CT SCAN FINDINGS IN ECLAMPSIA

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CERTIFICATE

This is to certify that the dissertation on "**CT Scan findings in Eclampsia"** is a bonafide work done by Dr.K.S.Rajarajeswari, in Institute of Obstetrics & Gynaecology, Egmore, Chennai - 8, Madras Medical College, Chennai - 3 under my supervision and guidance in partial fulfillment of the regulation laid down by the Tamil Nadu Dr.M.G.R.Medical University for MD (Obstetrics & Gynaecology) Branch - II Degree Examination to be held in March 2010.

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DECLARATION

I solemnly declare that the dissertation titled ***"CT scan findings in Eclampsia"*** *has been prepared by me at the Institute of Obstetrics and Gynaecology, Madras Medical college, Chennai.*

*This is submitted to T****he Tamilnadu Dr. M.G.R. Medical University****, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Examination in Obstetrics and Gynaecology. This has not been submitted previously by me for the award of any degree or diploma from any other university.*

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INDEX

INTRODUCTION

INTRODUCTION

Hypertensive disorders are one of the most common medical complications of pregnancy. These disorders continue to be a major cause of maternal and perinatal morbidity and mortality worldwide. Pre-eclampsia is a multisystem disorder affecting the cardiovascular, haematological, renal, hepatic and the central nervous system, of which CNS involvement is the most serious complication. [1]

Cerebrovascular involvement is the direct mechanism of death in 40% of these patients. [2]

When promptly recognised and treated, symptoms and radiological changes can be reversed. In some women it can progress to ischemia, massive infarction and death.

Besides clinical presentation, Neuroimaging is the only mode to assess the CNS involvement. Neuroimaging gives a more accurate assessment of the degrees of CNS involvement in these cases. [3]

Vascular changes in organs that occur during pregnancy have been the subject of intense study. Understanding how pregnancy and the

postpartum state affect the structure & function of the cerebrovascular bed may provide important clues as to how eclampsia develops and to potential treatments of this devastating condition.

To identify the prevalence of neurovascular complications and neurovascular changes in Eclampsia a prospective descriptive study was conducted in Institute of Obstetrics and Gynaecology, Madras Medical College, Chennai. CT scan Brain was done for 100 patients of eclampsia and the findings were analysed.

REVIEW OF LITERATURE

Hypertension is one of the most common medical complications that affect both fetal and maternal health and is often life threatening. Multiple maternal organs are affected by hypertension in pregnancy including brain in the form of eclampsia.

Eclampsia is a leading cause of maternal death, with classical neurological symptoms that include headache, nausea, vomiting, cortical blindness, coma and convulsions.

It is well known that pregnancy is associated with significant cardiovascular adaptation of both local and systemic circulation. The changes in cerebral blood flow autoregulation and cerebrovascular resistance enhance BBB permeability and hydrostatic brain edema.

DEFINITION

Eclampsia is defined as seizures that cannot be attributed to other causes in a woman with pre-eclampsia. [4]

Pre-eclampsia is defined as a rise in diastolic B.P of 90mm of Hg or more or systolic BP of 140mm of Hg or more recorded on at

least two occasions six hours apart and the development of proteinuria of 300mg or more in 24 hours or presence of 30mg or more per decilitre in random urine samples.

CLASSIFICATION

NATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAM WORKING GROUP (2000)[5]

GESTATIONAL HYPERTENSION

- BP >/= 140/90 mm of Hg for first time during pregnancy.
- No proteinuria.
- BP returns to normal < 12 weeks postpartum.
- Final diagnosis made postpartum.
- May have other signs and symptoms of pre-eclampsia for example, epigastric discomfort or thrombocytopenia.

PRE-ECLAMPSIA

Minimum criteria

- BP >/=140/90mm of Hg after 20 weeks gestation.
- Proteinuria>/=300mg/ 24 hours or >/= 1+ dipstick

Increased certainty of Pre-eclampsia

- BP >/= 160/100mm of Hg
- Proteinuria 2gm/24 hours or 2+ dipstick.
- Serum creatinine > 1.2mg/dl unless known to be previously elevated.
- Platelets < 1, 00, 000/mm^3
- Microangiopathic hemolysis (increased LDH)
- Elevated ALT or AST
- Persistent headache or other cerebral or visual disturbances.
- Persistent epigastric pain.

ECLAMPSIA

Seizures that cannot be attributed to other causes in a woman with pre-eclampsia.

SUPER IMPOSED PRE-ECLAMPSIA
(ON CHRONIC HYPERTENSION)

- New onset proteinuria >/=300mg/24 hrs in hypertensive women but no proteinuria before 20 weeks gestation.

- Sudden increase in proteinuria or blood pressure or platelet count <1,00,000/mm^3 in women with hypertension and proteinuria before 20 weeks gestation.

CHRONIC HYPERTENSION

- BP > 140/90 mm Hg before pregnancy or diagnosed before 20 wks gestation not attributable to gestational trophoblastic disease.

Or

- Hypertension first diagnosed after 20 weeks gestation and persistent after 12 weeks postpartum.

PROTEINURIA

Proteinuria is an important sign of pre-eclampsia. It reflects the degree of glomerular damage that causes leakage of protein through the basement membrane. The amount of proteinuria is used as an indicator for assessing the severity of pre-eclampsia. Significant proteinuria is described as 300mg per litre or more of urinary protein loss in 24 hours or persistent 30mg/dl (1+ dipstick) in random clean catch sample on at least 2 occasions collected 6 hours apart.

Dipsticks are routinely used to measure proteinuria and the colour changes correspond to

PROTEIN

Trace – 0. 1gm/L

1+ - 0. 3gm/L

2+ - 1. 0gm/L

3+ - 3. 0gm/L

4+ - 10gm/L

INCIDENCE

The incidence of pre-eclampsia is 7-10% depending on the population studied. The incidence of eclampsia in Institute of Obstetrics and Gynaecology, Chennai is 1%. Worldwide approximately 50,000 women are estimated to die annually because of eclampsia. The overall maternal death rate of eclampsia is 2% but varies geographically according to the available healthcare system. The perinatal mortality among babies born to eclamptic mother was 32.7% compared to 10.5% for total perinatal mortality. [6]

PATHOPHYSIOLOGY

Pre Eclampsia / Eclampsia is now considered to be primarily a placental disorder. Both poor placentation as well as hyper placentosis (e. g twin / molar pregnancies) is associated with this condition [22]. It is believed that either an unknown factor (factor X, as some call it) or villous debris from the malformed placenta manages to reach the maternal circulation, eliciting an immunological response from the mother's system [33]. Other variables like dietary factors namely protein and caloric deficiencies, lack of essential fatty acids, deficiencies of magnesium, calcium, zinc and an excess of sodium as well as genetic factors like abnormal alleles for the genes of TNF- alpha, angiotensinogen, Factor V and nitricoxide synthase[NOS] predispose to this event [23-26]. The maternal response is primarily immunological with increased activation and functional abnormalities of maternal neutrophils, high circulating levels of the cell adhesion molecule VCAM-1 and occurrence of immune complexes in the placenta, maternal serum and various organs [27, 28]. The effect of this maternal response is widespread cellular dysfunction with multisystem involvement [29]. There is

endothelial change with increased capillary permeability raised fibronectin levels and electro microscopic evidence of damaged endothelium [30, 31]. Coagulation abnormalities occur caused by increased activation and consumption of platelets, low antithrombin III levels, abnormal prostaglandin metabolism and in a few cases florid DIC[32]. These pathophysiological changes lead to hypertension, edema, proteinuria, bleeding tendencies, renal dysfunction, liver damage and the neurological abnormalities.

The brain is normally protected from extremes of blood pressure by an autoregulation system that ensures constant perfusion over a wide range of systemic pressures, in response to the systemic hypotension, cerebral arterioles dilate to maintain adequate perfusion. Whereas vessels constrict in response to high pressures. Above the upper limit of auto regulation, hypertensive encephalopathy may occur [7].

Two theories have been proposed to account for the clinical and radiological abnormalities associated with these cases. The first theory postulates that hypertensive encephalopathy results from spasm of cerebral vasculature in response to acute hypertension

(i.e. over regulation), resulting in ischaemia and cytotoxic edema involving mainly the border – zone arterial regions [8, 9].

A more recent hypothesis suggests that the syndrome results from breakthrough of auto regulation with passive over distension of cerebral arterioles [10, 11]. This would result in interstitial extravasation of protein and fluid producing focal vasogenic (hydrostatic) edema in peripheral vascular distribution of the involved vessel.

Belfort and associates (1999) found that pre-eclampsia was associated with increased cerebral perfusion pressure counter balanced by increased cerebrovascular resistance with no net change in cerebral blood flow [12].

In eclampsia, presumably due to loss of auto regulation of cerebral blood flow, there was hyper perfusion similar to that seen in hypertensive encephalopathy, unrelated to pregnancy. Women with headache frequently have increased cerebral perfusion.

Apollon and co-workers 2000, Cunningham and twickler 2000 concluded that eclampsia is caused by transient loss of

cerebral auto regulation. This is supported by the evidence of widespread hypodense areas confirmed by CT and MRI studies [13, 14].

Importantly Morris 1997, Rutherford 2003, Zeeman 2004b and all associates have not identified cerebral vasospasm [15, 16, 17].

Zeeman et al found in normal pregnancy cerebral blood flow decreases beginning early in pregnancy and is 20 percent less than non pregnant value by late pregnancy. Subsequently their investigations showed that with pre-eclampsia there is hyperperfusion which contribute to vasogenic edema which they identified using MRI.

The susceptibility of posterior circulation to the lesions is a well known [18, 19] but a poorly understood phenomenon, but one likely explanation involves the regional heterogeneity of the sympathetic vascular innervations.

In experimental studies, the sympathetic innervation of the intracranial arterioles has been shown to protect the brain from marked blood pressure [20]. Moreover, ultra structural studies have

shown that the internal carotid system is much better supplied with sympathetic innervations than in the vertebrobasilar system [21]. Acute hypertension would in this view stimulate the perivascular sympathetic nerves, which would protect the anterior but not the more poorly innervated posterior circulation. This would result in breakthrough of auto regulation with edema mainly in the occipital lobes.

Pregnancy and the post partum state also appear to affect endothelial vasodilator production and smooth muscle reactivity. Cerebral arteries from late pregnant and post partum animals constrict in a concentration dependent manner to serotonin, whereas arteries from non-pregnant animals dilate[2]. Because the dilatation was in the presence of both cyclo oxygenase and NO inhibition, there results suggest that arteries from non-pregnant animals produce endothelium dependent hyperpolarising factor in response to serotonin that is not present in arteries during pregnancy or the post partum state that are more dominated by NO. The significance of this change of vasodilator production by the cerebral endothelium is unclear; however it is possible that these changes

contribute to hemodynamic alteration during pregnancy similar to the periphery.

ANATOMICAL PATHOLOGY

There are 2 distinct types of cerebral pathology based on hypertension. The first is gross haemorrhage due to ruptured arteries caused by severe hypertension. Pre-eclampsia is not necessary for their development. The second type of cerebral lesion seen with eclampsia is a more widespread, focal and seldom fatal lesion. They are edema, hyperaemia, ischemia, thrombosis and haemorrhage.

Sheehan (1950) found haemorrhage ranging from petechiae to gross bleeding in 56 percent of the brains of 48 women with eclampsia, he examined very soon after death. In addition he reported that if the brain were examined within an hour after death, they most often displayed normal firmness without obvious edema [34].

In “Pathology of Toxemia of pregnancy” various histological features of autopsy findings in the brain of women with eclampsia were described.

They are

1. Pia arachnoid haemorrhage
2. Medium – sized haemorrhage in outer white matter
3. Sub cortical haemorrhage
4. Basal ganglia haemorrhage
5. Multiple focal softening (or) petechiae
6. Large haemorrhage in white matter
7. Cortical Petechiae

NEUROIMAGING STUDIES

Zeeman and colleagues (2004 a) reported that nearly all women with Eclampsia have abnormal brain findings [36].

Various techniques used for neuroimaging in eclampsia are Computed Tomography scanning of brain, Magnetic Resonance Imaging of the brain (MRI), Magnetic Resonance (MR) angiography, Magnetic Resonance (MR) Venography, Cerebral angiography, Transcranial Doppler study, Single Photon Emission Computed Tomography (SPECT).

Of these most common and most useful techniques are the CT scan of brain and MRI of brain. Other modalities such as MR angiography findings may be normal or may show reversible cerebral vasospasm.

MR venography findings are usually normal and are helpful in excluding the possibility of dural sinus thrombosis, an important differential diagnosis.

Cerebral angiography shows focal/segmental cerebral vasoconstriction. The venous phase of the study also helps to show normal flow pattern in dural sinuses helping to exclude sinus thrombosis.

Transcranial doppler study is a research tool used in the evaluation of eclampsia. It shows an increased velocity of blood flow in the middle cerebral artery, which is reversible. The change tends to parallel the severity of the clinical syndrome [37].

SPECT scanning reveals perfusion deficits [38].

Characteristic findings in MRI of the brain are hyper intense lesion on T2 – weighted, Fluid Attenuation Inversion Recovery

(FLAIR) or proton weighted sequences affecting the white matter and adjacent grey matter of the occipital / parietal lobes. However deep white matter structures, basal ganglia and white matter of the frontal or temporal lobes and brain stem can also be affected [39, 40].

Characteristic CT scan findings are

1. Normal
2. Cerebral edema
3. Diffuse white matter low density areas
4. Patchy area of low density
5. Occipital white matter edema
6. Loss of normal cortical sulci
7. Reduced ventricular size

Cerebral haemorrhage

1. Intraventricular haemorrhage
2. Parenchymal haemorrhage (high density)
3. Cerebral infarction

4. Low attenuation areas

5. Basal ganglia infarction

Posterior Reversible Encephalopathy Syndrome (PRES) is a potentially devastating neurologic syndrome characterised by rapidly progressive signs and symptoms which includes headache, seizures, consciousness disturbance and (or) visual disturbances. Imaging abnormalities of PRES are predominantly in bilateral posterior circulation regions mainly in the parieto – occipital areas. When unrecognised the patient's condition can progress to ischemia, massive infarction and death. Alternative term such as hypertensive encephalopathy, reversible posterior cerebral edema syndrome and posterior reversible leuko encephalopathy are used to describe this group of disorder [42]. A variety of causes such as pre-eclampsia / eclampsia, uremia, systemic lupus erythematosus (SLE) and immuno suppressant therapy have been associated with this syndrome [41-43].

Schwartz et al (1992) [44] used CT, MR imaging and SPECT imaging in 14 cases with hypertensive encephalopathy. 8 cases were obstetric patients, all with Pre-eclampsia – eclampsia

syndrome. All had abnormalities in the occipital lobes bilaterally involving the sub cortical white matter and often extending to the cortical surface. These lesion appeared hypo dense in CT. Additional abnormalities were noted bilaterally at the parieto occipital junction (three patients), cerebellum (two patients), superior frontal lobes (two patients), and left basal ganglia (one patient). Abnormalities in the occipital lobes were the most significant both radiologically and clinically. One patient with thrombocytopenia had evidence of an associated small haemorrhage. In no patient was an infarct or haematoma shown. The abnormalities in all patients resolved within 1-2 weeks after blood pressure was controlled at which time all neurologic signs and symptoms resolved completely without sequelae.

Chakravarthy et al (2000) [3] studied 19 patients with Eclampsia. All patients had recurrent generalised seizures. Neuroimaging (CT and or MRI) revealed symmetrical occipital lesion in all. One patient had a large pontine lesion. All patients recovered fully without any residual neurological deficit and radiological lesion resolved completely in all except one case.

Seizure control was achieved in all patients with intravenous phenytoin.

Dejana Jovanovic et al (1999)[45] investigated 31 women with Eclampsia in a 11 year period. CT scan brain was done for 29 patients and the pathological findings were discovered in 83% of patients. The most common findings were bilateral multifocal cerebral odema usually in occipital and posterior parts of parietal lobes. In all patients the odema resolved or regressed during two weeks of follow up.

Moodley et al (1993) [46] found that the most common CT scan findings associated with Eclampsia were transient reversible subcortical white matter hypodensities indicative of odema, and in the absence of clinical signs, central nervous system pathology did not alter the management.

Drislane and Wang (1997) [47] reported that CT abnormalities in eclamptics were not always benign and reversible and preceded long term neurological sequelae.

Mei-chun chou et al [48] reviewed 12 cases of PRES and described the imaging findings using MRI & Diffusion weighted imaging (DWI). PRES lesions were typically located in the territories of the posterior circulation, mainly in the parieto-occipital and posterior temporal region with white matter predominance. Only one patient had Eclampsia. MRI of that patient revealed bilateral occipital lobe involvement with white matter predominance over grey matter.

Cunningham F. Gary et al [49] studied the clinical course of 15 women with pre-eclampsia or eclampsia complicated by blindness .13 women underwent computed tomography, of them 8 had low-density areas predominantly in the occipital lobes. 5 out of these 13 patients subsequently underwent magnetic resonance imaging showed corresponding hyperintense lesion in the occipital areas. The conclusion was that cortical blindness associated with pre-eclampsia - eclampsia results from petechial haemorrhages and focal odema in the occipital lobes.

AIM OF THE STUDY

1. To analyse the findings of CT scan of brain in eclampsia.

2. To determine if these findings can be of value in determining the prognosis of this disorder and whether it adds to the understanding of the pathogenesis of this disorder.

3. To identify the prevalence of neurovascular complications in these cases.

MATERIALS AND METHODS.

TYPE OF STUDY

Prospective descriptive study.

PERIOD OF STUDY

August 2008 to August 2009.

SETTING

The study was conducted at INSTITUTE OF OBSTETRICS AND GYNAECOLOGY, EGMORE, CHENNAI. The study was approved by the Board of Ethical committee.

SAMPLE SIZE

Sample size – 100

METHODOLOGY

Screening

All patients in intensive care unit with a provisional diagnosis of Eclampsia were screened for enrolment into the study.

SUBJECT SELECTION CRITERIA

Inclusion criteria

Patients with eclampsia

Exclusion criteria

Patients with known history of

- ❖ Chronic hypertension alone
- ❖ Epilepsy
- ❖ Cerebral tumours
- ❖ Renal disorders.

