

Major Postpartum Blood Loss and Massive Transfusion in a Tertiary Hospital in North-Central, Nigeria: Case Report

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Received date: 18 Feb, 2015; Accepted date: 17 March, 2015; Published date: 19 March, 2015

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Abstract

Background: Postpartum haemorrhage (PPH) which may sometimes be massive is a common cause of maternal and neonatal mortality and morbidity all over the world, and is said to be increasing. It is the most common cause of maternal death in our environment. The availability of blood and safe blood transfusion practices in many countries of Sub-Saharan Africa is lacking

Case: We present two cases of major PPH following caesarean section resulting in massive transfusion at the Obstetrics Department of Benue State University Teaching Hospital, Makurdi, North Central Nigeria. There was observed massive blood loss in one, with sepsis and continuous bleeding in the second. They were effectively treated with massive fresh whole blood transfusion, hysterectomy (case1) and conservative management (case 2).

Conclusion: We report the problems associated with the diagnosis of PPH especially by visual assessment and the availability of blood in our environment. We further, highlight the need for the identification of early warning signs of potential clients, towards early diagnosis and recourse to hysterectomy for the prevention and reduction of PPH, so as to limit the hazards of massive blood loss and transfusion especially in Nigeria.

Keywords: Postpartum haemorrhage; Massive blood transfusion; Hysterectomy

Introduction

Bleeding from the female genital track after birth referred to as postpartum haemorrhage (PPH) may be primary or secondary, and is a major cause of maternal morbidity and mortality worldwide. The highest rate is seen in the developing world as a result of dearth of facilities, trained personnel and poverty. Major blood loss in obstetrics practice is a challenge to the doctor, the haematologist and blood transfusion services, sometimes causing avoidable friction between the obstetricians, those supplying blood and the laboratory leading to unnecessary waste of time and resources with possible adverse outcome for the patient and her survival in many clinical settings in developing countries of Sub-Saharan Africa.

According to World Health Organization (WHO), obstetrics haemorrhage causes 127,000 deaths annually worldwide and is the leading cause of maternal mortality [1]. In developed countries, haemorrhage causes 13% of maternal deaths and is the third leading cause of maternal death, with higher rate in other countries [2]. Death rate from PPH has increased from approximately 2% in 1994 to 3% in 2006 in the United States and from 4% to 5% in Canada during that time [3]. The major known direct causes of PPH may be classified into four namely; uterine atony is the most common cause of PPH; genital tract trauma ranging from lacerations to uterine rupture, inversion and hematomas; the presence of intrauterine remnants of conception and placenta invasion and hematological abnormalities resulting in coagulopathy (the diagnosis should be suspected after delivery when bleeding continues despite a well contracted uterus). In Africa, due to

increased prevalence of risk factors such as grand-multiparity, no routine use of prophylaxis against obstetric haemorrhage, coupled with poorly developed obstetrics services, obstetrics haemorrhage is responsible for 30% of the total maternal deaths [3]. Other factors such as lack of measures for drug and surgical management of atony are all well known.

Massive blood loss is arbitrarily defined as the loss of one blood volume within a 24 hours period [4]. On the other hand, major obstetric haemorrhage is defined as blood loss >2000 ml or rate of blood loss of 150 mls/min, or 50% blood volume loss within 3 hrs. It can also result in a decrease in Hb >4 g/dl, or acute transfusion requirement >4 units. A major obstetric haemorrhage that triggers the 'massive obstetric haemorrhage' protocol is defined as blood loss that is uncontrolled and ongoing with the rate of loss of 150 mls/minute [5,6]. Primary PPH is the most common form of major obstetric haemorrhage. The traditional definition of primary PPH is the loss of 500 ml or more of blood from the genital tract within 24 hours of the birth of a baby [7]. PPH can be minor (500-1000 ml) or major (more than 1000 ml). Major could be divided into moderate (1000-2000 ml) or severe (more than 2000 ml) [8]. On the other hand, secondary PPH is defined as abnormal or excessive bleeding from the birth canal between 24 hours and 12 weeks postnatal [9]. Haemorrhage emerges as the major cause of severe maternal morbidity in almost all 'near miss' audits in both developed and developing countries [10].

