ANALYSIS OF FETOMATERNAL OUTCOME IN ECLAMPSIA

A dissertation submitted to the

TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations for

the award of the degree of

M.D. (Branch II)

OBSTETRICS AND GYNECOLOGY

COIMBATORE MEDICAL COLLEGE

COIMBATORE – 641 014

APRIL 2012

CERTIFICATE

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ACKNOWLEDGEMENT

I solicit my humble thanks to God Almighty for guiding me throughout my project work.

I express my gratitude to Dr.Vimala, the Dean Coimbatore Medical College Hospital for providing facilities to carry out this project work successfully.

I am extremely thankful to Prof.Dr. M. Swathandra Devi M.D., DGO., Head of Department of Obstetrics & Gynecology and my Unit Chief Dr. Sundari M.D.,DGO., for their valuable guidance, support and advice rendered in completion of my project.

I express my sincere thanks to Prof. Dr. Revathy, Prof. Dr .Usha Rani, Prof. Dr. Vijaya for their valuable help in the course of my project.

I thank Prof. Dr. Neelambikai M.D., professor of physiology and Head of Ethics Committee Coimbatore Medical College for the invaluable suggestions and corrections.

I am extremely thankful to Dr. V. Geetha M.D., Dr. Nallichandra M.D., Dr.Manonmani M.D., DGO.,Dr.Savithri DGO., for their constant encouragement and support to carry out this study.

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ABSTRACT

A cress-sectional hospital base study was conducted to analyze fetomaternal outcome in eclampsia, clinical characteristics that predispose as risk factors, changes in serum magnesium level and its influence on maternal outcome. Study conducted from 1st March 2010 to 30th June 2011 in the department of Obstetrics and Gynecology, Coimbatore Medical College Hospital, Coimbatore.

In our study there were total of 104 eclamptic patients with an incidence of 1.23%. Only 65.38% were booked patients and with regular antenatal checkups. Primigravida constituted 76%. Maximum incidence of eclampsia was in the age group between 21 and 25 years (58%). Antepartum eclampsia constituted 81%. Mean blood pressure on admission was 156 ± 15 / 103 ± 12 mm Hg. Headache was the commonest imminent symptom. 57% had vaginal delivery. Serum magnesium level was significantly low (1.74 ± 0.28 mg/dl ; p value < 0.05) in eclamptic patients. Maternal complications included PPH, HELLP, renal failure, abruptio placenta, pulmonary edema, aspiration and intra cerebral hemorrhage in decreasing order of frequency. There were 4 maternal deaths with case fatality ratio of 3.84%, contributing 10.5% of total maternal deaths. Half of the deaths were due to HELLP-DIC. Age >25 years, multipara, unbooked patients, seizures ≥ 5 episodes, systolic BP ≥ 160 mm Hg were significantly associated with maternal

complications. Platelet less than 1 lakh and serum magnesium level less than 1.6 mg/dl were associated significantly with adverse maternal outcome. Perinatal complications included small for gestational age, preterm, jaundice, seizures, respiratory distress, sepsis. There were 30 perinatal deaths contributing to 6.96% of total perinatal mortality. Perinatal mortality rate was 3.56/1000 live births. Multiparity, gestational age ≤ 32 weeks, seizures >5 episodes were significantly associated with perinatal mortality.

Key words: Eclampsia, fetomaternal outcome, serum magnesium.

INTRODUCTION

Eclampsia has been a recognized pathological entity since the time of Hippocrates and ancient Greek.[1] It is derived from the Greek word meaning "flash out", in the sense of sudden event and dates back to seventeenth century. Eclampsia is perceived as the end of linear spectrum that stretches from the normal pregnancy through mild gestational hypertension, pre-eclampsia finally eclampsia.

It is an enigmatic disease and unique to pregnancy.A number of social, genetic, medical and obstetric conditions predispose to an increase risk of pre-eclampsia and eclampsia. It is the multisystem disorder. The exact etiology of pre-eclampsia is unknown. Several theories have been proposed over the years, most of which have not withstood the test of time. Some of these failed to stand up to further investigation, while others yielded conflicting results in different studies, and none could explain all the changes in this condition. As Boyd stated pre-eclampsia remains "die krankheit der theorian" – the disease of theories.

Worldwide it accounts for 50,000 maternal deaths annually.[2] It is said that pre-eclampsia and eclampsia contribute to death of a women every 3 minutes worldwide.[3]20 times more common in developing countries.Incidence of eclampsia in India is approximately 220 per 10,000 deliveries, contributing about 8% of maternal mortality[4]. It ranks second only to anemia in developing countries.

Pre-eclampsia is not preventable but eclampsia is preventable.In spite of the global and regional interventions and initiatives from government, its outcome in terms of maternal and perinatal mortality continues to be worse. Eclampsia is preceded by alarming symptoms and signs of pregnancy induced hypertension.The institution of vigilant antenatal care to detect risk factors and prompt treatment of cases of pre-eclampsia will ameliorate the disease burden.

Incidence, morbidities and mortalities of eclampsia remain unacceptably high in developing countries. This indicates the need for continued in depth studies into its characteristics and pattern. This study was undertaken to determine the incidence of eclampsia, identify the predisposing socio-demographic factors, demonstrate clinical profiles, changes in serum magnesium levels and analyze modes of management and resulting maternal and fetal outcome at Coimbatore Medical College Hospital.

AIM OF THE STUDY

1. To record the clinical profile of patients with eclampsia and to assess the incidence of eclampsia.
2. To study the various maternal and fetal outcome by means of morbidity and mortality in eclampsia.
3. To analyze the clinical characteristics that predisposes as risk factor for eclampsia and fetomaternal outcome.
4. To analyze the changes in platelet count, serum magnesium level and their influence on maternal outcome.

REVIEW OF LITERATURE

Hypertensive disorders complicate 5 to 10 % of all pregnancies and together they form the deadly triad along with hemorrhage and infection, contribute greatly to maternal morbidity and mortality rates. In developed countries, 16% of maternal mortality was due to hypertensive disorders[5]. Over half of these hypertension related deaths were preventable.[6]

The Working Group of the NHBPEP- National High Blood Pressure Education Program (2000) classification of hypertensive disorders complicating pregnancy is as follows:

1. Gestational hypertension
2. Pre-eclampsia
3. Eclampsia
4. Pre-eclampsia superimposed on chronic hypertension
5. Chronic hypertension

An important feature of this classification is differentiating preeclampsia and eclampsia from other hypertensive disorders because the former two are potentially more ominous. This concept is also important to interpret and appreciate studies that address the etiology, pathogenesis, and clinical management of pregnancy related hypertensive disorders.

- **GESTATIONAL HYPERTENSION**

The diagnosis of gestational hypertension is made in women whose blood pressure reaches 140/90 mm Hg or greater for the first time after

mid pregnancy (20 weeks), but in whom proteinuria is not identified. Almost half of these women subsequently develop preeclampsia. Gestational hypertension is reclassified as transient hypertension if evidence for preeclampsia does not develop and the blood pressure returns to normal by 12 weeks postpartum.

- **PREECLAMPSIA**

It is a pregnancy specific syndrome that can affect virtually every organ. Proteinuria is the surrogate objective marker that defines the system wide endothelial leak. It is defined by 24-hour urinary protein excretion exceeding 300 mg, persistent 30 mg/dL (1+dipstick) protein in random urine samples.[7]

- **ECLAMPSIA**

The onset of convulsions in a woman with preeclampsia that cannot be attributed to other causes is termed eclampsia. The seizures are generalized and may appear before, during, or after labor. 10 % of eclamptic seizures develop before overt proteinuria is identified.[8]

- **ETIOPATHOGENESIS**

Gestational hypertensive disorders are more likely to develop in women who

- Are exposed to chorionic villi for the first time.

- Are exposed to a superabundance of chorionic villi, as with twins or hydatidiform mole
- Have preexisting renal or cardiovascular disease
- Are genetically predisposed to hypertension developing during pregnancy.

Regardless of precipitating etiology, the cascade of events that leads to the preeclampsia is characterized by a host of abnormalities that result in vascular endothelial damage and subsequent vasospasm, transudation of plasma, and ischemic and thrombotic sequelae. Recently preeclampsia has been considered as two stage disorder. Stage 1 faulty endovascular trophoblastic remodeling that downstream causes the stage 2 clinical syndrome.[9]

❖ **Abnormal trophoblastic invasion**

In normal implantation, the uterine spiral arterioles undergo extensive remodeling as they are invaded by endovascular trophoblasts. These cells replace the vascular endothelial and muscular linings to enlarge the vessel diameter. In preeclampsia there may be incomplete trophoblastic invasion. With such shallow invasion myometrium, vessels are not lined by endovascular trophoblasts.[10]They do not lose endothelial lining and musculoelastic tissue, and their mean external diameter is only half that of vessels in normal placentas, abnormally narrow spiral arteriolar lumen impairs placental blood flow. The magnitude of defective trophoblastic

invasion of the spiral arteries correlates with the severity of the hypertensive disorder.

❖ **Immunological factors**

Diminished perfusion and a hypoxic environment eventually lead to release of placental debris that incites a systemic inflammatory response, results in dysregulation of immune tolerance . Formation of blocking antibodies to placental antigenic sites might be impaired. Beginning in early second trimester women who develop preeclampsia, Th_1 action increased and the Th_1/Th_2ratio changes. Contributors to an enhanced immunologically mediated inflammatory reaction are stimulated by placental microparticles.[11]

❖ **Endothelial cell activation**

Briefly, cytokines such as tumor necrosis factor-α(TNF-α), and the interleukins (IL) may contribute to the oxidative stress associated with preeclampsia. This is characterized by reactive oxygen species and free radicals that lead to formation of self-propagating lipid peroxides[12]. These in turn generate highly toxic radicals that injure endothelial cells, modify their nitric oxide production, and interfere with prostaglandin balance. These observations on the effects of oxidative stress in preeclampsia have given rise to increased interest in the potential benefit of antioxidants to prevent preeclampsia. Antioxidants are from a diverse family of compounds that function to prevent overproduction of and damage

caused by noxious free radicals. Dietary supplementation with antioxidants to prevent preeclampsia has thus far proven unsuccessful.

❖ **Genetic factors**

Preeclampsia is a multifactorial, polygenic disorder. An incidence risk factor for preeclampsia is 20 to 40 percent for daughter of preeclampsia. Thus the phenotypic expression of inherited gene will differ depending on interactions with environmental factors.

More than 70 genes have been studied for their possible association with preeclampsia. Seven of these have been investigated widely.[13]

GENETIC FACTORS:

GENE	FUNCTION AFFECTED	CHROMOSOME
MTHFR(C677T)	Methylene tetrahydrofolate Reductase	1p36.3
F5(Leiden)	Factor V	1q23
AGT(M235T)	Angiotensinogen	1q42-q43
HLA(Various)	Human Leukocyte Antigens	6p21.3
NOS3(Glu 298 Asp)	Endothelial nitricoxide	7q36
F2(G20210A)	Prothrombin	11p11-q12
ACE	Angiotensin converting enzyme	17q23

- Increased pressor response to infused norepinephrine and angiotensin II in women with early preeclampsia. Normal pregnant women develop refractoriness to vasopressor.

- Endothelial prostacyclin (PGI_2) production decreases in preeclampsia. Thromboxane A_2 increases. PGI_2/TXA_2ratio decreases. The net result favors increased sensitivity to infused angiotensin II and ultimately vasoconstriction.

- Nitric oxide – potent vasodilator is synthesized from L-arginine by endothelial cells. Withdrawal of nitric oxide results in a clinical picture similar to preeclampsia in a pregnant animal model[14]. It appears that preeclampsia is associated with decreased endothelial nitric oxide synthase expression, thus increased nitric oxide inactivation. These responses may be race related.[15]

- Soluble Fms –like tyrosine kinase 1(sFlt-1) is a variant of the Flt-1 receptor for placental growth factor (PlGF) and vascular endothelial growth factor (VEGF). Increased maternal sFlt-1 levels inactivate and decreased circulating free PlGF, VEGF concentrations leading to endothelial dysfunction.[16]

- Soluble endoglin (sEng) is a placenta- derived molecule that blocks endoglin which is a cofactor for TGF-β. This soluble form endoglin inhibits TGF hence decreased endothelial nitric oxide dependent vasodilatation.[17]

- Cardiac output in preeclampsia decreases likely due to increased peripheral vascular resistance.

- Hemoconcentration is hallmark of preeclampsia. Generalized vasoconstriction that follows endothelial activation and leakage of plasma into the interstitial space.

- THROMBOCYTOPENIA- it is common hence platelet count is routinely measured in any form of gestational hypertension. The frequency and intensity of thrombocytopenia vary and are dependent on the severity and duration of the preeclampsia. Overt thrombocytopenia defined by platelet count less than 1,00,000 cells/μL – indicates severe disease. In general lower the platelet count, higher the rates of maternal and fetal morbidity and mortality[18]. In most cases delivery is advisable because thrombocytopenia continues to worsen. After delivery, the platelet count may continue to decrease for the first day or so. It then usually increases progressively to reach a normal level usually within 3 to 5 days. In some instances platelet count continues to fall after delivery like HELLP syndrome.

- Other platelet abnormalities include platelet activationwith increased degranulation, thromboxane A_2 release and in vitro platelet aggregation is decreased compared with the normal

increase characteristic of pregnancy.[19]This is likely due to platelet exhaustion following in vivo activation. Although the cause is unknown, immunological process or simply platelet deposition at sites of endothelial damage may be implicated.Platelet bound and circulating platelet–bindable immunoglobulins are increased, which suggest platelet surface alterations. Severe thrombocytopenia does not develop in fetus or infant in preeclamptic women.

- KIDNEY- during normal pregnancy blood flow and glomerular filtration increase. With development of preeclampsia, there may be a number of reversible anatomical and pathophysiological changes. Renal perfusion and glomerular filtration are reduced. Glomerular endotheliosis blocking the filtration barrier. Intensive intravenous fluid management is not indicated for these women with oliguria, unless diminished urine output is caused by hemorrhage. Plasma uric acid is typically elevated in preeclampsia, due to reduction in filtration and enhanced tubular reabsorption. At least some degree of proteinuria will establish the diagnosis of preeclampsia. In 24-hour quantitative urinary specimen, the standard consensus threshold value used is>300 mg/24-hour. Urinary dip stick method is simple but with false positive and negative results. Worsening of proteinuria has been considered to

be a sign of severe disease, this may not be the case. It is currently being investigated.

- LIVER- hepatic changes in women with fatal eclampsia were described in 1856 by Virchow. The characteristic lesions commonly found in the liver periphery. Hemolysis, hepatocellular necrosis, and thrombocytopenia were later termed as HELLP syndrome. 10 percent HELLP had concurrent eclampsia[20]. They have worse outcome than only with preeclampsia. Liver involvement in preeclampsia is clinically significant in following circumstances. 1. Symptomatic involvement, typically manifest by moderate to severe right upper or midepigastric pain and tenderness, is usually only seen with severe disease. 2. Asymptomatic elevations of serum hepatic transaminase levels AST and ALT are considered markers for severe preeclampsia. Values seldom exceed 500 U/L. they inversely follow platelet levels. Both usually normalize within 3 days following delivery. 3. Hepatic hemorrhage from areas of infarction may extend to form a hepatic hematoma. These in turn may extend to form a subcapsular hematoma that may rupture. They can be identified using CT or MRI. Management usually consists of conservative and observation. Very few cases may require prompt surgical intervention.

