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Case Report

Management of Disseminated Intravascular Coagulation Following PPH: A Case Report

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Abstract

Disseminated intravascular coagulation is a life threatening complications of severe post-partum hemorrhage. That is the end point of a pathologically activated cascade leading to excessive consumption of platelets culminating bleeding. It results from washing out of all important procoagulants. A case of post-partum hemorrhage following caesarean section is reported. She was treated by emergency peripartum hysterectomy and aggressive blood and blood component therapy.

Keywords: Disseminated Intravascular Coagulations (DIC), Post-Partum Hemorrhage (PPH), Blood component therapy.

Introduction

Postpartum hemorrhage is a life threating obstretic emergency that is nightmare for an obstetrician during her professional life. It complications 3-6% of all deliveries and accounts for 15-20% of maternal deathin Bangladesh.[1] Disseminated intravascular coagulation (DIC) is a clinicopathological syndrome characterized by the formation of fibrin clots with concomitant consumption of platelets and coagulation factors that leads to organ failure and contributes to a high mortality if left tuntreated.[2] Anemia, malnutrition poorly supervised deliveries, delay in transfer of patient to tertiary care center and lack of adequate blood and blood components therapy contribute to the gross outcome.[3-5]

Although post-partum hemorrhage is anticipated in some high risk cases, it occurs unexpectedly in many others cases.[6] Unpredictability of primary PPH contributes the main hazard of home deliveries. Shock following severe hemorrhage and DIC are common life threating complications of post-partum hemorrhage.[7] Timely replacement of blood and blood products in an aggressive manner can save many young women dying from these serious but preventable complications of pregnancy and delivery.[8]

Case Presentation

30 years old woman with amenorrhea for 37 weeks presented with occasional lower abdominal pain. She hadD her first cesarean section 9 years back. She was under complete antenatal checkup. Now despite optimal medical therapy her pain did not reduce. Then her lower segment caesarean section (LUCS) was done on the next day of her admission at morning. After the operation she was reasonably well. Her uterus was contacted; urine output was good and no excessive per vaginal bleeding.

But after 2:30 hours of operation she developed excessive per vaginal bleeding. Then she was treated as per as treatment protocol of primary post-partum hemorrhage. Ballooncatheter was done. Bleeding was reduced in amount then after 1 hour again bleeding started, her peripartum hysterectomy was done at evening for saving her life. Patient was severely anemic so, combat her loss 3 unit blood was given on that day. She had no history of any bleeding and thrombosis or anticoagulant history. Before peripartum hysterectomy patient was receiving, oxytocin, methylergometrine, maleate, ceftriaxone, misoprostol. An emergent complete blood count was drawn and a significant platelet count drop (85000u/L) was noted then an emergent coagulations studies were taken .There were significant for an elevated international normalized ration (INR) of more than 2.70, clotting time 12 min 5 sec , D dimer 5.677 microgram, APTT 52.1sec . These findings indicated blood loss, fibrinogen and platelet depletion in the widespread thrombotic process and breakdown products of fibrinogen and hemoglobin due to extensive clotting and red blood cell sludging in thrombotic capillaries, respectively. To see end organ damage we performed renal and liver function test here. Creatinine was 1.20 mg/dl and S. ALT/ SGPT was 99.98 U/L.

Supportive therapy with blood products (fresh human blood) to replace lost blood components included 6 units fresh human blood was given. Over the next 24 hour with the above interventions. She improved clinically with stabilization of thehemoglobin and platelet counts. She was discharged from hospital after one week. During follow up she remained clinically stable with no evidence of recurrent haematogical abnormalities, residual end organ damage.

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Discussion

Disseminated intravascular coagulation (DIC) is a syndrome characterized by increased turnover of coagulation factors, platelet destruction, activation of fibrinolytic system, formation of thrombi in the micro circulation and uncontrolled thrombin activity. It is a life threatening complication seen during pregnancy or after delivery.[9] The conditions where DIC may occur are abruptio placenta, thrombocytopenic purpura, jaundice in pregnancy, HELLP (hemolysis elevated liver enzymes low platelet count) syndrome. Intrauterine death (IUD) of fetus, pre eclamsia, eclamsia, septicemia, hypovolemic shock, amniotic fluid embolism, vesicular mole etc.

These conditions trigger delicate hemostatic mehanismeither by endothelial injury or by release of thromboplastin and phospholipids. Because of a hypercoagulable state in pregnancy, prevalence of any provocative factor can easily unset the normal balance, culminating in disseminated intravascular coagulopathy. Following severe post-partum hemorrhage, DIC mayoccourue to diminished pro coagulants or increased fibrinolytic activity. DIC can be diagnosed by clinical signs and by laboratory investigations. Bleeding from venepuncture site, abdominal surgical wound site, gastric hemorrhage, appearance of petechial hemorrhage; raise suspicion of onset of DIC.

This involves maintenance of circulatory blood volume with appropriate fluid replacement. Rapid infusion of fresh frozen plasmas is recommended at 15ml/kg, with massive blood transfusion. One liter of fresh frozen plasma is recommended for 6 units of blood transfused. Platelet transfusion is recommended to maintain platelet count above 50,000/cumm and cryoprecipitate to be administered, if the fibrinogen level falls to less than 1 gm/dl. Recombinant factor VII has shown definitive role in the treatment of severe postpartum hemorrhage with disseminated intravascular coagulation.[10]

It is difficult to assess the particular disorders of coagulations, due to rapid changes in the disorder from one phase to another. Volume replacement by massive whole blood transfusion is the sheet anchor of treatment to replenish fibrinogen and other pro coagulants. A volume of 500 ml of fresh blood raises the fibrinogen level by 12.5mg/100ml. It also adds 10000-15000 platelets to the circulation. Fresh frozen plasma contains fibrinogen and other coagulation factors including V and VIII. It also contains anti thrombin III, which prevents intravascular clotting. Fresh frozen plasma must be ABO compatible but need not be Rh compatible.

Platelet concentrates may be given to the patient if platelet count is below 50,000/cumm. Platelets are administered rapidly over a period of 10 minutes. It should be ABO abd Rh compatible. One unit of platelet transfusionraises the platelet count by 5000 to 10,000/cumm; cryoprecipitates are rich in fibrinogen and factor VIII, XIII. One bag of cryoprecipitate will raise blood fibrinogen level by 5 mg/dl. Obstetric hysterectomy is a lifesaving procedure in intractable atonic postpartum hemorrhage.[11] At time the surgeon is in a dilemma; whether to sacrifice a women's reproductive capacity, especially if she is nulliparous or having less than 2 children. A timely decision to perform hysterectomy can make the difference between life and death of the patient.[12] A quick subtotal hysterectomy usually saves life in conditions of acute blood loss and shock. Training of resident doctors to perform obstetric hysterectomy in an emergency situation is important. Networking of regional blood blanks can help in timely procurement of requisite blood and its components in dire emergencies.

Conclusion

Disseminated intravascular coagulation is frequently found in primary postpartum hemorrhage and regarding this patient her life was saved in cost of hysterectomy. So early diagnosis and prompt action is the key to save life from the complications of postpartum hemorrhage.

Conflict of interest: No

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