In those with abnormal CT scan findings the commonest diagnosis was hypertensive encephalopathy/ PRES (70%) followed by intracranial hemorrhage (8%), multiple infarcts (6%).

In a similar study at Hubli, CT scan revealed normal findings in 48% of the women. In those with abnormal CT scan findings the commonest diagnosis was cerebral venous thrombosis (CVT) with infarct (23%) followed by infarct (14%), PRES (6%), hypertensive leucoencephalopathy (HLE) (5%), CVT (3%), cerebral atrophy (1%) [11].

Neuroimaging in eclampsia demonstrates a higher incidence of atypical distribution and the presence of cytotoxic edema than previously thought [32].

In our study, 42 cases had CT findings. Among these significantly higher number women, i.e. 39 women had neurological signs and symptoms and 3 cases did not have neurological signs and symptoms ( $p=0.000005$ ). The sensitivity, specificity, positive predictive value and negative predictive value were found to be 92.85%, 75%, 95.12% and 66.66 % and the diagnostic accuracy was found to be 90% .The kappa value showed good strength of agreement (Kappa = 0.646; SE of kappa = 0.145; 95% confidence interval: From 0.361 to 0.931) suggesting good correlation between the neurological signs and symptoms and CT diagnosis. These findings suggest that, the signs and symptoms at admission predict the neurological involvement and help to predict the likely diagnosis.

CT of 16 % patients had no abnormalities detected. Of these, 50% presented with headache, 37.5% with blurring of vision, 12.5% with vomiting. 1 patient presented with altered sensorium and another

with unconsciousness and 1 was drowsy and disoriented.

In most instances brain CT in eclamptics might be normal due to temporal relationship of scan to seizure. Neuroimaging within a short time after seizure in eclamptics may yield more abnormalities, presumably due to transient nature of lesions. Most common lesions detected on CT in eclampsia are focal areas of cerebral edema in subcortical white matter of parietal and occipital areas.

Hira B and Moodley J have shown that CT scan does change management in 27% of eclamptic mothers which is statistically significant [33].

## CONCLUSION

The limitations of the study were smaller sample size which limited the study from evaluating commonest neurological signs and symptoms in differential diagnosis. . Patients were not randomized, role played by clinical bias and other confounding variables like duration of hypertensive illness or vascular insult before, cannot be ruled out. Another limitation was the duration following admission when neuroimaging was conducted (48 hours). CT scan was the preferred method of evaluation due to cost and availability. Further studies including diagnosis of specific neurological manifestations would emphasize the precise clinical presentations in women with eclampsia. Neuroimaging therefore, will help in modifying management protocol in eclampsia. Some cases of eclampsia are without chronic neurologic sequelae and are transient.

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.

Patients unresponsive to conventional treatment should be screened by neuroimaging to exclude serious morbid CNS pathological conditions and should be considered in all patients who are unconscious, with poor Glasgow Coma Scale and focal neurologic deficit, not responding to conventional therapy.

## REFERENCES

1. World Health Organization, UNICEF. Reduction of maternal mortality: a joint WHO/UNFPA/UNICEF/World Bank Statement.
2. Duley, L. (1992). Maternal mortality associated with hypertensive disorders of pregnancy in Africa, Asia, Latin America and the Caribbean. *BJOG: An*

