"CLINICAL AND DIAGNOSTIC MARKERS IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION- HOSPITAL BASED OBSERVATIONAL STUDY."

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Dr. Ranjit Kangle MD,
Professor and HOD
Department of Pathology,
J. N. Medical College,
Belagavi, Karnataka

Date :
Place: Belagavi.

Dr.(Mrs) N. S. Mahantashetti MD (Paed).
Principal
J. N. Medical College,
Belagavi, Karnataka.

Date:
Place: Belagavi.

PLAGIARISM CERTIFICATE

JAWAHARLAL NEHRU MEDICAL COLLEGE

(Recognized by Medical Council of India, New Delhi)

Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

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Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BN0119002.
Postgraduate Student,
2019-20 Batch,
Department of Pathology,
J. N. Medical College, Belagavi.

LIST OF ABBREVIATIONS USED

/Cumm	-	Per cubic millimeter
AD	-	Anno Domini
ALP	-	Alkaline phosphatase
ALT	-	Alanine transaminase
AST	-	Aspartate amino transferase
aPTT	-	Activated partial thromboplastin time
BP	-	Blood Pressure
CBC	-	Complete blood count
CI	-	Confidence interval
DIC	-	Disseminated intravascular coagulation
e.g.	-	For example
etc.	-	Etcetera
ESR	-	Erythrocyte sedimentation rate
FDP	-	Fibrin degradation products
gm%	-	Gram percentage
GFR	-	Glomerular filtration rate
Hb	-	Hemoglobin
HELLP	-	Haemolysis, elevated liver enzymes, and low platelets
hrs	-	Hours
i.e,	-	Thatis,
IU/L	-	International units per liter
IUGR	-	Intrauterine growth retardation
IUD	-	Intrauterine death

kb	-	kilo byte
L	-	Liter
LFT	-	Liver function tests
LDH	-	Lactate Dehydrogenase
MPV	-	Mean platelet volume
meq/L	-	Milli equivalent per liter
mg/dL	-	Milligram per deciliter
mmHg	-	Millimeters of mercury
mm^3	-	Cubic millimeter
mmol/L	-	Millimole perliter
n	-	Total number
Nm	-	Nanometer
p	-	Probability
PCV	-	Packed cell volume
PIH	-	Pregnancy Induced hypertension
PT	-	Prothrombin time
PDW	-	Platelet Distribution Width
PGI2	-	Prostacyclin
RBC	-	Red blood cell
RFT	-	Renal function test
r	-	Correlation coefficient
SD	-	Standard deviation
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic pyruvic transaminase
TLC	-	Total leucocyte count

TT	-	Thrombin time
USA	-	United States of America
WBC	-	White blood cell
WHO	-	World Health Organization

ABSTRACT

Background and objectives

Pregnancy-induced hypertension (PIH) is one of medical disorder of pregnancy contributing significantly to maternal and fetal morbidity and mortality. It is progressive disease with a variable mode of presentation and rate of progression. Preeclampsia has contributed to both maternal and perinatal morbidity and mortality globally in developing as well as developed countries. This study is undertaken to know the haematological, biochemical and coagulative derangement in preeclampsia and further access the prognosis and severity of the complications and thus will help as a treatment guide for cases of preeclampsia and eclampsia.

Methodology

Blood and Urine samples were collected from 160 patients clinically diagnosed with PIH at Dr. Prabhakar Kore Charitable hospital Belgaum. Of these 160 patients

73 cases- mild PIH ,52 cases- severe PIH ,27 cases - eclampsia ,8 cases- HELLP

The patients were properly education about the procedure, an informed consent was taken. The collected samples were labelled with the sample number, date and time of collection and processed in the department of pathology and department of biochemistry for haematological, biochemical and coagulative studies in PIH.

Haematological parameters like haemoglobin, PCV, RBC count, total count (TC), erythrocyte sedimentation rate (ESR), retic count, platelet count (PLT), mean platelet volume (MPV)

Liver function test (LFT) [total bilirubin, SGOT, SGPT, ALP]

Renal function test (RFT) [serum creatinine, serum urea, serum uric acid].

Coagulation tests such as prothrombin time (PT), activated partial thromboplastin time(aPTT), thrombin time (TT), D-dimer and fibrinogen, routine urine examination for urinary proteins was done in all patients.

Results

PIH is frequent in 26 to 30 years, primigravida with previous and family history. Epigastric pain was most common symptom in patients. The mean haemoglobin was 10.92 g./. The platelet count, mean platelet volume decreased with severity of PIH. Total bilirubin, SGOT, SGPT, ALP, LDH And serum urea, creatinine, uric acid, urine protein increased with severity of disease. PT, aPTT, TT, D-dimer increased significantly with severity of PIH.

Interpretation and Conclusion

- PIH was in 26 to 30 years primigravida. Decrease in the haemoglobin level with the increase in severity of PIH.
- Platelet count decreased, SGOT, SGPT, ALP, LDH levels, Serum creatinine, serum uric acid was increased with the severity of PIH.
- PT, aPTT, TT and d-dimer were raised with severity of PIH.
- Urine proteins were elevated in PIH.

Keywords

Pregnancy induced hypertension (PIH), HELLP, prothrombin time (PT), activated partial thromboplastin time (aPTT), thrombin time (TT).

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INTRODUCTION

Pregnancy induced hypertension (PIH) is defined as BP > 140/90 mm Hg after 20 weeks of gestation, proteinuria >300mg/24hrs or >1+dipstick, in a previously non hypertensive, non proteinuric women. (1)

Pregnancy-induced hypertension is one of the common medical disorders of pregnancy leading to maternal/fetal morbidity and mortality. It is progressive disease with a variable mode of presentation and rate of progression. Hypertension, excessive weight gain, proteinuria, edema are some of the classic clinical features. (2)

Preeclampsia has contributed to both maternal and perinatal morbidity and mortality globally in developing as well as developed countries. (5,6)

Hypertensive disorders of pregnancy occur in about 8-10% of all pregnant women around the world and is the second leading cause of direct maternal and fetal deaths. Preeclampsia affects 3–5% of pregnancies of which the incidence in primigravida is about 10% and in multigravida 5%.

Preeclampsia and other hypertensive disorders of pregnancy will impact 5-8% of all child births in the United States. Incidence rates for preeclampsia in the United States, Canada and Western Europe, range from 2-5%. Severe forms of preeclampsia and eclampsia is seen in a range of 4% of all deliveries to as high as 18% in parts of Africa and other developing world. Around the world about ten million women develop preeclampsia ever year. Worldwide about 76,000 pregnant women die each year from preeclampsia and related hypertensive disorders. The number of fetal deaths from these disorders is calculated to be 500,000 per annum.

Women in developing countries are seven times more prone to develop preeclampsia than woman in a developed country. Maternal death occurring from hypertensive disorders of pregnancy is around 10-25% thus it is responsible for around one tenth of all maternal deaths in Asia and Africa.

India is among those countries which have a very high maternal mortality rate i.e. 122 per 100,000 live births. The major causes of maternal deaths in India are haemorrhage, sepsis, hypertension, obstructed labour, abortion and other conditions. Hypertension which can be a sign of pre-eclampsia accounts for 5% of maternal deaths in India.

In India, the incidence of preeclampsia is reported to be 8-10% among the pregnant women. According to a study, the prevalence of hypertensive disorders of pregnancy was 7.8% with preeclampsia being 5.4% of the study population in India. [(3)]

In Karnataka maternal mortality rate i.e. 103 per 100,000 live births. Thus Karnataka continues to remain at 11^{th} position in the country and fifth among southern states in terms of maternal mortality rate.

Various stages of preeclampsia have been explained by many pathophysiological mechanisms, alone or in combination. There can be impaired vascular remodelling of the maternal–fetal interface, excessive immune response to paternal antigens, systemic inflammatory response, and dysfunctional placental or endothelial response, all of these processes being modulated by genetic and environmental parameters. Increased systemic vascular resistance, enhanced platelet aggregation, activation and alteration of the coagulation system, and endothelial cell dysfunction can cause abnormal vascular response which is thought to be the pathogenesis of preeclampsia.

Intravascular coagulation leads consumption of platelet and fall in the platelet count.

The risk of pre-eclampsia significantly increases in women with nulliparous state; previous history of preeclampsia or eclampsia; age above 30 years those with either pre-existing vascular diseases or any other conditions which increase cardiovascular risk, hypertension, diabetes, known renal diseases, thrombophilia, obesity (body mass index > 29), multiple gestation, molar pregnancy and African American ethnicity.

Pregnant female with preeclampsia can have the following complications Eclampsia, Hemolysis, elevated liver enzymes, and low platelets (HELLP), placental abruption, stroke, pulmonary oedema, kidney failure, liver failure, disseminated intravascular coagulation (DIC), intracranial haemorrhage, retinal detachment, maternal death. Fetal complication include intrauterine growth retardation (IUGR), preterm birth, still birth.

Amongst these Elevated liver enzymes, and low platelets (HELLP) syndrome and eclampsia are the serious complications of the preeclampsia in pregnant mother. HELLP syndrome is a severe form of pre-eclampsia, which is dangerous to both mother and fetus. This abbreviation HELLP was first used in 1982 by Weinstein as this disease was a triad of hemolysis, elevated liver enzymes, and low platelets.

HELLP syndrome is classified into three classes according to the decreased maternal platelet count :-

Class 1 - If platelet count <50,000/cumm,

Class 2 - If platelet count is >50,000 and <100,000/cumm

Class 3 - If platelet count >100,000 and <150,000/cumm. [7]

Pathophysiologically, it is characterized by microangiopathic hemolytic anemia associated with liver and kidney damage that can progress to DIC having fetal termination. Thus morbidity and mortality related to preeclampsia can be prevented by providing timely accurate haematological , biochemical and coagulative studies in women presenting with such complications.

Hematological abnormalities in case of PIH range from thrombocytopenia, consumption coagulopathy and hemolysis. Thus evaluation of peripheral smear with a special reference to red blood cell morphology, platelet morphology, aggregation and number are simple hematological parameters. Abnormal and premature forms of erythrocytes can identify microangiopathic hemolytic anemia cases which can progress to levels which require aggressive therapy.

Some biochemical parameters have been proposed like angiogenic markers, placental tissue protein 13(PP-13), soluble endoglin and soluble fms-like tyrosine kinase1 by some investigators. However, these techniques are not suitable for simple, low cost and rapid routine screening, there is a need to develop simple methods specifically designed for use in a hospital environment. Coagulation tests with special emphasis on D-dimer testing which can be used as a sensitive screening and follow-up tool for pre-eclamptic coagulopathy helping to define a subset of patients with severe disease. The D-dimer testing has been preferred over the test for fibrin degradation products (FDPs) as it has been established as a more sensitive tool for fibrinolysis. Thus this study is undertaken to know the hematological, biochemical and coagulative derangement in preeclampsia and further access the prognosis and severity of the complications and thus will help as a treatment guide for cases of preeclampsia and eclampsia.

OBJECTIVES

The objective of this study was

1. To know the clinical and diagnostic markers in patients with pregnancy induced hypertension.
2. To predict the severity of the disease based on these clinical and laboratory markers.

REVIEW OF LITERATURE

Historical perspectives

Hippocrates around 400 BC, stated that headache accompanied by heaviness and convulsions during pregnancy was considered bad which years later was known as "preeclampsia-eclampsia". [12] It was earlier considered as an entity associated with early pregnancy.

Bossier de Sauvages (1710-1795) was the first to use the term, "eclampsia", a Greek word meaning "lightning", as the convulsions arise suddenly and unexpectedly. [13]

In 1739 seizures of eclampsia was differentiated from epilepsy, eclampsia is acute in nature and would resolve once the precipitating event was removed. Demanet (1797) recognized the extreme swelling in eclamptic women and Pierre Rayer (1793-1867), a Frenchman, is considered the first to describe proteinuria in eclamptic. [14]

In 1800's headache, temporary loss of vision, severe pain in the stomach, and edema in the upper body were the symptoms which helped in recognition of pre-eclampsia.[12]

In (1896) introduction of mercury manometer by Scipione Riva-Rocci's to measure blood pressure that led to the recognition that preeclampsia was a hypertensive disorder.

JC Webster, in 1903, defined pre-eclampsia as the syndrome of hypertension, proteinuria, and edema, which was earlier called as "toxaemia". By 20th century, examinations of the placenta and other affected organs and newer technology to

examine smaller components of cells helped realize the role of spiral arteries, endothelial cells, antioxidants, antiangiogenic proteins, and the tendencies toward inflammation and other systemic dysfunctions.

Incidence

The incidence of Pregnancy induced hypertension (PIH) shows great variation, which may be due to differences in definition, population composition, demographic and obstetric characteristics, or actual disease incidence. (9)

The incidence of PIH appears to range from 5 % to 9 % for gestational hypertension and from 5 % to 7 % for preeclampsia among nulliparous women without chronic hypertensive disease or diabetes mellitus.

The incidence of PIH is 4-5 times higher in nulliparous women than that in multipara.

The incidence of PIH is distributed unevenly throughout late gestation, increasing with advancing gestation. Approximately half of PIH cases occur at term (>37 weeks gestation), including most cases of gestational hypertension. (8)

Worldwide, the incidence of preeclampsia ranges between 2% and 10% of pregnancies. (10) Incidence of preeclampsia is estimated by WHO to be seven times higher in developing countries (2.8% of live births) than in the developed countries (0.4 %).

The incidence of eclampsia is estimated to be about 5–7 cases per 10,000 deliveries in the developed countries of North America and Europe. However, in developing nations incidence of eclampsia varies widely ranging from 1 case per 100 pregnancies to 1 case per 1700 pregnancies. (11) Among the countries like South Africa, Egypt, Tanzania, and Ethiopia the incidence varies from 1.8% to 7.1%.[11] In Nigeria, prevalence ranges between 2% to 16.7%.

In India, the incidence of preeclampsia is reported to be 8-10% and prevalence of hypertensive disorders of pregnancy was 7.8%.

In Karnataka maternal mortality rate i.e. 103 per 100,000 live births. Thus Karnataka continues to remain at 11th position in the country and fifth among southern states in terms of maternal mortality rate.

Definitions and diagnostic criteria for Hypertensive Disorders of Pregnancy

As given by American college of Obstetrician and Gynecologist

1. **Hypertension**-is defined as increase in systolic blood pressure more than or equal to 30 mm Hg or increase in diastolic blood pressure more than or equal to 15 mm Hg or blood pressure more than or equal to 140/90 mm Hg on two occasions 6 hours apart. (1)

2. **Chronic hypertension** is identified if hypertension precedes pregnancy, is present at < 20 weeks gestation, or persists for > 6 weeks (usually > 12 weeks) postpartum (even if hypertension is first documented at > 20 weeks gestation). Physiologic decrease in BP during early pregnancy can mask chronic hypertension in patients.

3. **Severe Hypertension**-is defined as increase in systolic blood pressure more than or equal to 160 mm Hg or increase in diastolic blood pressure more than or equal to 110 mm Hg.

4. **Gestational hypertension** is hypertension without proteinuria or other findings of preeclampsia; it first occurs at > 20 weeks gestation in women known not to have hypertension before pregnancy and is corrected by 12 weeks (usually by 6 weeks) postpartum.

5. **Preeclampsia** is new-onset hypertension (BP > 140/90 mm Hg) plus new unexplained proteinuria (> 300 mg/24 hours or urine protein/creatinine ratio ≥ 0.3) after 20 weeks of pregnancy.

6. **Mild Preeclampsia** – is defined as increase in systolic blood pressure more than or equal to 140 mm Hg or increase in diastolic blood pressure more than or equal to 90 mm Hg, over 20 weeks of gestation (in a women with previously normal blood pressure) and presence of proteinuria more than or equal 0.3 g/24 hours (dipstick more than equal to 1+)

7. **Severe Preeclampsia** – is defined as increase in systolic blood pressure more than or equal to 160 mm Hg or increase in diastolic blood pressure more than or equal to 110 mm Hg,(on two occasions 6 hours apart in a women on bed rest) and presence of proteinuria more than or equal to 5g (dipstick more than equal to 3+)in 2 random urine sample collected 2 hours apart and presence of other complications like

a) Thrombocytopenia: Platelet count less than 100,000/microliter.
b) Oliguria: less than 500 mL of urine in 24 hours
c) Cerebral or visual disturbances
d) Pulmonary edema or cyanosis

e) Epigastric or right upper quadrant pain

f) Intrauterine growth restriction.

g) Impaired liver function: Elevated blood levels of liver enzymes to twice normal concentrations

h) Renal Insufficiency: Serum creatinine concentration greater than 1.1 mg/dL or a doubling of the serum creatinine concentration in absence of other renal disease.

i) New-onset headache unresponsive to medication and not accounted for by alternative diagnoses or visual symptoms.

8. **Eclampsia** -Preeclampsia patients with new-onset tonic-clonic, focal, or multifocal seizures in the absence of other causative conditions such as epilepsy, cerebral arterial ischemia and infarction, intracranial hemorrhage, or drug use. [15]

9. **Preeclampsia superimposed on chronic hypertension** is diagnosed when new unexplained proteinuria develops or proteinuria worsens after 20 weeks in a woman known to have hypertension with BP elevations above the baseline or in a woman known to have hypertension and proteinuria develops preeclampsia with severe features after 20 weeks of gestation.

10. **Proteinuria-**is defined as more than or equal 0.3 g/24 hours (dipstick 1+) on two occasions 6 hours apart.

11. **Severe Proteinuria-** is defined as more than or equal 2 g/24 hours (dipstick 2+) on two occasions 6 hours apart.

Risk Factors

A variety of risk factors have been associated with increased probability of preeclampsia the emerging data suggest some genetic component play a major role in development of preeclampsia however known risk factors for preeclampsia are

- **Nulliparity**
- **Maternal age 35 years or older**
- **Personal or family history of preeclampsia**
- **Race -**increased risk in black women
- **Obesity-** Pre pregnancy body mass index greater than 30
- **Multifetal gestations**
- **Chronic hypertension**
- **Gestational diabetes, pre gestational diabetes**
- **Interval between pregnancies**-pregnancy less than two years or more than 10 years apart leads to a higher risk of preeclampsia. [16]
- **New paternity-** each pregnancy with a new partner increases the risk of preeclampsia more than does a second or third pregnancy with the same partner.
- The patients associated with **obstructive sleep apnea**, **thrombophilia (antiphospholipid syndrome, protein C, S deficiency, factor V leiden), systemic lupus erythematosus**, **antiphospholipid antibody syndrome** ,**chronic kidney disease and assisted reproductive technology** have increased chances of development of pre-eclampsia.

According to the study done in Maharashtra, India by Bharti Anup Rathi et al, the most common associated factors were family history, previous history of PIH, Age >34, Primigravida, K/C/O hypertension. [35]

PATHOGENESIS

Several mechanisms of disease have been proposed in preeclampsia including the following: chronic uteroplacental ischemia, immune maladaptation, very low-density lipoprotein toxicity, genetic imprinting, increased trophoblast apoptosis or necrosis and an exaggerated maternal inflammatory response to expelled trophoblasts. [33]

The basic underlying pathology is **Endothelial dysfunction** and **intense vasospasm** because of interaction of various vasoactive agents such as prostacyclin (vasodilator), thromboxane A2 (potent vasoconstrictor), nitric oxide (potent vasodilator), and endothelin (potent vasoconstrictor) leads to decreased placental perfusion.

The immunogenic factors lead to inadequate trophoblastic invasion of maternal spiral arteries. The increased number of trophoblast along with above mentioned factors leads to decreased placental perfusion which may lead to placental production of endothelial toxins which terminate into generalized endothelial cell injury which may lead to

a) Increased vascular permeability leading to peripheral and pulmonary edema.
b) There will be decreased maternal production of PGI2 and other endogenous vasodilators which may cause increased sensitivity to all pressors and decreased renal renin secretion.[9] This will lead to arterial and venous vasoconstriction with decreased aldosterone secretion, which will lead to increased blood pressure and decreased plasma volume.

c) The generalized endothelial cell injury will also lead to activation of intravascular coagulation and fibrin deposition in the kidney leading to proteinuria and decreased GFR, in liver abnormal liver function test and abdominal pain, in central nervous system there maybe seizure and coma ,there will be consumptive coagulopathy and placental changes may manifest as abruption and intrauterine growth retardation.

