IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

A STUDY ON ANALYSIS OF FETOMATERNAL OUTCOME IN ECLAMPSIA IN A TERTIARY CARE HOSPITAL

[1]C.Rohith Siri Subramanyam, [2]N.V.Bhuvaneswari, [3]N.NavyaSree, [4]M.Mamatha

[1]Assistant Professor, [2, 3, 4] Pharm D Interns,

[1]Department of Pharmacy Practice,

[1]Dr.K.V.Subba Reddy Institute Of Pharmacy, Kurnool, India.

Abstract: A cross-sectional hospital base study was conducted to analyze fetomaternal outcome in eclampsia, clinical Scharacteristics that predispose as risk factors, changes in serum magnesium level and its influence on maternal outcome. Study conducted from 1st September 2021 to 30th March 2022 in the department of Obstetrics and Gynecology, Kurnool Medical College Hospital, Kurnool.

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, abruption 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. Multiparty, gestational age ≤ 32 weeks, seizures >5 episodes were significantly associated with perinatal mortality.

Key Words: - Eclampsia, fetomaternal outcome, serum magnesium.

I. INTRODUCTION

Eclampsia has been a recognized pathological entity since the time of Hippocrates and ancient Greek. It is derived from the Greek word meaning "flash out", in the sense of sudden event and dates back to 17th century. Eclampsia is perceived as the end of linear spectrum that stretches from the normal pregnancy through mild gestational hypertension preeclampsia finally eclampsia.

It is a enigmatic disease. A number of social genetic medical and obstetric conditions predisposed 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 non would 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. It is said that pre-eclampsia and Eclampsia contribute to death of a woman every 3 minutes Worldwide. 20 times more common in developing countries. Incidence of eclampsia in India is approximately 220 per 10,000 deliveries, that contributing about 8% of maternal mortality. It ranks 2nd only to anemia in developing countries.

Pre-eclampsia is not preventable but Eclampsia is preventable. Inspite if the global and regional interventions and initiatives from government, it's 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.

Incidence, morbidities and mortalities of eclampsia remain unacceptably high in developing countries. This indicates the need for continued in deepth studies in to its characteristics and pattern. This study was undertaken to determine the incidence of eclampsia, identify the predisposing socio-demographic factors, demonstrate clinical profile, changes in serum magnesium levels and analyze the modes of management and resulting fetal and maternal outcome at Kurnool medical college hospital.

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

The working group of NHBPEP- National High Blood Pressure Education Program (2000) classification if 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 pre-eclampsia and Eclampsia from the 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.

1. GESTATIONAL HYPERTENSION:

The diagnosis of gestational hypertension is made in women whose blood pressure reaches 140/90 mmHg 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 pre-eclampsia. Gestational hypertension is reclassified as transistent hypertension if evidence for pre-eclampsia doesn't develop and the blood pressure returns to normal by 12 weeks post-partum.

- PRE ECLAMPSIA:

It is a pregnancy specific syndrome that can effect virtually every organ. proteinuria is the surrogate objective maker that defines the system wide endothelial leak. It is defined by 24 hours urinary protein excretion exceeding 300mg , persistent 30 mg/dL(1+dipstick)protein in random urine samples.

1. ECLAMPSIA:

The Onset of convulsions in a women with pre eclampsia that can't be attributed to other causes is termed eclampsia. The seizures are generalized and may appear before , during ,or after labour.10% of eclamptic seizures develop before over proteinuria is identified.

- PREECLAMPSIA SUPERIMPOSED WITH CHRONIC HYPERTENSION:

This condition occurs in women who have been diagnosed with chronic high blood pressure before pregnancy, but then develop worsening high blood pressure and protein in the urine or other health complications during pregnancy.

- CHRONIC HYPERTENSION:

It is high blood pressure that was present before pregnancy or that occurs before 20 weeks of pregnancy . But because of high blood pressure usually doesn't have symptoms ,it might be hard to determine when it began.

ETIOPATHOGENESIS:

Gestational hypertensive disorders are more likely to develop in women who

1. Are exposed to chorionic villi for the first time.
2. Are exposed to a super abundance of chorionic villi, has with twins or hydatidiform mole
3. Have pre-existing renal or cardiovascular disease.
4. Are genetically pre disposed to hypertension developing during pregnancy.

Regardless of precipitating etiology, the cascade of events that leads to the pre eclampsia is characterized by a host of abnormalities that result in vascular endothelial damage and subsequent vasospasm, transudation of plasma, and ischemic and thrombotic sequalae. Recently pre eclampsia has been considered as 2 stage disorder.

Stage 1-Faulty endovascular trophoblastic remodeling that downstream causes the stage -2 clinical syndrome.

Abnormal trophoblastic invasion:

In normal implantation, the uterine spiral arterioles undergo extensive remodeling as they are invaded by endovascular trophoblast. These cells replace the vascular endothelial and muscular linings to enlarge the vessel diameter. In pre eclampsia there may be incomplete trophoblastic invasion with such shallow invasion myometrium, vessels are not lined by endo vascular trophoblasts. They don't use endothelial lining and musculoelastic tissue, and their mean external diameter is only half that of vessels in normal placenta, abnormally narrow spiral arteriolar lumen impairs placental blood flow. The magnitude of defective trophoblastic invasion of spiral arteries Corelates with the severity of hypertensive disorders.

Immunological factors:

Diminished perfusion and a hypoxic environment eventually lead to release of placental debris that insights a systemic inflammatory response, results in dysregulation of immune tolerance. Formulation of blocking antibodies to placental antigenic sites might be impaired. Beginning in early 2nd trimester women who developed a eclampsia, Th 1 action increased and the Th 1 /Th 2 ratio changes .contributes to an enhanced immunologically mediated inflammatory reaction are stimulated by placental microparticles.

Endothelial Cell activation:

Briefly, cytokines such as tumor necrosis factor-(TNF-alpha),and the interleukins (IL)may contribute to the oxidative stress associated with preeclampsia. This is characterized by reactive oxygen species and free radicles that lead to formation of self - propagating liquid peroxide. These in turn generate highly toxic radicles 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 anti-oxidants to prevent pre-eclampsia. Antioxidants are from a diverse family of compounds that's function to prevent overproduction of and damage caused by noxious free radicles. Dietary supplementation with antioxidants to prevent pre eclampsia has thus far proven un successful.