- BRAIN- headaches and visual symptoms are common with severe preeclampsia, and associated convulsions define eclampsia. Principle lesions found in eclamptic women were cortical and subcortical petechial hemorrhages. Other frequently described major lesions were subcortical edema. The classical microscopic vascular lesions consist of fibrinoid necrosis of the arterial wall and perivascular microinfarcts and hemorrhage. Two general theories to explain cerebral abnormalities associated with eclampsia. Importantly, endothelial dysfunction that characterizes the preeclampsia syndrome likely plays a key role in both: 1. Response to acute and severe hypertension, cerebrovascular overregulation leads tovasospasm.[21]Deminished cerebral blood flow is hypothesized to result in ischemia, cytotoxic edema, and eventually, tissue infarction. 2. Sudden elevations in systemic blood pressure exceed the normal cerebrovascular autoregulatory capacity.[22] Forced vasodilation and vasoconstriction develops. At capillary level, disruption of hydrostatic pressure, hyperperfusion, and extravasation of plasma and red cells through endothelial tight-junctions openings leading to vasogenic edema. Clinical manifestations include 1. Headache and scotoma are thought to arise from cerebrovascular hyperperfusion that has a predilection for the occipital lobes. 50 to 70 percent of women have headaches and 20 to 30 percent have visual changes preceding eclamptic convulsions. 2. Convulsions are diagnostic for eclampsia.

3. Blindness is rare in preeclampsia but can in eclampsia. 4. Generalized cerebral edema may develop and is usually manifest by mental status changes that vary from confusion to coma. This situation is particularly dangerous because fatal supratentorial herniation may result.

- MAGNESIUM LEVELS IN ECLAMPSIA-the role of serum electrolytes in PIH has been studied. For long time it is known that sodium retention can cause hypertension and sodium restricted diet is advised to hypertensive patient. The serum potassium is also measured in hypertensive patient to screen mineralocorticoid induced hypertension and to provide a baseline before beginning a diuretic therapy. Recently other two electrolytes namely calcium and magnesium is gaining importance in preeclampsia. The decrease in serum magnesium level in preeclampsia is reported.[23] Calcium and magnesium can regulate blood pressure in a neurohumoral, renal and adrenal machanism[24], which can explain their decrease in preeclampsia. Magnesium is the 4th most abundant cation in the body and is present in more than 300 enzymatic systems where it is crucial for ATP metabolism.[25] Magnesium may influence blood pressure by modulating vascular tone and structured through its effects on myriad biochemical reactions that control vascular contraction/dilation, growth/apoptosis, differentiation and inflammation. Magnesium acts as calcium channel antagonist. It stimulates production of vasodilator

prostacyclin and nitric oxide and alters vascular responses to vasoconstrictors agents. Its deficiency level can also play a role in hypertension in pregnancy.[26]Regarding magnesium there are lots of contrast studies. Some studies do not support the hypothesis that low serum magnesium is a risk factor for developing hypertension and vascular dysfunction.[27]

1. Magnesium modulates the cardiovascular effect of sodium and potassium and it is the co-factor for the sodium-potassium ATPase activity.[28]
2. It affects the cardiac and smooth muscle cells by altering the transport of calcium and its binding to the membrane and organ cells.
3. It acts peripherally to produce peripheral vasodilatation and a fall in blood pressure. Thus, low levels of magnesium predispose to an increase in the arterial pressure.[29]
4. It is known to increase the prostacyclin release from the endothelial cells of the blood vessels, which acts as a potent vasodilator. Magnesium depletion increases the vasoconstrictor effect of angiotensin II and nor-adrenaline.
5. Magnesium also has a substantial beneficial effect in preeclampsia for the prevention and treatment of convulsions. Therapeutic magnesium sulphate which is used in PIH inhibits phosphatidylinositol-4,5-bisphosphonate specific phospholipase c activity and subsequent calcium release in the cells, thus leading to

decreased intracellular calcium levels and a decrease in blood pressure.

6. Hemodilution effect of estrogen and increased demand of fetus decreases the serum magnesium level and in preeclampsia urinary excretion of magnesium also increases so the level decreases further.[30]
7. Magnesium deficiency causes hemodynamic abnormalities such as arterial wall thickening, abnormal vascular tone and endothelial dysfunction which are due to alteration in the biology of cellular and non-cellular components of arterial wall. There may be a causal relationship between hypomagnesaemia and preeclampsia since magnesium is involved in blood pressure regulation through an intracellular inhibition of NO synthase in endothelial cells.[31]

- **RISK FACATORS**
  - AGE- risk of preeclampsia/eclampsia is increased at the extremes of reproductive age. 20-29 years, young primipara women. Maternal age over 40 years is associated with double the risk of preeclampsia irrespective of parity.[32]
  - PARITY- primigravida have 15 times greater risk compared to multigravida of developing preeclampsia.[33]Although at a lower risk for developing preeclampsia multiparous women often develop severe forms of the disease and are at a higher risk of developing

severe morbidity such as eclampsia, renal failure, and pulmonary edema and hence higher mortality.[34]

- PREVIOUS PREECLAMPSIA- history of preeclampsia in the previous pregnancy is a strong predictor of recurrence of the disease. Risk in a subsequent pregnancy to be increasing by more than 6 times.[35]Early onset preeclampsia run a greater risk of recurrence. It has been observed that there is an increased incidence of preeclampsia with a different partner or donor sperm pregnancy meaning that every first pregnancy with a new partner is at greater risk.

- LONG BIRTH INTERVAL- the risk of preeclampsia in a multipara is found to be directly related to the time elapsed since the last delivery. If the interval between 2 pregnancies was more than 10 years the risk of preeclampsia is equaled the risk in primigravidas.[36]

- GENETIC FACTOR- a positive family history is a known risk factor as it is observed that the daughters of eclamptic women are at eight times higher risk of developing preeclampsia and hence has a genetic predisposition.[37]

- MULTIPLE PREGNANCY- it is a high risk for developing preeclampsia. Risk for preeclampsia is four times in twin pregnancies compared to singletons.[38]It was found that the risk was irrespective of the chorionicity and zygosity.

- ASSISTED REPRODUCTION- has a higher risk of preeclampsia because of the higher incidence of multiple pregnancies. Gamete donation found to increase the risk of preeclampsia. Women who conceived with donor sperm, donor oocytes or donated embryos showed that the rate of preeclampsia was increased to 18.1 percent compared with controls who consisted of women who either conceived spontaneously or with intrauterine insemination with husband's sperm.[39]

- OBESITY-it is an established risk factor by itself.[40]In addition it is one of the components of syndrome X, the other components of this medical disorder being type II diabetes and hypertension. It has been established that the waist circumference in early pregnancy which is a measure of central obesity especially when associated with insulin resistance is directly associated with preeclampsia.[41]

- PRE-EXISTING HYPERTENSION-Incidence of preeclampsia is much higher in women with chronic hypertension. The risk of preeclampsia is related to degree of hypertension. Superimposed preeclampsia is 46 percent in severe hypertension (diastolic of 110 mmHg or more) as against an incidence of 14 percent among those with mild hypertension (diastolic BP 90-119 mmHg).[42]

- DIABETES MELLITUS- risk of preeclampsia is roughly doubled by diabetes. Pre-existing diabetes mellitus are at greater risk than with those having GDM. As in hypertension risk is proportionate to

degree of DM. women with micro vascular complications are at higher risk than those without it.[43]

- DYSLIPIDAEMIA- It has been observed that preeclampsia is associated with increased triglyceride synthesis by the liver and decreased catabolism of the same at the periphery leading to elevated triglyceride levels. It is thought that adipocyte lipolysis which is a feature of normal pregnancy that leads to elevated non-esterified fatty acids is exaggerated in women destined to develop preeclampsia. Elevated NEFA concentrations in general, cause dyslipidemia in human causing hypertriglyceridemia. It also enhances oxidative stress, inflammation and endothelial dysfunction, features characteristics of pathogenesis of preeclampsia.[44]Probably women destined to develop preeclampsia possess a preexisting lipolytic defect. Similar situation is seen in individuals with central obesity.

- THROMBOPHILIA- whether hereditary or acquired, thrombophilia is a well-established high risk factor.[45]Antiphospholipid syndrome characterized by the presence of antibodies like lupus anticoagulant and/or anticardiolipin antibodies in association with clinical history suggestive of thrombosis or adverse pregnancy outcome is a high risk factor for development of preeclampsia. Thrombophilia, there is definite association with early onset preeclampsia.[46]

- Smoking is found to be associated with reduced incidence of preeclampsia though otherwise it is associated with a higher perinatal loss.[47]Stress is another important factor that predisposes to the development of preeclampsia.

- Certain placental and fetal factors have a bearing on the occurrence of preeclampsia. A large placenta as is often seen in multiple pregnancy, placental hydrops and molar pregnancy are associated with higher incidence of preeclampsia. Certain chromosomal defects like triploidy, trisomy 13, and trisomy 16 mosaicism are also frequently associated with preeclampsia. All conditions associated with poor placentation have a higher risk of developing preeclampsia.

PREDICTORS OF PREECLAMPSIA

Measurement of variety of biological, biochemical, and biophysical markers implicated in the pathophysiology of preeclampsia has been proposed to predict its development. Attempts have been made to identify early markers of faulty placentation, impaired placental perfusion, endothelial cell activation and dysfunction, and activation of coagulation. But unfortunately these testing strategies are with poor sensitivity and with poor positive predictive value for preeclampsia. However, combinations of tests, some yet to be adequately evaluated, that may be promising. Most of the testingare cumbersome and time consuming.

- PROVOCATIVE PRESSOR TESTS- there are three tests extensively evaluated to assess blood pressure increase in response to a stimulus. The "*roll over test*" measures the hypertensive response in women at 28-32 weeks who are resting in the left lateral decubitus position and the "roll-over" to assume a supine position. The *isometric exercise test* employs the same principle by squeezing a handball. The *angiotensin II infusion test* is performed by giving incrementally increasing doses intravenously, and the hypertensive response is quantified. All three tests found to have sensitivities ranging 55-70 percent and specificities approximately 85 percent.[48]

- UTERINE ARTERY DOPPLER VELOCIMETRY- faulty trophoblastic invasion of the spiral arteries. This results in decreased placental blood flow and increased uterine artery impedance in upstream. Increase flow resistance results in an abnormal waveform represented by an increased diastolic notch.

- PULSE WAVE ANALYSIS- finger arterial pulse "*stiffness"* is an indicator of cardiovascular risk. Investigations have evaluated its usefulness in preeclampsia prediction.[49]

- ENDOCRINE DYSFUNCTION-number of serum analyses that have been proposed to help predict preeclampsia are human chorionic gonadotropin (hCG), alpha-fetoprotein (AFP), estriol,

pregnancy associated protein A (PAPP A), Inhibin A, activin A, placental protein 13, corticotrophin releasing hormone.

- SERUM URIC ACID-it is one of the earliest laboratory manifestations of preeclampsia. It is likely results from reduced uric acid clearance from diminished glomerular filtration, increased tubular reabsorption, and decreased secretion. But they have very low sensitivity and specificity.

- Microalbuminuria and urine albumin:creatinine ratio have unacceptable sensitivity and specificity.[50]

- FIBRONECTINS-these high molecular weight glycoproteins serve a variety of cellular functions that include adhesion and morphology, migration, phagocytosis, and hemostasis.[51]Fibronectins are released from endothelial cells and extracellular matrix following endothelial injury. Recent studies concluded that neither cellular nor total fibronectins was clinically useful to predict preeclampsia.[52]

- COAGULATION ACTIVATION- thrombocytopenia and platelet dysfunction are integral features of preeclampsia. Platelet activation causes increased destruction and decreased concentrations. Mean platelet volume increases because of young platelet age. Although markers of coagulation activation are increased, there is a substantiate overlap between normal pregnancies.

- OXIDATIVE STRESS- increased levels of lipid peroxidases coupled with decreased antioxidant activity have raised the possibility that markers of oxidative stress might predict preeclampsia.[53] Malondialdehyde is a marker of lipid peroxidation. Other pro-oxidation markers include iron, transferrin, and ferritin, free fatty acids, triglycerides.[54]

- ANGIOGENIC FACTORS- an imbalance between proangiogenic and antiangiogenic factors is related to the pathogenesis of preeclampsia. Proangiogenic factors like vascular endothelial growth factors, placental growth factors begin to decrease before clinical preeclampsia develops. Antiangiogenic factors like soluble fms-like tyrosine kinase -1, soluble endoglin are increased.[55]

- FREE FETAL DNA-using polymerase chain reaction, free fetal DNA can be detected in maternal plasma. It is hypothesized that free DNA is released by accelerated apoptosis of cytotrophoblast.[56]

- **PREVENTION**
  - LOW SALT DIET- one of the earliest research efforts to prevent preeclampsia was salt restriction.[57] But randomized control trial showed that a sodium restricted diet was ineffective in preventing preeclampsia.[58]

- CALCIUM SUPPLEMENTATION- women with low dietary calcium intake were at significantly increased risk for gestational hypertension. Trials have shown that unless women are calcium deficient, supplementation has no salutary effects.[59]

- FISH OIL SUPPLEMENTATION- the most common dietary sources are eicosapentaenoic acid and alpha-linoleic acid. With proclamations that supplementation with these fatty acids would prevent inflammatory mediated atherogenesis, it was not a quantum leap to posit that they might prevent preeclampsia.[60]

- ANTIHYPERTENSIVE DRUGS- women given diuretics had a decreased incidence of edema and hypertension, but not of preeclampsia. Because women with chronic hypertension are at high risk for preeclampsia, antihypertensive drugs reduce the incidence of superimposed preeclampsia.

- ANTIOXIDANTS- there are inferential data that an imbalance between oxidant and antioxidant activity may have an important role in the pathogenesis of preeclampsia. Vitamin C & E may decrease such oxidation. Moreover, women who developed preeclampsia were found to have reduced plasma levels of these two vitamins.[61]Thus dietary supplementation was propose as a

method to improve the oxidative capability of women at risk for preeclampsia.

- LOW-DOSE ASPIRIN- 50-150 mg daily, aspirin inhibits effectively platelet thromboxane A2 biosynthesis with minimal effects on vascular prostacyclin production.[62]In meta-analysis of 31 randomized trials, for women received antiplatelet agents, relative risk of preeclampsia was decreased significantly by 10 percent for development of preeclampsia, superimposed preeclampsia, preterm devlivery.[63]The number needed to treat was high. Because of marginal benefit, it seems reasonable to individualize use of low-dose aspirin to prevent preeclampsia. Collaborative Low Dose Aspirin Study in Pregnancy (CLASP), a large randomized controlled trial reported a reduction of 12% in preeclampsia.

