A DISSERTATION ON STUDY OF CORRELATION BETWEEN NEUROIMAGING AND NEUROLOGICAL PRESENTATION IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA AND ITS MATERNAL OUTCOMES

Submitted to

THE TAMIL NADU DR. MGR. MEDICAL UNIVERSITY,

CHENNAI–600032. TAMILNADU.

In partial fulfillment of the regulations

For the award of the degree of

M.S. DEGREE

OBSTETRICS AND GYNECOLOGY

May 2019

GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL COLLEGE, SALEM, TAMILNADU.

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE & HOSPITAL

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled **STUDY ON CORRELATION BETWEEN NEUROIMAGING AND NEUROLOGICAL PRESENTATION IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA AND ITS MATERNAL OUTCOMES** is a bonafide and genuine research work carried out by me under the guidance of **Prof Dr.S.S.SUBHA, M.D, D.G.O., Professor and Head of Department,** Department of obstetrics and gynaecology, Government Mohan Kumaramangalam Medical College Hospital, Salem, Tamil Nadu, India.

Date:
Place: Salem

Signature of the Candidate
DR. S.ESWARI

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE & HOSPITAL

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation **STUDY ON CORRELATION BETWEEN NEUROIMAGING AND NEUROLOGICAL PRESENTATION IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA AND ITS MATERNAL OUTCOMES** is a bonafide work done by **DR. S.ESWARI** in partial fulfillment of the requirement for the degree of M. S. in Obstetrics and Gynaecology, examination to be held in 2019.

Date:
Place:Salem

Signature of the Guide
Prof. Dr. S.S.SUBHA , MD, DGO.,
Professor and Head of Department,
Department of Obstetrics and Gynecology,
Government Mohan Kumaramangalam
Medical College& Hospital,
Salem, Tamil Nadu.

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE & HOSPITAL

ENDORSEMENT BY THE HEAD OF DEPARTMENT

This is to certify that this dissertation titled **STUDY ON CORRELATION BETWEEN NEUROIMAGING AND NEUROLOGICAL PRESENTATION IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA AND ITS MATERNAL OUTCOMES** is a bonafide work done by **Dr. S.ESWARI,** under overall guidance and supervision of **Prof. Dr.S.S.SUBHA, M.D., D.G.O., Professor and Head of Department**, Department of Obstetrics and Gynecology, Government Mohan Kumaramangalam Medical College Hospital, in partial fulfillment of the requirement for the degree of M. S. in Obstetrics and Gynecology, examination to be held in 2019.

Date :

Place : Salem

Seal & Signature of the HOD
Dr. S.S.SUBHA, MD.,DGO.,
Professor and Head
Department of Obstetrics and Gynaecology
Government Mohan Kumaramangalam Medical Colle
Hospital, Salem, Tamil Nadu, India

GOVERNMENT MOHAN KUMARAMANGALAM
MEDICAL COLLEGE & HOSPITAL

ENDORSEMENT BY THE DEAN OF THE INSTITUTION

This is to certify that this dissertation entitled **" STUDY ON CORRELATION BETWEEN NEUROIMAGING AND NEUROLOGICAL PRESENTATION IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA AND ITS MATERNAL OUTCOMES"** is a bonafide work done by **Dr. S.ESWARI** under guidance and supervision of **Prof. Dr. S.S.SUBHA , MD., DGO., Professor and Head of Department,** Department of Obstetrics and Gynaecology, Government Mohan Kumaramangalam Medical College Hospital, in partial fulfillment of the requirement for the degree of M. D. in Obstetrics and Gynaecology, examination to be held in 2019.

Date :

Place : Salem

Seal Signature of the Dean
DEAN
Government Mohan Kumaramangalam
Medical College and Hospital
Salem, Tamil Nadu, India

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE & HOSPITAL

COPYRIGHT

I hereby declare that the Government Mohan Kumaramangalam Medical College Hospital, Salem, Tamil Nadu, India, shall have the rights to preserve, use and disseminate this dissertation / thesis in print or electronic format for academic / research purpose.

Date:
Place: Salem

Signature of the Candidate
DR. S.ESWARI

ACKOWLEDGEMENT

It gratefully acknowledge and sincerely thank our beloved **Dean, Prof. Dr.M.K. RAJENDRAN, M.S., M.Ch.,** for permitting me to utilize the infrastructure and resources needed to conduct this study in Govt. Mohan Kumaramangalam Medical College Salem.

At the start, I would like to start by thanking my Head of the Department and guide **Prof. DR.S.S.SUBHA, MD., DGO.**, for her constant support and encouragement throughout my study. I am greatly indebted to her, for her timely suggestions and advise which has helped me to complete my study.

I thank my co-guide **Associate Prof. DR.D.MOHANA, MD., DGO.,** for their constant guidance and helping me to understand the basics of research and in correcting my manuscript.

I am greatly thankful to all my assistant professors who helped me throughout the study. I thank my co-postgraduates, who have been with me and helped me in many ways in this study.

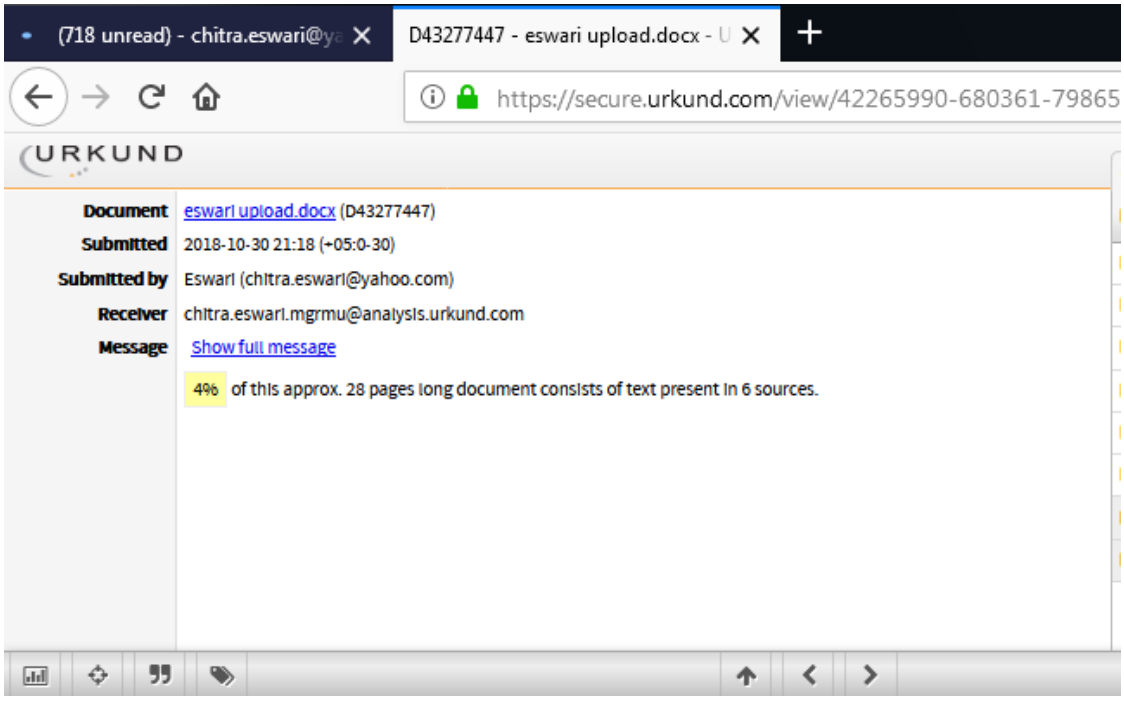
It would be incomplete if I don't thank my family, my parents, sister, husband and roommate for their constant support and encouragement.

Last but not the least, I'm very greatly indebted to my patients without whose co-operation it would have been impossible to do this study.

PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled **STUDY ON CORRELATION BETWEEN NEUROIMAGING AND NEUROLOGICAL PRESENTATION IN ANTEPARTUM AND POSTPARTUM ECLAMPSIA AND ITS MATERNAL OUTCOMES** of the candidate **Dr. S.ESWARI** with registration Number **221616702** for the award of **M.S.,** Degree in the branch of Obstetrics and Gynaecology. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **4%** percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.



URKUND

Urkund Analysis Result

Analysed Document: eswari upload.docx (D43277447)
Submitted: 10/30/2018 4:48:00 PM
Submitted By: chitra.eswari@yahoo.com
Significance: 4 %

Sources included in the report:

INTRO AND ROL FOR PLEG.docx (D42571165)
dhivya Final updated 15-10-2018.docx (D42583459)
A prospective study for the prediction of preeclampsia with serum prolactin level.docx (D27818210)
AARIFA DISS FINAL.docx (D42422891)
http://rguhs.ac.in/cdc/onlinecdc/uploads/01_M012_26366.doc
http://jfrh.tums.ac.ir/index.php/jfrh/article/view/634

Instances where selected sources appear:

18

CONTENTS

INTRODUCTION

Eclampsia and Pre eclampsia are two clinical situation exclusively associated with pregnancy. Eclampsia is one of the obstetric emergencies where resuscitation plays an important role and requires regular drills to optimise management. It may occur swiftly without any warning manifestations. However in majority of the cases eclampsia proceeded by features of preeclampsia. Preeclampsia is defined as new onset hypertension of blood pressure more than 140/90 mm Hg associated with proteinuria or end organ damage after 20 weeks of gestation .If it occurs in early trimester diagnosis of hydatiform mole has to be considered. Eclampsia currently defined as women with preeclampsia, a convulsion that cannot be attributed to another cause is termed as eclampsia[1]. Though it is multisystem complex disorder, central nervous system involvement is more common.

A multidisciplinary team has to be involved and should include anaesthetist, obstetric physician, and consultant obstetrician. The incidence of Eclampsia 1 in 2000 deliveries in developed countries , in developing countries varies from 1 in 100 to 1 in 1700. Although the incidence and mortality has comes down over the past several decades due to better antenatal care, early detection and management of preeclampsia ,associated maternal and fetal morbidity and mortality is still significant. Major maternal complications include 7-10% placental abruption, 7 -11%DIC, 9-20%HELLP, 2 -3% aspiration pneumonitis, 3- 5 % pulmonary edema, 2-5% cardiopulmonary arrest,

2- 3%cerebral haemorrhage,5-9% acute renal failure.The perinatal mortality associated with eclampsia accounts for 5-12% .The onset of Eclamptic convulsions may be antepartum(38-53%), intrapartum (18-36%), or postpartum (11-44%).

Neurological complication includes mental confusion, seizures, deficits in visual fields, headache, blurring of vision, coma, cerebrovascular accidents,hemiparesis, pappilloedema,cortical blindness. The recognition of premonitory symptoms lowers maternal mortality and morbidity.At neuroimaging the abnormal findings are cerebral edema, intra cerebral haemorrhage, cortical vein thrombosis, posterior reversible encephalopathy, Hypertensive leucoencephalopathy. This dissertation is designed to correlate neurological presentation with neuroimaging (CT/MRI) in antepartum and postpartum eclampsia patients which will help in proper arrival of diagnosis and management. CT is a quick early imaging method . MRI findings abnormality noted in most of the women. Findings includes an increased signal intensity at grey white matter junction on T2 weighted images, with cortical edema and hemorrhage. Posterior reversible syndrome has been increasingly reported in patients with Eclampsia which reflects central vasogenic edema. PRES was first described in 1996.In this dissertation several analysis is done to investigate various factors associated with eclampsia like blood pressure, gestational age at presentation,maternal complications.

AIMS AND OBJECTIVES

The aim of the study is to correlate neuroimaging and neurological presentation in antepartum and postpartum eclampsia and its maternal outcomes. The objectives of present study are given below.

1) To assess the utility of radiological (CT/MRI) imaging by correlating the clinical presentation and neuroimaging and to identify abnormalities which will help in proper arrival of diagnosis and treatment.

2) To evaluate maternal morbidity and mortality associated with eclampsia

REVIEW OF LITERATURE

Eclamptic convulsions eras back to 4000 years which was recognised in Indian, ancient Chinese and greek literature.Around 400 BC,Hippocrates stated that the headache with convulsions during pregnancy was considered bad. Bossier de saurages (1739) coined the term“Eclampsia”. John Lever(1843) reported that proteinuria was specific to preeclampsia condition.

Eclamptic hypertension was discovered by Vasquez and Nobcourt (1897).Writings showed eclampsia had been traced as far as back 2200 BC . Chesley (1984) said that sensory stimuli were decreased by keeping patients in quiet dark room. Horn (1906) was described first use of magnesium sulphate on pre-eclampsia and Eclampsia. Several studies demonstrate that magnesium sulphate is superior over other anticonvulsants.

Crawford *et al.,* (1987) performed serial MRIs on an eclamptic women, with findings compatible with neuropathologic changes. Magnetic resonance imaging (MRI) is a developed neuroimaging technique that appears superior to other processes for defining intracranial anatomy and pathophysiology .

Duncan *et al*., (1989) described three cases of cortical blindness in eclampsia women with magnetic resonance imaging (MRI) and CT. They concluded that the correspondence of MRI and low attenuation lesions on CT scan specified ischaemia than haemorrhage as the pathological mechanism .

Vandenplas *et al*., (1990) studied the magnetic resonance evaluation of severe neurological disorders in eclampsia. They explained that initial magnetic resonance imaging (MRI) exposed multiple hyperintense areas throughout the brain and brainstem that were consistent with ischemia and/or edema. MRI abnormalities were found to correlate more closely with clinical and electrophysiological data than CT findings.

Royburt *et al.*, (1991) reviewed the neurologic involvement in hypertensive disease of pregnancy. They summarized as, the current knowledge on the pathophysiologic changes in the central nervous system (CNS) is caused by pregnancy induced hypertension.

Digre *et al*., (1993) considered 16 patients with severe preeclampsia and 10 patients with eclampsia at University of Utah College of Medicine, Saltlake city and concluded that the women with eclampsia had specific changes in MRI like cortical edema and haemorrhage as against nonspecific changes in severe preeclampsia. Therefore, neuroimaging is useful in cases with diagnostic uncertainty, different presentation or focal neurological deficit.

Chang *et al*., (1996) concluded that the temporoparietooccipital junction is most frequently involved area. They stated about visual disturbance and headache that has a good correlation with radiological findings, that was occipital lobe involvement and diffuse brain edema, respectively.

Dittmar (1999) studied the neurological spectrum of preeclampsia and eclampsia. He explained preeclampsia and eclampsia by an endothelial dysfunction with increased vascular sensitivity to circulating pressure agents and structural endothelial lesion with fluid loss from the intravascular compartment

Richard et al., (2000) conducted study at University of Nebraska School of Medicine, Omaha and showed that the brain edema at MR imaging had a significantly greater incidence of abnormal red blood cell morphology and higher levels of lactic dehydrogenase than with normal MR imaging findings.

Study done at Department of Radiology, Izmir Ataturk Training and Reasearch Hospital, Izmir, Turkey in December 2005 to compare patients with and without MRI findings showed that increased permeability of the blood-brain-barrier in relation to endothelial injury plays a major role in the pathogenesis of preeclampsia/eclampsia.

Study at Department of Obstetrics &Gynecology, Istanbul University, Istanbul School of Medicine, Istanbul, Turkey on 120 patients with severe preeclampsia, eclampsia and HELLP syndrome between January 1998 to December 2005 showed that it was essential to perform cranial imaging in patients with symptoms and neurological deficits.

Bartynski and Boardman (2007) stated that vasogenic edema around 98 per cent present in the parietal or occipital regions, but other locations also common. Frontal, inferiorparietal, cerebellar hemispheres are affected often.

Kokila *et al*.,(2011) studied the correlation of clinical and neuro imaging findings affecting management in postpartum eclampsia . They observed that many abnormalities were detected on imaging without any chronic neurologic sequelae. Hence expensive neuroimaging had limited role in typical cases and cases which were responding to prompt therapy. They concluded that neuroimaging was indicated in atypical and fatal cases.[2]

Jindal *et al.,* (2013) conducted a prospective observational study to compare radiological findings of eclampsia patients with respect to neurological signs and symptoms.The symptoms ranged from headache,altered sensorium to coma .Study concluded symptoms like visual blurring , loss of vision, and ophthal findings in eclampsia suggested occipital lobe involvement.MRI imaging abnormalities correlate well with clinical findings in eclampsia .[3]

Khawla Abu Samra (2013) discussed the ophthalmic complications of preeclampsia/eclampsia with focus on the hypertensive retinopathy, exudative retinal detachment and cortical blindness. He concluded about 25 per cent of patients with severe preeclampsia and 50 per cent of patients with eclampsia had visual symptoms [4].

Paanu et al in the year 2014 studied maternal and perinatal outcome in eclampsia and factors affecting the outcome .They stated that inadequate and delayed initiation of treatment and preterm deliveries was found to linked with poor maternal outcome.Time interval between onset of fit and delivery directly

correlated with poor maternal and fetal outcome.Age,parity, onset of convulsions before, during, after delivery was not found to have any effect on maternal and fetal outcome.From all datas they concluded that better antenatal care,early recognition of disease, referral in time, early initiation of treatment and termination of pregnancy improved the outcome[5]

Ugran *et al.,* (2016) conducted a prospective study 100 patients were included in that study. The neurological presentation and neuroimaging findings correlated in eclampsia patient. Study showed that 52 patients have had CT findings. Out of 52, 35 patients had neurological signs and symptoms, 17 women did not have neurological symptoms and signs. Study concluded that neuroimaging has limited role in uncomplicated cases and neuroimaging is mainly necessary for atypical presentation, fatal cases, those cases resistant to antiepileptic therapy, where signs and symptoms are less likely to predict the diagnosis.[6]

Sharma *et al.*,(2016) studied eclampsia preeclampsia with respect to clinical and neurological correlations. They observed that the patients with severe preeclampsia and positive cerebral findings on MRI had more possibilities of buildup convulsions as compared with negative findings on MRI. They suggested that PRES was a predecessor to eclamptic convulsions. This study group had extreme benefit from aggressive therapy [7].

Mahalakshmi et al studied the maternal and perinatal outcome of eclampsia in a tertiary hospital in the year 2016. It was a retrospective study conducted from Jan 2015 –December 2015 on all eclampsia cases .Parameters like age, parity, gestational age, mode of delivery, admission to delivery interval, maternal and perinatal outcome were studied. From that they concluded eclampsia is associated with higher rates of maternal complications in developing countries like india[8]

Bhanu et al(2017) studied the maternal complications associated with eclampsia.They highlighted that eclampsia is still the major cause of maternal morbidity and mortality in India.Information about imminent signs and symptoms has to be obtainable to antenatal patients.They also emphasised on timely referral to the tertiary center[9] .

CLASSIFICATION OF HYPERTENSIVE DISORDERS IN PREGNANCY: National High Pressure Education program classification (2000)

Gestational Hypertension:

Hypertension that develops first time in pregnancy after 20 weeks of gestation, not accompanied by proteinuria.Blood pressure returns to normal within 12weeks postpartum,final diagnosis is made only after12 weeks postpartum.

Pre eclampsia, Eclampsia

Chronic hypertension:

Hypertension diagnosed before pregnancy or before20 weeks of gestation

Pre eclampsia superimposed on Chronic Hypertension :

When a pregnant woman with chronic hypertension develops proteinuria after 20 weeks , superimposed preeclampsia is diagnosed.

Conventional mercury sphygmomanometry is gold standard device for blood pressure measurement. Blood pressure has to be measured with mother seated or reclined at 45º, with her feet on the ground or well supported, her arm at the level of heart. The right arm should be used with the cuff of the appropriate size. Electronic blood pressure monitors may miscalculate the accurate pressure.

