# PREVALENCE AND CHARACTERIZATION OF THROMBOCYTOPENIA IN PREGNANCY

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*In partial fulfillment of the requirements for the degree of*

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**THANJAVUR MEDICAL COLLEGE**

**THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY**

**CHENNAI, TAMILNADU**

**May 2020**

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## CERTIFICATE – II

This is to certify that this dissertation work titled **"PREVALENCE AND CHARACTERIZATION OF THRMBOCYTOPENIA IN PREGNANCY"** of the candidate **DR.DEVANAYAGI . M** with registration Number **221716202** for the award of the degree of in the branch of M.S Obstetrics & Gynaecology. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from Introduction to conclusion pages and result shows 1 percentage of plagiarism in the dissertation.

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# INTRODUCTION

Thrombocytopenia is a common occurrence in pregnancy. It is the second most common hematological finding in pregnancy next to anemia.

Pregnancy is associated with numerous physiological and pathological changes in platelet number and its functions and which can be of clinical concern. Inherited qualitative and quantitative platelet disorders may also manifest during pregnancy with the risk of bleeding.

Thrombocytopenia affects 7-10%[5] of all pregnancies. Most studies report a reduction in platelet count about 10% lower than pre-pregnant values. The normal range of platelets is non-pregnant women is $150\text{-}400\times10^9/l$ [5]. Thrombocytopenia is defined as a drop in platelet count below $150\times10^9/l$. The modern recognition of the condition is mainly attributable to automated complete blood count, which routinely includes platelet count. Most of this decrease occurs during the third trimester and is associated with a shift in the histogram of platelet count distribution. There is a physiological fall in the platelet count with a leftward shift in platelet distribution. The cause for this decrease is multifactorial and is related to hemodilution, increased platelet consumption and increase in platelet aggregation driven by increased level of TXA2. This is supported by the fact that platelet count may also be lower in twin pregnancy when compared to singleton pregnancies. But pregnant woman with thrombocytopenia have lower bleeding complications when compared to non-pregnant

women because of procoagulant state induced by increased levels of fibrinogen, factor VIII, VWF, suppressed fibrinolysis and reduced protein S activity.

Thrombocytopenia may result from a variety of causes, spectrum of benign conditions such as gestational thrombocytopenia to life threatening syndromes such as HELLP syndrome(Hemolysis, Elevated liver enzymes, Low Platelet count)

A finding of thrombocytopenia during pregnancy poses an intriguing problem before the obstetricians. The Gestational thrombocytopenia is most common etiology, which accounts for almost three fourths of all cases Thrombocytopenia complicating hypertensive disorders of pregnancy are responsible for approximately 20% of all cases of thrombocytopenia during pregnancy. The thrombocytopenia in preeclampsia is mild to moderate, but severe thrombocytopenia can occur. Patients with eclampsia are at even greater risk for developing severe thrombocytopenia, and more likely to have HELLP syndrome, which is a subset of preeclampsia. Thrombocytopenia is a key and necessary component of this syndrome. Immune-mediated thrombocytopenia, including idiopathic thrombocytopenia purpura and neonatal alloimmune thrombocytopenia, is responsible for 4.1% of cases. These conditions, however, can cause considerable morbidity and mortality and must be managed closely. Other causes include Rheumatologic Disease (Eg, Systemic Lupus Erythematosus), Disseminated Intravascular Coagulation, Thrombotic Thrombocytopenia Purpura, Acute Fatty Liver of Pregnancy, Antiphospholipid Syndrome, Human Immunodeficiency Virus (HIV) Infection, Dengue ,Vitamin B 12 deficiency And Medications.

Thrombocytopenia results mainly from four processes –deficient platelet production, accelerated destruction, artifactual thrombocytopenia and pooling of platelets. The platelet count is a valuable rapid screening test in assessing acute obstetric hemostatic failure, particularly in helping the attendants together with other assessments to diagnose the presence and severity of disease.

During normal pregnancy, physiological changes in hemostasis include increasing concentrations of many clotting factors, decreasing level of some natural anticoagulants and diminishing fibrinolytic activity. These changes create a hypercoagulability state that is most marked around term and the immediate postpartum period, thereby decreasing bleeding complications that may be associated with delivery. The most important initial factors for hemostasis at delivery, however, are uterine muscle contractions and constriction of the spiral arteries that interrupt blood flow.

All blood clotting factors except XI and XIII increase during pregnancy. The plasma fibrinogen concentration increases from non-pregnant levels of 2.5–4 g/l to 6 g/l in late pregnancy and labour [4] The increase in the concentration of the two components of the factor VIII complex, Factor VIII and von Willebrand factor (VWF) antigen, occur in parallel in the first half of pregnancy, but then diverge because of a two-fold increase in von Willebrand factor antigen. Factor XI concentrations decrease to approximately 60% of the non-pregnant values. Factor XIII concentrations fall to about 50% of the normal non-pregnant value at term. The increase in factors VII and X is highest in mid-pregnancy and remains high in the third trimester. Most blood

coagulation inhibitors are unchanged. Antithrombin and protein C levels are normal during pregnancy. However, the plasma concentration of free protein S decreases markedly during pregnancy and may contribute to the hypercoagulable state. The level of tissue factor pathway inhibitor increases in pregnancy. Plasma fibrinolytic activity is reduced during pregnancy and labour, and returns to normal within 1 h after placental delivery. The diminished fibrinolysis is caused by increased concentrations of plasminogen activator inhibitor-1 derived from endothelial cells and plasminogen activator inhibitor-2 derived from the placenta.

The normal platelet count in non-pregnant women is $150\text{-}400\times10^9$/l. In uncomplicated pregnancies, recent studies shows that the platelet count decreases by an average of 10% during the third trimester as a result of hemodilution or accelerated destruction leading to younger and larger platelets. Incidental thrombocytopenia in pregnancy is usually benign. The mean platelet volume increases, suggesting that a compensated state of progressive platelet destruction occurs during the third trimester. The concentration of plasma ß-thromboglobulin (a specific protein in the α-granules that is secreted during platelet activation) increases in the second and third trimesters of pregnancy. Platelet activation, coagulation and fibrinolytic activity are enhanced during delivery. Significant increase in fibrinogen degradation products occur in 21% of parturients during labour, with 32% showing similar increase in the immediate postpartum period. At 24–72 hrs after delivery, fibrinogen degradation remains elevated in only 10% of women. Platelet count returns to normal 24–72 hours postpartum, and fibrinolytic activity decreases rapidly. During placental separation,

the clotting mechanism is activated and factor VIII activity transiently increases after delivery, shortening the coagulation times.

In the pregnant women, thrombocytopenia is defined as a platelet count of less than $150\times10^9$ /l .Counts of $100–150\times10^9$/l is defined as mild thrombocytopenia, counts of $50–100\times10^9$/l as moderate thrombocytopenia and counts of less than $50\times10^9$/l as severe thrombocytopenia. Thrombocytopenia is caused either by increased platelet destruction or decreased platelet production. In pregnancy, increased platelet destruction may be mediated by immunological mechanisms, abnormal platelet activation, or platelet consumption.

Increased destruction or utilization of platelets during pregnancy occurs in microangiopathies (exposure to abnormal blood vessels) such as thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, Hemolysis, Elevated Liver enzymes, Low Platelets (HELLP) syndrome and pre-eclampsia.

Gestational or incidental thrombocytopenia, the most common cause of thrombocytopenia during pregnancy accounts for 75% of pregnancy-associated thrombocytopenia. The decreased platelet count may be related to haemodilution and/or accelerated platelet turnover with increased platelet production in the bone marrow, and increased trapping or destruction at the placenta. The quantitative decrease in platelet count is balanced by enhanced platelet reactivity.

ITP is disorder that is immunologically mediated platelet destruction and an increase in circulating mega thrombocytes. However antiplatelet antibodies are present

in only 80% of cases. Incidence of ITP is one case of thrombocytopenia per 1000 pregnancies[28] and accounts for 5% of cases of pregnancy-associated thrombocytopenia. It is the most common cause of significant thrombocytopenia in the first trimester. ITP is a diagnosis of exclusion because there are no pathognomonic signs, symptoms or laboratory tests. The four consistent features that are associated with the condition are persistent thrombocytopenia (platelet count $<100\times10^9$/l) with or without peripheral mega thrombocytes; normal or increased number of megakaryocytes detected by bone marrow aspiration; absence of splenomegaly; and exclusion of systemic diseases or drugs that are known to cause thrombocytopenia.

Pre-eclampsia is present in 21% of cases[6] of maternal thrombocytopenia. Thrombocytopenia occurs in 50% of patients with preeclampsia and occasionally precedes other manifestations of the disease. The thrombocytopenia is usually moderate and clinical hemorrhage is uncommon unless the patient develops disseminated intravascular coagulopathy. A decreasing maternal platelet count is considered as an early sign of worsening of preeclampsia and may occur even before other clinical manifestations of the disease are apparent. The pathogenesis of thrombocytopenia in women with severe preeclampsia is unknown, although vascular endothelial damage, impaired prostacyclin production and increased deposition of fibrin within the vascular bed have been suggested. Accelerated platelet destruction, platelet activation, increased platelet volume and increased megakaryocyte production have been observed.

The HELLP (Hemolysis, Elevated Liver enzymes, Low Platelets) syndrome, a component of severe pre-eclampsia , is characterized by microangiopathic anaemia, SGOT>70 units and thrombocytopenia (<$100\times10^9$/l) and is associated with a maternal mortality of about 3.3%. Severe epigastric or right upper quadrant abdominal pain, which need not be associated by proteinuria and hypertension, are common symptoms. It is more common in multiparas, occurs in a slightly younger age group (19 years -25 years), and can manifest in approximately 30% of cases postpartum. It has been suggested that the underlying primary pathological lesion in the HELLP syndrome might be endothelial dysfunction and damage, which leads to platelet aggregation, consumption and eventually thrombocytopenia.

Thrombotic thrombocytopenic purpura is characterised by microangiopathic haemolytic anaemia, thrombocytopenia, central neurological abnormalities, fever and renal dysfunction. In some series, up to 10% of all cases of TTP occurred in pregnant patients, and pregnancy is considered to be a predisposing factor for this disease.

Mild microangiopathic haemolysis and thrombocytopenia are observed in acute fatty liver of pregnancy (AFLP).AFLP is most common in primiparas and affects one of 5000–10,000 pregnancies.

Before carrying out further investigations to determine the cause of thrombocytopenia, factitious platelet counts due to laboratory artefacts must be ruled out by reviewing the peripheral blood film using a citrated blood sample. This artefact, termed as pseudo-thrombocytopenia, is caused by in vitro platelet clumping when

EDTA is used as the anticoagulant, and is due to adhesion of platelets to the periphery of neutrophils in the presence of platelet agglutinins (IgG or IgM).

The onset of these disorders during pregnancy and their clinical manifestations often overlap, making the specific diagnosis difficult. Thrombocytopenia carries a risk for both the mother and her foetus, associated with substantial maternal or neonatal morbidity & mortality. However a specific therapy, if instituted promptly, improves the outcome of affected patients and their offspring.

Thrombocytopenia during pregnancy is an underexplored condition in Indian women. This study PREVALANCE AND CHARACTERIZATION OF THROMBOCYTOPENIA IN PREGNANCY A PROSPECTIVE STUDY' was conducted in the Department Of Obstetrics and Gynaecology, Govt. Raja Mirasudhar Hospital,Thanjavur Medical College,Thanjvur.

## AIM AND OBJCTIVES:

### AIM

The purpose of this study is to determine the various causes, prevalence of thrombocytopenia in pregnancy. Diagnosis , evaluation and management of thrombocytopenia at prompt time decreases the maternal mortality and morbidity. And to evaluate the fetal outcome in mothers with thrombocytopenia.

### OBJCTIVES:

1. To study various cause associated with thrombocytopenia in pregnancy, and to investigate various obstetric risk factors and complications ,outcomes of pregnancy associated with thrombocytopenia
2. To determine the feto-maternal outcome in pregnancies associated with thrombocytopenia

## REVIEW OF LITERATURE

Platelets are small, anuclear blood cells derived from bone marrow megakaryocytes. They circulate at levels of 150-400×$10^9$/l of blood for 7-10 days. Their primary role in hemostasis is the maintenance of vascular integrity, but they are involved in variety of processes including angiogenesis, inflammation and metastasis[1][2].Under physiological conditions platelets circulate in a quiescent state largely due to the antiplatelet properties of the endothelium (Prostaglandin $I_2$, nitric oxide and CD39-ectoADPase pathways)[3] and platelet tissue factor pathway[4]. Upon vascular damage, platelets rapidly adhere to the exposed extracellular matrix proteins, become activated then aggregate to form a hemostatic plug that prevents further blood loss.

The pregnant state must achieve a fine balance between hemorrhage and thrombosis. Platelets play a major role in these processes yet there is a lack of clear knowledge and consensus about platelet function in pregnancy[5]. The platelet has been implicated as a potential mediator in the pathogenesis of utero placental disorders such as hypertensive disorders of pregnancy, IUGR[6].

Platelets were first described by Bizzozero in the 1800s as BLUE PLATTEN when small cells were noted clumped together at the site of vascular injury. He correctly correlated the platelet in the role of hemostasis and thrombosis[7]. Glanzmann described a familial bleeding disorder in 1918[8]. Von Willibrand disease was described in 1920 [9]and Bernaud Soulier syndrome in 1940[10]. At the time there was

an appreciation of an underlying defect in both quantitative and qualitative aspects of platelet function. The description of platelet adhesiveness was reported in 1961 by Hellem et al. With further investigation the same group reported later in 1961 the aggregating ability of adenosine diphosphate (ADP)[11].The first platelet aggregation studies were reported by G. V. R. Born in 1962 and in the same year by O' Brien in the Journal of Clinical Pathology[12][13]. This involved adding a platelet aggregating agent to a suspension of platelets and using light transmission to assess clumping of platelets. The advent of aggregometry transformed research of platelet function. This technique helped to elucidate the underlying platelet defects in many of the primary platelet disorders identified in the earlier part of the century, to identify platelet agonists and antagonists and revealed many of the underlying molecular mechanisms in platelet biology throughout the 1980s[10][14].

During normal pregnancy there are significant changes in the hemostatic system with increase in several coagulation factors, such as fibrinogen, factors VII, VIII, X, XII and von Willebrand factor [15,16,17,18], alongside a reduction in the activity of the fibrinolytic system[16,19]. Early studies into platelet parameters in normal pregnancy observed enhanced platelet destruction accompanied by increased platelet production and activation[20,21]. Indeed, evidence continues to emerge regarding the role of platelet activation in the pathogenesis of abnormal pregnancy such as those complicated with hypertensive disorders, diabetes and preterm labour[22,23,24,25,26]

Normal platelet count during pregnancy is 150,000 to 400,000 per microliter. A platelet count of less than 150000 per microliter is called thrombocytopenia. The

risk for severe bleeding is present when the platelet count is less than 10,000 to 20,000. Mild bleeding occurs when platelet count is less than 50,000. Normal pregnancy is usually thought not to affect platelet count. But the normal range is somewhat lower in pregnancy[28,29,15].

Thrombocytopenia is mostly asymptomatic; however, mild to serious bleeding can occur. It may present as severe internal bleeding leading to a medical emergency or as external bleeding presenting as purpura and petechiae. Thrombocytopenia complicates upto 7-10% of all pregnancies[5]. A finding of thrombocytopenia during pregnancy poses an intriguing problem before the obstetricians. Thrombocytopenia complicating hypertensive disorders of pregnancy are responsible for approximately 20% of all cases of thrombocytopenia during pregnancy.

## HISTORY

Historically thrombocytopenia has been a cause for unnecessary, often invasive additional testing and caesarean deliveries. In 1977, a case of neonatal intracranial hemorrhage was reported (due to perceived trauma) after vaginal delivery of a thrombocytopenic infant to a mother with Immune Thrombocytopenia Pregnancy[30]. This led to the recommendation that women with Immune Thrombocytopenia Pregnancy be delivered by elective caesarean delivery but In late 1980's caesarean deliveries were recommended only for fetuses with known or suspected thrombocytopenia (<50,000/$mm^3$), but fetal platelet counts do not correlate with maternal platelet counts, splenectomy or presence of platelet associated antibodies. The only certain method of determining fetal platelet count would be by direct fetal blood sampling. By assessing platelet count in utero most caesarean deliveries could be avoided because most fetuses of thrombocytopenic mothers have normal platelet counts.

Fetal scalp sampling: This is the first direct fetal blood sampling technique. It requires ruptured membranes and a cervix dilated at least 3cm. Falsely low foetal platelet counts are encountered often. Due to procoagulants in amniotic fluid, foetal platelets start to clump immediately, resulting in falsely low platelet counts. Additionally, the capillary tube into which the fetal blood is drawn is lined with heparin, which can cause platelet clumping and spuriously low count. Clumping observed on the smear from scalp sampling usually indicates a platelet count of at least 20,000/$mm^3$. [31]

Percutaneous umbilical blood sampling (PUBS): comparatively PUBS is more accurate[32], but it is associated with a higher complication rate (2-3%).[33]

## PLATELET STRUCTURAL AND FUNCTIONAL ANATOMY:

### Light microscopy:

On Wright- Giemsa stained blood smear, platelet appear as small, anuclear, ovoid or round cells with a pale greyish blue cytoplasm that contains homogeneously distributed purple- red granule. After platelet aggregation, these dispersed granules become concentrated in the middle of the cell.

### Dimensions:

The volume of circulating platelets from a single individual is heterogeneous and exhibits a normal size distribution. Circulating platelets have a volume of 7.06 ± 4.85fl (femtolitres), a diameter of 3.6 ± 0.7mm, thickness of 0.9 ± 0.3mm. From one individual to another platelet size varies, although abnormally small or large platelets are present only in certain disease states. By scanning electron microscopy, circulating blood platelets appear as flat discs, with smooth contour and spiny filo podia. This also reveals random openings of a channel system, the surface connected canalicular system, which invaginates throughout the platelet and is the conduit by which granule contents exocytose after stimulation. Although the platelet is aneuclear, transmission electron microscopy reveals a cytoplasm packed with a number of different organelles essential to the maintenance of normal haemostasis.

The glycocalyx cell surface of the platelet contains key glycoprotein receptor that mediate events in primary haemostasis. GP Ib is the receptor for von Willebrand factor (VWF) the protein responsible for initial platelet contact (adhesion) with the injured vessel walls. After platelet adhesion and activation, the platelet GP IIb-IIIa complex is expressed and serves as the fibrinogen receptor mediating platelet aggregation.

Platelets contain two types of granules that mediate the platelet phase of haemostasis- Alpha granules that contain protein such as VWF, platelet derived growth factors, platelet factor- 4, and ß-thromboglobulin and delta or dense granules that contain adenine nucleotides (ADP,ATP), calcium and serotonin. ADP release from platelets activates additional platelets, recruiting them into the growing platelet plug. Serotonin acts as a vasoconstrictor at the site of vascular injury and reduce the blood loss.

## PLATELET PHYSIOLOGY

### Platelet adhesion:

Within an intact blood vessel, platelet circulates in the resting discoid shape. Vascular endothelium actively inhibits platelet activation by synthesis and secretion of prostacyclin and nitric oxide. With vascular injury, components of sub endothelium including collagen & microfibrils, promote platelet adhesion by inducing VWF binding to platelet GP lb. VWF is present in the subendothelial matrix as well as in blood plasma and also is secreted by platelets after activation. The initial GP Ib/VWF

mediated platelet contact with subendothelium is reinforced by the platelet GP IIb-IIIa complex binding VWF and other adhesive ligands to firmly attach a layer of platelets to the damaged vascular surface[34].

**Platelet activation & aggregation:**

If an activating stimulus is sufficient (threshold level), platelet activation occurs. This is associated with granule secretion (the release reaction) and stimulation of prostaglandin systhesis. Granule contents are released through canalicular system that connect the interior of the platelet with the external environment. Prostaglandin synthesis is initiated. When phsopholipase $A_2$ generates arachidonic acid from platelet phospholipids, arachidonic acid subsequently is converted by platelet cyclooxygenase to labile endoperoxides ($PGG_2$,$PGH_2$) that are then converted by thromboxane synthetase to $TXA_2$ [35], a potent platelet activator and vasoconstrictor. Platelet activation also leads to expression of the GP IIb-IIIa receptors, fibrinogen binding and platelet aggregation.

## LABORATORY EVALUATION OF PLATELET NUMBER & FUNCTION

In patients suspected of a disorder of haemostasis, defects in platelet number or function, impaired coagulation or abnormalities in vascular function should be considered.[36]

**Platelet count:** This screening test is performed routinely as a part of the complete blood count using automated particle counters. A typical reference range is 1,50,000-4,40,000/mc .

When thrombocytopenia is reported confirmation should be obtained by evaluation of the patients peripheral smear[37]. Approximately 1% of patients have artificial thrombocytopenia due to EDTA- induced platelet clumping and do not have true thrombocytopenia. The platelet count is a valuable rapid screening test in assessing acute obstetric hemostatic failure, particularly in helping the attendants together with other assessments to diagnose the presence and severity of disease.

**Bleeding time:** The length of time a small skin wound continues to bleed depends largely on the number and function of platelets, i.e their ability to form plugs at the site of injury (normal range upto 7-10 minutes)[34]. It is only rapidly performed invivo test of platelet vascular interaction. The bleeding time cannot predict bleeding, blood loss or transfusion requirements.[34]

**Bone marrow evaluation of thrombocytopenia:** If thrombocytopenia is detected and confirmed, a bone marrow evaluation is indicated unless the clinician is aware of an obvious reason for thrombocytopenia (recent chemotherapy, splenomegaly etc). If there is no obvious reason for thrombocytopenia, a bone marrow exam should be performed with a major focus of interest being whether megakaryocyte numbers are normal or increased versus decreased.

**Antiplatelet antibody tests:** Antiplatelet antibody test may be a greater clinical utility in the evaluation of neonatal alloimmune thrombocytopenia. Enzyme linked immunosorbent assays (ELISA) are available to diagnose platelet alloantibodies.[38]

**Thrombopoietin (TPO) assay:** Thrombopoietin is a recently described hormone essential for normal platelet production preliminary studies suggested that measurement of TPO levels might indicate whether thrombocytopenia in a given patients resulted from marrow failure or from platelet destruction. Very low TPO levels are associated with peripheral platelet destruction, including ITP, where as an elevated TPO levels are seen in marrow failure.[39]

If there results are confirmed in additional studies a low TPO level might replace the necessity for bone marrow examination in certain patients with thrombocytopenia.

**COAGULATION MECHANISM:**

There are three simple rapid in vitro tests of the integrity of the coagulation cascade.

1. Activated partial thromboplastin time (APTT) - intrinsic system.

2. Prothrombin time (PT) - extrinsic system.

3. Thrombin time (TT) - final common pathway.

**Activated partial thromboplastin time:** This test is also known as the partial thromboplastin time. It gives a crude assessment of the integrity of the intrinsic coagulation system. The normal range usually lies between 35 and 45 seconds, but all tests must always be compared with a known normal plasma[40]. Prolongation occurs with isolated factor deficiencies of factors VIII, IX and XI and also in the presence of therapeutic heparin levels.

**Prothrombin Time:** This test measure the clotting time of citrated plasma after the addition of an optimal concentration of tissue extract (thromboplastin). It is a measure of the overall efficiency of the extrinsic clotting system. Normal range is approximately 10-14 seconds. It is prolonged in isolated factor VII deficiency and in liver failure, and it is the test used to monitor dosage of warfarin (INR) in those on oral anticoagulants.

**Thrombin time (TT):** This test is a measure of the final common pathway of the extrinsic and intrinsic coagulation systems. Normal plasma will give a thrombin clotting time of 15-19 seconds. The commonest cause of delayed TT is the presence of heparin.

**Fibrinogen estimation:** It is noted that later part of pregnancy i.e. after 30 weeks gestation, the normal non pregnant range of fibrinogen, 2.0-4.5g/l is increased to 4.0-6.0g/l. Reduction of fibrinogen is seen in severe consumptive coagulopathy and in rare inherited deficiencies of fibrinogen.

**Detection of fibrinogen/ fibrin degradation products:** Healthy subjects have FDP concentration of less than 10mg/ml. Concentrations between 10 and 40mg/l are seen in acute inflammatory disorders, in acute venous and arterial thrombosis and after strenuous exercise or major surgery. High levels of FDPs are seen in association with severe acute disseminated intravascular coagulation and following thrombolytic therapy with streptokinase[41].

**Blood coagulation:** The end result of blood coagulation is the formation of an insoluble fibrin clot from the soluble precursor fibrinogen which is in the plasma. This involves a complex interaction of clotting factors and a sequential activation of a series of proenzymes[42]. The original enzyme cascade of the coagulation system proposed by Macfarlane has been modified as a result of the recognition of complexes which form between certain activated factors.

When a blood vessel is injured, blood coagulation is initiated by activation of factor XII by collagen (intrinsic mechanism) and activation of factor VII by thromboplastin release (extrinsic mechanism) from the damaged tissue. Both the intrinsic and extrinsic mechanisms are activated by components of the vessel wall and both are required for normal hemostasis. The two mechanisms are diagrammatically represented.