CONSENT

Informed consent in the form of written consent was obtained from the patients or relatives (in situations where patients is indisposed) after explaining the procedure.

PROTOCOL

This study was conducted in intensive care unit, postoperative ward and complicated postnatal ward.

Patient's medical history, obstetric history with complication, demographic details as detailed in preformed proforma was obtained. CT scan brain was done within 1 week in the postpartum period.

FOLLOW- UP

Patients with positive findings in the CT scan brain were followed up after 2 months.

DATA ANALYSIS AND RESULTS

Computerised tomographic scan of brain was taken for 100 patients with eclampsia. The CT scan findings and the data collected from these 100 patients were analysed. When comparison was done between the patients with positive CT scan findings and patients with negative CT findings Chi-square test was used for statistical analysis.

Table – 1: AGE DISTRIBUTION

AGE IN YEARS	N=100	%
<20	15	15%
20 – 30	78	78%
>30	7	7%

The most common age group in our study was in the range of 20 to 30 years.

78% of the patients were in the age group of 20 to 30 years. Only 7% were in the age group of more than 30 years.

TABLE – 2: PARITY

PARITY	N= 100	%
Primi	68	68%
G2	21	21%
G3	9	9%
G4	2	2%

Primigravida constituted the majority in our study. 68% of the patients were primigravida.

Table – 3: BODY MASS INDEX

BODY MASS INDEX	N=100	%
< 19. 8	4	4%
19. 8-26	69	69%
26-29	17	17%
>29	10	10%

Majority of the patients had a normal body mass index of 19.8 to 26.

69% of the patients in our study had a BMI of 19.8 to 26. 10% of patients had BMI of more than 29.

Table – 4: Gestational Age at Presentation

GESTATIONAL AGE IN WEEKS	***N=100***	***%***
20-28	7	7%
29-36	42	42%
37-40	24	24%
Postpartum	27	27%

Most of the patients were in the antenatal period at the time of presentation. 42% of the patients were in the gestational age of 29 to 36 weeks. 27% of the patients were in the postpartum period.

Table – 5: Duration of Hypertension since Diagnosis

DURATION IN WEEKS	**N=100**	**%**
0 (At presentation)	75	75%
</=4	19	19%
5-12	5	5%
13-20	1	1%

In our study 75% of the patients with eclampsia had hypertension diagnosed only at the time of presentation. Only 1% of patient had hypertension for more than 13 weeks.

Table – 6: IMMINENT SYMPTOMS

IMMINENT SYMPTOMS	N=100	%
Present	73	73%
Absent	27	27%

73% of patients had imminent symptoms. In 27% of patients there were no imminent symptoms.

Table – 7: HAEMOGLOBIN MEASUREMENT

HB IN GMS%	N=100	%
>/=11	14	14%
10 -10. 9	22	22%
7 – 10	62	62%
4 – 7	2	2%

62% of patients with eclampsia in our study had Hb% in the range of 7 to 10gms%.

14% of patients had Hb% above 11gms%.

Table -8: CONVULSIONS

CONVULSIONS	***N= 100***	***%***
ANTEPARTUM ECLAMPSIA	69	69%
1	21	
2	18	
3	16	
>/=4	14	
POSTPARTUM ECLAMPSIA	36	36%
1	22	
2	6	
3	6	
>/=4	2	
BOTH	5	5%

In our study 69% of patients had antepartum eclampsia. Out of 69 patients 14 had 4 or more convulsions. 36% of patients had postpartum eclampsia. Out of the 36 patients 2 had 4 or more convulsions. 5% of patients had both antepartum and postpartum eclampsia.

Table - 9: CT SCAN FINDINGS

FINDINGS	**N = 100**
NORMAL	85
ABNORMAL	15

Out of 100 eclamptic patients 15 patients had positive findings in the CT scan brain.

Table – 10:POSITIVE PAST HISTORY

POSITIVE PAST HISTORY	***NEGATIVE CT FINDINGS GROUP***	***%***	***POSITIVE CT FINDINGS GROUP***	***%***
NO	83	97.6%	13	86.7%
YES	2	2.4%	2	13.3%

p – 0. 045 significant

97.6% in the negative CT findings group and 86.7% in the positive findings group had no positive past history. 13.3% in the positive findings group and 2.4 % in the negative CT findings group had past history of eclampsia or thrombocytopenia.

Table – 11: CONSCIOUS LEVEL

CONSCIOUS LEVEL	***NEGATIVE CT FINDINGS GROUP***	***%***	***POSITIVE CT FINDINGS GROUP***	***%***
Conscious	68	80%	7	46.7%
Drowsy	17	20%	3	20%
Unconscious	0	0	5	33.3%

p – 0. 000 significant

33.3% of patients in the positive CT findings group & none in negative CT findings group were unconscious at the time of presentation.

Table – 12: BLOOD PRESSURE PARAMETERS

BLOOD PRESSURE PARAMETERS	***NEGATIVE CT FINDINGS GROUP***	***%***	***POSITIVE CT FINDINGS GROUP***	***%***
Systolic BP(mm of Hg)				
<140	3	3.5%	0	0
140 – 160	48	56.5%	12	80%
161 – 200	33	38.8%	3	20%
>200	1	1.2%	0	0
Diastolic BP(mm of Hg)				
90 – 100	31	36.5%	10	66.7%
100 – 110	29	34.1%	3	20%
>110	25	29.4%	2	13.3%
MAP (mm of Hg)				
96 -116	24	28.2%	4	26.7%
116 -126	23	27.1%	6	40%
>126	38	44.7%	5	33.3%

Systolic BP p value – 0.371

Diastolic BP p value – 0.088

MAP p value – 0.568

p value were not significant.

56.5% in negative CT findings group & 80% in positive CT findings group had systolic BP in the range of 140 – 160 mm of Hg.

None in the positive CT finding group had systolic BP <140 or >200mm of Hg. 44.7% in negative CT findings group & 33.3% in positive CT finding group had MAP >126mm of hg.

Table – 13: FUNDUS CHANGES

FUNDUS CHANGES	***NEGATIVE CT FINDINGS GROUP***	***%***	***POSITIVE CT FINDINGS GROUP***	***%***
NORMAL	77	90.6%	11	73.3%
GRADE-1	7	8.2%	0	0
GRADE-2	1	1.2%	1	6.7%
GRADE-3	0	0	2	13.3%
OTHERS	0	0	1	6.7%

p – 0.000 significant

Grade -3 fundus changes were seen in 13.3% of patients in positive CT findings group and none in negative CT findings group. 90.6% in negative CT findings group had normal fundus.

Table – 14: PROTEINURIA

PROTEINURIA	*NEGATIVE CT FINDINGS GROUP*	*%*	*POSITIVE CT FINDINGS GROUP*	*%*
1+	33	38.8%	3	20%
2+	26	30.6%	11	73.3%
3+	15	17.6%	1	6.7%
4+	11	12.9%	0	0

p – 0.015 significant. 73.3% of patients in positive CT findings group and 30.6% in negative CT findings group had proteinuria of 2+.

Table – 15: PLATELET COUNT

PLATELET COUNT Lakhs/cu mm	*NEGATIVE CT FINDINGS GROUP*	*%*	*POSITIVE CT FINDINGS GROUP*	*%*
< 1	0	0	3	20%
1 – 4. 5	85	100%	12	80%

p – 0.000 significant

20% in positive CT findings group & none in negative CT findings group had platelet count less than 1 lakh/mm^3.

Table – 16: LIVER FUNCTION TEST

LFT	***NEGATIVE CT FINDINGS GROUP***	***%***	***POSITIVE CT FINDINGS GROUP***	***%***
NORMAL	84	98.8%	12	80%
ALTERED	1	1.2%	3	20%

p – 0.001 significant. 20% in positive CT findings group & 1.2% in negative CT group had altered liver function test.

Table – 17: RENAL FUNCTION TEST

RFT	***NEGATIVE CT FINDINGS GROUP***	***%***	***POSITIVE CT FINDINGS GROUP***	***%***
NORMAL	84	98.8%	15	100%
ALTERED	1	1.2%	0	0

p – 0.673 not significant

Only 1.2% in negative CT group & none in positive group had altered renal function test.

Table – 18: MAGNESIUM SULPHATE REGIMEN

MGSO4	***NEGATIVE CT FINDINGS GROUP***	***%***	***POSITIVE CT FINDINGS GROUP***	***%***
LOW DOSE	22	25.9%	2	13.3%
STANDARD DOSE	61	71.8%	11	73.3%
INFUSION	0	0	2	13.3%
NOT GIVEN	2	2.4%	0	0

p – 0.006 significant. Only in positive CT group 13.3% were treated with $MgSo_4$ infusion. 73.3% in positive CT group & 71.87% in negative CT group were treated with standard dose regimen.

Table – 19 ANTICONVULSANT

PHENYTOIN	*NEGATIVE CT FINDINGS GROUP*	*%*	*POSITIVE CT FINDINGS GROUP*	*%*
YES	30	35.3%	11	73.3%
NO	55	64.7%	4	26.7%

p – 0.006 significant

73.3% in positive CT group & only 35.3% in negative CT group were treated with phenytoin in addition to $MgSo_4$ regimen.

Table – 20: TYPE OF CT CHANGES

CT CHANGES	***N = 100***	***%***
NORMAL	85	85%
CEREBRAL ODEMA	7	7%
HAEMORRHAGE	5	5%
INFARCTION	2	2%
NOT RELATED	1	1%

85% of patients with eclampsia had normal CT scan findings. Cerebral odema was the most common finding in the positive CT findings group. Out of 15 women 7 had cerebral odema. Next common finding was cerebral haemorrhage (5). 2 patients had cerebral infarction. 1 patient had an unrelated finding i.e. old calcified lesion in parietal region.

Table – 21: AREAS OF BRAIN AFFECTED

AREAS	**N = 15**	**%**
DIFFUSE	4	26.6%
FRONTO-TEMPORAL	2	13.3%
PARIETAL	6	40%
OCCIPITAL	2	13.3%
BRAINSTEM	1	6.6%

Parietal region of the brain was the most affected. 6 (40%) patients had lesion in the parietal region. 4(26.6%) patients had diffuse involvement of the brain. 2(13.3%) patients had fronto-temporal region involvement. 2(13.3%) patients had occipital lobe involvement. 1(6.6%) patient had brain stem lesion.

Table – 22: MATERNAL OUTCOME

MATERNAL OUTCOME	***NEGATIVE CT FINDING GROUP***	***%***	***POSITIVE CT FINDING GROUP***	***%***
RECOVERED	85	100%	7	46.7%
RECOVERED WITH SEQUELAE	0	0	1	6.7%
EXPIRED	0	0	7	46.7%

p – 0.000 significant

All patients who did not have any significant findings in the CT scan brain taken recovered fully. 46.7% of patients in positive CT finding group recovered. Another 46.7% in positive CT finding group expired. 1(6.7%) patient in positive CT finding group recovered with sequelae.

Table – 23: COMPARISON OF MATERNAL RECOVERY WITH TYPES OF CT CHANGES

Types	*Recovered*	*%*	*Recovered with sequelae*	*%*	*Expired*	*%*
NORMAL	85	100%	0	0	0	0
CEREBRAL ODEMA	6	85.7%	0	0	1	14.2%
HAEMORRHAGE	0	0	0	0	5	100%
INFARCTION	0	0	1	50%	1	50%
NOT RELATED	1	100%	0	0	0	0

$p < 0.05$ significant

All 5 patients i.e. 100% who had haemorrhage in brain expired.1 out of 2 patients i.e. 50% who had infarction in brain expired and the other patient with infarction in brain recovered with hemiparesis. 6 out of 7 patients i.e. 85.7% who had cerebral odema recovered.1 patient i.e. 14.2% with cerebral odema expired. 1 patient who had old calcified lesion in the brain recovered.

Table – 24: COMPARISON OF MATERNAL RECOVERY WITH AREAS AFFECTED

Areas	***Recovered***	***%***	***Recovered with Sequelae***	***%***	***Expired***	***%***
DIFFUSE	4	100%	0	0	0	0
FRONTO-TEMPORAL	0	0	0	0	2	100%
PARIETAL	2	33.3%	1	16.7%	3	56%
OCCIPITAL	1	50%	0	0	1	50%
BRAINSTEM	0	0	0	0	1	100%

p – 0. 000 significant

All 4 patients i.e. 100% with diffuse involvement recovered. Both of the 2 patients i.e. 100% with lesion in fronto-temporal region expired. Out of 6 patients with parietal lobe involvement 2 patients i.e. 33.3% recovered, 1 patient i.e. 16.7% recovered with hemi paresis, 3 patient i.e. 56% expired. Out of the 2 patients with occipital lobe involvement 1 patient i.e. 50% recovered, 1 patient i.e. 50% expired. 1 patient who had brainstem involvement expired.

Table – 25: FOLLOW – UP IN ALIVE POSITIVE CT FINDINGS GROUP

FOLLOW-UP	***N=8***	***%***
LESION DISAPPEARED	6	75%
LESION PERSISTED	1	12.5%
LOST FOLLOW-UP	1	12.5%

Out of 8 patients who were alive in the positive findings group, lesion disappeared in 6 i.e. 75%. In 1 patient i.e. 12.5% lesion persisted. 1 patient lost follow-up.

DISCUSSION

In this study 100 women with eclampsia were selected according to the inclusion exclusion criteria and Computed Tomographic Scan of brain was done. The findings obtained were analysed.

In our study (Table -1) the most common age group was 20 to 30 yrs. 78% of patients were in the age group of 20 to 30yrs. 7% of patients were above 30yrs of age. 15% of patients were below 20yrs of age.

In our study (Table-2) majority of the patients were primigravida. 68% of patients were primigravida.

In the study by Milliez et al [50] majority of the patients were multiparas.

In our study (Table-3) 69% of patients had body mass index in the range of 19.8 – 26. That is majority of the patients had normal body mass index. 10% of patients were obese with BMI greater than 29.4% of patients had low BMI of less than 19.8.

In our study (Table-4) majority of the patients were in the antepartum period at the time of presentation. 27% of patients were in the postpartum period at the time of presentation. Majority of the patients (42%) were in the gestational age group of 29 to 36 weeks.

In our study (Table-5) majority had HT diagnosed at the time of presentation i.e. 75% of patients had HT diagnosed only at the time of presentation with eclampsia.

In the study by Chakravarthy et al also, all patients except one did not show any evidence of pre-eclampsia till the last clinical visit.

This observation highlights the magnitude of undiagnosed preeclampsia and also the need for screening and effective treatment.

In our study (Table-6) majority of the patients (73%) had imminent symptoms of headache, vomiting, epigastric pain, blurring of vision, or decreased urine output. 27% of patients had no imminent symptoms.

In our study (Table-7) majority of the patients were anaemic. 62% of patients had Hb% in the range of 7-10 gms%. Only 14% of patients had Hb% greater than 11gms.

In our study (Table-8) majority of the patients (69%) had antepartum eclampsia. 36% of patients had postpartum eclampsia. 5% of patients had both antepartum and postpartum eclampsia. 16 patients had 4 or more convulsions.

In our study (Table -9) 15 patients i.e.15% had positive CT scan findings, remaining 85 patients had normal finding.

According to a study conducted by Dejana Jovanovic et al [45] CT scan brain was done for 29 women with Eclampsia in a 11 year period and pathological findings were discovered in 83% of patients.

In a study conducted by Milliez j et al [50] CT scan brain was done for 44 women with Eclampsia, 18(40%) had pathological abnormalities in CT scan.

In a study conducted by Chakravarthy et al [3] 19 patients with Eclampsia were analysed in a 3 year period. But in this CT or

MRI was used. All had recurrent generalised seizures. All had radiological brain lesion.

The main difference between these studies and our study is that, our study was a prospective study done in a 1 year period. Almost 70% of patients with eclampsia admitted to our hospital were included in the study to find out the prevalence of CT scan abnormalities in these patients.

In order to find out the prognosis and associated factors, patients were divided into 2 groups based on the positive or negative CT findings and the results obtained were compared and analysed.

In our study (Table-10) 13.3% in the positive CT group & none in negative group had positive past of Eclampsia or thrombocytopenia with the p value of < 0.05 which is statistically significant difference was observed between the groups in terms of positive past history.This indicates the severity and recurrence of preeclampsia and eclampsia.

In our study (Table-11) 33.3% of patients in positive CT findings group and none in negative CT group were unconscious at the time of presentation with p value of 0.000 which is statistically significant .

In our study (Table – 12) 56.5% of patients in the negative CT findings group & 80% of patients in positive CT group had systolic BP of 140 – 160 mm of Hg. Majority in both groups had diastolic BP of 90 – 100 mm of Hg. In 44.7% in negative CT group & 33.3% in positive CT group had mean arterial pressure >126mmof Hg. There was no statistically significant difference between the groups in terms of blood pressure parameters.

In the study by Chakravarthy et al all patients had BP over 140mm of Hg systolic and 100mm of Hg diastolic.

In our study (Table-13) Grade–3 hypertensive retinopathy fundus changes were seen in 13.3% in positive CT group & none in negative CT group with the p value of 0.000 which is statistically significant . 88% of patients had normal fundus on examination.

In the study by Chakravarthy et al all patients had fundus changes.

In our study (Table -14) 73.3% of patients in the positive CT findings group and 30.6% in negative CT findings group had proteinuria of 2+. p value is 0.015, thus statistically significant difference was observed between the groups.

In our study (Table-15) 20% of patients in positive CT findings group had platelet count less than 1 lakh per mm^3. None in negative CT group had thrombocytopenia. p value is 0.000, thus statistically significant difference was observed between the groups.

In a study by Hira.B and Moodley [46] 70% had thrombocytopenia.

In our study (Table-16) 20% of patients in positive CT findings group and 1.2% in negative CT group had altered liver function test. p value is 0.001, thus statistically significant difference was observed between the groups.

In the study by Hira.B and Moodley 7% had altered liver function test.

In our study (Table -17) only 1.2% of patients in negative CT findings group & none in positive CT group had renal impairment. p value is 0.672, thus there was no statistically significant difference.

