



A CASE REPORT ON ANAESTHETIC & OBSTETRIC MANAGEMENT OF ANTEPARTUM HAEMORRHAGE WITH SHOCK

Anaesthesiology

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ABSTRACT

Maternal mortality is a matter of great concern in developed and developing nations. More than 25% of maternal deaths can be attributed to haemorrhage. The new target of a global MMR of <70 deaths per 100 000 live births by 2030 is ambitious, yet achievable and to reach this target a significantly increased effort to promote and ensure universal, equitable access to reproductive, maternal and newborn services for all women and adolescents will be required. One of the major shortcomings for providing high quality emergency obstetric care is a serious shortage of specialists such as Obstetricians and Anaesthetists at various levels in rural setting. Anaesthetists, perhaps the only perioperative physicians in labour room and operating room with special training in resuscitation and critical care get involved early in the management of these patients. The differences in management options between antepartum haemorrhage (APH) and postpartum haemorrhage (PPH) are that there are two individuals to care for in APH (delivery of the fetus and placenta will help to arrest the bleeding). Thus a multidisciplinary approach and team effort can go a long way in averting impending death and disability.

KEYWORDS

Abruptio, APH, IOCS.

CASE REPORT

A 29 Years old female unbooked primigravida patient with 21 weeks of gestation + IVF conception + abruptio placenta referred from IVF centre in view of bleeding per vagina to our hospital. Patient came to ER with complaints of bleeding PV from morning approximately of 300ml, taken Inj Susten 100mg IM, Inj Proluton 500mg IM, Inj Tranexamic acid 1gm Iv at outside hospital. Patient doesn't have any comorbidities, H/O laparotomy was done for acute abdominal pain 10 years ago which was uneventful. On examination patient was conscious, oriented, per abdomen size was 21 weeks with tenderness, p/s shows minimal bleeding through OS, PV shows soft cervix and 1 finger dilatation. USG scan was done which shows retroplacental clot small in size and vitals were stable. Patient was on Tab Ecospirin 150mg, last dose was on previous night. Investigation reports one month ago shows Hb: 12.6g/dl, TLC: 9900, Platelet count: 1.92L/cumm, PT/INR: 12/1.35, B+ve blood group, TSH: 1.08, serum creatinine: 0.5.

After 10 minutes, patient c/o giddiness, altered consciousness, with PR: 50bpm, feeble; BP: 60/30mmHg, grbs: 97mg/dl. Call attended for patient being unresponsive and hypotension, reached Emergency Room in a minute. On examination patient was drowsy, arousable, irritable with altered sensorium, bp was not recordable, PR: 40-50bpm, carotid and radial artery pulses were feeble, Spo₂: 90-94% at RA. Immediately secured two 18G cannulas, iv fluid 500ml bolus given, oxygen 6lit/min started, Inj. Noradrenaline 4mg in 50ml DNS at the rate of 0.05 mcg/kg/min infusion started, blood samples taken and sent for major surgical profile + cross matching, reserved 2pint PRBC, 4pint FFP and requested to issue 2pint PRBC. PV shows ongoing severe bleeding with gush of blood clots around 250ml from OS, P/A shows tense abdominal wall of 30 weeks size. Emergency USG was done by radiologist, noted fetal bradycardia with retroplacental clot of 15x11x12cm. Foley's catheterisation done. Patient shifted to OT immediately with precautions. Audiovideo counseling was given to the patient attenders about the patient condition regarding APH, Fetal death, PPH, haemorrhagic shock, ionotrope support, General anaesthesia, post op ventilation, blood and blood products transfusion, haemodynamic instability etc; written informed High risk consent was taken. Case accepted under ASA grade 5E.

Current ECG shows normal sinus rhythm, pallor⁺⁺ seen, haemodynamics (BP: 90/40mmHg, HR: 140BPM, SPO₂: 90-94% at room air) RS: Bilateral normal vesicular breath sounds heard, no added sounds, CVS: S1S2- were heard. Patient was monitored with pulse rate, NIBP, ECG, SPO₂ and urine output. Ventilated with 100% O₂, patient was induced by RSI (rapid sequence induction) with Inj Etomidate 10mg iv; Inj Glycopyrrolate 0.2mg iv, Inj Atracurium 40mg

iv, direct laryngoscopy was done, intubated with 7.0mmID cuffed portex OETT, confirmed bilateral air entry equal and secured ET at No 20, surgery started simultaneously, maintained with sevoflurane: o₂: air: 0.5%: 2: 2. PR-150/min, BP-70/30mmHg on NIBP, peripheral pulses, carotids were feeble.

