STUDY OF PLATELET COUNT, PROTHROMBIN TIME AND ACTIVATED PARTIAL THROMBOPLASTIN TIME IN PREGNANCY INDUCED HYPERTENSION (PIH)

By

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In

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Under the guidance of

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A line from Sanskrit Shlokha Says “Guru brahma guru vishnu gurudevo maheshwaraha, guru sakshaat parabrahma tasmay shrigurave namaha” - meaning a teacher is next to god and without him knowledge is always incomplete

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ABSTRACT

BACKGROUND: Hypertensive disorders complicate 5-10% of pregnancies and together they form one member of the deadly triad along with hemorrhage and infection that contribute greatly to maternal and mortality rates Haematological abnormalities such as low platelet count and decrease in some plasma clotting factors may develop in preeclamptic women. Such subtle changes with disseminated intravascular coagulation (DIC) are potentially serious.

There is no universal agreement as to the need for further investigation if the platelet count comes normal .Thus coagulation testing is indicated in these patients for evidence of DIC and HELLP syndrome-Haemolysis. enzyme elevation and low platelet to prevent coagulation failure.

OBJECTIVES: To correlate the platelet count, prothrombin time (PT) and activated partial thromboplastin time (aPTT) in different trimesters of pregnancy in pregnancy induced hypertension (PIH).

METHODS :A crossectional study of 105 cases of all patients clinically diagnosed as PIH cases who have attended OPD & wards of department of obstetrics and gynaecology in BLDE University, Shri B.M.Patil Medical College, Hospital and Research centre, Bijapur from November 2011 to April 2013 were taken for the study.

A detailed history, complete general physical examination, systemic review of the patients and laboratory investigations was undertaken.

RESULTS: Out of 105 cases, 75% cases were in age group of 21-30yrs, 67 % cases were primigravidas and 92 cases were in third trimester .Among PIH cases, 22 cases were gestational hypertension,19 cases were mild preeclampsia, 35 cases were severe preeclampsia and 39 cases of eclampsia. Thrombocytopenia was seen in 24% cases ,81% with prolonged PT, and 6% with prolonged aPTT. Two cases of HELLP syndrome was observed. Thrombocytopenia, prolonged PT was most commonly observed in severe preeclampsia cases.

CONCLUSION: Preeclampsia and eclampsia remains a serious complication of pregnancy. This study gives an outline of the investigations to be carried out in PIH which can alert the physician of the severity of condition, so that appropriate and timely management can be iniated.

KEY WORDS:

Preeclampsia, eclampsia, coagulation failure

LIST OF ABBREVIATIONS USED

PIH	Pregnancy induced hypertension
GH	Gestational Hypertension
MP	Mild Preeclampsia
SP	Severe Precclampsia
E	Eclampsia
PT	Prothrombin time
aPTT	Activated partial thromboplastin time
DIC	Disseminated intravascular coagulation
HELLP	Haemolysis, elevated liver enzymes, low platelets
PG	Prostaglandins
FDP	Fibrin degradation products
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
LFT	Liver function test

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INTRODUCTION

Hypertensive disorders of pregnancy complicate 5-10% of pregnancies and these along with hemorrhage and infection form the deadly triad that contribute greatly to maternal and child mortality rates.[1]

Pregnancy induced hypertension (PIH) is defined as hypertension that develops as a result of gravid state after 20 weeks of gestation. It includes gestational hypertension, preeclampsia and eclampsia.[2]

Most common immediate maternal complications of preeclampsia can be eclampsia, accidental hemorrhage, blindness, preterm labour and HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelet count) during pregnancy, postpartum hemorrhage during labour and shock along with sepsis during puerperium.

Remote complications of preeeclampsia include residual hypertension, recurrent preeclampsia and chronic renal failure of the mother.

The fetal complications include intrauterine death, intrauterine growth restriction, prematurity and asphyxia.

Many hemostatic abnormalities have been reported in association with PIH. Studies have proved that these parameters show more abnormal result with increasing severity of PIH.

Thrombocytopenia, the most common condition, may at times become severe and life threatening. Thus the alterations in coagulation parameters including platelet count, prothrombin time (PT) and activated partial thromboplastin time (aPTT) have predictive value in detecting disseminated intravascular coagulation (DIC) in PIH and such subtle changes with DIC are potentially serious.[1]

Hence this study is aimed to analyse platelet count, prothrombin time (PT) and activated partial thromboplastin time (aPTT) in patients with pregnancy induced hypertension (PIH) which are helpful for early detection, careful monitoring and appropriate management in turn to reduce the morbidity and mortality of both mother and fetus.

OBJECTIVES

To correlate the platelet count, prothrombin time (PT) and activated partial thromboplastin time (aPTT) in different trimesters of pregnancy in pregnancy induced hypertension (PIH).

REVIEW OF LITERATURE

Haematological Abnormalities in PIH

Many haematological abnormalities develop in hypertensive disorders due to pregnancy. Among these are thrombocytopenia which at times may become so severe as to be life threatening, the level of few plasma clotting factors may be decreased and erythrocytes may be so traumatised that they display bizarre shapes and undergo rapid haemolysis.[1]

Platelet disorder:

Kate and Romero associates stated that maternal thrombocytopenia can be induced actually by preeclampsia and eclampsia. After delivery, the platelet count will increase progressively to reach the normal level within few days (Kate and associates 1990, Romero and collegues 1989).

The frequency and intensity of maternal thrombocytopenia vary and are dependent on the intensity of disease process, the length of delay between the onset of the disease process, the length of delay between the onset of preeclampsia and delivery ,and the frequency with which platelet count is less than 100,000/ul is an ominous sign. It indicates severe disease and delivery is usually indicated because the platelet count continues to decrease.[1]

Burrows et.al stated that thrombocytopenia is the most common hemostatic abnormality of PIH.[3]

Keltonet.al observed that approximately 20% of patients with preeclampsia who develop a consumptive thrombocytopenia is usually mild ,but can be severe.[4]

Mathur et.al and Keehan and Bell conducted a study on 30 eclampsia women and observed that platelet count was between 1,00,000/cumm to 1,30,000/ Cumm. The definite decrease was statistically significant.[5,6]

Schwartz et.al Observed that usually thrombocytopenia is an incidental finding and the platelet count does not decrease to a level that requires platelet transfusion.[7]

Rahim R et.al conducted a study on 100 cases of PIH to observe the platelet count .Out of the total cases, 47 % had low platelet count (<1,50,000/cumm). Among eclampsia group, 60 % had low platelet count .12% cases developed postpartum haemorrhage (PPH) and among them 66 % had low platelet count. Mortality was 3% and all were in eclamptic group. Those patients with low platelet count, 75% had low birth weight (LBW) babies. Study concluded that platelet count is very important investigation for antenatal mother having PIH, as it is directly related to maternal and perinatal outcome.[8]

Walker J et.al showed that in preeclampsia there are changes in platelet number, platelet survival and mean platelet volume, which have been interpreted as evidence of increased platelet consumption. Study had suggested that platelet count falls early in the disease and precedes hypertensive renal changes suggesting that platelet consumption plays an active role in the pathophysiology of this disorder.[9]

Mohapathra S et.al study included 30 normal pregnant women and 90 pregnant women with varying degrees of PIH. Platelet count were estimated during the second and third trimester. Platelet levels gradually decreased as the severity of the PIH

increased. The study concluded that there was an inverse relationship between the severity of PIH and platelet count.[10]

Ugur Kazimonglu et.al conducted a study to evaluate the clinical utility of the laboratory studies, especially coagulation tests in the evaluation of the patients with preeclampsia. Coagulation profile, platelet count, serum levels of LDH and transaminases of 120 preeeclamptic patients were evaluated. Thrombocytopenia and elevated liver enzymes were observed in 32 patients. The coagulation profile was within normal limits in 68 patients. Their study recommended that the LDH with or without platelet count should be assessed in the evaluation of coagulation abnormalities in the preeclamptic patients.[11]

Vamseedhar Annam et.al conducted study to evaluate platelet indices and platelet counts and their significance in preeclampsia and eclampsia. 82 cases of preeclampsia and 63 cases of eclampsia diagnosed were evaluated prospectively with 100 healthy pregnant women as the control group. The platelet counts were lower while the mean platelet volume, platelet distribution width and platelet large cell ratio were increased in preeclampsia and eclampsia as compared to control group. They also found a relationship between platelet indices and severity of preeclampsia.[12]

John G Kelton et.al conducted a prospective study on 26 preeclamptic patients and 17 pregnant control subjects. PT, aPTT, TT and platelet count tests were done. Study suggested that patients with preeclampsia can have a significant defect in platelet function as well as platelet number.[13]

Kulkarni RD et.al conducted study on 84 pregnant females, out of which 20 were normal pregnant women, 55 were cases of pregnancy with toxemia and 9 cases of

pregnant women in whom roll over test was positive. Platelet count in the normal pregnant women was found to be in the normal range. In mild and severe toxemia, there was fall in platelet count. The study concluded that lowering of platelet count was a feature of toxemia of pregnancy.[14]

Consumuptive Coagulopathy in PIH:

It has been well recognised that consumptive coagulopathy is concomitant with certain obstetrics entities like abruptio placenta, intrauterine death, amniotic fluid embolism, septic abortion, hydatiform mole etc. However concomitant involvement of coagulation system in toxaemia, particulary severe preeclampsia is less appreciated. The occurrence of coagulation defects in eclampsia is better documented than in preeclampsia. In 1964 Mckay presented definite evidence that there was slowly progressive intravascular coagulation in preeclamptic patients. These changes are often associated with fetal distress .Hence the diversity of organ system involvement is a reflection of variability of consumptive coagulopathy.[15]

Activation of both coagulation and fibrinolytic system leads to development of DIC. However clinically evident DIC occurs in only the most severe cases and measurement of PT, aPTT, FDP and fibrinogen levels in preeclamptic patients usually yields normal results . Nevertheless, more sensitive assays of procoagulation system becomes activated to a subtle degree in many preeclamptic patients , who manifest neither clinical nor classic laboratory manifestations of DIC.[16]

Studies were sought to determine whether a normal platelet count assures that no other clinically significant clotting abnormalities are present and the level of

thrombocytopenia predicts a risk of abnormalities in other coagulation indices provides a conflicting data.

