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Risk Association of the Development of Disseminated Intravascular Coagulation (DIC) in Obstetrical Cases

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Abstract

Background: Disseminated intravascular coagulation (DIC) is a syndrome that can be initiated by a myriad of medical, surgical, and obstetric disorders. Also known as consumptive coagulopathy, DIC is a common contributor to maternal morbidity and mortality and is associated with up to 25% of maternal deaths. **Aim of the study:** To determine the risk factors associated with the development of disseminated intravascular coagulation (DIC) in obstetrical cases. **Material & Methods:** This descriptive type of study was carried out in the Department of Obstetrics and Gynecology of Janaki Medical College Teaching Hospital, Ramdaiya Bhawadi, Janakpurdham, Nepal from January 2016 to December 2021. A total of 500 pregnant women complicated with DIC admitted for termination of pregnancy were included in this study. History and clinical examination were completed. The adverse obstetrical event that causes DIC is identified from clinical diagnosis and relevant investigations. Statistical analysis of the results was obtained by using windows computer software with Statistical Packages for Social Sciences (SPSS-version 26). **Results:** More than half (58.0%) of the patients did not receive any antenatal checkup. Regarding the risk factors for the development of DIC; abruptio placenta was associated with 185(37.0%) cases followed by PPH 147(29.4%) and preeclampsia and eclampsia 119(23.8%). Patients with risk factors of DIC were hypertension 360(72.0%), Antepartum heamorrhage 227(45.4%) and PPH 193(38.6%) these are the most common presenting features. More than two-thirds of the patients (68.0%) had spontaneous vaginal delivery. Almost two-thirds (64.0%) of patients stayed in the hospital for 8-14 days. Maternal death was found in 60(12.0%) cases and perinatal death in 121(24.2%) cases. **Conclusion:** Maternal and perinatal mortality in patients with DIC were 12.0% and 24.0% respectively. The major determinant of survival is prompt identification of the underlying trigger, elimination of the cause and appropriate management.

Keywords:- Disseminated intravascular coagulation, maternal morbidity, maternal mortality, placental abruption, postpartum hemorrhage, preeclampsia, perinatal mortality.

INTRODUCTION

The most important pregnancy-related condition leading to bleeding with high mortality and morbidity rates is disseminated intravascular coagulation (DIC).[1] The real incidence of obstetrical DIC is unknown since it represents a wide spectrum ranging from mild

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to severe. Various studies showed that the incidence of DIC in all pregnancies was 0.02-0.07%.[2] Maternal mortality associated with DIC varies from 6 to 24% and postpartum hysterectomy, massive blood transfusions and acute tubular necrosis are listed as the main maternal morbidity indicators.[3] DIC in obstetrics is a continuous state that is always secondary to an underlying mechanism.[4] This complication can start in its non-overt form and deteriorate if unattended to a life-threatening complication. They also emphasized that specific scores that have high sensitivity and specificity can assist in the diagnosis of non-overt and overt DIC during pregnancy. The incidence of DIC during pregnancy is not well defined and ranges from 0.03% to 0.35%.[3,5] The leading etiologies include placental abruption, especially when associated with stillbirth in developed countries, and preeclampsia and retained stillbirth in developing countries.[2] There is a considerable controversy between obstetricians and hematologists regarding what should be defined as obstetrical hemorrhage/postpartum hemorrhage with associated coagulation factor consumption, and which of these hemorrhagic complications should be defined as a "true" DIC. In pregnancy, DIC has been described in association with fetal demise; placental abruption; amniotic fluid embolism; hemolysis, elevated liver enzyme, low platelets syndrome; and septic abortion.[6,7] Over activation of the hemostatic system, leading to DIC may occur in two phases, non-overt and overt.[8,9] Non-overt DIC is characterized by a compensated over activation of the hemostatic system without organ failure or thrombosis of small- and medium-sized vessels.[3,9] Such phenomena can be observed in cases of retained stillbirth before the development of DIC, chronic abruption, and retained placenta accreta. During the pregnancy, substantial changes take place in the hemostasis mechanism. A significant rise in the majority of the coagulation factors, and a decrease in the level of natural anticoagulants and fibrinolytic activity are the most important physiological changes notable through pregnancy.[10] These alterations lead to a state of hypercoagulability and an increased risk of thromboembolism. Following the delivery, the period when the placenta is being retrieved has the highest level of thrombotic activity due to the release of thermoplastic substances. Fibrinogen levels double during the pregnancy compared to before the pregnancy. Also, D-Dimer levels rise during the pregnancy.[10,11] In response to the activation of cytokines, the release of procoagulant factors or exposure to pro-coagulant factors, all predisposing factors leading to DIC initiate the activation of the coagulation cascade as part of the systemic inflammatory response.[12] The DIC pathogenesis is a complex mechanism in which the invivo increased thrombin production plays a central role. Increased tissue factor production, anticoagulation system dysfunction, insufficient fibrinolysis and increased anionic phospholipid concentration lead to the development of DIC.[13] Obstetrical hemorrhage is a leading cause of maternal morbidity and mortality, and recent reports suggest that about 50% of these cases are preventable.[14,15] Thus, early identification of patients at risk for obstetrical bleeding that requires blood product transfusion can be a key step in reducing, at least in part, the rate of preventable maternal deaths due to hemorrhage. Finally, DIC is an independent predictor of mortality in obstetrical cases. The