Patient delivered a single live (deeply asphyxiated) female baby of weight 390gms at 1:57pm; as breech with APGAR 1. Gush of 1litre fresh blood along with clots seen. Vanishing twin along with placenta and membranes (196gms) delivered into toto.

Inj Fentanyl 100mcg Iv given after baby delivery, Inj Carbetocin 100mcg iv; Inj Oxytocin 10 units IM; 10 units added in 500ml RL; uterus was atonic; Inj carboprost 250mcg IM given; uterine massage was done, still atonic uterus seen, intramyometrial carboprost 250mcg given and Hayman sutures placed. Uterus tone was good, Mops and instrument count verified, haemostasis achieved.

Intra Op Investigations:

Hb-6.3gm%, TLC-7790, PLT-94000/cumm. As blood products were not arrived, managed with crystalloids and colloids initially. During the procedure blood loss was around 1500ml to 2 litres. Sent ABG, coagulation profile, serum electrolytes. Her vitals were improved with ionotrope support and blood products intraoperatively with HR 130-140/min and BP was 90-95/60 mm of Hg. During Intra Operative period, patient received 2pint PCV, 2pint FFP, 2pint RL, 1pint NS, 1pint Haemaccel, Inj Noradrenaline infusion @ 20 mcg/min was continued. Inj Calcium Gluconate 1gm (10ml of 10% calcium gluconate) & Antibiotics were given. After about an hour, HR settled to 120/min, BP: -110/70 (on ionotropic support). Urine output was 50ml. Inj Furosemide 20mg was given to prevent fluid overload. Hysterotomy was done in 2hrs and patient was extubated smoothly with reversal Inj Neostigmine 2.5mg + glycopyrrolate 0.5mg. Patient was conscious, respiration was regular and adequate. Haemodynamically stable and total urine output by the end of surgery was 750ml. Patient was shifted to ICU for further treatment and management as ionotrope support was still on flow at 8mcg/min. Her ABG report was as follows- pH: 7.38, pCO₂: -33, HCO₃: -19.5, BE: -5.2, PO₂: 54. Serum Calcium: 8.06, Na/k/cl-132/4.1/96. Ionotrope support was gradually reduced and then stopped, B/L lower limb stockings applied, Antibiotics were escalated.

During her stay in hospital she received total 3pint PCV, 3pint FFP and 500mg Ferric carboxy Maltose. She never had fever and her wound was healthy. Her subsequent recovery was uneventful. She was shifted to ward on 4th postoperative day and was discharged home on 6th postoperative day.

DISCUSSION

Antepartum haemorrhage (APH) is bleeding from the genital tract after 24 weeks gestation up to and including delivery of the fetus¹. Incidence of APH is 2-5% of all pregnancies³. Main causes are

1. Placenta praevia
2. Placenta abruption
3. Uterine rupture
4. Vasa praevia. Major complications of APH are perinatal mortality, preterm labour and PPH².

Abruptio placenta is defined as premature separation of a normally situated placenta. Incidence is 0.6-1% of all births. Maternal haemorrhage may be revealed (80%) or concealed (20%). Fetal compromise occurs because of loss of placental surface area for fetomaternal exchange of oxygen and nutrients. Risk factors include maternal hypertension, multiparity, uterine over distension, previous abruption, advanced maternal age and abdominal trauma. There is often associated increased abdominal pain, uterine tone, vaginal bleeding, uterine tenderness and premature labour¹. Fetal distress is common and can be the presenting feature⁸. In cases of concealed abruption, vaginal bleeding can be absent and an underestimation of maternal hypovolemia may occur^{9,10}. USG is highly specific for abruption (96%), but is not very sensitive (24%)^{10,12,13}.

Complications Of Abruption:

Maternal: haemorrhagic shock, Disseminated intravascular coagulation, acute renal failure, death. **Fetal:** prematurity, IUGR, IUD^{9,10}.