POSITIONING OF PATIENT FOR BP RECORDING

According to brown et 1998 korotkoff phase V be used as a measure of diastolic pressure

1. Korotkoff 4/ Korotkoff 5 difference is minor in hypertensive than in normotensive pregnant women.
2. Korotkoff 5 which is close to actual intra-arterial pressure, physiologically more accurate, is more consistently detected and is reproducible.
3. Korotkoff 4 has restricted reproducibility

PREDISPOSING FACTORS FOR ECLAMPSIA AND PRE ECLAMPSIA:

COUPLE RELATED RISK FACTORS

Primipaternity and primiparity: A new partner contributes to preeclampsia in women's second pregnancy is called as dangerous father. The incidence is about 24% in new paternity multipara because they have shorter period of sperm exposure preceding conception. Incidence of preeclampsia in nulliparous 3 to 10% and in multiparous less than that for nulliparous women[10]

Pregnancies after donor insemination, donor egg, donor embryo Dangerous male partner –men who fathered a preeclamptic pregnancy were nearly twice likely to father another such pregnancy with different women

MATERNAL RELATED RISK FACTORS

Age :

Patients <20 and >30 years displayed increased incidence of preeclampsia. In patients> 35 years Pre-eclampsia accounts for over 40% of pre-mature deliveries with increased of risk of low birth weight and small for gestational age babies

Race

Muslims, jews and arabs have higher incidence of preeclampsia and eclampsia. Myatt et al 2012a conducted a study showed that Africoamericans have higher incidence(11%) compared to 5% in white , 9% in hispanic[11]

Social status:

Women from low social economic status are said to have higher incidence of preeclampsia, Eclampsia. Baird and colleagues 1969, in their study said that socioeconomic status does not show any difference in incidence.

Smoking

Smoking causes a substantial reduction in HCG and estradiol level due to direct effect on the placental function

Previous H/O eclampsia

Preeclampsia is seen in subsequent pregnancies 35% when there was history of eclampsia in the previous pregnancies [12] Maternal age and interval between pregnancies directly proportional to the risk of preeclampsia and eclampsia.

Family history

Family history of eclampsia and pre eclampsia will have impact on the current pregnancy.

Urinary tract infection:

UTI results in increased production of inflammatory products such as cytokines, free radicals and proteolytic enzymes that causes endothelial dysfunction. Hence screening and treatment for urinary tract infection should be part of routine antenatal care[13]

PREGNANCY ASSOCIATED RISK FACTORS

Twin gestation:

Incidence of preeclampsia 25.3% due to hyperplacentosis with augmented secretion of placental hormone, accompanying placental ischemia and immunological reaction.

Molar pregnancy:

Incidence of preeclampsia is 70% in women with fast growing moles. There is no increased incidence of preeclampsia with slowly growing moles.

Congenital malformations:

There is risk of emerging preeclampsia is 35% due to placentomegaly in triploidy

Hydropsfetalis:

Hyperplacentosis in hydropsfetalis surges the risk.

Obesity and insulin resistance/ gestational diabetes

The risk of preeclampsia doubles with each 5 -7 kg/m^2 increase in prepregnancy BMI[35]

PRE EXISTING MEDICAL DISEASE

Pre gestational diabetes mellitus

Chronic hypertension or renal disease

Maternal immunological disease

Pre existing thrombophilia, antiphospholipid antibody syndrome.

There was evidence of higher rate of acquired and genetic thrombophilia in women with early onset preeclampsia and eclampsia compared with controls

PREMONITARY SYMPTOMS:

A) **Headache-** due to cerebral hyperperfusion that have more tendency towards occipital lobes. Sibai (2005) and Zwart (2008) stated that 50% to 75% of women have headache preceding eclamptic convulsion.Severity of headache varies from mild to severe and intermittent to constant.They do not respond to routine analgesics, but severity reduces after magnesium sulphate therapy.

B) **Epigastric or right upper quadrant pain-** Due to hepatocellular necrosis, ischaemia,edema that stretches Glissons capsule.This pain commonly associated with elevation in hepatic transaminase levels.

C) **Visual disturbances or scotoma**

D) **Reduced urine output**

CLINICAL FEATURES OF ECLAMPSIA:

Eclampsia is a clinical diagnosis grounded on occurrence of one or more generalised tonic clonic convulsions/coma in a preeclamptic women.All pregnant women with fits has to be considered to have eclampsia unless proved otherwise.Typical eclamptic convulsions do not have focal neurological deficits or continued coma

STAGES OF ECLAMPTIC FIT:

The convulsions are epileptiform , comprising of four stages

Stage1: Premonitory stage- This stage last for about 30seconds.The women become unconscious, twitching of muscles of the face, tongue and limbs.Eyeballs roll or turned to one side and fixed.

Stage 2: Tonic stage- Duration of this stage is about 30 seconds.The whole body goes for tonic spasm, trunk-ophisthotonus,limbs are flexed and hands clenched.Respiratory effort ceases, tongue protrudes between the teeth.Patient becomes cyanosed. Eyeballs becomes fixed.

Stage 3: Clonic stage – This stage lasts upto 1-4 minutes. All voluntary muscles go for alternate contraction and relaxation.Twitchings start in the face then involve one side of the extremities and ultimately whole body is involved.Biting of tongue follows.Breathing is stertorous , cyanosis progressively disappears.Blood stained frothy secretions fills mouth

Stage 4: stage of coma- Following convulsion patient develops stage of coma which last for short period. Some women develop deep coma persist or postictal confusion state and unable to remember the happenings.

The fits are often multiple, when it occur in quick succession called as status epilepticus. Following convulsions blood pressue ,temperature,pulse rate, respiratory rate, blood uric acid increased, urinary output may be significantly reduced, proteinuria pronounced,

BIOCHEMICAL CHANGES IN BRAIN:

Convulsions are diagnostic of eclampsia,caused by excessive release of excitatory neurotransmitters especially Glutamate.This neurotransmitter causes massive depolarization of neurons results in burst of action potential (Meldrum 2002)

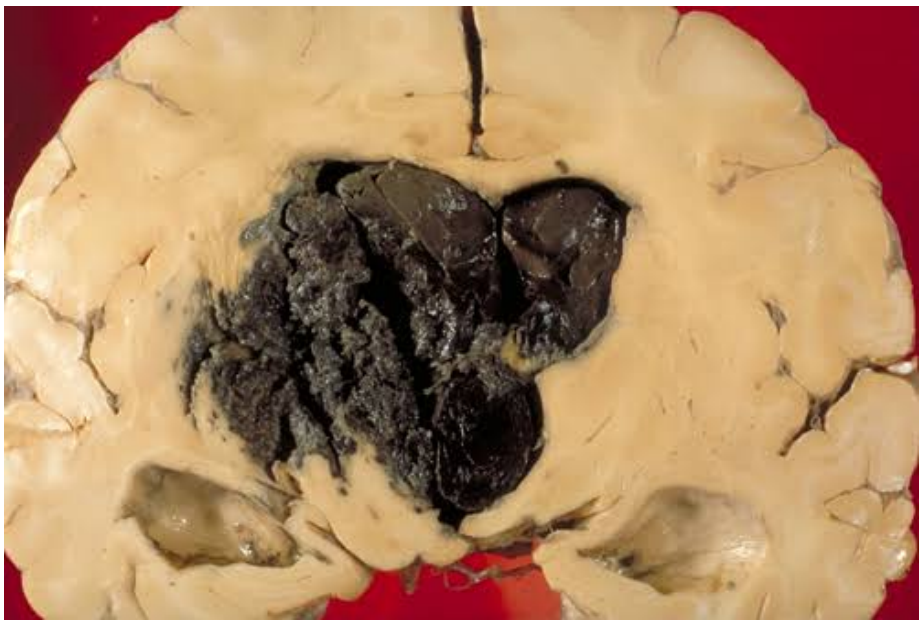
CEREBRO VASCULAR PATHOPHYSIOLOGY IN ECLAMPSIA:

In earlier period brain involvement in eclampsia and anatomical findings came from autopsy specimens. CT and MRI and Doppler studies in recent days added important insights into cerebrovascular involvement.

1) ANATOMICAL LESIONS IN BRAIN:

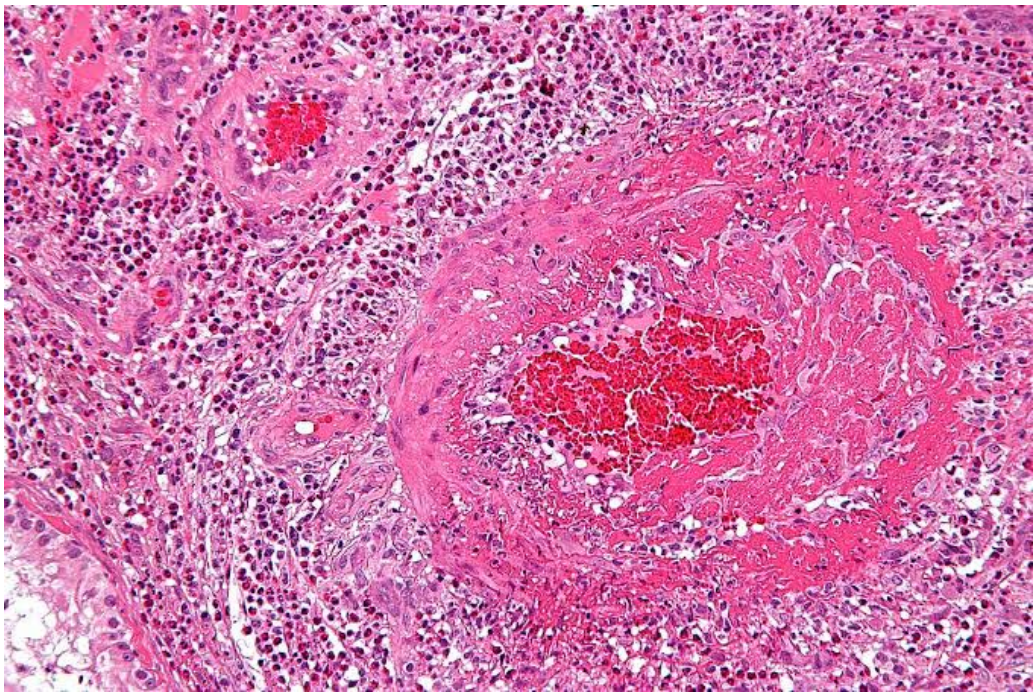
Accoring to Melrose ,1984; Richards ,1988; Sheehan 1973 gross intracerebral haemorrhage was seen upto 60% of eclamptic patients , it was fatal in only half of these.Other lesions found at autopsy were cortical and subcortical hemorrhages,subcorticaledema, non haemorrhagic areas of softening , hemorrhagic areas in white matter.

INTRACEREBRAL HAEMORRHAGE



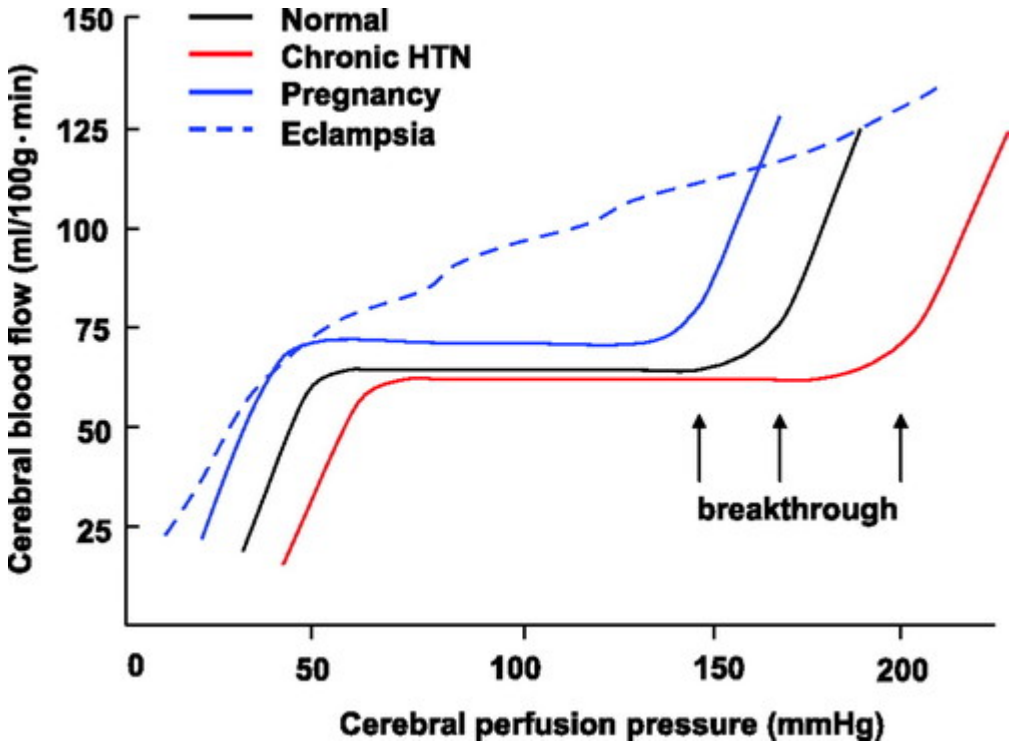
On microscopic examination the vascular lesions consist of fibrinoid necrosis of arterial wall with perivascular microinfarcts and haemorrhages.

FIBRINOID NECROSIS



2)CEREBRAL BLOOD FLOW:

Autoregulation is the mechanism , so that cerebral blood flow remains constant despite alteration in cerebral perfusion pressure. In normal non pregnant women this autoregulatory mechanism protects brain from hyperperfusion even though mean arterial pressure raises more than 160 mmHg. To explain eclamptic convulsions, it was hypothetised that autoregulation must be altered during pregnancy.



Regarding species differences Cipolla et al in the year of 2014 had demonstrated that autoregulation is unchanged in rodents thoughout pregnancy. Janzarik in the same year provided evidence of impaired autoregulation in preeclamptic women.[14]

Zeeman et al (2003) showed evidence that cerebral blood flow remains unaltered during first and second trimester. During third trimester it decreases by 20%, but in preeclamptic and eclamptic women they had documented increased cerebral blood flow results in endothelial damage which forces capillary fluid into the interstitial compartment , which leads to perivascular edema.

3) THEORIES TO EXPLAIN CEREBRAL ABNORMALITIES:

A)Endothelial cell dysfunction:-

Acute and severe hypertension

↓

Cerebral vasospasm

↓

Diminished cerebral blood flow

↓

ISCHEMIA,cytotoxic edema,

↓

Tissue infarction

There was less objective evidence to support this theory.

B) Vasodilation and vasoconstriction theory:

Sudden elevation in systemic blood pressure

(Blood pressure exceed normal cerebrovascular autoregulatory capability Hauser -1988; Schwartz 2000)

↓

Regions of enforced vasodilation and vasoconstriction

↓

At capillary level

↓

Interruption of end capillary pressure

↓

Increased hydrostatic pressure, hyperperfusion

↓

Extravasation of plasma and red cells through endothelial tight junction

↓

Vasogenic edema

This theory is inadequate because limited eclamptic women have mean arterial pressure that exceeds limits of autoregulation, around 160 mmHg.

To determine the most likely mechanism is combination of two above said theories

Zeeman(2009) stated that preeclampsia related interendothelial cell leakage develops at lower blood pressure level than those usually causing vasogenic edema , and combined with a loss of upper limit of autoregulation.

In imaging studies these changes are apparent as reversible posterior leukoencephalopathy syndrome, which consequently referred as posterior reversible encephalopathy syndrome- PRES. This term is misleading because PRES lesions predominantly involve posterior brain parietooccipital cortex which is boundary zone of anterior,middle, posterior cerebral arteries. According to Edlow 2013; and Zeeman 2004a in atleast third of cases other brain areas also involved.[15] These lesions are reversible.

One likely explanation for posterior predominance of brain lesions is that the anterior circulation is much better supplied by sympathetic innervations , better protected against the effects of elevated blood pressure.The level of cerebral perfusion pressure required to cause barotraumas and seizures varies between individuals.

4**.NEUROLOGICAL MANIFESTATIONS**:

A**)** **Headache and scotoma-** due to cerebral hyperperfusion

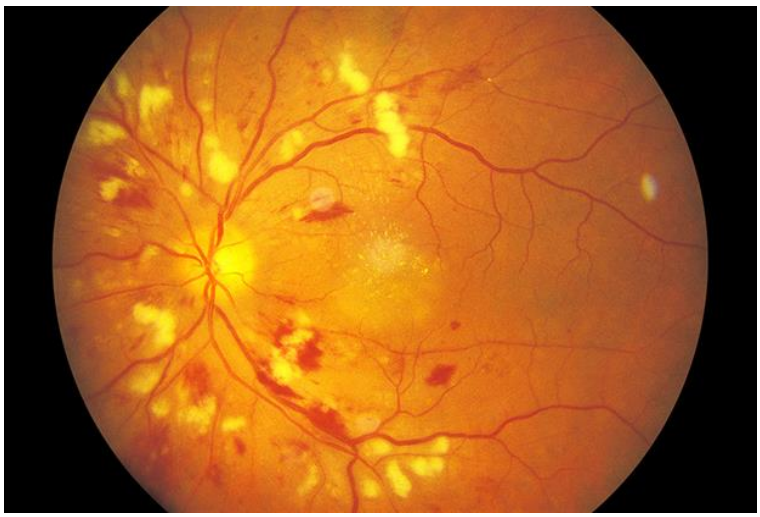
B) **Convulsions:**It is second likely manifestation and diagnostic neurological manifestation of eclampsia.Evidences suggested that extended convulsions cause significant brain injury and brain dysfunction later life.

C) **Blindness** is infrequent with pre eclampsia alone, but when complicated by eclamptic convulsions it may occur upto 15% of women(Cunningham 1995).Chambers in the year 2004 reported that blindness develop upto one week or more following delivery.Scotoma, blurred vision or diplopia are communal with severe preeclampsia and eclampsia, they typically improve with magnesium sulphate therapy, blood pressure control.Blindness is less common , usually reversible. Blindness may arise from 3 potential areas in brain. 1) visualcortex of occipital lobe 2)Lateral geniculate nuclei 3)Retina(Roos 2012)

There are atleast 2 types of blindness common with eclampsia.

1) **Occipital blindness** – otherwise called as amaurosis is derived from greek word dimming, Women will have evidence of occipital lobe vasogenic edema on neuroimaging studies.Some times extensive cerebral infarction may cause total or partial visual loss

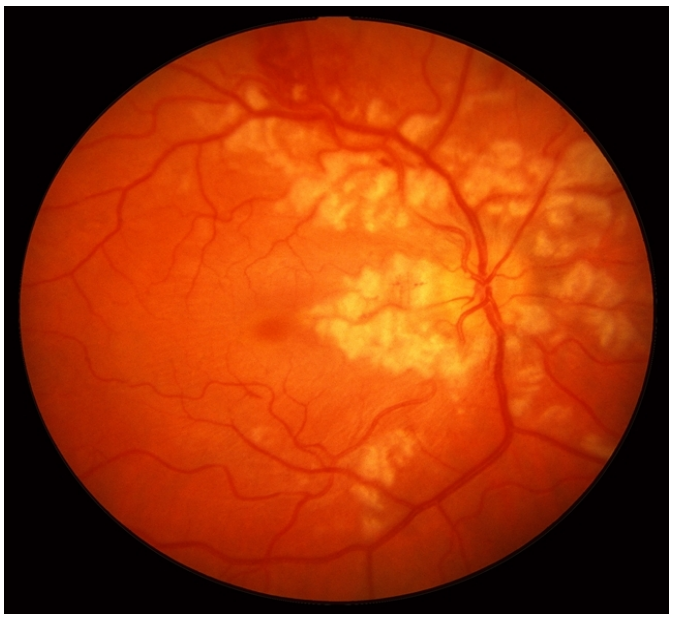
COTTON WOOL SPOTS AND HAEMORRHAGES



2) **Blindness from retinal lesions** caused either by serous retinal detachment or infrequently by retinal infarction which is known as Purtscher retinopathy.Serous retinal detachment generally unilateral, hardly ever cause total vision loss.Aymptomatic cases are common.