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presence of DIC may increase the risk of death by a factor of 1.5 to 2.0 in various studies. Increased severity of DIC is directly related to increased mortality. Therefore, the present study is aimed to determine the risk factors associated with the development of DIC in obstetrical cases which help the timely intervention of the risk patient and decrease mortality associated with DIC.

MATERIAL AND METHODS

Study Procedure

All pregnant women terminated their pregnancy with a deranged coagulation profile that was studied for identification of risk factors and alteration in any of the following laboratory parameters were included in the study that was complete blood cell count, prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen (mg/dl), and D-dimers (µg/dl). The information of all the laboratory results was obtained from the patient's file. The women who have deliveries complicated with DIC are comprised of the study group. The clinical diagnosis of DIC was based upon severe maternal hemorrhage associated with prolonged PT as well as APTT and low fibrinogen concentrations that required blood product transfusion. The adverse obstetrical event that was Pre-eclampsia, Eclampsia, Sepsis, intrauterine foetal death, Amniotic fluid embolism, Shock, Abortion, Abruptio placenta etc that causes DIC was identified from clinical diagnosis and investigations. Demographics of the affected women were collected, including age, parity, gestational age at delivery, mode of delivery, days in the hospital, and maternal weight.

Operational definition:

DIC: is a clinicopathological syndrome characterized by widespread intravascular fibrin deposition in response to excessive blood protease activity that overcomes the natural anticoagulant mechanism

Abruptio placenta: it is one form of antepartum haemorrhage where bleeding occurs due to premature separation of the normally situated placenta.

Intrauterine death: embraces all fetal deaths weighing 500gm or more occurring both during pregnancy and during labour.

Pre-eclampsia: is a multisystem disorder of unknown etiology characterized by the development of hypertension to the extent of 140/90 mm of Hg or more with proteinuria after 20 weeks in previously normotensive and non-proteinuric patients.

Eclampsia: preeclampsia when complicated with grandmal seizures (generalized tonic-clonic convulsion) and /or coma is called eclampsia.

Septic abortion: any abortion associated with a clinical shade of evidence of infection of the uterus and its contents is called septic abortion.

Statistical Analysis

Statistical analyses of the results were obtained by using window-based computer software devised with Statistical Packages for Social Sciences (SPSS-26.0). Quantitative variables were presented as means ± standard deviations and the quantitative observations were indicated by frequencies, and percentages and presented in tables, figures, and diagrams.