Genetic factors:

Pre eclampsia is a multifactorial ,poly genic disorder.an incidence risk factor for pre eclampsia is 20 to 40 percent for daughter of pre eclampsia .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 pre eclampsia . 7 of these have been investigated widely.

●Increased pressor responds to infused nor epinephrine and angiotensin -2 in women with early pre eclampsia . Normal pregnant women develop refractoryness to vaspressor .

●Endothelial prostacyclin (PGI2)production decreases in pre eclampsia .Thromboxane A2 increases. PGI2 /TXA2 ratio decreases. The net result favors increased sensitivity to infused angiotensin 2 and ultimately vasoconstriction.

●Nitric oxide-Potent vasodilator is synthesized from L-Argenine by endothelial cells .withdrawal of nitric oxide results in a clinical picture similar to pre eclampsia in a pregnant animal model. It appears that pre eclampsia is associated with decreased endothelial nitric oxide synthesis expression, thus increased nitric oxide inactivation. These responses may be race related.

●soluble Fms-soluble like tyrosine kinase -1 (sFIt-1) is a variant of the (FIt-1) receptor for placental growth factor (PIGF) and vascular endothelial growth factor(VEGF).Increased maternal sFIt 1 levels inactivate and decreased circulating free PIGF, VEGF, concentrations leading to endothelial dysfunction .

●Soluble endoglin (sEng) is a placenta derived molecule that blocks endoglin which is a co factor for TGF-beta. These soluble form endoglin inhibits TGF hence decreased endothelial nitric oxide dependent vasodilation.

●Cardiatic output in pre eclampsia decreases likely due to increase peripheral vascular resistance.

●Hemo Concentration is hallmark of pre eclampsia .Generalized vaso-constriction 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 pre eclampsia. over thrombocytopenia defined by platelet count less than 1 lakh cells /cu.L./ Indicates severe disease .In general lower the platelet count ,Greater the rates of maternal and fetal morbidity and mortality. In most cases delivery is advisable because thrombocytopenia continues to worsen .after delivery; the platelet count may continue to decrease for the 1st 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.

●**KIDNEY**-During normal pregnancy blood flow and glomerular filtration increase .With development of pre eclampsia 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 I dilated for these women with oliguria ,unless Diminished urine output is caused by hemorrhage. Plasma uric acid is typically elevated in pre eclampsia,due to reduction in filtration and enhanced tubular re absorption. Atleast some degree of proteinuria will establish the diagnose of pre eclampsia .In 24 hour quantitative urinary specimen ,the standard consensus threshold value used is greater than 300 mg/24hr.urinary dipstick method is simple but with false positive and negative results .Worsening of proteinuria has been considered to be a sign of severe disease, these may not be the case .it is currently being investigated.

●**LIVER**-Hepatic changes in women with fatal eclampsia were described in 1856 by Virchow.

These characteristic lesions commonly found in the liver periphery. Hemolysis, hepatocellular necrosis and Thrombocytopenia were later termed as HELLP SYNDROME.

10% HELLP had concurrent eclampsia. They have worse outcome than only with pre eclampsia .liver involvement in pre eclampsia is clinically significant in following circumstances.

1. Symptomatic involvement, typically manifest by moderate to severe right upper or mid epigastric pain and tenderness, is usually only seen with severe disease.

2. Asymptomatic evaluations of serum hepatic transaminase levels AST and ALT are considered markers for severe pre eclampsia. Values seldom exceed 500 U/L. They inversionally follow platelet levels. but usually normalize within 3 days following delivery.

3. Hepatic hemorrhage from areas of infraction may extend to form a hepatic hematoma .These inturn may extend to form a sub capsular hematoma they may rupture. They can be identified using CT or MRI .Management usually consists of conservative and observation .vary few cases may require prompt surgical intervention.

●**BRAIN**-Headaches and visual symptoms are common with pre eclampsia ,and associated convulsions define eclampsia. Principle lesions found 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.

II.NEED FOR THE STUDY:

1. To reduce the incidence and complications of eclampsia.
2. To improve the antenatal care at community level.
3. To avoid the recurrence of seizures and hypoxic injury to both mother and fetus.
4. To reduce the perinatal mortality due to eclampsia.
5. To validate the low serum magnesium level as predictor of maternal complications in eclampsia.

