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Case Report

Anaesthetic Management of Disseminated Intravascular Coagulation (DIC) in Pregnancy at a Tertiary Care Hospital

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ABSTRACT

Abruptio placenta can lead to rapid separation of the placenta and result in fetal demise. Secondary to abruption, Disseminated Intravascular Coagulation (DIC) can occur due to inappropriate activation of the coagulation and fibrinolytic system. Once DIC develops, early recognition, specific treatment and repeated tests with aggressive correction of coagulation and electrolyte imbalance along with avoidance of hypothermia and metabolic acidosis help in preventing multi organ failure and mortality.

Keywords: DIC, Abruptio placenta, Coagulation system, Rescue management, Anaesthesia care

INTRODUCTION

The Scientific and Standardization Committee (SSC) on Disseminated Intravascular Coagulation (DIC) of the International Society on Thrombosis and Haemostasis (ISTH) defined DIC as an acquired syndrome that is characterized by the intravascular activation of coagulation with a loss of localisation arising from different causes. It can originate from and cause damage to the microvasculature, which if sufficiently severe, can produce organ dysfunction.[1] The incidence of coagulopathy in Intrauterine fetal demise (IUFD) is higher and may present in ≥10% cases. DIC is always secondary to an underlying disorder. Indeed, it is associated with pregnancy complications such as placental abruption, haemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome, pre-eclampsia, retained stillbirth, sepsis, post-partum hemorrhage (PPH), acute fatty liver, and amniotic fluid embolism. The clinical presentation of DIC can be either thrombosis and/or bleeding as it is a thrombo-hemorrhagic disorder.[2] Beyond the clinical presentations, the main problem with DIC diagnosis lies in relation to coagulation test abnormalities. It is widely believed that in DIC, patients will have thrombocytopenia, prolonged prothrombin time (PT) and partial thromboplastin time (PTT), low fibrinogen and raised D-dimers.[3]

CASE HISTORY

A 25 year old female G6P5L5A0 at 35 weeks of gestation presented to the emergency room with chief complain of bleeding per vaginum. Patient was conscious and oriented with a pulse rate of 122 beats/min and a blood pressure of 102/60 mmHg. On examination, fetal heart sounds were absent. Ultrasound sonography (USG) examination confirmed fetal demise. Patient was planned for emergency lower segment caesarian section (LSCS). Point-of-care investigations revealed a haemoglobin (Hb) level of 5.7gm/dl and a platelet count of 1,62,000/Cumm. Samples were sent for blood grouping, cross-matching, PT/INR, APTT and D-dimer. The patient was transferred to the operation

theatre, standard pre-induction monitors and two-wide-bore cannula of 18G IV inserted. Patient's heart rate increased to 146 beats/min and her blood pressure was 100/60 mmHg. Rapid sequence induction was achieved with IV glycopyrrolate 0.2mg, propofol 80mg and succinylcholine 100mg. The patient was intubated with a 6.5 mm cuffed endotracheal tube. Anaesthesia was maintained with sevoflurane and atracurium. The child was delivered and placenta removed. Approx 500-600gm of blood clots were removed. The approximate blood loss was around 1.5L. Laboratory reports showed a PT/INR of 1.1, APTT of 32.3 sec, D-dimer >8000 mg/L. Intraoperatively blood pressure fell down to 80/60 mmHg and pulse rate was 150 beats/min, so ionotropic support was started with Inj. Noradrenaline 4mg/50cc NS at 0.48 mg/hr.

Intraoperatively 2 units of packed cell volume (PCV), 4 units of fresh frozen plasma (FFP), 2 units of whole blood were transfused. Intraoperative total urine output was 50 ml and drain output was 500 ml. The abdominal wound was closed after placing a drain. Patient was transferred to obstetric intensive care unit (ICU), intubated and on shifting patient was on ionotropic support. The complete blood count (CBC) report taken at the end of post operative day 0 showed Hb 4.3 gm/dl, platelet count of 1,18,000/cumm. Drain output at the end of post operative day 0 was 1780ml. CBC report on post operative day 1 was Hb 5.6mg/dl, platelet count of 74,000/cumm. Patient was transfused 7 units of random donor platelet (RDP) and 4 units of FFP. Drain output at the end of post operative day 1 was 1880ml. Abdominal drain output was decreased slowly over next 4 days and was nil by the post operative day 5. Patient was extubated on Day 2 and ionotropic support was tapered slowly and stopped on Day 2. Over the next 5 days patient was transfused with 9 units of RDP, 4 units of PCV, and 4 units of whole blood. CBC report on post operative day 5 showed Hb - 8.9mg/dl, platelets - 1,20,000/cumm. Abdominal drain was removed on post operative day 5 and adequate amount of urine output was also achieved and patient was vitally stable.

Total 40 products in which 6 units of PCV, 12 units of FFP, 16 units of RDP and 6 units of whole blood were transfused. Patient was discharged on the 11th day with Hb 9.3 gm/dL and platelet count of 1,37,000/cumm.

DISCUSSION

Abruptio placenta can lead to rapid separation of the placenta and result in foetal demise.[4,5] Secondary to abruption, DIC can occur due to unseemly activation of the coagulation and fibrinolytic system.[6,7] DIC is a consumption coagulopathy, and a high index of suspicion for deranged coagulopathy has to be kept in mind with pregnant female who came with bleeding per vaginum and on clinical examination diagnosed with IUFD and anti-partum haemorrhage. It can be confirmed by platelet count, PT, APTT, FDP, D-dimer and thromboelastography (TEG). FDP is a sensitive test, whereas the D-dimer is more specific for DIC.[8]

Early diagnosis and aggressive management with crystalloids, Red Cell Counts (RCCs), FFP, RDPs, cryoprecipitate and early surgical intervention under general anaesthesia are vital to avoid multi organ failure.[9] The need for viscoelastic point-of-care testing and the availability of blood products for managing patients with DIC are of paramount importance. TEG helps in the early diagnosis and treatment of coagulopathy as PT/INR and APTT delay resuscitation. Appropriate blood components as per the finding of TEG helps to avoid fluid overload in preeclamptic patients. Inline fluid warmer can be used to avoid hypothermia. The haemolysis of the transfused RBC can cause hyperkalemia, leading to arrythmias and cardiac arrest. Hence, follow up of the potassium and calcium values by blood report is advisable. The response to the therapy is monitored clinically and by repeated tests. If all measures fail, addition of recombinant activated factor 8 in management should be considered.

CONCLUSIONS

The rate of recurrence of abruptio placenta in the upcoming pregnancy is 5.8%, and, the pre-conceptional guidance, early diagnosis and treatment are needed to prevent maternal morbidity and mortality. In spite of, once DIC develops, if recognised early with repeated tests and specific treatment with aggressive correction of coagulation

and electrolyte imbalance along with avoidance of hypothermia and metabolic acidosis help in preventing multi organ failure and mortality.

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