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RESULTS

The mean age was 29.0±4.4 years varying from 20 to 35 years. Nearly a half (49.4%) of the patients belonged to age 26-30 years. Most (73.8%) of the patients came from rural area and 131(26.2%) from urban area. The majority (82.2%) patients were from lower-income were and 89(17.8%) in the middle-income group. Regarding the family history, 67(13.4%) patients had hypertension followed by 53(10.6%) with diabetes and 47(9.4%) with pre-eclampsia and eclampsia. Almost a half (43.0%) of the patients belonged to gestational age more than 29 weeks of gestation followed by 170(34.0%) who belonged to 12-28-weeks, 70(14.0%) in the postpartum period and 45(9.0%) in under 12 weeks of gestation. The majority (85.8%) of patients were multipara and 71(14.2%) were nullipara. More than half (51.0%) of patients were 3rd or more gravida followed by 205(41.0%) in 2nd gravida and 40(8.0%) were primi gravida. About the antenatal checkup 12.0% of patients received it regularly, 30.0% received it irregularly and 58.0% didn't receive any antenatal checkup during their current pregnancy. Regarding the obstetrics complications, it was observed that more than one third (34.0%) of patients had Abruptio placenta, 135(27.0%) had PPH, 110(22.0%) developed preeclampsia and eclampsia and 40(8.0%) patients presented with septic abortion and others 45(9.0%). Most them the (68.0%) patients had spontaneous vaginal delivery, 30(6.0%) had assisted vaginal delivery, 130(26.0%) underwent caesarian delivery. About the presenting features, almost three fourth (72.0%) patients had hypertension, 227(45.4%) presented with antepartum hemorrhage, 193(38.6%) had postpartum hemorrhage, 141(28.2%) had haematuria, 93(18.6%) with headache, blurred vision, 47(9.4%) with convulsion, 41(8.2%) intrauterine death, 38(7.8%) Oliguria, bleeding from caesarean section wound 33(6.6%), shock 30(5.4%) and Petechiae 17(3.4%). Regarding the duration of hospital stay it was observed that almost two-thirds (63.2%) of patients stayed in hospital for 8-14 days. 113(22.6%) for 1-7 days and 71(14.2%) for more than 14 days.

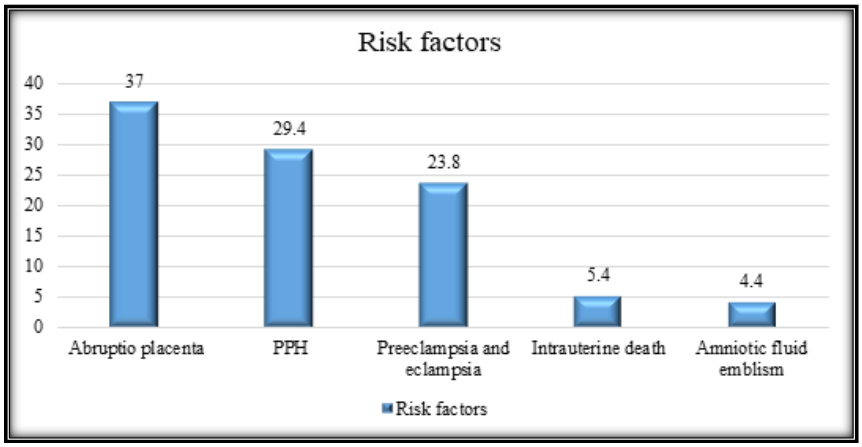

Figure 1: Bar diagram showing risk factors of the study patients.