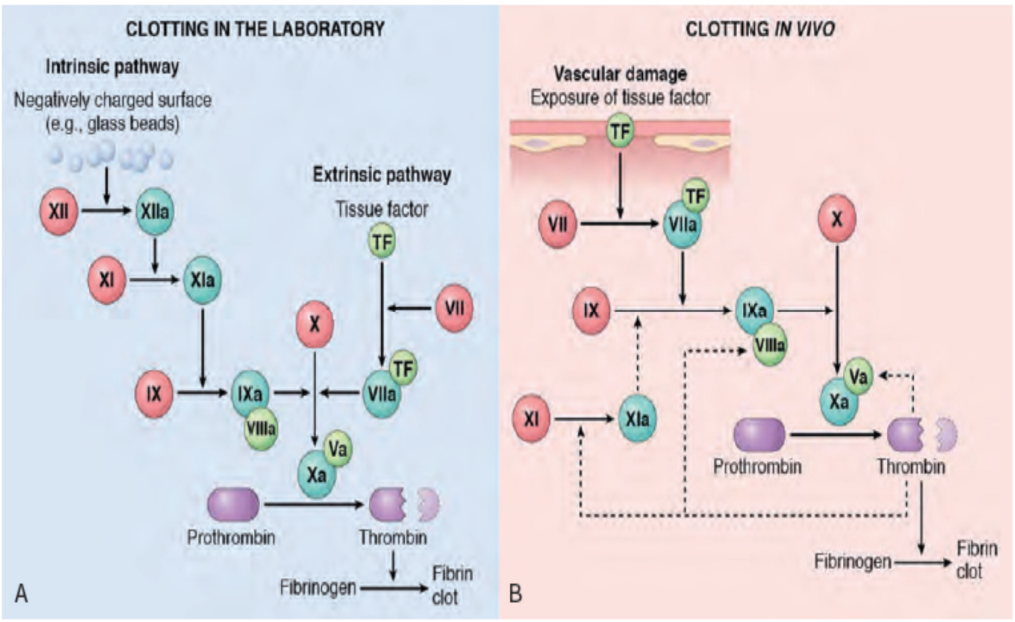



**FIGURE 1: COAGULATION PATHWAY**

## PATHOPHYSIOLOGY OF THROMBOCYTOPENIA

It is the most common cause of abnormal bleeding. The pathophysiology of thrombocytopenia is similar to that of anaemia. Thus, despite the number and diversity of disorders that may be associated etiologically, thrombocytopenia results from only four processes1. Accelerated platelet destruction,2. Deficient platelet production,3. Artifactual thrombocytopenia,4. Abnormal distribution or pooling of the platelets within the body.

**Artifactual thrombocytopenia:** Artifactual thrombocytopenia or falsely low platelet counts occurs ex-vivo, when platelets are not counted accurately. The mechanism should be considered in patients who have thrombocytopenia but no petechiae or ecchymosis. Although inaccurate counting may occur in the presence of giant platelets or with platelet satellitism, the most common cause of artifactual thrombocytopenia is platelet clumping (pseudo thrombocytopenia)[43]. Platelet clumping in pseudo thrombocytopenia appears to be caused by anticoagulant dependent platelet agglutinins that are immunoglobulins (Ig) of IgG, IgA, of IgM subtypes. Although clumping is most commonly seen when blood is collected into EDTA anticoagulant.

**Accelerated Platelet Destruction:** Accelerated platelet destruction is the most common cause of thrombocytopenia. It leads to stimulation of thrombopoiesis and consequently to an increase in the number, size, and rate of maturation of the precursor

megakaryocytes. When the rate of platelet destruction exceeds this compensatory increase in platelet production, thrombocytopenia develops.[44]

Platelet consumption in intravascular thrombi or on damaged endothelial surfaces is another cause of thrombocytopenia. This occurs in diffuse intravascular coagulation and in thrombotic thrombocytopenia purpura and other microangiopathic processes.

**Deficient platelet production:** Deficient platelet production may result from any of number of processes. Those that depopulate the stem cell or megakaryocyte compartments are the most common, such as marrow injury by myelosuppressive drugs or irradiation and aplastic anemia[45]. Deficient platelet production also may be the consequence of disordered proliferation within a precursor compartment of normal or even increased size.

**Abnormal pooling:** Abnormal pooling or abnormal in vivo distribution of an essentially normal total platelet mass may produce thrombocytopenia. This type of thrombocytopenia is seen in the various disorders associated with splenomegaly, in which platelet production is normal or even increased, but most of the platelets are sequestered in the vastly enlarged extravascular splenic pool. Thrombocytopenia may also be caused by dilution of platelets when patients are massively transfused during blood loss.

## PLATELETS IN PREGNANCY INDUCED HYPERTENSION:

Platelets play a crucial role in the pathophysiology of pre-eclampsia by promoting vascular damage and obstruction, leading to tissue ischaemia and further damage[46]. Thromboxane A2 the major product of arachidonic acid metabolism in platelets, is a potent vasoconstrictor and platelet- aggregating agent. As it has a short half-life it is normally measured as its stable hydration product, thromboxane $B_2$. The effects of thromboxane $A_2$ are normally counterbalanced by prostacyclin, a potent vasodilator and anti-platelet prostanoid which is the major product of arachidonic acid metabolism in vascular endothelium and which plays an important role in protecting the endothelium and limiting damage by inhibiting platelet aggregation and promoting vasodilation. These two substances function as local hormones and are thought to be important in the control of the platelet - endothelium interaction. They oppose each other through the regulation of platelet adenylate cyclase, which controls cAMP production and thereby platelet free calcium concentration; this links receptor occupancy with cellular response. Pro -aggregatory substances such as thromboxane $A_2$ inhibit adenylate cyclase , allowing free intracellular calcium to rise, while prostacyclin stimulates adenylate cyclase thus increasing cAMP, reducing free intracellular calcium and inhibiting platelet activation.There is considerable evidence implicating platelets in the pathophysiology of pre-eclampsia. The circulating platelet count is reduced, reflecting a reduced platelet life- span, and an inverse relationship between platelet count and fibrinogen and fibrin degradation products has been noted, suggesting that the reduction in platelet count is due to

increased platelet consumption associated with low- grade DIC. The platelet - specific protein ß- thromboglobulin , marker of platelet activation in vivo, has been found to be increased in pregnancy induced hypertension. This correlates with proteinuria and serum creatinine, and suggests a link between platelet activation with renal microvascular damage.

The platelet content of 5-hydroxytryptamine (5-HT) is reduced in pre-eclampsia, indicating platelet aggregation and stimulation of the platelet release reaction in vivo. Low platelet 5-HT levels have also been associated with loss of platelet responsiveness to various aggregating agents in vitro.

Increased platelet thromboxane $A_2$ production in vivo has been shown to occur in pre-eclampsia complicated by intrauterine growth retardation[47]. Londen et al found reduced platelet reactivity in whole blood in women with pre-eclampsia compared to normal controls, although there was no difference in thromboxane A2 production in vivo. The report of Londen et al would be in keeping differences in patient severity, as platelet reactivity may vary according to the stage of the disease process, with increased reactivity perhaps occurring in the early stages of the disease and platelet exhaustion in advanced disease[39].

The damage in the coagulation system and in platelet function support the concept that disseminated intravascular coagulation occurs in patients with pregnancy induced hypertension. A ‘coagulation index’ of serum fibrin-fibrinogen degradation

product, platelet count and plasma factor VIII has been shown to correlate with a 'clinical index' of disease severity, highlighting the association of the two conditions.

## CLASSIFICATION OF THROMBOCYTOPENIA

**TABLE 1 :ETIOLOGIC CLASSIFICATION:** The etiologic classification for thrombocytopenia can be divided into 3 categories-

1. Increased Sequestration:
2. Decreased production
3. Increased destruction or utilization:

| **Sequestration:** | **Decreased production:** | **Increased destruction or utilization:** |
|---|---|---|
| • Observed with splenic congestion (e.g. Cirrhosis) | • Leukaemia<br>• aplastic anaemia<br>• folate deficiency<br>• medications<br>• viral infections | • Immunologic – Immune Thrombocytopenic Purpura (ITP), Systemic Lupus Erythematosus (SLE)<br>• Microangiopathies (exposure to abnormal blood vessels).<br>• Haemolysis, Elevated liver enzymes, Low platelets (HELLP syndrome)<br>• Thrombotic thrombocytopenic purpura (TTP)<br>• Haemolytic Uremic Syndrome (HUS)<br>• Gestational thrombocytopenia (GT) |

**TABLE 2:CLINICAL CLASSIFICATION**

| Pregnancy specific | Non pregnancy specific |
|---|---|
| 1.Gestational (incidental) ,<br>2.Severe Preeclampsia,<br>3.HELLP syndrome ,<br>4.Acute fatty liver of pregnancy | ✓ Immune thrombocytopenic purpura<br>✓ Thrombotic thrombocytopenic purpura<br>✓ Haemolytic Uremic Syndrome<br>✓ Thrombotic microangiopathies<br>✓ Systemic lupus erythematosus<br>✓ Viral infection (HIV,CMV,EBV)<br>✓ Antiphospholipid antibodies.<br>✓ Disseminated intravascular coagulation(DIC)<br>✓ Bone marrow dysfunction<br>✓ Nutritional deficiencies<br>✓ Drug- induced thrombocytopenia<br>✓ Type IIb von Willibrand disease.<br>✓ Hypersplenism |

## GESTATIONAL THROMBOCYTOPENIA:

This condition has also been termed benign or incidental thrombocytopenia. There is no history of autoimmune disease and the other possible causes of thrombocytopenia such as pre eclampsia have been excluded. Accelerated platelet activation and consumption seen in normal pregnancy may be the cause for thrombocytopenia.[21,32] Typically, the condition is mild with a platelet count of 120-150 x $10^9$/l, although moderate (50-120 x $10^9$/1) and severe (<50 x $10^9$/1) thrombocytopenia have been described[48,49]. Many studies have documented the normal fetal and maternal outcome in such cases.

In the largest study involving over 15,000 women, Burrows and Kelton[50] noted the incidence of thrombocytopenia to be 4.8% having excluded autoimmune disease and preeclampsia; there was no neonatal morbidity. In another large study of 730 women, the incidence of thrombocytopenia was 3.6 % and no neonatal morbidity was noted and none of the 26 infants had a platelet count of less than 100x $10^3$/l. Although there appears to be no significant fetal morbidity, fetal and/or neonatal thrombocytopenia may occur in 4-13% of these cases. The mechanism of neonatal thrombocytopenia has not been determined and it has recently been suggested that in nearly 50% of such cases there may be an underlying maternal autoimmune condition. Clearly, the greater the intensity of investigation for possible autoimmune disease the greater the apparent detection rate. The benefits of thorough investigation in reaching a firm diagnosis and its implications for the current and future pregnancies must be weighed against the expense and whether the knowledge of this information confers

benefit on the patient. Sometimes, it may not be possible to distinguish gestational and autoimmune thrombocytopenia. The diagnosis of gestational thrombocytopenia relies on the exclusion of the other possible causes, and is helped by the knowledge that a platelet count was normal either before, or in the early part of a pregnancy. If this is the case, then prognosis appears to be extremely good and the platelet counts tend to return to a normal value following delivery. Aggressive medical or obstetric intervention should be avoided. Delivery should be attempted vaginally unless there are other obstetric indications for operative delivery. Provided the platelet count is >80 x $10^3$/l most anaesthetists will be happy to offer epidural anaesthesia. Postnatally, foetal cord blood should be sampled at the time of delivery and serial maternal counts undertaken to ensure that the maternal count returns to a normal value.

**HELLP SYNDROME**

TABLE 3: HELLP syndrome is diagnosed with the following laboratory abnormalities.

| **Haemolysis:** | **Elevated liver enzymes:** | **Low platelet count:** |
|---|---|---|
| ✓ Abnormal peripheral smear<br>✓ Totalbilirubin >1.2mg/dl<br>✓ Lactic dehydrogenase > 600U/L | ✓ Aspartate aminotransferase>70U/L<br>✓ Lactate dehydrogenase > 600 U/L | Platelets <100,000/$mm^3$ |

The finding of an abnormal peripheral smear is a sensitive although nonspecific test for HELLP syndrome. Lactic dehydrogenase and bilirubin also are included in the definition as additional indicators of the severity of hemolysis. Typically those patients with elevated lactic dehydrogenase levels tend to have abnormal peripheral smears. The appearance of injured red blood cells includes echinocytes (speculated red cells named from sea urchins), schizocytes (fragmented helmet cells resembling cut erythrocytes) and spherostomatocytes (spherical cells).

Martin et al at the University of Mississippi have further categorized HELLP syndrome into different classes based on the severity of the thrombocytopenia.

Class 1 -Platelets $\leq 50{,}000/mm^3$

Class 2 -Platelets $>50{,}000/mm^3$ to $\leq 100{,}000/mm^3$

Class 3 -Platelets $> 100{,}000/mm^3$

**AETIOLOGY AND PATHOPHYSIOLOGY OF HELLP SYNDROME:**

As with other microangiopathies a major component of the underlying pathology of HELLP syndrome involves endothelial injury. Vascular endothelium serves as an active barrier and contributes to vessel tone. Intimal injury stimulates fibrin deposition in the vessel lumen → subsequent disruption of the formed elements flowing in contact with the area of injury→Activate platelets release vasoconstrictive substances→ Platelet aggregation ca.uses further endothelial injury→impairing prostacyclin production.

HELLP syndrome can occur at any time during the second or third trimesters, but they are typically preterm. Fifteen per cent of HELLP syndrome occurs in the midtrimester: 8% between 17 and 20 weeks and 7% between 20 and 26 weeks gestation. Eighteen per cent of patients will have symptoms at term. Delivery is not uniformly curative because up to 30% of patients develop manifestations within 2 days after delivery (range, a few hours to 6 days postpartum). Seventy nine per cent of those patients developing HELLP syndrome in the postpartum period have an antepartum diagnosis of preeclampsia. About 15% of patients have neither high BP or proteinuria[51].

**Laboratory findings:** Platelet nadirs and transaminase elevations commonly occur after delivery. It has been found that despite abdominal symptoms, liver function readings may be normal initially, only to become elevated postpartum. In George et al's series of 158 patients[38], 13% experienced a nadir in platelet count on admission, 29% at the time of delivery, 30% on postpartum day one and 21% on postpartum day two. Both class 1 and Class 2 HELLP syndrome patients experienced a similar pattern of decline (43% decrease from baseline). Lactate dehydrogenase levels peaked on postpartum day 1 for class 2 patients and a day later in those patients with class 1 HELLP syndrome. There was an inverse relation between the increase in platelet count and the decrease in lactate dehydrogenase levels. A return to normal readings required a longer period of time for class 1 patients. Levels of the predominant elevated transaminase, the aspartate aminotransferase, peaked around the time of delivery and returned to baseline earlier than did the lactate dehydrogenase.

## THROMBOCYTOPENIA IN SEVERE PRE ECLAMPSIA:

The second most common cause of thrombocytopenia in pregnancy is thrombocytopenia complicating hypertension. Thrombocytopenia in the hypertensive disorders of pregnancy is responsible for 21% of thrombocytopenic mothers, which represents an incidence of 13-15 per 1000 live births (approximately 1-2% of all pregnancies). Thrombocytopenia by itself has long been associated with preeclampsia, with approximately 50% of patients manifesting thrombocytopenia. Thrombocytopenia can precede the hypertensive changes, suggesting that the platelet destruction is pathologically related to the hypertensive disorder[52,53]. As a result, ASA has been used to prevent preeclampsia[54]. Typically, the thrombocytopenia in a preeclamptic patient is mild to moderate, with platelet counts in the range 50,000-100,000/$mm^3$. The diagnosis of preeclampsia is usually clinically apparent, with the ultimate treatment being delivery. Typically, the platelet count returns to normal within days after delivery.

## ACUTE FATTY LIVER OF PREGNANCY (AFLP):

AFLP affects one of every 5000-10,000 pregnancies, and is most common in prim paras during the third trimester. . A clue to the pathogenesis of AFLP is provided by the observation that in some series, a foetal deficiency of long-chain 3-hydroxy-acyl CoA dehydrogenase (LCHAD) or other enzymes involved in mitochondrial fatty acid oxidation was commonly present[55]. A mutation of glutamic acid to glutamine at position 478 of LCHAD appears to be of particular importance in disease

development. However, comparison of the frequency of this mutation in the population with the incidence of AFLP suggests that it accounts for only a subset of all AFLP cases. Women with AFLP present with malaise, nausea, epigastric and right upper quadrant pain, dyspnoea, mental status changes and cholestatic liver abnormalities. Diabetes Insipidus may also occur, and hypoglycaemia is common and often severe. Levels of fibrinogen and antithrombin are severely depressed, and 75% of patients manifest a prolonged PT accompanied by laboratory evidence of disseminated intravascular coagulation, perhaps related to decreased hepatic synthesis of antithrombin. Up to 50% of patients with AFLP may also meet criteria for preeclampsia. The extent of microangiopathic haemolysis and thrombocytopenia is generally compared to that observed in HELLP, TTP or HUS. Management of patients with AFLP should be supportive, focusing on correction of hypoglycemia and electrolyte imbalances, as well as the underlying coagulopathy. Up to 10 days after delivery may be required for normalization of hemostatic abnormalities. Fetal mortality in this disorder approaches 15% though maternal mortality occurs in less than 5% of cases.[56]

**Idiopathic Thrombocytopenic Purpura (ITP):**

Idiopathic Thrombocytopenia Purpura (ITP) is the most common immunologically mediated thrombocytopenic condition during pregnancy[57].Idiopathic Thrombocytopenic Purpura often is referred to as Autoimmune Thrombocytopenic Purpura (ATP) and in adults is seen more frequently in women than in men, with a ratio of approximately 3:1. It presents in the second to

third decade of life with the clinical symptoms of easy bruising, bleeding, petechiae, ecchymosis, and/or menorrhagia. These symptoms are the result of a decrease in the number of platelets, but the platelets that remain in circulation are normal in function.

The clinical diagnosis is based on four parameters:

1) A platelet count persistently less than 100,000/ $mm^3$.

2) A bone marrow biopsy that shows increased number and size of megakaryocytes.

3) Exclusion of other conditions that may cause thrombocytopenia, including use of drugs associated with low platelet counts and

4) Absence of splenomegaly

The prothrombin time and activated partial thromboplastin time are normal. The bleeding time may be normal or increased depending on the platelet count. The presence of antiplatelet antibodies is not required for a diagnosis of ATP, but it is confirmatory. Autoantibodies that react with the major platelet antigens are present in approximately 80% of patients with ATP. The most common targets are the platelet membrane glycoproteins IIb/IIIa and Ib. Other sites for antibody targeting include platelet antigens Ib/V/IX, glycoprotein Ia/IIA, and glycoprotein IV.

**Antiplatelet antibody Assays:** There are many mechanisms by which antiplatelet antibodies can be detected; however, because of the complexity of platelet membranes, the precise identification and measurement of such antibodies has been difficult. There are three types of assays that are used to measure platelet –associated antibodies.

Variations of these assays are based on methodology and on the chronologic time at which each test was developed and clinically introduced. Each assay has its own sensitivity and specificity, advantages and pitfalls. Antibody testing, though confirmatory in the diagnosis of ATP, is not required for diagnosis or management. Indeed, only approximately 75% of patients with ATP have some form of detectable antiplatelet antibodies. The management of ATP depends on the patient's age, severity of disease, and the autoimmune nature of the disease. Patients who are discovered to have a mild thrombocytopenia should be followed expectantly. Platelet counts greater than 50,000/$mm^3$ are rarely associated with clinically significant bleeding. Women with ITP may have intrapartum bleeding when platelet count is < 30,000/$mm^3$.The cornerstone[57] of therapy, when indicated, is glucocorticoid administration, most commonly intravenous methyl prednisone in the initial dose of 1mg/kg body weight. Platelet counts generally increase to more than 50,000/$mm^3$ with glucocorticoid therapy within the first week. Most patients will have a relapse after discontinuation of therapy, especially if the taper is too rapid. Therefore, most patients are continued on oral steroids after discontinuation of intravenous therapy. The management of ATP in pregnancy poses a challenge because of the theoretical risk of bleeding not only in the mother, but in the fetus as well. Most obstetricians today manage ATP expectantly, treating the mother only if the platelet count decreases to less than 50,000/$mm^3$ or if spontaneous bleeding develops. The autoantibodies in ATP are of the IgG class, and this class of antibody crosses the placenta readily[58]. Therefore, the fetus may be severely affected despite a relatively mild maternal thrombocytopenia. The range of profound fetal thrombocytopenia[58,59] (platelet counts of less than 50,000/$mm^3$) is

from 4-15%. Noninvasive predictors of the degree of neonatal thrombocytopenia are notoriously inaccurate. Therefore, one must rely on invasive measurement of fetal platelet count before delivery. Under these circumstances, vaginal delivery is probably safe. When platelet count is <20,000/mm$^3$ then caesarean section is advised[59]. The cornerstone of therapy for ATP in pregnancy is glucocorticoids. Prednisone is generally well tolerated during pregnancy, although sleeplessness, acne, striae, and increased appetite are common side effects. Occasionally, glucose intolerance occurs, and serum glucose measurements should be taken after 24-26 weeks gestation and again in the third trimester. Treatment with prednisolone generally is initiated once platelets counts decrease to less than 50,000/mm$^3$ although often avoided during pregnancy because of untoward fetal effects. Splenectomy has been safely accomplished during pregnancy. If required, splenectomy should preferably be performed in the second trimester, as a surgery earlier in the pregnancy carries a high risk of abortion, and later in pregnancy.Splenectomy is successful in increasing platelet counts in 75% of cases. Intravenous immune globulin (IVIG) has been used in pregnancy but is less well studied for this disorder. This therapy has been shown to be safe and is very effective in most patients with ATP[63].

## OTHER CAUSES OF PREGNANCY–ASSOCIATED THROMBOCYTOPENIA

Approximately 25% of patients with systemic lupus erythematosus (SLE) develop thrombocytopenia secondary to platelet destruction due to antiplatelet antibodies, circulating immune complexes or other causes[64]. Antiphospholipid

antibodies (APLA) may be associated with preeclampsia in addition to thrombosis and recurrent fetal loss, and have been described in patients with syndromes resembling HELLP, HUS, or TTP which may not respond as well as expected to standard management approaches. Drug-induced thrombocytopenia occurs in the pregnant as well as the non-pregnant setting an updated list of offending drugs is maintained[66]. Surreptitious cocaine use has been associated with the development of a HELLP- like syndrome in pregnant women. Disseminated intravascular coagulation may complicate several obstetrical disorders, including preeclampsia, placental abruption, amniotic fluid embolism, uterine rupture, and retention of a dead foetus, and may result in thrombocytopenia. Finally, congenital platelet disorders, such as the Mayhegglin anomaly and other macrothrombocytopenias may be initially recognized during pregnancy; many of these may be diagnosed by careful examination of the peripheral blood film. Pseudo thrombocytopenia, an in vitro artefact attributable to platelet clumping caused by EDTA-dependent antiplatelet antibodies, may be transferred from mother to foetus following trans placental passage of the offending antibody. Finally, pregnancy-included increase in levels of an abnormal vWF molecule that binds to platelets with increased affinity and enhances their clearance accounts for the thrombocytopenia that may develop in pregnant women with underlying type IIb vWD. [67]

**THROMBOCYTOPENIA ASSOCIATED WITH INFECTION:**

As in the non-pregnant setting, HIV infection should be considered in any thrombocytopenic patient with risk factors. Human immunodeficiency virus positive

women may manifest thrombocytopenia similar to women with immune thrombocytopenia, increasing the risk of fetal thrombocytopenia and hemorrhages[68]. In a retrospective study of 890 HIV positive pregnant women in France, 25(2.8%) were thought to be thrombocytopenic as a result of their infection; 16 (64%) of these women were treated with zidovudine, corticosteroids or intravenous gamma globulins. Only 1 infant was noted to be thrombocytopenic at birth and sadly went on to develop early onset acquired immunodeficiency syndrome. Caesarean section with appropriate antiviral drug regimens given immediately prior to delivery is the current recommended mode of delivery of HIV positive women, irrespective of the foetal platelet count. Other viral infection being Dengue fever, Early detection and access to proper medical care reduces fatality from 20% to below 1 %. The clinical manifestations, treatment and outcome of dengue in pregnant women are similar to those of nonpregnant women. Misdiagnosis or delayed diagnosis are due to some of the overlapping clinical and/or laboratory features with the better recognized conditions of pregnancy. Ex HELLP syndrome, pneumonia, pulmonary embolism, various obstetric causes of per-vaginal bleeding and other infectious diseases.

**THROMBOTIC TROMBOCYTOPENIC PURPURA:**

This is caused by severely reduced activity of VWf and factor cleavage protein ADAMTS13. The findings are seen in acquired TTP: Median platelet count 10,000/microL,median hematocrit -21%,gastro intestinal symptoms in 45%,weakness ,major neurologic findings (coma,stroke,seizures,TIA),bleeding purpura.

