

# Management of major obstetric haemorrhage

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**ABSTRACT**

One of the most important causes of maternal mortality is major obstetric haemorrhage. Major haemorrhage can occur in parturients either during the antepartum period, during delivery, or in the postpartum period. Early recognition and a multidisciplinary team approach in the management are the cornerstones of improving the outcome of such cases. The management consists of fluid resuscitation, administration of blood and blood products, conservative measures such as uterine cavity tamponade and sutures, and finally hysterectomy. Blood transfusion strategies have changed over the last decade with emphasis on use of fresh frozen plasma, platelets, and fibrinogen. Point-of-care testing for treating coagulopathies promptly and interventional radiological procedures have further revolutionized the management of such cases.

**Key words:** Management, obstetric anaesthesia, obstetric haemorrhage, parturient

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**INTRODUCTION**

Major obstetric haemorrhage (MOH) refers to any kind of excessive bleeding (usually related to pregnancy) in a parturient. This could be during pregnancy, child birth, or in the postpartum period. Bleeding typically occurs vaginally but rarely in the abdominal cavity. Available evidence suggests that severe obstetric haemorrhage is the most frequent cause of maternal mortality and morbidity all over the world. In the year 2015, a total of 8.7 million cases of MOH have been reported with 83,000 deaths attributed to it.<sup>[1]</sup> There is no universal consensus on the exact definition of massive obstetric haemorrhage; however, in most institutions it is defined as a blood loss of more than 1500 ml, or a fall in haemoglobin of more than 4 g/dl after acute blood loss in a parturient or need transfusion of four or more units of blood.<sup>[2]</sup> Definitions based on haemodynamic values are not useful for diagnosing MOH as the physiological changes associated with pregnancy mask the clinical presentation of hypovolaemia that leads to delay in recognition of blood loss and initiation of treatment. Further visual estimation of blood loss is often erroneous and underestimated due to contamination with amniotic fluid, or blood loss being concealed internally or hidden in the drapes. Thus, meticulous clinical observation and a high index of suspicion are

required to detect and eventually treat MOH early. Poor outcomes following MOH have been attributed to delayed treatment, unavailability of blood and blood products, inaccurate estimation of blood loss, absence of treatment protocols, poor communication among the treating teams, and inadequate organisational support.<sup>[3-5]</sup>

**MAIN CAUSES OF MASSIVE OBSTETRIC HAEMORRHAGE**

MOH can be classified into three types [Table 1] depending on the time of the pregnancy the bleeding occurs – antepartum period, during delivery, and in the postpartum period. Any bleeding from the genital tract after 24 weeks (some consider after 20 weeks) of gestation till the onset of labor is defined as antepartum haemorrhage (APH). It is associated with low birth weight babies and can lead to maternal and foetal morbidity and even mortality. Common

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**Table 1: Classification and main causes of obstetric haemorrhage**

Period	Causes
Antepartum	Placenta previa Placental abruption Coagulopathies Uterine rupture
During delivery	Lacerations during vaginal delivery
Postpartum	Uterine atony
Primary: Within 24 h of delivery	Placenta accrete Placenta increta
Secondary: 24 h to 6 weeks after delivery	Placenta percreta Retained placenta
Local causes: Can lead to bleeding any time during pregnancy	Cervical lesions: Dysplasia, cervicitis, polyps, ectropion, carcinoma, following sexual intercourse, clinical examination

**Table 2: Complications of antepartum haemorrhage**

Foetal complications: Hypoxaemia
Premature delivery
Growth retardation
Intrauterine death
Congenital malformations
Birth asphyxia
Maternal complications: Severity depends on pre-existing anaemia and amount of blood loss
Acute haemorrhagic shock and its sequelae
Acute kidney injury - renal tubular necrosis
Premature labor
Postpartum haemorrhage
Retained placenta
Complications associated with massive blood transfusion
Coagulopathy
Transfusion-related acute lung injury
Associated with higher caesarean section rates
Peripartum hysterectomy
Puerperal infections
Maternal death

complications of APH are enumerated in Table 2. Placenta previa and abruptio placenta are the most common causes of severe haemorrhage. The former is a condition where the placenta is abnormally implanted wholly or partially into the lower segment of the uterus near the uterine os. Placenta accreta, increta, and percreta refer to the conditions of abnormal implantation of the placenta in which there is an increasing degree of abnormal invasion of the placenta into the myometrium.<sup>[6]</sup>