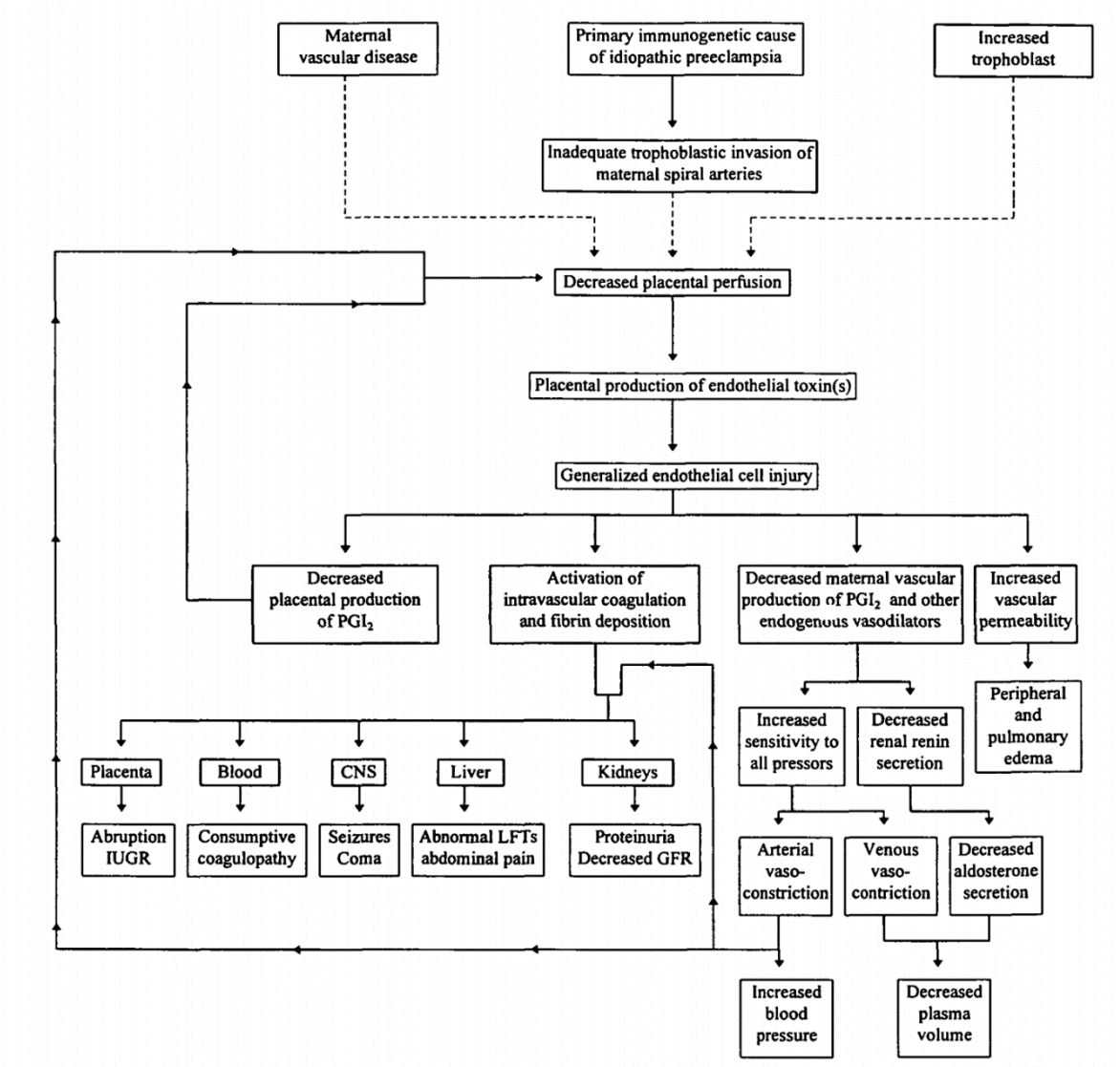


FIGURE 1:- MODEL OF THE PATHOPHYSIOLOGY OF PREECLAMPSIA

Clinical Presentation

Onset of Preeclampsia is insidious and runs a slow course. Rarely onset can become acute and follow a rapid course.

Preeclampsia is a syndrome of signs and symptoms which appear late.

Mild symptoms -mild ankle swelling which persist on rising from bed in the morning is early manifestations of edema due to preeclampsia. Gradually the swelling extends to the face, abdominal wall, vulva and then whole body.

Alarming symptoms- following ominous symptoms.

These are associated with acute onset of syndrome

1. Headache
2. Disturbed sleep.
3. Diminished urinary output -<400 ml/24hrs
4. Acute epigastric pain maybe associated with vomiting
5. Eye symptoms- blurring, scotoma, dimness of vision or at times complete blindness.

Signs

1. Abnormal weight gain within a short span of time. Rapid weight gain of >5 lb/month or >1 lb/week in later months of pregnancy is significant.
2. Rise in Blood Pressure- is insidious or abrupt. Diastolic blood pressure rises first followed by systolic blood pressure.

3. Edema-visible ankle edema on rising from bed in morning, sudden and generalized edema indicate imminent eclampsia.
4. Pulmonary edema
5. Abdominal edema- scanty liquor or growth retardation in fetus.

Order of manifestations of preeclampsia are:-

Rapid gain in weight ⟶ hypertension followed by visible edema ⟶ proteinuria.

Therefore, a proper diagnostic approach is required when signs and symptoms indicative of severe preeclampsia are absent in the setting of a clinical presentation similar to preeclampsia, alternative diagnoses like thrombotic thrombocytopenic purpura, hemolytic–uremic syndrome, molar pregnancy, renal disease or autoimmune disease to be considered at gestational ages earlier than 20 weeks.

Eclampsia is the convulsive manifestation of the hypertensive disorders of pregnancy it is one of the severe manifestations of the disease. Eclamptic patients always shows previous manifestations of acute fulminating preeclampsia, called as premonitory symptoms.

The eclamptic convulsions has four stages.

1. Premonitory stage
2. Tonic stage
3. Clonic stage
4. Stage of coma

Status eclampticus is term used when the fits are usually multiple, recurring at various intervals and occurs in quick succession.

COMPLICATIONS

Complications of preeclampsia may include:

Maternal complications :-

a) During pregnancy:- eclampsia ,accidental haemorrhage ,oliguria and anuria ,dimness of vision and even blindness, preterm labour, HELLP syndrome ,cerebral haemorrhage, acute respiratory distress syndrome.
b) During labour:- eclampsia, postpartum haemorrhage
c) Puerperium:- eclampsia, shock, sepsis

Fetal complication :-

a) Intrauterine death
b) Intrauterine growth restriction
c) Asphyxia
d) Prematurity

DIAGNOSIS

In the diagnosis of any disease, management is the most crucial step. In case of diagnosis of pregnancy induced hypertension, it is very important to recognize the nature of the disease. The clinical manifestations appear usually after the 20th week of gestation. Preeclampsia is principally a syndrome of signs and when symptoms appear, it is usually late. (1)

Regular antenatal screening is recommended to look for alarming signs and symptoms investigations should be carried to rule out the diagnosis of PIH.

The following laboratory tests can be performed -

- Complete blood count
- Urine examination
- Liver function tests
- Renal function test

Ophthalmoscopic examination in severe cases and Antenatal fetal monitoring for assessment of fetal well-being can done by clinical examination, daily fetal kick count, ultrasonography for fetal growth and liquor pockets, cardiotocography, umbilical artery flow velocimetry and biophysical profile

The reported hematologic findings in toxemia of thrombocytopenia, hemolysis, increased platelet adhesiveness, and increased FDPs are indicators compatible with intravascular coagulation. [(4)] There may be abnormal coagulation profile of varying degree and hepatic enzymes may be increased. Proteinuria is last feature of preeclampsia to appear.

A study in Egypt by Alkholy et al, the estimation of platelet indices was easy, reliable, economic and rapid method for detection of preeclampsia and assessment of severity.[(36)]

According to the study by Dhakre et al done in Madhya Pradesh, India the platelet count decreases while MPV and PDW increases as pregnancy advances, and these changes are more pronounced in preeclampsia than normotensive pregnancy.[(37)]

According to the study by Shweta Chaudhary et al done in Bhavnagar, India out of 100 patients with pregnancy induced hypertension, platelet count was decreased in 7.5% cases of mild preeclampsia,57% cases of severe preeclampsia and 100% cases of eclampsia. PT was prolonged in 28% cases of severe preeclampsia and 37.5%

cases of eclampsia. aPTT was prolonged in 2.8% cases of severe preeclampsia and 25% cases of eclampsia. Thus degree of thrombocytopenia increases with increased severity of disease. PT and aPTT are increased in PIH but do not differ with severity of PIH and are prolonged only in cases of thrombocytopenia. (38)

According to a study by Thakur Bhavana et al done in Raipur, India prolonged PT and aPTT are reliable indicators of consumptive coagulopathy seen in PIH. Of these aPTT is more reliable for clinical subgroups of preeclampsia. (39)

A study in Hubli, Karnataka, India by Jyothi Shetty et al, of the 200 patients diagnosed of PIH, D-dimer test is better than platelet count, PT, aPTT, fibrinogen and FDP in early screening and follow-up for coagulopathy in PIH and also help to define the subset of patients with severe disease. (4)

According to a study by Monteiro et al done in Mangalore, Karnataka, India platelet count may be a suitable marker in monitoring subjects with PIH at risk of IUGR and IUD. Anemia in PIH patients may be associated with IUD and IUGR. (40)

Blood values of serum uric acid level (biochemical marker of preeclampsia) of more than 4.5mg/dl indicates presence of preeclampsia. Blood urea level remains normal or slightly raised. Serum creatinine level maybe >1 mg/dl. According to a study by Jyothi Shetty et al mean AST was higher in HELLP cases compared to non-HELLP cases. However, there was no significant difference in mean bilirubin concentration between the mild and severe PIH groups.

A study in Mysore, Karnataka, India by Prathap T et al, concluded that test like serum LDH, uric acid, alkaline phosphatase and platelet count help to predict and deal with the adverse complications of PIH and are cost effective as well. (7)

METHODOLOGY

The present study was done from January 2020 – December 2020 in Jawaharlal Nehru Medical College and KLE's Dr. Prabhakar Kore Hospital and Research Centre, Belagavi.

Study design and duration

One year hospital based observational study.

Study period

January 2020 – December 2020

Place

The present study was done in the Department of Pathology in Jawaharlal Nehru Medical College and KLE's Dr. Prabhakar Kore Hospital and Research Centre, Belagavi.

Source of Data

All the patients clinically diagnosed with pregnancy induced hypertension at KLE's Dr. Prabhakar Kore Hospital and Medical research Centre, Belagavi and referred to the Department of pathology and Department of biochemistry for hematological, biochemical and coagulative studies in PIH cases.

Sample size

According to the previous three years data an average of 160 patients diagnosed with PIH at Dr. Prabhakar Kore Charitable hospital Belgaum and referred to the department of pathology and department of biochemistry for hematological, biochemical and coagulative studies.

Sampling procedure

Universal sampling that is all the pregnant female clinically diagnosed of having PIH.

Inclusion criteria

All the patients with gestation age ranging from 20 weeks till term and clinically diagnosed of having PIH.

Exclusion criteria

1.Gestational Hypertension- BP> 140/90 mmHg for the first time in pregnancy after 20 weeks, without proteinuria.[1]

2.Chronic Hypertension- known hypertension before pregnancy or hypertension diagnosed first time before 20 weeks of pregnancy.[1]

3. History of systemic illnesses like diabetes mellitus, renal disease, liver diseases.

Ethical clearance

Obtained from the Institutional Ethical Committee, J N Medical College, Belagavi.

Method of collection of data

Blood and Urine samples were collected from clinically diagnosed PIH patients at Dr. Prabhakar Kore Charitable hospital Belgaum. The patients were education about the procedure and an informed consent was taken. The collected samples were labeled with the sample number, date and time of collection and processed in the department of pathology and department of biochemistry for hematological, biochemical and coagulative studies in PIH cases.

The blood count was performed on fully automated hematology analyzer.

The biochemical investigations for liver and renal involvement were performed on automated biochemistry analyzer.

Investigations

The data regarding the following investigations of the selected patients clinically diagnosed Pregnancy induced hypertension documented in predesigned proforma.

- Complete hemogram
- Total bilirubin
- SGOT and SGPT
- Alkaline phosphatase
- LDH
- Serum urea
- Serum creatinine
- Serum uric acid
- Coagulation test
- D-dimer

Patient profile

The patient history including period of gestation, presenting complaints, history of present illness, history of present pregnancy, obstetric history, menstrual history, past medical/surgical history, family history, personal history including general physical examination build, nutrition, pallor, jaundice, oedema of legs, pulse, blood pressure systemic and obstetrical examination.

Hematological profile

Based on the case sheet the hematological profile was assessed and abnormalities like hemolysis and thrombocytopenia were looked for based on hemoglobin levels, total count, hematocrit levels and platelet count.

Liver profile

The liver profile was assessed by evaluating total bilirubin, SGOT and SGPT, alkaline phosphatase levels.

Renal profile

The assessment of renal profile was based on serum urea , serum creatinine and serum uric acid levels.

Urine examination

Proteinuria was determined by complete urinary examination including physical, biochemical and microscopic examination.

Coagulation profile

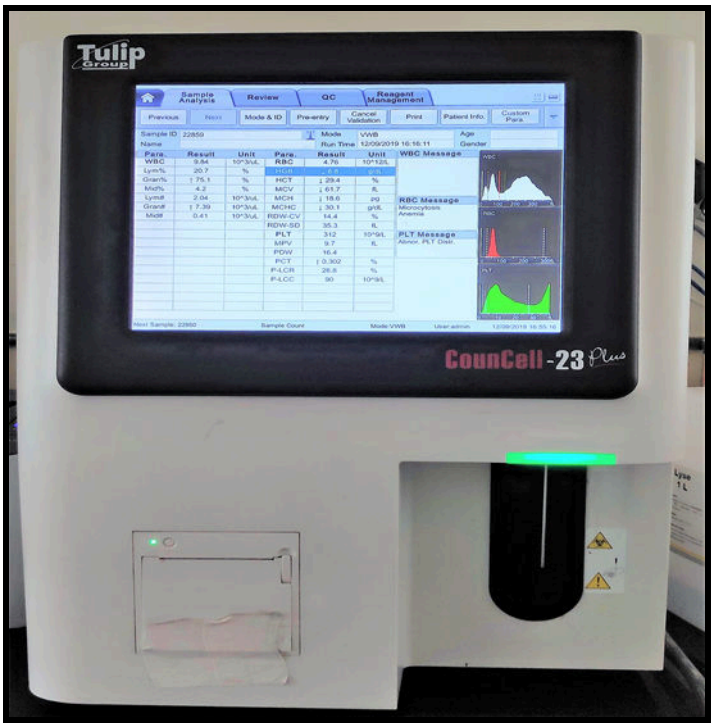
The assessment of consumptive coagulopathy was done by prothrombin time, activated partial thromboplastin time, thrombin time, D-dimer, fibrinogen.

Statistical analysis

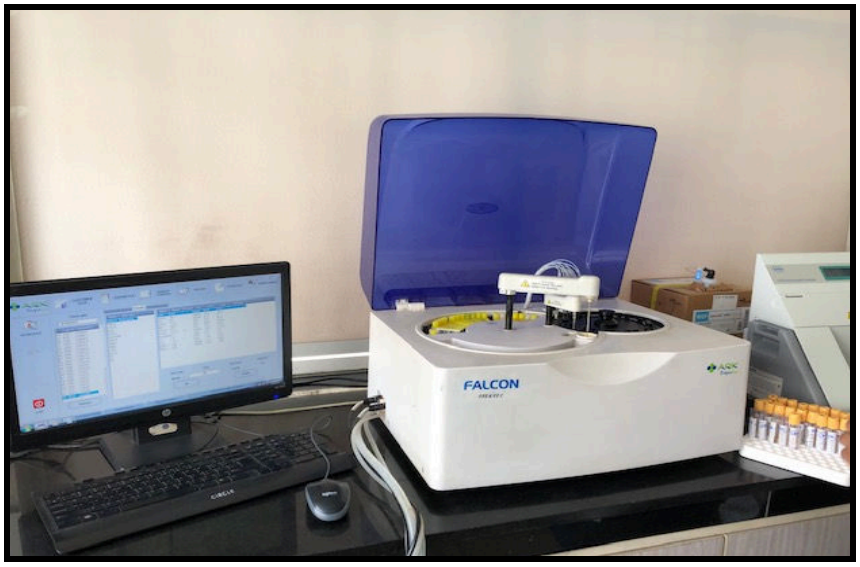
The data achieved was coded and entered into Microsoft Excel. Association between variables was determined using “chi-square test” or “Fisher’s exact test” and “Spearman’s correlation coefficient”. The continuous data was expressed as mean ± standard deviation (SD). A probability value i.e, ‘p’ value of equal to or less than 0.05 was considered as significant statistically.

Photograph 1: Equipments used for routine haematological investigations

Photograph 2: Sysmex Hematology Analyzer XN- 350



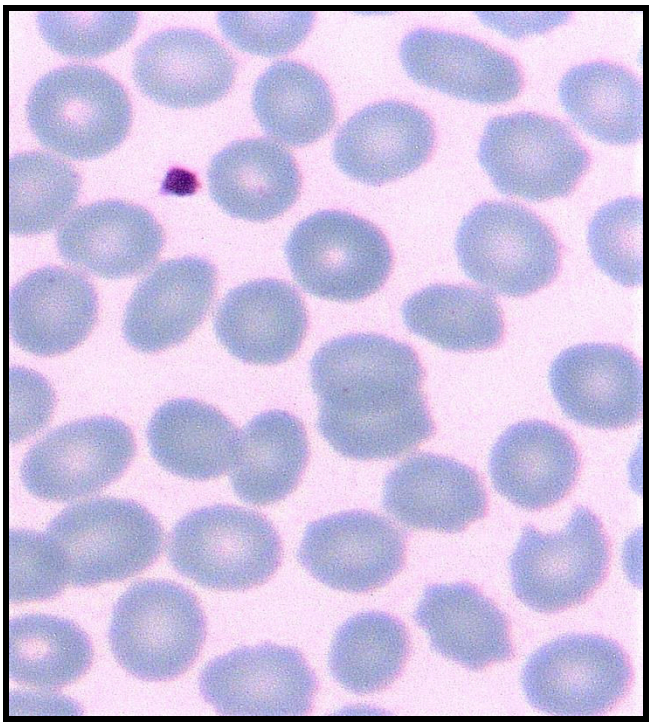
Photograph 3: Tulip Fully Automatic 5-Part Haematology Analyzer, Councell – 23 V2



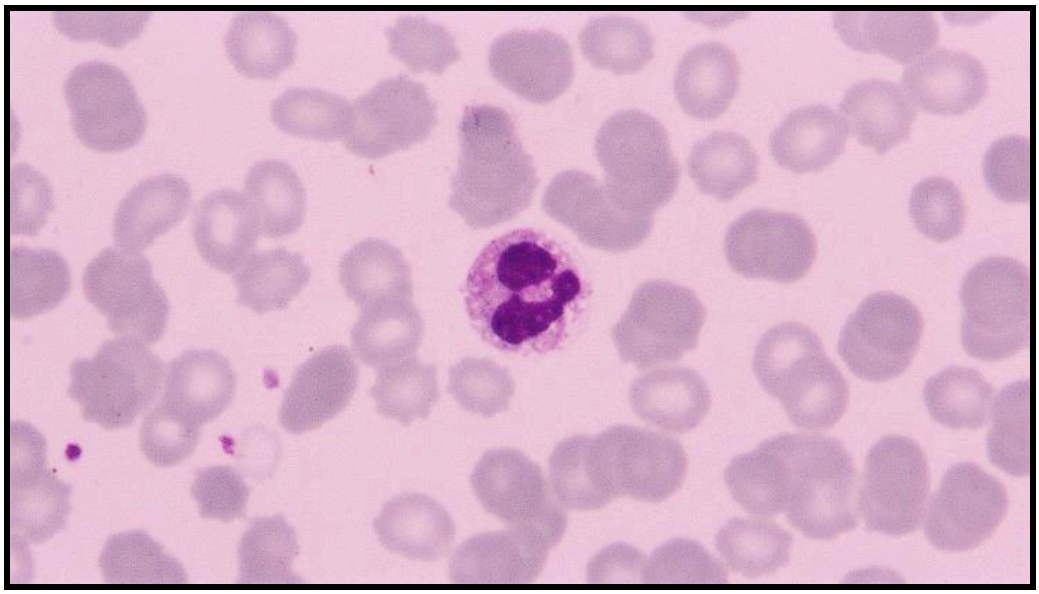
Photograph 4: Falcon mini- Fully Automated Random Access Chemistry Analyzer used for the estimation of LFT and RFT

Photograph 5: Instrumentation Laboratory's ACL Top 300/500 used for estimation of PT, aPTT, D-dimer, fibrinogen levels

Photograph 6: Mission U 120 Smart used for estimation of urine protein.



