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CASE REPORT AIRWAY MANAGEMENT

Anesthetic implications in managing a case of placenta percreta: A case report

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ABSTRACT

Placenta percreta is the extreme end of the placenta accreta spectrum. It involves the placenta which invades through out the wall of the myometrium, crossing the uterus and getting adherent to other pelvic or abdominal organs. Surgical and anaesthetic management of placenta percreta can be challenging in such scenarios, especially in the cases where placenta percreta is getting a direct feed from aorta. It carries a high risk of maternal and fetal mortality and morbidity. I present a case of placenta percreta, G5P4, with a history of previous 3 Caesarean sections and placenta previa. Admitted with severe abdominal pain at 23 weeks. Imaging was suggestive of placenta percreta with placenta extending beyond the uterus, getting adherent to the urinary bladder, and getting direct feed from the aorta along with neovascularisation in the pelvis. She was managed by a multidisciplinary approach, caesarean delivery followed by a hysterectomy, and urinary bladder dome repair was carried out. The intraoperative course was complicated by severe obstetric haemorrhage which was successfully managed with fluids and blood products.

Keywords: Placenta Accreta Spectrum; Placenta Percreta; Massive Obstetric Hemorrhage; Massive Transfusion Protocol

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1. INTRODUCTION

Placenta percreta is the extreme end of placenta accreta spectrum (PAS). It involves the invasion of uterine myometrium through and through crossing the serosa and involving the adjacent organs. Placena accreta involves the adhesion of villi to the endometrium whereas placenta increta involves the invasion of villi through the uterine myometrium.[1] In addition to placental invasion, abnormal neoangiogenesis also plays a role in the pathogenesis of this disease process. It involves overexpression of certain angiogenic growth factor proteins e.g vascular endothelial growth factor and angiopoietin-2. However, downregulation of certain antiangiogenic proteins can also be involved as well e.g VEGF receptor-2, endothelial cell tyrosine kinase receptor Tie-2, and soluble fms-like tyrosine kinase.[2]

The risk factors associated are particularly previous deliveries by cesarian sections, placenta previa, and advanced maternal age.[3] Antenatal diagnosis of placenta percreta is by ultrasound scan and magnetic resonance imaging (MRI).[4] Intraoperative management of bleeding placenta percreta involves the formation of a team to manage the bleeding, maintain the circulating volume, and optimize the oxygen-carrying capacity which involves maintaining adequate hemoglobin by transfusion of blood products, and antifibrinolytic therapy, correction of electrolytes, serial blood gases to guide the therapy coagulation studies and rotational thermoelectrometry (ROTEM).[5]

2. CASE REPORT

I present a case of 28 years old G5P4 with a history of previous 3 cesarean sections and placenta previa at 23/52 weeks of gestation who presented to obstetric emergency with severe right-sided lower abdominal pain. There was no history of trauma. No leaking or bleeding per vagina. Examination revealed tenderness in the right iliac fossa and fullness in the pouch of Douglas. Table 1 shows her

Table 1: Vitals of the patient at admission

Vitals	Value
Blood Pressure	75/30 mmHg
Pulse	85 / min
Temperature	37 ºC
Saturation	100 %
Respiratory rate	20 / min

vitals at admission. Whereas, Table 2 shows her labs at admission. Ultrasonography (USG) revealed an echogenic mass-like lesion measuring 12x7 cm seen in the right upper abdominal cavity as shown in Figure 1. MRI revealed a 17 x 10 x 11 cm large subhepatic collection extending along the iliopsoas muscle. The placenta was noted extending from the midline to the right wall and occupying the posterior wall and extending into the uterus ostium which could be partial placenta previa as shown in Figure 2.

Table 2: Lab tests of the patient at admission

Lab tests	Value	Reference values
WBC	13 x 10^3/µL	4-11 10^3/µL
Hb	9.9 g/dl	12-15 g/dl
Platelets	213 10^3/µL	100-400 10^3/µL
INR	1	≤ 1.1
PT	11 sec	9.2-11.9 sec
APTT	28.7 sec	24.1-32.3 sec
Creatinine	25 µmol/L	62-106 µmol/L
Sodium	141 mmol/L	135-145 mmol/L
Potassium	3.7 mmol/L	3.5-5.5 mmol/L
Fibrinogen	2.97 g/L	2-5 g/L

APTT: Activated partial thromboplastin clotting time; Hb: Hemoglobin; INR: International normalized ratio; PT: Prothrombin time; WBC: White blood cells

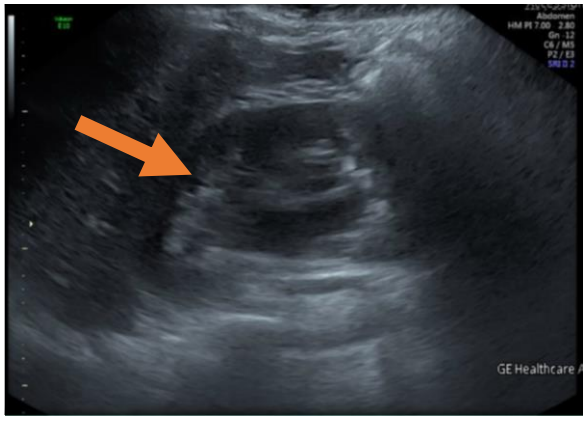

Figure 1: Ultrasound of the pelvis showing retroperitoneal hematoma as pointed by the arrow.