Leduc L et.al conducted a study on 100 women with severe preeclampsia.50 women had platelet counts below 150,000/µl, of whom 13 and 2 cases had a prolonged PT and aPTT respectively. Study concluded that thrombocytopenia was a strong indicator of severity of PIH .When monitoring intrapartum coagulation indices in preeclampsia, one can safely follow only the platelet count at admission and subsequently ,reserving PT, aPTT and fibrinogen levels for those cases complicated by counts less than 1,00,000/ul.[17]

Fitzgerald MP et.al conducted a retrospective study and surveyed patients with PIH in two hospitals and observed that 37% had thrombocytopenia, 16% prolonged PT, and 12% prolonged aPTT. These majority of abnormal results occurred in the group of patients with severe preeclampsia.[18]

Jahromi BN et.al conducted a study on coagulation factors in preeclampsia and concluded that platelet count >150,000/mm3 cannot assure the physician that no other significant clotting abnormalities are present. However, measurement of aPTT seems to be important for early detection of coagulation abnormalities in patients with severe eclampsia who have normal platelet counts. The study also stated that FDP (fibrin degradation products) measurement does not seem to be appropriate screening test since it is expensive and of little help in diagnosis. This can achieved by measuring aPTT and ongoing coagulopathy should be suspected and clinically judged if either thrombocytopenia or prolongation of aPTT is found in patients with severe preeclampsia.[19]

Jambhulkar S et.al conducted study on 174 cases of PIH and 50 controls and observed that mild preeclampsia showed no abnormality in platelet and coagulation parameters.In severe preeclampsia decrease in platelet count was highly significant ($p<0.01$) and PTTK were significantly prolonged ($p<0.05$). In eclampsia, platelet count was significantly decreased ($p<0.01$) and PTTK was significantly prolonged ($p<0.05$). Study concluded that abnormalities pertaining to coagulation parameters in pregnancy in PIH indicate intravascular coagulation. Platelet count and PTTK have predictive value in detecting DIC in PIH and these parameters show more abnormal result with increasing severity of PIH.[20]

Orlikowski et.al conducted a study on 49 patients. Among these 7 were diagnosed as mild pre-eclamptic, 33 severe pre-eclamptic and rest 9 were diagnosed with eclampsia. Abnormal platelet count was seen in 25 out of 49 patients and 2 patients had prolonged PT with thrombocytopenia.[21]

Agarwal S et.al conducted study on 100 subjects out of whom 10 were normal non pregnant, 20 normal pregnant, 40 cases of mild preeclampsia, 20 of severe preeclampsia and 10 of eclampsia. In eclampsia, platelet count and plasma fibrinogen levels decreased, and platelet adhesiveness and fibrinolytic activity markedly increased. Their study suggested that there was a slow process of DIC in toxemias of pregnancy, and this process occurs to a greater extent in eclampsia.[22]

Jack A Prichard et.al study on eclamptic patients identified thrombocytopenia in 29% of patients, prolonged bleeding time in 50%, abnormally elevated serum fibrinogen, FDP in 3%, and circulating fibrin monomer in 5% of patients. Study concluded that the coagulation changes when present in eclampsia, were effect rather than cause.[23]

Dube B et.al conducted a study on 12 patients with pre-eclampsia, 15 with eclampsia and 15 with normal pregnancy in their third trimester. Coagulation studies showed significant prolongation of TT, elevation of serum FDP and hypofibrinogenemia in patients with pre-eclampsia as well as eclampsia. In patients with eclampsia, significant thrombocytopenia was observed. With these findings, they suggested that there was occurrence of intravascular coagulation in patients with preeclampsia and eclampsia.[24]

Pathogenesis of Haematological Abnormalities In PIH

There are various studies put forward by different authors for the mechanisms of haematological abnormalities in PIH .

Mechanisms for Thrombocytopenia :

Thrombocytopenia occurs in approximately 15% of patients with preeclampsia and eclampsia. It occurs in these patients without any evidence of coagulation disorder .There are several possible mechanisms to explain the reduction in the platelet number.

First, it may occur after generation of thrombin in the presence of circulating immune complexes and vascular disruption. Second, an increase in platelet agglutination and aggregation may contribute. Third, an immune mechanism has been implicated in a patient, whose platelets agglutinated in vitro in the presence of serum, when the serum was pre incubated with placental cells.

Thus platelets may be involved in an immune mediated phenomenon in some cases of preeclampsia.[25]

McKay et.al observed in their study conducted on eclampsia cases that decrease in platelet count was due to increased consumption and increased platelet adhesiveness.[26]

Howeiet.al conducted study on PIH cases and showed that mechanism of thrombocytopenia was due to persistent impaired platelet disaggregation.[27]

Platelet Activation

Platelet activation plays an important role in the pathogenesis of preeclampsia as demonstrated by reduced platelet count, increased mean platelet volume and elevated plasma concentrations of beta-thromboglobulin and platelet factor 4 in preeclamptic patients. Thrombocytopenia precedes the onset of clinical symptoms of preeclampsia. Possibly an enhanced state of the platelets in the circulation is present some time before the onset of preeclampsia : platelet activation might then be used to predict preeclampsia.

So, flow cytometry is considered the most sensitive technique at this moment to measure the activation of platelets. Fluorescent-labelled antibodies are used to detect antigens that appear on the platelet surface or change their conformation upon activation. During the first and second trimester of pregnancy preeclamptic patients have an increased expression of some antigens on the surface their platelets such as CD63.There is no reliable platelet test yet to predict the onset of preeclampsia.[28]

Sunita Ahlawat et.al demonstrated platelet aggregation indicated the presence of both activated (hyperaggregable) as well as exhausted (hypoaggregable) platelets in

circulation. Platelet activating factor (PAF) was demonstratable in 45.4% of preeclampsia suggesting its role in the mechanism of platelet activation in PIH.[29]

Samuels et.al performed direct and indirect antiglobulin tests and found that platelet bound and circulating bindable were increased in preeclamptic women and their neonates. They interpreted these findings as platelets surface alteration.[30]

Barron et.al reported that platelets from preeclamptic women were more likely to have platelet associated IgG, even if thrombocytopenia did not develop. Although they also believed that this mechanism implied ,that an autoimmune process involving IgG, could be bound to platelets damaged by any mechanism.[31]

Yusuf Ahmed et.al conducted a study which included 428 women with normal pregnancy and 74 women with preeclampsia from whom platlet measurements were available between 27 and 30 weeks of gestation .Mean platelet volume (MPV) and platelet number remained constant in normal pregnancies between the first trimester and the end of pregnancy. A persistent increase in MPV was found in 1 out of 15 preeclamptic patients between 24 weeks of gestation and in only 13 of 428 normal pregnant individuals. Platelet numbers were decreased in 12 of the 15 patients with preeclampsia.10 % of normal pregnant individuals showed a similar decline in platelet numbers showing that changes in platelet numbers may be less accurate assesement of the development of preeclampsia.[32]

Pathogenesis of Coagulation in PIH :

Haematological changes consistent with intravascular coagulation and less often erythrocyte destruction, may complicate preeclamptic patients and specially

eclamptics. Renewed interest in these changes has lead to the concept by some investigators that DIC is not only a characteristic features of preeclampsia but also plays a dominant role in the pathogenesis.

There is increasing evidence that enhanced coagulation activity is involved in the pathogenesis of preeclampsia. The activities of several clotting factors may be altered significantly in the preeclampsia patient .The impact of these changes may be expressed in both the haemostatic and fibrinolytic system. Preeclampsia and eclampsia accentuates this state of hypercoagulability already produced by normal pregnancy.[1]

Intrinsic and common pathways

It has been suggested that intrinsic pathway activation may be altered by preeclampsia. Accelerated PT was found in preeclamptic patients that was associated with changes in fibrinogen and Factors II, V and X. Thus the common pathway appears to be hypercoagulable in the preeclamptic patients.

Fibrinogen

A significant fall in the plasma fibrinogen level has been found in patients with severe preeclampsia, a finding which suggests fibrin deposition .This constitutes a significant difference PIH patients.

Gokhan Acmaz et.al conducted study in preeclamptics based on gestational weeks to detect fibrinogen levels in groups which were comprised on the basis of lung maturity. Statistical difference between preeclamptic and healthy pregnant ($p=0.012$)

was found. Elevated levels of fibrinogen in healthy pregnancies and decreased levels of fibrinogen in preeclamptic patients were detected.[33]

Fibrinolytic System

Fibrin deposition as emphazised earlier is common in patients with severe preeclampsia and may be responsible for the various clinical manifestations. It would be reasonable to anticipate that the fibrinolytic system would become activated in the presence of fibrin deposition.

There is a tissue type plasminogen activator produced by endothelial cells that may be released secondary to endothelial damage in the preeclamptic patients and thereby induce the fibrinolytic system.

Belo L et.al conducted a study on two groups of women (normal and preeclamptic patients) in their third triimesters and measured platelet number ,plasma fibrinogen and tissue plasminogen activator(t-PA) and PAI-I and found that similar values were noted for fibrinogen, platelet counts but higher values for t-PA in preeclamtic women. t-PA correlated positively and significantly with degree of proteinuria in preeclamtic women.[34]

HELLP (Haemolysis,elevated liver enzymes,low platelets) syndrome

The HELLP syndrome has received much attention in recent years, However this syndrome does not appear to be a unique disorder but rather a variant of preeclampsia, first described in 1975.The phrase “HELLP” was first suggested in 1982 and the criteria for diagnosis of this syndrome includes

1. Microangiopathic haemolytic anemia with schistocytes on peripheral blood film.
2. Serum glutamic oxaloacetic transaminase(SGOT) ≥ 70U/L
3. Thrombocytopenia with a platelet count less than 1,00,000/ul

However many women with severe preeclampsia may have laboratory abnormalities like isolated thrombocytopenia or elevated liver enzymes without the complete HELLP syndrome. It remains unclear whether these women should be managed like any other women with severe preeclampsia.[35]

Based on the lowest observed maternal platelet count, HELLP syndrome is classified into 3 classes.[36]

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

Class 2- if platelet count >50,000/cumm and < 1,00,000cumm

Class 3- if platelet count >1,00,000/cumm and < 1,50,000cumm

The pathophysiological mechanism of the HELLP syndrome is incompleletly understood.