Thrombocytopenia occurs in TTP as a result from deposition of platelets in microthrombi, severe thrombocytopenia is more common.ADAMST assay comnfirms the diagnosis .several scoring systems are available for the diagnosis of TTP namely PLASMIC score.treated by plasmapheresis and monoclonal antibodies such as capacizumab and eculizumab (anti C3 antibodies).

**DISSEMINATED INTRAVASCULAR COAGULATION (DIC):**

The most common hemostatic defect in obstetric disorders is thrombocytopenia, occurring in 17% of eclamptic patients[(69)]. DIC appears to be uncommon in this group of obstetric patients. Abnormal vascular endothelial cell function appears to be an important factor in preeclampsia. Primary therapy is based on rapid delivery of the fetus and placenta, although severely thrombocytopenic patients may require platelet transfusion.[(70)]

**Pathophysiology:** The pathophysiology of DIC is complex. The mechanisms that activate or "trigger" DIC act on processes that are involved in normal hemostasis, namely, the processes of platelet adhesion and aggregation and contact-activated (intrinsic) and tissue factor activated (extrinsic) pathways of coagulation[(71)]. These mechanisms have in common, the capacity in terms of either the magnitude or the duration of the activating stimulus, to exceed normal compensatory processes. Thrombin is persistently or recurrently elaborated and fibrin is formed in the circulating blood. Fibrinogen, various other coagulation factors and platelets are consumed. The fibrinolytic enzyme system is activated and large amounts of FDP are

produced, which further impair haemostatic function. Bleeding, shock and vascular occlusion commonly supervene and produce profound alterations in the function of various organ systems. Normal compensatory processes may become impaired, creating a self-perpetuating "vicious cycle". The ultimate outcome is determined by a dynamic interplay between the various pathologic processes and compensatory mechanisms, in other words, fibrin deposition versus fibrinolysis, depletion versus repletion of coagulation factors and platelets; and production versus clearance of fibrin, FDP, and other products of coagulation[69].

TABLE 4:DIFFERNTIAL DIAGNOSIS OF THROMBOCYTOPENIA

| **Symptoms/sign** | **TTP** | **HUS** | **HELLP** | **AFLP** |
|---|---|---|---|---|
| Abdominal pain | ++ | ++ | ++ | ++ |
| Anemia | ++ | ++ | + | + |
| >LDH | ++++ | ++++ | ++ | +/++ |
| Elevated transaminase | -/+ | -/+ | ++ | ++ |
| Fever | + | - | - | + |
| Headache / visual disturbannes | ++ | - | ++ | -/+ |
| Hypertension | +/++ | ++ | ++ | -/+ |
| Jaundice | - | - | + | + |
| Nausea and vomiting | ++ | ++ | ++ | ++ |
| Proteinuria | +/hematuria | ++ | ++ | ++ |
| Thrombocytopenia | ++ | ++ | ++ | + |
| Hypoglycemia | - | - | -/+ | ++ |

## ANAESTHETIC CONSIDERATION

The choice of anaesthetic technique in the pregnant women with thrombocytopenia largely depends on the proposed method of delivery, gestational age of the fetus, coagulation status, associated obstetric complications, history of recent or current bleeding and other significant medical history. The risk of epidural hematoma in the general population is estimated to be 1 in 150 000 after epidural analgesia[72]. There are eight reports of spinal hematomas following obstetric epidural analgesia/ anaesthesia in the literature but some of these are questionable[73,74]. The incidence of epidural hematoma is 0.2–3.7 in 1,00,000 obstetric epidural blocks[75].

Circulating platelet numbers and their function determine the safety of regional anaesthesia. A lower limit of 100 $\times 10^9$/l for platelet count is suggested as 'safe' for performing an epidural blockade, although there are no supporting data. Several studies have attempted to address the issue of the risk of epidural hematoma when the platelet count is between $50\times10^9$/l and $100\times10^9$/l. Two retrospective studies have suggested that epidural anaesthesia may be safely undertaken when the platelet count is $<100 \times 10^9$/l.[76] In a study of 2929 parturients, no complications associated with regional anaesthesia were recorded in the 24 women who had a platelet count $<100\times10^9$/l[77]. Beilin and colleagues reported that, over a 3-year period, 30 parturients who had platelet counts ranging from $69\times10^9$/l to $98\times10^9$/l safely received epidural anaesthesia. A survey of American anaesthetists reported that 66% in academic practice and 55% of those in private practice would place an epidural anaesthetic when the platelet count is between $80\times10^9$/l and $100\times10^9$/l.[79] Most haematologists suggest

that a platelet count >$50\times10^9$/l is safe for surgery and neuraxial blockade, provided platelet function is normal.[80]

The bleeding time test, a simple bedside test that evaluates the quality and quantity of platelets, is not considered to be reliable to determine the safety of epidural catheter placement because of wide observer variation. It is affected by technical (length and size of cut, occlusion pressure) and patient (ethnicity, diabetes, hypercholesterolemia, etc.) factors.

Coagulation tests performed several weeks before delivery are not reliable in predicting coagulation abnormalities during labour.[83] In a study of 797 women, prothrombin time and the activated partial thromboplastin time were normal in all patients including those with low platelets and plasma fibrinogen concentrations (<2.9 g/l) and it was concluded that these tests are unnecessary in clinically normal parturients. It has been reported that the percentage of women with platelet count values <$100\times10^9$/l increased from 0.5% to 1.4% between blood sampled during the 9th month of pregnancy and that obtained in labour.[84]

Large prospective studies with an estimated sample size of >2,00,000 patients are required to definitively determine whether it is safe to place an epidural or spinal anaesthetic in patients with a platelet count <$100\times10^9$/l.[78] The entire clinical presentation of the patient must be considered when deciding on the appropriate choice of anaesthesia. It is important to ensure that there is no clinical evidence of bleeding and that the platelet count is not decreasing when epidural catheter placement is

contemplated. A decreasing platelet count is considered a contraindication to neuraxial blockade, especially in dynamic conditions such as pre-eclampsia and Pseudo-thrombocytopenia must be excluded. A manual platelet count is more accurate in patients with recent thrombocytopenia because automated counters are not reliable at low platelet counts. Specific questions about medications that might interfere with platelet numbers and function should be asked. A physical examination of the patient should include looking for evidence of bruising and bleeding at venepuncture sites or petechiae at the blood pressure cuff site. Consumptive coagulopathy associated with placental abruption and other conditions must be ruled out. When considering regional anaesthesia in patients with thrombocytopenia, spinal anaesthesia may be safer.[82] Careful monitoring of the patient in the postpartum period to detect early signs and symptoms of an epidural haematoma should be undertaken. General anaesthesia for urgent Caesarean section becomes necessary if coagulation is abnormal or there is bleeding.[84]

Abdul Rahim Gari-Bai et al in their study of thrombocytopenia during pregnancy which was a prospective surveillance study of 1357 healthy women, who presented at the end of normal pregnancies in Hamilton, Ontario, Canada, reported thrombocytopenia in 8.3% of those women. The platelet counts were only very mildly reduced(mean $135x10^9$/L).[85]

Keith R. McCrae et al in their study of Thrombocytopenia in Pregnancy showed that Thrombocytopenia affects 6% to 10% of all pregnant women and other than anemia is the most common hematologic disorder in pregnancy. Gestational

thrombocytopenia, also known as incidental thrombocytopenia of pregnancy, is the most common cause of thrombocytopenia in pregnant women, accounting for approximately 75% of all cases.[86](2010)

Sarah L. Janes et al in their study of Thrombocytopenia in pregnancy, showed that normal pregnancy is generally thought not to affect the platelet count but it has been suggested that the normal range is lower in pregnancy, and that the count falls in the third trimester[87].

Levy JA et al in their study of Thrombocytopenia in pregnancy showed that Thrombocytopenia is the second most common hematologic abnormality during pregnancy and is usually a benign condition[88].

Michal Parnas et al in their study of Moderate to severe thrombocytopenia during pregnancy comparing 199 pregnant women with moderate to severe thrombocytopenia (platelet count below $100\times10^9$/l) with 201 pregnant women without thrombocytopenia found that the main causes of thrombocytopenia were gestational thrombocytopenia (59.3%), immune thrombocytopenic purpura (11.05%), preeclampsia (10.05%), and HELLP (Hemolysis, elevated liver enzymes and low platelet count) syndrome (12.06%) and concluded as moderate to severe maternal thrombocytopenia points to a higher degree of severity of the primary disease, which increases perinatal complications. However, the adverse outcome is specifically attributed to preeclampsia, HELLP syndrome, and rare causes, while the perinatal outcome of GT and ITP is basically favorable. Women with thrombocytopenia were

significantly older , compared with patients without thrombocytopenia, and had higher rates of labor induction and preterm deliveries . Even after controlling for labor induction, using the Mantel–Haenszel technique, thrombocytopenia was significantly associated with preterm delivery. Higher rates of placental abruption were found in pregnant women with thrombocytopenia. In a comparison of perinatal outcomes, higher rates of Apgar scores<7 at 5 min, intrauterine growth restriction , and stillbirth were noted in infants of mothers with thrombocytopenia. These adverse perinatal outcomes were found in rare causes of thrombocytopenia such as disseminated intravascular coagulation (DIC), familial thrombotic thrombocytopenic purpura (TTP), anti-phospholipid antibodies (APLA) syndrome, and myeloproliferative disease, and not among patients with GT[(89)].(2006)

Nadine Ajzenberg et al Pregnancy-Associated Thrombocytopenia Revisited:Assessment and Follow-Up of 50 Cases Whatever the severity of thrombocytopenia, it was found that biological features of an autoimmune disorder in 48% of the women, and chronic thrombocytopenia in 55%. A familial thrombocytopenia was evidenced in 1 case. These 50 women gave birth to 63 neonates, among whom 24 were thrombocytopenic, either at birth or during the first week of life[(90)].

Hisanori Minakami et al in their study of gestational thrombocytopenia showed that although the development of thrombocytopenia in women with pre-eclampsia is well documented, as acknowledged by George, changes in platelet count in an apparently normal pregnancy has not been adequately considered[(91)].

Sainio S et al in their study of Maternal thrombocytopenia at term: a population-based study, showed that Women with gestational thrombocytopenia do not require alteration of their treatment. Fetal blood sampling is not considered necessary when thrombocytopenia is discovered unexpectedly at term. Of 317 women studied, 81% was due to GT, 16% due to HT disorders, 3% due to ITP . There was no correlation between maternal and fetal platelet counts in this study[92].

A retrospective case-control study comparing all pregnant women with moderate to severe thrombocytopenia with 201 pregnant women without thrombocytopenia, delivered during the same study period, was conducted at the Soroka University Medical Centre, which is the sole hospital of the Negev, the southern part of Israel in 2003-2004. Out of 17,499 deliveries at the time of the research, moderate to severe thrombocytopenia was observed in 1.14% of patients.The study population included all pregnant women with moderate to severe thrombocytopenia.[92]

In a study of Gestational thrombocytopenia among pregnant Ghanaian women by Edeghonghon Olayemi et al & Frederick William Akuffo et al at Department of Haematology, University of Ghana Medical School, Ghana,in the year 2011-2012, the prevalence of thrombocytopenia in pregnant women was 15.3% compared with 4% in controls[93].

In a study conducted by Suna Ozdemir and Huseyn Gorkeml in Konya, Turkey in 2006-2007, of 135 women with a platelet count below 100,000/$mm^3$ There were 48

women with GT(35,5%), 32 with ITP (23,7%), 28 with HELLP syndrome(20,7%), and 18 with severe preeclampsia (13,3%) and 9women with other rare causes (6,6%). These women in the study group delivered 147 infants during the study period; there were 8 twins and two triplets. Systemic steroid treatment was used in the patients with ITP or HELLP syndrome. Blood products were transfused to the patients due to severe anemia and increasing the platelet values in operative deliveries[94].

In gestational thrombocytopenia: a prospective study conducted by Marco Ruggeri et al, Corrado Schiavotto et al at Vizenza , Italy with a total of 41 pregnancies observed, vaginal delivery was carried out in 80% patients; two patients were transfused with packed red cells for obstetric hemorrhage (post-partum uterine atony). Neonatal bleeding did not occur. [95]

In a prospective observational study done in the Department of Obstetrics and Gynaecology, VMMC and Safdarjung Hospital in New Delhi, India, for a period of 6 months in 2008, on the basis of the platelet count of these women, they have found that 62% are having milder forms of thrombocytopenia, 31% are in moderate thrombocytopenia group, and 7% are with severe thrombocytopenia. The prevalence of thrombocytopenia is12.82%.In their study, it has been found that around 188 (94%) patients delivered vaginally[96] .

In a study of Prevalence of Thrombocytopenia during Pregnancy & Its Effect on Pregnancy & Neonatal Outcome by Monica Arora, Lajja Goyal in Guru Gobind Singh Medical College, Faridkot, Punjab in 2016, among 1450 deliveries, total 137

women were having thrombocytopenia in third trimester. The commonest etiology was gestational thrombocytopenia (61%), thrombocytopenia due to severe preeclampsia and HELLP syndrome in this study was 24%, placental abruption 6.6%, PPH 4.3%, and wound hematoma 3.6% were noted. Fetal complications – stillbirth 11(8%), low birth weight 14(10.21%), low APGAR 22(16.2%) and neonatal thrombocytopenia 6(4.3%) were noted. Thrombocytopenia in pregnancy did not affect the mode of delivery and pre-term delivery rate. [97]

In a study conducted at Department of Pathology, LN Medical College, J. K. Hospital, Bhopal, Madhya Pradesh, India - Thrombocytopenia during pregnancy: an institutional based prospective study of one year by Anubha Pandey et al, the incidence of thrombocytopenia was 11.68%. This figure was higher than figures of 11.6% reported by Boehlen et al in and 7.2% reported by Sainio et al. They proposed that the higher prevalence in their study might be because of malaria and dengue infections. This study found no influence of age and religion on prevalence of thrombocytopenia in pregnancy like Mathews et al. In this study, gestational thrombocytopenia occurred across the three trimesters. No case of severe thrombocytopenia was seen in first trimester. In the study, maximum cases (41%) of thrombocytopenia were seen in 30-34 weeks of gestation followed by 20% cases in 35-39 weeks of gestation. This was in accordance with the report of Crowther et al who reported that gestational thrombocytopenia in pregnancy develops primarily in the late second or third trimester. This contrasted with the study done by Parnas et al in which maximum cases were in the gestational age 37-40 weeks. Out of 100 cases of thrombocytopenia in this

study, moderate and severe degree of thrombocytopenia was seen in 58% and 20% cases respectively. Gestational thrombocytopenia was the most common etiological factor with 44% cases followed by 23% for hypertensive disorders including HELLP syndrome followed by 21% for Malaria followed by 7% for dengue[98].

In a study of Risk factors of thrombocytopenia in pregnancy by Ayisha Begam et al in Department of Obstetrics and Gynecology, Government Medical College, Trivandrum, Kerala, India in 2016-2017, in 49% of subjects, the cause was identified as gestational thrombocytopenia, 39.5% cases were due to hypertensive disorders of pregnancy, 10.4% was due to ITP. SLE, AFLP, Dengue infection, HUS and APLA were rare causes of thrombocytopenia in their hospital. Of the 96 cases enrolled in the study, 88 were diagnosed during pregnancy. Amongst the hypertensive disorders, 16.7% was due to gestational hypertension, 10.4% due to preeclampsia and 7.4% were due to HELLP syndrome. 82.3% of patients with thrombocytopenia in this study were asymptomatic.

In Burrows' study of thrombocytopenia in pregnancy, 8 mothers had SLE, accounting for 0.8% of all thrombocytopenic gravidas. None of their infants had thrombocytopenia.Although both SLE and APS can cause fetal/neonatal complications (eg, heart block, second and third trimester fetal demise), thrombocytopenia plays no significant perinatal role.In Burrows' study, 19 pregnancies were complicated by alloimmune thrombocytopenia. Nine infants were born with severe thrombocytopenia. Intracranial hemorrhage was observed in 3 fetuses, one with fetal demise; no new cases occurred in neonates. Alloimmune

thrombocytopenia accounted for all the thrombocytopenia-related fetal morbidity and mortality in this large study. [50]

In a study of Neonatal outcome in pregnancies after preterm delivery for HELLP syndrome by Halil Aslan et al, Ahmet Gul et al, they compared neonatal outcome after preterm delivery of infants where pregnancy had been complicated by the HELLP syndrome. There were 518 pregnancies complicated by hypertensive disorders and 93 by HELLP syndrome. The incidence of HELLP syndrome among women with severe preeclampsia was 19.5%. They found a significant difference in the incidence of intrauterine growth restriction, intrauterine fetal death, abruptio placenta, and fetal distress between the two groups. There were no significant differences in complications (respiratory distress syndrome, intraventricular hemorrhage, necrotizing enterocolitis and sepsis) between the HELLP syndrome group and controls. However, the neonatal death rate and the need for mechanical ventilation and neonatal intensive care were greater in the HELLP syndrome group. [100]

In a study of Thrombocytopenia in pregnancy with different diagnoses by Wang et al, clinical data from 195 pregnant women with thrombocytopenia attending 2 tertiary hospitals from January 2014 to October 2016 were retrospectively studied. There were 60%, 28.2%, and 11.8% cases of pregnancy-associated thrombocytopenia (PAT), idiopathic thrombocytopenia (ITP), and hypertensive disorder in pregnancy (PIH), respectively. The percentage of nulliparous women, gestational age at delivery, date of diagnosis of thrombocytopenia, and delivery mode significantly differed

between the patients in these 3 groups (P <0.05). Patients with PIH had a higher percentage of premature delivery and of lower birth weight infants than patients in the other 2 groups. The 3 groups had similar incidences of postpartum hemorrhage, rates of stillbirth, and neonatal Apgar scores at 5minutes. PAT and PIH patients had different platelet counts after delivery compared with at diagnosis, whereas the platelet counts of the ITP patients were similar at diagnosis and after delivery. ITP patients in the non-treatment group and the treatment group had significantly different platelet counts (P <0.05), and in the treatment group, the maternal platelet count did not differ for treatment with intravenous immunoglobulin (IVIg) versus corticosteroids. [101]

In a study of Thrombocytopenia in hypertensive disorders of pregnancy by Rupakala et al, at Department of Obstetrics and Gynecology, Rajarajeswari Medical College and Hospital, Bangalore, Karnataka, India in 2008 , hypertensive disorders of pregnancy cases were classified into: gestational hypertension, mild preeclampsia, severe preeclampsia, Hemolysis, Elevated Liver enzymes, and Low Platelet levels (HELLP) syndrome and eclampsia. The incidence and severity of thrombocytopenia along with maternal and fetal complications encountered in the five groups were analysed. Preeclampsia- mild (29.25%) and severe (22.5%), accounted for most of the cases followed by eclampsia (3%) and gestational HTN (1.5%). Among these hypertensive patients, mild thrombocytopenia was noted in 40% cases, moderate thrombocytopenia in 32% cases, severe thrombocytopenia in 8% cases, and normal platelet counts in 20% cases. Poor maternal outcome was seen in 10.67% cases due

to HELLP syndrome and postpartum hemorrhage. Poor fetal outcome was seen in 16% cases due to intrauterine growth restriction and perinatal mortality. [102]

In a study conducted in the Department of Hematology and Immunohematology, School of Biomedical and Laboratory Sciences, College of Medicine and Health Sciences, University of Gondar, Gondar, Ethiopia, where a total of 217 women receiving antenatal care service at Gondar University Hospital participated. Thrombocytopenia among 19 pregnant women showed a prevalence of 8.8%.[103]

In a study of Thrombocytopenia in pregnancy by Douglas B. Cines and Lisa D at Maternal and Child Health Research Center, Department of Obstetrics and Gynecology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, the incidence of thrombocytopenia was 8%[38].

Cook in 1991 reviewed a 10 year experience (1980-1990) with ITP treatment which included 25 women and 32 infants. Platelet counts were obtained in 23 out of 32 infants. Of 8 infants with low platelets at birth 3 were mild, 3 were moderate and 2 were severe. A total of 6 infants had severe thrombocytopenia at birth or during the neonatal period. Median platelet nadir occurred 4 days following delivery. Eighteen cesarean deliveries were performed with 6 cases having complications(infection, haematoma and transfusion). [104]

Payne in 1997 reviewed a 10 year experience with 55 newborns to 41 women who had ITP. A total of 16 scalp samplings were performed. Three PUBS were

performed. Two were associated with complications. Of 24 (44%) cesarean deliveries reported half were performed solely due to ITP. Five of the cesarean deliveries were associated with PPH and 3 were associated with blood transfusions. Four (8%) infants had severe thrombocytopenia with platelet count less than 50,000/mcl at birth. 2 of 4 were delivered vaginally, 2 by cesarean section and all had normal results on head ultrasound. 3 neonates developed severe thrombocytopenia during first week of life. One experienced intracranial hemorrhage on fourth day of life. Scalp counts did not correlate with neonatal platelet count. [105]

In Sibai et al's series of 442 pregnancies complicated by HELLP syndrome pulmonary edema was reported in 6% and liver hematoma and retinal detachment was reported in 0.9%. Maternal mortality was 1.1%. Acute renal failure was reported in 7.4% of patients with this syndrome. HELLP syndrome puts the patient for increased risk for both antenatal and postnatal eclampsia[106].

In Audibert et al's series, blood transfusions(25% versus 4%), disseminated intravascular coagulation (15% versus 0%) and cesarean delivery (75% versus 52%) were more frequent among patients with HELLP syndrome when compared with partial HELLP syndrome[6].

Magann et al randomized 12 patients with class 2/3 HELLP syndrome to intravenous dexamethasone every 12 hours until delivery. Thirteen control patients were included. Improvement in platelet count and liver function tests enabled a mean delay in delivery of 25 hours. Urine output was increased in steroid used group. [107]

Martin et al examined the use of plasma exchange for the postpartum treatment of HELLP syndrome. Two groups of patients were identified. Those with slow resolution of HELLP syndrome beyond 72 hours postpartum(n=9) and those with worsening HELLP syndrome and with single or multiple organ dysfunction(n=9). The patients in the first group responded promptly to one or two plasma exchanges. Whereas the latter group did much worse including 2 maternal deaths. [108]

In a retrospective study by Shin et al indicated that in pregnant women with aplastic anemia, obstetric and disease complications are more prevalent in those with severe thrombocytopenia than in those with nonsevere thrombocytopenia. The study, which included 61 patients with aplastic anemia, found that in women with severe thrombocytopenia, the incidence of transfusion during pregnancy or the postpartum period (72.7% and 45%, respectively) was greater than in those with nonsevere thrombocytopenia (15.4% and 2.7%, respectively). It was also found that 25% of women with severe thrombocytopenia underwent bone marrow transplant after delivery, compared with 0% of those in the nonsevere thrombocytopenia group. Moreover, the odds ratios for composite disease complications and composite obstetric complications were higher in the women with severe thrombocytopenia than in the non-severe thrombocytopenia patients. In addition, among patients in the severe thrombocytopenia group, gestational age at the platelet count's nadir and at delivery was lower than in the women with non-severe thrombocytopenia. [109]

In a study Lescale evaluated 8 different platelet antibodies in 250 gravid women with thrombocytopenia (160 with presumed GT, 90 with ITP) to determine if any

antibodies could distinguish the 2 conditions. Platelet-associated IgG was comparably elevated in most women with GT (69.5%) and ITP (64.6%), P =0.24. A significantly higher proportion of patients with ITP had indirect IgG compared with patients with GT (85.9% vs 60.3%, P< 0.001), but significant overlap existed, limiting its clinical value. Antiplatelet antibody tests, either alone or in combination, cannot be used to distinguish ITP from GT. [38]

## MATERIALS AND METHODS

**PLACE OF STUDY:** Department of Obstetrics and Gynaecology, Thanjavur medical college,Raja mirasudhar hospital,Thanjavur

A total number of 273 women with platelet count <1,50,000/$mm^3$ admitted in their third trimester( >32 weeks) in the Department of Obstetrics and Gynaecology, RMH were taken up for the study from December 2017 to January 2018.

**STUDY DESIGN:** Hospital based prospective observational study.

**STUDY PERIOD:** 1 year

**STUDY POPULATION:** Patients admitted in RMH in their third trimester of pregnancy who were diagnosed to have thrombocytopenia.

**INCLUSION CRITERIA:**

Antenatal patients with platelet count less than $150\times10^9$/l in the third trimester who were willing to participate in the study were enrolled for study

**EXCLUSION CRITERIA:**

- Refused participation
- Women with known haematological disorders
- Women with history of Diabetes Mellitus, collagen disorders, Tuberculosis, Epilepsy,Women with previous CS.

**DATA COLLECTION TECHNIQUE:** The present study was approved by the Institutional Ethics Committee of Thanjavur Medical College and Hospital,Thanjavur.