In a study by Zhu XW [51] those patients with renal impairment and retinal changes were found to be more susceptible to cerebral lesions ($p<0.05$). This was observed by comparative study of relationship between either the function of liver or kidney and retinal changes with cerebral lesions.

Our study has also shown that patients with altered liver function and retinal changes were more susceptible for cerebral lesions.

In our study (Table-18) only 2 patients were not treated with magnesium sulphate regimen because of renal impairment in one patient, and because of severe hypotension in another patient. Both of them did not have any positive CT finding. 73.3% in positive CT finding group and 71.8% in negative CT group were treated with standard dose magnesium sulphate regimen.

But in the study by Chakravarthy et al none were treated with magnesium sulphate regimen. All the patients in the study by Chakravarthy et al were managed by neurologists and not by obstetricians.

In our study (Table-19) 73.3% of patients in positive CT finding group and 35.3% in negative CT group needed anticonvulsant phenytoin in addition to $MgSO_4$ regimen for control of seizures. p value is 0.006, thus statistically significant difference was observed between the groups.

In the study by Chakravarthy et al all patients were treated with intravenous phenytoin.

In our study (Table-20) out of 15 patients with positive findings the most common positive CT scan finding was cerebral odema, it was seen in 7 patients (46%). Next common finding was cerebral haemorrhage observed in 5 patients (33%). 2 patients (13.3%) had cerebral infarction. 1 patient (6%) had an old calcified lesion. out of the 7 with cerebral odema in 4 patients the findings were suggestive of Posterior Reversible Encephalopathy Syndrome.

In the study by Chakravarthy et al all 8 patients (100%) had cerebral odema, 3 patients (37%) had cerebral haemorrhage.

In a study by Dejana Jovanovic et al out of 29 patients 83% had pathological findings. The most common findings were cerebral odema.

In the study by Milliez et al out of 18 patients with positive findings 3 had features of cerebrovascular damage, 6 had cerebral odema, 9 had exaggerated cortical sulci suggesting the diagnosis of communicating hydrocephalus with cortical atrophy.

In the study by Moodley et al (1993) most common finding was cerebral odema. In our study also the most common finding was cerebral odema.

In our study (Table-21) parietal region was the most common area affected. 6 patients i.e. 40% had lesion in parietal region. 4 (26.6%) had diffuse involvement. 2(13.3%) had lesion in fronto-temporal region. 2(13.3%) had lesion in occipital region. 1(6.6%) had lesion in brain stem.

In the study by Dejana Jovanovic et al [1] occipital and parietal regions were the most common affected areas.

In the study by Chakravarthy et al occipital region was the most affected. There was no lesion in brainstem or basal ganglia.

In a study by Zhu XW cerebral lesions were involved at cortical and subcortical area of bilateral parietal and occipital lobes, secondly at deep basal ganglia and the superior sagittal sinus.

In a study by Milliez et al none of the patients had diffuse involvement.

As it has been already discussed the cause for such presentation is that acute hypertension would stimulate the perivascular sympathetic nerves which would protect the anterior but not the poorly innervated posterior circulation.

In our study (Table -22) all 85(100%) patients who did not have any positive findings in CT scan brain recovered completely. whereas in the positive CT findings group only 7 (46.7%) patients recovered completely, 1(6.7%) patient recovered with hemiparesis

& 7(46.7%) patients expired. p value is 0.000, thus statistically significant difference was noted between the groups.

In the study by Chakravarthy et al all patients recovered fully. In the study by Milliez et al 3 (16%) out of 18 patients expired, remaining 15 recovered fully. In a study by Hira.B and Moodley out of 30 patients 15(50%) died, 10(33%) recovered, 2(7%) had paresis, 3(10%) had psychosis.

In our study (Table-23) when we compared the maternal outcome with the type of CT scan changes we found that all 5(100%) patients who had haemorrhagic lesion had expired. Out of 7 patients with cerebral odema 6(85.7%) recovered & 1(14.2%) patient whose lesion was suggestive of PRES expired. In the 2 patients with cerebral infarction, 1(50%) recovered with hemiparesis & 1(50%) expired. 1 patient who had a calcified lesion also recovered fully. p value is <0.05, thus statistically significant difference was noted between the groups.

In the study by Milliez et al all 6 patients with cerebral odema recovered, all 9 patients with not related findings also recovered, all 3 patients who had features of cerebrovascular

damage, a total middle cerebral artery thrombosis, a massive Intraventricular haemorrhage, a temperoparietal hematoma expired.

In our study (Table-24) when we compared the maternal outcome with areas of brain affected all 4 (100%) patients who had diffuse involvement recovered. All 2(100%) patients who had frontotemporal region involvement expired. Of the 6 patients with parietal region involvement 2(33.3%) patients recovered, 1(16.7%) recovered with hemiparesis, 3(50%) patients expired. Of the 2 patients with occipital region involvement, 1(50%) recovered, 1(50%) expired. The 1 (100%) patient with brainstem involvement expired. P=0.000 thus statistically significant difference was noted between the groups.

In our study (Table-25) when the alive patients with positive CT findings were followed up, out of the 8 alive patients, lesion disappeared in 6(75%) patients. All the 6 were the patients who earlier had cerebral odema. In 1(12.5%) patient the lesion persisted. That was the patient with old calcified lesion. 1(12.5%) patient lost follow up. That was the patient with infarct in left frontoparietal region who recovered with hemiparesis.

In the study by Chakravarthy et al out of 8, 7 patients had follow up. In all except 1 patient the lesions detected earlier resolved completely. It included both cerebral odema and haemorrhage. In 1 patient with pontine lesion, the lesion persisted suggesting it to be an infarct or a post hemorrhagic gliotic area.

In the study by Dejana Jovanovic et al all patients who had cerebral odema resolved or regressed in two weeks follow up.

SUMMARY

100 patients with eclampsia between August 2008 to August 2009 at the Institute of Obstetrics and Gynaecology, Madras Medical College, Chennai were selected according to the inclusion and exclusion criteria already stated in the methodology were taken for this prospective descriptive study.

Computed Tomography scan of brain was done for these patients within 1 week of postpartum period. Patients with positive CT findings were followed up at 1 to 2 months interval.

The results obtained were tabulated, analysed and summarised as follows.

1. Out of the 100 patients 15% had pathological findings in the Computed Tomography scan of brain taken.

2. Majority of the patients (78%) were in the age group of 20-30 yrs.

3. Majority of the patients (68%) were primigravida.

4. Majority of the patients (69%) had normal body mass index of 19.8 - 26.

5. Most of patients around 70% were in the antepartum period at the time of presentation.

6. Majority of the patients (75%) had diagnosed hypertension only at the time of presentation.

7. Most of the patients (73%) had imminent symptoms.

8. Most of the patients (69%) had antepartum eclampsia.

9. Only in positive CT finding group 13.3% had past history of either eclampsia or thrombocytopenia. p value is 0.04 hence statistically significant.

10. Only in positive CT finding group 33.3% were unconscious at the time of presentation. p value is 0. 000, hence statistically significant.

11. Most common blood pressure was systolic 140-160mm of Hg in around 60% and diastolic 90-100mm of Hg in both groups.

12. Only in the positive CT finding group 13.3% had Grade–3 hypertensive retinopathy changes. p value is 0.000, hence statistically significant.

13. All patients had proteinuria. In the positive CT findings group 73.3% had proteinuria of 2+. p value is 0.015, hence statistically significant.

14. Only in the positive CT findings group 20% had thrombocytopenia. p value is 0.000, hence statistically significant.

15. 20% of patients in positive CT findings group had altered liver function test. p value is 0.001, hence statistically significant

16. Cerebral odema 46% was the most common positive CT finding. Parietal region 40% was the most common area of the brain affected.

17. Maternal mortality was high 46.7% in the positive CT finding group. p value is 0.000, hence statistically significant.

18. 85.7% of patients with cerebral odema recovered and in all of them the lesion resolved completely. 100% of patients with cerebral haemorrhage expired.

19. 100% of patients with diffuse involvement recovered. 100% of patients with frontotemporal or brainstem involvement expired. p value is 0.000 hence statistically significant.

CONCLUSION

1. 15% of patients with eclampsia had pathological abnormalities of brain detected through Computed Tomography scan of brain.

2. Maternal mortality was high in patients with positive CT scan brain findings.

3. Patients with altered liver function, retinal changes, and thrombocytopenia are more prone for developing cerebral lesions.

4. Cerebral odema was the most common pathological abnormality detected. In all but one of these patients recovery and resolving of the lesions were complete.

5. Maternal mortality was high in patients with cerebral haemorrhage, involvement of frontotemporal region or brainstem region. Hence prognosis in these patients is poor.

6. Since majority of patients had hypertension diagnosed only at the time of presentation the need for effective screening of hypertension and its management is emphasised.

7. Thus it is emphasised that CT scan of brain should be included in the investigation protocol for eclampsia if not for all at least for those patients with complications.

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NAME:

AGE: REGISTER NO:

ADDRESS: SOCIOECONOMIC STATUS:

DATE OF ADMISSION:

BOOKED/UNBOOKED: HEIGHT: WEIGHT:

PARITY:

GESTATIONAL AGE/POSTPARTUM:

HYPERTENSION DIAGNOSED AT:

IMMINENT SYMPTOMS: YES NO

- Headache
- Vomiting
- Blurring of vision
- Epigastric pain
- Oliguria

NUMBER OF SEIZURES

Antepartum

Intrapartum

Postpartum

PAST HISTORY: Hypertension

Preeclampsia

Eclampsia

Thrombocytopenia

CONSCOUS LEVEL: Conscious

Drowsy

Unconscious

BLOOD PRESSURE: SYSTOLIC DIASTOLIC MAP

At admission

Maximum BP

FUNDUS EXAMINATION: NORMAL ABNORMAL

URINE ALBUMIN:

HB%

PCV%

PLATELETS:

RENAL FUNCTION TEST: Normal

Abnormal

LIVER FUNCTION TEST: Normal

Abnormal

MAGNESIUM SULPHATE THERAPY:

ANTICONVULSANT:

CT SCAN FINDINGS:

PATIENT’S CONDITION:

Recovered

Recovered with sequelae

Expired

FOLLOW UP:

ABBREVIATION

ALT – Alanine transaminase

AST – Aspartate aminotransferase

BBB – Blood brain barrier

BMI – Body mass index

CNS – Central nervous system

CT SCAN – Computerised Tomographic Scan

DIC – Disseminated intravascular coagulation

DWI – Diffusion weighted imaging

FLAIR – Fluid attenuation inversion recovery

GA – Gestational age

HT – Hypertension

LDH – Lactate dehydrogenase

MgSo4 – Magnesium sulphate

MRI – Magnetic Resonance Imaging

NO – Nitric oxide

NOS – Nitric oxide synthase

PRES – Posterior Reversible Encephalopathy syndrome

SLE – Systemic Lupus Erythematosus

SPECT – Single photon emission computed tomography

TNF – Tumour necrosis factor

VCAM – Vascular Cell Adhesion Molecule

KEY TO MASTER CHART

AP – Antepartum

BMI – Body mass index

PRIMI – Primigravida

GA – Gestational age

PP – Postpartum

H – Headache

V – Vomiting

E – Epigastric pain

B – Blurring of vision

C – Conscious

D – Drowsy

UC – Unconscious

MAX BP – Maximum Blood pressure

MAP – Mean arterial pressure

FUNDUS – Fundus changes

N – Normal

GRADE – Grading of hypertensive retinopathy

U.ALB – Urine albumin

HB% - Haemoglobin percentage

LFT – Liver function test

RFT – Renal function test

AN – Abnormal

MGSO4 – Magnesium sulphate regimen

LD – Low dose regimen

SD – Standard dose regimen

MGSO4 IN – Magnesium sulphate infusion

NO – Magnesium sulphate regimen not given

P – Phenytoin

AN-1 – Haemorrhage in right frontal & subarchanoid haemorrhage

AN-2 – Subacute infarct in left fronto-parietal region

AN-3 – Haemorrhage in parietal region

AN-4 – Brainstem haemorrhage

AN-5 – Haemorrhagic infarct in left parietal region

AN-6 – Hypodensity in bilateral high parietal region ? venous infarct

AN-7 – Old calcified lesion seen in right high parietal region

AN-8 – Left fronto temporal intracerebral haemorrhage with intraventricular extension with mass

effect

AN-9 – Diffuse cerebral odema

AN-10 – Hypodensity in right post parietal region ? PRES(Posterior reversible encephalopathy

Syndrome)

AN-11 – shows white matter hypodensity suggestive of PRES

AN-12 – Features suggestive of diffuse cerebral odema

AN-13 – Diffuse cerebral odema

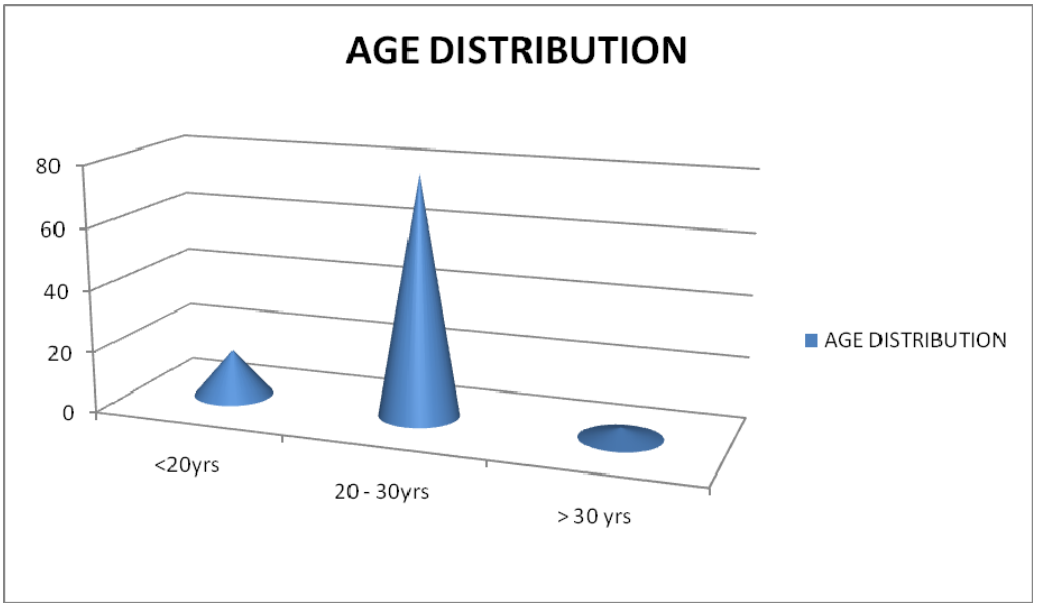
AN-14 – White matter hypodensity over periventricular region & post region suggestive of PRES

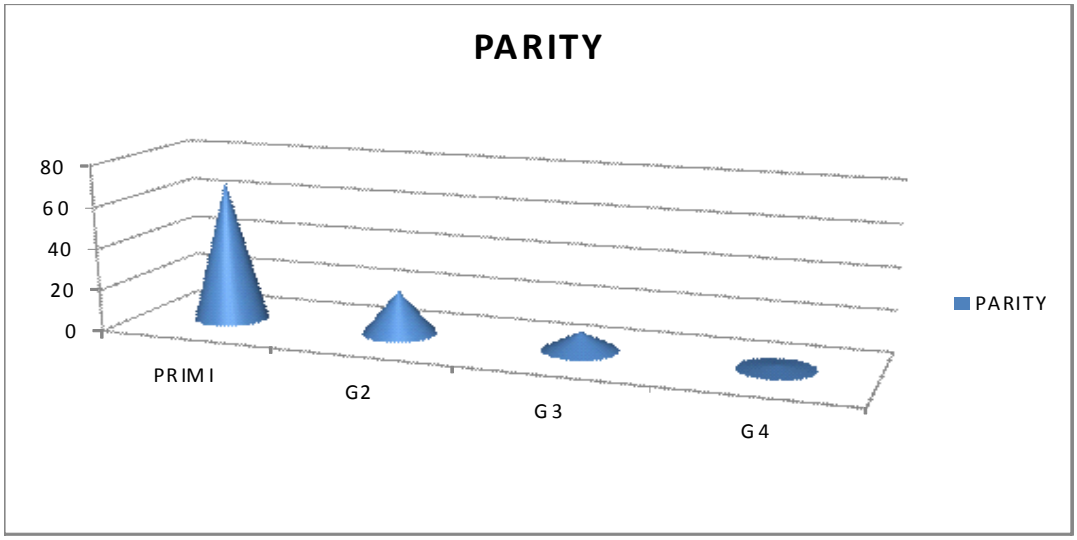
AN-15 – Bilateral occipital lobe hypodensity suggestive of PRES

