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*International Journal of Current Research*
*Vol. 6, Issue, 12, pp.11062-11075, December, 2014*

**INTERNATIONAL JOURNAL OF CURRENT RESEARCH**

**ISSN: 0975-833X**

## RESEARCH ARTICLE

# ASSESSMENT OF SEVERITY OF PREGNANCY INDUCED HYPERTENSION BY ESTIMATION OF PLATELETS, LIVER ENZYMES AND EARLY DETECTION OF HELLP SYNDROME

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**ARTICLE INFO**

***Article History:***
Received 25th September, 2014
Received in revised form
27th October, 2014
Accepted 08th November, 2014
Published online 30th December, 2014

***Key words:***

Pregnancy induced hypertension (PIH),
Preeclampsia,
HELLP syndrome,
Platelet count,
Liver enzymes,
Peripheral smear.

**ABSTRACT**

**Introduction:** In any community mother and children constitute a priority group. They comprise approximately 71.14 percent of the Population of the developing countries. Hypertensive disorders complicating pregnancy contribute greatly to maternal morbidity and Mortality. The present study was undertaken to assess the severity of pregnancy induced hypertension in the antenatal mothers at Government Raja Mirasudhar Hospital, Thanjavur.

**Aim and Objectives:** To study early assessment of severity of pregnancy induced hypertension by estimating platelets, liver enzymes, peripheral smear and haematocrit values and early detection of HELLP syndrome.

**Materials and Methods:** The study design was cross sectional study. This study was carried out in the Department of Physiology Thanjavur Medical College, Thanjavur. The study was carried out in 40 normotensive pregnant women, as control and 40 pregnancy induced hypertensive women, as study group. Both groups were correlated with age, parity and period of gestation. The subjects were collected from antenatal clinic, in patients ward and labour room from Department of obstetrics & Gynaecology, Raja mirasdhar Hospital, Thanjavur. The subjects were categorized as normotensive control, mild pregnancy induced hypertension and severe pregnancy induced hypertensive group by the presence of varying degrees of blood pressure and proteinuria. The presence of associated features of HELLP syndrome (Hemolysis, elevated liver enzymes – SGOT, SGPT > 70 U/L, LDH > 600 U/L) was assessed in patients in all the three classes of Thrombocytopenia.

**Results:** Statistical analysis was done by using the Statistical Package for Social Sciences (SPSS) X version. The results were analyzed by the Chi - Square test and ANOVA study. The mean age of control group ,study group was 24.95 ± 3.55 yrs,24.78 ± 3.59 yrs respectively. The mean gestational age in the control group, study group was 34.45 ± 3.37 wks. 35.45 ± 3.71 wks respectively. In normotensive control, the mean systolic & diastolic BP was 116.70 ± 6.19, 75.80 ± 5.04 mmHg respectively. In mild PIH, the mean systolic & diastolic BP was 144.21 ± 6.24, 94.41 ± 4.35 mmHg respectively. In severe PIH, the mean systolic & diastolic BP was 168.55 ± 12.62, 113.64 ± 6.38 mmHg respectively. In severe PIH the mean platelet count was 1.3900 ± 0.5909 lakhs / $mm^3$.The test results has shown significant reduction in platelet count in the mild & severe PIH ($P < 0.0005$). The mean difference in the SGOT, SGPT, LDH value in the normotensive control, mild & severe PIH were significant ($P < 0.0005$). The mean SGOT, SGPT, LDH in normotensive were 27.58 ± 9.28, 18.12 ± 7.03, 322.20 ± 84.46 U/L respectively. The mean SGOT, SGPT, LDH in mild PIH were 32.42 ± 17.64, 23.61 ± 14.61, 560.72 ± 224.50 U/L respectively. The mean SGOT, SGPT, LDH in severe PIH were, 90.60 ± 122.62, 76.85 ± 82.64, 801.73 ± 281.38 U/L respectively. These liver enzymes were markedly elevated in severe PIH. In the Haematocrit estimation the mean difference between these groups were significant ($P < 0.0005$). Hct % were raised in mild PIH, reduced in severe PIH, with normal results in control group. The mean Hct % in normotensive, 35.65, in mild PIH, 36.27 ± 4.5%. In severe PIH, 29.18 ± 4.75%. The peripheral smear study for hemolysis was significantly present in PIH when compared with normotensives ($P = 0.011$). The platelet estimation between control, mild PIH & severe PIH were statistically significnat (P value = 0.013). All degrees of thrombocytopenia were present in severe PIH & mild thrombocytopenia was present in mild PIH. Out of 10 cases of thrombocytopenia 6 cases were present with features of HELLP syndrome.

**Conclusion:** The progression of PIH from mild to life threatening diseases cannot be predicted. The aim of this study to draw attention to the life threatening complication such as hepatic dysfunction, haematological abnormalities and HELLP syndrome that may occur in cases of preeclampsia. The early diagnosis and early assessment of severity by platelet estimation, peripheral smear study, liver enzyme assays & urine analysis for proteinuria would be the most effective approach to enhance both maternal and fetal well being, as well as the successful outcome of pregnancy.



## INTRODUCTION

In any community mother and children constitute a priority group. They comprise approximately 71.14 percent of the Population of the developing countries. Mother and children not only constitute a large group but they are also a 'Vulnerable' or special risk group (Park *et al*., 2009). Hypertensive disorders complicating pregnancy are common and form one of the deadly Triad, along with haemorrhage and infection that contribute greatly to maternal morbidity and Mortality (Gary Cunningham *et al.,* 2005). Hypertensive disease of pregnancy account for 20% of all maternal deaths in India and 15% of maternal deaths globally (Vatsla Dadhwal,