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Table 1: Distribution of the study patients by the requirement of blood and blood product transfusion (N=500)

Blood and blood product transfusion	Frequency	Percentage
Whole blood transfusion Fresh frozen plasma (FFP) Platelet	61	12.2
Whole blood transfusion Fresh frozen plasma (FFP)	391	78.2
Whole blood transfusion	48	9.6

Table 2: Biochemical variable (coagulation profile) about a risk factor (N=500)

Risk factors	Platelet count (cumm) Mean±SD	Bleeding time (sec.) Mean±SD	Clotting time (sec.) Mean±SD	Prothombin time (sec.) Mean±SD
Abruptio placenta	65249.13±24831.98	12.63±2.06	16.31±2.04	18.56±2.03
Range(min-max)	20000-105000	16-Sep	13-20	15-22
pre-eclampsia & eclampsia	100419.33±26998.11	12.92±2.96	18.99±1.92	23.31±2.93
Range(min-max)	50000-150000	18-Aug	15-22	18-28
Amniotic fluid embolism	78456.12±21118.19	13.69 ±2.30	17.41±1.48	20.92±1.78
Range(min-max)	45000-120000	18-Oct	15-20	18-24
Intrauterine death	96304.59±28613.47	13.56±2.59	19.30±3.09	19.59±2.47
Range(min-max)	50000-140000	18-Sep	14-25	16-24
Postpartum hemorrhage	155653.34±52971.09	13.87±2.39	19.32±2.93	19.94±1.22
Range(min-max)	50000-250000	18-Oct	14-25	18-22

Table 3: Biochemical variable (coagulation profile) about risk factors (N=500)

Risk factors	APTT (sec.) Mean±SD	FDP (µg/dl) Mean±SD	D-dimer (µg/dl) Mean±SD	Serum fibrinogen (mg/dl) Mean±SD
Abruptio placenta	90.97±32.79	194.65±110.23	565.04±205.58	193.56±59.00
Range(min-max)	30-50	18-410	200-900	95-300
pre-eclampsia & eclampsia	90.97±32.79	247.45±97.18	394.74±118.49	206.23±55.79
Range(min-max)	30-50	80-410	200-600	110-300
Amniotic fluid embolism	34.52±5.39	221.34±80.71	519.38±171.03	209.83±61.59
Range(min-max)	25-45	100-350	230-800	100-320

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Intrauterine death	90.97±32.79	114.10±17.53	154.10±50.57	210.58±69.53
Range(min-max)	30-50	80-140	170-350	100-325
Postpartum hemorrhage	34.83±6.26	221.34±80.71	410.18±166.60	276.31±41.72
Range(min-max)	25-45	100-350	100-700	200-350

Table 4: Biochemical variable (for hepatic and renal function) about a risk factor (N=500)

Risk factors	**Serum bilirubin (mg/dl) Mean±SD**	**SGPT (mg/dl) Mean±SD**	**Serum creatinine (mg/dl) Mean±SD**
Abruptio placenta	2.28±0.46	113.96±15.22	2.72±1.06
Range(min-max)	1.5-3	87-140	1.09-4.4
pre-eclampsia & eclampsia	3.39±0.83	126.11±41.10	2.35 ±0.68
Range(min-max)	2.00-5.00	50-200	1.09-3.5
Amniotic fluid embolism	3.35±1.01	100.09±29.15	3.61±1.32
Range(min-max)	1.5-5.0	50-150	1.0-6.0
Intrauterine death	1.92±0.59	110.92±18.19	1.01±0.29
Range(min-max)	3-Jan	80-140	.5-1.5
Postpartum hemorrhage	1.92±0.59	70.20±16.36	1.01±0.29
Range(min-max)	3-Jan	40-100	.5-1.5