- LOW-DOSE ASPIRIN PLUS HEPARIN- because of high prevalence of placental thrombotic lesions found with severe preeclampsia, there have been several observational trials to evaluate heparin treatment for affected women. Prophylaxis with low-molecular-weight heparin plus low-dose aspirin on pregnancy outcomes in women with a history of severe early-onset preeclampsia and low-birth weight infants. There were better pregnancy outcomes in women given low-dose aspirin plus low-

molecular-weight heparin compared with those given low-dose aspirin alone.[64]

CLINICAL FEATURES OF ECLAMPSIA:

- Headache – persistent occipital or frontal headaches
- Visual disturbance – blurred vision and photophobia.
  Examination of the optic fundi will show marked retinal edema and in the severe stages haemorrhage, exudates and papilledema may also be present.
- Restless and agitation
- Epigastric and/or right upper quadrant pain
- Nausea and vomiting
- Oliguria
- Laboratory evidence of disseminated intravascular coagulation

MATERNAL COMPLICATION:

- Abruptio placenta is an important complication of hypertension in pregnancy and a massive unheralded bleed can have disastrous consequences.
- Cerebrovascular accident – usually intracerebral haemorrhage, occasionally ruptured intracranial aneurysm or cerebral thrombosis. This may be fatal or the woman may be left with a

residual neurological deficit such as hemiplegia, dysphasia or visual disturbance.

- HELLP syndrome- hemolytic anemia, elevated liver enzymes, low platelets
- Acute left ventricular failure with pulmonary edema
- Aspiration pneumonia
- Acute renal failure
- Microangiopathic hemolyticanemia. The patient usually has evidence of severe widespread intravascular coagulation, which may be associated with eclampsia, renal failure, hepatic failure, cerebral thrombosis and circulatory collapse.
- Cardiopulmonary arrest.

- **FETAL COMPLICATIONS**
  - Intrauterine death
  - Intrauterine growth restriction. It does not appear to be related to the actual level of blood pressure, the duration of hypertension and the presence of proteinuria being more important factors
  - Prematurity and its hazards: neonatal death, pulmonary, renal and hepatic dysfunction. Early labour may occur spontaneously, abruption, iatrogenic.
  - Antepartum and intrapartum asphyxia is more likely to be due to anoxia associated with poor placental blood flow, maternal

hypoxia induced by eclampsia or excessive therapy with sedatives and anticonvulsants.

- Neonatal encephalopathy – a systemic inflammatory response in the fetus, perhaps secondary to oxidative stress, could explain the link between maternal preeclampsia and neonatal encephalopathy, and this may occur through cerebral vasoconstriction.
- Effect of magnesium sulphate- hypermagnesaemia is seen. Lethargy, hypotonia, apnoea, respiratory depression, poor sucking reflex, decreased intestinal motility and delayed passage of meconium.

- **MANAGEMENT OF ECLAMPSIA:**
  - In a patient who is convulsing or just had a convulsion, the first priority is to avoid maternal injury and immediate attention to the airways.
  - The patient should be nursed in the left lateral position and any secretions or vomitus suctioned to avoid aspiration.
  - A padded tongue blade or airway is inserted between the teeth to avoid tongue bite and maintain airway.
  - Oxygen should be given by mask at 8-10 L/min to correct maternal and fetal hypoxia, and the oxygen saturation monitored with a transcutaneous pulse oximeter.

- Arterial blood gas analysis is indicated if the oximeter shows a saturation <92%.
- An intravenous access is secured and treatment commenced to prevent recurrent seizures and control blood pressure.
- Once the patient is a little stable and not restless, she should be catheterized with an indwelling catheter and a vaginal examination performed to access the cervical status.

- **CONTROL OF SEIZURES:**

MAGNESIUM SULPHATE - The drug of choice for the control of seizures in eclampsia is magnesium sulfate.[65] A total loading dose of 14 g is given, 4 g as intravenous infusion (20 ml of 20 % solution) over 15 to 20 minutes and 10 g deep intramuscular, 5 g in each buttock (10 ml of 50 % solution).This is followed by 5 g intramuscularly every 4 hours.

Magnesium toxicity can lead to respiratory paralysis, cardiac conduction defects and cardiac arrest. It is however rare in clinical practice provided the drug is administered with proper monitoring.The suggested therapeutic plasma level of magnesium sulfate for treatment of eclamptic convulsions is 1.8-3.0mmol/L.The first sign of impending toxicity occurs with loss of patellar reflex which corresponds to a plasma level of 3.5-5mmol/L.Therefore, repeat doses should be given only if the patellar reflex is present, respiratory rate is greater than 12 per minute and urine output has been greater than 100 ml in previous 4 hours.Routine

monitoring of serum magnesium levels is not required when these signs are monitored, although it is desirable in women with impaired renal function who are at greater risk of magnesium toxicity.If a patient develops signs of toxicity, magnesium should be immediately discontinued, oxygen should be administered and serum magnesium level obtained.10 ml of 10% calcium gluconate solution should be infused slowly over a period of 3 minutes if magnesium toxicity occurs. Other anticonvulsant drugs and regimens which were used commonly for the treatment of eclamptic convulsions before magnesium sulphate became popular include phenytoin, diazepam and the "lytic cocktail".

- **CONTROL OF BLOOD PRESSURE:**

Persistent and severe elevation in blood pressure (>= 160/110 mm Hg) should be treated to prevent cerebro-vascular accidents, pulmonary edema and renal failure. The hypertensive crisis is managed by acute-acting anti-hypertensives.

- **DELIVERY**

The definitive treatment of eclampsia is delivery, irrespective of gestational age. Once the mother is stabilized, the status of the fetus is assessed. Fetal bradycardia lasting for 3 to 5 minutes is common observation during and immediately after an eclamptic seizure and does not necessitate an emergency caesarean delivery. However fetal

bradycardia lasting for 10 minutes necessitates evaluation for uterine hyperstimulation or placental abruption. The gestational age, cervical status, fetal condition and position need to be considered before determining the most appropriate route of delivery. Vaginal delivery is safe option resulting in low maternal mortality rates as long as fetal presentation and status are appropriate and labour progresses in an orderly fashion. Cervical ripening agents can be used to improve the Bishop score. Prolonged inductions should however be avoided.

MATERIALS AND METHODS

PATIENTS AND METHODS

All the patients admitted with eclampsia at the obstetric and gynecological department of Coimbatore Medical College Hospital, Coimbatore between 1st of March 2010 to 30th of June 2011, who fulfill the inclusion criteria were included as study population.

Forms which captured the required clinical information were used. Such information used included age, parity, booking status, gestational age at presentation, time of onset of fits (antenatal, intrapartum, post natal), blood pressure at time of fits / arrival at the hospital. Biochemical parameters included platelet count, serum magnesium level. Treatment modality employed, mode of delivery, fetal morbidity and mortality, maternal morbidity and mortality were recorded.

INCLUSION CRITERIA

Patients with generalized tonic-clonic convulsions during pregnancy / labour / within 7 days of delivery were included.

EXCLUSION CRITERIA

- Previous history of epilepsy
- Previous history of neurological disorders
- Features suggestive of encephalitis/ meningitis
- Any other secondary causes underlying seizures

WORKING DEFINITIONS:

ECLAMPSIA – women with convulsions associated with signs of preeclampsia during pregnancy, labour or within 7 days of delivery, not attributed to other causes of seizures.

BOOKED PATIENTS - those who were registered at any health care providers for antenatal care and delivery (PHC/District hospitals/institutions/private hospitals).

ANTEPARTUM ECLAMPSIA- seizures occurring in preeclamptic women after 20 weeks of gestation before onset of labour.

INTRAPARTUM ECLAMPSIA - seizures occurring in pregnant women during labour.

POSTPARTUM ECLAMPSIA - seizures occurring after delivery and within seven days postpartum.

RECURRENCE OF FITS – considered when seizure occurs even after starting magnesium sulphate regimen.

BLOOD PRESSURE – taken in right arm, left lateral position, first and fifth korotkofff sound was taken as systolic and diastolic blood pressure at the time of admission.

PLATELET COUNT – normal range 1,50,000 – 3,00,000 cells/μL. overt thrombocytopenia when platelet count < 1,00,000 cells/μL.

SERUM MAGNESIUM LEVEL – sample taken on admission, before starting magnesium sulphate regimen.Normal value 1.8 – 3.0 mg/dl.

HELLP SYNDROME – hemolysis (schistocytes in blood smear, bilirubin >1.2 mg/dl), low platelet (<1 lakh) and elevated liver enzymes (AST ≥72IU/L, LDH >600 IU/L). All three parameters should be present for a diagnosis of HELLP syndrome.

PULMONARY EDEMA – diagnosis is made clinically in a woman with tachypnea, tachycardia, lung signs such as crepitation and with or without fall in saturation, in the absence of other explainable causes.

CEREBRAL HEMORRHAGE – diagnosed with CT brain imaging.

RENAL FAILURE – decreased urine output of <30 ml/hr for more than 6 hours in spite of fluid challenge if the patient was not susceptible to pulmonary edema, elevated creatinine of >1.2mg/dl.

STILL BIRTH OR FETAL DEATH – the absence of signs of life at or after birth.

EARLY NEONATAL DEATH – death of a live born neonate during the first 7 days after birth.

PERINATAL MORTALITY RATE – the number of stillbirths plus early neonatal deaths per 1000 total births.

SMALL FOR GESTATION – below 10^{th} percentile of growth for that gestational age using WHO nomogram chart.

IUGR – fetus less than 10^{th} percentile of growth for that gestational age and with corroborative signs of compromised intrauterine environment such as oligohydramnios or an elevated head-abdomen circumference ratio, clinically loss of subcutaneous fat, loose skin folds, a wasted appearance.

PROTOCOLS FOR MANAGEMENT OF ECLAMPSIA AT CMCH

- On admission air ways and intravenous lines secured
- Foley's either inserted into bladder
- Convulsions controlled with intravenous loading dose magnesium sulphate (Pritchard's regimen)
- Further convulsions after initiation of treatment controlled with an intravenous bolus of 2 grams of $MgSO_4$
- Hypertension controlled with antihypertensives, alphamethyldopa and nifidipine (through ryles tube if patient unconscious)
- Baseline investigations include complete blood cell count, serum urea, creatinine, liver function test and platelet count, serum electrolytes (including serum magnesium).
- Pregnancy was terminated irrespective of gestational age. Pelvic assessment and Bishop's scoring done. Labour accelerated if patient had spontaneous labour. Induction was given for unfavorableBishop's score. LSCS done for obstetrical indications.

- After delivery, patientskept under close monitoring and$MgSO_4$regimen should be continued for 24 hours since last seizure or delivery depending on which occurred last.
- 48 hours after last fit and diastolic BP less than 100 mm Hg patients were shifted out of ICU and monitored in postnatal ward till discharge.
- Mothers were followed up till discharge and all babies referred to the neonatal ward were followed up for 7 days to collect data on perinatal complications, early neonatal deaths and cause of neonatal deaths. Cause of maternal death was selected from the case notes as recorded by the doctor who certified the death. A post-mortem examination was not performed. The admission book was used to check if all admitted cases were included.

RESULTS AND ANALYSIS

STASTICAL ANALYSIS

All the data were analyzed with SPSS software (version 13.0). Categoricalvariables were compared by Chi square test (χ^2) or Fischer exact test and continuous variables were presented as mean ± SD and were compared by student "t" test. A probability value of <0.05 was considered statistically significant.

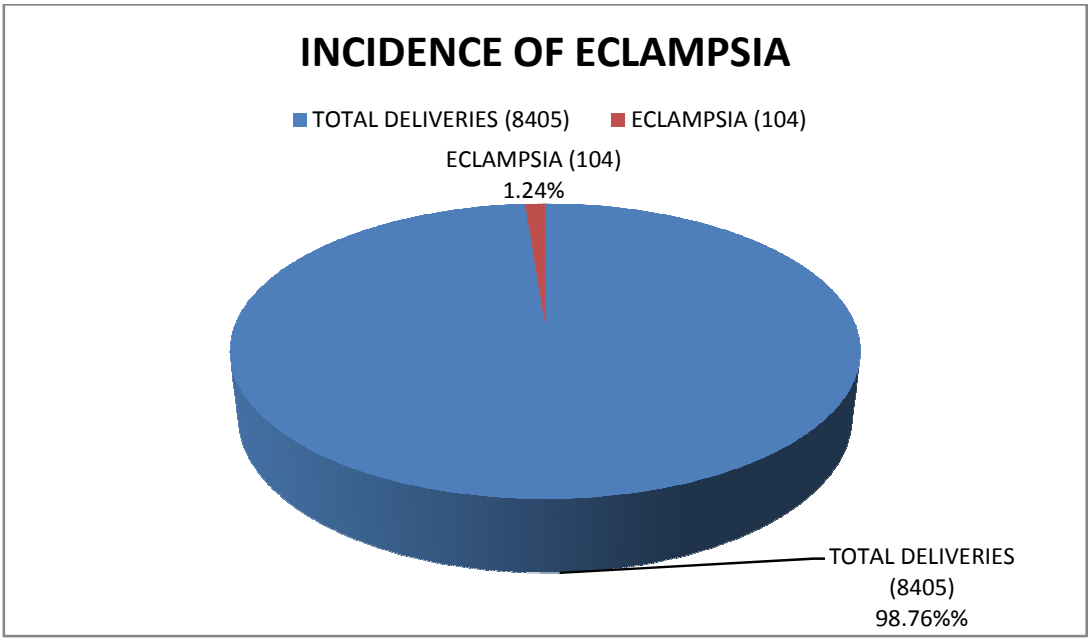
RESULTS AND ANALYSIS

INCIDENCE:

- ✓ Patients included in the study were those with the diagnosis of eclampsia, admitted in labour ward ICU between first of March 2010 and 30th of June 2011.
- ✓ There were total of**8405** deliveries during the study period.
- ✓ **104** eclamptic patients were admitted. This accounts for **124** per **10,000** deliveries
- ✓ Incidence of eclampsia at CMCH during the study period was **1.23%**.
- ✓ 101 singleton and 3 twin deliveries.

This is shown in the figure: 1

FIGURE: 1 INCIDENCE OF ECLAMPSIA



AGE:

Figure: 2show Age Distribution of Eclamptic patients.

The age range from 16 to 38 years.

58% of the patients were between 21 to 25 years, peak incidence occurred in this group.

75% of the patients were less than 25 years.

Teenage pregnancy accounts for 11%.

