"CLOT OBSERVATION TEST FOR EARLY DETECTION OF COAGULOPATHY IN OBSTETRIC EMERGENCY: A PROSPECTIVE STUDY."

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LIST OF ABBREVIATIONS:

PT/INR	:	Prothrombin time/international normalized ratio
PT	:	Prothrombin time
aPTT	:	Activated partial thromboplastin time
DIC	:	Disseminated Intravascular Coagulation
COT	:	Clot observation test
PPH	:	Postpartum hemorrhage
PIH	:	Pregnancy-induced hypertension
APACHE	:	The Acute Physiology and Chronic Health Evaluation
BCSH	:	British Committee for Standards in Haematology
TEG	:	Thromboelastography
AT	:	Antithrombin
TAT	:	Thrombin- antithrombin
TAFI	:	thrombin activatable fibrinolysis inhibitor
PAI	:	Plasminogen activator inhibitor
VLDL	:	Very low-density lipoprotein particles (VLDL)
TF	:	Tissue factor
tPA	:	Tissue plasminogen activator
HELLP	:	Haemolysis Elevated Liver Enzymes Low Platelets
VWF	:	von willebrand factor

ABSTRACT

Background: Multiple organ failure is connected to Disseminate intravascular coagulopathy (DIC) due to systemic coagulation activation, which causes disseminated fibrin deposition in microscopic blood vessels due to thrombin activation. Fibrinogen levels have been linked to the early diagnosis of abnormal pregnancies caused by coagulation abnormalities. Diagnosis of fibrinolysis can be done by using clot observation test, which involves observing the clot to detect presence of fibrinolysis.

Aim: Present study was aimed to compare clot observation test (COT) with serum fibrinogen level and to assess the role of the clot observation test, a simple bedside test, vs DIC profile procedure in the early stages of DIC diagnosis.

OBJECTIVES OF THE STUDY:

(i) To compare clot observation test with serum fibrinogen level.

(ii) To assess the role of the clot observation test, a simple bedside test, vs the sophisticated DIC profile for diagnosis of DIC.

Methodology: The study population consisted of pregnant women suspected to have coagulation disorders and admitted in labor room during the study period fulfilling the inclusion criteria and consenting to participate in the study. Bedside clot observation test is done and at the same time blood sample is sent for DIC profile, then clot observation test is compared with Serum fibrinogen level and DIC profile.

Results:

Out of 175 patients, 17 had bedside clot observation test of less than 6 minutes and 19 patients had bedside cot of more than and equal to 6 minutes with serum fibrinogen level of

less than 150 mg/dl. 134 patients had bedside cot of less than 6 minutes and 4 patients had bedside cot of more than and equal to 6 minutes with serum fibrinogen level more than and equal to 150 mg/dl. There is significant inverse correlation of Sr fibrinogen with COT.

Conclusion:

Significant difference in the distribution of Sr fibrinogen, Platelet count, aPTT PT/INR and D dimer with bedside COT is observed.

Keywords: DIC, Coagulation, Haemostasis

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CHAPTER-1

INTRODUCTION

Haemostasis is a complex mechanism involving various processes which operate at multiple levels, and encompass intricate systems, which are not well defined in the traditional coagulation pathway. This process is rendered even more complicated during pregnancy because of occurrence of changes like alterations in the concentration of coagulating factors and phenomenon like physiological anaemia. These disturb the balance between bleeding and clotting, thereby resulting in conditions like coagulopathy[1].

Recent research has proposed a cell-based model for coagulation which involves functioning of both systems together resulting in thrombin formation either intrinsically on surface of platelets or extrinsically at surface of injury. Formation of thrombin is initiated mainly by tissue factor, followed by amplification by platelet activation. To platelet receptors and arterial wall components, thrombin and circulating von willebrand factor (VWF) play a significant role. On the surface of platelet the activated factors form the tenase complex, which leads to formation of prothrombinase complex, generates thrombin burst, eventually forming fibrin from the activated factors. The anticoagulant system, which includes pathway factor inhibitor, protein C and protein S as well as the fibrinolytic processing or pasting something here, keeps the coagulation pathway in check[2].

During pregnancy various changes are observed in the coagulation system which include decrease in number of platelets, occurrence of physiological anaemia, which is brought about by a rise in plasma volume by 40 and rise in RBC volume by 25% as well as changes in concentration of coagulation factors. Fibrinogen levels climb from 2-4 gm/L to 4-6 gm/L (normal level), and factors VII, VIII, and X grow

by 20-100 percent. The combined effect of these alterations results in double the normal coagulation activity, due to which pregnancy is termed as a hypercoagulable state[3]. Moreover, there is a decrease in fibrinolytic activity. The largest increase in clotting activity occurs during childbirth, as the placenta is expelled, releasing thromboplastic substances. To prevent maternal blood loss, these substances promote the formation of clots. If clotting fails, significant haemorrhage can occur because placental blood flow can reach 700 ml per minute. 3–4 weeks after delivery, coagulation and fibrinolysis typically return to pre-pregnancy stages[4].

"The various coagulation disorders associated with pregnancy include disseminated intravascular coagulation, congenital coagulation disorders like Von Willebrand's disease, Haemophilia A and B, as well as non-inherited illness such as thrombocytopenia and immunologic (idiopathic) thrombocytopenic purpura, prothrombotic / microangiopathic events such as HELLP syndrome, other microangiopathies such as deep venous thrombosis (DVT) and multiple pregnancy loses are also included"[5].

"Disseminated intravascular coagulopathy (DIC) is considered to be one of the worst among all the coagulopathies as it has a significantly higher mortality as well as morbidity rate (Bhave, 2019). It is a fatal syndrome characterized by coagulation activation that is dispersed and sometimes uncontrolled. This syndrome is associated to an increased risk of macro- and microvascular thrombosis, as well as a gradual coagulopathy that increases the risk of bleeding[6].Activation of the coagulation pathway as a result of fibrin buildup in the vasculature, organ dysfunction, and clotting factor and platelet consumption all contribute to life-threatening haemorrhage. DIC occurs as a result of parallel development of certain mechanisms.

Excessive thrombin production is not effectively controlled by a defective anticoagulation system, such as antithrombin and C protein, resulting in an increase in fibrin formation and deposition in the vascular system. During the early stages of DIC, fibrinolysis is sustained by plasmin, which is released by endothelial cells activating plasminogen activators. However, high blood levels of plasminogen activator inhibitor-1, a fibrinolytic inhibitor, neutralise this impact rapidly.[6].

Many disease processes, which are often organ-specific, use disseminated intravascular coagulation as an intermediate mechanism. This devastating syndrome affects all aspects of medicine and has a wide clinical range that can be confounding. Consumptive coagulopathy was a term used in the early literature to describe DIC, however, this term is no longer considered a proper description since very little research has been done on it. The majority of components and plasma constituents are plasmin-biodegraded. Defibrination syndrome was coined later. The current terminology, Disseminated Intravascular Coagulation, is a useful descriptive pathophysiology term when coagulation is disseminated intravascularly[8].

Sepsis, trauma, cancer, liver illness, obstetric diseases, vascular abnormalities, and significant transfusion responses are all linked to DIC as a secondary disorder. In obstetrical patients, DIC is a widely recognised complication in patients with pre-eclampsia and placental abruption and amniotic fluid embolism, all of which are uncommon but life-threatening conditions. Women who survive an acute amniotic fluid embolism are more likely to develop DIC[9]. Intrauterine foetal death, missed abortion, septic abortion, excessive blood loss, puerperal sepsis, infections, neoplasms, and major tissue injury are some of the other problems linked to DIC[10]. Placental abruption was the leading cause of DIC in studies from Canada and Israel,

especially in combination with antepartum foetal death and post-partum haemorrhage, while foetal death and post-partum haemorrhage were found to be the major cause of DIC in reports from Pakistan and Thailand. DIC causes life-threatening diseases in mothers, including as major blood product transfusions, hysterectomy, and even death, necessitating thorough research, prompt diagnosis, and appropriate treatment[11].

DIC can take one of two forms, depending on the underlying condition, the degree of coagulation activity, and the presence or lack of natural anticoagulant pathways. These two forms are latent form and the overt form. The former is accompanied by lack of obvious symptoms, in spite of presence of haemostatic dysfunction and increases in thrombosis risk. This syndrome is characterised by an imbalance in the activation and inhibition of the coagulation mechanisms. It's more common during pregnancy or when someone has an immune disease. Overt DIC involves lack of regulatory mechanism and includes both bleeding and thrombosis. Bleeding may be due to reduced haemostatic ability caused by high consumption of coagulation factors during activation of coagulation and hence is also termed consumption coagulopathy[6]. DIC is also described as a progressive condition characterised by three phases 1, is an activation state without apparent use of platelets and coagulation factors, phase 2, a decompensated state which involves disturbances in coagulation resulting in lengthening of clotting times and decreasing platelet counts, and the last where intense utilization of clotting factors and platelets occurs, resulting in haemolysis[9].

Successful handling of this condition requires precise and timely detection methods. However, clinical examination of the patient is usually used to diagnose DIC. Furthermore, there is a scarcity of sensitive and specific laboratory or clinical

tests for DIC diagnosis. To close this gap and provide doctors with a means for early diagnosis and understanding of DIC, researchers have focused their efforts on developing scoring systems that can classify individuals who are at higher risk for this potentially fatal illness. Simple coagulation tests such as platelet count, PT prolongation, fibrinogen, and fibrin split products/D-dimer concentrations are used in this scoring system. If the score is five or higher, DIC is suspected, whereas lower numbers imply nonovert DIC. For less severe DIC presentations, a complicated composite score has been developed[12]. Platelet count, prothrombin time or PT, fibrin-related marker, and fibrinogen must all be assessed on a continuous basis in order to detect DIC[11].

Thrombocytopenia is a typical DIC symptom, however even without DIC, thrombocytopenia and obstetrical disorders like preeclampsia or HELLP can produce low platelet counts during pregnancy. The prolongation of PT and APTT is also used to diagnose DIC. However, normal ranges for both tests are substantially lower in pregnant women than in non-pregnant people. As a result, this detection test may reveal inaccuracies. The PT and APTT are not prolonged until the disease have progressed sufficiently. Because of the high amounts of factor VIII in the blood during pregnancy, the APTT can be shortened. The concentration of maternal plasma fibrinogen doubles during normal pregnancy. Although low fibrinogen concentration is a well-known DIC detection measure, the definition of low fibrinogen concentration during the pregnancy is the same as that of nonpregnant women. Thus, pregnant women with severe postpartum haemorrhage (PPH) may have normal fibrinogen concentration with use of nonpregnant levels.At the same time, fibrinogen concentration falls below the normal pregnancy range quicker than the other clotting

factors, according to research, and a decline in serum fibrinogen level is a good biomarker for progression from mild to severe PPH[11].

To aid in DIC diagnosis, a DIC score system was devised. In order to make it easier to determine which ISTH DIC score to use, additional parameters were added by the Japanese Association for Acute Medicine (JAAM) in 2005. An abnormal result has been connected to the prognosis of patients hospitalised to critical care units and has shown great diagnostic performance for diagnosing DIC in non-pregnant women. It has a good predictive value for DIC diagnosis and detection in non-pregnant critically unwell individuals. These scores, on the other hand, are not adjusted for the haemostatic alterations that occur during pregnancy, limiting their usefulness in pregnant patients[11].Using this as a foundation, a modified DIC score was created, which contained three parameters: platelet count, fibrinogen concentration and PT difference (ISTH DIC score). It exhibited a 96 percent specificity and an 88 percent sensitivity[13].

Since concentration of fibrinogen drastically reduces in pregnancy associated DIC, the bedside blood clot observation test, can be useful in diagnosis in patients with hypofibrinogenemia, which is a condition associated with heavy bleeding. Observation of the clot is one of the most valuable and reliable tests for fibrinopenia and fibrinolysis . The blood clot is typically deficient in bulk or consistency, appears soft, and is likely to be physiologically ineffective in this state. On standing, the clot disappears from blood samples; this disappearance can take hours before it is noticed, but in clinically important cases, the clot is likely to dissolve within an hour[14]. Fibrinolysis during early stages can be managed by proper therapy. However, without timely detection, the disease progresses rapidly into a stage, where treatment is

difficult. Hence a simple method of early diagnosis is desirable. However, there is no one laboratory test that can diagnose or rule out DIC, particularly in the early stages hence a simple and reliable test which could predict DIC early is the need of the day.

OBJECTIVES OF THE STUDY

The following are the study's aims, which are based on a gap in the literature:

(i) To compare clot observation test with serum fibrinogen level.

(ii) To assess the role of the clot observation test, a simple bedside test, vs the sophisticated DIC profile for diagnosis of DIC.

CHAPTER-2

REVIEW OF LITERATURE

Haemostasis is derived from two Greek words: haeme, which means blood and stasis which means motionless. It is a state of dynamic equilibrium between coagulation and fibrinolysis. In case of coagulation, vessel walls, platelets and the coagulation factors interact with each other. In case of endothelial injury, platelets attach themselves to the subendothelium resulting in a plug (platelets) that is hardened subsequently by deposition of fibrin. Antithrombin (AT) and proteins C and S are responsible for limiting clot formation. The liquid state of blood is maintained by the fibrinolytic system, where plasmin produced from plasminogen with the help of tissue plasminogen activator (t-PA), results in breakdown of fibrin[4]. Haemostatic mechanisms are complex functioning at multiple levels, with nuanced feedback systems and this becomes even more convoluted in pregnancy due to physiological changes like physiological anaemia and changes in coagulation factor quantity in blood, which affects balance between bleeding and clot formation[15]. Patients with a coagulation disorder during pregnancy may be difficult to classify in terms of whether they are facing thrombotic or haemorrhagic risk[1].

The intrinsic and extrinsic pathways of the classical coagulation model together with the common pathway at the level of factor X (FX) is useful for interpretation results of the core coagulation tests including prothrombin time (PT) and activated partial thromboplastin time (APTT) as is given in figure 2-1 below

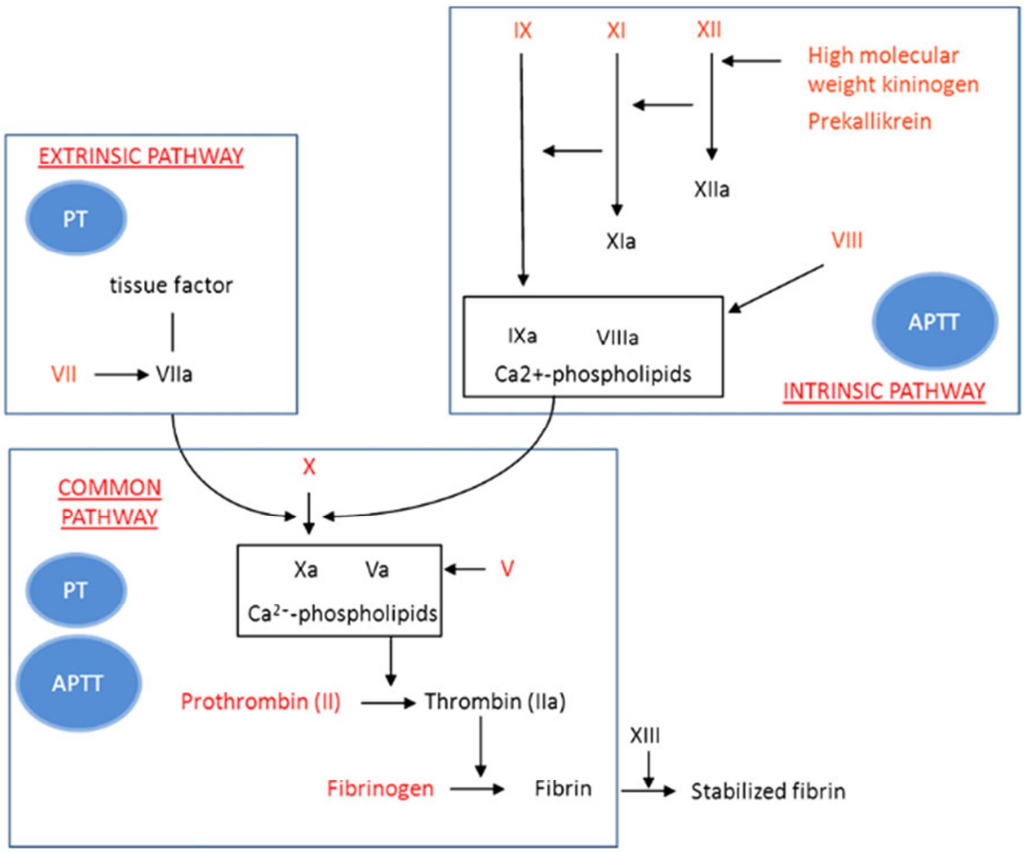

Figure 2-1. Coagulation pathway

2.1 CHANGES IN THE COAGULATION SYSTEM DURING PREGNANCY

Various haemostatic changes occur during pregnancy, including a drop in anticoagulant levels, an increase in clotting factor concentrations, and a decrease in fibrinolytic activity, all of which contribute to a hypercoagulable state and can be linked to hormonal changes. These alterations, which cause hypercoagulability and increase the risk of thromboembolism, are most likely attributable to hormonal changes[16]. This increase in clotting activity peaks during delivery and is responsible for the mother's blood loss prevention. In 3–4 weeks after delivery, this haemostatic aberration returns to normal.

