MATERNAL OUTCOMES IN PREGNANCY COMPLICATED WITH DISSEMINATED INTRVASCULAR COAGULATION AT A TERTIARY CARE CENTRE – AN OBSERVATIONAL STUDY

By

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This is to certify that the dissertation entitled "**Maternal Outcomes In Pregnancy Complicated With Disseminated Intravascular Coagulation At A Tertiary Care Centre – An Observational Study**" is a bonafide research work done by **Reg No: BJ0119010.**

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ABBREVIATIONS

DIC	-	Disseminated Intravascular Coagulation
IUFD	-	Intra Uterine Foetus Death
PRES	-	Posterior Reversible Encephalopathy Syndrome
ARDS	-	Acute Respiratory Syndrome
AKI	-	Acute Kidney Injury
ARF	-	Acute Renal Failure
SAH	-	Sub arachnoid Haemorrhage
HIE	-	Hypoxic Ischemic Encephalopathy
ANC	-	Ante Natal Care
TF	-	Tissue Factor
IL	-	Interleukin
TFPI	-	Tissue Factor Pathway Inhibitor
PAI	-	Plasminogen activator Inhibitor
PPH	-	Postpartum Hemorrhage
PT	-	Prothrombin Time
APTT	-	Activated prothrombin time
INR	-	International Normalised Ratio
ICU	-	Intensive Care Unit
ISTH	-	International society for thrombosis and haemorrhage
USG	-	Ultrasound
CT	-	Computed tomography

MRI	-	Magnetic Resonance Imaging
CPAP	-	Continuous positive airway pressure
SGOT	-	serum glutamic-oxaloacetic transaminase
SGPT	-	serum glutamic-pyruvic transaminase
LDH	-	lactic acid dehydrogenase
MgSo4	-	Magnesium Sulphate
PCV	-	Packed cell volume
RDP	-	Random Donor Platelets
SDP	-	Single donor platelets
FFP	-	Fresh frozen plasma
t test	-	Two sample t test
MC	-	Chi square test with Monte Carlo simulation
MW	-	Mann Whitney U test
MODS	-	Multi organ dysfunction
DVT	-	Deep venous thrombosis
AFLP	-	Acute fatty Liver in Pregnancy
Sl.No.	-	Serial Number
KLE’s	-	Karnataka Lingayat Educational Society
KAHER	-	KLE Academy of Higher Education and Research center
JNMC	-	Jawaharlal Nehru Medical College
SD	-	Standard Deviation

ABSTRACT

Background: Disseminated Intravascular coagulation (DIC) Is a consumptive coagulopathy responsible for almost a quarter of all maternal deaths. The etiological factors of DIC are most of the times are preventable causes. This study is conducted to analyse the causes of DIC at a tertiary care centre.

Objectives: a) To analyse maternal outcomes in Pregnancy complicated with DIC. b) To know the etiological factors of DIC in pregnancy

Material & methods: - Pregnant women admitted & diagnosed as DIC, who were delivered at Dr.Prabhakar kore Hospital & also who were delivered outside & referred & were in DIC were analysed prospectively between January 2020 and September 2021.Pregnancy modified ISTH DIC Score was used to diagnose DIC.

Results: 50 patients were diagnosed with DIC in pregnancy during the study period. Mortality rate was 16%.The most common cause for DIC is PPH (26.5%) followed by Abruption (12.24%).The most common complication raised in these patients was AKI (10%) & shock (10%).Among the patients who had PPH 69% had Atonic PPH, 23% had Traumatic PPH & 7% had secondary PPH. Out of all patients 60% had antepartum DIC & 40% had Postpartum DIC.Antepartum DIC has better recovery rate than Postpartum DIC.

Conclusion: DIC caused by obstetric conditions is associated with high mortality and morbidity.The management should be prompt & aggressive especially in postpartum DIC.Transfusion of blood & blood products should be done vigilantly while correcting DIC as adverse reactions due to transfusion might be the cause of mortality in DIC patients.

Key words – DIC ,maternal outcomes, etiology,complications**.**

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INTRODUCTION

Disseminated Intravascular Coagulation (DIC) is a clinic-pathological syndrome characterized by excess intravascular fibrin formation in response to excessive blood protease activity that overcomes the natural anticoagulant mechanism. Disseminated intravascular coagulation (DIC) is the endpoint of uncontrolled systemic activation of the hemostatic system, leading to a simultaneous widespread micro vascular thrombosis that can compromise the blood supply to different organs, and may lead to organ failure. In severe DIC diffuse multi organ bleeding and hemorrhagic necrosis, micro thrombi in small, medium & large blood vessels are seen[6,7,8,9]. Untreated or uncontrolled DIC leads to exhaustion of platelets, coagulation and anti-coagulation factors, which in turn leads to uncontrolled bleeding and death. DIC is one of the leading causes for maternal morbidity and mortality in the peripartum period.[1]

DIC is diagnosed in one half of pregnant women with abruption- placenta, amniotic fluid embolism, HELLP syndrome, AFLP1.Its prevalence among nations varies from 0.03 to 0.35%[10] and in KAHER'S Dr. Prabhakar Kore hospital is 0.93%. Prevalence is little high as it is a tertiary care Centre.

KAHER'S Dr. Prabhakar kore charitable hospital being a tertiary Centre we will be seeing patients of complicated pregnancies with pre-eclampsia, Eclampsia, HELLP Syndrome ,Placental Abruption ,IUFD, Pregnancy associated with other illness like Acute fatty liver in pregnancy, septicemia, shock, PRES Posterior Reversible Encephalopathy Syndrome) etc., All necessary active interventions will be taken(conservative, surgical & supportive treatment with blood and blood products and necessary drugs).Despite that some may land up into mortality and some with morbidity. This study is mainly aimed to see the maternal outcomes in mothers

diagnosed with DIC during pregnancy and look into the critical measures and interventions taken which prevented mortality and morbidity and also the role of Blood and blood products transfusion.

Many women with complicated pregnancies develop DIC. Some of them recover well, but some ends up with morbidities & some with death. This study focuses mainly on maternal outcomes in patients who developed DIC & secondarily analyzing the Etiological factors of DIC in a tertiary care Centre in North Karnataka.

Even if patients present with DIC in a tertiary care Centre like DR. Prabhakar Kore hospital we are able to prevent mortality to some extent but death in the due course or later is inevitable in some patients due to some other complications like Thrombo-embolism, respiratory failure, renal failure or sudden cardiac arrest etc., which might be preventable if expert management is done. Here this study looks for maternal outcomes in patients with DIC at a tertiary care center and also to figure out the causes of death thus focusing on that area might be helpful in future management of DIC cases.

Transfusion of blood and blood products is massive which might control the bleeding temporarily but later results in adverse effects on the entire system like overload, acute respiratory distress syndrome (ARDS), acute kidney injury (AKI) which are not given a thought at the time of transfusion. Here we try to figure out when and what blood products to be transfused and also the number of volumes, thus it might help in preventing the transfusion related morbidities.

OBJECTIVES

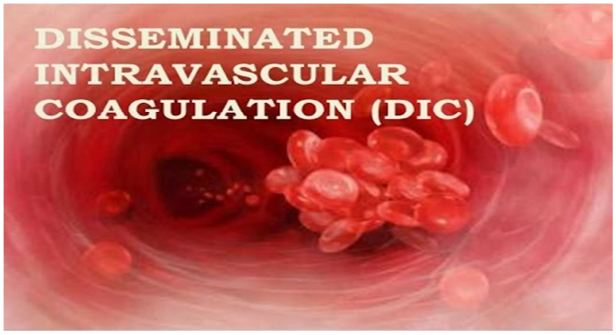
Primary Objectives: To analyze maternal outcomes in pregnancy complicated with DIC.

Secondary Objectives:

1. To know the Etiological factors

2. To assess the role of transfusion of blood and blood products in preventing mortality

REVIEW OF LITERATURE



Definition: DIC is an acquired intravascular activation of coagulation associated with consumption of coagulation factors, loss of localization, damage to microvasculature leading to bleeding, multi organ failure & risk of mortality.[11]

PHYSIOLOGY AND PATHOGENESIS

During pregnancy, a substantive increase in plasma volume is concomitantly augmented by production of most procoagulants.[12] importantly, fibrinogen (factor I) concentration increases approximately 50% above non-pregnant values, and during late pregnancy, it ranges from approximately 375 to 620 mg/dL.[13] Thus, virtually all clotting factors increase. At the same time, there is a reduction in levels of natural anticoagulants protein C and S and tissue factor pathway inhibitor-1 as well as an acquired resistance to protein C.[14] In addition, pro-fibrinolysin or plasminogen levels increase but there is also increased inhibition of fibrinolysis.[12]As a result of all of these alterations, the net result is that pregnancy is a procoagulant state.

The literature describing the physiologic process of coagulation continues to evolve. For many years it was proposed that there was a coagulation cascade or waterfall.[15] Instead, the current theory is that coagulation is primarily initiated by tissue factor, or thromboplastin, that forms complexes with factors VII and VIIa.[16] Tissue factor is an integral membrane glycoprotein that is found in highly vascularized organs such as the brain, lungs, and placenta, and it also can be

expressed constitutively by certain cell types.[17]In brief, the development of tissue factor FVIIa complexes ultimately generates activated factor X to initiate clotting, whereas the previously labeled intrinsic pathway is responsible for the amplification of this process. This main role of tissue factor FVIIa complex in coagulation is depicted in Figure 1. The end result of this coagulation process is fibrin formation, which is then counterbalanced by the fibrinolytic system—dedicated to the removal of excess fibrin. Also shown in the schematic is the fibrinolytic system with plasminogen activated by tissue factor, and this is augmented by thrombin to produce plasmin, which lyses fibrin and fibrinogen. The end result is production of fibrinogen-fibrin split products, which include D-dimers.

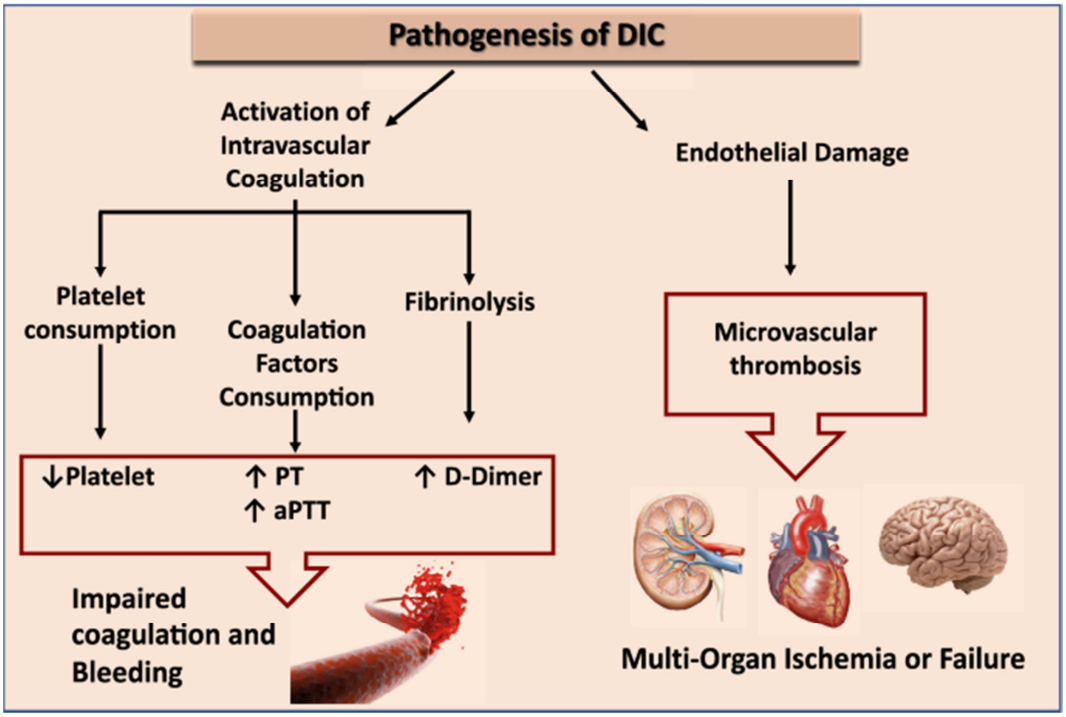


Figure 1 – Pathogenesis of DIC

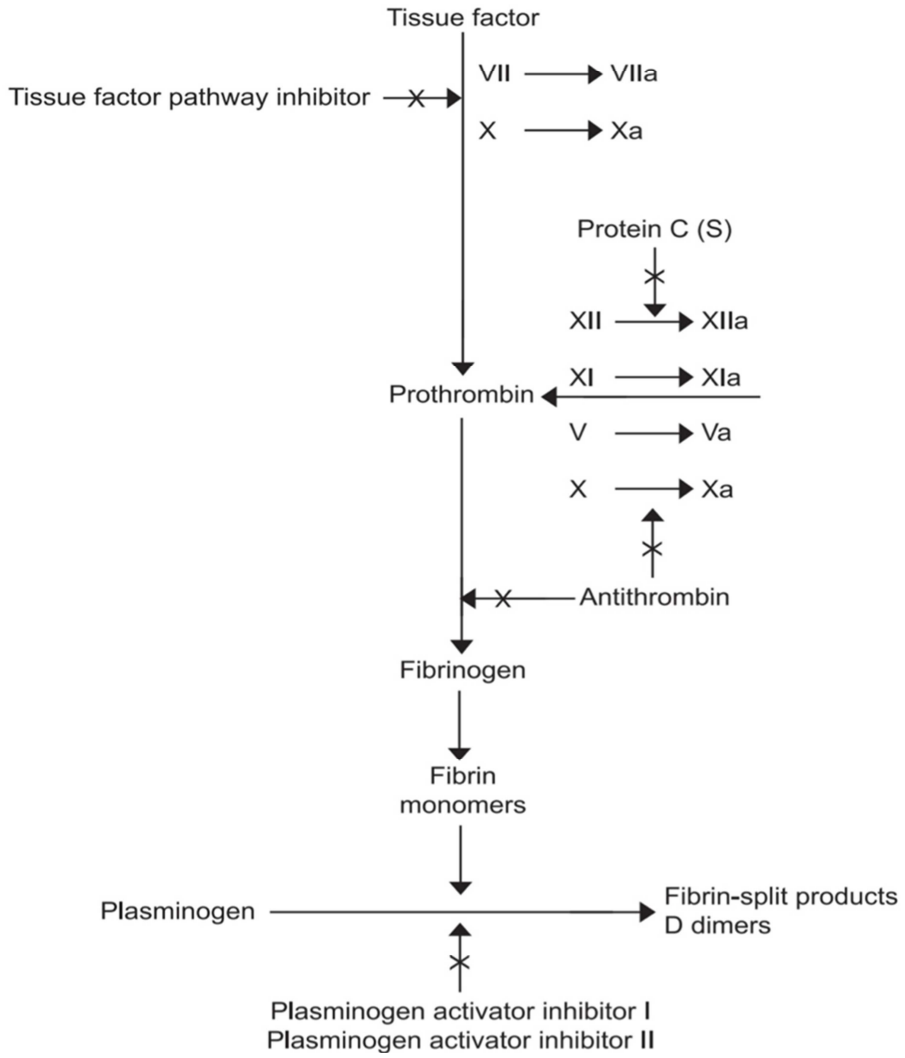


Figure 2 – Activation of coagulation process beginning with tissue factor and contrasting fibrinolytic system

The initiation of DIC begins with the release of tissue factor by any number of pathologic conditions. In most cases, tissue factor is released by damaged sub endothelial tissue and stimulated monocytes, which in turn provoke release of cytokines from the endothelium. In this scenario, with focal injury, there is attraction of monocytes and sub endothelium with platelets that promotes localized coagulation, viz the vessel plug. To the contrary, with generalized endothelial activation, there is diffuse activation of coagulation.