FIGURE: 2 AGE DISTRIBUTION

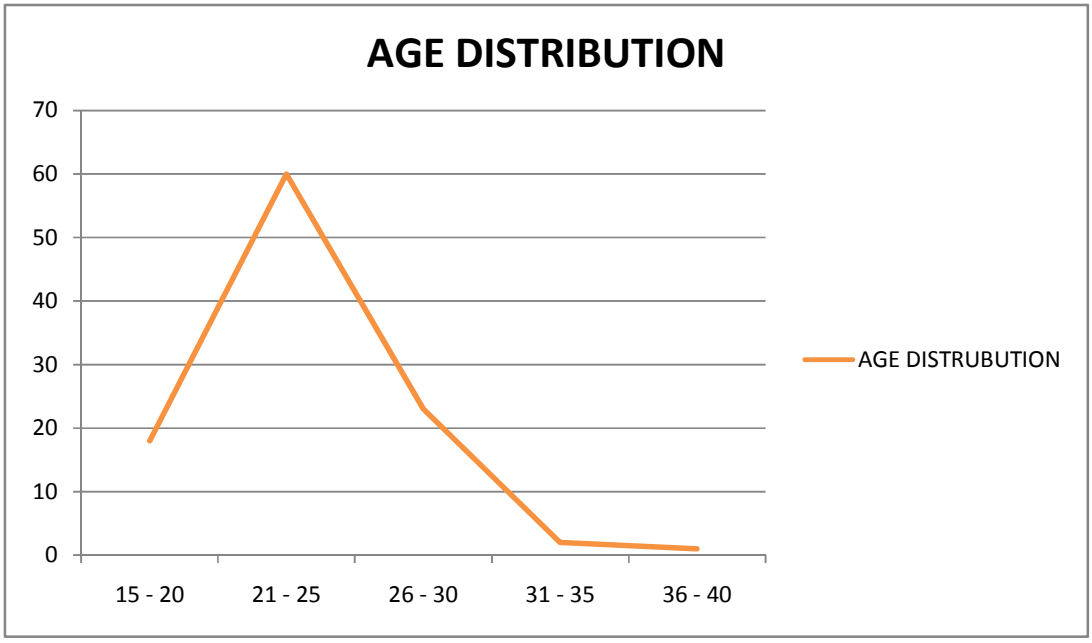


TABLE: 1 AGE DISTRIBUTION

AGE IN YEARS	NUMBERS (n=104)	PERCENTAGE (%)
15 – 20	18	17%
21 – 25	60	58%
26 – 30	23	22%
31 – 35	2	2%
36 – 40	1	1%

BOOKING STATUS

Booked patients with regular antenatal checkup(ANC) - 68

Booked patients with improper ANC follow up - 27

Unbooked patients - 9

Among those patients with intrauterine fetal demise (n=24),only 40% had regular antenatal checkup.

Perinatal complications were considerably high in those with either irregular ANC or unbooked patients constituting 55%.

27% of patients with maternal complications never been had any antenatal visits.

IMMINENT SYMPTOMS BEFORE ECLAMPSIA:

Among 104 eclamptic patients 80 of them had imminent symptoms before onset of seizures. 11 patients did not have symptoms and 13 patients could not recollect the event before seizures.

HEADACHE- The most common symptom prior to eclampsia, predominantly in occipital region.Usually occur at least 6 hours before the onset of seizures. In some it had been for 3 to 4 days. 80 women with eclampsia had headache (77%).

VOMITING – it usually follows headache rather than occurring as isolated sign of imminent eclampsia. 22 patients out of eighty women

with symptoms of imminent eclampsia had vomiting along with headache.

BLURRING OF VISION – was present in 7 patients. Of which one had retinal detachment in one quadrant and resolved in one week after delivery.

OLIGURIA – 3 patients had decreased urine output before occurrence of eclampsia. among them 2 had renal failure.

EPIGASTRIC PAIN – one of the patients had epigastric pain as an imminent symptom.

Percentage of women with imminent symptoms has been given in the table: 2.

TABLE : 2 IMMINENT SYMPTOMS OF ECLAMPSIA

IMMINENT SYMPTOMS	PERCENTAGE
HEADACHE	77%
VOMITING	21%
BLURRED VISION	7%
OLIGURIA	3%
EPIGASTRIC PAIN	0.96%

CLINICAL FINDINGS AT PRESENTATION

Clinical findings at the time on arrival at the hospital are listed in table: 3.

The majority of the patients had seizures several times before arrival at the hospital and only 34 (32.6%) of the patients arrived in the hospital within six hours of onset of convulsions. 12 patients had ≥ 5 seizures episodes, of which seven patients had been referred from places other than Coimbatore district. These patients had either no loading dose of magnesium sulphate or improper loading dose before referring. Two patients had native treatment in the form of heat application on forehead.

Ninety one patients had pedal edema (87.5%). Two patients had anemia (1.2%).

Totally 98 patients were in postictal drowsiness or confusion on admission (94.2%).

Ninety five patients had proteinuria on admission (91.3%).

TABLE : 3 CLINICAL FINDINGS

SIGNS	NO OF PATIENTS (Total patients n=104)	PERCENTAGE
Seizures ≥5 episodes	12	11.5%
Pedal edema	91	87.5%
Post ictal drowsiness/ confusion at admission	98	94.2%
Anemia	2	1.9%
Proteinuria	95	91.3%

PARITY:

As mentioned in all studies, most of them were primigravida. This accounted for 76%, whereas multigravida- 24%.

Parity distribution is illustrated in table: 4 as follows.

Among the multipara (n= 25) 15 had previous history of gestational hypertension /preeclampsia.

One had recurrence of eclampsia and 1 had chronic hypertension. One patient had history of second marriage where previous pregnancy was uneventful.

TABLE : 4 PARITY

PARITY	NUMBER (n=104)	PERCENTAGE
PRIMIGRAVIDA	79	76%
G2P1	16	15%
G3P2	5	5%
G4P3 and above	4	4%

TYPE OF ECLAMPSIA:

Classification of eclampsia is based on onset of fit relative to delivery.

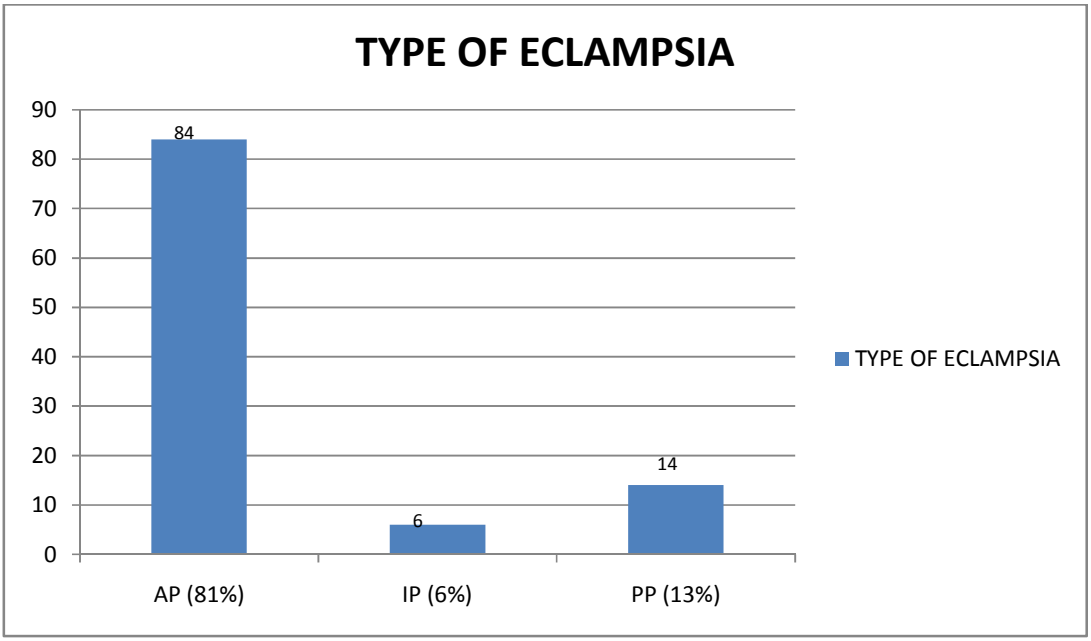
Antepartum Eclampsia contributed 81% (n = 84) of total cases.

While 6 (6%) had intrapartum and 14(13%) in postpartum period.

In this study none had seizures beyond 48 hours of delivery irrespective of mode of delivery.

Classification of eclampsia by onset of fits has been depicted in figure: 3.

FIGURE: 3 TYPE OF ECLAMPSIA



BLOOD PRESSURE:

Twenty patients had diastolic blood pressure >110 mm Hg on admission.

Nine patients had systolic blood pressure of more than or equal to 180 mm Hg.

Average diastolic pressure 103.38 ± 12.74 mm Hg

Systolic pressure on an average 156.25 ± 15.71 mm Hg

Three patients were normotensive on admission.

The diastolic pressure as a continuous variability has been put up in table: 5.

TABLE: 5 DIASTOLIC BLOOD PRESSURE AT THE TIME OF ADMISSION.

BP in mm Hg	NUMBER (104)	PERCENTAGE
≤90	8	7.69%
91 – 100	27	25.96%
101 – 110	49	47.11%
111 – 120	18	17.30%
121 – 130	2	1.91%

TABLE: 6 SYSTOLIC BLOOD PRESSURE AT THE TIME OF ADMISSION

BP in mm Hg	NUMBER (104)	PERCENTAGE
≤ 140	18	17.30%
141 – 150	25	24.03%
151 – 160	24	23.07%
161 – 170	18	17.30%
171 – 180	15	14.42%
≥ 181	4	3.84%

GESTATIONAL AGE:

Onset of eclampsia less than 24 weeks of gestation was found in three patients, of which one known to have chronic hypertension.

Eighteen (17%) patients had eclampsia at term, gestational age ≥37 weeks.

Gestational age at admission was more than 32 weeks in 75 (73%) patients.

Fifty patients had disparity in their gestational age and their clinical examination.

Gestational age of the patients on admission in weeks has been put up in table: 7.

TABLE :7 GESTATIONAL AGE DISTRIBUTION

GESTATIONAL AGE IN WEEKS	NUMBER(104)	PERCENTAGE
<24	3	3%
25-32	26	25%
33-36	57	55%
≥37	18	17%

MODE OF DELIVERY:

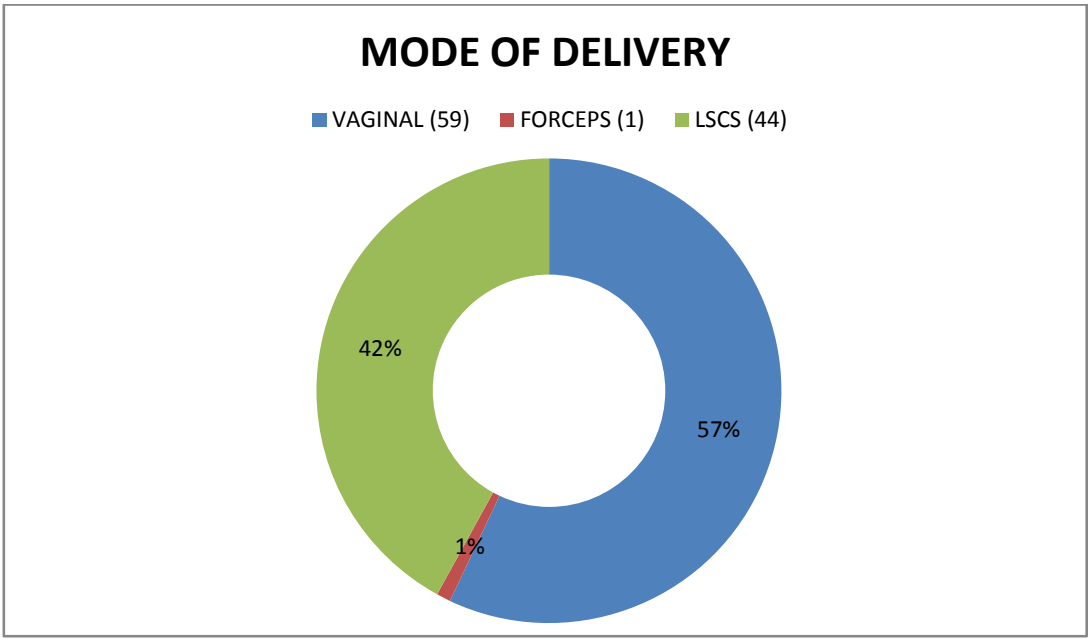
Figure: 4 project the mode of delivery in eclampsia patients.

Fifty Nine (57%) had successful vaginal delivery.

Forty four (42%) patients had Lower Segment Caesarian Section.

Indications included were failed induction (10), unfavorable cervix (16), and fetal distress (18).

FIGURE: 4 MODE OF DELIVERY



DEMOGRAPHIC CHARACTERISTICS – IN RELATION TO TYPES OF ECALMPSIA:

The demographic analysis of the study population in relation to types of eclampsia would be useful in comparing the outcome and to derive the recommendations at various level of health care providing system.

Age group distribution and occurrence of eclampsia types on analysis showed not much of significance in antepartum eclampsia. 82%(64) of women ≤25 years and 76%(20) of women >25 years had

antepartum eclampsia. 78% (11) of postpartum eclampsia had women ≤25 years.

Twenty one multipara (84%) had antepartum eclampsia against 79%(63) of primigravida had antepartum eclampsia.

Systolic blood pressure more than 160 mmHg were 37 in numbers, of which 33 had antepartum eclampsia. These features are shown in table: 8.

TABLE: 8 DEMOGRAPHIC CHARACTERISTICS – TYPE OF ECLAMPSIA

CHACTERISTICS	ANTEPARTUM	INTRAPARTUM	POSTPARTUM
Age ≤25 (n=78)	64	3	11
Age >25 (n=26)	20	3	3
Diastolic BP >110 (n=20)	19	1	NIL
Systolic BP>160 (n=37)	33	1	3
Primigravida (n=79)	63	3	13
Multigravida (n=25)	21	3	1

PLATELETS:

Fifty nine patients had thrombocytopenia, of which 30 patients had overt thrombocytopenia (<1 lakh). 45 patients had normal platelet count.

Platelet count in eclamptic patients has been shown in table: 9.

TABLE:9 PLATELET COUNT IN ECLAMPSIA.

PLATELET COUNT	NUMBERS (104)	PERCENTAGE
< 50,000	3	3%
50,000-1,00,000	27	26%
1,00,000-1,50,000	29	28%
>1,50,000	45	43%

SERUM MAGNESIUM LEVEL:

Serum magnesium level was measured in all eclamptic patients on admission.

The mean value of serum magnesium in eclamptic women studied was 1.74 ± 0.28 (mean ± SD). This was significantly lower than the normal value of 1.8 – 3.0 mg/dl, ($p < 0.05$).

Those with maternal complications the serum magnesium levels were 1.41 ± 0.15. This was statistically significant of $p < 0.001$ (t value- 10.25).

Patients without complications of eclampsia had magnesium level of 1.84 ± 0.16.

Magnesium level in those who had recurrence of seizures (47.6%) is 1.60 ± 0.12.

Serum magnesium level associated with and without complications is shown in table: 10.

TABLE: 10 SERUM MAGNESIUM LEVEL

Normal magnesium level	1.8 – 3.0 mg/dl
Average of total patients (n=104)	1.74 ± 0.28 mg/dl
With complications (n=18)	1.41 ± 0.15 mg/dl
Without complications (n=86)	1.84 ± 0.16 mg/dl
Recurrence of seizures (n=49)	1.60 ± 0.12 mg/dl

FETAL OUTCOME:

BIRTH WEIGHT:

Birth weight of all neonates irrespective of outcome was recorded. This is shown in table: 11.

Sixty One neonates were with birth weight ≤ 2kg.

Only 17 were above 2.5Kg birth weight.

One hundred and one(101) singleton and 3 twins were delivered, total of 107 babies. Three deliveries were less than 24 weeks of gestation.