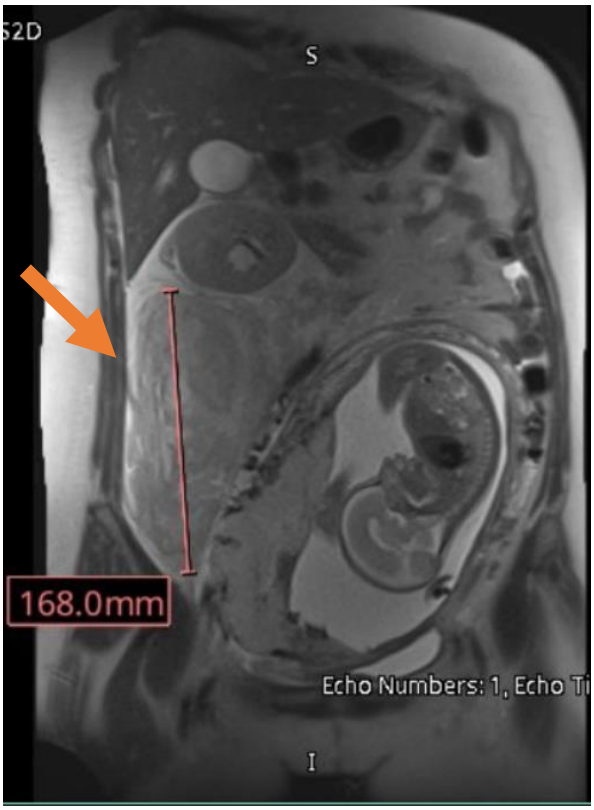

Figure 2: MRI of pelvis showing retroperitoneal hematoma as shown by arrow.

The patient was stabilized with IV fluids and one unit of PRBCs and transferred to ICU for hemodynamic monitoring along with a plan of doing a CT scan to know the origin of retroperitoneal bleeding. In ICU she was transfused with a further 4 units of PRBCs. CT scan showed placenta percreta supplied from a prominent tortuous arterial branch originating from the aorta anteriorly superior to the inferior mesenteric artery likely representing an anomalous ovarian artery as shown in Figures 3 and 4. No evidence of arterial blush to suggest active arterial extravasation.

Multi-disciplinary team (MDT) meeting was carried out involving personnel from obstetrics, anesthesia, intensive care, vascular surgery, general surgery, neonatology, urology, and radiology. The decision was made for laparotomy hysterotomy/delivery of the fetus plus minus hysterectomy plus-minus to proceed. Massive transfusion protocol (MTP) was initiated and she was pushed to the operation theatre for the planned surgery.

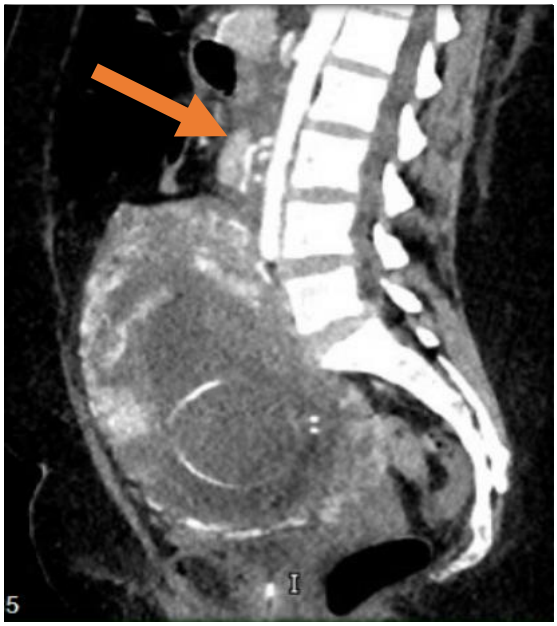

Figure 3: CT scan of the pelvis showing the origin of the artery supplying the placenta previa directly from the aorta as shown by the arrow.

