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Case Report

Disseminated intravascular coagulation in pregnancy: a case report

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ABSTRACT

Disseminated intravascular coagulation (DIC) is a life-threatening situation arising from a variety of obstetrical and non-obstetrical causes. Obstetrical causes include postpartum hemorrhage, placental abruption, preeclampsia, HELLP (hemolysis, elevated liver enzymes and low platelets) syndrome, retained stillbirth, septic abortion and intrauterine infection, amniotic fluid embolism and acute fatty liver of pregnancy. Prompt diagnosis and understanding of the underlying mechanism is essential for a favorable outcome. We report a successful pregnancy outcome in a multiparous woman with severe atonic postpartum hemorrhage following normal vaginal delivery managed by obstetric hysterectomy complicated with disseminated intravascular coagulation. Massive blood and blood products transfusion given. Serial monitoring of coagulation profile done- improved on postoperative day 3. Identification of risks and early diagnosis of DIC can prevent maternal morbidity and mortality. Team work and prompt treatment are essential for successful management.

Keywords: DIC, Pregnancy, Maternal mortality

INTRODUCTION

Disseminated intravascular coagulation (DIC) during pregnancy constitutes one of the leading causes of maternal mortality worldwide, rate varies from 0.03 to 0.35%.[1] Disseminated intravascular coagulation is a syndrome involving activation of both coagulation and fibrinolytic systems, often secondary to common obstetric condition.[2] Diagnosis of DIC is in relation to coagulation test abnormalities. During pregnancy, the balance between coagulation and fibrinolysis changes to create a procoagulant state. Factors I, VII, IX and X increase along with plasminogen but plasmin, platelet count declines until after delivery and platelet activation is enhanced and hence D-dimer raised.[3,4] Despite the above causes, consumptive coagulopathy as a sole cause of maternal mortality is uncommon accounting for only 0.2% of maternal deaths in the United States.[5] The British Committee of Standards in Hematology recommendations for maintaining coagulation parameters on massive hemorrhage include: hemoglobin >8 g/dl, platelet count >75×10^9, fibrinogen >1 g/l, prothrombin <1.5 mean control, and activated partial thromboplastin time (aPTT) <1.5 mean control.[6] Due to physiological changes in pregnancy, International Society on Thrombosis and Hemostasis (ISTH) has made changes in the DIC scoring algorithm which uses 3 components – platelet count, fibrinogen and differences in prothrombin time (PT).[7]

CASE REPORT

29 years old G2P1L1 with a gestational diabetes mellitus on oral hypoglycemics and history of VP shunt placement for hydrocephalus was admitted at 37 weeks for induction of labor. Her obstetric history included one prior uneventful full term normal delivery. She was induced with 1 dose of oral PGE1 50 mcg. She delivered a term live female baby of weight 3.254 kg with APGAR scores of 7 and 8 at 1 and 5 minutes respectively. The placenta and

membranes were delivered completely. However, shortly after delivery, she developed profuse vaginal bleeding.

Initial management included uterine massage, administration of oxytocin, methylergometrine, carboprost and misoprostol. Despite these, bleeding persisted. Examination revealed no retained products or genital tract trauma. Manual uterine compression and bilateral uterine artery clamping were attempted.

Due to unremitting hemorrhage and signs of hemodynamic instability, a decision was made to perform an emergency obstetric hysterectomy under general anesthesia in view of severe atonic postpartum hemorrhage. Intraoperatively, she was transfused with 3 units of packed red blood cells (PRBCs) and 4 units fresh frozen plasma (FFP). Postoperatively, she was shifted to MICU and planned for elective mechanical ventilation until hemodynamically stable.

Initial and serial investigations revealed changes of early DIC: hemoglobin dropped from 10.2 g/dl to 6 g/dl, platelet count 1.4 lakhs/cu.mm to 1 lakh/ cu.mm, prothrombin time/international normalized ratio (PTINR) was prolonged 1.9 which then gradually improved to 0.97, aPTT was 49.7 seconds improved to 27.3 seconds, D-dimer 10 µg/ml, serum glutamic-oxaloacetic transaminase (SGOT) – 42 IU/l, serum glutamic-pyruvic transaminase (SGPT) – 22 IU/l, total bilirubin 1.69 mg/dl, and procalcitonin 31 ng/ml.

Laboratory findings confirmed the diagnosis of overt DIC based on the International Society on Thrombosis and Hemostasis (ISTH) criteria: thrombocytopenia (50,000-100,000/cu.mm)- 1point, prolonged INR (>1.5) – 2 points, and D-dimer (elevated) – 3 points. A score ≥5 suggested overt DIC. Fibrinogen is typically decreased in DIC but it can be falsely normal or elevated in pregnancy. Additionally, liver enzymes were elevated suggesting hepatic dysfunction possibly from hypoperfusion or DIC. Markedly elevated procalcitonin indicating possible sepsis/ systemic inflammation (due to DIC trigger).

Clinically, the patient manifested classical signs of DIC including bleeding diathesis, falling hemoglobin and progressive coagulopathy. Prompt recognition and activation of a massive transfusion protocol with correction of INR and platelet deficits resulted in reversal of coagulopathy. A total of 8 pints PRBC, 4 pints platelets, 8 pints FFP and 5 pints cryoprecipitate were transfused.

A multidisciplinary team comprising obstetricians, anesthesiologists, intensivists, and transfusion medicine specialists was actively involved in the patient's care. With prompt intervention, her clinical and hematological parameters gradually stabilized—hemoglobin improved to 8.1 g/dl, platelet count to 1.9 lakh/cu.mm, INR normalized to 0.97, total bilirubin reduced to 0.6 mg/dl, and SGPT to 15 IU/l. She was successfully extubated on postoperative day one and made a steady recovery. The patient was discharged on postoperative day eight in a stable condition with advice for follow-up after two weeks. This case underscores the importance of early recognition, timely surgical intervention, and coordinated multidisciplinary management in improving maternal outcomes in life-threatening obstetric DIC.

