International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

Life-Saving Role of Factor VIII in Refractory Obstetric Hemorrhage Associated with Abruptio Placentae and HELLP Syndrome: A Case Report

Dr Swapnil Wasekar, Dr Shrinivas Gadappa, Dr Rupali Gaikwad, Dr Meghna Malu

Abstract: ***This case report describes a 27-year-old woman with previous cesarean delivery who presented with abruptio placentae, intrauterine fetal death, HELLP syndrome, severe anemia, and consumptive coagulopathy. Emergency laparotomy was performed because of maternal deterioration and transverse fetal lie. Persistent hemorrhage continued despite uterotonics, uterine artery ligation, compression sutures, massive transfusion, and intensive supportive care. Following administration of the coagulation Factor VIII was administered intravenously at a dose of 250 IU diluted in 10 ml NS, hemostasis improved substantially and bleeding was controlled. The patient recovered gradually and was discharged in stable condition. This case highlights the potential role of t coagulation factor VIII therapy as rescue treatment in carefully selected patients with refractory obstetric hemorrhage associated with severe placental abruption and HELLP syndrome.***

Keywords: Abruptio placentae, HELLP syndrome, Disseminated intravascular coagulation, Obstetric hemorrhage, Postpartum hemorrhage, Coagulation factor therapy, Maternal morbidity

1. Case Report

A 27-year-old gravida 2 para 1 living 1 (G2P1L1) woman at term gestation with a history of one previous lower segment cesarean section (LSCS) was referred from a district hospital to our tertiary care institute in view of severe anemia and hypertension. The patient had presented with abdominal pain and decreased fetal movements prior to referral.

On admission, the patient's general condition was poor. She was afebrile, markedly pale, and tachycardic with pulse rate of 124/min. Blood pressure was 154/104 mmHg. Cardiovascular examination revealed normal heart sounds, while respiratory examination showed bilateral equal air entry.Per abdominal examination revealed a uterus corresponding to approximately 36 weeks gestation with increased uterine tone and tenderness. The lower uterine pole was empty and fetal heart sounds could not be localized clinically or on Doppler examination. Per vaginal examination showed the cervix to be 1 cm dilated and 10–20% effaced, with membranes present and station –1. Pelvis was found to be adequate.

Ultrasonography confirmed intrauterine fetal death (IUFD) with a large hyperechoic retroplacental clot measuring approximately 6 × 8 cm suggestive of abruptio placentae. The fetus was in transverse lie with the fetal head towards the maternal left side.

A diagnosis of term pregnancy with previous LSCS complicated by abruptio placentae, IUFD, severe preeclampsia with HELLP syndrome, and transverse lie was made.

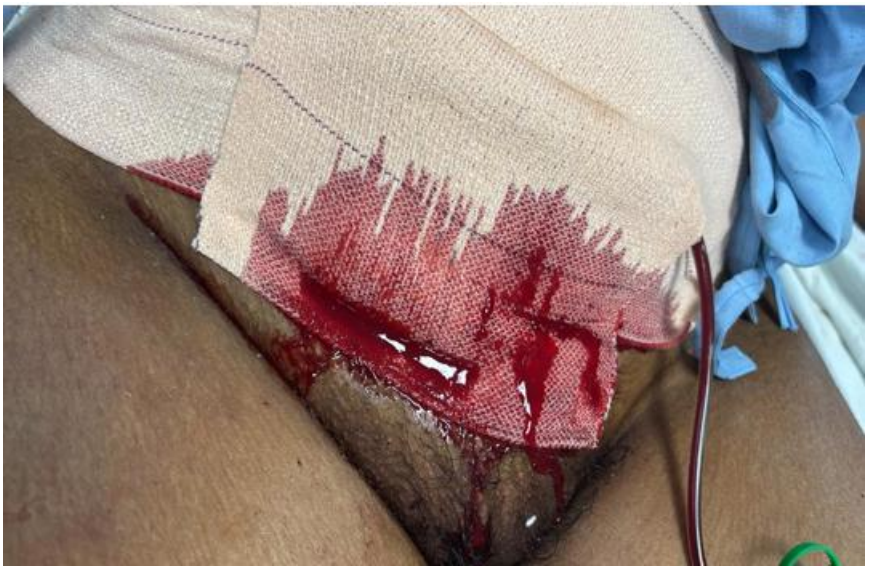
Laboratory investigations revealed severe anemia and consumptive coagulopathy. Hemoglobin was 5 g/dL, total leukocyte count 24,000/mm³, and platelet count 88,000/mm³. Bleeding time was 4 minutes and clotting time was prolonged to 16 minutes. Liver enzymes were elevated with SGOT 188 IU/L and SGPT 129 IU/L. PT was 19.01 seconds with INR 1.43. Renal function tests were within normal limits.