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Deepika Deka). In India about two-thirds of perinatal deaths are due to low birth weight, among one of the cause is toxemia of pregnancy (Park *et al.,* 2009). It is associated with a 20 fold increase in perinatal mortality (Charles *et al.,* 1997). How pregnancy incites or aggravates hypertension remains unsolved despite decades of intensive research. In addition to essential hypertension, a unique form of hypertension that occurs only during pregnancy, called as pregnancy induced hypertension, also commonly known as preeclampsia. It is characterized by the onset of hypertension, proteinuria and edema, usually during III trimester. Management of this disorder differs significantly from that of chronic hypertension. Therefore, it is important to distinguish the two entities as clearly as possible. Preeclampsia complicates 5-7 percent of all pregnancies. The disorder occurs with increased frequency among young, nulliparous women. Normal pregnancy is associated with decreased maternal sensitivity to vasopressor, expansion of intravascular space, increased intravascular volume and decline in BP in the early gestation, then gradual rise in blood pressure to normal level near term. Women destined to develop preeclampsia do not exhibit normal refractoriness to vasopressors. As a result, normal expansion of the intravascular space and intravascular volume is reduced. In addition to the classic findings of hypertension, proteninuria, and edema, the laboratory abnormalities include elevation in the hematocrit, lactate dehydrogenase, serum transaminases (David *et al.,* 4th edition). The HELLP syndrome (Hemolysis, Elevated liver enzymes and low platelets) is a variant of severe preeclampsia. It was identified in almost 20 percent of women with severe preeclampsia (Gary Cunningham *et al.,* 2005). As understood from its definition, the syndrome is together with disorder of liver functions.

Increased maternal and fetal morbidity and mortality due to thrombocytopenia and hemolytic anemia. Maternal mortality is reported in between 0-24% in patients with HELLP syndrome. Perinatal mortality is reported in between 37% and 85% in babies of these pregnancies (Selahattin kumru *et al.,* 2005). The importance lies in early diagnosis, the early and timely diagnosis, especially that of severe preeclampsia, is quite essential in view of the significant complications that can Ensue (Chee Jing Jye *et al.,* 2009).

**Aim and Objectives**

To study early assessment of severity of pregnancy induced hypertension by estimating platelets and liver enzymes, peripheral smear and haematocrit values. To study the influence of varying degrees of Blood pressure and proteinuria on severity of pregnancy induced hypertension.
To study early detection of HELLP syndrome in pregnancy induced hypertension.

## MATERIALS AND METHODS

The study design was cross sectional study. This study was carried out in the Department of Physiology, Thanjavur Medical College, Thanjavur. The study was carried out in 40 normotensive pregnant women, as control and 40 pregnancy induced hypertensive women, as study group. Both groups were correlated with age, parity and period of gestation. The subjects were collected from antenatal clinic, in patients ward and labour room from Department of obstetrics and Gynaecology, The subjects were categorized as normotensive control, mild pregnancy induced hypertension and severe pregnancy induced hypertensive group by the presence of varying degrees of blood pressure and proteinuria.

The criteria for mild PIH,

- Blood pressure $\geq \frac{140}{90}\text{mmHg} < \frac{160}{110}\text{mmHg}$
- Proteinuria 1+ (Dispstick test).

The criteria for severe PIH,

- Blood pressure $\geq \frac{160}{110}\text{mmHg}$
- Proteinuria 3+, 4+
- Thrombocytopenia
- Elevated transaminases

To assess the severity of pregnancy induced hypertension, the following laboratory investigations were undergone.

- Urine analysis (Evidence of proteinuria)
- Platelet count
- Peripheral smear study (Evidence of hemolysis)
- Liver enzymes study (SGOT, SGPT, LDH)
- Haematocrit estimation.

The presence of hepatic abnormalities, altered haematological parameters & occurrence of HELLP syndrome were assessed. In platelet count, values > 1,50,000 cells /$mm^3$ was considered as normal.

Platelet count < 50,000 cells/$mm^3$ considered as class 1 (More severe Thrombocytopenia)

Platelet count between 50,000 to 1,00,000 cells/$mm^3$ as class 2 (Moderate severe Thrombocytopenia)

Platelet count between 1,00,000 to 1,50,000 cells/$mm^3$ as class 3 (Less severe Thrombocytopenia)

The presence of associated features of HELLP syndrome (Hemolysis, elevated liver enzymes – SGOT, SGPT > 70 U/L, LDH > 600 U/L) was assessed in patients in all the three classes of Thrombocytopenia.

**Inclusion Criteria**

Pregnant women of varying degrees of PIH in the age group of 19-35 years, in the II and III trimester were selected. Healthy normotensive women in the age group of 19-35 years, in the II and III trimester were selected as control.

**Exclusion Criteria**

The subjects were excluded from study with following histories and findings. History of Anaemia, cardiovascular

disease, diabetes, essential hypertension or secondary hypertension due to some other pathology, hepatic disorders, haemorrhagic disorders, asthma.

**Materials for the Study**

- Proforma to record the subject details and clinical examination findings.
- Semi auto analyser for recording the enzyme levels.
- Grease free glass slides for preparing peripheral smear study for platelet estimation and to rule out haemolysis.
- Glass tubes for collecting Blood samples for enzyme study.
- EDTA coated glass tubes for haematocrit estimation and wintrobe's haematocrit tube.
- Disposable syringes.
- 14% Magnesium sulphate
- Sphygmomanometer
- Stheoscope.
- Diagnostic reagent strips for urine analysis.

**Methodology**

The study was carried out after explaining the procedure in detail and getting informed consent from the subjects.
The Experimental protocol included,

- Recording of a detailed History from the study subjects.
- A thorough clinical examination of the study subjects.

A Detailed history regarding name, address, IP/OP Number, Age, occupation, LMP, EDD, Parity, Chief complaints, obstetric history, Menstrual, Marital History, Past history regarding previous medical illness, Family history regarding hypertension and Family onset pregnancy induced hypertension. A detailed General Examination was carried out in the subjects height, weight, pulse Rate, BP, nourishment, Built, Examination of thyroid, Pedal or generalized Edema, Cyanosis, Clubbing, icterus, Lymphadenoparthy, Examination of CVS, RS, CNS. Local examination included per abdominal examination, for Determining GA, fetal viability. Laboratory investigations include Hb%, BT, CT, Blood urea, sugar, serum creatinine, bilirubin were recorded in addition to investigations under study.

**Measurement of Blood Pressure**

The participants were instructed regarding the procedure and getting informed consent.

- Blood Pressure measured by using sphigmomanometry
- Appropriate sized cuff, 1.5 times the mid arm circumference was used.
- Subjects were positioned lying at 45° angle or in sitting position with cuff at heart level.
- Recorded either in the right or left arm,
- For recording systolic BP Korotkoff phase 1 sound was taken
- For recording diastolic BP, Korotkoff Phase 5 was taken.
- The blood pressure varies greatly during every day and isolated readings must be checked after a period of rest. For the diagnosis of hypertension it is accepted practice to require two consecutive abnormally high measurements made at least 4 to 6 hours apart.