The objective of this study is to help guide health care professionals in the recognition of major obstetric haemorrhage and the challenges in management towards its early recognition and prompt and effective treatment if shock and its consequences are to be prevented in similar settings.

Case 1

A 35-years old, booked, P₃⁺¹ (3A) woman with 2 previous caesarean sections presented to the emergency Obstetrics unit of Benue State University Teaching Hospital (BSUTH), Makurdi, at 37 weeks of gestation with vagina bleeding. She booked index pregnancy at 22 weeks gestation, and had four [4] regular visits but was unable to do her booking ultrasound scan due to financial reasons. She was diagnosed with major degree placenta praevia in a late ultrasound scan at 33 weeks of gestation. The ultrasound scan details showed a 33 weeks viable, singleton intrauterine fetus, with the placenta crossing over the internal cervical os (placenta praevia type IV), and the fetus weighed 1.86 kgs. She was counselled on the nature of the condition and offered admission but she declined. She was then advised to come to the hospital any time she noticed vagina bleeding or contractions. Her booking blood pressure was 110/70 mmHg, packed cell volume (PCV) 36%, and urinalysis was essentially normal. Her blood group was B Rh+ve, genotype AA. Retroviral screening test was not reactive and results of HBcAg, HCV, and VDRL tests were all negative. Her booking weight and height were 80 kg and 1.62 m respectively.

She presented with five [5] hours history of painless bleeding per vagina at 37⁺⁵ weeks gestation. The bleeding was spontaneous with no preceding trauma to her abdomen or any other part of her body. There was associated passage of blood clots but no dizziness, fainting or loss of consciousness. There was no associated abdominal pain or labour pains. The bleeding was first noticed while defecating some days earlier and persisted over a few days as spotting per vaginam before the present episode which was said to have increased in volume. She had two previous deliveries which were all by caesarean section, in 1998 and 2002. The first caesarean section was due to placenta praevia, and she was transfused one unit of blood and was delivered of a live male baby that weighed 3.2 kg while the second was a live female baby with birth weight of 3 kg. She had a voluntary termination of pregnancy in her fifth week of gestation in 2003 with no adverse sequelae. She was not a known diabetic or hypertensive and had no known drug allergy. She is the only wife to her husband who is a civil servant. She is a health worker with a primary health care centre in one of the Local Government in Benue State. She had no family history of diabetes mellitus, hypertension, or sickle cell disease. Her mother and elder sister had a set of twins each.

Physical examination at presentation revealed a young woman who was not ill looking, pale, or febrile. She had mild pitting pedal edema and the temperature was 37.1°C. Her pulse rate was 92 beats per minute, regular and full volume. The Blood pressure was 110/80 mmHg and the 1st and 2nd heart sounds were heard. The respiratory rate was 20 cycles per minute and the chest was clinically clear. The abdomen was uniformly enlarged and the symphysio-fundal height was 35 cm; compatible with an Expected gestational age of 37 weeks. It was a live singleton fetus presenting breech. There was no uterine contraction in 10 minutes. Vagina examination showed a blood stained Perineal pad on inspection. A diagnosis of antepartum Haemorrhage due to major degree placenta praevia (type IV) was made. She was admitted into the antenatal ward and counselled for elective caesarean section the following day. The tests whose results appear below were done:-

Urgent PCV (35%), Urgent repeat obstetric ultrasound scan (placenta praevia type IV), Urinalysis (Normal), four [4] pints of blood were grouped and cross-matched and she was asked to keep a fetal kick chart while electronic monitoring of the fetus and uterine activity was initiated at 4 hourly intervals. She was also advised on strict bed

rest with an intravenous line of dextro-saline one litre 8 hourly for 24 hours using a wide bore needle (size 16G) set up. The next morning of the scheduled surgery, she had elective lower segment caesarean section (ELSCS) with the following findings:

There was a normal gravid uterus, with well-formed lower uterine segment. Moderate fibrous adhesions were seen between the uterus, bladder and the anterior abdominal wall. A live male baby in transverse lie weighing 2.8 kg with Apgar scores of 6 and 8 in first and fifth minutes respectively was delivered. A partially morbidly adherent placenta (accreta) was overlying the lower segment anteriorly and completely covered the cervix (praevia type IV). There was an estimated blood loss (EBL) of 1.7 litres.