Currently endothelial or trophoblastic dysfunction in uteroplacental system is considered to play a central role in initiation of the syndrome.[37]

De.Boer K et.al using a set of sensitive and specific coagulation assays studied providing convincing evidence that compensated DIC is present in all HELLP patients. Parmer V.M Patil and Deshpande A.K stated that HELLP syndrome occurs in nearly 10% of severely preclampticpatients.They also suggested that considering the high incidence of preeclampsia in our country ,this syndrome obviously appears to be under diagnosed .[37]

Martin J.N et.al found that 50 % of pregnancies complicated by class1 HELLP syndrome exhibited significant maternal morbidty compared with only 11% of those complicated by severe preeclampsia without HELLP syndrome. Maternal morbidity can be frequent and significant when HELLP syndrome occurred in association with eclmapsia. The need for blood products and infection were the two most common forms of maternal morbidity.[38]

Jaleel A, Baseer A study observed that there was significant ($p<0.01$) reduction in platelet count of preeclamptic and highly significant ($p<0.001$) in eclamptic women as compared to controls. It was concluded that there is need to do platelet count in all pregnancy induced hypertensive women, which can be an earlier detector for HELLP syndrome.[39]

One must be aware of HELLP syndrome as well as its clinical and laboratory findings, so that proper therapy can be initiated soo that maternal and fetal deaths can be held to a minimum.[40]

Introduction To Pregnancy Induced Hypertension (PIH)

Pregnancy induced hypertension (PIH) is defined as hypertension that develops as a result of gravid state after 20 weeks and it includes Gestational hypertension, Pre-eclampsia & Eclampsia.

According to schema of the Working Group of the NHBPEP-National High Blood Pressure Education Programe (2000), hypertensive disorders complicating pregnancy describes four types of hypertensive disease.[1]

1. Gestational hypertension-formerly termed pregnancy induced hypertension.

2. Preeclampsia and Eclampsia syndrome.
3. Preeclampsia syndrome superimposed on chronic hypertension.
4. Chronic hypertension.

Pregnancy induced hypertension(PIH) is defined as hypertension that develops as a result of gravid state.It includes

- Gestational hypertension
- Pre-eclampsia
- Eclampsia

1. **Gestational hypertension is defined as :**
   1 Systolic BP≥140 mmHg or Diastolic BP≥90mmHg for first time during pregnancy.
   2 No proteinuria.
   3 Blood pressure returns to normal before 12weeks postpartum.
   4 May have other signs or symptoms of preeclampsia.Eg-epigastric discomfort or thrombocytopenia.

Calculation is based on mean arterial pressure(MAP) advocated by **Page**[2]

$$MAP = \frac{\text{Systolic pressure}+(\text{diastolic pressure} \times 2)}{3}$$

A rise of 20mmHg MAP over previous reading ,or when MAP is 105mmHg or more should be considered significant.

The rise of blood pressure should be evident at least on two occasionsatleast 6 hours apart.

2. Preeclampsia:

Preeclampsia is a pregnancy specific syndrome of reduced organ perfusion secondary to vasospasm and endothelial activation. Proteinuria is an important sign of preeclampsia and the diagnosis is questionable in its absence. Proteinuria is defined as 300gm or more urinary protein per 24 hrs or persistent 30 mg/dl (1+dipstick) in random urine samples.

The minimum criteria for diagnosis of eclampsia are hypertension plus minimal proteinuria. The more severe the hypertension or proteinuria the more certain is the diagnosis of preeclampsia. Similarly abnormal laboratory findings in tests of renal ,hepatic and haematological function increases the certainity of Pre eclampsia. Persistent premonitory symptoms of eclampsia like headache and epigastric pain also increases the certainity of pre eclampsia.

Minimum criteria:

(1)BP≥140/90mmhg after 20 weeks gestation

(2)Proteinuria ≥300mg/24hrs or ≥1+dipstick

Increased certainity of preeclampsia

1 BP≥160/110mmhg.

2 Proteinuria 2g/24hrs or ≥2+dipstick.

3 Serum creatinine>1.2mg/dl unless known to be previously elevated.

4 Platelets<100,000/ul.

5 Microangiopathic hemolysis-increased LDH.

6 Elevated transaminase levels-ALT, AST.

7 Persistent headache or visual disturbance, epigastric pain.

Table 1.The severity of pre eclampsia is assessed by frequency and intensity of abnormalities listed below. [1]

Abnormality	Mild	Severe
Systolic blood pressure	<160mmHg	≥160mmHg
Diastolic Blood pressure	<110mmHg	≥110mmHg
Proteinuria	Trace to 1+	Persistent 2+ or more
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsions	Absent	Present
Serum creatinine	Normal	Elevated
Thrombocytopenia	Absent	Present
Liver enzyme elevation	Minimal	Marked
Fetal growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present

Importantly the differentiation between mild and severe preeclampsia can be misleading because apparently mild disease may progress rapidly to severe disease.

Eclampsia:

Eclampsia is defined as occurrence of seizures in women with preeclampsia that cannot be attributed to other causes. The seizures are generalised and may appear before, during or after labor.

Incidence and risk factors[1]

Gestational hypertension more often affects nulliparous women. Older women who acquire an increasing incidence of chronic hypertension with advancing age are at greater risk for superimposed eclampsia. Thus women at either end of reproductive age are considered to be more susceptible.

The incidence of preeclampsia is about 5%, which is markedly influenced by parity, race, ethnicity, genetic predisposition and environmental factors .Other risk factors associated with preeclampsia include multiple pregnancy ,history of chronic hypertension ,maternal age over 35 years and obesity. The relationship between maternal weight and risk of preeclampsia is progressive.

Moreover women with twins and hypertensive disorder due to pregnancy experience higher rates of adverse neonatal outcomes than do those with singletons.[1]

Pathophysiology :

Pathological deterioration of function in a number of organs and systems presumably as a consequence of vasospasm and ischemia has been identified in severe pre eclampsia and eclampsia. The major cause of fetal compromise occurs as a consequence of reduced uteroplacental perfusion. [1]

Haemodynamic changes:

With preeclampsia there is a marked reduction in cardiac output and increased peripheral resistence. By contrast women with gestational hypertension have significantly elevated cardiac output before and during the development of chronic hypertension.

Blood volume:

It has been known for over 100 years that haemoconcentration is a hallmark of eclampsia. In eclamptic women the normally expected hypervolemia is usually absent. Women of average size should have a blood volume of nearly 5000ml during the last several weeks of a normal pregnancy compared with about 3500ml when non pregnant.

With eclampsia, however much or all of the anticipated 1500ml of blood normally present in late pregnancy is absent. The virtual absence of an expanded blood volume is likely the consequence of generalised vasoconstriction, is made worse by increased vascular permeability. In women with preeclampsia these differences are not as marked and women with gestational hypertension usually have a normal blood volume .

Vasoconstriction results from relative deficiency of vasodilating prostaglandins (PG). Previous investigators have reported that renal excretion of prostacyclin metabolites (PGI_2) or the production of other eicosonoides by blood vessels or by the placenta is lower in preeclamptic women.[41]

Coagulation and Fibrinolysis in Pregnancy

Placental separation during the third stage of labour represents a major haemostatic challenge to the mother. Although myometrial contraction is of major importance in constricting the blood vessels in the placental bed, adequate fibrin generation is also required .

Physiological adaptations which occurs during pregnancy to help the mother meet this haemostatic challenge, taken together, the change in coagulation and fibrinolysis in pregnancy represents a hypercoagulable state. [1]

The Coagulation System

The concentration of many clotting factors increases during pregnancy.

1. II-prothrombin
2. VII-proconvertin which increases by more than 200% in the 2nd trimester till the 3rd trimester.
3. VIII-antihaemophilic factor increases with a peak and plateau therafter in the 3rd trimester.
4. IX-christmas factor
5. XII-Stuart prower factor increases 200% in the 3rd trimester

Clotting factors that decrease during pregnancy:

1. XI-Plasma thromboplastin antecedent
2. XIII-fibrin stabilizing factor
3. Both of these decrease by 70% in the 3rd trimester.[42]

MATERIALS AND METHODS

Source of data

All the patients clinically diagnosed as pregnancy induced hypertension (PIH) cases who attended OPD & wards of department of obstetrics and gynaecology in BLDE University, Shri B. M. Patil Medical College, Hospital and Research centre, Bijapur from November 2011 to April 2013 were taken for the study.

Method of collection of data.

A cross -sectional study of 105 patients satisfying the inclusion and exclusion criteria, managed by department of obstetrics and gynaecology, BLDE University Shri B.M. Patil Medical College, Hospital & RC Bijapur, Karnataka were studied.

A detailed history of included patients were elicited and a complete general physical examination and systemic review of the patients was undertaken.

The following investigations were carried out for the patients:

1. Complete haemogram
2. Urine sugar,protein& microscopy.
3. Prothrombin time(PT)
4. Activated partial thromboplastin time (aPTT)
5. Random blood sugar
6. Serum uric acid
7. Serum creatinine
8. LFT
9. Fundus examination(when indicated)

Inclusion criteria:

All the patients who attended OPD & admitted in wards of department of obstetrics and gynanecology, BLDE University shri B M Patil Medical College Hospital and Research center with PIH.

Exclusion criteria:

- Known case of hypertension.
- Patients on oral anticoagulant therapy.
- Liver disorders.
- Vitamin k deficiency.
- Disseminated intravascular coagulation.
- Any other major disorders which will alter the coagulation profile other than PIH.

Sample Size:

With incidence rate of hypertensive disorders of pregnancy as 5% to 10%[1] with 95% confidence interval and 20% allowable error, required sample size is calculated using formula:

Statistical formula $n = \frac{(1.96)^2 pxq}{L^2}$

Where n: sample size, L: margin of error.