Initial stabilization was started with blood and blood component therapy. Two units packed cell volume (PCV), four units fresh frozen plasma (FFP), and four unit's random donor platelets (RDP) were transfused. In view of worsening maternal condition, previous LSCS, abruptio placentae, and transverse lie, emergency laparotomy was planned for maternal salvage.

Under general anesthesia, the abdomen was opened through a midline vertical incision. A male fetus was delivered in transverse lie along with approximately 600 grams of retroplacental clots, confirming severe placental abruption. Complete postpartum haemorrhage prophylaxis was administered after placental delivery.

Despite aggressive uterotonic therapy, significant bleeding persisted intraoperatively. Bilateral uterine artery ligation was performed followed by Hayman uterine compression sutures to achieve haemostasis. Vaginal examination and swabbing performed intraoperatively showed no active vaginal bleeding. During surgery, the patient received an additional 2 units PCV, 4 units FFP, and 4 units RDP.

Postoperatively, the patient was shifted to Trauma Intensive Care Unit (TICU) for ventilatory support and close monitoring. Although her hemodynamic parameters stabilized initially, abdominal dressings continued to soak with blood, indicating persistent diffuse bleeding from the operative wound and suture sites.

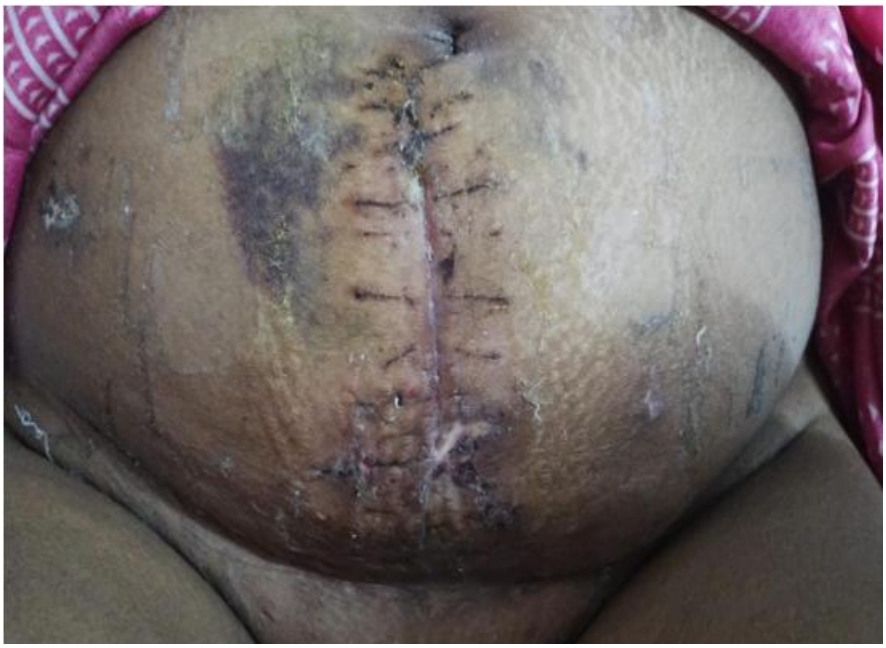


Repeat investigations showed hemoglobin 6 g/dL, TLC 20,000/mm³, and platelet count 52,000/mm³. Additional blood component therapy with 2 units PCV and 4 units FFP was administered. However, bleeding continued due to severe consumptive coagulopathy.

Despite administration of all indicated blood products and corrective measures. Bleeding continued. The coagulation profile of patient BT 4 min CT 12 min PT 17 INR 1.4. comprehensive coagulation factor assays could not be performed. Therefore, In view of refractory haemorrhage, a multidisciplinary decision was taken to administer Factor VIII rescue therapy. Dried Factor VIII Fraction was administered intravenously at a dose of 250 IU diluted in 10 ml NS IV bolus. Three doses were administered at 12-hourly interval

Following administration of Factor VIII bleeding was controlled effectively with rapid improvement in hemostasias. No further active bleeding episodes occurred thereafter. The patient gradually improved clinically with stabilization of coagulation parameters and correction of haematological abnormalities.

