Review Article

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Anesthetic Considerations and Management of Obstetric Hemorrhage

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Abstract

Massive obstetric hemorrhage (MOH) remains a significant cause of maternal morbidity and mortality worldwide. Causes may be antepartum, intrapartum, and postpartum bleeding. This review highlights the various factors responsible for MOH, its management and the most recent treatment guidelines. Team responses that emphasize the accurate estimation of blood loss, early warning signs of shock, rapid response to blood loss, and coagulopathy will lead to reduction in maternal morbidity. The hemostatic management of major obstetric hemorrhage is a major challenge for both anesthesiologist and obstetricians. Bleeding leads to consumption of coagulation factors which may be exacerbated by dilutional coagulopathy after volume resuscitation. Many laboratory-based tests like plasma fibrinogen concentration and platelet count are unsuitable for emergency use due to their long turnaround times, so they have limited value for the management of PPH. Current evidence suggests that viscoelastic monitoring using thromboelastography or thromboelastometry based tests may be useful for rapid assessment and for guiding hemostatic therapy during acute hemorrhage. Intraoperative blood salvage may be life-saving in cases of intractable hemorrhage if banked blood is not available. The safety and efficacy of recombinant activated factor VII therapy have not been fully evaluated in the treatment of obstetric hemorrhage.

Key words: Fibrinogen, Operative blood salvage, Postpartum hemorrhage, Pregnancy, Thromboelastography

INTRODUCTION

A major obstetric hemorrhage remains one of the leading causes of maternal mortality and morbidity worldwide.[1] A study by world health organization revealed that 25-30% of maternal deaths are due to peripartum hemorrhage globally.[2] Despite the widespread reduction in maternal deaths due to improved antepartum, intrapartum and postpartum care in developed nations, mortality rates are persistently high in many countries which are unable to provide advanced medical care. Postpartum hemorrhage accounts for a substantial proportion of maternal deaths in developing countries.[3] A lot of literature is available regarding causes, prevention, and management of massive obstetric hemorrhage (MOH). We have tried to compile the literature and include the latest developments in this field. This article aims to provide a practical and pragmatic approach to the management of MOH, coagulopathy associated with it, although it is recognized that due to limited evidence, expert opinion will vary.

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Management of MOH is often challenging due to many contributing factors. Blood loss can be underestimated because bleeding may be concealed and the presence of amniotic fluid makes accurate measurement difficult. The physiological changes of pregnancy mask the magnitude of the blood loss.[4] Serious morbidity resulting from hemorrhage includes adult respiratory distress syndrome, coagulopathy, shock, loss of fertility, and pituitary necrosis.[5]

METHODS

An extensive literature search was performed through Medline, PubMed, and Google scholar using the keywords

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such as postpartum hemorrhage, thromboelastography (TEG), pregnancy, fibrinogen, and operative blood salvage. Abstracts from potentially relevant publications were also studied. In this narrative review, we have discussed the various causes of MOH, management of unanticipated hemorrhage, massive transfusion protocols, and anesthesia concerns, especially if emergency hysterectomy is to be done.

CAUSES OF OBSTETRIC HEMORRHAGE

Obstetric hemorrhage is classified into antepartum, intrapartum, and postpartum hemorrhage (Table 1).

Antepartum Hemorrhage

Antepartum hemorrhage is defined as bleeding from genital tract after 24 weeks of gestation and has an incidence of 2-5% of all pregnancies beyond 24 weeks.[5] APH represents a greater threat to fetus than mother and fetal compromise may precede maternal complications.[6] Placenta previa and abruption are major causes of significant hemorrhage in the third trimester.

Placenta previa

In placenta previa, there is an abnormal implantation of placenta in the lower uterine segment.[2] Placenta accreta, increta, and percreta are conditions of abnormal placentation in which there is an increasing degree of abnormal invasion of the placenta into the myometrium.[4] The presence of placenta previa increases the likelihood that the patient will require a peripartum hysterectomy.[6] The classic sign of placenta previa is painless vaginal bleeding during the second or third trimester.