The surgery was well tolerated with the placenta delivered by gentle separation using the ulna border of the surgeon's right hand in a see-saw fashion. Oxytocin was given intravenously 10 i.u.stat and 40 units to run in drip at 125 ml per hour for the next 4 hours. One unit of blood was transfused intra-operatively. About an hour after surgery, she was noticed to be bleeding (at the recovery room) with blood soaked perineal pad and bed sheets. She was immediately evaluated by the surgeon with finding of uterine bleeding from atony. While rubbing up a contraction, there was administration of another bolus injection of 10 i.u intravenous oxytocin with a second unit of blood set up. About 1-hour later, she went into hypovolaemic shock while on her second pint of blood. The pulse (radial) was non-recordable while BP was 60/40 mmHg; the uterus was then packed with sterile gauze while resuscitation continued with arrangements being finalized to return to the theatre for detailed examination with the husband counselled for possible sub-total hysterectomy. Meanwhile a second IVF line of 0.9% normal saline 1 L was setup and blood transfusion continued at a faster rate. She was also worn anti-shock garment (Non-pneumatic).

About 4 hours post caesarean section, she was stable and the husband gave informed consent and she had a sub-total hysterectomy after which she was taken to the intensive care unit (ICU) for close monitoring. She was on her 4th pint of fresh blood by the end of the sub-total hysterectomy. By the first day post sub-total hysterectomy, she had another hypovolaemic shock with no recordable pulse (radial) and BP 50/30 mmHg, respiratory rate 26 cycles per minute with dyspnea and urine output was only 300 ml. She was again resuscitated with oxygen by face mask at 5 L/min, IV dexamethasone 8 mg start, IV frusemide 120 mg start and continued on blood transfusion (7th pint) and IV normal saline alternating with 5% dextrose in water. She had received injection calcium gluconate after the fourth pint of blood and the haematologist was invited to review her in view of ongoing massive blood loss and transfusion. The Physician was also invited to review her. Results of clotting profile were deranged as follows: PT=30 secs [11-16] seconds. Control=14 secs, PTTK=54 seconds (30-50) seconds. Control=37 secs.

By the 2nd day post subtotal hysterectomy, she again had exploratory laparotomy after resuscitation due to haemoperitonium because of disseminated intravascular coagulation (DIC) when she was noticed to be severely pale with a distending abdomen. Intra-operatively, 2.5 litres of blood and clots were evacuated from the abdomino-pelvic cavity; there was no active bleeding from the vagina stump and angles. By the end of the exploratory laparotomy, she was on the 15th pint of blood and received two additional units in the ICU where she was nurse for three days and the third in the post natal ward before discharge from the hospital following satisfactory clinical

progress. Post transfusion PCV (after 18 units of blood) at discharge was 31%.

She was seen in the post natal clinic one week after discharge and two weeks thereafter. She defaulted on her postnatal clinic review three weeks later.

Case 2

20 years old P₁⁺⁰ (not alive) woman of Christian origin who lived with her husband at Adikpo in Kwande Local Government Area of Benue State was referred to us after an emergency caesarean section 3 weeks prior to presentation at a missionary hospital in the same town due to suspected big body at Term. She was delivered of a live male neonate with a poor 5 minute Apgar score who eventually died 4 hours later. She had been transfused with one pint of blood after the operation and transfused three more pints the next day before she was referred to another health facility in a nearby Local Government headquarter (Konshisha) about 30 kilometres away due to abdominal distension and 'poor recovery' after surgery.

She was finally referred to us from this hospital on the seventh day after the caesarean section with complaints of generalized body weakness, dyspnea and chest pain, abdominal pain and distension, fever and vagina bleeding. General examination revealed a young woman, in moderate respiratory distress (respiratory rate 20 cycles/minute), febrile (38°C), not pale, anicteric, acyanosed, not dehydrated and with no pedal oedema. The abdomen was uniformly distended and moved with respiration. She had a fresh Sub-umbilical longitudinal midline scar. There was wound dehiscence just below the umbilicus (3 cm). There was generalized tenderness and guarding with signs of fluid collection and hypoactive bowel sounds. On vagina examination, the external genitalia were healthy with minimal lochia. Speculum and sterile digital examination showed a closed cervical os, with no active bleeding. The pouch of Douglas was full but the pelvis could not be examined adequately due to tenderness. Examining finger was stained with frank blood.