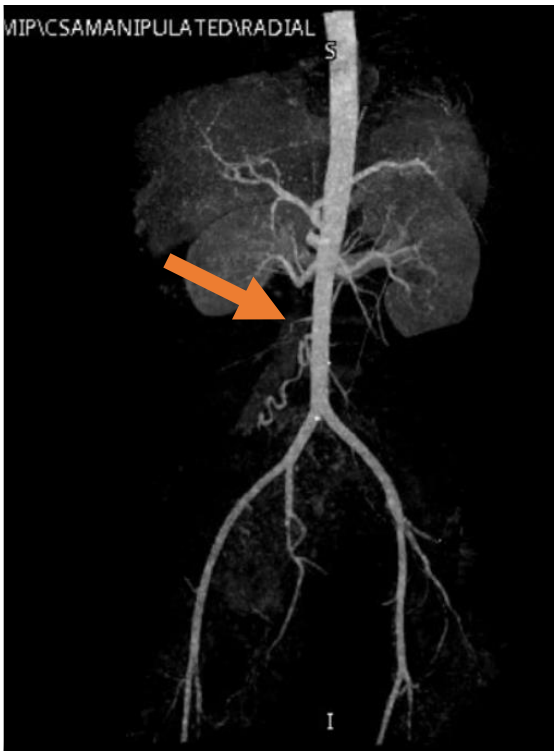

Figure 4: CT angiogram showing the origin of the artery supplying the placenta previa directly from the aorta just above the origin of the inferior mesenteric artery as shown by the arrow

3. ANESTHESIA MANAGEMENT

Before the induction obstetricians, vascular surgeons, and neonatologists were made to be present in the room. The team was organized with proper assignment of roles to team members. The team was comprised of two anesthesia consultants, one anesthesia trainee, and five anesthesia technicians. Another Anesthesia consultant apart from the team was assigned the role to communicate with the blood bank. Inside the operation room (OR) she was connected to standard American society of anesthesiologists (ASA) monitors.

Adequate preoxygenation was done. She was induced with fentanyl 200mcg and etomidate 20mg. 100mg rocuronium was given. Size 7 ETT was inserted and fixed at 20cm at the lip. Anesthesia was maintained with remifentanil infusion along with sevoflurane. Intermittent boluses of rocuronium were given throughout the surgery. The right radial arterial line and right internal jugular vein central line were inserted. Level one was connected to the central line. 2 large bore IV cannulas, a foley catheter, and a right nasal gastric tube were inserted as well. Right after line placement 10mg of morphine was given. Cell saver was kept on standby. She received 2 units of uncross-matched O-negative packed red blood cells before the incision. She was ran on 0.01 mcg/kg/min of noradrenaline. Seventeen minutes into the incision alive baby girl was born weighing 600 grams. Handed over to the neonatologist, intubated, ventilated, and transferred to the neonatal intensive care unit. Serial arterial blood gases (ABGs) and rotational (ROTEM) studies were carried out to guide hemodynamics management. Forty-five minutes into the surgery, she had severe bradycardia with a heart rate (HR) of 30 /min, for which she was given adrenaline.

During the dissection of the placenta percreta, a massive hemorrhage started which persisted for around 2 h and 20 min with a total blood loss of 15L. During this period the patient was resuscitated with IV crystalloids, colloid, blood, and blood products. She was managed with 10 L of crystalloids, 600 ml of colloids, 8 g fibrinogen, 2 g of tranexamic acid, 22 units of PRBCs, 9 units of fresh frozen plasma (FFP), and 12 units of platelets. She had received 4000 mg of calcium chloride and 2000 mg of calcium gluconate. Vascular surgeons were challenged to secure hemostasis as sutures were not holding due to tissue friability. It took them 2 h and 20 min to secure the bleeding. During the placental dissection phase bladder dome was injured which was repaired by the urologist towards the end of the surgery. The total surgical time was 4hr and 30 min after which the patient was transferred to ICU successfully. Her blood gas before transfer to ICU is shown in Table 3.

Table 3: ABG of the patient before transfer to the intensive care unit

ABG	Value	Reference values
pH	7.421	7.35-7.45
PCO_2	42 mmHg	35-45 mmHg
Na	142 mmol/L	136-146 mmol/L
K	3.9 mmol/L	3.7-4.7 mmol/L
Lactate	1.8 mmol/L	0.0-2.0 mmol/L
Hb	10.8 g/dL	12-14 g/dL
Glucose	7 mmol/L	3.5-5.4 mmol/L
SpO_2	100%	95-100%
HCO_3	27.5 mEq/L	22-28 mEq/L
BE	2.7 mmol/L	–3-3 mmol/L

ABG: Arterial blood gas; BE: Base excess; Hb: Hemoglobin; K: Potassium; Na: Sodium; PCO2: Partial pressure of carbon dioxide; pH: Potential of Hydrogen; SpO2: Oxygen saturation

She was extubated the next day with an uneventful hospital course later on. She was transferred to the ward a couple of days later and was discharged home finally.