Hence, a minimum of **105** prospective cases were included in the study

Statistical analysis:

Data will be analysed by using

1) Chi-square test or correlation

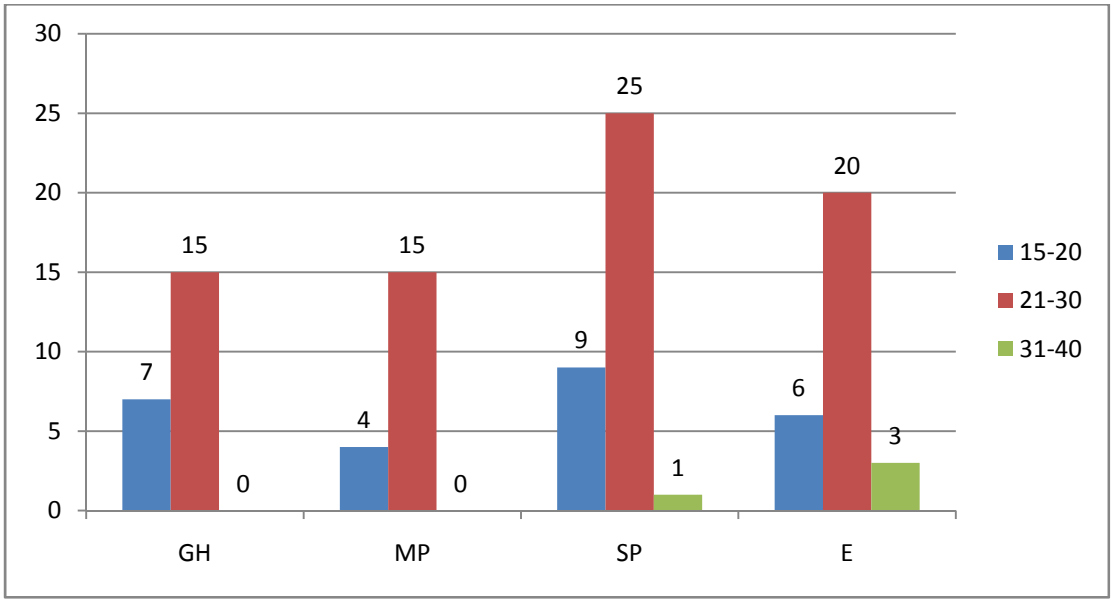
2) Diagrammatic presentation

RESULTS

Table No. 2: Table showing age distribution of PIH cases.

Age (yrs)	GH	Percentage	MP	Percentage	SP	Percentage	E	Percentage
15-20	07	32	04	21	9	26	06	20
21-30	15	68	15	79	25	72	20	69
31-40	00	00	00	00	1	02	03	11
Total	22	100	19	100.0	35	100.0	29	100.0

Figure No.1: Bar graph showing age distribution of PIH cases.



Seven cases (32%) of GH, 04 cases (21%) of MP, 09cases (26%) of SP and 06 cases (20%) of E were in the age group of 15-20yrs.

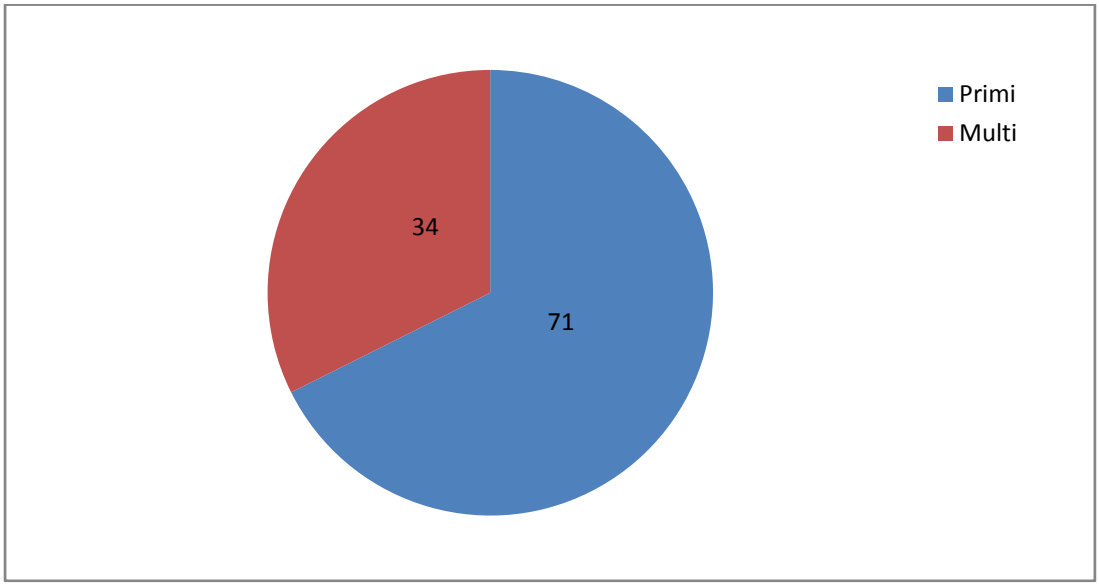
Fifteen cases (68%) of GH, 15 cases (79%) of MP, 25 cases (72%) of SP and 20 cases (69%) of E were in the age group of 21-30yrs. (Table-2)

The youngest patient was18yrs and eldest was 36yrs in PIH.

Table No. 3:Table showing Gravida status of PIH cases

Gravida	No of cases	Percentage
Primigravida	71	67.6
Multigravida	34	32.4
Total	105	100.0

Figure No.2: Pie chart showing Gravida status of PIH cases

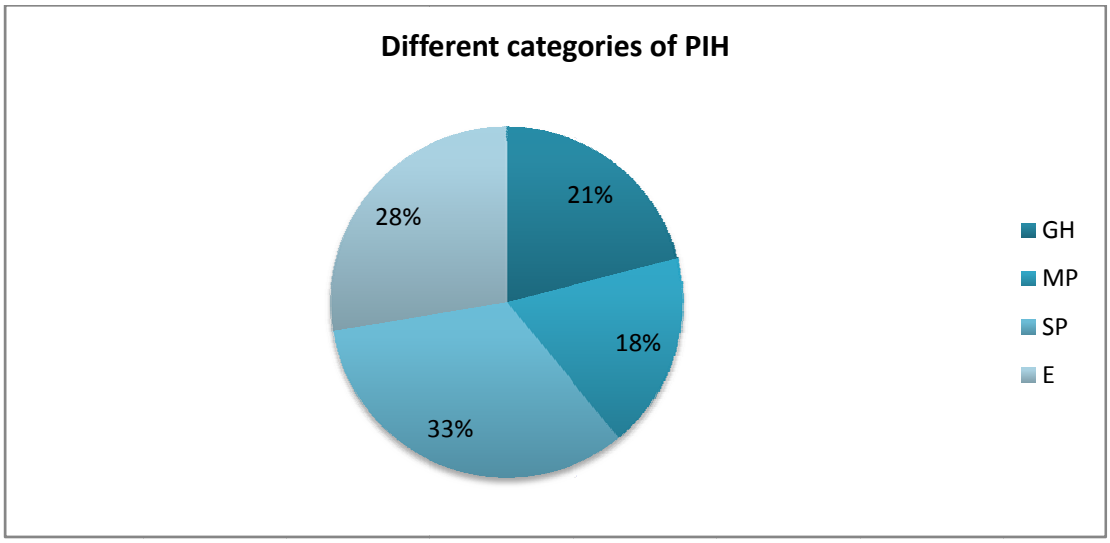


Out of total 105 cases 73 cases (67.6%) were primigravidas and 34 (33.4%) were multigravidas (Table-3)

Table No.4: Table showing different categories of PIH cases.

PIH cases	No of cases	Percentage
Gestational hypertension	**22**	**20.9**
Mild Preeclampsia	**19**	**18.2**
SeverePreeclmpsia	**35**	**33.3**
Eclampsia	**29**	**27.6**
Total	**105**	**100**

Figure No.3: Pie chart showing different categories of PIH cases

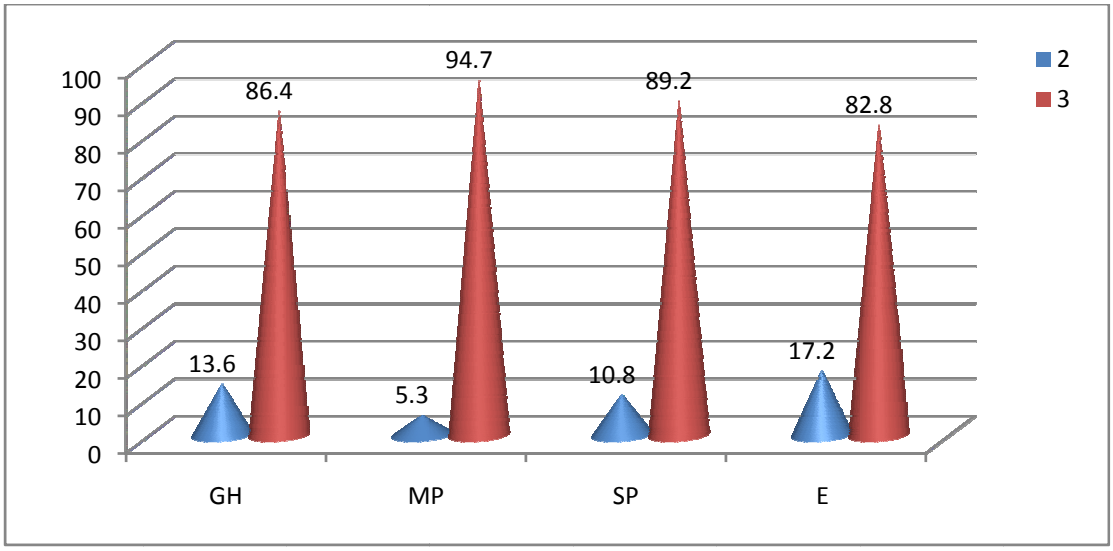


Out of total 105 cases, 22 cases (20.9%) were GH, 19 cases (18.2%) MP, 35 cases (33.3%) SP and 29 cases (27.6%) were E. (Table-4)