Cesarean section is the recommended mode of delivery. Patients with placenta previa are at significantly increased the risk for high intraoperative blood loss due to the possibility of the obstetrician incising through the placenta and the increased risk for placenta accrete.[7] The uterine site of abnormal implantation does not contract as efficiently as a normal uterine segment which leads to increased bleeding.

Table 1: Causes of obstetric hemorrhage

Causes of hemorrhage
Antepartum hemorrhage
Placenta previa
Placental abruption
Genetic coagulopathies
Postpartum hemorrhage
Uterine atony
Placenta accreta/increta/percreta
Retained placenta
Lacerations during vaginal delivery

Placental abruption

Hemorrhage arising from premature separation of a normally situated placenta is known as placental abruption. Risk factors include maternal hypertension, multiparity, uterine over distension, previous abruption, advanced maternal age, and abdominal trauma. There is often associated increased uterine tone, abdominal pain, and premature labor. Fetal distress is a common and can be the presenting feature.[4]

In cases of concealed abruption, vaginal bleeding can be absent and an underestimation of maternal hypovolemia may occur.[8] The major complications of abruption include hemorrhagic shock, acute renal failure, coagulopathy, and fetal demise. Abruption is the most common cause of disseminated intravascular coagulation in pregnancy.[9]

Uterine rupture

Uterine rupture is the most life-threatening emergency in obstetrics associated with high maternal and perinatal morbidity and mortality.[10] Risk factors include prior uterine surgery, trauma, uterine anomalies, dystocia, use of uterotonic drugs, and abnormal placentation. Clinical presentation can vary from subtle findings such as uterine tenderness and non-reassuring fetal heart rate patterns to severe localized abdominal pain and a rapid onset of maternal hypovolemic shock.[11]

Fetal delivery with repair of ruptured uterine wall is the definitive treatment. Uterine and internal iliac arterial ligation may be done in anticipated cases.[12] In intractable blood loss as a last resort, obstetric hysterectomy is advocated without wasting time in decision making.[13]

Postpartum Hemorrhage

Postpartum hemorrhage is defined as blood loss of 500 ml or more from genital tract in the first 24 h of delivery. Massive PPH is defined as the blood loss of 1000 ml or more.[14] It can be further subdivided into minor (500-1000 ml) or major (>1000 ml).

Blood loss is frequently underestimated and physiological variables especially that of systolic blood pressure (BP) may change little until 30-40% of circulating blood volume has been lost. High index of suspicion for major obstetric hemorrhage must be maintained.[15]

MANAGEMENT OF MOH

MOH is defined as blood loss from the uterus or genital tract >1500 ml or decrease in hemoglobin of >4 g/dl or acute loss requiring transfusion of more than 4 units of blood.[1]

Prevention

- Avoidance of prolonged labor.
- Minimal trauma during assisted vaginal delivery.
- Detection and treatment of anemia during pregnancy.
- Identification of placenta previa by antenatal ultrasound examination.
- Magnetic resonance imaging (MRI) to determine placenta accreta/percreta. If present then multidisciplinary planning is required.
- Active management of the third stage as below

    1. Early clamping of umbilical cord
    2. Controlled cord traction for placental delivery and prophylactic administration of uterotonics at delivery[16] (e.g., oxytocin).
    3. A long acting oxytocin derivative carbetocin (single dose of 100 μg) is at least as effective as oxytocin.[17,18]