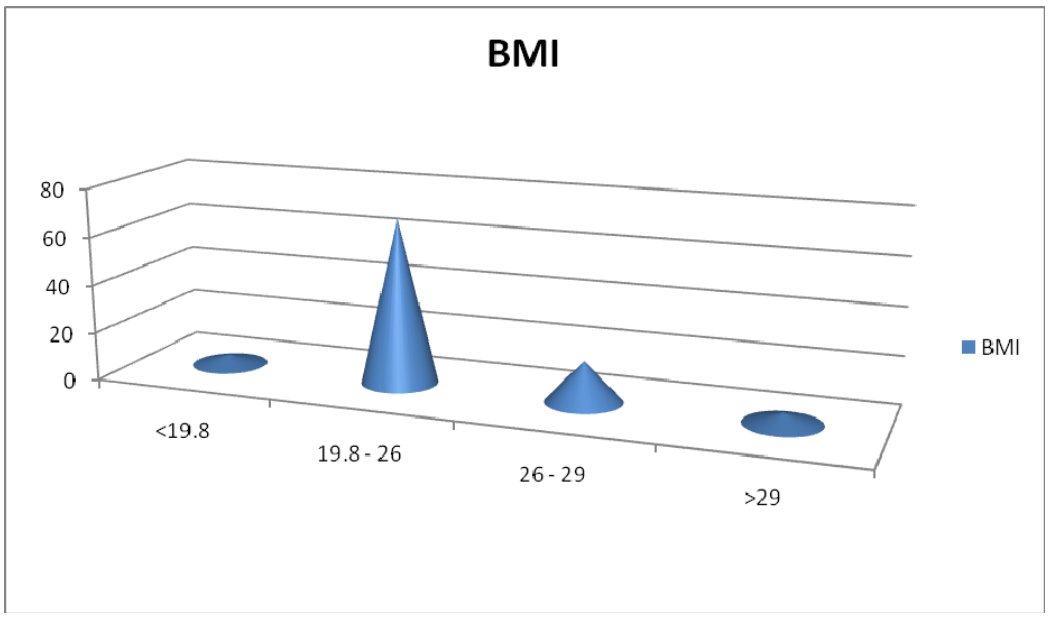
R – Recovered

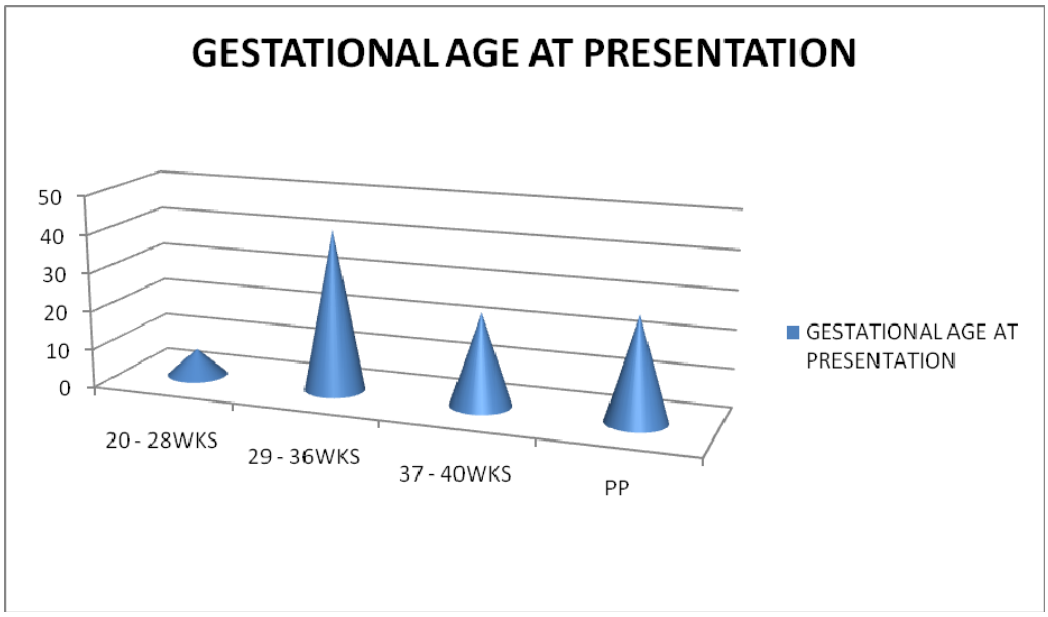
E – Expired

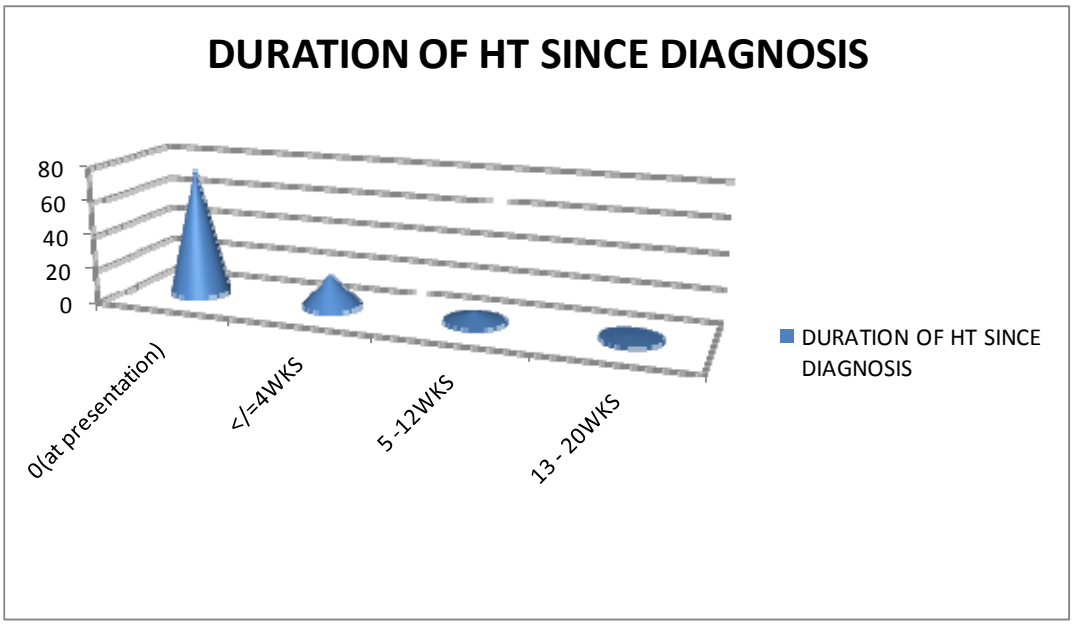
R-S – Recovered with sequelae

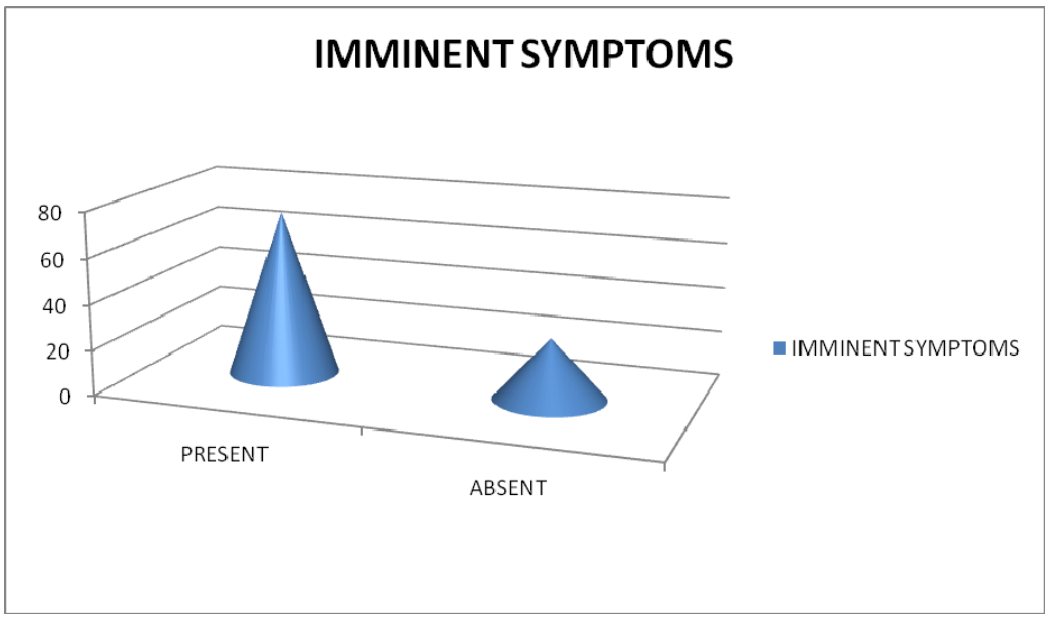

AGE DISTRIBUTION
80
60
40
20
0
<20yrs
20 - 30yrs
> 30 yrs
AGE DISTRIBUTION

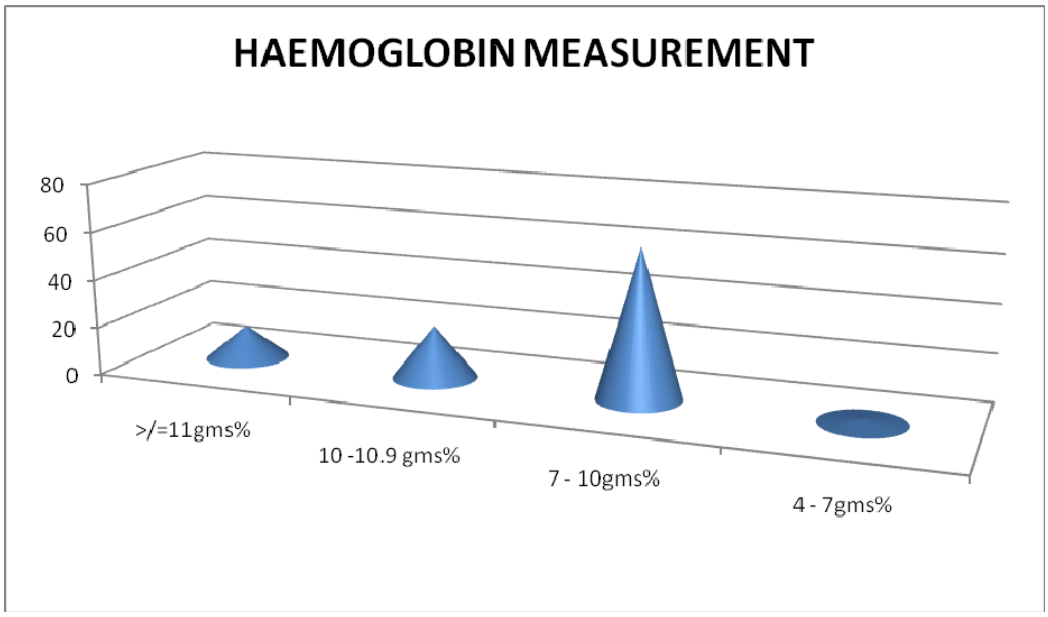

PARITY
80
60
40
20
0
PRIMI
G2
G3
G4
PARITY


BMI
80
60
40
20
0
<19.8
19.8 - 26
26 - 29
>29
BMI

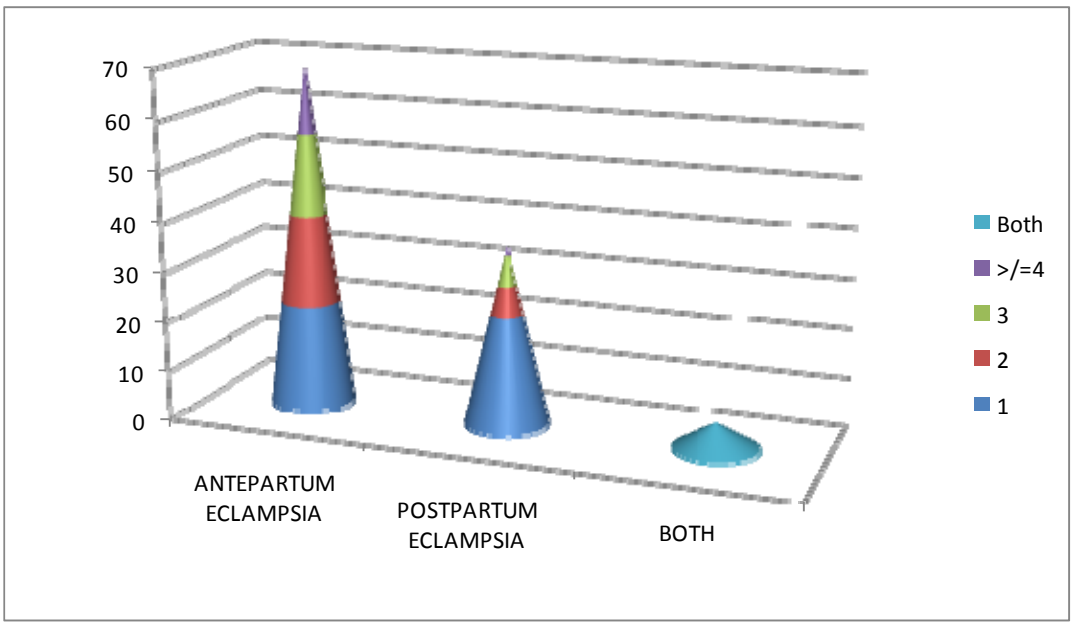

GESTATIONAL AGE AT PRESENTATION
50
40
30
20
10
0
20 - 28WKS
29 - 36WKS
37 - 40WKS
PP
GESTATIONAL AGE AT PRESENTATION

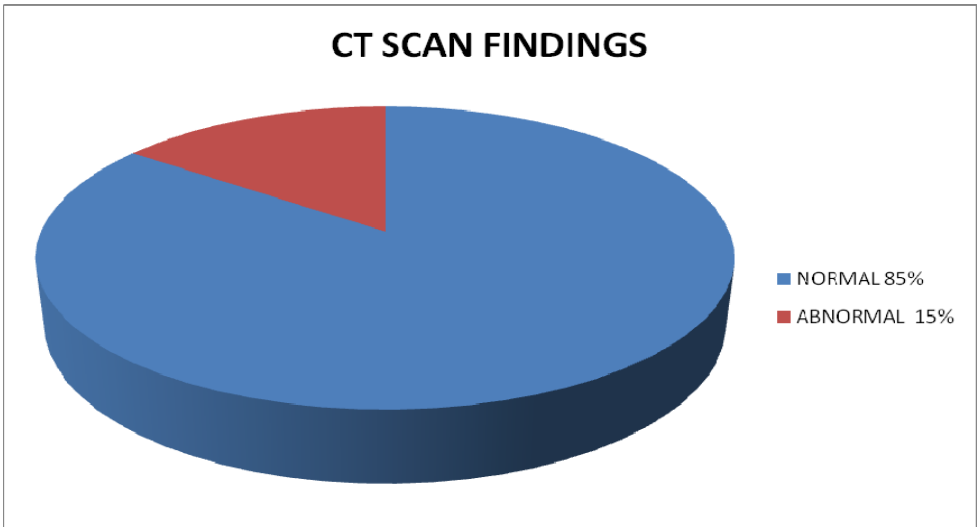

DURATION OF HT SINCE DIAGNOSIS
80
60
40
20
0
0(at presentation)
</=4WKS
5 -12WKS
13 - 20WKS
DURATION OF HT SINCE DIAGNOSIS


IMMINENT SYMPTOMS
80
60
40
20
0
PRESENT
ABSENT
IMMINENT SYMPTOMS

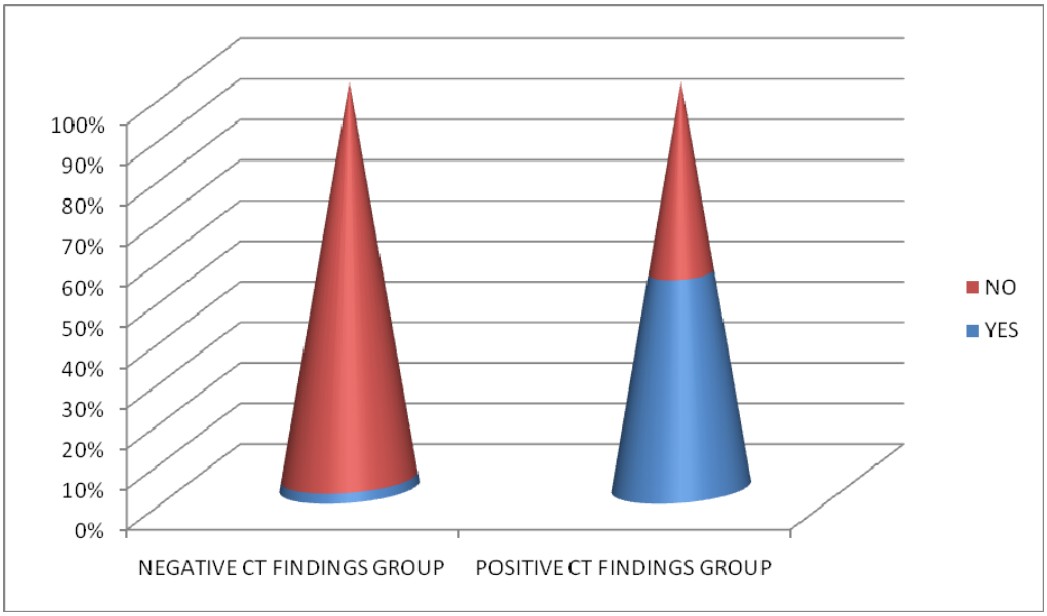

HAEMOGLOBIN MEASUREMENT
80
60
40
20
0
>/=11gms%
10 -10.9 gms%
7 - 10gms%
4 - 7gms%