Her postoperative recovery was uneventful. Serial investigations demonstrated gradual improvement in haemoglobin, platelet count, liver enzymes, and coagulation profile. Sutures were removed on postoperative day 14, and the patient was discharged in stable condition.



2. Discussion

Abruptio placentae remains one of the most serious obstetric emergencies and is associated with high maternal and perinatal morbidity and mortality. Severe placental abruption can rapidly progress to hypovolemic shock, disseminated intravascular coagulation (DIC), acute renal injury, HELLP syndrome, and massive obstetric hemorrhage. The condition becomes even more catastrophic when associated with intrauterine fetal death, previous cesarean section, severe anemia, and abnormal fetal lie, as seen in the present case.

Hypertensive disorders of pregnancy are among the strongest risk factors for abruptio placentae. Chronic vasospasm and endothelial dysfunction result in placental ischemia and premature placental separation. In our patient, severe preeclampsia complicated by HELLP syndrome significantly contributed to endothelial damage, platelet consumption, and coagulation abnormalities. The large retroplacental clot observed intraoperatively confirmed severe placental abruption with extensive placental separation.

HELLP syndrome itself is a life-threatening obstetric complication characterized by hemolysis, elevated liver enzymes, and low platelet count. When combined with abruptio placentae, it can precipitate profound consumptive coagulopathy. Release of thromboplastin from the damaged placenta activates the coagulation cascade, leading to widespread fibrin deposition and depletion of clotting factors and platelets. As a result, patients may develop diffuse uncontrolled bleeding from surgical sites, venepuncture sites, and raw surfaces despite adequate surgical hemostasis.

Massive obstetric hemorrhage requires prompt multidisciplinary management involving obstetricians, anesthesiologists, intensivists, hematologists, and transfusion medicine specialists. Standard management includes aggressive fluid resuscitation, blood and blood component replacement, uterotonic agents, correction of coagulopathy, and surgical interventions such as uterine artery ligation, compression sutures, devascularization procedures, or hysterectomy when necessary.

In the present case, several conventional methods were employed including uterotonics, bilateral uterine artery ligation, Hayman compression sutures, and massive transfusion protocol. However, persistent diffuse bleeding continued due to severe coagulopathy. At this stage, the patient was at extremely high risk for irreversible shock, multiorgan dysfunction, and possible hysterectomy. Factor VIII has emerged as an important adjunctive therapy in severe refractory obstetric hemorrhage. Originally developed for treatment of haemophilia patients with inhibitors, it acts by activating factor X on the surface of activated platelets, thereby enhancing thrombin generation and promoting formation of a stable fibrin clot at the site of vascular injury. The rationale behind administration of recombinant Factor VIII in this patient was to augment intrinsic pathway coagulation activity and improve thrombin generation at sites of vascular injury. By increasing availability of functional Factor VIII, adequate activation of Factor X and subsequent fibrin clot stabilization could occur, thereby reducing diffuse microvascular bleeding and persistent surgical site oozing. After administration of coagulation Factor VIII, the patient showed dramatic clinical improvement. Diffuse bleeding reduced significantly, dressing soakage decreased markedly, and hemodynamic parameters stabilized. No further active hemorrhage occurred following therapy. The rapid cessation of bleeding following administration strongly suggested that correction of coagulation factor deficiency played a major role in achieving hemostasis. The successful use of Factor VIII in this case highlights several important clinical implications:

1) Correction of Consumptive Coagulopathy:
   Factor VIII supplementation helped restore intrinsic pathway coagulation activity in a patient with severe depletion of clotting factors due to DIC and massive hemorrhage.
2) Control of Diffuse Surgical Site Bleeding:
   Persistent oozing from wound and suture sites often reflects coagulation failure rather than surgical

bleeding. Factor VIII contributed to stabilization of fibrin clot formation and effective control of diffuse hemorrhage.

3) Reduction in Further Blood Product Requirement: Effective hemostasis following administration reduced ongoing blood loss and likely minimized the need for further massive transfusion, thereby decreasing risks associated with transfusion-related complications.
4) Avoidance of More Radical Surgical Procedures: Successful control of hemorrhage potentially prevented the need for peripartum hysterectomy, which might otherwise have been required as a life-saving procedure.
5) Potential Life-Saving Rescue Therapy: In critically ill obstetric patients with refractory hemorrhage unresponsive to conventional management, recombinant coagulation factor therapy may serve as an important rescue modality.