Women experiencing obstetric hemorrhage are in good health, young and initially compensate well for losses due to the hypovolemia until the circulating blood volume is very low. Modified early obstetric warning system (MEOWS) is a useful bedside tool for predicting morbidity of these patients and is recommended in all obstetric patients. It is helpful to track maternal physiological parameters and to aid early recognition and treatment.[1] Blood loss is generally underestimated both in volume and rapidity.[19] Blood loss may be concealed and difficult to calculate. Thus, MEOWS includes looking for signs such as tachycardia, hypotension, decreased urine output, pallor, lower abdominal pain, and cold peripheries.[1] The "rule of 30" is useful if the patients systolic BP drops by 30 %, the heart rate rises by 30%, the respiratory rate increase to more than 30/min, the hemoglobin or hematocrit drops by 30% and the urine output decreases to <30 ml/h, the patient is likely to have lost 30% of her blood volume. The "shock index" defined as the heart rate divided by systolic BP (normal up to 0.9 in obstetrics) has been shown to be an accurate indicator of compensatory changes in the chorionic villus sampling due to blood loss. Active periodic estimation improves the accuracy of estimation.[20] According to one study, there was 16% underestimation at 300 ml blood loss which rose to 41% at 2000 ml loss.[21]

Management depends on whether the MOH is anticipated/unanticipated.

Anticipated MOH

If anticipated as in patient of low lying placenta or uterine scar, placenta accreta, we should keep two large bore intravenous cannulae ready with rapid infusion device/pressure bags, cross-matched blood, and blood warmer. Invasive monitoring, cell salvage, and interventional radiological procedures should be considered. Antenatal diagnosis of placenta accreta is associated with less maternal and neonatal morbidity including decreased blood loss at delivery and transfusion of fewer units of blood products. Ultrasonography is a useful screening procedure but may be imperfectly sensitive/specific. MRI may help in such patient.

Unanticipated MOH

Bonnar describes a five-step management plan for MOH.[22]

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

Communication and teamwork are essential in case of both anticipated and unanticipated MOH.

Steps of management include:

- Alerting blood transfusion service and hematologist. Ensuring the availability of O negative, 2-4 units of blood for emergency use. Allocation of roles to the team members is essential for the effective management.
- Assessment of airways, breathing and circulation in accordance with advanced life support guidelines.[15]
- Attach monitoring lines, BP, electrocardiography, oxygen saturation.
- Give high flow oxygen via face mask with a reservoir bag.
- Head down tilt to increase the venous return and preserve cardiac output.
- Intravenous access with two large bore cannulae and take blood for cross matching.
- Foley's catheter to monitor urine output.
- Consider arterial cannulation for arterial blood gas, invasive monitoring of BP and blood samples for evaluation of coagulation.
- Fluid resuscitation gives warm crystalloids up to 2 L (ringer lactate, normal saline) with rapid infuser or pressure bags and colloids up to 1-2 L until blood arrives.
- Large volumes of cold fluids place the patients at risk of hypothermia which induces shivering and subsequently increases oxygen consumption in a patient with already decreased oxygen carrying capacity and decreased oxygen reserves. Hypothermia also impairs coagulation, affects renal and liver function and delays wound healing.

Prompt communication between anesthesiologist, obstetrician and gynecologist, nursing and laboratory staff and blood bank is essential for effective evaluation and management of excessive blood loss.

Coagulopathies in Pregnancy

Pathophysiology

The etiology of coagulopathy in obstetrical hemorrhage may be due to dilutional coagulopathy, localized consumption, disseminated consumption, and increased fibrinolysis.[23]

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.[2]

Biomarkers to diagnose coagulopathy of pregnancy

Hypercoagulable state of pregnancy is marked by increases in fibrinogen concentration, Von Willebrand Factor, F VII, FVIII and FIX concentrations.[24]

1. Fibrinogen: Fibrinogen levels fall below normal pregnancy range sooner than other coagulation factors,[25] and in some circumstances may rapidly fall to <2 g/l in PPH. There is strong evidence that a low clauss fibrinogen is an accurate biomarker for progression from moderate to severe PPH.
2. Rotational thromboelastometry (ROTEM) and TEG: Current evidence suggests that targeted goal-directed therapy using coagulation factor concentrates guided by visco-elastic methods such as ROTEM or TEG enables the effective correction of coagulopathy and is associated with a decreased incidence of allogenic blood transfusion and thrombotic/thromboembolic events and with reduced costs.[26]