The following table (Table 2-1) shows the changes in the factors responsible for haemostasis[1].

Table 2-1. Changes in parameters of haemostasis in pregnancy

Haemostatic parameter	Change at term pregnancy (% change)
Factors II and V	No change
Fibrinogen	Rises more than 100%
Factor VII	Increase upto 1000%
Factors VIII, IX, X, XII and VWF	Increase more than 100%
Factor XI and Variable Factor XIII	50% decrease
Protein C	No change
Protein S	Decrease upto 50%
D dimer	Increase till 400%
Platelet count	20% decrease

2.2 COAGULATION DISORDERS IN PREGNANCY

"Coagulopathies in pregnancy include prothrombotic / disorders such as HELLP (Haemolysis Elevated Liver Enzymes Low Platelets), TTP (thrombotic thrombocytopenia purpura), and other microangiopathies such as DIC (disseminated intravascular coagulopathy), DVT (deep venous thrombosis), and recurrent pregnancy losses, as well as conditions characterized by excessive bleeding such as antepartum haemorrhage ,postpartum haemorrhage[5]. Various coagulation disorders are described below.

"Disseminate intravascular coagulopathy (DIC) is one of the most dangerous coagulopathies, with high rates of morbidity and mortality. Multiple organ failure is connected to DIC due to systemic coagulation activation, which causes disseminated fibrin deposition in microscopic blood vessels due to thrombin activation. Because of the hypercoagulability, platelets and clotting factors are reduced, resulting in heavy bleeding later. This condition is termed consumptive coagulopathy"[5].

"Thrombocytopaenia is a condition that affects 6–10% of all pregnancies and is marked by a reduction in platelet count. Pregnancy-related thrombocytopaenia includes gestational thrombocytopaenia, pre-eclampsia, including HELLP syndrome, acute fatty liver of pregnancy, DIC, and thrombocytopenic purpura. Thrombocytopenia can be caused by severe sepsis, some drugs (e.g., SH), and viral infections"[17].

"Von Willebrand disease and haemophilia carrier states are the most frequent coagulopathies caused by clotting factor deficits."[18].

A. Von Willebrand's disease (vWD) is a congenital (vWD). It is the most prevalent congenital bleeding condition, affecting 1% of the population and caused by autosomal dominant transmission of quantitative or qualitative abnormalities in v Wf (a glycoprotein that plays a crucial role in primary and secondary haemostasis). v Wf is essential for proper platelet adherence to the injury site and protects circulating FVIII from proteolysis. The clinical condition in v WD is caused by a significant decrease in FVIII activity.

B. Factor VIII Deficiency, often known as haemophilia A, is an X-linked, recessive disorder that affects only a small percentage of women. When the activity of FVIII is 35 percent, haemophilia A is diagnosed.

C. Factor IX deficiency: FIX deficiency also termed haemophilia B, is also a X linked, recessive condition, hence its occurrence in women is very uncommon. A decrease in its concentration during pregnancy results in abnormalities in coagulation.

D. Factor I, II, V, VII, X, and XIII anomalies are among the more uncommon coagulation diseases.

2.3 DISEMINATED INTRAVASCULAR COAGULATION

"Disseminated intravascular coagulation (DIC) occurs when the carefully balanced process of haemostasis is disrupted, turning coagulation from a protective mechanism to a potentially harmful occurrence that is associated with significant mortality and morbidity. It is associated with disseminated and unbridled occurrence of coagulation, resulting not only in macro- and micro vascular thrombosis but also a higher risk of haemorrhage due to presence of consumption coagulopathy. Because standardized scoring methods for DIC are rarely regularly used in clinical practice, the overall incidence of DIC in hospitalized patients is unclear. Furthermore, the incidence of DIC varies depending on the kind of hospitalization and is higher among critically ill patients admitted to intensive care units (ICUs). Depending on the detection methods and diagnostic scoring system utilized, the frequency of DIC in this group of patients ranges from 8.5 percent to 34 percent"[19].

"Absence of localised initiation of clotting and the inability of natural anticlotting factors to reduce thrombin production is the characteristic feature of DIC. As a result, the scientific subcommittee of the international Society of Thrombosis and Haemostasis has described DIC as an acquired illness that causes intravascular coagulation activation without localisation and is thought to be induced by a variety of factors. It may also affect microvasculature with high intensity resulting in organ

dysfunction[6]. High amount of thrombin is produced and maintained in DIC to disrupt the perfectly balanced hemostatic process followed by its dissemination.[20].

DIC is not a disease in and of itself; it always arises as a result of another problem. DIC causes intravascular fibrin development and the process of fibrin formation can be indirectly monitored in a blood sample by currently available laboratory methods. DIC can be associated with deranged fibrinolysis[9]. DIC not only activates coagulation, resulting in fibrin deposition in small blood arteries due to thrombin generation, but it also causes consumption coagulopathy due to a decrease in platelet and coagulation factor levels, which is presented in Figure 2-2.

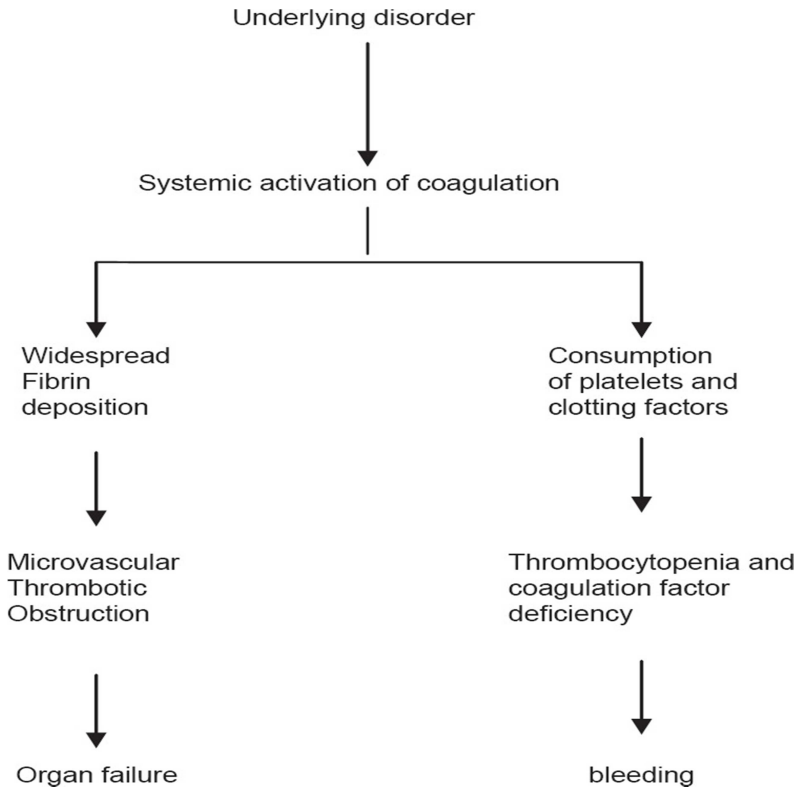

Fig. 2-2 Mechanism of DIC(Source : Venugopal, 2014)

2.4 FACTORS RESPONSIBLE FOR CAUSING DIC

The number of ailments associated with DIC that have been documented is significant. Despite their differences, these illnesses are likely to be serious illnesses with signs of systemic inflammatory involvement, a high chance of multiorgan failure and high mortality rate. DIC is a symptom of a significant underlying illness in general. DIC has been linked to a variety of clinical diseases with sepsis, cancer and obstetric situations being the most common. In the presence of sepsis, the frequency of DIC is extremely high. Patients with sepsis have DIC in 30 % to 50 % of cases, but patient with solid tumors, trauma or obstetric emergences have DIC in 10 % of cases. The numerous clinical disorders that cause DIC in pregnancies are listed below in Table 2-2[6].

Table 2-2. Causes of DIC

Malignancies: solid tumors and acute leukemias
Obstetrical complications: amniotic fluid embolism and abruptio placentae
HELLP syndrome
Pre-eclampsia/eclampsia
Vascular disorders: giant hemangioma and major aortic aneurysms
Sepsis (bacterial infections and viral infections)
severe hemolysis (transfusion reaction, sickle cell crisis), heat stroke, snake bites, vasculitis, transplantation
Liver cirrhosis and other liver disease

The most frequent clinical conditions linked with DIC are infectious illnesses, particularly bacterial septicemia in 30 – 50 % of patients with gram negative bacterial septicemia. DIC develops, and this illness has been a well-studied model for DIC (caused by bacterial lipopolysaccharide) in experimental investigations. However, DIC is also associated with Gram positive infections. The most widely known infectious disease associated with DIC is meningococcal septicemia where haemorrhagic manifestations are observed and multiorgan failure is attributed to disseminated fibrin clots. Severe trauma, especially when the brain is involved (gunshot wounds), is very commonly linked to DIC, with a frequency of 50–70% Tissue trauma combined with the production of thrombogenic material (phospholipid-containing tissue factor), hemolysis, and an inflammatory reaction can cause DIC[21]. DIC occurred in 10–15% of patients with metastasised tumors. In such cases DIC manifests as macrovascular thromboembolism (Trousseau syndrome). Substances derived from tumor cells, specific proteases, or vascular expression of tissue factor in tumor beds may all stimulate fibrin formation. In 13–20 percent of patients, DIC is linked to hematological malignancies, notably acute leukemias. A systemic bleeding condition, which is different from other types of DIC and predominantly manifests as a hyperfibrinolytic state, is common in promyelocytic leukemias. DIC is also a common secondary complication in patients with pre-eclampsia, placental abruption and amniotic fluid embolism. Where 50% of patients affected with it develop DIC. Presence of DIC is also seen in patients with HELLP.Vascular diseases such as giant hemangiomas (Kasabach-Merrit syndrome) and big aortic aneurysms have been linked to DIC (0.5–1.0 percent in patients with aneurysms to 25 percent in individuals with giant hemangiomas). Other disorders linked to DIC include chronic liver insufficiency, particularly liver cirrhosis, and it has been suggested that DIC is to

blame for the tendency to bleed seen in these patients. Although decreased coagulation protein synthesis is the most evident cause of extended clotting times in these individuals, additional mechanisms such as poor endotoxin clearance are likely to be to blame for the increased coagulation activity, which might represent a genuine reaction of DIC[9].

2.5 CLINICAL MANIFESTATIONS OF DIC

Disseminated intravascular coagulation (DIC) is a severe form of haemostatic activation that can develop in a variety of clinical settings, the majority of which are associated with some level of local or systemic inflammation. Coagulatory activation in the systemic circulation, which is aggravated by a diminished physiological fibrin-degrading capability and is ineffectively addressed by coagulation inhibitors, could lead to fibrin production and microvascular thrombotic microangiopathy. This constant and excessive thrombin production would result in a decrease in the amount of coagulation proteases and platelets, raising the risk of haemorrhagic consequences[6].

The basic illness, level of coagulation activation, and inefficiency of natural anticoagulant pathways are used to classify DIC as latent or overt.

Latent DIC: Despite the absence of clinical symptoms, this phase involves latent and compensated clotting with very modest haemostatic dysfunction and a probable increase in thrombotic risk. It is caused by a coagulation system imbalance. By reducing the stimulus that activates coagulation and strengthening the anticoagulation mechanism, this phase can be reversed to a normal state. It is frequently seen in cases requiring obstetrics or acute immunologic reactions[22].

Overt DIC: This phase is associated with reduction in haemostasis and shows a marked absence of regulatory mechanism. Moreover, its clinical symptoms include bleeding as well as thrombosis, which may be microvascular or involve larger blood vessels. Overt DIC can develop from latent, compensated DIC, which is a chronic disease that can develop as a result of the evolution of latent DIC.Clinical symptoms depend on the primary disease and include multiorgan failure caused by thrombosis as well as bleeding[6].

tenCate et al., termed DIC as a progressive condition and classified the manifestations of DIC into three phases, Phase 1 is a compensated activation state (with no obvious consumption of platelets and coagulation factors), phase 2 is a decompensated state (with prolonged clotting times and lower platelet counts), and phase 3 is a full-blown consumption of a number of clotting factors and marked depletion of platelets, as well as evidence of hemolysis[9]. Due to a decrease in the amount of circulating blood cells (platelets and red cells) and coagulation factors, DIC becomes clinically obvious at an advanced stage and a tendency to bleed is an early sign. Either it becomes apparent on its own due to formation of hemangiomas, petechiae, purpura, gastrointestinal bleeding, or hematuria), or during invasive processes. Internal organs, such as the adrenals, may bleed, leading in adrenal insufficiency, a fairly typical consequence in sepsis, or other potentially dangerous regions, such as the retroperitoneum or the cerebrum. Unless there is macrovascular thrombosis, such as in metastasized malignancies, clinical evidence of intravascular fibrin development is seldom seen. Intravascular fibrin formation has been found responsible for multiorgan failure[23].

2.6 PATHOLOGY OF DIC

"Thrombin is important for keeping the balance between coagulation and fibrinolysis in check. Clotting begins when tissue factor (TF) is exposed, allowing factor VIIa to activate factor X, resulting in the creation of thrombin (IIa). Thrombin promotes clotting by converting fibrinogen (Fgn) to fibrin, as well as anticoagulation by producing protein C (aPC), which degrades active factor V and VIII. It also plays an important role in anticoagulation by inducing tissue plasminogen activator (tPA) which generates fibrin-degradation products (fdp) regulates fibrinolysis by activation of fibrinolysis inhibitor (TAFI)[24]. As a result, increased thrombin production in vivo plays a key role in the development of DIC".

The following are the mechanisms involved in the pathophysiology of DIC.

- "High levels of thrombin production. The tissue factor (TF) factor over-exposure associated with the underlying disease process stops it. Tissue factor is an important part of the coagulation process. Inflammatory agents improve the appearance of TF in monocytes and endothelial cells, particularly through transcriptional methods. Excessive exposure to TF by activated monocytes and endothelial cells triggers activation. It is also highly exposed to other cancer cells. In meningococcal sepsis, phospholipid microparticles (MPs) are produced by active monocytes and platelet-monocyte complexes identified as the source of TF. FVa / phospholipids / Ca) and intrinsic tenase (FIXa / FVIIIa / phospholipids / Ca), leading to a proliferation of thrombin production.[25]. In addition, in severe sepsis, low-density lipoprotein particles (VLDL), especially VLDL complexes and C-reactive protein, are elevated and responsible for thrombin synthesis".

- "Platelet Function: DIC is characterized by a decrease in platelet count caused by platelet use during the fusion process. Platelet performance is attributed to this use of platelets. Because platelets are used during coagulation process, distributed intravascular coagulation interactions are often associated with decreased platelet count. Platelet utilization is a process related to platelet function. During platelet activation, the formation and release of procoagulant MPs, as well as the release of procoagulant and pro-inflammatory proteins and vasoactive molecules (i.e., FV, platelet factor 4, serotonin, adrenaline, prostaglandins), contributes to hypercoagulable and thrombus condition distribution. Platelets also contribute to the pathophysiology of DIC. Increased expression of membrane glycoprotein IIb / IIIa in its activated form, along with P-selectin, may contribute to the inhibition of microvascular occlusions in DIC patients"[26].
- "Anticoagulant pathway abnormalities, including tissue factor pathway inhibitor (TFPI), antithrombin (AT), and protein C (PC): Natural anticoagulant Tissue factor pathway inhibitor (TFPI), Antithrombin (AT), and protein C (PC) were severely damaged during the development of DIC. The tissue factor pathway inhibitor is a serine protease inhibitor that blocks Tomb-induced thrombin synthesis. Elevated TFPI levels have been observed in patients with DIC-induced sepsis, as well as improved TF levels, indicating TFPI-related deficiency to reduce TF pathway activation Antithrombin (FIIa), FXa, FVIIa, FIXa, and FXIa -serine protease inhibitors active thrombin (FIIa), FXa, FVIIa, FIXa, and FXIa. The high mortality rate in sepsis patients with DIC is due to low AT concentration. The decrease in AT during DIC is related to both hypercoagulable pregnancy status and increased use after thrombin-antithrombin (TAT) production. Capillary leakage, volume enlargement, and narrowing of the joints are all associated with AT deficiency in sepsis. AT inactivity

and depletion of AT with neutrophil elastase and other enzymes has been suggested as a possible cause of AT deficiency in DIC patients. C is a type of protein. The role of PC anticoagulant pathway in the development of DIC has been evaluated in experimental models of DIC caused by sepsis. Protein C deficiency is more common in patients with sepsis, due to factors such as increased appetite, decreased hepatic synthesis, and vascular leakage. Deficiency of a detected PC is linked to a hypercoagulable condition and high mortality in DIC caused by sepsis"[26].

