THE MANAGEMENT OF MASSIVE POSTPARTUM HAEMORRAHAGE IN A TERTIARY HOSPITAL IN NIGERIA. A REPORT OF TWO CASES

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ABSTRACT

Major obstetric haemorrhage still remains one of the leading causes of maternal mortality in the west African subregion and worldwide and hence close monitoring of the vital signs of patients will ensure early detection and prompt management. Massive Obstetric haemorrhage when detected early and adequately managed will prevent severe maternal morbidity and mortality. We report the anaesthetic challenges of two cases of massive obstetric haemorrhage that were successfully managed.

INTRODUCTION

Haemorrhage is a leading cause of maternal mortality. It is the underlying cause in at least 25% of maternal deaths in the developing world.[1] Blood loss following uterine incision to delivery of the baby and placenta can be a challenge for the managing team. This period is characterised by large amounts of blood loss and hence prompt intervention and resuscitation should be the goal to ensure haemodynamic stability. Major obstetric haemorrhage may be defined as blood loss >1500 ml; a decrease in haemoglobin of more than 4 g/dl; or an acute transfusion requirement of more than 4 units of packed red blood cells in 6hours are suggested criteria. [2] Definitions based on haemodynamic deterioration are unhelpful as maternal physiology often allows compensation until haemorrhage is significant. Careful clinical observation and a high index of suspicion are required to detect bleeding early.

We present a case of sudden cardiovascular collapse resulting in loss of consciousness following placenta separation and another case of a major haemorrhage that was detected in the post anaesthetic care unit.

CASE ONE

A 36 year old booked G4P2+1 woman weighing 78kg, with a height of 1.67m and a BMI of 28 was scheduled for an elective caesarean section on account of two previous CS at term. The Indications for the previous caesarean sections was breech presenting foetus at term. The preoperative review by the duty anaesthetist revealed a fit young woman without any intercurrent medical illnesses. There was also no history of previous drug allergies or blood transfusion. The systemic review did not reveal any abnormality. Ultrasound findings was in keeping with a placenta previa type II.

Examination findings revealed a woman who was not pale, afebrile, anicteric. Preoperative vital signs were within normal limits with pulse

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rate of 94bpm, Blood pressure 130/80mmHg. Heart sounds I and II were normal and chest was clear on auscultation. Airway assessment was in keeping with Mallampatti II. The patient was classified ASA I(American Society of Anesthesiologists) physical health status. Laboratory investigations which included a full blood count, electrolytes and urea, urinalysis and random blood sugar were all within normal limits. The PCV was 31%. She was premedicated with ranitidine 50mg and methochlopramide 10mg intravenously.

Multiparameter monitor was attached and baseline vital signs were obtained. Blood pressure was 132/94mmHg. Pulse rate was 96bpm while the oxygen saturation was 99% in room air. Electrocardiography showed sinus rhythm. Patient was preloaded with 750ml of normal saline after establishing an intravenous line Subarachnoid block was established at the L3/4 interspace in the sitting position using aseptic technique with a 25G pencil point needle. With clear reflux of cerebrospinal fluid, a combination of 2.0mls of bupivacaine and 10mg of pethidine was deposited. A block height of T6 was obtained. A live female neonate was delivered with APGAR score of 8 and 9 in 1and 5 minutes respectively.

As soon as the Placenta was removed there was massive blood loss which resulted in severe cardiovascular collapse and loss of consciousness. There was a sudden drop in blood pressure to 54/40mmHg and a pulse rate of accompanied by loss of consciousness. The woman's trachea was immediately intubated with a size 7mm cuffed endotracheal tube and connected via the breathing circuits to the anaesthetic machine and then ventilated with carbondioxide absorber. Pancuronium 6mg was used as muscle paralysis. The rate of administration of Intravenous fluids was increased and blood transfusion was commenced. Oxytocin was changed to ergormetrine and ephedrine 30mg was given in aliquots till the blood pressure rose to 100/60mmHg. A decision was taken to do a subtotal hysterectomy which lasted for 2hours and patient's vital signs were stable throughout the surgery. Calcium gluconate 10mg, hydrocortisone 200mg, and fresh frozen plasma, platelet concentrates and tranexamic acid 500mg were also given as recommended by the haematologists. The estimated blood loss was 6litres.

At the end of surgery, residual neuromuscular block was antagonised and tracheal extubated following signs of adequate reversal. The vital signs were stable with BP of 120/70mmHg and pulse rate of 87bpm and urine output was 300ml. She was then transferred to the recovery room with oxygen via nasal prongs.

One hour after the procedure, hypotension was observed. Examination revealed frank bleeding from the vaginal and hence a decision was taken for a total hysterectomy. The procedure lasted for 3hours. Estimated blood loss was 4litres. She had a total of 4 units of fresh whole blood, 1 unit of platelet concentrate and 4 units of fresh frozen plasma.

A total of 10 units of blood was transfused.

