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Post Partum Hemorrhage and A Saga Through Sequentiality

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Abstract

Post partum hemorrhage is an obstetric emergency complicating 1-10% of all deliveries [1]. It continues to be the leading cause (35%) of maternal deaths [2]. PPH is defined as blood loss of 500 ml or more within 24 hours after birth while severe PPH is defined as a blood loss of 1000 ml or more within the time frame by the World Health Organisation. Obstetric hemorrhage and especially DIC is a leading cause for maternal mortality, often secondary to maternal and/or fetal complications including placental abruption, amniotic fluid embolism, HELLP syndrome, retained stillbirth, acute fatty liver of pregnancy and sepsis [3]. Here, we present to you a young P_1L_2 who underwent elective LSCS for DCDA twin/IVF conception and the sequential management.

Introduction

PPH is a complication of delivery and the most common cause of maternal death accounting for 35% of all maternal deaths worldwide [4]. Every year 14 million women suffer from PPH. Primary PPH occurs within first 24 hours after delivery, while Secondary or late PPH occurs 24 hours to 12 weeks postpartum [4].

The maternal mortality ratio in developing countries in 2015 is 239 per 100,000 live births vs 12 per 100,000 live births in developed countries [5]. Thus, 99% of all maternal deaths occur in developing countries. WHO statistics suggest that 25% of maternal deaths are due to PPH; which could kill a healthy women, if left untreated. Shock following severe haemorrhage and DIC are common life threatening complications of PPH.

DIC is characterised by a concomitant over-activation of the coagulation and fibrinolytic systems, leading to widespread microvascular thrombosis, disruption of blood supply to different organs and thereby leading to multi-organ failure. Thus extensive activation of coagulation cascade leads to consumption and depletion of platelets and coagulation protein, which can provoke concurrent severe bleeding.ISTH criteria for DIC takes into account the platelet count, elevated levels of fibrin - related marker, prolonged prothrombin time and the fibrinogen levels. A score of ≥5 is compatible with overt DIC.

Case report

A young lady, para one live two (P_1L_2) who underwent elective LSCS following IVF conception on presented to the emergency department with features of DIC. 7 hours following elective LSCS, she went into shock where USG abdomen revealed hemoperitoneum and Hb drop of 7.9 gm/dl. She was taken up for emergency laparotomy and 1500 ml of collection was drained. 2 pint PRBC was transfused intra operatively. It was followed by visual disturbances and seizures 3 hours post extubation; she was then reintubated and managed with magnesium sulphate, vitamin K and blood products; was initiated on ionotropic support and was shifted to our hospital.

On admission, her level of consciousness was altered but was opening eyes to command and was responding to painful stimulus. She was in a state of shock with tachycardia, feeble pulse and hypotension. Uterus was well contacted and retracted along with soakage of dressing. Diffuse oozing was noted from drain site and nostrils. Investigations revealed Hb: 8 gram/dl, platelet: 25,000, prothrombin time: 66.9 seconds, PT INR 5.24, D dimer > 20,000, elevated fibrinogen: 53.2; a diagnosis of Disseminated Intravascular Coagulation (DIC) was made as per the ISTH scoring system. She developed severe metabolic acidosis (pH 6.903, bicarb 13.8, lactate 8.3). Vasopressors, ionotropes, Anti-fibrinolytic agent and hydrocortisone infusion was started. CT brain revealed no evidence of bleed while CT Abdomen revealed liver and bowel hyperperfusion changes. An MDT was initiated with the hematologist, intensivist, neurologist, gynecologist, nephrologist and infectious disease control. SLED was initiated and multiple blood products and Octoplex injection were given accordingly as per the EXTEM report. She then developed pulmonary edema/ARDS and multiple episodes of hypoglycemia. Sepsis protocol was activated; started on ulinastatin, antibiotics were escalated to meropenem and teicoplanin, and supported with hydrocortisone. Along with the blood products, she received 3 units of Octoplex injection. ABG improved to 7.434/110.5/40.9/26.8 on day 3 following 2 sessions of SLED. She had persistent oozing from the drain site. Peripheral smear revealed schistocytes, platelets: 20,000. She received 34 units of platelets,11 units of packed red cell, 11 units of plasma, 27 units of cryoprecipitate and 3 units of Octoplex. She received 5 sessions of SLED in total. She was shifted out of the ICU on post op day 11 and was discharged on the 20th day.

Discussion

DIC is a dynamic situation that requires a continuous assessment of clinical and laboratory parameters including decreasing concentration of fibrinogen and platelet count, prolongation of prothrombin time and increased concentration of fibrin split products or D-dimers until it resolves. The hallmark of successful management depends on the prompt and accurate recognition of DIC. Bedside tests like bleeding time, coagulation time, clot retraction time, peripheral smear and circulatory fibrinolysis test helps in the diagnosis. Standard laboratory tests have limitations including absence of real time data and incapacity to determine the functionality of the hemostatic system of whole blood. Viscoelastic tests or TG and rotational thromboelastography (ROTEM) are the most widely used viscoelastic tests which provide rapid assessment of in vivo. A shorter R&K, higher alpha angle and maximum amplitude suggest that clot formation is faster and bigger (as in pregnancy; especially in the 3rd trimester). Consumptive Coagulopathy is a serious complication of massive PPH. Management should be concentrated on removing the trigger. It involves maintenance of circulatory blood volume with appropriate fluid replacement. Volume replenishment by massive blood transfusion is the sheet anchor of treatment to replenish fibrinogen and other pro-coagulants. Platelet transfusion is recommended to maintain platelet count above 50,000/cubic mm and cryoprecipitate to be administered if fibrinogen levels fall less than one gm/dl.

OCTAPLEX is a plasma derived, virally inactivated concentrate of clotting factors II, VII, IX and X. It also contains the naturally occurring anticoagulants, Protein C and S.The dosing is adjusted according to the INR value. Use of recombinant activated factor 7 or RF 7A to start with low dose of 40 to 60 microgram/kilogram requires correction of metabolic acidosis, hypothermia,

hypofibrinogenemia and thrombocytopenia. A volume of 500 ml fresh blood raises the fibrinogen level by 12.5 milligram/ml. 1 unit of platelet transfusion raises platelet count by 5000 to 10,000 per cubic millimeter. Cryoprecipitate is rich in fibrinogen and factor VIII, XIII. Tranexamic acid, an anti-fibrinolytic agent prevents the activation of plasminogen by plasmin. Human fibrinogen concentrate have been used for substitution therapy in hypofibrinogenemia. Recombinent Human Soluble Thrombomodulin decreases excessive thrombin activation and regulates the imbalance of coagulation system which improves platelet count, D-dimer, fibrinogen concentrations, prothrombin time and decreases the need for platelet transfusion.

Conclusion

The basic principles of treatment of obstetric DIC include:

a) Identifying & treatment of the underlying condition leading to DIC,
b) Fast and prompt delivery or termination of pregnancy options should be discussed by MDT and consider the fastest mode of delivery to the mother,
c) Treatment with blood product transfusion, surgical care and related matters,
d) Rigorous clinical and laboratory patient surveillance,
e) Prompt involvement of needed consultants such as hematologist, gynecologist, anesthesiologist and others,
f) Shifting the patient to a higher center where blood bank can support massive blood transfusion.

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