The collection of the sample was after taking informed consent of the patient, blood specimen was withdrawn from the ante-cubital vein using a dry sterile disposable syringe and needle. 2ml of blood was dispensed into EDTA anticoagulant tube. The specimens were labelled with subject's name, age, sex and hospital number and sent to laboratory for platelet count estimation, and analyzed within 4 hours.

Data of the patients who were diagnosed to have thrombocytopenia were collected in a proforma from time to time. A written informed consent was obtained from the patient/attendants/legal guardian of the patient before enrolment.

Antenatal women were enrolled in the study in third trimester. All women had platelet count estimation at the time of enrolment. Platelet count assessment was done through automated blood count analyser with routine antenatal haematological evaluation of the patient. Detailed menstrual, obstetric history was taken. Presenting complaints if any, findings of general, systemic and obstetric examination including pelvic examination if required of all the patients were recorded in an approved proforma. Investigations including urine for albumin/sugar, CBC, LFT, RFT, peripheral blood smear, coagulation profile, detection of malaria (by malarial antigen detection or peripheral blood smear) , dengue IgG and IgM antibodies were done as and when required. Gestational age was established by menstrual history and clinical examination confirmed by USG. The diagnosis was inferred from the above

investigations. Platelet count of 1,00,000/$mm^3$ to 1,50,000/$mm^3$ was classified as mild thrombocytopenia, 50,000/$mm^3$ to <1,00,00/$mm^3$ as moderate thrombocytopenia and <50,000/$mm^3$ as severe thrombocytopenia.

All the cases were followed till delivery to record any complications like preterm labour, abruption, preeclampsia, postpartum hemorrhage or any other morbidities. Duration of pregnancy at the time of delivery, indication of induction and method (if required) and mode of delivery including indication for instrumental delivery or caesarean section were recorded. Progress of labour was monitored with partograph. Neonates of all cases were looked up for 1 minute and 5 minutes APGAR score, NICU admission and neonatal deaths. The platelet count was repeated 10 days after delivery. The diagnosis of ITP was made according to the guidelines of the American Hematology Association and preeclampsia according to those of the International Society for Study of Hypertension in Pregnancy*.

**DATA ANALYSIS:** The data were coded and entered in MS-excel office 2010. The data were analyzed using Graph Pad Prism version 5. The categorical data were represented as n and numerical data in mean with SD. Fisher's exact test was used to compare the proportions between the groups for sample less than 30. Chi square test was used to compare the proportions between the groups for sample more than 30. Mann Whitney U test was used to compare the means of quantitative data with non-normal distribution. $p<0.05$ was considered statistically significant.

## DIAGNOSTIC CRITERIA

### DIAGNOSIS OF GESTATIONAL THROMBOCYTOPENIA

Diagnosis of exclusion with platelet count <1,50,000 and >50000 /L in the absence of other causes or disorder associated with thrombocytopenia.

### DIAGNOSIS OF ITP (AMERICAN HEMATOLOGICAL SOCIETY):

Primary ITP was defined as a platelet count less than 50000/L in the absence of other causes or disorders that may be associated with thrombocytopenia.

### DIAGNOSIS OF SEVERE PREECLAMPSIA:

Hypertension developing after 20 weeks gestation and the coexistence of one or more of the following new onset conditions:

1. Proteinuria
2. Other maternal organ dysfunction: renal insufficiency (creatinine >90 μmol/L) liver involvement (elevated transaminases and/or severe right upper quadrant or epigastric pain) neurological complications (examples include eclampsia, altered mental status, blindness, stroke, or more commonly hyper reflexia when accompanied by clonus, severe headaches when accompanied by hyperreflexia, persistent visual scotomata) haematological complications (thrombocytopenia, DIC, haemolysis)
3. Uteroplacental dysfunction foetal growth restriction

**DIAGNOSIS OF HELLP(BY MISSISSIPI CLASSIFICATION)**

1. Elevated liver enzymes AST or ALT >/= 2 times the upper level of normal .
2. Low platelet <1,00,000 cells/L
3. Peripheral smear with schitocytes and burr cells.
4. S. Bilirubin >1.2mg/dl
5. LDH >2 times the upper level of normal (200-400IU/L)
6. Severe anemia unrelated to blood loss.

**DIAGNOSIS OF AFLP**

1. Elevated liver enzymes AST or ALT >/= 2 times the upper level of normal .
2. Elevated serum bilirubin levels
3. Low serum creatinine
4. Elevated white blood count
5. Elevated ammonia level
6. Elevated uric acid
7. Prolonged PT/INR,aPTT
8. Low platelet count
9. Low fibrinogen
10. Fragmented RBC and burr cells
11. Proteinuria

## RESULTS AND OBSERVATIONS

The study comprises of 273 cases who were admitted with thrombocytopenia (platelet count <150×$10^9$/l) in their third trimester in the Department of Obstetrics and Gynaecology, RMH,Thanjavur, during the 1 year study period. The following are observations made at the end of the study.

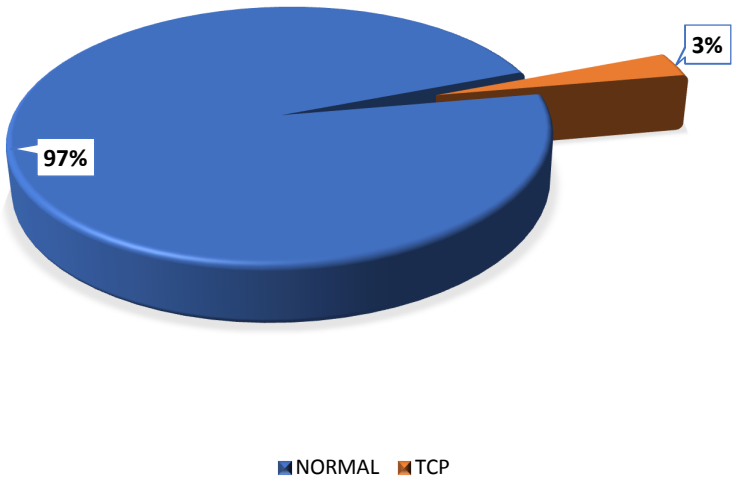
### PREVALENCE OF THROMBOCYTOPENIA:

Study population- 8544

Sample size- 273

Prevalence of thrombocytopenia in this study- 3.1%

**FIGURE 2:PREVALENCE OF THROMBOCYTOPENIA**



**AGE**

TABLE 5: AGE WISE DISTRIBUTION OF THE PATIENTS

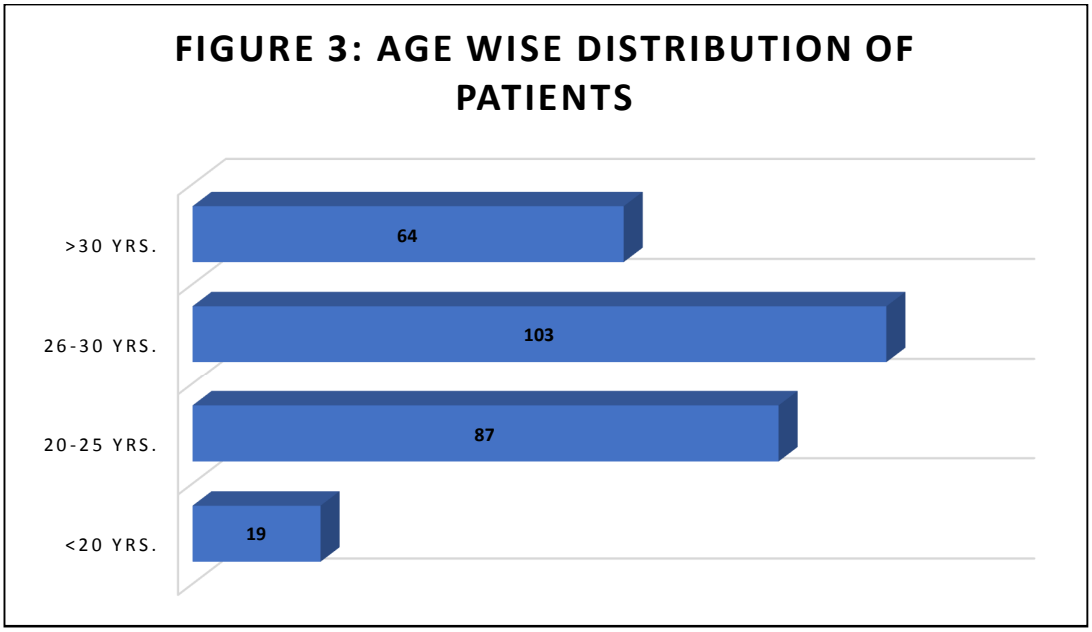
| AGE | No. of patients | % |
|---|---|---|
| <20 years | 19 | 6.9 |
| 20 – 25 years | 87 | 31.86 |
| 26 – 30 years | 103 | 37.72 |
| >30 years | 64 | 23.44 |

Of the total 273 cases of thrombocytopenia the mean age group of antenatal mothers was 26.5yrs. with SD of 4.5.

The maximum age was 39 years and minimum was 18 years.

Patients <20 years constitute 6.9%(19) of total cases,20-25 years were 31.86%(87),26-30 years constitute 37.72%(103) and >30 years were 23.44%(64).

Age of the patient has no influence on the occurrence of thrombocytopenia (p value-0.701).



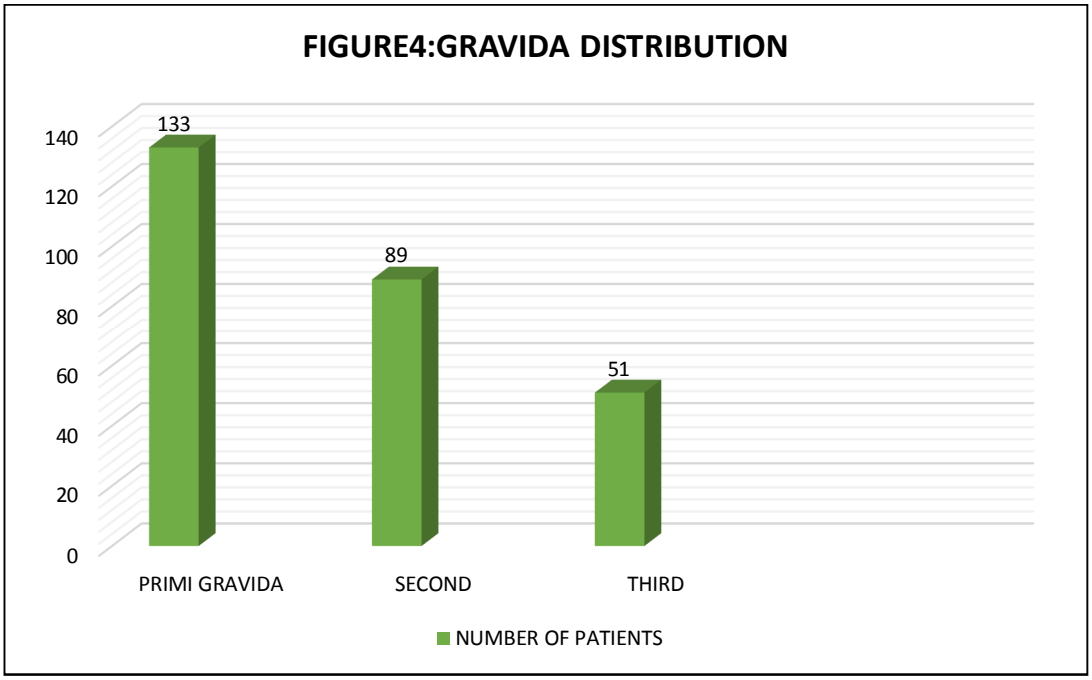
**GRAVIDA**

TABLE 6: GRAVIDA DISTRIBUTION AMONG PATIENTS

| GRAVIDA | NO. OF PATIENTS | % |
|---|---|---|
| Primi gravida | 133 | 48.71 |
| Second gravida | 89 | 32.60 |
| Multi gravida | 51 | 18.68 |

Of the total 273 cases of thrombocytopenia, 48.71% ( 133) were Primi Gravida and 32.60%(89) were second gravida and 18.68%(51) Multi Gravida.

Gravida has no influence on the occurrence of thrombocytopenia (p=0.698).



**GESTATIONAL AGE:**

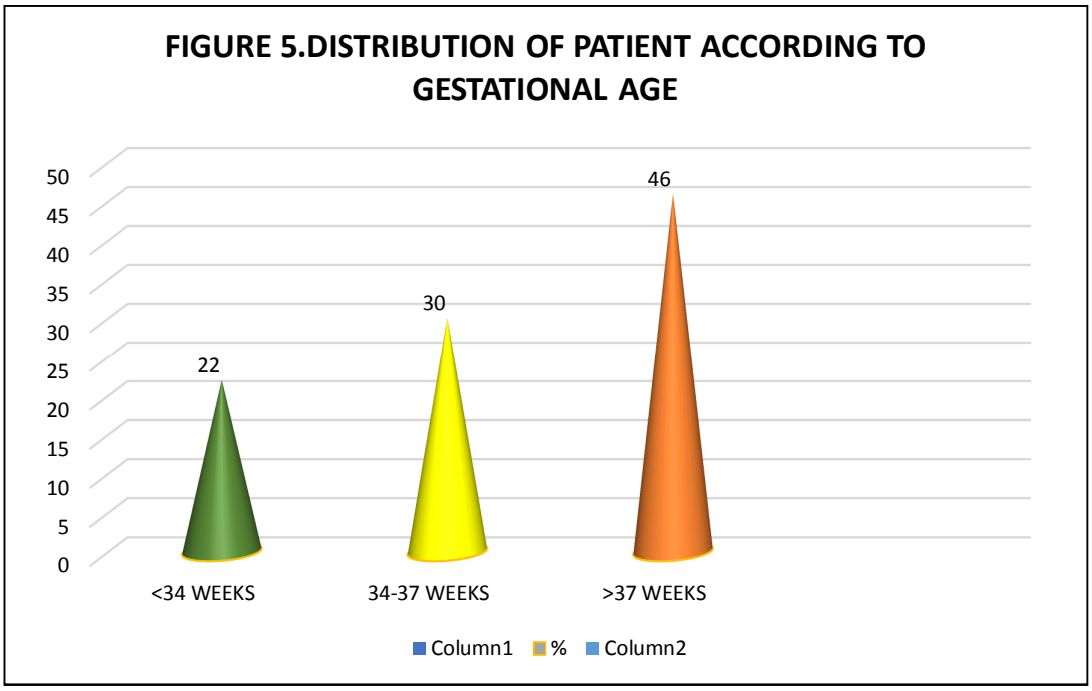
TABLE 7: DISTRIBUTION OF PATIENTS ACCORDING TO

| Gestational age | Number of patients | % |
|---|---|---|
| <34 weeks | 62 | 22.71 |
| 34 to 37 weeks | 84 | 30.76 |
| >37 weeks | 127 | 46.52 |

GESTATIONAL AGE:

Of the total 273 cases, the mean gestational age was 36.5 weeks,with minimum of 28 weeks and maximum of $40^{+2}$ weeks.

22.71 %(62) were less than 34 weeks of gestation,30.76%(84) were in 34-37 weeks category, and 46.52%(127) were with gestational age more than 37 weeks.



**SEVERITY OF THROMBOCYTOPENIA:**

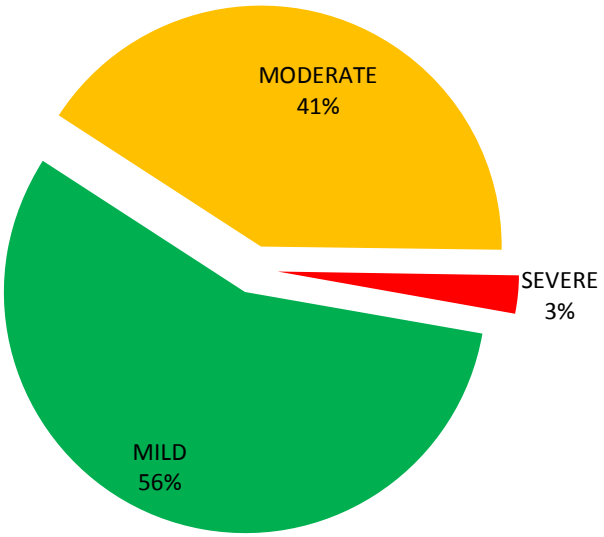
TABLE 8: DISTRIBUTION OF PATIENTS ACCORDING TO SEVERITY

| SEVERITY | NUMBER OF PATIENTS | % |
|---|---|---|
| Mild | 155 | 56.77 |
| Moderate | 111 | 41.39 |
| Severe | 7 | 1.83 |

Of the total 273 cases studied, the mean platelet count was 1,03,836/$mm^3$ with maximum of 1,48,000/$mm^3$ and minimum of 20000/$mm^3$.

Mild thrombocytopenia was noted in 56.77%( 155) of the total cases, moderate thrombocytopenia in 41.39 %(111) and severe thrombocytopenia in 1.83%(7) of cases.

**FIGURE 6: SEVERITY OF THROMBOCYTOPENIA**



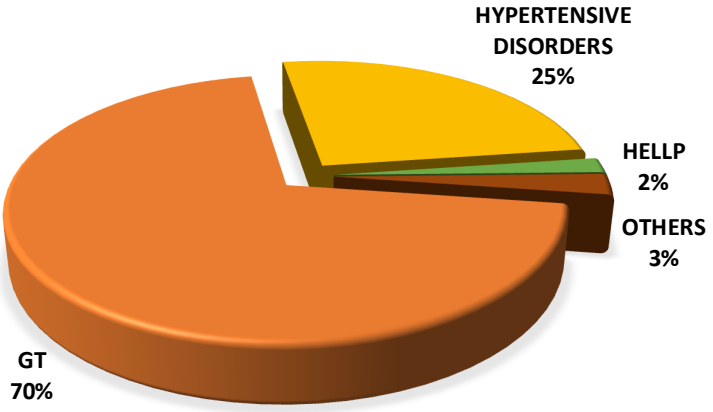
**ETIOLOGY**

TABLE 9: DISTRIBUTION ACCORDING TO ETIOLOGY

| ETIOLOGY | NO. OF PATIENTS | % |
|---|---|---|
| Gestational thrombocytopenia | 191 | 69.9 |
| Hypertensive disorders | 69 | 25.27 |
| HELLP | 5 | 1.83 |
| Pancytopenia | 2 | 0.007 |
| Dengue | 2 | 0.007 |
| AFLP | 1 | 0.003 |
| ITP | 3 | 0.01 |

Of the total 273 cases studied, 69.9% (191) of cases were found to be Gestational Thrombocytopenia, 25.27%(69) were associated with hypertensive disorders of pregnancy and 1.83%(5) associated with HELLP syndrome. Pancytopenia , ITP,Dengue ,AFLP contributed to 0.003%(1),0.01%(3),0.007%(2),0.003%(1) respectively.

**FIGURE 7: DISTRIBUTION ACCORDING TO ETIOLOGY**



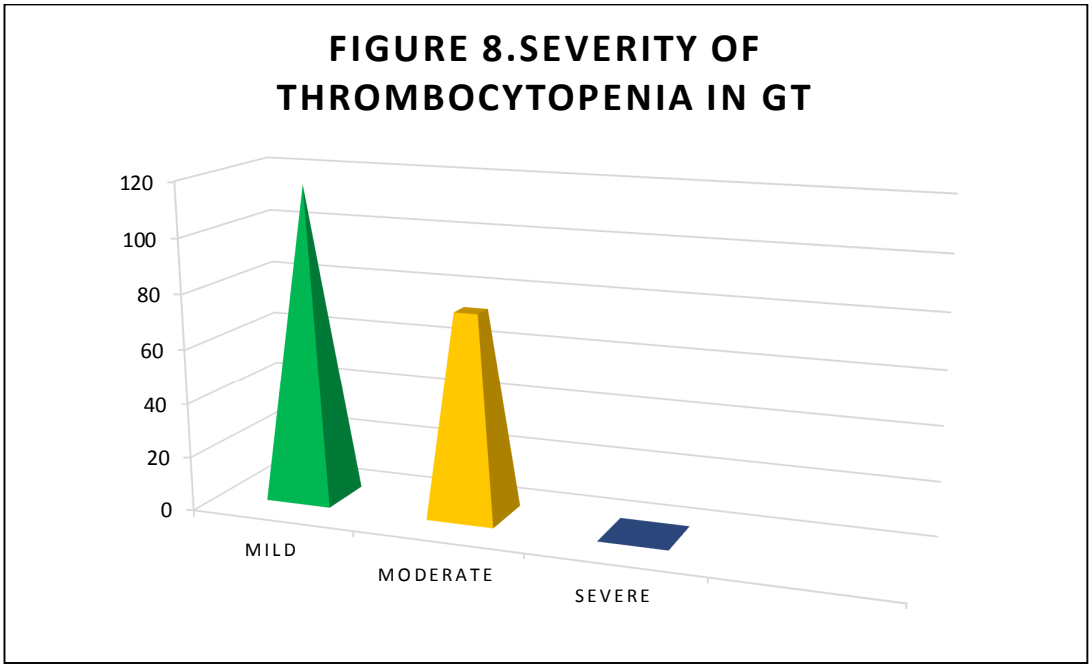
**SEVERITY IN CASES OF GESTATIONAL THROMBOCYTOPENIA:**

TABLE 10: DISTRIBUTION OF PATIENTS WITH GT ACCORDING TO SEVERITY

| SEVERITY | NUMBER OF PATIENTS | % |
|---|---|---|
| Mild | 116 | 60.7 |
| Moderate | 75 | 39.3 |

Of the total 191 cases of gestational thrombocytopenia studied, 60.7%(n=116) had mild thrombocytopenia, with p=0.014 which is statistically significant, 39.3%(n=75) had moderate thrombocytopenia and no severe thrombocytopenia were reported.

This analysis shows that gestational thrombocytopenia is usually associated with mild thrombocytopenia.

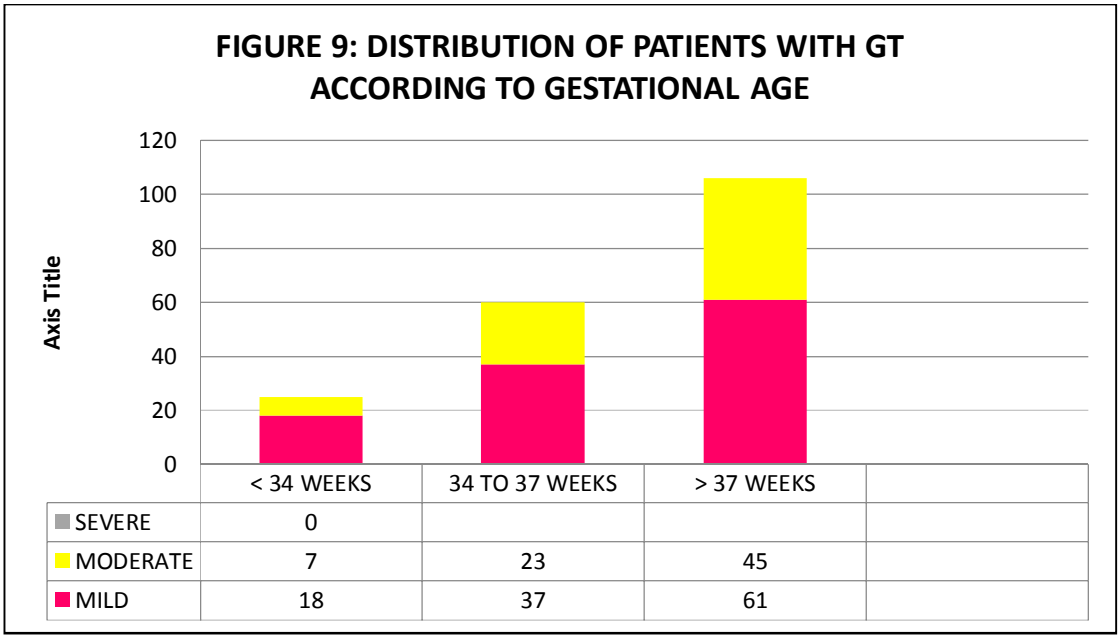


**SEVERITY IN CASES OF GESTATIONAL THROMBOCYTOPENIA WITH REFERENCE TO GA**

TABLE 11: DISTRIBUTION OF PATIENTS WITH GT ACCORDING TO GESTATIONAL AGE

| Gestational age | Thrombocytopenia | | | |
|---|---|---|---|---|
| | Mild | | Moderate | |
| | n | % | N | % |
| <34 weeks | 18 | 11.6 | 7 | 6.4 |
| 34 to 37 weeks | 37 | 24 | 23 | 20.1 |
| >37 weeks | 61 | 39.6 | 45 | 40.3 |

Of the total 191 cases of gestational thrombocytopenia, among the 116 mild severity cases, 11.6%(n=18) were of <34 weeks gestation, 24% (n=37) belonged to 34-37 weeks category, and 39.6%(n=61) were >37 weeks gestation. Among 75 moderately severe cases, 6.4 %( n=7) were of <34 weeks gestation, 20.1% (n=23) belonged to 34-37 weeks category and 40.3% (n=45) were>37 weeks gestation. And no severe thrombocytopenia cases were reported in gestational thrombocytopenia group.