There are many limitations of coagulation monitoring using TEG and ROTEM. By direct addition of an activator, such as tissue factor or kaolin ROTEM and TEG automatically bypasses primary hemostasis and therefore cannot detect disorders of primary hemostasis. Most viscoelastic tests also cannot diagnose the cause of coagulapathy involving platelet function defects, for example, abnormal/deficient platelets due to antiplatelet drugs such as clopidogrel.[27] Parallel assessment using point of care testing (POC) platelet function essays may, therefore, improve diagnosis.

3. ROTEM FIBTEM A5 assay: There is good evidence that the ROTEM FIBTEM A5 assay (available within 10 min) can be used as a surrogate for clauss fibrinogen during PPH.[25] This assay does not measure the same hemostatic parameter as clauss fibrinogen but provides similar measures of hemostatic competence.[28] As a rough guide, an FIBTEM A5 of 15 min equates to a clauss fibrinogen of about 3 g/L; 10 min to 2 g/L and 6 min to 1 g/L.

A recent audit of an algorithm to manage obstetric hemorrhage (>1500 ml and ongoing) based on FIBTEM A5 has been published. It showed that the use of fibrinogen concentrate in place of fresh frozen plasma (FFP)/platelet "shock packs" when the FIBTEM A5 fell below 7 mm (and considered if below 12 mm in clinically severe bleeding), led to a substantial reduction in transfused red cells, FFP, cryoprecipitate, platelets and transfusion-associated circulatory overload and a nonsignificant reduction in hysterectomy.[29] These data support the use of POC testing during PPH and provide evidence for a potentially appropriate intervention trigger (FIBTEM <12 mm and/or fibrinogen of 2.2 g/L). If the bleeding has stopped, the hemostatic blood products need not be given whatever the results. Unmonitored use of shock packs is unlikely to be beneficial for the majority of women.[25] (Figure 1)

CONSERVATIVE MANAGEMENT-UTEROTONIC DRUGS

Uterotonic drugs such as oxytocin, ergotamine, methyl ergot, and 15-methyl Prostaglandin F2α are used.