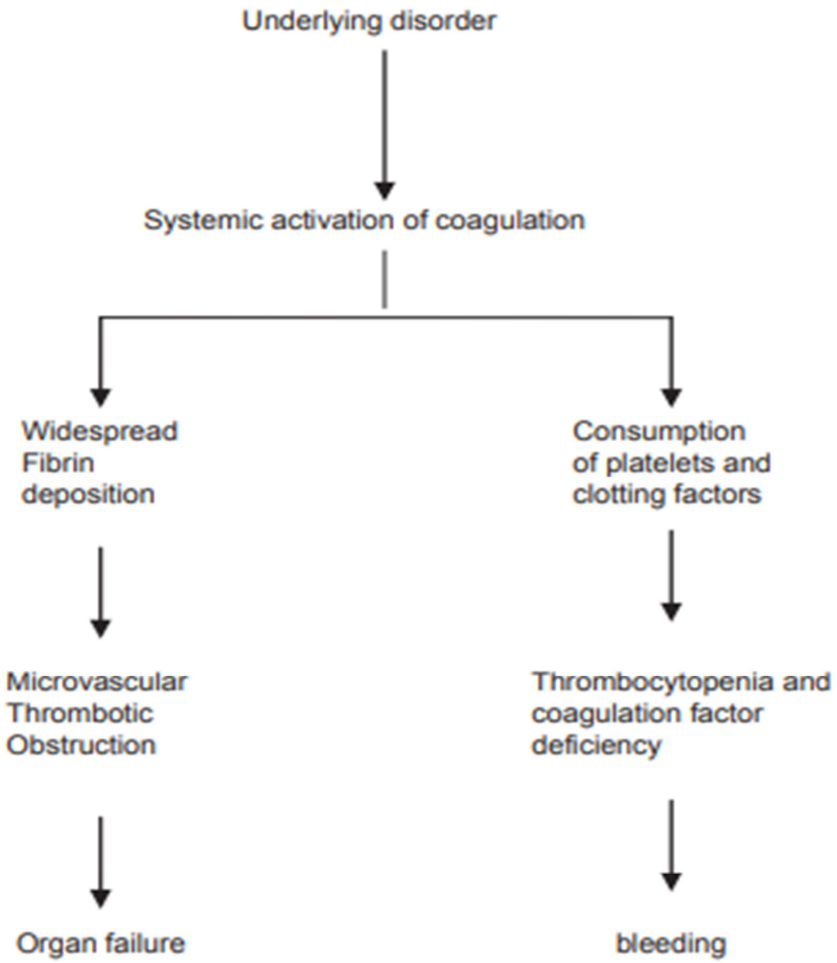


Figure 3 – Flow chart showing consequences of DIC

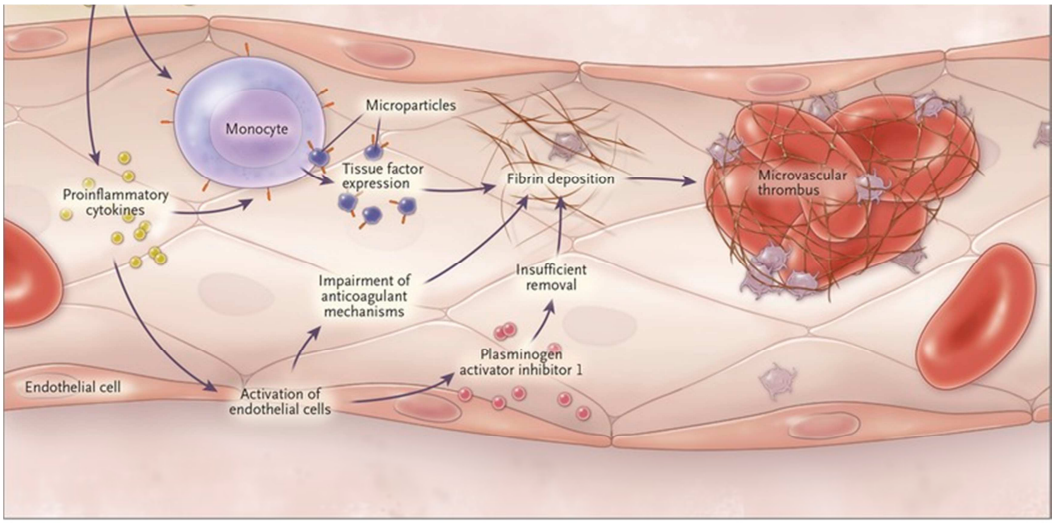


Figure 4 – Showing the mechanism of micro thrombus formation

DIC although tissue factor is found in endothelial cells, it is also in abundant supply in trophoblastic tissue and amniotic fluid.[18-20] Thus, in obstetric syndromes, some of the most profound coagulopathies are stimulated by release of tissue factor from these sources. This pathologically activated cycle of coagulation and fibrinolysis becomes clinically important when coagulation factors and platelets are sufficiently depleted, resulting in a consumptive coagulopathy.

Clinical presentation of DIC may be the results of the following mechanisms.

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1. Endothelial dysfunction and platelet activation

Intact, dysfunctional, or activated cells, as well as remnants of cell surfaces, inflammatory mediators, and coagulation proteins are all part of an interplay in which uncontrolled activation of coagulation cascade leads to DIC.[21]

Endothelial cells, platelets, but in some cases also leucocytes and cancer cells can participate in the genesis of the process leading to DIC by releasing pro-inflammatory cytokines, propagating the activation of coagulation on their surface or inducing tissue factor (TF) expression on their membrane.[22]A systemic inflammatory response that is associated with markedly increased circulating pro- inflammatory cytokines such as tumor necrosis factor-a, interleukin-1 (IL-1), and interleukin-6 (IL-6) can lead to exaggerated expression of TF by leukocyte and endothelial cells.[23]This will generate an uncontrolled coagulation response that will eventually deteriorate into DIC. Lastly, the initiation of coagulation leading to thrombin generation in DIC, is mediated by the TF/factor Vila pathway, also known as the extrinsic coagulation pathway.[24]

The most significant source of TF is not completely clear in all situations. Tissue factor may be expressed not only in mononuclear cells in response to

proinflammatory cytokines (mainly IL- 6) but also by vascular endothelial or cancer cells.[25]Despite the potent initiation of coagulation by TF, the activation of coagulation cannot be propagated if the physiological anticoagulant pathways function properly. However, in DIC all major natural anticoagulant pathways (i.e., antithrombin III, protein C system, and TF pathway inhibitor [TFPI]) appear to be impaired.[26]

Plasma concentrations of antithrombin III, the most important inhibitor of thrombin, are markedly reduced during DIC because of a combination of consumption[27] degradation by elastase from activated neutrophils[28] and impaired synthesis.[31]

A significant depression of the protein C system may further compromise an adequate regulation of activated coagulation.[29]This impaired function of the protein C system is caused by a combination of impaired protein synthesis, cytokine-mediated down-regulation of endothelial thrombomodulin, and a fall in the concentration of the free fraction of protein S (the essential cofactor of protein C), resulting in reduced activation of protein C.[29]

Lastly, there seems to be a misbalance of TFPI function in relation to the increased TF-dependent activation of coagulation.[30]

All these anticoagulant pathways are linked to the endothelium, and it is likely that endothelial cell activation and dysfunction are an important component of the imbalance between coagulation and anticoagulation systems. Of interest, experimental and clinical studies indicate that during DIC, the fibrinolytic system is largely suppressed at the time of maximal activation of coagulation.[31'']This inhibition of fibrinolysis is caused by a sustained rise in the plasma concentrations of plasminogen activator inhibitor (PAI)-1, the principal inhibitor of the fibrinolytic system.

Activation of platelets may also accelerate fibrin formation.[32]The expression of TF in monocytes is markedly stimulated by the presence of platelets and granulocytes in a P-Selectin dependent reaction.[33]This effect may be the result of nuclear factor kappa B activation induced by binding of activated platelets to neutrophils and mononuclear cells.[3]During pregnancy maternal leukocytes are in a higher state of activation than in non-pregnant women[35]and have characteristics akin to sepsis[36] .However, they are well controlled during pregnancy, and it has been proposed that the trophoblast plays a role in the maintenance of the balanced systemic maternal inflammation during gestation.[37]Nevertheless, in cases of sepsis caused by an infectious agent or septic abortion and at least in some of the cases of amniotic fluid embolism[38] this equilibrium is disturbed and the mother develops DIC.

2. Trophoblast properties and activation of the coagulation system

During normal gestation the trophoblast has 2 hemostatic functions: (1) to allow the laminar flow of maternal blood in the intervillous space and prevent it from clotting during that time and (2) to prevent bleeding at the maternal fetal interface.[45]

To address these contradicting challenges, the syncytio-trophoblast acquires endothelial-cell like properties (Figure 2). As a consequence, first, in human normal placenta, syncytiotrophoblast strongly expresses TF, and its activity is higher if compared with human umbilical vein endothelial cells. On the contrary, the trophoblast is able to synthesize protein C, protein S, and protein Z as well as a specific inhibitor of the tissue factor pathway known as TFPI-2 (placental protein 5)[39]that will prevent unnecessary activation of the coagulation cascade.[40]

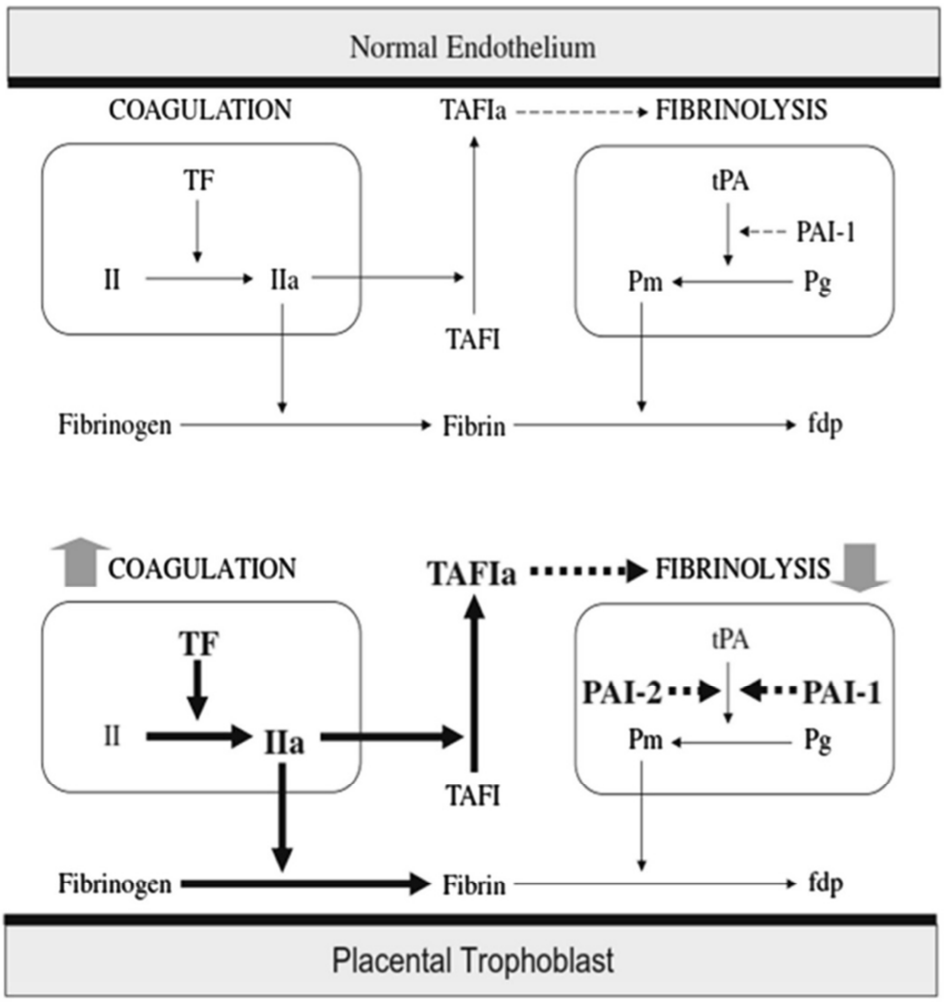
Second, the placenta produces PAI-2 in addition to the gradual increase of PAI-1, observed during normal pregnancy and becomes markedly elevated in the third

trimester to prevent fibrinolysis.[41]These changes are associated with relatively unchanged tissue plasminogen activator concentrations, contributing to a state of reduced clot Lysis and a prothrombotic bias in the pregnant woman.[42]This mechanism is further mediated through thrombin activatable fibrinolysis inhibitor.[43]

The evidence herein supports the fact that any condition that disrupts the integrity of the trophoblast (Figure 2) can lead to a release of a large amount of potent TF that will activate the coagulation cascade and propagate an inflammatory response that can easily become systemic, leading to uncontrolled thrombin generation and the subsequent development of DIC.[44]

There are several conditions that are associated with DIC in which the current evidence suggests that the systemic maternal response is the result of endothelial activation. The classical one is abruption, especially that with concealed bleeding and fetal demise. These patients have a combination of consumption coagulopathy and discharge of thromboplastin (tissue factor) into the maternal circulation.[31]

Although the DIC developed in patients with placental abruption is regarded as a problem of consumption coagulopathy, it seems that there is more to it, meaning that often patients with a retro- placental cot have a much lower blood loss than those who developed PPH, yet the DIC of in these patients is much more severe. A probable explanation is that this complication is associated with the release of procoagulating factors, such as thromboplastin, into the maternal circulation.[45]



Comparison between normal endothelium (*top*) and placental trophoblast (*bottom*). The placenta is in a heightened state of coagulation activation through increased TF production. This increases pro-thrombin (II) to thrombin (IIa) conversion for cleavage of fibrinogen into fibrin. Increased amounts of TAFIa is generated, which together with increased levels of PAI-1 and PAI-2 reduce fibrinolytic activity that would normally occur through tPA-induced generation of Pm from Pg in generating fdp. The *bold arrows* signify increased generation, and the *dotted arrows* signify inhibition.

Figure 5 – comparison between normal endothelium and placental trophoblast

In addition, local hypoxia and hypovolemia trigger endothelial response leading to increased expression of vascular endothelial growth factor, which causes an increased endothelial expression of TF. Evidence in support of this view is brought by Erez etal[22] who demonstrated that in women with fetal death, those who had abruption had a higher amniotic fluid of TAT complexes. These events result in the

consumption of coagulating factors, fibrin deposition in microcirculation, and thrombus formation on maternal surface of the placenta at the site of abruption. This is followed by fibrinolysis and the release of fibrin degradation products further contributing to the development of DIC.