Coagulopathies In Pregnancy:

The etiology of coagulopathy in obstetrical hemorrhage may be due to dilutional coagulopathy, disseminated consumption and increased fibrinolysis¹⁸. Other than obstetric causes of coagulopathy in pregnancy (abruptio placentae, preeclampsia) bleeding disorders should also be kept in mind as an important differential diagnosis. PPH may be the first indication in patient suffering from Von Willebrand disease. A patient with menorrhagia when screened in antenatal care may be timely diagnosed for such types of bleeding diathesis. Hypercoagulable state of pregnancy is marked by increases in fibrinogen concentration, Von Willebrand Factor, FVII, FVIII and FIX concentrations. One third of coagulopathies in pregnancy are attributable to abruption and coagulopathy is associated with fetal demise¹⁴. In abruptio placenta the extrinsic pathway is triggered by tissue destruction. Thromboplastin is liberated from the placenta leading to consumption of clotting factors and platelets. Fibrinolytic system gets activated in response to fibrin deposition. Plasminogen in turn converts to plasmin causing lysis forming fibrinogen, fibrin monomer, fibrin polymer and fibrinogen degradation products-FDP. These in turn cause bleeding. Plasma fibrinogen levels are diagnostic in severe haemorrhage. Rapid detection and treatment of hypofibrinogenaemia is essential to stabilize the situation. The use of near-patient testing of coagulation using ROTEM allows monitoring of trends of coagulopathy^{15,16,19}.

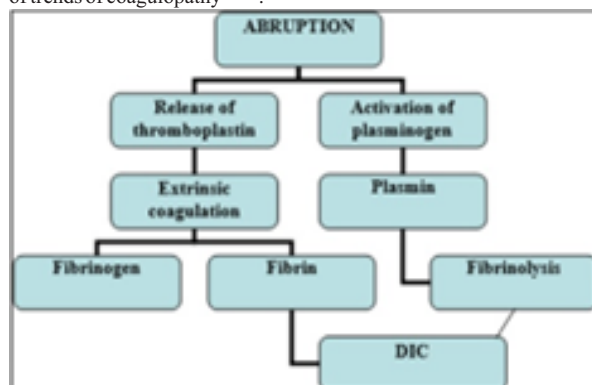


Figure 1: Abruptio placenta and DIC

Bonnar describes a five-step management plan for major obstetric haemorrhage (MOH)¹⁷

1. Organization of multidisciplinary team
2. Treating the underlying cause of bleeding
3. Restoration of blood volume
4. Correction of defective coagulation
5. Evaluation of response to treatment

Resuscitation of bleeding obstetric patients with severe hemorrhage may decompensate rapidly, so they require a TEAM (together everybody achieves maximum) approach. A successful resuscitation needs a multidisciplinary team effort, including senior obstetricians, anesthesiologists, general physician, haematologists, neonatologists, nurses, paramedical staff should be organized early in the course of obstetric hemorrhage, involving other services as necessary in order to achieve good outcome. The focus of resuscitation should be the preservation of the woman's life rather than preservation of her uterus²⁰. The anesthesiologists & obstetricians should have thorough knowledge of normal physiological changes in pregnancy and hence their role is crucial in the management of obstetric hemorrhage. Multiple tasks have to be carried out quickly and with expertise in the event of massive hemorrhage. Hence timely and effective communication between the obstetrician and anesthesiologist is adjutory.

Resuscitation:

The urgency and measures taken to resuscitate and arrest hemorrhage need to be tailored to the degree of shock.² A recumbent pregnant woman can maintain a normal pulse and blood pressure until she has lost one third of her blood volume due to relative hemodilution and high cardiac output. Therefore haemodynamic status may not correspond with apparent blood loss in obstetric haemorrhage. As visual blood loss estimation does not accurately judge the blood loss⁴, other reliable methods such as blood collection drapes and weighing swabs, floor spills and suction bottles are preferred. A laparotomy sponge if 50% soaked with blood gives an estimate of 25ml blood loss and in case it is dribbling with blood the estimated loss is 100ml⁵. Multidisciplinary observations of estimated blood loss revealed that these were grossly miscalculated by 30-50%⁴. Accurate estimation of blood loss along with measurement of vital signs can be used to classify haemorrhagic shock into 4 different stages. Royal College of Obstetricians and Gynaecologists recommends MEOWS (maternal obstetric early warning score) based on below clinical parameters for early identification of the shock². Thus, MEOWS includes looking for signs such as tachycardia, hypotension, decreased urine output, pallor, lower abdominal pain, altered conscious level, tachypnea, fetal demise and cold peripheries⁶. The "rule of 30" is useful if the patient's systolic BP drops by 30%, the heart rate rises by 30%, the respiratory rate increases to more than 30/min, the hemoglobin or hematocrit drops by 30% and the urine output decreases to <30ml/hr, the patient is likely to have lost 30% of her blood volume⁷.

Table 1: Classification Of Shock.