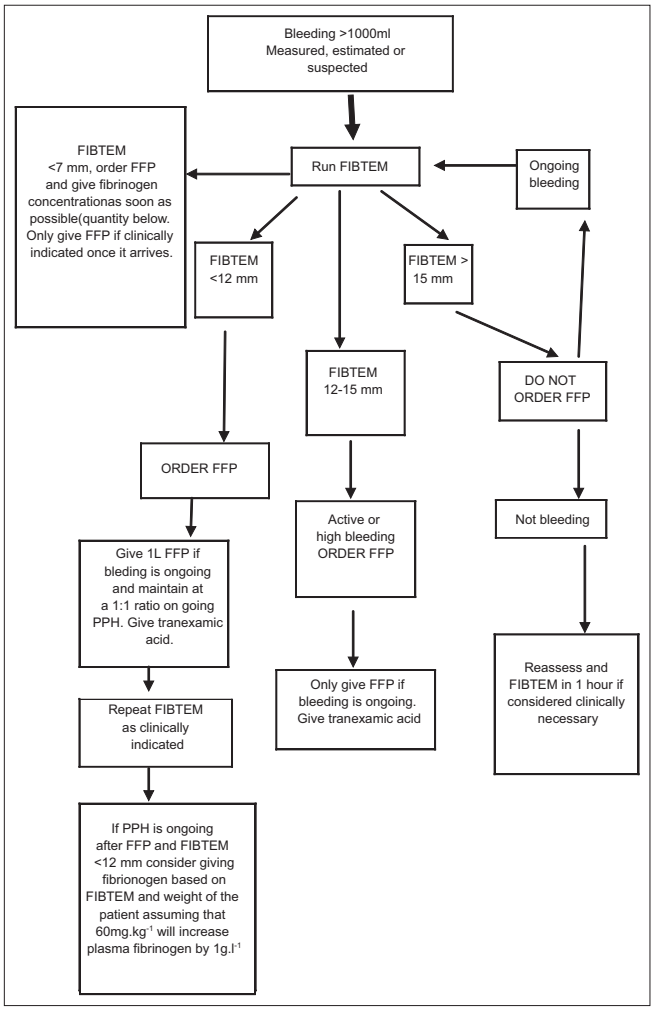

Figure1: Point of care FIBTEM algorithm used at the University hospital of wales in 2014. Reproduced with permission from author

Misoprostol, a prostaglandin E1 analog has shown great promise as an uterotonic agent. In a study rectal administration of 1000μg of misoprostol lead to sustained uterine contraction and control of hemorrhage within 3 min.[30]

Invasive Management

These are needed to be performed promptly in cases of failure of conservative management.

1. Intrauterine balloon tamponade:
   Sengestaken Blakemore esophageal catheter is most often used. The catheter is inserted in the uterine cavity and balloon is filled with warm sterile water/saline until uterus is firm on palpation and bleeding is arrested. This technique is least invasive, most rapid, and lacks significant complications.[31]
2. Uterine compression sutures-(b-lynch sutures):
   They are most useful in refractory uterine atony.[31] Advantages include high success rate, ease of placement and fertility preservation.[32]
3. Angiographic arterial embolization-interventional radiology:
   Uterine artery and ovarian artery contribute significantly to uterine blood flow during pregnancy.[31] Embolization of these arteries with angiographic occlusion balloon catheters requires fluoroscopic guidance and expert interventional radiologist.[32,33] The patient should be stable enough to be transferred to radiological unit and monitoring should continue along with the facility to proceed with the surgical intervention if the patient becomes unstable.
4. Arterial ligation:
   Surgical ligation of uterine, ovarian and internal iliac artery is useful when above methods have failed. Ligation of bilateral uterine arteries is easier than internal iliac artery ligation.[34]
5. Hysterectomy:
   Hysterectomy is a definitive treatment for PPH resulting from uterine atony and placenta accrete.[31] Peripartum hysterectomy is estimated to occur in 0.8/1000 deliveries.[13] If alternative interventions fail and bleeding continues, hysterectomy should not be delayed.

TRANSFUSION OF BLOOD AND BLOOD PRODUCTS

According to British Committee for Standards in Hematology, the therapeutic aim for the management of massive blood loss is to maintain.[33]

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

Cryoprecipitate

It contains a higher concentration of fibrinogen than FFP. Existing risk of immunological reactions and the transmission of infectious agents have led to its withdrawal in several European countries.[35] In the setting of PPH, cryoprecipitate is used to replace fibrinogen, which is rapidly consumed during obstetric hemorrhage. Guidelines recommend the use of cryoprecipitate to maintain the fibrinogen level above 1-1.5 g/L if FFP has not been successful.[36,37] Cryoprecipitate has been shown to successfully increase fibrinogen levels during PPH.[38] One pool (1 bag of cryoprecipitate contains 325 mg of fibrinogen. 5 bags make one pool or 1625 mg of fibrinogen) of cryoprecipitate is expected to raise the fibrinogen level by about 0.5 g/L in the average women although this will vary depending on consumption. Cryoprecipitate also contains a high concentrate of factor VIII, Von Willibrand factor and factor XIII, which will be depleted in established hemostatic failure.[25]

Dose of fibrinogen = (desired increase in g/L) × (plasma volume in L).

