Puerperal uterine inversion and shock

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Uterine inversion is an unusual and potentially life-threatening event occurring in the third stage of labour. It is associated with significant blood loss, and shock, which may be out of proportion to the haemorrhage, although this is questionable. When managed promptly and aggressively, uterine inversion can result in minimal maternal morbidity and mortality. A recent case is described, followed by a short review of the literature.

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Uterine inversion is a potentially life-threatening complication of childbirth that occurs between one in 2148 and one in 6407 births.1,2 There is little published information on the pathophysiology or anaesthetic management of this
problem. We describe a recent case and present a short review of published work on uterine inversion, particularly relating to its clinical features and management.

Case history
A 30-yr-old woman, 12 days beyond her expected date of delivery, was admitted for induction of labour. She had no medical problems and had had two previous normal vaginal deliveries. All investigations were within normal limits (haemoglobin 11 g dl⁻¹ and weight 78 kg). After a 4-h labour, with intramuscular pethidine 100 mg and Entonox for pain relief, she gave birth to a healthy boy. The placenta remained adherent despite intermittent controlled cord traction, but after 20 min was felt to be at the cervical os. The patient gave a push and passed approximately 500 ml fresh blood. On examination, the placenta had passed through the introitus and the uterus was thought to be partly inverted. The placenta was removed with further heavy blood loss of approximately 500 ml. There was no evidence of placental abnormality such as placenta accreta. Attempts by the obstetricians to reduce the uterus manually and using hydrostatic pressure (a litre bag of warm crystalloid attached to a silicone suction cap held firmly in the vagina) were unsuccessful. No tocolytic was administered. An anaesthetist was called to anaesthetize the patient for urgent reduction of the uterus. Total blood loss was estimated to be 1000 ml.

At this time, the patient was almost unconscious and only groaning in response to painful stimuli. She was pale, with a heart rate of 150 beats min⁻¹, a thready pulse, prolonged capillary refill time, and an unrecordable arterial pressure. The bed was soaked with fresh blood. There was no evidence for a neurogenic component to the hypotension such as bradycardia or peripheral vasodilatation. After rapid infusion of 3 litres of crystalloid and incremental doses of ephedrine (total 12 mg), her arterial pressure rose to 90/60 mm Hg and she was transferred to the operating theatre. Standard monitoring was instituted and rapid sequence induction with etomidate 8 mg and succinylcholine 100 mg was followed by tracheal intubation and maintenance of anaesthesia with isoflurane (end-tidal concentration 0.6–0.9%) in 50% oxygen and nitrous oxide. The uterus was reduced without difficulty. A bolus dose of oxytocin 5 u was followed by an infusion of 40 u over 4 h as per unit protocol. Ephedrine (total 18 mg), methoxamine (total 8 mg), and a further 3 litres of crystalloid were required to maintain adequate cardiovascular function. Her haemoglobin concentration estimated using a Hemocue was 8 g dl⁻¹ and 5 u of blood were infused in theatre. Her post-transfusion haemoglobin measured in the recovery room was 8 g dl⁻¹. Her subsequent recovery was uneventful.

Discussion
Puerperal uterine inversion is due to displacement of the fundus of the uterus, usually occurring during the third stage of labour. It is classified as complete if the fundus passes through the cervix, or incomplete if it remains above this level.² It is associated with placenta praevia and fundal implantation, antepartum use of magnesium sulphate, and umbilical cord traction with vigorous fundal pressure.² Three The underlying causes are not completely understood.² Three

The classical presentation is of an obviously displaced uterus while delivering the placenta, usually in association with post-partum haemorrhage and clinical shock (hypotension and inadequate tissue perfusion), out of proportion to the blood loss. The shock is thought to be due to the parasympathetic effect of traction on the ligaments supporting the uterus and may be associated with bradycardia.³ This description of the shock is not well supported by the literature, and may mislead emergency management.

The clinical features of uterine inversion have been investigated in several retrospective studies. Platt and colleagues reviewed 28 cases, of which eight patients were diagnosed as clinically shocked, although no definition was given. The average estimated blood loss was 1260 ml in primiparous women and 800 ml in multiparous women.¹ Brar and colleagues later reviewed 56 cases from the same hospital and the range of estimated blood loss was 500–2500 ml.² The average amount of blood transfused was 2 u (range 0–6), and one-third of the patients were diagnosed as clinically shocked, defined as a systolic arterial pressure of less than 90 mm Hg and a pulse rate of more than 120 beats min⁻¹ at any time during the episode. None were considered to be shocked out of proportion to the estimated blood loss. It was noted that removing the placenta increased the blood loss.

Details of 11 uterine inversions were published from a retrospective study in Rhode Island.² Eight of the 11 patients had a calculated blood loss of over 1000 ml, and three patients lost over 2000 ml. Blood loss was calculated using the formula: Loss in ml = (pre-delivery haematocrit – pre-discharge haematocrit) × 150 ml + ml blood replaced. Only one patient was diagnosed as clinically shocked, again without definition and she had a calculated loss of 4300 ml. Only three of these patients were given a blood transfusion, of between 2 and 5 u of packed red cells. These surveys do not support the classical description of shock out of proportion to blood loss. Indeed, it must be remembered that blood loss is frequently underestimated.²

Management of uterine inversion has two important components: the immediate treatment of the haemorrhagic shock and replacement of the uterus. Resuscitation should start immediately while attempts are made to replace the uterus manually. The chance of immediate reduction is between 22 and 43%.¹⁵ Six If unsuccessful, further attempts should start until the patient is haemodynamically stable.³ If possible, the placenta should be left in place to reduce bleeding.⁵ If the uterus remains inverted, contraction of the cervix may require relaxation by general anaesthesia or tocolytic therapy. Severe cases require laparotomy.⁷
Regional anaesthesia does not provide relaxation but may be helpful by providing analgesia.6 They recommended terbutaline as a drug of rapid onset and short duration. It was used successfully in five out of eight patients. In another study, terbutaline 0.25 mg was used successfully in 16 out of 18 patients, but the authors recommended that it should not be used in patients with significant hypotension and shock.5 In these patients, magnesium was used successfully in seven of eight patients. An i.v. dose of 4 G is recommended.8,9 Nitroglycerine has also been used successfully.10 A small i.v. dose of 100 μg has the advantage of a quick onset and short duration, causing little haemodynamic disturbance, and is familiar to anaesthetists in the UK. If, despite tocolytic therapy, reduction is unsuccessful, the patient will require a general anaesthetic. Surgical repositioning via an abdominal or vaginal approach may be necessary.

In summary, puerperal uterine inversion is a serious but infrequent complication of childbirth. Haemorrhage may be rapid and patients require aggressive resuscitation. The degree of blood loss is usually underestimated and the classical description of shock out of proportion to blood loss is probably due to this underestimation. Tocolysis may enable avoidance of general anaesthesia, although this remains the treatment of choice if reduction is difficult, or the patient becomes severely haemodynamically compromised.

References