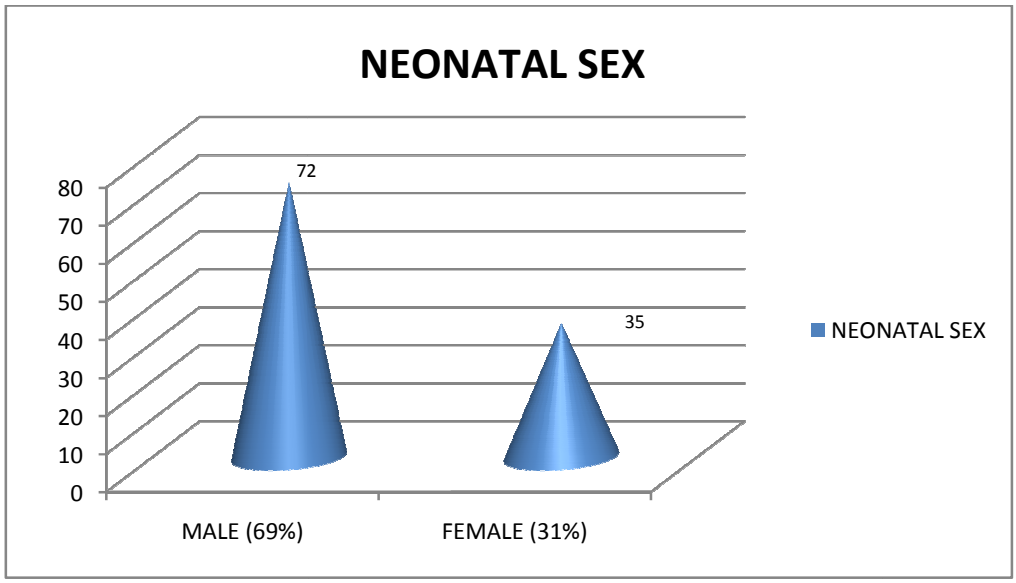
TABLE: 11 BIRTH WEIGHT

BIRTH WEIGHT IN Kg	NUMBER (n=107)	PERCENTAGE
≤1.0	9	8.41%
1.1 – 1.5	14	13.08%
1.6 – 2.0	38	35.51%
2.1 – 2.5	29	27.10%
2.6 – 3.0	17	15.88%

SEX:

As mentioned in review of literature, in this study also male sex preponderance is seen.Male baby – 72 (69%), female baby – 35 (31%). This is shown in following figure: 5.

FIGURE: 5 NEONATALSEXES



PERINATAL MORBIDITY:

There were 83 live births among 104 eclampsia patients.Neonatal wellbeing was assessed by Apgar score at 5min.40 (49%) babies had Apgar of ≥7 at five minutes.11 (12%) babies had score of <3. There were 41 NICU admissions among live births.

Thirty Nine(39) of 83 live births were small for gestation. 16 small for gestation babies were admitted in NICU.8 were preterm babies, including 3 twins. 16 babies had respiratory distress, which was settled in 24 -48 hrs.11 had jaundice, required phototherapy.6 neonates had seizures during NICU admission due to hypoxia.2 had sepsis.6 neonatal deaths were recorded.Apgar score at 5 minutes is shown in figure: 6.

FIGURE: 6 APGAR SCORE AT 5 MINUTES

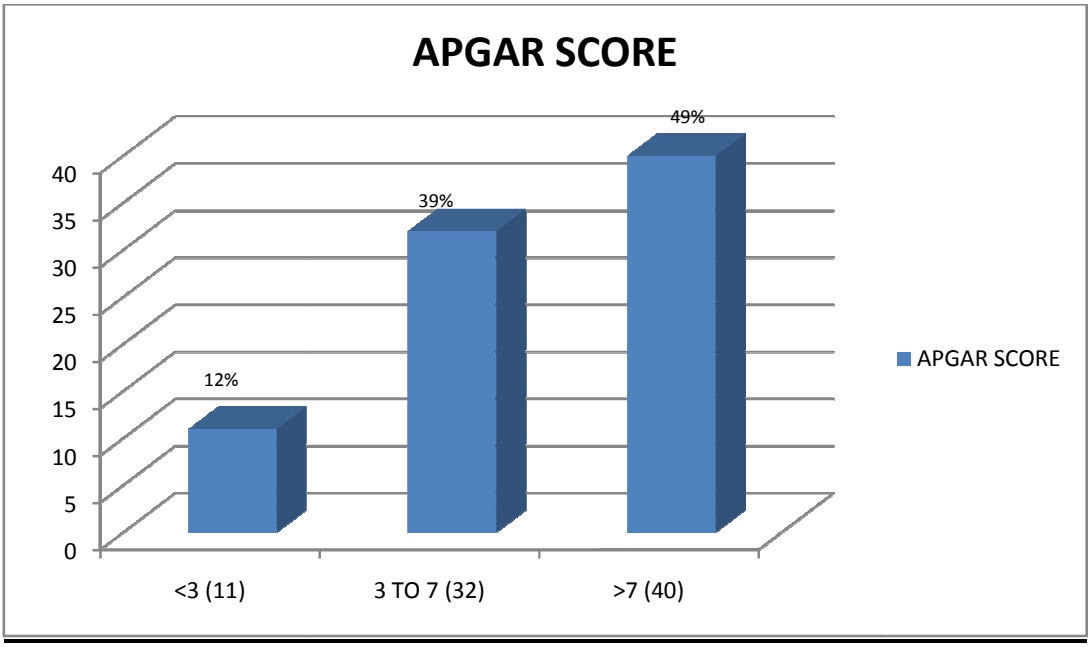


Table: 12 show the perinatal morbidity in 80 live births.

TABLE: 12 PERINATAL MORBIDITY

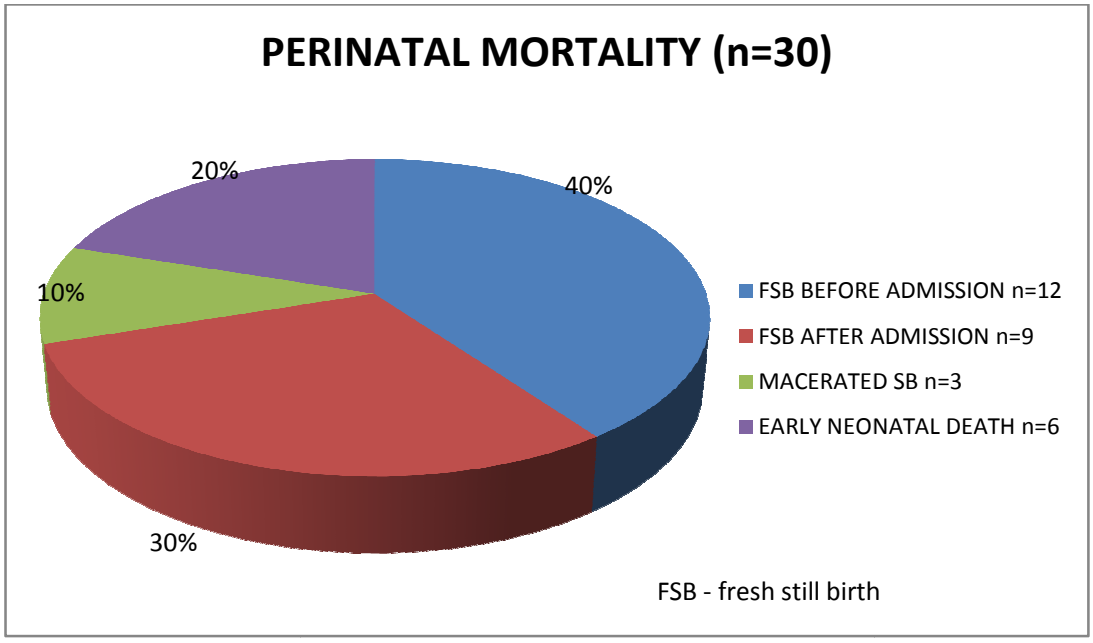
PERINATAL MORBIDITY	NUMBER (n=83)	PERCENTAGE
NICU admissions	41	49.39%
Small for gestation	39	46.98%
Preterm	8	9.63%
Jaundice	11	13.25%
Seizures	6	7.22%
Respiratory distress	16	19.27%
Sepsis	2	2.40%

PERINATAL MORTALITY:

There were 431 perinatal deaths during the study period, 30 were due to eclampsia constituting 6.96%. There were 24 still births, of which 12 were before admission and 9 after admission. 3 were macerated still birth.Six neonatal deaths were registered; all were less than 48 hrs.of admission in NICU.

Perinatal mortality rate to eclampsia was **3.56/1000** live births.Perinatal mortality was figured out in figure: 7.

FIGURE: 7 PERINATAL MORTALITY



SMALL FOR GESTATION:

Thirty nine (39) of 107 babies (36.4%) were small for gestation.

As maternal age increases number of small babies were also increased.53% of babies born to mothers of age more than 25 were small for gestation.($P<0.05$)

Longer the duration of preeclampsia as marked by proteinuria more than 4 weeks increased babies with small for gestation. 62% patients with proteinuria more than 4 weeks had small for gestation. ($P<0.01$)

Earlier the onset of preeclampsia higher the rate of small for gestation. 30 patients among small for gestation group had onset of preeclampsia between 28-30 weeks of gestation.(P<0.01)

Maternal comorbid conditions like chronic hypertension, anemia, twin pregnancy were associated with increase in small for gestation babies. Out of 26 small for gestation 21 babies were IUGR.

TABLE: 13 RISK FACTORS FOR SMALL FOR GESTATION IN ECLAMPSIA

Risk factors	Small for gestation (n=39)	Appropriate for gestation (n = 68)	P value
Age > 25 (n = 26)	14	12	<0.05
Onset of preeclampsia < 28 weeks (n = 29)	19	10	<0.01
Proteinuria > 4 weeks (n = 57)	35	22	<0.01
Maternal comorbidity (n = 16)	12	4	<0.01

NUMBER OF SEIZURES AND PERINATAL OUTCOME:

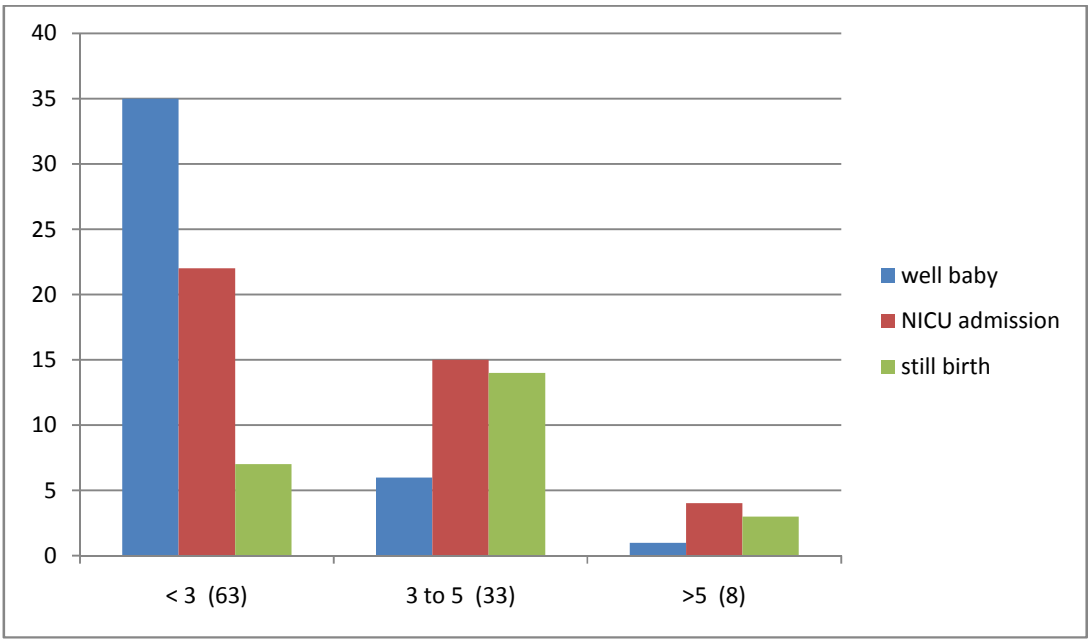
As number of seizures increased, it had impact on perinatal outcome.Total number of babies 107.63 patients had seizures less than 3. Of which 35 (54%) were well babies, 7 (11%) were stillbirth, 22 (33%) had NICU admissions.8 patients had seizures more than five episodes. Only 12% (1) was well baby, 50% (4) had NICU admissions, 38% (3) of

babies were still birth.The impact of the repeated seizures on perinatal outcome is shown in table: 14.

TABLE: 14NUMBER OF SEIZURES AND PERINATAL OUTCOME

NO OF SEIZURES	NO OF PATIENTS (n=104)	WELL BABY (n=42)	NICU ADMISSION (N=41)	STILL BIRTH (n=24)
< 3	63	35 (54%)	22 (33%)	7 (11%)
3 – 5	33	6 (16%)	15 (42%)	14 (42%)
>5	8	1 (12%)	4 (50%)	3 (38%)

FIGURE: 8 NUMBER OF SEIZURES AND PERINATAL OUTCOME



RISK FACTORS FOR PERINATAL MORTALITY:

Twenty two of 78 women with age less than 25 years had perinatal mortality which includes 18 still births and 4 early neonatal deaths.10 of 13 multipara women (79.92%) had 7 still births and 3 neonatal deaths.

Nearly 50% of those women with either irregular ANC or unbooked had perinatal mortality.Gestational age at eclampsia also had impact. 15 perinatal mortalities occured among 29 women with gestational age less than 32 weeks.These parameters has been put in table: 15.

TABLE: 15 RISK FACTORS FOR PERINATAL MORTALITY

Risk factors	Total (n)	Perinatal mortality(n=30)	Percentage	P value
Age ≤ 25	78	22	28.20 %	< 0.05
Multiparity	13	10	79.92 %	< 0.001
Irregular ANC/ Unbooked	36	18	50.00 %	< 0.01
Gestational age ≤ 32 weeks	29	15	51.72 %	< 0.01
Seizures > 5 episodes	8	3	38%	<0.05
AP eclampsia	84	23	27.38%	<0.05

MATERNAL COMPLICATIONS

Major complications of mothers with eclampsia were recorded.

Top on the list was postpartum hemorrhage (6), in spite of active management of third stage of labour. There were four (4) HELLP syndrome.Two (2) patients had renal failure, both required hemodialysis. Two patients had abruptio placenta. One (1) patient had aspiration during fits before admitting to hospital.One (1) had intracerebral hemorrhage, with admitting BP of 178/116 mm of Hg. Two patients had pulmonary edema.

Maternal complications are listed in table: 16

TABLE: 16 MATERNAL COMPLICATIONS

MATERNAL COMPLICATIONS	NUMBER (n=18)	PERCENTAGE (%)
Post PartumHemmorhage	6	5.76
HELLP syndrome	4	3.84
Renal failure	2	1.92
Abruptio placenta	2	1.92
Aspiration	1	0.96
Intracerebral hemorrhage	1	0.96
Pulmonary edema	2	1.92

MARTERNAL MORTALITY:

Total number of maternal deaths during the study period was 38. Maternal deaths due to eclampsia were four (4).10.5% of total maternal deaths were due to eclampsia.3.84% maternal mortality was seen in 104 eclampsia patients during study period.25% was due to neurological cause and 50% was due to HELLP syndrome, 25% was due to pulmonary edema.Cause of maternal mortality is shown in table: 17.

TABLE: 17 MATERNAL MORTALITY – CAUSES

CAUSES	NUMBER (n=4)	PERCENTAGE
HELLP syndrome-DIC	2	50%
Intra cerebral hemorrhage	1	25%
Pulmonary edema	1	25%

RISK FACTORS ANALYSIS OF MATERNAL OUTCOME:

Age less than 25 – among 26 eclampsia patients 16 were associated with maternal complications. This constituted 61.5% ($p < 0.001$) of total women less than 25 years.