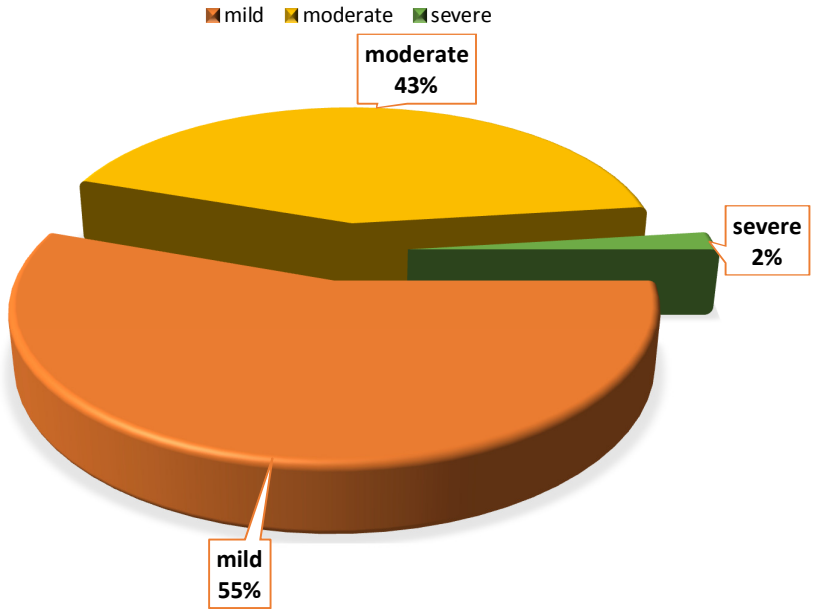
**SEVERITY OF THROMBOCYTOPENIA IN HYPERTENSIVE DISORDERS/HELLP SYNDROME**

TABLE 12: SEVERITY IN CASES OF HYPERTENSIVE DISORDERS/HELLP SYNDROME

| SEVERITY | NO. OF PATIENTS | % |
|---|---|---|
| Mild | 39 | 52.7 |
| Moderate | 30 | 40.5 |
| Severe | 5 | 6.7% |

Of the total 74 hypertensive cases studied, 52.7 %(n=39) had mild thrombocytopenia, 40.5%(n=30) had moderate thrombocytopenia and 6.7%(n=5)had severe thrombocytopenia.

**FIGURE 10: SEVERITY IN CASES OF HYPERTENSIVE DISORDERS/HELLP SYNDROME**



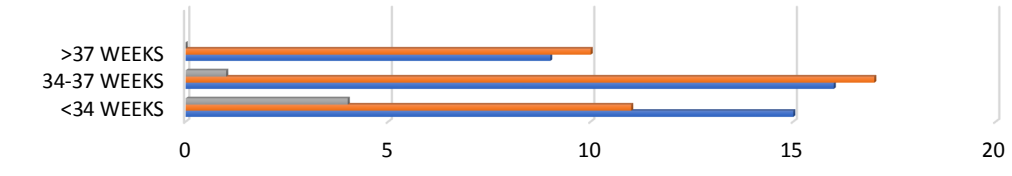
**SEVERITY OF THROMBOCYTOPENIA IN CASES OF HYPERTENSIVE DISORDERS OF/ HELLP SYNDROME ACCORDING TO GA:**

TABLE 13: DISTRIBUTION OF PATIENTS WITH HYPERTENSIVE DISORDERS /HELLP SYNDROME ACCORDING TO GA:

| Gestational age | Thrombocytopenia | | | | | |
|---|---|---|---|---|---|---|
| | Mild | | Moderate | | Severe | |
| | N | % | n | % | N | % |
| <34 weeks | 15 | 9.7 | 11 | 10.1 | 4 | 47.1 |
| 34 to 37 weeks | 15 | 5.1 | 9 | 7.3 | 1 | 14.3 |
| >37 weeks | 9 | 4.5 | 10 | 9.2 | 0 | 0 |

Of the total 74 cases studied, among the 39 mild severity cases,9.7%(n=15) were of <34 weeks gestation, 5.1% (n=15) belonged to 34-37 weeks gestation, and 4.5%(n=9) were >37 weeks gestation.Among 30 moderate severity cases, 10%(n=11) were of <34 weeks gestation, 7.3% (n=9) belonged to 34-37 weeks and 9.2% (n=10) were>37 weeks gestation. Among 5 severe thrombocytopenia cases, 47%(n=4) were of <34 weeks with p=0.015 which is statistically significant, 14.3%(n=1) were 34-37 weeks. No severe thrombocytopenia was noted in >37 weeks group.

**FIGURE 11: SEVERITY OF THROMBOCYTOPENIA IN HYPERTENSIVE DISORDERS/HELLP SYNDROME WITH RESPECT TO GESTATIONAL AGE**



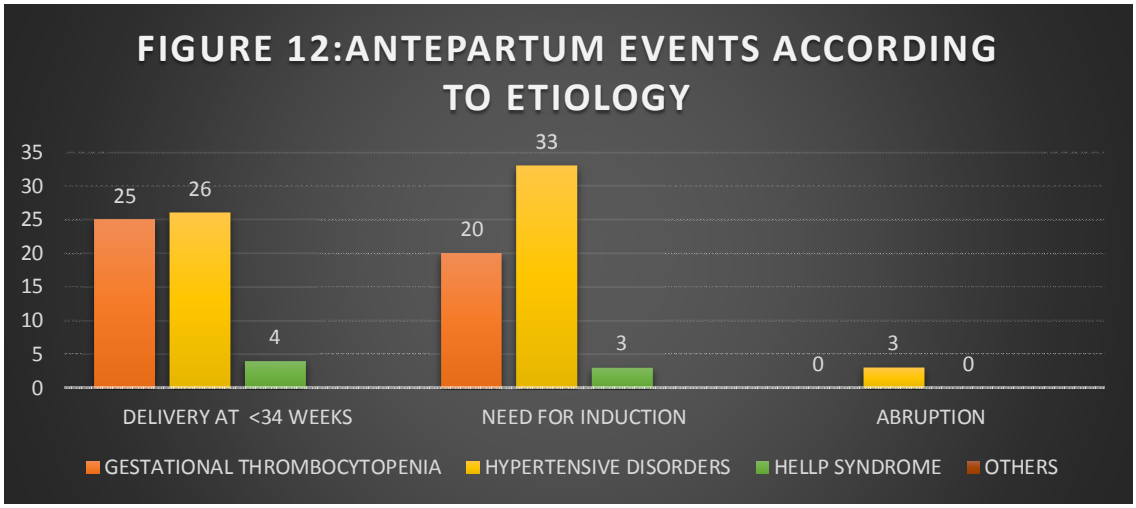

| | <34 WEEKS | 34-37 WEEKS | >37 WEEKS | |
|---|---|---|---|---|
| severe | 4 | 1 | 0 | |
| moderate | 11 | 17 | 10 | |
| mild | 15 | 16 | 9 | |

**ANTEPARTUM EVENTS:**

TABLE 14: ANTEPARTUM EVENTS ACCORDING TO ETIOLOGY

| Etiology | Total Number Of Patients | Delivery At GA<34 Weeks | Need For Induction | Abruption |
|---|---|---|---|---|
| Gestational thrombocytopenia | 191 | 2 | 20 | 0 |
| Hypertensive disorders | 69 | 26 | 33 | 3 |
| HELLP | 5 | 4 | 3 | 0 |
| OTHERS | | | | |
| Pancytopenia | 2 | 0 | 0 | 0 |
| Dengue | 2 | 0 | 0 | 0 |
| AFLP | 1 | 0 | 0 | 0 |
| ITP | 3 | 0 | 0 | 0 |

5 patients with gestational thrombocytopenia, 26 patients with hypertensive disorders OIand 4 patients with HELLP syndrome delivered at GA<34 weeks. 20 patients with Gestational thrombocytopenia, 33 patients with hypertensive disorders and 3 patients with HELLP needed induction of labour. 3 patients with hypertension had abruptio placenta.No adverse antepartum events were noted in patients with thrombocytopenia due to other causes.



**MODE OF DELIVERY**

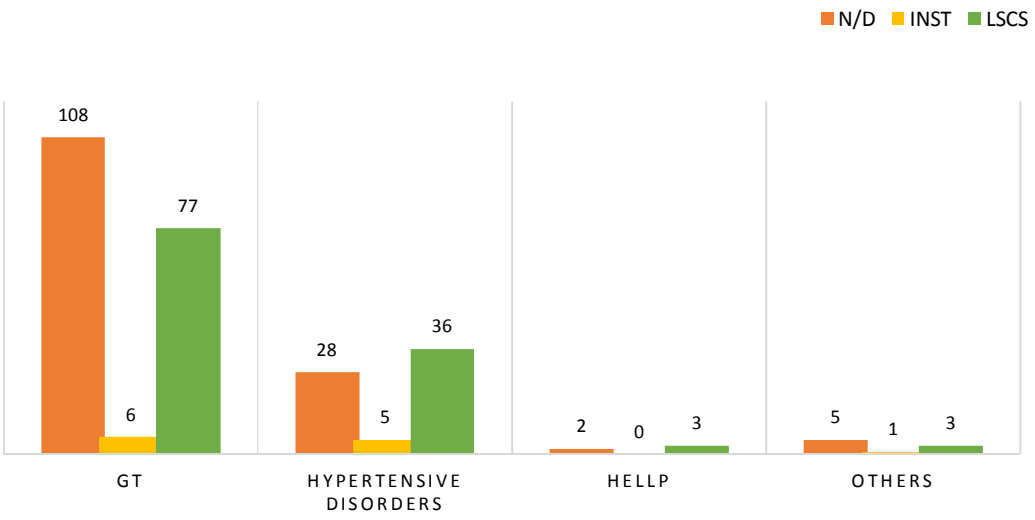
TABLE 15: MODE OF DELIVERY ACCORDING TO ETIOLOGY

| Etiology | No. Of Patients | N/D | Instrumental Delivery | LSCS |
|---|---|---|---|---|
| Gestational thrombocytopenia | 191 | 108 | 6 | 77 |
| Hypertensive disorders | 69 | 28 | 5 | 36 |
| HELLP | 5 | 2 | 0 | 3 |
| Pancytopenia | 2 | 2 | 0 | 0 |
| Dengue | 2 | 2 | 0 | 0 |
| AFLP | 1 | 0 | 0 | 1 |
| ITP | 3 | 1 | 0 | 2 |

Among 191 gestational thrombocytopenia cases, 108 delivered normally vaginally, 6 had instrumental delivery and 77 had LSCS.Among 69 hypertensive cases, 28 delivered normally vaginally, 5 had instrumental delivery and 36 had LSCS.Among 5 cases with HELLP syndrome, 2 delivered vaginally and 3 by LSCS. 2 pancytopenia cases and 2 dengue cases were delivered normally vaginally. 1 case of AFLP delivered by LSCS.

Among 3 ITP cases, 1 delivered by labour natural and 2 by LSCS. By applying Fisher's exact test; Chi square value = 18.24; df=10; p = 0.051 (Not Significant). This implies that the presence of thrombocytopenia has no influence on the mode of delivery.

## FIGURE 13: MODE OF DELIVERY ACCORDING TO ETIOLOGY

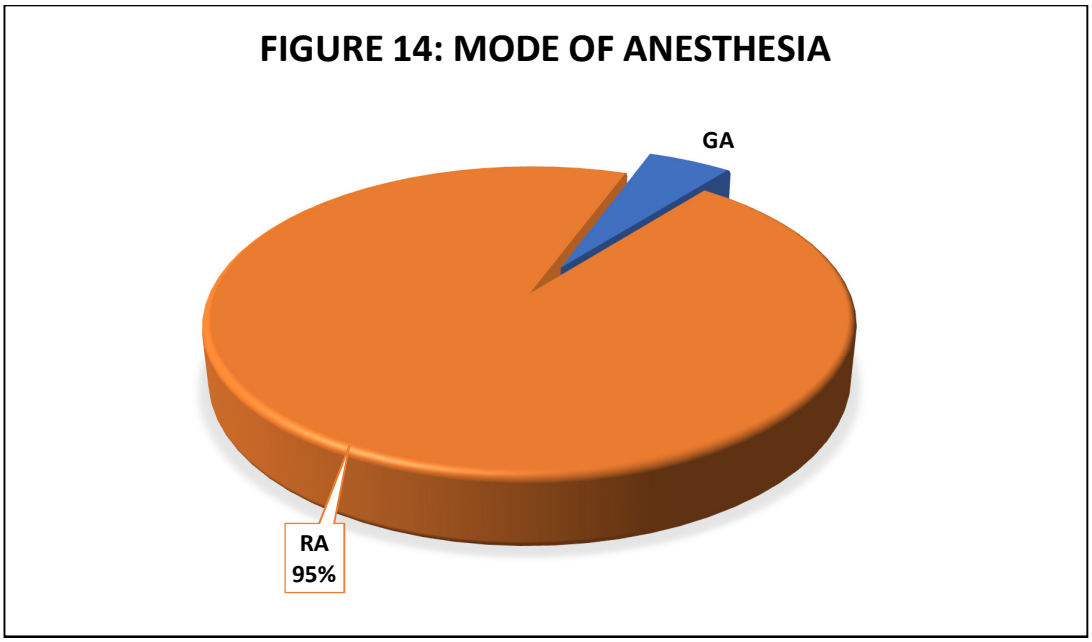


**MODE OF ANESTHESIA**

TABLE 16: MODE OF ANESTHESIA ACCORDING TO ETIOLOGY

| Etiology | No. Of Patients | LSCS | GA | SA |
|---|---|---|---|---|
| Gestational thrombocytopenia | 191 | 77 | - | 77 |
| Hypertensive disorders | 69 | 36 | 2 | 34 |
| HELLP | 5 | 3 | 2 | 1 |
| Pancytopenia | 2 | 0 | 0 | 0 |
| Dengue | 2 | 0 | 0 | 0 |
| AFLP | 1 | 1 | 1 | 0 |
| ITP | 3 | 2 | 2 | 0 |

Of the total 273 cases 119 delivered by LSCS. 113(94.95%) was taken up by regional anesthesia(spinal anesthesia) and 6 (5.04%)cases taken under general anesthesia



**BLEEDING TENDENCIES :**

TABLE 17: BLEEDING TENDENCIES INLUDING PPH ACCORDING TO THE TIME OF OCCURENCE

| **Total (N)** | **Patients With Bleeding Tendencies** | | **Antepartum** | | **Intrapartum** | | **Postpartum** | |
|---|---|---|---|---|---|---|---|---|
| 273 | n | % | n | % | n | % | n | % |
| | 16 | 6.2% | 3 | 17.6 | 10 | 64.7% | 3 | 17.6 |

Of the total 273 thrombocytopenia cases,6.2%(16)developed bleeding manifestations in their course of stay, of which 17.6%(3) occurred in antepartum period ,64.7%(10) in intrapartum period, and 17.6%(3) in post-partum period.

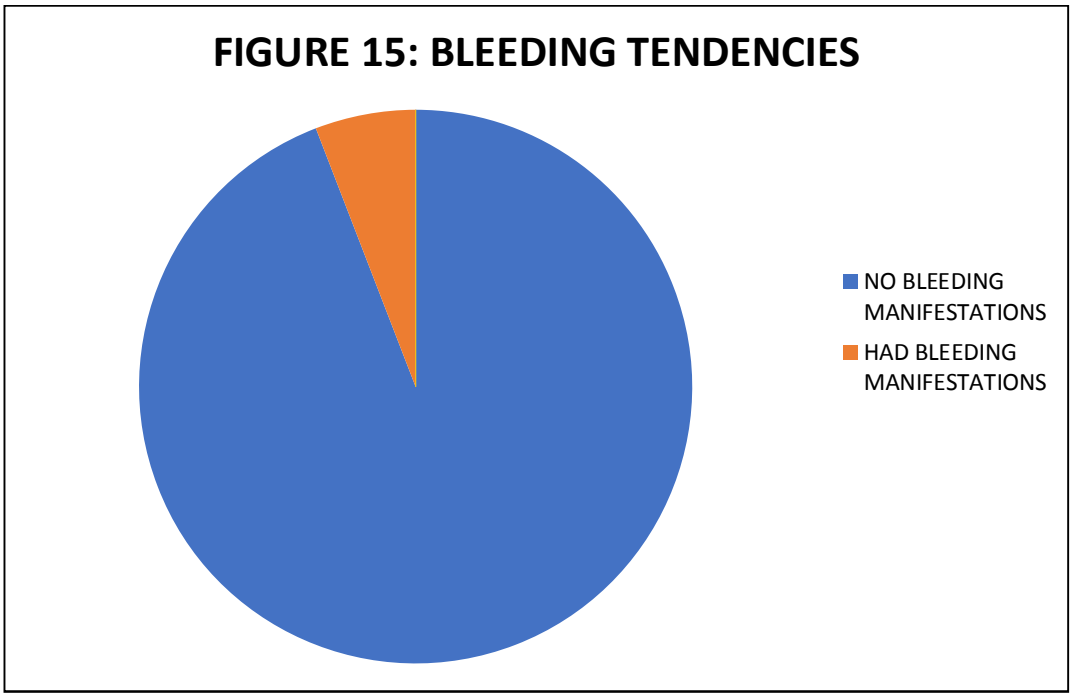


**FIGURE 16: BLEEDING TENDENCIES ACCORDING TO THE TIME OF OCCURENCE**

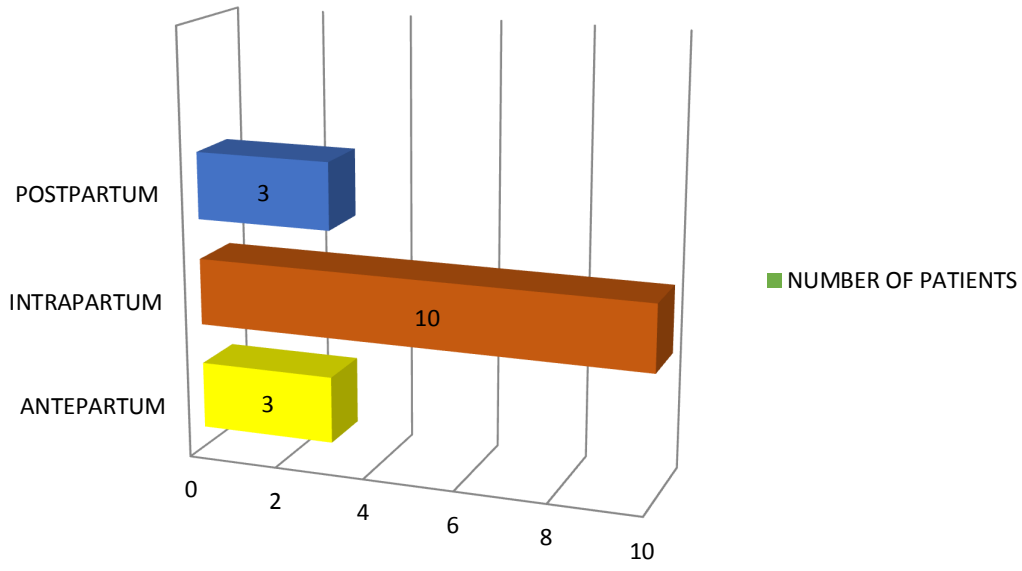
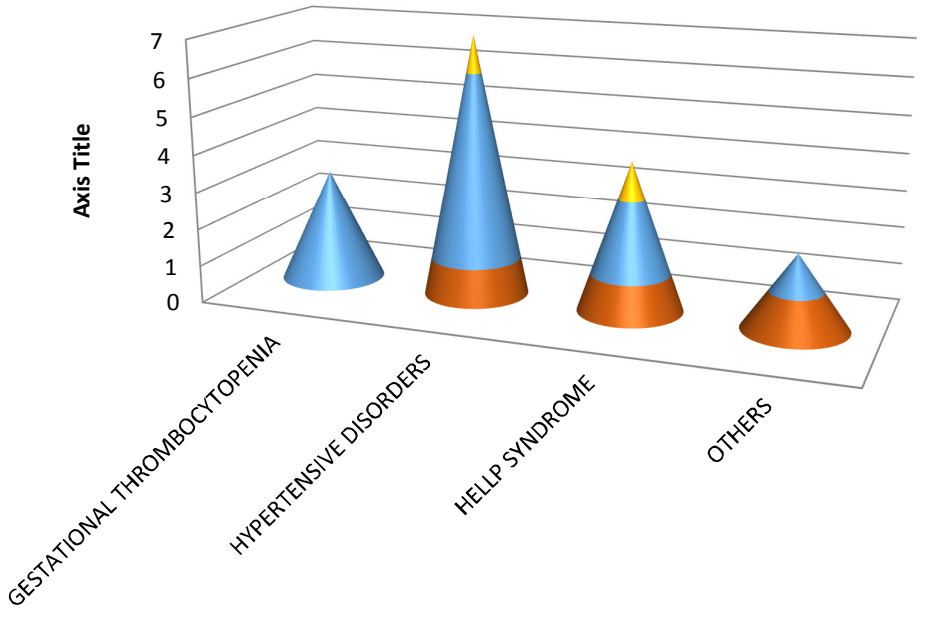


**TABLE 18: BLEEDING TENDENCIES ACCORDING TO ETIOLOGY:**

| Etiology | Ante-Partum | Intra-Partum Including PPH | Post Partum | Total |
|---|---|---|---|---|
| Gestational thrombocytopenia | 0 | 3 | 0 | 3 |
| Hypertensive disorders | 1 | 5 | 1 | 7 |
| HELLP syndrome | 1 | 2 | 1 | 4 |
| OTHERS | | | | |
| Pancytopenia | 1 | 0 | 0 | 1 |
| Dengue | 0 | 0 | 0 | 0 |
| Liver disease | 0 | 0 | 0 | 0 |
| ITP | 0 | 1 | 0 | 1 |

Of the total 191 patients with gestational thrombocytopenia, 3 patients had bleeding tendencies all of which occurred during the intrapartum period.Among the patients of hypertensive disorders, 1 patient had bleeding tendency in her antepartum period, 5 during their intrapartum period and 1 in her postpartum period.Among the patients with HELLP syndrome, 1 had bleeding tendency in her antepartum period, 2 in their intrapartum period and 1 in her postpartum period.Patients who had thrombocytopenia due to other causes developed bleeding tendencies- 1 in her antepartum period and 1 in her intrapartum period.

**FIGURE 16: BLEEDING TENDENCIES ACCORDING TO ETIOLOGY**




| | GESTATIONAL THROMBOCYTOPENIA | HYPERTENSIVE DISORDERS | HELLP SYNDROME | OTHERS |
|---|---|---|---|---|
| POSTPARTUM | 0 | 1 | 1 | 0 |
| INTRAPARTUM | 3 | 5 | 2 | 1 |
| ANTEPARTUM | 0 | 1 | 1 | 1 |

**BLEEDING MANIFESTATIONS:**

TABLE 19: BLEEDING MANIFESTATIONS ACCORDING TO ETIOLOGY

| | **Gestational Thrombocytopenia** | **Hypertensive Disorders** | **HELLP Syndrome** | **Others** |
|---|---|---|---|---|
| Hematuria | 0 | 1 | 1 | 0 |
| PPH | 3 | 4 | 2 | 0 |
| Vulval Hematoma | 0 | 1 | 0 | 0 |
| DIC | 0 | 1 | 1 | 0 |
| Bleeding Gums And Purpura | 0 | 0 | 0 | 1 |

Of the 191 gestational thrombocytopenia cases, 3 patients developed bleeding manifestations, all of which occurred during the intrapartum period. They presented as PPH.Among the patients with hypertensive disorders,1 patient had bleeding in her antepartum period in which she presented with haematuria, 1 in her post-partum period and she presented with features of DIC and 5 patients in their intrapartum period – 4 as PPH, and 1 patient presented with vulval haematoma. Among the 5 patients with HELLP syndrome, 4 patients had bleeding manifestations- 1 patient presented antenatally with haematuria, 2 patients had intrapartum bleeding presented with PPH and one with DIC.One of the patient with pancytopenia presented with gum bleeding and purpura in antenatal period and settled down with blood and blood products transfusion. One of the ITP patient presented with PPH which was managed medically

**BLOOD AND BLOOD PRODUCTS TRANSFUSION:**

TABLE 20: PATIENTS REQUIRING TRANSFUSION OF BLOOD/ BLOOD PRODUCTS

| Total Cases | No Transfusion | Blood Transfusion | | Blood Products Transfusion | |
|---|---|---|---|---|---|
| 273 | 248 | AP | IP/PP | AP | IP/PP |
| | | 5 | 12 | 1 | 7 |

Of the total 273 cases 6.2% (17) needed blood transfusion and 2.9% (8) needed transfusion of blood products.