Of interest, if the abruption in concealed or it is severe enough to cause fetal demise, it is at much higher risk for the development of DIC because of a continuous release of TF in the maternal circulation. The probable mechanism leading to this observation is similar to that observed in amniotic fluid embolism with systemic release of TF that leads to systemic activation of coagulation and subsequent DIC.

This view is supported by the experiment reported by Schneider[46] which demonstrated that the intravenous injection of placental extracts into mice leads to the death of the animal through DIC, which can be prevented by the administration of heparin.[46]he author identified thromboplastin as the causative agent through its effect on the clotting time and its chemical properties and measured its activity by the 1-stage prothrombin-time method.[46]

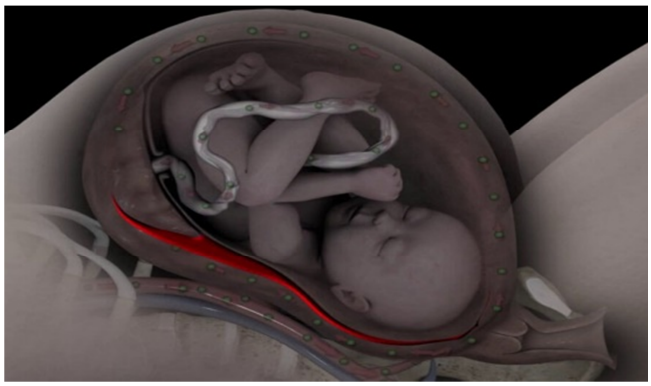
Hemorrhage

Acute obstetrical bleeding is being considered by many[45]as a leading cause for DIC. This form of consumption coagulopathy is classically related to PPH as a result of uterine atonicity, retained placenta or membranes, uterine rupture, placenta accreta, or severe cervical or vaginal lacerations. In all of these cases, the mother is losing a large volume of blood and coagulation factors in a short time interval, and these patients are usually hemodynamically compromised.

Currently there is a debate whether this form of consumption coagulopathy is truly DIC or just a massive blood loss that depletes the patient's coagulation factors and can lead to death because of exsanguination.[10]However, massive maternal bleeding may not be that straightforward as a pure loss of coagulation factors. During the time of parturition and postpartum period, there is substantial activation of coagulation cascade and generation of thrombin as a result of the release of TF to the maternal circulation following the separation of the membranes and the placenta.[47]Thus, these women already have increased thrombin generation and indeed are regarded as high-risk patients for the development of deep vein thrombosis during the puerperium.[47]

The evidence brought here in, that parturient with PPH have a higher activation of coagulation cascade even above the physiological threshold, suggests

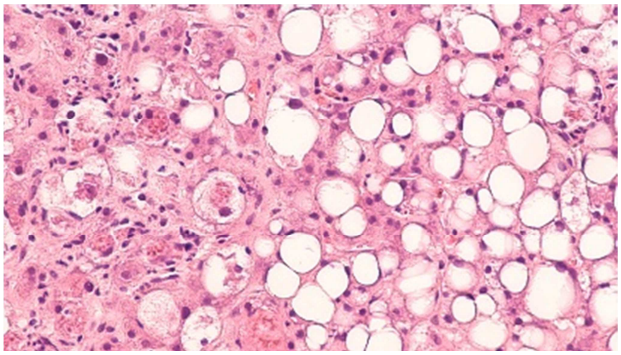
that the clinicians who treat these patients must regard them as a high-risk group for DIC, even though the fundamental pathology is a rapid and massive loss of blood as well as coagulation factors.[44]Therefore, patients with PPH need to be treated promptly, medically, and/or surgically and by blood products as well as volume expanders to sustain the maternal circulation and perhaps to prevent the subsequent development of DIC.



Placental Abruption

The reported incidence of placental abruption varies but averages approximately 0.5% or one in 200 births.[48]It is a common cause of perinatal mortality and approximately 10% of third-trimester stillborn neonates are attributed to abruption. According to the Centers for Disease Control and Prevention, placental abruption was the direct cause of maternal mortality in 1.1% of pregnancy-related deaths in the United States from 2006 to 2010.[49] Extensive placental abruption causes immediate and frequently profound DIC. This is initiated by large amounts of decidual and placental-derived tissue factor that rapidly enters the maternal circulation to activate widespread coagulation with depletion of procoagulants. Clotting intensity and plasma fibrinogen depletion are related to several important factors. The first of these is the amount of placental tissue involved, and thus total abruptions typically cause more intense DIC than partial ones. Specifically, one third of women with an

abruption severe enough to kill the fetus will have a plasma fibrinogen less than 150 mg/dL.[50] Second, a woman with a concealed abruption partial or complete more likely will exhibit DIC because the intrauterine pressure is higher than in those patients with external vaginal bleeding. The third important factor is the baseline fibrinogen level recall that plasma fibrinogen levels are elevated substantively in late pregnancy and range from approximately 400 to 650 mg/dL.[31]Thus, a woman with a fibrinogen level of 600 mg/dL might have a level of 300 mg/dL post abruption, which signifies massive intravascular utilization of fibrinogen, but at the same time, plasma fibrinogen concentration is sufficient to maintain hemostasis. Lastly, the duration of ongoing DIC caused by an abruption appears to be self-limited. Although the plasma fibrinogen nadir will usually be manifest by 8 hours, continuing blood loss from the implantation site will result in procoagulant deficiency if only packed red cells are transfused.[50]



Acute fatty liver of pregnancy

Acute fatty liver of pregnancy is a rare (an estimated incidence between 6 and14 per 100,000 pregnancies[51]) but potentially fatal complication of pregnancy. It is characterized by fatty microvascular infiltration of hepatocytes with progressive loss of liver function[52] without alteration of the overall structure of the liver. Women who develop this complication have abnormal renal function and DIC[52].The mechanisms by which DIC develops in this complication is a combination of reduced liver

production of fibrinogen as well as coagulation proteins and hemorrhage.

Evidence in support of this view is presented by Nelson et al studied 51 women with acute fatty liver of pregnancy. Their hemostatic condition was classified according to the International Society of Thrombosis and Hemostasis DIC score, and 80% of these women had unequivocal DIC defined as composite score of 5 or greater. The authors studied the hepatic and hemostatic function of these patients including fibrinogen, fibrin-fibrinogen split products, coagulation studies, and cholesterol. Those who developed DIC had abnormally low plasma fibrinogen concentrations that persisted for the first several days after delivery along with only mild to moderately elevated fibrin degradation products.[53]

At the same time, there was also evidence for continuing increased procoagulant consumption caused by ongoing DIC provided by the modestly elevated levels of fibrin degradation products in the face of depressed plasma fibrinogen concentrations. This observation was in contrast to that of patients with abruption in whom the fibrinogen concentration recovered into normal range several hours after the acute event. Collectively the continuous low fibrinogen concentration and abnormal function of the coagulation cascade is the result of the liver dysfunction associated with acute fatty liver of pregnancy, leading to a lower production of coagulation factors, anticoagulation proteins, and fibrinogen by the liver.

HELLP Syndrome

This condition is an additional cause for DIC in obstetric patients that may involve the liver. The relationship between acute fatty liver of pregnancy and HELLP syndrome has not been clearly established. There are obviously common clinical and biological features between these 2 entities.[52]Indeed, some authors have suggested that acute fatty liver of pregnancy and HELLP syndrome are the 2 faces of the same coin. However, others found that a difference in liver histopathology (fatty microvascular infiltration of hepatocytes vs fibrin deposition or hemorrhage in the periportal areas) makes an overlap between these 2 entities not possible.[54]

One of the major differences between acute fatty liver of pregnancy and HELLP syndrome is the prevalence of DIC. In a study by Vigil-De Gracia[52] DIC was present in more than 70% of patients with acute fatty liver of pregnancy and less than 15% of those with HELLP syndrome. Thus, although women with HELLP syndrome have a reduced production of fibrinogen and other coagulating as well as anticoagulation factors that can lead to the development of DIC[54] this is not the central feature of this disease. From the evidence brought herein, DIC is a central feature of acute fatty liver and in a way reflects the severity of the hepatic injury, whereas in HELLP syndrome, it is present in only a fraction of the patients, probably those with a more severe form of the microangiopathic hemolytic anemia associated with this syndrome.

The following are reviews from various articles:

1) In a Retrospective multiple logistic Analysis done between 2006-2013 it was found that
   - 30.4% patients were discharged successfully
   - 69.6% ended up with either mortality or morbidity.

Morbidities among these were as follows

- Respiratory Failure in 10 patients (19.22%)
- Acute Renal Failure in 5 patients (9.6%)
- PRES in 4 patients (7.7%)
- SEPSIS in 2 patients
- Hematoma of liver in one patient (1.9%)
- Intra cranial Hemorrhage in one patient (1.9%)
- ARDS in one patient (1.9%)
- Hepatic Failure (1.9%)

Mortality Causes include ARF, Sepsis, SAH, Hypovolemic shock, HIE, ARDS. They concluded that the lung edema in ARDS is due to rapid transfusion of massive volumes of blood & blood products. Also, incidence of DIC is more in women who doesn't attend ANC regularly. Therefore, suggesting DIC can be preventable with early diagnosis.[2]

2) In a prospective one-year study done in 2016 using Modified ISTH (International Study on Thrombosis and Hemostasis) DIC Score where only 3 components (platelet count, fibrinogen, PT) of DIC score were used to establish the diagnosis of overt DIC, they found out there are unnecessary blood transfusions in 179 of the postpartum women (64.1%). Thus, ultimately it might help in preventing morbidity (ARDS, AKI, Thromboembolism etc.,)[3]

3) In a descriptive case series over a 6-month period done in Karachi, Pakistan found out various causes of DIC of which eclampsia and pre-eclampsia (56%), abruption (20%), placenta Previa (18%), Postpartum hemorrhage (2%). Also, prevalence of anemia is more in these patients-Hb-7-9gms is 68%. Thus, suggesting major causes of DIC are preventable if

- Diagnosed early
- Proper Antenatal care given to women
- Created awareness regarding importance of approaching a health facility regularly [4]

The present study considered Karachi study as review as the socioeconomic and health sector set up matches to the area of study to some extent.

4 A Retrospective study of 25 patient records from 1993-2005 in Songklanagarind University concluded early diagnosis and Prompt treatment which includes quick decision for surgical intervention and eradicating the predisposing conditions will minimize the maternal morbidity and mortality.[5]

5. A Retrospective study of 49 cases over a period of 30years (1980-2009) at Dalhousie university, Nova Scotia, Canada showed the following findings: -

- Etiology – Placenta abruption (37%), Postpartum Hemorrhage (29%), Pre-eclampsia/HELLP (14%), acute fatty liver (8%), sepsis (6%) and amniotic fluid embolism (6%).
- Maternal morbidity included transfusion of >_ 5 units (59%), hysterectomy (18%), ICU admission (41%) and Acute tubular necrosis requiring dialysis (6%).
- Maternal mortality rate is 61.2 % (n=3), case fatality rate was 1 in 16.
- Prompt recognition and treatment with timely administration of blood products is crucial in the management of this life-threatening disorder.

MATERIALS AND METHODS

Source of Data

All the admitted women who are diagnosed as DIC at KAHER'S DR. Prabhakar Kore Charitable hospital.

Method of collection of data

a) Study design: An observational study

b) Study setting:

KAHER's Dr. Prabhakar Kore Charitable Hospital, Belagavi.

C) Duration of data collection: 1 year 9 months

d) Study Period: January 2020 – September 2021

e) Study Population:

Pregnant women admitted and diagnosed as Disseminated Intravascular coagulation at KAHER'S DR. Prabhakar Kore Charitable Hospital, Belagavi**.**

Selection criteria

Inclusion Criteria:

- Pregnant Women diagnosed as DIC irrespective of the Gestational Age.
- Post-delivery (post-natal & post-operative) patients who are in DIC
- Patients who delivered outside and referred to our Centre and were diagnosed as DIC.
- DIC is diagnosed using International society for Thrombosis & Hemostasis (ISTH) criteria for diagnosing DIC

Exclusion Criteria:

- DIC due to non-Obstetric causes
  - a) Vasculitis-causes consumption of platelets
  - b) Thrombotic thrombocytopenic purpura (TTP)
  - c) Hemolytic Uremic syndrome
  - d) Hematological malignancies causing DIC
  - e) Patients on treatment with anti-neoplastic drugs
  - f) Crush injuries
  - g) Patients with Malignancies (Breast, lung, pancreatic, GI malignancies)
  - h) Chronic inflammatory disorders.
- Known case of bleeding disorders

f) Sample Size:

Chi square test is used to calculate the minimum sample size required for this study. R 4.1.1 software has been used for calculating the same.

Assuming moderate effect size between ISTH standard DIC score and outcome, at 90% power and 5% level of significance, the minimum sample size required is $42.029 \approx 42$.

Larger the sample size, better the precision. Hence, 50 subjects are considered in this study.

g) Statistical Analysis:

Since the study is of observational study the plan of analysis will be as follows. Data is analyzed using R software version 4.1.1 and Excel. Categorical variables are given in the form of frequency table. Continuous variables are given in Mean ± SD/ Median (Min, Max) form. Two sample t test/Welch's t test is used to

compare means of variables over outcome. Mann Whitney U test is used to compare the distributions of variables over outcome. Chi-Square test is used to check the association between attributes. Logistic regression is used to find the effect of ISTH standard and pregnancy modified DIC score on predicting mortality. P-value less than or equal to 0.05 indicates statistical significance.

METHODOLOGY:

All the patients selected as per the inclusion criteria will be enrolled in the study. Women as per the exclusion criteria will not be included in the study.

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 (platelet count, prothrombin time (PT), fibrinogen, soluble fibrin monomers or fibrin degradation products)

3. Score global coagulation test results

DIC is diagnosed using Pregnancy Modified International society for Thrombosis & Hemostasis (ISTH) criteria.

Parameter	Pregnancy Modified ISTH DIC SCORE
Platelet Count (X 10^9/L)	
>185000	0
>100000	1
50000-100000	2
<50000	1
PT Difference(s)	
>1.5	25
1-1.5	12
0.5-1	5
<0.5	0
Fibrinogen(g/L)	
≤3	25
3-4	6
4-4.5	1
≥4.5	0
score	>26 – Overt DIC

Sensitivity 91%, Specificity 97%

The eligible candidates will be closely followed right from the day of admission to the day of discharge.

Details of the following will be taken

- Details of the patient- Age, Address
- Obstetrics History in detail

- Presentation at the time of admission(condition)
- Mode of delivery, Interventions taken (surgical & medical)
- Investigations –

a) Hematological- Hemoglobin (Hb), Peripheral Smear, PCV, Platelets, Total count, Differential Count, Liver function tests (SGPT, SGOT, Alkaline phosphatase, LDH, Total bilirubin, Total protein), Renal function Tests (urea, creatinine, uric acid), Bed side Clotting Time, Coagulation studies (D-Dimer, APTT, PT, INR, Serum fibrinogen)

b) Radiological – obstetric scan, USG Abdomen- pelvis, CT, MRI.

c) Urine Routine & microscopy, Urine Culture and sensitivity

- Treatment given like-

    a) Antihypertensive, use of MgSo4

    b) Antibiotics for infection or sepsis

    c) Supportive therapy -ventilator support, CPAP, Ionotropic drugs

- Blood transfusion details –

    a) volumes and type of blood or blood product

    b) Transfusion related Problems like overload, citrate toxicity, Reactions developed if any etc.,

- Type of intervention required if any
- Post discharge morbidities
    - Causes of mortality if patient ends up in death.