Primigravida as already proven, has strong correlation with eclampsia but only 12 of 91 primigravida had complications. This did not have statistical significance.

On the other hand multigravida had higher proportion of maternal complications. 6 out of 13 multigravida patients had maternal complications, contributing 46.15% with p value <0.01.

Unbooked and patients with irregular antenatal checkup had more maternal complications.Recurrence of seizures ≥ 5 episodes had higher complications than with less than five episodes (33.33% Vs 15.21% p<0.01).

Admission systolic blood pressure more than 160 mm Hg had significance in maternal complications. 13 among 35 patients with systolic blood pressure >160 mm Hg had complications, contributing 37.14%.

On the other side even with diastolic blood pressure of >110 mm Hg did not have significant impact on maternal complications. (4 of 20 patients, 20.06%).

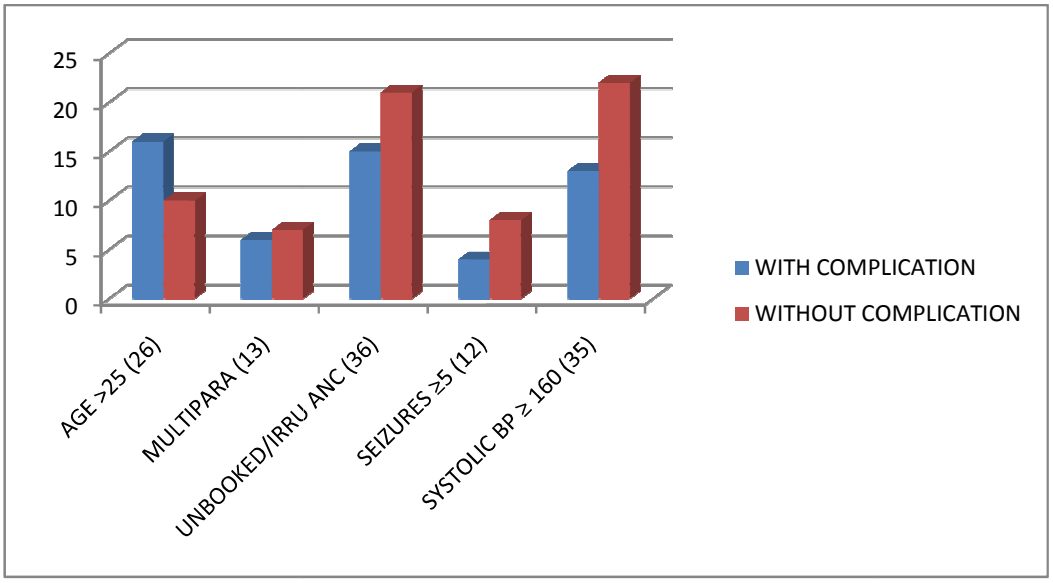
Influence of various factors over maternal complications is enlisted in table: 18

TABLE: 18 RISKFACTORS FOR MATERNAL COMPLICATIONS

RISKFACTORS	MATERNAL COMPLICATIONS	PERCENTAGE	P VALUE
Age > 25 (n=26)	16	61.5%	<0.001
Multipara (n=13)	6	46.15%	<0.01
Primipara (n=91)	12	13.18%	NS*
Unbooked/ Irregular ANC (n=36)	15	41.66%	<0.01
Recurrence of Seizure ≥5 (n=12)	4	33.33%	<0.01
Systolic BP ≥ 160 mm hg (n=35)	13	37.14%	<0.01
Diastolic BP > 110 mm hg (n=20)	4	20.06%	NS*

*NS – NOT SIGNIFICANT

FIGURE: 9 RISKFACTORS FOR MATERNAL COMPLICATIONS



PLATELET COUNT AND MATERNAL COMPLICATIONS:

Platelet count had impact on maternal outcome in eclampsia.14 of 30 women with platelet count less than 1,00,000 had maternal complications. Constituting 46.66%, p value <0.01.Only 2 of 45 women with platelet count more than 1,50,000 had complications.This is shown in table: 19.

TABLE: 19 PLATELET AND MATERNAL COMPLICATIONS

PLATELET COUNT	TOTAL (n=104)	NO OF MATERNAL COMPLICATIONS (n=18)	PERCENTAGE (%)	P VALUE
< 1,00,000	30	14	46.66%	< 0.01
1,00,000-1,50,000	29	2	6.89%	< 0.01
> 1,50,000	45	2	4.44%	< 0.01

SERUM MAGENESIUM LEVEL AND MATERNAL COMPLICATIONS:

Normal serum magnesium level as already mention is taken as 1.8 – 3 mg/dl.Lower the magnesium level more the maternal complications.13 of 30 (43.33%) women with serum magnesium level 1.00-1.59 mg/dl had complications, with p value less than 0.01. But only 3 of 63 of women with serum magnesium level 1.60-2.09 mg/dl had complications.When magnesium level was 2.10 – 2.50 mg/dl two patients had renal failure hence elevated serum magnesium due to impaired excretion. The relation with serum magnesium level is shown in table: 20.

TABLE: 20 SERUM MAGNESIUM AND MATERNAL COMPLICATIONS

SERUM MAGNESIUM (mg/dl)	TOTAL n=104	MATERNAL COMPLICATIONS		PERCENTAGE	P VALUE
		WITH (n=18)	WITHOUT (n=96)		
1.00 – 1.59	30	13	27	43.33%	< 0.01
1.60 – 2.09	63	3	60	4.76%	< 0.01
2.10 – 2.50	11	2	9	18.18%	< 0.05

TYPE OF ECLAMPSIA –PERINATAL ANDMATERNAL OUTCOME:

Most common type was antepartum eclampsia, contributing 81% of total eclampsia patients. Among 24 still births 23 occurred in antepartum eclampsia and 1 in postpartum eclampsia (95.83% vs 4.16%). 34 of 41 NICU admissions were due antepartum eclampsia compared to 3 and 4 admissions in intrapartum and postpartum eclampsia respectively . But intrapartum eclampsia had higher proportion of NICU admissions than antepartum and postpartum eclampsia (50.00% vs 40.47% and 28.57%). 82.92% of NICU admissions were from antepartum group.There were no maternal mortality or morbidity in intrapartum eclampsia group. All four maternal mortality were in antepartum eclampsia group.16 maternal complications in antepartum eclampsia and 2 in postpartum eclampsia group (19.04% vs 14.28%). 88.88% of maternal complications occurred in antepartum eclampsia.The above data is shown in following table: 21.

TABLE: 21 TYPE OF ECLAMPSIA – PERINATAL AND MATERNAL OUTCOME

	ANTEPARTUM		INTRAPARTUM		POSTPARTUM	
Stillbirth(n=24)	23	95.83%	NIL	NIL	1	4.16%
Nicu admissions(n=41)	34	82.92%	3	7.31 %	4	9.75%
Maternal complications(n=18)	16	88.88%	NIL	NIL	2	11.11%
Maternal mortality(n=4)	4	100%	NIL	NIL	NIL	NIL

DISCUSSION

Incidence of eclampsia:

In our study over a period of 16 months 104 patients of eclampsia had been studied, with the incidence of 1.24%. Incidence in India varies between 0.5% and 1.8% [66].This incidence is similar to other African countries, 1.32%[67]. But still far behind the developed countries whose incidence is 8.4/10,000 deliveries[68].

Clinical profile:

Eclampsia occurred in both extremes of age group in reproductive women. In our studyage group between 21 and 25 years constitute the maximum percentage of eclampsia(58%) similar to other studies[69].

The incidence of eclampsia is higher than figures from developed countries where there is better compliance with antenatal clinic attendances and easier access to specialist care, this is further demonstrated by the finding that only 65.38% of eclampsia patients had regular antenatal attendances in our study.

The significant difference observed in the incidence of eclampsia amongst the very young and the primigravida (76%) compared to multigravida is in keeping with the findings of other studies[70].

Antepartum eclampsia accounted for the majority (81%) of the cases in our study. This was the finding in some earlier works[71]. But

study from UK showed relatively higher proportion of post partum eclampsia (44%)[72] which was explained due to better antenatal care.

The need for vigilance and close monitoring of patients in the immediate postpartum period especially those with features of preeclampsia is highlighted by the fact that 88% of first convulsions in postpartum period occurred within 12 hours of delivery[73].

Premonitory imminent symptoms were present in variable duration before seizures. 80 patients had imminent symptoms. Most common was headache, which was there in all patients who had imminent symptoms.

Regarding gestational age maximum distribution was between 32 – 37 weeks (57%), comparable to other studies[74]. 17% of women presented at term, ≥ 37 weeks.

Overt Thrombocytopenia (< 1 lakh) seen in 29% eclamptic women in our study

Serum magnesium levels of eclamptic patients were significantly lower than normal value (1.74 ± 0.28 mg/dl vs 1.8 – 3.0).

Although the definitive management of eclampsia is delivery of the fetus and placenta as early as possible but as the eclampsia patient goes into labour quickly most of the patients delivered vaginally (57% vs 42% LSCS) consistent with other study from Pakistan[74]. Demographic picture is compared in table: 22 with study from Orrisa[75] and Canada[76].

TABLE: 22 DEMOGRAPHIC COMPARISION

CHARACTERS	OUR STUDY - 2011	Singh S et al (ORRISA-2011)	CANADIAN STUDY -2011
Incidence	124/10000	320/10000	8.4/1000
Unbooked	28.84%	97.4%	-
Age <25	69%	46%	30.5%
AP eclampsia	81%	81.64%	70%
Primiparity	76%	73%	54.96%

Perinatal morbidity and mortality:

Perinatal mortality in our study is 3.56/1000 total births.6.96% of perinatal deaths were due to eclampsia.This is comparable to studies from India (30 – 40 %) [77] and neighbor countries like Nepal and Bangladesh[78].

Antepartum eclampsia was strongly associated with preterm eclampsia and hence is also associated with increased risk of perinatal complications and small for gestation.

As the number of fits increased rate of NICU admission increased. 50% NICU admissions and 38% mortality when number of seizures were more than 5.

Age ≤ 25 years, early onset preeclampsia, proteinuria more than four weeks duration and maternal comorbidity were significantly associated with small for gestation ($p < 0.01$).

Perinatal mortality was significantly associated with multiparity (79.92% vs 20.08% $p<0.001$).

Unbooked and patients with defaulting ANC care had 50% perinatal mortality ($p<0.01$).

Gestational age less than 32 weeks had higher perinatal mortality ($p<0.01$). This is due an obvious reason of prematurity.

Perinatal complications included NICU admissions, prematurity, birth asphyxia, seizures, respiratory distress, IUGR and sepsis.

Late arrival of patients after onset of fits and recurrence result in severe intrauterine hypoxia and intrauterine death. Eclampsia occurring preterm necessitates preterm delivery. Available neonatal care facilities also determine the perinatal outcome.

Basic characteristics and perinatal outcome is compared between Indian study from Karnataka[79] and Canadian study[76] in table: 23.

TABLE: 23PERINATAL OUTCOME COMPARISION

	OUR STUDY - 2011	RAJSRI et al – (KARNATAKA 2011)[79]	CANADIAN STUDY – (2011)[76]
NICU admissions	51.25%	47.88%	24.5%
SGA*	37.5%	-	20.6%
Seizure	7.5%	10%	0.3%
RDS†	20%	13%	0.7%
Sepsis	2.5%	4%	0.24%
Jaundice	13.5%	22%	-
Stillbirths	23%	20%	0.18%
Preterm	10%	31%	17%
Perinatal death	3.56/1000	3.50/1000	-

*SGA – small for gestation.

†RDS – respiratory distress syndrome.

Maternal outcome:

In our study 17.30% (n=18) of eclampsia patients had complications.

Eclampsia continues to be associated with significant maternal morbidity and mortality.

Leading cause of maternal complications in our study was postpartum hemorrhage(33% of complications contributed by PPH)

similar to other study[74]. But in some studies pulmonary causes were leading morbidity,Renal failure, abruption, HELLP syndrome, rupture uterus, aspiration, intracerebral hemorrhage were the other causes of mortality.

As in other studies patient who suffered intracranial hemorrhage did not survive.

When age and parity increased the outcome of eclamptic mothers was bad.

Thirty six (n=36) 41.6% of unbooked and patients with irregular ANC had complications.

Twenty nine, n= 29 (28%) women had blood pressure more than or equal to 160/110 mmHg at admission. This group of patients developed most complications[80].

There were 4 maternal deaths during our study period. Constituting 3.8% of case fatality rate and contributing to 10.5% of total maternal deaths during the same period in our institution.

Maternal deaths were high among those who experienced antepartum eclampsia compared to those who had intrapartum or postpartum eclampsia. This could be partly explained by the relatively longer duration or possibly repeated episodes of convulsions from the onset of the first fit that often increases the mortality.

Maternal outcome is compared in table: 24. This is done with an Indian[75] and Nigerian study[81].

TABLE: 24 COMPARISION OF MATERNAL OUTCOME.

	OUR STUDY - 2011	Singh S et al (ORRISA-2011)	NIGERIAN STUDY -2010
Case fatality rate	3.8%	4.4%	8.5%
Maternal mortality ratio	8%	10.44%	12%
Maternal complications	17.3%	26.58%	30%
Pulmonary edema	25%	71%	-
Acute renal failure	NIL	14.2%	-
Cerebral cause	25%	14.2%	-
Hellp-DIC	50%	NIL	-

Last four rows indicate causes for maternal deaths.

Platelet and serum magnesium in maternal outcome:

Thrombocytopenia is associated with preeclampsia and eclampsia. As the platelet count dropped below 1 lakh (overt thrombocytopenia) the maternal complications increased. 46.66% had maternal complications compared to platelets more than 1,50,000 were only 2 of 45 patients had complications[81].

Serum magnesium is low during pregnancy but reduces further during preeclampsia[82]. In our study the serum magnesium level was significantly low1.74 ± 0.28 mg/dl ($p<0.01$).

Furthermore low level of magnesium was found in patients with maternal complications than without complications (1.41 ±0.15 vs 1.84 ± 0.16 p value <0.001).But 18.18%(n=11)patients(including 2 patients with renal failure) had magnesium values between 2.10 - 2.50mg/dl which may be due to renal dysfunction as explained by ShaliniM et al.,[83].

CONCLUSION

- To reduce the incidence andcomplications of eclampsia, there is dire need to improve antenatal care at community level.This includes booking of all antenatal mothers,identify the high risk mothers and ensureproper antenatal care with regular BP monitoring, which will result in early diagnosis of gestational hypertension. Health care providers and pregnant women with high BP should be sensitized for the imminent symptoms and early referral if it is present.
- Multiparity is associated with adverse maternal and perinatal outcome hence it advisable to limit the family size.
- Eclamptic patients should be given full loading dose before referring them to tertiary care to avoid recurrence of seizures and its hypoxic injury to both mother and fetus.
- Vigilant postpartum care is required since postpartum eclampsia is common in first 12 hours of delivery and PPH is the most common maternal complication.
- Early diagnosis and treatment of preeclampsia, appropriate measures to prevent recurrence of seizures and prompt referral to centre with newborn care facilities will reduce the perinatal mortality due to eclampsia.
- Low platelet and low serum magnesium level were associated with adverse maternal outcome in eclamptic patients. Further studies are needed to validate the low serum magnesium level as predictor of maternal complications in eclampsia.

