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Case Report

Intra-operative cardiac arrest from undiagnosed incretic placenta praevia precipitated haemorrhage reversed via external jugular access: a case report

Alfred T. Aggo*, Samuel B. Olamuyiwa

Department of Anaesthesia, University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria

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***Correspondence:**
Dr. Alfred T. Aggo,
E-mail: alfred.aggo@uniport.edu.ng

ABSTRACT

A 32-year old obese multipara with undiagnosed incretic major degree placenta praevia was scheduled to have an elective repeat Caesarean for two previous caesarean. Under subarachnoid anaesthesia, placental severance and myometrial avulsion precipitated torrential haemorrhage occasioning intraoperative cardiac arrest which necessitated external jugular access for successful resuscitation. Following peripartum hysterectomy under general anaesthesia, she received intensive care and was discharged to the ward after 48 hours. This report highlights the necessity of accurate prenatal diagnosis of invasive placentation and the grave sequelae of a missed diagnosis, as well as the strategic significance of external jugular access in resuscitation post-cardiac arrest from massive haemorrhage.

Keywords: Incretic placenta, Jugular access, Massive haemorrhage

INTRODUCTION

An increasing number of high risk obstetric surgical patients has been recorded over the years by the University of Port Harcourt Teaching Hospital, a tertiary healthcare facility sited in the South-South region of Nigeria. This is attributable partly to the facility being the foremost training and referral institution and partly to its location in a city with rapidly growing population.

This case report is about severe obstetric haemorrhage from antenatally undiagnosed major degree placenta praevia with incretism, in a patient who had undergone two Caesarean sections, resulting in cardiac arrest that was successfully reversed with the aid of an external jugular venous (EJV) access required for circumventing the limitations of difficult peripheral access.

Uvelin and colleagues have documented the indispensability of EJV access in many conditions including massive haemorrhage precipitating severe hypovolaemia.[1]

CASE REPORT

A 32-year old obese multipara was scheduled to have an elective repeat Caesarean section at a gestational age of 38 weeks and 3 days, for two previous Caesarean deliveries. She had a major degree placenta praevia with incretism that missed prenatal diagnosis. On preanaesthetic assessment the previous day, she had no detectable comorbidity, was conscious, alert and afebrile (temperature 36.8°C). Her weight and height recorded during her last antenatal visit one week earlier were 88 kg and 1.63 m respectively (BMI 33.12 kg/m^2). Her cardiovascular system examination findings were normal-pulse rate (PR) 84 b/min, blood pressure (BP) 120/70 mmHg while 1st and 2nd heart sounds only were heard. Bilaterally, her breath sounds were vesicular and she had mild pitting pedal oedema. She had no contraindication to subarachnoid block (SAB) and her foetal heart rate was within normal limits. Her last ultrasonography (USG) done 14 days prior to admission for an elective caesarean reported a normal foetus in oblique lie at a gestational age of 36 weeks and 3 days, adequate volume of liquor amnii,

and an anterior, mid-uterine placenta with grade III maturity. Her other laboratory investigations in her 36th week of gestation showed a haemoglobin of 11.2 g/dl, a fasting blood glucose of 5.3 mmol/l and mild proteinuria (1^+). She was instructed to withhold solid food for 8 hours but take clear fluid up to 2 hours prior to surgery, assigned to American Society of Anesthesiologists (ASA) class II and premedicated with tablet ranitidine 150 mg orally 10 p. m. the previous night and at 6 a. m. in the morning of surgery. A written informed consent was obtained for SAB and she was advised to provide 1 unit of whole blood.