Table No.5:Table showing different trimesters in PIH cases

Trimester	GH	Percentage	MP	Percentage	SP	Percentage	E	Percentage
2	03	13.6	1	5.3	4	10.8	5	17.2
3	19	86.4	18	94.7	33	89.2	24	82.8
Total	22	100	19	100.0	35	100.0	29	100.0

Figure No.4: Bar graph showing different trimesters in PIH cases



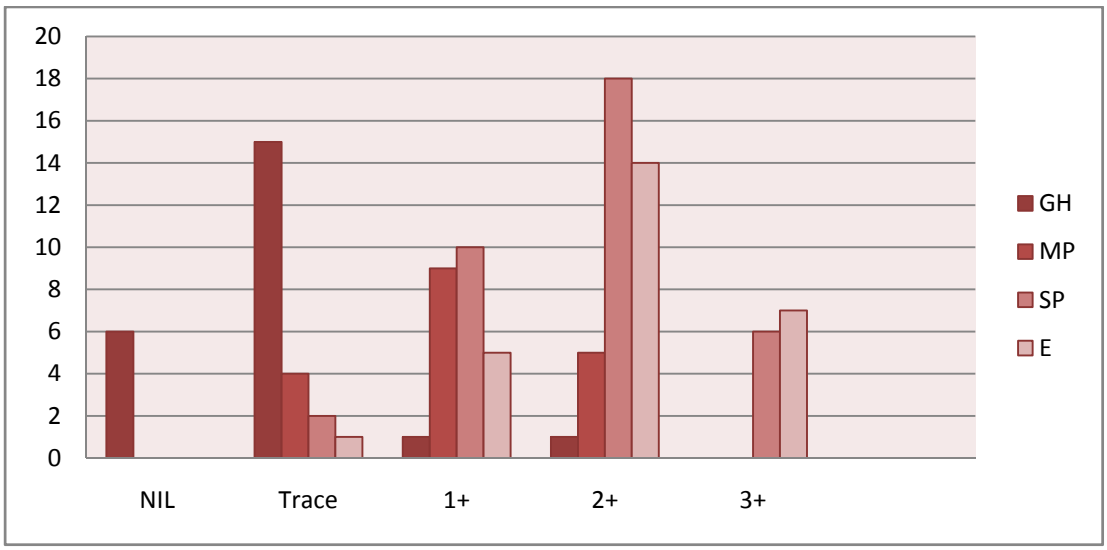
Out of 105 cases, 03 cases (13.6%) of GH,01 cases (5.3%) of MP, 04cases (10.8%) of SP and 05 cases(17.2%) of E were in 2nd trimester of gestational period .

Nineteen cases (86.4%) of GH, 18 cases (94.7%) of MP, 33cases (89.2%) of SP and 24 cases (82.8%) of E were in 3rdtrimester of gestational period.(Table-5)

Table No. 6 : Table showing proteinuria in PIH

Urine Albumin	GH	MP	SP	E
Nil	06	00	00	00
Trace	15	04	02	01
1+	01	09	10	05
2+	01	05	18	14
3+	00	00	06	07
Total	23	18	36	17

Graph No.5 :Bar graph showing proteinuria in PIH



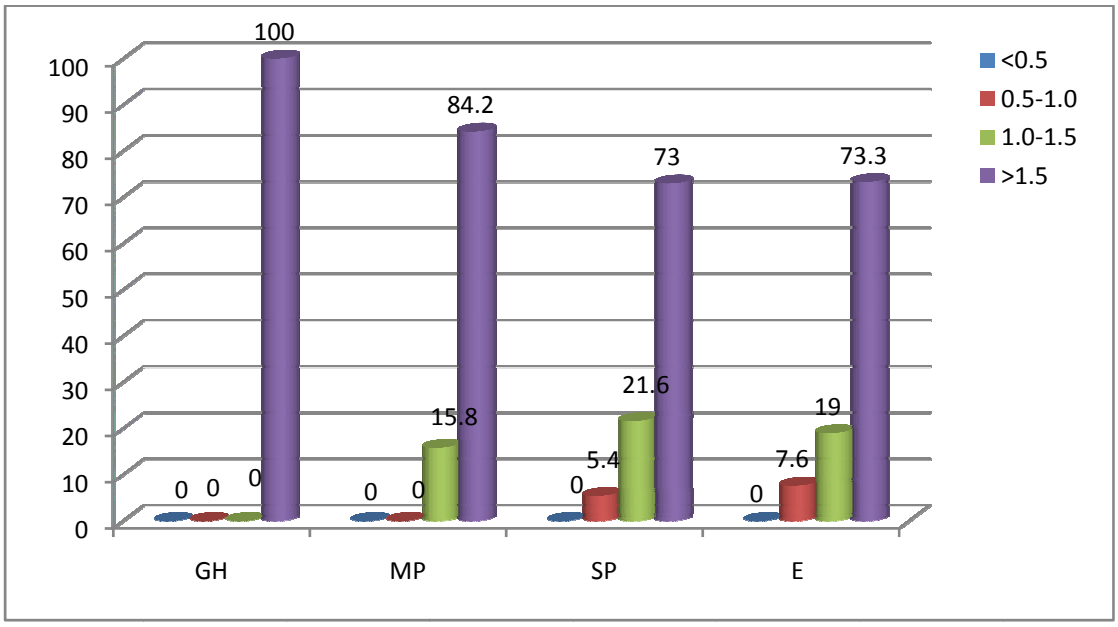
Majority of GH showed traces of urine albumin. MP showed 9 cases of 1+ proteinuria. Eighteen cases of SP showed 2+ proteinuria followed by 1+ of 10 cases.

Eclampsia cases showed 14 cases of 2+ proteinuria followed by 7 cases with 3+ proteinuria.(Table-6)

Table No.7: Table showing platelet count in PIH cases.

Platelet	GH	Percentage	MP	Percentage	SP	Percentage	E	Percentage
<0.5	00	00	00	00	00	00	00	00
0.5-1.0	00	00	00	00	02	5.4	06	7.6
1-1.5	00	00	03	15.8	08	21.6	09	19.0
>1.5	22	100	16	84.2	25	73.0	14	73.3
Total	22	100	19	100.0	35	100.0	29	100.0

Figure No.6: Bar graph showing platelet count in PIH cases



Platelet count was normal (>1.5 lakhs) in all 22 cases (100%) of GH, 16 cases (84.2)of MP, 25 cases (73%) SP and 14cases(73.3%) of E.

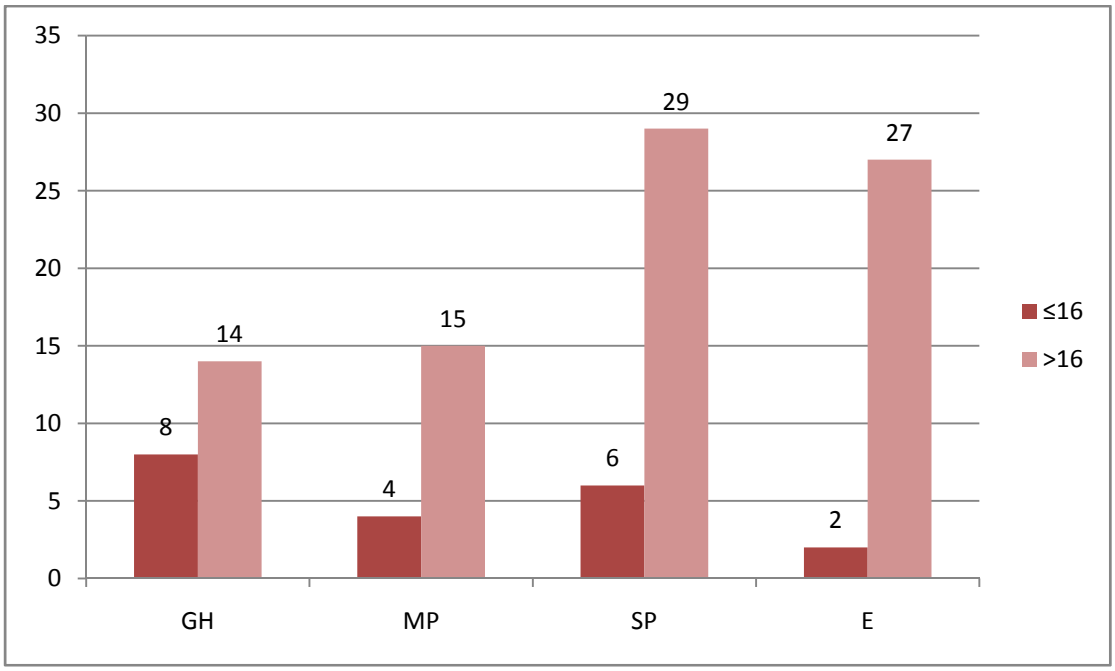
Platelet count between 1-1.5lakhs was seen in 3 cases (15.8) of MP, 8 cases (21.6) SP and 9 cases (19%) of E.

Platelet count between 50,000 -1lakhs was seen in 2 cases (5.4%) of SP and 6 cases (7.6%) of E. (Table-7)

Table No.8: Table showing Prothrombin time in PIH cases

PT	GH	Percentage	MP	Percentage	SP	Percentage	E	Percentage
≤16	8	36.4	4	21.1	6	21.6	2	6.9
>16	14	63.6	15	78.9	29	78.4	27	93.1
Total	22	100.0	19	100.0	35	100.0	29	100.0

Figure No.7: Bar graph showing Prothrombin time in PIH cases



Normal Prothrombin time (≤16secs) was observed in 8 cases (36.4%) of GH, 4cases (21.1%) of MP, 6 cases (21.6%) of SP and 2 cases (6.9%%) of E.