Moseman and Shelton in the year 2002 described permanent blindness was due to combination of retinal infarction and bilateral lesions in lateral geniculate body.

PURTSCHER RETINOPATHY



GRADINGS OF HYPERTENSIVE RETINOPATHY:

GRADE 1 : Minimal narrowing of retinal arteries

GRADE 2 : Narrowing of retinal arteries in association with regions of focal narrowing and arteriovenous nipping

GRADE 3 : Abnormalities in Grade 1 & 2 with retinal hemorrhages, hard exudation, and cotton wool spots

GRADE 4 : Papilloedema which warrants termination of pregnancy.

D) Generalised cerebral edema:

Symptoms ranged from lethargy, confusion, and coma, symptoms are usually waxing and wanning type.These women are susceptible to sudden change in blood pressure that is severe blood pressure elevation, which can

worsen the vasogenic edema.Thus cautious blood pressure control is very crucial. Death may occur from transtentorial herniation.

E) Long term neurocognitive sequelae:

In the past eclamptic seizures were believed to have no significant long term sequaelae. But Zeeman (2004) reported that all eclamptic women have multifocal areas of perivascular edema, with cerebral infarction.

Aukes et al in the year 2012 founded that long term persistence of brain white matter lesions that were acquired during eclampsia,and formerly eclamptic women had impaired cognitive functioning.

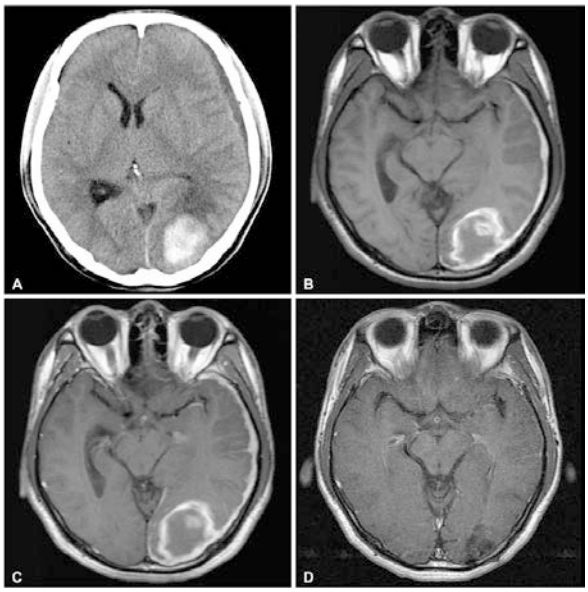
Postma in the year 2009 reported that women with multiple seizures had impaired attention compared to normotensive women.

Weigman et al -2012 described that eclamptic patients after approximately 10 years had lower vision compared to control groups.

NEUROIMAGING STUDIES

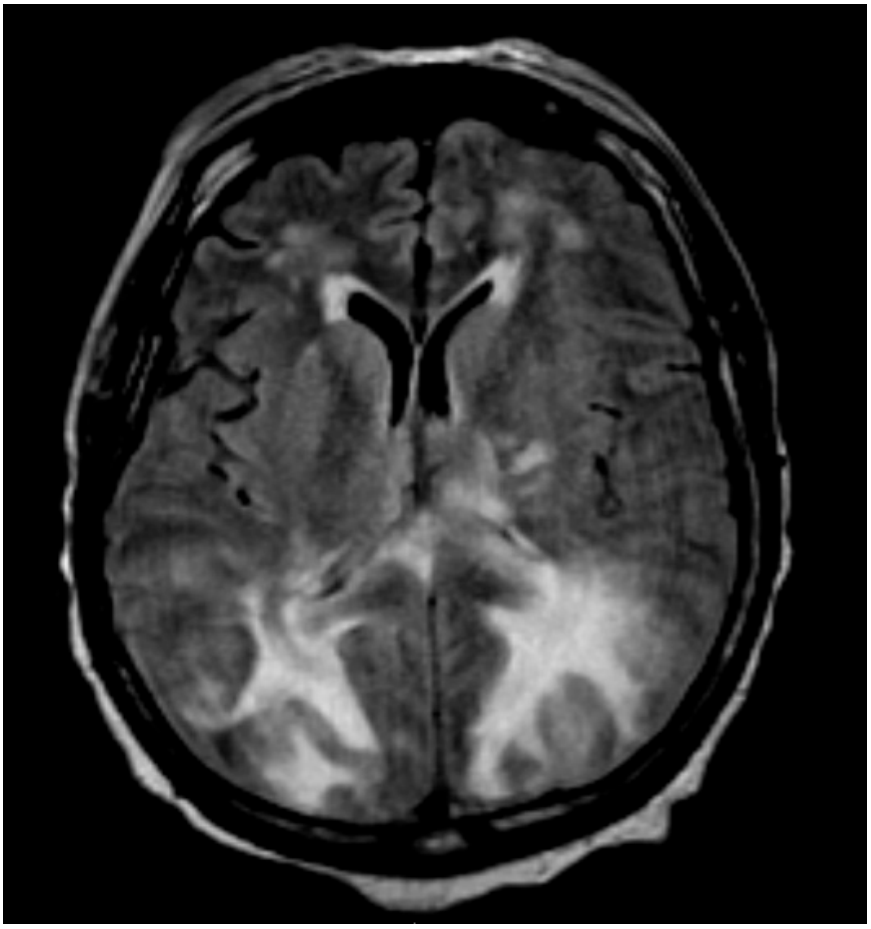
The spectrum of brain involvement wide. CT imaging localised hypodense lesions at greywhite matter junction,concerning parieto occipital areas are characteristically found in eclampsia.Similar lesions may also found in frontal and inferior parietal lobes, thalamus and basal ganglia.Edema of occipital lobes or diffuse cerebral edema may casuse blindness, lethargy, confusion.In advanced cases, diffuse edema will result in marked compression or obliteration of cerebral ventricles.

Regarding MRI common findings are isointense to hypointense on T1W1 and on hyperintenseT2W1 and FLAIR sequences that is posterior reversible encephalopathy syndrome in cortical and subcortical areas of brain involving parieto occipital lobes.Lesions are more common in white matter than in grey matter which is correalting with vasogenic edema and bilaterally symmetrical in nature.



Brewer et al 2013 stated as basal ganglia, brainstem, and cerebellum are other frequently involved brain areas[16] .The PRES lesions are virtually universal in eclampsia, their incidence in preeclampsia is less.They are most commonly seen in women with severe disease and those who have neurological symptoms.

MRI SHOWING PRES LESION:



DIAGNOSIS

Diagnosis of eclampsia is very clear when antenatal women presents with seizure , increased blood pressure, and associated proteinuria. Elevated blood pressure is the hallmark for the diagnosis of eclampsia.Regrettably 15% of women, hypertension and proteinuria may not be present.Presence of premonitory symptoms along with laboratory findings may helpful in arriving correct diagnosis when blood pressure and protenuria were not present.

INVESTIGATIONS:

Complete blood count:

Patients with mild preeclampsia there will be elevation in haemoglobin and haematocrit due to decrease in plasma volume .Haemoconcentration has been considered hallmark of eclampsia .This was explained by Zeeman et al (2009) and showed in eclamptic women that the normally expected hypervolemia is severly shortened. Haemoconcentration results from generalised vasoconstriction which leads to endothelial cell activation and plasma leakage into interstitial space .

Thus women with eclampsia were excessively sensitive to forceful fluid therapy. Eclamptic women are sensitive to amount of blood loss during delivery that were considered normal for normotensive women.

Thrombocytopenia with eclampsia had been described in the year 1922 by Stancke. Leduc in the year 1992 stated that platelet count and maternal , fetal

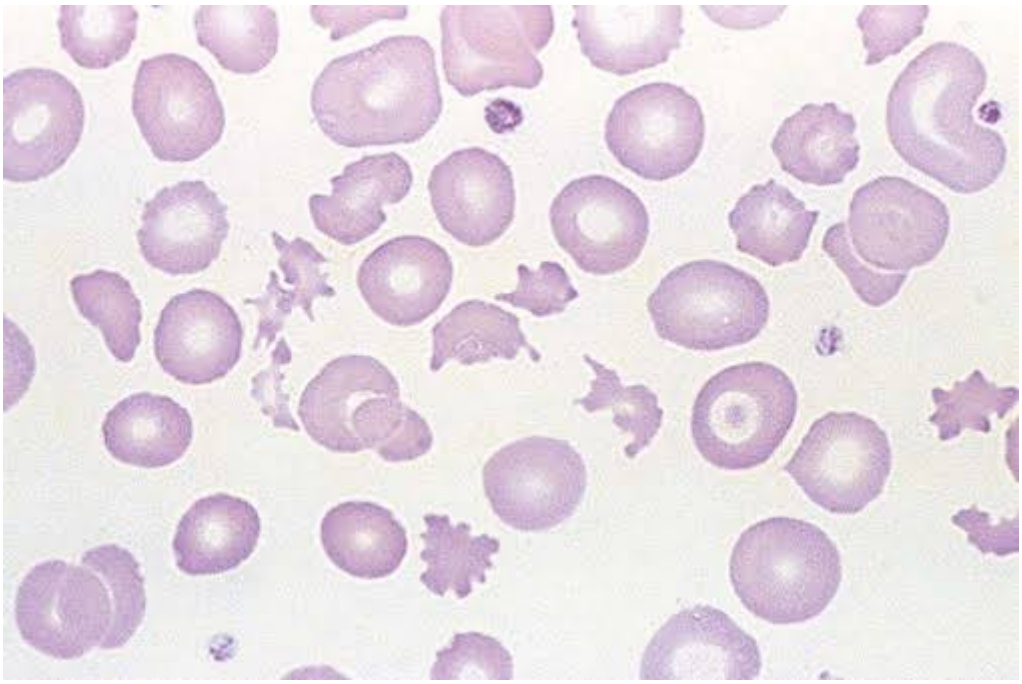
morbidity and mortality were inversely related. Delivery is recommended for thrombocytopenia.After delivery platelet count progressively improved to reach the normal level within 3-5 days.In women with HELLP syndrome , thrombocytopenia continues after delivery.

Kenny etal (2014) reviewed the platelet abnormalities stated that there were platelet activation with increased alpha degranulation which produces beta thromboglobulin and factor 4 and increased their clearance[17]

Peripheral smear study

In women with HELLP syndrome peripheral smear study may expose spherocytes, schistocytes,triangular cells and burr cells.

PERIPHERAL SMEAR:



Renal function test:

In normal pregnancy renal plasma flow and glomerular filtration rate are increased producing fall in blood urea, serum creatinine, serum uric acid.In women with preeclampsia there will be 25% reduction in GFR. The upper limits for serum creatinine 1.2 mgs% and for Blood urea nitrogen 20-25 mg/dl.Renal functions are altered in late stage of preeclampsia or HELLP syndrome. Serum creatinine elevation is associated with worsening of disease. Hyperuricemia is associated with renal dysfunction,glomerular endotheliosis and decreased tubular secretion.

LIVER FUNCTION TEST:

Liver involvement occurs in about 10% of cases of severe preeclamptic women.Bilirubin level elevation warrants diagnosis of HELLP syndrome.

RCOG reviewed guidelines stated that elevation in asparatate transaminase or alanine transaminase of about 70IU/L is considered significant and elevation above 150 IU/L has been associated with increased morbidity and mortality of women[18] . Haemolysis is evidenced by increased serum lactate dehydrogenase ,decreased haptoglobin levels.

Coagulatory changes:

Kenny (2014) stated that subsidiary changes consistent with intravascular coagulation are frequently found in women preeclampsia and eclampsia. The changes are increased factor8 consumption, elevated fibrinopeptides A and B ,

D-Dimer, diminished level of antithrombin 3,protein C and protein S. Plasma fibrinogen levels tends to fall in abruption with DIC

PROTEINURIA:

Presence of proteinuria establishes the diagnosis of preeclampsia. According to Zwart (2008) reported that 17% eclamptic women did not show proteinuria at the time of convulsions.

Chen et al (2008) showed that clean catched catheterised urine samples correlate well.

URINE DIPSTICK TESTS:

GRADINGS:

Trace: 0.1g/ L

1+ :0.3 g/L

2+ :1.0 g /L

3+ :3.0 g /L

4+ :10.0 g/L

Dipstick tests were depend on urinary protein concentration , and more prone for false positive and false negative reports.

24 hour quantitative specimen Task Force (2013) recommended the consensus threshold value of> 300 mg/24 hours[19] .Tun et al (2012) showed a

study stating that equivalent efficacy of using protein excretion of 165 mgs% in a 12 hour period sample.

Estimating the urine protein :creatinine ratio may displace the burdensome 24 hour quantification. Papanna et al (2008) showed that random urine protein: creatinine ratio between0.13 to 0.15 indicate low chance of proteinuria exceeding 300mg per day

Fundus examination:

Grade 3 Grade 4 changes are indication of termination of pregnancy irrespective of gestational age.

MANAGEMENT

General management

Control of convulsion

Control of hypertension

Obstetric management

Early detection and management of complications

GENERAL MANAGEMENT

The first step is call for help.Eclamptic convulsions may be violent ,so eclamptic patients never be left alone. ABC resuscitation principle should be followed. Initial step aimed at preventing maternal injury and to maintain cardiorespiratory function.Eclamptic women has to be kept in quiet room The bedside rails are elevated . Patient must be in left lateral position- 15 º tilt to prevent aspiration and oral suctioning done if required Airway to be kept to prevent tongue fall. Nasal oxygen 10-12 litres provided through face mask.,ventilator must be kept standby .Vitals monitoring via multipara monitor. Continuous bladder drainage-hourly urine output .I/o chart has to be maintained. Appropriate antibiotics has to be started to prevent infection

CONTROL OF CONVULSIONS:

MgSo4 is the drug of choice .Its mechanism of action is neuroprotective and anticonvulsant action on cerebral cortex

MAGPIE TRIAL 2002

Magnesium Sulphate for Prevention of Eclampsia. It was a large international study on randomised comparison of MgSo4 with placebo for preeclamptic women . From 33 countries 10,000 women with preeclampsia over 2 years from 1998 to 2001 were given MgSo4 regime . The study results were significantly fewer eclamptic fits seen in control group that is 58% risk reduction of eclampsia and also lower risk of placental abruption.

Magpie trail recruited women followed by Magpie trail followup collaborative study group in the year 2007 which concluded that the risk reduction of eclampsia followed by magnesium sulphate prophylaxsis was not associated with an additional death or morbidity for the women after two years.

MgSO4.7H2O REGIME:

Mechanism of Action

Anticonvulsant action on cerebral cortex

Reduced presynaptic release of the neurotransmitter glutamate

Blockade of NMDA receptors

Potentiation of adenosine action

Blocks entry of calcium into synaptic terminals [20]

ECLAMPSIA TRAY:

It has to contain 20 ampoules of MgSo4,20ml syringe-1,10ml syringe-2,Airway,2 amps of Calcium gluconate ,Normal saline/ distilled H20,Venflon-green (18 G) / grey (16G),Knee hammer,Adult AMBU bag,Foley's catheter,BP apparatus,Suction catheter

PRITCHARD' S REGIME

LOADING DOSE :

4 gm of 20% $MgSo_4$ slow IV over 10-15 mins

5 gm of 50% $MgSo_4$ IM(20G needle) in both buttocks

MAINTENANCE DOSE

5gm of 50% $MgSo_4$ IM in alternate buttock every 4hr

IF SEIZURE REOCCURS

2gm of 20% $MgSo_4$ slow IV

TO MONITOR

Urine output – >25ml/hr

Patellar reflex - present

Respiratory rate – >16/min

MgSo4 to be continued for 24hours following delivery or last seizure whichever is later.

ZUSPAN REGIME

LOADING DOSE **:**

4gm of 20% $MgSo_4$ slow IV over 15 minutes

MAINTENANCE DOSE**:**

1gm/hr infusion

RECURRENCE OF SEIZURE**:**

Increase the infusion rate to 1.5-2gm/hr

ANTIHYPERTENSIVE THERAPY:

The aim of antihypertensive therapy is to maintain systolic blood pressure between 140-160mmHg, diastolic blood pressure between 90- 105 mmHg.

IV LABETALOL DOSE

INDICATION

- If BP>160/110 mm of Hg and not controlled with oral treatment with labetalol and Nifedipine.

  Dose 20 mg/hr (1 amp-20 ml=100 mg=5 mg/ml)

  1. **IV BOLUS METHOD:**
     - 10 mg IV over 2 minutes initially;
     - Double every 10 minutes till BP<=150/100 mm Hg
     - Maximum 220 mg/treatment cycle
     - Monitor BP and FHS every 5 minutes
  2. **SYRINGE DRIVER:**
     - Take 2 ampoule (40 ml) in 50 ml syringe
     - Start at 4 ml/hr(20 mg/hr); 0.2-0.4 ml/min
     - Monitor BP and FHS every 5 min
     - Double the dose every 20 minutes a maximum of 220 mg/treatment cycle
     - Stop it if BP<150/100 mm Hg.

3. **INFUSION PUMP:**

- Take 400 ml of ns+100 ml of Labetalol (5ampoule)=1 mg/ml
- Start at 20 mg/hr=20 ml/hr=5 drops/Min
- Monitor BP and FHS every 5 minutes
- Double the dose every 20 minutes to a maximum of 220 mg/treatment cycle
- Stop if BP <150/100 mm Hg.

SIDE EFFCETS

- Hypotension
- Flushing

CONTRA INDICATION

- Bronchial Asthma
- Congestive Cardiac Failure

COMMON ORAL ANTIHYPERTENSIVE AGENTS IN PREGNANCY

S. NO	DRUG	ONSET OF ACTION	MECHANISM OF ACTION	DOSAGE	COMMENTS
1	LABETOLOL	2 to 5 mins (IV) 30mins – 2 hours (oral)	Non selective β blocker , Selective ά1 blocker	200-2400 mg/day orally in two or three divided doses 10-20 mg IV upto 220 mg/ 1-2 mg/min IV	Orthostatic hypotension, Bronchoconstriction. Avoid in patients with asthma and CHF
2	NIFEDIPINE	30 mins (oral)	Calcium channel blocker	10-20 mg ORALLY QID 30-120 mg / day orally as slow release preparation	Do Not Use Sublingual Form, Reflex Tachycardia ,Headache
3	HYDRALAZINE	5 – 20 mins	Peripheral vasodilator	5mg IV followed by 5-10mg IV at 20-40mins interval	High dose or frequent dose associated with maternal hypotension, headache, fetal distress
4	METHYLDOPA	6 to 8 hours	Centrally acting ά2 agonist	0.5 -3 gms /day orally in two to three divided doses	May not be as effective in control of hypertension

FETAL RESPONSE TO MATERNAL SEIZURE:

Maternal hypoxemia, lactic acidosis, hypercarbia together will cause fetal bradycardia, loss of fetal heart rate variability, late decelerations.In most of the time these situation brief, fetal heart rate returns to normal soon after seizure.Sometimes persistent uterine contraction will cause placental abruption and abnormal fetal heart rate pattern continue until fetus die.

FETAL MONITORING

Non stress test,Obstetric USG, Biophysical profile, Fetal umbilical artery Doppler

OBSTETRIC MANAGEMENT

Delivery is the only conclusive treatment for eclampsia, after the patient is stablisied with senior obstetrician

If GA<34 weeks of gestation corsticosteroids has to be administered to improve fetal outcome.