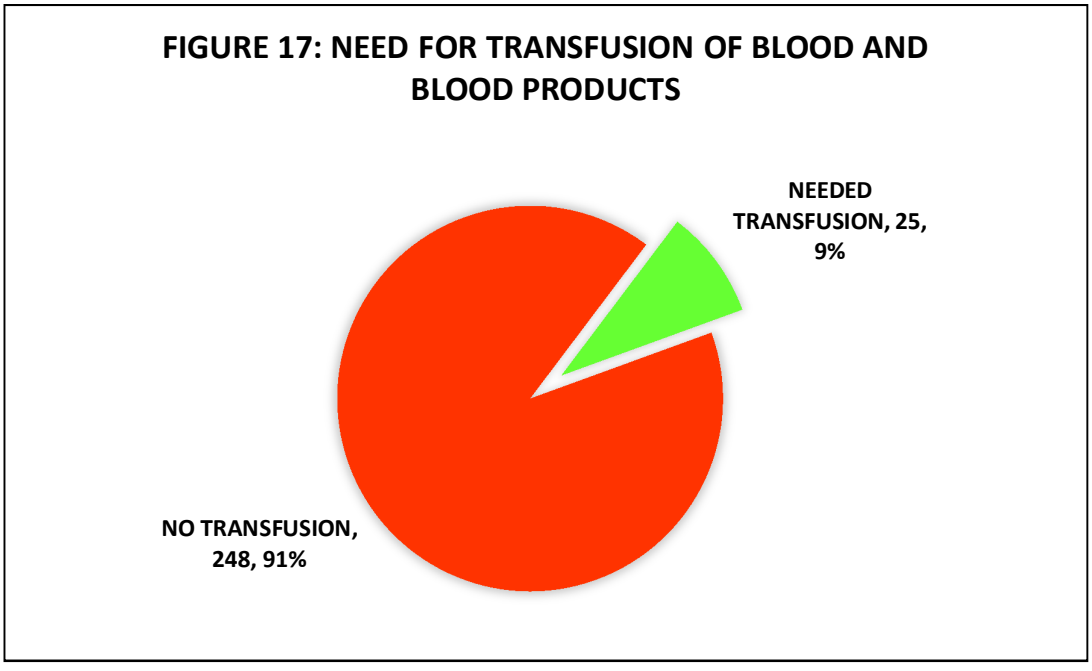
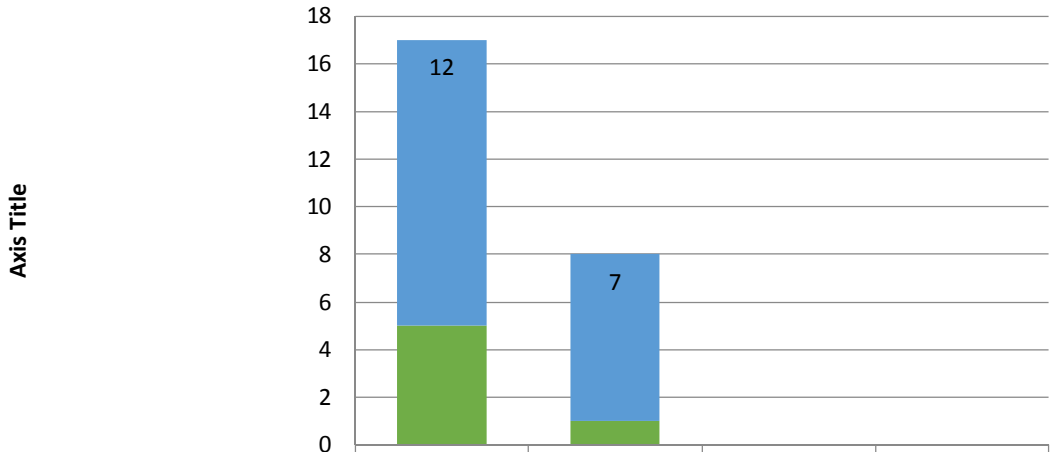


**FIGURE 18: NEED FOR TRANSFUSION ACCORDING TO THE TIME OF DELIVERY**



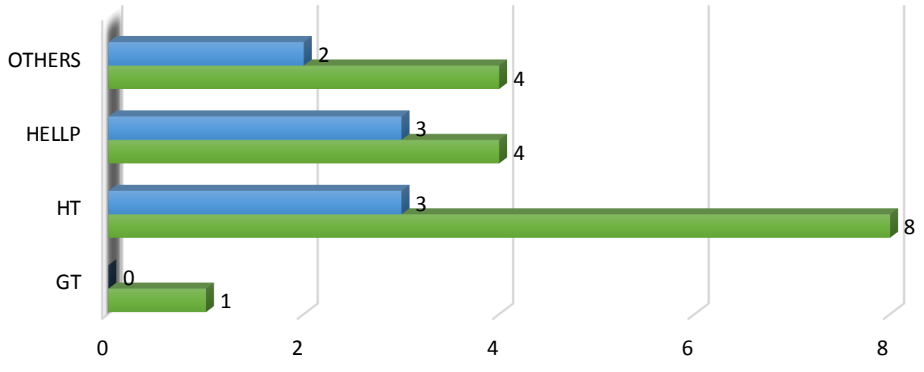

| | | | | |
|---|---|---|---|---|
| Column1 | | | | |
| INTRAPARTUM/POSTPARTUM | 12 | 7 | | |
| ANTEPARTUM | 5 | 1 | | |

**TABLE 21: BLOOD AND BLOOD PRODUCTS TRANSFUSION ACCORDING TO ETIOLOGY:**

| Etiology | Overall (N=273) | Blood | Blood Products |
|---|---|---|---|
| Gestational thrombocytopenia | 191 | 1 | 0 |
| Hypertensive disorders | 69 | 8 | 3 |
| HELLP | 5 | 4 | 3 |
| Pancytopenia | 2 | 2 | 1 |
| Dengue | 2 | 0 | 0 |
| AFLP | 1 | 1 | 0 |
| ITP | 3 | 1 | 1 |
| TOTAL | 273 | 17 | 8 |

1 patient with gestational thrombocytopenia(among 191 patients) received blood transfusion.Of the 69 patients with hypertensive disorders, 8 patients received blood transfusion and 3 patients received blood products transfusion.Among the 5 patients with HELLP syndrome, 4 patients received blood transfusion and 3 patients received blood products transfusion.Among the cases of thrombocytopenia due to other causes, 2 patients who had pancytopenia, 1 patient with AFLP and 1 patient with ITP received blood transfusion. 1 patient with pancytopenia received and 1 with ITP received platelet transfusion.3 patients who had HELLP syndrome and 1 patient with pancytopenia needed both blood and blood products transfusion.

**FIGURE 19: BLOOD AND PLATELET TRANSFUSION ACCORDING TO ETIOLOGY**




| | GT | HT | HELLP | OTHERS |
|---|---|---|---|---|
| Column1 | | | | |
| BLOOD PRODUCTS | 0 | 3 | 3 | 2 |
| BLOOD | 1 | 8 | 4 | 4 |

BLOOD PRODUCTS BLOOD

**FOETAL OUTCOME:**

TABLE 22: FETAL OUTCOME ACCORDING TO ETIOLOGY

| Etiology | No. of patients | Healthy | Mortality | Morbidity (NICU Admission) |
|---|---|---|---|---|
| Gestational thrombocytopenia | 191 | 170(89%) | 2(1%) | 19(9.9%) |
| Hypertensive disorders including HELLP | 74 | 40(56.5%) | 9(11%) | 25(31.8%) |
| Pancytopenia | 2 | 0 | 0 | 2(100%) |
| Dengue | 2 | 1 | 0 | 1(33%) |
| AFLP | 1 | 1 | 0 | 0 |
| ITP | 3 | 0 | 0 | 3(100%) |
| TOTAL | 273 | 212 | 11 | 50 |

Of the total 273 cases 77.65%(212) had healthy babies, perinatal mortality occured in 4.02%(11) of cases , and NICU admissions in 17.94%(49) of cases.

Among the babies born to mothers with gestational thrombocytopenia(n=191), 89% (n=170) were healthy,1%(n=2) had perinatal mortality, 9.9%(n=19) had admission to NICU.

56.5% babies of hypertensive mothers were healthy. 31.8% babies of hypertensive mothers had NICU admissions and this is statistically significant with p=0.0028. Perinatal mortality occurred in 11% (n=9) of babies.

In ITP and pancytopenia group, all babies needed admission and with this low number of cases statistical significance could not be calculated

**FIGURE 20: FETAL OUTCOME**

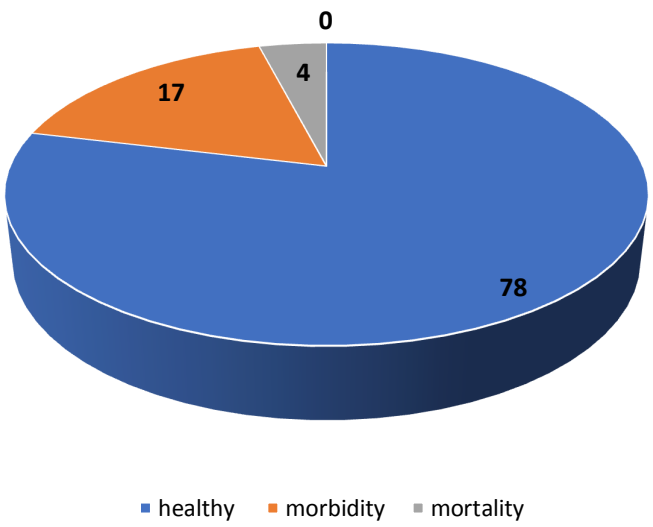



**FIGURE 21: FETAL OUTCOME ACCORDING TO ETIOLOGY**

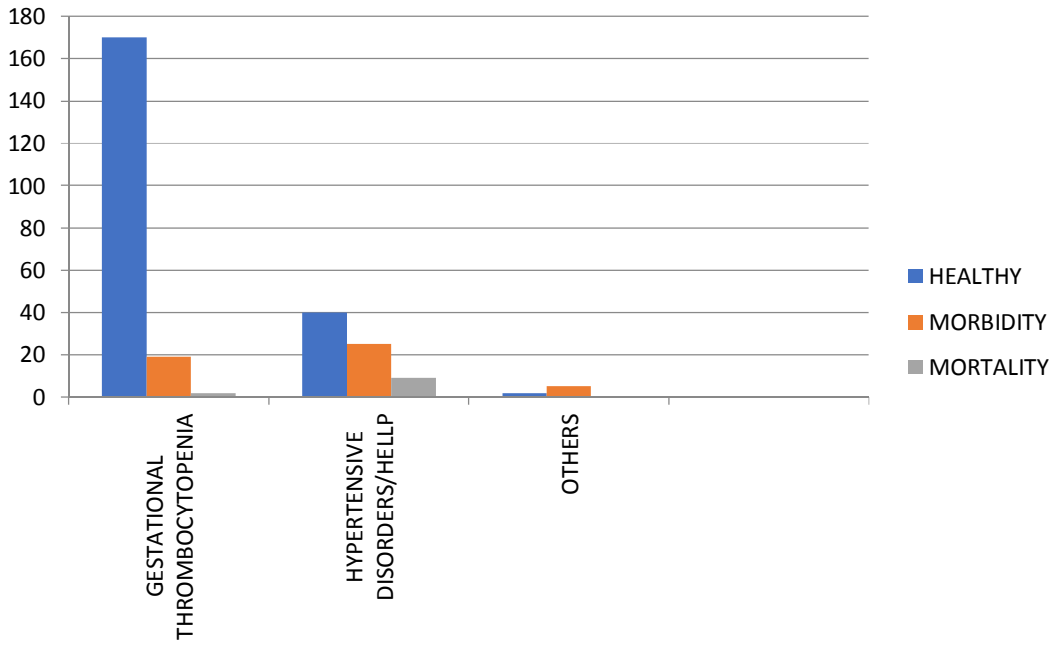


**TABLE 23 : NEONATAL THROMBOCYTOPENIA AND THE NEONATAL OUTCOME WITH ETIOLOGY:**

| Etiology | No. of patients | Newborn with thrombocytopenia | New Born with ICH/bleeding manifestations |
|---|---|---|---|
| Gestational thrombocytopenia | 191 | 0 | 0 |
| Hypertensive disorders including HELLP | 74 | 12(4.3%) | 2(0.73%) |
| Pancytopenia | 2 | 1(0.36%) | 0 |
| Dengue | 2 | 0 | 0 |
| AFLP | 1 | 0 | 0 |
| ITP | 3 | 3(1.09%) | 1(0.36) |
| TOTAL | 273 | 16(5.08%) | 3 |

Of the total 273 cases no thrombocytopenia mothers about 5.08% (16) newborn developed thrombocytopenia of which no cases associated with gestational thrombocytopenia ,and 4.3%(12) and (3)1.09% of the hypertensive disorder complicating pregnancy and ITP respectively associated with neonatal thrombocytopenia .with 3 cases presented with bleeding manifestations which includes 0.73%(2) in hypertensive cases associated with pulmonar hemorrhage and 0.36%(1) with Intra Cerebral Hemorrhage in ITP .

**POSTPARTUM FOLLOW UP:**

TABLE 24: NORMALISATION OF PLATELET COUNT

| **Etiology** | **Overall (N=273)** | **Immediate postpartum** | **Postpartum day 10** | **Thrombocytopenia in postpartum day 10** |
|---|---|---|---|---|
| Gestational thrombocytopenia | 191 | 78 | 191 | 0 |
| Hypertensive disorders | 69 | 12 | 68 | 0 |
| HELLP | 5 | 0 | 4 | 0 |
| Pancytopenia | 2 | 0 | 0 | 2 |
| Dengue | 2 | 0 | 2 | 0 |
| Liver disease | 1 | 0 | 1 | 0 |
| ITP | 3 | 0 | 1 | 2 |
| TOTAL | 271 | 90 | 267* | 4 |

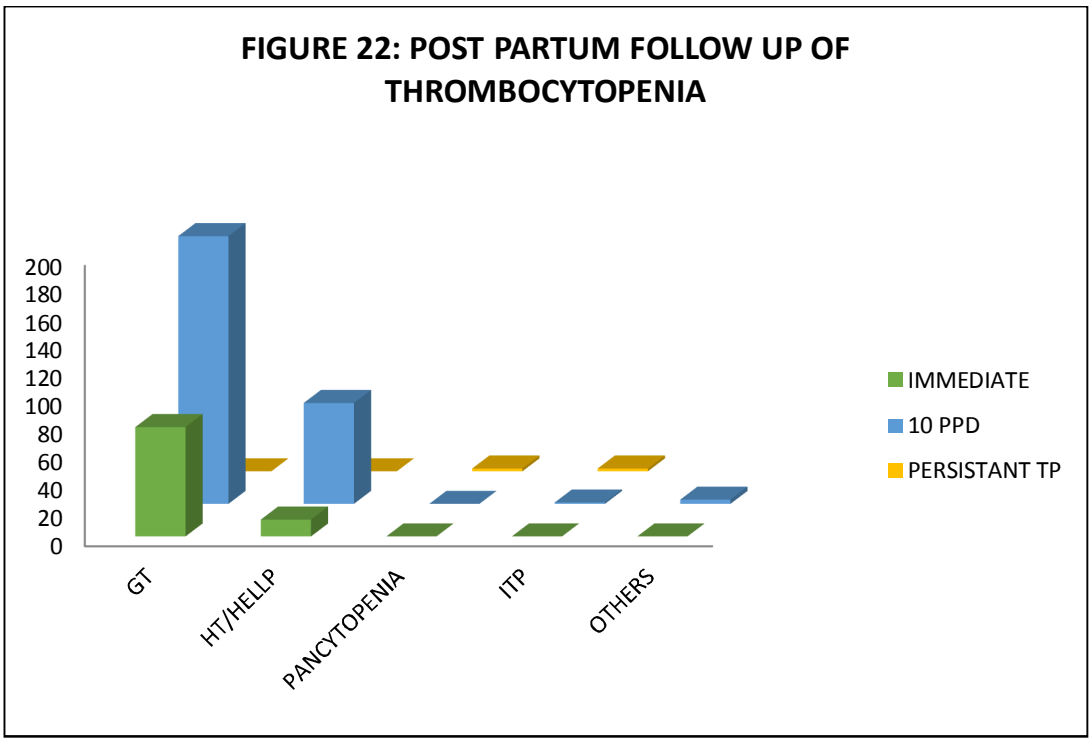
* Mortality occurred in 2 patients and hence could not be evaluated on the 10th postpartum day.

Among 191 patients with gestational hypertension, platelet counts returned to the normal range in the immediate postpartum period. By the 10th postpartum day all the 191 patients with gestational thrombocytopenia had platelet counts within normal range.

Among 69 patients with hypertensive disorders, 12 patients had normal platelet counts in the immediate postpartum period. 1 patient died in her 3rd postpartum day. All remaining 68 patients had platelet counts in the normal range by 10 days postpartum.

Among the patients with HELLP syndrome, 4 patients had normal platelet counts by 10th postpartum day. 1 patient died on the 2nd postpartum day.

Thrombocytopenia persisting after 10th postpartum day occurred in 4 patients – 2 patients with pancytopenia and 2 patients with ITP.




**MATERNAL MORTALITY:**

Of 273 cases of thrombocytopenia, maternal mortality occurred in two cases.. They were associated with severe pre eclampsia and HELLP syndrome.

1. P1L1/LSCS done/severe preeclampsia/HELLPwith MODS died on 3rd postpartum day. Her platelet count was 24,000/mm$^3$.
2. P2L2 /Emergency LSCS/Severe Pre eclampsia/MODS/AKIdied in 7th POD .her platelet count was 85,000/mm$^3$

# DISCUSSION

## PREVALENCE OF THROMBOCYTOPENIA:

In the present study, the prevalence of thrombocytopenia was 3.1%. This is consistent with the finding of 4.1% reported by Lin et al[116] in 2013. However, the prevalence is higher than the figure of 1.8% by Shital et al [117] and 1.9% by Brohi et al[118] in 2013. Sainio et al[110] reported a higher figure of 7.2%. Much higher figures were reported by Vijay et al[97] who reported 12.8% .

| Prevalence | My Study | Lin et al (consistent) | Brohi et al |
|---|---|---|---|
| | 3.1% | 4.1% | 1.9% |

## CAUSES OF THROMBOCYTOPENIA:

Our present study was aimed at investigating the causes of decreased platelet count and the obstetric outcome in various causes of thrombocytopenia. Of the total 273 cases studied, 69.9% of cases were found to be Gestational Thrombocytopenia, 25.27% were associated with hypertensive disorders of pregnancy and 1.83% associated with HELLP syndrome. Pancytopenia ,ITP ,Dengue ,AFLP contributed to 0.003%, 0.01%, 0.007%, 0.003% respectively.

In a study conducted by Wang et al[101] in 2016, the incidence of gestational thrombocytopenia was 60%, hypertensive disorders was 28.2%and other causes including ITP making 11.8%. The results in our study are comparable with this study.

In a study conducted by Sainio et al[110] in 2001, gestational thrombocytopenia was 81%, preeclampsia was 16%and ITP was 3%.This study included only term patients and this may be the reason for a slightly higher incidence of preeclampsia in our study.

In a study conducted by Michal Parnas et al[89] in their study of Moderate to severe thrombocytopenia during pregnancy comparing 199 pregnant women with moderate to severe thrombocytopenia (platelet count below 100×109/l) with 201 pregnant women without thrombocytopenia found that the main causes of thrombocytopenia were gestational thrombocytopenia (59.3%), immune thrombocytopenic purpura (11.05%), preeclampsia (10.05%), and HELLP (Hemolysis, elevated liver enzymes and low platelet count) syndrome (12.06%)(3).This study included only moderate and severe thrombocytopenia cases and hence there is an increase in incidence of HELLP syndrome and a decreased incidence of gestational thrombocytopenia.

In study conducted by Vanaja et al[111] at Gandhi medical college Secunderabad in 2017 – prevalence and characterization of thrombocytopenia in Indian women, gestational thrombocytopenia included 64%, hypertensive disorders making 21% and other disorders making 13% .

In a study of Prevalence of Thrombocytopenia during Pregnancy & Its Effect on Pregnancy & Neonatal Outcome by Monica Arora, Lajja Goyal in Guru Gobind Singh Medical College, Faridkot, Punjab[97] in 2016, Among 1450 deliveries, total 137

women were having thrombocytopenia in third trimester. The commonest etiology was gestational thrombocytopenia (61%). Thrombocytopenia due to severe preeclampsia and HELLP syndrome in this study was 24%.

Ajzenburg et al[113] assumed that gestational thrombocytopenia occurs due to increased platelet consumption within the placental circulation and/or normal inhibition of megakaryocytopoiesis. It followed a benign course without any adverse effect and need for intervention during pregnancy.

| | **My study** | **Wang et al** | **Saino et al** | **Monica arora** |
|---|---|---|---|---|
| GT | 69.9% | 60%(consistent) | 81% | 61%(consistent) |
| HYPERTESIVE DISODER | 25.27% | 28.2%(consistent) | 16% | 24%(consistent) |
| ITP | 1.83% | 11.8% | 3%(consistent) | |

**SEVERITY OF THROMBOCYTOPENIA:**

In our study, Mild thrombocytopenia was noted in 56.77 % of the total cases, moderate thrombocytopenia in 41.39% and severe thrombocytopenia in 1.83% of cases.

**TABLE 19: SEVERITY OF THROMBOCYTOPENIA IN DIFFERENT STUDIES**

| Study | Mild | Moderate | Severe |
|---|---|---|---|
| Present study | 56.67% | 41.39% | 1.83% |
| Vijay Zutshi et al | 62%(consistent) | 31% | 7% |
| Asrie et al | 74% | 20.7% | 5.3% |

In a prospective observational study done in the Department of Obstetrics and Gynaecology, VMMC and Safdarjung Hospital[96] in New Delhi, India, for a period of 6 months in 2008, On the basis of the platelet count of these women, they have found that 62% are having milder forms of thrombocytopenia, 31% are in moderate thrombocytopenia group, and 7% are with severe thrombocytopenia. There is a less incidence of severe thrombocytopenia in our study when compared to this study.

In a study conducted by Khatke et al in Mumbai at Sir J.J group of hospitals in 2014, 70.9%were with moderate thrombocytopenia and 29.1%were with severe thrombocytopenia. In our study mild thrombocytopenia was noted in 56.77 % of the total cases, moderate thrombocytopenia in 41.39% and severe thrombocytopenia in 1.83% of cases. This is because we have included mild thrombocytopenia cases in the study.

In a study conducted in Ethiopia Gondar university by Asrie et al[103] in 2014, 74% were mild thrombocytopenia, 20.7% were moderate, and 5.3% were severe thrombocytopenia. In our study the incidence of moderate thrombocytopenia was more when compared with this study.

## AGE OF THE PATIENTS AND GRAVIDA:

Asrie et al[103] conducted a study in Ethiopia in 2014 which reported 35% of study group were primigravida and 65% were multigravida. Most of the women in this study were between 30-35 years. In our study, 48.7% were primigravida and 52.3% were multigravida. Primigavida were more in our study when compared to this study and most of the cases falls under 25-30 years in our study .

In our study, age has no influence on the occurrence of thrombocytopenia.(p=0.698)

## SEVERITY OF CASES IN GESTATIONAL THROMBOCYTOPENIA:

In our study, among those with gestational thrombocytopenia, 60.7% had mild thrombocytopenia, 39.3% had moderate thrombocytopenia and no severe thrombocytopenia was reported. This shows that Gestational thrombocytopenia is usually of mild severity. This is comparable with Olayemi et al[93] study on Gestational thrombocytopenia among pregnant Ghanaian women in 2011 in which 65% had mild thrombocytopenia.

Boehlen et al[59] also reported that gestational thrombocytopenia is usually mild.

**SEVERITY IN CASES OF HYPERTENSIVE DISORDERS AND HELLP SYNDROME:**

In our study, of the total 74 hypertensives including 5 cases of HELLP syndrome cases studied, 52.7% had mild thrombocytopenia, 40.5% had moderate thrombocytopenia and 6.7% had severe thrombocytopenia. Of this HELLP syndrome compromises the 6.7% cases of severe thrombocytopenia. The results are comparable with the study conducted by Rupakala et al[102] on thrombocytopenia in hypertensive disorders in pregnancy in which severe thrombocytopenia is seen in 5.8%, moderate thrombocytopenia in 35.5% and mild thrombocytopenia in 58.7%. Incidence of HELLP in this study was 6.6% which is also comparable with our study.

Ozdemir et al's[94]study on a comparison between thrombocytopenia in hypertensive disorders and other causes shows the incidence of thrombocytopenia in hypertensives were 24.2% and severe thrombocytopenia was seen in 8.1% of cases.

Magann et al's[107] study on thrombocytopenia in pregnancy reported that severe thrombocytopenia is seen in 12% of HELLP syndrome and 30% of eclampsia cases and 18% of severe preeclampsia.

**MODE OF DELIVERY:**

In our study 56.38% patients delivered vaginally, out of which 11 patients had instrumental delivery. LSCS had been done for obstetric indications. Among the cases of severe thrombocytopenia, 2 delivered normally vaginally and 3 patients underwent

CS. Our study results are comparable with Ozdemir et al's[94] study in which vaginal delivery rate is 56.3%.