Although recombinant activated Factor VIIa is more commonly described in refractory obstetric hemorrhage, this case demonstrates the potential role of Factor VIII replacement in severe coagulopathy-associated bleeding where deficiency or dysfunction of intrinsic pathway factors contributes significantly to failure of haemostasis. However, its use should remain individualized, multidisciplinary, and carefully monitored because of potential thromboembolic risks.

This case therefore emphasizes the importance of early recognition of coagulation factor deficiency and the possible role of advanced coagulation factor therapy in selected cases of catastrophic obstetric hemorrhage associated with DIC and HELLP syndrome.

3. Conclusion

Factor VIII can serve as an effective life-saving rescue therapy in cases of refractory obstetric hemorrhage where conventional medical, surgical, and transfusion measures fail to achieve adequate hemostasis. Its timely administration may help control bleeding rapidly, reduce transfusion requirements, prevent hysterectomy, and improve maternal survival.

This case report has certain limitations. As this was an emergency clinical situation, comprehensive coagulation factor assays could not be performed. Therefore, the patient may have had an underlying coagulation factor deficiency ***or a previously*** undiagnosed bleeding disorder, including a rare form of hemophilia or other coagulation abnormality, which was not identified at the time of management. Consequently, the favorable response observed cannot be attributed solely to Factor VIII with absolute certainty.

This report is not intended to establish efficacy but rather to share a successful clinical experience with the use of Factor VIII in refractory obstetric hemorrhage. We hope that this success story encourages further research and larger studies to evaluate the potential role of Factor VIII in selected cases of severe obstetric bleeding where conventional management fails.

Massive obstetric hemorrhage associated with abruptio placentae, HELLP syndrome, and consumptive coagulopathy requires prompt multidisciplinary management. In this patient, coagulation factor VIII therapy was associated with successful control of refractory bleeding after failure of conventional measures. Although a favorable outcome was achieved, larger studies are required to clarify the safety, indications, and effectiveness of this approach in severe obstetric hemorrhage

References

[1] American College of Obstetricians and Gynecologists. ACOG Practice Bulletin No. 183: Postpartum Hemorrhage. Obstet Gynecol. 2017; 130: e168–e186.
[2] Royal College of Obstetricians and Gynaecologists. Prevention and Management of Postpartum Hemorrhage. Green-top Guideline No. 52. London: RCOG; 2016.
[3] Cunningham FG, Leveno KJ, Bloom SL, et al. Williams Obstetrics. 25th ed. New York: McGraw Hill; 2018.
[4] Sibai BM. Diagnosis and management of HELLP syndrome. Obstet Gynecol. 2004;103(5 Pt 1):981–991.
[5] Levi M, Ten Cate H. Disseminated intravascular coagulation. N Engl J Med. 1999; 341: 586–592.
[6] Franchini M, Mannucci PM. The use of clotting factor concentrates in obstetric hemorrhage. Thromb Res. 2013;131(Suppl 1): S55–S59.
[7] Charbit B, Mandelbrot L, Samain E, et al. The decrease of fibrinogen is predictive of severe postpartum hemorrhage. J Thromb Haemost. 2007; 5: 266–273.
[8] James AH. Bleeding disorders in pregnancy. Obstetrics and Gynecology. 2010;115(2 Pt 1):393–402.
[9] Collins PW, Cannings-John R, Bruynseels D, et al. Viscoelastometric-guided early fibrinogen concentrate replacement during postpartum hemorrhage: OBS2 randomized controlled trial. British Journal of Anaesthesia. 2017;119(3):411–421.
[10] Levy JH, Goodnough LT. How I use fibrinogen replacement therapy in acquired bleeding. Blood. 2015;125(9):1387–1393.
[11] Peyvandi F, Garagiola I, Menegatti M. Treatment of rare factor deficiencies in women. Haemophilia. 2013; 19 (Suppl 4):180–184.
[12] Erez O, Mastrolia SA, Thachil J. Disseminated intravascular coagulation in pregnancy: insights in pathophysiology, diagnosis, and management. Am J Obstet Gynecol. 2015;213(4):452–463.
[13] Franchini M, Lippi G. Factor VIII and von Willebrand factor in hemorrhagic disorders and thrombosis. Semin Thromb Hemost. 2010;36(5):540–546.