Vaginal bleeding in placenta previa is classically painless and is usually seen in second or third trimester of pregnancy. Caesarean delivery is the procedure of choice in such cases with an increased risk of severe blood loss due to inadvertent incision through the placenta during surgery.

Abruptio placentae refers to the abnormal separation of the normally sited placenta; the bleeding occurs due to separation of the placental lining from the uterus. This bleeding may occur per vagina or may be concealed in the form of a retroplacental clot. Clinical features include abdominal pain, increased uterine tone, vaginal bleeding, and premature labor with signs of foetal distress. In case the bleeding is concealed, clinical presentation could be of haemorrhagic shock, acute renal failure, and foetal death.

Placenta accreta is a condition when the placenta is abnormally attached to the myometrium. Rarely blood vessels within the placenta or the umbilical cord traverse the foetal membranes overlying the lower uterine segment, and this condition is known as vasa previa.

One of the most devastating causes of APH is uterine rupture, and it is associated with a very high incidence of foetal and maternal mortality. Clinical features include abdominal pain, uterine tenderness, nonassuring foetal heart rate, and ultimately hypovolaemic shock, which could lead to maternal death. Maternal resuscitation along with emergency surgery is the only definitive treatment. Surgical procedures needed could vary from any of the following: foetal delivery with repair of the ruptured uterine wall, ligation of the uterine and internal iliac arteries, or hysterectomy.

Haemorrhage occurring in the antepartum period can result in postpartum haemorrhage (PPH) also. PPH results from any one or combination of four processes (remembered by the acronym of “4Ts”): atony of the uterus (tone), retained placenta (tissue), trauma of the genital tract (trauma), and coagulation disorders (thrombin). PPH can be primary (during the first 24 h) or secondary (occurring between 24 h to 6 weeks after child birth) and can be classified as minor (blood loss of 500–1000 ml) or major (blood loss more than 1000 ml).

Major PPH has been further divided into moderate (blood loss of about 1000–2000 ml) and severe (blood loss more than 2000 ml). Certain risk factors increase the possibility for developing PPH; the best treatment for the same is prevention, early recognition, and planned multidisciplinary management [Table 3]. Parturients usually are young and at term have a physiological increase in blood volume and this compensates for haemorrhage initially. As already mentioned, in addition, the blood loss may be concealed; therefore, the signs of obstetric haemorrhage may only be evident when the

circulating volume is very low. These signs are: cold periphery, pallor, tachycardia, and hypotension. It has been suggested that while treating massive haemorrhage the main therapeutic goals are: haemoglobin of more than 8 g/dl, a platelet count of more than  $75 \times 10^9/l$ , a prothrombin time of less than 1.5 times the control, activated prothrombin time of less than 1.5 times the control, and fibrinogen levels of more than 1.5–2.0 g/l.<sup>[7]</sup> The same goals should hold true for MOH also.

The treatment of parturients with MOH has two components: first, initial resuscitation and management of haemorrhage/hypovolaemic shock [Table 4], and the second includes identification and management of the underlying cause.