- International Journal of Obstetrics & Gynaecology*, 99(7), 547-553.
3. Sing, K., Medhi, R., & Bhattacharjee, A. K. (2010). Book of Abstract, 53rd AICOG.
  4. Chandriole, N., Singh, S., & Dhillon, B. S. (2010). Eclampsia, care hood and management practices at tertiary hospital in India (ICMR), New Delhi. *Book of Abstract AICOG, Guwahati*, 33.
  5. Pal, A., Bhattacharjee, R., & Bannerjee, C. (2001). Maternal mortality over a decade in a referral Medical College Hospital, West Bengal. *Indian J Perinatol Reprod Biol*, 4, 10-3.
  6. Pal, A., Bhattacharjee, R., & Bannerjee, C. (2001). Maternal mortality over a decade in a referral Medical College Hospital, West Bengal. *Indian J Perinatol Reprod Biol*, 4, 10-3.
  7. Gupte, S., & Wagh, G. (2014). Preeclampsia–eclampsia. *The Journal of Obstetrics and Gynecology of India*, 64(1), 4-13.
  8. Chakravarty, A., & Chakrabarti, S. D. (2002). The neurology of eclampsia: some observations. *Neurology India*, 50(2), 128.
  9. Edlow, J. A., Caplan, L. R., O'Brien, K., & Tibbles, C. D. (2013). Diagnosis of acute neurological emergencies in pregnant and post-partum women. *The Lancet Neurology*, 12(2), 175-185.
  10. Jindal, M. A., Gaikwad, H. S., Hasija, B. D., & Vani, K. (2016). Comparison of neuroimaging by CT and MRI and correlation with neurological presentation in eclampsia. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*, 2(1), 83-87.
  11. Ugran, S. M., & Donimath, K. V. (2016). Correlation between neuroimaging (CT scan) and neurological presentation in antepartum and postpartum eclampsia. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*, 5(2), 419-424.
  12. Kokila, M. S., & Dwivedi, A. D. (2011). Correlation of clinical and neuroimaging findings affecting management in postpartum eclampsia: A prospective study. *J South Asian Federation Obstet Gynaecol*, 3(3), 125-30.
  13. Dahiya, K., Rathod, M., Rohilla, S., & Dahiya, P. (2016). Correlation between Neuroimaging and Clinical Presentation in Eclampsia. *International Journal of Obstetrics and Gynaecology Research*, 3(6), 375-384.
  14. Halimi, S., & Halimi, S. M. A. (2010). Eclampsia and its association with external factors. *Journal of Ayub Medical College Abbottabad*, 22(3), 110-112.
  15. <http://www.who.int/reproductivehealth/news/antenatal-care/en/>
  16. Das, R., & Biswas, S. (2015). Eclampsia: the major cause of maternal mortality in eastern india. *Ethiopian journal of health sciences*, 25(2), 111-116.
  17. Chakravarty, A., & Chakrabarti, S. D. (2002). The neurology of eclampsia: some observations. *Neurology India*, 50(2), 128.
  18. Kaur, P. (2012). A clinical study in eclampsia on a referral hospital. *J South Asian Feder Obst Gynaecol*, 4(2), 113-115.
  19. Chames, M. C., Livingston, J. C., Ivester, T. S., Barton, J. R., & Sibai, B. M. (2002). Late postpartum eclampsia: a preventable disease?. *American journal of obstetrics and gynecology*, 186(6), 1174-1177.
  20. Hirshfeld-Cytron, J., Lam, C., Karumanchi, S. A., & Lindheimer, M. (2006). Late postpartum eclampsia: examples and review. *Obstetrical & gynecological survey*, 61(7), 471-480.
  21. Lubarsky, S. L., Barton, J. R., Friedman, S. A., Nasreddine, S., Ramadan, M. K., & Sibai, B. M. (1994). Late postpartum eclampsia revisited. *Obstetrics and gynecology*, 83(4), 502-505.
  22. Sibai, B. M., Schneider, J. M., Morrison, J. C., Lipshitz, J. E. F. R. E. Y., Anderson, G. D., Shier, R. W., & Dilts, J. P. (1980). The late postpartum eclampsia controversy. *Obstetrics and gynecology*, 55(1), 74-78.
  23. Minnerup, J., Kleffner, I., Werschling, H., Zimmermann, J., Schäbitz, W. R., Niederstadt, T., & Dziewas, R. (2010). Late onset postpartum eclampsia: it is really never too late—a case of eclampsia 8 weeks after delivery. *Stroke Research and Treatment*, 2010.
  24. Zatik, J., Major, T., Aranyosi, J., Molnár, C., Limburg, M., & Fülesdi, B. (2001). Assessment of cerebral hemodynamics during roll over test in healthy pregnant women and those with pre-eclampsia. *BJOG: An International Journal of Obstetrics & Gynaecology*, 108(4), 353-358.
  25. Oehm, E., Hetzel, A., Els, T., Berlis, A., Keck, C., Will, H. G., & Reinhard, M. (2006). Cerebral hemodynamics and autoregulation in reversible posterior leukoencephalopathy syndrome caused by pre-/eclampsia. *Cerebrovascular Diseases*, 22(2-3), 204-208.
  26. Chames, M. C., Livingston, J. C., Ivester, T. S., Barton, J. R., & Sibai, B. M. (2002). Late postpartum eclampsia: a preventable disease?. *American journal of obstetrics and gynecology*, 186(6), 1174-1177.
  27. Sibai, B. M. (2005). Diagnosis, prevention, and management of eclampsia. *Obstetrics & Gynecology*, 105(2), 402-410.
  28. Cunningham, F. G., & Twickler, D. (2000). Cerebral edema complicating eclampsia. *American journal of obstetrics and gynecology*, 182(1), 94-100.
  29. Felz, M. W., Barnes, D. B., & Figueroa, R. E. (2000). Late postpartum eclampsia 16 days after delivery: case report with clinical, radiologic, and



- pathophysiologic correlations. *The Journal of the American Board of Family Practice*, 13(1), 39-46.
30. Dahmus, M. A., Barton, J. R., & Sibai, B. M. (1992). Cerebral imaging in eclampsia: magnetic resonance imaging versus computed tomography. *American Journal of Obstetrics & Gynecology*, 167(4), 935-941.
31. Beeson, J. H., & Duda, E. E. (1982). Computed axial tomography scan demonstration of cerebral edema in eclampsia preceded by blindness. *Obstetrics and gynecology*, 60(4), 529-532.
32. Junewar, V., Verma, R., Sankhwar, P. L., Garg, R. K., Singh, M. K., Malhotra, H. S., ... & Parihar, A. (2014). Neuroimaging features and predictors of outcome in eclamptic encephalopathy: a prospective observational study. *American Journal of Neuroradiology*.
33. Gurjar, B., & Rawat, R. P. (2017). CT scan findings in patients of eclampsia. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*, 6(8), 3405-3408.