**Urine Analysis (Dipstick Testing)**

Uristrix – Bayer India diagnostic reagent strips were used. The impregnated end of the strip was dipped into the freshly collected, well mixed urine. The results were read within 60 sec. The change in strip color were matched with the standard color blocks & the results were documented as negative, trace, 1+, 2+ , 3+, 4+ proteinuria according to the change in strip colors.

**Haematocrit Estimation**

With all aseptic precautions, 2 ml of blood is withdrawn by venepuncture with no regard, to fasting state. The wintrobe's tube filled upto the 100 mark on top with a pasteur pipette, ensuring that there are no air bubbles in the blood column. Centrifuge the tube for 15 min at 3500 rpm until packing is complete. After centrifuging, the blood is separated into three layers, the bottom column constitute the red cells. The percentage of the height of the column of blood occupied by packed Red cells constitute the haematocrit.

**Pepripheral smear (hemolytic evidence and platelet estimation)**

Blood samples were collected from fingertips by pricking with a sterile needle after placing a drop of 14% magnesium sulphate solution on the fingertips, which prevented clumping, and disintergration of platelets. Blood smears were drawn and stained with Leishman stain as done for differential count of white blood cells. Platelet estimation was done by accepted manual method. This consists of counting platelets in 10 oil immersion fields in an ideal stained smear, (ie. A smear with proper stains and free from dust particles, should not have clumping of Cells). When the smear was not an ideal it was discarded and another smear prepared. The total number of platelets in Lacs/$mm^3$ is calculated as average number of platelets / oil immersion field x 20,000. According of platelet estimation the subjects were categorized and severity assessed. Hemolysis were recorded by presence of abnormal peripheral smear with schistocytes, Burr cells, fragmented RBCs and reticulocytosis (Mohapatra *et al.,* 2007; Ramnik Soud *et al.*, 1999; Raymond *et al.,* 1969; Karen *et al.,* 2002).

**Liver Enzyme Estimation**

With all aseptic precautions, 2 ml of blood was collected by vene puncture with no regard to fasting state. The blood was allowed to clot, and retract, the separated serum was stored at 2-8°c. The enzyme levels ( SGOT,SGPT,LDH) is reported to be stable in serum for 3 days at 2-8°C. The serum should be free from haemolysis. The haemolysed serum was discarded and repeat samples obtained. The enzyme assay done in the serum without any hemolysis (Michael et al., 2001; Raymond *et al*., Raymond *et al*., 1962).

**The method and KIT used were,**

| | Method | Kit |
|---|---|---|
| 1. SGOT Estimation | Modified IFCC method | SGOT (ASAT) Kit |
| 2. SGPT Estimation | Modified IFCC Method | SGPT ( ALAT) Kit |
| 3. LDH Estimation | Modified IFCC Method | LDH (P-L) Kit |

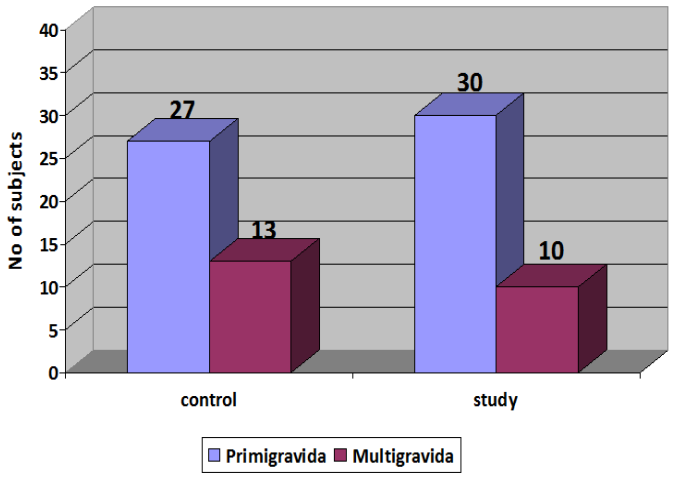
**Instrument was used:** Semi Auto analyser.

**Normal Reference values** – SGOT – Upto 31 U/L at 37°C
SGPT – Upto 34 U/L at 37°C
LDH –Upto230–460U/L at 37°C

## RESULTS

### Statistical Analysis

Statistical analysis was done by using the Statistical Package for Social Sciences (SPSS) X version. The results were analyzed by the Chi - Square test and ANOVA study. Data's were expressed as mean with standard deviation. The control normotensive group were compared with mild and severe pregnancy induced hypertension group. The statistical significance was considered at $P < 0.05$. The present study was conducted in the Department of Physiology, Thanjavur. 80 subjects [40 normotensive pregnant women and 40 pregnancy induced hypertensive women] were recruited for the study.




**Among the study and control groups, the primi gravida constitutes the major proportion**

**Table 1. Demographic and Clinical parameters of Study and Control groups**

| Group | | Age | Ga | Systolic bp | Diastolic bp | Platelet count | Sgot | Sgpt | Ldh | Hct |
|---|---|---|---|---|---|---|---|---|---|---|
| Control | Mean | 24.95 | 34.45 | 116.70 | 75.80 | 2.3668 | 27.58 | 18.12 | 322.20 | 35.6500 |
| | N | 40 | 40 | 40 | 40 | 40 | 40 | 40 | 40 | 40 |
| | Std. Deviation | 3.55 | 3.37 | 6.19 | 5.04 | 0.3895 | 9.28 | 7.03 | 84.46 | 1.8612 |
| Study | Mean | 24.78 | 35.45 | 150.90 | 99.70 | 1.8622 | 48.42 | 38.26 | 627.00 | 35.3250 |
| | N | 40 | 40 | 40 | 40 | 40 | 40 | 40 | 40 | 40 |
| | Std. Deviation | 3.59 | 3.71 | 13.78 | 9.98 | 0.5215 | 69.07 | 49.84 | 261.47 | 5.5395 |
| Total | Mean | 24.86 | 34.95 | 133.80 | 87.75 | 2.1145 | 38.00 | 28.19 | 474.60 | 34.9875 |
| | N | 80 | 80 | 80 | 80 | 80 | 80 | 80 | 80 | 80 |
| | Std. Deviation | 3.55 | 3.56 | 20.22 | 14.36 | 0.5230 | 50.08 | 36.79 | 246.56 | 4.1598 |

The mean age of control group was 24.95 ± 3.55 yrs.
The mean age of study group was 24.78 ± 3.59 yrs.
The mean GA in the control group was 34.45 ± 3.37 wks.
The mean GA in the study group was 35.45 ± 3.71 wks.