Mode of delivery –

Considering the presentation of the fetus, fetal condition and likelihood of success of induction of labour after assessment of cervix mode of delivery determined. There were no randomized trials for comparing the perfect method for delivery in women with eclampsia. Vaginal delivery should be attempted for all women provided that there is no other indication for caesarean section.

Eclampsia per se is not an indication for LSCS that has to be reserved for only obstetric indications [21]

INTRAPARTUM MANAGEMENT

1. MgSO4 has to be continued in labour .
2. If cervix is favourable , oxytocin augmentation has be initiated.
3. If it is unfavourable, caesarean section has to be considered because of more incidence of abruption, fetal distress and other complications.
4. ANALGESIA

   Provided by use of 25-50 mg of pethidine (parentral) or segmental epidural analgesia.

   Local infitration in vaginal delivery.

   Nonstop epidural or balanced GA indicated for caesarean
5. Input and output monitoring

   Hourly urine output chart has to be maintained

   Restricted fluid intake to 150 ml / hr.

   If oliguria (<100ml / 4 hrs), fluids & MgSO4 has to be adjusted accordingly.
6. Antihypertensive therapy

POSTPARTUM MANAGEMENT

Intensive monitoring done for 2-4 days. Vitals, input output monitoring and 10 units syntocin IM preferred in 3rd stage of labour.Ergometrine is

contraindicated . Antihypertensive medication can be continued upto 3 months postpartum due to risk of late seizures

Women with persistent hypertension and proteinuria at 6 weeks postpartum must be considered for further investigations. Ensure that women will have a careful review after discharge from the hospital

MATERNAL OUTCOME

1) **CEREBRAL HAEMORRHAGE (7%)**

Leading cause of death, severe occipital headaches and convulsions followed by coma may occur. It is a indication for aggressive treatment with antihypertensive agent

2. POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

3. HYPOXIC ISCHEMIC ENCEPHALOPATHY

4. ABRUPTIO PLACENTA

Abruption is one of the most leading cause of antepartum hemorrhage in patients with eclampsia. Premature separation of normally situated placenta prior to the delivery of the fetus occurs mostly in pre eclamptic and eclamptic patients which is indicated by antepartum hemorrhage typically associated with uterine tenderness. Abruption may be concealed or revealed, externally concealed hemorrhage frequently diagnosed after delivery as retroplacental clot.

The degrees of placental separation may be

1. Mild: Maternal vitals stable,concealed hemorrhage with retrospective diagnosis
2. Moderate:Maternal tachycardia, postural hypotension fetal distress present
3. Severe: Patient may be in shock, dead fetus and features of coagulopathy present. Management mainly aims at preventing, reducing complications maternal : DIC, hypovolemic shock, AKI fetal: low birth weight, preterm labor, fetal hypoxia

   Labour natural is preferably carried out in tertiary care centre after securing 2 large bore IV cannula, bed side clotting time assessment, drawing blood , reserving adequate blood . Experienced neonatologist may be needed to resuscitate the baby. Strict monitoring of maternal vitals with special importance to pulse, urine output and BP. Caesarean section may be taken up in conditions like fetal distress, failure of progress of labor. Couvelaire uterus is bleeding into the myometrial layers of the uterus which leads to release of thromboplastins causing DIC. Uterine laxity leading to atony of uterus causing further bleeding.

5. HELLP SYNDROME (10- 15%)

- MMR 2-24% , PMR 9-39%
- Hemolysis (H)- passage of RBCs through the partially obliterated blood vessels (microangiopathic hemolysis) – Schizocytosis, Spherocytosis, Reticulocytosis.

- Elevated liver enzymes (EL)- excessive Fibrin-fibrinogen deposition in the hepatic sinusoids Parenchymal necrosis of the liver, subcapsular hematoma formation
- Low platelet count (LP)- aggregation and deposition of platelets at the site of endothelial damage

CLASSIFICATION OF HELLP SYNDROME

MISSISSIPPI classification

Class 1:

Platelets $\leq$ 50,000

AST or ALT > 70IU/L

LDH>600IU/L

Class2:

Platelets 50,000 – 100000

AST or ALT >70IU/L

LDH>600IU/L

Class 3:

Platelets 1,00,000 to 1,50,000

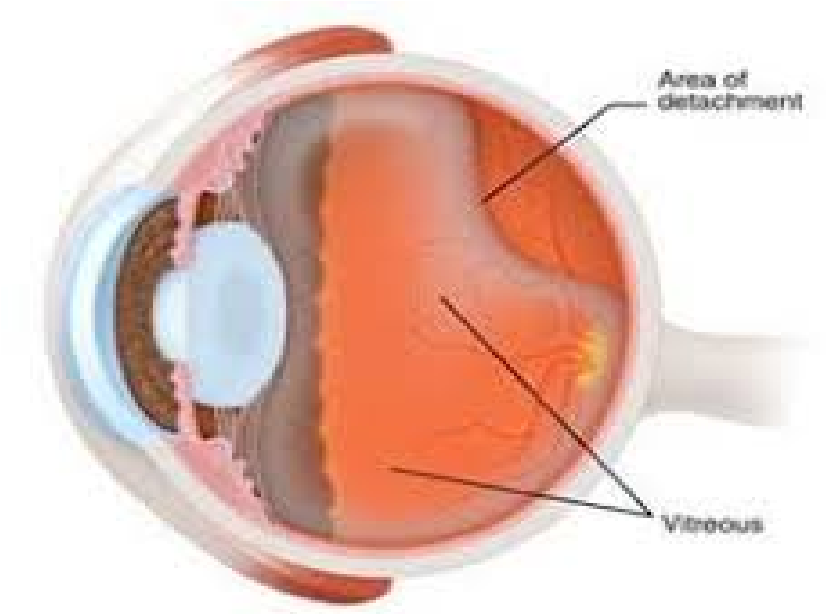
AST or ALT >40IU/L

LDH>600IU/L

Management is termination of pregnancy,

6. HYPERTENSIVE RETINOPATHY, RETINAL DETACHMENT

Serous retinal detachment is a very uncommon cause for blindness in eclampsia, produced by alteration in choroidal vascularization. The management is conservative and the prognosis is good.



RETINAL DETACHMENT

7. PULMONARY EDEMA (5%)

It is a common complication of eclampsia and severe preeclampsia The clinical signs and symptoms include bilateral basal crepts, respiratory distress, hypoxia. The fluid overload in postpartum period which leads to edema expansion and left ventricular failure resulting in pulmonary edema in eclamptic patients. Typically the patients presented with no prior history of heart disease, normal ECG, no cardiomyopathy on echo or chest Xray

Treatment:

Propped up position, back rest, nasal oxygen administration, fluid restriction.

Diuretics: frusemide 40mg iv 6hourly

Continuous positive airway pressure may be used in severe condition of pulmonary edema associated withrespiratory distress to shift the fluid to interstitial compartment and thus into capillaries. Frusemide acts by profuse diuresis and thus reducing the intravascular fluid volume. Central venous pressure monitoring may be necessary in patients with associated co morbid illness

8. ASPIRATION PNEUMONITIS

- Follows inhalation of gastric contents, prompt diagnosis and management by suction.

9. ACUTE RENAL FAILURE(4%)

- Usually due to volume depletion (pre renal), and do not respond to fluid challenge, in rare cases it may be due to Acute Tubular Necrosis.

- May require dialysis, but recovery is the rule.

10 DIC

11.HEPATIC FAILURE AND HEPATIC RUPTURE

12.CARDIAC FAILURE

13.MATERNAL MORTALITY (8-36%)

LONG TERM SEQUELAE

Development of chronic hypertension (6.4 fold increase)

IHD (24%)

CVA (9.5%)

Type 2 Diabetes (3.5 fold increase)

Thromboembolism

Renal sequelae (4 fold increase)

Impaired memory

NEED FOR THE STUDY

Eclampsia is a very serious complication of pregnancy which is responsible for high maternal and perinatal mortality. Eclampsia is an acute life-threatening complication of pregnancy characterized by the appearance of tonic clonic seizures (convulsions), usually in a woman who has developed pre-eclampsia. The incidence of eclampsia in India has been quoted as 1.56%.

Though not all cases of eclampsia can be prevented , majority of cases can be prevented by early detection and effective treatment of preeclampsia, for which good ANC services are needed. Once eclampsia started the prognosis of mother and baby becomes gloomy. Various complicating causes are responsible for maternal death in eclampsia

Computed tomography (CT) and MRI of brain have revolutionized visualization of lesions in eclampsia and other organic conditions. CT is a rapid initial imaging tool preferred to MRI in some conditions, like haemorrhage and space occupying lesions and complementary to MRI in others. Hence the need to study correlation and emphasis on the need for neuroimaging in accurate diagnosis and proper management of eclampsia

MATERIALS AND METHODS

5.1 **Source of data:** Patient admitted in Government Mohan Kumaramangalam Medical college hospital , Salem with antepartum and post partum eclampsia between January 2017- December 2017

5.2 **Study Design:** Prospective study

5.3 **Sample Size :**100 women with antepartum or postpartum eclampsia

5.4 **Place Of Study :**GMKMCH, Salem

5.5 **Period Of Study:** January 2017-December 2017

5.6 **Consent:** Informed written consent from patient or her attenders.

5.7 **Methods of collection of data**

1. History taking , clinical and neurological examination.
2. Blood pressure on admission
3. Haemoglobin,PCV, platelet count
4. Renal Function Tests
5. Liver Function Tests
6. Coagulation profile
7. urine analysis
8 Fundoscopy
9. CT BRAIN
10. MRI – Brain MRA and MRV

All patients of eclampsia admitted at Government Mohan Kumaramangalam Medical College, Salem who fulfilled the inclusion and exclusion criteria were first stabilized with magnesium sulfate as anticonvulsant and antihypertensives according to blood pressure. Detailed history was elicited. Maternal demographic, clinical, laboratory and neuroimaging data, maternal outcome and associated morbidities collected and analyzed. The categorical data expressed in terms of rates, ratios and proportions and continuous data expressed as mean ± 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.

A probability value (p value) of < 0.05% level of significance considered as statistically significant. IBM SPSS version 23 was used for statistical analysis.

INCLUSION CRITERIA

Patients with Eclampsia (atleast one episode of seizure in women with more than 20 weeks gestation or less than 6 weeks postpartum with blood pressure more than 140 mm of Hg systolic and 90 mm of Hg diastolic or increase in diastolic pressure by 15 to 25 mm of Hg compared to prepregnant state with urine albumin of more than 0.3gm/L) both antepartum and postpartum

EXCLUSION CRITERIA

1) Women who are known case of hypertension and epilepsy
2) Seizures due to metabolic disturbances, space occupying lesions or intracerebral infections

RESULTS

A total of 100 women with eclampsia who satisfied selection criteria were studied. The data was analyzed and various observations were given below.

6.1 Age distribution

Study sample consists of 100 women with eclampsia whose average age was 23.95 years while mean age of patients from age group above 21 years and below 21 years was found to be 26.03 years and 19.52 years respectively. 59% of patients fall under 25 to 29 years age group. Out of the sample, 10 % found to be less than 19 years while 11 % found to be above 30 years age group.

Chi square value -3.109

Degree of freedom- 3

P value is 0.375 which is statistically not significant.

Table :1 Age distribution

Age Distribution	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
< = 19 Years	6	14.29	4	6.90	10	10
20 - 24 Years	21	50.00	38	65.52	59	59
25 - 29 Years	9	21.43	11	18.97	20	20
> = 30 Years	6	14.29	5	8.62	11	11
Total	**42**	**100**	**58**	**100**	**100**	**100**

6.2 Onset of eclampsia -distribution

42 cases were Antepartum eclampsia while 58 cases were Postpartum eclampsia. Out of 42 antepartum eclampsia cases, 50% (21) had age group between 22 to 24 years and 21.43 % (9) fall under age group of 25 to 29 years. Similarly, under 58 postpartum eclampsia cases, 65.52% (38) had age group between 20 to 24 years and 18.97% (11) fall under age group of 25 to 29 years.

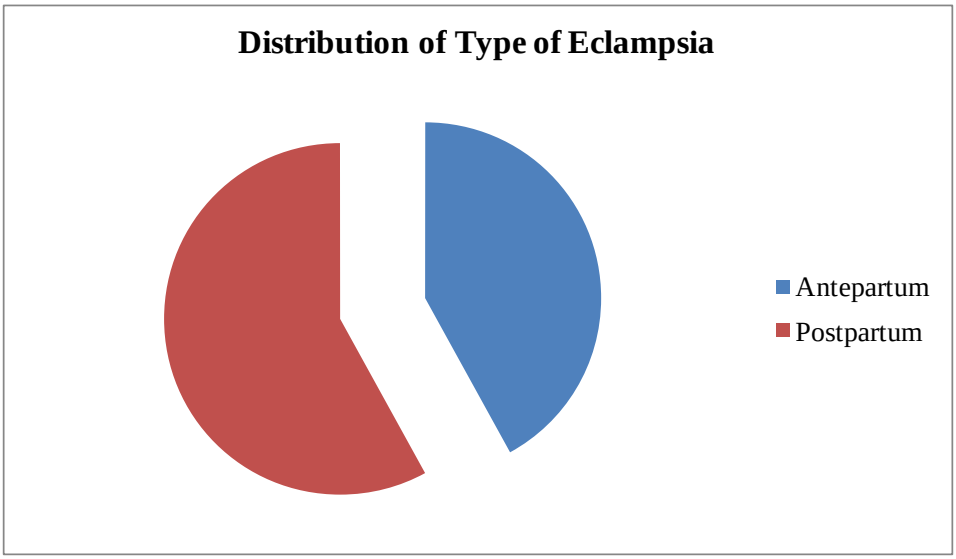


Fig.1. Distribution of type of Eclampsia

6.3 Gestational Age at the Onset of Antepartum Eclampsia

Onset of antepatrum eclampsia was observed at an average gestational age of 35.07 weeks. 38.10% (16) had gestational age greater than 37 weeks while, 4.76% (2) had gestational age less than 28 weeks. Percent of cases with average gestational age of 28-34 weeks and 34 to 37 weeks are found to be 26.19% (11) and 30.95%(13) respectively.

Table 2 Gestational Age at the Onset of Antepartum Eclampsia

Gestational Age at the Onset of Antepartum Eclampsia	Antepartum (n=42)	
	No.	**%**
< 28 Weeks	2	4.76
28 - 34 Weeks	11	26.19
34 - 37 Weeks	13	30.95
> 37 Weeks	16	38.10
Total	**42**	**100**

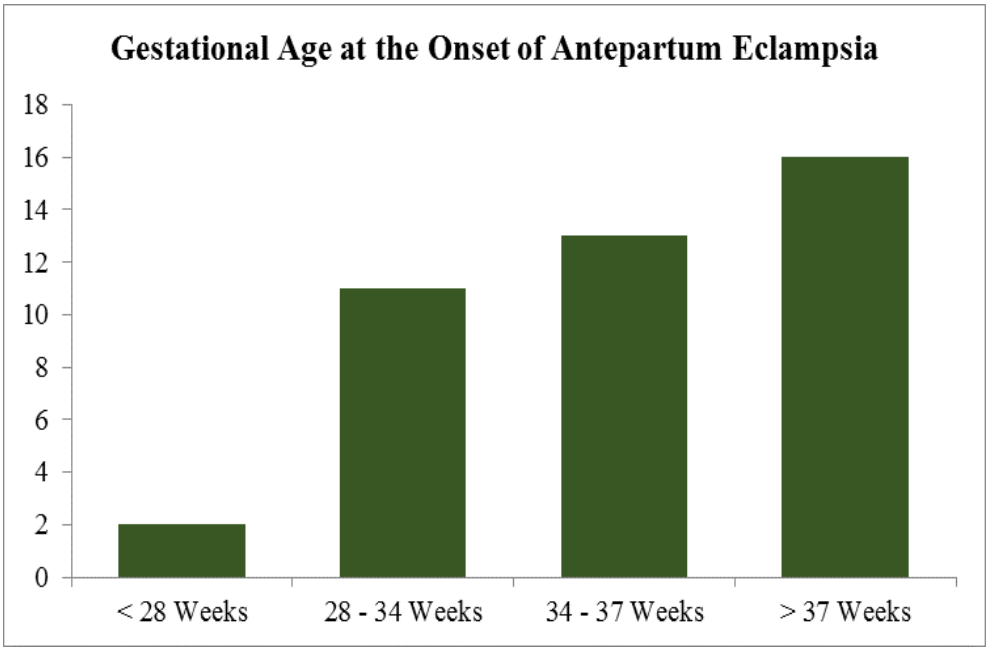


Fig.2. Gestational Age at the Onset of Antepartum Eclampsia

Out of 42 Antepartum cases, 69.05 percent (29) were Primi Gravida while 30.95 percent (13) were Multi Gravida.

Table 3 Distribution according to gravida in antepartum eclampsia

Gravida	Antepartum (n=42)	
	No.	**%**
Primi Gravida	29	69.05
Multi Gravida	13	30.95
Total	**42**	**100**

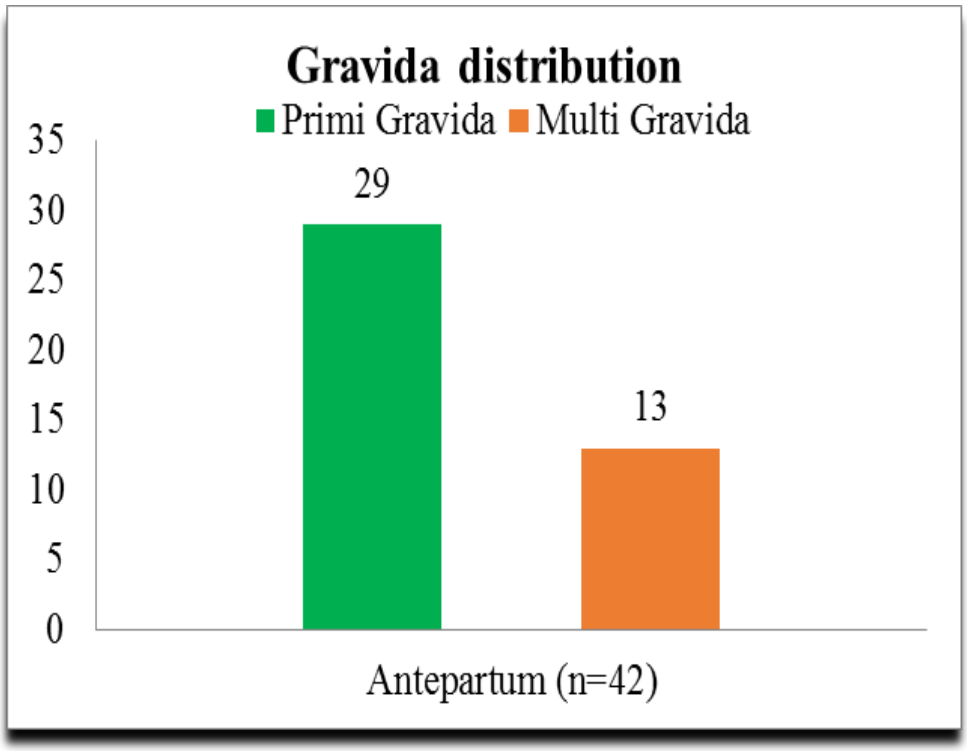


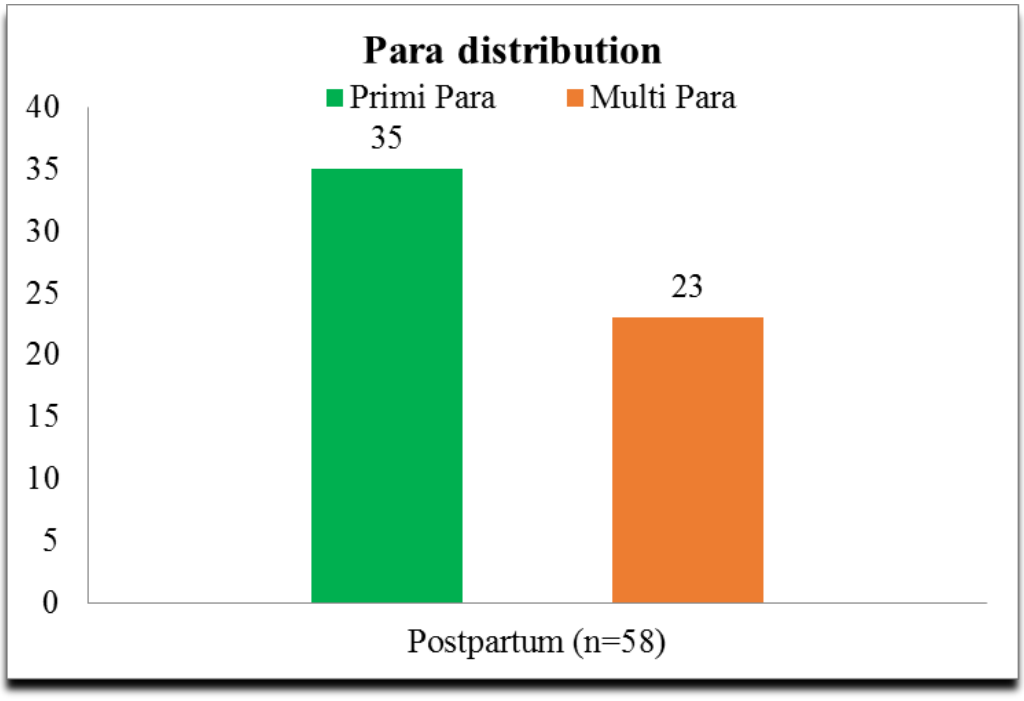
Fig.3. Gravida distribution

6.4 Distribution according to parity in post partum eclampsia

While 58 report cases of Postpartum, 60.34% (35) were Primi Para while 39.66% (23) were Multi Para.