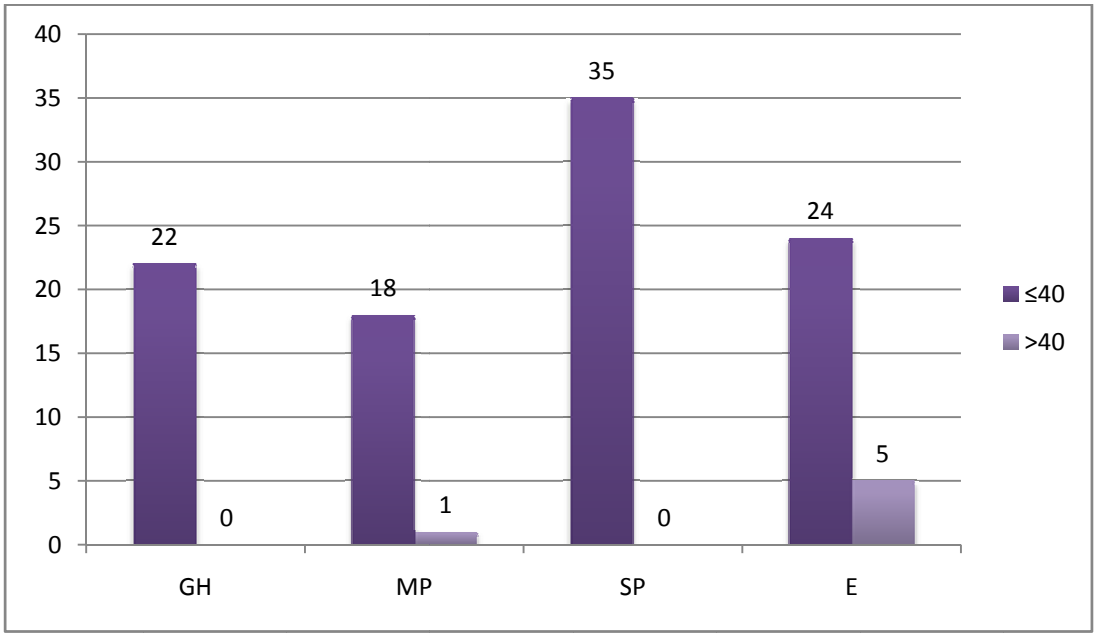
Prolonged Prothombin time (>16secs) was observed in 14 cases(63.6%) of GH,15cases (78.9%) of MP, 29cases(78.4%) of SP and 27cases (93.1%) of E. (Table8)

The prothrombin time in severe PIH was significantly prolonged ($p<0.05$)

Table No.9: Table showing aPTT in PIH cases

aPTT (Secs)	GH	Percentage	MP	Percentage	SP	Percentage	E	Percentage
≤40	22	100	18	94.7	35	100	24	82.8
>40	00	00	01	5.3	00	00	05	17.2
Total	22	100	19	100	35	00	29	100

Figure No.8: Bar graph showing aPTT in PIH cases



Normal aPTT (≤40sec) was observed in all 22 cases (100%) of GH, 18cases (94.7%) of MP, all 37cases(100%) of SP and 24cases (82.8%) of E.

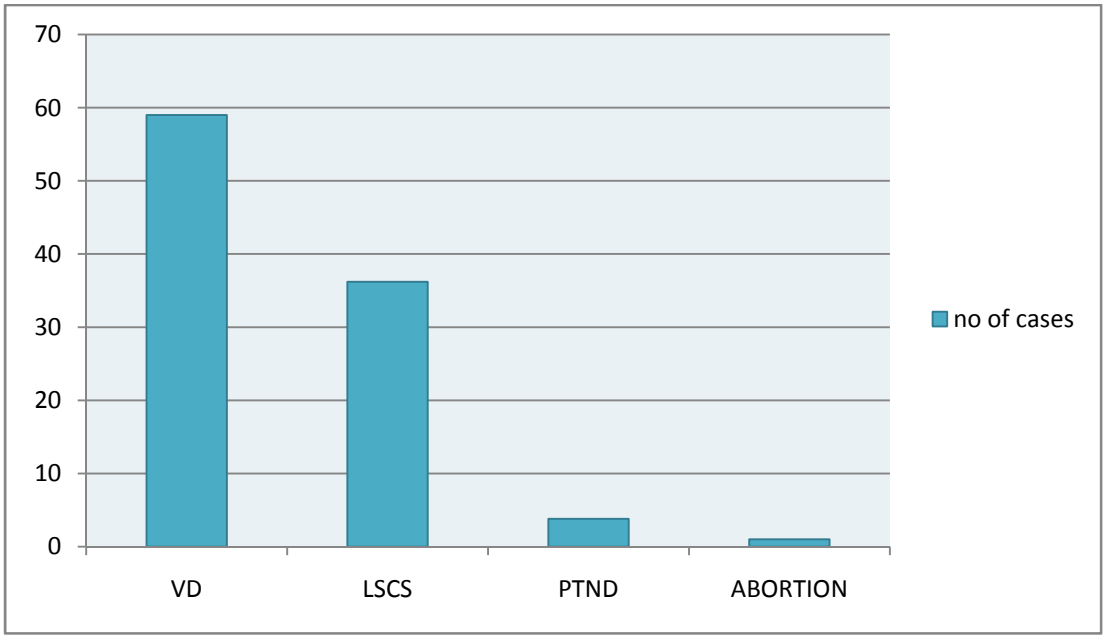
Prolonged aPTT (>40sec) was observed in 1 case (5.3%) of MP and 5 cases (17.2%) of E.

aPTT was not significantly prolonged in PIH(p>0.05).(Table-9)

Table No .10: Table showing MOD in PIH cases.

MOD	No of cases	Percentage
VD	62	59.0
LSCS	38	36.2
PTND	01	3.8
ABORTION	04	1.0
TOTAL	105	100

Graph No.9 : Bar graph showing MOD in PIH cases.



Majority of cases of PIH who underwent LSCS were 38 cases. Of which 14 were E,16 SP and 8 were of MP (Table-10).

DISCUSSION

PIH is a major complication during pregnancy which if left unattended might lead to severe problems for the mother as well as the fetus. Thus it is advisable to check for further complications that arise from PIH to protect the mother and the baby.Hence this study was done to correlate the plaletet count and coagulation parameters like PT and aPTT in different trimesters of pregnancy in PIH.

Platelet count:

Thrombocytopenia occurs in 15% of preeclampsia .A significant reduction in platelet count is seen frequently during and even before the onset of disease. [1]

Table No.11: Table showing comparision of percentage of PIH cases having thrombocytopenia in different studies.

Authors	Percentage
Kelton J.G.et al	34%
Burrows R.F et al	50%
Thomas A.et al	16%
Present study	24%

Present study showed thrombocytopenia in 24% of PIH cases with chi square p value of 0.001indicating statistically highly significant similar to **Burrows R.F et al and Kelton J.G et al**.[3,4]

Our study showed that platelet count was inversely proportional to the severity of PIH. This was similar to study done by **Mohapathra et al**. [10]

In the present study 24% of the patients showed thrombocytopenia thus co-relating with the study by **Ugur Kazimonglu et al**[11] conducted showing 27% patients with thrombocytopenia..

In our study 24% of patients had thrombocytopenia which is slightly lower than that observed by **Jack A Prichard et al**[23] where his study showed thrombocytopenia in 29% patients of PIH.

Coagulation Studies :

The abnormalities of coagulation parameters like PT, aPTT and fibrinogen levels are usually observed in severe pre-eclampsia and eclampsia[17] and even in the presence of normal platelet count.[18,19]

DIC has been suggested as a cause or an important secondary mechanism in toxemia of pregnancy .The reported haematologic findings in toxemia of pregnancy include thrombocytopenia, hemolysis, increased platetet adhesiveness, cryofibrinogenemia, prolonged PT, aPTT, TT and increased fibrin degradation products are indicators compatible with intravascular coagulation.

In the present study an attempt was made to determine if the clinical categories of toxemia of pregnancy could be related to the syndromes of DIC on the basis of plasma assays of PT and aPTT.

Present study had 24% of patients with thrombocytopenia and 6% patients with prolonged aPTT which is similar to the study conducted by **Fitzgerald et al**[18] on

PIH and showed 37% of patients had thrombocytopenia and 10% had prolonged aPTT. However in our study 77% patients had prolonged PT, much higher than that observed by Fitzgerald in his study (16%).

Present study observed that there is significant difference in platelet count in 3rd trimester in PIH at $p<0.001$ and PT was significantly prolonged at $p<0.05$ in 3rd trimester in severe preeclampsia and eclampsia cases. **Jambhulkar et al**[20] stated that, in severe preeclampsia decrease in platelet count was highly significant ($p<0.01$) but PTTK were significantly prolonged ($p<0.05$). In eclampsia, platelet count was highly significantly decreased ($p<0.01$) and PTTK was significantly prolonged ($p<0.05$). This variation is seen cause we have co-related the parameters in different trimesters. Smaller sample size and non-inclusion of control subjects might also be the reasons for variation.

Our study showed 19 out of 105 patients were diagnosed as mild preeclamptic while 35 were severe preeclamptic and 29 had eclampsia. Thrombocytopenia was seen in 32 patients while both prolonged PT and thrombocytopenia were seen in 24 patients. Similar study conducted by **Orlikowski et la**[21] showed that out of 49 cases 7 were diagnosed as mild pre-eclamptic, 33 were severe pre-eclamptic and rest 9 were diagnosed with eclampsia. Abnormal platelet count was seen in 25 out of 49 patients and 2 patients had prolonged PT with thrombocytopenia.

In our study 24% patients have thrombocytopenia which is less compared to the study conducted by Line Leduce et al in which 50% had thrombocytopenia. However, in our study 24% of patients have both thrombocytopenia and prolonged PT which is more than the 13% found in the study of **Line Leduce et al.**[17]

Other observations made in our study was also on

Age :

In the present study 75 cases were in the second and third decades .Similar observation was made by **O' Brein WF et.al**[43] in his study.

Proteinuria :

The severity of proteinuria in preeclampsia has been regarded by some as a predictor of adverse outcomes for the mother .This correlated with our study where the degree of proteinuria was found to increase with severity of preeclampsia and eclampsia.[44]

SUMMARY

This study entitled "**Study of Platelet count, Prothrombin time and activated partial thromboplastin time in Pregnancy induced hypertension** " was conducted on 105 patients attending wards and OPD of department of obstretics and gynaecology of Shri B.M. Patil medical college, Bijapur during the period of Nov 2011 to April 2013, diagnosed with PIH, were taken for the study.

