

Intracerebral Changes Detected by CT Scan of Brain in Eclampsia

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Abstract

Objective: To evaluate the different neurological changes in brain in eclampsia by CT scan in relation to neurologic symptoms. **Method:** This is prospective observational study in a tertiary hospital. CT scan of brain is performed within 48 hours of eclampsia after confinement of fetus and after stabilising the mother with standard MgSO₄ protocol. The CT scans of brain are performed with 5mm and 10mm section in the axial plain. **Results:** CT scan of brain shows, 31.6

Index terms— computed tomography, eclampsia.

1 Introduction

clampsia is defined as occurrence of convulsion, not caused by any co-incidental neurological disorder (e.g. epilepsy) in a woman whose condition also meets the criteria for preeclampsia which is a complex multi-organ disorder characterised by pregnancy induced hypertension and proteinuria after 20 wks of pregnancy (exception- gestational trophoblastic disease or multiple pregnancy). The diagnostic criteria are being blood pressure $\geq 140/90$ mm Hg and proteinuria (≥ 300 mg/24 hours or $\geq 1+$ dipstick). The incidence of eclampsia in developing country like India is 1 in 100 to 1 in 1700 deliveries. Cerebral complications are the major cause of deaths in eclampsia; still the neuropathophysiology of eclamptic seizure is mostly unknown. There are two distinct but related types of cerebral pathology. The first is gross haemorrhage due to ruptured arteries caused by severe hypertension of any cause, not necessarily only by preeclampsia or eclampsia. The second type of post-mortem lesions are edema, hyperaemia, ischemic microinfarcts and petechial haemorrhages. The mechanism of the cerebral lesions in eclampsia is unclear. The neurologic manifestations of severe eclampsia are identical to those of hypertensive encephalopathy, which is clinically manifested as generalised tonic-clonic seizure and usually preceded by neurological symptoms like hyper-reflexia, altered sensorium, headache, visual changes and even coma.

There are two theories to describe pathogenesis of hypertensive encephalopathy:-1. Theory of vasospasm: Due to increased hypertension cerebral autoregulation causes intense cerebral vasospasm, followed by local anoxic damage to capillary endothelium and disruption of blood-brain barrier which leads to cerebral edema (cytotoxic edema) 2. Theory of hyperperfusion: Sudden fluctuation in blood pressure exerts pressure on blood vessel wall leads to extravasations of fluid and protein and pericapillary ring haemorrhage (vasogenic edema) with increased blood-brain permeability The recent advances in radiologic imaging including the use of computed tomography (CT) scans and magnetic resonance imaging (MRI), have greatly enhanced our understanding about the correlation between neurologic manifestations and neuro-anatomic and pathological characteristics of eclampsia 4. Harandou M et al 6; showed that 73.68% cases of eclamptic mothers who are still symptomatic after 24 hours have cerebral edema and 10.5% have cerebral hemorrhage and 15.7% have normal CT scan study.

The aim of the study is to evaluate the different neurological changes in brain in eclampsia in relation to neurologic symptoms by CT scan. In this study, CT scan methodology has been adopted because it is less expensive, easily available and results are almost same but MRI reflects more and minute information.

2 II.

3 Methodology

This is a prospective study of CT scan finding of brain on cases of eclampsia admitted in a tertiary hospital. The study population are chosen by random samplings who are patient of eclampsia admitted through emergency and also indoor patients who develop eclampsia after admission. a) Inclusion Criteria If the mother is not already delivered, assessment of cervix and delivery of the fetus is done accordingly either by induction of labour or Caesarean section. CT scan of brain is performed within 48 hours of eclampsia after confinement of fetus and after stabilising the mother. The CT scans of brain are performed with 5mm and 10mm section in the axial plain without intravenous contrast. The CT scan findings are evaluated with clinical characteristics. Level of consciousness is classified according to Glasgow coma scale (<8 severe, 9-12 moderate and >13 minor) 5 . Statistical analysis is performed with aid of Statistical Package for the Social Sciences (SPSS 16, SPSS Inc., Chicago, IL, USA). P value <0.05 is considered for statistical significance.

4 III.

5 Results

Total 38 eclamptic mothers are included in this study. Median age of the mothers is 23 years with standard deviation (SD) of 3.8years. 47.4% eclamptic mothers are primigravida and 52.6% eclamptic mothers are multigravida. Among them 28.9% have postpartum eclampsia, 39.8% have intrapartum eclampsia and 31.6% have antepartum eclampsia. 39.47% mothers delivered by normal delivery and 60.53% mothers have undergone LSCS.