**Table 2. Parity distribution among control and study group**

| | | GRAVIDA | | |
|---|---|---|---|---|
| | | MULTI | PRIMI | Total |
| | NORMOTENSIVE | 13 | 27 | 40 |
| | | 32.5% | 67.5% | 100.0% |
| | MILD PIH | 6 | 23 | 29 |
| | | 20.7% | 79.3% | 100.0% |
| | SEVERE PIH | 4 | 7 | 11 |
| | | 36.4% | 63.6% | 100.0% |
| Total | | 23 | 57 | 80 |
| | | 28.8% | 71.3% | 100.0% |

**Table 3. Systolic & diastolic blood pressure in normotensive, mild pih and severe PIH**

| | | SYSTOLIC BP | DIASTOLIC BP |
|---|---|---|---|
| NORMAL | Mean | 116.70 | 75.80 |
| | N | 40 | 40 |
| | Std. Deviation | 6.19 | 5.04 |
| MILD PIH | Mean | 144.21 | 94.41 |
| | N | 29 | 29 |
| | Std. Deviation | 6.24 | 4.35 |
| SEVERE PIH | Mean | 168.55 | 113.64 |
| | N | 11 | 11 |
| | Std. Deviation | 12.62 | 6.38 |
| Total | Mean | 133.80 | 87.75 |
| | N | 80 | 80 |
| | Std. Deviation | 20.22 | 14.36 |

**Table 4. Analysis of systolic & diastolic bp between normotensive, mild & sever PIH by anova study**

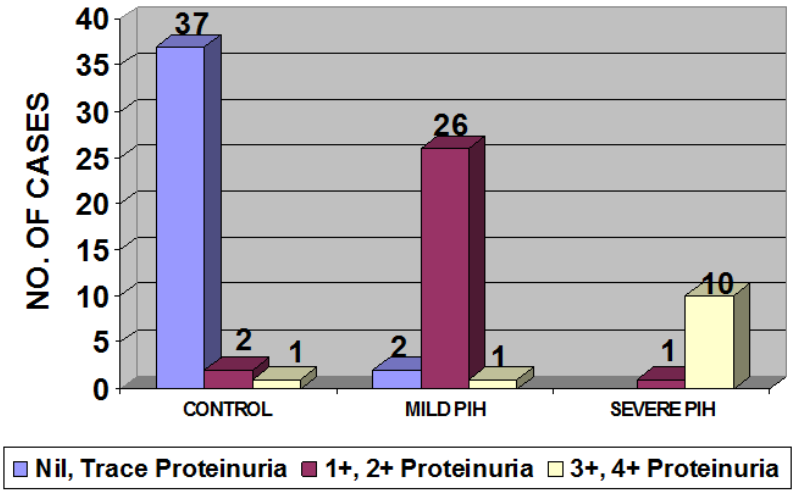
| | | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|---|
| SYSTOLIC BP | Between Groups | (Combined) | 28116.914 | 2 | 14058.457 | 259.227 | < .0005 |
| | Within Groups | | 4175.886 | 77 | 54.232 | | |
| | Total | | 32292.800 | 79 | | | |
| DIASTOLIC BP | Between Groups | (Combined) | 14371.020 | 2 | 7185.510 | 286.976 | <.0005 |
| | Within Groups | | 1927.980 | 77 | 25.039 | | |
| | Total | | 16299.000 | 79 | | | |

In normotensive control, the mean systolic & diastolic BP was 116.70 ± 6.19, 75.80 ± 5.04 mmHg respectively.
In mild PIH, the mean systolic & diastolic BP was 144.21 ± 6.24, 94.41 ± 4.35 mmHg respectively.
In severe PIH, the mean systolic & diastolic BP was 168.55 ± 12.62, 113.64 ± 6.38 mmHg respectively.
The mean difference in Blood Pressure between these 3 groups was significant (P value < 0.0005).

**Table 5. Presence of proteinuria in mild PIH & severe PIH**

| URINE ANALYSIS (Evidence of Proteinuria) | Control | Mild PIH | Severe PIH | Total |
|---|---|---|---|---|
| Nil, Trace | 37 | 2 | - | 39 |
| 1+, 2+ | 2 | 26 | 1 | 29 |
| 3+, 4+ | 1 | 1 | 10 | 12 |
| Total | 40 | 29 | 11 | 80 |

Chi Square P <0.001




In this study 1+ proteinuria were more in the mild PIH. 3+, 4+ proteinuria were present in severe PIH when compared with normotensive control patients. The study results were significant (P < 0.001).

**Table 6. Laboratory results of normotensive control, mild & severe PIH**

| | | PLATELET COUNT | SGOT | SGPT | LDH | HCT |
|---|---|---|---|---|---|---|
| NORMAL | Mean | 2.3668 | 27.58 | 18.12 | 322.20 | 35.6500 |
| | N | 40 | 40 | 40 | 40 | 40 |
| | Std. Deviation | .3895 | 9.28 | 7.03 | 84.46 | 1.8612 |
| MILD PIH | Mean | 2.0414 | 32.42 | 23.61 | 560.72 | 36.2759 |
| | N | 29 | 29 | 29 | 29 | 29 |
| | Std. Deviation | .3650 | 17.64 | 14.61 | 224.50 | 4.5111 |
| SEVERE PIH | Mean | 1.3900 | 90.60 | 76.85 | 801.73 | 29.1818 |
| | N | 11 | 11 | 11 | 11 | 11 |
| | Std. Deviation | .5909 | 122.62 | 82.64 | 281.38 | 4.7501 |
| Total | Mean | 2.1145 | 38.00 | 28.19 | 474.60 | 34.9875 |
| | N | 80 | 80 | 80 | 80 | 80 |
| | Std. Deviation | .5230 | 50.08 | 36.79 | 246.56 | 4.1598 |