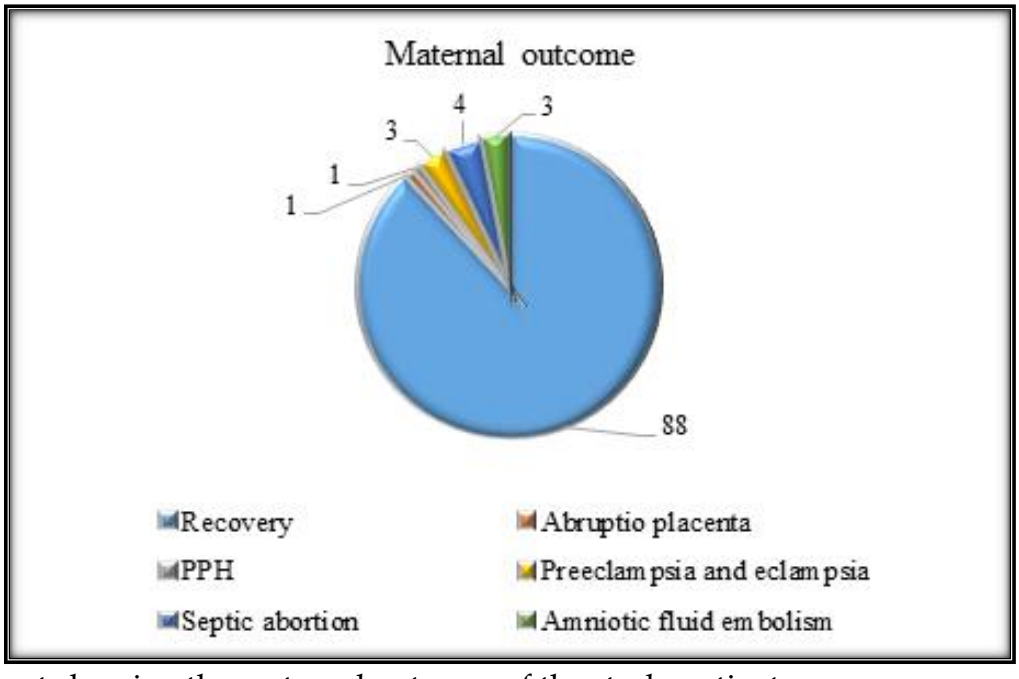

Figure 2: Pie chart showing the maternal outcome of the study patients.

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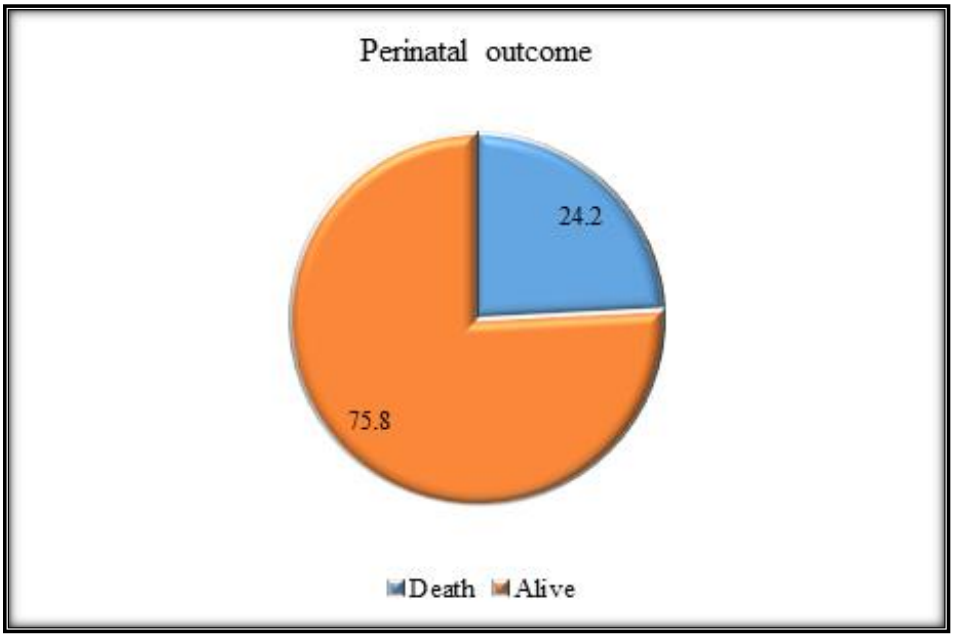

Figure 3: Bar diagram showing the perinatal outcome of the study patients.