### MONITORING OF PATIENTS WITH MOH

Parturients with MOH show features of haemorrhagic shock and while being resuscitated can develop coagulopathy; this is caused by consumption of clotting factors and haemodilution due to transfusion of crystalloids and colloids. Other than haemodynamics and urine output, haemoglobin, haematocrit, and coagulation profile (platelet count, prothrombin time, activated partial thromboplastin time, and plasma fibrinogen levels) should be repeatedly measured and if point-of-care monitors are available, they should be utilized. There is enough evidence to recommend use of thromboelastography and rotational thromboelastometry for correction of coagulopathy effectively and quickly in parturients. Targeted goal-directed therapy with specific coagulation factors based on such viscoelastic methods are associated with less incidence of allogenic blood transfusions and thromboembolic events. Though these tests have been recommended by the obstetric and anaesthesia societies of UK, USA, and Europe, the initial cost of these monitors is a cause for concern.<sup>[8-10]</sup>

### USE OF BLOOD PRODUCTS IN TREATMENT OF MOH

Ideally cross-matched specific blood should be given to a parturient with MOH. In case of nonavailability of specific blood and a collapsing patient, O-negative blood can be used. Infusion of large volume of crystalloids and colloids can cause dilutional coagulopathy, metabolic acidosis, tissue oedema, and tissue hypoxia. Current advances in the management of haemorrhage in war-wounded army personnel have been extrapolated to MOH also.

**Table 3: Risk factors associated with and prevention of postpartum haemorrhage**

Risk factors for developing PPH	Prevention strategies for PPH
1. Uterine atony Over distension of uterus Polyhydramnios, multiple Gestation, macrosomia	1. Avoid prolonged labour
2. Prolonged third stage of labour	2. Atraumatic-assisted vaginal delivery
3. Use of tocolytic agents	3. Active management of third stage of labor
4. Abnormal placentation Placenta accreta Placenta increta Placenta percreta Uterine inversion	4. Early diagnosis of placenta previa and abnormal placentations - use MRI if possible
5. Placental abruption	5. Early clamping of umbilical cord, prophylactic judicious use of uterotonics
6. History of previous PPH	6. Planned multidisciplinary management of placenta previa and abnormal placentations
7. Obese parturient	
8. Previous caesarean delivery	
9. Instrumentation-assisted delivery	
10. Retained products of conception	
11. Puerperal sepsis	

MRI – Magnetic resonance imaging; PPH – Postpartum haemorrhage

**Table 4: Immediate plan for management of MOH**

- Recognize early warning signs of MOH and establish basic noninvasive monitoring
- Administer 100% oxygen by face mask
- In case the patient is pregnant nurse her in left lateral position, head down position
- Call for appropriate help/team
- Establish at least two large bore iv lines and/or a large central venous line
- Send blood for cross match, arrange for 4-6 units of blood, 4-6 units of fresh frozen plasma
- Start rapid infusion of warm saline, replace by red cells as soon as available
- Combination of crystalloids (1.5-2.0 l) and colloids 1-1.5 l can be given till blood is available
- Send blood for basic investigations - CBC, RFT, and coagulation parameters
- Use blood/fluid warmers and warming blankets - avoid hypothermia
- O Rh negative blood may be used if specific blood group is not available and the parturient is collapsing
- Aim is to maintain normovolemia, monitor hematocrit, urine output and haemodynamics
- Prompt treatment of coagulopathy, if present
- Early planning of surgical interventions, if conservative measures and pharmacologic measures fail
- Further management would depend upon the cause of the MOH

MOH – Major obstetric haemorrhage; CBC – Complete blood count; RFT – Renal Function Tests

The recommendations are to use packed cells and fresh frozen plasma in the ratio of 1:1 with early use of platelets.<sup>[11]</sup> A massive blood loss treatment protocol or massive blood transfusion protocol should be present in all delivery areas, keeping in view local expertise and facilities available. It is essential that all obstetric units should have immediate access to a “massive transfusion ‘pack’ consisting of four units of O-negative blood, four units of fresh frozen plasma, and one apheresis pack of platelets.” Ideally, fibrinogen concentrate and prothrombin complex concentrate should also be available with this pack.