4. DISCUSSION

PAS is a general term that consists of the following three subtypes. Placenta accreta - placental villi attach to the myometrium (rather than decidua). Placenta increta - placental villi penetrate through the myometrium. Placenta percreta -placental villi penetrate through the myometrium to the uterine serosa or adjacent organs.

The International Federation of Gynecology and Obstetrics (FIGO) Placenta Accreta Spectrum Disorders classified PAS as follows.1 Grade 1 - abnormally adherent placenta: placenta adherent. Grade 2 - abnormally invasive placenta: increta. Grade 3 - abnormally invasive placenta: percreta. Subtype 3a - limited to the uterine serosa. Subtype 3b - urinary bladder invasion. Subtype 3c - the invasion of other pelvic tissue/organs.

In addition to placental invasion, abnormal neoangiogenesis also plays a role in the pathogenesis of this disease process. It involves overexpression of certain angiogenic growth factor proteins e.g vascular endothelial growth factor and angiopoietin-2. However, downregulation of certain antiangiogenic proteins can also be involved as well e.g VEGF receptor-2, endothelial cell tyrosine kinase receptor Tie-2, and soluble fms-like tyrosine kinase.[2] Risk factors for PAS include placenta previa following the previous C-section and advanced maternal age among others.6 Complications include coagulopathy, ARDS, AKI, and so on. The diagnosis of antenatal PAS can be made reliably by ultrasound scan and MRI.

Management options include either primary hysterectomy or conservative management or additional techniques e.g Interventional radiology-guided intraoperative internal iliac artery embolization or post-operative internal iliac artery or abdominal balloon occlusion along with uterine artery embolization.[7] Placenta percreta is one of the other causes of massive obstetric hemorrhage leading to maternal morbidity and mortality if not managed promptly. Various definitions of obstetric hemorrhage exist such as loss of blood > 2500 ml, drop in Hb > 4g/dl, need for transfusion of more than 5 units of PRBCs, or coagulopathy secondary to bleeding.[8]

The definition of massive transfusion includes a large volume of blood products that are transfused to the patient over a short period, transfusion of more than 10 units of PRBCs in 24 h, transfusion of more than 4 units of PRBCs in one hour with anticipation of further transfusion, replacement of more than 50% of total blood volume by blood products within 3 h.[9]

Once the Massive transfusion protocol is activated, blood products are provided by the blood bank in a 1:1:1 ratio (6 units of PRBCs, 6 units of FFP, and one unit which includes 6 packs of platelets).[10] The use of cell salvage was traditionally contraindicated during such scenarios for the concern of amniotic fluid embolism. however, the American College of Obstetricians and Gynecologists (ACOG) and Royal College of Obstetricians and Gynecologists(RCOG) support its use while ASA recommends its use if blood products are not available.[9] Tranexamic acid is an antifibrinolytic drug whose mechanism of action includes antifibrinolytic i.e prevention of fibrin lysis by plasmin. The WOMAN trial favors the use of Tranaxemic acid within 3h of delivery.[11]

Disseminated intravascular coagulation (DIC) is one of the known complications of hemorrhage which can be prevented by augmentation with fibrinogen which itself is an independent predictor of mortality.[12] Fibrinogen can be provided by FFP, cryoprecipitate, and fibrinogen concentrate each having its disadvantages.[13] Prothrombin complex concentrate (PCC) which contains factors II, VII, IX, and X along with protein C and protein S has been used in case of severe bleeding, especially in the setting of vitamin K antagonism and reversal. However the obstetric population is not prescribed warfarin due to its concerns regarding teratogenicity but PCC can be transfused in patients with liver cirrhosis, HELLP (hemolysis, elevated liver function tests, and low platelets), or in cases of acquired factor deficiency.[14] Recombinant factor VIIa (rFVIIa) should be considered as a last resort to manage bleeding

because it causes thromboembolism and platelets and fibrinogen should be optimized before transfusing rFVIIa.[15]

Moreover serial ABGs and ROTEM studies guide toward a specific therapy. Electrolytes should be managed to prevent arrhythmia as hyperkalemia can occur during the phase of large-volume resuscitation, and hypocalcemia can occur due to chelation by citrate. In both hyperkalemia and hypocalcemia, 1 to 2 g of CaCl2 should be given to stabilize the myocardium.[10]

5. CONCLUSION

Bleeding placenta percreta requires a multidisciplinary approach of management requiring an anesthetist, obstetrician, intensivist, neonatologist, vascular surgeon, and urologist in cases of bladder involvement like ours. Massive obstetric hemorrhages present a unique challenge to anesthesiologists given massive transfusion and electrolytes correction. Teamwork is a key to success. Adequate resuscitation and perioperative hemodynamics management lead to positive patient outcomes.

6. Conflict of interest

None

7. Ethical considerations

Written informed consent was obtained from the patient to publish this report as well as the pictures.

8. Author contribution

Um I. Rubab is the sole author of this case report

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