Classification of shock	STAGE 1	STAGE 2	STAGE 3	STAGE 4
BLOOD LOSS	<15%	15-30%	30-40%	>40%
HEART RATE	<100/min	>100/min	>120/min	>140/min
SYSTOLIC BLOOD PRESSURE	Normal	Normal	decreased	decreased
RESPIRATORY RATE	14-20/min	20-30/min	30-40/min	>40/min
MENTAL STATUS	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

Monitoring & Assessment Of Intravascular Depletion:

The extent of bleeding is almost always underestimated in obstetric patients.² Signs suggestive of hypovolemia in APH patients should be monitored carefully.²¹ Clinical signs of shock have to be continuously monitored like colour of the patient, pallor, urine output, temperature and capillary refill time. Invasive blood pressure monitoring may be done for fragile patients². All the parameters recorded have to be documented well on a flow chart for further references.

1. Hypotension
2. Heart rate > 120 beats/minute
3. Urine output < 0.5 ml/kg/minute
4. Capillary refill time < 5 seconds

Irrespective of the degree of hypovolemic shock, fluid therapy is best guided by continual assessment of maternal vital signs, urine output, hemoglobin and acid base balance^{22,23}. The main aims of management are rapid resuscitation to restore tissue oxygen delivery while predicting, preventing and correcting hemostatic disorders.

Anaesthetic & Surgical plan in APH:

If surgery is required, one should remember to ensure that routine

safety precautions are taken including anesthetic history, airway assessment, antacid prophylaxis and preoxygenation with 100% oxygen. Best anesthetic plan for abruptio placenta is general anesthesia with endotracheal tube, even in hemodynamically stable patients. This is because of the high propensity of post partum hemorrhage in patients of abruptio placenta. Rapid sequence induction with cricoid pressure has to be done considering all pregnant patients to be full stomach. The choice of induction agent strongly depends on the hemodynamic stability of the patient. Ketamine is the agent of choice in patients presenting with shock. Etomidate is another good alternative. However, thiopentone and propofol have to be used cautiously because of their deleterious effect on circulatory system. For maintenance, a volatile agent has to be added to prevent awareness with a concentration of not more than 0.5 MAC, else the chances of uterine atony and postpartum hemorrhage become high. Nitrous oxide should not be used for maintenance in case the indication for cesarean section is fetal distress. Pain has to be addressed well. Oxytocin should be given to prevent uterine atony. Tranexamic acid 1gm IV is given when uterotonics fail to control bleeding. Selective embolisation of these vessels may lead to cessation of bleeding. In cases that fail to respond to these conservative methods, hysterectomy may be necessary.

Restoration Of Blood Volume

The cornerstones of resuscitation in peripartum hemorrhage are restoration of both blood volume and oxygen carrying capacity.² All delivery units, especially small units without the blood bank facility, should maintain a supply of O Rh negative blood, as this might offer the only means of restoring oxygen carrying capacity within an acceptable timescale. The clinical picture is the main determinant for the need of blood transfusion and time should not be wasted for laboratory results. Volume replacement must be undertaken on the basis that blood loss is often grossly underestimated. Compatible blood in the form of red cell concentrate is the best fluid to replace major blood loss and should be transfused whenever deemed necessary.²

Choice of fluid/blood/blood products:

There is controversy as to the appropriate fluids for volume resuscitation. The nature of fluid infused is of less importance than rapid administration and warming of the infusion.²³

Table 2: Fluids & Blood Products For Volume Resuscitation

IV Fluid	Use
Crystalloid	Up to 2 lit of Hartmann's solution
Colloid	Up to 1-2 lit colloid until blood arrives
Blood	Crossmatched. If crossmatched blood unavailable, give uncrossmatched group-specific blood OR 'O-negative' blood
Fresh frozen plasma (FFP)	4 units for every 6 units of red cells or PT/APTT > 1.5 x mean control (12-15 ml/kg or total 1 lit)
Platelet concentrates	If platelet < 75 x 10 ³ /mm ³
Cryoprecipitate	If fibrinogen < 1 g/l

According to British Committee for Standards in Hematology, the therapeutic aim for the management of massive blood loss is to maintain.