RESULTS

Data contains measurements on 50 subjects whose age ranged from 19 years to 34 years with mean age 25.15 ± 4.35 years. The following table gives the comparison of different variables with outcome.

Table 1 – Distribution of age

Age(years)	Total
≤20	9 (18.37%)
21-25	18 (36.73%)
26-30	17 (32.65%)
31-35	6 (12.24%)

Out of 50 patients 10 %(n=5) belong to upper class, 26% (n=13) belong to upper middle class, 42% (n=21) belong to lower middle class & 22% (n=11) belong to lower class. The distribution is not statistically significant.so there is no relation between patients developing DIC & socioeconomic status.

Table 2 – Distribution according to socioeconomic status

Variable	Number	Percentage
Upper class	5	10
Upper middle class	13	26
Lower middle class	21	42
Lower class	11	22

Out of 50 patients 91.8% (n= 45) patients were referred cases to our institute, but were taking antenatal visits regularly at their respective registered hospitals or clinics.8.1% (n=5) patients were registered at our institute.

Table 3 – Registered cases and referred cases

Variable	Number	Percentage
Referred cases	45	91.84
Registered cases	5	8.16

Table 4- The distribution of various blood parameters in patients

Variables	**Sub Category**	**Mortality**	**Recovered**	**Total**	**P-value**
Platelets	Mean ± SD Median (Min, Max)	55000 ± 29722.53 63500 (14000, 97000)	92268.29 ± 62109.99 79000 (9000, 218000)	86183.67 ± 59474.67 72000 (9000, 218000)	0.0164[WT]
Total WBC count	Mean ± SD Median (Min, Max)	27500 ± 19098.69 30050 (8500, 66000)	17613.02 ± 8881.94 16200 (5200, 49800)	19227.22 ± 11513.77 17006 (5200, 66000)	0.1913[WT]
Urea	Mean ± SD Median (Min, Max)	78.29 ± 66.3 74 (14, 203)	29.8 ± 15.24 26.5 (12, 65)	37.02 ± 32.78 28 (12, 203)	0.1015[WT]
S. Creatinine	Mean ± SD Median (Min, Max)	2.31 ± 1.78 1.42 (0.57, 4.5)	1.12 ± 0.67 0.9 (0.45, 3.8)	1.3 ± 0.99 0.9 (0.45, 4.5)	0.1556[MW]
Uric Acid	Mean ± SD Median (Min, Max)	6.71 ± 2.58 7 (3.1, 10.6)	6.19 ± 1.53 6.3 (2.7, 9.4)	6.28 ± 1.7 6.4 (2.7, 10.6)	04649[t]
Direct bilirubin	Mean ± SD Median (Min, Max)	2.69 ± 3.16 0.48 (0.3, 7.8)	2.06 ± 3.94 0.45 (0.02, 17.13)	2.16 ± 3.81 0.48 (0.02, 17.13)	0.3092[MW]
Total protein	Mean ± SD Median (Min, Max)	4.19 ± 0.92 4.5 (2.3, 5.1)	5.07 ± 1.13 5.2 (1.8, 7)	4.94 ± 1.14 4.9 (1.8, 7)	**0.029[MW]***
S. Albumin	Mean ± SD Median (Min, Max)	4.34 ± 5.15 2.6 (1.8, 16)	2.69 ± 0.62 2.8 (1, 3.9)	2.93 ± 2.03 2.7 (1, 16)	0.3605[MW]
SGOT	Mean ± SD Median (Min, Max)	260.57 ± 272.46 140 (27, 784)	101.75 ± 195.4 35 (6, 1044)	125.4 ± 212.89 44 (6, 1044)	**0.0259[MW]***
SGPT	Mean ± SD Median (Min, Max)	148 ± 112.26 143 (13, 372)	65.62 ± 120.98 16 (3, 569)	77.89 ± 122.19 16 (3, 569)	**0.0136[MW]***
LDH	Mean ± SD Median (Min, Max)	1747.43 ± 1985.1 890 (358, 5525)	682.71 ± 407.57 616.5 (138, 2018)	848.33 ± 910.68 630 (138, 5525)	0.2103[MW]
ALP	Mean ± SD Median (Min, Max)	318.86 ± 329.31 133 (45, 896)	219.2 ± 200.46 162.5 (30, 792)	234.04 ± 222.49 162 (30, 896)	0.8812[MW]

The mean hemoglobin among 50 patients is 8.33gm/dl, the minimum hemoglobin is 1gm/dl & maximum hemoglobin is 14gm/dl, this might be because of hemoconcentration due to severe preeclampsia, abruption & other factors. The mean hemoglobin among the mortality group is 6.18gm/dl & in recovered group is 8.75gm/dl.

The mean platelets among 50 patients is 86,183, the minimum is 9,000 & maximum is 2,18,000. The mean platelets among the recovered group is 92,268 & in mortality group is 55,000. The distribution of platelets between the two groups is not statistically significant.

The mean total count among 50 patients is 19,227, the minimum is 5,200 & maximum is 66,000. The mean total count among the recovered group is 17,613 & in mortality group is 27,500. The distribution of total count between the two groups is not statistically significant.

The mean urea among 50 patients is 37, the minimum is 12 & maximum is 203. The mean among the recovered group is 29.8 & in mortality group is78.29. The distribution of urea between the two groups is not statistically significant.

The mean serum creatinine among 50 patients is 1.3, the minimum is 0.45 & maximum is 4.5. The mean among the recovered group is 1.12& in mortality group is 2.31. The distribution of serum creatinine between the two groups is not statistically significant.

The mean uric acid among 50 patients is 6.28, the minimum is2.7 & maximum is 10.6. The mean among the recovered group is 6.19 & in mortality group is 6.71. The distribution of uric acid between the two groups is not statistically significant.

The mean direct bilirubin among 50 patients is 2.16, the minimum is 0.02 & maximum is 17.13. The mean among the recovered group is 2.06 & in mortality

group is 2.69. The distribution of direct bilirubin between the two groups is not statistically significant.

The mean total protein among 50 patients is 4.94, the minimum is 1.8 & maximum is 7. The mean among the recovered group is 5.2 & in mortality group is 4.5. The distribution of total protein between the two groups is not statistically significant.

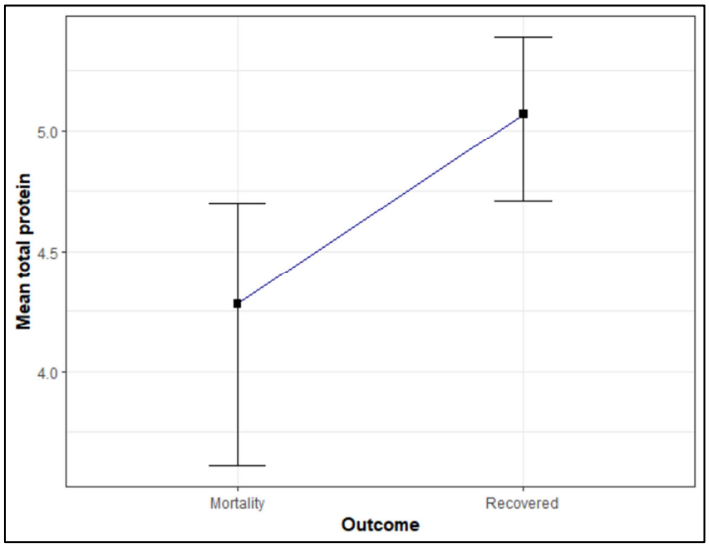


Figure 6: Mean plot of total protein over outcome.

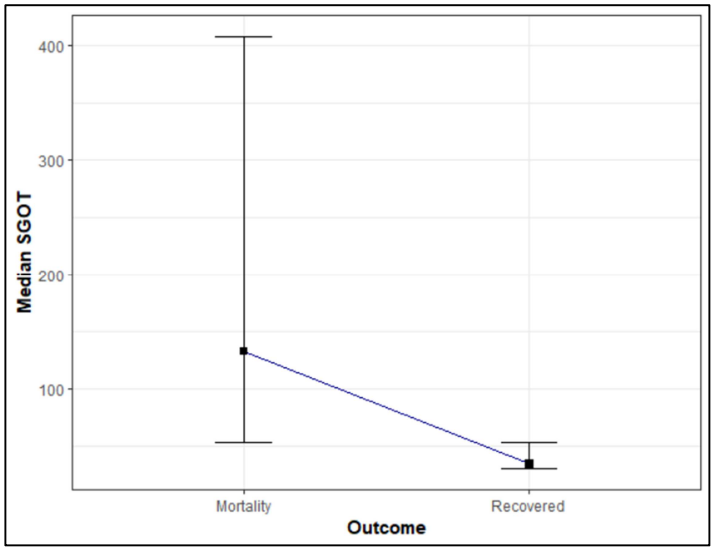


Figure 7: Median plot of SGOT with outcome.

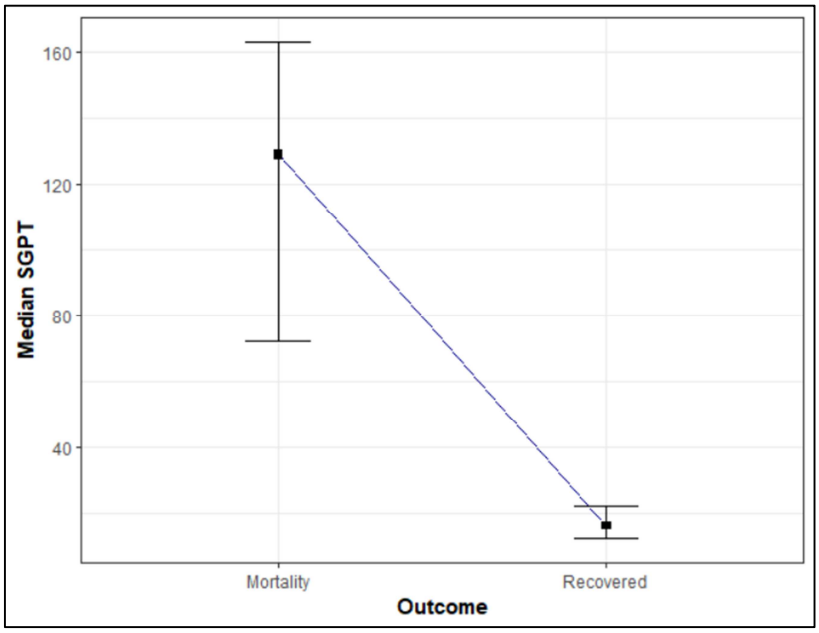


Figure 8: Median plot of SGPT over outcome.

Table 5 – Comparison of DIC Profile in mortality and recovered group.

Variables		Mortality	Recovered	Total	P-value
D-Dimer	<5000	2 (25%)	12 (29.27%)	14 (28.57%)	1^{MC}
	≥5000	6 (75%)	29 (70.73%)	35 (71.43%)	
APTT		30.18 ±61.94	11.55 ±31.91	14.59 ±38.16	0.0149^{MW}*
PT		44.06 ±48.67	14.03 ±25.68	18.93 ±31.95	0.008^{MW}*
PT difference		37.51 ±44.49	129.55 ±779.84	114.53 ±712.93	0.001^{MW}*
INR		4.15 ±3.67	1.85 ±1.93	2.23 ±2.41	$< 0.001^{MW}$*
S. Fibrinogen		69.5 ±22.19	117.1 ±103.53	109.33 ±96.54	0.1132^{MW}

There is significant difference in the distribution of total protein, SGOT, SGPT, APTT, PT, PT difference and INR over outcome.

Table 6- Comparison of Pregnancy Modified DIC Score over maternal outcome

	Variables	Mortality	Recovered	Total	P-value
Pregnancy Modified DIC Score	>26	8 (100%)	36 (85.37%)	44 (87.76%)	0.5752[MC]
	≤26	0	6 (14.63%)	6 (12.24%)	

Total 44 patients (87.76%) patients had DIC Score of >26 and out of these 8 patients had ended up in mortality.6 patients had DIC Score of ≤26 & all were recovered. Among non-survivors all patients had DIC Score of >26, i.e., all patients who ended up in mortality had DIC Score of >26.Among survivors 85.37% patients (n =36) had DIC Score of >26 and 14.63% patients (n=6) had DIC Score of ≤ 26.

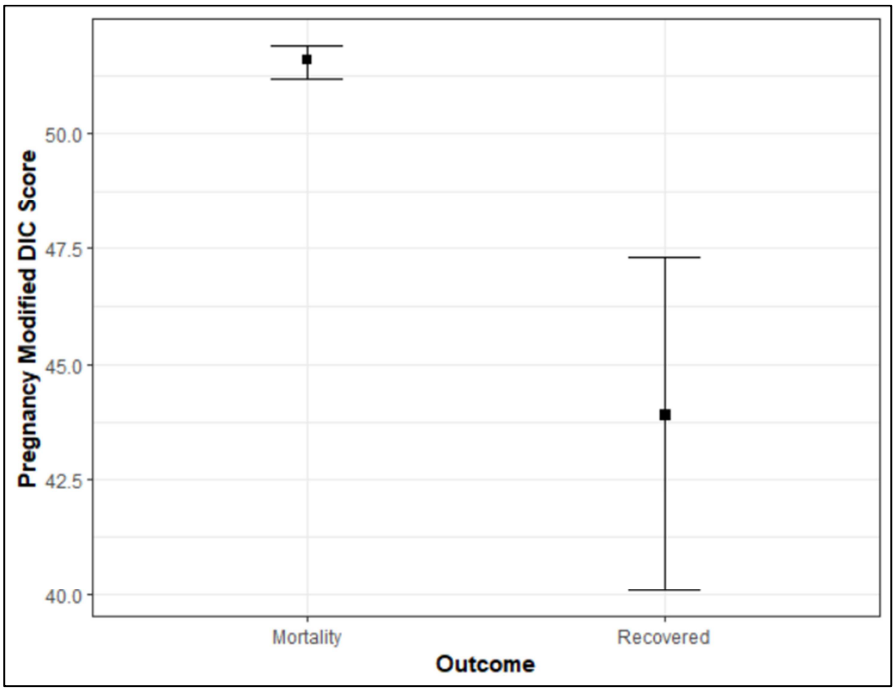


Figure 9: Mean plot of pregnancy modified DIC Score over outcome.