Table. 4 Parity in postpartum eclampsia- distribution

Parity	Postpartum (n=58)	
	No.	%
Primi Para	35	60.34
Multi Para	23	39.66
Total	**58**	**100**



Fig, 4. Parity in postpartum eclampsia- distribution

6.5 Proteinuria

The range of Proteinuria was assessed using urine albumin dipstick test. Proteinuria upto 100 mg/dl (2^+) was found in 35% of study group followed by 30mg/dl (1^+) was found in 23%, 300 mg/dl (3^+) found in 12%, and greater than 1000 mg/dl (4^+) found in 6% respectively. 24% of women had nil urine albumin. Regarding antepartum eclampsia 1^+ was observed in 14 cases (33.33%) followed by 2^+as 13 cases (30.95%), 3^+ and 4^+ were 2 cases (4.76%) each. In postpartum eclampsia highest number of observations (22 women) was found in 2^+ category(37.93%) followed 3^+ with 10 cases (17.24%), 1^+ with 9 cases (15.52%) respectively. Urine albumin was found to be negative in 11 cases of antepartum eclampsia i.e. 26.19 % and 13 cases of postpartum eclampsia .Chi square P value 0.126 statistically not significant.

Table 5 Range of Proteinuria

Urine Albumin	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
1 +	14	33.33	9	15.52	23	23
2 +	13	30.95	22	37.93	35	35
3 +	2	4.76	10	17.24	12	12
4 +	2	4.76	4	6.90	6	6
Nil	11	26.19	13	22.41	24	24
Total	**42**	**100**	**58**	**100**	**100**	**100**

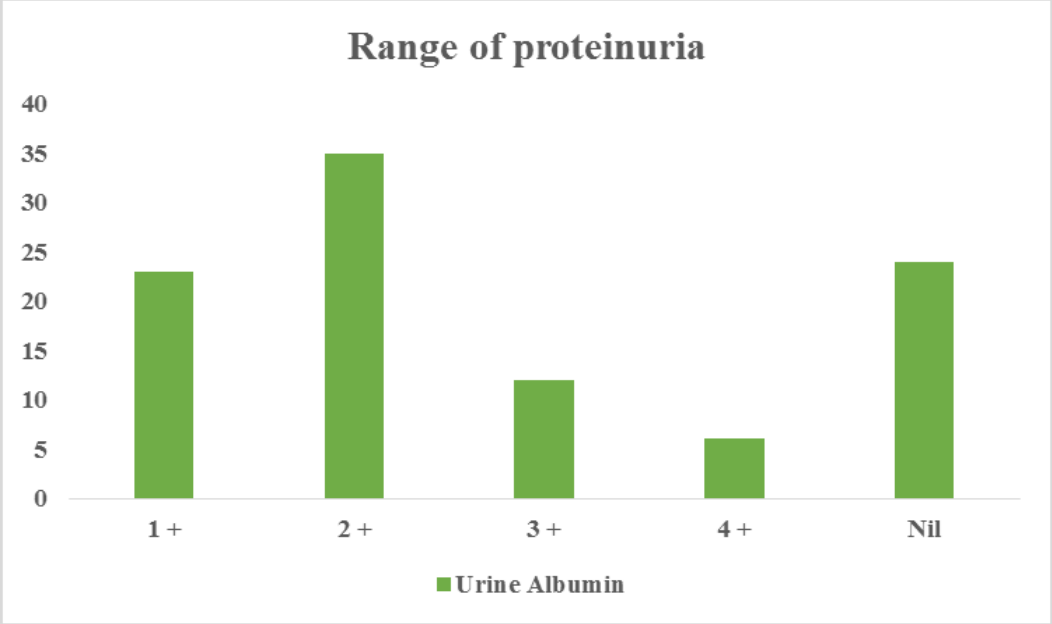


Fig.5. Range of Proteinuria

6.6 Fundus examination findings

Majority of the patients had normal ophthalmic examination findings. Cortical blindness was found in 2 women whereas grade I changes found in 18% of women followed by grade II (2 %), grade III and IV had 1% each. The grade IV changes i.e.,papilloedema found in antepartum eclampsia woman for which termination of pregnancy done by emergency LSCS .

Chi square P value0.269 statistically not significant

Table 6 Ophthal findings

Ophthal findings	Ante partum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
Grade I	8	19.05	10	17.24	18	18
Grade II	0	0.00	2	3.45	2	2
Grade III	0	0.00	1	1.72	1	1
Grade IV	1	2.38	0	0.00	1	1
Cortical Blindness	2	4.76	0	0.00	2	2
Normal	31	73.81	45	77.59	76	76
Total	**42**	**100**	**58**	**100**	**100**	**100**

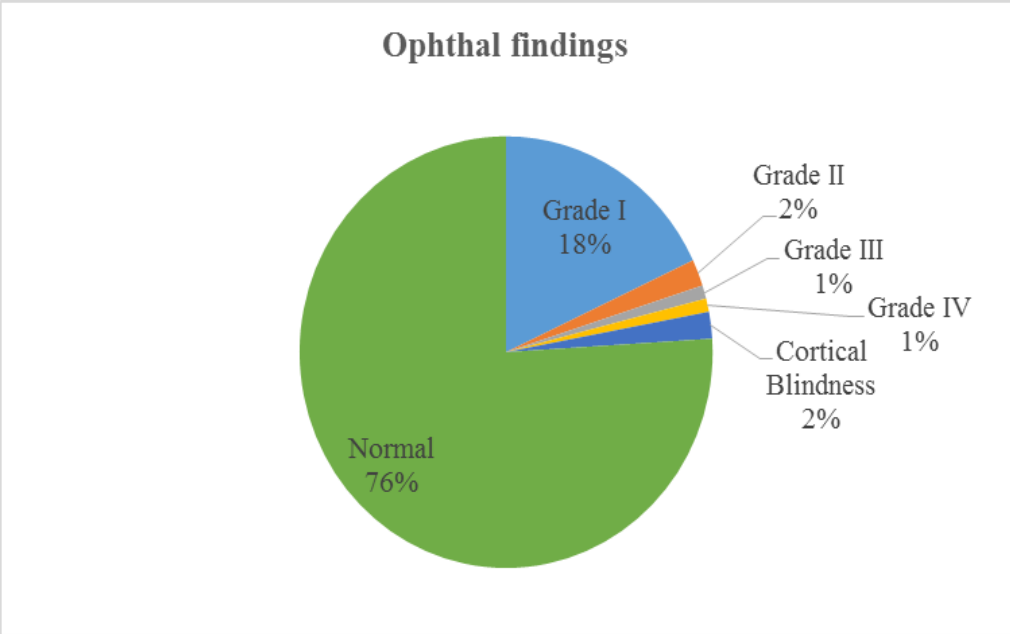


Fig.6. Ophthal findings

6.7 Clinical presentation

Commonest clinical presentation was found to be Unconsciousness accounting to 65 percent of which 40 % (26) were reported under Antepartum eclampsia while 60% (39) was reported under postpartum eclampsia. Altered Sensorium falls next to unconsciousness with 19% contribution in clinical presentation.

Within Antepartum eclampsia cases, Unconsciousness, Altered Sensorium and Frothing were reported as 61.90% (26), 25.19% (11), 9.52 % (4) respectively. Single case of Incontinence was reported under Antepartum eclampsia. Similarly, within Postpartum eclampsia cases, Unconsciousness and Incontinence were reported as 67.24% (39) and 5.17% (3) respectively. Frothing and Altered Sensorium shared an equal contribution of 13.79% (8) each.

Chi square value – 2.922

Degree of freedom-3

P value 0.404 not significant

Table 7 Clinical presentation

Clinical Presentations	Ante partum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	**%**	**No.**	**%**	**No.**	**%**
Unconscious	26	61.90	39	67.24	65	65
Altered Sensorium	11	26.19	8	13.79	19	19
Frothing	4	9.52	8	13.79	12	12
Incontinence	1	2.38	3	5.17	4	4
Total	**42**	**100**	**58**	**100**	**100**	**100**

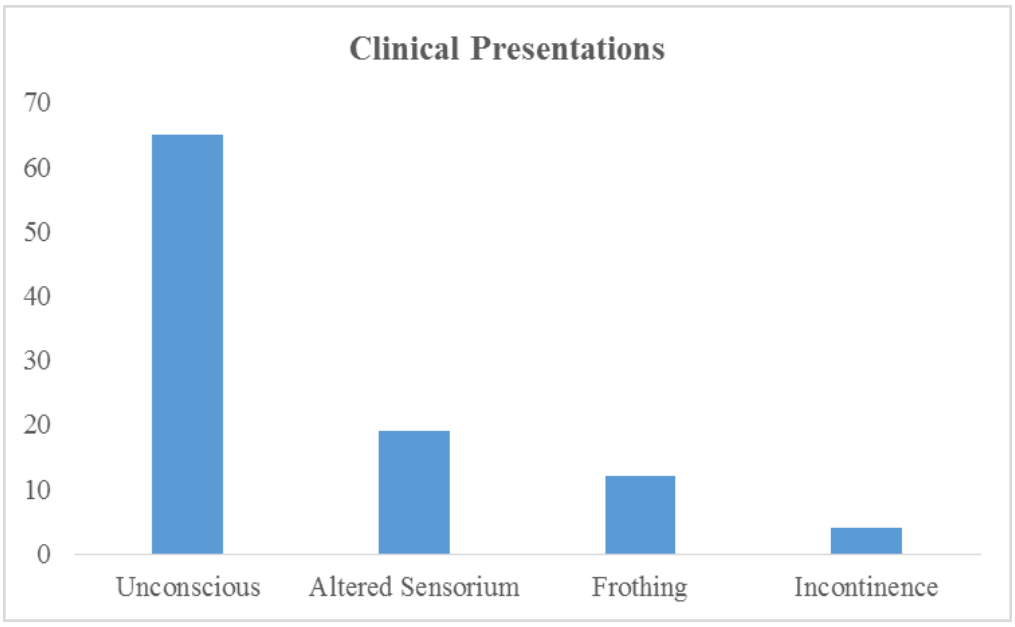


Fig. 7. Clinical presentation

6.8 Neurological Examination Findings- consciousness

In this study, 4.76% of antepartum eclampsia women were unconscious and 7.14% of women presented in drowsy state. In postpartum eclampsia, unconsciousness was found in 1.72% of women as well as drowsy state seen in 5.17% of women.

The difference was statistically not significant(P value 0.614)

Table 8 Neurological Examination Findings - Consciousness

Neurological Examination Findings – Consciousness	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
Unconsious	2	4.76	1	1.72	3	3
Drowsy	3	7.14	3	5.17	6	6
Conscious	37	88.10	54	93.10	91	91
Total	**42**	**100**	**58**	**100**	**100**	**100**

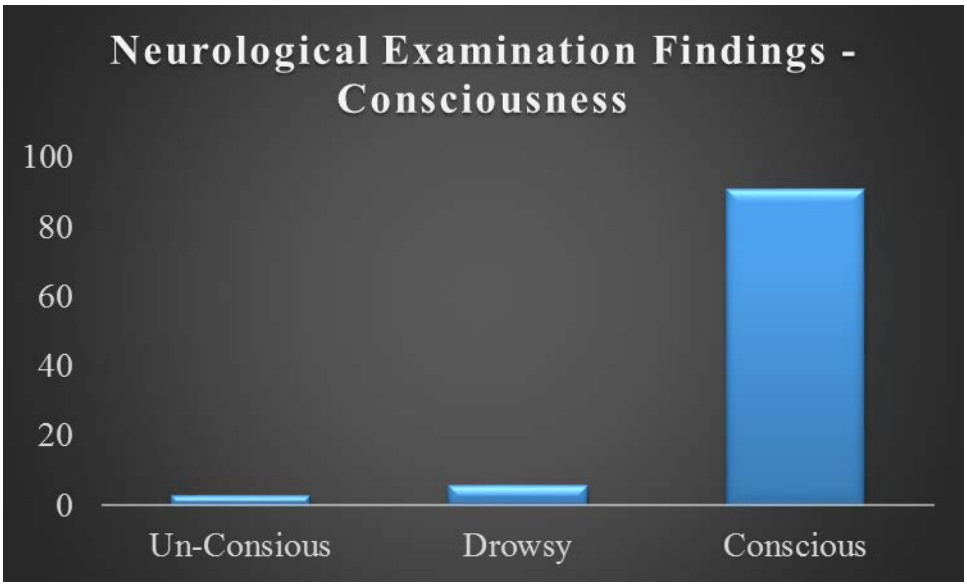


Fig.8. Neurological Examination Findings - Consciousness

6.9 Neurological Examination Findings – Orientation

At the time of neurological examination 91% of women were well oriented whereas 9% found to be disoriented in the present study. Also,11.90% women were under disoriented category of antepatrum eclampisa and 6.90% women of postpartum eclampsia were found to be disoriented and this difference ws statistically not significant(P value 0.486)

Table 9 Neurological Examination Findings – Orientation

Neurological Examination Findings – Orientation	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
Well Oriented	37	88.10	54	93.10	91	91
Disoriented	5	11.90	4	6.90	9	9
Total	**42**	**100**	**58**	**100**	**100**	**100**

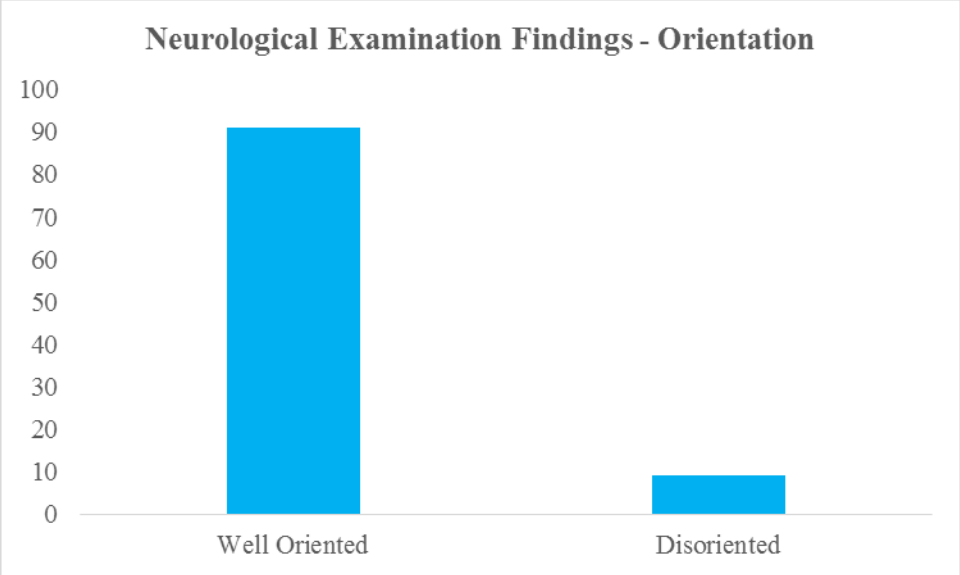


Fig.9. Neurological Examination Findings – Orientation

6.10 Symptoms

The commonest symptoms found in antepartum eclampsia was headache and it was observed in 35.71% (15.) whereas in postpartum eclampsia no imminent symptoms were found in 41.38% of women (24). The blurring of vision and vomiting were the other symptoms of eclampsia in which 19.05% (8) and 7.14% (3) of women in antepartum eclampsia had symptoms of blurring of vision and vomiting

In postpartum eclampsia 37.93% of women had headache followed by blurring of vision (6.9 %) and vomiting (12.075%). The reduced urine output was observed in 3 women as a symptom of imminent eclampsia. In that, 2 women were falls under the category of antepartum eclampsia and remaning one woman came under post partum eclampsia.

While considering the overall symptoms of eclampsia, 38% of women had no imminent symptoms. 37% of women had the symptoms of head ache followed by blurring vision (12%) and Vomiting (10%).

Table 10 Imminent symptoms

Symptoms	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
Headache	15	35.71	22	37.93	37	37
Blurring of Vision	8	19.05	4	6.90	12	12
Vomiting	3	7.14	7	12.07	10	10
No Imminent Symptoms	14	33.33	24	41.38	38	38
Reduced Urine Output	2	4.76	1	1.72	3	3
Total	**42**	**100**	**58**	**100**	**100**	**100**

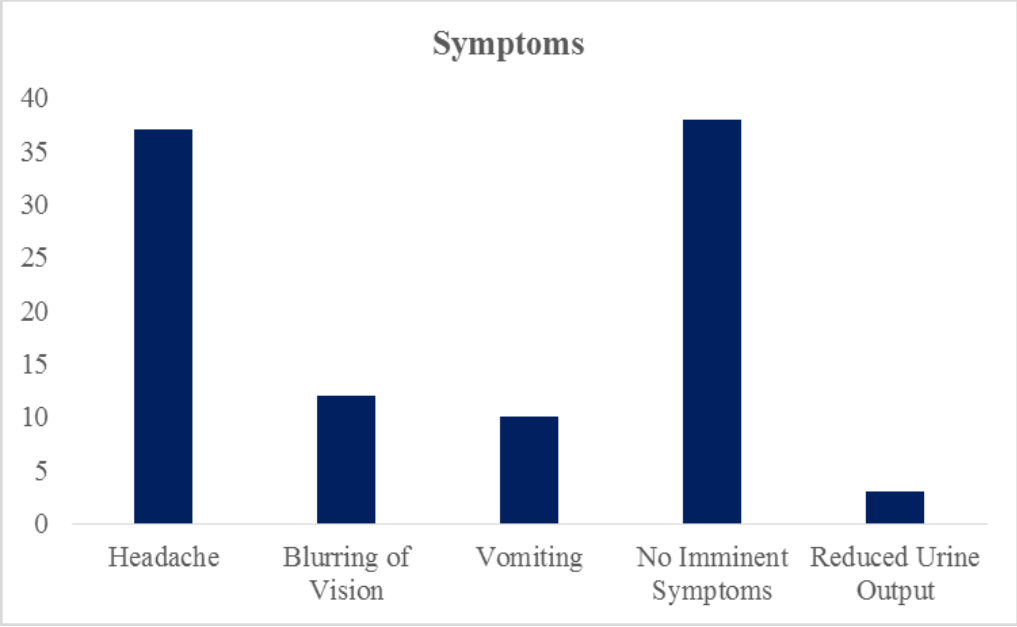


Fig.10. Imminent symptoms

6.11. Radiological findings

Radiological findings were found in 61 patients in which, 27 patients had Posterior Reversible Encephalopathy Syndrome followed by CVT with infarct (12 patients), Hypertensive Leucoencephalopathy (4 patients), Sub arachnoid hemorrhage and Cerebral atrophy (one patient each).