She was then transferred to the intensive care unit for close monitoring where she made significant improvement. In the ICU, she received an additional 3 units of fresh whole blood after post transfusion revealed a haemoglobin concentration of 7g and PCV was 21%. She was closely monitored throughout the time in the ICU and the PCV appreciated to 27%. She was in the ICU for 36hours from where she was then transferred to the ward from where she

was discharged home after 2wks in a satisfactory condition.

CASE TWO:

This was a case of a booked 30yr old G4p3+1(2 alive) RVD positive patient who had emergency caesarean section on account of obstructed labour.The past medical history was not significant except being diagnosed for retroviral disease 6years ago prior to presentation and was stable on HAART. Physical examination revealed a young woman who was not pale or jaundiced or dehydrated. The pulse rate was 75bpm with a blood pressure was 110/70mmHg. Airway assessment was in keeping with Mallampati II. Packed cell volume was 32% while urinalysis was normal. The patient was classed as ASA II using the American Society of Anaesthesiologist physical health status classification. An intravenous access was established with a 16G cannula as soon as patient arrived in the theatre and baseline vital signs were obtained and recorded. Pulse rate was 68bpm while the blood pressure was 120/70mmHg. SPO_2 was 98% in room air. She was premedicated with IV Ranitidine 50mg and metoclopramide 10mg.

Preloading was achieved with 1L of normal saline. Observing strict asepsis, Subarachnoid block was established in the sitting position with 2.2ml of heavy bupivacaine under aseptic technique. Surgery was commenced and live male neonate was extracted 10minutes later with an APGAR score of 8 in the first minute and 9 in 5 minutes. Surgery lasted for 130minutes. Estimated blood loss was 500ml. she was then transported to the recovery room for close monitoring.

It was, however, noted that 5hrs after surgery that the vital signs of the patient became unstable. She developed hypotension and tachycardia, blood pressure 66/40mmHg and pulse was 136bpm. Abdominal parecentesis yielded 20mls of serosaguinous blood which was free flowing.

She was taken back to the theatre where resuscitation continued. She was then taken for re-exploration under GA. Rapid sequence Induction was achieved after preoxygenation with ketamine 75mg and suxamethonium 100mg to aid tracheal intubation with a 7.5mmHg cuffed endotracheal tube. After correct placement was confirmed by auscultation, she was then connected via the circle circuit to the anaestheic machine and ventilated with carbondioxide absorber. She was then ventilated to normocabia with carbondioxide absorber. Maintenance was achieved with midazolam and pancuronium with IPPV at 4l of oxygen. Analgesia achieved with intravenous paracetamol 600mg and tramadol 100mg.

Exploratory laparatomy findings was haemoperitonium of 3.5l. Estimated blood loss was 4.5litres. She subsequently had hysterectomy.she was given 4l of crystalloid and 3units of blood Intraop and had another 2units in the immediate post operative period. urine output was 500ml and adequate. Her vital signs were stable throughout the intraoperative period.

Surgery lasted 2hour 30mins. Residual muscle paralysisi was reversed with glycopyrrolate 0.4mg and neostigmine 2.5mg. when the patient resumed spontaneous respiration and became conscious, the trachea was extubated.

The Immediate post operative finding was satisfactory. In the recovery room, she was managed with oxygen via nasal prongs and analgesics. Analgesia was achieved with

intravenous paracetamol 600mg and pentazocin 30mg. When the Postop vital signs were stable, she was then transferred to the ward. She was in the ward for one week. The post transfusion pcv was 28% and she was subsequently discharged home.

DISCUSSION

Postpartum Haemorrhage (PPH) is commonly defined as a blood loss of 500 ml or more within 24 hours after vaginal birth or greater than 1000ml following caesarean section[2] Massive obstetric haemorrhage is defined as blood loss from the uterus or genital tract >1500ml, a decrease in haemaglobin of > 4 g/dl or acute transfusion of > 4 units blood.[2] The first patient had a total blood loss of 10L and had 11 units of blood within a 24hr period and hence met the criteria for massive obstetric haemorrhage. The second patient had a blood loss of 3.5l within a 4hr period.

Postpartum haemorrhage affects approximately 2% of all women who give birth: it is associated not only with nearly one quarter of all maternal deaths globally but is also the leading cause of maternal mortality in most low-income countries.[3,4] World Health Organization reported that Nigeria has one of the highest rates of maternal mortality in the developing World.[5] with a maternal mortality rate at 800 per 100,000 live births (National Planning Commission, 2001). Haemorrhage accounted for the major cause of death.

Massive PPH can be caused by uterine atony, genital lacerations, retained placenta and uterine inversions. Massive PPH may lead to serious consequences such as severe maternal morbidity and long-term disability as well as to a number of other severe maternal conditions generally associated with more substantial blood loss, including shock and organ dysfunction. Both patients had severe cardiovascular collapse which required prompt resuscitation(ventilation, blood transfusion and the use of vasopressors) to prevent long term disability.Case 1 developed unconsciousness as a result of severe cardiovascular collapse.