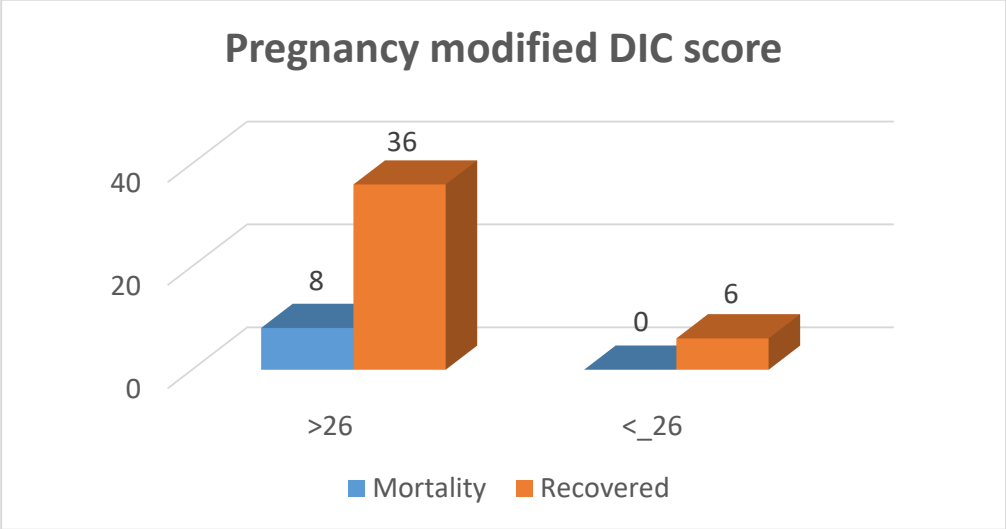


Figure 10 – Graphical representation of pregnancy modified DIC Score and maternal outcomes

Table 7 – Maternal outcomes

Total No. of patients	Recovered		Mortality
N= 50	42(84%)		8 (16%)
	Morbidity	24 (48%)	
	No Morbidities	18 (36%)	

Out of 50 patients 84% recovered and 16% ended up in mortality. Out of the recovered patients 48% had developed morbidities during the hospital stay, but eventually recovered and discharged without any complications.

Out of 50 patients 12.24% had Abruption , 10.2% (n=5) had abruption with IUD ,10.2%(n=5) had AFLP.8.16%(n=4) patients had DIC due to retained products of conception, among these 4%(n=2) are due to second trimester abortion and 4% (n=2) had retained products of conception post Caesarean section(outside delivered), for which exploratory laparotomy was done at our Centre and immediate resuscitation & management was done & were recovered.6.12% (n=3) patients developed DIC due to

HELLP Syndrome and 4% (n=2) patients due to severe preeclampsia. Ruptured ectopic pregnancy, placenta Previa contributes 4% (n=2) patients each. 2% (n=1) patients developed DIC due to placenta percreta, spontaneous abortion, dengue with thrombocytopenia each.

Table 8 – List of etiological factors distribution

Etiological factor	Number & Percentage
Post-Partum Haemorrhage	13 (26.52%)
Abruption	11 (12.24%)
IUFD	5 (10.2%)
AFLP	5 (10.2%)
Retained products of conception	4 (8.16%)
HELLP Syndrome	3 (6.12%)
Severe PE	2 (4.08%)
Ruptured Ectopic Pregnancy	2 (4.08%)
Placenta Previa	2 (4.08%)
Placenta percreta	1 (2.04%)
Spontaneous abortion	1 (2.04%)
Dengue with Thrombocytopenia	1 (2.04%)

In the present study the most common etiology causing DIC is Postpartum Hemorrhage followed by abruption, IUFD and AFLP. Out of 26.5% (n=13) cases of PPH 69% (n=9) had atonic PPH, 23% (n=3) had traumatic PPH & 7% (n=1) had secondary PPH.

Table 9 – Types of PPH

Type of PPH	Number (n=13)
Atonic	9 (69.2%)
Traumatic	3 (23.07%)
Secondary	1 (7%)

Maximum number of units of transfusion among the patients enrolled in the study is as follows: -

- Whole blood – 5 pints
- Packed cell volume – 15
- RDP - 21
- SDP – 4
- FFP- 24
- Cryoprecipitate- 10

From Mann Whitney U test, there is no significant difference in the distribution of blood transfusion variables over outcome. So, this implies that the outcome doesn't depend up on the number of units of blood products transfused but intern depends on timely intervention & correction of DIC in the early stage might prevent mortality.

Table 10 - Comparison of Blood Transfusion Variables over Outcome

Variables	Mortality	Recovered	Total	P-value
Whole blood	1 ± 1.93	0.15 ± 0.36	0.29 ± 0.87	0.3327 MW
Packed cells	4.38 ± 4.63	2.39 ± 2.35	2.71 ± 2.88	0.1458 MW
RDP 's	7.5 ± 7.01	3.41 ± 3.81	4.08 ± 4.65	0.0971 MW
SDP's	0.25 ± 0.71	0.37 ± 0.94	0.35 ± 0.9	0.6884 MW
FFP's	5.88 ± 5.08	5.29 ± 4.93	5.39 ± 4.91	0.7329 MW
Cryoprecipitate	4.12 ± 3.87	2.83 ± 3.22	3.04 ± 3.32	0.3583 MW

Out of 50 patients, 48% (n=24) developed complications. The most common complication is AKI & shock - 10% (n=6).6.12% (n=3) developed sepsis with MODS. Severe anemia is seen in 4% (n=2) patients. Common Femoral artery embolism, dengue shock syndrome, dilated cardiomyopathy, pulmonary thromboembolism, DVT, Sepsis with hepatic encephalopathy, sub segmental atelectasis were seen in 2% patients (n=1) each.

Table 11 – Distribution of Maternal Outcomes

Maternal Complication	Percentage
Acute Kidney Injury	6 (10%)
Shock	6 (10%)
Sepsis with MODS	3 (6.12%)
Common Femoral Artery Embolism	1 (2.04%)
Dengue Shock Syndrome	1 (2.04%)
Dilated cardiomyopathy	1 (2.04%)

Pulmonary Thromboembolism	1 (2.04%)
Pulmonary Thromboembolism & DVT	1 (2.04%)
Sepsis with Hepatic encephalopathy	1 (2.04%)
Severe Anaemia	2 (4.08%)
Sub segmental Atelectasis	1 (2.04%)

On looking into further details of AKI 4% (n=2) developed simple AKI, 2% (n=1) developed AKI with COVID 19 positive status, AKI with ARDS, AKI with toxic ischemic Nephropathy with pleural effusion, AKI with retinal detachment.

Table 12 – Elaboration of AKI

Acute Kidney Injury	n = 6 (10%)
AKI	2 (4.08%)
AKI with COVID positive	1 (2.04%)
ARDS with AKI	1 (2.04%)
AKI secondary to ischemic & Toxic Nephropathy, Pleural Effusion	1 (2.04%)
Retinal detachment, AKI	1 (2.04%)

Total 2 patients were COVID 19 positive. This study was done from January 2020 to September 2021 (1 year 9 months). During this period there was COVID 19 pandemic which started in the study region from April 2020.Out of these 2 patients one patient developed AKI and the other patient didn't had any morbidities.

Table 13- Comparison of type of DIC over maternal outcome.

	Variables	Mortality	Recovered	Total	P-value
Type of DIC	Antepartum	1 (3.33%)	29 (96.66%)	30 (100%)	0.0075
	Postpartum	7 (35%)	13 (65%)	20 (100%)	

There is significant association between the type of DIC and outcome.

In the present study 60% had antepartum DIC and 40% had postpartum DIC. Out of the antepartum DIC 96.66% recovered and 3.33% ended up in mortality. Among the postpartum DIC 65% recovered and 35% ended up in mortality.

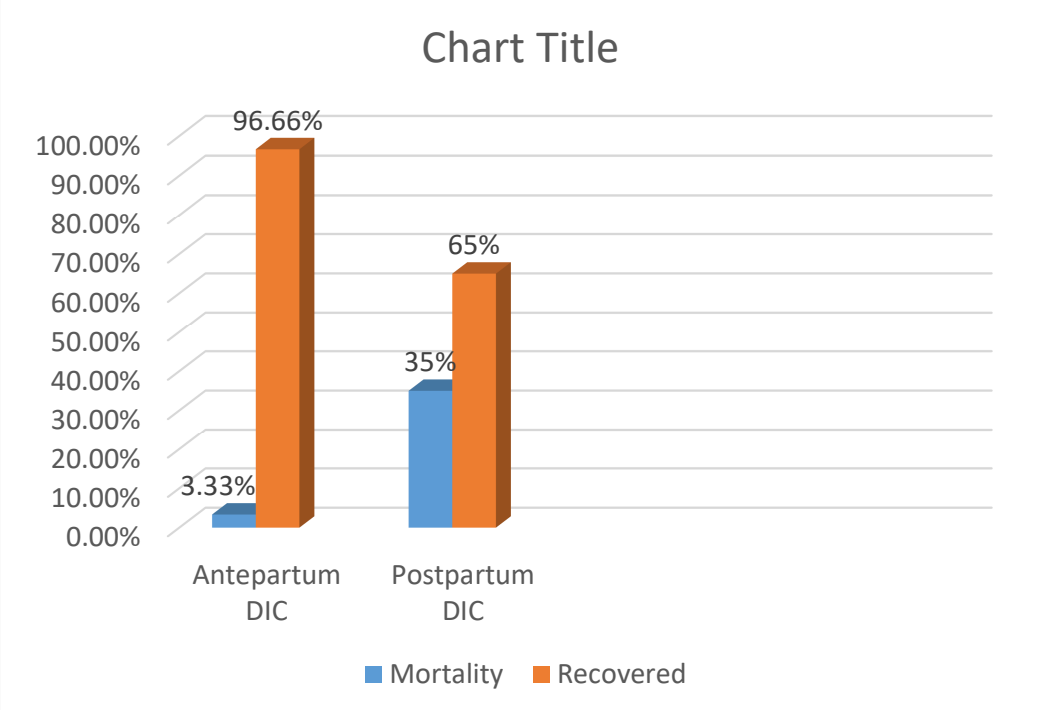


Figure 11- Showing mortality & recovery rate in antepartum and postpartum DIC

Table 14 – Mortality rate and etiology

Etiology	Patients who died	Patients who lived	Mortality Rate (%)	Recovery rate (%)
Postpartum Hemorrhage	5	8	38.46%	61.5%
Abruption	0	11	0	100%
IUFD	0	5	0	100%
AFLP	0	5	0	100%
Hypertensive disorders	0	5	0	100%
Placenta Previa	1	2	33.33%	66.67%
Retained products of conception	1	3	25%	75%

One patient died due to hemoperitoneum due to Thrombocytopenia with Dengue. This can't be included in PPH because uterus & cervix were absolutely fine. Uterus was well contracted. Also, this was not compared in the above table because we had only one such case & other patients who had dengue & thrombocytopenia alone were not included in the present study because they didn't develop DIC.

DISCUSSION

DIC is an acquired coagulation disorder due to various etiological factors. The incidence of DIC in the present study is 1.4%. The incidence is little higher compared to literature. This might be because the study was conducted at a tertiary care Centre with advanced technology and ICU services and so eventually receives high risk and complicated referral patients from the surrounding areas. In a study conducted by Serdar Basaranoglu et al. the incidence is 32%.The mortality rate is 25%[2]. In the present study the incidence of DIC is 1.4% and maternal mortality is 16%. In other recent studies conducted by various centers varies the maternal mortality is between 6.25% - 24%.

Fibrinogen is an acute phase reactant, which is also used in DIC scoring. In the study conducted by, Wada et al. suggested that high fibrinogen levels were associated with organ failure. They reported that DIC patients with high fibrinogen levels exhibited less activation of secondary fibrinolysis, resulting in organ failure and poor prognosis[55]. On the other hand, Sawamura et al. reported that reduced fibrinogen levels were associated with decreased survival[56]. In the present study, fibrinogen levels were found to be lower in non-survivors compared to survivors; however, difference between the two groups of patients was not statistically significant.

D-Dimer levels increase when there is breakdown of fibrin & indicate coagulation activation. Also note that D-dimer levels are elevated in pregnancy & so won't be of much help.so in this study pregnancy modified ISTH DIC Score is used for the diagnosis of DIC.

PT, INR, APTT & PT difference has significant statistical difference.PT, INR, APTT are increased in mortality group. Surprisingly PT difference was more in recovered group which might also imply that immediate correction of DIC &

necessary management at our Centre might had helped these patients to recover. In a study conducted in Turkey by Serdar Basangoulu et al. PT & INR were higher in non-survivors which is similar to the present study.[2]

Coming to the biochemical parameters in the present study SGOT, SGPT were increased in mortality group compared to recovered group & the difference is statistically significant. Total Protein (TP) is decreased in non-survivors. Renal function Tests had no significant difference in the two groups. Serum creatinine and urea were increased in mortality group but the difference between mortality and recovered is not statistically significant. In a study conducted by Serdar Basaranoglu et al. found that increased SGOT , SGPT , LDH, urea, creatinine, PTT and INR values on admission serve as a warning signal for mortality in DIC. Among these factors, urea is the most important one affecting mortality. But in the present study there was no significant difference in urea levels.[2]

Transfusion of blood & blood products didn't show any significant statistical difference.4 patients had developed pulmonary Edema, 1 patient developed fever and 1 patient developed Laryngeal Edema & rashes.

In the present study the most common etiological factor causing DIC in pregnancy is Postpartum hemorrhage (26.52%), out of which atonic PPH (69.2%) followed by traumatic PPH (23%) and secondary PPH (7%). In a conducted by , Mehmet Sıddık Evsen et.al. study, the most common conditions causing DIC were postpartum bleeding, hypertensive diseases of pregnancy, placental abruption and some others.[2]

Out of the patients who had PPH 61.5% recovered & 38.4% ended up in mortality. Total 13 patients had PPH, out of them 5 people landed up in mortality and 8 people recovered. Out of these 5 patients 2 patients delivered vaginally in the

periphery and had atonic PPH and were referred to our center in irreversible shock and unconscious state and succumbed to death with in 24hours.One patient delivered through caesarean delivery following which she developed breathlessness and hypotension and was referred to our center in shock on inotropes. She was taken up for Exploratory laparotomy in view of hem peritoneum and was found to be bleeding from uterine incision site along with retained products of conception.24 hours post-operative period of laparotomy she succumbed to death. One patient who died due to secondary PPH delivered in periphery presented with breathlessness and per vaginal bleeding after one week of caesarean section was admitted under cardiology & on further evaluation patient had hem peritoneum and was taken up for exploratory laparotomy where bleeding noted from uterine rent seen at the uterine incision which was previously sutured with catgut 1-0, repaired the uterine rent but eventually patient developed cardiac arrest and succumbed to death after 34 days. The other patient was admitted and delivered vaginally after induction of labor in view of post datism developed Atonic PPH ,managed medically and transfusion of blood and blood products was done and patient recovered, but on Post-natal day one patient died due to pulmonary thromboembolism.

12.24% patients developed DIC due to abruption. On further analysis the cause of abruption is hypertension in pregnancy, anemia and most importantly the lack of knowledge to approach a health facility or inappropriate management led to DIC in pregnancy. This implies that adequate awareness regarding high risk pregnancies & training of ground level health workers & most importantly to educate the pregnant patient regarding warning signs in pregnancy & high-risk factors in pregnancy might contribute in reduction in DIC in pregnancy & thus maternal mortality.

Most common complication seen in patients who developed DIC in pregnancy is AKI & shock (10%).6.12% patients developed Sepsis with MODS ended up in mortality. All patients who had complications among the recovered group developed these complications during hospital stay. The duration of stay in hospital was little longer compared to others who had no complications, but eventually fully recovered & are leading a normal life without any residual morbidities.