**Table 7. Analysis of laboratory results by anova study between normotensive control, mild PIH & severe PIH**

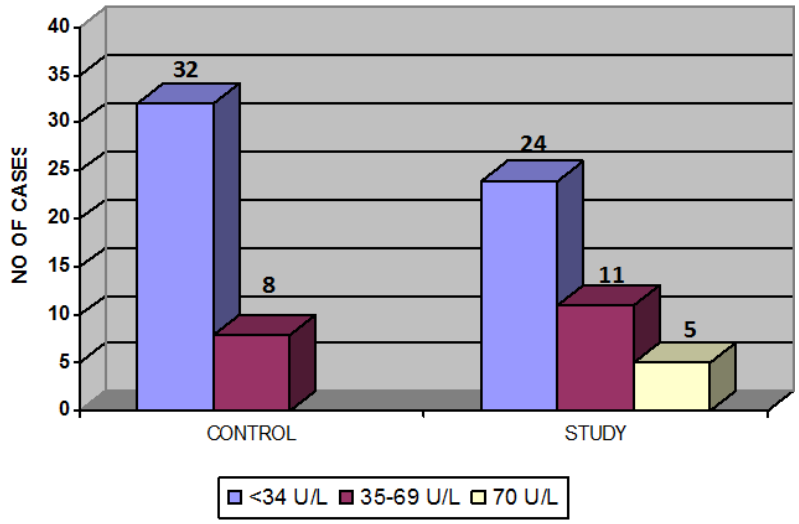
| | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| PLATELET COUNT | Between Groups | 8.474 | 2 | 4.237 | 24.832 | < .0005 |
| | Within Groups | 13.138 | 77 | .171 | | |
| | Total | 21.613 | 79 | | | |
| SGOT | Between Groups | 35683.483 | 2 | 17841.742 | 8.458 | < .0005 |
| | Within Groups | 162429.9 | 77 | 2109.479 | | |
| | Total | 198113.3 | 79 | | | |
| SGPT | Between Groups | 30714.946 | 2 | 15357.473 | 15.519 | < .0005 |
| | Within Groups | 76198.912 | 77 | 989.596 | | |
| | Total | 106913.9 | 79 | | | |
| LDH | Between Groups | 2321269 | 2 | 1160634.413 | 36.018 | < .0005 |
| | Within Groups | 2481230 | 77 | 32223.771 | | |
| | Total | 4802499 | 79 | | | |
| Hct | Between Groups | 436.458 | 2 | 218.229 | 18.058 | < .0005 |
| | Within Groups | 930.529 | 77 | 12.085 | | |
| | Total | 1366.987 | 79 | | | |

In normotensive control, mean platelet count was 2.3668 ± 0.3895 lakhs / mm3.
In mild PIH, mean platelet count was 2.0414 ± 0.3650 lakhs / mm3.
In severe PIH, mean platelet count was 1.3900 ± 0.5909 lakhs / mm3.
The test results show significant reduction in platelet count in the mild and severe PIH ($P < 0.0005$).
The mean difference in the SGOT, SGPT, LDH value in the normotensive control, mild & severe PIH were significant ($P < 0.0005$).
The mean SGOT, SGPT, LDH in normotensive were 27.58 ± 9.28, 18.12 ± 7.03, 322.20 ± 84.46 U/L respectively.
The mean SGOT, SGPT, LDH in mild PIH were 32.42 ± 17.64, 23.61 ± 14.61, 560.72 ± 224.50 U/L respectively.
The mean SGOT, SGPT, LDH in severe PIH were, 90.60 ± 122.62, 76.85 ± 82.64, 801.73 ± 281.38 U/L respectively.
These liver enzymes were markedly elevated in severe PIH.
In the Haematocrit estimation the mean difference between these groups were significant ($P < 0.0005$). Hct% were raised in mild PIH, reduced in severe PIH, with normal results in control group.
The mean Hct% in normotensive, 35.65 ± 1.86%
In mild PIH, 36.27 ± 4.5%
In severe PIH, 29.18 ± 4.75%

**Table 8. The sgot values in normotensive control, mild PIH & severe PIH**

| GROUP | | | NORMAL | MILD PIH | SEVERE PIH | Total |
|---|---|---|---|---|---|---|
| CONTROL | SGOT | < 34 U/L | 32 | | | 32 |
| | | | 100.0% | | | 100.0% |
| | | 35 to 69 U/L | 8 | | | 8 |
| | | | 100.0% | | | 100.0% |
| | Total | | 40 | | | 40 |
| | | | 100.0% | | | 100.0% |
| STUDY | SGOT | < 34 U/L | | 21 | 3 | 24 |
| | | | | 87.5% | 12.5% | 100.0% |
| | | 35 to 69 U/L | | 7 | 4 | 11 |
| | | | | 63.6% | 36.4% | 100.0% |
| | | >70 U/L | | 1 | 4 | 5 |
| | | | | 25.0% | 75.0% | 100.0% |
| | Total | | | 29 | 11 | 40 |
| | | | | 74.4% | 25.6% | 100.0% |

Chi square P = 0.019

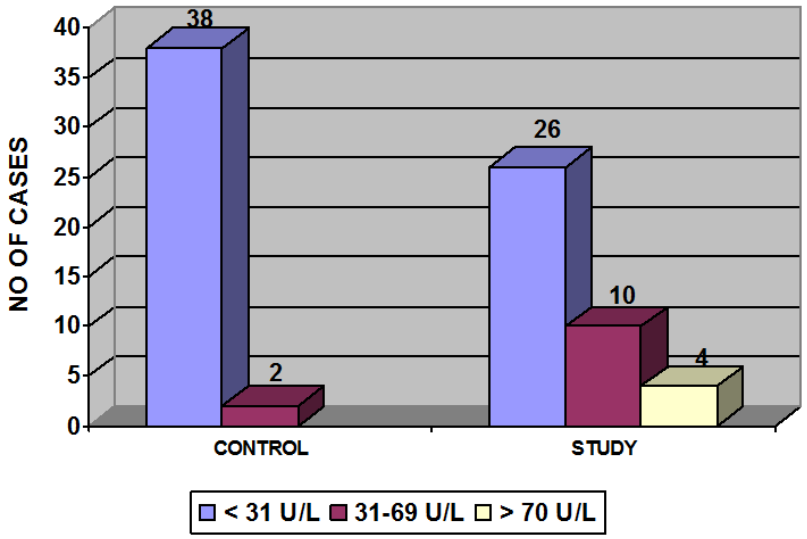



**By chi square study, the test results of SGOT was significant (P = 0.019), marked elevation ( > 70 U/L) was more among severe PIH.**