In a study by Shevell et al, advanced maternal age, previous Caesarean section and presence of sponge-like findings in the cervix were risk factors for massive bleeding during Cesarean section in cases of placenta praevia, regardless of whether placental adherence is present. Placental location on the scar of a previous Caesarean section and lack of a clear zone are risk factors for placental adherence.[6] The Risk factors identified in the first patient for the development of major obstetric haemorrhage were advanced maternal age, 2 previous caesarean sections and placenta adherence. The risk factor in the second patient was slipped ligature.

Anaesthetic management involved the use of 2 large bore IV canula, grouping and crossmatch of 2 units of blood, FFP, volume preloading and availability of drugs for resuscitation. Though these were in place in the first patient, the sudden blood loss following placenta separation was so massive that there was a sudden loss of consciousness. This loss of autonomic outflow due to severe cerebral hypoxia, led to loss of consciousness in this patient. Hence regional technique was converted to general anaesthesia with airway management. When mean arterial pressure falls below 60 mmHg, cerebral perfusion decreases because the pressure is below the autoregulatory range. Cerebral ischemia produces very intense sympathetic discharge that is several-fold greater than the maximal sympathetic activation caused by the

baroreceptor reflex which is a compensatory mechanism in haemorrhagic shock.

The degree of placenta adherence was not anticipated in this patient and hence there was. no prior plan for its management. The second patient was monitored with a multiparameter patient's monitor in the Post Anaesthetic Care Unit. The continuous monitoring contributed significantly to the early recognition of hypotension.

Management of obstetric haemorrhage utilizes a multidisciplinary approach usually consultants in such fields. This includes the anaesthetists, obstetricians, haematologists, midwives and supportive staff. Prompt resuscitative measures and the cause-directed management is the mainstay of treatment for PPH, and includes fluid, whole blood and blood products administration as indicated. In addition, the use of uterotonics, uterine massage, repair of lacerations, removal of retained products of conception and intrauterine balloon tamponade by the radiologist. If these measures are unsuccessful in controlling the bleeding, the next step is usually surgical either conservative or aggressive like uterine bracing, suture application, internal iliac artery ligation, or hysterectomy [7] the first patient had right internal artery ligation but bleeding persisted and hence hysterectomy was done for her

The management of both patients involved the consultation with the haematologist that provided the fresh frozen plasma and platelet concentrates. The use of interventional radiology is not yet developed in our centre and hence was not utilized. Uterotonics such as oxytocin was changed to ergormetrine due to hypotension which was observed in both patients. The switch to ergometrine and misoprostol is usually influenced by the amount of blood loss which may be grossly underestimated due to the presence of liquor. [8,9]

Tranexamic acid 500mg was also given to both patients and this had been found beneficial. In cases of massive haemorrhage unsuccessfully treated with surgical measures and its combination with recombinant factor VIIa, and local vasopressin have been reported for postpartum haemorrhage.[10] Recombinant factor VIIa and vasopressin are not available in our centre.

Both patients had hysterectomy. Hysterectomy is often the definitive treatment for PPH with the most common indications being uterine atony and placenta accreta. [11]The first patient initially had a subtotal hysterectomy initially then total hysterectomy in the second surgery as the placenta was also noted to be morbidly adhered to the cervix. A markedly increased risk of placenta accreta is associated with an increasing number of prior Caesarean deliveries with and without placenta praevia. [12]Antenatal diagnosis of placenta accreta can be made by ultrasound or MRI and facilitates effective planning.[13] The diagnosis of plancenta accreta was not made in this patient as only a placenta praevia type II was diagnosed by ultrasound and hence consent for hysterectomy had to be given by the spouse as patient was already under general anaesthesia. Prior diagnosis of the degree of placenta adherence to the uterine wall would have necessitated early planning of the choice of anaesthesia and surgical technique in this patient. General anaesthesia would have been the choice of anaesthesia abinitio in the first patient and planned hysterectomy would have prevented the occurrence of this episode of massive blood loss leading to deterioration in her conscious level. Early recognition by

experienced and skilled manpower saved the situation in both patients. Both patients had blood transfusion immediately. This is a challenge in our centre where blood availability is unreliable. Also Consultants in all the fields of specialty concerned were on ground to participate and give expert opinion.

Patients are often monitored in the recovery room of the obstetric theatre for a minimum of 4hrs in our centre in order to detect early postoperative complications. The deterioriation in the vital signs was promptly detected in case two by the anaesthetic team that resumed duty in the morning, about 4hrs after the surgery.

It has also been observed that massive obstetric haemorrhage may precipitate disseminated intravascular coagulopathy (DIC) in some patients as part of the shock cascade. Both patients, however, did not develop DIC. Disseminated intravascular coagulopathy can result from massive haemorrhage due to prolonged shock with catastrophic outcomes.[14] This is further compounded by the hypercoagulable state in pregnancy. The early transfusion of blood and blood products prevented the development of DIC in these patients.

CONCLUSION

We recommend the need for use of additional tools for investigation of patients with previous uterine scars and the provision of a mini blood bank in all obstetric units and increased time of monitoring in the post anaesthetic recovery room especially for patients at risk.

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