Photograph 7: Wright's stain photomicrograph showing Normocytic Normochromic anaemia with thrombocytopenia and leucopenia (100x)



Photograph 8: Wright's stain photomicrograph showing Normocytic normochromic anaemia with thrombocytopenia (100x)

RESULTS

The present study is an observational study of one year, conducted in the Department of Pathology, JNMC and KLE's Dr. Prabhakar Kore Hospital and Research Centre, Belagavi. A total of 160 patients clinically diagnosed with pregnancy induced hypertension from January 2020 – December 2020 were studied.

Of the 160 cases:

73 cases had **mild PIH** (diastolic BP ≥90 mmHg)

52 were **severe PIH** (diastolic blood pressure [BP] ≥110 mmHg)

27 cases were of **eclampsia**

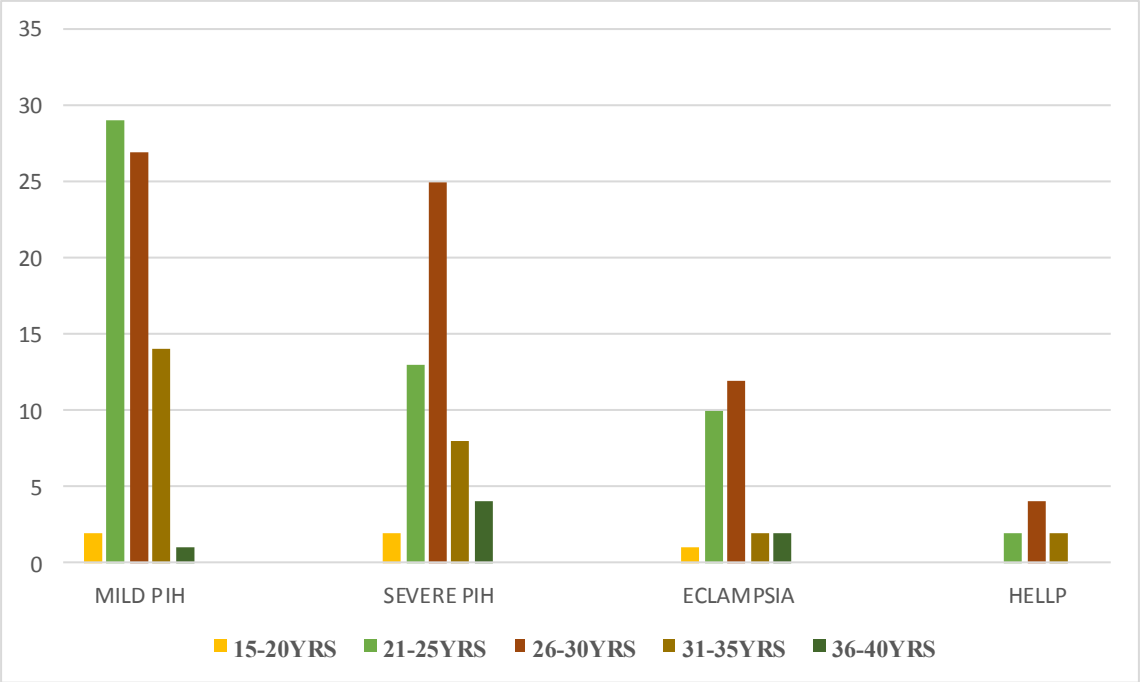
8 cases had features of **HELLP** syndrome.

The data obtained was analysed and the observations are interpreted below.

TABLE 1: AGE DISTRIBUTION IN PIH PATIENTS.

AGE DISTRIBUTION OF PIH CASES				
AGE GROUP	**MILD PIH**	**SEVERE PIH**	**ECLAMPSIA**	**HELLP**
15-20YRS	2	2	1	0
21-25YRS	29	13	10	2
26-30YRS	**27**	**25**	**12**	**4**
31-35YRS	14	8	2	2
36-40YRS	1	4	2	0
TOTAL NO OF PATIENTS	73	52	27	8

GRAPH 1:-AGE DISTRIBUTION IN PIH PATIENTS.

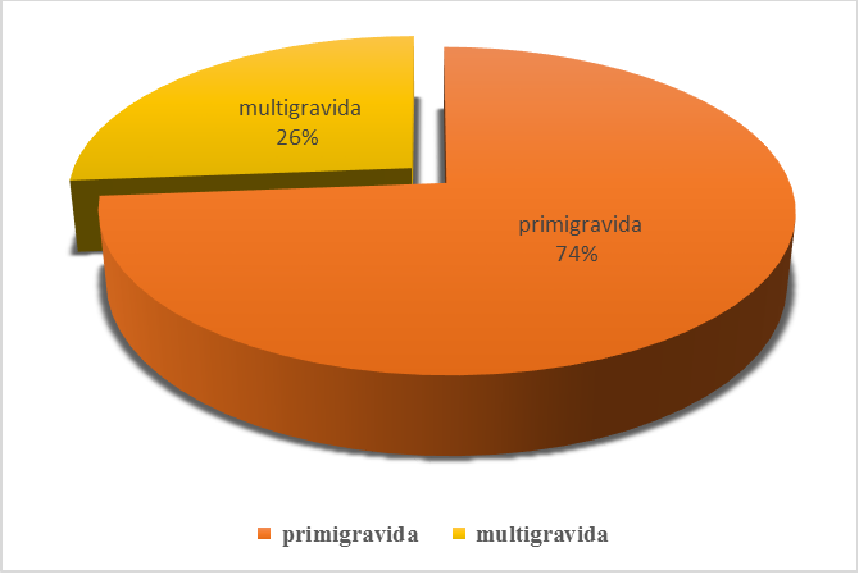


Of the 160 cases maximum cases were seen in mild PIH (29 cases) between 21-25 years whereas in severe PIH (25 cases), eclampsia (12 cases) and HELLP (4 cases) were seen between the age group of 26 to 30 this probably indicates that severity of complications increases with the age of patient. The increase in age with the severity of PIH was found to be statistically significant (p value of age =0.0008).

TABLE – 2 : GRAVID STATUS OF PIH PATIENT.

GRAVID	NUMBER OF PATIENTS	PERCENTAGE
Primigravida	118	73%
Multigravida	42	26%
Total	160	100%

GRAPH 2: GRAVID STATUS OF PIH PATIENTS

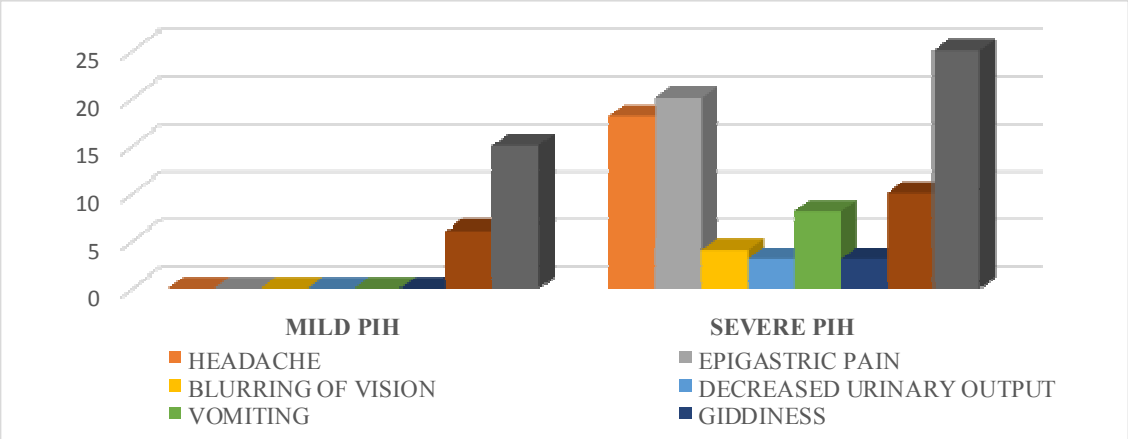


Of the 160 PIH cases, 118(73%) were primigravida and 42 (26%) were multigravida. Thus cases of preeclampsia, eclampsia and HELLP syndrome was found to be more common in primigravida.

TABLE 3: SYMPTOMATOLOGY OF PIH PATIENTS.

SYMPTOMS	MILD PIH	SEVERE PIH
HEADACHE	0	18
EPIGASTRIC PAIN	0	20
BLURRING OF VISION	0	4
DECREASED URINARY OUTPUT	0	3
VOMITING	0	8
GIDDINESS	0	3
PREVIOUS H/O PIH	6	10
FAMILY H/O PIH	15	25

GRAPH 3: SYMPTOMATOLOGY OF PIH PATIENTS.



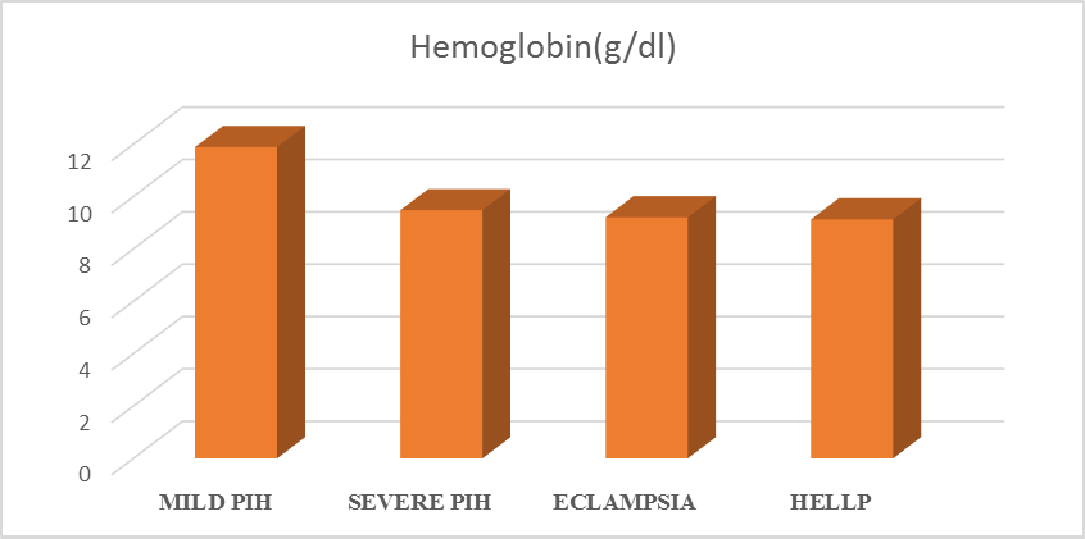
Cases with mild PIH were mostly asymptomatic, whereas all cases of severe PIH were symptomatic with epigastric pain being the predominant symptom present in 20 cases followed by headache in 18, vomiting in 8, blurring of vision in 4, reduced urine output in 3 and giddiness in 3 cases. Previous history of mild PIH was positive in 6 cases, severe PIH was positive in 10 cases and 8 cases were positive for eclampsia. Thus 15% of the cases had a significant past history of PIH. Family history of mild PIH was positive in 15 cases, severe PIH was positive in 25 cases and eclampsia was positive in 4 cases. Thus family history was important in 27.5% cases.

HEMATOLOGICAL INVESTIGATIONS:

TABLE 4: HAEMOGLOBIN LEVELS IN PIH PATIENTS.

SEVERITY OF PIH	MEAN HEMOGLOBIN (g/dl)
MILD PIH	11.89
SEVERE PIH	9.48
ECLAMPSIA	9.21
HELLP	9.13

GRAPH 4: HAEMOGLOBIN LEVELS IN PIH PATIENTS.

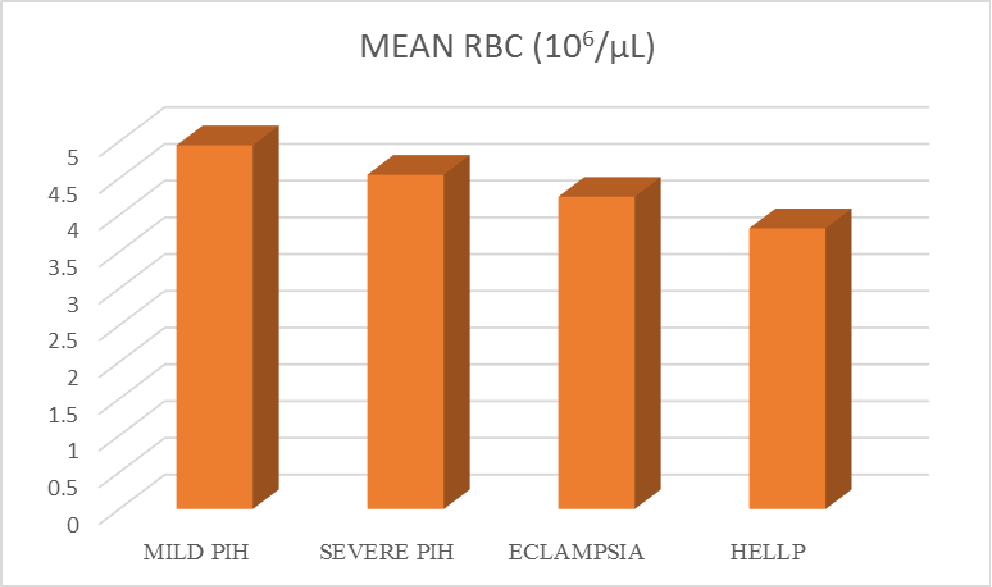


Hemoglobin levels of all these 160 patients varied from 7.1 to 15.5 g/dl , the overall mean being 10.92g/dl . The mean hemoglobin of mild PIH cases was 11.89 g/dl while that of severe PIH was 9.48 g/dl, mean hemoglobin of eclampsia and HELLP was 9.21 g/dl and 9.13 g/dl respectively. The decrease in hemoglobin levels with the severity of PIH was found to be statistically significant (p value of hemoglobin =0.0024).

TABLE 5: MEAN RBC COUNT IN PIH PATIENTS.

SEVERITY OF PIH	MEAN RBC (10^6/µL)
MILD PIH	4.95
SEVERE PIH	4.55
ECLAMPSIA	4.25
HELLP	3.82

GRAPH 5 : MEAN RBC COUNT IN PIH PATIENTS.

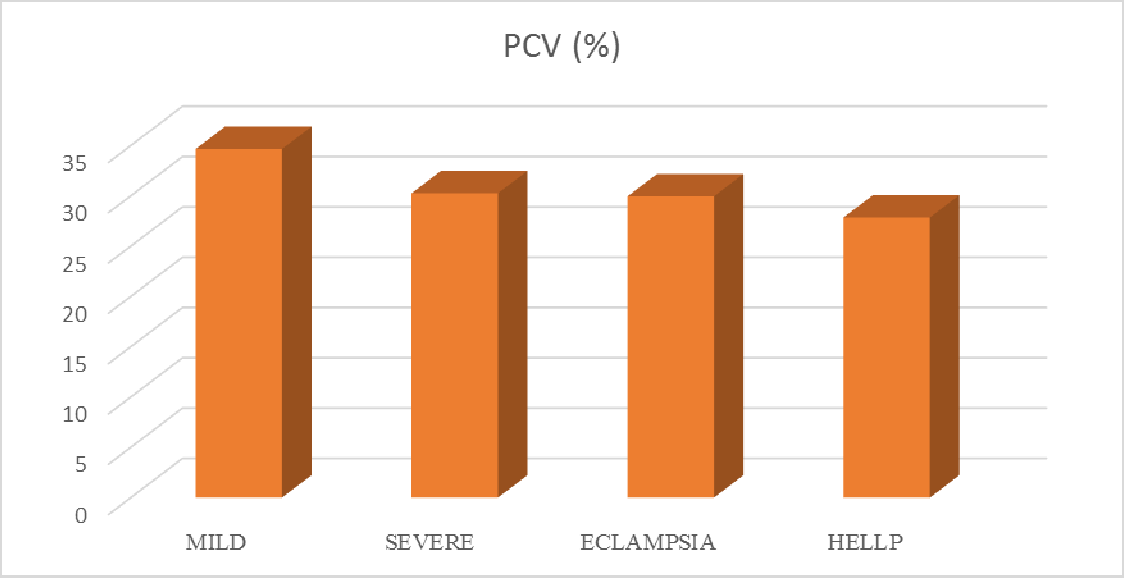


The mean RBC count of mild PIH cases was 4.95 x 10^6/µL while that of severe PIH was 4.55x10^6/µL, mean RBC count of eclampsia and HELLP was 4.25 10^6/µL and 3.82 10^6/µL respectively.

TABLE 6 : MEAN PCV VALUES IN PIH PATIENTS.

SEVERITY OF PIH	MEAN PCV (%)
MILD	34.66
SEVERE	30.26
ECLAMPSIA	29.95
HELLP	27.85

GRAPH 6: MEAN PCV VALUES IN PIH PATIENTS.

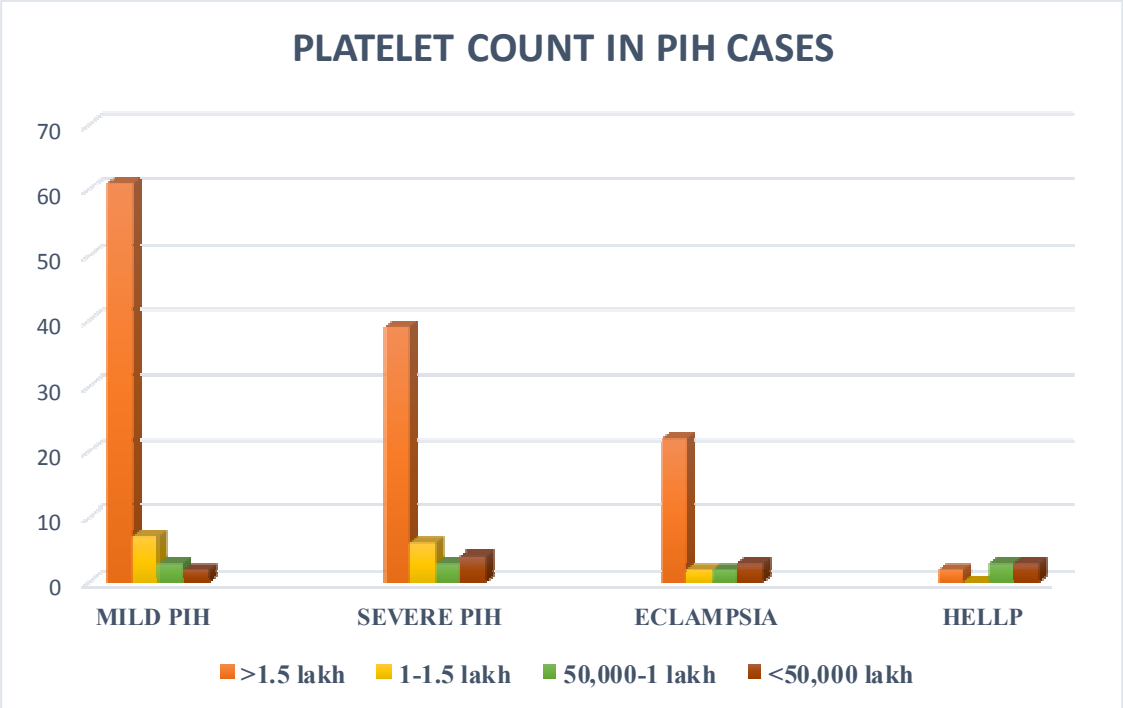


The mean PCV value of mild PIH cases was 34.66 % while that of severe PIH was 30.26 %, mean PCV value of eclampsia and HELLP was 29.95 % and 27.85 % respectively.

TABLE 7 : PLATELET COUNT IN PIH PATIENTS

PLATELET COUNT	MILD PIH	SEVERE PIH	ECLAMPSIA	HELLP
>1.5 lakhs (normal count)	40	25	15	0
1-1.5 lakhs (mild thrombocytopenia)	23	15	4	2
50,000-1 lakhs (moderate thrombocytopenia)	7	9	5	3
<50,000 lakhs (severe thrombocytopenia)	3	3	5	3

GRAPH 7: PLATELET COUNT IN PIH PATIENTS



Of the 160 PIH cases, 80 cases showed normal platelet count and 80 cases showed deranged platelet count.

Of these 73 cases of mild PIH, 23 cases showed mild thrombocytopenia, 7 cases showed moderate thrombocytopenia, and 3 cases had severe thrombocytopenia.

In the 52 cases of severe PIH, 15 cases had mild thrombocytopenia, 9 cases showed moderate thrombocytopenia, and 3 cases had severe thrombocytopenia.