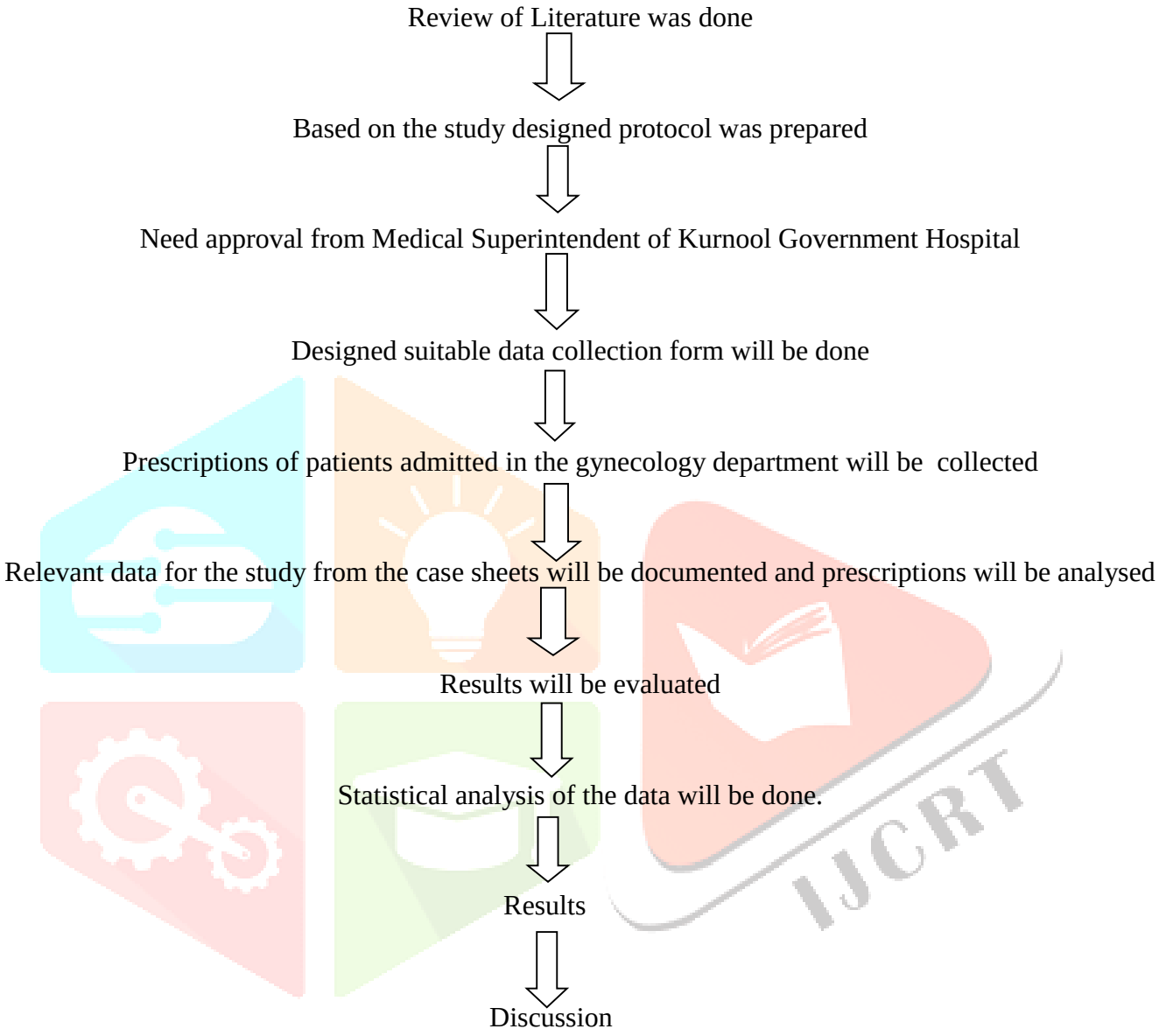
III. AIM OF THE STUDY

A Study on analysis of feto-maternal outcome in eclampsia

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 feto-maternal outcome.
4. To analyze the changes in platelet count, serum magnesium level and their influence on maternal outcome.

IV. Plan of work

- The present study will be conducted in Department of Gynocology at Kurnool Govt.General Hospital. The patients will be selected based on the inclusion criteria. Written consent will be obtained and confidentially the data will be assured to the patient. All the necessary and relevant data will be collected from the patient case notes, treatment charts, laboratory reports and patient interview. It will be recorded in a specially designed patient proforma / quesionarries



V. Methodology

Study design:

A Prospective observational study

Study duration:

The study will be conducted over a period of 6 months.

Study site:

The study will be conducted at inpatient Department of gynecology at Government general hospital, Kurnool, Andhra Pradesh.

Source of data:

All the patients satisfying the inclusion criteria were selected from a Gynecology department in government general hospital, Kurnool.

Inclusion criteria:

- Reporting in third trimester of pregnancy.
- Single term and multiple term pregnancy.
- Patients with generalized tonic-clonic convulsions during pregnancy/labour/ within 7 days of delivery were included.

Exclusion criteria:

- Previous history of epilepsy are excluded.
- Previous history of neurological disorders.
- Features of suggestive of encephalitis/meningitis.

Patient consent: The study procedure will be completely explained to the patient and a patient consent will be collected from them.

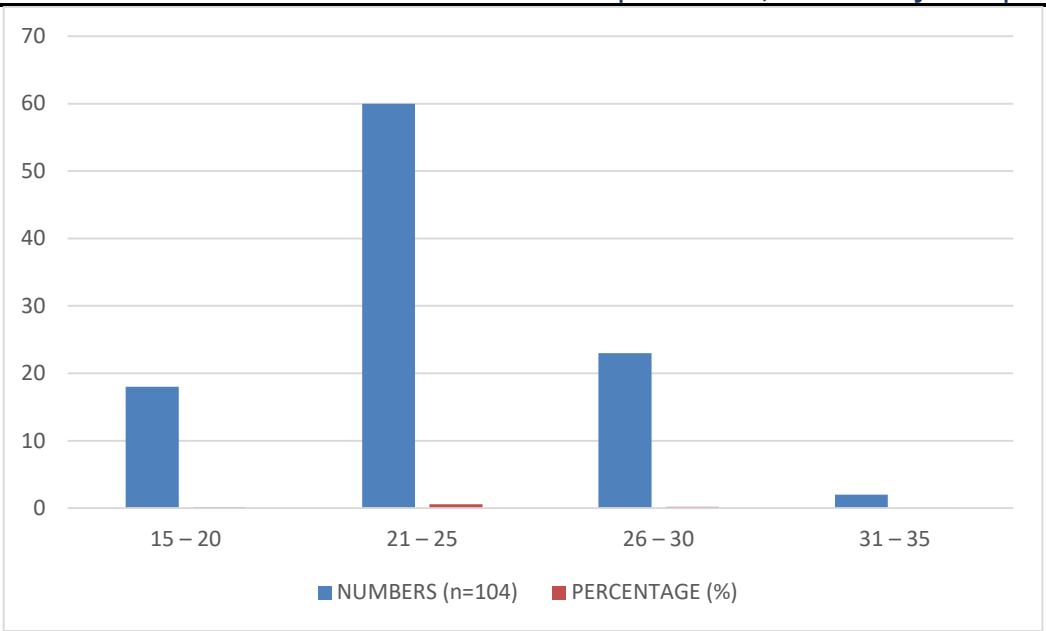
METHOD OF COLLECTION OF DATA:

- All the patients were satisfying the inclusion criteria were selected from the Gynecology department in government general hospital, Kurnool.
- All the data of the subject are collected by using the proforma.
- The data collection includes Demographic details, History of present illness, Treatment history, past medication history, Family history and allergies, Laboratory investigations, Diagnosis, Drug chart, Risk factors, and patient counselling.