**Table 9. Study results of SGPT in normotensive control, mild & severe PIH**

| GROUP | | NORMAL | MILD PIH | SEVERE PIH | Total |
|---|---|---|---|---|---|
| CONTROL | SGPT U/ L-31 | 38<br>100.0% | | | 38<br>100.0% |
| | 32-69 | 2<br>100.0% | | | 2<br>100.0% |
| | Total | 40<br>100.0% | | | 40<br>100.0% |
| STUDY | SGPT U/ L 0-31 | | 24<br>92.3% | 2<br>7.7% | 26<br>100.0% |
| | 32-69 | | 4<br>40.0% | 6<br>60.0% | 10<br>100.0% |
| | > 70 | | 1<br>50.0% | 3<br>50.0% | 4<br>100.0% |
| | Total | | 29<br>76.3% | 11<br>23.7% | 40<br>100.0% |

Chi square P = 0.003

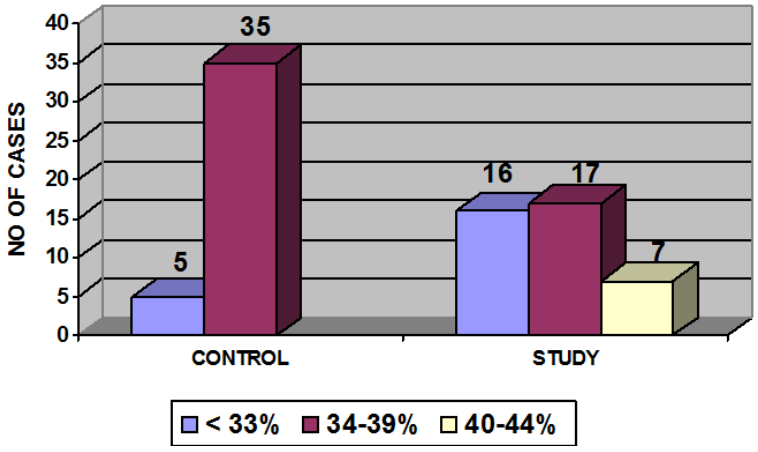



The results of SGPT among normotensive, mild & severe PIH were significant (P = 0.003), marked elevation of SGPT was present in severe PIH when compared with normotensive control & mild PIH

**Table 10. Haemotocrit in normotensive compared with mild PIH & severe PIH**

| GROUP | | | NORMAL | MILD PIH | SEVERE PIH | Total |
|---|---|---|---|---|---|---|
| CONTROL | HCT | <33 % | 5 | | | 5 |
| | | | 100.0% | | | 100.0% |
| | | 34-39 % | 35 | | | 35 |
| | | | 100.0% | | | 100.0% |
| | Total | | 40 | | | 40 |
| | | | 100.0% | | | 100.0% |
| STUDY | HCT | <33 % | | 6 | 10 | 16 |
| | | | | 37.5% | 62.5% | 100.0% |
| | | 34-39 % | | 16 | 1 | 17 |
| | | | | 94.1% | 5.9% | 100.0% |
| | | 40 - 44 % | | 7 | | 7 |
| | | | | 100.0% | | 100.0% |
| | Total | | | 29 | 11 | 40 |
| | | | | 72.5% | 27.5% | 100.0% |

Chi square P < 0.0005




The mean differences in Hct% in all these 3 groups (normotensive, mild & severe PIH) were significant (P < 0.0005). Hct% was elevated in mild PIH & reduced in severe PIH.

**Table 11. Peripheral smear report [evidence of hemolysis] between normotensive control & PIH**

| HEMOLYSIS | GROUP | | Total |
|---|---|---|---|
| | CONTROL | STUDY | |
| Not Present | 40 | 34 | 74 |
| | 54.1% | 45.9% | 100.0% |
| Present | | 6 | 6 |
| | | 100.0% | 100.0% |
| Total | 40 | 40 | 80 |
| | 50.0% | 50.0% | 100.0% |

Chi square P=0.011

The peripheral smear study for hemolysis was significantly present in PIH when compared with normotensives (P = 0.011).

**Table12. Platelet count in normotensives, mild & severe PIH**

| | Platelet Count (Cells/mm3) | Control | Mild PIH | Severe PIH | Total |
|---|---|---|---|---|---|
| Normal Count | > 1,50,000 | 40 | 27 | 3 | 70 |
| Thrombocytopenia | Class1 (more severe) < 50,000 | - | - | 1 | 1 |
| | Class 2 (moderate severe) 50,000 – 1,00,000 | - | - | 2 | 2 |
| | Class 3(less severe) 1,00,000 – 1,50,000 | - | 2 | 5 | 7 |
| Total | | | 40 | 4 | 80 |

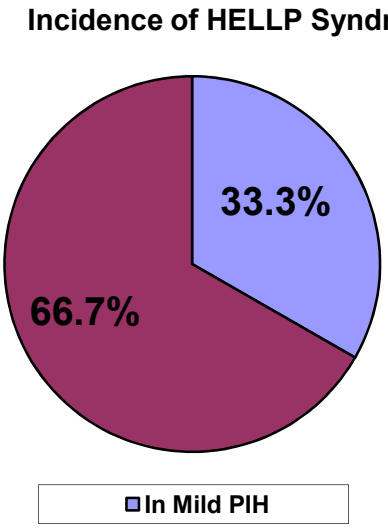
Chi square P = 0.013

The platelet estimation between control, mild PIH & severe PIH were statistically significnat (P value = 0.013). All degrees of thrombocytopenia were present in severe PIH & mild thrombocytopenia was present in mild PIH. Out of 10 cases of thrombocytopenia 6 cases were present with features of HELLP syndrome.