She was admitted in the postnatal ward as a case of puerperal sepsis due to haemoperitonum and peritonitis to rule out abscess and placed on broad spectrum intravenous antibiotics and metronidazole, with intravenous fluid resuscitation for the first 48 hours before laparotomy. Details of her condition and the mode of treatment were explained to her and her husband. The surgical team on call was invited to review the patient and an assessment of puerperal sepsis with haemoperitonum to rule out abscess was made with an advice to continue the outlined management. She had urgent PCV (30%), abdominopelvic USS showed intra-abdominal collection of abscess/blood, full blood count (FBC) showed leucocytosis WBC 20.3×10⁶ cells/l, platelet count 110,000×10⁶/l. The recovery was slow and on the 3rd day she became restless with difficulty in breathing, pale with increasing abdominal distention. The PCV was 24% and 3 units of blood were grouped and cross-matched with 2 units transfused. Retroviral screen was negative.

A day prior to laparotomy, IV Rocephin was changed to IV Augmentin 1.2 g 12 hourly and continued postoperatively with metronidazole 500 mg 8 hourly for 72 hours. She had exploratory laparotomy the next day with finding of 2.3 litres of intraperitoneal abscess and multiple areas of fibrinoid adhesions in the abdominal cavity with abrasions on many areas of the proximal and mid-jejunum. There was however, no noticeable breach in the integrity of the lumen. A sample of pus was taken for microscopy, culture and antibiotic

sensitivity and copious peritoneal lavage was done with 3 L of warm saline and metronidazole after which a pelvic drain was inserted through a stab wound and the abdominal wound sutured by mass wound closure with nylon-1. She was continued on IV antibiotics, analgesics and intravenous fluids and a third unit of blood transfused (7th) on the ward. She was commenced on graded oral sips and encouraged to ambulate on the third postoperative day with the fever completely subsided. The abdominal drain was removed on the second (day fifth day post operative) after it had stopped draining (it drained 250 ml of purulent discharge).

She developed fever (38.8°C), abdominal pain and tenderness again two days later and repeat ultrasonographic examination of the abdomen and pelvis showed significant intraperitoneal fluid collection although antibiotic culture report showed staphylococcus aureus bacteria growth which was highly sensitive to augmentin. A second exploratory laparotomy on the 9th day yielded 350 ml of purulent discharge from the lesser sac with noticeable erosion of medial lobe of the liver by the abscess cavity. Abdominal and pelvic drains were then inserted after lavage of the abdomen as above. Post operatively, she was admitted into the intensive care unit (ICU) where she received 4 more units of fresh whole blood (making a total of 11 units) based on PCV of 18% on the second day. The blood clotting profile before transfusion was [Bed side clotting time was 18 minutes, PT=20 seconds [11-16] seconds. Control=14 secs, PTTK=54 seconds (30-50) seconds. Control=37 seconds] slightly deranged. She was given calcium gluconate 10 ml IV injection slowly and remained stable with steady recovery and was moved back to the female surgical ward on the fourth day after repeat laparotomy. Her tests remained essentially within normal range and she was discharged to the out-patient clinic after two weeks of admission.

Repeat follow-up visits in both the surgical and obstetric and gynaecological clinics two months later were uneventful and she was subsequently discharge.

Discussion

In developing countries, pregnancy and complications from child birth account for 18% of the diseases among females [11]. About 40% of the pregnant Nigerian women experience pregnancy related health problems during or after pregnancy and child birth, with 15% estimated as suffering from serious or long term complications. Although antenatal clinic attendance is expected to reduce the mortality and morbidity rates, it has been shown that women who had an apparently normal antenatal period develop complications during labour, delivery and the puerperium, and some of them die [12]. This was clearly seen in our patients as the first case had 2 previous CS and placenta praevia with antepartum bleeding but refused initial medical advice and admission, while the second had no reported risk factor or incident in the antenatal period and yet had haemorrhage and sepsis.