CONVULSIONS

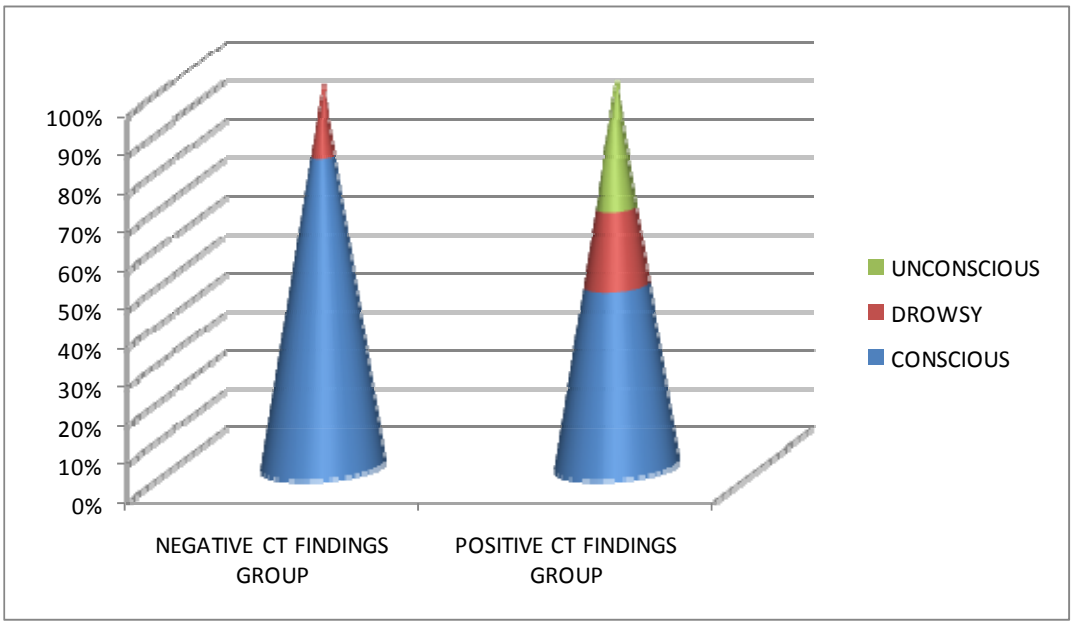




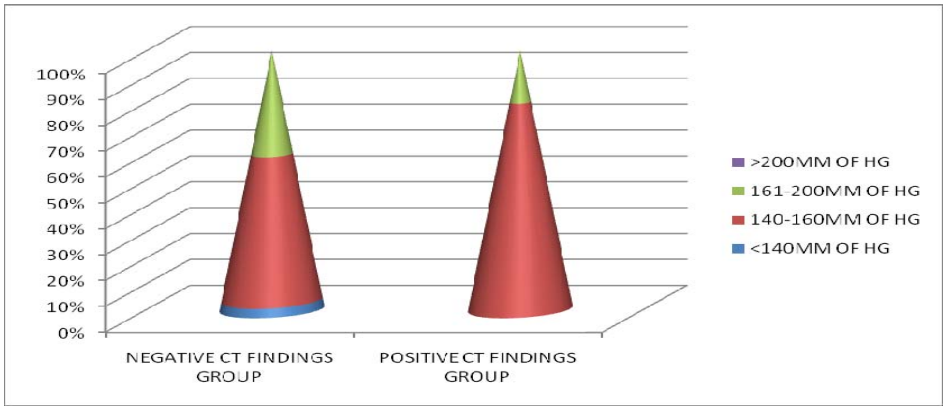
POSITIVE PAST HISTORY



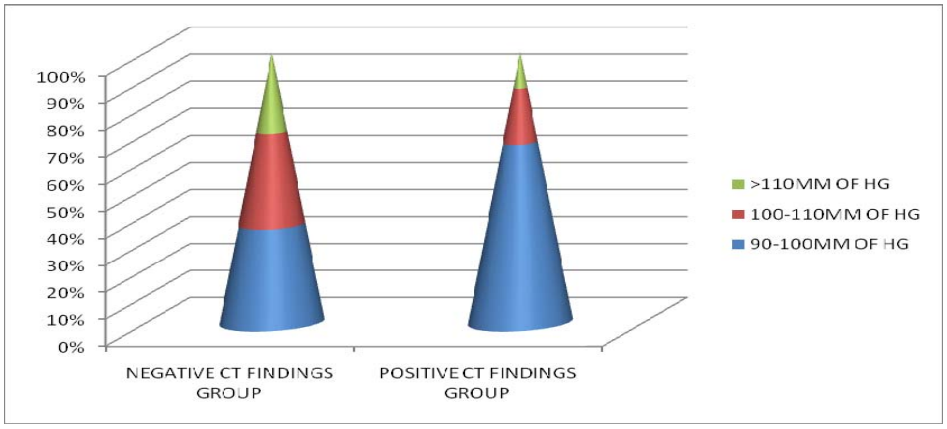
CONSCIOUS LEVEL



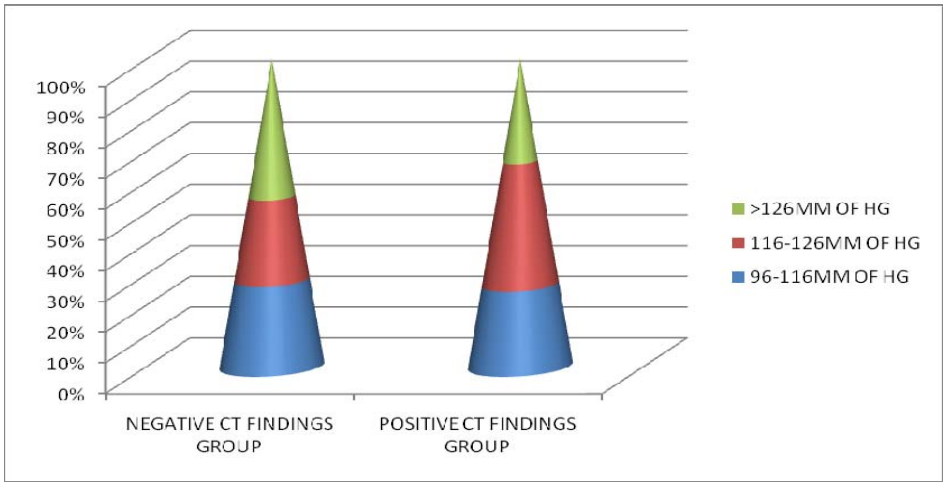
SYSTOLIC BP



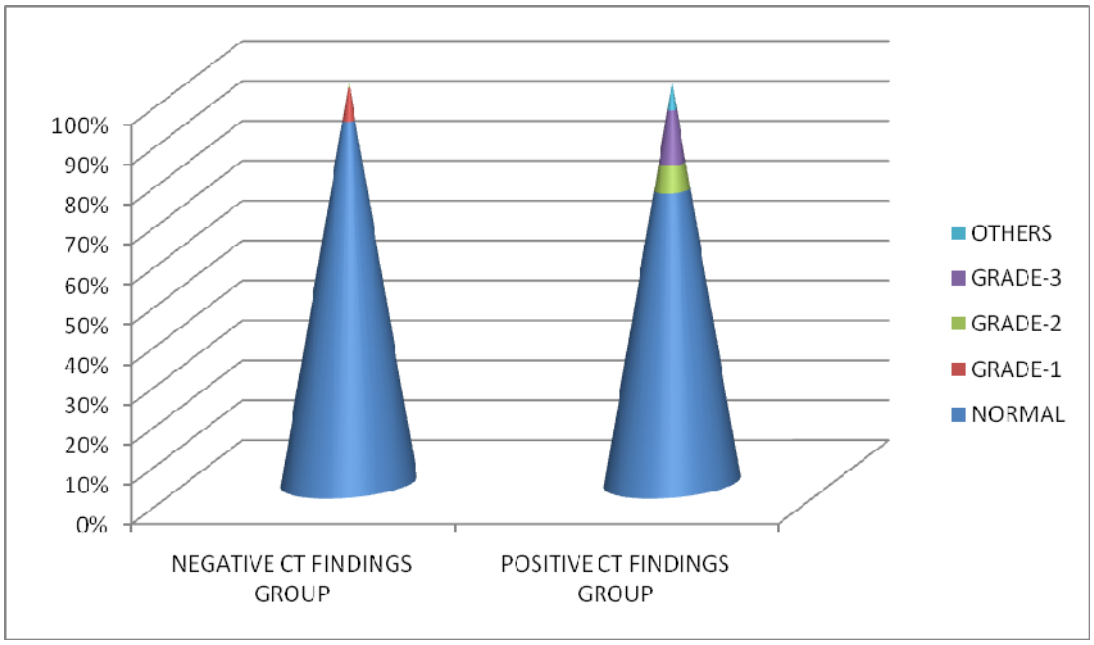
DIASTOLIC BP



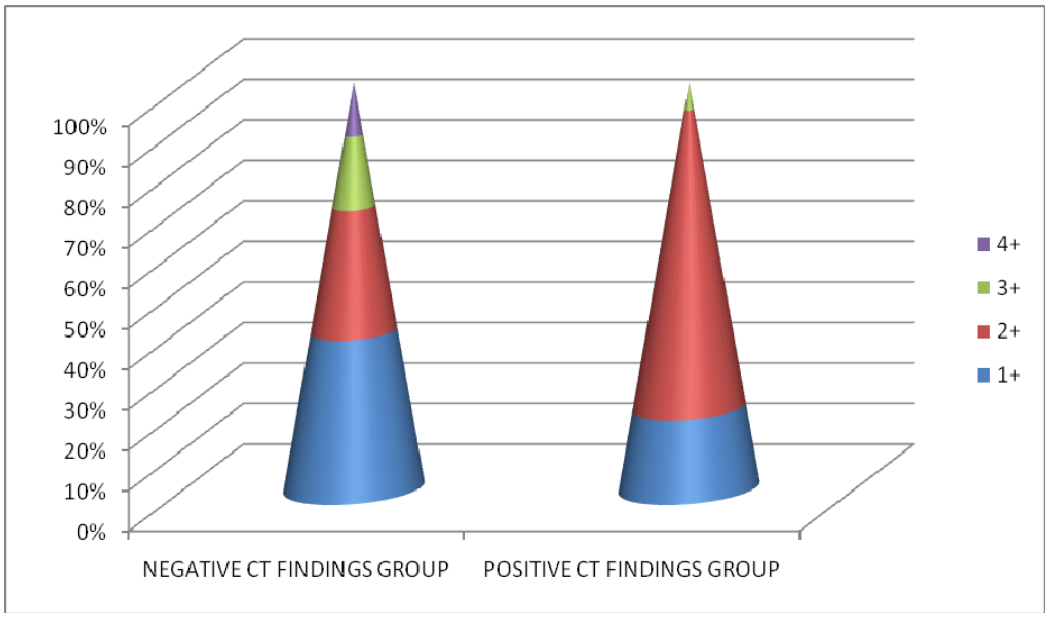
MEAN ARTERIAL PRESSURE



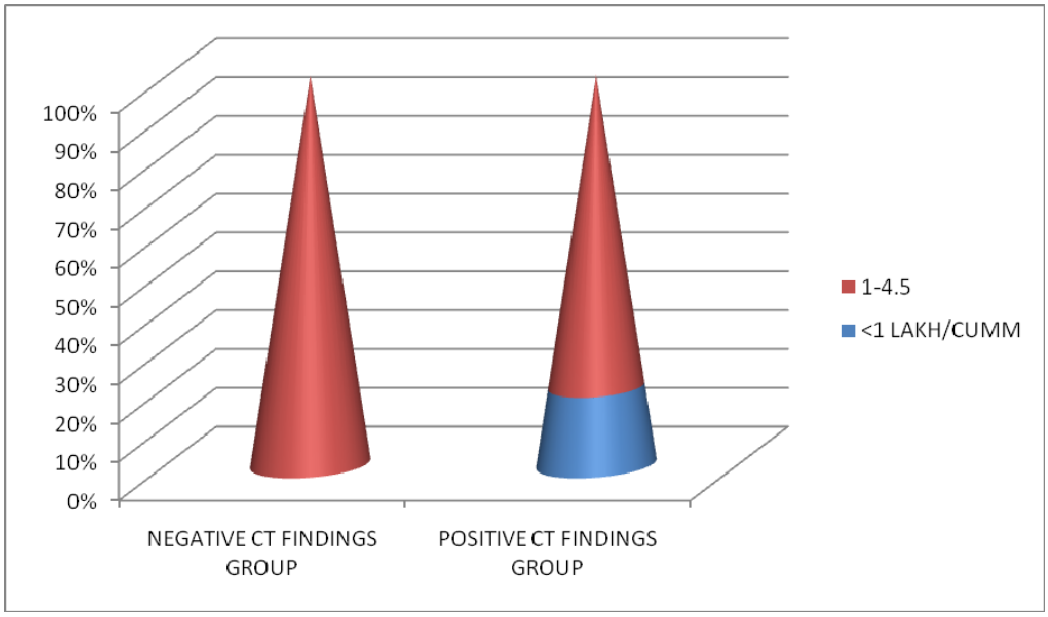
FUNDUS CHANGES



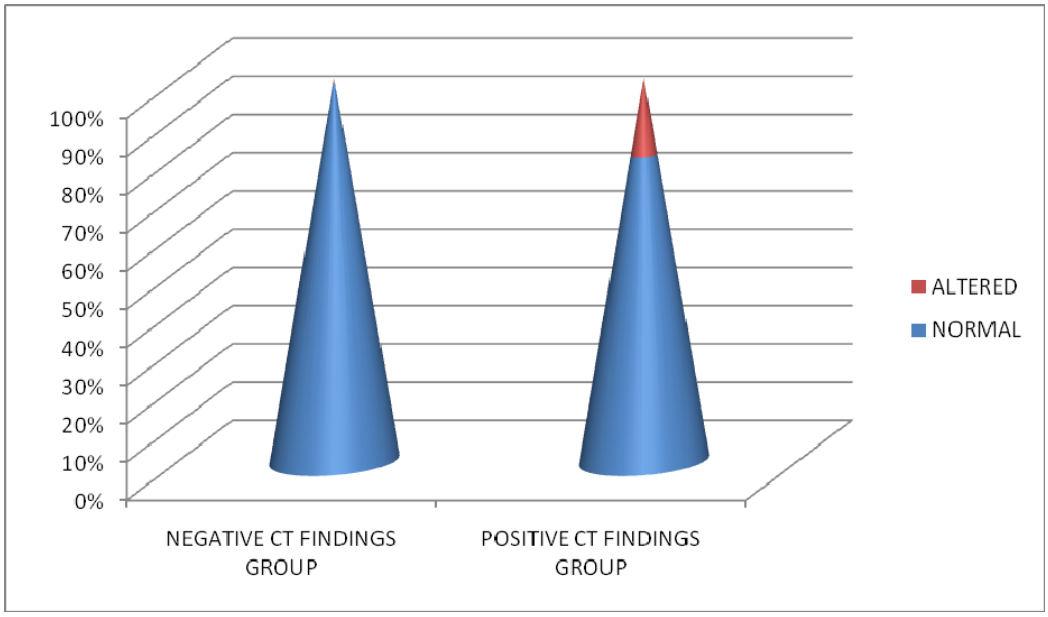
PROTEINURIA



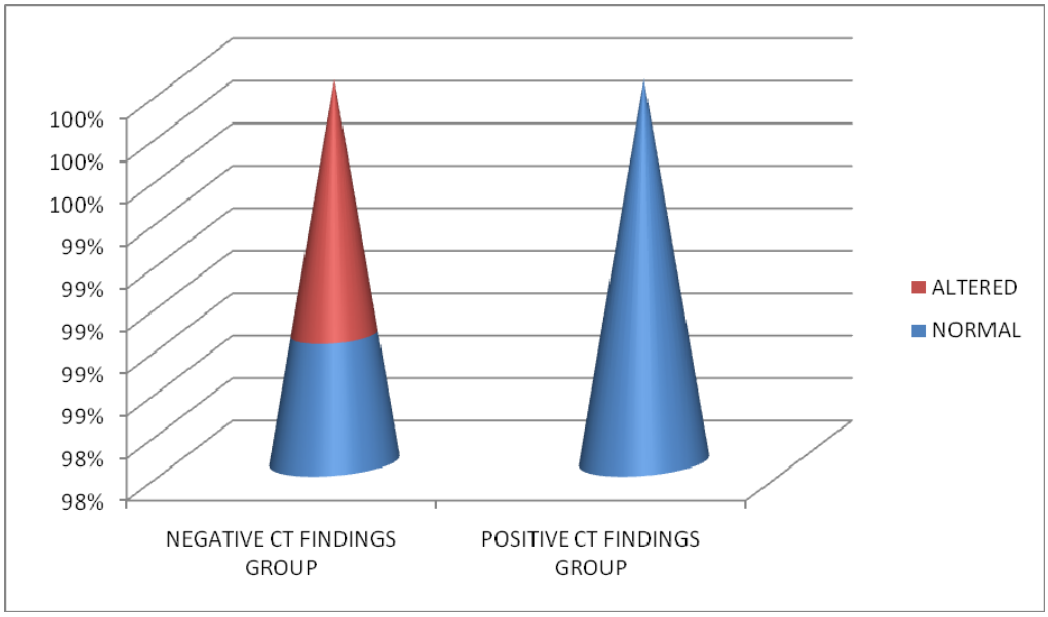
PLATELET COUNT



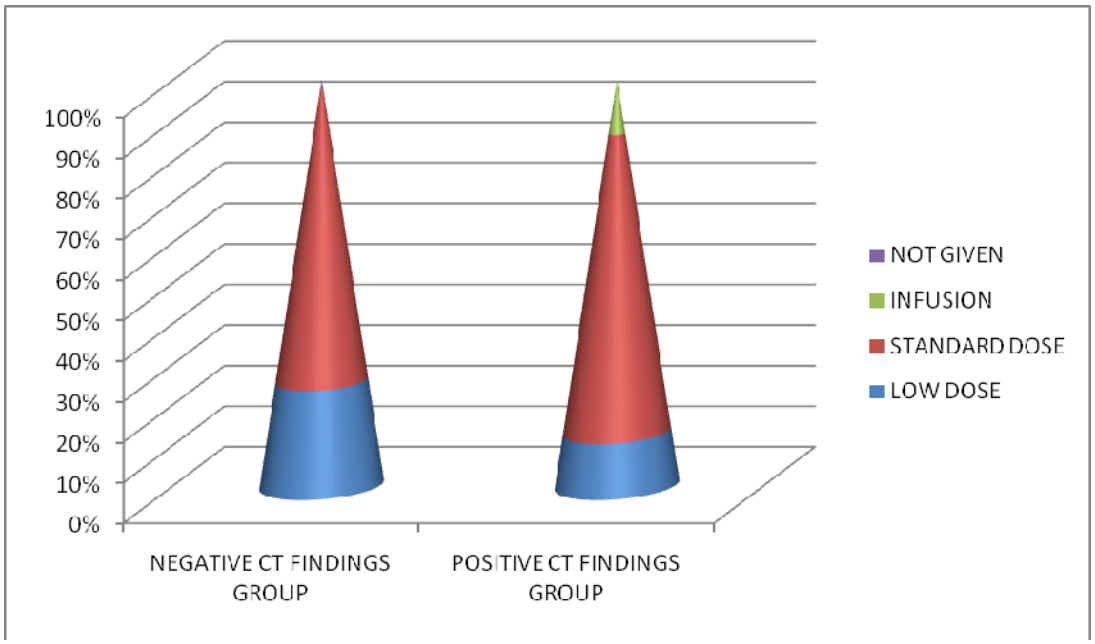
LIVER FUNCTION TEST



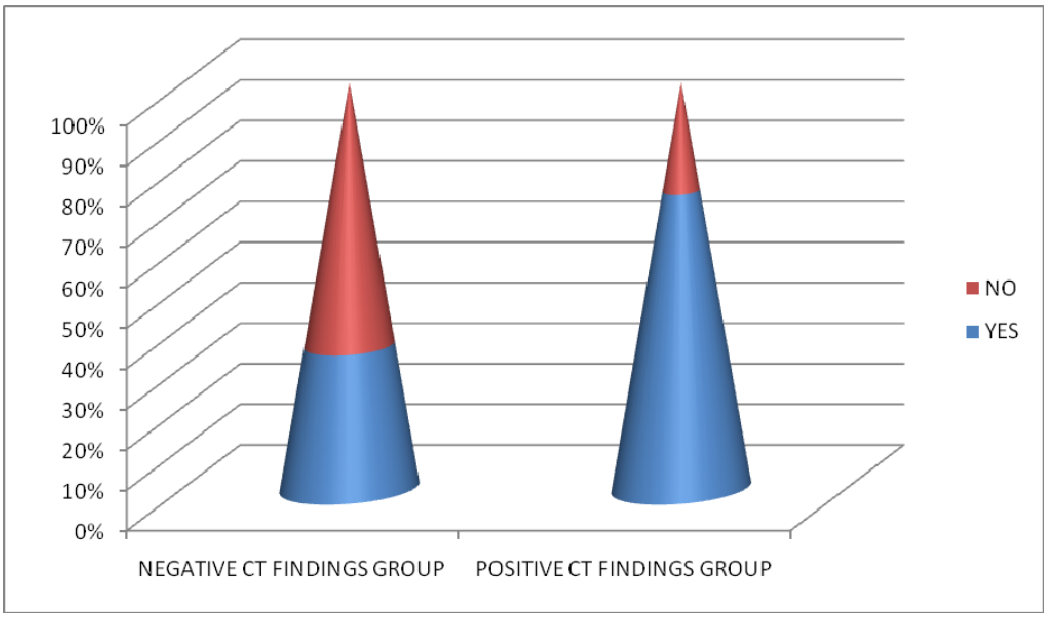
RENAL FUNCTION TEST

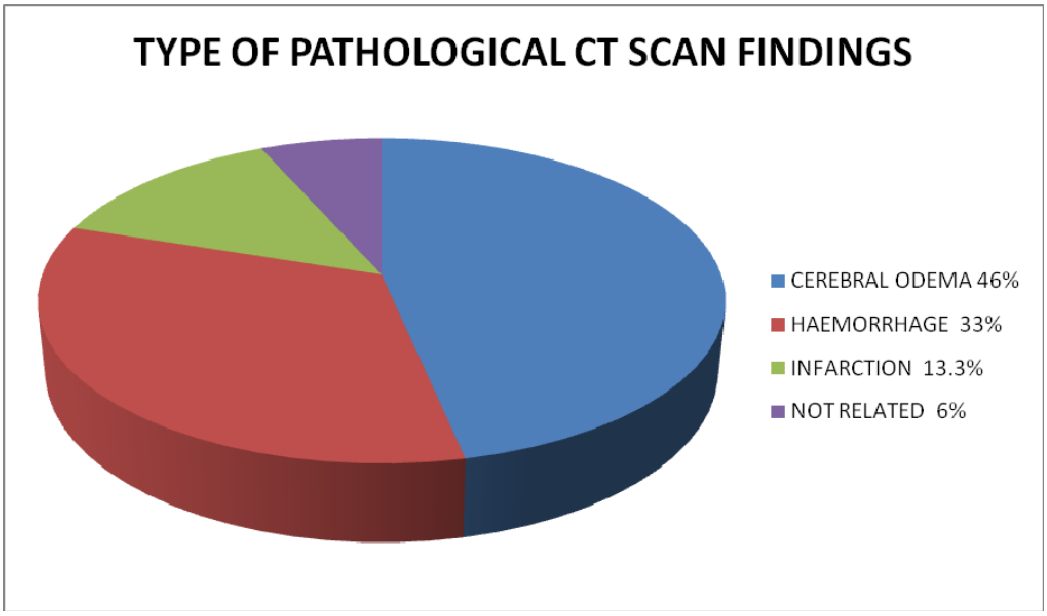


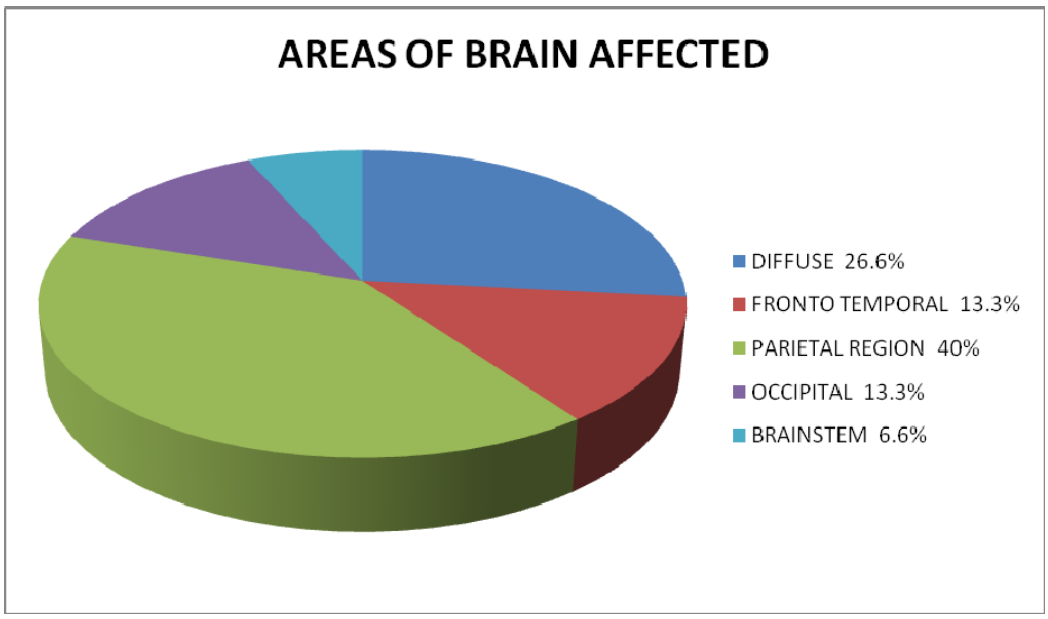