Thus, administration of 3 g of fibrinogen concentrate in a 70 kg patient increases the plasma fibrinogen concentration by 1.0 g/L (assuming 0.04 L/kg plasma volume) approximately.[39]

Platelets

Guidelines recommend that the platelet count should be kept >$(50 \times 10^9)^{-L}$ during ongoing PPH and to achieve this they should be infused when the count falls below $(75 \times 10^9)^{-L}$.[36,39] With the exception of placental abruption, amniotic fluid embolus, severe preeclampsia or inherited or immune thrombocytopenia, a platelet count < $(75 \times 10^9)^{-L}$ is common during PPH. The strategy of 1:1:1 of red cells: FFP: Platelet transfusion would result in multiple platelet transfusions well above recommended levels and cannot be justified on current evidence.

FFP

In some centers red cells: FFP: Platelets are advocated into a 1:1:1 protocol.[40] 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, and Von Willebrand factor due to coagulation. Early empirical FFP may be justified if significant consumption is likely (e.g., placental abruption or amniotic fluid embolus), or very large volume of blood loss is expected (e.g., uterine rupture or placenta accreta). By contrast uterine atony or surgical/genital tract trauma are unlikely to have early hemostatic impairment and early unmonitored FFP administration is more difficult to justify.[25]

VII a

Recombinant factor VIIa (rVIIa) is not the first line treatment for hemorrhage and is effective only once major source of bleeding have been controlled. Before administration patient should ideally have platelet count 20,000/mm^3, 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.[13] 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.[41] It is recommended to give deep vein thrombosis (DVT) prophylaxis once the bleeding risk is considered to be low.

Seighton *et al.* advise against rVIIIa administration in the setting of amniotic fluid embolism because tissue factor may play a role in its pathophysiology and thrombotic complications may be increased.[42] The ASA guidelines recommended considerations of rVIIIa therapy of traditional well-tested options for treating microvascular bleeding (i.e., coagulopathy) have been exhausted.[43]

Tranexamic ACID

This has been shown to reduce bleeding and transfusion requirement in massive hemorrhage secondary to a number of non-obstetric causes. Its role in obstetric bleeding is not established.[44]

The WOMAN trial (world maternal antifibrinolytic) is currently attempting to further assess the role of antifibrinolytic therapy in post-partum hemorrhage. This randomized and double-blinded trial started recruitment worldwide in 2009 in a design similar to the CRASH 2 trial in trauma. The trial has recruited >4000 women to date and is aiming for a total of 15,000. This data set should be able to elucidate the impact of tranexamic acid on mortality and morbidity of postpartum hemorrhage and possible complications. Furthermore, it includes developing countries where the need for a pragmatic and cost-effective treatment of post-partum hemorrhage is greatest.[45]

At present, the evidence for antifibrinolytics in obstetrics is limited. The EXADELI trial suggests a benefit of tranexamic acid in ongoing postpartum hemorrhage after vaginal delivery. There may also be a reduction in blood loss by prophylactic administration of tranexamic acid after cesarean section.[45] Tranexamic acid may be most beneficial for women who demonstrate hyperfibrinolysis based on hemostatic monitoring such as TEG.[27,46]

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 (LDF). Indeed use of a LDF and the requirement for dedicated suction for blood have not been questioned and abandoned by some advocates of technology.[47] Furthermore, authors have stated that reinfusion of blood harvested by cell salvage and passage through LDF, results in clinically insignificant bacteremia. Further LDFs are also known to remove bacteria from blood.[47] IOCS for autologous transfusion is already being used in cardiac, orthopedic and vascular surgeries with relative reduction of blood transfusion by 39% and absolute risk reduction by 23%.[1] The use of IOCS has an undisputed role in patients who refuse blood or blood components transfusion (Jehovah's witness) and in patients where massive blood loss is anticipated (placenta accreta, percreta).[2] 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.[48,49] Early identification of patients at risk for obstetric hemorrhage and storage of autologous blood has been attempted for pre-operative autologous blood donation. Since most patients do not have identifiable risk factors and many patients do not donate more than one unit

of blood, its utility in acute severe hemorrhage is uncertain. Its utility and safety however have been questioned as it may cause anemia, does not eliminate transfusion risks and cannot be used in an emergency.[50] It also subtracts the potential risks of homologous transfusions is cost-effective and decreases dependence on donors.[4] 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.[51]

Anesthesia Concerns in Patients with MOH

Senior anesthesiologist and intensive care team should be involved early as obstetric patients with severe hemorrhage may decompensate rapidly.