Blood samples were collected and investigations were done which included platelet count, prothrombin time and activated partial thromboplastin time along with the routine investigations (as per the proforma given below).

The patients were aged between 18-36yrs of which 75% were between 21-30yrs and 20% were between 15-20yrs. 67% of the patients were primigravida and 33% were multigravida. Among 105 patients, 92 cases were diagnosed as PIH in their 3rd trimester while 13% were diagnosed in their 2nd trimester.

Diagnosed PIH cases belonged to different groups, 22% were gestational hypertension, 19% were mild preeclampsia, 35 % were severe eclampsia and 29% with eclampsia.

Low platelet count was seen in 24 cases out of 105 patients, among which 5 were diagnosed in their 2nd trimester and 19 were diagnosed in their 3rd trimester. Prolonged PT was seen in 81cases of 105 PIH cases, 8 cases were diagnosed in 2nd trimester and 73cases were diagnosed in 3rd trimester.

Prolonged aPTT was seen in 6 cases of 105 cases, 3 cases were from 2nd trimester and 3 cases from 3rd trimester of pregnancy. Decreased platelet count with prolonged PT and aPTT was observed in 5 out of 105 patients.

The results were tabulated and analysis was done.

CONCLUSION

Pregnancy induced hypertension is a significant cause of maternal and fetal morbidity and mortality during pregnancy. There is no single reliable, cost-effective screening test and there are no well-established measures for primary prevention for the same.

In the present study, thrombocytopenia and prolonged PT was present in severe preeclampsia with majority in primiparas and in 3rd trimester, which can detect severity of PIH to some extent.

However, only the above said parameters cannot be used to determine the correlation between the severity of PIH and derrangements in coagulation system.

Hence, analysis and subsequent correlations of other hemostatic parameters in different trimesters of pregnancy with diagnosed PIH will help in early detection of complications, prognosis and selection of operative procedures in PIH and thus monitoring the health of foetus and mother leading to reduced morbidity and mortality rate.

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ANNEXURES

Proforma for PIH cases

NAME :

AGE :

OP/IP No. :

D.O.A :

D.O.D :

History of present illness:

Past history:

Family history:

General physical examination:

- PR
- BP
- RR
- Pallor / Icterus / LN /Pedal Edema

Systemic examination:

- Per Abdomen
- Cardiovascular system
- Respiratory system:
- CNS

Clinical diagnosis:

Investigations:

Complete hemogram

Urine sugar, protein and microscopy

Prothrombin Time (PT)

Activated Partial Thromboplastin Time (aPTT)

Random blood glucose

Serum uric acid

Serum creatinine

Liver Function Tests (LFT)

Fundus examination

MOD:

ANNEXURE

Following Investigations were analysed by following method

- Platelet count- Blood samples were analysed by automated hematology analyser (Sysmex KX- 21)
- Prothrombin time (PT)-This test is used to measure the extrinsic pathway, factor VII as well as factors in the common pathway.
- Activated partial thromboplastin time(APTT)- This test is used to measure the intrinsic system factors (VIII, IX,XI and XII) as well as factors common to both intrinsic and extrinsic systems(factors X,V, prothrombin and fibrinogen).
- Both PT and aPTT were measured by semi automated method (Trinity Biotech PIc. Model –KC 1 delta Co-agulometer).

The following values are taken as abnormal in following investigations

Platelet count : <1,50000 cells /cmm(Thrombocytopenia)[45]

Prothrombin time : above the reference range (between11 and 16s)[45]

Activated partial thromboplastin time:above the reference range (between 30 and 40s)[45]

ETHICAL CLEARANCE CERTIFICATE

Ethical Committee
BIJAPUR-586103
OUT WARD
No 27/2011
Date 27-10-11
B.L.D.E. Shri B. M. Patil Medical College

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 20-10-2011 *at* 10.30 am *to scrutinize the Synopsis/Research projects of postgraduate/undergraduate student/Faculty members of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis/Research project has been accorded Ethical Clearance.*

Title "Study of platelet count, prothrombin time and activated partial thromboplastin time in pregnancy induced hypertension (PIH)"

Name of P.G./U.G. student/Faculty member Dr. Santhoshi Mata
Dept of Pathology.

Name of Guide/Co-investigator Dr. S. B. Hipparagi, prof of Pathology

DR.M.S.BIRADAR,
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Chairman
Ethical Committee
BLDEA'S Shri. B.M. Patil
Medical College
Bijapur-586103

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

KEY TO MASTER CHART

Am	- Amenorrhoea(in weeks)
GH	- Gestational hypertension
MP	- Mild preeclampsia
SP	- Severe preeclampsia
E	- Eclampsia
Trim	- Trimester
BP	- Blood Pressure
UR(alb)	- Urine routine(albumin)
HB%	- Haemoglobin in gram percentage
PT(T)	- Prothrombin time (Test)
PT(c)	- Prothrombintime(control)
aPTT(T)	- Activated partial thromboplastin time(Test)
aPTT(c)	- Activated partial thromboplastintime(control)
MOD	- Mode of delivery

MASTER CHART

MASTER CHART

SL. No	IPD	AGE	AM	PARITY	GH	MP	SP	E	TRI	BP (SBP)	DBP	UR (alb)	HB	PC	PT (T)	PT©	aPTT (T)	aPTT©	MOD	Baby (wt)	Indication
1	2751/11	28	35	primi			y		3	150	100	1+	10	2.42	24.6	14.6	31.7	26.2	LSCS	2.1	SP
2	27905/11	26	35	primi			y		2	170	140	1+	16	1.89	16.3	14.5	28.6	24	Abortion	2	IUD
3	518/12	22	36	primi		y			3	130	80	1+	10.8	1.69	16.5	13.6	24	22	VD	3.2	
4	580/12	26	31	multi				y	3	160	120	2+	12.5	2.88	18.2	13.5	26.8	25	VD	1.2	
5	609/12	21	36	primi			y		3	170	120	1+	8.7	2.67	19	13.8	31	24	LSCS	2.7	FD
6	2355/12	20	28	primi				y	2	150	90	3+	10.6	2.49	22	15	30	22	VD	1.3	
7	4601/12	28	36	multi			y		3	160	100	2+	8.7	2.45	16	14.2	30	25	VD	2.8	
8	2929/12	20	36	primi			y		3	160	110	3+	11	3.25	29.9	14.8	32.6	26.8	VD	2.7	
9	2924/12	20	37	primi		y			3	150	70	2+	10.5	1.95	26.8	14.8	34	26.8	VD	2.8	
10	3319/12	30	36	multi				y	3	150	110	2+	11.4	2.34	17.7	16	24	22	VD	2.52	
11	4477/12	18	26	primi				y	2	150	110	2+	13.8	1.5	15.3	14.8	33	24	LSCS	2.1	MSL
12	4519/12	21	36	primi			y		3	140	100	2+	9.2	1.2	20	16	32	24	VD	3.15	Anemia
13	4867/12	25	36	primi				y	3	160	90	2+	10.8	80,000	18.5	15	30	20	LSCS	2	HELLP
14	7045/12	22	34	primi			y		3	154	104	2+	10.9	1.7	19	14.3	26	22	Abortion	1.2	IUD
15	6301/12	21	36	multi			y		3	164	112	2+	11.6	1.3	17.5	15.4	26.1	22.2	VD	2.1	IUGR
16	7122/12	26	36	primi			y		3	160	110	2+	11.4	2.1	18	15	26	24	LSCS	2.8	FD
17	6974/12	25	36	primi		y			3	150	104	Trace	11	3.66	17	14.3	26	24	LSCS	3.2	CPD
18	7285/12	23	36	primi				y	3	140	100	3+	15.1	70,000	25.8	14.2	32	29	VD	2.1	
19	7983/12	22	36	primi	y				3	170	110	Absent	12.2	2.2	19	14	24	22	VD	3.4	
20	8436/12	26	33	multi			y		3	152	108	2+	11.2	1.21	15.1	14.2	31	27	LSCS	2.4	IUGR
21	8691/12	22	32	multi			Y		3	160	100	2+	9.5	1.3	15.2	13.9	27.3	25	LSCS	2.6	SP

SL. No	IPD	AGE	AM	PARITY	GH	MP	SP	E	TRI	BP (SBP)	DBP	UR (alb)	HB	PC	PT (T)	PT©	aPTT (T)	aPTT©	MOD	Baby (wt)	Indication
22	9192/12	19	36	primi			y		3	136	100	2+	11.3	2.16	16	14	27	25	LSCS	2.3	SP
23	9297/12	30	36	primi		y			3	144	90	1+	10.7	1.67	16	14	25	22	VD	2.8	
24	9390/12	19	36	primi			y		3	160	120	3+	8.4	2.86	22	15	30	24	VD	1.9	IUGR
25	9905/12	22	32	primi			y		3	160	110	Trace	11.5	2.04	17.9	14	25.7	24.6	Abortion	1.2	IUD(twins)
26	9965/12	20	36	primi	y				3	140	90	Absent	11	1.8	17	15	22	20	VD	2.6	
27	9998/12	23	36	primi				y	3	160	110	3+	5.8	1.16	19	15	28	24	LSCS	1.8	Anemia
28	11386/12	26	36	multi	y				3	140	90	Trace	11	2.06	18.6	13.9	35.2	25.8	VD	3.36	
29	10792/12	23	37	multi		y			3	140	90	Absent	12.5	2.24	17.4	13.9	28.4	27	LSCS	2.4	Oligo
30	147859/12	26	36	multi		y			3	140	70	1+	10.4	1.68	18	15	26	24	VD	2.8	
31	11291/12	24	27	primi			y		2	190	124	3+	8	2.4	50.5	14.6	38	32	LSCS	2.2	SP
32	12067/12	26	38	multi		y			3	146	70	1+	11.8	1.9	16	14	22	20	VD	2.6	
33	16656/12	28	32	multi				y	3	140	96	1+	5.9	1.5	39	14.8	45.4	28.5	VD	2	SA
34	14791/12	24	28	multi			y		2	130	110	2+	13.5	1.14	16.5	13.8	24	22	VD	2.3	
35	183934/12	30	37	multi				y	3	160	110	2+	6	1.13	16.2	13.4	24	22	Abortion	1.3	IUD
36	17586/12	21	36	multi	y				3	118	90	Trace	10.6	2.64	13.1	14	24	28	VD	2.4	
37	17608/12	22	24	primi			y		2	200	180	1+	10	2.74	14.5	13.7	26.4	28.8	VD	2	
38	18990/12	21	41	primi				y	3	200	110	2+	10.8	3.54	13.2	14.2	25.9	24.1	VD	1.9	IUGR
39	184911/12	25	34	primi				y	3	130	90	2+	13.4	3.23	18	14.5	25	26	VD	2.2	
40	18453/12	26	36	primi				y	3	150	100	2+	10.2	2.25	24	16	28	26	VD	2.3	
41	19345/12	22	36	multi			y		3	136	90	2+	14	85000	15.2	13.6	30.7	26.8	FTND	2	
42	19392/12	25	40	primi			y		3	146	100	3+	11.5	3.3	18.2	15.1	28	24.5	FTND	2.5	
43	19441/12	20	36	primi			y		3	150	90	2+	11.5	2.1	14.5	13.1	25.3	24.2	FTND	2.6	