VI. RESULTS

6.1 AGE DISTRIBUTION

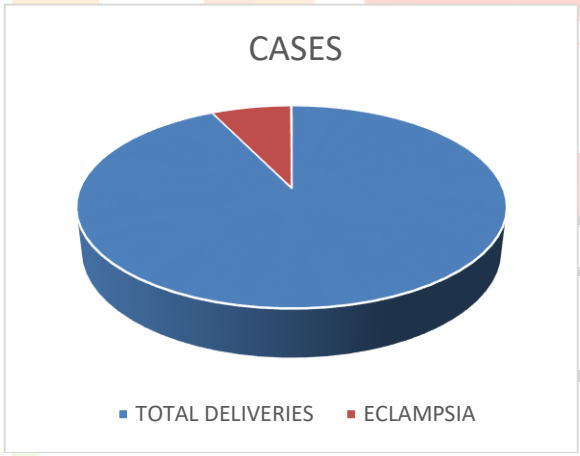
AGE IN YEARS	NUMBERS (n=104)	PERCENTAGE (%)
15 – 20	18	17%
21 – 25	60	58%
26 – 30	23	22%
31 – 35	2	2%
36 – 40	1	1%



6.1.a AGE DISTRIBUTION

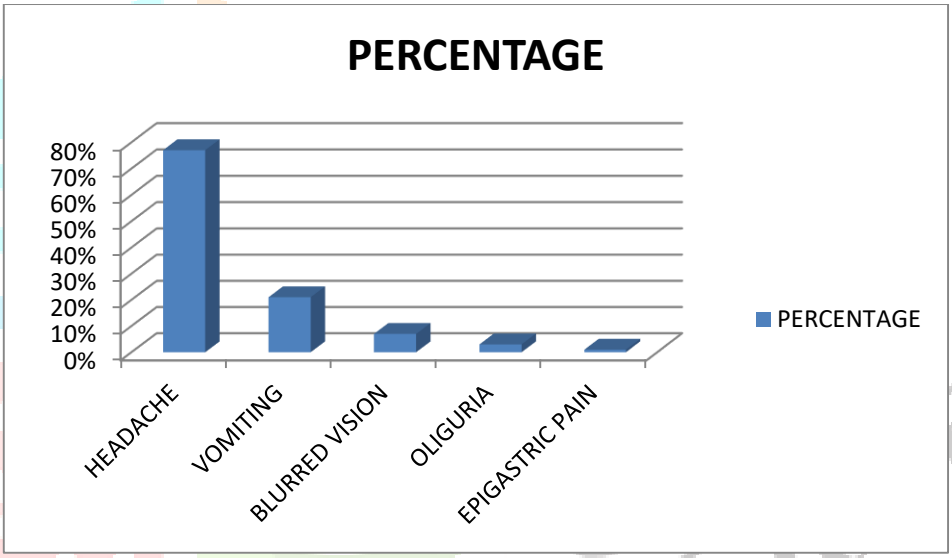
INCIDENCE: Patients included in the study were those with the diagnosis of eclampsia, admitted in labor ward ICU between first of September 2021 and 30th of March 2022.

- ✓ There were total of **1350** deliveries during the study period.
- ✓ **104** eclamptic patients were admitted.
- ✓ 101 singleton and 3 twin deliveries
- ✓ Incidence of eclampsia at KMH during the study period was **1.23%**.



6.2 IMMINENT SYMPTOMS OF ECLAMPSIA

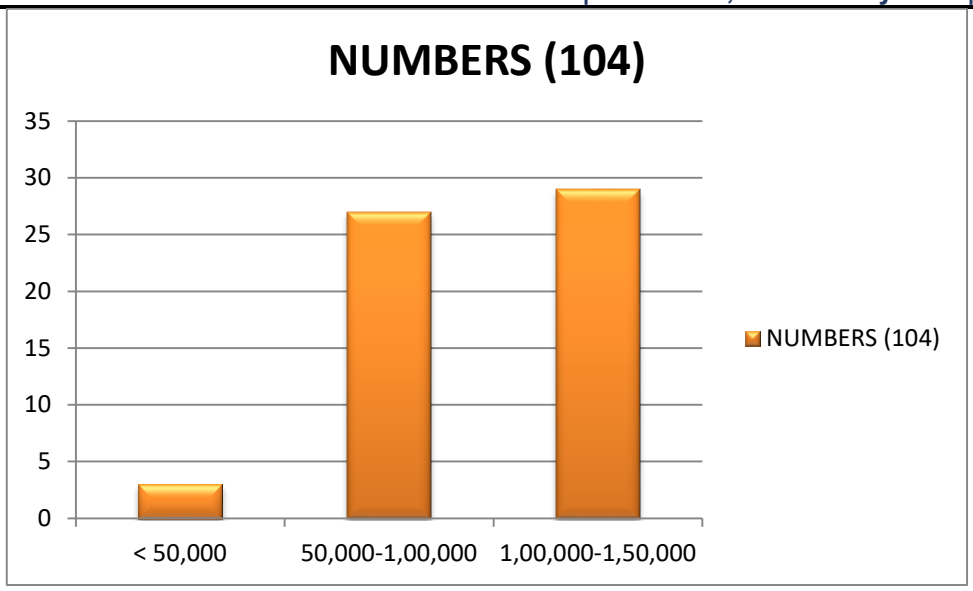
IMMINENT SYMPTOMS	PERCENTAGE
HEADACHE	77%
VOMITING	21%
BLURRED VISION	7%
OLIGURIA	3%
EPIGASTRIC PAIN	0.96%