CT scan of brain shows, 31.6% have cerebral edema (diffuse white matter low density areas, patchy area of low density, loss of normal cortical sulci) 23.7% have cerebral infarct (hypo attenuating brain tissue), 7.9% have cerebral haemorrhage (intraventricular/ parenchymal hemorrhage), while 36.8% have no detectable findings. Parietal region of the brain is affected in 67% followed by parieto-occipital area (17%), occipital area (8%) and brain stem (8%). (Figure ??) In this study there is no difference between blood pressure distributions between those who have CT scan findings than those who have not positive CT scan findings. (Figure ??) Figure ??

6 Discussion

In this study cerebral edema is most common lesion (31.6%) detected by CT scan, but most importantly 37.8% eclamptic mothers have no CT scan finding. These finding is corroborative with the findings of Harandou M et al 6 and Akan H et al 22 (Figure 4). Regarding area of distribution parietal and occipital area is the most frequent site of brain lesions in CT scan; supported by observation of Naidu et al 7 . They found parieto-occipital involvement in 97.4% of cases. Sometimes diffuse brain edema is associated with compression or dilatation of 3rd and 4th ventricles. There is two such cases in our study. One rare case of lacunar infarct and another rare subarachnoid haemorrhage is found in this study.

The CT scan findings observed in this study is similar to that observed in patients have severe hypertensive encephalopathy 9 or more similar to its variant Posterior reversible encephalopathy syndrome (PRES) . PRES is characterized by headache, altered mental status, visual disturbances, and seizures. Although hypertensive encephalopathy can arise in patients with conditions in which there is acute systemic hypertension alone, it most commonly occurs in patients also having pre-existing endothelial dysfunction or damage. The Year 2014 E combination of acute hypertension and endothelial damage results in hydrostatic edema -a specific form of vasogenic edema characterised by the forced leakage of serum through capillary walls and into the brain interstitium-which, if severe enough, will be radiographically evident. 10,11 Vasogenic edema is most common finding in eclampsia which explain the reversible nature of most eclampsia. The patients which show no significant finding in CT scan may have very mild vasogenic edema not enough for radiologic detection. The CT scan findings of cerebral infarction are originating from anoxia and cytotoxic edema. This may represents the spectrum of eclampsia ranges from an initially reversible phase of vasogenic edema formation to a later phase of ischemic damage and hemorrhage, which carries a worse prognosis with residual neurologic effect 21 . In fact, laboratory studies of hypertensive encephalopathy, suggest that as vasogenic edema progresses, local tissue pressure increases. This causes a decrease in regional perfusion pressure and a reduction of blood flow to ischemic levels. Subsequently, areas surrounding marked vasogenic edema may progress to infarction and cytotoxic edema. 21 Brain perfusion is maintained by an auto regulatory system of small arteries and arterioles that has myogenic and neurogenic component 11 . In PRES cases direct toxic effect on endothelium or vessel distension decrease the effect of myogenic mechanism. Then neurogenic mechanisms take over regulation of cerebral perfusion. The perivascular sympathetic nerves travel in the adventitial layer of cerebral blood vessels and are relatively protected from agents that cause endothelial damage. Since the vertebra-basilar system and posterior cerebral arteries are sparsely innervated by sympathetic nerves 12 ; the occipital lobe and other posterior brain regions may be particularly susceptible to breakthrough of auto-regulation with elevated systemic pressure. Vasoconstriction induced by sympathetic innervations, moderately protects anterior circulation areas from over perfusion.

Headache is most common neurologic symptoms in this study (68.4%). Akutsu T et al(1992) 13 and Chang WN et al(1996) 14 also get similar results. Eclamptic mothers with visual symptoms and coma have more lesions

102 in parieto-occipital region and parietal region respectively is corroborative with the findings of Chakravarty A,
103 Chakrabarty SD(2002) 15 and Chang WN et al(1996) 14 . Mothers who have develop coma with Glasgow coma
104 scale <8 and with recurrent episode of convulsion (>5 times in number) develop more findings in CT scan. This
105 finding is correlated to study of Richards et al 16 showing severity of edema is related to duration of intermittent
106 seizures. Also, mothers who become fully oriented within 24 hours have significantly less chance of having
107 brain lesions in CT scan. As cerebral mass effect along with diffuse white matter hypo-densities is associated
108 significantly more with coma (p0.034); these mothers recovered later from their eclamptic episodes 17 . In this
109 study preterm eclamptic mother are significantly having pronounced CT scan finding than term mother (p<0.05);
110 as preterm mothers are more severely affected in respect to more prodromal symptoms, multiple seizures, major
111 maternal complication 18 . In our study, there is no statistical significant difference in blood pressure values
112 between cases of positive CT scan findings and cases with normal CT scan findings. Brain edema detected in
113 preeclampsia/eclampsia is thought to be secondary to endothelial injury, rather than hypertension. This finding
114 is correlated with the findings of Schwartz et al. 19 V.