SL. No	IPD	AGE	AM	PARITY	GH	MP	SP	E	TRI	BP (SBP)	DBP	UR (alb)	HB	PC	PT (T)	PT©	aPTT (T)	aPTT©	MOD	Baby (wt)	Indication
44	19501/12	24	28	primi		y			2	160	90	2+	8.5	1	27.2	13.3	42	22	LSCS	1.9	
45	19656/12	22	40	primi				y	3	180	130	3+	8	78,000	48.2	16.5	45	27.5	LSCS	1.8	
46	19731/12	25	38	multi				y	2	170	110	3+	9	97,000	24.5	15.5	40.5	30	LSCS	2.2	
47	19845/12	22	38	primi	y				3	130	100	2+	10.5	2.3	17.2	13.7	24	20	LSCS	2.5	
48	16432/12	25	30	multi				y	2	170	110	3+	9	97000	24.5	20	40.5	31	LSCS	1.8	
49	20203/12	19	37	primi			y		3	140	90	trace	8.4	1.7	20.5	17.6	38.5	25.7	FTND	2.5	
50	16434/12	22	38	primi	y				3	130	100	2+	10.5	2.3	17.2	13.7	24	20	FTND	2.1	
51	10941/13	20	33	primi				y	3	190	110	1+	9.2	2.7	22	15	36	30	VD	1.7	E
52	11106/13	23	36	primi		y			3	140	90	1+	7.3	4.3	20	14	26	22	LSCS	2	oligo
53	11379/13	21	37	primi		y			3	146	90	1+	6	1.2	18	14.5	28	26	FTND	2.1	oligo
54	11476/13	19	38	primi				y	3	200	130	2+	11.9	1.69	20.6	16	38.1	35	LSCS	2.4	
55	11531/13	20	37	primi	y				3	130	90	Trace	11	2.59	17	14	24	22	FTND	3.1	
56	11712/13	26	35	primi	y				3	150	100	trace	9.9	2.09	18	15	24	22	FTND	2.5	
57	11737/13	19	38	primi	y				3	140	90	trace	9.7	2.9	20	14	24	20	FTND	2.75	
58	1418/13	21	38	primi				y	3	150	100	2+	12	2.18	17.5	14.7	28	26.1	LSCS	2.5	
59	1263/13	24	34	multi			y		3	160	100	1+	10	1.4	19.2	15.1	28	25.6	LSCS	2	
60	1997/13	23	35	primi			y		3	140	100	1+	12	4.8	21	15	30	24	LSCS	2.1	
61	2951/13	20	38	primi				y	3	170	110	3+	11.7	2.6	19.5	14.2	28.1	24	LSCS	2.4	
62	4391/13	18	37	multi			y		3	170	110	2+	9.2	4.2	20	13.8	27	23	FTND	2.2	
63	4934/13	20	36	primi		y			3	140	90	trace	12	2.98	22	15	30	26	FTND	2.1	
64	5143/13	18	34	primi			y		3	130	90	1+	9.9	1.7	20	14	29.5	28	LSCS	2.2	FD
65	5299/13	20	26	primi	y				2	140	90	trace	11	2.5	15	13.8	26	28	VD	2.2	

SL. No	IPD	AGE	AM	PARITY	GH	MP	SP	E	TRI	BP (SBP)	DBP	UR (alb)	HB	PC	PT (T)	PT©	aPTT (T)	aPTT©	MOD	Baby (wt)	Indication
66	6685/13	20	38	primi			y		3	180	110	2+	12.1	3.2	17	14.1	32	28.5	FTND	3.1	
67	7407/13	24	37	primi	y				3	140	90	trace	11.2	3.1	15.1	13.7	32	28	LSCS	2.8	FD
68	8071/13	19	30	multi			y		3	200	120	2+	12.7	5.5	14.8	13.1	27	26	FTND	1.8	
69	8184/13	21	36	primi	y				3	130	90	Trace	9.4	2.9	16.7	14.5	30	28	FTND	3.2	
70	8239/13	20	38	multi	y				3	160	120	nil	11.8	2.1	18	15	28	26	FTVD	3.1	
71	8862/13	22	38	primi	y				3	140	170	Trace	12	1.8	16	14.5	30	28	FTND	3.2	
72	8997/13	18	37	primi	y				3	140	180	trace	10	2.9	16	14	28	26	FTND	2.5	
73	9178/13	22	30	multi			y		3	160	100	1+	7.1	1.8	20	15	30	28	FTND	1.6	IUGR
74	9556/13	23	38	primi			y		3	150	110	3+	9.1	1.2	24	15	26	24	LSCS	3	FD
75	10110/13	21	35	primi	y				3	160	110	trace	10.5	3.9	18	14	28	24	FTVD	2	
76	10223/13	26	32	multi				y	3	170	110	2+	12.4	1.79	22	15	34	24	FTVD	1.1	IUGR
77	10330/13	21	37	primi	y				3	140	90	Trace	10.3	1.78	16	14	32	30	FTND	2.1	
78	10343/13	30	38	multi			y		3	160	100	2+	8.2	1.1	16.9	14.8	37.4	26	FTND	2.2	
79	10349/13	27	36	primi			y		3	160	110	2+	11.1	2.63	20	15	36	28	LSCS	2.5	
80	10544/13	22	37	multi		y			3	180	110	2+	13.2	1.3	23	15.2	36	32	FTND	1.1	
81	10978/13	30	33	primi	y				3	140	90	Trace	10.5	2.13	16.1	14.5	30	28	FTND	2.8	
82	12132/13	35	38	multi			y		3	160	100	2+	13.1	2.1	19	13.1	32	26	LSCS	2	
83	12407/13	34	33	multi				y	3	140	100	2+	10.1	1.1	24	15.2	34	23	LSCS	1.8	IUGR
84	12404/13	28	36	multi				y	3	140	90	Trace	14.8	1.5	22	15	32	24	LSCS	2	
85	12466/13	22	36	primi				y	3	130	90	1+	10.5	90,000	24	15.4	36	22	LSCS	2.2	HELLP
86	12808/13	23	37	primi		y			3	140	90	1+	9.5	2.8	20	14.1	28	26	FTND	1.6	IUGR
87	12983/13	27	36	primi		y			3	170	80	1+	10.3	2	18	15	32	26	LSCS	3.3	

SL. No	IPD	AGE	AM	PARITY	GH	MP	SP	E	TRI	BP (SBP)	DBP	UR (alb)	HB	PC	PT (T)	PT©	aPTT (T)	aPTT©	MOD	Baby (wt)	Indication
88	12953/13	20	38	primi		y			3	150	100	1+	11	1.9	19.1	14.1	26	24	FTND	3.2	
89	13595/13	28	28	primi	y				2	150	100	Trace	11.4	3.1	16	14	32	30	FTND	2.9	
90	13645/13	23	33	multi				y	3	140	100	2+	10.5	1.2	22	15	32	24	LSCS	2.5	
91	14244/13	35	34	multi				y	3	144	90	1+	10.2	1.3	22	16	28	24	FTND	1.8	
92	14785/13	25	38	primi		y			3	130	90	2+	11	2	20	15	30	28	FTND	2.2	
93	14894/13	21	36	primi				y	3	160	100	2+	10.2	3.9	20	15	32	28	LSCS	2.7	
94	15278/13	22	36	primi		y			3	150	100	2+	6.8	3.4	15	14.1	28	22	FTND	2.9	
95	15561/13	28	38	multi	y				3	140	90	1+	11.1	2.5	16	13.9	29	26	FTND	2	
96	15584/13	20	32	primi				y	3	140	90	1+	9.7	2.6	22	15	32	28	FTND	2.5	
97	15666/13	23	37	primi			y		3	220	110	3+	10.8	3.2	28	14.1	36	30	LSCS	2.7	
98	16453/13	20	34	primi		y			3	170	100	Trace	9.8	1.7	19	14.1	32	30	LSCS	2.6	
99	16466/13	22	38	primi			y		3	160	110	1+	10.2	1.8	20	14	32	29	LSCS	2.3	
100	16705/13	28	30	primi			y		3	160	100	1+	12.5	1.6	24	15	28	26	LSCS	1.5	Preterm
101	17921/13	22	36	primi			y		3	150	100	2+	8.9	2.2	35.9	13.8	39.1	26.8	FTND	2.7	
102	17271/13	22	34	primi			y		3	160	100	1+	3.7	70,000	23.8	12.8	28.6	25.7	FTVD	2.4	SA
103	17288/13	24	37	multi				y	3	150	120	traces	9.4	1.3	22	15.1	42	30	FTND	2.5	
104	17383/13	26	28	multi	y				2	140	100	trace	9.1	2.6	20	16	32	28	PTND	2.1	
105	18859/13	30	39	multi		y			3	150	100	trace	9.5	1.6	14.2	13.1	26	22	FTND	2.5	