The recently observed increases in PPH incidence in the US and other high resource countries underscores the importance of increasing knowledge of transfusion practice among obstetricians [13]. It has been observed that even in developed countries with sophisticated systems of providing medical care, improperly executed transfusion contributes to morbidity and mortality associated with obstetric haemorrhage [14]. Increasing incidence of, PPH over the years, imbalance between resource-rich and resource-poor areas are probably due to a combination or increased prevalence of risk factors such as grand multiparity, lack of safe blood banking, non-routine use

of prophylaxis against haemorrhage, and lack of measures for drug and surgical management of atony [15]. Other risk factors include multiparity, multiple gestation, caesarean section, placenta adherence, antepartum haemorrhage, genital tract lacerations in labour, uterine inversion and disseminated intravascular coagulation (DIC). Trained medical personnel may also be involved in the causation of PPH through negligence or ignorance as seen in acute uterine inversion (due to excessive traction on the cord of a fundal implanted placenta), routine use of episiotomies and inadequate aseptic procedures.

Our hospital is a major referral centre in a society with people of low socioeconomic status where case 1 presented in the antenatal clinic after two previous caesarean sections and was diagnosed with major degree placenta praevia which are major risk factors for PPH, intractable massive haemorrhage and or massive blood transfusion. She subsequently had a third CS followed by sub-total hysterectomy as a result of intractable haemorrhage.

Some patients with massive haemorrhage are also at risk of consumptive coagulopathy commonly seen in obstetrics haemorrhage, and particularly associated with placenta abruption, amniotic fluid embolism and sepsis (as was seen in our second case). These are liable to develop haemostatic failure which may be consumptive or dilutional (due to transfusion of fluids) which may have contributed in these patients. Patients being treated for massive haemorrhage are at risk of dilutional coagulopathy leading to reduced platelets, fibrinogen and other coagulation factors [16]. This occurs if volume replacement is with red cells, crystalloids and platelets [16]. This can be prevented with early infusion of fresh frozen plasma (FFP). All our cases had transfusions of many litres of crystalloids to ensure tissue perfusion and haemodynamic stability to wade off shock and its consequences. In massive haemorrhage as seen here, coagulopathy is likely to occur rapidly and regular monitoring and haematological tests are necessary to ensure good outcome.

Rapid recognition of clients at risk of PPH and early diagnosis is essential to successful management and favourable outcome of labour, ensuring optimal maternal care and prevention of complications as clearly elucidated by Olowokere et al. [15]. Many publications have reported that placenta accreta has become the most common cause of intractable PPH [17]. This may have significantly contributed to excessive bleeding in case 1.

The use of active management of the third stage of labour and avoidance of routine episiotomies have been shown to be effective in the reduction of PPH. The key to the successful prevention and management of massive/major or intractable PPH is the identification of risk factors for PPH, and its early diagnosis and treatment. Treatment options for PPH include conservative management with uterine massage, use of uterotonic drugs, selective devascularization by ligation or embolization of the uterine artery, external compression with uterine sutures (B-Lynch, Hayman, Cho), and intrauterine packing, [14-16] sometimes including hysterectomy. The choice of treatment is said to depend on several factors such as; delivery mode, the site of origin and volume of bleeding, the patients haemodynamic tolerance including the facilities and the skills available. However, postpartum hemorrhage also occurs in women with no risk factors, so physicians must be prepared to manage this condition at every delivery [18]. All our patients were successfully managed with blood transfusion, intravenous fluids and surgery for the control of bleeding and sepsis respectively, and ICU care. Secondary PPH is often associated with infection and generally treatment involves the use of antibiotics and uterotonics.

Other supportive measures for patients with massive PPH receiving massive transfusion are the use of central venous pressure monitoring by skilled anaesthetist if available and avoidance of hypothermia by warming the patient and all blood and fluids meant for transfusion. The absence of blood components in our institution was a major drawback and fresh whole blood was employed in all cases.

It is known that PPH and its sequelae are largely avoidable through skilled attendance at childbirth through proper training and retraining of all those involved with the possible use of drills, and the avoidance of delays at home, during transportation and in the hospital. It is therefore, our desire that our initial experience with these cases will help doctors and all those involved in the care of birthing women and the transfusion chain, practicing in similar environments towards the early diagnosis and treatment of PPH to avoid its ugly consequences.

Acknowledgements

We are highly grateful to those who helped to make this work possible.

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