One patient developed femoral artery embolism following uterine artery embolization for placenta percreta and ended up amputating the right lower limb above knee.

In the present study 60% patients had antepartum DIC out of which 3.33% ended up in mortality & 96.66% recovered and went home. Out of 40% patients of postpartum DIC 35% ended up in mortality and 65% patients recovered. So, the type of DIC might help in predicting mortality and also if properly managed most of the DIC patients might recover.

CONCLUSION

DIC caused by obstetrical conditions is associated with high mortality & morbidity rates. Patient's presenting with conditions known to be causing DIC should be assessed meticulously. All the necessary investigations & transfusion of blood & blood products should be done promptly, as deranged parameters like increased SGOT, SGPT, PT, APTT, INR, PT difference values are associated with multiorgan failure & increased morbidity and mortality. Outcomes in Antepartum DIC is better than Postpartum DIC. Transfusion of blood & blood products should be done vigilantly as adverse reactions due to these might be the cause of morbidity and mortality in DIC Patients. Patients presenting early and receiving a multidisciplinary treatment would recover better.

SUMMARY

The present study was an observational study conducted to analyze the maternal outcomes in pregnancy complicated with disseminated intravascular coagulation at a tertiary care Centre. The study was carried out at the department of Obstetrics and Gynecology of KAHER's Dr.Prabhakar Kore Charitable Hospital, Belagavi, Karnataka for a period of one year nine months. Patient population included all pregnant women admitted and diagnosed as Disseminated Intravascular coagulation. A total of 50 patients were enrolled in the study as per inclusion criteria. Data regarding sociodemographic, clinical, investigations and treatment has been collected in the form of structured proforma and analyzed statistically.

Key findings of this study have been summarized as follows:

- The age of the participants ranged from 19 years to 34 years with mean age 25.15±4.35 years. Maximum number of women were in the age group of 21-25 years (36.7%).
- Maximum proportion of study participants belong to lower middle class (42%).There is no relation between socioeconomic status and patients developing DIC.
- Maximum participants were referred cases to our institute contributing 91.8% and only 8.16% were registered cases.
- Among the laboratory parameters, there is statistically significant difference in the distribution of total protein, SGOT, SGPT, APTT, PT, PT difference and INR over outcome.
- Among non-survivors all patients had DIC Score of >26, i.e., all patients who ended up in mortality had DIC Score of >26.Among survivors 85.37% patients (n =36) had DIC Score of >26 and 14.63% patients (n=6) had DIC Score of ≤ 26.

- There is no significant difference in the distribution of blood transfusion variables over outcome. This implies that the outcome doesn't depend up on the number of units of blood products transfused but intern depends on timely intervention & correction of DIC in the early stage might prevent mortality.
- Out of 50 patients 84% recovered and 16% ended up in mortality. Out of the recovered patients 48% had developed morbidities during the hospital stay, but eventually recovered and discharged without any complications.
- Postpartum Hemorrhage (26.5%) followed by Abruption(12.2%), AFLP(10.2%), IUD(10.2%) are the main etiological factors of DIC.Other etiological factors include retained products of conception (8.1%),HELLP Syndrome(6.1%),severe Pre-eclampsia, ruptured ectopic pregnancy, placenta Previa each contributing to 4%.Placenta percreta, spontaneous abortion, dengue with thrombocytopenia each contributing to 2%.
- Out of 26.5% cases of PPH 69% had atonic PPH, 23% had traumatic PPH & 7% had secondary PPH.
- Out of 50 patients, 48% (n=24) developed complications. The most common complication is AKI & shock - 10% (n=6).6.12% (n=3) developed sepsis with MODS. Severe anemia is seen in 4% (n=2) patients. Common Femoral artery embolism, dengue shock syndrome, dilated cardiomyopathy, pulmonary thromboembolism, DVT, Sepsis with hepatic encephalopathy, sub segmental atelectasis were seen in 2% patients (n=1) each.
- Out of all patients who developed AKI- 4% (n=2) developed simple AKI, 2% (n=1) developed AKI with COVID 19 positive status, AKI with ARDS, AKI with toxic ischemic Nephropathy with pleural effusion, AKI with retinal detachment.

- Out of 50 patients 60% (n=30) had Antepartum DIC. Out of these 60%, 3.33% patients (n=1) ended up in mortality & 96.66% (n=29) patients recovered and went home.
- Out of 40% patients of postpartum DIC 35% ended up in mortality and 65% patients recovered.
- Among non-survivors 12.5% patients (n=1) had Antepartum DIC and 876.5% (n=7) had postpartum DIC. Among survivors 68.29% (n=29) patients had Antepartum DIC and 31.71% (n=13) had postpartum DIC.
- Out of 50 subjects who were in DIC, 84% recovered, 16% ended up in Death.Incidence of DIC is 1.4%maternal mortality rate due to DIC 16%.

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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)
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Ref: MDC/DOME/ 172 **Date: 24/12/2019**

To,

REG. NO. BJ0119010

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 **"MATERNAL OUTCOMES IN PREGNANCY COMPLICATED WITH DISSEMINATED INTRAVASCULAR COAGULATION AT A TERTIARY CARE CENTRE – AN OBSERVATIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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

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

ANNEXURE II – INFORMED CONSENT FORM.

Title of Research Study: "Maternal outcomes in pregnancy complicated with Disseminated Intravascular Coagulation at a Tertiary Care Centre – an Observational Study"

Principal Investigator: -

REG. NO. BJ0119010

Post Graduate student

Department of Obstetrics & Gynaecology

J.N Medical College, Belagavi.

Co-Investigator

Dr. _____

PROFESSOR

Dept. of Obstetrics & Gynaecology

J.N Medical College, Belagavi.

CONSENT FOR PARTICIPATION IN THE RESEARCH STUDY

Mrs. ---------- We are requesting you to enroll yourself in the study titled: "Maternal outcomes in pregnancy complicated with Disseminated Intravascular Coagulation at a Tertiary Care Centre –an Observational Study" conducted by **REG. NO. BJ0119010,** Post Graduate in M.S Obstetrics and Gynaecology under the guidance of Dr._____, Department of Obstetrics & Gynaecology. J.N.Medical College, KAHER; Belagavi.

The Purpose of this study is to see for the maternal outcomes in pregnant women with DIC.I will be the investigator for our study. The 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.

PURPOSE OF THE STUDY: To see for the maternal outcomes in women who were diagnosed as DIC during Pregnancy or following delivery and also to assess the expectant management given to these patients. Therefore to know the etiological factors causing DIC & role of blood & blood product transfusion and their after effects in these patients.

TYPE OF THE STUDY

This is an observational study. It involves noting down the investigations & treatment given to the patient from the time of admission to the time of discharge or death or patient leaving the Hospital (AMA).And also follow up to 3rd month after leaving the hospital.

PARTICIPANT SELECTION

We welcome all the admitted pregnant women who were diagnosed as DIC to participate in the study.

VOLUNTARY PARTICIPATION

Your participation in the Research is voluntary .It is your choice whether to participate or not .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 Dr.Prabhakar kore Hospital or J.N medical college. If you choose not to participate in the study, you will still be offered the treatment necessary for you. If you decide to participate in the study then you are free to withdraw at anytime.

PROCEDURE INVOLVED:

If you agree to enroll yourself in my study then your detailed present & past history will be taken to know if you are eligible for this study or not. If you have even one of the exclusion criteria then you will not be enrolled into this study. If you are eligible to participate in this study then along with the history, all the investigations done will be noted down and your treatment course also will be taken down on the proforma sheet till the time you leave the hospital. You will receive calls from me enquiring about your health condition till 3 months after leaving the hospital.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No financial incentives are being offered to enrolled patients. It is purely done with the idea of research and all the cost of the study will be borne by the investigator.

PRIVACY & 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:

1. In Emergency to protect your rights and welfare.
2. If required by law.

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 out come.

RIGHT TO REFUSE OR WITHDRAW FROM THE STUDY:

You do not have to participate in the study if you do not wish to. You can withdraw at any time from the study. There will be no penalty for withdrawal .Your treatment and care in this hospital will not change irrespective of whether you agree to participate or not. You can be removed from the study if necessary.

INSTITUTIONAL / SPONSOR'S POLICY:

In the event of any injury related to the study, treatment will be made available through KAHER; Belagavi. There is no compensation or payment for such medical treatment by the law. If you are injured you may contact **REG. NO. BJ0119010,** Post graduate student, Department of Obstetrics and Gynaecology, KAHER or by ph.No-8500921291**.**

CONTACT DETAILS:

In case you have any questions related to the study, in future or in case of study related problems you can contact **REG. NO. BJ0119010,** Post graduate student, Department of Obstetrics and Gynaecology, KAHER, Ph.No:_____ or Dr. _____ (OBG), Dept. of Obstetrics and Gynaecology, KAHER, Belagavi.

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

CONSENT STATEMENT:

I, _____ voluntarily agree for participating in this study. By signing this consent form I am not giving any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read the form 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 of Investigator:_____

Witness Name: _____ Signature: _____

Date:_____

ANNEXURE III - SCREENING FORM.

Screening number: | | | | | | |

Date of screening: _____

First name: _____

Middle name: _____

Last name: _____

Husband's name: _____

Age (years): _____

IP number: _____

Address: H.no _____

Street _____

Taluka _____

District _____

Phone number: | | | | | | | | | | |

Inclusion criteria satisfied: YES /NO

ANNEXURE III -PROFORMA

Name	
Age	
Address	
Phone number	
Date of admission	
Date of delivery Date of discharge	
IP No	

Level of Education

1= no formal schooling, illiterate ☐

2= no formal schooling, literate ☐

3= schooling ☐

4= PUC (11^{th} & 12^{th}) ☐

5= Degree holder ☐

Socio-economic status (according to modified B.J Prasad classification)

1 =upper class ☐

2=upper middle class ☐

3=middle class ☐

4=Lower middle class ☐

5=lower class ☐

Registered	
Unregistered	

Details of referral:-

Presenting Complaint :-

Obstetric history:

Gravida : para: Living: Abortions:

Trimester	Uneventful	Eventful	Details if eventful
T1			
T2			
T3			

Menstrual history:
LMP:-
EDD:-
POG:-

5. Past history:

CONDITION	YES	NO	IF YES DETAILS
Thyroid			
Hypertension			
Diabetes mellitus			
Asthma			
Tuberculosis			
Cardiac disease			
Previous surgeries			
Blood Transfusions			
Drug allergies			
Jaundice			
Others if any			

General physical examination- At Admission

Pulse rate	
Blood pressure	
Pallor	
Icterus	
Pedal edema Grade	

SYSTEMIC EXAMINATION:-

Respiratory system: _____

Cardiac system: _____

Central nervous system: _____

Abdominal examination:

UTERUS:-

FHS :-

Other significant findings:-

Provisional clinical diagnosis:-

Investigations:

DATE	
HAEMOGLOBIN	
PCV	
PLATELETS TOTAL COUNTS DIFFERENTIAL COUNT	
TOTAL BILIRUBIN DIRECT BILIRUBIN TOTAL PROTEIN S. ALBUMIN SGOT	

SGPT ALP LDH	
UREA URIC ACID CREATININE	
D-DIMER	
APTT	
PT	
INR	
FIBRINOGEN	
URINE ROUTINE & MICROSCOPY	
BED SIDE CLOT OBSERVATION TEST:	

Parameter	**Pregnancy Modified ISTH DIC SCORE**	Patient Score
Platelet Count(X 10^9/L)		
>185000	0	
>100000	1	
50000-100000	2	
<50000	1	
PT Difference(s)		
>1.5	25	
1-1.5	12	
0.5-1	5	
<0.5	0	
Fibrinogen(g/L)		
≤3	25	
3-4	6	
4-4.5	1	

≥4.5	0	
score	>26 – Overt DIC	

Parameter	**ISTH DIC SCORE**	Patient Score
Platelet Count(X 10^9/L)		
>100000	0	
50000-100000	1	
<50000	2	
Prolonged PT(s)		
≥6	2	
≥3 to 6	1	
<3	0	
Fibrinogen(g/L)		
<1	1	
≥1	0	
Fibrin Split products		
No Increase	0	
Moderate Increase	2	
Strong Increase	3	
score	>5 - overt DIC	

Established Diagnosis:-

Blood transfusion details -

DATE						TOTAL
WHOLE BLOOD						
PACKED CELLS						
RANDOM DONOR PLATELETS						
SINGLE DONOR PLATELETS						
FRESH FROZEN PLASMA						
CRYOPRECIPITATES						
OTHERS						

Period of transfusion	Details of blood & blood products transfused	Reason for transfusion	Any reactions / eventful findings
Pre operative period			
Intra operative period			

Post operative period			
Others if any			

Any reaction after transfusion:

- **Surgical interventions taken :-** Yes /No

If YES Details:-

Etiology		
Day of shifting out of ICU care		
Condition on discharge		
Number of days of hospital stay		
Condition after 3 months of delivery / discharge		
Result	Recovered	
	Mortality	

ANEXXURE IV

KEY TO MASTER CHART

DIC	-	Disseminated Intravascular Coagulation
ISTH	-	International society for thrombosis and haemorrhage
SGOT	-	serum glutamic-oxaloacetic transaminase
SGPT	-	serum glutamic-pyruvic transaminase
LDH	-	lactic acid dehydrogenase
PCV	-	Packed cell volume
RDP	-	Random Donor Platelets
SDP	-	Single donor platelets
FFP	-	Fresh frozen plasma
PT	-	Prothrombin Time
APTT	-	Activated prothrombin time
INR	-	International Normalised Ratio
ICU	-	Intensive Care Unit