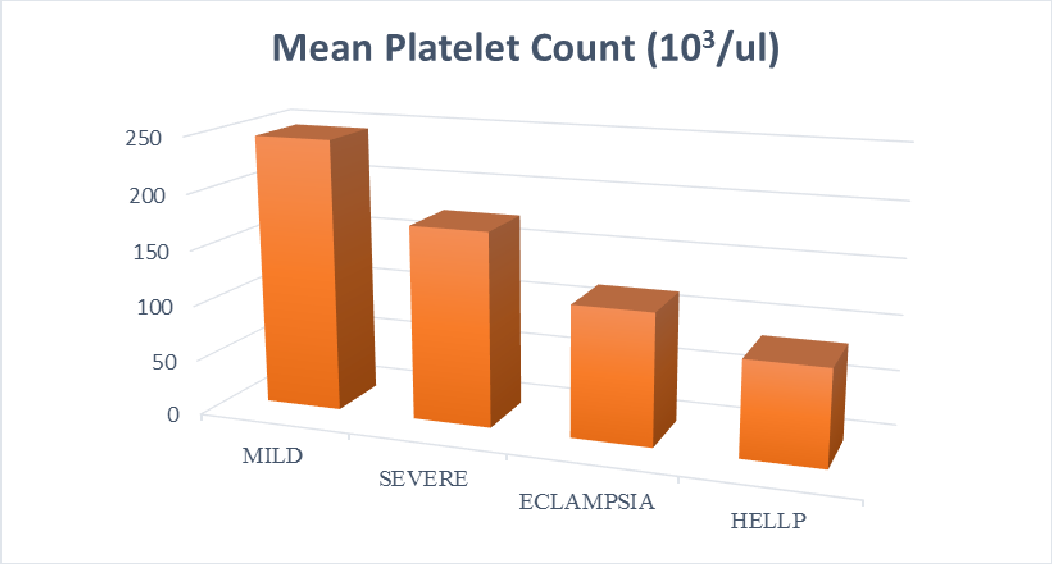
Of the 27 cases of eclampsia, 4 cases showed mild thrombocytopenia, 5 showed moderate thrombocytopenia, and 5 cases were severe thrombocytopenia.

8 cases of HELLP syndrome, all had thrombocytopenia i.e. 2 cases showed mild thrombocytopenia, 3 cases showed moderate thrombocytopenia, and 3 cases had severe thrombocytopenia.

TABLE 8: MEAN PLATELET COUNT IN PIH PATIENTS.

SEVERITY OF PIH	MEAN PLATELET COUNT ($10^3/\mu L$)
MILD	244.93
SEVERE	174.76
ECLAMPSIA	118.07
HELLP	86.125

GRAPH 8: MEAN PLATELET COUNT IN PIH PATIENTS.

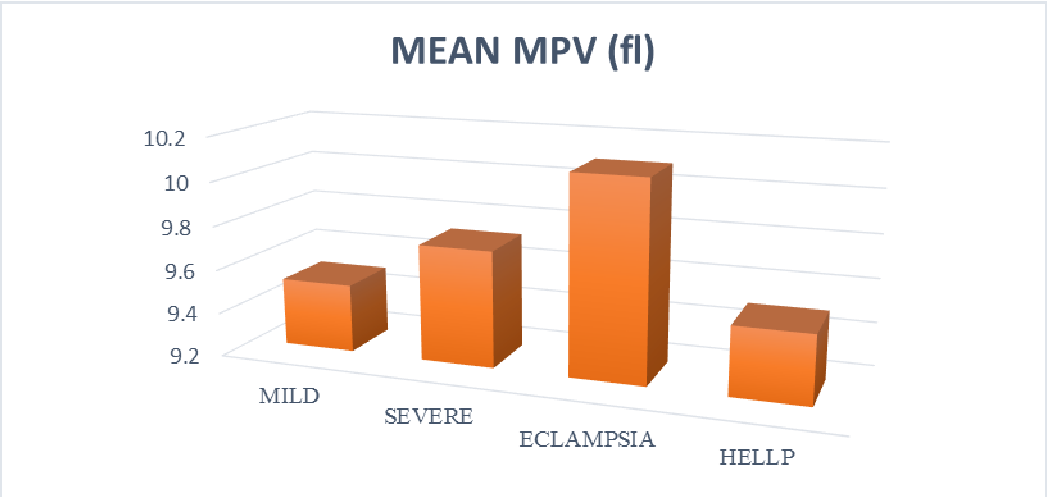


The mean platelet count among the mild pre-eclampsia and severe pre-eclampsia cases was 244.93 x $10^3/\mu L$ and 174.77 x$10^3/\mu L$ respectively. In case of eclampsia the mean platelet count was 118.07 x $10^3/\mu L$ and in case of HELLP the mean platelet count was 86.125 x$10^3/\mu L$. The platelet count decreased significantly with the increase in severity of pregnancy induced hypertension. The decrease in platelet count with the severity of PIH was found to be statistically significant p value of platelet count =0.0001

TABLE 9: MEAN MPV IN PIH PATIENTS

SEVERITY OF PIH	MEAN MPV (fl)
MILD	9.51
SEVERE	9.73
ECLAMPSIA	10.1
HELLP	9.51

GRAPH 9: MEAN MPV IN PIH PATIENTS



Of 160 cases mean platelet volume (MPV) in mild PIH was 9.51fl , mean platelet volume (MPV) of severe PIH was 9.73 fl and HELLP was 9.51 fl and mean platelet volume (MPV) of eclampsia was 10.1fl

ESR of these patients ranged from 30 to 140 mm/h The mean ESR value was 60 mm/h. Total leukocyte count varied from 4000 to 25,000 cells/mm^3. The mean of total leukocyte count being 12,000 cells/mm^3.

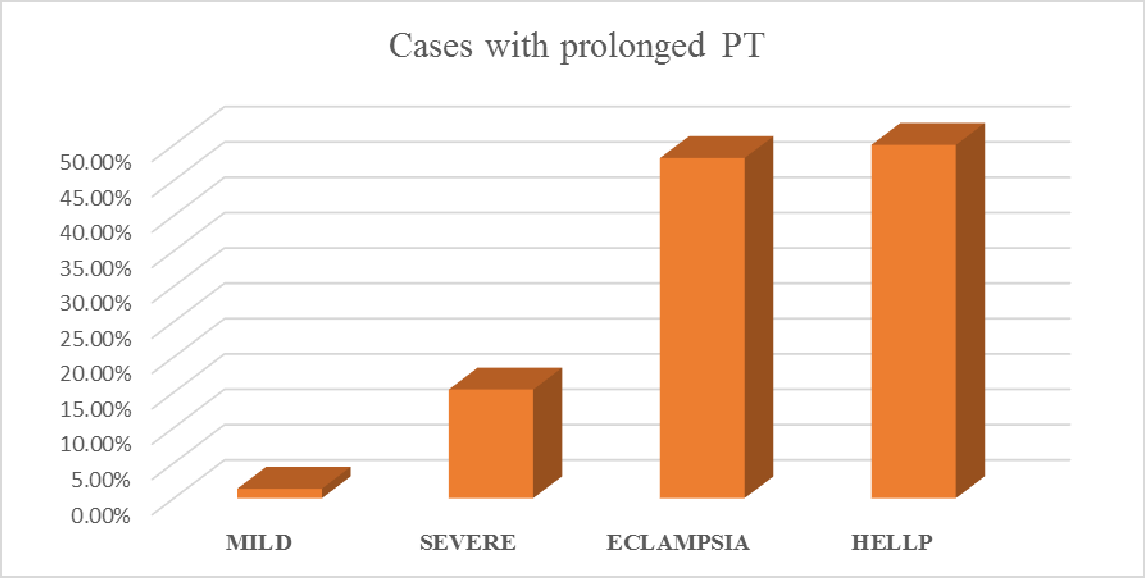
The **Reticulocyte Count** varied between 0.5% and 8%. It was raised (>2.5%) in 10 cases (13.69%) of mild PIH and 18 cases (34.66%) of severe PIH and 15 cases (55.56%) of eclampsia and all cases (100%) of HELLP syndrome indicating hemolysis.

COAGULATIVE PROFILE –

TABLE 10: PROLONGED PT IN PIH PATIENTS

SEVERITY OF PIH	CASES WITH PROLONGED PT
MILD	1 (1.36%)
SEVERE	8 (15.38%)
ECLAMPSIA	13 (48.14%)
HELLP	4 (50%)
TOTAL	26

GRAPH 10: PROLONGED PT IN PIH PATIENTS



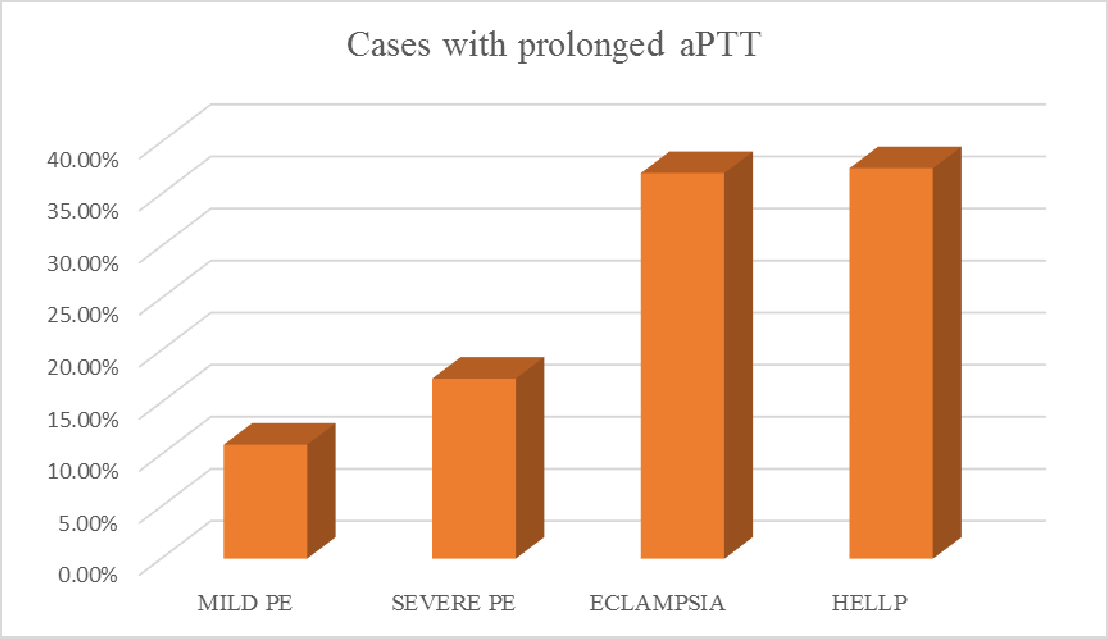
Prolonged PT was seen in 1case (1.36%) of mild preeclampsia, 8 cases (15.38 %) of severe preeclampsia, 13 cases (48.14%) of eclampsia and 4 cases (50%) of HELLP syndrome.

The increase in PT with the severity of PIH was found to be statistically significant. (p value of PT=0.0017*).

TABLE 11: PROLONGED aPTT IN PIH PATIENTS

SEVERITY OF PIH	CASES WITH PROLONGED APTT
MILD PE	8 (10.95%)
SEVERE PE	9 (17.30%)
ECLAMPSIA	10 (37.03%)
HELLP	3 (37.5%)
TOTAL	30

GRAPH 11: PROLONGED aPTT IN PIH PATIENTS

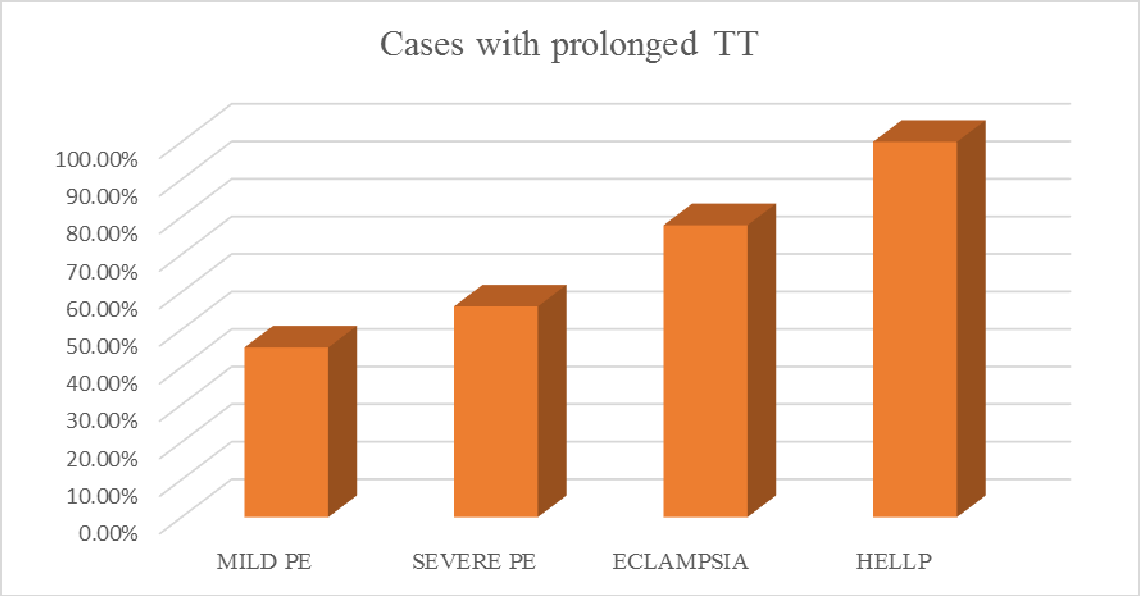


Prolonged aPTT was seen in 8 cases (10.95%) of mild preeclampsia, 9 cases (17.30 %) of severe preeclampsia, 10 cases (37.03%) of eclampsia and 3 cases (37.5%) of HELLP syndrome The increase in aPTT level with the severity of PIH was found to be statistically significant. (p value of aPTT= 0.0002*).

TABLE 12: PROLONGED TT IN PIH PATIENTS

SEVERITY OF PIH	CASES WITH PROLONGED TT
MILD PE	33 (45.20%)
SEVERE PE	32 (56.14%)
ECLAMPSIA	21 (77.78%)
HELLP	8 (100%)
TOTAL	94

GRAPH 12: PROLONGED TT IN PIH PATIENTS



Prolonged TT was seen in 33 cases (45.20%) of mild preeclampsia, 32 cases (56.14 %) of severe preeclampsia, 21 cases (77.78%) of eclampsia and 8 cases (100%) of HELLP syndrome

The increase in TT with the severity of PIH was found to be statistically significant. (p value of TT=0.0001*).

TABLE 13: D-DIMER LEVELS IN MILD PIH, SEVERE PIH, ECLAMPSIA AND HELLP SYNDROME

	Number of cases with increased D-dimer (ng/ml)				
	MILD PIH (73 cases)	**SEVERE PIH** (52 cases)	**ECLAMPSIA** (27 cases)	**HELLP** (8 cases)	Total 160 cases
>457 ng/ml in second trimester	18(24.65%)	15(28.84%)	8 (29.62%)	1(12.5%)	42 cases (26.25%) in second trimester
>644 ng/dl in third trimester	32(43.83%)	30(57.69%)	12(44.44%)	6(75%)	80 cases (50%) in third trimester
TOTAL	50(68.48%)	45(86.53%)	20(74.06%)	7(87.5%)	**Total 122 cases(76.25%)**

Normal D-dimer value in first trimester of pregnancy is upto 286 ng/ml, in second trimester of pregnancy is upto 457 ng/ml, and in third trimester of pregnancy upto 644 ng/ml.

Total 122 cases(76.25%) showed raised D-dimer values. The D- dimer was raised in 42 cases of second trimester. The values were significantly raised in 80 cases of third trimester. The increase in d-dimer values with the severity of PIH was found to be statistically significant. (p value for D-dimer=0.0001*).

TABLE 14: FIBRINOGEN LEVELS IN MILD PIH, SEVERE PIH, ECLAMPSIA AND HELLP SYNDROME

	Number of cases with increased fibrinogen (mg/dl)				
	MILD PIH (73 cases)	**SEVERE PIH** (52 cases)	**ECLAMPSIA** (27 cases)	**HELLP** (8 cases)	Total 160 cases
>853 mg/dl in second trimester	10(13.69%)	12 (23.07%)	6 (22.23%)	1(12.5%)	29 cases (18.13%) in second trimester
>914 mg/dl in third trimester	28(38.36%)	25 (48.07%)	10(37.03%)	5(62.5%)	68 cases (42.5%) in third trimester
TOTAL	38(52.05%)	37(71.15%)	16(59.25%)	6(75%)	**Total 97 cases (60.62%)**

The normal fibrinogen reference value range in first trimester of pregnancy is 264-656 mg/dl, in second trimester of pregnancy is 340-853 mg/dl, and in third trimester of pregnancy 363-914 mg/dl.

Total 97 cases (60.62%) showed raised fibrinogen values. The fibrinogen was raised in 29 cases of second trimester. The values were significantly raised in 68 cases of third trimester. The increase in fibrinogen values with the severity of PIH was not found to be statistically significant. (p value for fibrinogen =0.1306).

BIOCHEMICAL EXAMINATION –

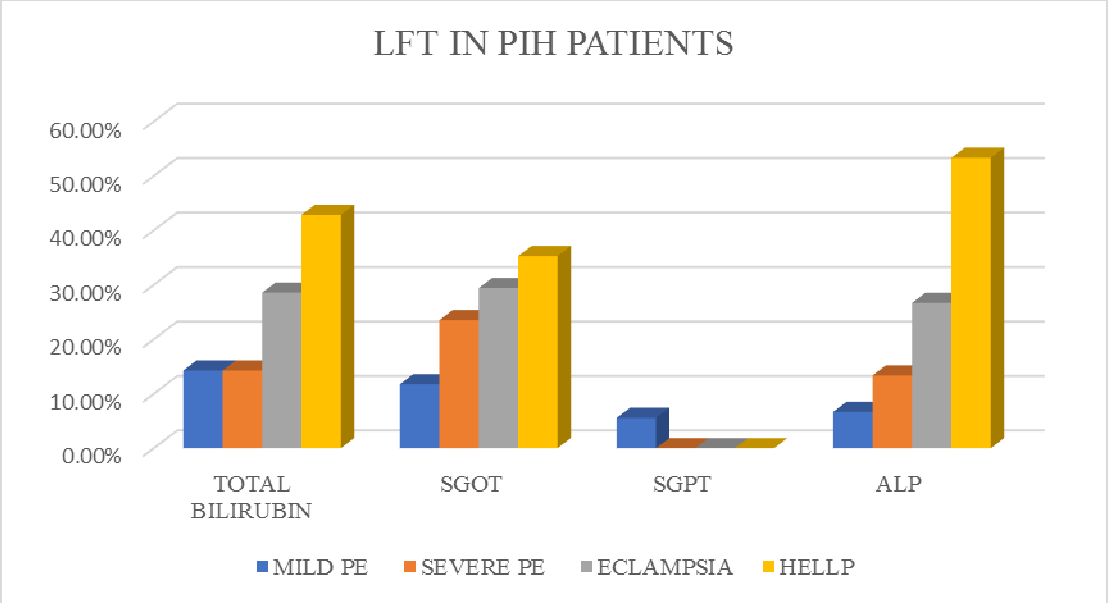
Total of 160 cases showed proteinuria of varying range. Mild proteinuria more than or equal 0.3 g/24 hours (dipstick 1+) was seen all 73 cases (45.63%) of mild PIH while severe proteinuria more than or equal 2 g/24 hours (dipstick 2+) was seen in 75 cases (46.88%) of these 52 cases of severe PIH and 3 cases of eclampsia and HELLP had 3+ proteinuria, 24 cases of eclampsia and 5 cases of HELLP had 4+proteinuria. Increase in proteinuria with increase in severity of PIH was statistically significant (p value of 0.0001*).

TABLE 15: DISTRIBUTION OF SEVERITY OF PREGNANCY INDUCED HYPERTENSION CASES ACCORDING TO LIVER FUNCTION TESTS

TOTAL BILIRUBIN (0.1-1.1 mg/dl)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	1	14.28%
SEVERE PE	1	14.28 %
ECLAMPSIA	2	28.57%
HELLP	3	42.85%
TOTAL	7	100%
SGOT (4-33U/L)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	2	11.76%
SEVERE PE	4	23.52%
ECLAMPSIA	5	29.42%
HELLP	6	35.29%
TOTAL	17	100%
SGPT (3-33U/L)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	1	5.57%
SEVERE PE	4	22.23%
ECLAMPSIA	6	33.37%

HELLP	7	38.87%
TOTAL	18	100%
ALP (38-229U/L)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	1	6.67%
SEVERE PE	2	13.37%
ECLAMPSIA	4	26.67%
HELLP	8	53.33%
TOTAL	15	100 %

GRAPH 13: DISTRIBUTION OF SEVERITY OF PREGNANCY INDUCED HYPERTENSION CASES ACCORDING TO LIVER FUNCTION TESTS



Total bilirubin values was raised in 7 cases predominant being in HELLP 3 (42.85%) followed by eclampsia 2 cases (28.57%). The increase in total bilirubin values with the severity of PIH was not found to be statistically significant. (p value =0.8913).

