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Case Report

Interesting rare case of recurrent puerperal uterine inversion: a case report

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ABSTRACT

Uterine inversion is a rare obstetric emergency that may lead to severe haemorrhage, shock and eventually death. The incidence of uterine inversion varies from one in 2,000-20,000 deliveries. Recurrent uterine inversion is still rare with no exact reported incidence so far. We report a case of a near miss patient, 28 years old primiparous with complete uterine inversion with atonic postpartum haemorrhage and shock, managed by manual repositioning and tamponade insertion which got corrected. Patient had recurrent uterine inversion twice in the puerperal period on 5th and 7th postpartum day. A new approach to management was taken. Rather than going for laparotomy, vaginal manual correction followed by intrauterine tri-way foley catheter insertion was done. Gradual deflation was done leading to final permanent correction. Uterine inversion is a rare but potentially deadly complication post vaginal delivery. Mortality and morbidity can be reduced by rapid recognition and immediate replacement. For recurrent inversion prolonged intrauterine balloon placement may be needed in rare cases.

Keywords: Manual uterine re-position, Recurrent uterine inversion, Tamponade, Tri-way foley catheter, Postpartum haemorrhage, Near miss

INTRODUCTION

Uterine inversion is defined as the passage of the uterine fundus inferiorly into the uterine cavity and cervix, turning the uterus inside out and is a rare obstetric emergency that may lead to severe haemorrhage and shock resulting in maternal death. The incidence of uterine inversion ranges from one in 2,000 to 1 in 20,000 vaginal deliveries.[1] Immediate active management of uterine inversion is recommended as the massive and often underestimated blood loss is reported to be fatal in as much as 15% cases.[2] Based on timing, uterine inversion can be classified into 3 – acute (within 24 hours of delivery), subacute (>24 hours but <4 weeks) or chronic (>1 month postpartum). Uterine inversion can be puerperal or non-puerperal. The treatment includes immediate uterine repositioning, fluid resuscitation and control of haemorrhage to restore maternal hemodynamic stability. Manual replacement under general anaesthesia is usually successful. If the manual replacement fails, surgical replacement by laparotomy needs to be performed.

CASE REPORT

A 28-year-old primipara female, 4 hours post-partum, was referred to the emergency department from district women hospital with history of complete uterine inversion, postpartum haemorrhage and hypovolemic shock with A-negative blood group. On arrival at the emergency department, the patient was anaemic and unresponsive with active vaginal bleeding. Physical examination revealed blood pressure non recordable, tachycardia (178/min) and tachypnoea (26/min) with severe pallor, cold and clammy extremities and prolonged capillary refill

time (>3 seconds). On per abdomen examination uterine fundus was not felt and per speculum examination revealed active bleeding with cervical stitches in entire circumference and uterine fundus was felt through the os on per vaginal examination. Immediate blood workup at time of admission was performed which revealed anaemia and leucocytosis. Normal thrombocyte level was found with deranged coagulation profile (Table 1).

Table 1: Blood workup at admission.

Parameters	Values
Haemoglobin (g/dl)	6.9
Haematocrit (%)	18.1
Leucocytes (cells/ul)	39300
PT (seconds)	45.2
INR	3.28

The patient was treated by fluid resuscitation with crystalloids, vasoactive drugs and blood transfusion. We successfully performed manual repositioning of the uterus under general anaesthesia followed by tight intrauterine and vaginal packing. Post procedure she was kept on oxytocin drip. The patient was stabilized. Post-transfusion of 4 packed red cells and 2 units fresh frozen plasma, blood investigations were repeated which showed haemoglobin of 7.1 g/dl, haematocrit 19.2%, erythrocyte 2.24 million/ul, leucocyte 18,700/ul and thrombocyte 1.96 lac/ul. Patient's haemodynamics became stable and anti-D injection was given. On 3rd day postpartum packing was removed and ultrasound revealed normal contour of the postpartum uterus. Re-inversion occurred on 5th postpartum day, uterine contour was not felt and uterine fundus was felt through the os. Condom catheter tamponade was inserted inflated with 350 ml of normal saline and vaginal packing done. Tamponade was deflated and on 7th day postpartum, on removal of packing, there was re-inversion, no further intervention was done, she tested covid positive and was shifted to COVID dedicated hospital for management. She was discharged with oral antibiotics and pain medications.

Patient got readmitted with recurrent uterine inversion on 26th day postpartum. Transvaginal sonography revealed uterine inversion with thickened endometrium (Figure 1).

Manual repositioning was done under spinal anaesthesia followed by tri-way foley catheter insertion inflated with 150 ml of normal saline and tight vaginal packing. Postoperatively she was kept on uterotonics and broad-spectrum antibiotics. Tri-way foley catheter was gradually deflated by 10-20 ml over a period of 7 days. Post procedure ultrasound revealed normal contour of the uterus. Postoperative period was uneventful and she was discharged on 16th postoperative day in good condition. Follow-up postnatal visit after 2 weeks revealed normal involuted uterus. At her follow up visit at 6 weeks, the patient had resumed her menstruation and her local examination revealed no abnormality.