Fibrinogen is an important component of the coagulation pathway. Its levels decrease very rapidly during haemorrhage, and in parturients the aim should be to keep fibrinogen levels more than 1.5–2 g/l. Fibrinogen concentrates have been used for correction of hypofibrinogenaemia in cases of PPH, although in many countries it is an off-label use. Fibrinogen is also present in fresh frozen plasma, cryoprecipitate, and in prothrombin concentrate. These should be used whenever required. Prothrombin complex concentrates that are available contain clotting factors II, IX, and X, and sometimes even VII. It has been used in parturients with PPH. They are associated with thrombotic events when used in nonobstetric population; therefore, they are still under investigation in parturients and their regular use is still not advocated in this subgroup of patients.

### CELL SALVAGE IN MOH

Cell salvage is an attractive option for managing obstetric haemorrhage, but there have been issues that make this modality controversial. These are possibilities of maternal alloimmunization, mixing of blood with amniotic fluid, and cell debris. More importantly, such a modality can be useful only if a 24-h service during emergency hours is available. Cell salvage can be very useful in parturients who land up in severe haemorrhage because of conditions such as placenta previa, accreta, abruption, and uterine rupture. It is also an attractive modality for parturients with severe anaemia, rare blood groups, and refusal of blood transfusion. A recent review analysing the use of cell salvage in obstetrics concluded that this modality has an “acceptable safety profile and should be considered when treating patients at high risk for obstetric haemorrhage and transfusion.”<sup>[12]</sup> The American College of Obstetricians and Gynecologists recommend consideration of this modality in

parturients, who have massive bleeding due to PPH.<sup>[13]</sup> American Society of Anesthesiologists endorses its use in PPH when banked blood is not available or in cases where patient refuses banked blood.<sup>[14]</sup>

### USE OF CRYSTALLOIDS AND COLLOIDS IN MOH

Infusion of large volumes of cold crystalloids and colloids in parturients with MOH can cause coagulopathy and hypothermia and that should be prevented.

#### Tranexamic acid

Tranexamic acid is a synthetic analog of lysine. It has antifibrinolytic activity and World Health Organization recommends early use of intravenous tranexamic acid in PPH regardless of the cause. Tranexamic acid should be administered within 3 h following vaginal birth or caesarean section. Delayed use of tranexamic acid reduces the benefit with no benefit seen after 3 h.<sup>[15]</sup>

Calcium concentrations should be measured and corrected in the event of massive transfusion. Maintenance of calcium concentration in such cases prevents coagulopathy and as a “thumb rule” 10 ml of 10% calcium chloride after every four units of packed red cells should be administered. Complications associated with massive blood transfusion that should be looked for and treated adequately are citrate toxicity, hyperkalaemia, hypothermia, hypomagnesaemia, and acidosis.

#### Recombinant activated factor VII (rFVIIa)

There are numerous reports of successful management of parturients with MOH with rFVIIa. It is expensive and its use in obstetrics is still “off-label.” Parturients with MOH can develop disseminated intravascular coagulation. Delayed correction of these coagulation problems is associated with high morbidity and mortality. Recombinant factor VIIa promotes homeostasis at the site of the injury. It either acts via a tissue factor-dependent pathway or via a tissue factor independent pathway. Ultimately, factors IX and X are activated to form thrombin. The small amounts of thrombin activate factors V, VIII, XI and platelets, which lead to further thrombin and fibrin generation. Earlier recommendations suggest that it should be used after failure of conventional methods, and should be administered only when the patients’ hematocrit is adequate, platelet count is greater than  $50 \times 10^9/l$ , fibrinogen levels are greater than 1 g/l, and there is no acidosis or hypothermia.<sup>[16]</sup> Latest guidelines, however, do not recommend the routine use of rFVIIa in the management of major PPH.<sup>[17]</sup>



## SURGICAL INTERVENTIONS FOR MOH

Surgical interventions are often required to treat MOH. The intervention chosen would depend on the cause of the bleeding, experience of the treating team, and the hospital setup where the parturient is being treated. Commonly performed interventions are manual removal of the placenta, uterine packing, intrauterine balloon tamponade (Bakri/Rusch balloon, Foley's/condom catheter, Sengstaken-Blakemore tube), uterine compression sutures (B lynch sutures), pelvic vessel ligations (internal iliac, uterine, hypogastric, or ovarian arteries), uterine and hypogastric artery ligation, interventional radiology (intraarterial balloon occlusion and arterial embolization), and hysterectomy.<sup>[18]</sup>