S.No	NAME	AGE	IP NUMBER	ADRESS	PARITY INDEX	BOOKING STATUS	LMP AGE	PA AGE	DIFF	SYSTOLIC BP
1	SATHYA	16	52572	gobi	primi	IRRU ANC	28	28	_	180
2	RUBIDEVI	17	9518	MADUKARAI CBE	primi	REG ANC	36	36	_	152
3	BINU	17	34897	CBE	primi	REG ANC	32	30	2WKS	160
4	SUGANTHY	18	69989	SALEM	primi	REG ANC	28	28	_	140
5	RABIYA	18	69986	CBE	primi	REG ANC	36	36	_	152
6	MOHANA	19	58016	AVINASHI	primi	IRRU ANC	34	28	6WKS	160
7	SANGEETHA	19	59011	SATHY	primi	REG ANC	36	32	4WKS	170
8	SUGANYA	19	71348	TIRUPUR	primi	REG ANC	30	26	4WKS	162
9	SUGANYA	19	71345	PALLADUM	primi	REG ANC	40	36	4WKS	160
10	PUSHPALATHA	19	70824	UDUMALAI	G2A1	UB	34	34	_	170
11	GEETHA	19	15147	gobi	primi	IRRU ANC	22	22	_	154
12	poornima	20	9436	cbe	primi	REG ANC	38	34	4WKS	150
13	RANJITHA	20	30399	CBE	primi	REG ANC	37	32	5WKS	170
14	RABIYA	20	55317	CBE	primi	REG ANC	38	38	_	150
15	ANDICHI	20	24000	UDUMALAI	primi	REG ANC	34	34	_	114
16	SARATHY	20	6780	CBE	primi	REG ANC	36	32	4WKS	170
17	SUDHA	20	2724	METTUPALAYAM	primi	REG ANC	40	36	4WKS	140
18	SARASU	20	74565	CBE	primi	IRRU ANC	34	32	2WKS	172
19	DEVI	21	111545	SULUR CBE	PRIMI	REG ANC	38	38	_	150
20	BAGAVATHY	21	24470	ANAIMALAI	PRIMI	REG ANC	32	32	_	140
21	UMADEVI	21	39700	UDUMALAI	primi	REG ANC	36	32	4WKS	150
22	SARADHA	21	34918	SATHY	primi	IRRU ANC	36	30	6WKS	174
23	MEENA	21	11324	CBE	primi	REG ANC	36	36	_	160
24	MUMTAJ	21	14332	ERODE	primi	IRRU ANC	32	28	4WKS	170
25	KAMATCHI	21	26544	ERODE	primi	UB	36	36	_	174
26	JOTHIMANI	21	31241	CBE	primi	IRRU ANC	38	34	4WKS	190
27	JANAKI	21	32215	CBE	primi	REG ANC	34	34	_	130
28	KRITHIGA	21	45413	CBE	primi	REG ANC	36	36	_	160
29	ISWARYA	21	56732	CBE	primi	REG ANC	36	36	_	162
30	mallika	22	9941	gobi	primi	IRRU ANC	36	30	6wks	140
31	GAYATHRI	22	9272	CBE	PRIMI	IRRU ANC	37	30	7WKS	150
32	VENI	22	24582	UDUMALAI	G2P1L1	IRRU ANC	39	36	3WKS	152
33	SIVAGAMI	22	54780	SATHY	primi	IRRU ANC	28	28	_	160
34	SUBULAKSHMI	22	64908	ERODE	primi	IRRU ANC	28	28	_	148
35	JAYANTHI	22	2661	AVINASHI	primi	REG ANC	22	22	_	150

S.No	NAME	AGE	IP NUMBER	ADRESS	PARITY INDEX	BOOKING STATUS	LMP AGE	PA AGE	DIFF	SYSTOLIC BP
36	VANITHA	22	34545	CBE	primi	REG ANC	36	30	6wks	178
37	KALAI	22	67578	CBE	primi	REG ANC	36	32	4WKS	160
38	AMUTHA	22	34567	CBE	primi	REG ANC	36	36	_	154
39	CHITRA	23	10987	CBE	PRIMI	REG ANC	38	36	2WKS	160
40	RANI	23	39763	POLLACHI	primi	REG ANC	36	36	_	180
41	HEMAVATHY	23	36777	CBE	G2P1L1	REG ANC	34	28	6WKS	124
42	DEVI	23	32425	CBE	primi	REG ANC	36	36	_	160
43	KAMALEESHWARI	23	34528	KOTHAGIRI	primi	IRRU ANC	36	30	6WKS	162
44	RANGANAYAKI	23	34568	PEELAMEU CBE	primi	REG ANC	38	38	_	130
45	JOTHI	23	11562	CBE	primi	REG ANC	36	36	_	140
46	NAGAMANI	23	12358	CBE	primi	REG ANC	28	26	2WKS	178
47	VAITHESHWARI	23	21981	gobi	primi	REG ANC	38	38	_	160
48	muthuselvi	23	26586	dharapuram	primi	UB	38	38	_	170
49	GEETHA	23	3425	KOTHAGIRI	primi	IRRU ANC	32	26	6wks	178
50	PANDEESHWARI	23	62630	SULUR CBE	primi	REG ANC	36	34	2WKS	150
51	PANDISELVI	23	33422	CBE	primi	IRRU ANC	36	30	6WKS	178
52	CHITRA	23	56458	CBE	primi	REG ANC	36	36	_	150
53	JAYALAKSHMI	23	34289	TIRUPUR	G2P1L1	IRRU ANC	36	30	6wks	180
54	muthuselvi	23	676899	CBE	primi	REG ANC	36	36	_	172
55	HEMAPRIYA	23	3556	CBE	primi	REG ANC	34	34	_	140
56	MANI	23	54672	CBE	primi	REG ANC	34	34	_	130
57	PRIYA	24	114435	OOTY	G4P1L1A2	REG ANC	36	36	_	142
58	NASERA	24	112323	UDUMALAI	PRIMI	REG ANC	38	38	_	180
59	PREMA	24	52686	ERODE	primi	IRRU ANC	38	34	4WKS	150
60	SARASWATHY	24	43678	CBE	G2A1	REG ANC	28	28	_	152
61	SATHYA	24	61871	TIRUPUR	primi	REG ANC	28	28	_	150
62	AMUTHA	24	60714	OOTY	G2A1	IRRU ANC	36	30	6WKS	190
63	THERAISA	24	45644	METTUPALAYAM	G2P1L1	IRRU ANC	36	36	_	132
64	MEENA	24	21298	UDUMALAI	primi	REG ANC	32	32	_	170
65	FARITHA BANU	24	31562	CBE	primi	REG ANC	36	36	_	176
66	ANDICHI	24	65867	gobi	primi	REG ANC	32	32	_	140
67	ARIFA	24	31657	TIRUPUR	primi	REG ANC	36	36	_	170
68	VALLI	24	65432	CBE	primi	REG ANC	36	36	_	148
69	MANIMEGALAI	24	55655	TIRUPUR	primi	IRRU ANC	34	28	6wks	160
70	CHINNU	24	43433	CBE	G2A1	REG ANC	32	32	_	150

S.No	NAME	AGE	IP NUMBER	ADRESS	PARITY INDEX	BOOKING STATUS	LMP AGE	PA AGE	DIFF	SYSTOLIC BP
71	SELVI	25	71021	CBE	G2A1	UB	22	22	_	148
72	mallika	25	19887	POLLACHI	primi	REG ANC	36	30	6wks	160
73	KAVITHA	25	21307	TIRUPUR	G2A1	IRRU ANC	28	26	2WKS	182
74	CHITRA DEVI	25	3979	TIRUPUR	primi	UB	36	32	4WKS	150
75	ESWARI	25	43531	TIRUPUR	primi	REG ANC	36	34	2WKS	140
76	DEVI	25	7895	ERODE	primi	REG ANC	36	32	4WKS	168
77	SEETHALAKSHMI	25	10453	OOTY	primi	IRRU ANC	34	26	8W	160
78	MEENA	25	43244	TIRUPUR	primi	REG ANC	36	36	_	140
79	SIVAGAMI	**26**	60266	TIRUPUR	primi	REG ANC	28	28	_	164
80	KATHAYEE	26	40622	CBE	primi	IRRU ANC	36	30	6wks	160
81	dhanalakshmi	26	65075	PERUR CBE	G2A1	REG ANC	38	34	4WKS	140
82	SAJITHA	26	48289	CBE	primi	REG ANC	36	36	_	158
83	VASANTHA	27	10161	TIRUPUR	primi	IRRU ANC	36	32	4WKS	170
84	CHITRA	27	23371	KUNIYAMUTHR CBE	primi	REG ANC	32	32	_	162
85	SANUMA	27	37999	CBE	primi	REG ANC	34	32	2WKS	136
86	LATHAMANI	28	109877	SIRUMUGAI CBE	G2P1L1	REG ANC	28	32	4WKS	150
87	MAHESHWARI	28	23029	CBE	primi	IRRU ANC	38	32	6WKS	170
88	SUMATHY	28	6173	AVINASHI	G3P2L1	REG ANC	28	28	_	172
89	KARTHIGA DEVI	28	5007	TIRUPUR	primi	UB	32	30	2WKS	160
90	RANJITHA	28	45432	CBE	G2A1	REG ANC	36	32	4WKS	160
91	SACHU	28	32436	CBE	primi	REG ANC	34	34	_	148
92	dhanalakshmi	29	11144	arulpuram cbe	primi	IRRU ANC	36	32	4WKS	150
93	RAMALAKSHMI	30	58257	VALPARAI	G3P2L1	REG ANC	28	28	_	200
94	SELVI	30	58023	TIRUPUR	G3P1L1A1	UB	32	32	_	156
95	KASTHURI	30	49289	ERODE	G2P1L1	IRRU ANC	38	32	6wks	140
96	MYLAL	30	70144	ANNAMALAI	primi	UB	36	32	4WKS	164
97	ANANDHI	30	14745	TIRUPUR	G2P1L1 TWIN	REG ANC	34	34	_	142
98	SELVI	30	30053	MADUKARAI CBE	G2P1L0	REG ANC	34	34	_	150
99	SARANYA	30	50032	MADUKARAI CBE	primi	REG ANC	34	34	_	150
100	GOWRI	30	34345	CBE	G3P1L1A1	REG ANC	32	30	2WKS	144
101	VENILLA	30	34564	CBE	G4P1L1A2	REG ANC	34	30	4WKS	150
102	ESWARI	32	1940	CBE	G3P2L2	REG ANC	36	36	_	150
103	PREMA	35	34770	OOTY	G10P9L8	UB	26	20	6wks	164
104	PARIMALA	38	71101	MADUKARAI CBE	G5P3L3A1	REG ANC	28	28	_	180

S.No	NAME	AGE	IP NUMBER	diastolic BP	TYPE	TIMES	MODE	WT IN KG	PLAT	MAGNESIUM LEVEL	BABY
1	SATHYA	16	52572	132	AP	1	LSCS	1	87000	1.94	IUD
2	RUBIDEVI	17	9518	110	AP	4	LSCS	2.7	180000	1.53	NICU
3	BINU	17	34897	102	AP	3	VAGINAL	2.1	280000	2.1	NICU
4	SUGANTHY	18	69989	90	AP	1	VAGINAL	1.1	207000	2.3	NICU
5	RABIYA	18	69986	104	AP	1	LSCS	2.5	264000	2.5	WB
6	MOHANA	19	58016	100	PP	2+1	VAGINAL	2	120000	1.43	IUD
7	SANGEETHA	19	59011	102	PP	2	LSCS	2.2	290000	2.12	WB
8	SUGANYA	19	71348	100	AP	2	VAGINAL	1	200000	1.94	NICU
9	SUGANYA	19	71345	104	AP	1	LSCS	2.5	210000	1.89	WB
10	PUSHPALATHA	19	70824	110	AP	5	VAGINAL	1	74000	1.33	NICU
11	GEETHA	19	15147	100	AP	3	VAGINAL	0.6	250000	1.45	IUD
12	poornima	20	9436	100	AP	1	VAGINAL	2.1	30000	1.35	IUD
13	RANJITHA	20	30399	112	AP	3	VAGINAL	3	180000	1.92	NICU
14	RABIYA	20	55317	110	AP	7	LSCS	2.5	300000	1.35	WB
15	ANDICHI	20	24000	72	AP	3	LSCS	1.75	180000	1.36	NICU
16	SARATHY	20	6780	110	AP	3	LSCS	1.9	100000	2.34	NICU
17	SUDHA	20	2724	114	AP	1	VAGINAL	2	230000	2.19	NICU
18	SARASU	20	74565	110	AP	3	VAGINAL	2	100000	1.33	NICU
19	DEVI	21	111545	110	AP	2	LSCS	2.6	200000	1.67	WB
20	BAGAVATHY	21	24470	112	AP	4	LSCS	2.1	110000	1.89	IUD
21	UMADEVI	21	39700	100	AP	4	VAGINAL	2	100000	1.88	IUD
22	SARADHA	21	34918	110	AP	3	VAGINAL	1.3	140000	1.93	WB
23	MEENA	21	11324	110	PP	2	LSCS	2.3	120000	1.99	WB
24	MUMTAJ	21	14332	106	AP	3	VAGINAL	1.4	40000	1.4	IUD
25	KAMATCHI	21	26544	110	PP	1	LSCS	2.5	122000	1.25	WB
26	JOTHIMANI	21	31241	90	AP	2	VAGINAL	2	111000	2.33	WB
27	JANAKI	21	32215	90	AP	2	VAGINAL	2	120000	1.98	NICU
28	KRITHIGA	21	45413	102	PP	1	VAGINAL	2.8	200000	2.13	WB
29	ISWARYA	21	56732	114	AP	1	VAGINAL	2.7	340000	1.89	WB
30	mallika	22	9941	110	AP	5	VAGINAL	1.9	150000	1.46	WB
31	GAYATHRI	22	9272	116	AP	1	VAGINAL	1.8	120000	2.34	WB
32	VENI	22	24582	100	AP	2	LSCS	2.3	120000	1.76	NICU
33	SIVAGAMI	22	54780	136	AP	2	VAGINAL	1.3	290000	1.66	NICU
34	SUBULAKSHMI	22	64908	90	IP	2	VAGINAL	1.5	100000	2.45	WB
35	JAYANTHI	22	2661	100	AP	3	VAGINAL	0.5	42,000	1.96	IUD