"Insufficient fibrin degradation due to insufficient fibrinolysis or excessive fibrin and fibrinogen degradation: Fibrin breakdown is reduced when the fibrinolytic pathway is inhibited, which contributes to the pathophysiology of many kinds of DIC. During an inflammatory response, levels of Plasminogen activator inhibitor 1 (PAI-1), which plays an essential role in inhibiting fibrinolysis, rise. As a result, sepsis, which is connected to an intensive inflammatory response, leads to hypofibrinolytic DIC, microvascular thrombosis, and organ failure. Furthermore, patients with genetic variations that result in high PAI-1 concentrations have a higher mortality rate in severe sepsis. High thrombin concentrations activate a thrombin activatable fibrinolysis inhibitor (TAFI). Activated TAFI is a carboxypeptidase that cleaves lysine residues in fibrin that are necessary for tPA, plasminogen, and plasmin binding, making it more resistant to fibrinolysis. Reduced TAFI activity in patients with overt DIC is biological evidence of enhanced fibrinolysis. In individuals with low TAFI activity, death and multiple organ dysfunction syndrome are important predictors"[27]. Stimulation of the inflammatory process at the same time.

The pathology of DIC mediated by the various factors is presented in Figure 2-3[6].

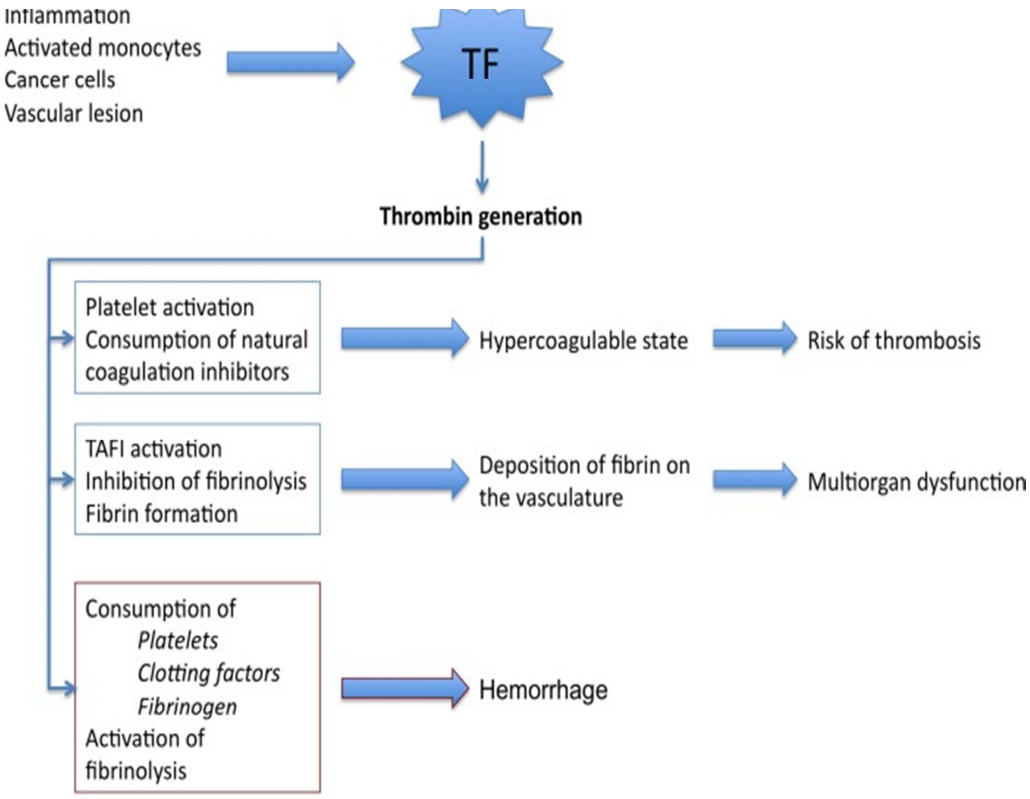

Figure 2-3. Pathology of DIC

The clinical presentation of DIC as seen in different primary diseases which lead to development of DIC[11].

Endothelial dysfunction and platelet activation. The vascular endothelium is engaged in the coagulation-inflammation interaction. A systemic inflammatory response with significantly raised circulating pro-inflammatory cytokines such as tumour necrosis factor, interlukin -1 (IL -1) and interlukin – 6 (IL-6) occurs in settings of abnormal endothelial function, such as in HELLP syndrome. This could result in an uncontrolled beginning of the coagulation cascade, which, combined with a decrease in anticoagulant protein activity found in systemic inflammation, could lead to DIC.

Trophoblast properties and coagulation system activation: Any condition that affects trophoblast results in the production of potent TF, which activates the coagulation cascade and causes uncontrolled thrombin production, which leads to the development of DIC, As seen in placental abruption and amniotic fluid embolism.

Hemorrhage: Acute obstetrical bleeding is connected to consumption coagulopathy and is an important cause of DIC. Since in this process, the mother loses lot of of blood and coagulation factors it could lead to development of DIC.

Disruption of liver function: The mother's vulnerability to bleeding and thrombosis increases as a result of the liver damage, which causes a decrease in the synthesis of anticoagulation proteins and coagulation factors.

2.7 DIAGNOSIS OF DIC

The diagnosis of DIC is based on the examination of numerous haemostatic measures rather than a single isolated test as well as information of the patients underlying disease. Prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT, low platelet count, low fibrinogen and high fibrin breakdown products such as D –dimer are some of the most often utilised tests for DIC diagnosis. Due to lack of specific guidelines for detection, the clinician and the specialized laboratory select the array of assays used. And as specification of limiting values per assay is difficult, due to lack of gold standard, the clinician has to analyse the combination of test results for the individual patient in order to reach a decision. There are three reasons for screening people who are at risk of having DIC: (a) to be informed about current bleeding risks, (b) to estimate the clinical status at the time (knowing that DIC might signal a bad clinical course), and (c) to decide on specific therapeutic treatments. Since DIC can be detected at various time points of its

development, use of overall clotting tests like APTT may not be useful at that particular point in time. Hence global coagulation screening (APTT, PT and thrombin clotting time as optional test) including a platelet count should be carried out and repeated at regular intervals during the disease. Any worsening of clinical course requires more screening for DIC. Early diagnosis of DIC requires employment of tests with high sensitivity for measuring coagulation activation such as F112, TAT, fibrin monomers, Fibrin degradation products (FDP), D-dimers. In clinical practice, the FDP is the most commonly utilized test.Quantification of antithrombin or protein C, or measurement of fibrinolytic inhibitor PAI-1 would be instrumental in measuring DIC progression[9].

Parameters for diagnosing DIC

Fibrinogen

Fibrinogen level in plasma level remains high for extensive periods inspite of its consumption in DIC. Hence, hypofibrinogenaemia has very low sensitivity in diagnosis of DIC and is only seen in severe forms of DIC.

Thrombocytopenia: A major finding in DIC is low platelets or quickly developing thrombocytopenia. The majority of individuals have moderate to severe thrombocytopenia (50,000/mm3), and those with 50,000 have a 4-5 times higher risk of haemorrhagic consequences compared to those with normal counts. It's a hallmark indication of DIC, but it's not specific because illnesses like acute leukaemia and sepsis, which are associated to DIC, can cause thrombocytopenia even when DIC isn't present. However, a stable platelet count implies that thrombin production has come to a standstill[28].

D-dimer and fibrin degradation products: Fibrin degradation product refers to fibrinolytic activity that is rapidly increasing and is higher in DIC. Assays that can detect neo antigens on these can detect the breakdown product D dimer. Its concentration rises in circumstances like trauma, surgery, or venous thromboembolism, making it non-specific for DIC. It also rises when there is a problem with the liver or kidneys. As a result, when used in conjunction with a falling platelet count and prolonged PT and aPTT, it is successful Because soluble fibrin monomer is exclusively produced intravascularly and is undetectable even in local inflammation and damage, it may be a better option for diagnosing DIC than FDP. It has a mediocre specificity despite its high sensitivity (90-100 percent). However, employing it instead of D-dimer in the ISTH scoring system can improve DIC diagnosis specificity[29].

Prothrombin time and activated partial thromboplastin time

In 50% of DIC cases, PT and aPTT are prolonged, which is mostly due to coagulation factor consumption but can also be linked to defective coagulation factor synthesis as well as massive bleeding, while in the other 50 % PT and aPTT are normal or shortened due to activated clotting factors thrombin or Xa. As a result, the existence of a normal PT or aPTT does not always imply that DIC is not present[30].

The parameters employed in diagnosis of DIC are presented in Table 2-3.

Table 2-3. Parameters employed in DIC diagnosis

Test	Changes	Causes other than DIC contributing to test result
Platelet count	Decreased	Sepsis, impaired production, major blood loss, hypersplenism
Prothrombin time	Prolonged	Vitamin K deficiency, liver failure, major blood loss
aPTT	Prolonged	Liver failure, heparin treatment, major blood loss
Fibrin degradation products	Increased	Hematoma
Protease inhibitors	Decreased	Liver failure, capillary leakage

Aside from the tests indicated above, only a few other markers have been found to be useful in the diagnosis of DIC. These molecular markers such as Thrombin- antithrombin (TAT) complexes and prothrombin fragments are not commonly used and standardised since they are not readily available. These include:

(i) Protein C and antithrombin (AT)

Reduced levels of these anticoagulants are associated with DIC severity and unfavorable outcomes, however sensitivity for early DIC is unclear because DIC can be detected by global coagulation tests at levels below 50 percent. Indeed, decreasing trends in PT platelets and D-dimer provide adequate robustness in the absence of PC or AT levels. When looked at as continuous variables, major clinical studies have demonstrated that PT and D-dimer changes not PC/AT are highly linked to death.

(ii) Thromboelastography

Automation of thromboelastography (TEG) allows for point-of-care coagulation testing, resulting in a more accurate image of the hemostatic situation. Sivula et al[31] discovered hypocoagulation in people with overt DIC, but hypercoagulation in people who didn't have it. TEG alterations in DIC-relevant illnesses have been associated to both hypercoagulability and hypocoagulability, and a comprehensive evaluation of TEG in sepsis corroborated this variability

(iii) Potential assays

"levels of circulating histones and histone – DNA complexes as well as double stranded cell free DNA, may have translational importance in DIC patient because DAMPs are becoming more significant in cross talk between coagulation, inflammation and innate immunity in 199 people with suspected DIC, histone-DNA complexes and cell free DNA levels were shown to be linked to the severity of DIC scores"[32].

2.7 DIAGNOSIS OF DIC IN PREGNANCY

Although these tests are used to diagnose DIC in pregnant women, they are difficult to perform since the concentrations of most coagulation factors with the exception of factor XI are significantly increased during pregnancy, resulting in a significant shortening of the PT and aPTT. Even if coagulation were used, the overall clot time would most likely be within non-pregnant norms. To interpret the continuing DIC process, serial variations in the PT and aPTT must be examined. Similarly, physiological thrombocytopenia of pregnancy must be taken into account when assessing the platelet count in DIC. For recording increased thrombin production, a

serial decline is more illuminating than a single test. In addition, fibrinogen analysis may be problematic during pregnancy. Only 46 patients (8.6%) with overt DIC (not related to pregnancy) had low plasma fibrinogen levels (less than 1 g/L), indicating that it is not sensitive for DIC, according to Levi et al. This could be a concern during pregnancy because the level of fibrinogen is double what it is normally, making the reading ambiguous. According to Charbit et al[33] fibrinogen can be used to predict the severity of PPH. The French PPH study group collected from 128 women, 50 with PPH. From admission till 24 hours afterward, 72 serial coagulation tests were done which showed that fibrinogen was the only marker related to PPH development.

D-Dimer levels, which anticipate cross-linked fibrin production and so function as a diagnostic for DIC, are already high in normal pregnancy, making DIC detection in pregnancy ineffective.

2.8 DIC SCORING SYSTEM

"Diagnostic scoring techniques that assess clinical and laboratory factors are now used to diagnose DIC. The ISTH, the Japanese Ministry of Health and Welfare (JMHW), the Japanese Association for Acute Medicine (JAAM), the British Committee for Standards in Haematology (BCSH), and the Italian Society of Thrombosis and Hemostasis (SISET) recommended five distinct diagnostic scoring systems for DIC[6]. An ISTH panel has established a grading system for overt DIC (Table 2-4), which is derived using a five-step diagnostic approach based on laboratory data. For the diagnosis of overt DIC, a score of 5 or above from four criteria was considered relevant, and it was proven to be sensitive to both infectious and non-infectious causes of DIC. It had a 91 percent sensitivity and a 97 percent specificity. Platelet count, PT, a fibrin breakdown product (such as D-dimer), and

fibrinogen levels were all used in the ISTH system. Five or more points showed DIC, while lower grades suggested non overt DIC. Because DIC is linked with fewer clinical characteristics, it necessitated a more complex composite score that has yet to be validated. The INR was used in place of the PT in this scoring system, making consistency and standardization easier. By identifying the best threshold value for the fibrin-related marker, the system's accuracy was increased. The Acute Physiology and Chronic Health Evaluation (APACHE-II), which combines intensive care measurement techniques with the DIC score, can be a powerful technique for predicting DIC prognosis .Japan has developed and assessed composite ratings comparable to those used in the United States. The most significant difference between ISTH and Japanese scores is the presence of a greater number of people with haemato-oncological disorders[20].

Table 2-4: ITSH diagnostic scoring system for overt DIC.

1.Risk assessment: Does the patient have an underlying disorder known to be associated with overt DIC?
If yes: proceed
If no: do not use this algorithm
2. Order global coagulation tests (prothrombin time, platelet count, fibrinogen, fibrin related marker)
3. Score the test results
Platelet count (>100 = 0, 3 but 6 s = 2)
Elevated fibrin marker (D-dimer, fibrin-degradation products) (no increase = 0, moderate increase = 2, strong increase = 3)
Prolonged prothrombin time (3 but 6 s = 2)
Fibrinogen level (>1 g/L = 0)
4. Calculate score

2.9 DIC SCORING SYSTEM IN PREGNANCY

The ISTH DIC score was created in 2001 to solve the issue of facilitating DIC diagnosis. An abnormal result has been linked to the prognosis of patients admitted to intensive care unit and has a high diagnostic performance for the detection of DIC in the non pregnant condition. In normal state, these scores serve not only as a diagnostic tool but also help in prediction of DIC although the correlation between them is low. However, these scores are not adjusted for the physiological changes observed in pregnancy, hence affecting their efficiency during pregnancy. Erez et al. Taking these considerations into account, researchers created a pregnant modified DIC score using only three ISTH DIC score parameters (platelet count, fibrinogen levels and PT difference) and found that it had an 88 percent sensitivity and a 96 percent specificity.Erez et al. evaluated the diagnostic characteristics of both scores in women with abruption to verify the score vs. the ISTH DIC score. 150 (21.93 percent) of 684 patients with placental abruption required blood transfusions, while 43 (6.29 percent) had DIC. At a cutoff point of 26, the pregnancy modified DIC score had an 88 percent sensitivity and a 96 percent specificity. Table 2-5 depicts evaluation of pregnancy modified DIC score[11].

Table 2-5. Evaluation of pregnancy modified score

Parameters	ISTH score	Pregnancy Modified score Erez et al.
Platelet count (109/L)	>100 = 0 <100=1 <50=1	>185 = 0 100-185 =1 50-100=2 <50=1
Fibrin-related markers	no increase: 0 moderate increase: 2 strong increase: 3	
Prothrombin time (value of patient/normal value)	<3s=0 ≥3 s but <6s=1 ≥6s=2	<0.5 = 0 0.5–1 = 5 1.0–1.5 = 12 1.5 = 25
Fibrinogen level (g/L)	<1.0 = 1 >1.0 = 0	3.0 = 25 3.0–4.0 = 6 4.0–4.5 = 1 >4.5 = 0
Calculated score	>5: compatible with overt DIC; repeat scoring daily <5: suggestive (not affirmative) for non-overt DIC; repeat next 1–2 days	>26 high probability

2.10 DIFFRENTIAL DIAGNOSIS OF DISSEMENATED INTRAVASCULAR COAGULOPATHY

Differential diagnosis of DIC includes:

1. Excessive Blood loss
2. Thrombotic microangiopathy
3. Heparin induced thrombocytopenia
4. Vitamin k deficiency
5. Liver impairment

Additional diagnostic clues which help improving its difference from DIC is shown in Table 2-6[7].

