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ACUTE PUERPERAL UTERINE INVERSION: CASE REPORT

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ACUTE PUERPERAL UTERINE INVERSION: CASE REPORT

E. SEQUEIRA and Y. PATEL

SUMMARY

Acute puerperal uterine inversion is a rare but potentially fatal obstetric emergency in which the uterine fundus collapses into the uterine cavity. Maternal mortality is high unless the condition is recognised and quickly corrected. The duration of time elapsed from moment of diagnosis to that of correction, along with rapid resuscitation measures are of utmost importance in its prognosis. A recent case managed successfully is described followed by a short review of the literature.

CASE REPORT

A 25 year old woman, six days beyond her expected date of delivery, was admitted in active phase of labour. She had no antecedent medical problems and had one previous normal vaginal delivery five years back. Her clinical examination and all investigations done earlier were within normal limits. Her antenatal haemoglobin level was 12.6 g/dl. Her initial vaginal examination revealed a well effaced cervix, 4 cm dilated and an amniotomy was done. Five hours after admission, following augmentation with syntocinon, she gave birth to a healthy boy weighing 2.9 kg with good Apgar scores. The delivery of the placenta was difficult despite controlled cord traction, but after about 25 minutes was felt to be in the vagina. It was delivered in toto with the membranes and was noted to have a succenturiate lobe. She passed approximately 500 ml of fresh blood with the delivery of the placenta. There was no evidence of placenta accreta. On examination, it was noted that there was a fleshy mass in the introitus, firm and bleeding. A diagnosis of an acute uterine inversion was made and assistance called for. Attempts by the obstetrician to reduce the uterus manually in the labour room were futile. No tocolytic was administered. By this time,

the total blood loss was estimated to be 700-800 ml, and the patient was pale, drowsy with a heart beat of 140 beats per minute, a thready pulse, prolonged capillary filling time and an unrecordable arterial blood pressure.

There was no obvious evidence of any neurogenic component to the hypotension, such as bradycardia or peripheral vasodilation. The patient was rapidly transfused crystalloids and she was transferred to the operation theatre for urgent repositioning of the inverted uterus.

Standard intraoperative management was instituted and rapid sequence induction of general anaesthesia was followed by endotracheal intubation and maintenance of anaesthesia with enflurane. The oxytocin infusion was stopped since the diagnosis was made. Her arterial pressure was still unrecordable despite rapid crystalloid infusion and colloid infusion as well. Four units of grouped and cross-matched whole blood were requested. The uterine inversion was confirmed to be complete by an examination under anaesthesia, and was reduced without difficulty, after relaxation with enflurane. An infusion of 20 units of oxytocin was started in 500 ml normal saline. A bolus of 0.5 mg ergometrine was also given and 250µg of Hemabate (Carboprost-15 methyl derivative of PgF₂ α) was given later to contract the

uterus while the corrected position was maintained by a hand in the uterine cavity and the other hand holding the fundus up abdominally for at least five minutes. Her arterial pressure slowly picked up from an initial 40/30 mmHg to 120/70 mmHg by the time of reversal from general anaesthesia. The patient was observed in the operating theatre for almost one hour post-procedure till the vitals stabilised to a radial pulse rate of 110/min, and a blood pressure of 120/70 mmHg.

Her post operative haemoglobin concentration estimated was 5.4 g/dl and three units of whole blood were transfused. Her post-transfusion haemoglobin was 8.1 g/dl. Her subsequent recovery was uneventful and she was discharged on the second postpartum day on antibiotics.

Her follow up visit was one week later. She was in good condition still on haematinics.

DISCUSSION

Acute puerperal uterine inversion is a rare but potentially life threatening complication in which the uterine fundus collapses within the endometrial cavity. The reported incidence varies considerably in literature, ranging between approximately one in 2000 deliveries to one in 10,000 deliveries (1-3). Maternal mortality has been reported to be as high as 15% if not corrected rapidly (3,4).

Uterine inversion is classified both by the degree of inversion as well as by the onset of inversion (2,4). The uterine fundus that is inverted and lies within the endometrial cavity without protruding beyond the external os is called an incomplete inversion (4,5). A complete inversion is one in which the inverted fundus extends beyond the vaginal introitus (4), while a total inversion involves the uterus and vaginal wall as well.

In terms of onset of the inversion, acute describes an event occurring before the contraction of the cervical ring. If the cervical ring has contracted, a subacute inversion is diagnosed. It is described as chronic if four weeks have elapsed from the event (1-4).