DISCUSSION

Disseminated Intravascular Coagulation (DIC) is an acute emergency characterized by inappropriate activation of coagulation and fibrinolytic system and manifested by severe bleeding. DIC is always a secondary phenomenon and is often encountered in obstetric practice. Common conditions predisposing to DIC include abruptio placentae, amniotic fluid embolism, sepsis, severe pre-eclampsia and eclampsia. Diagnosis is often made from clinical manifestation and estimation of coagulation profile though the histological diagnosis of fibrin deposits is the definitive feature of DIC.[16] The significant increase in hemorrhage with age above 25 years emphasizes the importance of not deferring pregnancy to an older age.[17] This high incidence attributed to age may be due to increased parity, placenta previa, abruption placenta, uterine atony and increased incidence of cesarean section. Ganatra et al.[16] studies found that the majority of the patients fell in the age group of 20-29 years and the mean age was 25.01 years, which is similar to the present study. Almost similar maternal mean age was observed by Rattary et al.[3] where they found the average age of their study patients was 28.1 years. According to Al-Zirqi et al.[18] family history of hypertension was 20.0%, diabetes 15.0% which is comparable with the present study. Bangash et al.[19] studies found the gestational age of the patients with DIC had mostly at term followed by postnatal patients. Ganatra et al.[16] studies found the mean gestational age was 33.07 weeks and the median gestational age was 34.41 weeks. In another study, Rattray et al.[3] study observed the average gestational age at delivery was 35.5 weeks. A large proportion of the patients (85.8%) were multipara, 3rd or more gravida (51.0%) which was comparable to Al-Zirqi et al.[18] It was seen that occurrence of postpartum

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hemorrhage increased with increasing parity and gravida. This was comparable with the other study by Limaye et al.[20] The antenatal care status is an important contributing factor for DIC. As 58.0% of the patients did not receive any antenatal check-ups during their current pregnancy. Khatri et al,[21] observe 71.0% of women with no antenatal visit, which reflects the very poor standard of obstetric care and referral of all complicated cases in the tertiary level hospitals all over Nepal. The most common incidence of DIC in the present study was abruptio placenta which is in occurrence with the previous reports.[22] Abruptio placenta rarely produces severe maternal complications while the fetus is alive in utero. The event of fetal death indicates a severe form of abruptio placentae and the risk of maternal death from an overt coagulopathy was also observed in the present study. Abruptio placenta in all of the presented cases was severe enough to cause DIC. Haemorrhagic complication leading to hypovolemic shock was the cause of renal insufficiency. Regarding the mode of delivery Ganatra et al.[16] studies observed 100 cases, out of which 72.0% delivered vaginally, 1 had an instrumental vaginal delivery and 26.0% had to undergo a caesarean section. Regarding the presenting problem, Cavkaytar et al.[23] showed that 58.7% of patients presented with hypertension, and antepartum haemorrhage in 40.2% of patients which supports the present study. Regarding blood and blood product transfusion platelet suspensions are administered to patients with platelet count < 50×109 and actively bleeding. So platelet concentrate is less frequently required which was only 12.2% in the present study. More required fresh blood and FFP as prolonged PT and APTT about 80.0% which compares with Preston FF et al in which about 78.0%. Thrombocytopenia is the most common hemostatic abnormality. Physiological thrombocytopenia associated with pregnancy should be considered while diagnosing DIC. In serial analyses, the decline in platelet counts provides information about the increase in thrombin formation and the associated development of DIC.[24] In a study reported by Spero et al.25 thrombocytopenia was noted in 98% of patients, while severe thrombocytopenia (<50 x 109/l) was noted in 50% of patients. In another study, it was postulated that low platelet counts could be an indicator of increased thrombin production.[24] Thrombocyte aggregation related to thrombin is the main reason for thrombocyte consumption.[26] Normal bleeding time is 3 to 10 minutes (ivy method). Prolonged bleeding time was found in patients with thrombocytopenia and progressive prolongation with platelet count <80x109/1 (R.J Macdoncyh). Bleeding time is significantly increased in abruptio placenta and amniotic fluid embolism. This study is supported by Takenaka et al.[27] Clotting time is prolonged in coagulation factor deficiency. Normal clotting time is 5 to 11 minutes. In Khooharo et al.[28] clotting time was raised in 100.0% of patients which is consistent with the present study. Normal prothrombin time is 16 to 18 sec. Prolonged prothrombin time indicates extrinsic pathway defect mainly factor VII. Prothrombin time increased in DIC. During pregnancy even if consumption of coagulation factors associated with DIC leads to pronglongation of PT time, they may still be within normal limit. For this reason, serial measurements are vital for the diagnosis of evolving DIC. In this study average, PT time is 18 to 24 sec.0. supported by Sahin et al.[29]