115 7 Conclusion

116 It is evident from this study that cerebral edema is most common cerebral lesions followed by infarction and
117 hemorrhage and parieto-occipital regions of brain is the most common affected area. Although almost 38%
118 eclamptic mother s do not have cerebral lesions, those who have lesions are significantly related to level of
119 consciousness, number of convulsive episode and time taken to recover fully oriented state. Most common
120 neurological finding is headache followed by altered sensorium and hyper-reflexia, visual disturbances and coma.
121 CT scan of brain can provide useful intracerebral information to detect different brain lesions in eclampsia which
122 may have different prognosis with residual effect and may need specific modification in management protocol to
123 prevent long term neurologic sequels and reduce maternal mortality and morbidity; although these parameters
124 are not included in this study. Hira B and Moodley J (2004) have shown that CT scan does change management
125 in 27% of eclamptic mothers which is statistically significant. 20 CT scan of brain in eclampsia can provide useful
126 intracerebral information and should be done in cases with severe neurologic manifestations, if possible for every
eclamptic mother.



Figure 1: Figure

and 90 mm of Hg diastolic with urine albumin of more than 0.3gm/L). both antepartum and postpartum

b) Exclusion Criteria

1. Women who are known case of Hypertension, Epilepsy.

Dr. S. Khandaker ? , Dr. M. Haldar ? & Dr. S. Munshi ? 2. Seizures due to metabolic disturbances, space occupying lesions or intracerebral infections.

Total 38 eclamptic mothers is chosen according to inclusion criteria. Basic information including age, parity and gestational age, previous medical or obstetric history is taken.

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1. Patients with Eclampsia (at least one episode of

seizure in women with more than 20 weeks

gestation or less than 06 weeks postpartum with

blood pressure more than 140 mm of Hg systolic

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Figure 2:

Volume XIV Issue III Version I	1:	Cerebral edema	Cerebral haemorrhage	Cerebral infract
Different areas of brain involvement by CT scan				
Basal ganglia and internal capsule		4.1%	0%	4.1%
Cerebral cortex: occipital		8.3%	0%	0%
Cerebral cortex: parietal		37.5%	8.3%	20.8%
Cerebral cortex: both parieto-occipital		0%	4.1%	12.5%
Among different neurologic symptoms mothers have headache, 18.4% have visual disturbances, 34.2% have altered sensorium with hyperreflexia and 36.6% have coma. Among different neurologic symptoms the CT scan findings are shown in figure 2. Eclamptic mother who presented with visual disturbances (7/38) mostly have brain lesions in parieto-		68.4%	occipital and occipital region (6/7), which is statistically significant.(p<0.005) Similarly, mothers presented coma(14/38) mostly have lesions in parietal cortex (10/14) also, significant.(p 0.002) But no association found with area of lesions and other symptoms like headache and hyperreflexia.	
Figure 2: CT scan findings among different neurologic symptoms				
Neurologic symptoms	CT Scan of brain	Normal CT findings	Cerebral edema	Cerebral infarction
	Altered sensorium and hyperreflexia	38.5%	46.2%	15.4%
	Headache	26.9%	30.8%	30.8%
	Visual disturbances	14.3%	28.6%	42.9%
	Coma	7.1%	35.7%	35.7%
			0%	21.4%

[Note: Year()53.3% eclamptic mothers are preterm (< 37 weeks completed gestational age); among them 42.]

Figure 3:

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- 128 [Richards et al. ()] 'Active management of unconscious eclamptic patient'. A M Richards , J Moodley , D I
129 Graham , M R Bullock . *Br J Obstet Gynaecol* 1986. 93 p. .
- 130 [Teasdale and Jennett ()] 'Assessment of coma and impaired consciousness'. G Teasdale , B Jennett . *Lancet*
131 1974. p. .
- 132 [Beausang and Bill ()] 'Cerebral circulation in acute arterial hypertension: protective effects of sympathetic
133 nervous activity'. L M Beausang , A Bill . *Acta Physiol Scand* 1981. 111 p. .
- 134 [Barton and Sibai ()] 'Cerebral pathology in eclampsia'. J R Barton , B M Sibai . *Clin Perinatol* 1991. 18 p. .
- 135 [Richards et al. ()] 'Clinicopathological study of neurological complications due to hypertensive disorders of
136 pregnancy'. A M Richards , D Graham , R Bullock . *J Neuro Neurosurg Psychiatry* 1988. 51 (3) p. .
- 137 [Lewis et al. ()] 'CT and angiographic correlation of severe neurological disease in toxemia of pregnancy'. L K
138 Lewis , D B Hinshaw , A D Will , A N Hasso , J R Thompson . *Neuroradiology* 1988. 30 p. .
- 139 [Chang et al. (1996)] 'CT and MRI findings of eclampsia and their correlation with neurologic symptoms'. W N
140 Chang , C C Lui , J M Chang . *Zhonghua Yi Xue Za Zhi(Taipei)* 1996 Mar. 57 (3) p. .
- 141 [Koch et al. ()] 'Diffusion-weighted imaging shows cytotoxic and vasogenic edema in eclampsia'. S Koch , A
142 Rabinstein , S Falcone , A Forteza . *AJNR Am J Neuroradiol* 2001. 22 (6) p. .
- 143 [Douglas and Redman ()] 'Eclampsia in the United Kingdom'. K A Douglas , C G Redman . *Br Med Journal*
144 1994. 309 p. .
- 145 [Cunningham et al. ()] 'Hypertensive disorder of pregnancy'. F G Cunningham , P C Macdonald , N F Gant
146 . ' *obstetrics*. 18, F G Cunningham, P C Macdonald, Nf Eds.-Williams Gant (ed.) (Norwalk, Conn) 1989.
147 Appleton & Lange. p. . (th ed.)
- 148 [Schwartz et al. ()] 'Hypertensive encephalopathy: findings on CT, MR-imaging and SPECT-imaging in 14 cases'.
149 R B Schwartz , K M Jones , P Kallina , R L Gajakian , M T Mantello , B Garada , B L Holman . *Am J*
150 *Radiol* 1992. 159 p. .
- 151 [Harandou et al. ()] 'Neuroimaging findings in eclamptic patients still symptomatic after 24 hours: a descriptive
152 study about 19 cases'. M Harandou , N Madani , S Labibe , O Messouak , S Boujraf . *Ann Fr Anesth Reanim*
153 2006. 25 (6) p. .
- 154 [Akutsu et al. ()] 'Neurological and neuroimaging studies of eclampsia'. T Akutsu , F Sakai , T Hata . *Rinso*
155 *Shinkeigaku* 1992. 32 p. .
- 156 [Marques et al. ()] 'Neurological involvement in preeclampsia/eclampsia: the role of neuro-imaging'. R Marques
157 , J Braga , I Leite , C S Jorge . *Acta Med Port* 1997. 10 (8-9) p. .
- 158 [Covarrubias et al. ()] 'Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion
159 weighted MR imaging'. D J Covarrubias , P H Luetmer , N G Campeau . *Am J Neuroradiol* 2002. 23 p. .
- 160 [Schwartz et al. ()] 'Preeclampsia/eclampsia: clinical and neurological correlates and insights into the pathogen-
161 esis of hypertensive encephalopathy'. R B Schwartz , S K Feske , J F Polak . *Radiology* 2000. 217 p. .
- 162 [Schwartz et al. ()] 'Preeclampsia/eclampsia: clinical and neuroradiographic correlates and insights into the
163 pathogenesis of hypertensive encephalopathy'. R B Schwartz , S K Feske , J F Polak . *Radiology* 2000.
164 217 p. .
- 165 [Hira and Moodley ()] 'Role of cerebral computerised tomography scans in eclampsia'. B Hira , J Moodley .
166 *Journal of Obstetrics and Gynaecology* 2004. 7 (24) p. .
- 167 [Naidu et al. ()] 'Single photon emission and cerebral computerised tomographic and transcranial Doppler
168 sonographic findings in eclampsia'. K Naidu , J Moodley , P Corr , M Hoffmann . *Br J Obstet Gynaecol*
169 1997. 104 (10) p. .
- 170 [Akan et al. (1993)] 'The diagnostic value of cranial computed tomography in complicated eclampsia'. H Akan ,
171 M Küçük , O Bolat , M B Selçuk , G Tunali . *J Belge Radiol* 1993 Oct. 76 (5) p. .
- 172 [Chakravarty and Chakrabarty ()] *The neurology of eclampsia: some observations*, A Chakravarty , S D
173 Chakrabarty . 2002. 50 p. .
- 174 [Naheedy et al. (1985)] 'Toxemia of pregnancy: cerebral CT findings'. M H Naheedy , J Biller , M Schiffer , B
175 Azar-Kia . *J Comput Assist Tomogr* 1985 May-June. (3) p. .