6.2.b IMMINENT SYMPTOMS OF ECLAMPSIA

6.3 TYPES OF ECLAMPSIA

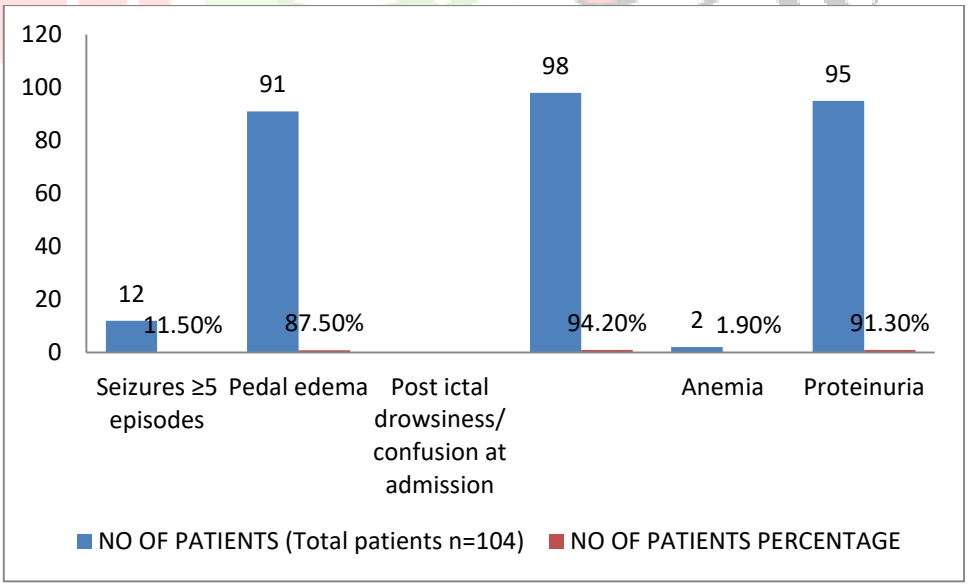
TYPE OF ECLAMPSIA	TOTAL NUMBER OF SUBJECTS
Antepartum Eclampsia	**81**
Intrapartum Eclampsia	**6**
Postpartum Eclampsia	**14**



6.3.c TYPES OF ECLAMPSIA

6.4 SIGNS OF ECLAMPSIA

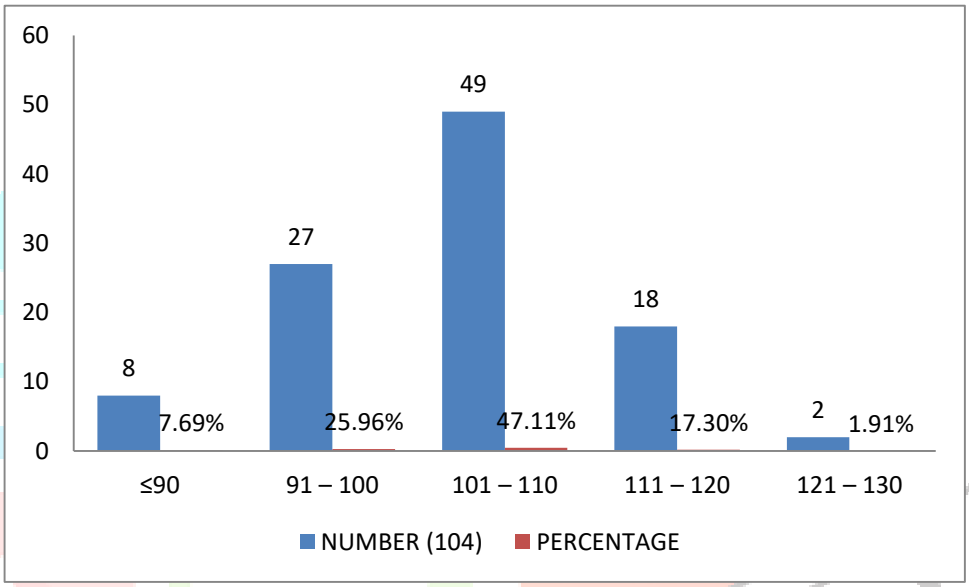
SIGNS	NO OF PATIENTS (Total patientsn=104)	PERCENTAGE
Seizures ≥5 episodes	12	11.5%
Pedal edema	91	87.5%
Post ictal drowsiness/ confusion at admission	98	94.2%
Anemia	2	1.9%
Proteinuria	95	91.3%



6.4.d SIGNS OF ECLAMPSIA

6.5 DIASTOLIC BLOOD PRESSURE AT THE TIME OF ADMISSION

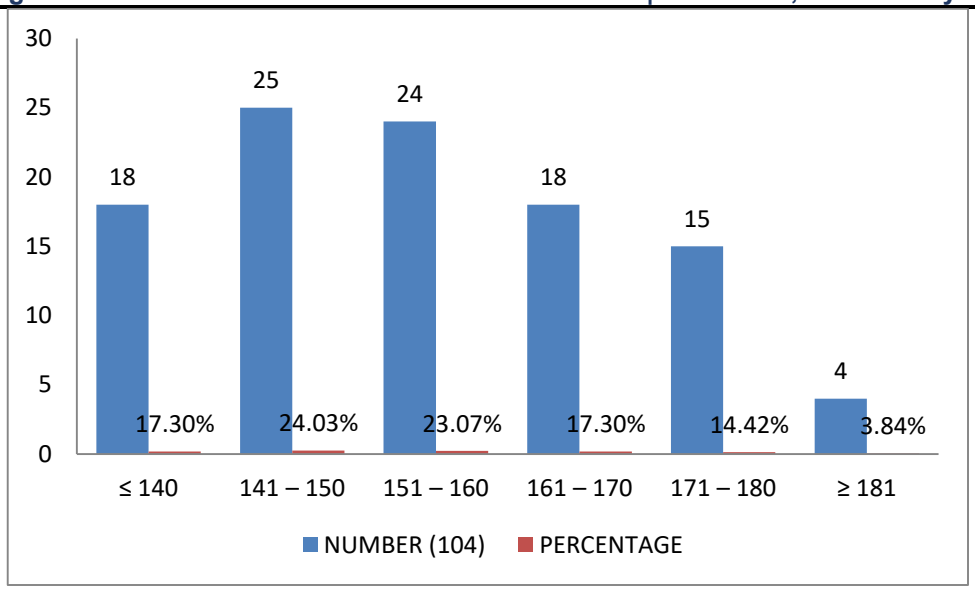
BP in mm Hg	NUMBER (104)	PERCENTAGE
≤90	8	7.69%
91 – 100	27	25.96%
101 – 110	49	47.11%
111 – 120	18	17.30%
121 – 130	2	1.91%