MAGNESIUM SULPHATE REGIMEN



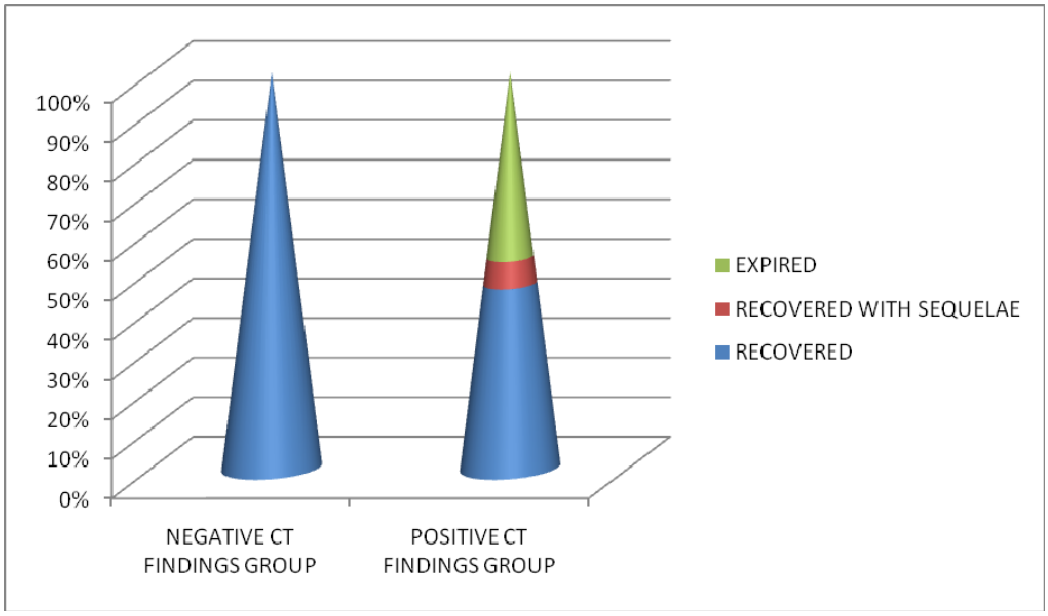
ANTICONVULSANT



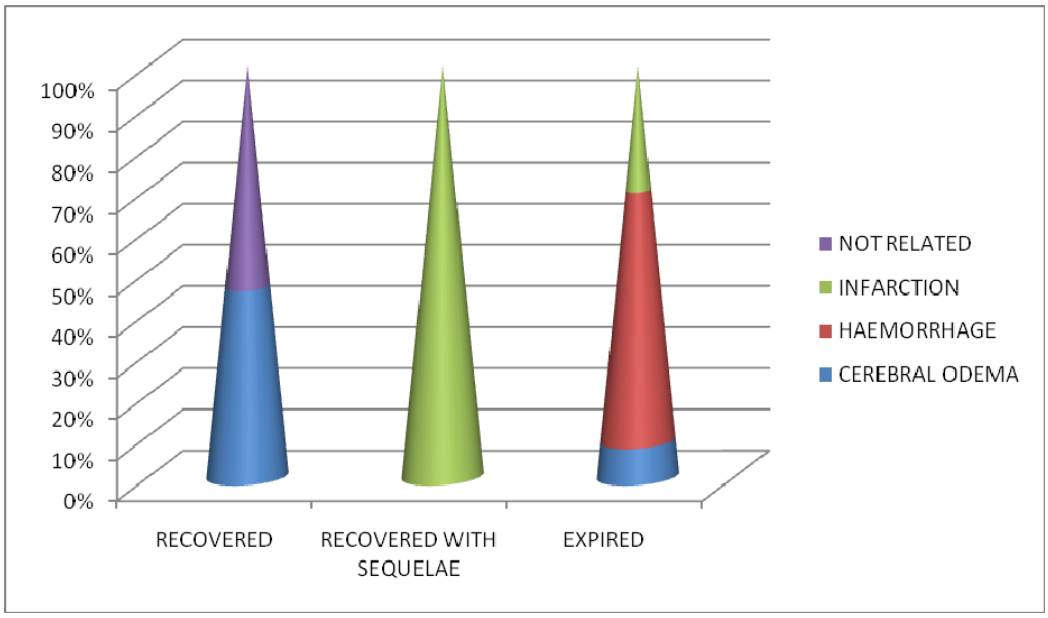




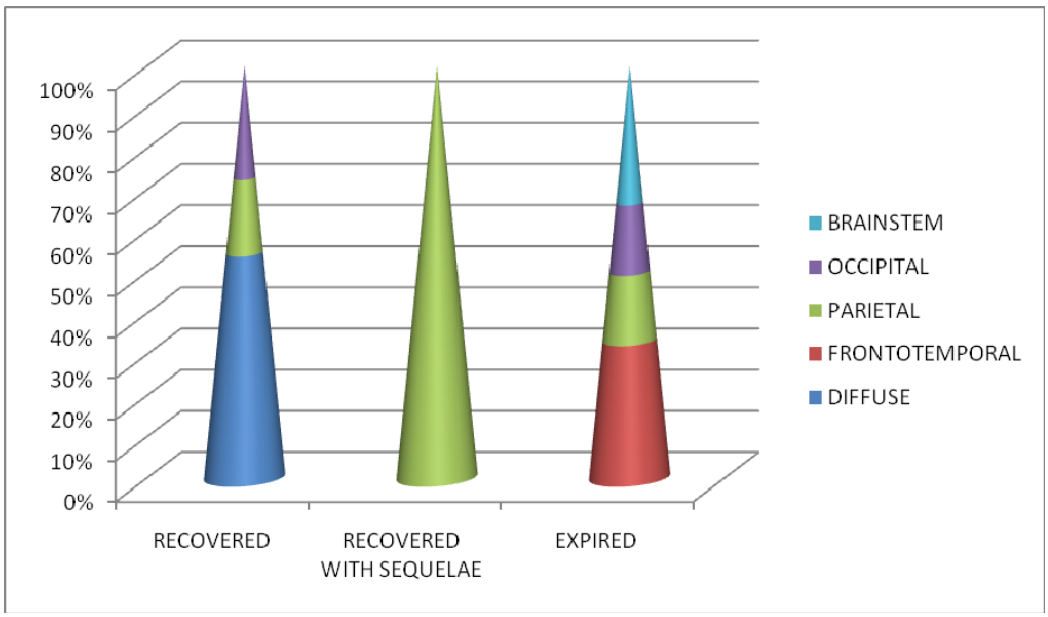
MATERNAL OUTCOME

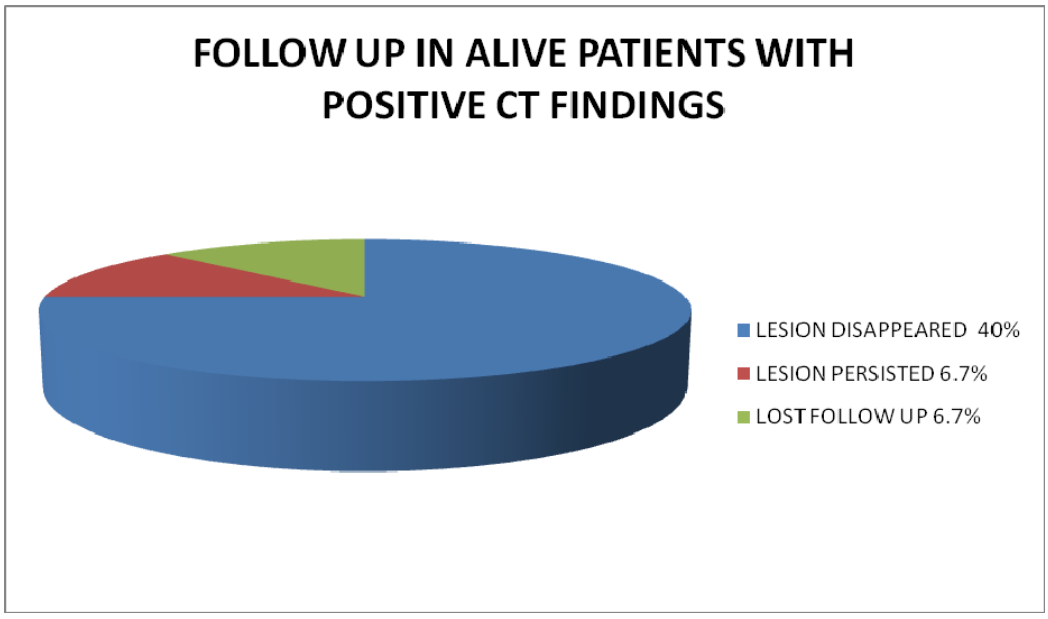


COMPARISON OF MATERNAL OUTCOME WITH TYPE OF CT CHANGES

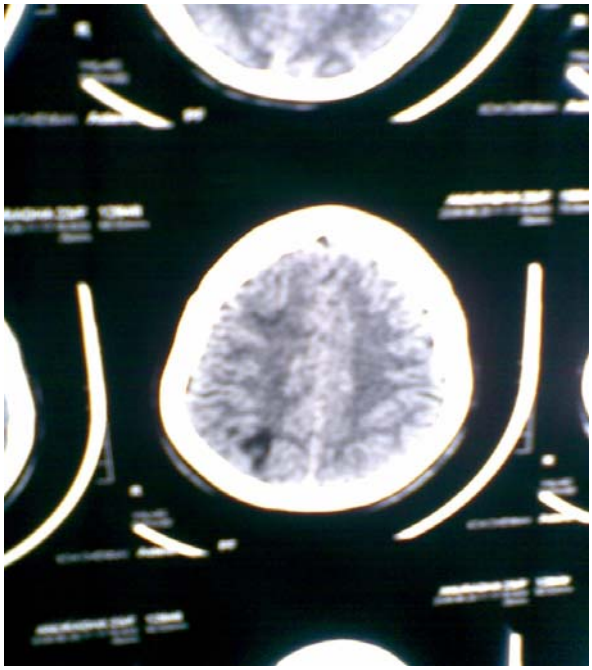


COMPARISON OF MATERNAL OUTCOME WITH AREAS OF BRAIN AFFECTED

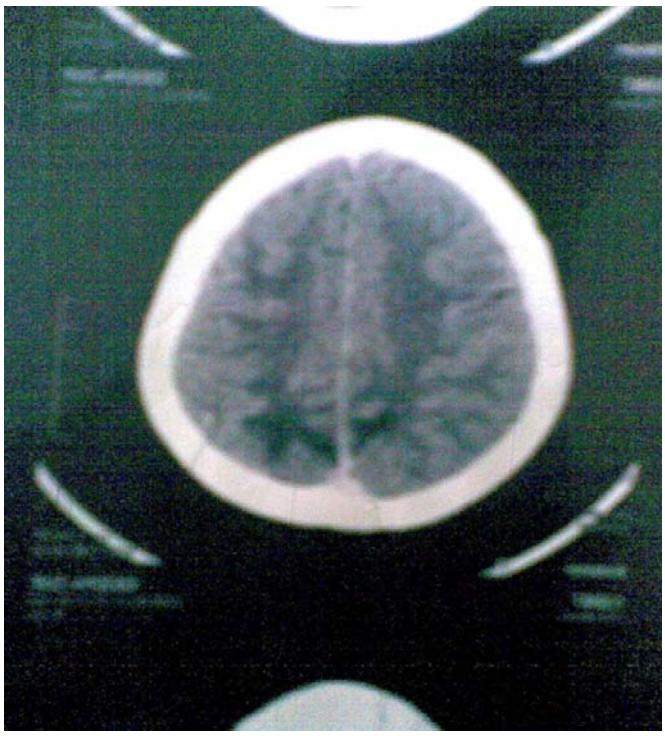



FOLLOW UP IN ALIVE PATIENTS WITH POSITIVE CT FINDINGS
LESION DISAPPEARED 40%
LESION PERSISTED 6.7%
LOST FOLLOW UP 6.7%

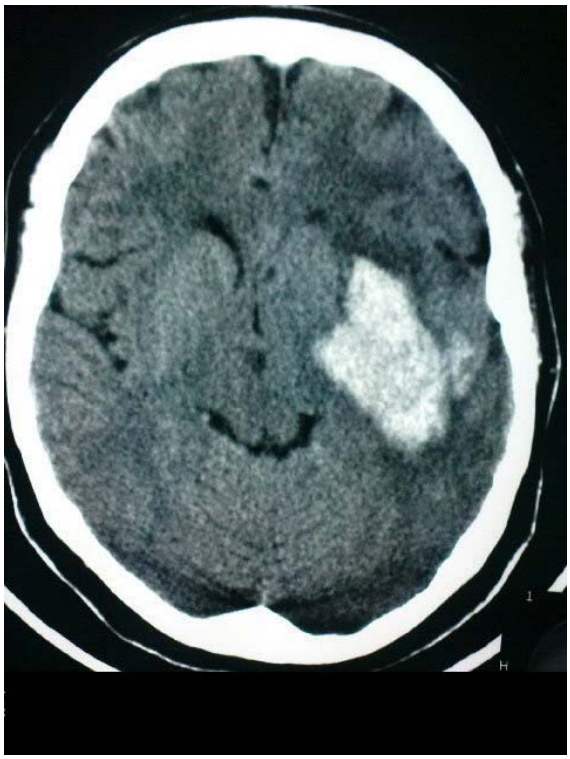
CT SCAN PICTURES OF PATHILOGICAL CHANGES OF BRAIN IN ECLAMPSIA



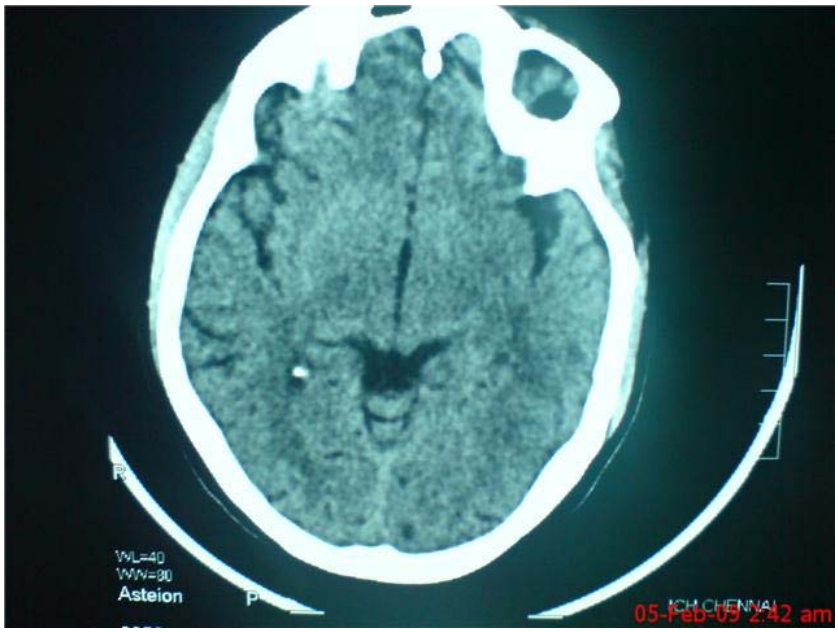
Hypodensity in right post parietal region ?PRES