1. Hemoglobin ≥ 8 g/dl
2. Platelet count $\geq 75 \times 10^9$ /L
3. Prothrombin time ≤ 1.5 mean control
4. Activated prothrombin time ≤ 1.5 mean control
5. Fibrinogen ≥ 1.0 g/dl

FFP In some centers red cells: FFP: Platelets are advocated into a 1:1:1 protocol. The products are frequently issued as "shock packs" on activation of a major obstetric hemorrhage protocol. The rationale for this approach is to maintain thrombin generation and fibrinogen by the replacement of coagulation factors as early as possible, and that it takes too long in practice to obtain lab results and issue components. The disadvantage of unmonitored "shock packs" is that the majority of woman will have completely normal coagulation and platelets at the time of administration and will be receiving blood products with less fibrinogen and other coagulation factors than they have circulating. FFP is donated from nonpregnant population and has fibrinogen level of around 2 g/L and will therefore lead to reduction in fibrinogen, factor VIII, Von Willebrand factor due to coagulation. In order to raise the fibrinogen level by 1 g/L, 30 ml/kg of FFP needs to be given, compared with 3 ml/kg of cryoprecipitate and 60mg/kg of fibrinogen concentrate.

Recombinant FVIIa is a controversial option to be given as last resort just before hysterectomy. Before administration patient should ideally have platelet count 20,000/mm³, fibrinogen >1 g/dl, temperature >32°C, pH >7.2 and normal ionized calcium. These preconditions will facilitate adequate functioning of clotting cascade. Optimal dose in obstetric hemorrhage is unknown though dose of 90 mg/kg is used. Despite having a very short half-life (2-6 h), concerns about thromboembolism with factor VIIa as a complication are real. A recent systematic review showed a higher risk of arterial thrombosis (not venous) among patients who received factor VII as an adjunct therapy for life-threatening bleeding. Leighton et al advised against rVIIa administration in the setting of amniotic fluid embolism because tissue factor may play a role in its pathophysiology and thrombotic complications may be increased.²⁵ It is recommended to give deep vein thrombosis (DVT) prophylaxis once the bleeding risk is considered to be low.

Tranexamic acid this has been shown to reduce bleeding and transfusion requirement in massive hemorrhage.

Intraoperative Cell Salvage (IOCS) IOCS is now an established technique in the management of hemorrhage complicating caesarean section. IOCS was adopted late in obstetrics relative to other surgical interventions as a result of concerns regarding the potential for harvest and retransfusion of amniotic fluid causing so-called "amniotic fluid embolus." But now, it has been proved that these fears were unfounded and that amniotic fluid is effectively removed by the salvage process and administration via a leukocyte depletion filter. This technique can also reduce exposure to allogenic blood transfusion along with its risks as well as is cost-effective.

Blood Conservation Strategies: Autologous blood transfusion (donation, storage and retransfusion) has been shown to be safe in pregnancy. Autologous transfusion is generally reserved for situations with a high chance of transfusion in a patient with rare antibodies. Early identification of patients at risk for obstetric hemorrhage and storage of autologous blood has been attempted for pre-operative autologous blood donation. It is important to identify women who refuse blood or blood product transfusion. For example, Jehovah's witness in which autologous transfusion is the only option.

After care: The increasingly important issues in blood transfusion are adverse effects associated with transfusion, including potential infection and transmission of prions, rising costs and the possible future problems of availability. Patients should be monitored carefully for diagnosing the complications (fever, hyperkalemia, hypocalcemia, citrate toxicity etc) seen after massive blood transfusion. Regular monitoring of hemoglobin level and coagulation is needed. FFP, platelets and cryoprecipitate transfusion may be necessary if coagulopathy develops. All women following major APH require intensive monitoring for at least first 24-48 hours. Post-operative management includes transfer to intensive care unit/high dependency unit. Equal attention should be given to rebound hypercoagulation and thromboembolism that follows blood transfusion especially in pregnancy which is a hypercoagulable state by itself. This can be prevented by usage of graduated compression stockings and pharmacological thromboprophylaxis. Once the bleeding is arrested and any coagulopathy is corrected, thromboprophylaxis is administered as there is a high risk of thrombosis. Pneumatic compression devices can be used if thromboprophylaxis is contraindicated in cases of thrombocytopenia. It is important to remember that thrombo-embolic disease (TED) is still one of the commonest cause of maternal morbidity.

CONCLUSION

The role of anesthesiologist & obstetrician, Prompt communication between them in the management of obstetric hemorrhage is crucial. Teamwork is essential in case of both anticipated and unanticipated MOH. Team responses that emphasize the accurate estimation of blood loss, early warning signs of shock, rapid response to blood loss, coagulopathy, a clear cut protocol are associated with less maternal morbidity & mortality. Urgent access to definitive care remains a major stumbling block in limited resource areas in the developing countries. However, recent advances in prediction and assessment of blood loss, a better understanding of coagulation mechanisms, should improve the efficacy of management of MOH. Central to success is the flair and leadership skills of anesthesiologist in coordination of resuscitation and communication among team members.

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