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Normal APTT is 30 to 50 sec. In pregnancy, APTT levels shorten based on the increase in coagulation factors. During pregnancy even if consumption of coagulation factors associated with DIC leads to pronglongation of APTT time, they may still be within normal limit. For this reason, serial measurements are vital for the diagnosis of evolving DIC. In this study average APTT time was 30 to 50 sec. supported by Sahin et al.[29] FDP also known as fibrin split product are components of the blood produced by blood clot degeneration. Normal FDP level (<10 µg/dl). FDP is extremely sensitive but non-specific. It informs the amount of plasmin that formed in our body cleaved soluble fibrinogen, fibrin and soluble cross-linked fibrin (other conditions trauma, inflammation, recent surgery increased FDP). The level of FDP was raised in all cases of DIC. This study is similar to Bonnar et al.[30] D-diamer is a fibrin degradation product (FDP), a small protein fragment present in the blood after a blood clot is degraded by fibrinolysis. It is so named because it contains two D-fragments of the fibrin protein joined by a cross-linked. It is less sensitive and highly specific. It measures prior thrombin and plasmin formation. Normal D-diamer level <200 µg/dl. Since D-diamer levels are already elevated in pregnancy, only a significant rise in serial measurement may aid in the diagnosis of DIC. D-diamer level raised above normal in the 2nd trimester of pregnancy. In abnormal pregnancy and other obstetrical conditions, the D-diamer level is elevated in all conditions. This study supported by Bonnar et al.[30] Fibrinogen (Factor-I) is a glycoprotein that circulates in the blood of vertebrates. During tissue and vascular injury, it is converted enzymatically by thrombin to fibrin and subsequently a fibrin-based blood clot. Normal range of fibrinogen is 150 to 400 mg/dl. As fibrin activation is a central component of DIC, it would seem logical to assume that if soluble fibrin is elevated, the diagnosis of DIC can be made with confidence. However, soluble fibrin is not available to most clinicians within a relevant time frame. Laboratory assays aimed at differentiating between cross-linked fibrin, fibrinogen and soluble fibrin have been developed, but are not routinely available to clinicians. The massive fibrin deposition in DIC suggested that the fibrinogen level would be decreased. Accordingly, the measurement of fibrinogen has been widely advocated as a useful tool for the diagnosis of DIC. But it is not always helpful. Fibrinogen as a positive acute-phase reactant is increased in inflammation whereas values may decrease as the illness progress, but they are rarely low. One study demonstrated that in up to 50.0% of DIC patients the level of fibrinogen may remain within normal limit.[31] In this study fibrinogen levels are in the normal range in PPH (200-350 mg/dl). So sequential measurement of fibrinogen is more useful and might be more likely to provide diagnostic clues. Gillisen et al.[32] showed that where fibrinogen levels <150 mg/dl due to severe postpartum haemorrhage the maternal mortality and morbidity increase up to 75.0%. Considering the double fold increase in the baseline fibrinogen level during pregnancy, it is not surprising to encounter fibrinogen levels which are within the normal limits in patients with a suspicion of DIC. Hepatic dysfunction in 19.0% of patients complicated the prolonged PT, APTT.[33] Rathi et al.[34] supported 52.3% of cases with liver dysfunction due to preeclampsia, abruptio placenta, PPH, and amniotic fluid embolism. Average serum bilirubin is 2 to 5 mg/dl which