OP Number / IP Number	Age in years	Registered / unregistered	Socioeconomic status	Gravida	Parity	Obs Score	Presented with complaint of	Diagnosis	Investigations	Time from onset of complaints to complication	Haemoglobin	Platelets	Total counts	Urea	S.creatinine	Uric Acid	Direct bilirubin	Total protein	S.Albumin	SGOT	SGPT	LDH	ALP	D- Dimer	APTT	PT	Prolonged PT	INR	S.fibrinogen	ISTH Standard DIC Score	Pregnancy Modified DIC Score	Bedside COT	Blood Transfusion	Whole blood	Packed cells	RDP 's	SDP's	FFP's	Cryoprecipitate	Pre -operative	Intra- operative / partum	Post operative	Any Reaction to Transfusion	Surgical interventions	Mode of delivery	Morbidities arised or associated	Length of ICU Stay	Condition on discharge	Condition after 3 months of Delivery	Discharge Against Medical Advice	Etiology For DIC	Antepartum DIC /Postpartum DIC	OUTCOME
5561484	28	UR	Upper Middle class	1	0	Primigravida	Diarrhoea with jaundice & pallor	Primigravida with 34 weeks POG with Cephalic Presentation with AFLP with DIC with Sepsis		24hours	12.3	2E+05	25,400	13	1.39	6.8	9	5.8	2.9	93	175	443	426	>5000	1.61	16.2	4.9	1.42	82	5	50	6 mins		0	0	0	0	15	0	10 FFP	4FFP	1FFP	Pulmonary Edema	Emergency LSCS	LSCS	AFLP	4 days	Stable	Stable	NO	AFLP	Antepartum	Recovered
5564951	28	UR	Upper Middle Class	2	1	P2L2	Traumatic PPH	P2L2 with PND 1 with Traumatic PPH with DIC with SHOCK with ARDS		12 hours	4.4	77	32,500	34	1.42	5.5	7.8	3.9	1.8	79	115	358	408	>5000	2.45	42.3	31	3.65	81	7	52	10 mins		0	1	0	0	6	4	0	0	1 PCV , 4 Cryo , 6 FFP	Nil	Haemostatic Sutures in Episiotomy	Vaginal	SHOCK	12 hours	Death	Death	NO	Traumatic PPH	Postpartum	Mortality
994113	28	UR	Upper Middle Class	3	2	P3L3	Pulmonary Thromboembolism with secondary PPH	Secondary PPH with Pulmonary Thromboembolism & DVT with Post Emergency Hysterectomy with DIC with Hypoxic Ischemia Brain Injury		6 hours	7	74,000	10,000	74	0.75	4.5	0.31	4.6	2.6	333	158	986	73	4245	4.66	27.5	16.2	2.46	94	7	52	6 mins 55 sec		5	15	21	0	15	6	5 WB , 1 Pcv	7 PCV , 9 FFP, 5 RDP	5 PCV , 12 RDP,,2 FFP , 2 cryo	Fever to 11th PCV on POD-0	Emergency Hysterectomy	LSCS	Pulmonary Thromboembolism & DVT	32 days	Death	Death	NO	Secondary PPH	Postpartum	Mortality
5615113	30	UR	Lower Middle class	2	1	P2L2	C/o Pain Abdomen	Ectopic pregnancy with Pulmonary Thromboembolism		4 hours	11.3	1E+05	7350	20	0.72	6.9	0.87	6.1	3.4	31	16	552	61	>5000	0.81	1.15	1.7	1.14	<50	3	52	4 mins		0	0	0	0	0	0	0	0	0	nil	Laparotomy with left salpingectomy	Ectopic - Laparotomy	Pulmonary Thromboembolism	5 days	Stable	Stable	NO	Ectopic Pregnancy	Antepartum	Recovered
1001534	20	UR	LowerMiddle class	2	1	P2L1D1	Post emergency LSCS with DIC	P2L1D1 with Preterm Emergency LSCS done on 14/02/2020 in DIC with severe Aneamia (f- Abruption with IUD)		12 hours	8.3	69,000	21,800	62	2.59	7.1	0.33	4.8	2.6	190	71	656	61	>5000	0.95	15.5	3.9	1.34	<50	6	52	5 mins 40 sec		1	2	1	0	5	9	0	0	2 pcv , 1 RDP, 5 FFP, 9Cryo ,1 WB	ARDS	Nil	LSCS	ARDS with AKI	40 days	Stable	Stable	NO	Abruption with IUD	Antepartum	Recovered
1000721	19	UR	Lower middle class	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 with post ASD Closure		6 hours	13.4	2E+05	14450	24	1.1	5.5	6.5	7	3.7	148	151	436	792	>5000	1.69	2.99	23.1	2.93	<50	5	52	8 mins 20 sec		0	2	0	0	12	0	8 FFP	4 FFP , 1 PCV	2 PCV	Nil	Nil	Vaginal delivery	AFLP	5 days	Stable	Stable	NO	AFLP	Antepartum	Recovered
1003854	26	UR	Lower middle class	5	4	G5P4L3D1	C/o PV bleeding	G5P4L3D1 with 37weeks with vertex presentation with Abruptio Placenta with Severe Anaemia in Active Labour		6 hours	5.8	94000	18600	40	1.2	7	0.1	5.4	2.7	44	6	899	703	>5000	1.14	1.43	5.1	1.43	38	5	52	6mins 50 secs		0	5	2	0	4	0	1 PCV ,2RDP	1 PCV , 4 FFP	3 PCV, 2 FFP	Nil	Nil	Preterm Delivery	ASD	3 days	Stable	Stable	NO	Abruption	Antepartum	Recovered
1002241	22	UR	Lower middle class	2	1	G2P1L1	False labor pains	G2P1L1 with 40weeks 2 days POG with cephalic presentation with fetal VSD with TGA with False labour pains		4 hours	5.2	62,000	9,400	115	4.41	10.6	0.37	4.1	16	407	143	3479	45	>5000	1.36	18.6	7	1.6	56	7	52	8 mins		0	5	10	0	4	5	0	2 Pcv4 RDP,4 FFP,5 cryo	3 PCV, 6 RDP	Pulmonary Edema	Nil	Vaginal delivery	Pleural Effusion ,AKI secondary to ischemic & Toxic Nephropathy	2 days	Death	Death	NO	Atonic PPH	Postpartum	Mortality
5661529	23	UR	Upper Middle Class	2	1	G2P1L1E1	Ruptured Ectopic with Haemorrhagic Shock	G1P1L1 with Ruptured Ectopic with Haemorrhagic Shock		4 hours	2.6	110	10.7	12	0.54	Not sen	0.02	1.8	1	6	3	Not sen	33	243	1.39	34.4	22.8	2.9	46	5	52	6 min 40 sec		1	3	2	0	2	4	2 PCV	1 WB, 2 RDPS,2 FFP, 4 Cryo	1 pcv	Nil	Emergency Laparotomy with Right Salpingectomy under GA	Ectopic - Laparotomy	Nil	3 days	Stable	Stable	NO	Ruptured Ectopic	Antepartum	Recovered
5669725	19	UR	Lower middle class	1	0	Primigravida	C/o Blurring of vision & Non Reactive NST	Primigravida with 36 weeks POG with Cephalic Presentation with Severe PE with HELLP in DIC with Fetal Distress with RH negative pregnancy with Hypothyroidism		6 hours	6	35,000	23,200	60	2.45	7.7	11.67	4.3	2	202	49	2018	175	>5000	1.02	1.4	4.6	1.39	94	6	51	7 mins 40 secs		0	1	7	0	6	0	4RDP, 4 FFP	3 RDP,1 FFP	1 FFP, 1 PCV	Nil	Preterm Emergency LSCS With Hayman's sutures with B/L Uterine Artery Ligation	LSCS	Retinal detachment , Sub conjuctival Haemorrhage , AKI	9 days	Stable(corrected AKIwith dialysis) but drain insitu ,AMA on POD 9	Stable	YES	Severe Pre eclampsia with HELLP	Postpartum	Recovered
1009724	23	R	lower Middle class	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		6 hours	10.5	97,000	19,000	15	0.5	4.6	0.3	6.2	3.4	31	12	456	162	>5000	0.96	12.7	1.1	1.09	95	5	39	5mins 30secs		0	0	0	0	8	5	5 Cryo	0	8 FFP	Nil	Full Term Emergency	LSCS	Severe PE with DIC	3 days	Stable	Stable	NO	Severe PE	Antepartum	Recovered
5685790	30	UR	Lower middle class	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		4 hours	1.7	25,000	7400	65	1.86	9.3	0.17	4.9	2.7	35	22	729	130	>5000	0.97	12.2	0.8	1.05	57	6	31	6 Mins 40 secs		1	5	1	0	6	6	0	1WB,2PCV,6FFP,6cryo,1 RDP	3 PCV	Nil	Preterm Emergency LSCS	LSCS	DIC with SHOCK	3 days	Stable	Stable	YES	Abruption with IUD	Antepartum	Recovered
5696770	21	UR	Lower Middle class	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		6 hours	1	24,000	8500	Not sen	Not sen	Not sen	Not Sen	Not sen	Not sen	Not sen	Not sen	Not sen	Not se	>5000	5.7	>120	108	>10	<50	8	51	12 mins		0	1	0	0	0	0	0	0	1 pcv	Nil		Vaginal delivery	Irreversiblr Shock	4 hours	Death	Death	NO	Post Partum Haemorrhage	Postpartum	Mortality
10111634	24	UR	Upper Middle Class	1	₹0	Primigravida	C/o Jaundice , Pruritis	Primigravida with 38weeks 3 days POG with AFLP with Moderate Anaemia		12 hours	10.4	210	19700	13	1.04	7.4	13	5.8	3	155	140	413	612	>5000	>180	>120	>5000	>10	<50	5	50	6 mins 20 sec		0	2	0	0	10	10	10 FFP, 10Cryo, 2 PCV	0	0	Nil	Full Term Emergency LSCS	LSCS	Nil	3 days	Stable	Stable	NO	AFLP	Antepartum	Recovered
5814261	34	UR	Lower Middle class	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		6 hours	8.2	48,000	23,300	24	1.29	4.2	0.4	4.2	2.7	32	10	716	92	>5000	0.96	13.5	2.6	1.16	67	6	51	6 mins 15 sec		0	3	0	0	0	10	0	0	3 PCV, 10 cryo	Nil	Vaginal delivery	Vaginal delivery	Hypovolemic shock & DIC	4 days	Stable	Stable	NO	Abruption with IUD	Antepartum	Recovered
5828718	26	UR	Upper Middle Class	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		4 hours	6	54,000	21,100	27	1.08	7.3	0.22	5.8	3	31	12	412	381	>5000	0.93	1.18	3	1.18	97	5	52	3 mins		0	3	4	0	6	0	0	1 PCV,6FFP, 4 RDP	2 PCV	Nil	Nil	Vaginal delivery	Nil	2 days	Stable	Stable	NO	Abruption	Antepartum	Recovered
5834862	23	UR	Lower middle class	3	2	P3L3	P3L3 /FTVD/Female/2.9Kg/8.16 am on 15/09/2020 with Traumatic PPH			10 hours	11.4	69,000	27200	16	0.62	2.7	0.92	4.4	2.3	34	8	804	118	>5000	0.91	1.13	1.5	1.13	116	4	39	5 mins 40secs		0	3	0	0	0	4	0	0	3 PCV, 4 cryo	Nil	Cervical exploration for cervical tear	Vaginal delivery(outside)	Nil	1 day	Stable	Stable	NO	Traumatic PPH	Postpartum	Recovered
5832394	33	UR	Upper Middle Class	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		6 hours	9.6	97000	29000	21	0.9	7	0.48	5.1	2.7	27	13	534	133	5000	1.02	1.58	6.7	1.57	40	6	52	7 mins 10 sec		3	4	4	0	12	10	2 PCV, 6 Cryo , 8 FFP	2 PCV,2 WB,4 RDP,4 FFP	0	Nil	Emergency LSCS with Peripartum Hysterectomy	LSCS	DIC with SHOCK	1 day	Death	Death	NO	Placenta previa	Antepartum	Mortality
5834886	34	UR	Lower middle class	4	2	G4P2L2A1	IUD with Pre eclampsia	G4P2L2A1 with 33 weeks 3 days POG with severe Pre eclampsia with HELLP with AKI		12 hours	4.2	2E+05	49800	36	1.06	6.9	0.12	4.6	2.4	24	9	414	143	>5000	0.84	1.3	3.5	1.3	68	4	52	4 Mins 30sec		0	2	8	0	4	4	1 PCV ,8RDP,4 Cryo	0	1 PCV,4 FFP	Nil	Vaginal delivery	Vaginal delivery	AKI	3 days	Stable	Stable	YES	Severe Pre eclampsia with HELLP	Antepartum	Recovered
5861576	28	UR	Lower Middle class	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		6 hours	4.2	1E+05	27,100	36	1.06	6.5	0.12	4.6	2.4	24	9	414	143	>5000	0.84	1.3	3.5	1.3	68	4	52	6 Mins		0	7	0	0	10	4	1 PCV, 4FFP	2PCV,6FFP,2Cryo	4PCV, 2 cryo	Nil	LSCS	LSCS	DIC	4 days	Stable	Stable	NO	Abruptio Placenta	Antepartum	Recovered
1020561	26	UR	Upper Middle Class	1	0	Primigravida	C/o Pain Abdomen & Fever	Primigravida with 40weeks 2 days POG with Cephalic presentation with Dengue fever in Latent Labour		4 hours	8.1	65000	#####	14	0.57	3.1	0.3	4.5	2.2	54	72	5525	896	2146	5.69	2.12	13	2.1	60	6	52	5 mins 45 secs		0	5	11	2	4	0	1SDP, 1 PCV	4RDPS	7 RDPs, 4 PCV,1SDP ,4 FFP	Nil	Nil	LSCS	Dengue Shock Syndrome	2 days	Death	Death	NO	Dengue with Thrombocytopenia	Postpartum	Mortality
1026095	26	UR	Upper Middle Class	4	3	G4P3L3	IUD with Pre eclampsia	G4P3L3 with 40 weeks POG with vertex presentation with IUD with Sepsis with DIC with Hepatic encephalopathy		6 hours	13.8	1E+05	20,900	45	1.82	9.4	17.13	5.9	2.7	48	67	424	683	6354	1.81	26.6	15	2.27	107	4	52	7 mins 40 secs		0	0	0	0	0	6	6 cryo	0	0	Nil	Nil	Vaginal delivery	Sepsis with DIC with Hepatic encephalopathy	4 days	Stable	Stable	NO	IUD	Antepartum	Recovered
1026296	23	UR	Lower middle class	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		8 hours	4.3	19,000	5,200	29	0.9	7.7	0.6	2.2	1.1	24	10	603	53	2512	1.54	15.4	3.8	1.32	72	6	51	6 mins 10 secs		0	2	8	0	8	8	2 PCV,6 RDP,4FFP, 4 CRYO,	1 RDP	1 RDP,4CRYO, 4 FFP	NIL	Check curretage	Vaginal delivery	DIC with Severe Anaemia	3 days	Stable	Stable	NO	Atonic PPH	Postpartum	Recovered
1026444	20	UR	Lower middle class	1	1	P2L1	Traumatic PPH	P2L1 with VBAC outside delivery on 27/10/21 with HELLP with DIC with AKI		6 hours	9.3	90,000	11,800	34	2.09	6.7	3.98	4.1	2.1	75	11	862	188	>5000	1.4	1.36	4.2	1.36	102	5	52	4 mins		0	0	4	0	4	0	0	0	4FFPS , 4 RDPS	NIL	NIL	Vaginal delivery (VBAC)	DIC with AKI	1 day	Stable	Stable	NO	Traumatic PPH	Postpartum	Recovered
1028003	22	UR	Lower middle class	1	0	A1	Excessive PV bleeding	A1 with Abnormal uterine blleding with severe Anaemia in Early DIC		10 hours	3	9000	8,100	15	0.46	4.5	0.7	2.9	1.9	10	8	138	42	80	0.9	1.66	7.7	1.66	79	5	51	7 mins		1	1	6	0	4	0	1 WB , 1 PCV, 4 FFP,6 RDPS	0	0	Nil	Nil	D&C	Severe anemia & Thrombocytopenia	2 days	Stable	Stable	NO	Retained products of conception	Postpartum	Recovered