In a study at VMMC and Safdarjung Hospital[96] in New Delhi, India it has been found that around 94% patients delivered vaginally; among these, 9 patients had instrumental delivery, In our study 56.4%of patients delivered vaginally which is lower when compared to this study.

In a study of thrombocytopenia in pregnancy induced hypertension by Manjula et al in 2005, the LSCS rate among thrombocytopenic mothers with hypertension was 59% and is slightly higher when compared with the LSCS rate of 52.7% among hypertensive thrombocytopenic mothers in our study.

ACOG recommends that the definitive treatment of maternal thrombocytopenia in the setting of PIH with HELLP syndrome is termination of pregnancy.

**BLEEDING MANIFESTATIONS:**

In our study,6.2% developed bleeding manifestations in their course of stay, of which 17.6% occurred in antepartum period , 64.7% in intrapartum period, and 17.6% in post-partum period. In our study PPH occurred in 3.2% cases, placental abruption in 1.09% cases.

In a study of Prevalence of Thrombocytopenia during Pregnancy & Its Effect on Pregnancy & Neonatal Outcome conducted by Monica Arora, Lajja Goyal in Guru Gobind Singh Medical College, Faridkot, Punjab[97] in 2016 , Placental abruption-

6.6%, PPH -4.3%, Wound hematoma- 3.6% were noted. When compared with this study, the occurrence of complications was less in our study.

In a retrospective study by Shin et al[109] it was inferred that in pregnant women with aplastic anemia, obstetric and disease complications are more prevalent in those with severe thrombocytopenia than in those with nonsevere thrombocytopenia. In our study only 2 cases of pancytopenia were studied and hence comparison could not be made. Large population is needed to compare the results.

Platelet transfusions are less effective in women with PIH and HELLP syndrome because of accelerated platelet destruction. Two situations in which additional platelets should be given are:

• To treat severe thrombocytopenia with ongoing bleeding, and

• To increase the platelet count to more than 50,000/L for an operative delivery[114].

**FETAL OUTCOME:**

In our study of the total 273 cases 77.65% were healthy babies, mortality occured in 4.02% of cases, and NICU admissions 17.94% cases.

In a systematic review and meta-analysis by Mohammad Mohseni et al[110] on thrombocytopenia among pregnant women, it was noted that the neonatal mortality and morbidity rate was 0.30% - 13.20%. The rate in our study is higher when compared to this meta-analysis.

**NORMALISATION OF PLATELET COUNTS:**

In our study, of the total 273 cases of thrombocytopenia in 98.89% cases the platelet count returned to normal by 10th postnatal day and cases of 1.47% had persistent thrombocytopenia. This was noted in 2 cases of pancytopenia and 2 cases of ITP. This is comparable with Ruggeri et al's [95] study on gestational thrombocytopenia in which all the cases of gestational thrombocytopenia had normal platelet count after 6 weeks. This is also comparable with Vijay Zutshi et al[96] 2019 study in which all the cases with gestational thrombocytopenia the platelet count returned to normal in the postpartum period.

The platelet count usually begins returning to normal within 72 hours of delivery

## SUMMARY

- Prevalance of thrombocytopenia was 3.1% in third trimester, with no influence on age and gravida .The distribution of thrombocytopenia with etiology is 69.9% with gestational thrombocytopenia,25.25% with hypertensive disorder and 1.83% with HELLP and 0.01% with ITP and 0.007% in pancytopenia and dengue .
- The gestational thrombocytopenia mostly of mild variety and predominantly in latter third trimester, thrombocytopenia in hypertensive disorder and HELLP is mild to moderate type, which was predominantly in early third trimester.
- There was no influence of thrombocytopenia In the mode of delivery. anaesthesia preferred in thrombocytopenia was general anaesthesia but nearly 96% was done under regional Anesthesia preferably spinal anesthesia. Severe thrombocytopenia were done surgery under General anesthesia.
- Bleeding manifestations occurred in about 6.2%(16) of the study population and majority during the intrapartum period mainly the PPH.Blood and blood products transfused for 17 cases ostly associated with HELLP and ITP,there was no transfusion needed in study population of gestational thrombocytopenia.
- Fetal outcome in study population with mortality rate is 4.02%(11) majority in cases in hypertensive disorders associated was 17%(49) cases neonatal thrombocytopenia was associated in al cases of ITP and 12 cases of hypertensive disorder and HELLP ,where the platelet count resumed normal in the babies which was associated with other causes than ITP.

- Normalisation of platelet count occurred in 267 cases of the study population within 10 days post partum period.

# CONCLUSION

The prevalence of thrombocytopenia in pregnancy is 4.1% in third trimester in patient attending RMH. The most common cause of thrombocytopenia being Gestational Thrombocytopenia and next being hypertensive disease complicating pregnancy. Thrombocytopenia has no influence on age, parity ,gestational age,mode of delivery.

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| S.NO | AGE | gravida | PARA | LIVE | ABORTION | GA | PLATELET COUNT | BLEEDING MANIFESTATION | PLATELET TRANSUSION | BLOOD TRANSFUSION | CO MORBIDITIES | DIAGNOSIS | MODE OF DELIVERY | NB WT | NB STATUSADMISSION | NB STATUS | LENGTH OF STAY | TREATMENT | MATERNAL DEATH |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 21 | 1 | 0 | 0 | 0 | 38 | 100000 | 0 | 0 | 0 | E PRE ECLA | SP | LN | 2.7 | 0 | 1 | 7 DAYS | ANHT | 0 |
| 2 | 30 | 1 | 0 | 0 | 0 | 38 | 120000 | 1 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 3 | 26 | 1 | 0 | 0 | 0 | 39 | 110000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |
| 4 | 25 | 2 | 1 | 1 | 0 | 28 | 86000 | 0 | 0 | 0 | SP | SP | LN | 1.1 | 1 | 0 | 7 | ANHT | 0 |
| 5 | 27 | 1 | 0 | 0 | 0 | 40 | 120000 | 0 | 0 | 0 | | GT | LN | 2.2 | 0 | 1 | 3 | | 0 |
| 6 | 25 | 2 | 1 | 1 | 0 | 37 | 120000 | 0 | 0 | 0 | | GT | LN | 2.2 | 0 | 1 | 3 | | 0 |
| 7 | 18 | 1 | 0 | 0 | 0 | 37 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.8 | 0 | 1 | 5 | | 0 |
| 8 | 26 | 3 | 0 | 0 | 3 | 36 | 120000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 9 | 23 | 1 | 0 | 0 | 0 | 38 | 120000 | 0 | 0 | 0 | SP | SP | LSCS | 2.8 | 0 | 1 | 7 | | 0 |
| 10 | 26 | 3 | 1 | 1 | 1 | 34 | 84000 | 0 | 0 | 1 | SP | SP | LN | 2.3 | 1 | 0 | 12 | ANHT | 0 |
| 11 | 22 | 3 | 2 | 2 | 0 | 36 | 98000 | 0 | 0 | 0 | FEVER | DENGUE | LN | 2.6 | 0 | 1 | 21 | | 0 |
| 12 | 26 | 2 | 1 | 1 | 0 | 38 | 110000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 13 | 31 | 1 | 0 | 0 | 0 | 38 | 110000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 14 | 25 | 2 | 0 | 0 | 1 | 36 | 140000 | 0 | 0 | 0 | SP | SP | LSCS | 2.6 | 0 | 1 | 7 | ANHT | 0 |
| 15 | 28 | 1 | 0 | 0 | 0 | 39 | 110000 | 0 | 0 | 0 | | GT | LN | 2.3 | 0 | 1 | 3 | | 0 |
| 16 | 26 | 1 | 0 | 0 | 0 | 40 | 115000 | 1 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 17 | 27 | 3 | 1 | 1 | 1 | 37 | 110000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 18 | 27 | 2 | 1 | 1 | 0 | 36 | 106000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 19 | 21 | 2 | 1 | 0 | 0 | 36 | 94000 | 0 | 0 | 1 | | PANCYTOPEN | VBAC | 2.2 | 0 | 1 | 7 | | 0 |
| 20 | 26 | 2 | 1 | 1 | 0 | 37 | 92000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 3 | | 0 |
| 21 | 25 | 1 | 0 | 0 | 0 | 38 | 100000 | 0 | 0 | 0 | SP | SP | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 22 | 32 | 1 | 0 | 0 | 0 | 38 | 97000 | 0 | 0 | 0 | | GT | LSCS | 2.8 | 1 | 0 | 5 | | 0 |
| 23 | 33 | 1 | 0 | 0 | 0 | 34 | 130000 | 0 | 0 | 0 | SP | SP | LN | 2.2 | 1 | 0 | 12 | ANHT | 0 |
| 24 | 28 | 2 | 1 | 1 | 0 | 38 | 77000 | 0 | 0 | 0 | | GT | LSCS | 2.1 | 0 | 1 | 5 | | 0 |
| 25 | 27 | 1 | 0 | 0 | 0 | 39 | 107000 | 0 | 0 | 0 | | GT | LN | 2.4 | 0 | 1 | 3 | | 0 |
| 26 | 29 | 1 | 0 | 0 | 0 | 37 | 116000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 27 | 32 | 1 | 0 | 0 | 0 | 38 | 97000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 28 | 28 | 3 | 2 | 2 | 0 | 38 | 101000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 29 | 18 | 1 | 0 | 0 | 0 | 39 | 108000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 3 | | 0 |
| 30 | 28 | 1 | 0 | 0 | 0 | 39 | 109000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |
| 31 | 30 | 3 | 1 | 1 | 1 | 34 | 110000 | 0 | 0 | 0 | SP | SP | LSCS | 2.1 | 1 | 0 | 7 | ANHT | 0 |
| 32 | 32 | 1 | 0 | 0 | 0 | 38 | 110000 | 1 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 33 | 31 | 2 | 1 | 1 | 0 | 34 | 130000 | 0 | 0 | 0 | SP | SP | LN | 1.9 | 1 | 0 | 21 | ANHT | 0 |
| 34 | 28 | 2 | 1 | 1 | 0 | 34 | 101000 | 0 | 0 | 0 | | GT | LN | 1.8 | 0 | 1 | 12 | | 0 |
| 35 | 31 | 3 | 0 | 0 | 2 | 40 | 96000 | 0 | 0 | 0 | | GT | LSCS | 2.4 | 0 | 1 | 5 | | 0 |
| 36 | 19 | 1 | 0 | 0 | 0 | 38 | 125000 | 0 | 0 | 0 | SP | SP | LSCS | 2.75 | 0 | 1 | 5 | ANHT | 0 |
| 37 | 31 | 1 | 0 | 0 | 0 | 37 | 90000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 38 | 19 | 1 | 0 | 0 | 0 | 38 | 107000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |
| 39 | 33 | 1 | 0 | 0 | 0 | 28 | 100000 | 0 | 0 | 0 | SP | SP | LN | 900 | 1 | 0 | 5 | ANHT | 0 |
| 40 | 29 | 2 | 1 | 1 | 0 | 34 | 101000 | 0 | 0 | 0 | | GT | LN | 1.8 | 0 | 1 | 12 | | 0 |
| 41 | 26 | 1 | 0 | 0 | 0 | 38 | 90000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 42 | 29 | 2 | 1 | 1 | 0 | 39 | 110000 | 0 | 0 | 0 | | GT | LN | 3.1 | 0 | 1 | 3 | | 0 |
| 43 | 26 | 3 | 2 | 2 | 0 | 39 | 76000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 44 | 28 | 3 | 0 | 0 | 2 | 36 | 80000 | 0 | 0 | 0 | | GT | LSCS | 2.8 | 0 | 1 | 5 | | 0 |
| 45 | 27 | 1 | 0 | 0 | 0 | 40 | 91000 | 0 | 0 | 0 | HD | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 46 | 21 | 1 | 0 | 0 | 0 | 39 | 98000 | 0 | 0 | 0 | SP | SP | LN | 2.7 | 0 | 1 | 7 | ANHT | 0 |
| 47 | 26 | 2 | 1 | 1 | 0 | 38 | 107000 | 0 | 0 | 0 | | GT | LN | 3.1 | 0 | 1 | 3 | | 0 |
| 48 | 27 | 3 | 2 | 2 | 0 | 40 | 102000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |
| 49 | 23 | 1 | 0 | 0 | 0 | 38 | 110000 | 0 | 0 | 0 | SP | ECLAPSIA | LSCS | 2.6 | 0 | 1 | 14 | ANHT | 0 |
| 50 | 26 | 3 | 2 | 2 | 0 | 37 | 103000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 51 | 29 | 2 | 1 | 1 | 0 | 39 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.4 | 0 | 1 | 5 | | 0 |
| 52 | 29 | 3 | 2 | 2 | 0 | 39 | 96000 | 0 | 0 | 0 | | GT | LN | IUD | 0 | 1 | 5 | | 0 |
| 53 | 18 | 1 | 0 | 0 | 0 | 39 | 92000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 54 | 21 | 1 | 0 | 0 | 0 | 37 | 95000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 55 | 32 | 2 | 1 | 1 | 0 | 34 | 127000 | 0 | 0 | 0 | SP | SP | LN | 2.1 | 0 | 1 | 7 | ANHT | 0 |
| 56 | 28 | 2 | 1 | 1 | 0 | 40 | 98000 | 0 | 0 | 0 | | GT | LSCS | 2.5 | 0 | 1 | 5 | | 0 |
| 57 | 29 | 4 | 0 | 0 | 3 | 38 | 112000 | 0 | 0 | 0 | | GT | LSCS | 2.8 | 0 | 1 | 5 | | 0 |
| 58 | 22 | 1 | 0 | 0 | 0 | 37 | 96000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 59 | 32 | 4 | 2 | 2 | 1 | 40 | 108000 | 0 | 0 | 0 | | GT | LSCS | 3 | 0 | 1 | 5 | | 0 |
| 60 | 27 | 2 | 1 | 1 | 0 | 34 | 130000 | 0 | 0 | 0 | SP | SP | LSCS | 2.2 | 1 | 0 | 14 | ANHT | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 61 | 19 | 1 | 0 | 0 | 0 | 36 | 86000 | 0 | 0 | 0 | SP | SP | LSCS | 2.4 | 0 | 1 | 7 | ANHT | 0 |
| 62 | 27 | 2 | 1 | 1 | 0 | 40 | 110000 | 0 | 0 | 0 | | GT | LN | 3.2 | 0 | 1 | 3 | | 0 |
| 63 | 34 | 3 | 0 | 0 | 2 | 37 | 110000 | 0 | 0 | 0 | | GT | LSCS | 3.2 | 0 | 1 | 5 | | 0 |
| 64 | 26 | 2 | 1 | 1 | 0 | 39 | 130000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |
| 65 | 22 | 1 | 0 | 0 | 0 | 39 | 92000 | 0 | 0 | 0 | SP | SP | LSCS | 2.7 | 0 | 1 | 7 | ANHT | 0 |
| 66 | 37 | 3 | 2 | 2 | 0 | 38 | 116000 | 0 | 0 | 0 | | GT | LSCS | 3.3 | 0 | 1 | 5 | | 0 |
| 67 | 26 | 3 | 0 | 0 | 2 | 34 | 120000 | 1 | 0 | 0 | SP | SP | LN | 2.3 | 1 | 0 | 17 | ANHT | 0 |
| 68 | 21 | 1 | 0 | 0 | 0 | 39 | 97500 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 69 | 28 | 2 | 1 | 1 | 0 | 41 | 120000 | 0 | 0 | 0 | | GT | LN | 3.4 | 0 | 1 | 3 | | 0 |
| 70 | 31 | 1 | 0 | 0 | 0 | 39 | 97000 | 0 | 0 | 0 | | GT | LN | 2.2 | 0 | 1 | 5 | | 0 |
| 71 | 25 | 2 | 1 | 1 | 0 | 32 | 120000 | 0 | 0 | 0 | SP | SP | LSCS | 1.7 | 1 | 0 | 14 | ANHT | 0 |
| 72 | 24 | 1 | 0 | 0 | 0 | 36 | 126000 | 0 | 0 | 0 | SP | SP | LSCS | 2.6 | 0 | 1 | 7 | ANHT | 0 |
| 73 | 27 | 2 | 1 | 1 | 0 | 32 | 130000 | 0 | 0 | 0 | | GT | LN | 1.1 | 0 | 1 | 14 | | 0 |
| 74 | 36 | 3 | 2 | 2 | 0 | 36 | 92000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 75 | 27 | 2 | 1 | 1 | 0 | 38 | 112000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 76 | 22 | 1 | 0 | 0 | 0 | 40 | 85000 | 0 | 0 | 0 | | GT | LN | 2.3 | 0 | 1 | 3 | | 0 |
| 77 | 32 | 3 | 2 | 2 | 0 | 32 | 35000 | 1 | 4 | 3 | HELLP | SP | LN | 1.7 | 1 | 0 | 14 | ANHT | 0 |
| 78 | 26 | 2 | 1 | 1 | 0 | 37 | 110000 | 0 | 0 | 0 | SP | SP | LSCS | 2.5 | 0 | 1 | 5 | ANHT | 0 |
| 79 | 31 | 3 | 0 | 0 | 2 | 37 | 102000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 80 | 27 | 1 | 0 | 0 | 0 | 37 | 110000 | 0 | 0 | 0 | | GT | LN | 2.4 | 0 | 1 | 3 | | 0 |
| 81 | 30 | 4 | 0 | 0 | 3 | 34 | 180000 | 0 | 0 | 0 | | GT | LN | 1.8 | 0 | 1 | 10 | | 0 |
| 82 | 31 | 2 | 0 | 0 | 1 | 38 | 100000 | 0 | 0 | 0 | SP | SP | LN | 2.6 | 0 | 1 | 7 | ANHT | 0 |
| 83 | 39 | 4 | 3 | 3 | 0 | 32 | 100000 | 0 | 0 | 0 | GDM | GT | LN | 1.3 | 0 | 1 | 5 | | 0 |
| 84 | 26 | 2 | 1 | 1 | 0 | 40+2 | 81000 | 0 | 0 | 0 | | GT | LN | 3.1 | 0 | 1 | 3 | | 0 |
| 85 | 23 | 1 | 0 | 0 | 0 | 36 | 130000 | 0 | 0 | 0 | SP | SP | LSCS | 2.5 | 0 | 1 | 5 | ANHT | 0 |
| 86 | 28 | 4 | 0 | 0 | 3 | 37 | 110000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 87 | 34 | 5 | 4 | 4 | 0 | 26 | 100000 | 0 | 0 | 0 | SP | SP | LN | 800 | 1 | 0 | 7 | ANHT | 0 |
| 88 | 28 | 2 | 1 | 1 | 0 | 30 | 110000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 89 | 27 | 1 | 0 | 0 | 0 | 38 | 120000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 90 | 19 | 1 | 0 | 0 | 0 | 38 | 140000 | 1 | 0 | 0 | SP | SP | LN | 2.6 | 0 | 1 | 5 | ANHT | 0 |
| 91 | 26 | 2 | 1 | 1 | 0 | 40 | 97000 | 0 | 0 | 0 | | GT | LSCS | 1.9 | 0 | 1 | 5 | | 0 |
| 92 | 26 | 1 | 0 | 0 | 0 | 39 | 94000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 93 | 24 | 1 | 0 | 0 | 0 | 40 | 130000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 5 | | 0 |
| 94 | 26 | 2 | 1 | 1 | 0 | 32 | 99000 | 0 | 0 | 0 | SP | SP | LSCS | 1.8 | 0 | 1 | 12 | ANHT | 0 |
| 95 | 23 | 4 | 0 | 0 | 3 | 37 | 140000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |
| 96 | 26 | 1 | 0 | 0 | 0 | 36 | 90000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 97 | 31 | 3 | 1 | 1 | 1 | 30 | 100000 | 0 | 0 | 0 | SP | SP | LN | 1.4 | 1 | 0 | 11 | ANHT | 0 |
| 98 | 26 | 2 | 1 | 1 | 0 | 38 | 103000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 99 | 24 | 4 | 0 | 0 | 3 | 38 | 108000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |
| 100 | 22 | 1 | 0 | 0 | 0 | 36 | 98000 | 0 | 0 | 0 | SP | SP | LSCS | 2.6 | 0 | 1 | 7 | ANHT | 0 |
| 101 | 29 | 2 | 1 | 1 | 0 | 39 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 102 | 25 | 2 | 1 | 1 | 0 | 34 | 130000 | 0 | 0 | 0 | SP | ECLAP | LSCS | 2.2 | 1 | 0 | 14 | ANHT | 0 |
| 103 | 25 | 1 | 0 | 0 | 0 | 37 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.4 | 0 | 1 | 3 | | 0 |
| 104 | 24 | 1 | 0 | 0 | 0 | 38 | 110000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 105 | 29 | 2 | 1 | 1 | 0 | 38 | 103000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 106 | 31 | 2 | 1 | 1 | 0 | 37 | 120000 | 0 | 0 | 0 | | GT | LN | 2.4 | 0 | 1 | 4 | | 0 |
| 107 | 27 | 3 | 0 | 0 | 2 | 34 | 142000 | 1 | 0 | 0 | SP | SP | LN | 2.1 | 1 | 0 | 7 | ANHT | 0 |
| 108 | 21 | 1 | 0 | 0 | 0 | 36 | 96000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 109 | 27 | 2 | 1 | 1 | 0 | 36 | 86000 | 0 | 0 | 0 | | GT | LSCS | 2.2 | 0 | 1 | 5 | | 0 |
| 110 | 24 | 2 | 0 | 0 | 1 | 38 | 98000 | 0 | 0 | 0 | SP | SP | LN | 2.7 | 0 | 1 | 7 | ANHT | 0 |
| 111 | 32 | 2 | 1 | 1 | 0 | 38 | 110000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 5 | | 0 |
| 112 | 18 | 1 | 0 | 0 | 0 | 34 | 84000 | 0 | 0 | 0 | | GT | LN | 2.1 | 0 | 1 | 3 | | 0 |
| 113 | 21 | 1 | 0 | 0 | 0 | 39 | 110000 | 0 | 0 | 0 | SP | SP | LN | 2.8 | 0 | 1 | 5 | ANHT | 0 |
| 114 | 26 | 2 | 1 | 1 | 0 | 34 | 96000 | 0 | 0 | 0 | | GT | LSCS | 1.9 | 0 | 1 | 5 | | 0 |
| 115 | 34 | 3 | 2 | 2 | 0 | 37 | 26000 | 0 | 8 | 4 | HELLP | SP | LSCS | 2 | 1 | 0 | 7 | ANHT | 0 |
| 116 | 34 | 2 | 1 | 1 | 0 | 39 | 102000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 5 | | 0 |
| 117 | 28 | 1 | 0 | 0 | 0 | 40 | 86000 | 0 | 0 | 0 | | GT | LN | 3.1 | 0 | 1 | 3 | | 0 |
| 118 | 25 | 1 | 0 | 0 | 0 | 38 | 38000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 119 | 26 | 2 | 1 | 1 | 0 | 39 | 97000 | 0 | 0 | 0 | | GT | LSCS | 2.8 | 0 | 1 | 5 | | 0 |
| 120 | 25 | 2 | 0 | 0 | 1 | 34 | 84000 | 1 | 0 | 0 | SP | SP | LN | 2.1 | 1 | 0 | 19 | ANHT | 0 |
| 121 | 27 | 2 | 1 | 1 | 0 | 37 | 96000 | 1 | 0 | 0 | SP | SP | LSCS | 2.6 | 0 | 1 | 7 | ANHT | 0 |
| 122 | 32 | 1 | 0 | 0 | 0 | 39 | 131000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 123 | 33 | 2 | 1 | 1 | 0 | 40 | 107000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 10 | | 0 |
| 124 | 19 | 1 | 0 | 0 | 0 | 40+3 | 112000 | 0 | 0 | 0 | | GT | LSCS | 3 | 0 | 1 | 5 | | 0 |