6.5.e DIASTOLIC BLOOD PRESSURE AT THE TIME OF ADMISSION

6.6 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%



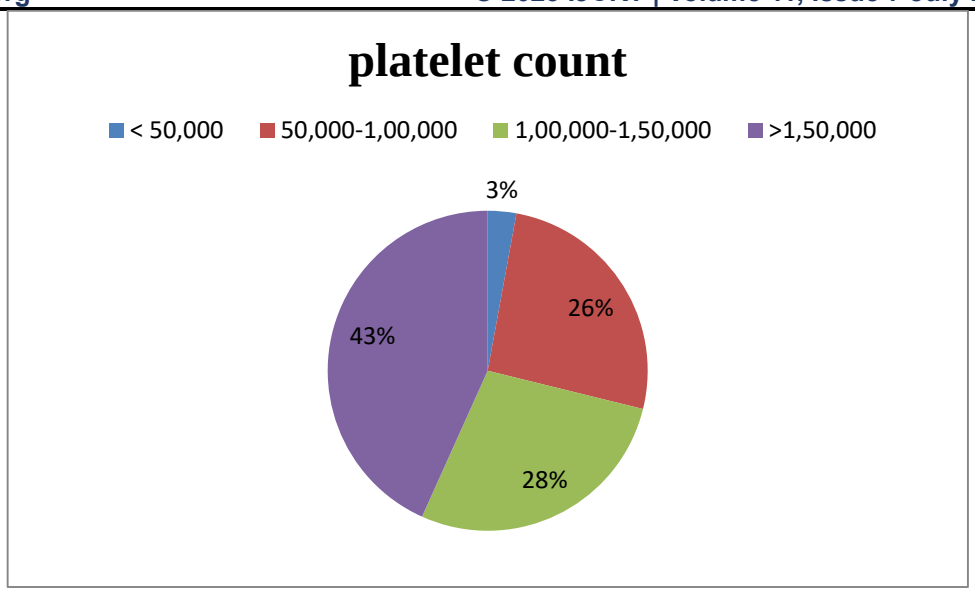
6.6.f SYSTOLIC BLOOD PRESSURE AT THE TIME OF ADMISSION

6.7 DEMOGRAPHIC CHARACTERISTICS – IN RELATION TO TYPES OF ECALMPSIA:

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

6.8 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%



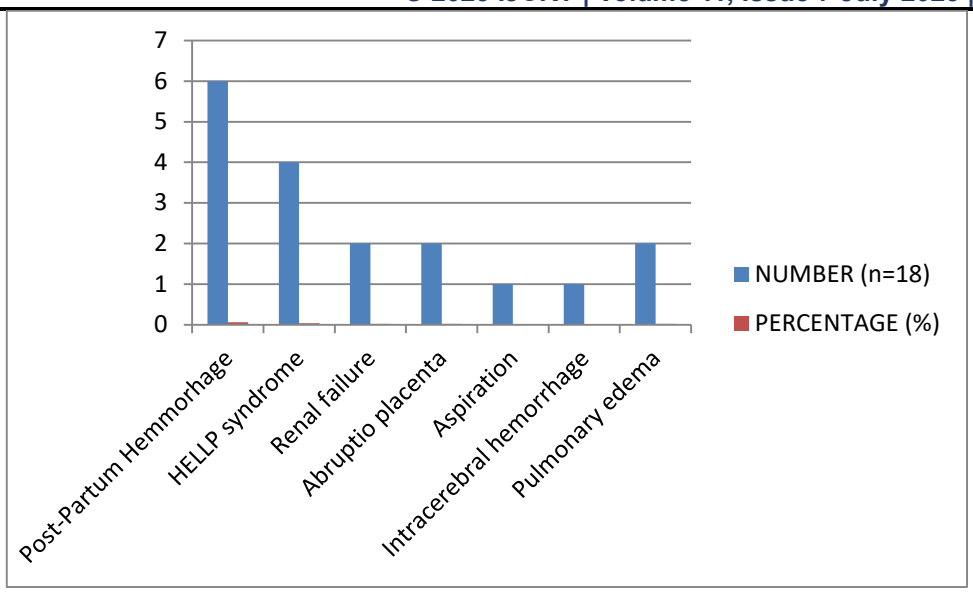
6.8.g PLATELET COUNT IN ECLAMPSIA

6.9 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

6.10 MATERNAL COMPLICATIONS

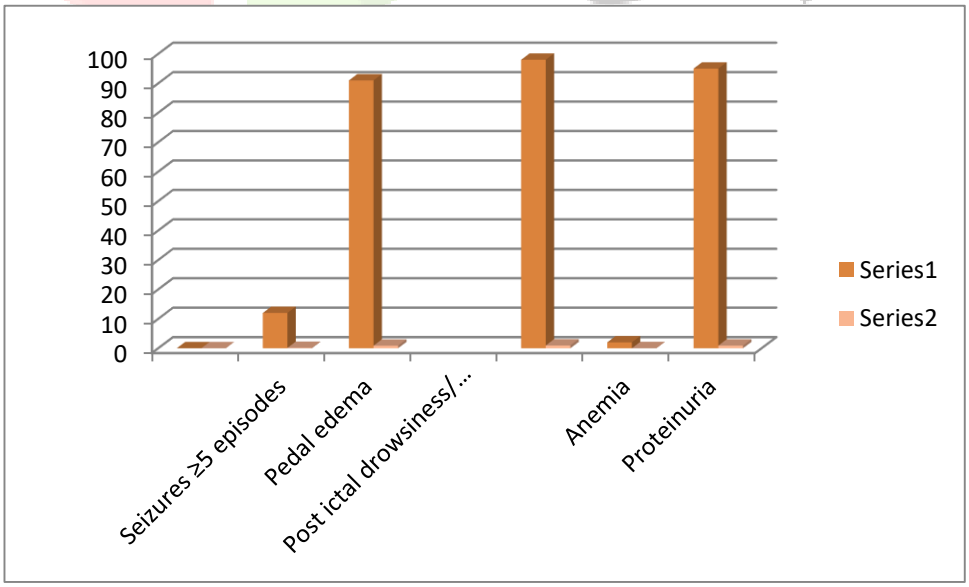
MATERNAL COMPLICATIONS	NUMBER (n=18)	PERCENTAGE (%)
Post-Partum Hemmorhage	6	5.76%
HELLP syndrome	4	3.84%
Renal failure	2	1.92%
Abruptio placenta	2	1.92%
Aspiration	1	0.96%
Intracerebral hemorrhage	1	0.96%
Pulmonary edema	2	1.92%