The SGOT values was raised in 17 cases with majority being in HELLP 6 (35.29%) followed by eclampsia 5 cases (29.42%) The increase in SGOT values with the severity of PIH was found to be statistically significant. (p value=0.0099*).

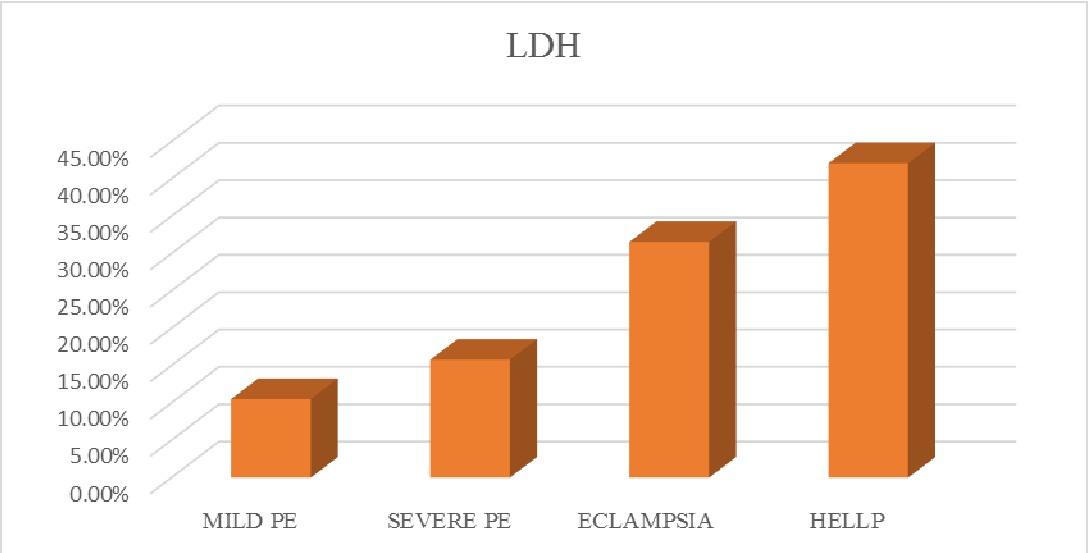
18 cases had raised SGPT values with predominance seen in HELLP 7 (38.87%) followed by eclampsia 6 cases (33.37%). The increase in SGPT values with the severity of PIH was not found to be statistically significant. (p value =0.1114).

15 cases showed raised ALP values predominantly raised in HELLP 8 (53.33%) followed by eclampsia 4 cases (26.67%). The increase in ALP values with the severity of PIH was not found to be statistically significant. (p value =0.9519).

TABLE 16: DISTRIBUTION OF SEVERITY OF PREGNANCY INDUCED HYPERTENSION CASES ACCORDING TO LDH LEVELS

LDH (200-400 IU/L)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	2	10.52%
SEVERE PE	3	15.78%
ECLAMPSIA	6	31.57%
HELLP	8	42.10 %
TOTAL	19	100%

GRAPH 14: DISTRIBUTION OF SEVERITY OF PREGNANCY INDUCED HYPERTENSION CASES ACCORDING TO LDH LEVELS

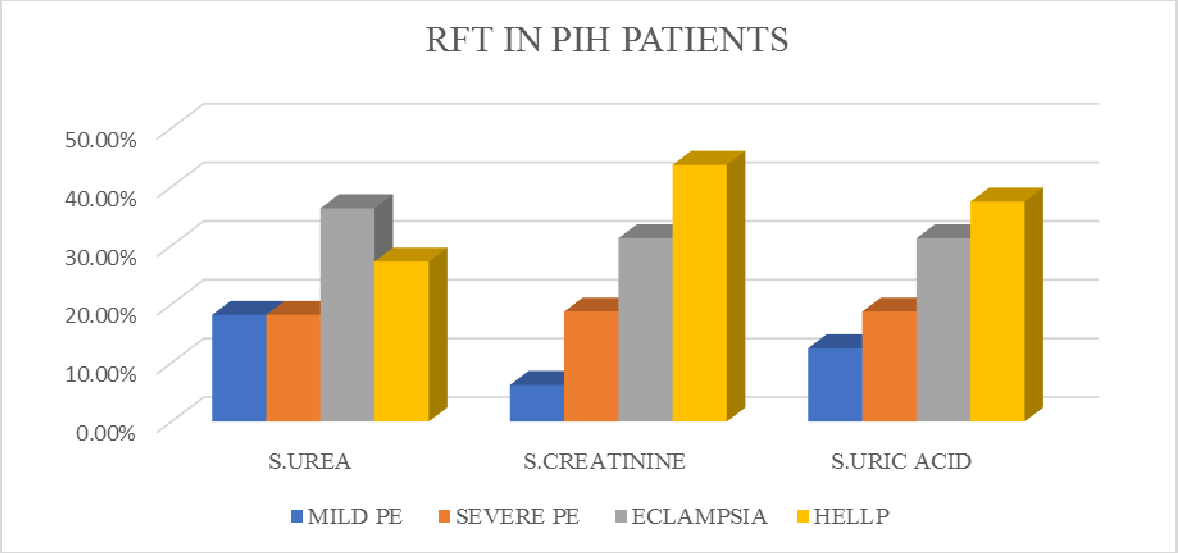


The present study showed raised LDH in 19 patients of these maximum cases were of HELLP 8 cases (42.10%) followed by eclampsia 6 cases (31.57%). The increase in LDH values with the severity of PIH was found to be statistically significant. (p value =0.0011*).

TABLE 17: DISTRIBUTION OF SEVERITY OF PREGNANCY INDUCED HYPERTENSION CASES ACCORDING TO RENAL FUNCTION TESTS

SERUM UREA (19-44 mg/dl)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	2	18.18%
SEVERE PE	2	18.18%
ECLAMPSIA	4	36.36%
HELLP	3	27.27%
TOTAL	11	100 %
SR. CREATININE (0.4mg/dl)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	1	6.25%
SEVERE PE	3	18.75%
ECLAMPSIA	5	31.25%
HELLP	7	43.75%
TOTAL	16	100%
SR. URIC ACID (3.5-7.2mg/dl)	**ELEVATED IN NUMBER OF CASES**	**PERCENTAGE**
MILD PE	2	12.5 %
SEVERE PE	3	18.75%
ECLAMPSIA	5	31.25 %
HELLP	6	37.5%
TOTAL	16	100%

GRAPH 15: DISTRIBUTION OF SEVERITY OF PREGNANCY INDUCED HYPERTENSION CASES ACCORDING TO RENAL FUNCTION TESTS



11 cases showed increased serum urea values out of these maximum cases were of eclampsia 3 (36.36%) followed by HELLP 4 cases (27.27%). Increase in serum urea values with the severity of PIH was not found to be statistically significant. (p value =0.5173).

Serum creatinine was raised in 16 cases predominantly in HELLP 7 cases (43.75%) followed by eclampsia 5 cases (31.25%). The increase in serum creatinine values with the severity of PIH was found to be statistically significant. (p value =0.0236*).

6 cases showed increased serum uric acid out of these HELLP had maximum cases 6 cases (37.5%) followed by eclampsia 5 cases (31.25%). The increase in serum uric acid values with the severity of PIH was found to be statistically significant. (p value of 0.0105*).

TABLE 18: SUMMARY OF HEMATOLOGICAL, COAGULATIVE AND BIOCHEMICAL PROFILES OF THE PATIENTS.

No	Summary	Min	Max	Range	Mean	SD	Median
1	Age (Years)	19.00	38.00	19.00	26.43	4.04	26.00
2	Hemoglobin(gm/dl)	7.30	15.90	8.60	11.44	1.67	11.50
3	RBC(million/cumm)	2.33	6.02	3.69	4.25	0.68	4.22
4	PCV(%)	18.90	46.50	27.60	34.91	4.95	35.40
5	TLC(cells/cumm)	2.90	30.00	27.10	12.93	4.47	12.00
6	Platelet (cells/cumm)	24.00	489.00	465.00	192.21	90.82	160.50
7	MPV(fL)	7.10	13.50	6.40	9.69	1.35	9.50
8	Total bilirubin(mg/dl)	0.04	6.30	6.26	0.46	0.52	0.34
9	SGOT(IU/L)	10.00	1645.00	1635.00	42.55	140.46	21.00
10	SGPT(IU/L)	5.00	644.00	639.00	27.97	59.09	14.00
11	ALP(IU/L)	68.00	463.00	395.00	182.86	70.67	174.50
12	Serum urea(mg/dL)	10.00	77.00	67.00	18.13	10.01	15.00
13	SR. creatinine(mg/dL)	0.30	2.60	2.30	0.70	0.37	0.60
14	SR. uric acid	1.90	10.40	8.50	5.67	1.73	5.60
15	PT	8.30	18.00	9.70	11.16	1.79	10.60
16	aPTT	18.70	48.30	29.60	29.32	3.76	29.40
17	TT	9.80	25.90	16.10	15.58	2.20	15.35
18	D-dimer	120.00	5000.00	4880.00	883.47	976.29	623.50
19	Fibrinogen	105.00	800.00	695.00	399.71	106.16	396.00
20	LDH	101.00	2612.00	2511.00	405.09	283.04	321.50
21	Urine protein	0.00	4.00	4.00	1.95	1.28	2.00

TABLE 19: CORRELATION BETWEEN HEMATOLOGICAL, BIOCHEMICAL AND COAGULATIVE PARAMETERS WITH PIH PATIENTS USING SPEARMAN'S CORRELATION COEFFICIENT.

Parameters	N	R	t-value	p-level
Age (Years)	160	0.2614	3.4048	0.0008*
Haemoglobin(gm/dl)	160	-0.2409	-3.0903	0.0024*
RBC(million/cumm)	160	0.0761	0.8932	0.3733
PCV(%)	160	0.0434	0.5413	0.5891
TLC(cells/cumm)	160	0.1574	1.8857	0.0614
Platelet count (cells/cumm)	160	-0.6109	-9.7003	0.0001*
MPV(fL)	160	0.1284	1.4245	0.1569
Total bilirubin(mg/dl)	160	-0.0112	-0.1369	0.8913
SGOT(IU/L)	160	0.2085	2.6111	0.0099*
SGPT(IU/L)	160	0.1296	1.6012	0.1114
ALP(IU/L)	160	0.0049	0.0605	0.9519
SR.urea(mg/dL)	160	0.0527	0.6490	0.5173
SR. creatinine(mg/dL)	160	0.1829	2.2860	0.0236*
SR. uric acid	160	0.2180	2.5956	0.0105*
PT	160	0.2741	3.2115	0.0017*
aPTT	160	0.3249	3.8714	0.0002*
TT	160	0.4395	5.4485	0.0001*
D-dimer	160	0.3357	4.0003	0.0001*
Fibrinogen	160	0.1359	1.5217	0.1306
LDH	160	0.2765	3.3309	0.0011*
Urine protein	160	0.8239	18.1609	0.0001*

R = Correlation Coefficient, **N** = number of study participants/patients, **p** = p value (probability value) based on independent sample t-test, *Statistically significant at *p value<0.05

Interpretation:

Hematological, Biochemical and Coagulative parameters were correlated with patients who were clinically diagnosed with pregnancy induced hypertension using "Spearman's correlation".

Correlation coefficient (R) was seen between -1 to +1. A negative 'R' value indicated negative correlation and positive 'R' value indicated positive correlation. Zero signifies no correlation. 'p' value of less than 0.05 was considered to be statistically significant.

Age of these patients clinically diagnosed with pregnancy induced hypertension was found to have a positive correlation and has a statistically significant p value. ($p<0.05$)

Hematological variables like RBC, PCV, TLC and MPV showed positive correlations with clinically diagnosed patients of pregnancy induced hypertension while hemoglobin and decreased platelet count with severity of disease was found to have statistically significant p value ($p<0.05$). Variables like hemoglobin and platelet count have a negative correlation and RBC, PCV, TLC, MPV have statistically insignificant p values.

Coagulative variables like PT, aPTT, TT, D-dimer and fibrinogen showed positive correlations with clinically diagnosed patients of pregnancy induced

hypertension while PT, aPTT, TT, D-dimer were found to have statistically significant p values fibrinogen was found to have a statistically insignificant p value.

Biochemical variables like SGOT, SGPT, ALP in liver function test, variables like serum urea, serum creatinine, serum uric acid in renal function test, LDH and Urine protein showed positive correlations with clinically diagnosed patients of pregnancy induced hypertension while SGOT, serum creatinine, serum uric acid, LDH and urine protein were found to have statistically significant p values. Total bilirubin is found to have a negative correlation and has a statistically insignificant p values while SGPT, ALP, serum urea also had statistically insignificant p values.

DISCUSSION

The present study is an observational study of one year, conducted in the Department of Pathology, J N. Medical College and KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi in the year 2020 from January - December. A total of 160 patients clinically diagnosed with pregnancy induced hypertension from January 2020 – December 2020 were studied. Pregnant women with pregnancy induced hypertension develop a variation in hematologic, biochemical and coagulative profile which have an impact on the outcome of these patients so that aggressive therapy can be initiated to prevent maternal and neonatal morbidity and mortality. Simple investigations such as complete hemogram, urine examination, and liver enzymes were done on all the cases that can detect platelet abnormalities, red cell abnormality, and detect patients likely to progress to HELLP syndrome. Coagulation profile was done only on patients with thrombocytopenia which is an important parameter for detecting DIC.

TABLE 20: COMPARISON OF AGE GROUP OF PIH PATIENTS WITH OTHER STUDIES

S.NO.	STUDY CONDUCTED	COMMON AGE GROUP AFFECTED IN MILD PIH	COMMON AGE GROUP AFFECTED IN SEVERE PIH
1.	Shetty et al[4]	25-30 years (39.77 %)	20-25 years (45.53%)
2.	Chaudhari, et al[17]	>34 years (50%)	>34 years (50 %)
3.	Kumar P. et al[41]	26-30 years (55.3%)	26-30 years (44%)
4.	Thalor et al[87]	26– 29 years (50%)	26-29 years (50%)
5.	Present study	26-30 years (36.98%)	26- 30 years (48.07%)

The common **age** group in the present study was 26-30 years for mild PIH and severe PIH 36.98% cases and 48.07% cases respectively. Study done by Shetty et al correlated well with our study with regards to mild PIH. The other three studies showed a higher age group involvement. In severe PIH commonest age group was 26-30 years (48.07%). All the other studies mentioned in the above table correlated well with regards to severe PIH. This indicates that the severity of complications increases with the age of patient which can be attributed to low nitric oxide levels and high oxidative stress which are signs of ageing and adversely affect the relaxation of the endothelium. This could cause the development of pregnancy-induced hypertension in older mothers, because pregnancy increases cardiac output.[34]

TABLE 21 : COMPARISON OF GRAVIDA STATUS IN PIH PATIENTS

S.NO.	STUDY CONDUCTED	PRIMIGRAVIDA	MULTIGRAVIDA
1.	Kumar P. et al[41]	61(61%)	39 (39%)
2.	Shetty et al[4]	109 (54.5%)	91(45.5%)
3.	Chaudhari et al[17]	25 (39.06%)	39 (60.94%)
4.	Present study	118 (73%)	42 (26%)

Pregnancy induced hypertension is mainly disease of **primigravida**.118(73%) were primigravida and 42 (26%) were multigravida in our study which correlated well with Kumar P et al[41] and Shetty et al[4] whereas maximum cases were seen in multigravida in the study done by Chaudhary et al [17]. Increased risk of PIH in young or elderly primigravida can be due to failure of trophoblast invasion as there is first time exposure to chorionic villi.[1]

The most common associated factor was **family history** seen in 40 cases (32%) of mild and severe PIH, similar observation was made by Rathi et al., who reported family history of PIH in 90% cases.

Epigastric pain was the predominant symptom seen in 20 cases (38.46%) of severe PIH patients in this study, similar observation was made by Weinstein et al. [18]

HEMATOLOGICAL PARAMETERS:

TABLE 22: COMPARISON OF HAEMATOLOGICAL PARAMETERS OF PIH PATIENTS WITH OTHER STUDIES (VALUES ARE MEAN)

S.NO.	STUDY CONDUCTED		HAEMOGLOBIN MEAN (g/dl)	RBC mean (10^6/mm^3)	PCV mean(%)	WBC count mean 10^3(cells/mm^3)
1.	AlSheeha et al[54]	Mild PE	--	--	35.7	8.2
		Severe PE	--	--	35.1	8.1
2.	Monteiro et al[40]	Mild PE	11.39	--	31.42	12.679
		Severe PE	8.54	--	28.01	13.248
3.	Al-Nimer et al[53]	PE	12.07	4.47	35.75	7.17
4.	Present study	Mild PE	11.89	4.95	34.66	12,000
		Severe PE	9.48	4.55	30.26	

In the present study the mean hemoglobin concentration of these 160 patients was 10.92g% which is slightly lower than mean hemoglobin concentration of normal pregnancy, similar results was seen in a study conducted by Shetty et al.[4]

In our study the mean **hemoglobin** concentration of mild PIH patients was 11.89 gm%, severe PIH was 9.48 gm%, eclampsia was 9.21% and HELLP was 9.13gm%,

thus it revealed a significant decrease in the hemoglobin level with the increase in severity of the disease which is consistent with a study done by Jhajharia N et al[19] ,Monterio et al[40] and Al-Nimer et al.[53] This can be attributed to microangiopathic intravascular haemolysis.

In our study the **RBC** mean was 4.95 x 10^6/mm^3 for mild PIH and 4.55x10^6/µL for severe PIH this correlated with Al-Nimer et al[53] and the mean **PCV** in present study was 34.66 % in mild preeclampsia and 30.26% in severe preeclampsia which correlated well with AlSheeha et al [54], Monteiro et al[40] and Al- Nimer et al.[53]

The mean **WBC count** was more than 12000 cell/mm^3 which correlated well with Monteiro et al. where as studies done by AlSheeha et al[54] and Al Nimer et al[53] showed normal range of WBC count. The increase in absolute neutrophil count in preeclampsia as compared with normal pregnancy is the cause of increase in mean WBC.[19]

TABLE 23: PLATELET COUNT MEAN IN RELATION TO SEVERITY OF PIH

S.NO	STUDY CONDUCTED	MILD PE (Lac/cmm)	SEVERE PE (Lac/cmm)	ECLAMPSIA (Lac/cmm)
1.	Gupta A et al[52]	2.3	1.6	0.99
2.	Prathap T et al[7]	2.31	1.68	1.27
3.	Chaudhary S et al[17]	2.35	1.24	0.86
4.	Mohapatra S et al[51]	2.23	1.82	1.21
5.	Present study	2.44	1.74	1.18

In the present study, it is seen that **platelet count** reduced as per the severity of PIH in mild preeclampsia it was 2.44 lakh/cumm it was 1.74 lakh/cumm and eclampsia showed 1.18 lakh/cumm. Our study correlated well with Gupta A et al[52], Prathap P et al[7], Mohapatra S et al [51]and Chaudhary S et al.[17] The low platelet count was attributed to immunologically mediated destruction, platelet aggregation and consumption, which appear to be due to endothelial damage. Platelet activation leads to increase in generation of thromboxane A2 and release of serotonin which in turn increase vasoconstriction and platelet aggregation. [20]

TABLE 24:-MPV IN RELATION TO SEVERITY OF PIH

S.NO	STUDY CONDUCTED	MILD PE (fL)	SEVERE PE(fL)	ECLAMPSIA (fL)
1.	Manchanda J et al [49]	10.46		11.52
2.	Nooh et al[22]	10.8	12.1	--
3.	Dadhich et al[50]	8.8	10.6	--
4.	Present study	9.51	9.73	10.1

The **mean platelet volume (MPV)** in PIH cases in our study were 9.51 ,9.73 and 10.1 in mild preeclampsia, severe preeclampsia and eclampsia there was a gradual rise in MPV as the severity of disease progressed. MPV values correlated well with Dadhich et al [50], Manchanda J et al[49] and Nooh et al.[22] The p value in our study was 0.1569 which was statistically not significant.

COAGULATIVE PARAMETERS:

TABLE 25- COMPARISON OF PROLONGED PT, APTT, TT (SEC) IN PIH CASES WITH OTHER STUDIES.