S.No	NAME	AGE	IP NUMBER	diastolic BP	TYPE	TIMES	MODE	WT IN KG	PLAT	MAGNESIUM LEVEL	BABY
36	VANITHA	22	34545	110	AP	4	LSCS	2	110000	1.88	NICU
37	KALAI	22	67578	110	AP	2	VAGINAL	2.3	240000	1.76	WB
38	AMUTHA	22	34567	112	AP	2	VAGINAL	2.4	130000	1.98	WB
39	CHITRA	23	10987	100	PP	1	VAGINAL	2.1	110000	1.67	WB
40	RANI	23	39763	108	AP	3	LSCS	1.7	100000	1.45	IUD
41	HEMAVATHY	23	36777	92	AP	1	LSCS	1.80	100000	1.33	IUD
42	DEVI	23	32425	120	AP	2	LSCS	2.4	230000	1.81	WB
43	KAMALEESHWARI	23	34528	110	AP	12	LSCS	1.4	80000	1.23	IUD
44	RANGANAYAKI	23	34568	84	AP	4	LSCS	2.8	240000	1.91	WB
45	JOTHI	23	11562	100	IP	1	OUTLET	3	200000	1.95	WB
46	NAGAMANI	23	12358	116	AP	5	VAGINAL	0.6	55000	1.35	IUD
47	VAITHESHWARI	23	21981	110	AP	3	LSCS	2.7	65000	1.84	WB
48	muthuselvi	23	26586	102	PP	5+1	LSCS	3	154000	1.32	NICU
49	GEETHA	23	3425	120	AP	5	VAGINAL	0.7	374000	1.56	IUD
50	PANDEESHWARI	23	62630	110	PP	2	VAGINAL	2	230000	1.82	WB
51	PANDISELVI	23	33422	100	AP	2	VAGINAL	2	120000	1.87	NICU
52	CHITRA	23	56458	100	PP	1	VAGINAL	2.6	250000	1.86	WB
53	JAYALAKSHMI	23	34289	110	IP	4+1	LSCS	2.1	88,000	1.89	NICU
54	muthuselvi	23	676899	106	AP	1	LSCS	2.4	200000	1.92	WB
55	HEMAPRIYA	23	3556	110	AP	1	LSCS	2.4	120000	1.94	WB
56	MANI	23	54672	90	AP	1	VAGINAL	2.3	270000	1.96	WB
57	PRIYA	24	114435	110	AP	1	VAGINAL	2.3	100000	1.74	WB
58	NASERA	24	112323	106	AP	2	LSCS	2.7	180000	1.72	WB
59	PREMA	24	52686	110	AP	4	LSCS	1.5	100000	1.35	IUD
60	SARASWATHY	24	43678	110	AP	2	VAGINAL	1	200000	1.67	NICU
61	SATHYA	24	61871	102	AP	2	LSCS	1.3	140000	1.79	NICU
62	AMUTHA	24	60714	110	AP	2	VAGINAL	2	145000	1.76	NICU
63	THERAISA	24	45644	80	AP	1	LSCS	2.5	100000	1.49	IUD
64	MEENA	24	21298	116	AP	1	VAGINAL	1.6	140000	1.78	NICU
65	FARITHA BANU	24	31562	100	AP	2	VAGINAL	2.6	123000	1.82	WB
66	ANDICHI	24	65867	100	AP	2	VAGINAL	2	260000	1.85	NICU
67	ARIFA	24	31657	100	AP	2	LSCS	2.6	123000	1.84	WB
68	VALLI	24	65432	110	PP	2	VAGINAL	2.6	260000	1.86	WB
69	MANIMEGALAI	24	55655	100	AP	3	LSCS	1.8	170000	1.64	NICU
70	CHINNU	24	43433	110	AP	2	LSCS	2.3	180000	1.71	NICU

S.No	NAME	AGE	IP NUMBER	diastolic BP	TYPE	TIMES	MODE	WT IN KG	PLAT	MAGNESIUM LEVEL	BABY
71	SELVI	25	71021	106	AP	5	VAGINAL	0.8	100000	1.21	IUD
72	mallika	25	19887	110	AP	3	VAGINAL	1.5	150000	1.73	WB
73	KAVITHA	25	21307	110	AP	2	VAGINAL	1.2	112000	1.75	IUD
74	CHITRA DEVI	25	3979	100	AP	4	VAGINAL	2	200000	1.71	NICU
75	ESWARI	25	43531	102	AP	2	VAGINAL	2.1	180000	1.77	NICU
76	DEVI	25	7895	110	AP	4	VAGINAL	2	200000	1.79	NICU
77	SEETHALAKSHMI	25	10453	116	AP	4	VAGINAL	1	180000	1.61	IUD
78	MEENA	25	43244	110	PP	1	VAGINAL	2.5	150000	1.63	WB
79	SIVAGAMI	**26**	60266	112	AP	2	VAGINAL	1.75	280000	1.65	NICU
80	KATHAYEE	26	40622	100	AP	1	LSCS	1.8	100000	1.67	IUD
81	dhanalakshmi	26	65075	104	AP	2	LSCS	2	94000	1.69	WB
82	SAJITHA	26	48289	100	AP	2	VAGINAL	2.6	280000	1.77	WB
83	VASANTHA	27	10161	112	AP	5	LSCS	1.8	120000	1.49	IUD
84	CHITRA	27	23371	104	AP	1	LSCS	1.7	55000	1.36	NICU
85	SANUMA	27	37999	100	AP	1	VAGINAL	2.3	130000	2.32	WB
86	LATHAMANI	28	109877	106	PP	4+6	LSCS	1.7	180000	1.47	NICU
87	MAHESHWARI	28	23029	112	AP	4	VAGINAL	1.2	59000	1.5	IUD
88	SUMATHY	28	6173	100	AP	3	LSCS	0.7/0.8	122000	1.85	IUD
89	KARTHIGA DEVI	28	5007	104	PP	4+1	LSCS	1.9/2	92000	1.43	NICU
90	RANJITHA	28	45432	100	AP	1	VAGINAL	2.4	120000	2.01	NICU
91	SACHU	28	32436	106	AP	1	LSCS	2.6	180000	1.67	WB
92	dhanalakshmi	29	11144	102	AP	2	LSCS	2	100000	1.97	NICU
93	RAMALAKSHMI	30	58257	120	AP	3	VAGINAL	2.2	280000	1.83	NICU
94	SELVI	30	58023	110	AP	2	LSCS	2	120000	1.33	NICU
95	KASTHURI	30	49289	110	AP	2	LSCS	2.5	150000	1.38	WB
96	MYLAL	30	70144	100	PP	1+1	VAGINAL	1	90000	1.6	NICU
97	ANANDHI	30	14745	100	IP	2+1	VAGINAL	1.8	200000	1.57	NICU
98	SELVI	30	30053	116	IP	2	LSCS	2	98000	1.65	NICU
99	SARANYA	30	50032	100	IP	2	VAGINAL	2.2	280000	2.03	WB
100	GOWRI	30	34345	100	AP	1	VAGINAL	2.3	280000	1.65	WB
101	VENILLA	30	34564	112	AP	2	LSCS	2	100000	1.98	NICU
102	ESWARI	32	1940	110	AP	3	VAGINAL	1.75	270000	1.87	NICU
103	PREMA	35	34770	120	AP	1	VAGINAL	0.7	95000	1.37	IUD
104	PARIMALA	38	71101	100	AP	3	VAGINAL	1	100000	1.33	IUD

S.No	NAME	AGE	IP NUMBER	NEONATAL COMP	MATERNAL COMP	SEX	IMM SIGNS	AN DIAGN
1	SATHYA	16	52572		RENAL FAILURE	F	H1WK	BP +
2	RUBIDEVI	17	9518	TWINS		M/F	_	BP(-)
3	BINU	17	34897			M	H	BP(-)
4	SUGANTHY	18	69989			M	H	BP(-)
5	RABIYA	18	69986			M	H,V	BP(-)
6	MOHANA	19	58016			M	H	BP +
7	SANGEETHA	19	59011	SGA	RENAL FAILURE	M	H	BP(-)
8	SUGANYA	19	71348	PRETERM		M	H	BP(-)
9	SUGANYA	19	71345	SGA		M	H	BP(-)
10	PUSHPALATHA	19	70824	SEIZURES	PPH	M	H	BP +
11	GEETHA	19	15147		CHT	M	H	BP +
12	poornima	20	9436		HELLP,MM	M	_	BP +
13	RANJITHA	20	30399	SGA		M	_	BP(-)
14	RABIYA	20	55317			M	_	BP(-)
15	ANDICHI	20	24000		PPH	M	H	BP(-)
16	SARATHY	20	6780	SGA		M	H,V	BP(-)
17	SUDHA	20	2724	SGA		M	H,V	BP(-)
18	SARASU	20	74565	sepsis	PPH	M	H	BP(-)
19	DEVI	21	111545			F	_	BP +
20	BAGAVATHY	21	24470			M	_	BP(-)
21	UMADEVI	21	39700			M	H(+)	BP(-)
22	SARADHA	21	34918	SGA		M	_	BP +
23	MEENA	21	11324			F	H	BP(-)
24	MUMTAJ	21	14332		HELLP,MM	M	H	BP +
25	KAMATCHI	21	26544		ABRUPTION	M	H	BP +
26	JOTHIMANI	21	31241	SGA		M	H,V	BP(-)
27	JANAKI	21	32215			M	H,V	BP(-)
28	KRITHIGA	21	45413			M	_	BP +
29	ISWARYA	21	56732			M	H,V	BP +
30	mallika	22	9941	SGA		M	_	BP(-)
31	GAYATHRI	22	9272	SGA		F	_	BP +
32	VENI	22	24582	LBW		M	_	BP(-)
33	SIVAGAMI	22	54780	preterm DIED -1DAY		M	H10D	BP +
34	SUBULAKSHMI	22	64908			M	H	BP +
35	JAYANTHI	22	2661		PPH	M	H	BP +

S.No	NAME	AGE	IP NUMBER	NEONATAL COMP	MATERNAL COMP	SEX	IMM SIGNS	AN DIAGN
36	VANITHA	22	34545	SGA		M	H,V	BP +
37	KALAI	22	67578	SGA		M	H	BP +
38	AMUTHA	22	34567			F	H	BP(-)
39	CHITRA	23	10987			M	_	BP +
40	RANI	23	39763			M	H-12	BP(-)
41	HEMAVATHY	23	36777		PULMONARY EDEMA MM	F	_	BP(-)
42	DEVI	23	32425			F	H,V,BV	BP +
43	KAMALEESHWARI	23	34528			M	H	BP +
44	RANGANAYAKI	23	34568			M	H	BP(-)
45	JOTHI	23	11562			F	H	BP(-)
46	NAGAMANI	23	12358		ICH,MM	M	H	BP(-)
47	VAITHESHWARI	23	21981			M	H	BP(-)
48	muthuselvi	23	26586		PULMONARY EDEMA	M	H,V,BV	BP +
49	GEETHA	23	3425			M	H,V	BP +
50	PANDEESHWARI	23	62630			M	H	BP +
51	PANDISELVI	23	33422	SEIZURES		M	H	BP(-)
52	CHITRA	23	56458			M	H	BP +
53	JAYALAKSHMI	23	34289	DIED -1DAY		F	H,V,BV	BP +
54	muthuselvi	23	676899			M	H	BP(-)
55	HEMAPRIYA	23	3556			M	H	BP +
56	MANI	23	54672			F	H	BP(-)
57	PRIYA	24	114435			M	_	BP(-)
58	NASERA	24	112323			M	_	BP +
59	PREMA	24	52686		ABRUPTION	F	H	BP +
60	SARASWATHY	24	43678	DIED -1DAY		F	H	BP(-)
61	SATHYA	24	61871	preterm,DIED -1DAY		M	H,V,BV	BP(-)
62	AMUTHA	24	60714	SGA		F	H	BP +
63	THERAISA	24	45644	MACERATED	ASPIRATION	M	_	BP(-)
64	MEENA	24	21298			M	H	BP(-)
65	FARITHA BANU	24	31562			F	H,V	BP +
66	ANDICHI	24	65867	SEIZURES		M	H	BP(-)
67	ARIFA	24	31657			F	H,V	BP +
68	VALLI	24	65432			M	H	BP +
69	MANIMEGALAI	24	55655	SEIZURES		F	H,V	BP +
70	CHINNU	24	43433	PRETERM		F	H	BP +

S.No	NAME	AGE	IP NUMBER	NEONATAL COMP	MATERNAL COMP	SEX	IMM SIGNS	AN DIAGN
71	SELVI	25	71021			M	H	BP(-)
72	mallika	25	19887	SGA		M	H2D	BP(-)
73	KAVITHA	25	21307			F	H	BP(-)
74	CHITRA DEVI	25	3979	SEIZURES		M	H	BP(-)
75	ESWARI	25	43531	sepsis		F	_	BP +
76	DEVI	25	7895	SGA		M	H	BP +
77	SEETHALAKSHMI	25	10453			F	H,V	BP +
78	MEENA	25	43244			F	H	BP +
79	SIVAGAMI	**26**	60266	PRETERM		M	H,V	BP(-)
80	KATHAYEE	26	40622			F	H	BP(-)
81	dhanalakshmi	26	65075	SGA		M	H	BP(-)
82	SAJITHA	26	48289			M	H	BP(-)
83	VASANTHA	27	10161			M	_	BP(-)
84	CHITRA	27	23371	SEIZURES	HELLP	F	_	BP +
85	SANUMA	27	37999			M	H	BP(-)
86	LATHAMANI	28	109877	PRETERM		M	_	BP(-)
87	MAHESHWARI	28	23029		PPH	F	_	BP +
88	SUMATHY	28	6173	TWINS		F/F	H,V,BV	BP +
89	KARTHIGA DEVI	28	5007	TWINS		M/F	H,V,BV	BP +
90	RANJITHA	28	45432	SGA		M	H	BP(-)
91	SACHU	28	32436			F	H	BP(-)
92	dhanalakshmi	29	11144	SGA		M	_	BP +
93	RAMALAKSHMI	30	58257	PRETERM		M	H,V,BV	BP +
94	SELVI	30	58023	PRETERM		M	H	BP(-)
95	KASTHURI	30	49289		HELLP	F	H	BP(-)
96	MYLAL	30	70144	DIED -1DAY	PPH	F	H2D	BP(-)
97	ANANDHI	30	14745			M	_	BP +
98	SELVI	30	30053	DIED -1DAY		F	H,V	BP +
99	SARANYA	30	50032			F	H	BP +
100	GOWRI	30	34345			F	H	BP +
101	VENILLA	30	34564	SGA		M	H,V	BP +
102	ESWARI	32	1940			F	H	BP(-)
103	PREMA	35	34770		CHT	M	_	BP +
104	PARIMALA	38	71101			M	H	BP +

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PROFORMA

ANALYSIS OF FETOMATERNAL OUTCOME IN ECLAMPSIA

NAME AGE IP NO:

ADRESS:

BOOKING STATUS: PARITY:

GESTATIONAL AGE:

TYPE OF ECLAMPSIA: AP/IP/PP NUMBER OF FITS:

ADMISSION BP: TIME SINCE FITS: <6HRS / >6HRS

ANTENATAL DIAGNOSIS OF PREECLAMPSIA: YES/NO.

INDUCTION: YES/NO. MODE OF DELIVERY:

SEX:

APGAR AT 5MIN: LIVE/STILLBIRTH:

BIRTH WEIGHT:

NICU ADMISSION: YES/NO.

PERINATAL COMPLICATIONS:

MATERNAL COMPLICATIONS:

IF MATERNAL DEATH – CAUSE:

ABBREVIATIONS

AP – antepartum

BP – blood pressure

CHT – chronic hypertension

DIC – disseminated intravascular coagulation

FBS – fresh still birth

ICH – intra cerebral hemorrhage

IP – intrapartum

IUGR – intra uterine growth restriction

LSCS – lower segment caesarian section

MgSo4 – magnesium sulphate

MM – maternal mortality

PIH – pregnancy induced hypertension

PP – postpartum

PPH – post partum hemorrhage

SGA – small for gestation

WB – well baby