Chi square value- 14.395

Degree of freedom – 6

P value 0.026 which is statistically significant.

Table 11 Radiological findings

Findings	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
CVT with Infarct	3	7.14	13	22.41	16	16
Infarct	4	9.52	8	13.79	12	12
Posterior Reversible Encephalopathy Syndrome	16	38.10	11	18.97	27	27
Hypertensive Leucoencephalopathy	4	9.52	0	0.00	4	4
Sub arachnoid haemorrhage	0	0.00	1	1.72	1	1
Cerebral atrophy	0	0.00	1	1.72	1	1
No Abnormality	15	35.71	24	41.38	39	39
Total	**42**	**100**	**58**	**100**	**100**	**100**

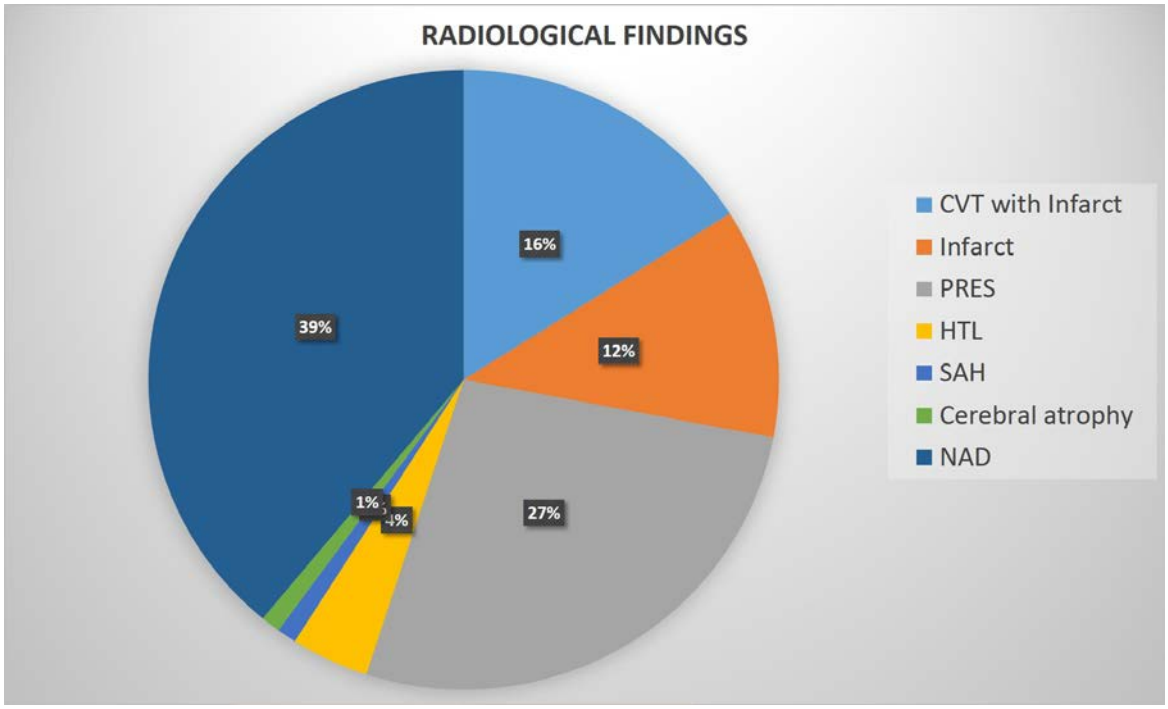


Fig .11. Radiological findings

Posterior Reversible Encephalopathy Syndrome (PRES) is a specific radiological finding which is found in 27 per cent of women (27 Nos.). Out of which, antepartum eclampsia women had higher number of findings (16) rather than post partum eclampsia (11). 48.15% of patients with PRES had headache, followed by blurring of vision (18.52%), vomiting and reduced urine output had 7.41% each respectively. While considering clinical presentation, 59.26% women presented with unconscious state followed by altered sensorium (25.93 %), frothing (11%) and incontinence (3.70%) respectively.

CVT with Infarct had 16%, out of which 3 cases of antepartum eclamspia (18.75%) and 13 cases of postpartum eclampsia (81.25%) were found to have CVT with infarct. While concern to imminent symptoms, headache present in 87.5% followed by vomiting and blurring of vision was 6.25% each was

presented. In clinical presentation, 62.5% were presented in unconscious state followed by 25% had altered sensorium and frothing had 12.5%.

Infarct was found in 12% of women out of which, 4 patients belongs to antepartum eclamspia (33.33%) and remaining 8 patients belongs to postpartum eclampsia (66.66%). The most common neurological imminent symptom was headache i.e. 58.33%. In clinical presentation, 83.3.% presented in unconsiuous state.

Table: 12 Radiological Findings Vs Imminent Symptoms

Radiological Findings vs Imminent Symptoms	CVT with Infarct	Infarct	PRES	HTL	SAH	Cerebral atrophy	NAD	Total
Headache	14	7	13	2	1			37
Blurring of Vision	1	1	5				5	12
Vomiting	1	1	2	2			4	10
No Imminent Symptoms		3	5			1	29	38
Reduced Urine Output			2				1	3
Total	16	12	27	4	1	1	39	100

Table 13 Radiological Findings Vs Clinical presentation

Radiological Findings Vs Clinical presentation	CVT with Infarct	Infarct	PRES	HTL	SAH	Cerebral atrophy	NAD	Total
Unconsious	10	10	16	1	1		27	65
Altered Sensorium	4	1	7	1		1	5	19
Frothing	2	1	3	1			5	12
Incontinence	0	0	1	1			2	4
Total	16	12	27	4	1	1	39	100

6.12 Accuracy of Neurological Signs and Symptoms in determining the radiological diagnosis

In the present study 61 cases had the radiological (CT/MRI) findings. Out of which 52 had neurological signs and symptoms and 9 cases did not have the neurological signs and symptoms.

The sensitivity, specificity positive predictive value and negative predictive value i.e., 85.25%, 74.36%, 83.8%, and 76.32% respectively. The diagnostic accuracy was found to be as 82.22%.

Chi square value -35.874

Degree of freedom -1

P value of 0.0005 which is stastistically significant,

Table 14 Neurological Signs and Symptoms

Neurological Signs and Symptoms	CT & MRI Findings		Total
	Positive	Negative	
Present	52	10	62
Absent	9	29	38
Total	61	39	100
P value= 0.0005			
Sensitivity	Specificity	PPV	NPV
85.25%	74.36%	83.87%	76.32%

6.13 Co Morbid Conditions

Anaemia was found to be major Co Morbid factor both in antepartum and postpartum eclampsia. Obesity and GDM contributes 6% in the present study. 3 % of women had previous history of eclampsia.

Table 15 Co Morbid Conditions

Co Morbid Conditions	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
Anaemia	11	26.19	21	36.21	32	32
GDM	3	7.14	3	5.17	6	6
Heart Disease	1	2.38	0	0.00	1	1
Obesity	4	9.52	2	3.45	6	6
Pre-History of Eclampsia	2	4.76	1	1.72	3	3
Sepsis	0	0.00	1	1.72	1	1
Nil	21	50.00	30	51.72	51	51
Total	**42**	**100**	**58**	**100**	**100**	**100**

6.14 Mode of delivery

Out of 100 eclamptic women, 56% patients delivered vaginally. Cesarean section were performed in 42% of women and one patients expelled spontaneously whereas one patient underwent hysterotomy.

Chi square value- 22.981 Degree of freedom -3

P value 0.0005 which is statistically significant

Table 16 Mode of delivery

Mode of Delivery	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
Vaginal	12	28.57	44	75.86	56	56
LSCS	28	66.67	14	24.14	42	42
Hysterotomy	1	2.38	0	0.00	1	1
Spontaneous Expulsion	1	2.38	0	0.00	1	1
Total	**42**	**100**	**58**	**100**	**100**	**100**

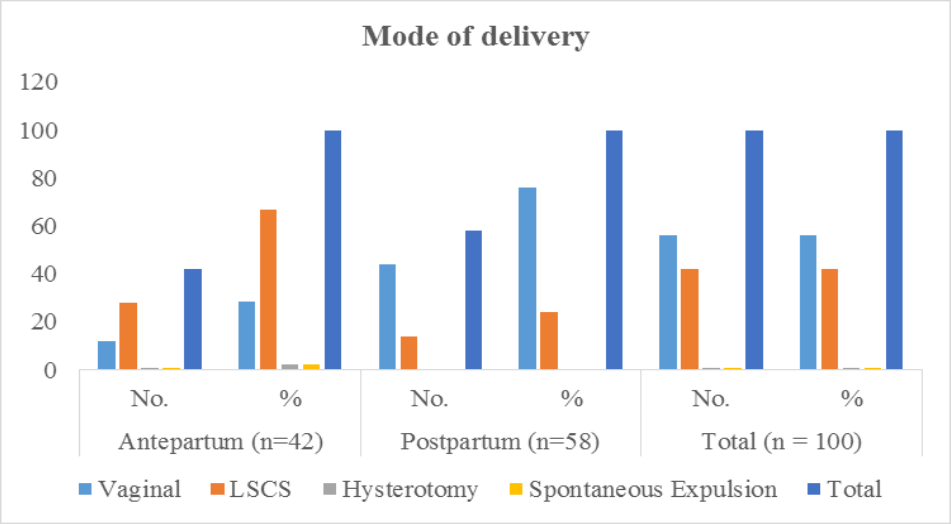


Fig. 12 Mode of delivery

6.15 Admission to delivery interval distribution

Most of the patient delivered in less than 12hours (31) and remaining patients delivered in the interval of 12-24 hours.

Table 17 Admission to delivery interval distribution

Admission-Delivery Interval	Antepartum (n=42)	
	No.	**%**
< 12 Hours	31	73.81
12-24 Hours	11	26.19
Total	**42**	**100**

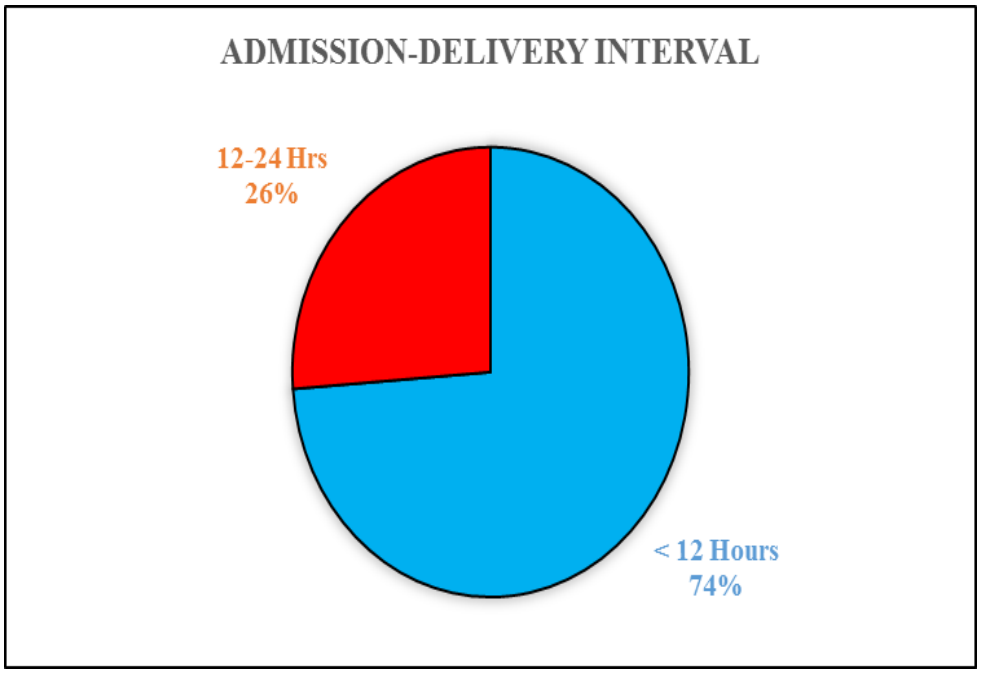


Fig. 13 Admission to delivery interval distribution

6.16 Maternal Outcome

48% women had nil complications. In present study 12% women suffered from DIC, HELLP was observed in 8% of women, 7% developed PPH, 6% had CVA with residual paralysis, 5% developed pulmonary edema, and 4% had acute kidney injury, 2% of placental abruption and blindness were reported.There were 5% maternal deaths in this study .

Chi square value-23

Degree of freedom -9

P value 0.005 statistically significant

Table 18 Maternal Outcome

Outcome	Antepartum (n=42)		Postpartum (n=58)		Total (n = 100)	
	No.	%	No.	%	No.	%
Placental Abruption	2	4.76	0	0.00	2	2
DIC	2	4.76	10	17.24	12	12
CVA	3	7.14	3	5.17	6	6
AKI	3	7.14	1	1.72	4	4
Blindness	2	4.76	0	0.00	2	2
HELLP	2	4.76	6	10.34	8	8
Pulmonary Edema	3	7.14	2	3.45	5	5
PPH	7	16.67	0	0.00	7	7
Death	1	2.38	4	6.90	5	5
Aspiration Pneumonitis	1	2.38	0	0.00	1	1
No Complications	16	38.10	32	55.17	48	48
Total	**42**	**100**	**58**	**100**	**100**	**100**

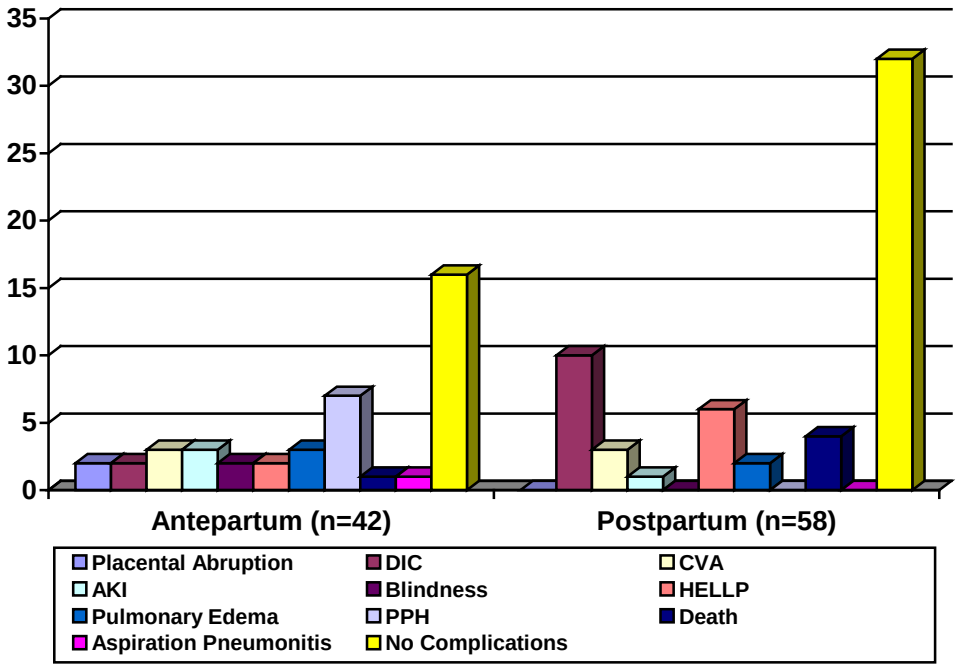


Fig.14. Maternal Outcome

6.17 Causes of death

Totally five patients were expired out of which 40% was due to Cerebral Venous Thrombosis (2.) The remaining 3 patients expired due to ARDS , Sub arachnoid haemorrhage, Acute Renal Failure / DIC.

Table 19 Cause of Death:

Causes of Death	No. of Cases	%
ARDS	1	20
Acute Renal Failure / DIC	1	20
Sub Arachnoid Hemorrhage	1	20
Cortical Venous Thrombosis	2	40
Total	**5**	**100**

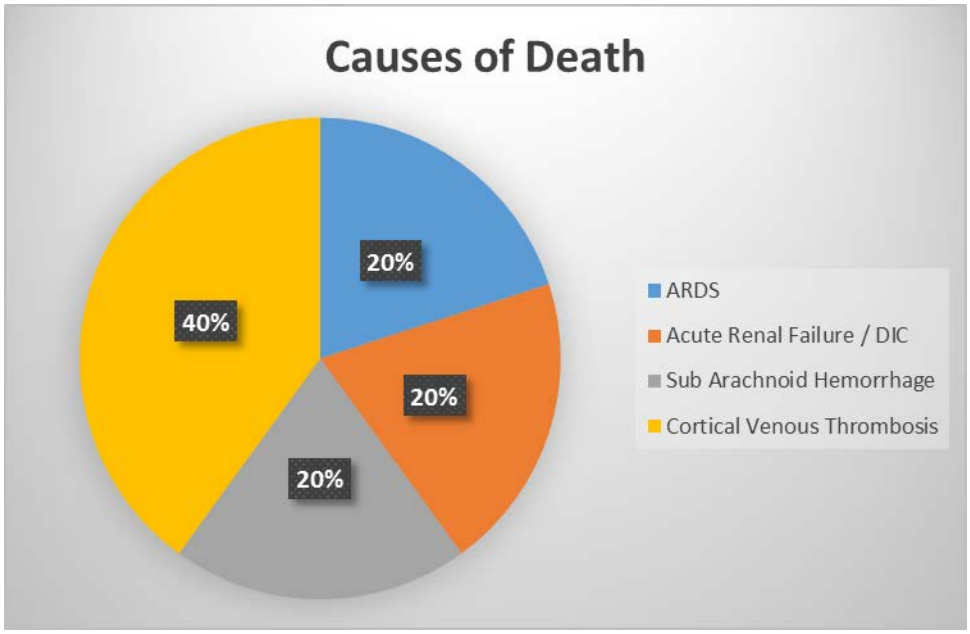


Fig 15. Causes of death

Table 20 Information of maternal deaths:

S.No	Onset of Eclampsia	Gestational Age (in weeks)	Mode of Delivery	Seizure to Delivery Interval	Delivery time and onset of eclampsia (in days)	Seizure to Death Interval (in days)
1	Postpartum		Vaginal		5	6
2	Antepartum	31	LSCS	< 12 Hours		11
3	Postpartum		Vaginal		1	4
4	Postpartum		LSCS		18	1
5	Postpartum		Vaginal		3	1

Total number of maternal deaths were 5 %, Most of deaths occurred in postpartum eclampsia women, most common cause was cortical vein thrombosis

DISCUSSION

Neurologic manifestation in eclampsia were headache, blurring of vision, confusion, visual hallucinations and blindness. The central nervous system changes characterise a form of hypertensive encephalopathy. Loss of cerebral autoregulation results in disruption of blood brain barrier with end result of cerebral edema.Such variations are responsible for many symptoms in eclamptic patients and evident on CT,MRI as witnessed in our study. Sudden cause for death in eclampsia due to massive cerebral haemorrhage or edema.