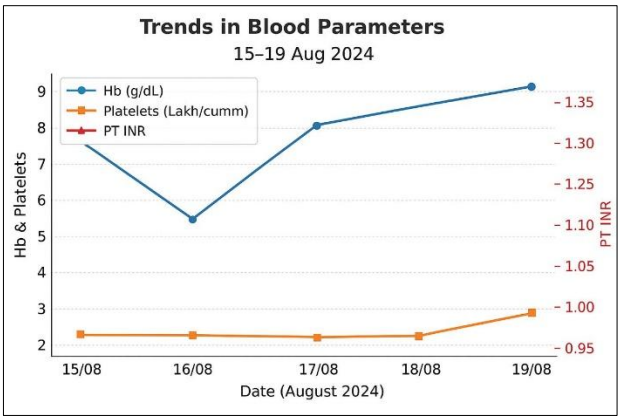

Figure 1: Trends in blood parameters.

DISCUSSION

DIC is a complex, acquired syndrome characterized by systemic activation of coagulation pathways, leading to widespread fibrin deposition in the microvasculature, consumption of clotting factors and platelets, and subsequent bleeding tendencies.

Tissue factor, the main initiator of the extrinsic coagulation pathway, is expressed in high amounts in placental trophoblasts and amniotic fluid. In conditions like placental abruption or amniotic fluid embolism, massive release of tissue factor leads to explosive thrombin generation, fibrin deposition, and subsequent consumption of clotting elements.[8] Concurrently, the fibrinolytic system becomes overwhelmed, contributing to microvascular thrombosis and multiorgan dysfunction.

DIC in pregnancy arises from an imbalance between the coagulation and fibrinolytic systems, often triggered by endothelial damage or release of procoagulant substances such as tissue factor into circulation.[8] Pregnancy itself induces a procoagulant state, with elevated levels of fibrinogen and clotting factors VII, VIII, X, and XII, as well as increased platelet turnover.[9] These changes are meant to prevent hemorrhage during delivery but can accelerate pathological clot formation when an obstetric insult occurs.

DIC may present acutely or insidiously. Clinical features include excessive bleeding from intravenous sites, mucosal hemorrhages, hematuria, petechiae, and purpura. In severe cases, multiorgan dysfunction due to microvascular thrombosis may occur. Laboratory findings typically reveal thrombocytopenia, prolonged PT and

aPTT, elevated D-dimer, low fibrinogen, and schistocytes on peripheral smear.[8]

There is no single definitive test for DIC. Diagnosis relies on clinical suspicion supported by a constellation of laboratory abnormalities. The ISTH developed a scoring system that includes platelet count, fibrin degradation products (e.g., D-dimer), PT prolongation, and fibrinogen level.[7] However, physiological changes in pregnancy necessitate a modified scoring algorithm, as fibrinogen levels are normally elevated in pregnancy and may mask early DIC.[9]

A pregnancy-specific DIC score was developed by Erez et al., incorporating gestation-adjusted laboratory thresholds. This tool demonstrated improved sensitivity and specificity for detecting overt DIC in pregnant women.[10]

Management of obstetric DIC requires prompt identification and treatment of the underlying cause, supportive care, and correction of coagulopathy. Early delivery or surgical intervention may be necessary in cases related to placental abruption, IUFD, or uterine rupture.[1,8]

Massive transfusion protocols are central to management, typically involving PRBC, FFP, platelets, and cryoprecipitate in defined ratios. The British Committee for Standards in Hematology recommends maintaining: hemoglobin >8 g/dl, platelet count >75×10⁹/l, fibrinogen >1.5 g/l, and PT and aPTT <1.5× control.[6]

The use of tranexamic acid has shown benefit in PPH-related coagulopathy, especially when given early, as evidenced in the WOMAN trial.[11] Anticoagulation with heparin is generally reserved for chronic compensated DIC with predominant thrombosis rather than bleeding.

Timely and effective management can reverse DIC and prevent end-organ damage. However, delay in diagnosis or inadequate transfusion support increases the risk of maternal death. Studies report that maternal mortality rates in DIC vary from 10% to 40%, depending on the underlying etiology and healthcare resources.[12] Early warning systems, standardized obstetric emergency protocols, and multidisciplinary team involvement have been shown to improve outcomes in obstetric DIC.[13] Prophylactic strategies include close monitoring of high-risk pregnancies and immediate availability of blood products.[1,14]

Emerging research focuses on identifying early biomarkers of DIC, such as soluble thrombomodulin and tissue factor pathway inhibitor, to allow earlier intervention. Additionally, viscoelastic testing (e.g., thromboelastography) is gaining popularity in obstetric hemorrhage management for real-time coagulation assessment.[15] Artificial intelligence-based predictive models and integration of point-of-care testing may further enhance early detection and treatment efficacy in the near future.

CONCLUSION

This case highlights the critical importance of early recognition and prompt multidisciplinary management of obstetric disseminated intravascular coagulation. Severe atonic postpartum hemorrhage can rapidly progress to life-threatening coagulopathy, requiring timely surgical intervention and massive transfusion support. Close monitoring of coagulation parameters, rapid correction of deficits and coordinated team work between obstetric, anesthesia, intensive care and transfusion services are essential to optimize outcomes. Early diagnosis and intervention remain the cornerstone in reducing maternal morbidity and mortality associated with obstetric DIC.

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