S.NO	STUDY CONDUCTED	SEVERITY OF PIH	PT(sec)	aPTT(sec)	TT(sec)
1.	Kumar P et al[41]	MILD PE	6 cases (20.7%)	10 cases (34.5%)	--
		SEVERE PE	4 cases(26%)	14 cases (56%)	--
		ECLAMPSIA	--	--	--
2.	Chaware S A et al[61]	MILD PE(n=50)	13.92	28.5	--
		SEVERE PE(n=40)	14.22	30.6	--
		ECLAMPSIA(n=30)	14.4	31.03	--
3.	Shetty et al[4]	MILD PE	2 cases (28.57%)	0 cases	1 case(14.29%)
		SEVERE PE	29 cases(82.86%)	8 cases (22.86%)	7 cases (20%)
		ECLAMPSIA	--	--	--
4.	PRESENT STUDY	MILD PE	1 case (1.36%)	8 cases (10.95%)	33 cases (45.20%)
		SEVERE PE	8 cases (15.38%)	9 cases (17.30%)	32 cases (56.14%)
		ECLAMPSIA	13 cases (48.14%)	10 cases (37.03%)	21 cases (77.78%)

In the current study, **PT** was prolonged with the severity of PIH with the maximum affected cases in HELLP 4 cases (50%) followed by eclampsia 13 cases (48.14%) and severe preeclampsia 8 cases (15.38%) this correlated with other Shetty et al[4] , Kumar P et al[41] Chaware S A et al [61] .The PT value of PIH was statistically significant with p value of 0.0017*. The prolongation of PT reflects picture of utilization of clotting factors due to mild intravascular coagulation.

aPTT was prolonged with the severity of PIH in the present study with the maximum affected cases in HELLP 3 cases (37.5%) followed by eclampsia 10cases (37.03%) and severe preeclampsia 9 cases (17.30%) this correlated with other Shetty et al[4] , Kumar P et al[41] and Chaware S A et al [61] The aPTT value of PIH was statistically significant with p value of 0.0002*. Significant prolongation of aPTT in severe PIH indicates consumption of coagulation factors, especially factor VIII.

In the current study, **TT** was prolonged with the severity of PIH with the maximum affected cases in HELLP 8 cases (100%) followed by eclampsia 21 cases (77.78%) and severe preeclampsia 32 cases (56.14%) this correlated with study by Shetty et al. [4] The TT value of PIH was statistically significant with p value of 0.0001*. Prolonged TT is due to low concentration of substrate for thrombin, i.e., hypofibrinogenemia

TABLE 26:- COMPARISON OF INCREASED D DIMER LEVELS WITH SEVERITY OF PIH IN VARIOUS STUDIES

S.NO	STUDY CONDUCTED	MILD PE	SEVERE PE	ECLAMPSIA
1.	**Shetty et al [4]**	>200ng/ml in 1 case(14.2%)	>200ng/ml in 10 cases (28.57%)	**--**
		Undetectable in 4 cases(57.14%)	Undetectable in 9 cases(25.71%)	
2.	**Kumar et al[41]**	**7 cases (24.1%)**	**10 cases(40%)**	**--**
3.	**PRESENT STUDY**	>457ng/dl in 18 cases (24.65%) of second trimester.	>457ng/dl in 15 cases (28.84%) of second trimester.	>457ng/dl in 8 cases (29.62%) of second trimester
		>644 ng/dl in 32 cases(43.83%) of third trimester.	>644 ng/dl in 30 cases(57.69%) of third trimester.	>644 ng/dl in 12 cases(44.44%) of third trimester

In our study cases of mild preeclampsia the **D-dimer levels** of >457ng/dl in 18 cases (24.65%) of second trimester and >644 ng/dl in 32 cases (43.83%) of third trimester.
Similarly in cases of severe preeclampsia, D-dimer >457ng/dl in 15 cases (28.84%) of second trimester and >644 ng/dl in 30 cases (57.69%) of third trimester.

In cases of eclampsia D-dimer levels >457ng/dl in 8 cases (29.62%) of second trimester and >644 ng/dl in 12 cases (44.44%) of third trimester. The above pattern of D-dimer value can be explained by activation of coagulation and fibrinolytic system. The increase in d-dimer values with the severity of PIH was found to be statistically significant. (p value for D-dimer=0.0001*) this correlated with the study by Shetty et al[4] and Kumar et al.[41]

Thus it was observed in the other studies that D-dimer positive women had greater risk of cesarean section, premature delivery, and low birth weight. [23] Hence our cases need to be followed up. Thus D-dimer was a better indicator of DIC compared to all other tests and correlated well with the outcome of pregnancy in other studies where cases were followed up.

In present study the **fibrinogen levels** in mild pre-eclampsia were >853mg/dl in 10 cases (13.69%) of second trimester and >914mg/dl in 28 cases (38.36%) of third trimester.
Similarly in cases of severe preeclampsia, fibrinogen levels >853mg/dl in 12 cases (23.07%) of second trimester and >914mg/dl in 25 cases (48.07 %) of third trimester.
In cases of eclampsia fibrinogen levels >853mg/dl in 6 cases (22.23%) of second trimester and >914mg/dl in 10 cases (37.03%) of third trimester.

The increase in fibrinogen values with the severity of PIH was found to be statistically insignificant. p value =0.1306.

Anuradha R[24] in her study found that there was a significant increase in fibrinogen levels in pregnant women when compared to non-pregnant women, and increase in fibrinogen levels has a lesser significance among preeclampsia when compared to normal pregnancy. Chatterjee T et al[26] has also reported that there is increase in plasma fibrinogen levels in preeclampsia and eclampsia about 70% and 143% respectively. Yusuf Ustun et al[25] found higher levels of Fibrinogen and CRP in Preeclampsia.

Hence in our study we emphasize that raised parameters of PT, aPTT, TT and D-Dimer are alarming signs for aggressive treatment which aid in prognostic outcome.

BIOCHEMICAL INVESTIGATIONS-

Proteinuria is an important sign of pre-eclampsia and diagnosis of pre-eclampsia is doubtful in its absence. In our study, Mild proteinuria more than or equal 0.3 g/24 hours (dipstick 1+) was seen all 73 cases (45.63%) of mild PIH while severe proteinuria more than or equal 2 g/24 hours (dipstick 2+) was seen in 75 cases (46.88%) of these 52 cases of severe PIH and 3 cases of eclampsia and HELLP had 3+ proteinuria, 24 cases of eclampsia and 5 cases of HELLP had 4+proteinuria.Thus proteinuria was present in all 160 patients (100%). This was found to be statistically significant with p value of 0.0001*. Shetty et al[4] and Jambhulkar et al[48] also had similar statistically significant findings which correlated well with our study.

Total bilirubin levels in our study showed mild elevation as per the severity of PIH and was not found to be statistically significant p value of 0.8913 Hassanpour et al[42] and Shetty et al[4]also found no significant correlation with regards to bilirubin levels.

In our study **Serum SGOT and SGPT** levels was found to have a positive correlation with clinically diagnosed patients of pregnancy induced hypertension. SGOT and SGPT values were more elevated with the severity of disease. SGOT was elevated in 2 cases (11.76%) of mild PE, 4 cases (23.52%) of severe PE ,5 cases (29.42%) of eclampsia and 6 cases (35.29%) of HELLP thus SGOT values are elevated in percentage of patients as the severity of disease increases this was found to be statistically significant (p value = 0.0099*).

SGPT was elevated in 1case (5.57%) of mild PE, 4 cases (22.23%) of severe PE ,6 cases (33.37%) of eclampsia and 7 cases (38.87%) of HELLP thus SGOT values is elevated in percentage of patients as the severity of disease increases but SGOT was found to be statistically insignificant (p value = 0.1114).

Menzies J et al[31] , Shetty et al[4] , Hazari NR et al[11] and Kozic R et[32] al in there study observed that liver enzymes were elevated as per the severity of PIH . In our study SGPT was raised but statistically nonsignificant as compared to SGOT which was statistically significant.

There are raised levels of liver enzymes mainly transaminases which are released from the damaged liver tissue. Liver changes are found in 60-70% of women. The enzymes level are not specific to preeclampsia or eclampsia but are manifestations of vasoconstriction and disseminated intravascular coagulation. Thus no specific therapy

is required for Liver involvement in preeclampsia, but it is an indicator to prevent more serious disorders such as eclampsia, hepatic rupture, or necrosis. [30]

ALP was elevated in 1case (6.67%) of mild PE, 2 cases (13.37%) of severe PE ,4 cases (26.67%) of eclampsia and 8 cases (53.33%) of HELLP thus ALP values are elevated in patients as the severity of PIH increases thus it is observed that values are significant in eclampsia and HELLP but taking ALP levels in PIH as a whole it was found to be statistically insignificant (p value = 0.9519). Makuyana D et al[43], Wong et al[44] and Prathap T et[7] al in their study found that ALP levels increased with the severity of PIH.

Serum LDH levels in our study was found to have a positive correlation. Serum LDH was elevated in 2 cases (10.52%) of mild PE, 3 cases (15.78%) of severe PE ,6 cases (31.57 %) of eclampsia and all the 8 patients of HELLP syndrome thus in our study Serum LDH values were more elevated with the severity of disease. Serum LDH is found to be statistically significant (p value = 0.0011*). The observations made in our study was similar to Prathap T et al[7] and Jaiswer SP et al.[47]

Renal function test-

Serum urea, serum creatinine, serum uric acid levels in our study was found to have a positive correlation.

Serum urea was elevated in 2 cases (18.18%) of mild PE, 2 cases (18.18%) of severe PE ,4 cases (36.36%) of eclampsia and 3 cases (27.27%) of HELLP thus it is seen that

serum urea values also mildly raised as per severity of PIH but is found to be statistically insignificant p value = 0.5173.

Serum creatinine was elevated in 1 case (6.25%) of mild PE, 3 cases (18.75%) of severe PE ,5 cases (31.25%) of eclampsia and 7 cases (43.75%) of HELLP serum creatinine was found to be statistically significant p value = 0.0236*.

Serum uric acid was elevated in 2 cases (12.5 %) of mild PE, 3 cases (18.76%) of severe PE ,5 cases (31.25%) of eclampsia and 6 cases (37.5 %) of HELLP serum uric acid was found to be statistically significant p value = 0.0105*.

In the study done by Ekun et al.[45] Makuyana D et al [46] and Pratap t et al[7] the uric acid levels, urea and creatinine were significantly raised and statistically significant. In our study serum creatinine and uric acid levels were raised and showed statistical significance as per severity of PIH but blood urea levels though raised did not show statistical significance this is probably because of non-sensitive and non-specific nature of urea levels as compared to creatinine.

CONCLUSION

- PIH was commonly seen in 26 to 30 years age group .
- PIH was commonly seen in primigravida
- Family history was more significant
- Epigastric pain was one of the common symptom.
- There was decrease in the hemoglobin level with the increase in severity of PIH.
- Platelet count decreased with the severity of the PIH.
- Coagulation parameters like PT, aPTT, TT and d-dimer were also raised with the as per the severity of PIH.
- Urine proteins are found and are elevated in various degrees of PIH.
- SGOT, SGPT, ALP and LDH levels increased with the severity of PIH .
- Serum creatinine, serum uric acid was raised with severity of PIH.

SUMMARY

Pregnancy-induced hypertension is common medical disorder of pregnancy leading to maternal/fetal morbidity and mortality. It is progressive disease with a variable mode of presentation and rate of progression. Thus there is a need to develop simple methods specifically designed for use in a hospital environment for simple, low cost and rapid routine screening,

Hence, this study was undertaken to know the hematological, biochemical and coagulative derangement in preeclampsia and further access the prognosis and severity of the complications and thus will help as a treatment guide for cases of preeclampsia and eclampsia.

This observational study was done for a period of one year in the Department of Pathology, J N Medical College and KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 160 patients clinically diagnosed with PIH from January 2020 – December 2020 were studied. The important observations and implications summarized as below.

- Out of 160 patients, 73 cases had mild PIH,52 cases were severe PIH,27 cases of eclampsia and 8 cases of HELLP syndrome.

- Most of the pregnant women with PIH were aged between 26-30 years (42.5%). This probably indicates that severity of complications increases with the age of patient.

- 118 cases (73%) were primigravida and 42 cases (26%) were multigravida.

- Of these 15% of the cases had a significant past history of PIH. Family

history was important in 27.5% cases. Epigastric pain was the predominant symptom seen in 20 cases (38.46%) of severe PIH patients

- Mean hemoglobin concentration of these 160 patients was 10.92g% while mild PIH, severe PIH, eclampsia, HELLP had a mean hemoglobin concentration of 11.89 gm%, 9.48 gm%, 9.21% and was 9.13gm% respectively thus it revealed a significant decrease in the hemoglobin level with the increase in severity of the disease.

- The mean RBC count of mild PIH, severe PIH, eclampsia, HELLP cases was 4.95 x 10^6/µL, 4.55x10^6/µL,4.25 10^6/µL and 3.82 10^6/µL respectively.

- The mean PCV value of mild PIH, severe PIH, eclampsia and HELLP was 34.66 %, 30.26 %, 29.95 % and 27.85 % respectively.
- Mean WBC count was more than 12000 cell/mm^3. The mean ESR value was 60 mm/h.
- The reticulocyte count varied between 0.5% and 8%. It was raised (>2.5%) in 10 cases (13.69%) of mild PIH, 18 cases (34.66%) of severe PIH ,15 cases (55.56%) of eclampsia and all cases (100%) of HELLP syndrome.
- Of the 160 PIH cases, 80 cases showed normal platelet count and 80 cases showed deranged platelet count.
- The mean platelet count among the mild pre-eclampsia and severe eclampsia, eclampsia and HELLP cases was 244.93 10^3/µL and 174.77 10^3/µL ,118.07 10^3/µL, 86.125 10^3/µL. respectively. The platelet count decreased significantly with the increase in severity of pregnancy induced hypertension.
- Mean platelet volume (MPV) in mild PIH, severe PIH, eclampsia, HELLP

was 9.51fl , 9.73 fl , 10.1fl and 9.51 fl respectively.

- Prolonged PT was seen in 1case (1.36%) of mild preeclampsia, 8 cases (15.38 %) of severe preeclampsia, 13 cases (48.14%) of eclampsia and 4 cases (50%) of HELLP syndrome. Prolonged aPTT was seen in 8 cases (10.95%) of mild preeclampsia, 9 cases (17.30 %) of severe preeclampsia, 10 cases (37.03%) of eclampsia and 3 cases (37.5%) of HELLP syndrome. Prolonged TT was seen in 33 cases (45.20%) of mild preeclampsia, 32 cases (56.14 %) of severe preeclampsia, 21 cases (77.78%) of eclampsia and 8 cases (100%) of HELLP syndrome. The increase in PT, aPTT, TT with the severity of PIH was found to be statistically significant.
- Total 122 cases (76.25%) showed raised D-dimer values while total 97 cases (60.62%) showed raised fibrinogen values.
- Mild proteinuria (dipstick 1+) was seen all 73 cases (45.63%) while severe proteinuria (dipstick 2+) was seen in 75 cases (46.88%) of PIH.
- In liver function tests, total bilirubin was raised in 7 cases, SGOT was raised in 17 cases, SGPT was raised in 18 cases and ALP levels were raised in 15 cases of PIH. LDH was raised among 19 cases.
- With regard to renal profile, serum creatinine levels was raised in 16 cases, serum urea was raised in 11 cases while serum uric acid was raised in 16 cases.

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ANNEXURE I

INFORMED CONSENT

"CLINICAL AND DIAGNOSTIC MARKERS IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION".

Purpose of the study: This study is undertaken to know the hematological, biochemical and coagulative derangement in preeclampsia and further access the prognosis and severity of the complications and thus will help as a treatment guide for cases of preeclampsia and eclampsia.

You are requested to participate in this study which will help to provide appropriate and effective treatment in clinically diagnosed PIH patients. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge.

Your participation in the study is voluntary, you are free to withdraw from the study at any time. We assure there won't be alteration in quality of treatment you receive, irrespective of your participation in study.

Procedure: Blood and urine sample will be collected from all the patients who are clinical diagnosed as PIH and referred to the Department of pathology and Department of biochemistry for hematological, biochemical and coagulative studies in PIH over a period of 1 years will be included in the study. Complete hemogram, LFT, RFT, Coagulation tests, routine urine examination will be done in all patients.

Procedure for collecting the sample: Total of 4 cc of blood will be collected for complete hemogram using venipuncture method. The collected blood will be placed in EDTA vials and send for hematological investigations. For urine sample 10-15 ml of urine is to be collected into a sterile wide mouthed container provided which will be send for routine urine examination.

Risks and benefits There are no risks/minimal risks involved and benefits are to be evaluated.

Alternatives: Taking part in this study is voluntary. You may choose not to take part in this study or if you decide to take part now, you can later change your mind and withdraw from the study. The study doctor or sponsor may terminate your participation in this study anytime.

Privacy and confidentiality: The only people to know that you are a research subject are members of the research team. No information about you or provided by you during research will be disclosed to others without your written permission, except in emergency to protect your rights and welfare.

Financial incentives for participation: You will not be paid / offered any gift /incentives for participating in this study. You will not be charged for the test to be carried out on your blood and urine sample.

Financial incentives for participation: You will not receive any remuneration for participating in this study.

Authorization to publish results: When the results of research are published or discussed, no information will be displaced that would disclose your identity. Any information that is obtained in connection with this study and that can be identified

with you will remain confidential. However the results of this study would be forwarded to the KLE University, Belagavi as a part of requirement towards the completion of MD degree, review and publishing.

CONSENT STATEMENT

I undersigned _____have been explained in my vernacular language about the study and my participation in the study is voluntary. If I want, I can withdraw at any time. Also I have been given enough time to clear my doubts and rights as study participant.

In case of the queries during the study or in future you may contact following person.

Principal Investigator:

Guide:

Signature or left thumb print of participant or legally authorized representative.

Participants

Name _____signature_____

Witness

Name

_____signature_____

Experimenters

Name _____signature_____

Date:_____

Address: _____

Phone number:_____

ANNEXURE-II- ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/257 **Date: 24/12/2019**

To,

BM0119002
PG student in Pathology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CLINICAL AND DIAGNOSTIC MARKERS IN PATIENTS WITH PREGNANCY INDUCED HYPERTENSION – HOSPITAL BASED OBSERVATIONAL STUDY "**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE-III

PROFORMA

PATIENT HISTORY

Name:

Age:

IP no:

Period of gestation:

Presenting complaints:

History of present illness:

History of present pregnancy:

Obstetric history:

Menstrual history:

Past medical/surgical history:

Family history:

Personal history:

General Physical Examination

Build:

Nutrition:

Height:

Weight:

Pallor:

Jaundice:

Oedema of legs:

Pulse:

Blood pressure:

Systemic Examination

CNS:

CVS:

RS:

PA:

Obstetrical Examination

Abdominal, vaginal.

Routine investigation

Blood, urine.

Hematological investigation

- HB concentration
- PCV
- RBC
- ESR
- reticulocyte count
- white blood cell count
- platelet count
- Mean platelet volume (MPV)

ROUTINE URINE EXAMINATION

- Urine proteins

LIVER FUNCTION TEST

- SGOT
- SGPT
- ALP
- total bilirubin

— LDH

RENAL FUNCTION TEST

— Serum creatinine

— Serum urea

— Serum uric acid

COAGULATION TESTS

— Prothrombin time

— Activated partial thromboplastin time

— Thrombin time

— D-dimer

— Fibrinogen

ANNEXURE IV

WRIGHT'S STAINING PROTOCOL

Procedure:

1. Allow the slides to dry and place on the staining rack.
2. Put sufficient Wrights stain on the smear and wait for 3 minutes.
3. Add equal quantity of buffer (distilled water or phosphate buffer pH-6.5)
4. Blow gently
5. Wait for 5 minutes and then discard the stain and the buffer.
6. The back of the slide is cleaned with gauze
7. Excess of water is drained off
8. The stained smear is dried.