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| 125 | 28 | 2 | 1 | 1 | 0 | 38 | 95000 | 0 | 0 | 0 | | GT | LSCS | 2.4 | 0 | 1 | 5 | | 0 |
| 126 | 23 | 1 | 0 | 0 | 0 | 38 | 125000 | 0 | 0 | 0 | SP | SP | LN | 2.5 | 0 | 1 | 5 | ANHT | 0 |
| 127 | 32 | 2 | 1 | 1 | 0 | 28 | 128000 | 0 | 0 | 0 | SP | SP | LN | 1 | 0 | 1 | 7 | | 0 |
| 128 | 26 | 3 | 1 | 1 | 1 | 28 | 94000 | 0 | 0 | 0 | SP | SP | LSCS | 980 | 1 | 0 | 10 | ANHT | 0 |
| 129 | 26 | 1 | 0 | 0 | 0 | 38 | 86000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 130 | 31 | 2 | 1 | 1 | 0 | 36 | 106000 | 0 | 0 | 0 | | GT | LSCS | 2.4 | 0 | 1 | 5 | | 0 |
| 131 | 30 | 1 | 0 | 0 | 0 | 37 | 90000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 132 | 31 | 2 | 1 | 1 | 0 | 36 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.3 | 0 | 1 | 5 | | 0 |
| 133 | 26 | 1 | 0 | 0 | 0 | 38 | 84000 | 0 | 0 | 0 | SP | SP | LSCS | 2.4 | 0 | 1 | 7 | ANHT | 0 |
| 134 | 34 | 2 | 1 | 1 | 0 | 38 | 130000 | 0 | 0 | 0 | GDM | GT | LSCS | 2.8 | 0 | 1 | 5 | | 0 |
| 135 | 24 | 1 | 0 | 0 | 0 | 36 | 94000 | 0 | 0 | 0 | SP | SP | LN | 2.5 | 0 | 1 | 7 | ANHT | 0 |
| 136 | 26 | 2 | 1 | 1 | 0 | 39 | 120000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 137 | 18 | 1 | 0 | 0 | 0 | 39 | 77000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 138 | 27 | 2 | 1 | 1 | 0 | 40 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 139 | 26 | 2 | 1 | 1 | 0 | 40+2 | 96000 | 0 | 0 | 0 | | GT | LSCS | 3.1 | 0 | 1 | 5 | | 0 |
| 140 | 27 | 1 | 0 | 0 | 0 | 40 | 84000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 3 | | 0 |
| 141 | 29 | 2 | 1 | 1 | 0 | 38 | 97000 | 0 | 0 | 0 | | GT | LSCS | 2.4 | 0 | 1 | 5 | | 0 |
| 142 | 22 | 1 | 0 | 0 | 0 | 40+3 | 88000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 143 | 35 | 1 | 0 | 0 | 0 | 37 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 144 | 21 | 1 | 0 | 0 | 0 | 38 | 92000 | 0 | 0 | 0 | SP | SP | LN | 2.7 | 0 | 1 | 5 | ANHT | 0 |
| 145 | 27 | 2 | 1 | 1 | 0 | 37 | 94000 | 0 | 0 | 0 | | GT | LSCS | 2.5 | 0 | 1 | 6 | | 0 |
| 146 | 23 | 1 | 0 | 0 | 0 | 38 | 86000 | 0 | 0 | 0 | | GT | LSCS | 2.1 | 0 | 1 | 5 | | 0 |
| 147 | 28 | 2 | 1 | 1 | 0 | 39 | 95000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 7 | | 0 |
| 148 | 26 | 3 | 2 | 2 | 0 | 39 | 75000 | 0 | 0 | 0 | PANCYTO | | LN | 2.7 | 0 | 1 | 14 | STEROID | 0 |
| 149 | 22 | 1 | 0 | 0 | 0 | 39 | 77000 | 0 | 0 | 0 | | GT | LSCS | 1.9 | 0 | 1 | 15 | | 0 |
| 150 | 21 | 1 | 0 | 0 | 0 | 40 | 120000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 151 | 31 | 1 | 0 | 0 | 0 | 38 | 117000 | 0 | 0 | 0 | HD | GT | LSCS | 2.6 | 1 | 0 | 5 | | 0 |
| 152 | 22 | 2 | 1 | 1 | 0 | 36 | 96000 | 0 | 0 | 0 | | GT | LSCS | 2.3 | 0 | 1 | 10 | | 0 |
| 153 | 23 | 1 | 0 | 0 | 0 | 37 | 111000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 154 | 19 | 1 | 0 | 0 | 0 | 34 | 136000 | 0 | 0 | 0 | SP | SP | LN | 2 | 1 | 0 | 17 | ANHT | 0 |
| 155 | 26 | 2 | 0 | 0 | 1 | 34 | 46000 | 0 | 0 | 0 | SP | SP | LN | 2.2 | 1 | 0 | 21 | ANHT | 0 |
| 156 | 21 | 1 | 0 | 0 | 0 | 34 | 102000 | 0 | 0 | 0 | | GT | LN | 1.2 | 0 | 1 | 5 | | 0 |

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| 157 | 22 | 1 | 0 | 0 | 0 | 32 | 100000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 5 | | 0 |
| 158 | 19 | 1 | 0 | 0 | 0 | 31 | 108000 | 0 | 0 | 0 | | GT | LN | 1.3 | 0 | 1 | 10 | | 0 |
| 159 | 23 | 1 | 0 | 0 | 0 | 39 | 112000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 5 | | 0 |
| 160 | 31 | 4 | 3 | 3 | 0 | 35 | 98000 | 0 | 0 | 0 | SP | SP | LSCS | 2.01 | 1 | 0 | 17 | ANHT | 0 |
| 161 | 21 | 1 | 0 | 0 | 0 | 39 | 109000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 162 | 33 | 2 | 1 | 1 | 0 | 38 | 88000 | 0 | 4 | 1 | | GT | LSCS | 2.8 | 0 | 1 | 5 | | 0 |
| 163 | 22 | 1 | 0 | 0 | 0 | 36 | 126000 | 0 | 0 | 0 | SP | SP | LSCS | 2.3 | 0 | 1 | 7 | ANHT | 0 |
| 164 | 32 | 2 | 1 | 1 | 0 | 36 | 86000 | 0 | 0 | 0 | | GT | LSCS | 2.5 | 0 | 1 | 5 | | 0 |
| 165 | 21 | 1 | 0 | 0 | 0 | 30 | 106000 | 0 | 0 | 0 | | GT | LN | 1.1 | 0 | 1 | 17 | | 0 |
| 166 | 32 | 1 | 0 | 0 | 0 | 30 | 120000 | 0 | 0 | 0 | | GT | LN | 1.1 | 0 | 1 | 12 | | 0 |
| 167 | 34 | 2 | 1 | 1 | 0 | 32 | 86000 | 0 | 0 | 0 | | GT | LSCS | 1.6 | 0 | 1 | 10 | | 0 |
| 168 | 34 | 2 | 1 | 1 | 0 | 39 | 76000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 169 | 18 | 1 | 0 | 0 | 0 | 38 | 130000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |
| 170 | 21 | 1 | 0 | 0 | 0 | 38 | 110000 | 1 | 0 | 0 | | HELLP | LSCS | 2.3 | 0 | 1 | 10 | ANHT | 0 |
| 171 | 37 | 2 | 1 | 1 | 0 | 38 | 86000 | 0 | 0 | 0 | | GT | LSCS | 3 | 0 | 1 | 6 | | 0 |
| 172 | 23 | 1 | 0 | 0 | 0 | 39 | 110000 | 0 | 0 | 0 | | GT | LN | IUD | 0 | 1 | 3 | | 0 |
| 173 | 37 | 2 | 1 | 1 | 0 | 40 | 84000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 174 | 26 | 1 | 0 | 0 | 0 | 37 | 110000 | 0 | 0 | 0 | | DENGUE | LN | 2.7 | 0 | 1 | 21 | | 0 |
| 175 | 36 | 2 | 1 | 1 | 0 | 37 | 130000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 7 | | 0 |
| 176 | 24 | 1 | 0 | 0 | 0 | 40 | 120000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 5 | | 0 |
| 177 | 36 | 1 | 0 | 0 | 0 | 37 | 107000 | 0 | 0 | 0 | | GT | LSCS | 2.5 | 0 | 1 | 7 | | 0 |
| 178 | 33 | 2 | 1 | 1 | 0 | 34 | 45000 | 1 | 4 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 179 | 25 | 2 | 1 | 1 | 0 | 30 | 118000 | 0 | 0 | 0 | ECLAM | | LSCS | 1.4 | 1 | 0 | 7 | ANHT | 0 |
| 180 | 31 | 1 | 0 | 0 | 0 | 39 | 108000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 7 | | 0 |
| 181 | 32 | 2 | 1 | 1 | 0 | 36 | 72000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 5 | | 0 |
| 182 | 30 | 1 | 0 | 0 | 0 | 32 | 100000 | 0 | 0 | 0 | SP | SP | LSCS | 1.25 | 1 | 0 | 7 | ANHT | 0 |
| 183 | 36 | 2 | 1 | 1 | 0 | 38 | 120000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |
| 184 | 27 | 3 | 1 | 1 | 1 | 30 | 120000 | 0 | 0 | 0 | SP | SP | LN | 1.5 | 1 | 0 | 10 | ANHT | 0 |
| 185 | 19 | 1 | 0 | 0 | 0 | 32 | 109000 | 0 | 0 | 0 | | GT | LN | 1.5 | 0 | 1 | 10 | | 0 |
| 186 | 26 | 1 | 0 | 0 | 0 | 30 | 106000 | 0 | 0 | 0 | | GT | LN | 1.1 | 0 | 1 | 3 | | 0 |
| 187 | 25 | 2 | 1 | 1 | 0 | 24 | 98000 | 0 | 0 | 0 | SP | SP | LN | 2 | 1 | 0 | 14 | ANHT | 0 |
| 188 | 28 | 1 | 0 | 0 | 0 | 37 | 121000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 189 | 31 | 2 | 1 | 1 | 0 | 39 | 120000 | 0 | 0 | 0 | GDM | GT | LSCS | 2.6 | 0 | 1 | 6 | | 0 |
| 190 | 25 | 1 | 0 | 0 | 0 | 34 | 90000 | 0 | 0 | 0 | | GT | LN | 1.7 | 0 | 1 | 7 | | 0 |
| 191 | 24 | 1 | 0 | 0 | 0 | 37 | 96000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 192 | 31 | 2 | 1 | 1 | 0 | 40 | 120000 | 0 | 0 | 0 | | GT | LSCS | 2.9 | 0 | 1 | 5 | | 0 |
| 193 | 36 | 1 | 0 | 0 | 0 | 39 | 91000 | 0 | 0 | 0 | | GT | LSCS | 2.8 | 0 | 1 | 3 | | 0 |
| 194 | 22 | 2 | 1 | 1 | 0 | 39 | 86000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 195 | 23 | 1 | 0 | 0 | 0 | 30 | 140000 | 0 | 0 | 0 | SP | SP | LN | 1.4 | 1 | 0 | 9 | ANHT | 0 |
| 196 | 23 | 2 | 1 | 1 | 0 | 40 | 87000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 5 | | 0 |
| 197 | 33 | 4 | 2 | 2 | 1 | 36 | 100000 | 0 | 0 | 0 | SP | SP | LSCS | 2.2 | 1 | 0 | 11 | ANHT | 0 |
| 198 | 33 | 1 | 0 | 0 | 0 | 33 | 106000 | 0 | 0 | 0 | | GT | LSCS | 1.7 | 0 | 1 | 10 | | 0 |
| 199 | 24 | 2 | 1 | 1 | 0 | 39 | 81000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |
| 200 | 27 | 1 | 0 | 0 | 0 | 38 | 131000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 201 | 26 | 2 | 1 | 1 | 0 | 34 | 36000 | 0 | 4 | 2 | SP | SP | LSCS | 2.1 | 1 | 0 | 12 | ANHT | 0 |
| 202 | 18 | 1 | 0 | 0 | 0 | 40 | 86000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 203 | 24 | 2 | 1 | 1 | 0 | 38 | 94000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 3 | | 0 |
| 204 | 27 | 1 | 0 | 0 | 0 | 40 | 117000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 205 | 23 | 2 | 1 | 1 | 0 | 38 | 101000 | 0 | 0 | 0 | HD | GT | LN | 2.5 | 0 | 1 | 10 | | 0 |
| 206 | 23 | 1 | 0 | 0 | 0 | 39 | 120000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 3 | | 0 |
| 207 | 32 | 1 | 0 | 0 | 0 | 40 | 110000 | 0 | 0 | 0 | | GT | LSCS | 3 | 0 | 1 | 5 | | 0 |
| 208 | 21 | 1 | 0 | 0 | 0 | 40 | 98000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 209 | 25 | 2 | 1 | 1 | 0 | 39 | 92000 | 0 | 0 | 0 | | GT | LN | 2.1 | 0 | 1 | 6 | | 0 |
| 210 | 36 | 1 | 0 | 0 | 0 | 36 | 107000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 211 | 24 | 2 | 0 | 0 | 1 | 39 | 100000 | 0 | 0 | 0 | SP | SP | LSCS | 2.7 | 0 | 1 | 7 | ANHT | 0 |
| 212 | 26 | 1 | 0 | 0 | 0 | 32 | 110000 | 0 | 0 | 0 | SP | ECLAM | LSCS | 1.8 | 1 | 0 | 14 | ANHT | 0 |
| 213 | 29 | 1 | 0 | 0 | 0 | 37 | 96000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 214 | 31 | 2 | 1 | 1 | 0 | 36 | 120000 | 0 | 0 | 0 | | GT | LN | 2.2 | 0 | 1 | 3 | | 0 |
| 215 | 34 | 1 | 0 | 0 | 0 | 37 | 108000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 216 | 24 | 1 | 0 | 0 | 0 | 38 | 110000 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 3 | | 0 |
| 217 | 26 | 2 | 1 | 1 | 0 | 30 | 120000 | 0 | 0 | 0 | SP | SP | LN | 1.3 | 1 | 0 | 12 | ANHT | 0 |
| 218 | 26 | 1 | 0 | 0 | 0 | 34 | 107000 | 0 | 0 | 0 | | GT | LSCS | 2 | 0 | 1 | 7 | | 0 |
| 219 | 21 | 1 | 0 | 0 | 0 | 32 | 180000 | 0 | 0 | 0 | | GT | LSCS | 1.8 | 0 | 1 | 10 | | 0 |
| 220 | 25 | 2 | 1 | 1 | 0 | 40 | 72000 | 0 | 0 | 1 | | GT | LN | 3 | 0 | 1 | 3 | | 0 |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 221 | 23 | 1 | 0 | 0 | 0 | 32 | 121000 | 0 | 0 | 0 | | GT | LSCS | 1.1 | 0 | 1 | 5 | | 0 |
| 222 | 27 | 1 | 0 | 0 | 0 | 38 | 110000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 223 | 32 | 3 | 2 | 2 | 0 | 36 | 96000 | 0 | 0 | 0 | SP | SP | LN | 2.5 | 0 | 1 | 7 | ANHT | 0 |
| 224 | 18 | 1 | 0 | 0 | 0 | 37 | 140000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |
| 225 | 21 | 2 | 1 | 1 | 0 | 40 | 72000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 3 | | 0 |
| 226 | 28 | 1 | 0 | 0 | 0 | 36 | 96000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 227 | 22 | 1 | 0 | 0 | 0 | 37 | 98000 | 0 | 0 | 0 | SP | SP | LSCS | 2.7 | 0 | 1 | 7 | ANHT | 0 |
| 228 | 29 | 3 | 2 | 2 | 0 | 34 | 98000 | 0 | 0 | 0 | SP | SP | LSCS | 2 | 1 | 0 | 14 | ANHT | 0 |
| 229 | 19 | 2 | 1 | 1 | 0 | 37 | 70000 | 0 | 0 | 0 | | GT | LN | 2.3 | 0 | 1 | 3 | | 0 |
| 230 | 27 | 1 | 0 | 0 | 0 | 34 | 88000 | 0 | 0 | 0 | | GT | LN | 2.1 | 0 | 1 | 5 | | 0 |
| 231 | 20 | 2 | 1 | 1 | 0 | 37 | 120000 | 0 | 0 | 0 | | GT | LN | 2.4 | 0 | 1 | 3 | | 0 |
| 232 | 31 | 1 | 0 | 0 | 0 | 31 | 102000 | 0 | 0 | 0 | | GT | LSCS | 1.7 | 0 | 1 | 8 | | 0 |
| 233 | 23 | 1 | 0 | 0 | 0 | 38 | 115000 | 0 | 0 | 0 | SP | SP | LN | 2.6 | 0 | 1 | 7 | ANHT | 0 |
| 234 | 19 | 2 | 1 | 1 | 0 | 37 | 90000 | 0 | 0 | 0 | | GT | LN | 2.25 | 0 | 1 | 3 | | 0 |
| 235 | 33 | 1 | 0 | 0 | 0 | 38 | 113000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |
| 236 | 22 | 2 | 1 | 1 | 0 | 37 | 110000 | 0 | 0 | 0 | GDM | GT | LN | 2.7 | 0 | 1 | 4 | | 0 |
| 237 | 25 | 1 | 0 | 0 | 0 | 37 | 94000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 238 | 28 | 2 | 1 | 1 | 0 | 30 | 131000 | 0 | 0 | 0 | SP | SP | LN | 1.6 | 1 | 0 | 7 | ANHT | 0 |
| 239 | 22 | 1 | 0 | 0 | 0 | 40 | 77000 | 0 | 0 | 0 | | GT | LN | 3 | 0 | 1 | 3 | | 0 |
| 240 | 23 | 2 | 1 | 1 | 0 | 39 | 108000 | 0 | 0 | 0 | | GT | LN | 3.1 | 0 | 1 | 3 | | 0 |
| 241 | 21 | 1 | 0 | 0 | 0 | 38 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.7 | 0 | 1 | 5 | | 0 |
| 242 | 31 | 3 | 1 | 1 | 1 | 36 | 110000 | 0 | 0 | 0 | SP | SP | LSCS | 2.7 | 0 | 1 | 5 | ANHT | 0 |
| 243 | 22 | 1 | 0 | 0 | 0 | 36 | 96000 | 0 | 0 | 0 | | GT | LSCS | 2.3 | 0 | 1 | 10 | | 0 |
| 244 | 27 | 2 | 0 | 0 | 1 | 32 | 98000 | 0 | 0 | 0 | SP | ECLAMP | LSCS | 1.7 | 0 | 1 | 21 | ANHT | 0 |
| 245 | 28 | 1 | 0 | 0 | 0 | 40 | 89000 | 0 | 0 | 0 | | GT | LN | 3.1 | 0 | 1 | 3 | | 0 |
| 246 | 25 | 2 | 0 | 0 | 1 | 38 | 89000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 5 | | 0 |
| 247 | 25 | 1 | 0 | 0 | 0 | 37 | 120000 | 0 | 0 | 0 | | GT | LSCS | 2.5 | 0 | 1 | 5 | | 0 |
| 248 | 30 | 2 | 1 | 1 | 0 | 30 | 128000 | 0 | 0 | 0 | SP | SP | LN | 1.5 | 1 | 0 | 7 | ANHT | 0 |
| 249 | 26 | 1 | 0 | 0 | 0 | 36 | 110000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 250 | 22 | 1 | 0 | 0 | 0 | 34 | 38000 | 1 | 4 | 3 | | HELLP | LSCS | 2.1 | 0 | 1 | | | 1 |
| 251 | 23 | 1 | 0 | 0 | 0 | 38 | 94000 | 0 | 0 | 0 | SP | SP | LSCS | 2.7 | 0 | 1 | 5 | ANHT | 0 |
| 252 | 26 | 1 | 0 | 0 | 0 | 32 | 105000 | 0 | 0 | 0 | | GT | LN | 1.6 | 0 | 1 | 7 | | 0 |

| | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 253 | 22 | 1 | 0 | 0 | 0 | 36 | 100200 | 0 | 0 | 0 | | GT | LN | 2.3 | 0 | 1 | 5 | | 0 |
| 254 | 35 | 2 | 0 | 0 | 1 | 35 | 110000 | 0 | 0 | 0 | | GT | LN | 2.4 | 0 | 1 | 5 | | 0 |
| 255 | 27 | 1 | 0 | 0 | 0 | 36 | 106000 | 0 | 0 | 0 | | GT | LSCS | 2.6 | 0 | 1 | 5 | | 0 |
| 256 | 22 | 3 | 0 | 0 | 2 | 37 | 110000 | 0 | 0 | 0 | | GT | LN | 2.7 | 0 | 1 | 3 | | 0 |
| 257 | 24 | 2 | 1 | 1 | 0 | 36 | 120100 | 0 | 0 | 0 | | GT | LSCS | 2.4 | 0 | 1 | 5 | | 0 |
| 258 | 26 | 1 | 0 | 0 | 0 | 34 | 110000 | 0 | 0 | 0 | | GT | LSCS | 1.8 | 0 | 1 | 12 | | 0 |
| 259 | 32 | 3 | 1 | 1 | 1 | 38 | 108000 | 0 | 0 | 0 | | SP | SP | 2.8 | 0 | 1 | 7 | | 0 |
| 260 | 34 | 1 | 0 | 0 | 0 | 36 | 110000 | 0 | 0 | 0 | | GT | LN | 2.5 | 0 | 1 | 3 | | 0 |
| 261 | 24 | 2 | 1 | 1 | 0 | 37 | 140000 | 0 | 0 | 0 | SP | SP | LN | 2.6 | 0 | 1 | 5 | ANHT | 0 |
| 262 | 26 | 1 | 0 | 0 | 0 | 35 | 89000 | 0 | 0 | 0 | | GT | LN | 2.4 | 0 | 1 | 5 | | 0 |
| 263 | 22 | 2 | 1 | 1 | 0 | 37 | 110000 | 0 | 0 | 0 | | GT | LSCS | 2.5 | 0 | 1 | 5 | | 0 |
| 264 | 27 | 1 | 0 | 0 | 0 | 36 | 120000 | 0 | 0 | 0 | | GT | LN | 2.6 | 0 | 1 | 3 | | 0 |
| 265 | 28 | 2 | 1 | 1 | 0 | 32 | 110000 | 0 | 0 | 0 | HELLP | | LN | 1.4 | 0 | 1 | 10 | ANHT | 0 |
| 266 | 25 | 1 | 0 | 0 | 0 | 36 | 64000 | 0 | 0 | 1 | ITP | | LSCS | 2.4 | 0 | 1 | 12 | | 0 |
| 267 | 22 | 1 | 0 | 0 | 0 | 40+2 | 102000 | 0 | 0 | 0 | | GT | LN | 2.9 | 0 | 1 | 3 | | 0 |
| 268 | 21 | 2 | 1 | 1 | 0 | 36 | 120000 | 0 | 0 | 0 | SP | SP | LN | 2.4 | 0 | 1 | 5 | ANHT | 0 |
| 269 | 32 | 2 | 1 | 1 | 0 | 36 | 77000 | 1 | 0 | 2 | ITP | | LN | 2.3 | 0 | 1 | 9 | | 0 |
| 270 | 25 | 2 | 0 | 0 | 1 | 34 | 95000 | 0 | 0 | 1 | AFLP | | LSCS | 2.4 | 0 | 1 | 9 | | 0 |
| 271 | 26 | 1 | 0 | 0 | 0 | 38 | 103000 | 1 | | 0 | HELLP | | LSCS | 2.6 | 0 | 1 | | | 1 |
| 272 | 21 | 1 | 0 | 0 | 0 | 35 | 100200 | 0 | 0 | 0 | | GT | LN | 2.8 | 0 | 1 | 7 | | 0 |
| 273 | 23 | 2 | 1 | 1 | 0 | 38 | 120000 | 0 | 0 | 0 | 0 | GT | LSCS | 2.5 | 0 | 1 | 5 | | 0 |

**THROBOCYTOPENIA IN PREGNANCY**
**RAJA MIRASUDHAR HOSITAL,THANJAVUR**

NAME: AGE: IP NO:
LMP: G/P/L/A:
EDD: DOA:
GA: DOD:
BLOOD GROUPING:

## HISTORY:

-GHT YES ☐ NO ☐ IF yes _____

-BLEEDING MANIFESTATION YES ☐ NO ☐ IF YES _____

-JAUNDICE YES ☐ NO ☐ IF YES _____

-PAST H/O THROMBOCYTOPENIA YES ☐ NO ☐ IF YES _____

## EXAMINATION:

## INVESTIGATIONS:

| INVESTIGATIONS | | | | | | |
|---|---|---|---|---|---|---|
| PLT | | | | | | |
| HB | | | | | | |
| BT | | | | | | |
| CT | | | | | | |
| TC | | | | | | |
| DC | | | | | | |
| PT | | | | | | |
| INR | | | | | | |
| TB | | | | | | |
| DB | | | | | | |
| INB | | | | | | |
| UREA | | | | | | |
| CREATININE | | | | | | |
| APTT | | | | | | |
| SGOT | | | | | | |
| SGPT | | | | | | |
| ALP | | | | | | |
| LDH | | | | | | |
| PDW | | | | | | |
| MPV | | | | | | |
| DENGUE | | | PSS | | | |
| OTHERS | | | | | | |

## DELIVERY OUTCOME:

GESTATION AGE : TERM ☐ PRE-TERM ☐ POST-TERM ☐

MODE OF DELIVERY: LABOUR NATURAL ☐ CS ☐

INDICATION FOR CS _____

BLOOD LOSS IN LABOUR _____(ml)

BLOOD/ BLOOD PRODUCTS

BLOOD ☐ PLATELETS ☐ FFP ☐ IRON SUCROSE ☐

INDICATION FOR TRANSFUSION:_____
_____
_____
_____

## NEONATAL FOLLOW UP:

| |
|---|
| |

## CONCLUSION:

| | |
|---|---|
| GESTATIONAL: | |
| GHT | |
| AUTOIMMUNE | |
| HEMOLYTIC | |
| LIVER DISEASE | |
| DRUG INDUSED | |
| OTHERS | |

**CONSENT FOR PARTICIATING IN STUDY:** I here by giving my consent for participating in this study.

SIGNATURE OF PATIENT SIGN OF INVESTIGATOR