Table 2-6. Differential diagnosis of suspected DIC

Differential diagnosis	Additional diagnostic clues
DIC	Prolonged aPTT and PT, high concentration of fibrin split products, reduced levels of physiological anticoagulant factors (antithrombin, protein C)
Massive blood loss	Extensive bleeding, low hemoglobin, prolonged aPTT and PT
Thrombotic microangiopathy	Schistocytes in blood smear, Coombs-negative hemolysis, fever, neurologic symptoms, renal insufficiency, coagulation times mostly normal, PT and aPTT normal
Heparin-induced thrombocytopenia	Heparin use, venous or arterial thrombosis, positive HIT test (typically an ELISA for heparin-platelet factor IV antibodies), platelet rebound following heparin discontinuation; coagulation times are usually normal; PT is normal (aPTT may be prolonged due to heparin)
Vitamin K deficiency	PT prolonged, aPTT normal or slightly prolonged, normal platelet count
Liver insufficiency	PT and aPTT prolonged, platelets (moderately) low, liver test abnormalities, hypersplenism, jaundice

2.11 CLOT OBSERVATION TEST

Diagnosis of fibrinolysis can also be done by other methods like clot observation test, which is an age old test and involves observing the clot to detect presence of fibrinolysis. In fibrinolytic patients the clot lacks in bulk is soft and dissolves within 4 hours whereas in more intense conditions, dissolution of blood is observed in an hour. It has been proved to be a trusted method for detection of hypofibriniogenemia [34]. In this method 5 mL of the patients' blood into is withdrawn into a clear dry tube and monitored for clot formation and stability Unlike normal blood, which clots in 8 to 12 minutes and is stable for at least 24 hours, fibrinogen shortage causes the blood to either not clot or dissolve completely in 30 to 60 minutes. This observation indicates hypofibrinogemia and fibrinolytic activity are indicated. In a study by Poe 125 patients undertook clot observation test to show the correlation between fibrinolytic activity and excessive surgical bleeding. 23 of the 125 individuals evaluated had atypical clots, and two of them had significant bleeding. Clot observation tests were used to diagnose five more individuals who appeared to have bleeding tendencies. The blood either didn't clot or formed soft, unstable clots that retracted abnormally. Freshly obtained venous blood failed to clot or form a stable clot of normal size which indicated the presence of defective clotting mechanism.

Hypofibrinogenemia is studied by measuring fibrinogen level. But instead of these tests a simple clot observation test can be employed. According to Pattinson, 1976[14], this test is easy to carry out, inexpensive, requires no special equipment and only a small amount of blood, and provides useful information. When paired with a regular platelet count,bleeding time, activate partial thromboplastin time and a

detailed history, the clot observation test is a valuable screening tool for bleeding patients.

"Fibrinogen levels have been linked to the early diagnosis of abnormal pregnancies caused by coagulation abnormalities in several investigations. In the year 2016, a study was conducted to discover the levels of fibrinogen in abnormal pregnancy, such as pregnancy-induced hypertension (PIH), intrauterine fetal death (IUFD), missed abortion, and abruptio placenta. It involved 150 patients, 50 of whom were controls and 100 of whom were patients, with 40 of them having PIH, 25 having IUFD, 25 having missed abortions, and 10 having Abruptio placentae. The patient's fibrinogen levels were checked. The fibrinogen levels in PIH (Control mean 442.0, S.D. 43.38, Test mean 296.0, S.D. 48.03, p0.001), IUFD (Control mean 442.0, S.D. 43.38, Test mean 262.4, S.D. 20.06, p0.001), missed abortion (Control mean 442.0, S.D. 43.38, Test mean 250.80, S.D. 26.13, p0.001), and abrupt .This led to the conclusion that plasma fibrinogen estimate is useful not only in the early detection of haemostatic failure but also in guiding replacement therapy during the fibrinopenic state"[10].

CHAPTER– 3

METHODOLOGY

3.1 STUDY DESIGN

The present study was a hospital based prospective, observational study to compare clot observation test with serum fibrinogen level in pregnant women diagnosed with coagulation disorder previously or during pregnancy. This study was conducted at KLES Dr. Prabhakar Kore Charitable Hospital, Belgaum for a period of 18 months. Data and samples were collected from eligible pregnant women who were briefed about the nature of the study. Patients expressing their willingness to participate in the study were enrolled after obtaining a written informed consent.

3.2 STUDY SETTING

The study was conducted at the Department of Obstetrics and Gynecology of KLES Dr. Prabhakar Kore Charitable Hospital, Belgaum, Karnataka. The hospital is a clinical training facility that provides free health care to the underprivileged in basic specialties. KLES Dr. Prabhakar Kore Charitable Hospital is recognized by the Medical Council of India, and Government of Goa along with industries in Maharashtra and Karnataka.

3.3 STUDY PERIOD

The study was conducted for a period of 18 months (from January, 2020 to June, 2021). The study period included enrolment of participants, data collection, analysis and reporting.

3.4 STUDY POPULATION

The study population consisted of pregnant women suspected to have coagulation disorders and admitted in labor room at the Department of Obstetrics and Gynecology wards and wards of Intensive Coronary Care Unit (ICCU) at KLES Dr. Prabhakar Kore Charitable Hospital, Belgaum during the study period fulfilling the inclusion criteria and consenting to participate in the study.

3.5 SAMPLE SIZE

Sample size was obtained by the formula:

$$n = Z^2pq/d^2$$

Where:

n = sample size

Z = 1.96 corresponding to 95% confidence interval

p = proportion of participants (0.093% for this study, based on statistics for medical records department [MRD] of the hospital).

q= 1-p

d = margin of error set at 5% (0.05)

The minimum sample size was calculated as follows:

n = 1.96 x 1.96 x 0.093 x 0.907 / 0.0025

=129.6 (rounded off to 130).

Hence, the minimum sample size was taken to be 130.

3.6 SAMPLING METHODS

Universal sampling method was adopted for this study.

3.7 SELECTION CRITERIA

3.7.1 INCLUSION CRITERIA:

Pregnant women admitted to the hospital and diagnosed with the following conditions were included in the study:

- Intra uterine fetal death (IUFD)
- Abruptio placentae
- Eclampsia
- Postpartum hemorrhage (PPH)
- Sepsis
- HELLP syndrome
- Missed Abortion.

3.7.2 Exclusion criteria:

- Patients not willing for participation in the study
- Pregnant women admitted with treatment history of drugs that can alter coagulation profile including heparin, aspirin

3.8 DATA COLLECTION AND SAMPLING TECHNIQUES

Data was collected conveniently among the eligible women participating in the study. Eligibility was sought by checking their files for the diagnosis. Investigations for clot observation test and DIC profile were carried out and correlated in order to determine the modality accurate for early detection of pregnancy complicated with DIC.

The followings parameters and methods were used for data collection and sampling:

3.8.1 Socio-demographic characteristics

Data on socio-demographic and clinical characteristics like age, obstetric history and detailed history about other associated conditions were collected via interview and use of structured questionnaires prior to collection of blood samples for clinical investigations. Findings of clinical examination and subsequent systemic examination were recorded on a predesigned and pretested proforma.

3.8.2 Bedside Clot observation test (COT)

COT was performed by taking 5ml of venous blood from the patient in a plain tube. The tube was held in fist to maintain body temperature and inverted three to four times and the following observations were made (Table 3-1):

Table 3-1: Interpretation of COT

Sl. No.	Observation	Interpretation
1	Clotting time < 6 mins.	Fibrinogen level > 150 mg per 100 ml
2	No clot formed within 30 mins.	Fibrinogen level probably < 100 mg per 100 ml

3.8.3 Serum fibrinogen levels

Serum fibrinogen levels were determined from COT assay as described above.

3.8.4 Disseminated Intravascular Coagulation (DIC) profile

The followings tests were carried out for DIC profile of study participants:

i. Platelet count

Platelet count was carried out by automated hematology analyzer. Venous blood was collected with anticoagulant and the sample was mixed by inverting. The instrument's analysis mode was set to 'Whole Blood' and it was ensured that 'Ready' was displayed in the status indicator of the instrument. Sample tube was placed in the sample probe after removing the cap and analysis was started. Results were displayed after 60 seconds of analysis and were noted down.

ii. D-Dimer levels

D-Dimer levels were estimated in the collected blood sample by an enzyme-linked immunoassay using an automated analyzer.

iii. **Activated partial thromboplastin time (aPTT)**

aPTT was analyzed in blood sample of study participants using actin and calcium chloride as reagents in an automated aPTT analyzer. 50 µl of venous blood collected with anticoagulant was added to the cuvette of the analyzer and analysis was started. After 1 minute, aPTT reagent was added followed by waiting for 3 minutes. After 3 minutes, reading was noted down.

iv. Prothrombin time/international normalized ratio (PT/INR)

For estimation of prothrombin time (PT), 2 ml of venous blood collected with anticoagulant trisodium citrate was centrifuged to obtain plasma. 50 µl of plasma was added to the cuvette of automated blood coagulation analyzer and analysis was started. After 1 minute, 100 µl of prothrombin time reagent was added and reading displayed was noted down. INR was calculated from PT values of the study participants using the following formula:

$INR = (\text{Patient PT}/\text{Mean PT})^{ISI}$

Where:

Mean PT= Geometric mean of PT values of 20 healthy persons tested in the same laboratory.

ISI= International sensitivity index, derived by comparing with a reference thromboplastin of known ISI. ISI is specific to each reagent and instrument combination.

3.9 FOLLOW UP

Participants of the study were followed up from the time of diagnosis of coagulation disorders post admission till the delivery in order to evaluate the complications developed due to coagulation disorders.

3.10 STATISTICAL ANALYSIS

Analysis of collected data was done using descriptive statistics.The information gathered was coded and entered into a Microsoft Excel spreadsheet. Statistical Package for the R and graph pad prism version 9 was employed for statistical analysis and interpretation of collected data.Rates, ratios, and proportions were used to express categorical data. Chi-square test was used to measure the strength of associations between the categorical data. Continuous data were summarized as mean ± standard deviation (SD) and independent sample 't' test was used to compare the data. Probability values ('p' values) of <0.05 at 95% confidence interval were considered to be statistically significant.

3.11 ETHICAL ISSUE AND ETHICAL CLEARANCE

An informed choice was given by each participant based on the participant's full understanding of the method or procedure, including its characteristics, action and possible risks and benefits. The participant's consent sought and obtained after adequate information about all aspect covered by the study. During the process of obtaining consent, the rights to decline participation or to withdraw participation at any time of the study should they wish to do so, were emphasized. Information regarding privacy and confidentiality of the patient was provided. It was also ensured that the participants were educated about warning signs and the need for follow- ups.

Ethical clearance for this study was obtained from the Institutional Ethics and Research Committee, Jawaharlal Nehru Medical College, Belgaum, Karnataka in prescribed format.

OBSERVATION AND RESULTS

Data contains measurements on 175 subjects whose age ranged from 18 years to 45 years with mean age 26.25 ± 5.07 years. Table 4-1 shows the baseline parameters of all the subjects included in the study.

More than 80% (141 cases) of the subjects had more than or equal to 150 serum fibrinogen level with 19.43% (34 cases) having serum fibrinogen level of less than 150. Around 24% (45 cases) of pregnant women has less than or equal to 1.5 lac platelet count and more than 133 cases (n=76) had more than 1.5 lac platelets. Similarly, D-dimer level was less than or equal to 5000 in 21.71% (n=38) cases. Majority of the pregnant women had less than 1.5 sec aPTT and PT/INR whereas 5.7% of them had more than or equal to 1.5 sec aPTT and PT/INR respectively. The bed side COT time were less than 6 in 86.8% (n=152) cases and more than or equal to 6 in 13.14% (n=23) cases (Table 4-1)

Table 4-1: Baseline coagulation parameters of pregnant women included in the study

Variables	Sub-Category	Number of subjects (%)
Serum Fibrinogen	<150	34 (19.43%)
	≥150	141 (80.57%)
	Mean ± SD Median (Min, Max)	324.06 ± 153.3 357 (38,727)
Platelet count	≤1.5lacs	42 (24%)
	>1.5 lacs	133 (76%)
	Mean ± SD Median (Min, Max)	172.03 ± 122.37 180 (0.09,629)
D-dimer	< 5000	137 (78.29%)
	≥ 5000	38 (21.71%)
	Mean ± SD Median (Min, Max)	1806.08 ± 1902.2 868 (80, 7066)
aPTT	Mean ± SD Median (Min, Max)	1.007 ± 0.68 0.9(0.18, 5.7)
	≥1.5	10 (5.7%)
	<1.5	165 (94.2%)
PT/INR	Mean ± SD Median (Min, Max)	1.04 ± 0.41 0.93 (0.12, 3.65)
	≥1.5	12 (6.86%)
	<1.5	163 (93.1%)
Bedside COT (min)	<6	152 (86.8%)
	≥6	23 (13.14%)
	Mean ± SD Median (Min, Max)	4.35 ± 12.1 5.2 (3.1, 12.55)

From Spearman's rank Correlation test, we observe that, serum Fibrinogen level and Platelet count have significant negative correlation with COT. There is significant positive correlation of D-dimer, aPTT and PT/INR with COT. Correlation of different variables with COT are given in Table 4-2.

Table 4-2: Correlation of different variables with COT.

Variables	**Spearman's rank Correlation Coefficient**	**p-value**
Serum Fibrinogen	-0.26	**0.0005***
Platelet count	-0.21	**0.004***
D-dimer [@]	0.15	**0.04***
aPTT	0.20	**0.005***
PT/INR	0.19	**0.01***

*Abbreviation: S-Spearman's rank correlation test, * indicates statistical significance. In D-dimer >5000 is replaced by 5000 while calculating Correlation*

From Welch's t test, we observe that there is significant difference in mean COT with Serum Fibrinogen. Further, we observe that, COT is more if Serum Fibrinogen is < 150. There is significant difference in the mean COT with D-dimer. Further, we observe that, COT is more if D-dimer is ≥ 5000. (Table 4-3).

Table 4-3: Comparison of COT with different variables.

Variables	**Sub-Category**	**Mean ± SD**	**Median (Min, Max)**	**p-value**
Serum Fibrinogen	<150	6.06 ± 1.6	6 (4,10)	**<0.0001^{WT}***
	≥150	4.3 ± 0.6	4 (3,5)	
Platelet count	≤1.5lacs	5. 27 ± 1.6	5 (3, 12)	0.008*
	>1.5 lacs	4.5 ± 0.8	5 (3,8)	
D-dimer	< 5000	4.93 ± 0.6	5 (4, 6)	**<0.0001^{WT}***
	≥ 5000	6.25 ± 2.2	6 (3,12)	
aPTT	≤1.5	4.6 ± 0.8	5 (3,8)	**0.016**
	>1.5	6.9 ± 2.4	6 (4,12)	
PT/INR	≤1.5	4.5 ± 0.9	5 (3,12)	0.004*
	>1.5	6.5 ± 1.9	7 (3,10)	

*Abbreviation: WT – Welch's t test, WA – Welch's ANOVA, **

From Mann Whitney U test, we observe that, there is no significant difference in distribution of COT over registration status

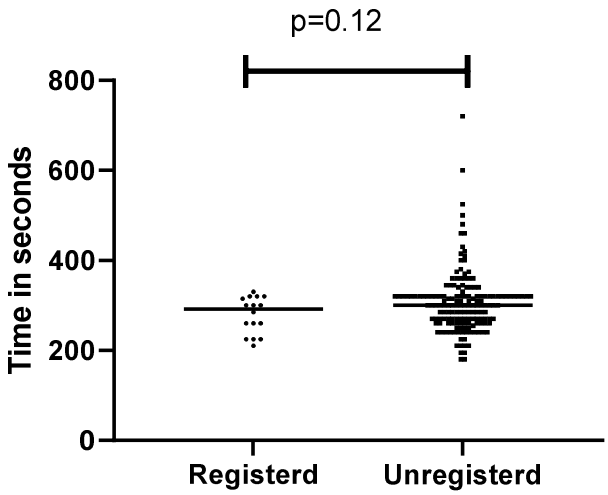

Figure 4-1. Distribution of COT over registration status

From Chi square test, we observe that, there is significant association of Serum Fibrinogen with Bedside COT. We also observe that; D-dimer is significantly associated with bedside COT. From Mann Whitney U test, we observe that, there is significant difference in the distribution of platelet count, aPTT and PT/INR with bedside COT. There is no significant difference in distribution of age with bedside COT. The following table (Table 4-4) gives the comparison of different variables with bedside COT.