## INTERVENTION RADIOLOGY IN MOH

Intervention radiological techniques are being utilized frequently for anticipated or unanticipated cases of MOH.<sup>[19]</sup> Catheters with balloons at the distal end are placed in the uterine or iliac arteries; the balloons may be prophylactically inflated during the surgical procedure (after delivery of the foetus in case of caesarean sections), or whenever needed.

After the bleeding is controlled the balloons can be deflated, the catheters are removed, and alternatively embolization (with gel foam, glue, coils, or other particles) of the arteries could be carried out if the bleeding is not controlled. Such procedures can avoid caesarean hysterectomy, and preserve fertility; however, they need to be carried out in the radiology room which makes monitoring and management of bleeding difficult. Nonavailability of an intervention radiologist during emergency hours is another drawback that has limited the routine use of this procedure. Common complications are local haematoma formation, infection, formation of a false aneurysm, and rarely uterine necrosis.<sup>[20,21]</sup>

## MEDICAL MANAGEMENT – UTEROTONIC DRUGS

Many drugs are available to treat bleeding because of uterine atony. The most commonly used are: oxytocin, ergotamine, methyl ergot, and 15-methyl prostaglandin F2 $\alpha$  [Table 5]. Oxytocin is used prophylactically in the third stage of labor and is mostly very effective. It causes vascular smooth muscle relaxation that can lead to hypotension and reflex tachycardia. It is given as a slow bolus followed by an infusion. Ergometrine is a second

Table 5: Uterotonic drugs

Drug	Salient features
Oxytocin (syntocinon)	Slow IV bolus (5 units), followed by infusion not more than 10 units per hour Causes vascular smooth muscle relaxation, hypotension, reflex tachycardia Onset: 2-3 min, duration: 30 min
Ergometrine	Slow IV or IM, dose: 500 mcg Causes nausea, vomiting, and headache, precipitates severe hypertension, contraindicated in eclampsia and hypertensive disorders
Prostaglandin F2 $\alpha$ (carboprost)	IM dose 0.25 mg can be repeated to a maximum of 2 mg Also administered directly into the myometrium (off label use) Can cause bronchospasm
Prostaglandin E1 analogs (misoprostol)	Can be given orally, rectally, or sublingually, dose: 800 mcg Common side effects: Shivering and transient rise in temperature

IV – Intravenous; IM – Intramuscularly

line uterotonic medication and acts on uterine and other smooth muscle. It causes severe nausea and vomiting and increase in blood pressure; hence it is contraindicated in preeclampsia and other hypertensive conditions.

## ANAESTHETIC MANAGEMENT OF PATIENTS WITH MOH

If parturients with MOH require anaesthetics for any surgical intervention, then the main concerns would be: need for rapid efficient resuscitation, ensuring wellbeing of the unborn baby and the mother, though the mother would gain precedence. Adequate noninvasive and invasive haemodynamic monitoring is of paramount importance; the latter should be instituted early. In a haemodynamically unstable pregnant woman, general anaesthesia is the preferred technique, though problems with pregnant airway and foetal effects of the anaesthetics should always be considered. Regional anaesthesia is always preferred in parturients, however, the presence of coagulopathy may be a contraindication for its use. Ketamine and etomidate may be preferred induction agents in unstable patients. Intravenous fluid and blood replacement should follow principles that have been mentioned above. It is imperative to monitor such parturients after surgery in intensive care till they are haemodynamically stable.

## SUMMARY

Delay in recognition and management of obstetric haemorrhage can result in severe maternal morbidity and

preventable maternal mortality. Prompt resuscitation, identification of the cause, and subsequent treatment are essential for improving the outcomes.

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#### Conflicts of interest

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