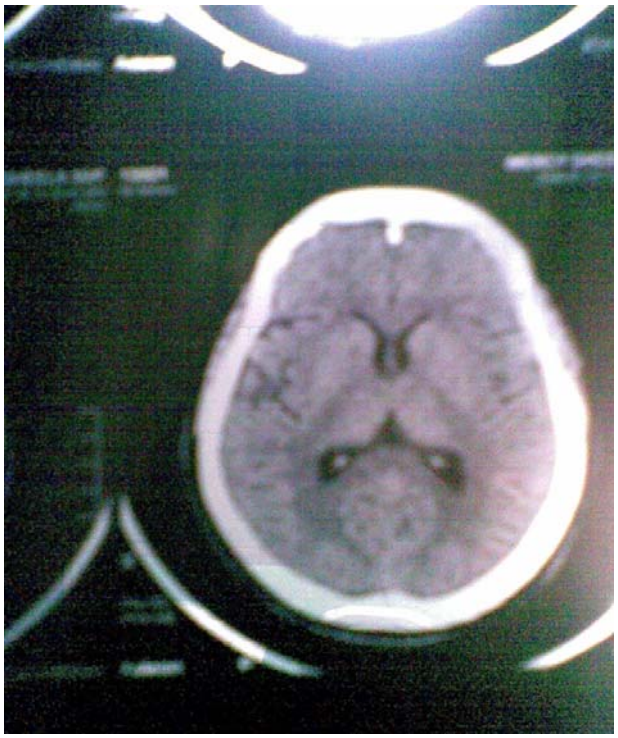
Bilateral occipital lobe hypodensity suggestive of PRES



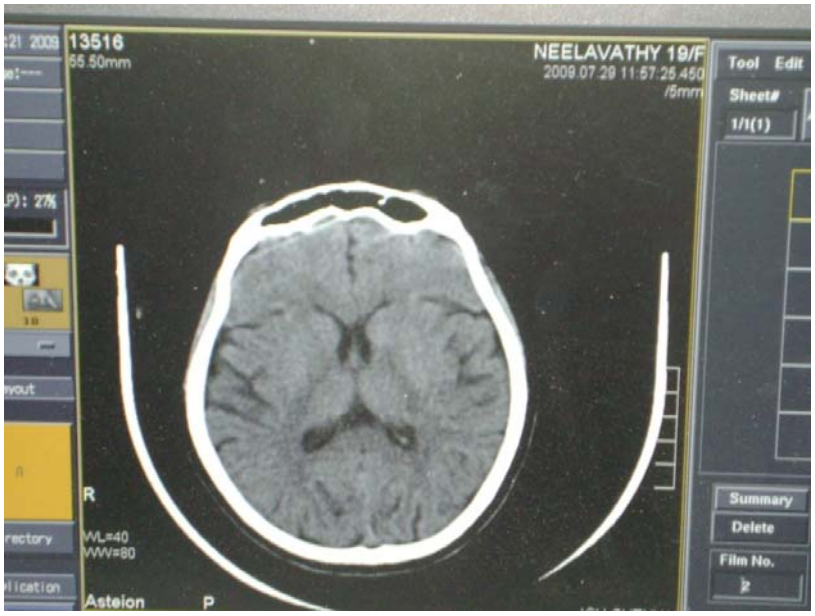
Haemorrhage in parietal region



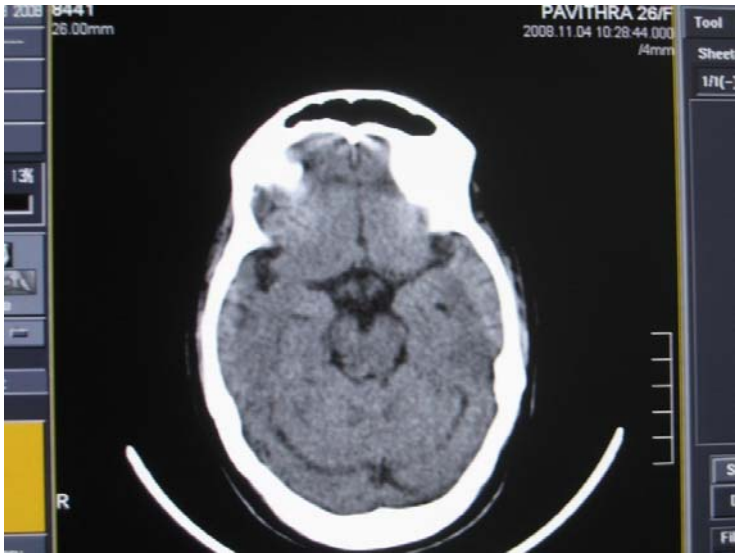
Hypodensity in high parietal region ? venous infarct



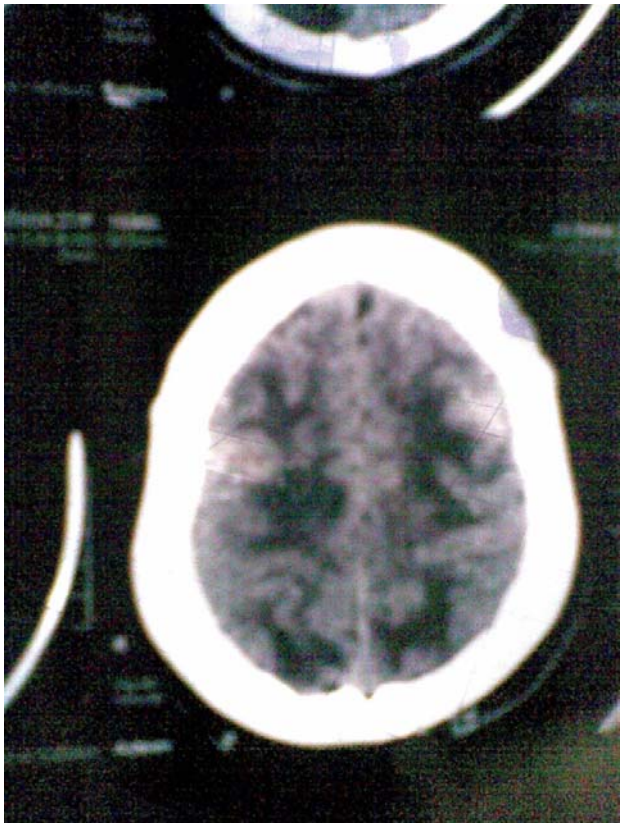
Shows whitematter hypodensity suggestive of PRES



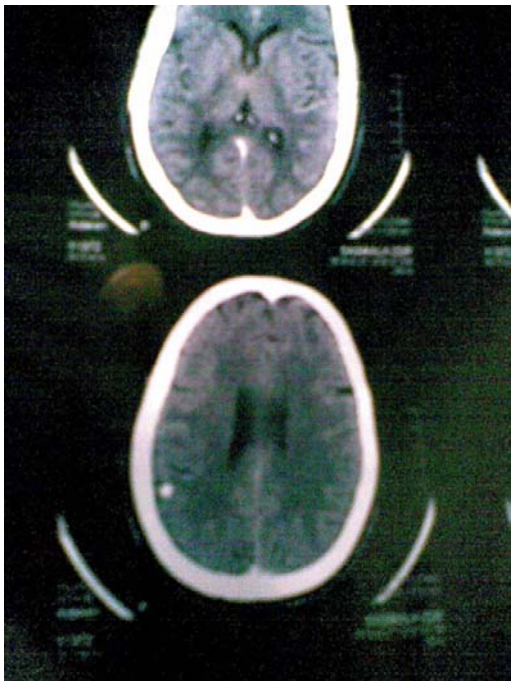
Diffuse cerebral odema



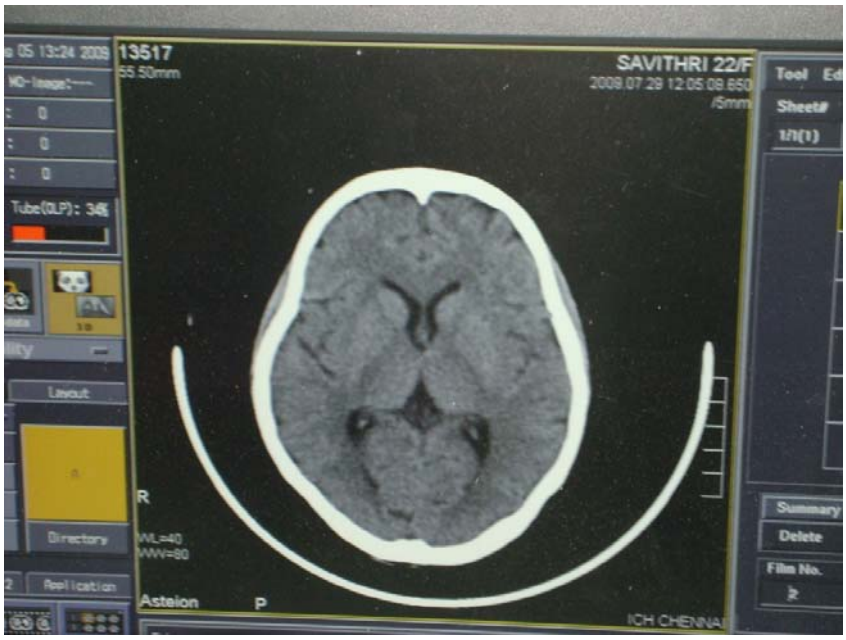
Subacute infarct in left fronto parietal region



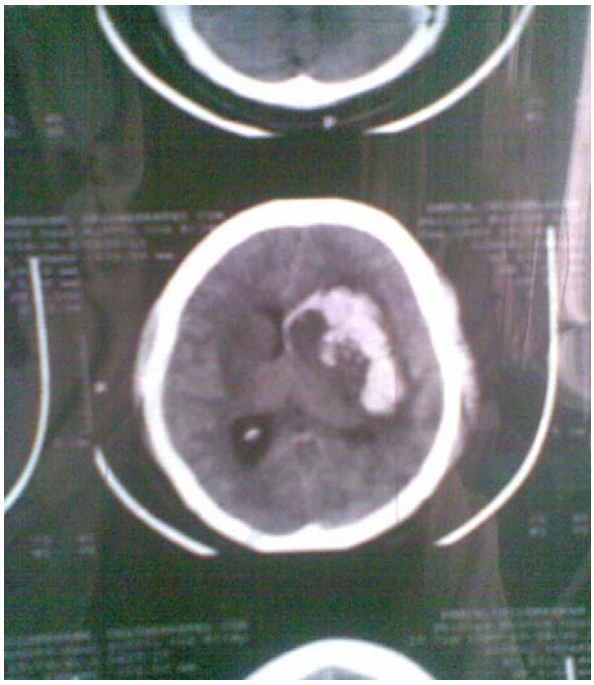
Diffuse cerebral odema



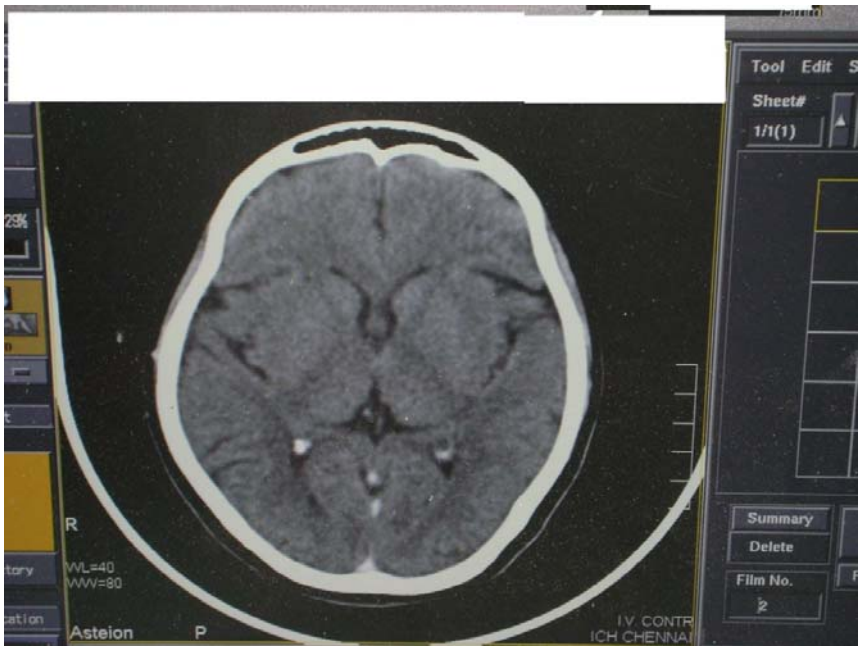
Old calcified lesion seen in right high parietal region



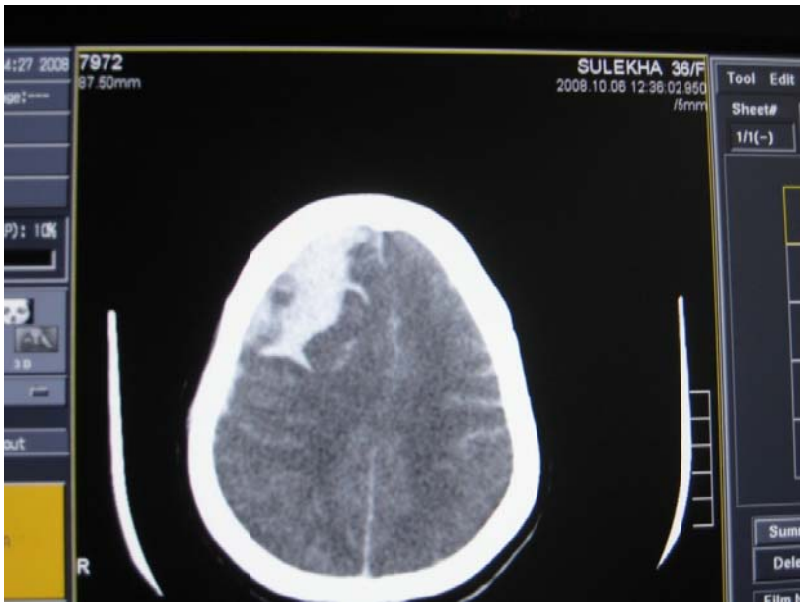
Features suggestive of diffuse cerebral odema



