CASE REPORT

Cardiac arrest following acute puerperal uterine inversion

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SUMMARY

Although uterine inversion is a potentially life-threatening complication of childbirth, there are only six case reports of cardiac arrest due to acute inversion to date. We report a successful outcome in a patient with sickle cell disease who had a witnessed cardiac arrest due to acute puerperal uterine inversion. Revival from cardiac arrest and resuscitation was followed by manual repositioning of the uterus. She gradually recovered from the acute kidney injury that developed as sequelae of the cardiac arrest and subsequent circulatory shock. Awareness of this rare complication and timely intervention is the key to a successful outcome in these patients.

BACKGROUND

The reported incidence of uterine inversion is extremely variable; it ranges from one in 2148 to 6407 births¹ to 13 in 358 874 deliveries.² Although it is considered to be a 'potentially life-threatening complication of childbirth',¹ only one case of haemorrhagic death associated with uterine inversion was reported in 1461 270 pregnancies in the USA between 2000 and 2006,³ whereas three maternal deaths in 36 acute inversions out of 57 036 deliveries were reported from Pakistan between 1995 and 2002.⁴ A literature search using PubMed revealed only six case reports^{5–10} of cardiac arrest due to uterine inversion, two of which were after caesarean deliveries.^{6,7}

CASE PRESENTATION

A 27-year-old primigravida was admitted to our hospital for safe confinement and delivery at 35 weeks gestation with intra uterine growth retardation and borderline oligohydramnios. The estimated fetal weight was 1.8 kg and amniotic fluid index was 8 cm on fetal ultrasonography. The patient also had sickle cell anaemia confirmed by the Sickling Test and haemoglobin electrophoresis. Except for anaemia (haemoglobin 9 g/dL), all her vital observations and laboratory investigations were normal.

Over the next few days she developed hypertension with blood pressures ranging from 150/100 mm Hg to 146/96 mm Hg on more than two occasions greater than 4 h apart with proteinuria two plus on dipstick measurement. Anticipating the need for early delivery, she was given two 12 mg doses of β -methasone to accelerate fetal lung maturity. After discussing the available therapeutic options with the patient, induction of labour at 36 weeks gestation was planned with 0.5 mg intracervical prostaglandin E2 gel.

Eight hours later, the patient's cervix was 5 cm dilated, 80% effaced and the degree of descent of the presenting part of the fetus through the maternal pelvis was indicated by the fetal head being at minus one station. The fetal heart remained reassuring and the patient's cervix was fully dilated 3 h later. She pushed effectively for 45 min and had a normal spontaneous vaginal delivery of a 1.55 kg baby boy over a right mediolateral episiotomy. Intravenous oxytocin at 6 milliunits/min had been used for augmentation of labour and 5 units of oxytocin bolus was given after the delivery of the anterior shoulder. The intact placenta was delivered spontaneously within 15 min and had no evidence of placenta accreta on inspection. The baby had an APGAR score of 7 at 1 min and 9 at 5 min. Immediately after delivery, the mother had a heart rate of 82 bpm with a non-invasive blood pressure (NIBP) of 132/83 mm Hg. The episiotomy was repaired.

About an hour later, post partum haemorrhage (PPH) was noted and approximately 800 mL of blood clot was removed from the vaginal passage. The patient's heart rate was 115 bpm and NIBP was 110/80 mm Hg. An intravenous oxytocin infusion was started at 10 Units/h and 0.25 mg prostaglandin F2 α was administered intramuscularly. Rapid infusion of intravenous crystalloids was initiated. Clinical examination revealed a very sensitive soft necrosed mass exteriorised at the vulva. On an abdominal examination, the uterine fundus could not be palpated. On vaginal examination, a rim of cervix was felt all around the protruding mass. An emergent pelvic ultrasound in the labour room demonstrated the absence of the uterus in the pelvic cavity. The constellations of these findings were consistent with the diagnosis of complete uterine inversion. By this time the NIBP had dropped to 100/60 mm Hg and the heart rate was 96 bpm. Within minutes, severe bradycardia for a few seconds was followed by cardiac arrest with a non-shockable rhythm.

TREATMENT

The patient was managed as per hospital protocol for cardiac arrest based on the 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation. Although atropine is not routinely recommended for cardiac arrest with a non-shockable rhythm, considering the possibility of vagal stimulation causing the bradycardia and cardiac arrest, the patient also received 0.6 mg atropine intravenously. About 8 min of cardiopulmonary resuscitation resulted in sinus rhythm with restoration of spontaneous circulation (ROSC).

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By this time, tracheal intubation was achieved and the patient was being ventilated by manual intermittent positive ventilation with 100% oxygen by a Bain circuit. Infusion of epinephrine at 6 µg/min and dopamine at 6 µg/kg/min was started to maintain a mean arterial pressure of >65 mm Hg. The patient's PPH continued and by now her estimated blood loss was about 2000 mL. Her arterial blood gases immediately after ROSC revealed severe metabolic acidosis with a pH of 6.84 and lactate levels of 13.1 mmol/L with a base deficit of 24.3 mmol/L. The haemoglobin level was 5.8 g/dL. Despite a sinus rhythm and ROSC, she was unconscious and unresponsive with a Glasgow Coma Scale (GCS) score of E1, V-T, M1) 3/15. She was immediately taken to the operating theatre and the episiotomy was reopened, the uterus was repositioned and the episiotomy was again closed in layers under general anaesthesia, which was maintained with intravenous ketamine, fentanyl and intermittent positive pressure ventilation with nitrous oxide and oxygen. The patient was subsequently transferred to the critical care unit (CCU). The blood loss was replaced with packed red blood cells and fresh frozen plasma. Supportive care was continued in the CCU with mechanical ventilation and inotropic support. Over the next 2 days her level of consciousness gradually improved, inotropes were tapered and she was weaned from mechanical ventilation. Finally, tracheal extubation was achieved on the third day.