Result :

RBC -dark pink

Neutrophils -dark purple nucleus, pale pink cytoplasm with purple granules Eosinophils - blue nucleus, pale pink cytoplasm with reddish orange granules Basophils - dark blue nucleus and cytoplasm, dark purple black granules Lymphocyts -dark purple to deep bluish nucleus, sky blue cytoplasm Platelets - Violet to purple granules

ANNEXURES V - MASTER CHART

MASTER CHART

S.NO	Patient # no.	Diagnosis	trimester	Age	Gravida	Family H/O	Previous H/O	Hb	PCV	RBC	TC	PLT	MPV	ESR	RETIC	PT			APTT			TT			D-dimer	Fibrinogen	LFT				LDH	RFT				URINE
																Cont	Test	INR	Cont	Test	Ratio	Cont	Test	Ratio			SGOT	SGPT	T.Bilirubin	ALP		S.Creat	Uric acid	Urea	Protein
1	976809	MILD PE	3	22	p	-	-	13.8	41.4	5.39	13.1	208		67	0.8	11.3			30			13.9					32	31	0.11	159	292	0.2	6.8	20	1+
2	976067	MILD PE	2	20	p	+	-	10.4	31.2	4.15	12.9	344	9		0.6	11.3	10.6	0.94	30	28.7	0.96	13.9	13.2	0.95	495	484	11	10	0.8	159		0.3	7.2	20	1+
3	1048821	MILD PE	3	25	p	-	-	11.1	34.7	4.74	11.1	233	9		0.5	11.2	10.3	0.92	32.5	26.4	0.81	14.4	15.5	1.08	663	404	12	20	0.36	102	339	0.2	5.9	16	1+
4	1049040	MILD PE	2	22	p	-	-	12.2	33.7	3.28	10.97	384			0.6	11.2	10.8	0.96	32.5	25.7	0.79	14.4	14.4	1	332	431					329				1+
5	1048657	MILD PE	3	21	p	-	-	11.5	35.7	4.78	10.36	289	9.1	30	0.5	11.2	11.1	0.99	32.5	29.4	0.9	14.4	13	0.9	1116	574	27	17	1	225	286	0.3	4.7	16	1+
6	1051364	MILD PE	3	25	p	-	-	12.4	36.2	3.93	11.19	157	7.4		0.5	11.2	11	0.98	32.5	30	0.92	14.4	16.9	1.17	791	385	25	14	0.4	220	636	0.2	1.9	16	1+
7	1051147	MILD PE	3	22	p	-	-	12.3	37.7	5.03	15.36	374	8.3		0.7	11.2	11.1	0.99	32.5	26.8	0.82	14.4	12.5	0.87	345	409	32	32	0.9	84	306	1.3	7.2	14	1+
8	1050738	MILD PE	3	23	p	-	-	12	36.7	4.17	10.29	293			0.8	11.2			32.5			14.4					19	14	0.3	160	195	0.4	6.1	17	1+
9	1046873	MILD PE	3	26	p	-	-	12.3	37.7	5.03	15.36	374	8.3	65	0.6		11.1	0.99	32.5	26.8	0.82	14.4	12.5	0.87	345	409	32	32	0.9	84	306	0.3	7.9	14	1+
10	1047604	MILD PE	2	26	p	-	-	12.4	36.2	3.93	11.19	157	7.4		0.5		11	0.98	32.5	30	0.92	14.4	16.9	1.17	791	385	25	14	0.4	200	326	0.4	1.9	16	1+
11	1049593	MILD PE	2	22	p	-	-	12.6	36.9	4.37	15.1	247			0.8	11.2	11.6	1.03	32.5	26.8	0.82	14.4			4421		32	31	0.3	92	389	0.4	7.8	15	1+
12	1046386	MILD PE	3	25	p	+	-	12	36.7	4.17	10.29	293			0.8												19	14	0.3	160	195	0.4	6.1	17	1+
13	1046372	MILD PE	3	21	p	-	+	12.6	36.9	4.37	15.1	247			0.8	11.2	11.6	1.03	32.5	26.8	0.82				4421		32	31	0.3	92	230	0.3	7.8	15	1+
14	989324	MILD PE	3	26	m	-	+	12.4	36.8	3.9	11.8	246	7.1		0.8	11.3	8.8	0.78	30	30.4	1.01	13.9	15.4	1.11	510	297	18	12	0.24	163	207	0.3	5.3	21	1+
15	990514	MILD PE	3	23	p	+	+	11.6	36.2	4.38	15.3	253	10		0.9	11.3	10.5	0.93	30	29	0.97	13.9	14.7	1.06	1073	367	30	17	0.37	262	359	0.4	4.1	12	1+
16	990755	MILD PE	3	24	p	-	-	11.8	35.4	4.65	10.1	186		80	0.5	11.3			30			13.9					16	14	0.27	221	281	0.3	3.5	10	1+
17	990934	MILD PE	2	25	p	-	-	9.3	29.1	3.81	16.9	421	9		2.8	11.3	11.2	0.99	30	29.5	0.98	13.9	12.7	0.91	494	516	12	15	0.21	202	258	0.4	6.2	12	1+
18	991079	MILD PE	3	31	m	+	-	8				218	9		2.6	11.3	10.7	0.95	30	31.2	1.04	13.9	13.1	0.94	550	105	21	12	0.7	169	396	0.4	4.9	24	1+
19	998708	MILD PE	3	23	p	-	-	12.4	35.3	4.08	14.7	351	8.5		0.4	11.6	11.1	0.96	31.6	20	0.65	13.9	13.7	1.01	433	192	32	32	0.29	105	301	0.3	3.7	19	1+
20	999616	MILD PE	3	25	p	+	+	9.5				390	8.2		2.8	11.3	10.5	0.91	31.6	29.4	0.93	13.9	12.7	0.93	396	471	17	15	0.42	211		0.4		13	1+
21	1000023	MILD PE	3	23	p	+	+	8.5	27.4			275	10.9		2.7	11.3	9.9	0.85	31.6	29.4	0.93	13.9	13.4	0.99	1278	449								14	1+
22	999557	MILD PE	3	21	p	-	-	10.6				184	9.8		2.6	11.3	10.3	0.89	31.6	29.3	0.93	13.9	14.4	1.06	612	388	14	10	0.61	107	238	0.3	6.3	13	1+
23	1000353	MILD PE	2	23	p	-	-	10.5	28.7	5.1	12.2	364			0.7	11.3			31.6			13.9					19	10	0.51	230	306	0.4	6.5	16	1+
24	1000908	MILD PE	3	23	p	-	+	12.2	37.2	4.59	11.3	95			0.6	11.3			31.6			13.9					11	10	0.44	184	186	0.34	4.1	11	1+
25	1000939	MILD PE	3	19	p	+	-	12.4	37.5			128	13	50	0.8	11.3	10.1	0.87	31.6	26.9	0.85	13.9	13	0.96	463	315	25	12	0.3	220	291	0.45	6.9	16	1+
26	1001135	MILD PE	3	24	m	-	-	14.3	43.1	4.67	17.5	291	8.5		0.9	11.3	10.2	0.88	31.6	31.1	0.98	13.9	12.4	0.91	582	311	38	13	0.56	211	338	0.34	4.4	10	1+
27	1001050	MILD PE	3	22	p	-	-	9.3	28.9	3.11	9.6	90		65	2.9	11.3			31.6			13.9					14	8	0.62	196	389	0.45	5.7	21	1+
28	1001245	MILD PE	3	26	p	-	-	12.6	37.8	4.26	13.4	261			0.7	11.3			31.6			13.9					18	18	0.33	112	164	0.47	5.7	11	1+
29	994776	MILD PE	3	32	p	-	-	12.2	35.3		12.1	293	9.1		1	11.3	9.6	0.85	30	31.3	1.04	13.9	12.8	0.92	633	449	31	12	0.5	211	350	0.3	5.2	15	1+
30	995110	MILD PE	3	31	m	-	-	13.9	41.7	4.57	11.7	263	8.5		0.8	11.3	10.7	0.95	30	24	0.8	13.9	14.7	1.06	465	449	25	14	0.4	200	378	0.5	1.9	16	1+
31	995411	MILD PE	3	30	m	+	-	12.1	36.3		12.7	287	10.9		0.6	11.3	10.6	0.94	30	29.4	0.98	13.9	14.3	1.03	217	401	14	31	0.9	105		0.3		45	1+
32	995639	MILD PE	3	22	p	-	-	9	27	4.45	13.5	489	8.9	55	2.6	11.3	10.5	0.93	30	26.3	0.88	13.9	16.9	1.22	281	263	27	17	1	217	286	0.3	4.7	16	1+
33	995816	MILD PE	3	23	p	-	-	12.2	36.6	4.06	11.8	142			0.7	11.3			30			13.9					28	11	0.5	192	389	0.5	7.3	19	1+
34	996264	MILD PE	3	24	p	+	-	13.1	36.7	4.36	8.6	329	9.2		0.8	11.3	10.1	0.89	30	36.7	1.22	13.9	15.7	1.13	481	371	12	20	0.36	102	339	0.3	5.9	16	1+
35	996261	MILD PE	2	25	p	-	-	13.2	39.6	3.97	9.6	183	10		0.9	11.3	10.8	0.96	30	27.1	0.9	13.9	16.1	1.16	1053	390	18	10	0.2	212	188	0.3	4.3	12	1+
36	996902	MILD PE	3	27	p	+	-	9.2	28.2	3.92	7.9	367	10		2.7	11.3	9.5	0.84	30	26.9	0.9	13.9	14.6	1.05	167	377	22	11	0.3	172	273	0.46		11	1+
37	997852	MILD PE	3	23	p	-	-	11.4	34.2	3.6	12.6	290	7.9		0.6	11.3	10.2	0.88	30	28.7	0.91	13.9	13.9	1.02	745	372	14	10	0.29	133	242	0.4	6	10	1+
38	998936	MILD PE	3	26	p	-	-	9.4	28.2	3.31	11.6	133			2.6	11.3	10.3	0.89	30	23.1	0.75	13.9	12.3	0.9	280	246	12	10	0.26	127	349	0.52	5.1	19	1+
39	991405	MILD PE	2	30	p	+	-	13.6	40.8	4.37	12.3	178	12.8	45	0.7	11.3	10.1	0.89	30	28.4	0.95	13.9	16.1	1.16	1176	260	30	32	0.31	90	337	0.3	7.3	45	1+
40	991781	MILD PE	3	31	m	-	+	15.5	46.5	6.02	25.2	212	9.1		0.8	11.3	9	0.8	30	30.5	1.02	13.9	14.7	1.06	128	323	12	10	0.19	149	101	0.3	5.4	14	1+
41	992110	MILD PE	3	33	m	-	+	11.7	35.1	3.77	18.1	163	10.7		0.6	11.3	9.3	0.82	30	25.6	0.85	13.9	15.2	1.09	209	315	31	12	0.5	225	360	0.4	5.2	15	1+
42	992113	MILD PE	2	23	m	+	+	8.5	25.5	4.16	18.6	341	8.9		2.7	11.3	10.5	0.93	30	33.3	1.11	13.9	14.4	1.04	527	454	25	14	0.4	200	395	0.5	1.9	16	1+
43	992394	MILD PE	2	24	m	-	+	11.7	33.8	4.22	10.5	184	13		0.8	11.3	10.2	0.9	30	26	0.87	13.9	14.3	1.03	498	421	14	32	0.9	105	267	0.3	4.7	42	1+
44	992828	MILD PE	3	28	p	-	-	7.3	21.9	3.67	9.3	217	9		0.6	11.3	11.1	0.98	30	26.1	0.87	13.9	16.9	1.22	336	315	28	14	0.7	183	365	0.6	5.5	10	1+
45	993335	MILD PE	3	24	p	-	-	11.2	24.6	3.25	9.4	131	9.9		0.7	11.3	11.6	1.03	30	30.7	1.02	13.9	14.6	1.05	964	290	19	14	0.3	160	195	0.4	6.1	17	1+
46	993333	MILD PE	3	26	p	-	-	10.7	32.1	3.36	10.7	80		65	0.5	11.3			30			13.9					14	20	0.34	95	292	0.53	4.3	14	1+
47	993682	MILD PE	3	27	p	-	-	11.5	34.5	4.35	9.7	305	9.9		0.8	11.3	9.3	0.82	30	30.9	1.03	13.9	15.8	1.14	615	454	32	32	0.45	225	389	0.3	7.3	15	1+
48	993782	MILD PE	3	30	m	-	-	13.5	41.8			217	11.3		0.9	11.3	10.4	0.92	30	31.7	1.06	13.9	16.3	1.17	120	290	16	22	0.3	152	275	0.3		11	1+
49	988270	MILD PE	2	31	m	-	-	10.9	32.7	4.51	12.3	251			0.7	11.3			30			13.9					14	10	0.3	142	300	0.4	5.7	19	1+
50	987660	MILD PE	3	32	m	+	-	10.9	32.7	4.24	10.3	232	8.5		0.8	11.3	9	0.8	30	18.7	0.62	13.9	14	1.01	210	542	16	8	0.3	188	236	0.7	7.1	19	1+
51	1006285	MILD PE	3	23	p	-	-	11.7	34.8	3.94	9.9	204			0.6	11.6	10.9	0.94	31.6	23.4	0.74	13.6	15	1.1	1120	511	18	8	0.7	179	266	0.54	6.5	12	1+
52	1002451	MILD PE	2	22	p	-	-	13.9	41.1	4.99	8.2	168			0.7												102	55	1.2	312	866	0.4	4.8	21	1+
53	1001522	MILD PE	3	26	p	-	-	14.9	44	4.9	12	239			0.8												30	17	0.37	220	359	0.4	4.1	12	1+
54	1003575	MILD PE	3	27	p	+	-	11.5	34.5	3.93	8.2	221	10.6		0.7	11.6	10.5	0.91	31.6	26.4	0.84	13.6	14.2	1.04	280	456	12	15	0.21	202	258	0.62	6.2	12	1+
55	983509	MILD PE	3	29	p	-	+	13.3	39.9	5.05	10.3	266	7.6		0.5	11.3	9.3	0.82	30	32	1.07	13.9	14.2	1.02	1161	532	31	30	0.47	358	337	0.4	7.1	31	1+
56	983577	MILD PE	3	23	p	-	+	11.4	34.2			238	9.9	45	0.4	11.3			30			13.9					23	25	0.44	165		0.62	6	10	1+
57	983562	MILD PE	3	24	p	+	+	12.5	37.5	4.53	11.6	307			0.6	11.3	9.5	0.84	30	33.6	1.12	13.9	15.1	1.09	539	430	16	13	0.48	220	306	0.43	5	10	1+
58	983956	MILD PE	2	32	p	-	-	10.8	32.4	4.39	13.2	232	10.2		0.7	11.3	10.9	0.96	30	29.3	0.98	13.9	15.3	1.1	895	406	17	12	0.51	219	317	0.56	4.5	13	1+
59	983980	MILD PE	2	33	m	-	-	11.6	34.8			177	10.4		0.6	11.3	9.7	0.86	30	30.8	1.03	13.9	14.5	1.04	400	375	18	21	0.2	214		0.54	4.7	20	1+
60	986004	MILD PE	2	27	m	-	-	10.3	27.9	2.79	5.8	34		70	0.5	11.3	11.3	1	30	27	0.9	13.9	16.7	1.2	380	320	19	22	0.82	150	1282	0.44	4.9	15	1+
61	986244	MILD PE	2	22	p	+	-	12.5	38.6			146	10.1		0.7	11.3	9.8	0.87	30	28.4	0.95	13.9	15.7	1.13	562	455	14	10	0.3	142	300	0.75	5.7	19	1+