1032103	22	UR	Lower middle class	4	2	G4P2L2A1	Loose stools	G4P2L2A1 with 37 weeks 4 days POG with cephalic presentation with Acute fatty liver In pregnancy With DIC		6 hours	12	2E+05	30900	24	1.38	8.1	3.77	5.7	3.2	184	211	359	624	7016	1.28	1.55	6.4	1.54	85	5	52	8 mins 45secs		0	0	0	0	8	6	4 cryo, 4 FFPS	2 cryo, 2 FFP	2 FFP	Nil	Nil	LSCS	AFLP with DIC	3 day	Stable	Stable	NO	AFLP	Antepartum	Recovered
1031922	33	R	Upper Middle Class	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		4 hours	11.8	1E+05	17900	45	0.66	6.2	1.54	4.9	3	81	33	638	49	1288	1.22	1.36	4.2	1.36	154	3	52	3mins 45 secs		1	9	6	1	11	6	2 PCV	6PCV,6RDPS,3FFPs	1WB, 3PCV,8FFP,1SDP,6CRYO	Pulmonary Edema	LSCS	LSCS	Dilated cardiomyopathy with DIC	8 days	Stable	Stable	NO	Placenta previa with increta & percreta	Postpartum	Recovered
1040663	28	R	Lower Middle Class	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		6 hours	10.2	79000	26500	32	0.7	5.2	0.22	6.3	3	753	480	630	165	>5000	1.25	1.48	5.4	1.48	105	5	52	4 mins 20 secs		0	4	4	0	4	0	Nil	2 PCV, 4 RDPs	4PCV, 4 FFP	Nil	LSCS f/b Above Knee amputation	LSCS	Common Femoral Artery Embolism	10 days	Stable	Stable	NO	Placenta previa	Postpartum	Recovered
1039482	23	UR	Upper Middle Class	1	0	Primigravida	PV leak	Primigravida with 39weeks 1 day POG in Latent Labour		8 hours	10.3	2E+05	13900	17	0.76	5.1	0.12	5.1	3	19	9	223	163	>5000	0.65	0.93	0.8	0.93	207	2	32	5 mins		0	4	4	0	4	4	2 PCV	2 PCV, 4RDPs, 4FFPs , 4 Cryo	0	Nil	Peripartum Hysterectomy	Vaginal	DIC	2 days	Stable	Stable	NO	Atonic PPH	Antepartum	Recovered
1042994	19	UR	Lower Middle Class	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		6 hours	8.3	52000	15200	28	0.9	5.2	0.9	5.3	3.2	43	15	697	336	7066	1.11	1.19	2.1	1.19	58	4	52	5 mins 20 secs		0	0	4	1	10	0	4 RDPs , 6 FFPs	0	4 FFPs , 1 SDP	Nil	Nil	Vaginal	Partial HELLP	3 days	Stable	Stable	NO	Abruption with Severe PE	Antepartum	Recovered
1043185	21	UR	Lower Middle Class	1	0	P1L1	AFLP with Hypovolemic Shock	P1L1 Post LSCS WITH AFLP IN DIC IN Hypovolemic Shock With Multi Orgna Dysfuction Syndrome		6 hours	4.9	24000	25000	49	1.5	2.7	3.5	3.9	2	47	18	596	76	947	2.66	2.9	21.6	2.9	54	6	51	4mins 30secs		0	9	17	4	24	2	6 PCV, 13RDPs, 3SDPs,	3 PCV, 1 SDP, 4RDPs, 4 FFPs, 2 Cryo	0	Nil	Exploratory laparotomy & Evacuation of Rectus Sheath Heamatoma	LSCS	DIC with Sepsis with MODS	10 days	Stable	Stable	NO	Rectus sheath Haematoma with AFLP	Postpartum	Recovered
1043401	20	UR	Lower Middle Class	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		6 hours	12.8	31000	11800	22	0.72	6.4	0.76	6.5	3.9	1044	569	1699	254	4112	1	1.39	4.4	1.39	148	5	51	5mins 10 secs		1	1	10	2	2	0	4RDPs, 1 SDPs	2RDPs, 2 FFPs,	1 WB,1PCV, 4 RDPs, 1 SDP	Nil	LSCS	LSCS	DIC with HELLP Syndrome	5 days	Stable	Stable	NO	HELLP Syndrome	Antepartum	Recovered
1043831	26	UR	Lower Middle Class	1	0	P1L1	Atonic PPH	P1L1with Full Term Emergency LSCS (outside) on 11/03/2021/Female/3.5Kg/ with Atonic PPH		8 hours	5.7	83000	29000	38	0.92	6.2	0.54	4.2	2.3	24	8	657	144	>5000	0.7	1.05	0.6	1.05	350	3	13	4mins 10 secs		0	4	2	0	2	0	0	0	4 PCV, 2 FFP, 2 RDP	Nil	Nil	LSCS	Sub segmental Atelectatsis	3 days	Stable	Stable	YES	SEVERE PE	Antepartum	Recovered
1047750	32	R	Lower Middle Class	1	0	Primigravida	Post datism in latent labour	Primigravida with 41weeks 1 day POG with Cephalic presentation with Postdatism in Latent Labour		4 hours	11.9	1E+05	17006	Not sen	Not sent	Not sen	Not Sen	Not sen	Not sent	Not sen	Not sen	Not sen	Not sen	1204	0.77	1.54	6.3	1.54	104	5	52	3 mins 30 secs		0	2	4	0	0	4	0	0	2PCV, 4 Cryo,4RDPs	Nil	Nil	LSCS	Nil	2 days	Stable	Stable	NO	Atonic PPH	Postpartum	Recovered
1048700	24	UR	Lower Middle Class	1	0	Primigravida	PV bleeding	Primigravida with 31weeks 1 Day POG with severe PE with Abruptio Placenta with Intrauterine Fetal Demise		5 hours	8.3	43000	22000	65	3.8	7.1	0.4	5.5	2.9	49	16	600	221	>5000	0.8	1.01	0.1	1.01	199	4	26	4mins 30secs		0	4	8	0	2	0	2PCV,6RDPs,2FFP	1 PCV, 2 RDPs	1 PCV	Rashes, Laryngeal Edema	LSCS	LSCS	AKI with COVID positive	2 days	Stable	Stable	YES	Abruptio Placenta	Antepartum	Recovered
1051537	23	UR	Lower Middle Class	3	2	G3P2L2	No fetal movements felt	G3P2L2 with 35weeks POG with Cephalic Presentation with IUD with Severe Thrombocytopenia		12 hours	10.4	10,000	9900	39	0.88	4.3	0.57	5.5	3.1	19	79	1109	221	1290	1.4	11.5	0.3	1.03	57	5	26	5 mins 40 sec		0	0	0	1	0	6	1 SDP, 6 cryo	0	0	Nil	Nil	Vaginal	Nil	3 days	Stable	Stable	NO	IUD	Antepartum	Recovered
1052643	27	UR	Lower Middle Class	2	1	G2P1L1	PV bleeding	G2P1L1 with 20 weeks 1 day POG with IUD with previous LSCS with PV bleeding with COVID 19 positive		12 hours	11.9	72000	6240	22	0.8	5.3	0.1	6	3.3	35	10	Not sen	94	6874	0.97	11.9	0.7	1.06	105	4	32	4 Mins 30sec		0	0	2	1	6	0	6 FFP, 1 SDP	2RDP	0	Nil	Nil	Vaginal - 2 nd Trimester Abortion	Nil	3 days	Stable	Stable	NO	IUD	Antepartum	Recovered
1052267	24	UR	Upper Middle Class	1	0	Primigravida	Decreased fetal movements with Haematuria	Primigravida with 34 weeks 2 days POG with IUD with cephalic presentation with COVID Positive in DIc in Latent Labour		8 hours	12.6	1,50,000	11,700	12	0.45	Not sen	0.2	6	2.9	33	54	942	250	6893	1.14	12.5	1.3	1.1	134	3	39	5 mins 10 sec		0	0	0	0	8	0	4FFP	4 FFP	0	Nil	LSCS	LSCS	Nil	3 days	Stable	Stable	NO	IUD	Antepartum	Recovered
1052318	30	UR	Upper Middle Class	4	3	G4P3L3	C/o pain Abdomen	G4P3L3 with 34 weeks 6 days POG with MCDA Twins with Threatened Preterm Labour With Anaemia With Thrombocytopenia		12 hours	7.4	33,000	6,600	12	0.74	7.5	0.27	5.5	2.8	77	16	544	201	>5000	1.41	11.5	0.3	1.03	106	5	26	4 mins 20 sec		0	3	4	4	0	4	2 PCV , 4 RDP , 3 SDP , 4 Cryo	1 SDP , 1 PCV	0	Nil	LSCS	LSCS	Nil	6 days	Stable	Stable	No	Severe Anaemia	Antepartum	Recovered
1053935	22	UR	Lower Middle Class	2	0	G2A1	No Fetal movements felt	G2A1with 33 weeks 6 days POG with Cephalic presentation with IUD in DIC		6 hours	12.2	55,000	7500	14	0.6	6.1	0.2	6.4	3.1	54	16	752	117	1378	1.27	11.9	0.7	1.06	78	4	32	4 mins		0	0	6	0	10	2	10FFP, 6 RDP, 2 Cryo	Nil	Nil	Nil	LSCS	LSCS	NIL	3 days	Stable	Stable	NO	IUD	Antepartum	Recovered
1056127	33	UR	Lower Middle Class	4	2	G4P2L2A1	Pain Abdomen	G4P2L2A1with 40weeks 2 days POG with cephalic presentation with post datism for safe confinement		4 hours	14	34,000	10570	36	1.47	8.1	0.9	4.9	2.8	71	20	680	102	5000	1.39	23	12	2.01	<50	7	51	7 mins		0	4	7	0	6	6	Nil	Nil	4PCV,6FFP,7RDP,6cryo	Nil	Cervical Exploration for cervical tear	Vaginal	DIC	3 days	Stable	Stable	NO	Atonic PPH	Postpartum	Recovered
1056153	20	UR	Lower Middle Class	1	1	Primigravida	PV bleeding	Incomplete Abortion		24 hours	8.7	27,000	33,500	87	3.6	7.4	6.1	4.8	2.9	784	372	460	75	>5000	>180	>120	109	>10	71	7	51	8 mins		0	1	4	0	4	0	Nil	Nil	1PCV,4RDP,4FFP	Nil	Nil	Vaginal	Sepsis with MODS	1 day	Death	Death	NO	Retained products of conception	Postpartum	Mortality
1060018	20	UR	Lower Middle Class	1	1	Primigravida	PV bleeding	Primigravida with 35weeks 5 days POG with Cephalic Presentation with Revealed Abruption with IUD		4 hours	5.2	46,000	12,920	16	0.7	6.2	0.2	5.6	3.0	30	13	339	165	>5000	45	21	10	1.90	49	7	51	6 mins 40 sec		0	4	6	0	8	6	2PCV, 8FFP,6RDP,6 Cryo	1 PCV	1PCV	Nil	Vaginal delivery	Vaginal	DIC	2 days	Stable	Stable	NO	Abruption with IUD	Antepartum	Recovered
1064315	27	UR	Lower Middle Class	2	0	G2A1	Pain Abdomen	G2A1 With 39 weeks 1 day POG with cephalic presentation with Chronic RHD , Severe MR,PAH & Moderate MS with Mitral valve Replacement with Clinical IUGR in Latent Labour with deranged Coagulation Profile		6 hours	12.5	2,18,000	14,078	38	0.8	5.2	0.2	6.6	3.0	20	12	302	248	288	90	>120	109	>10	598	4	25	7 mins		0	1	0	0	3	0	2 FFP	1 FFP		NIL	LSCS	LSCS	Nil	3 days	Stable	Stable	NO	Deranged coagulation profile	Antepartum	Recovered
1067596	24	UR	Lower Middle Class	3	2	G3P2L1	Pain Abdomen	G3P2L1 with 34 weeks 6 days POG with Cephalic presentaion with previous LSCS with IUGR with IUD with Severe Pre Eclampsia with HELLP Syndrome		4 hours	11.1	16,000	16,200	26	1.04	6.5	0.5	4.8	2.3	170	211	1895	173	>5000	27	10.6	0.6	0.95	340	6	12	6 mins		0	0	10	1	1	0	6RDP, 1 SDP	NIL	1 FFP, 4 RDP	Nil	LSCS	LSCS	DIC	2 days	Stable	Stable	NO	Abruption with IUD	Antepartum	Recovered
1070729	22	UR	Lower Middle Class	3	1	G3P1A1D0	PV bleeding	G3P1A1D1 with 11 weeks POG with Incomplete Abortion with Previous LSCS		10 hours	6.8	2,06,000	12,100	23	0.69	5.6	0.8	4.7	2.7	22	17	678	33	1500	33.7	16.9	5.7	1.51	111	4	50	5 mins 20 secs		0	3	1	0	2	0	3 PCV,2 FFP,1 RDP	Nil	Nil	Nil	Check currettage	Vaginal	DIC	1 day	Stable	Stable	NO	Retained products of conception	Postpartum	Recovered
1070972	28	UR	Lower Middle Class	3	2	P3L2D1A1	PV bleeding	P3L2D1A1 with post Hysterotomy with Previous 2 LSCS with PPH with Hypovolemic Shock		12 hours	5.5	36,000	14,000	32	0.9	4.6	0.3	3.8	1.6	33	15	480	30	>5000	35	16	5	1.44	127	4	51	5 mins		0	1	2	0	0	0	1PCV, 2 RDP	Nil	Nil	NIL	Laparotomy	LSCS	DIC with Shock	3 days	Stable	Stable	NO	Retained products of conception	Postpartum	Recovered
1070986	22	UR	Lower Middle Class	1	1	P1L1	PV bleeding	P1L1 with Post Partum with Secondary Haemorrhage		10 hours	6.4	1,77,000	29,020	16	0.8	5.6	0.3	4.8	2.4	25	14	734	104	1368	23	12.7	1.4	1.13	194	3	38	4 mins		0	4	0	0	2	0	4 PCV, 2 FFP	Nil	Nil	Nil	NIL	Vaginal	Severe Anaemia	2 days	Stable	Stable	NO	Post Partum Haemorrhage	Postpartum	Recovered
1070290	25	UR	Lower Middle Class	1	1	Primigravida	PV bleeding	P1L1 Post LSCS on 20/9/21 with intraperitoneal bleeding with Sepsis with DIC with MODS		12 hours	5.4	14,000	66,000	203	4.5	8.9	3.5	2.3	2.2	140	163	890	602	>5000	40.6	20.4	9.2	1.82	104	7	51	7 mins 30 secs		0	3	10	0	2	8	2 FFP,2PCV, 8 cryo	1 PCV,6 RDP	4 RDP	Pulmonary Edema	Laparotomy	LSCS	Sepsis with MODS	3 days	Death	Death	NO	Reactionary Haemorrhage - Post Partum	Postpartum	Mortality
1075371	24	UR	Lower Middle Class	1	1	Primigravida	Pain Abdomen	Primigravida with 37 weeks 6 days POG with Severe PE with IUD with Abruption with DIC		8 hours	3.8	28000	16000	44	1.42	10.2	0.88	5.8	3.5	54	25	1105	195	1416	23.6	11.7	0.5	1.04	177	4	32	6 mins 20 secs		1	3	10	1	4	0	10RDP,1 SDP	1WB,1PCV	4FFP 2 PCV	Nil	LSCS	LSCS	Nil	2 days	Stable	Stable	NO	Abruption	Antepartum	Recovered