On the morning of surgery, she was wheeled to the obstetric theatre on a trolley and transferred to the operating table on which she was placed supine with a wedge under her right hip to ensure left lateral uterine displacement of 15-30 degrees. A multiparameter monitor (Dash 4000®, GE Medicals) was attached and her noninvasive baseline vital signs recorded were: PR 94 b/min., BP 122/76 mmHg, SpO_2 98-99% and axillary temperature 36.7ºC; the 5-lead electrocardiogram (ECG) displayed normal P-Q-R-S-T tracing. Antecubital venous access secured with a 16 gauge Mediflon cannula, ceftriaxone 1g and dexamethasone 8 mg were administered intravenously. Following preloading with 15 ml/kg prewarmed 0.9% normal saline and bladder catheterization, observing asepsis, SAB was done at first attempt though the L3/L4 intervertebral space in her sitting position, using 2 ml of 0.5% hyperbaric bupivacaine and fentanyl 25 µg injected over 15-20 sec via a 25 gauge Quincke spinal needle. The lumbar puncture site was covered with sterile gauze and the patient returned to the supine position with slight head-up. Post SAB, her BP was checked every 2 min for 10 min, then every 2-3 min subsequently. In the first 15 min post SAB, she had stable BP (98/63 to 107/70 mmHg), PR (68-74 b/min.), SpO_2 (97-99%) and axillary temperature (36.3-36.5ºC); there was normal 5-lead ECG tracing.

Surgery commenced 8 minutes after SAB, the patient having achieved adequate sensory block height (T6-T7) and spontaneously breathing supplemental oxygen delivered at 2.5 l/min. via nasal prongs. A lower segment uterine incision, 15 min after SAB, for foetal extraction severed the placenta; oxytocin 2.5 units bolus was administered intravenously at the delivery of the anterior shoulder followed by 10 units titrated via each litre of 0.9% normal saline. Torrential haemorrhage occurred from placental severance and myometrial avulsion upon manual removal of the placenta.

The diagnosis of major degree placenta praevia with incretism was then made and a decision taken for an immediate peripartum hysterectomy for which a separate consent was sought for the avoidance of litigation. Transfusion was ordered while repeated intravenous boluses of ephedrine 5 mg were given as necessary along with 10 mg titration in each litre of 0.9% normal saline to counter hypotension. A second peripheral venous access attempted was unsuccessful due to severe hypovolaemia and peripheral venous collapse.

Seeing the fast declining haemodynamic parameters, her trachea was swiftly intubated with size 7.0 mm cuffed tracheal tube facilitated by intravenous ketamine hydrochloride 100 mg and suxamethonium chloride 100 mg, with Sellick's manoeuvre; intermittent positive pressure ventilation (IPPV) was commenced with 100% oxygen delivered via the anaesthetic machine, excluding volatile agent. About 8 min after tracheal intubation, flat ECG tracing appeared as patient developed cardiac arrest from continuing haemorrhage. Chest compressions were begun promptly at 100 to 120 per min at the lower half of the sternum. Transfusion was started as soon as blood arrived but stopped within few seconds due to evidently extravenous migration of cannula tip. EJV access decided, she was tilted into Trendelenburg position; with chest compressions paused briefly for about 30 sec, right EJV access was secured at first attempt using 16 gauge Mediflon cannula.

Epinephrine 1 mg bolus was administered intravenously, the first 2 units of whole blood were transfused in quick succession over 5-6 min and replaced with 0.9% normal saline while awaiting more units. Chest compressions and IPPV were continued till the 7th min post-cardiac arrest when return of spontaneous circulation (ROSC) was detected as evidenced by palpable central pulses and appearance of P-Q-R-S tracing on the monitor. Having obtained a second consent, a peripartum hysterectomy was completed within 2 hours. She received a total of 9 pints of whole blood, 7 l of 0.9% normal saline and 5 g of calcium gluconate (1 g given intravenously after every 2 pints of blood) intraoperatively. She was mechanically ventilated postoperatively for 24 hours in the ICU and discharged to the ward after 48 hours.

DISCUSSION

An unacceptably high maternal morbimortality rate yet plagues underdeveloped countries raising serious global concern among healthcare professionals. Of the different identifiable aetiologies, Say and colleagues in a WHO systematic analysis documented that postpartum haemorrhage (PPH) was responsible for about 1,00,000 maternal deaths annually, with the majority occurring in poorly developed nations.[2] Musarandega et al similarly reported that PPH causing severe hypovolaemia was the leading cause of maternal mortality especially in sub-Saharan Africa.[3] Although physiological, pregnancy and parturition constitute potential risks of morbidity and mortality to the parturient and foetus, posing significant challenge to care givers. To prenatally identify and reduce this risk to zero or barest minimum through the provision of quality health services underpins antenatal care.