62	986419	MILD PE	2	21	p	-	-	13.7	32.1	4.17	12.6	324			0.8	11.3			30			13.9									7.2		1+
63	986693	MILD PE	2	20	p	-	-	12.8	22.5	2.8	12.7	387	7.8	65	0.8	11.3	10.4	0.92	30	20	0.67	13.9	13.6	0.98	500	324	14	10	0.38	177	199	0.52	7.3	11	1+
64	984556	MILD PE	3	22	p	-	-	11.5	34.5	4.25	7.3	237			0.8	11.3			30			13.9					28	10	0.45	198	300	0.41	2.7	19	1+
65	985221	MILD PE	2	23	p	+	-	13.1	39.3	4.15	10.4	304	10.9		0.9	11.3	10.7	0.95	30	26.3	0.88	13.9	15.5	1.12	200	396	20	9	0.24	171	265	0.52	4.5	12	1+
66	985250	MILD PE	2	24	p	-	-	10.9	32.7	3.41	14.9	149	11.7		0.7	11.3	10.4	0.92	30	26.4	0.88	13.9	14.8	1.06	565	430	28	24	0.2	116	185	0.56	5.7	34	1+
67	988988	MILD PE	2	27	p	+	-	13.3	38.7			230	10.4		0.8	11.3	10.9	0.97	30	24.9	0.83	13.9	14.3	1.03	200	398	12	10	0.26	127	449	0.52	5.1	19	1+
68	1002103	MILD PE	2	22	p	-	-	11.1	23.5	2.33	7.2	33	10.2	85	0.5	11.6	9.2	0.79	31.6	28.7	0.91		16.6	1.22	924	331	32	13	0.92	229	389	0.6	7.3	11	1+
69	976088	MILD PE	3	30	p	-	+	15.9	35.7	4.39	14.8	347	8.3	45	0.3	11.3	11.3	1	30	29.1	0.97	13.9	13.1	0.94	547	507	16	8	0.3	227	240	0.4	4.7	13	1+
70	978073	MILD PE	2	36	m	+	+	11	33	4.13	9.9	267	9.8	67	0.5	11.3	15.8	1.4	30	36.8	1.23	13.9	19.1	1.37	343	424	11	14	0.2	124	153	0.47	3.7	18	1+
71	983549	MILD PE	2	27	p	+	+	11.7	35.1			282	8	70	0.6	11.3	10.4981		30	28.187		13.9	14.6481		700.593	387.865	17	7	0.58	192	326	0.65	4.6	21	1+
72	987999	MILD PE	2	24	p	-	+	11	34.4	4.57	10.5	230	9.5	65	0.5	11.3			30			13.9					22	12	0.32	220		0.4	3.5	16	1+
73	982802	MILD PE	3	34	m	+	+	12	34.5	4.81	8.8	150	9.51	45	0.5	11.3			30			13.9					22	12	0.24	165	309	0.52	3.3	14	1+
S.NO	Patient IP no.	Diagnosis	rimeste	Age	Gravida	amily H	/ious	Hb	PCV	RBC	TC	PLT	MPV	ESR	RETIC	PT			APTT			TT			D-dimer	Fibrinoge	LFT				LDH	RFT			URINE
																Cont	Test	INR	Cont	Test	Ratio	Cont	Test	Ratio			SGOT	SGPT	T.Bilirubi	ALP		S.Creat	Uric acid	Urea	Protein
1	975007	Severe PE	2	35	m	+	+	10.8	37.2	4.1	11.1	138	9.2	80	0.8	11.3	10.6	0.94	30	30.4	1.01	13.9	15.7	1.13	721	348	43	25	0.38	279	477	0.68	5.8	15	3+
2	975283	SEVERE PE	2	25	p	-	-	10.5	31.5	5.01	16.7	336	8.3		0.9	11.3	9.9	0.88	30	24.9	0.83	13.9	13.5	0.97	689	647	23	24	0.21	118		0.95	4.5	24	3+
3	1049595	SEVERE PE	3	28	m	-	-	9.1	38		16.8	150	8	56	0.6	11.2	9.9	0.88	32.5	24.8	0.76	14.4	14.8	1.03	941	354	11	21	0.3	177	351	0.48	2.4	10	3+
4	976590	SEVERE PE	3	20	p	-	-	10.1	34.8	4.17	9.8	150	7.5		0.5	11.3	11	0.97	30	30.5	0.84	13.9	11.2	0.81	509	442	16	12	0.2	192	235	0.73	5.2	17	3+
5	976489	SEVERE PE	3	25	p	-	-	11	33	4.22	9.3	132	12.5	65	0.8	11.3	10.1	0.89	30	30.7	0.64	13.9	12.3	0.88	762	375	11	10	0.3	146	227	0.63	6	12	3+
6	976774	SEVERE PE	3	23	p	-	-	9.6	37.8	5	10.9	215	10.4		2.6	11.3	9.9	0.88	30	29.3	0.98	13.9	9.8	0.71	938	484	18	7	0.28	224	326	0.79		10	3+
7	976755	SEVERE PE	3	24	p	-	-	10.8	38.4			136	9.1	50	0.7	11.3	9.2	0.81	30	28.9	0.96	13.9	15.4	1.11	764	406									3+
8	976105	SEVERE PE	3	27	p	+	-	11.5	38.7	4.95	10	150	9.9	67	0.8	11.3	8.3	0.73	30	28.5	0.95	13.9	13.9	1	230	366	14	17	0.04	72		0.6		23	3+
9	980678	SEVERE PE	3	29	p	-	-	10.8	38.4	4.79	12	150	10.2		0.9	11.3	12.5	1.11	30	30	1	13.9	15.9	0.9	443	773	27	18	0.25	155	311	0.52		10	3+
10	980781	SEVERE PE	2	32	p	-	-	9.8	35.4	3.88	8.2	97			2.6	11.3			30			13.9					20	11	0.47	110	321	0.74	6.2	23	3+
11	980089	SEVERE PE	3	30	p	-	-	9.6	28.8	4.01	7.5	391	7.7	58	1.2	11.3	11.5	1.02	30	29.7	0.78	13.9	13.6	0.98	782	412	15	9	0.22	150	203	0.51	5.9	10	3+
12	983323	SEVERE PE	3	26	m	+	-	8.2	39.3	5	11.3	185	11.9		0.9	11.3	10.3	0.91	30	24.8	0.83	13.9	13.4	0.96	555	424	13	7	0.52	215	203	0.55	5.1	12	3+
13	984328	SEVERE PE	3	32	m	-	-	10.7	44.4	5.27	20.9	150	10.3	75	0.7	11.3	11	0.97	30	29	0.97	13.9	18.8	0.9	354		33	19	0.44	152	492	0.64	6.6	15	3+
14	978117	SEVERE PE	3	23	p	-	+	7.9	41.7	4.79	12.2	150	11.7		2.7	11.3	10.7	0.95	30	28.7	0.96	13.9	14.6	1.05	519	484	22	18	0.2	191	285	0.61	3.9	12	3+
15	978739	SEVERE PE	2	25	p	-	+	8.9	32.7			150	8.4		2.5	11.3	11.8	1.04	30	33.4	1.11	13.9													3+
16	978710	SEVERE PE	3	29	p	+	+	10.8	32.4	3.4	14.9	150	12.1	57	0.8	11.3	9.5	0.84	30	24.3	0.81	13.9	15.9	1.14	886	424	22	20	0.3	113	287	0.55	5.6	26	3+
17	979637	SEVERE PE	2	28	p	-	+	9.6	31.5	4.38	12.7	112	11.3		2.6	11.3	10.9	0.96	30	30.9	1.03	13.9	15.7	1.13	767	351	23	11	0.31	154	335	0.75	5.1	13	3+
18	987707	SEVERE PE	2	37	m	+	+	10.9	39.4			108	9.1	67	1.8	11.3	9.5	0.84	30	29	0.9	13.9	15.4	1.11	316	168	19	22	0.82	150	1282	0.44	4.9	15	3+
19	987092	SEVERE PE	3	32	m	-	+	9.5	42.1	4.45	11.2	189	13.5		2.6	11.3	9.9	0.88	30	29.9	1	13.9	18	0.86	1106	476	33	19	0.44	152	492	0.64	6.6	15	3+
20	986367	SEVERE PE	3	22	p	+	-	10.8	33.8	3.75	7.7	150			0.3	11.3			30			13.9					28	10	0.45	262	515	0.41	2.7	19	3+
21	987675	SEVERE PE	3	26	m	-	-	11.9	35.1	4.22	12	150		56	0.4	11.3	10.6	0.94	30	30.7	0.92	13.9	19	1.08	503	385	20	9	0.24	171	265	0.52	4.5	12	3+
22	987623	SEVERE PE	3	27	m	+	-	8.5	40.9	4.8	15.8	179			0.5	11.3	10.5	0.84	30	30.3	1.01	13.9	16.8	1.21	232	491	28	24	0.2	116	185	0.56	5.7	34	3+
23	989320	SEVERE PE	3	25	p	-	-	10.8	32.8	3.75	18.2	152	9.3		0.6	11.3	9.5	0.84	30	28	0.93	13.9	17.1	1.23	1036	224	102	12	0.3	312		0.8	4.8	21	3+
24	988099	SEVERE PE	3	31	m	+	-	11.4	34.5		11.6	141	9.4		0.7	11.3	12.9	0.79	30	30	1	13.9	15.4	1.11	386	240	22	11	0.3	172	273	0.46		11	3+
25	988073	SEVERE PE	3	26	p	+	-	10.3	30.9	3.72	15.1	147	11.8		0.8	11.3	13.9	0.88	30	29.7	0.76	13.9	16.1	1.16	336	362	14	10	0.29	133	242	0.6	6	10	3+
26	989257	SEVERE PE	2	28	m	-	+	11.6	40.2	4.5	13	179		65	0.9	11.3			30			13.9					11	5	0.24	81		0.5	2.9	10	3+
27	988628	SEVERE PE	2	29	m	-	+	10.5	31.5	4.08	10.2	150			0.6	11.3			30			13.9					32	43	0.29	105	301	0.54	3.7	19	3+
28	1001955	SEVERE PE	3	24	p	+	+	12.9	38.7	4.18	9.6	150	9.3		0.8	11.6	13.4	0.9	31.6	30.5	0.97	13.6	15.7	1.06	301	348	25	12	0.91	188	297	0.73	6.2	19	3+
29	1002664	SEVERE PE	3	22	p	+	+	9.4	29.8	5.16	10.2	150	8.4	78	2.6	11.6	10.6	0.91	31.6	34.8	0.85		15.8	0.99	588	377	18	12	0.24	163	207	0.79	5.3	21	3+
30	1003194	SEVERE PE	2	24	p	+	-	11.7	36.6	4.57	30	150	10.7		0.7	11.6	11.6	1	31.6	28.1	0.92	13.6	14.1	1.04	698	436	16	14	0.27	221	281	0.57	3.5	10	3+
31	1006211	SEVERE PE	3	22	p	+	-	12.8	39.6	4.12	12.8	150	9		0.9	11.6	11.6	0.9	31.6	40.5	0.94	13.6	15.2	1.12	1190	511	16	15	0.15	235	452	1.12	6.6	31	3+
32	1008186	SEVERE PE	3	24	p	-	-	10.5	31.5	4.48	14.9	150	8.6		0.8	11.6	10.9	0.94	31.6	28.6	0.91	13.6	13.3	0.98	675	478	18	15	0.15	233	218	0.57	5.7	12	3+
33	1007803	SEVERE PE	2	22	p	-	-	9.5	34.5	4.11	9.9	150	10.6	56	2.5	11.6	13.7	0.84	31.6	40.8	1.29	13.6	18	0.96	650	471	16	11	0.3	154	283	0.57	6.5	20	3+
34	1009524	SEVERE PE	2	30	p	-	-	9.7	30.7	4.51	9.15	200	11.4		2.6	11.6	10.4	0.9	31.6	27.4	0.87	13.6	14	1.03	367	393	21	11	0.37	156	229	0.47	4.5	11	3+
35	1010033	SEVERE PE	3	33	p	-	-	10.3	39.2	4.87	11.6	167	9.5		0.9	11.6	13.8	0.93	31.6	28.5	0.9	13.6	17.5	1.29	1795	297	126	125	0.38	248	408	0.85	7.6	12	3+
36	1047657	SEVERE PE	3	23	p	+	-	8.1	27.6	3.87	22.28	220			1.9		18	0.98	32.5	27.1	0.83	14.4	15.9	1.1	1527	389	31	12	0.5	280	360	0.8	5.2	15	3+
37	1009530	SEVERE PE	3	31	p	+	+	9.6	39	4.73	8.7	150	8.1	65	3	11.6	10.3	0.89	31.6	29.4	0.93	13.6	13.6	1	357	357	19	14	0.23	134	321	0.7	6.1	12	3+
38	1048683	SEVERE PE	3	27	p	-	+	7.9	24.2	2.85	15.2	50	9.5		2.8		11.4	1.02	32.5	29.7	0.77	14.4	17.5	1.22	511	335	14	34	0.9	105	267	2.6	4.7	45	3+
39	1050915	SEVERE PE	2	27	m	-	+	10.6	36.4	3.59	12.9	234	8.5		0.8	11.2	10.3	0.92	32.5	27.8	0.8	14.4	18	0.97	1445	311	14	20	0.34	95	292	0.53	4.3	14	3+
40	1007246	SEVERE PE	3	27	m	-	+	9.4	34.8	4.18	10.24	165	8.9	79	2.7												20	11	0.3	208		0.8	4.9	15	3+
41	1051191	SEVERE PE	3	24	p	-	+	11.4	37.2	3.98	12.1	254	9.9		0.8	11.2	11.2	1	32.5	29.4	0.9	14.4	14.9	1.03	446	303	16	22	0.3	152	275	0.56		11	3+
42	1051410	SEVERE PE	3	24	p	-	+	8.1	27.6	3.87	22.28	194			0.5	11.2	13	0.98	32.5	27.1	0.83	14.4	15.9	1.1	1527	389	31	12	0.5	280	350	0.8	5.2	15	3+
43	1051390	SEVERE PE	3	29	m	-	-	7.9	24.2	2.85	15.2	42	9.5	65	2.9	11.2	11.4	1.02	32.5	25.1	0.77	14.4	17.5	1.22	511	335	14	34	0.9	105		2.6		45	3+
44	1048794	SEVERE PE	2	23	m	-	-	7.3	36.8	4.35	11.99	158	8.8		0.6	11.2			32.5			14.4					28	11	0.5	192	431	0.5	8.3	19	3+
45	1046841	SEVERE PE	3	30	p	-	-	8.4	36.4	3.59	12.9	271	8.5		2.7		10.3	0.92	32.5	25.9	0.8	14.4	19	0.97	1445	311	14	20	0.34	95	282	0.53	4.3	14	3+
46	1047077	SEVERE PE	3	24	p	-	-	11.4	37.2	3.98	12.1	254	9.9	56	0.8		11.2	1	32.5	29.4	0.9	14.4	14.9	1.03	446	303	16	22	0.3	152	275	0.56		11	3+
47	1004114	SEVERE PE	2	29	p	+	-	10.9	38.4	4.41	17.8	151	8.1		0.6	11.6	8.9	0.77	31.6	29.8	0.85	13.6	15	1.1	561	372	21	12	0.7	169	416	0.67	4.9	24	3+
48	1002547	SEVERE PE	3	26	p	+	-	8.5	39.6	4.92	17.2	335	9.2		2.8	11.6	8.6	0.74	31.6	35	0.85	13.6	14.2	1.04	>5000	417	30	24	0.19	126	377	0.97	7.9	30	3+
49	1005060	SEVERE PE	3	22	p	-	-	11.1	33.3	4	18	204	8.9	60	0.8	11.6	10.1	0.87	31.6	23.2	0.73	13.6	17.7	1.3	1130	199	30	39	0.31	90	437	0.79	8.3	45	3+
50	1005854	SEVERE PE	3	26	p	-	-	9.4	35.1	4.05	12.3	151	10.4		2.5	11.6	12.5	1.08	31.6	24.8	0.78	13.6	18.7	0.93	245	478	12	10	0.19	149	101	0.49	5.4	14	3+
51	1002187	SEVERE PE	3	24	p	-	-	8	33.5	4	9.2	240			3												23	9	0.49	233	246	0.82	6.3	15	3+
52	1002160	SEVERE PE	3	25	m	-	-	9.2	39.5	4.24913		234	11.3	70	2.6	11.6	11.6	0.83	31.6	34.2	0.89	13.6	14.2	1.04	640	379	10	9	0.4	138	235	0.65	6.4	31	3+
S.NO	Patient IP no.	Diagnosis	rimeste	Age	Gravida	amily H/O		Hb	PCV	RBC	TC	PLT	MPV	ESR	RETIC	PT			APTT			TT			D-dimer	Fibrinoge	LFT				LDH	RFT			URINE
																Cont	Test	INR	Cont	Test	Ratio	Cont	Test	Ratio			SGOT	SGPT	T.Bilirubi	ALP		S.Creat	Uric acid	Urea	Protein
1	1005707	ECLAMPSIA	3	24	p	-	+	9.1	39.3	4.48	11.7	120			1.8																	0.61		13	4+

2	1008861	ECLAMPSIA	3	25	p	-	-	9.7	30.5	4.43	21.4	150	10.6	86	3.1	11.6	10.9	0.94	31.6	34.4	0.77	13.6	19.5	1.04	1035	463	30	10	0.3	212	409	0.93	9.5	15	4+
3	1001733	ECLAMPSIA	3	26	p	-	-	8.4	40.1	4.24	13.5	148	9.1		2.9	11.6	13.5	0.83	31.6	29.8	0.88	13.6	18.4	1.01	722	344	16	11	0.51	122	260	0.43	4.9	10	4+
4	994787	ECLAMPSIA	3	34	m	-	-	9.8	40.8	4.17	14.7	41	10.9	75	3	11.3	15.4	0.81	30	31.1	1.04	13.9	16.9	1.22	841	201	48	40	0.9	84	306	1.3	7.9	14	3+
5	991023	ECLAMPSIA	3	29	p	+	+	10.6	37.2	4.21	12.3	73	11.3		0.6	11.3	14.7	0.82	30	29.9	1	13.9	15.1	1.09	526	179	19	11	0.28	236	655	0.69	6.1	23	4+
6	989552	ECLAMPSIA	3	24	p	+	-	9.8	39.8	5.08	15.3	100			2.6	11.3	13.4	0.94	30	30.6	1.02	13.9	14.5	0.87	632	483	17	15	0.42	211		0.45		13	3+
7	1008855	ECLAMPSIA	3	27	p	+	-	10.6	43.8	5.42	19.2	150	9.6	68	0.7	11.6	15.8	0.81	31.6	32.9	0.89	13.6	15.7	1.01	1177	485	40	19	0.24	463	462	0.82	7.6	18	3+
8	1046555	ECLAMPSIA	3	22	p	-	-	8.7	34.4	4.1	7.48	90	10.9		0.7	11.2	14.5	0.97	32.5	30.7	0.82	14.4	18.9	0.86	660	442	28	14	0.7	183	443	0.6	5.5	10	4+
9	1009030	ECLAMPSIA	3	28	p	-	-	9.1	28.3	2.73	9.1	93	11.6		0.6	11.6	15.6	1.19	31.6	32.8	0.91	13.6	14.9	1.1	180	377	40	28	0.73	118	465	0.7	10.3	17	2+
10	1006867	ECLAMPSIA	2	25	p	+	-	9.3	28.9	3.06	13.79	120			0.5												28	17	0.49	227	374	0.55	5.7	12	3+
11	1046857	ECLAMPSIA	3	38	m	-	+	10.1	41.9	5.95	24.8	45	9.9		0.6		15.6	0.83	32.5	29.6	0.86	14.4	18.5	1.07	>5000	311	42	87	0.45	236	834	0.77	9.1	15	3+
12	1048854	ECLAMPSIA	3	29	m	-	-	8.8	28			150	8.1	65	2.6	11.2	10.2	0.91	32.5	30.6	0.78	14.4	19.3	0.99	914	404	18	10	0.2	212	188	0.3	4.3	12	3+
13	1049600	ECLAMPSIA	2	25	p	+	-	10.7	33.4	4.1	7.48	150	10.9		0.7	11.2	14.6	0.97	32.5	29.7	0.82	14.4	18.4	0.86	660	442	28	14	0.7	183	443	0.6	5.5	10	4+
14	976132	ECLAMPSIA	3	29	m	-	-	7.25	36.4	4.13	11.6	120	8.3		2.6	11.3	10.6	0.94	30	32.8	0.92	13.9	18.5	0.92	830	449	12	13	0.4	214	221	0.53	4.2	12	3+
15	976532	ECLAMPSIA	2	37	m	-	-	9.6	40.8	4.96	13.8	150	8.5		2.6	11.3	13.6	0.81	30	32.1	1	13.9	14.7	1.06	680	335	28	19	0.2	195	413	0.79	7.7	47	4+
16	1050454	ECLAMPSIA	2	26	p	+	-	10.7	34.4	4.1	7.48	93	10.9		0.7	11.2	13.4	0.97	32.5	26.7	0.82	14.4	16.4	0.86	660	442	28	14	0.7	183	443	0.6	5.5	10	3+
17	977758	ECLAMPSIA	3	34	m	+	-	9.6	39.9	4	11.7	150		85	2.6	11.3			30			13.9												3+
18	980560	ECLAMPSIA	3	30	p	-	-	8.9	38.4	4.6	14.9	120	9.5		2.6	11.3	14.8	0.85	30	31.8	1.06	13.9	18.6	0.99	582	689	30	23	0.49	221	322	0.49		11	3+
19	986132	ECLAMPSIA	2	26	p	-	+	9.8	36.9	4.55	8.4	150			2.7	11.3			30			13.9					22	12	0.32	220		0.4	3.5	16	4+
20	983603	ECLAMPSIA	3	28	p	-	-	8	34.8	~~5.35~~	12	80	12.6	80	2.6	11.3	13.8	0.86	30	30.2	1.01	13.9	18.3	1.03	418	442	22	8	0.41	282	411	0.73	6.5	15	4+
21	1006146	ECLAMPSIA	3	23	p	-	-	10.8	35	4.69	12.3	150	10.7	50	0.6	11.6	14.6	0.72	31.6	30.7	0.88	13.6	19.3	1.06	1238	478	52	34	6.3	396	563	0.87	8.4	21	2+
22	1002041	ECLAMPSIA	3	21	p	-	+	8.5	36.2	5.59	17.5	150	9.4	66	2.6	11.6	13.6	0.77	31.6	32.8	0.72	13.6	14.6	1.07	635	486	29	36	0.32	149	494	0.64	5.8	16	3+
23	998829	ECLAMPSIA	3	28	p	-	-	10.7	32.1	4.03	7.6	111		76	0.6	11.3			30			13.9					11	5	0.24	81		0.5	2.9	10	4+
24	977567	ECLAMPSIA	3	25	p	-	-	8.5	37.5			100	11.2	65	2.6	11.3	10.6	0.85	30	30.7	0.92	13.9	19.5	1.05	737	455	15	9	0.25	255	233	0.6	4.6	18	1+
25	979751	ECLAMPSIA	3	32	m	-	-	10.5	36.5	5.36	21.7	92	8.9		0.5	11.3	14	0.8	30	31.3	0.84	13.9	19.3	1.17	1379	359	175	108	0.76	216	938	1.28	8.7	60	3+
26	979911	ECLAMPSIA	3	25	p	-	-	8.9	31.2			150	9.4		2.6	11.3			30			13.9												negative
27	1051071	ECLAMPSIA	3	28	p	-	+	7.3	41.9	5.95	24.8	150	9.9	70	2.6	11.2	10.2	0.83	32.5	32.6	0.86	14.4	19.6	1.07	>5000	311	42	87	0.45	236	834	0.77	9.1	15	4+
S.NO	Patient IP no.	Diagnosis	rimeste	Age	Gravid	amily H	vious I	Hb	PCV	RBC	TC	PLT	MPV	ESR	RETIC	PT			APTT			TT			D-dimer	Fibrinoge	LFT				LDH	RFT			URINE
																Cont	Test	INR	Cont	Test	Ratio	Cont	Test	Ratio			SGOT	SGPT	T.Bilirubi	ALP		S.Creat	Uric acid	Urea	Protein
1	1049636	HELLP	3	35	m	-	-	9.5	28.5	3.01	15.8	24	9.1	80	2.6	11.6	9.6	0.83	31.6	28.5	0.9	13.6	15.4	1.13	1162	480	38	36	1.25	361	883	0.65	7.8	28	3+
2	1009230	HELLP	3	28	p	+	-	9.5	28.5	3.01	15.8	24	9.1	60	2.6	11.6	9.6	0.83	31.6	28.5	0.9	13.6	15.4	1.13	1162	320	30	25	0.25	341	883	0.65	7.9	28	3+
3	1004845	HELLP	2	30	m	+	+	7.6	22.8	3.15	8.1	80	8.6	140	8	11.6	9.9	0.85	31.6	27.6	0.87	13.6	16.6	1.22	569	450	49	53	0.28	245	655	0.69	6.1	53	3+
4	993094	HELLP	3	21	p	-	-	10.3	36.9	4.34	25.4	200	11.9	75	2.6	11.3	10.2	1.43	30	48.3	1.61	13.9	25.9	1.86	>5000	580	96	135	0.3	432	517	0.64	7.8	15	3+
5	978197	HELLP	3	32	m	-	+	9.3	37.8	4.72	8.6	156	8.7	70	2.5	11.3	9.7	0.86	30	28.2	0.94	13.9	16.1	1.16	852	490	199	83	0.46	278	811	1.05	9.3	77	4+
6	1001343	HELLP	3	30	p	-	+	7.9	18.9	2.57	2.9	34	10.4	127	2.6	11.6	9	0.78	31.6	34.2	1.08	13.6	14.5	1.07	427	800	18	50	0.6	268	829	0.4	8.5	18	4+
7	984148	HELLP	3	25	p	+	-	9.8	37.2	4.5	16.8	92	8.7	50	2.6	11.3	12	1.06	30	29.3	0.98	13.9	14.4	1.04	1373	325	567	255	1.12	233	1049	0.72	9.6	29	4+
8	1009740	HELLP	3	30	m	-	+	9.2	29	5.25	28.5	79	9.6	55	2.8	11.6	10.6	0.91	31.6	30.5	0.97	13.6	16.4	1.21	3670	560	1645	644	1.58	361	2612	1.5	10.4	67	4+

ANNEXURE-VI

KEY TO MASTER CHART

ALP	-	Alkaline phosphatase
aPTT	-	Activated partial thromboplastin time
ESR	-	Erythrocyte sedimentation rate
HB	-	Hemoglobin
LFT	-	Liver function test
LDH	-	Lactate Dehydrogenase
MPV	-	Mean platelet volume
M	-	Multigravida
P	-	Primigravida
PCV	-	Packed cell volume
PLT	-	Platelet count
PT	-	Prothrombin time
RBC	-	Red cell count
RFT	-	Renal function test
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic pyruvic transaminase
Sr	-	Serum
TLC	-	Total leucocyte count
TT	-	Thrombin time
3	-	Third trimester
2	-	Second trimester
-	-	Negative
+	-	Positive