**Table 13. Incidence of hellp syndrome in normotensives, mild PIH & severe PIH**

| | | NORMAL | MILD PIH | SEVERE PIH | Total |
|---|---|---|---|---|---|
| HELPP SYND | NO | 40 | 27 | 7 | 74 |
| | | 54.1% | 36.5% | 9.5% | 100.0% |
| | YES | | 2 | 4 | 6 |
| | | | 33.3% | 66.7% | 100.0% |
| Total | | 40 | 29 | 11 | 80 |
| | | 50.0% | 36.3% | 13.8% | 100.0% |

Chi square P < 0.0001




The incidence of HELLP syndrome was more among severe PIH group. The occurrence rate was significant (P < 0.0001)

**Table 14. Mean systolic & diastolic bp in those with hellp syndorme & without hellp syndrome**

| HELPP SYND | | SYSTOLIC | DIASTOLIC |
|---|---|---|---|
| NO | Mean | 130.95 | 86.08 |
| | N | 74 | 74 |
| | Std. Deviation | 17.42 | 13.38 |
| YES | Mean | 169.00 | 108.33 |
| | N | 6 | 6 |
| | Std. Deviation | 20.54 | 9.83 |
| Total | Mean | 133.80 | 87.75 |
| | N | 80 | 80 |
| | Std. Deviation | 20.22 | 14.36 |

The mean systolic & diasotlic BP in HELLP syndrome was 169 ± 20.54, 108.73 ± 9.83 mmHg respectively.

**Table 15. Analysis of bp between hellp syndrome, PIH & normotensive groups by anova study**

| | | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|---|
| SYSTOLIC * HELPP SYND | Between Groups | (Combined) | 8037.016 | 1 | 8037.016 | 25.845 | <0.0005 |
| | Within Groups | | 24255.784 | 78 | 310.972 | | |
| | Total | | 32292.800 | 79 | | | |
| DIASTOLIC * HELPP SYND | Between Groups | (Combined) | 2748.153 | 1 | 2748.153 | 15.819 | <0.0005 |
| | Within Groups | | 13550.847 | 78 | 173.729 | | |
| | Total | | 16299.000 | 79 | | | |

The mean difference in the systolic & diastolic BP were statistically significant between HELLP syndorme and without HELLP syndrome groups (P < 0.0005)

**Table 16. The laboratory results with hellp syndrome and without hellp syndrome (normotensive, PIH)**

| HELPP SYND | | PLATELET COUNT | SGOT | SGPT | LDH | HCT |
|---|---|---|---|---|---|---|
| NO | Mean | 2.2023 | 29.97 | 22.93 | 432.64 | 35.7432 |
| | N | 74 | 74 | 74 | 74 | 74 |
| | Std. Deviation | .4271 | 12.56 | 21.44 | 193.24 | 3.0880 |
| YES | Mean | 1.0317 | 136.98 | 92.97 | 992.17 | 25.6667 |
| | N | 6 | 6 | 6 | 6 | 6 |
| | Std. Deviation | .3711 | 156.87 | 96.06 | 260.34 | 4.6332 |
| Total | Mean | 2.1145 | 38.00 | 28.19 | 474.60 | 34.9875 |
| | N | 80 | 80 | 80 | 80 | 80 |
| | Std. Deviation | .5230 | 50.08 | 36.79 | 246.56 | 4.1598 |

The mean platelet count was markedly reduced in HELLP syndrome. The mean values of SGOT, SGPT, LDH were markedly elevated in HELLP syndrome & Hct% was reduced when compared with normotensive and PIH.

**Table 17. Analysis of laboratory results between hellp syndrome, PIH and normotensives by anova study**

| | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| PLATELET COUNT | Between Groups | 7.606 | 1 | 7.6060 | 42.353 | <0.0005 |
| | Within Groups | 14.007 | 78 | 0.180 | | |
| | Total | 21.613 | 79 | | | |
| SGOT | Between Groups | 63555.219 | 1 | 63555.219 | 36.841 | <0.0005 |
| | Within Groups | 134558.1 | 78 | 1725.104 | | |
| | Total | 198113.3 | 79 | | | |
| SGPT | Between Groups | 27219.821 | 1 | 27219.821 | 26.641 | <0.0005 |
| | Within Groups | 79694.037 | 78 | 1021.718 | | |
| | Total | 106913.9 | 79 | | | |
| LDH | Between Groups | 1737569 | 1 | 1737569.218 | 44.220 | <0.0005 |
| | Within Groups | 3064930 | 78 | 39293.974 | | |
| | Total | 4802499 | 79 | | | |
| Hct | Between Groups | 563.533 | 1 | 563.533 | 54.708 | <0.0005 |
| | Within Groups | 803.455 | 78 | 10.301 | | |
| | Total | 1366.987 | 79 | | | |

The mean difference of laboratory values (Platelet count, SGOT, SGPT, LDH, Hct%) between normotensives, PIH and HELLP syndrome were significant ($P < 0.0005$).

## DISCUSSION

The present study was done on 40 pregnancy induced hypertensive women and 40 healthy normotensive pregnant women. The present study has shown that the assessment of severity of PIH and early detection of HELLP syndrome was significantly influenced by platelet estimation, peripheral smear study (evidence of hemolysis), haemotocrit estimation and urine analysis for proteinuria. Both groups had similar demographic characteristics. In the study, the mean maternal age was 24.78 ± 3.59 years in the range of 19 to 35 years. The mean GA was 35.45 ± 3.71 wks, in the range of 28 wks to 40 wks, most of the study groups were fall between 32 to 40 wks of gestation. (i.e., late 3$^{rd}$ trimester)

Prakash *et al.* (2006) showed the mean maternal age were 24.75 ± 3.36 years in the range of 19 to 32 years. The vast majority (84%) of patients were found to be hypertensive in the III trimester (Prakash *et al*., 2006). The present study congruent with this literature cited. In the present study the majority of patients were primigravida 30 (75%) and 10 (25%) were multigravida. In many studies the frequency was more among primigravida. Ahmed. F A et al showed the occurrence of PIH were more among primigravida (71.7%) than in multigravida (28.3%) Ahmed *et al* (2007). The present study has parity distribution were as that of literature quoted.