Table 4-4: Comparison of different variables with bedside COT.

Variables	**Sub-Category**	**Bedside COT**		**p-value**
		<6	**≥6**	
Serum Fibrinogen	<150	17 (9.7%)	19 (10.8%)	**0.001**[C]
	≥150	134 (76.5%)	4 (2.2%)	
Platelet count (‘000s)	Mean ± SD Median (Min, Max)	185.45 ± 120.9 202 (0.16, 629)	83.93 ± 92.7 0.97 (0.09, 230)	**< 0.0001**[MW]*****
	≤1.5lacs	49 (32.2%)	16 (69.5%)	**0.0006**
	>1.5 lacs	103 (67.7%)	7 (30.4%)	
D-dimer	<5000	129 (73.7%)	8 (4.5%)	**0.0001**[C]
	≥5000	23 (13.14%)	14 (8%)	
aPTT	Mean ± SD Median (Min, Max)	0.92 ± 0.45 0.88(0.18, 5.69)	1.57 ± 1.2 1.1(0.72, 5.7)	**< 0.0001**[MW]*****
	≤1.5	149 (98.02%)	16 (69.5%)	**<0.0001**
	>1.5	3 (1.9%)	7 (30.4%)	
PT/INR	Mean ± SD Median (Min, Max)	0.96 ± 0.25 0.92(0.12, 2.9)	1.5 ± 0.7 1.39 (0.83, 3.6)	**< 0.0001**[MW]*****
	≤1.5	149 (98.02%)	14 (60.8%)	**<0.0001**
	>1.5	3 (1.9%)	9 (39.1%)	

*Abbreviation: C – Chi square test, MW – Mann Whitney U test, * indicates statistical significance.*

Correlation between bedside COT values and distribution of platelet count, serum fibrinogen, a PTT and PT/INR revealed significant association in almost all cases (Table 4-5). Increased platelet count had more subjects in <6 bedside COT time and more than 150 serum fibrinogen level had less than 6 bedside COT time in 89.4% (n=135 cases). Similarly, 65.21% (n=15) cases had serum fibrinogen in the range of 50-100. As per the (0.5-1 sec) aPTT and PT/INR values nearly 86.1% (n=131) and 74.3% (n=113) had in less than 6 bedside COT time (Table 4-5)

Table 4-5. Correlation between coagulation parameters and Bedside COT

Variables	**Sub-Category**	**Bedside COT**		**p-value**
		<6	**≥6**	
Serum Fibrinogen	<50	0	3 (13.04%)	**<0.001**
	50−100	11(7.2%)	15 (65.21%)	
	100−150	6 (3.9%)	1 (4.3%)	
	>150	135 (89.4%)	4 (17.3%)	
Platelet count	<50000	12(7.9%)	7(30.4%)	**0.001**
	50000−100000	18 (11.8%)	5 (21.7%)	
	100000−185000	40 (26.3%)	6 (26.09%)	
	>185000	83 (52.9%)	5 (21.7%)	
aPTT	<0.5	2(1.3%)	0	<0.001
	0.5-1	131(86.1%)	7 (30.4%)	
	1-1.5	16(10.5%)	9(39.1%)	
	>1.5	3(1.9%)	7(30.4%)	
Pt/INR	<0.5	2(1.3%)	0	<0.001
	0.5-1	113(74.3%)	3(13.0%)	
	1-1.5	34 (22.3%)	10(43.4%)	
	>1.5	3 (1.9%)	10(43.4%)	

DISCUSSION

Pregnancy is complicated by conditions like Intra uterine fetal death (IUFD), abruptio placentae, eclampsia, postpartum hemorrhage (PPH), sepsis, HELLP syndrome and abortion can lead to terminal complication like DIC which is responsible for maternal morbidity and mortality.

So, proper diagnosis and treatment is required to minimize morbidity and mortality. The combination of multiple assays such as thrombin time, Activated partial thromboplastin time, prothrombin time, platelet count and D-dimer assay were performed to score DIC in obstetric patients[35].

From the onset of clinical manifestation of the DIC till the diagnosis of the DIC by laboratory test it might take as long as one to two hours, such a delay may lead to worsening of the condition. So that it is necessary to diagnose DIC at early subclinical stage and therapeutic measures should be initiated at earliest. Bedside clot observation test is used for early diagnosis of coagulopathy in obstetric emergency.

In this study, we have studied 175 patients with obstetric emergency; the mean bedside clot observation test is 4 minutes 35 seconds. The mean serum fibrinogen level in 175 patients is 324 mg/dl. Out of 175 patients, 9.7% patients had bedside clot observation test of less than 6 minutes with serum fibrinogen level less than 150mg/dl and 10.8% patients had bedside cot of more than and equal to 6 minutes with serum fibrinogen of less than 150mg/dl. 76.5% patients had bedside cot of less than 6 minutes with serum fibrinogen level more than and equal to 150mg/dl and 2.2% patients had bedside cot of more than and equal to 6 minutes with serum fibrinogen level more than and equal to 150 mg/dl.According to this study prolonged bedside COT is associated with low fibrinogen level. Thus, bedside COT can be used as an early measure to diagnose DIC rather than waiting for the laboratory DIC profile

which usually takes 1-2 hours in obstetric emergency patients and prompt treatment can be started , which can help to prevent patient from further worsening of clinical condition.

Several studies have reported role of fibrinogen levels in early detection of abnormal pregnancies arising from coagulation disorders. In a study conducted by Kanchana A. et al they found fibrinogen levels decreased significantly in PIH, IUFD, DIC, missed abortion and abruption placentae. It was concluded that the estimation of plasma fibrinogen is helpful not only in the early diagnosis of haemostatic failure but also to guide replacement therapy during the fibrinopenic state[10].

In our study, platelet count has a direct correlation with bedside clot observation test i.e. When there is low platelet count bedside COT is prolonged. So, when a patient presents with a condition associated or leading to coagulopathy then instead of wasting the precious time in waiting for lab results decision can be taken based on bedside COT & prompt treatment like transfusion of blood and blood products can be started morbidity and mortality can be prevented.

Although In literature search we didn't find any studies comparing the reliability of bedside COT & platelets ,we at our center daily face the problem of waiting for lab results & in the meantime patient condition getting worsened .So, in order to see how far we can rely on bedside COT in cases suspected coagulopathy we have conducted this study and found that Bedside COT is good at predicting low platelets counts. aPTT and PT/INR has significant positive correlation with bedside clot observation test in detecting abnormal coagulation states. When aPTT, PT/INR is prolonged bedside COT is also prolonged. But usually aPTT, PT/INR alone is not useful in diagnosing coagulation disorders like DIC but they tell us the abnormal coagulation state of the patient thus giving us the clue for further evaluating the case.

But sometimes in overt DIC PT/INR will be prolonged. In such cases where we are clinically sure that the patient is in DIC & need prompt transfusion or correction of coagulation bedside COT can be helpful as evidence to our clinical decision despite waiting for the lab results which takes 1-2 hours.

In most of the cases in our study, patients with prolonged bedside clot observation test has prolonged aPTT and PTINR.In the study conducted by vani et all most of the DIC patients had elevated aPTT and PT. The aPTT alone did not have statistical significance with DIC scores[45]. According to study done by O Erhabor et al the PT and aPTT were significantly higher in women with obstetric haemorrhage compared to those of non haemorrhagic pregnant women. PT and aPTT are used in the management of coagulopathy in obstetric hemorrhage.PT and aPTT may identify significant coagulation impairment, but they test limited parts of coagulation and do not help diagnose the underlying defect[44].

In a study conducted by Cunningham et al found prolongation of PT and aPTT is observed in 50% of DIC cases which is mostly caused by consumption of coagulation factors but can also be associated with defective synthesis of coagulation factors as well as massive bleeding in the other 50 % cases PT and aPTT are normal or shortened because of activated clotting factors like thrombin or Xa. Hence presence of normal PT or APTT does not always indicate an absence of DIC [37].

Normal pregnancy causes the maternal plasma D-dimer concentration to increase progressively from conception until delivery. Hence, D-dimer is of little importance in the early detection of DIC in pregnancy. However, in our study there is positive correlation of D-dimer with bedside COT for early detection of coagulopathy in obstetric emergency. According to Katrine K. Hedengram et al D-dimer is of limited use during pregnancy due to high numbers of false positives. An elevated D-

dimer threshold throughout the pregnancy cannot be recommended. A gestational age –specific reference interval is required, and even then, D-dimer values should be interpreted with precaution due to large number of outliers[46].

In the present study we have found that when the D-Dimer is >5000 bedside COT is also prolonged. But when D-Dimer is <5000 we didn't find any statistically significant difference between bedside cot & D-dimer .Generally in pregnancy D-dimer is seen to be moderately elevated and thus D-Dimer alone is not so significant is diagnosing coagulation abnormalities but still it tells about the severity of the state. Thus, when we do a bedside COT in conditions where patients are not in coagulopathy but that condition is known to be associated with increased D-dimer levels like thromboembolisms, in such cases when we get a prolonged bedside clot observation test we have to think of the ongoing fibrin degradation & accumulation of degraded fibrin products and take measures accordingly.

With our study we conclude that the each parameters of DIC profile has significant correlation with bedside COT. Thus, from our study we found that overall DIC profile has got association with simple bedside clot observation test which can be used as an assay for measuring the severity of clinical condition and accordingly treatment measure can be started at earliest despite waiting for laboratory result.

CONCLUSION

DIC that occurs due to stimulation or activation of the coagulation process leads to secondary fibrinolysis or thrombotic microangiopathy. DIC is caused by multiple factors including obstetric problems, infection, malignancy, tissue destruction, release of tissue factors and severe trauma. A single analytical technique is not enough to judge and diagnose DIC. The combination of multiple assays such as peripheral blood smear, fibrin degradation product assay, thrombin time, activated partial thromboplastin time, prothrombin time, platelet count and D-dimer assay were performed to score DIC obstetric patients. Due to complexity of the analysis, Bedside COT analysis was compared with the laboratory tests (DIC profile). Significant difference in the distribution of Platelet count, aPTT and PT/INR with bedside COT was observed. Correlation between bedside COT values and distribution of serum fibrinogen, platelet count, aPTT and PT/INR revealed significant association in almost all cases. Bedside COT values can thus be an alternative to much time consuming laboratory test for early diagnosis of DIC.

SUMMARY

The study was designed to evaluate the role of clot observation test, a simple bedside test, as compared to the time consuming laboratory method of serum fibrinogen level in the diagnosis of DIC during early stages. The study included 175 subjects ranging from 18 years to 45 years with mean age 26.25 ± 5.07 years was characterized for the baseline coagulation parameters. More than 80% (141 cases) of the subjects had more than or equal to 150 serum fibrinogen level with 19.43% (34 cases) having serum fibrinogen level of less than 150.Majority of the patients (76%) had more than 1.5 lac platelets followed by around 24% (45 cases) of pregnant women having less than or equal to 1.5 lac platelet count. Similarly, D-dimer level was less than or equal to 5000 in 21.71% (n=38) cases. Majority of the pregnant women had less than 1.5 sec aPTT and PT/INR whereas 5.7% of them had more than or equal to 1.5 sec aPTT and PT/INR respectively. The bed side COT time were less than 6 in 86.8% (n=152) cases and more than or equal to 6 in 13.14% (n=23) cases. These baseline coagulation parameters were correlated with clot observation time, where significant positive correlation of D-dimer, aPTT and PT/INR with COT and negative correlation of platelet count and serum Fibrinogen was observed. No significant association between age and COT was observed based on Spearman's rank Correlation test. Whereas based on Welch's t test, a significant difference in the mean COT with D-dimer was observed. COT is found to be higher with Serum Fibrinogen is < 150 and D-dimer being ≥ 5000. Chi square test showed the significant linkage with serum fibrinogen, D-dimer and Bedside COT.

Based on Mann Whitney U test a significant difference in the distribution of Platelet count, aPTT and PT/INR with bedside COT was seed. No significant association with age and bedside COT. Since nearly no consensus was observed with

coagulation parameter range with DIC severity. Therefore, the parameters are distributed under different ranges to find their association with Bedside COT and DIC occurrence. Majority of the pregnant women (73.14%) had more than 250 serum fibrinogen level, nearly 25.71% (n=25) cases had more >250000 platelet, less than 1000 D-dimer level was found in 56% of cases. The aPTT and PT/INR distribution was similar among the study population whereas a majority of 78.8 and 66.28% of cases having 0.5-1 level of a PTT and PT/INR respectively. The second majority of them had 1-1.5 unit of a PTT and PT/INR ie., 14.28% and 25.71% respectively. Correlation between bedside CoT values and distribution of serum fibrinogen, platelet count, aPTT and PT/INR revealed significant association in almost all cases. Increased platelet count and more than 150 serum fibrinogen level had more subjects in <6 bedside COT time and a less than 6 bedside COT time in 89.4%. Similarly, 65.21% (n=15) cases had serum fibrinogen in the range of 50-100. As per the (0.5-1 sec) aPTT and PT/INR values nearly 86.1% (n=131) and 74.3% (n=113) had in less than 6 bedside COT time.

Based on the above coagulation parameters, distribution and correlation analysis reveals bedside COT analysis could be considered for the diagnosis of obstetric DIC patients due to its significant association with serum fibrinogen levels, platelet count and aPTT

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ANNEXURE - I - ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
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Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 205 **Date: 24/12/2019**

To

REG. NO. BJ0119007
PG student in Obstetrics & Gynaecology,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CLOT OBSERVATION TEST FOR EARLY DETECTION OF COAGULOPATHY IN OBSTETRIC EMERGENCY – A ONE YEAR PROSPECTIVE STUDY"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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

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

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ANEXXURE II- CONSENT FORM

Title of Research Study: "CLOT OBSERVATION TEST FOR EARLY DETECTION OF COAGULOPATHY IN OBSTETRIC EMERGENCY**:**A PROSPECTIVE STUDY"

Principal Investigator: -	Co-Investigator
REG. NO. BJ0119007	Dr. _____
Post Graduate student	PROFESSOR
Department of Obstetrics Gynaecology	Dept. of Obstetrics & & Gynaecology
J.N Medical College, Belagavi.	J.N Medical College, Belagavi.

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

"CLOT OBSERVATION TEST FOR EARLY DETECTION OF COAGULOPATHY IN OBSTRETICS EMERGENCY: A PROSPECTIVE STUDY".

Mrs. _____ we are requesting you to enroll yourself in study **"CLOT OBSERVATION TEST FOR EARLY DETECTION OF COAGULOPATHY IN OBSTRETICS EMERGENCY: A PROSPECTIVE STUDY".** Conducted by **REG. NO. BJ0119007**, Post Graduate in M.S. Obstetrics and Gynaecology under the guidance of DR._____, Department of Obstetrics and Gynaecology, J.N. Medical College, Belgaum under KAHER, Belagavi. The purpose of research study is to early detection of coagulopathy in obstretics emergency. I will be the investigator for our study. This study is not being

funded.I am going to give you information about this research project. Before you decide, you can talk to anyone you feel comfortable with about the research.

Objectives /purpose of study:

a) Respected Madam we request you to participate in our study as you are eligible for participating and your participation in this study is important as it helps us to study the early detection of coagulopathy disorder in obstetrics emergency, it would help us improve the perinatal outcome and would also help us to further prevent any complications in obstetrics patients with same condition.

b) Your participation in research is voluntary. Your decision whether to participate in the study or not will not change present or future health care services offered to you and will not affect your relationship with KLE's Dr. Prabhakar Kore Hospital, Belagavi. If you decide to participate you are free to withdraw at any time. All pregnant women meeting the inclusion criteria will be recruited in our study.

c) The purpose of research study is to know the perinatal outcome in early detection of coagulopathy in obstetric patients. I will be the investigator for our study. This study is not being funded.

Type of Study:

This is a hospital based observational study for early detection of coagulopathy in obstetrics emergency.

Participant selection

We are inviting all women with coagulation disorder after applying the exclusion criteria.

Voluntary Participation

Your participation in research is voluntary. Your decision whether to participate in the study or not will not change present or future health care services offered to you and will not affect your relationship with J.N. Medical College.

Procedure Involved:

If you agree to enroll yourself in this study, you will be interviewed regarding your present, past and family history, then you will be clinically examined in detail and investigated which may or may not cause pain. The procedures don't cause any temporary or lasting problems to you. Your co-operation is necessary as the investigations may be repeated number of times as required.

Risks and Benefits:

There are no potential risks and discomforts associated with any procedure involved in our study. The benefits of taking part in this research is that you can contribute to medical research to improvise treatment currently practiced.