Mean age of study population was 23.95. 59% of women between the age group of 20-24 years. This result correlates with the Ugran SM et al[25] where mean age was 23.89 years, 50% aged between 22-25years. A similar study from Mishra R showed majority of women belonged to age group between 20-25 years which was comparable with this study .

	<19 years	20-24years	25-29years	>30 years
Bhanu BT et al[9]	40.8%	68%	20.8%	8.6%
Our study	10%	59%	20%	11%

Mean systolic blood pressure was 154.54, mean diastolic blood pressure was 94.86 Worldwide studies showing that postpartum eclampsia more common. In this study 58% women has postpartum eclampsia and 42% has antepartum eclampsia. Similar study from Krishna Dahiya et al showed 66% women presented with postpartum eclapmsia and 34% had antepartum eclampsia[22] .

GESTATIONAL AGE:

Most cases presented in third trimester 38.10% had gestational age more than 37 weeks.

	<28weeks	28-34weeks	34 -37 weeks	>37weeks
Bhanu BT et al[9]	-	26%	48%	26%
Our study	4.76%	26.19%	30.95%	38.10%

PARITY:

In this study 64 % are primiparous which is comparable with Ugran SM et al study.Two cases of twin gestation had eclampsia , one patient had normal radiological finding, another woman had subarachnoid haemorrhage.

	Primiparous	Multiparous
UgranSM et al[6]	59%	41%
Bhanu BT et al[9]	45%	55%
Jindal MA et al[3]	56%	44%
Our study	64%	36%

PROTEINURIA:

In our study proteinuria grade 2+ found in most of the women

	1+	2+	3+	4+	Nil
Jindal MA et al[3]	16%	40%	24%	20%	-
Our study	23%	35%	12%	6%	24%

FUNDOSCOPY EXAMINATION:

12% women presented with blurring of vision , most of them had only blurring of vision , no visual field defect.2 cases presented with cortical blindness.

	Normal	Grade 1	Grade 2	Grade3	Grade4	Cortical blindness
Jindal MAet al[3]	48%	16%	16%	8%	12%	-
Our study	76%	18%	2%	1%	1%	2%

Cortical blindness is loss of vision with intact pupillary reflexes. Fundus examination will be normal.Cortical blindnessis due damage to primary visual cortex.This may be permanent or temporary depending on the cause.

In this study one woman recovered well with in 24 hours of treatment,whose MRI showed PRES.Another woman evolved to permanent blindness with the CT findings of multiple infarct.

There is no correlation found between onset of eclampsia and severity of hypertension , fundoscopy findings and amount of proteinuria with Chi square p value is not statistically significant.

RADIOLOGICAL FINDINGS

In this study 61% has positive radiological findings, 39% women has normal findings.The cause for normal finding might be due to temporal relationship of scan to seizure.CT enable early noninvasive diagnosis of CVT. Plain CT or MRI will be helpful in confirmation of pituitary apoplexy.

The most common abnormal finding is PRES accounts for 27% followed by CVT with infarct- 16%, infarct -16%, Hypertensive leucoencephalopathy -4%, cerebral atrophy -1%, Sub arachnoid haemorrhage accounts for 1%.The commonest neurological presentation in PRES was unconsciousness (16%) followed by headache (13%) In PRES with hypertension the myogenic cerebral autoregulation effect decreased depending on elevated blood pressure.The neurogenic autoregulatory mechanism take over the regulation of cerebral perfusion, became more sensitive to elevation in blood pressure leads to vasogenic edema. In PRES without hypertension pathogenesis thought to be direct endothelial injury which might surges permeability of blood brain barrier. If clinical and radiological results in PRES lesion are easily identified and treated, they might be totally reversible.

Radilogical findings	Our study	Ugran et al[6]
CVT with infarct	16%	23%
Infarct	12%	14%
PRES	27%	6%
HTL	4%	5%
Sub arachnoid haemorrhage	1%	-
Cerebral atrophy	1%	1%
No abnormality	39%	48%

Kokila et al from Karnataka, India reported 46.4% of women had noneclamptic organic cause for postpartum convulsions , 28.6%of postpartum convulsions were due to CVT.

In this study 61% of women had abnormal radilological findings, among these considerably higher number 52 women had neurological signs and symptoms and 9 cases did not have neurological signs and symptoms.(p=0.0005). The sensitivity, specificity,positive predictive value ,Negative predictive value were found to be 85.25%, 74.36% , 83.87% , 76.32% respectively.These findings suggest that, signs and symptoms during admission helps to predict the neurological involvement and aiding to arrive the likely diagnosis.A prospective study from Jindal et al to compare CT and MRI findings in eclampsia patients in relation to neurological signs and symptoms. This study concluded that MRI found to co- relating more than CT with respect to neurological signs and symptoms and had 90% sensitivity and 100% specificity .MRI can be superior compared to CT in eclampsia patients[22.]

CO MORBID CONDITION

32% of women has anaemia as associated comorbidity, 6% were obese, 3% has history of eclampsia in the previous pregnancy

CLINICAL PRESENTATION

In this study commonest clinical presentation is unconsciousness 65% followed by 19% altered sensorium , 12% frothing,4% presented with incontinence. The unconscious state more common with postpartum eclampsia(67.24%)

	Unconsciousness	Altered sensorium	Frothing	Incontinence
Ugran SM et al[6]	35%	14%	11%	6%
Our study	65%	19%	12%	4%

IMMINENT SYMPTOMS:

In this present study commonest imminent symptom is headache.

A similar study from Mishra R reported as headache was most common symptom that is 76%, slurring of speech in 44% of women, 32% were disoriented. In this study headache is more commom in postpartum eclampsia that is 37.93%.

MODE OF DELIVERY :

Out of 100 women 56% delivered vaginally, 42% delivered by caesarean section. 2 cases presented less than 28 weeks out of which one women expelled foetus spontaneously, eclampsia itself induced labour pains. Another one underwent hysterotomy.

	Vaginal	Cesarean section	Hysterotomy	Spontaneous expulsion
Mahalakshmi et al[8]	52.9%	47.1%	-	-
Our study	56%	42%	1%	1%

ADMISSION TO DELIVERY INTERVAL

Most of the women (73.81%) delivered with in 12 hours of admission.

	< 12 hours	>12hours
Mahalakshmi et al[8]	64.7%	30.3%
Our study	73.81%	26.19%

DELIVERY TO SEIZURE INTERVAL IN POSTPARTUM ECLAMPSIA

The mean was 3.59 days in this study.Postpartum eclampsia occurs usually first 24 hours of delivery and hardly it may occur 48-72 hours after delivery

MATERNAL OUTCOME:

In this study 12% of women had DIC, 8% had HELLP syndrome

Complications	Our study	Bhanu BT et al[9]	Mahalakshmi et al[8]
Placental abruption	2%	15%	10.78%
DIC	12%	5.7%	2.94%
CVA	6%	1.7%	4.9%
AKI	4%	3%	1.96%
Blindness	2%	1.3%	-
HELLP	8%	12.1%	7.84%
Pulmonary edema	5%	1.7%	1.96%
PPH	7%	3.9%	1.96%
Death	5%	5.7%	6.9%

CAUSE OF MATERNAL DEATH

In this study, maternal death were 5%,most common cause was cortical vein thrombosis , occurred in cases of postpartum eclampsia.Ghimire S studied the fetomaternal outcome in eclampsia , maternal death rate was found to be 5.36%[23]

Study	Maternal death
Ghimire S	5.36%
Bhanu BT et al	5.7%
Our study	5%

SUMMARY

This prospective study was conducted in the department of obstetrics and gynaecology , Govt Mohan Kumaramangalam medical college Hospital,Salem from the period January 2017- December 2017. A total of 100 women presented with antepartum or post partum eclampsia were included .

1. Mean age of study population 23.95,were primiparous women
2. Mean gestational age 35.07 weeks.
3. Antepartum eclampsia 42%, Postpartum eclampsia 58%
4. All women had generalised tonoic clonic convulsions.
5. Mean systolic BP 154.54 mmHg, Diastolic BP 94.86 mmHg
6. Proteinuria 2+ found in most of the women
7. 76% had normal fundoscopy findings
8. Most of them had headache, presented in unconscious state.
9. Most common comorbidity was anaemia
10. Most of them delivered vaginally within 12 hours of admission
11. In postpartum eclampsia delivery to seizure interval is 3.59 days.
12. PRES was seen as a major radiological finding
13. Out of 61 patients with radiological abnormality 52 had neurological signs and symptoms.
14. 48% of women recovered without any complications, DIC seen in 12%
15. Maternal death was 5%, Common cause was cortical vein thrombosis.

CONCLUSION

Eclampsia is preventable, still remains a major cause for maternal morbidity and mortality. Maternal morbidity includes placental abruption with resulting DIC, pulmonary edema, acute renal failure, aspiration pneumonia, postpartum haemorrhage. Neuroimaging may be indicated in all eclampsia patients, specific attention has to be given to atypical cases with onset of eclampsia before 20 weeks of gestation or more than 48 hours after delivery and those resistant to anticonvulsant therapy, where neurological signs and symptoms fails to predict the diagnosis. More awareness should be created for the women while attending the antenatal clinics. Women from low socio economic status should be educated about nutrition, pregnancy, information about premonitory symptoms and the need for regular antenatal and postnatal check ups.

BIBLIOGRAPHY

1. Williams obstetrics 24 th edition ; 40:730

2. Kokila,M.S, Dwivedi AD. Correlation of clinical and neuroimaging findings affecting management in postpartum eclampsia: A prospective study.J.South Asian Federation Obstet Gynaecol.2011; 3(3):125-30

3. Jindal MA., Harsha S. Gaikwad, Bhagat D. Hasija, Kavita Vani Comparison of neuroimaging by CT and MRI and correlation with neurological presentation in eclampsia. Int J Reprod Contracept Obstet Gynecol. 2013 Mar;2(1):83-87

4. Khawla Abu Samra. 2013. The eye and visual system in the preeclampsia/ eclampsia syndrome: what to expect?. Saudi J Opthalmol.27(1):51-53

5. Pannu,D, Banashree Das, Paramita Hazari, Shila. Maternal and perintal outcome in eclampsia and factots affecting the outcome: a study in north Indian population. J. of reproduction, contraception, obstetrics and gynecology. 2014 Jun;3(2):347-351

6. Ugran SM Kasturi V.Donimath Correlation between neuroimaging(CTscan) and neurological presentation in antepartum and postpartum eclampsia Int J Reprod Contracept Obstet Gynecol. 2016 Feb;5(2):419-424

7. Sharma,G,Deepali Bhatt, Kamlesh Yadav, Sudesh agarwal, Swati Kochar,Asmita Nayak, Shahnaz Chandad. Eclampsia Preeclampsia; clinical and neurologicalcorrelates, J. of dental and medical sciences, 2016 May;15(5),87-91.

8. Mahalakshmi G., A.Krishnaveni, WinnieNimma, K.Vinusha. The study of maternal and perinatal outcomeof eclampsia in a tertiary hospital. J. of dental and medical sciences. 2016 April;15(4):123-128.

9. Bhanu,BT, Amudha S, Sarojini, Clinical study of maternal complications associated with eclampsia, J. of reproduction, contraception, obstetrics and gynecology, 2017 may;6(5):1905-1908

10. Staff AC , Sibai BM,Cunningham FG: Prevention of eclampsia and pre eclampsia.In Taylor RN, Roberts JM, Cunningham FG (eds): Chesley s Hypertensive Disorders in Pregnancy. Amsterdam Academic Press, 2014

11. Myatt L, Clifton RG, Roberts JM, et al: First trimester prediction of preeclampsia in nulliparous women at lowrisk. Obstet Gynecol119(6):2012a.

12. Arias high risk pregnancy and delivery 4 th edition 13(221)

13. Minassian C, Thomas SL, Williams DJ, et al.Acute maternal infection and risk of preeclampsia: a population based case-control study.PLoS One 2013;8:e73047.

14. Janzarik WG, Ehlers E, Ehmann R, et al: Dynamic cerebral autoregulation in pregnancy and the risk of preeclampsia. Hypertension 63:161,2014

15. Edlow JA, Caplan LR, O'Brien K, et al: Diagnosis of aute neurological emergencies in pregnant and postpartum women. Lancet Neurol 12(2): 175,2013

16. Brewer J, Owens MY , Wallace k, et al: Posterior reversible encephalopathy

syndrome in 46 of 47 patients with eclampsia.Am J Obstet Gynecol208(6):468.el,2013

17. Kenny L, McCrae K, Cunningham FG: Platelets, coagulation and the liver.In Taylor RN,Roberts JM, Cunningham FG (eds): Chesley's Hypertensive Disoredrs in Pregnancy, 4 th ed. Amsterdam, Academic press 2014

18. Royal College of sObstetrician and Gynecologist. The management of severe preeclampsia /eclampsia .Evidence based clinical guide line No 10(A). London: RCOG Press reviewed in 2010

19. Task Force : Hypertension in pregnancy.Report of the American College of Obstetric and Gynaecologist's Task Force on Hypertension in pregnancy. Obstet Gynecol 122:1122,2013

20. Wang LC, Huang CY,Want HK et al: Magnesium sulphate and nimesulide have synergistic effects on recuing brain damage after transient focal ischemia.J Neurotrauma 29(7):1518,2012a

21. Seal SL, Ghosh D, Kamiya G et al.Am J Obstet Gynecol. 2012;206:484.e1-7

22. Krishna Dahiya , Mahesh Rathod, Seema Rohilla, Pushpa Dahiya et al: Correlation between Neuroimaging and Clinical presentation in Eclampsia;International Journal of Obstetrics and Gynaecology Research, Vol .3(2016) No 6, pp.375-384

23. Ghimire S. Eclampsia : fetomaternal outcomes in a tertiary care centre in eastern Nepal.J Nepal Med Assoc.2016:54(201):24-8

PATIENT CONSENT FORM

Study Title: **Study on correlation between neuroimaging and neurological presentation in antepartum and postpartum eclampsia and its maternal outcomes**

Department of Obstetrics and Gynaecology, GMKMCH, Salem

PARTICIPANT NAME:

AGE:

I.P. NO:

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during and after the study. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study.

Date : Signature of the Patient

PROFORMA

NAME AGE IP NO DOA

OBSTETRIC CODE LMP EDD

Type of Eclampsia- antepartum/ postpartum

COMPLAINTS

Present H/o

Period of amenorhea

Headache

Oliguria

Pain abdomen

Vomiting

Blurring of vision

History of seizure

PAST H/O:

H/o eclampsia in previous pregnancy

Menstrual History:

Marital History:

Medical/ surgical H/O:

H/O Epilepsy,Head injury,Neurological disorder

Family History:

Personal History:

EXAMINATION

Consciousness

Orientation

Anaemia

Pedal edema

Icterus

BP

PR

Temperature

CVS

RS

P/A

P/V

CNS

INVESTIGATIONS

BLOOD- Hb, pcv, platelet

Blood urea, serum creatinine

Blood glucose

Serum electrolytes

LFT

Coagulation profile

URINE ANALYSIS

Albumin

Sugar

Deposits

Ophthal findings

NEUROIMAGING

ADMISSION TO DELIVERY INTERVAL

MODE OF DELIVERY

Vaginal

LSCS

MATERNAL OUTCOME

ABBREVIATIONS

AKI- ACUTE KIDNEY INJURY

ARDS- ACUTE RESPIRATORY DISTRESS SYNDROME

AST- ASPARTATE TRANSAMINASE

ALT-ALANINE TRANSAMINASE

ANC-ANTENATAL CARE

BMI- BODY MASS INDEX

CT – COMPUTED TOMOGRAPHY

CNS- CENTRAL NERVOUS SYSTEM

DIC- DISSEMINATED INTRAVASCULAR COAGULATION

GA- GESTATIONAL AGE

GDM- GESTATIONAL DIABETES MELLITUS

GFR- GLOMERULAR FILTRATION RATE

HCG- HUMAN CHORIONIC GONADOTROPHIN

HTL- HYPERTENSIVE LEUCOENCEPHALOPATHY

IHD-ISCHAEMIC HEART DISEASE

LDH- LACTATE DEHYDROGENASE

LSCS- LOWER SEGMENT CAESAREAN SECTION

MMR – MATERNAL MORTALITY RATE

MRI- MAGNETIC RESONANCE IMAGING

NAD- NO ABNORMALITY DETECTED

PMR-PERINATAL MORTALITY RATE

PRES- POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

PPH-POSTPARTUM HAEMORRHAGE

SAH- SUB ARACHNOID HAEMORRHAGE

UTI- URINARY TRACT INFECTION

GOVERNMENT MOHAN KUMARAMANGALAM
MEDICAL COLLEGE & HOSPITAL
SALEM, TAMILNADU
College: Phone No.0427-2383313 Fax No:0427-2383193
E-Mail ID: deangmkmcslm@gmail.com
Hospital: Phone No: 0427 – 2210674, 2210757 Fax : 0427 – 2210876
E-Mail ID: msgmkmchsalem@gmail.com

Communication of Decision of the Institutional Ethics Committee(IEC)

Ref.No.GMKMCH/2623/IEC/01/2016 - 2 **Dated:30.12.2016**

Protocol title	**"STUDY ON CORRELATION BETWEEN NEUROIMAGING AND NEUROLOGICAL PRESENTATION IN AP AND PP ECLAMSIA AND ITS MATERNAL OUTCOME"**
Principal Investigator	Dr. S. Eswari, I Year, Post Graduate Student of MS (Obstetrics and Gynaecology)
Name of the Guide / Co - Investigator	Dr. B.JEYAMANI MD., DGO., Professor and HOD of Obstetrics and Gynaecology
Name & Address of Institution	Government Mohan Kumaramangalam Medical College & Hospital, Salem, Tamil Nadu.
Type of Review	☑ New review ☐ Revised review ☐ Expedited review
Date of review (D/M/Y)	25.11.2016
Date of previous review, if revised application:	Nil
Decision of the IEC	☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions/ Reasons/ Remarks:	Nil
Recommended for a period of :	3 years

PLEASE NOTE:

- Inform IEC immediately in case of any Adverse events and Serious adverse events.
- Inform IEC in case of any change of study procedure, site and investigator.
- This permission is only for period mentioned above. Annual report to be submitted to IEC.
- Members of IEC have right to monitor the trial with prior intimation.

Signature of Member Secretary

30/12/16

for DEAN,
Govt. Mohan Kumaramangalam
Medical College,
SALEM-636 030.