Following admission to CCU, the patient developed oliguria with rising blood urea and serum creatinine levels, which were 93.8 (normal 20–40 mg/dL) and 3.12 mg/dL (normal 0.5–1.5 mg/dL), respectively, on the fourth day of her CCU admission. She needed intermittent haemodialysis (total 8 cycles) and her urine output gradually improved by the 12th postpartum day.

OUTCOME AND FOLLOW-UP

The patient was discharged on the 30th day without residual neurological damage. Her urine output was adequate with serum creatinine of 1.9 mg/dL and a glomerular filtration rate of 41.3 mL/min on a radioisotope renal scan at discharge. She was followed up for the next 6 months at our outpatient department and her subsequent recovery was uneventful.

DISCUSSION

None of the suggested possible risk factors^{2 11 12} of acute uterine inversion mentioned in box 1 could be established in our case, reaffirming the fact that the causes are not yet clearly understood.² However, it has been suggested that active management of the third stage of labour can reduce the incidence of uterine inversion.¹³ Possibly, this awareness may be responsible for the lower incidence of puerperal uterine inversion and its potentially life-threatening complications.

Box 1 Causes of uterine inversion^{2 11 12}

1. Mismanagement of the third stage of labour
2. Inappropriate fundal pressure
3. Uterine atony due to relaxed lower segment or relaxed cervix
4. Premature or strong traction on the cord
5. Adherent placenta
6. Chronic uterine over-distension

The classical teaching was that diagnosis of uterine inversion is essentially clinical and is manifested by shock disproportionate to the blood loss.² As in this case, complete inversion could be easily identified by palpating the uterine fundus in the vaginal introitus or cervical os.¹⁴ Incomplete inversions need a clinical examination to palpate the fundal wall in the lower uterine segment or the cervix.¹⁴ Our hospital is a tertiary care centre for obstetric care. Ultrasound is readily available in the labour room and all doctors are trained in obstetric ultrasound. It merely acted as a tool to confirm the clinical finding while transfer to the operating theatre was being arranged. However, ultrasound confirmation of uterine inversion should not delay the management of acute uterine inversion. In our case, the sequence of events developed rapidly, with cardiac arrest developing within 20 min of the clinical diagnosis of uterine inversion. Although it is presumed that the shock is primarily due to bradycardia caused by the parasympathetic stimulation occurring during traction on supporting uterine ligaments, there is scarce clinical evidence to support this view.² In fact, there is little evidence to prove that the shock is disproportionate to the estimated blood loss, the actual loss often being underestimated.² As in our case, the estimated blood loss of about 2000 mL and drop in haemoglobin to 5.8 g/dL (from 9 g/dL) also indicate severe PPH as the primary cause of shock. However, with bradycardia preceding the cardiac arrest, intense parasympathetic stimulation cannot be ruled out as a possible cause of the cardiac arrest. In fact, severe blood loss with bradycardia preceding the cardiac arrest indicates that sudden severe hypovolemia and parasympathetic stimulation caused by the traction of the uterine ligaments could have contributed to the cardiac arrest in our patient.

The treatment involves simultaneous uterine repositioning and aggressive management of shock. Despite the guidelines not recommending routine use of atropine during bradycardic or asystolic cardiac arrest,¹⁵ atropine was used in this patient as it is established that uterine inversion causes intense parasympathetic stimulation² and atropine 'reverses cholinergic-mediated decreases in heart rate and atrioventricular nodal conduction'.¹⁵ Besides, there is 'no evidence that atropine has detrimental effects during bradycardic or asystolic cardiac arrest'.¹⁵ Although various modalities such as deepening of general anaesthesia, β-2 adrenergic agonists (terbutaline), nitroglycerine or magnesium sulphate have been suggested to relax the uterus to facilitate its repositioning,¹⁶ use of any of these options is associated with the risk of further aggravating the haemodynamic

Learning points

- ▶ Puerperal uterine inversion is a rare but potentially life-threatening complication of childbirth.
- ▶ Appropriate management of the third stage of labour, along with avoiding inappropriate fundal pressure and premature or strong cord traction can help reduce the incidence of puerperal uterine inversion and its life-threatening complications.
- ▶ Uterine inversion can potentially lead to hypovolemic and neurogenic shock. Early recognition and rapid management are important for a good recovery.
- ▶ Awareness about the life-threatening complications of uterine inversion and ability to rapidly manage its clinical consequences are crucial for a successful outcome.

instability. Although we successfully achieved manual replacement of the uterus in our patient, it is possible in only 30–40% of the patients.¹¹ The O'Sullivan Replacement Technique of pressurised infusion 2–3 L of warm saline into the vagina has a success rate of nearly 80%.¹¹ If these fail, the patient would require surgical correction with laparotomy.

In our patient, vasoocclusive and other insults due to sickle cell disease¹⁷ possibly aggravated the renal impairment due to decreased kidney perfusion caused by the circulatory shock, and this led to the acute kidney injury. Aggressive management of shock and the subsequent Renal Replacement Therapy until return of intrinsic kidney function adequate to meet patient's need ensured a successful outcome in our patient.

Contributors DSN was primarily responsible for the conduct of the case, its management, literature review and manuscript preparation. MRD was responsible for management of the case, literature review and manuscript preparation. DPS was responsible for the critical care management of the case and manuscript preparation. BP was responsible for the conduct of the case, its management, literature review and manuscript preparation.

Competing interests None.

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