Shyam Sunder Sud *et al.* (2006) explained the minimum criteria for PIH was BP≥ 140/90mmHg and increased certainty of preeclampsia was BP ≥160 / 110 mmHg (Shyam Sunder Sud *et al.,* 2006). In the present study, the mean systolic and diastolic BP was in mild PIH about 144.21 ± 6.24 mmHg, 94.41 ± 4.35 mmHg respectively and in severe PIH, the mean systolic and diastolic BP was 168.55 ± 12.62 mmHg, 113.6 ± 6.38 mmHg respectively. This study values were coincide with the reference cited.

Gary Cunningham *et al.* (2005) showed that proteinuria is a sign of worsening hypertensive disease. It increases perinatal morbidity and mortality. Proteinuria 1+ dipstick or more in random urine samples greatly bolters the diagnosis (Gary Cunningham *et al*. 2005). In the present study 65% of cases were present with 1+ proteinuria, 35% cases were present with 3+, 4+ proteinuria. The study results coincides with the reference cited. A significant decrease in platelet numbers were observed as the mean blood pressure increases in the study group. The degree of thrombocytopenia increases with severity of disease. In the present study the mean platelet count were found to be 2.3668 ± .3895 lakhs/mm$^3$ in the normotensive control group, 2.0414 ± 0.3650 lakhs/mm$^3$ in the mild PIH and 1.3900 ± 0.5909 lakhs/mm$^3$group in severe PIH group. This indicates that thrombocytopenia is directly proportional to the severity of PIH.

Mohapatra *et al.* (2007) compared the values of platelet estimation between study and control groups. They showed that the platelet estimation in the control group was 2.38 lakhs/$mm^3$. In mid PIH the estimation was around 2.23 ± 0.19 lakhs/$mm^3$, and in severe PIH it was 1.83 ± 0.45 lakhs/$mm^3$. They observed a significant decrease in platelet numbers as the mean blood pressure increases in the study group (Mohapatra *et al*., 2007). So the present study congruent with what is said in this literature.

Theodore E Warkentin explained an isolated maternal thrombocytopenia without microangipathic hemolysis or liver dysfunction regardless of the degree of hypertension indicates a worst prognosis and need prompt treatment (Theodore *et al.,* 2nd edition). Courtney Reynolds observed an incidence of 15 to 20% thrombocytopenia in the PIH group (Courtney Reynolds *et al*., 9th edition). In the present study, thrombocytopenia (< 1,50,000/$mm^3$) found to be in 25% of the PIH group. The study result coincides with the reference cited.

Prakash *et al.* (2006) states that when liver dysfunction occurs, mild elevation of transaminases in common. He observed majority of PIH had mild rise, (76.39%) and small numbers of patients (555%) had marked rise in SGOT level. The SGPT level mildly elevated in most of the patients (59.72%) and a marked rise in 8.33% of cases. LDH levels was markedly elevated in 9.72% of patients of which 5 patients had HELLP syndrome (Prakash *et al*., 2006).

Wayne R Cohen showed liver dysfunction defined by elevated SGOT was identified in 21% of PIH (Wayne *et al*., 2000). In the present study majority (27.5%) had mild rise in SGOT (35 to 70 U/L) and a small number (12.5%) of cases had marked rise in SGOT (> 70 U/L). Related to SGPT, most patients (25%) had a mild rise (31 to 70 U/L) & 10% of patients had a marked rise (>70 U/L). LDH levels were mildly elevated (460 – 600 U/L) in 35% of patients and a marked rise (> 600 U/L) in 42.25% of cases of which 6 had HELLP syndrome. The finding of this present study correlated with the reference quoted.

Rebecco W. Van Dyke showed liver involvement was more common upto 70% in severe preeclampsic, accounting for 20% MMR in preeclampsia (Rebecca *et al.,* 2006).

In the present study the elevation of SGOT observed in 72.5% of severe PIH cases. Again thin finding also coincide with the reference cited.

Courtney Reynolds observed microangipathic hemolytic anemia without other signs of disseminated intravascular coagulation in 5% of PIH (Courtney Reynolds *et al.,* 9th editon). In the present study 15% of PIH cases showed features of hemolysis. This study congrue with the literature finding. Dorothee Perloff showed that in normal pregnancy the Hct% was reduced. In PIH it may be normal or elevated, reflecting hemoconcentration due to reduced plasma volume or further reduced owing to hemolysis (Kalyan barmade *et al*., 2003). Kalyan Barmade showed in mild PIH, there is an elevation of Hct % (Kalyan barmade *et al*., 2003).

Nick-Anim-Nyame et al observed mean Hct% were higher in the preeclampsia group (control = 31± .02, precclamspia = 0.33 ± 0.03) (Nick Anim *et al*., 2001).

In the present study the mean Hct% in the control was 35.6500 ± 1.8612. In the mild PIH, it was 36.2759 ± 4.5111. In severe PIH, it was 29.1818 ± 4.7501. So, this study results also tally with the literatures cited.

HELLP syndrome in generally thought to be a progression or complication of PIH.

Rodger L Bick showed an incidence of HELLP syndrome in 15 to 26% of PIH (Proger *et al.,*2002) .

Shyam Sundar Sud showed an incidence of 10-20% of HELLP syndrome in PIH (Shyam Sunder Sud *et al.,* 2006).

In the present study, the HELLP syndrome was observed in 15% of PIH.

Rebecca W. Van Dyke showed the relationship between HELLP syndrome and preeclampsia was variable, as hypertension was mild in 25% of patients with HELLP syndrome (Rebecca *et al*., 2006).

In the present study the 33.3% of mild PIH showed HELLP syndrome. This result also coincides with the literature cited.

**Conclusion**

Pregnancy is one of the wonderful and noble services imposed by nature. More than 16-17% of pregnant women face various problems related to pregnancy and child birth. The preeclampsia and HELLP syndrome are still the leading causes of maternal, perinatal mortality & morbidity. The progression of PIH from mild to life threatening diseases cannot be predicted. The aim of this study to draw attention to the life threatening complication such as hepatic dysfunction, haematological abnormalities and HELLP syndrome that may occur in cases of preeclampsia. The early diagnosis and early assessment of severity by platelet estimation, peripheral smear study, liver enzyme assays & urine analysis for proteinuria would be the most effective approach to enhance both maternal and fetal well being, as well as the successful outcome of pregnancy.

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