S.no	IP.No	Name	Age	Gravida	Gestational age	Co Morbid conditions	Type of eclampsia	delivery time and onset of eclampsia	Imminent symptoms	clinical presentation	Urine albumin	Admission BP		neurological examination		Radiology findings	Ophthal findings	Onset of Fit and Delivery Interval	mode of delivery	outcome
												Systolic	Diastolic	Conscious	orientation					
					WEEKS			DAYS												
1	96	Lakshmi	19	Primi	37.57	Anaemia	Antepartum		Headache	Unconsious	Nil	142	98	Un-Conscious	Disoriented	PRES	Normal	< 12 Hours	LSCS	No Complications
2	1290	Boomathi	28	G2P1L1	36.43	Nil	Antepartum		Reduced Urine Output	Unconsious	1+	150	90	Drowsy	Disoriented	PRES	Normal	12-24 Hrs	LSCS	AKI
3	21823	Vellaiyammal	35	G2P1L1	37.29	Anaemia	Antepartum		Blurring of Vision	Unconsious	Nil	160	110	Un-Conscious	Disoriented	No Abnormality	Normal	< 12 Hours	Vaginal	PPH
4	23086	Govindammal	22	G2P1L1	38.14	Nil	Antepartum		Vomiting	Altered Sensorium	2+	148	100	Drowsy	Disoriented	Hypertensive Leuko Encephalopathy	Normal	< 12 Hours	Vaginal	Pulmonary Edema
5	21413	Janani	19	P1L1		Anaemia	Postpartum	3.21	Headache	Incontinence	2+	168	90	Conscious	Well Oriented	PRES	Normal		Vaginal	No Complications
6	20640	Sangeetha	30	P2L2		Nil	Postpartum	2.08	Headache	Unconsious	Nil	152	98	Conscious	Well Oriented	CVT with Infarct	Grade 1		LSCS	No Complications
7	21459	Ranjitham	28	P1L1		GDM	Postpartum	4.08	Headache	Unconsious	2+	162	94	Conscious	Well Oriented	Infarct	Grade 1		Vaginal	No Complications
8	26893	Aiswarya	23	Primi	34.14	Heart Disease	Antepartum		Headache	Unconsious	1+	160	100	Conscious	Well Oriented	PRES	Normal	< 12 Hours	Vaginal	No Complications
9	21682	Muthulakshmi	23	P1L1		Nil	Postpartum	1.08	No Imminent Symptoms	Frothing	4+	170	96	Conscious	Well Oriented	No Abnormality	Normal		LSCS	No Complications
10	29875	Ambika	25	Primi	37.71	Obesity	Antepartum		Headache	Unconsious	Nil	180	110	Conscious	Well Oriented	Infarct	Normal	12-24 Hrs	Vaginal	No Complications
11	30451	Usha	22	P1L1		Sepsis	Postpartum	5.25	Headache	Unconsious	2+	170	100	Drowsy	Disoriented	CVT with Infarct	Normal		LSCS	No Complications
12	30724	Sheelarani	20	Primi	36	Anaemia	Antepartum		Headache	Altered Sensorium	1+	160	100	Conscious	Well Oriented	Infarct	Grade 1	< 12 Hours	LSCS	No Complications
13	26183	Rekha	23	P1L1		Nil	Postpartum	1.13	Vomiting	Altered Sensorium	2+	146	92	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	CVA
14	31210	Amreen	25	G3P2L2	34	Pre-History of Eclampsia	Antepartum		No Imminent Symptoms	Unconsious	Nil	154	96	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	Placental Abruption
15	41449	Chitra	35	Primi	26	Nil	Antepartum		No Imminent Symptoms	Frothing	4+	150	100	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	Hysterotomy	PPH
16	42277	Sasikala	24	Primi	37.43	Nil	Antepartum		Vomiting	Incontinence	1+	160	100	Conscious	Well Oriented	Hypertensive Leuko Encephalopathy	Grade 1	12-24 Hrs	LSCS	Placental Abruption
17	23483	Priyadharshini	21	P1L1		Nil	Postpartum	1.42	Blurring of Vision	Unconsious	3+	170	110	Conscious	Well Oriented	PRES	Grade 2		Vaginal	Pulmonary Edema
18	25612	Munilakshmi	22	P1L1		Obesity	Postpartum	4.13	Headache	Unconsious	2+	166	98	Drowsy	Disoriented	CVT with Infarct	Grade 1		Vaginal	AKI

19	26654	Bharathi	23	P2L2		Nil	Postpartum	2.33	Headache	Unconsious	2+	148	98	Conscious	Well Oriented	Infarct	Normal		LSCS	DIC
20	41656	Eswari	22	Primi	35	Anaemia	Antepartum		No Imminent Symptoms	Unconsious	Nil	180	120	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	PPH
21	42918	Dharani	23	Primi	34.43	Nil	Antepartum		No Imminent Symptoms	Unconsious	1+	150	110	Conscious	Well Oriented	No Abnormality	Normal	12-24 Hrs	LSCS	No Complications
22	43906	Roshmidas	22	P1L1		Anaemia	Postpartum	5	Headache	Altered Sensorium	2+	164	96	Conscious	Well Oriented	PRES	Grade 1		Vaginal	Death
23	31038	Gomala	21	P1L1		Nil	Postpartum	3.42	Headache	Unconsious	1+	160	100	Conscious	Well Oriented	PRES	Normal		Vaginal	No Complications
24	34498	Parameswari	29	P3L3		Nil	Postpartum	2.42	No Imminent Symptoms	Frothing	3+	160	90	Conscious	Well Oriented	Infarct	Normal		Vaginal	HELLP
25	35175	Dhivya	24	P1L1		Anaemia	Postpartum	3.08	Vomiting	Unconsious	4+	150	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
26	3543	Kamali	22	P2L2		GDM	Postpartum	4.21	Headache	Altered Sensorium	Nil	148	90	Drowsy	Disoriented	CVT with Infarct	Grade 1		Vaginal	No Complications
27	2957	Sathya	21	P2L2		Nil	Postpartum	7.25	Vomiting	Unconsious	Nil	160	100	Conscious	Well Oriented	PRES	Normal		LSCS	DIC
28	35800	Kanaga	21	P1L1		Nil	Postpartum	6.08	No Imminent Symptoms	Unconsious	1+	168	94	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
29	36241	Nithya	18	P1L0		Anaemia	Postpartum	12	Headache	Frothing	2+	170	90	Conscious	Well Oriented	PRES	Normal		Vaginal	Pulmonary Edema
30	44032	Gomathi	21	Primi	37.71	GDM	Antepartum		Blurring of Vision	Altered Sensorium	3+	140	96	Conscious	Well Oriented	PRES	Normal	< 12 Hours	LSCS	No Complications
31	3158	Sudha	24	P1L1		Anaemia	Postpartum	5	No Imminent Symptoms	Unconsious	2+	164	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
32	44200	Bhuvaneshwari	20	Primi	37.29	Nil	Antepartum		Blurring of Vision	Unconsious	1+	150	100	Conscious	Well Oriented	PRES	Normal	12-24 Hrs	LSCS	AKI
33	46445	Jothi	23	P1L2		Anaemia	Postpartum	3	Headache	Unconsious	Nil	140	90	Conscious	Well Oriented	Subarchnoid haenorrhage	Normal		Vaginal	Death
34	52317	Ayisha	27	P1L1		Nil	Postpartum	2.42	No Imminent Symptoms	Unconsious	4+	144	92	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	DIC
35	638	Menaga	25	Primi	36.71	Anaemia	Antepartum		No Imminent Symptoms	Altered Sensorium	Nil	146	90	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	Aspiration Pneumonitis
36	47813	Kalpana	19	Primi	30	GDM	Antepartum		No Imminent Symptoms	Unconsious	Nil	152	90	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	Vaginal	PPH
37	9759	Yogapriya	19	P1L1		Anaemia	Postpartum	4.5	Headache	Altered Sensorium	2+	170	90	Conscious	Well Oriented	CVT with Infarct	Grade 2		Vaginal	DIC
38	13628	Manjula	26	P1L1		Nil	Postpartum	3.25	Headache	Frothing	2+	168	90	Conscious	Well Oriented	PRES	Normal		Vaginal	HELLP
39	16006	Madhammal	41	P6L4		Anaemia	Postpartum	2.17	Vomiting	Incontinence	1+	144	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications

40	51332	Rohini	24	Primi	32	Nil	Antepartum		No Imminent Symptoms	Unconsious	2+	160	100	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	No Complications
41	83659	Vijayalkashmi	27	P2L2		Anaemia	Postpartum	7	No Imminent Symptoms	Unconsious	3+	140	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
42	1171	Shanthi	29	P2L2		Nil	Postpartum	8.08	No Imminent Symptoms	Unconsious	Nil	150	90	Conscious	Well Oriented	Infarct	Normal		Vaginal	No Complications
43	1574	Gayathri	19	G2P1L1	33	Nil	Antepartum		Headache	Altered Sensorium	2+	160	90	Conscious	Well Oriented	CVT with Infarct	Grade 1	< 12 Hours	Vaginal	No Complications
44	53711	Vimala	39	Primi	37.14	Nil	Antepartum		Headache	Frothing	2+	170	100	Conscious	Well Oriented	Hypertensive Leuko Encephalopathy	Normal	< 12 Hours	LSCS	HELLP
45	45868	Chitra	25	G2P1L1	31	Anaemia	Antepartum		Headache	Unconsious	2+	180	100	Conscious	Well Oriented	PRES	Grade 4	< 12 Hours	LSCS	Death
46	55801	Muthamilselvi	20	P2L2		GDM	Postpartum	6.17	Blurring of Vision	Altered Sensorium	1+	170	90	Conscious	Well Oriented	CVT with Infarct	Grade 1		Vaginal	No Complications
47	57091	Anushaya	26	P2L1		Nil	Postpartum	3	No Imminent Symptoms	Unconsious	3+	140	100	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
48	60825	Manimegali	21	Primi	31.57	Nil	Antepartum		No Imminent Symptoms	Altered Sensorium	Nil	142	92	Conscious	Well Oriented	No Abnormality	Normal	12-24 Hrs	Vaginal	No Complications
49	46437	Indumathi	27	P2L2		Anaemia	Postpartum	3.33	Headache	Unconsious	3+	156	100	Conscious	Well Oriented	CVT with Infarct	Normal		Vaginal	No Complications
50	1563	Saranya	20	Primi	36.71	Nil	Antepartum		No Imminent Symptoms	Unconsious	2+	140	90	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	HELLP
51	64955	Vidhya	19	Primi	38	Nil	Antepartum		Headache	Unconsious	1+	140	90	Conscious	Well Oriented	PRES	Grade 1	< 12 Hours	LSCS	DIC
52	57039	Monisha	20	P1L1		Anaemia	Postpartum	4.58	No Imminent Symptoms	Unconsious	2+	168	92	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
53	57654	Singaram	38	P4L4		Anaemia	Postpartum	3.08	No Imminent Symptoms	Altered Sensorium	3+	170	100	Conscious	Well Oriented	Cerebral Atrophy	Normal		Vaginal	No Complications
54	68137	Thulasimani	20	G2P1L1	32.43	Nil	Antepartum		Reduced Urine Output	Frothing	2+	160	90	Conscious	Well Oriented	PRES	Grade 1	< 12 Hours	LSCS	AKI
55	60231	Ramya	23	P1L1		Nil	Postpartum	1	Headache	Unconsious	Nil	150	90	Conscious	Well Oriented	PRES	Normal		Vaginal	No Complications
56	69129	Premalatha	20	Primi	33.14	Anaemia	Antepartum		Blurring of Vision	Unconsious	Nil	170	100	Conscious	Well Oriented	No Abnormality	Grade 1	< 12 Hours	LSCS	PPH
57	69342	Kayalvizhi	29	P1L1		Anaemia	Postpartum	3.42	Headache	Unconsious	2+	140	90	Conscious	Well Oriented	CVT with Infarct	Grade 1		LSCS	CVA
58	61173	Mythili	24	P1L1		Nil	Postpartum	5.38	No Imminent Symptoms	Unconsious	2+	160	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	DIC
59	61808	Manjula	20	P1L1		Nil	Postpartum	1.17	No Imminent Symptoms	Unconsious	1+	150	90	Conscious	Well Oriented	Infarct	Normal		LSCS	No Complications

60	74068	Menaga	27	Primi	35.29	Obesity	Antepartum		No Imminent Symptoms	Unconsious	3+	130	100	Conscious	Well Oriented	No Abnormality	Normal	12-24 Hrs	LSCS	No Complications
61	60867	Chellakili	21	P2L2		Pre-History of Eclampsia	Postpartum	2	Blurring of Vision	Unconsious	Nil	148	100	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
62	8254	Madhammal	23	P2L2		Nil	Postpartum	3.08	Headache	Frothing	2+	140	92	Conscious	Well Oriented	CVT with Infarct	Grade 1		Vaginal	CVA
63	73708	Archanamary	22	Primi	38.14	Anaemia	Antepartum		Vomiting	Altered Sensorium	2+	160	110	Conscious	Well Oriented	PRES	Normal	< 12 Hours	LSCS	No Complications
64	77534	Mangalam	30	G2P1L1	36.57	Nil	Antepartum		Headache	Unconsious	1+	160	90	Conscious	Well Oriented	PRES	Grade 1	< 12 Hours	Vaginal	Pulmonary Edema
65	78143	Sangeetha	23	Primi	32.43	Obesity	Antepartum		Headache	Unconsious	2+	170	100	Conscious	Well Oriented	PRES	Grade 1	12-24 Hrs	LSCS	No Complications
66	83903	Chinnaponnu	24	P2L2		Anaemia	Postpartum	4.13	No Imminent Symptoms	Altered Sensorium	2+	160	90	Conscious	Well Oriented	PRES	Normal		Vaginal	No Complications
67	81651	Sripriya	26	Primi	31.57	Nil	Antepartum		Headache	Altered Sensorium	Nil	160	100	Conscious	Well Oriented	PRES	Normal	< 12 Hours	LSCS	DIC
68	30935	Devaiyani	18	Primi	36.14	GDM	Antepartum		Blurring of Vision	Unconsious	1+	152	90	Conscious	Well Oriented	Infarct	Normal	12-24 Hrs	LSCS	No Complications
69	84284	Shayamala	20	P1L2		Anaemia	Postpartum	3.46	No Imminent Symptoms	Unconsious	3+	148	90	Conscious	Well Oriented	No Abnormality	Normal		LSCS	DIC
70	32819	Reshma	22	Primi	35.71	Nil	Antepartum		No Imminent Symptoms	Unconsious	1+	151	90	Conscious	Well Oriented	PRES	Normal	< 12 Hours	Vaginal	Pulmonary Edema
71	80224	Dhalalakshmi	20	P1L1		Nil	Postpartum	2.08	Headache	Unconsious	2+	150	90	Conscious	Well Oriented	CVT with Infarct	Normal		LSCS	No Complications
72	920	Sasikala	22	P1L1		Anaemia	Postpartum	1.33	Vomiting	Frothing	Nil	134	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
73	4775	Janani	20	Primi	32.71	Nil	Antepartum		No Imminent Symptoms	Altered Sensorium	2+	168	90	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	PPH
74	37669	Poornima	24	P2L2		Obesity	Postpartum	3.21	No Imminent Symptoms	Unconsious	2+	152	90	Conscious	Well Oriented	No Abnormality	Grade 3		Vaginal	DIC
75	37754	Bakhiyalkshmi	30	G2P1L0	37.57	Pre-History of Eclampsia	Antepartum		No Imminent Symptoms	Altered Sensorium	1+	170	112	Conscious	Well Oriented	PRES	Normal	< 12 Hours	Vaginal	No Complications
76	43319	Selvi	20	P1L1		Nil	Postpartum	4	No Imminent Symptoms	Incontinence	2+	151	100	Conscious	Well Oriented	No Abnormality	Normal		LSCS	HELLP
77	8628	Dhanam	24	P2L2		Anaemia	Postpartum	3.25	No Imminent Symptoms	Frothing	3+	142	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
78	46437	Sandhiyaa	23	Primi	38.14	Nil	Antepartum		Headache	Unconsious	Nil	166	90	Conscious	Well Oriented	CVT with Infarct	Normal	< 12 Hours	LSCS	CVA
79	44796	Deepa	21	P1L0		Nil	Postpartum	2.33	No Imminent Symptoms	Unconsious	Nil	148	92	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	HELLP

80	9154	Chennammal	24	P2L2		Anaemia	Postpartum	3.38	No Imminent Symptoms	Unconsious	1+	142	96	Conscious	Well Oriented	No Abnormality	Grade 1		Vaginal	DIC
81	47160	Selvi	25	Primi	37.86	Nil	Antepartum		Headache	Unconsious	1+	150	90	Conscious	Well Oriented	Hypertensive Leuko Encephalopathy	Normal	12-24 Hrs	LSCS	CVA
82	24929	Vanitha	36	P1L1		Nil	Postpartum	4	Headache	Unconsious	2+	140	90	Conscious	Well Oriented	Infarct	Normal		Vaginal	No Complications
83	20965	Chandra	28	Primi	27	Nil	Antepartum		Blurring of Vision	Altered Sensorium	2+	160	100	Conscious	Well Oriented	PRES	Normal	< 12 Hours	Spontaneous Expelsion	CVA
84	21832	Meera	32	G4P3L3	38.71	Anaemia	Antepartum		Headache	Unconsious	2+	158	90	Conscious	Well Oriented	Infarct	Cortical Blindness	< 12 Hours	Vaginal	Blindness
85	35653	Sowmiya	20	P1L1		Nil	Postpartum	3.29	No Imminent Symptoms	Unconsious	Nil	142	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
86	45175	Manonmani	23	P2L1		Nil	Postpartum	2	No Imminent Symptoms	Unconsious	3+	148	90	Conscious	Well Oriented	PRES	Normal		Vaginal	HELLP
87	6162	Saroja	25	P2L2		Nil	Postpartum	6.08	No Imminent Symptoms	Altered Sensorium	4+	160	90	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
88	7943	Priyanka	20	P1L1		Anaemia	Postpartum	1.13	No Imminent Symptoms	Unconsious	1+	150	92	Conscious	Well Oriented	No Abnormality	Normal		LSCS	DIC
89	55367	Kavitha	25	P1L1		Nil	Postpartum	1	Reduced Urine Output	Frothing	Nil	140	92	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	No Complications
90	43442	Kaliyammal	24	G2A1	37.57	Anaemia	Antepartum		Headache	Frothing	1+	150	100	Drowsy	Disoriented	CVT with Infarct	Normal	12-24 Hrs	LSCS	No Complications
91	10332	Saranya	19	P2L2		Nil	Postpartum	0.25	No Imminent Symptoms	Unconsious	2+	148	100	Conscious	Well Oriented	PRES	Normal		Vaginal	No Complications
92	8272	Nandini	23	P2L2		Nil	Postpartum	0.42	Vomiting	Unconsious	1+	140	90	Conscious	Well Oriented	Infarct	Normal		LSCS	No Complications
93	57801	Amudha	22	P1L1		Anaemia	Postpartum	1	Headache	Unconsious	2+	152	90	Conscious	Well Oriented	CVT with Infarct	Normal		Vaginal	Death
94	58432	Lakshmi	23	G2A1	32.43	Anaemia	Antepartum		Blurring of Vision	Unconsious	2+	160	90	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	PPH
95	3412	Swathi	19	Primi	38	Nil	Antepartum		Blurring of Vision	Unconsious	1+	150	82	Conscious	Well Oriented	PRES	Cortical Blindness	< 12 Hours	Vaginal	Blindness
96	64987	Arulmozhi	20	P1L1		Nil	Postpartum	3.08	Vomiting	Unconsious	1+	150	90	Conscious	Well Oriented	CVT with Infarct	Normal		LSCS	DIC
97	64450	Malathi	21	P1L1		Nil	Postpartum	0.33	Headache	Unconsious	Nil	142	90	Conscious	Well Oriented	Infarct	Normal		Vaginal	No Complications
98	62632	Tamilselvi	23	P1L1		Nil	Postpartum	0.42	Blurring of Vision	Unconsious	Nil	140	98	Conscious	Well Oriented	No Abnormality	Normal		Vaginal	HELLP
99	51793	Jayasudha	23	G3P1L1A1	36.14	Obesity	Antepartum		No Imminent Symptoms	Unconsious	4+	150	90	Conscious	Well Oriented	No Abnormality	Normal	< 12 Hours	LSCS	No Complications
100	4526	Valarmathi	35	P2L1		Anaemia	Postpartum	18	Headache	Unconsious	3+	162	98	Un-Conscious	Disoriented	CVT with Infarct	Grade 1		LSCS	Death