It is unclear why inversion occurs (4,6). The most likely cause is strong traction on the umbilical cord in the third stage of labour particularly if the placenta is fundal in position. Other related factors include Crede's method of placental delivery, excessive fundal pressure, relaxed uterus, morbidly adherent placenta especially involving the fundus, a short

umbilical cord, congenital weakness of the uterus and its supports and ante-partum use of magnesium sulphate or oxytocin (2,4,6).

The classical presentation is of an obviously displaced uterus while delivering the placenta, usually in association with post partum haemorrhage and clinical shock, usually out of proportion to the blood loss. The shock is also thought to be due to the parasympathetic effect of traction on the ligaments supporting the uterus and may be associated with bradycardia (2,4).

When there is complete inversion, the diagnosis can be easily made by palpating the inverted fundus at the cervical os or vaginal introitus, and a dimpling of the uterine fundus can be made out abdominally. Associated profuse bleeding, absence of uterine fundus or an obvious defect of the fundus on abdominal examination provide further diagnostic clues (2,4).

Clinical symptoms and examination provide the diagnosis in most cases, though radiographic methods have also been described in literature. Hsieh and Lee (7) have described sonographic findings of uterine inversion. MRI of inversion has also been reported (5). The appearance of the uterus is similar to that found in sonography. Neither of these methods is routinely advocated for diagnosis. Immediate recognition and prompt attention to its management is the key to successful management of this potentially fatal obstetric emergency, and this doesn't allow for time to confirm a diagnosis radiographically, except if uncertain of the diagnosis, with the patient being stable clinically (5,7).

There are two major components of proper management of acute uterine inversion: the immediate treatment of the haemorrhagic shock and the replacement of the uterus (4). Resuscitation should start immediately while preparations and attempts are made to replace the uterine fundus. Pharmacologic agents are needed both to assist in uterine relaxation for correction, as well as to cause uterine contraction to prevent re-inversion and to decrease blood loss once successful replacement is done.

Once inversion is recognised, all oxytocic agents should be withheld until correction is achieved. Manual correction of inversion vaginally, known as Johnson manoeuvre consists of pushing the inverted fundus through the cervical ring with pressure directed towards the umbilicus (8). The "last out — first in" principle has to be maintained to ease the correction (2,4,8). Myometrial relaxation

can be implemented using several pharmacologic agents. Commonly used are magnesium sulphate (4), terbutaline and intravenous nitroglycerine (9,10) though other agents have also been used. If correction is not achieved immediately with the use of tocolytic agents, (as would be the case if reposition is not done immediately thereby allowing a tight cervical ring to form above the inverted fundus) general anaesthesia with halothane (or similar halogenated gases) may be induced to provide adequate uterine relaxation (4). This is also the preferred route if the patient is haemodynamically unstable, because halogenated gases have fewer potential adverse effects on haemodynamics than the β -adrenergic tocolytics do (8).

O'Sullivan's hydrostatic method of reposition of the uterus is also described, in which a bag of warm saline is held up above the level of the patient and allowed to flow, via tubing, into the vagina. The pressure of the fluid, held in place by the clinician's hands over the introitus, results in correction of the inversion (4). Ogueh and Ayida (9) described a novel way of creating adequate hydrostatic pressure by maintaining a proper water seal by using an intravenous tubing attached to a silicon cup used in ventouse extraction. By placing the cup in the vagina, an adequate seal is created allowing enough hydrostatic pressure to develop to correct the inversion.

If all these attempts at manual and hydrostatic reposition of the uterus fail, surgical correction may be necessary. The two common procedures described include Huntington's and Haultain's procedures, both being abdomino-vaginal procedures (4,8). Huntington's procedure involves a laparotomy to locate the cup of the uterus formed by the inversion. Clamps are then placed in the cup as well as on the round ligaments below the cervical ring and a gentle upward traction applied. Repeated clamping and traction continues till inversion is corrected (4).

Haultain's procedure involves an incision on the posterior portion of the cervical ring to increase the size of the ring and allow repositioning of the uterine fundus (4). Occasionally, as a life saving measure, emergency peripartum hysterectomy is needed to control haemorrhage.

Recently, a successful laparoscopic reduction was reported as a new method of management of acute uterine inversion (11). Another novel technique was described using obstetric ventouse inserted into the inverted uterus via a laparotomy. A vacuum is then

created and gentle traction given to achieve reduction with minimal trauma to surrounding tissue (12).

In conclusion, puerperal uterine inversion is a life threatening obstetric complication, but fortunately rare; however, manual reposition vaginally aided by tocolysis or halogenated gases is usually successful in most cases. In recalcitrant cases, surgical correction via a laparotomy may be needed.

Most important in proper management of this obstetric emergency is rapid recognition and prompt attempts in resuscitation and reposition of the inverted uterus.

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