6.10.h MATERNAL COMPLICATIONS

6.11 NUMBER OF SEIZURES AND PERINATAL OUTCOME

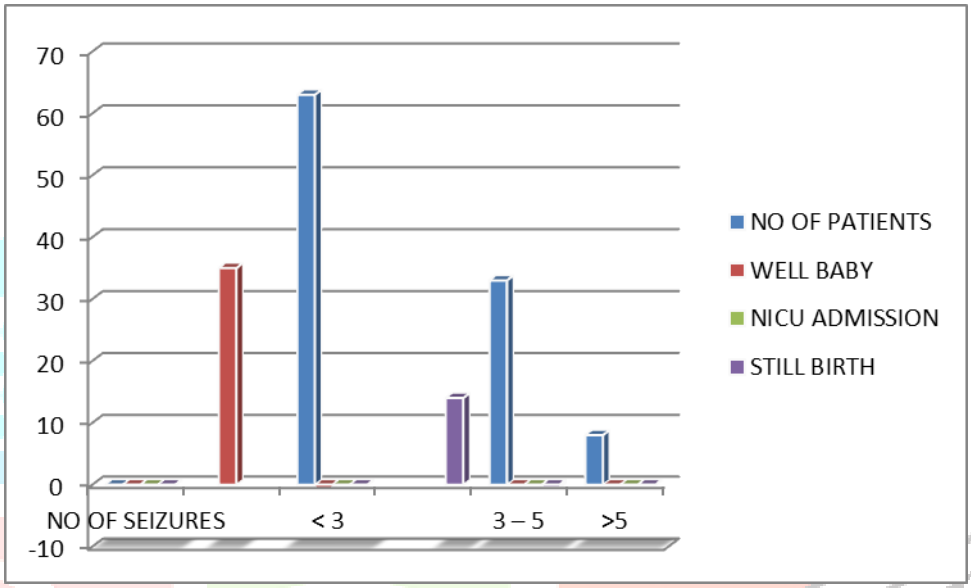
PERINATAL MORBIDITY	NUMBER (n=83)	PERCENTAGE
NICU admissions	41	49.39%
Small for gestation	39	46.98%
Preterm	8	9.63%
Jaundice	11	13.25%
Seizures	6	7.22%
Respiratory distress	16	19.27%
Sepsis	2	2.40%



6.11.i NUMBER OF SEIZURES AND PERINATAL OUTCOME

6.12 SEIZURES ACTIVITY:

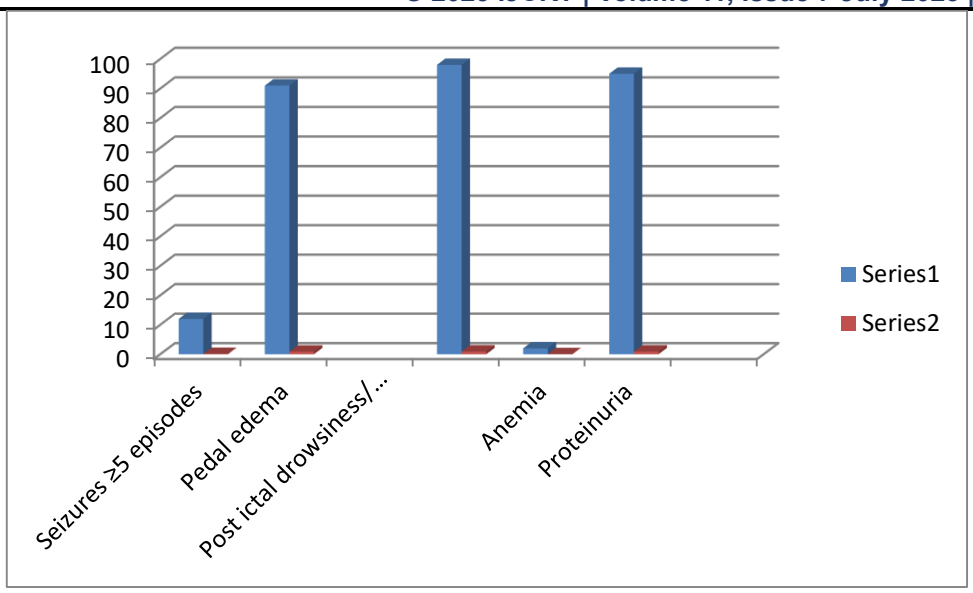
NO OF SEIZURES	NO OF PATIENTS (n=104)	WELL BABY (n=42)	NICU ADMISSION (N=41)	STILL BIRTH (n=24)
< 3	63	35 (54%)	22 (33%)	7 (11%)
3 – 5	33	6 (16%)	15 (42%)	14 (42%)
>5	8	1 (12%)	4 (50%)	3 (38%)



6.12.j. SEIZURES ACTIVITY

6.13 MATERNAL COMPLICATIONS

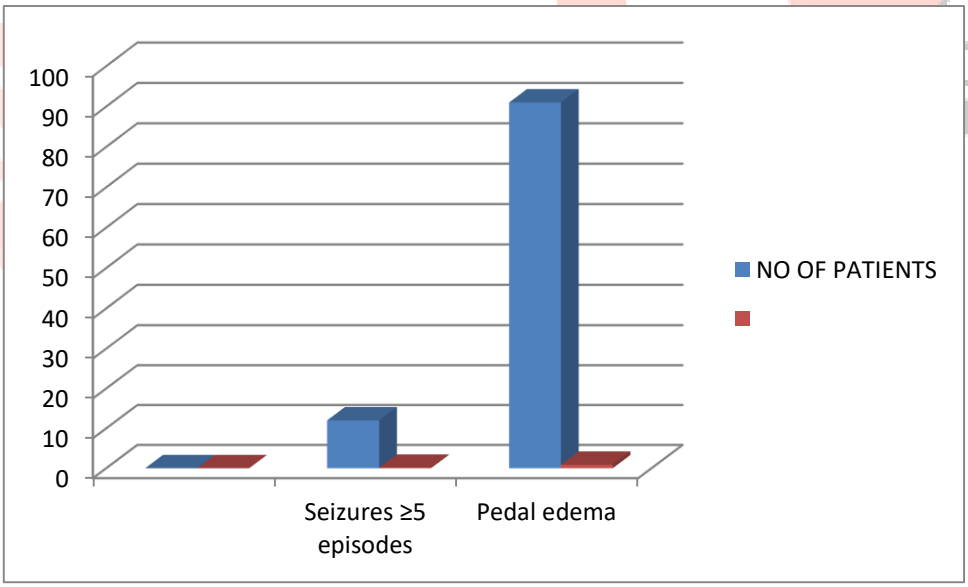
MATERNAL COMPLICATIONS	NUMBER (n=18)	PERCENTAGE (%)
Post Partum Hemmorhage	6	5.76
HELLP syndrome	4	3.84
Renal failure	2	1.92
Abruptio placenta	2	1.92
Aspiration	1	0.96
Intracerebral hemorrhage	1	0.96
Pulmonary edema	2	1.92