Alternative:

There are no other options of treatment. If you decline to participate it will affect the results of our study and you will get the routine line of management. You will be informed about any new information that may affect your decision to participate in the study.

Withdrawal from study:

You can withdraw at any time from the study. There will be no penalty for withdrawal. You can be removed from the study if necessary

Privacy and Confidentiality:

The only people who will know that you are the research subject will be the members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission except:

a) In emergency to protect your rights and welfare.

b) If required by law.

Institutional/sponsor's policy:

In the event of any injury related to the study, treatment will be made available through KLE's Charitable Hospital & MRC, Belgaum. There is no compensation or payment for such medical treatment by law. If you come across any complications due to the study, you may contact **REG. NO. BJ0119007**, Post graduate student, Department of Obstetrics and Gynaecology, KLE's Hospital & MRC or by Ph. No: _____.

Financial Incentives for participation:

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator. You will not be reimbursed for any expenses for participation in this research.

Contact details:

In case you have any questions related to the study, in future or in case of study related injury or illness, you can contact **REG. NO. BJ0119007,** Post graduate student, Department of Obstetrics and Gynaecology, KLE's Hospital and MRC, Ph. No:_____ or Dr. _____, Dept. Of Obstetrics and Gynaecology, KLE's Hospital and MRC, Belagavi, Ph. No: _____.

If you have any queries about your rights as a study participant, you may contact Dr. Roopa M Bellad, Prof. of Pediatrics as Chairman of J. N. Medical College Institutional Ethics Committee on Human Subjects Research, Phone No.0831 2473777 ext-1527 at J. N. Medical College.

Authorization to Publish Results:

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential. Results of the study will be used to improve maternal and perinatal outcome.

Consent statement:

I, _____ voluntarily agree for participating in this study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read in my own vernacular language, including the risks and the benefits and having all my questions answered.

Participant Name : _____

Signature or the Left Thumb Print of Participant : _____

Investigators Name: _____ Signature: _____

Witness Name: _____ Signature: _____

Date: _____

SCREENING FORM

ID: | | | | | |

Date of screening (dd -mm-yyyy): _____

First name: _____

Middle name: _____

Last name: _____

Husband's name: _____

Age (years): _____

IP number: _____

Address: 1) House no-_____

2) Street-_____

3) Taluka-_____

4) District-_____

Phone number-_____

Inclusion criteria	
Diagnosed with coagulation disorder	
Intra uterine fetal death	
Abruption placentae	
Pre-eclampsia	
Postpartum haemorrhage	
Hydatidiform mole	

Exclusion criteria	
On treatment with apirin	
Heparin	
Tranexamic acid	
Other anticoaugulants	

"CLOT OBSERVATION TEST FOR EARLY DETECTION OF COAGULOPATHY IN OBSTRETICS EMERGENCY A 1 YEAR PROSPECTIVE STUDY".

PROFORMA

1. Name : _____
2. I.P. no : _____

3.

Registered	
Unregisterd	

3. Age :
    1. <18 ☐
    2. 18 – 35 ☐
    3. >35 ☐
4. Parity **:**
    1. Primi: ☐
    2. Multi: ☐
5. Chief complaints:

headache	☐	☐
Epigastric pain	☐	☐
Blurring of vision	☐	☐
Convulsion	☐	☐
Bleeding per vaginum	☐	☐
Other	☐	☐

6. Gestational age:

    LMP- _____

    EDD- _____

    POG- _____

7. Past history :

	Y	N
a. History of Bleeding disorders during pregancy	☐	☐
b. History of blood transfusion	☐	☐
c. History of PIH	☐	☐
d. Any other specify	☐	☐

8. Obstetric conditions :

	Y	N
a. Pre-eclampsia	☐	☐
b. Previous LSCS	☐	☐
c. Abruptio placenta	☐	☐
d. HELLP syndrome	☐	☐
e. Placenta previa	☐	☐
f. Septic abortion	☐	☐
g. Hydatidiform mole	☐	☐
h. IUFD	☐	☐
i. Others	☐	☐

9. Clot observation test : _____

10. DIC profile

a. Platelet count	☐
b. Sr.Fibrinogen levels	☐
c. D-dimer	☐
d. aPTT	☐
e. PT/INR	☐

ANNEXURE IV – MASTERCHART

OP Number	Age in year	Registered/unregistered	Gravida	Parity	Obs score	Presented with complaints	Diagnosis	Past history	Bedside COT	Platelet	Sr fibrinogen	D-dimer	aPTT	PT/INR
1021556	27	R	3	2	G3P2L1	Decrease fetal movement	G3P2L1 with 40 weeks 1 day pog with cephalic presentation with previous LSCS with IUFD in labour	Nothing significant	5 mins	269	411	780	0.8	0.91
1021496	28	R	3	2	G3P2L2	headache, vomiting	G3P2L2 with 29 weeks 2 days pog with previous LSCS with pre-eclampsia with breech presentation with foetal growth restriction	Nothing significant	5 mins 20 secs	242	295	367	1.14	0.85
1021799	34	UR	4	2	G4P2L2A1	Absent fetal movement	G4P2L2A1 with 33 weeks 6 days pog with cephalic presentation with IUFD with revealed abruption with pre eclampsia in active labourr	History of 2 pint pcv transfusion in previous pregnancy	4 Mins 30sec	76000	<50	>5000	>180	>120
1021874	25	UR	1	0	Primigravida	Bleeding per vagina	Primigravida with 35 weeks pog with cephalic presentation with revealed abruption with pre eclampsia	Nothing significant	4mins 10 secs	179	380	611	0.86	0.9
1021866	36	UR	3	1	G3P2L1A1	High BP recordings	G3P2L1A1 with 34weeks 6 days pog with previous LSCS with severe PE with FGR	History of imminent eclampsia in previous pregnancy	5mins 10 secs	172	417	840	180	>120
1022587	35	UR	2	1	G2P1L1	pain in abdomen with high BP reading	G2P1L1 with 35 weeks 6 days pog with cephalic presentation with previous LSCS with Severe pre eclampsia with clinical FGR in latent labour	Nothing significant	5mins 30secs	162	292	507	0.88	0.92
1022646	20	UR	1	0	Primigravida	Bilateral pedal edema	Primigravida with 37 weeks 1 day pog with cephalic presentation with severe pre eclampsia with clinical FGR	Nothing significant	4 mins	392	261	487	0.9	0.86
1022672	21	UR	2	1	G2P1L0	Absent fetal movement	G2P1L0 with 33 weeks 4 days pog with cephalic presentation with severe pre eclampsia with IUFD with abruption	History of PIH in previous pregnancy	4 Mins 30sec	252	306	5000	0.8	0.92
1022759	27	UR	1	0	Primigravida	Decrease fetal movement	Primigravida with 38 weeks 1 day pog with cephalic presentation with IUFD with absent left kidney hydronephrosis	History of hysterosalpingography	5 mins	158	417	378	0.82	1.09
1022844	26	UR	1	1	Primigravida	pain in abdomen with pv bleeding	Primigravida with 36 weeks 1 day of pog with cephalic presentation with IUD with PIH with moderate aneemia with DIC	Nothing significant	5 mins 20 secs	62000	97	1514	0.93	1.18
1023183	28	UR	3	1	G2P2L1A1	headache,blurring of vision	G2P1L1A1 with 33 weeks pog with cephalic presentation with imminent eclampsia with abruption	Nothing significant	5 mins	228	417	752	0.79	0.84
1023193	28	R	2	0	G2A1	High BP recordings	G2A1 with 38 weeks 2 days pog with cephalic presentation with severe pre eclampsia with moderate anemiaa	Nothing significant	4 mins 20 secs	232	393	617	0.82	0.94
1023351	30	UR	4	3	G4P3L2D1	Headache, blurring of vision	G4P3L2D1 with 31 weeks 1 day pog with cephalic presentation with IUFD with severe FGR with severe pre eclampsia	Nothing significant	5 mins	119	131	1225	0.79	0.8
1023626	27	UR	3	2	G3P2L1	headache, vomiting	G3P2L1 with 32 weeks 6 days pog with cephalic presentation with gestational hypertension with abruption with IUFD	Nothing significant	4mins 10 secs	137	198	1368	0.84	0.87
5561484	28	UR	1	0	Primigravida	Diarrhoea with jaundice	Primigravida with 34 weeks POG with Cephalic Presentation with AFLP with DIC with Sepsis	Nothing significant	6 mins	200000	82	>5000	1.61	1.42
5564951	28	UR	2	1	P2L2	Traumatic PPH	P2L2 with PND 1 with Traumatic PPH with DIC with SHOCK with ARDS	Nothing significant	10 mins	77000	81	>5000	2.45	3.65

994113	28	UR	3	2	P3L3	Pulmonary Thromboembolism with secondary PPH	Secondary PPH with Pulmonary Thromboembolism with Post Emergency Hysterectomy with DIC with Hypoxic Ischemia Brain Injury	Nothing significant	6 mins 55 sec	74,000	94	4245	4.66	2.46
5615113	30	UR	2	1	P2L2	Pain Abdomen	Ectopic pregnancy with Pulmonary Thromboembolism	Nothing significant	4 mins	141000	<50	>5000	0.81	1.14
1001534	20	UR	2	1	P2L1D1	Post emergency LSCS with DIC	P2L1D1 with Preterm Emergency LSCS done on 14/02/2020 in DIC with severe Aneamia (I-Abruption with IUD)	Nothing significant	5 mins 40 sec	69,000	<50	>5000	0.95	1.34
1000721	19	UR	1	0	Primigravida	C/o pain Abdomen & Yellowish discolouration of eyes	Primigravida with 36 weeks 4 days POG with cephalic presentation with Preterm Labour with AFLP	Nothing significant	8 mins 20 sec	150000	<50	>5000	1.69	2.93
1003854	26	UR	5	4	G5P4L3D1	Bleeding per vagina	G5P4L3D1 with 37weeks with vertex presentation with Abruptio Placenta with Severe Anaemia in Active Labour	Nothing significant	6mins 50 secs	94000	38	>5000	1.14	1.43
1002241	22	UR	2	1	G2P1L1	False labor pains	G2P1L1 with 40weeks 2 days POG with cephalic presentation with fetal VSD with TGA with False labour pains	Nothing significant	8 mins	62,000	56	>5000	1.36	1.6
5661529	23	UR	2	1	G2P1L1E1	Ruptured Ectopic with Haemorrhagic Shock	G1P1L1 with Ruptured Ectopic with Haemorrhagic Shock	Nothing significant	6 min 40 sec	110	46	243	1.39	2.9
5669725	19	UR	1	0	Primigravida	C/o Blurring of vision	Primigravida with 36 weeks POG with Cephalic Presentation with Severe PE with HELLP in DIC with Fetal Distress with RH negative pregnancy with Hypothyroidismm	Nothing significant	7 mins 40 secs	35,000	94	>5000	1.02	1.39
1009724	23	R	2	1	G2P1L1	C/o vomiting , Headache,Giddiness& Blood tinged Urine	G2P1L1 with 37weeks 6 days POG with cephalic presentation with Severe PE with DIC	Nothing significant	5mins 30secs	97,000	95	>5000	0.96	1.09
5685790	30	UR	1	0	Primigravida	C/o Epigastric Pain	Primigravida with 32 weeks 1 day POG with cephalic presentation with severe PE with GDM with IUD with Abruption with DIC with Shock	Nothing significant	6 Mins 40 secs	25,000	57	>5000	0.97	1.05
5696770	21	UR	4	3	P4L2A2	Unconscious , not responding to commands	P2L2A2 with Full Term Vaginal Delivery (outside) on 14/05/2020 with DIC with Hypovolemic Shock secondary to PPH	Nothing significant	12 mins	24,000	<50	>5000	5.7	>10
10111634	24	UR	1	￥0	Primigravida	C/o Jaundice , Pruritis	Primigravida with 38weeks 3 days POG with AFLP with Moderate Anaemia	Nothing significant	6 mins 20 sec	210	<50	>5000	>180	>10
5814261	34	UR	4	2	G4P2L1D1	C/o Pain Abdomen & PV Bleeding	G4P2L1A1 with 33 weeks 6 days POG with vertex presentation with IUD with Revealed Abruption with Severe Pre Eclampsia with DIC	Nothing significant	6 mins 15 sec	48,000	67	>5000	0.96	1.16
5828718	26	UR	1	0	Primigravida	C/o Pain Abdomen & PV Bleeding	Primigravida with 36 weeks 1 day POG with cephalic presentation with IUD with Abruption with moderate	Nothing significant	3 mins	54,000	97	>5000	0.93	1.18
5834862	23	UR	3	2	P3L3	Traumatic PPH	PL3/FTVD/Female/2.9Kg/8:16am on 15/09/2020 with traumatic PPH	Nothing significant	5 mins 40secs	69,000	116	>5000	0.91	1.13
5832394	33	UR	3	1	G3P1L1A1	C/o PV bleeding	G3P1L1 A1 with 38weeks 1 day POG with Transverselie with placenta previa with previous LSCS in DIC with Shock	Nothing significant	7 mins 10 sec	97000	40	5000	1.02	1.57
5834886	34	UR	4	2	G4P2L2A1	IUD with Pre eclampsia	G4P2L2A1 with 33 weeks 3 days POG with severe Pre eclampsia with HELLP with AKI	Nothing significant	4 Mins 30sec	150000	68	>5000	0.84	1.3
5861576	28	UR	3	2	G3P2L2	C/o Pain Abdomen & PV Bleeding	G3P2L2 with 34weeks POG with previous 2 LSCS with IUD with Severe Anaemia with Rh Negative Pregnancy with Abruption with DIC with Hypotension	Nothing significant	6 Mins	120000	68	>5000	0.84	1.3
1020561	26	UR	1	0	Primigravida	C/o Pain Abdomen & Fever	Primigravida with 40weeks 2 days POG with Cephalic presentation with Dengue fever in Latent Labour	Nothing significant	5 mins 45 secs	65000	60	2146	5.69	2.1

1026095	26	UR	4	3	G4P3L3	IUD with Pre eclampsia	G4P3L3 with 40 weeks POG with vertex presentation with IUD with Sepsis with DIC with Hepatic encephalopathy	Nothing significant	7 mins 40 secs	136000	107	6354	1.81	2.27
1026296	23	UR	2	3	P3L3	Atonic PPH	P3L3(outside delivery) with female/2.4kg with Atonic PPH secondary to retained bits of placenta with DIC with severe anaemia	Nothing significant	6 mins 10 secs	19,000	72	2512	1.54	1.32
1026444	20	UR	1	1	P2L1	Traumatic PPH	P2L1 with VBAC outside delivery on 27/10/21 with HELLP with DIC with AKI	Nothing significant	4 mins	90,000	102	>5000	1.4	1.36
1028003	22	UR	1	0	A1	Excessive PV bleeding	A1 with Abnormal uterine blleding with severe Anaemia in Early DIC	Nothing significant	7 mins	9000	79	80	0.9	1.66
1032103	22	UR	4	2	G4P2L2A1	Loose stools	G4P2L2A1 with 37 weeks 4 days POG with cephalic presentation with Acute fatty liver In pregnancy With DIC	Nothing significant	8 mins 45secs	169000	85	7016	1.28	1.54
1031922	33	R	3	1	G3P1L1A1	Pv bleeding	G3P1L1A1 with 33 weeks POG with cephalic presentation with previous LSCS with central placenta previa with placenta Increta with Focal Percreta with APH	Nothing significant	3mins 45 secs	147000	154	1288	1.22	1.36
1040663	28	R	3	2	G3P2L2	PV bleeding	G3P2L2 with 31 weeks 4 days POG with Cephalic presentation with previous 2 LSCS With Central placenta previa with Antenatal Haemorrhage with Threatened Preterm labour with Hypothyroidism	Nothing significant	4 mins 20 secs	79000	105	>5000	1.25	1.48
1039482	23	UR	1	0	Primigravida	PV leak	Primigravida with 39weeks 1 day POG in Latent Labour	Nothing significant	5 mins	154	207	>5000	0.65	0.93
1042994	19	UR	1	0	Primigravida	Pain Abdomen	Primigravida with 34 weeks 4 days POG with Vertex presentation with IUFD with Revelaed Abruption In DIC with Severe PE with partial HELLP	Nothing significant	5 mins 20 secs	52000	58	7066	1.11	1.19
1043185	21	UR	1	0	P1L1	AFLP with Hypovolemic Shock	P1L1 Post LSCS WITH AFLP IN DIC IN Hypovolemic Shock With Multi Orgna Dysfuction Syndrome	Nothing significant	4mins 30secs	24000	54	947	2.66	2.9
1043401	20	UR	2	1	G2P1L0	High BP recordings	G2P1L0 with 41 weeks 4 days POG with vertex presentation with Severe PE with HELLP Syndrome with short stature	Nothing significant	5mins 10 secs	31000	148	4112	1	1.39
1043831	26	UR	1	0	P1L1	Atonic PPH	P1L1with Full Term Emergency LSCS (outside) on 11/03/2021/Female/3.5Kg/ with Atonic PPH	Nothing significant	4mins 10 secs	83000	350	>5000	0.7	1.05
1047750	32	R	1	0	Primigravida	Post datism in latent labour	Primigravida with 41weeks 1 day POG with Cephalic presentation with Postdatism in Latent Labour	Nothing significant	3 mins 30 secs	122000	104	1204	0.77	1.54
1048700	24	UR	1	0	Primigravida	PV bleeding	Primigravida with 31weeks 1 Day POG with severe PE with Abruptio Placenta with Intrauterine Fetal Demise	Nothing significant	4mins 30secs	43000	199	>5000	0.8	1.01
100621	22	UR	1	0	Primigravida	Pv leak	Primigravida with 39 weeks 6 days POG with vertex presentatiom with severe pre eclampsia with prolonged PROM	Nothing significant	4 Mins 30sec	283	511	785	0.93	0.89
995411	35	UR	2	1	G2P1L1	Headache	G2P1L1 with 28 weeks 1 day pog with with severe pre eclampsia with imminent signs	Nothing significant	5 mins 40secs	287	401	217	0.98	0.94
996264	34	UR	3	2	G3P2L0	Decreased fetal movement	G3P2L0 with 29 weeks 6 days pog with severe pre eclampsia with FGR with hypothyroidism with APLA IgM positive	APLE positive	4 Mins 30sec	329	372	460	1.12	0.87
992828	25	UR	3	2	G3P2L2	Lower limb swelling	G3P2L2 with 32 weeks 1 day pog with severe pre-eclampsia with moderate anemia with hypothyroidism	Nothing significant	5 mins	214	315	336	0.87	0.98