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is comparable with the present study. Pregnancy-specific disorders are the leading cause of abnormal liver function tests during the pregnant state, particularly in the third trimester. The pre-eclampsia-related disorder is the commonest among these. The average range of derangement of various liver function tests in pregnancy-associated liver dysfunction is as follows: bilirubin <5 mg %, transaminases over one and a half times above normal. Aminotransferase elevations are the hallmark of abnormal liver function. AST elevation in pre-eclampsia-related liver function which similar to the present study.[33] Serum creatinine levels raised in 15.0% of patients in abnormal pregnancy.[35] Acute renal failure is a rare life-threatening complication of severe pre-eclampsia and HELLP syndrome. Obstetric complications are the most common cause of (50.0%-70.0%) renal failure. The incidence of AKI has sharply elevated from 0.5/1000 pregnancies to 1-20000 in developed countries. On other hand, in developing countries pregnancy is still responsible for 15.0%-20.0% of AKI.[36] Mostly due to late referral of pregnancy-related complications. Pre-eclampsia, abruption placenta is the cause in the 2nd half of pregnancy, PPH in the after delivery. ARF in pregnancy is associated with a high risk of maternal mortality (9.0-55.0%). DIC is associated with AKI due to PPH, pre-eclampsia and eclampsia about 18.5% of obstetrical conditions. The average serum creatinine level is 3.7mg/dl which is comparable with the present study.[37] In this current study, it was observed that almost two-thirds 64.0% of patients had hospital stays 8-14 days, 22.0% 1- 7 days and 14.0% more than 14 days. Regarding the duration of hospital stay Ganatra et al.[16] studies found the mean hospital stay was 8.2 days and it is 10.9 days in patients with DIC. In another study, Rattray et al.[3] found the average stay in hospital was 12.2 days. DIC is an independent predictor of mortality in patients with sepsis and severe trauma (Sawamura et al. 2009; Sivula et al. 2009; Duchesne et al. 2009).[35,38,39] Siegal et al.[40] studies showed that septic patients with DIC had higher mortality than trauma patients with DIC did 34.7% and 10.5% which supports the current study. Obstetrical DIC is an uncommon condition associated with high maternal and perinatal morbidity and mortality. Rattray et al.[3] study enrolled the perinatal outcomes included stillbirth 25.0%, neonatal death 5.0%. A study suggested a higher rate of adverse perinatal outcomes (APGAR scores < 7 at 5 minutes, intrauterine growth restriction, and stillbirth) in infants born to mothers with thrombocytopenia secondary to DIC.[41]

CONCLUSIONS

Obstetrical DIC is an uncommon condition associated with high maternal and perinatal morbidity and mortality. The majority of the patients didn't receive any antenatal checkups during their current pregnancy and Spontaneous vaginal delivery was predominant. Hypertension, Antepartum hge, Postpartum hge and Haematuria were the more common presenting problems of disseminated intravascular coagulation and most of the patients received a blood transfusion and hospital stay for more than 7 days. Maternal and perinatal mortality was found at 12.0% and 24.0% respectively. Abruptio placental, PPH and Preeclampsia were the more common causes. Removal of the triggering mechanism and supportive treatment is the key to the successful management of DIC. In obstetrical

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patients, the outcome depends primarily on the ability to deal with the trigger and not on direct attempts to correct the coagulation deficit. This study has some limitations including only one centre study as well as the sample was taken by purposive method, so there was a chance of personal biases. Further studies can be undertaken by including a large number of patients.

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