6.13.k MATERNAL COMPLICATIONS

6.14 MATERNAL MORTALITY – CAUSES

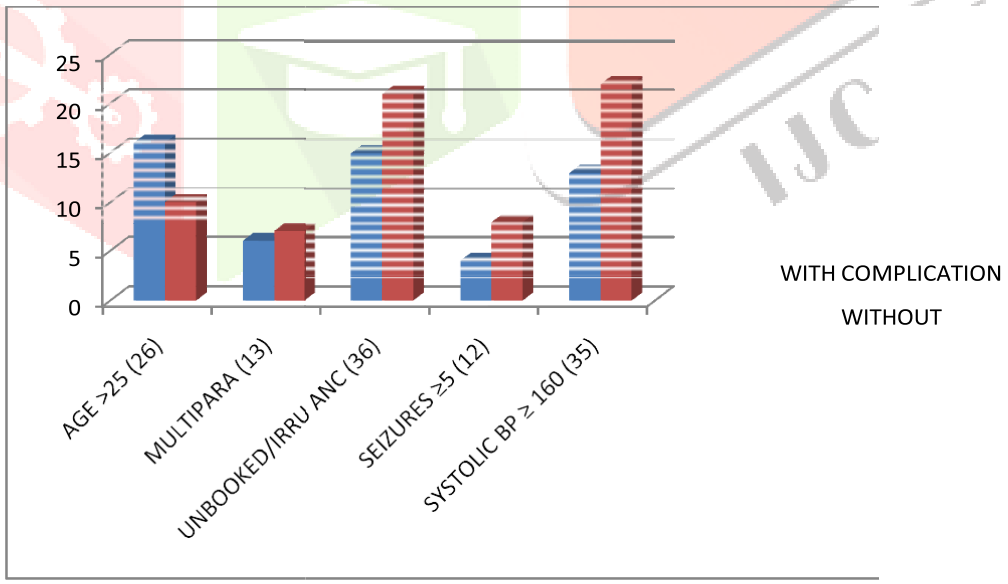
CAUSES	NUMBER (n=4)	PERCENTAGE
HELLP syndrome-DIC	2	50%
Intra cerebral hemorrhage	1	25%
Pulmonary edema	1	25%



6.14.l MATERNAL MORTALITY – CAUSES

6.15 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*
Un booked/ 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*



6.15.m RISKFACTORS FOR MATERNAL COMPLICATIONS

VII.DISCUSSION

Incidence of eclampsia: In our study over a period of 6 months 104 patients of eclampsia had been studied, with the incidence of 1.24%. Incidence varies between 0.5% and 1.8% .

Clinical profile: Eclampsia occurred in both extremes of age group in reproductive women. In our study age group between 21 and 25 years constitute the maximum percentage of eclampsia (58%) similar to other studies.

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.

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

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. 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%)

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).

Perinatal morbidity and mortality:

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 inceased. 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% $p<0.001$).

Un-booked 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.

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).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.

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.

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.

VIII. CONCLUSION

- To reduce the incidence and complications 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 ensure proper 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 center 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.

IX. REFERENCES

1. Navlaspur R, Narendra GS. A study on blood flow in uterine artery, umbilical artery and middle cerebral artery using Doppler ultrasound in a group of patients with pregnancy induced hypertension.
2. Duley L. Maternal mortality associated with hypertensive disorders of pregnancy in Africa, Asia, Latin America and the Caribbean. BJOG: An International Journal of Obstetrics & Gynaecology. 1992 Jul;99(7):547-53.
3. Myers JE, Baker PN. Hypertensive diseases and eclampsia. Current opinion in Obstetrics and Gynecology. 2002 Apr 1;14(2):119-25.
4. Shaheen B, Hassan L, Obaid M. Eclampsia, a major cause of maternal and perinatal mortality: a prospective analysis at a tertiary care hospital of Peshawar. JPMA. The Journal of the Pakistan Medical Association. 2003 Aug 1;53(8):346-50.
5. Khan KS, Wojdyla D, Say L, Gülmezoglu AM, Van Look PF. WHO analysis of causes of maternal death: a systematic review. The lancet. 2006 Apr 1;367(9516):1066-74.
6. Berg CJ, Harper MA, Atkinson SM, Bell EA, Brown HL, Hage ML, Mitra AG, Moise Jr KJ, Callaghan WM. Preventability of pregnancy-related deaths: results of a state-wide review. Obstetrics & Gynecology. 2005 Dec 1;106(6):1228-34.
7. Conrad KP, Karumanchi SA. Renal physiology and disease in pregnancy. InSeldin and Giebisch's The Kidney 2013 Jan 1 (pp. 2689-2761). Academic Press.
8. Chesley LC. Diagnosis of preeclampsia. Obstetrics and gynecology. 1985 Mar 1;65(3):423-5.
9. Taylor RN, Roberts JM, Cunningham GF, Lindheimer MD, editors. Chesley's hypertensive disorders in pregnancy. Elsevier; 2014 Aug 28.
10. Prefumo F, Güven MA. PRE-EKLAMPS NN PATOF ZYOLOJ S NDE ESK VE YEN GÖRÜ LER. Journal of Turkish Obstetric and Gynecology Society. 2007;4(4):237-45.
11. Mostello D, Catlin TK, Roman L, Holcomb Jr WL, Leet T. Preeclampsia in the parous woman: who is at risk?. American journal of obstetrics and gynecology. 2002 Aug 1;187(2):425-9.
12. Manten GT, van der Hoek YY, Sikkema JM, Voorbij HA, Hameeteman TM, Visser GH, Franx A. The role of lipoprotein (a) in pregnancies complicated by pre-eclampsia. Medical hypotheses. 2005 Jan 1;64(1):162-9.