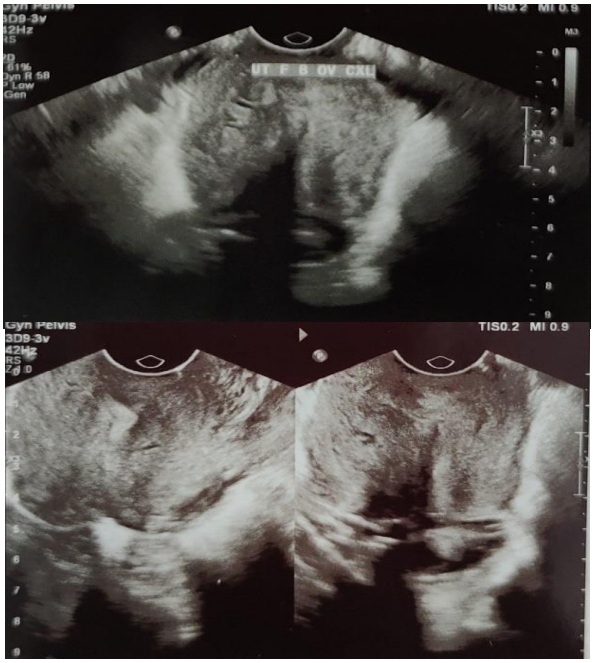

Figure 1: Ultrasound TVS with F/S/O deformed uterine fundus and non-visualisation of cervix s/o uterine inversion.

DISCUSSION

The exact etiology of uterine inversion remains elusive, however strong traction of the umbilical cord particularly during the third stage of labour when the placenta is in a fundal location is hypothesized to be the most likely cause. Other factors include relaxed uterus; primiparity; uterine fibroids; placenta accreta; excessive fundal pressure; short umbilical cord; congenital weakness or uterine anomalies; Ehlers-Danlos syndrome and foetal macrosomia as well as use of magnesium sulphate and oxytocin are risk factors for uterine inversion.[2]

The pathophysiology of acute uterine inversion involves relaxation of part of the uterine wall which causes a part of the wall to prolapse through the dilated cervix along with simultaneous downward traction on the uterine fundus leading to inversion of the uterus.[3]

The clinical presentation of uterine inversion differs according to its degree and timing. Incomplete uterine inversion may be subtle clinically while complete inversion often presents with profuse vaginal bleeding and maternal hemodynamic instability. Clinical diagnosis is made using bimanual examination wherein the uterine fundus is palpated in the lower uterine segment or within the vagina. Ultrasonography is useful to confirm the diagnosis when clinical examination is suspicious but unclear.[2]

The treatment includes immediate uterine repositioning, fluid resuscitation and control of haemorrhage to restore maternal hemodynamic stability. Uterine repositioning can

be performed either by Johnson manoeuvre or by hydrostatic method. Johnson manoeuvre is a manoeuvre wherein manual pressure is applied on the fundus through the vagina. It must be carried out as soon as possible to minimize blood loss and increase chances of resolution due to the involution of the cervix which induces a rigid ring that makes restoration of normal uterus position difficult.[4] Hydrostatic reduction was originally described by O'Sullivan in 1945 wherein pressure of warm fluid infused into the vagina was used to achieve reduction. If the placenta has not separated before the replacement operation is attempted, the best plan is to leave it undisturbed until the patient is in the operating room to minimise the blood loss.

Other conservative interventions include administration of utero-relaxant drugs. Magnesium sulphate, terbutaline and salbutamol are most commonly prescribed due to their availability and frequent use. General anaesthesia with halothane, isoflurane, desflurane and sevoflurane may be used particularly in patients with hemodynamic instability.[5]

Some reports suggest the use of balloons and obstetric vacuum placed intravaginally to increase pressure on the uterine fundus to push it to its original position. When conservative management fails, it is essential to perform a surgical intervention. Several techniques have been described – Huntington, Haultain, Spinelli and laparoscopic with the first 2 most commonly reported.[4] Rarely as a life saving measure peripartum hysterectomy maybe performed to achieve control of haemorrhage. A case report of successful management of recurrent puerperal uterine inversion was managed first by Johnson's manoeuvre for acute inversion and for recurrent uterine inversion exploratory laparotomy was done to reposit by Huntington technique (progressive upward traction on each round ligament by application of atraumatic clamps with simultaneous upward pressure transvaginally) and multiple Cho suture; bilateral uterine arteries and utero ovarian arteries were ligated prophylactically. Vaginal pack with roller gauze kept prophylactically to prevent reinversion.[6] Soleymani et al and Elósegui et al have reported first successful management of recurrent uterine inversion using surgical obstetric silicone bakri balloon.[7,8] Sharma et al and Kabir et al have reported acute uterine inversion managed by Johnson's manoeuvre; then subacute uterine inversion by Haultain's operation where cervical ring is posteriorly incised to facilitate uterine replacement by Huntington method.[9,10]

After repositioning of the uterus administration of uterotonic agents (oxytocin or misoprostol) is essential to prevent recurrence. Broad spectrum antibiotics are also recommended to prevent endometritis or sepsis.

Our case highlights the role of conservative management in uterine inversion. The combination of a fundal implanted placenta, flaccidity of the myometrium around the implantation site, and a dilated cervix is thought to predispose to puerperal inversion. In this case, it can be assumed that she had an intrinsic flaccidity of the myometrium. The possible cause of recurrence in this case could have been fundal myometrial weakness or placental defects. This case was unique as all standard protocols were followed after reposition earlier 2 times but still got inverted, during third time correction, gradual deflation over a period of time was done which gave time for remodelling of myometrium, leading to permanent correction.

CONCLUSION

Uterine inversion is a rare but potentially deadly complication post vaginal delivery. Its low incidence leads to sparse experience among health professionals in managing this obstetrical emergency. Early fluid resuscitation, manual reposition and balloon tamponade is essential in order to obtain the best prognosis. If the manual replacement fails, surgical replacement by laparotomy needs to be performed. In rare case of recurrent inversion prolonged intrauterine balloon placement using tri-way foley followed by gradual deflation may be an effective management strategy.

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