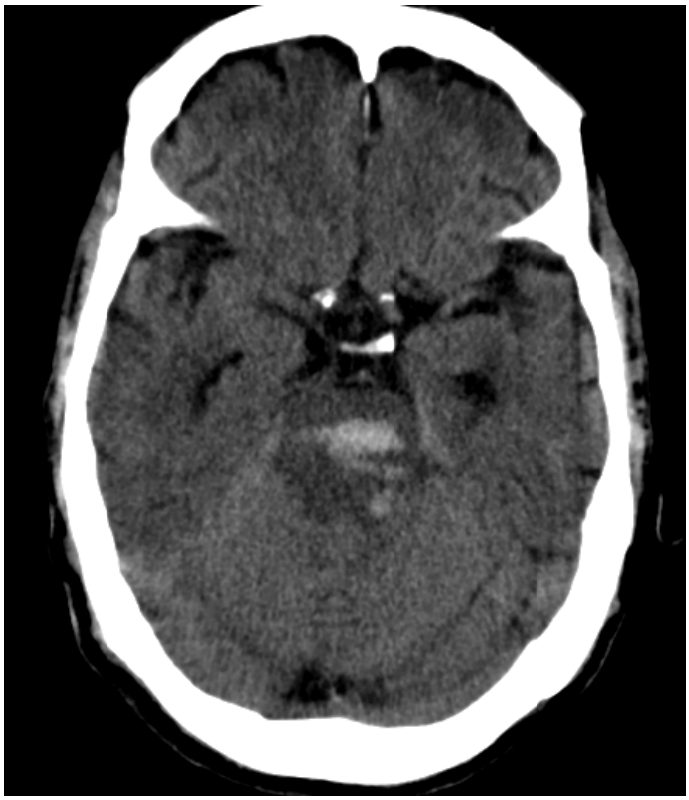
Left fronto temporal intracerebral haemorrhage with Intraventricular extension with mass effect



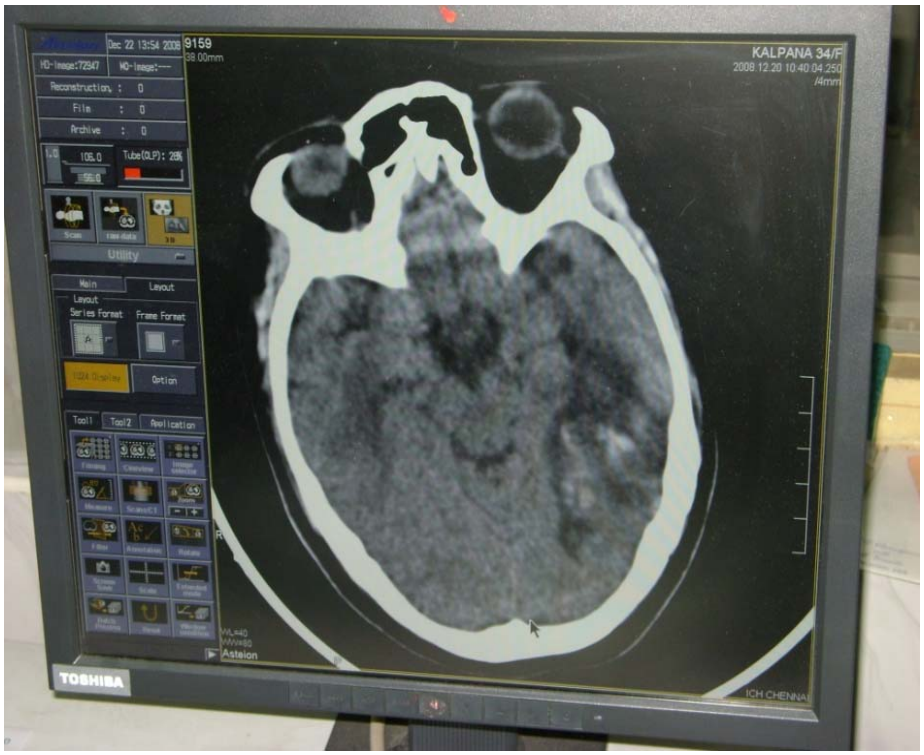
White matter hypodensity over periventricular region & post region suggestive of PRES



Haemorrhage in right frontal and subarachanoid region



Brainstem haemorrhage



Haemorrhagic infarct in left parietal region

SN NO	AGE	BMI	PARITY	GA/PP	HT AT	SYMPTOM	SEIZURE	PAST H/O	CONSCIOUS LEVEL	MAX BP	MAP	FUNDUS
1	20	22.8	primi	38	38	H & V	1/AP	No	C	160/120	113	N
2	18	22.6	primi	35	35	H	5/AP	No	D	140/110	120	N
3	25	20	primi	30	30	V	3/AP	No	C	200/120	146	N
4	25	21.6	G3P2L1	36	36	V&E	3/AP	No	C	170/130	143	N
5	35	32	G2P1L1	35	35	H&B&V	3/AP1/PP	NO	C	150/96	114	N
6	27	21	P2L2	PP	39	NO	3/PP	NO	D	160/110	126	N
7	27	21.7	P1L1	PP	PP	NO	3/PP	NO	C	140/100	113	N
8	24	22	P1L1	PP	28	NO	1/PP	No	C	130/90	103	N
9	30	22.2	G2A1	29	29	H&B	2/AP	NO	C	200/120	146	Grade-1
10	36	27.1	P3L3	PP	39	E	1/PP	No	UC	170/110	130	Grade-3
11	21	23.4	primi	30	30	H&B	1/AP1/PP	No	D	200/140	160	N
12	18	24.6	P1L0	PP	PP	NO	3/PP	No	D	150/120	130	N
13	23	27.4	primi	24	24	H	15/AP	No	D	180/120	140	N
14	23	24.6	primi	35	35	H	3/AP	No	C	170/120	136	N
15	20	27.2	P1L1	PP	PP	H	3/PP	No	C	140/100	116	N
16	24	21.54	primi	30	30	NO	5/AP	No	C	140/100	116	N
17	20	23.3	primi	40	40	V	1/AP	No	C	170/140	150	N
18	35	26.6	P2L2	PP	38	H&B	1/PP	No	D	160/110	126	N
19	22	22.8	primi	39	39	H	2/AP	No	C	160/100	120	N
20	23	32.4	primi	39	32	H	1/AP	No	C	180/120	140	N
21	19	18.6	P1L1	35	35	NO	1/PP	No	C	150/90	110	N
22	18	21	primi	28	28	H&V	5/AP	No	C	160/110	126	Grade-1
23	23	22.9	P2L2	39	38	H	1/PP	No	C	160/110	126	N
24	20	23.8	G2P1L1	36	32	B	1/AP5/PP	No	D	150/120	130	N
25	42	28.8	G2A1	21	21	H&B	3/AP	No	D	170/110	130	N
26	28	20	G2P1L1	29	29	H&V	3/AP	No	C	150/100	116	N
27	20	30.5	P1L1	PP	37	NO	1/PP	No	c	160/110	126	Grade-1
28	23	25.3	primi	32	32	NO	2/AP	No	C	160/100	130	N
29	24	22	P1L1	PP	PP	H	2/PP	No	C	150/100	116	N
30	25	24.8	primi	38	37	H	2/AP	No	C	150/100	116	N
31	23	23.8	primi	29	29	NO	2/AP	No	D	150/100	110	N

32	19	32.5	P1L1	38	38	NO	1/PP	No	C	140/100	113	N
33	24	19.7	G2P1L1	34	34	H&V&B	2/AP	No	C	170/110	130	Grade-1
34	26	24.7	G4P3L3	28	28	H	1/AP	No	C	160/120	133	N
35	25	24.5	P1L1	PP	PP	NO	5/PP	No	UC	160/100	120	N
36	24	32.3	G2P1L1	40	40	NO	1/AP	No	C	130/100	110	N
37	25	28.9	G2P1L0	PP	24	H	1/PP	ITP	UC	160/100	120	N
38	35	22.2	P1L0A3	PP	36	H	1/PP	No	C	160/110	126	N
39	18	20.9	primi	37	36	H	2/AP	No	C	180/120	140	N
40	24	20.3	P3L2	PP	PP	H	3/PP	E	D	150/100	116	N
41	28	23.8	G3P2L2	31	31	H&V	1/AP	No	C	170/120	136	N
42	24	25.1	G2P1L1	36	36	H	1/AP	No	C	180/120	150	N
43	24	17.3	primi	37	37	H&v&B	3/AP	No	C	140/100	116	N
44	30	27.3	P3L3	PP	PP	H	2/PP	No	D	140/100	116	N
45	20	24.2	primi	38	37	H&V	1/AP	No	C	180/110	133	N
46	18	25.7	primi	39	39	H&B&E	1/AP	No	C	170/110	130	Grade-1
47	20	25.1	primi	39	39	NO	2/AP	No	D	160/110	126	N
48	20	31.1	primi	32	32	H	2/AP	No	D	180/120	140	N
49	25	25.6	primi	30	28	B	5/AP	No	C	160/120	133	N
50	22	25.3	P2L2	PP	37	H	1/PP	No	C	140/110	126	N
51	19	31.1	primi	37	37	H	1/AP	No	C	180/110	133	N
52	19	21.4	primi	30	30	NO	2/AP	No	D	160/100	120	N
53	26	22.7	P2L2	PP	PP	NO	1/PP	No	C	150/90	110	N
54	23	22.2	P1L1	PP	PP	H	2/PP	No	C	160/100	120	N
55	25	24	primi	39	39	H	1/AP	No	C	190/130	150	N
56	22	22.8	primi	pp	pp	NO	1/pp	No	C	160/100	120	N
57	27	26.5	G2P1L1	38	38	H&V	6/AP	No	C	160/110	126	N
58	25	31.1	primi	30	30	H	3/AP	No	C	160/100	120	N
59	23	26.6	G3P2L1	39	39	NO	3/AP	AP/PP	C	140/90	116	N
60	36	22.2	P1L1A1	PP	34	H	1/PP	No	C	170/120	136	N
61	18	25.3	P1L1	PP	PP	NO	3/PP	No	C	150/100	126	N
62	20	22.4	primi	27	27	H	8/AP	No	D	180/120	140	N
63	22	21.6	primi	35	35	H	3/AP	No	C	190/100	130	N
64	21	24.6	P2L2	PP	35	H	1/PP	No	C	150/90	106	N

65	23	28	primi	34	34	H	4/AP	No	C	170/110	133	N
66	22	22.2	primi	35	24	H&B	1/AP	No	C	160/110	130	N
67	28	31.2	P1L1	PP	39	H	1/PP	No	C	140/100	113	N
68	20	21.3	primi	38	38	H&V	1/AP	No	C	140/110	123	N
69	28	23.1	P1L1	40	40	NO	1/PP	No	C	160/100	120	N
70	34	24.8	primi	24	24	H	5/AP	No	UC	180/130	146	Grade-3
71	19	30.4	primi	38	38	H	4/AP	No	C	150/100	126	N
72	25	23.8	G2P1L0	35	35	NO	1/AP	AP	C	150/100	116	N
73	22	25	P1L1	PP	28	H	1/PP	No	C	170/130	140	Grade-1
74	18	22	primi	36	36	NO	1/AP	No	C	150/100	116	N
75	20	27	primi	36	28	H&V	5/AP	No	D	210/120	150	N
76	17	20.5	primi	31	30	v	1/AP2/PP	No	C	140/110	126	N
77	20	22.7	primi	39	39	H&V	3/AP1/PP	No	C	170/110	123	N
78	23	24	G2P1L1	36	36	H&B	3/AP	No	C	180/110	133	N
79	21	25.9	primi	35	34	H&V	3/AP	No	C	150/100	126	N
80	20	27.5	primi	36	36	H	2/AP	No	C	150/90	110	N
81	22	20.7	P1L1	PP	PP	H	1/PP	No	C	150/110	123	N
82	22	24.2	primi	40	40	H&E	2/AP	No	C	160/100	120	N
83	22	22	primi	29	29	H&E	3/AP	No	C	170/100	130	N
84	20	22.8	P1L1	PP	PP	NO	2/PP	No	C	180/110	133	N
85	22	20.3	primi	36	36	H	2/AP	No	C	180/130	146	Grade-2
86	22	26.5	primi	21	21	H&E	1/AP	No	C	140/100	113	Grade-2
87	21	23.8	primi	36	36	H&B&V	2/AP	No	D	150/110	126	Grade-2
88	28	19.4	G3A2	37	37	NO	1/AP	No	C	180/120	140	Grade-2
89	32	28.8	primi	29	29	H&B	3/AP	No	C	170/110	130	Grade-2
90	21	25.3	primi	34	34	NO	2/AP	No	C	130/110	116	Grade-2
91	27	26.8	primi	30	30	H&V&B	5/AP	No	D	200/150	166	Grade-2
92	23	21.6	P1L0	PP	28	H&V	2/PP	No	C	150/100	116	N
93	30	25.6	primi	33	33	H	3/AP	No	D	160/110	130	N
94	22	21.2	P1L1	PP	37	NO	1/PP	No	C	150/90	110	PP CAT
95	19	24	primi	35	35	NO	2/AP	No	C	160/100	120	Grade-2
96	22	27.3	G3P1L1	30	30	H&B&V	2/AP	No	C	190/130	150	Grade-1
97	27	25	G3P1L0	30	30	H&V	1/AP	No	C	170/110	130	N

98	20	20	G2A1	32	32	H	6/AP	No	D	150/110	123	N
99	20	20	primi	36	36	V	10/AP	No	UC	140/100	113	N
100	25	27	G2P1L0	33	33	NO	2/AP	No	C	170/110	130	N

U.ALB	HB%	PLATELET	LFT	RFT	MGSO4	ANTI CON	CTFINDING	RECOVERY
4	9	2	N	N	L D	No	N	R
3	10	1.5	N	N	L D	P	N	R
3	6.5	1.8	N	N	S D	No	N	R
3	10	1.7	AN	N	L D	No	N	R
4	12.5	1	N	N	S D	NO	N	R
1	9.5	1.8	N	N	L D	NO	N	R
1	9.5	1.6	N	N	L D	P	N	R
2	9	1.5	N	N	S D	NO	N	R
3	9.8	1.5	N	N	S D	No	N	R
2	6	1	AN	N	L D	P	AN-1	E
4	9.8	1.5	N	N	L D	P	N	R
3	11	2	N	N	L D	P	N	R
4	8	2.1	N	AN	L D	P	N	R
4	8	1.4	N	N	S D	No	N	R
4	9	2.2	N	N	L D	P	N	R
4	11.5	1.6	N	N	L D	No	N	R
2	10.5	2.5	N	N	L D	No	N	R
1	10.5	1.8	N	N	L D	No	N	R
2	9	1.5	N	N	S D	No	N	R
1	10.5	1.8	N	N	L D	No	N	R
1	10.5	2	N	N	S D	No	N	R
3	10	1.6	N	N	S D	No	N	R
1	11	1.1	N	N	S D	P	N	R
1	12.5	1.6	N	N	S D	P	N	R
1	9.5	2	N	N	S D	P	N	R
2	11	2.3	N	N	S D	No	N	R
1	9.5	1.7	N	N	S D	No	N	R
1	10	2.4	N	N	S D	No	N	R
1	9	2.8	N	N	S D	P	N	R
1	11	1.8	N	N	S D	No	N	R
4	9	1.3	N	N	S D	No	N	R

1	9	1.5	N	N	L D	No	N	R
3	7.5	1.6	N	N	S D	No	N	R
2	9	1.4	N	N	L D	P	AN-2	R-S
1	10	1.1	AN	N	S D	P	AN-3	E
1	10	1.3	N	N	S D	P	N	R
2	9	0.7	N	N	MGSO4 I	P	AN-4	E
1	10	1.6	N	N	S D	P	N	R
3	8	2	N	N	L D	P	N	R
2	12	1.9	N	N	S D	P	AN-5	E
3	9	2.4	N	N	NO	P	N	R
1	9	1.2	N	N	S D	No	N	R
2	12	1.5	N	N	S D	P	N	R
1	9	2	N	N	S D	P	N	R
1	8.5	2.3	N	N	S D	No	N	R
1	9	1.5	N	N	S D	No	N	R
1	9	1.65	N	N	L D	No	N	R
2	9	1.8	N	N	S D	No	N	R
2	9	1.9	N	N	S D	No	N	R
2	10	2.1	N	N	S D	No	N	R
4	10	2.3	N	N	S D	No	N	R
2	9.5	0.92	N	N	S D	P	AN-6	E
1	9	1.8	N	N	S D	P	N	R
1	7.5	1.58	N	N	L D	No	N	R
3	11	1.37	N	N	S D	No	N	R
1	9.5	1.6	N	N	S D	P	AN-7	R
2	9	1.8	N	N	S D	P	N	R
1	10.5	1.5	N	N	L D	No	N	R
1	9.5	2	N	N	S D	No	N	R
2	7	1.5	N	N	S D	P	N	R
1	11	1.5	N	N	NO	P	N	R
2	8	2.2	N	N	S D	No	N	R
1	10	1.6	N	N	L D	P	N	R
2	10	1.8	N	N	L D	No	N	R

1	9	1.5	N	N	L D	No	N	R
2	10	1.7	N	N	S D	No	N	R
2	9	1.8	N	N	L D	No	N	R
1	10	1.7	N	N	S D	No	N	R
1	10	1.6	N	N	S D	P	N	R
2	9.5	2	N	N	S D	P	AN-8	E
3	9	1.7	N	N	S D	No	N	R
2	10	1.6	N	N	S D	No	N	R
3	10	1.8	N	N	S D	No	N	R
1	9	2	N	N	S D	No	N	R
3	11.5	2.1	N	N	S D	No	N	R
2	9	1.8	N	N	S D&L D	P	N	R
2	9	1.3	N	N	S D	P	N	R
2	9	2	N	N	S D	No	N	R
1	9.5	1.6	N	N	S D	No	AN-9	R
3	9	1.6	N	N	S D	No	N	R
1	8.6	1.8	N	N	S D	P	N	R
1	8	2.1	N	N	S D	P	N	R
2	9	1.8	N	N	S D	P	N	R
2	8.5	1.4	N	N	S D	No	N	R
4	9.5	1.8	N	N	S D	No	N	R
2	9	1.4	N	N	S D	No	N	R
2	8.5	3	N	N	S D	No	N	R
1	8.5	1.4	N	N	S D	No	N	R
3	11	1.5	N	N	S D	No	N	R
2	9.5	1.4	N	N	S D	No	N	R
2	9.5	2.6	N	N	S D	P	N	R
2	9	2	N	N	S D	P	AN-10	R
3	10.6	1.1	N	N	S D	No	AN-11	R
2	9	4	N	N	S D	P	AN-12	R
2	8.5	1.6	N	N	S D	No	AN-13	R
4	9.5	1.15	N	N	S D	No	N	R
2	11	0.37	AN	N	MGSO4 IN	No	AN-14	E

2	9	1.6	N	N	S D	P	N	R
2	9	1.4	N	N	S D	P	AN-15	R
2	9	1.3	N	N	S D	P	N	R