1008726	22	UR	1	0	Primigravida	Pain in abdomen	Primigravida with 37 weeks 4 days POG with vertex presentatiom with severe pre eclampsia	Nothing significant	4mins 10 secs	119	416	757	0.98	0.86
993682	22	UR	1	0	Primigravida	Headache	Primigravida with 32 weeks 3 days POG with cephalic presentation with severe PE with Imminents signn with FGR with cervical stitch insituu	Nothing significant	5 mins 20 secs	305	454	615	1.09	0.9
994777	29	UR	1	0	Primigravida	Asymptomatic	Primigravida with 25 weeks 4 days POG with sever pre eclampsia with hypothyroidism	Nothing significant	5 mins 20 secs	193	283	328	0.88	1.04
994121	23	UR	2	1	G2P1L1	Decreased fetal movement	G2P1L1 with 34 weeks 5 days POG with vertex presentation with IUD with previous LSCS with Abruption in active labour	Nothing significant	5 mins	276	151	>5000	0.75	0.88
993707	24	UR	2	1	G2P1L1	Decreased fetal movement	G2P1L1 with 38 weeks 6 days POG with vertex presentation with IUFD with previous LSCS with with hypothyroidism	Nothing significant	4 mins 20 secs	104	304	480	0.95	0.98
1000939	25	UR	1	0	Primigravida	Pain in abdomen,blurring of vision	Primigravida with 39 weeks 3 days POG with cephalic presentation with severe PE with Imminents signn in latent labour w	Nothing significant	4 min 45 sec	278	315	463	0.85	0.87
1000610	21	UR	1	0	Primigravida	Headache,vomiting	Primigravida with 33 weeks 1 day POG with vertex presentatiom with severe pre eclampsia with imminent signs	Nothing significant	4 Mins 30sec	249	429	649	0.92	0.84
1000023	45	UR	1	0	Primigravida	High BP recordings	Elderly Primigravida with 35 weeks 5 days POG with DCDA twins with anemia with GDM with mild PE	Nothing significant	3 mins 30 secs	275	449	1278	0.93	0.8
999979	23	UR	1	0	Primigravida	Decreased fetal movement	Primigravida with 29 weeks POG with vertex presentatiom with severe IUGR with IUFD	Nothing significant	4 mins	296	399	342	0.98	1.04
999616	24	UR	1	0	Primigravida	Pain Abdomen	Primigravida with 40 weeks POG with vertex presentatiom with severe pre eclampsia	Nothing significant	4 mins 20 secs	341	471	478	0.93	0.91
998708	36	UR	4	2	G4P2L2A1	Frontal headache	G4P2L2A1 with 26 weeks 6 days pog with severe pre eclampsia with previous LSCS with severe FGR With absent end diastolic flow	H/O PIH	5 mins 20 secs	350	192	433	0.65	0.96
997852	18	UR	1	0	Primigravida	Generalised body swelling	Primigravida with 32 weeks 4 days POG with cephalic presentation with severe pre eclampsia with hypothyroidism	Nothing significant	4 min 45 sec	275	372	752	0.77	0.82
996934	24	UR	3	2	G3P2L2	Bleeding per vagina	G3P2L2 with 32 weeks pog with vertex presentation with ? revealed abruption	Nothing significant	5 mins 20 secs	176	257	789	0.9	0.9
995979	21	UR	1	0	Primigravida	High BP recordings	Primigravida with 36 weeks 4 days POG with vertex presentatiom with severe pre eclampsia in latent labourr	Nothing significant	4 mins	270	286	876	0.91	0.88
993782	31	UR	3	2	G3P2L2	Pain in abdomen,pedal oedema	G3P2L2 with 36 weeks 4 days pog with severe pre-eclampsia with breech presentation with anaemia in active labour	Nothing significant	5 mins 20 secs	217	290	1285	1.06	0.92
1006146	21	UR	1	0	Primigravida	one episode of convulsion	Primigravida with 35 weeks 6 days POG with cephalic presentation with antepartum eclampsia in latent labour	Nothing significant	4 mins 20 secs	287	478	1238	0.89	0.72
1005313	33	UR	4	1	G4P1L1A2	Vomiting	G4P1L1A2 with 36 weeks 4 days POG with vertex presentation with IUD with Severe Pre Eclampsia GDM with hypothyroidism	Nothing significant	4 mins 20 secs	169	436	595	0.77	0.8
1004550	28	UR	3	2	G3P2L2	Absent fetal movement	G3P2L2 with 40 weeks 6 days pog with cephalic presentation with IUFD with overt DM with gestational HTN	Nothing significant	4 Mins 30sec	205	423	759	0.92	0.84
1004114	22	UR	2	1	G2P1L1	Pain in abdomen	G2P1L1 with 32 weeks 6 days pog with breech presentation with severe pre eclampsia with previous LSCS in latent labour	Nothing significant	5 mins 20 secs	219	406	375	0.83	0.75

1002547	21	UR	1	0	Primigravida	Vomiting,fever	Primigravida with 31weeks 5 days POG with cephalic presentation with clinical IUGR with pre eclampsia with anemia with less liquor	H/O blood transfusion	4 min 45 sec	301	417	466	0.78	0.84
1002160	30	UR	6	2	G6P2L1D1A3	High BP recordings	G6P2L1D1A3 with 33 weeks 6 days POG with vertex presentation with previous LSCS with IUGR Severe PE	H/O PIH	5 mins 20 secs	234	379	428	0.89	0.83
1002041	20	UR	1	0	Primigravida	Headache,fever,convulsion	Primigravida with 38 weeks 5 days pog with antepartum eclampsia	Nothing significant	5 mins 20 secs	287	486	427	0.72	0.77
1001538	21	UR	1	0	Primigravida	Decreased fetal movement	Primigravida with 40 weeks 5 days POG with vertex presentation with IUFD	Nothing significant	4 mins 20 secs	249	254	1231	0.85	0.9
1001343	20	UR	1	0	Primigravida	Pain in abdomen	Primigravida with 40 weeks 4 days POG with cephalic presentation with severe anemia with severe PE with IUGR with Rh negative pregnancy with HELLP syndrome	H/O blood transfusion	5 mins 20 secs	4400	184	427	0.9	0.84
1009530	24	UR	1	0	Primigravida	Absent fetal movement	Primigravida with 30 weeks 5 days POG with Breech presentation with IUD with Severe PE	Nothing significant	5 mins 45 secs	266	357	385	0.82	0.9
1010939	28	R	1	0	Primigravida	High BP recordings	Primigravida with 31 weeks 5 day POG with severe pre eclampsia with FGR with increased resistance on UA doppler	Nothing significant	5 mins 20 secs	270	442	562	0.9	0.98
1009530	24	UR	1	0	Primigravida	Decrease fetal movement	Primigravida with 30 weeks 5 days POG with vertex presentatiom with severe pre eclampsia with IUFD		4 min 45 sec	266	357	>5000	0.86	0.79
1009270	24	UR	4	3	G4P3L1	B/L pedal edema	G4P3L1 with 32 weeks 1 day pog with breech presentation with Previous 2 LSCS with severe pre eclampsia	Nothing significant	5 mins 20 secs	152	455	291	0.98	0.9
1009230	21	UR	1	0	Primigravida	Absent fetal movement	Primigravida with 33 weeks 4 days POG with cephalic presentation with IUD with severe PE with HELLP syndrome in latent labor	Nothing significant	5 mins	43000	327	1162	0.96	0.83
1019642	35	UR	3	2	G3P2L2	one episode of convulsion	G3P2L2 with 33 weeks 1 day pog with cephalic presentation with antepartum eclampsia with IUD with HELLP	Nothing significant	4 min 45 sec	96,000	204	5749	1.01	0.9
1023641	20	R	1	0	Primigravida	Pain in abdomen	Primigravida with 40 weeks 2 days POG with cephalic presentation with severe PE with Hbsag positive	Nothing significant	5 mins	341	387	494	0.94	0.94
1023718	27	UR	1	0	Primigravida	Headache,vomiting,pedal oedema	Primigravida with 38 weeks 2 days pog with cephalic presentation with Partial HELLP in latent labor with meeconium satined liqourr	Nothing significant	5 mins 20 secs	74000	322	1413	0.77	0.9
1023814	23	UR	1	0	Primigravida	Pain Abdomen	Primigravida with 35 weeks 4 days pog with cephalic presentation with severe pre eclampsia with established preterm labour	Nothing significant	4 mins	310	423	365	1.07	1.06
1023497	23	UR	1	0	Primigravida	High BP recordings	Primigravida with 36 weeks 3 days POG with DCDA twins with severe pre ecamlampsia with Rh negative pregnancy	Nothing significant	5 mins 20 secs	150	334	749	0.95	0.91
1024110	23	UR	1	0	Primigravida	Headache	Primigravida with 33weeks POG with severe PE with oligohydraminos	Nothing significant	4 mins 20 secs	274	343	870	0.92	0.83
1024335	26	UR	1	0	Primigravida	Epigastric pain and vomiting	Primigravida with 32 weeks 6 days POG with cephalic presentation with severe PE with Imminents signn	Nothing significant	5 mins 20 secs	155	480	125	0.76	0.87
1024780	43	UR	1	0	Primigravida	High BP recordings	G2A1 with 36 weeks 5 days pog with cephalic presentation with severe pre eclampsia with precious pregnancyy	History of myomectomy done	5 mins 40 sec	237	473	868	0.94	0.89
1024944	21	UR	2	0	G2A1	Pain Abdomen with pv leak	G2A1 with 36 weeks 5 days pog with cephalic presentation with severe pre eclampsia in active labour	Nothing significant	5 mins	175	352	947	0.91	0.87

1024877	34	UR	3	2	G3P2L2	High BP recordings	G3P2L2 with 31 weeks 4 days pog with chronic HTN with superimposed pre eclampsia with absent end diastoli flow on umbilical artery doppler with partaial HELLP	Nothing significant	4 min 45 sec	18400	352	347	0.98	0.79
1025004	30	UR	2	1	G2P1L1	Pain Abdomen with pv leak	G2P1L1 with 39 weeks 6 days pog with cephalic presentation with with Severe pre eclampsia with PROM in latent labour	Nothing significant	5 mins 20 secs	203	387	585	0.92	0.9
1024998	25	R	1	0	Primigravida	High BP recordings	Primigravida with 34 weeks 1 day POG with Vertex presentation with Severe PE	Nothing significant	3mins 45 secs	140	288	640	0.87	0.99
1025273	22	UR	2	1	G2A1	Absent fetal movement	G2A1with 33 weeks 6 days POG with Cephalic presentation with IUD in DIC	Nothing significant	4 min 45 sec	120	78	>5000	0.8	1.3
1025279	22	UR	1	0	Primigravida	High BP recordings	Primigravida with 39 weeks 4 days pog with cephalic presentation with severe pre eclampsia in latent labour	Nothing significant	5 mins 20 secs	179	320	940	0.92	0.9
1025473	23	UR	3	2	G3P2L1	High BP recordings	G3P2L1 with 31 weeks 3 days pog with breech presentation with chronic hypertension with superimposed pre eclampsia with hypothyroidismm	Known case of chronic hypertension	6 Mins	163	443	1263	0.86	0.85
1025653	28	UR	2	0	G2A1	Pv bleeding	G2A1 with 36 weeks pog with DCDA twins with severe pre eclampsia with central placenta previa	Nothing significant	5 mins 20 secs	92000	338	1141	1.07	0.97
1025996	27	UR	3	2	G3P2L0	Facial puffiness and pedal edema	G3P2L0 with 36 weeks 6 days pog with DCDA twins with previous 2 LSCS with severe PE	Nothing significant	5 min 10 secs	159	423	794	0.59	0.94
1026011	42	UR	3	2	G3P2L2	Headache,blurring of vision,epigastric pain	G3P2L2 with 30 weeks pog with cephalic presentation with IUFD with imminent eclampsia	Nothing significant	5 mins 45 secs	201	317	1274	0.83	0.7
1027016	21	UR	1	0	Primigravida	Decrease fetal movement, pv leak	Primigravida with 40 weeks 3days POG with compound presentation with severe PE with IUD in active labor	Nothing significant	3 mins	228	347	1200	0.88	0.94
1027026	34	UR	3	1	G3P1L1A1	High BP recordings	G3P1L1 A1 with 37 weeks 4 days POG with Vertex presentation with severe pre eclampsia with imminent signs in latent labor	Nothing significant	4 mins	164	325	732	0.83	0.9
1027093	23	UR	2	0	G2A1	High BP recordings	G2A1 with 37 weeks pog with cephalic presentation with severe pre eclampsia with IUFD in active labor with Rh negative pregnancy with RVD positive	Known case of RVD positive	4 Mins 45 secs	313	310	501	0.87	0.9
1026769	31	UR	3	1	G3P2L1A1	Pedal edema	G3P1L1 A1 with 33 weeks 3 days POG with Vertex presentation with severe pre eclampsia with hypothyroidism	Nothing significant	5 mins 20 secs	136	423	497	0.93	0.9
1027286	26	UR	3	0	G3A2	High BP recordings	G3A2 with 29 weeks 6 days pog with PIH with IUFD	Nothing significant	5 min 10 secs	297	276	680	0.81	1.49
1027771	28	UR	2	1	G2P1L1	High BP recordings	G2P1L1 with 38 weeks 6 days pog with cephalic presentation with previous LSCS with Severe pre eclampsia with Rh negative pregnancy	Nothing significant	4 mins 20 secs	179	371	455	0.8	0.96
1027923	24	UR	3	0	G3A2	High BP recordings	G3A2 with 36 weeks 6 days pog with severe pre eclampsia with FGR with oligohydraminos	Nothing significant	6 Mins	191	443	866	1.02	0.83
1028055	21	UR	1	0	Primigravida	Vulval and pedal edema, decrease frequency of urination	Primigravida with 39 weeks POG with cephalic presentation with PE with acute kidney injury	Nothing significant	3 mins 30 secs	363	399	1437	1	0.9
1028063	25	UR	3	2	G3P2L2	Headache,blurring of vision	G3P2L2 with 32 weeks pog with cephalic presentation with clinical FGR with severe pre eclampsia with previous 2 LSCSs	H/O blood transfusion	5 mins 20 secs	223	302	875	0.89	0.84
1028401	23	UR	1	0	Primigravida	High BP recordings	Primigravida with 39 weeks 5 days POG with cephalic presentation with severe PE in latent labor	Nothing significant	5 mins 20 secs	238	409	290	0.7	0.85