Modern day high quality obstetrics entails the use of medical imaging in enhancing prenatal evaluation and precision of diagnosis as an integral component, because

necessary planning and decision taking toward achieving safe foetomaternal outcome are enabled thereby. The usefulness of grayscale and colour/power Doppler USG, and Magnetic resonance imaging in the prenatal diagnosis of placental adherencial pathology (PAP) is documented.[4,5] PAP, a spectrum of three conditions characterized by abnormal trophoblastic invasion of the uterus and classified, based on the degree of invasion, as accretism, incretism or percretism, is a reported cause of significant increase in maternal morbimortality.[4] Pilloni et al noted a statistically significant more favourable maternal outcome in the group of parturients with antenatally diagnosed PAP, attributing this to several factors: (i) preoperative planning and adequate preparation enabled by an antenatal diagnosis, (ii) fundal uterine incision circumventing placental severance, (iii) avoidance of manual removal of placenta, and (iv) performance of peripartum hysterectomy.[4]

Research findings have linked a higher incidence of invasive placentation to the combined occurrence in same patient of placenta praevia and previous uterine surgery, especially Caesarean, with an increasing incidence corresponding to the number of Caesarean sections.[4,6] In this report, the parturient had two previous Caesarean and no antenatal diagnosis of her adherent placentation. The consequent failure of specific adequate planning leading to a lower segment incision, placental severance, manual removal and precipitation of massive haemorrhage occasioning significant maternal morbidity, lend support to previous documentations.[4,6] Therefore, a prenatal diagnostic radiology to exclude invasive placentation is highly indicated in obstetrics.

Uncorrected or poorly corrected severe blood loss triggers a pathophysiology progressing eventually to cardiac arrest as occurred in this parturient. To achieve ROSC, timely and adequate transfusion during resuscitation is demanded, requiring a wide bore access in a central or large peripheral vein. Hypovolaemic shock causes difficulty in securing peripheral venous access, especially in the obese, and central line insertion, compared to EJV access, is more challenging and time consuming. Given this background, a right EJV access was secured in this patient with a 16 gauge cannula, facilitated by a Trendelenburg position, enabling the transfusion of two units of blood within 5-6 min post cardiac arrest. The ROSC noted in the 7th min post arrest made evident the strategic significance of EJV access in the successful resuscitation.

EJV access has been described as capable of serving all the functions of a central venous access and indispensable in many conditions including massive haemorrhage.[1,7] While manoeuvring into Tendelenburg position prior to EJV cannulation is a procedural necessity to enhance venous filling and visibility, in the event of a failed intravenous access, an intraosseous access is recommended.[8,9] In an arrest with nonshockable rhythm as witnessed in this patient, prompt commencement of continuous, effective external chest compression, a secured airway and IPPV constitute critical steps to successful resuscitation. Timely administration of epinephrine causing systemic vasoconstriction and increasing coronary and cerebral perfusion pressures is contributory to ROSC.[10] Administering calcium during multiple citrated blood/blood product tranfusions is essential to prevent iartrogenic hypocalcaemia induced cardiac dysrhythmias, vasoplegia and coagulopathy which are associated with increased mortality in patients suffering haemorrhagic shock; additionally, deranged hepatic metabolism during haemorrhagic shock increases citrate levels, further aggravating hypocalcaemia.[11,12] In place of calcium chloride, therefore, calcium gluconate 5 g was administered. Scientific literature recommends 1 g of calcium gluconate for every 1-2 pints of blood transfused.[13]

Obtaining written informed consent prior to surgery and anaesthesia is imperative as an evidence that the consentee has been adequately informed about the purpose, nature, consequences, alternatives and risks of the intervention and has freely consented to it.[14] This depicts a satisfactory patient-doctor communication, the breach of which dominantly underpins litigation. The missed prenatal diagnosis of PAP in this parturient disabled fulfillment of the tenets governing preoperative informed consent for perinatal hysterectomy, occasioning avoidable intraoperative double consent. Post resuscitation, continued care in the ICU for survivors of cardiac arrest has been documented as very vital to successful outcome.[15]

CONCLUSION

Prenatal diagnostic radiology deficient in accurate assessment of placentation contributes to increased maternal morbimortality, especially in sub-Saharan countries. EJV access post-cardiac arrest is achievable and significantly strategic to successful resuscitation.

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