The focus of resuscitation should be the preservation of the woman's life rather than preservation of her uterus.[15]

- Continue resuscitation with warmed fluids and avoid hypothermia.
- Consider arterial line, central line and urinary catheter but only after definitive treatment has commenced. Their insertion must not delay resuscitation and fluid management.[1]
- The choice of anesthetic technique depends on the indication and urgency for delivery (as in the case of APH) the severity of maternal hypovolemia and the obstetric history, e.g., prior caesarean delivery.[8]
- The main aims of management are rapid resuscitation to restore tissue oxygen delivery while predicting, preventing, and correcting hemostatic disorders.
- The presence of cardiovascular instability is a relative contraindication to regional anesthesia. Blockage of the sympathetic system can potentially lead to worsening of hypotension due to hemorrhage. If cardiovascular stability has been achieved and there is no evidence of coagulation failure regional anesthesia can be used.[36]

This may be particularly appropriate where a working epidural has been in place during labor. Continuous epidural block is preferred over spinal as it allows better control of BP and can be used for prolonged surgery.

If surgery is required, one should remember to ensure that routine safety precautions are taken including anesthetic history, airway assessment, antacid prophylaxis, and preoxygenation.[15]

General anesthesia following a rapid sequence induction with cricoid pressure is the technique of choice in hemodynamically unstable patients.[15]

Etomidate or ketamine may be preferable to thiopentone or propofol in the presence of severe hypovolemia.[4] Ketamine 0.5-1.0 mg/kg has an excellent record of safety and efficacy in obstetric anesthesia practice.[8]

The volatile agents cause uterine relaxation and excessive concentrations should be avoided especially in the case of uterine atony.[15]

- Regular monitoring of hemoglobin level and coagulation using near patient devices if available (e.g., hemacue). FFP, platelets, transfusion, and cryoprecipitate may be necessary if coagulopathy develops.[1] Early liaison with hematology department for optimal and timely blood product replacement is of utmost importance.
- Perioperative monitoring of all vital parameter and recording of this parameter on a flow chart such as the modified obstetric early warning system charts.[1]
- There is a consensus that fibrinolytic inhibitors seldom if ever have a place in the management of obstetric hemorrhage.
- Post-operative management includes transfer to intensive care unit/high dependency unit.
- Anticipate coagulopathy and treat clinically until coagulation results are available.
- 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.

CONCLUSION

Obstetric hemorrhage is the most common cause of maternal morbidity worldwide and a leading contributor to the maternal mortality in developing nations. Major obstetric hemorrhage is managed by multidisciplinary approach. Clinicians should identify risk factors before and during labor so that the care may be optimized for high-risk women. Team responses that emphasize the accurate estimation of blood loss, early warning signs of shock and rapid response to blood loss and coagulopathy, a clear cut protocol are associated with less maternal morbidity. Urgent access to definitive care remains a major stumbling block in limited resource areas in the developing world. However, recent advances in prediction and assessment of blood loss, a better understanding of coagulation mechanisms, POC monitoring and the availability of minimum trading price 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. POC testing using viscoelastic methods such as ROTEM or TEG should be performed to guide the rapid identification of specific coagulation disorders and more accurate evaluation of coagulation. The role of tranexamic acid in obstetric hemorrhage is yet to be established. The guidance however will need to be updated as on-going clinical trials are updated.

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