1028685	24	UR	1	0	Primigravida	Headache,vomiting	Primigravida with 31 weeks 4 days POG with cephalic presentation with Imminents signs with moderate anemia	Nothing significant	4 Mins 30sec	182	392	931	0.92	0.95
1029528	35	R	2	1	G2P1L1	Absent fetal movement	G2P1L1 with 40 weeks 3 days pog with cephalic presentation with previous LSCS with with chronic HTN with IUFD	Known case of chronic hypertension	5 mins 20 secs	554	409	708	0.99	0.96
1029830	26	UR	3	2	G3P2L2	Headache,blurring of vision,vomiting	G3P2L2 with 34 weeks 3 days pog with cephalic presentation with imminent eclampsia	Nothing significant	4 Mins 30sec	124	352	1132	0.88	0.92
1016422	27	UR	2	0	G2A1	High BP recordings	G2A1 with 29 weeks 1 day POG with cephalic presentation with severe pre eclampsia	Nothing significant	5 mins	229	411	758	0.82	0.94
1029504	25	UR	2	1	G2P1L1	Headache	G2P1L1 with 33 weeks 2 days pog with cephalic presentation with Severe pre eclampsia with hypothyroidism with increase resistance on dopplerr	Nothing significant	4 mins	285	392	582	0.86	0.9
1030647	23	UR	1	0	Primigravida	Vomiting,Pv leak	Primigravida with 30 weeks 2 days POG with MCDA twins with PPROM with cord prolapse with severe PE	Nothing significant	3mins 45 secs	327	516	>5000	0.86	0.95
1031142	28	UR	3	2	G3P2L2	Headache,vomiting	G3P2L2 with 40 weeks pog with severe pre-eclampsia with imminent signs	Nothing significant	5 mins	245	403	608	0.89	0.97
1031604	28	UR	5	2	G5P2L2A2	PV bleeding	G5P2L2A2 with 34weeks 5 days with vertex presentation with revealed Abruption with Severe Pre eclampsia with IUD	Nothing significant	6 mins	230	204	702	0.89	0.99
1031929	28	UR	5	3	G5P3L2A1	Epigastric pain	G5P3L2A1 with 27 weeks 3 days with breech presentation with previous 2 LSCS with severe IUGR with absent diastolic flow with Severe Pre eclampsia	Nothing significant	4 mins	141	351	892	0.91	0.85
1033393	28	UR	1	0	Primigravida	PV leak	Primigravida with 39 weeks 6 days pog with cephalic presentation with severe preeclampsia with PROM with hypothyroidism with meconium satined liqour in latent labor	Nothing significant	5 mins 20 secs	233	293	975	0.89	0.89
102066	32	UR	2	1	G2P1L1	High BP recordings	G2P1L1 with 34 weeks 5 days POG with vertex presentation with previous LSCS with Severe PE with partial HELLP Syndrome	History of PIH in previous pregnancy	4 mins	96000	595	1398	0.9	1
1020714	26	UR	2	1	G2P1L0	High BP recordings	G2P1L0 with 29 weeks 2 days POG with Severe PE with partial HELLP Syndrome with FGR	Nothing significant	4 Mins 30sec	52000	366	1295	0.9	0.85
1020829	20	UR	1	0	Primigravida	1 episode Convulsion	Primigravida with 40 weeks 1 day POG with cephalic presentation with antepartum eclampsia	Nothing significant	3 mins 15 secs	161	417	697	0.92	0.86
1021063	26	UR	2	1	G2P1L1	High BP recordings	G2P1L1 with 37 weeks pog with cephalic presentation with previous LSCS with severe anemia with Severe pre eclampsia in active labour	Nothing significant	5 mins 20 secs	74000	338	1015	0.83	0.92
1021066	24	UR	1	0	Primigravida	High BP recordings	Primigravida with 41 weeks 1 day POG with cephalic presentation with severe pre eclampsia with hypothroidism in latent labor	Nothing significant	5 mins	228	465	374	0.91	0.92
1021052	38	UR	4	3	G4P3L3	High BP recordings	G4P3L3 with 28 weeks 6 days POG with breech presentation with severe PE with hypothyroidism	Nothing significant	3mins 45 secs	156	399	1068	0.84	0.83
1021181	26	UR	2	1	G2P1L1	Decrease fetal movement	G2P1L1 with 33 weeks 1 day POG with vertex presentation with IUD with previous LSCS with IUGR Severe PE with hypothyroidism	History of PIH in previous pregnancy	5 mins 45 sec	629	436	828	0.9	0.97
1020904	23	UR	2	1	G2P1L1	High BP recordings	G2P1L1 with 29 weeks 3 days pog with previous LSCS with Severe pre eclampsia with absent EDF	Nothing significant	5 mins 20 secs	413	455	425	0.9	0.9
1046841	35	UR	2	1	G2P1L1	High BP recordings	G2P1L1 with 30 weeks 4 days pog with cephalic presentation with with Severe pre eclampsia with severe FGR	Nothing significant	5 mins 20 secs	271	311	1145	0.8	0.93

1033292	27	UR	2	1	G2P1L0	Absent fetal movement	G2P1L0 with 30 weeks 6 days pog with breech presentation with previous LSCS with IUFD with typhoid fever	Nothing significant	3 mins 15 secs	365	465	633	0.95	1.1
1033571	19	UR	1	0	Primigravida	High BP recordings with vomiting	Primigravida with 38 weeks 6 days pog with cephalic presentation with pre eclampsia with imminent sign in second stage of labourr	High Bp recording and vomiting	3 mins 30 secs	217	465	313	0.73	0.92
1033345	26	UR	1	0	Primigravida	High BP recordings	Primigravida with 32weeks 3 days POG with severe PE with hypothyroidism	High Bp recording	4 Mins 30sec	206	419	655	0.98	1
1033964	25	UR	2	1	G2P1L1	Headache,vomiting	G2P1L1 with 33 weeks 4 day POG with vertex presentation with IUD with previous LSCS with chronic hypertension with superimposed eclampsia with Rh negative pregnancy	H/O blood transfusion	4 mins	196	472	318	0.83	0.88
1034120	26	UR	1	0	Primigravida	High BP recordings	Primigravida with 27 weeks 2 days POG with DCDA twins with One twin IUD with other twin severe FGR with absent end diastolic flow with mild pre eclampsia with hypothroidism	Nothing significant	5 mins 20 secs	247	437	574	0.9	0.86
1034740	32	UR	5	0	G5A4	Facial puffiness and pedal edema	G5A2 with 31weeks 5 days with Severe Pre eclampsia with mild oligohydraminos with clinical FGR	History of hysterolaparoscopy	5 min 10 secs	179	444	1116	0.76	0.87
1035363	24	UR	2	1	G2P1L1	Absent fetal movement	G2P1L1 with 37 weeks 2 days POG with vertex presentation with IUD with previous LSCS	Nothing significant	6 Mins	221	441	811	0.72	1.04
1036166	23	UR	1	0	Primigravida	High BP recordings	Primigravida with 26 weeks 3 days POG with vertex presentatiom with severe pre eclampsia with hypothroidism	Nothing significant	5 mins	241	440	430	0.82	0.96
1037071	28	UR	2	1	G2P1L1	Pedal edema	G2P1L1 with 30 weeks 1 day pog with MCDA twin with previous LSCS with Severe pre eclampsia with HELLP syndrome with FGR with increase resistance on dopplerr	Nothing significant	4 min 45 sec	25000	259	>5000	0.78	0.96
1043221	25	UR	1	0	Primigravida	Absent fetal movement	Primigravida with 24 weeks 6 days POG with vertex presentatiom with abruption with IUFD	Nothing significant	5 mins	155	165	>5000	0.87	1
1043401	20	UR	2	1	G2P1L0	Chest pain,pv leak	G2P1L0 with 41 weeks 4 days POG with Severe PE with HELLP Syndrome with short stature	Nothing significant	5 mins	31000	220	4112	0.78	0.96
1043188	23	UR	3	0	G3A2	Absent fetal movement	G3A2 with 37 weeks 2 days pog with IUFD with thrombocytopenia in latent labor	nothing significant	5 mins 20 secs	58000	476	1556	2.37	1.03
1043635	28	UR	4	2	G4P2L2A1	High BP recordings	G4P2L2A1 with 27 weeks 3 days pog with severe pre eclampsia with previous 2LSCS with FGR With absent end diastolic flow	Nothing significant	4 mins 20 secs	250	283	237	0.86	0.92
1057570	22	UR	3	0	G3A2	High BP recordings	G3A2 with 40 weeks 2 days pog with cephalic presentation with severe pre eclampsia with Short stature	Nothing significant	5 mins	2.11	493	306	0.8	0.9
1057514	28	UR	2	0	G2A1	High BP recordings	G2A1 with 34 weeks 3 days pog with cephalic presentation with severe pre eclampsia with FGR with oligoamnios with absent EDF with Rh negative pregnancy	Nothing significant	5 mins 15 secs	332	614	455	0.84	0.9
1057714	27	UR	2	1	G2P1L1	Pain Abdomen with pv leak with absent fetal movement	G2P1L1 with 38 weeks 3 days pog with cephalic presentation with IUFD with FGR in latent labour	Nothing significant	4 Mins 30sec	187	449	384	1.04	1.04
1058406	38	UR	2	1	G2P1L1	Absent fetal movement with pain in abdomen	G2P1L1 with 35 weeks 6 days pog with cephalic presentation with IUFD with severe FGR in active labourr	Nothing significant	4 Mins 45 secs	282	401	2497	0.64	0.95
1058432	27	UR	4	3	G4P3L3	High BP recordings	G4P3L3 with 38 weeks 6 days pog with cephalic presentation with severe pre eclampsia with pancytopenia	History of PIH in previous pregnancy	4 mins	73000	321	1438	0.91	0.87

1002664	40	UR	1	0	Primigravida	Headache	Primigravida with 36 weeks 4 days POG with vertex presentatiom with severe pre eclampsia in latent labour	nothing significant	5 mins 20 secs	180	388	321	0.79	0.79
1058775	20	UR	2	1	G2P1L1	Pain in abdomen	G2P1L1 with 35 weeks 1 day pog with cephalic presentation with IUFD in active labour	Nothing significant	4 Mins 30sec	262	417	3240	0.76	1.02
1058945	25	UR	1	0	Primigravida	PV leak	Primigravida with 30 weeks 4 days POG with DCDA twins with Twin A IUD with PROM in early labour	Nothing significant	5 mins	224	565	1360	0.94	1.1
1059073	21	UR	3	1	G3P1L0A1	Pain Abdomen	G3P1L0 A1 with 37 weeks 1 day POG with cephalic presentation with severe pre eclampsia with FGR in latent labor	Nothing significant	4 min 15 secs	211	485	402	0.92	0.9
1060018	20	UR	1	0	Primigravida	PV bleeding	Primigravida with 35 weeks 5 days POG with cephalic presentatiom with revealed abruption with IUFD	Nothing significant	5 mins 20 secs	46000	292	1211	0.69	0.92
1060665	21	UR	2	1	G2P1L1	Headache, blurring of vision ,1 episode of convulsion	G2P1L1 with 32 weeks pog with cephalic presentation with antepartum eclampsia with IUFD with hypothyroidism with imminent signs	Nothing significant	5 mins 15 secs	2	576	1084	0.81	0.85
1060729	25	UR	2	0	G2A1	High BP recordings	G2A1 with 27 weeks 6 days pog with breech presentation with severe pre eclampsia with FGR with oligoamnios with IUD with Hypothyroidism with imminent signs of eclampsia	Nothing significant	4 mins 20 secs	2.38	492	440	0.9	0.96
1060887	25	R	1	0	Primigravida	pain in abdomen with high BP reading	Primigravida with 38 weeks 3 days POG with cephalic presentation with mild preeclampsia with hypothyroidism	Nothing significant	4 min 45 sec	3.19	431	485	0.9	1.02
1060895	29	R	3	2	G3P2L2	High BP recordings	G3P2L2 with 35 weeks pog with cephalic presentation with severe pre eclampsia with Rh negative pregnanacy for safe confinement	Nothing significant	5 mins	1.07	605	1105	0.83	0.9
1061239	28	R	2	1	G2A1	High BP recordings	G2A1 with 37 weeks 5 days pog with cephalic presentation with severe pre eclampsia with FGR with PROM in latent labour	Nothing significant	4 mins 20 secs	1.96	379	676	0.62	0.86
1062237	32	UR	2	1	G2P1L1	Pain Abdomen with pv bleed	G2P1L1 with 34 weeks 1 day pog with cephalic presentation with previous lscs with IUFD with APH with revealed abruption	Nothing significant	5 mins 15 secs	2.04	391	1192	0.83	0.93
1062640	28	UR	4	3	G4P3L3	Absent fetal movement	G4P3L3 with 34 weeks 6 days pog with cephalic presentation with IUFD with central placenta previa with APH	Nothing significant	3 mins 30 secs	1.75	354	778	0.96	0.98
1063287	28	UR	2	0	G2A1	Absent fetal movement	G2A1 with 30 weeks 5 days pog with cephalic presentation with IUFD	Nothing significant	4 min 45 sec	2.75	369	294	1.03	0.96
1063286	36	UR	2	1	G2P1L1	High BP recordings	G2P1L1 with 28 weeks 4 days pog with previous LSCS with chronic hypertension with hypothyroidismm	Nothing significant	4 mins 20 secs	2.89	402	604	0.91	0.91
1064726	27	UR	2	0	G2A1	High BP recordings	G2A1 with 31 weeks 1 day pog with cephalic presentation with severe PE with FGR with AEDF in few loops	Nothing significant	5 mins	222	487	550	1.04	0.78
1070511	26	UR	3	2	G3P2L2	PV bleeding	G3P2L2 with 36 weeks 6 days POG with Cephalic presentation with previous 2 LSCS With Complete placenta previa with Antepartum Haemorrhagee	Nothing significant	4mins 30secs	219	408	639	0.88	1.01
1071163	20	R	1	0	Primigravida	Decrease fetal movement	Primigravida with 40 weeks 3 days POG with cephalic presentation with IUD in latent labour	Nothing significant	3mins 45 secs	259	484	706	0.65	0.99
1069883	27	R	1	0	Primigravida	High BP recordings	Primigravida with 34 weeks 4 days POG with cephalic presentation with Severe PE with FGR with oligohydramnios with partial HELLP	Nothing significant	5 mins 15 secs	120	402	825	0.88	0.88
1069310	24	UR	4	2	G4P2L2A1	Vomiting,epigastric pain	G4P2L2A1 with 35 weeks 4 days pog with breech presentation with severe pre eclampsia with FGR With oligohydraminos	Nothing significant	5 mins 20 secs	118	376	119	0.8	0.8

1068727	27	UR	1	0	Primigravida	Pain Abdomen with pv leak	Primigravida with 37 weeks 6 days POG with cephalic presentation with IUD in latent labour	Nothing significant	4 mins	115	592	501	0.96	1.13
1068252	31	UR	1	0	Primigravida	Decrease fetal movement	G2P1L0 with 33 weeks 2 days pog with cephalic presentation with IUFD with Rh negative pregnancy with previous LSCS	Nothing significant	5 mins 20 secs	451	727	727	0.71	1.07
1067596	24	UR	3	2	G3P2L1	Pain Abdomen with decrease fetal movement	G3P2L1 with 34weeks 6 days POG with Cephalic presentation with previous LSCS with FGR with IUD with severe pre eclampsia with HELLP syndrome	Nothing significant	4 min 45 sec	16000	340	>5000	0.85	0.95
1067328	33	UR	1	0	Primigravida	Absent fetal movement	Primigravida with 26 weeks 4 days POG with IUFD with severe anemia	Nothing significant	5 mins	328	331	668	0.82	0.93
1057288	29	UR	2	1	G2P1L1	Pv bleeding	G2P1L1 with 39 weeks POG with cephalic presentation with IUD with previous LSCS with IUGR in latent labour	Nothing significant	5 mins 40 secs	267	153	>5000	0.74	1
1056850	23	UR	1	0	Primigravida	High BP recordings	Primigravida with 28 weeks 6 days POG with severe pre eclampsia with IUFD	Nothing significant	5 mins 30 secs	23900	564	340	0.71	0.8
1056893	28	UR	2	1	G2P1L1	Decrease fetal movement	G2P1L1 with 33 weeks 5 days pog with cephalic presentation with previous Lscs with IUD with FGR	Nothing significant	4 mins 20 secs	305	546	349	0.7	1.14
1055884	26	UR	2	1	G2P1L1	Pain Abdomen with pv bleed	G2P1L1 with 32 weeks pog 1 day with DCDA twins with both twins in breech with both IUFD with revealed abruption	H/O blood transfusion	6 mins 15 sec	40000	73	5968	1.26	1.12
1054807	22	UR	1	0	Primigravida	High BP recordings	Primigravida with 38 weeks with cephalic presentation with late onset FGR with IUD with Severe PE with partial HELLP	Nothing significant	4 mins 20 secs	115	406	1198	0.79	0.9