Case Report

Acute Puerperal Uterine Inversion: A Case Report and Narrative Study

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Abstract

The postpartum uterine inversion represents a life-threatening emergency mostly occurring in the third stage of labour and burdened with the high morbidity. The work is comprehensively devoted to the issue of acute inversion of the uterus arising immediately after the childbirth. A rare case report of acute partial puerperal inversion is described in detail. Moreover, a literature review of the papers published from 1872 and meeting an inclusion criterion according to PRISMA guidelines is presented. The critical parameters related to the acute postpartum inversion of the uterus are evaluated in a detailed analysis.

INTRODUCTION

The uterine inversion (UI) represents a rare, life-threatening emergency described as a collapse of the uterine fundus into the endometrial cavity, cervix, vagina, or even beyond the vaginal introitus. The incidence of UI reported in the literature varies from 1:2000 to 1:23000 childbirths [1].

The inversion of the uterus might be classified according to either degree of fundal descent or the timing of the occurrence. First degree of inversion is described as fundus reversal into the uterine cavity. If the uterine fundus reaches the external cervical os, the conditions for the second degree are met. The third degree of UI is characterised by fundus reaching the vaginal introitus and when the uterine fundus is beyond the introitus, it is defined as the fourth degree inversion [2]. The UI may also be described as acute if occurring within 24 hours of labour; subacute appearing between 24 hours and 4 weeks of delivery and chronic if appears after 4 weeks of labour or in non-pregnant women. It is most observed in the postpartum period as an acute UI, although may also be of non-puerperal origin [3].

The aetiology of puerperal UI is still unclear; however, it may often be associated with uncontrolled cord traction or excessive fundal pressure during the third stage of labour [4]. The known risk factors include foetal macrosomia, previous UI, nulliparity, uterine anomalies, difficult removal of the placenta, placenta accrete, precipitous delivery, short umbilical cord, ligaments laxity, and the use of uterine relaxants such as magnesium sulphate [5]. The non-puerperal UI may most commonly be caused by submucosal leiomyomas, but the potential causes also include endometrial polyps and carcinomas [6].

The diagnosis of acute puerperal UI is mostly clinical, frequently suspected in case of lower abdominal pain, massive loss of blood after labour, or absence of the uterine fundus during bimanual abdominal palpation, with the greatest diagnostic difficulties prompted by the first degree inversion. In the case of the fourth degree inversion, a mass is observed beyond the vaginal introitus. The UI may be followed by neurogenic shock and subsequently by haemorrhagic shock due to the uncontrolled bleeding. In a patient with inconclusive physical examination, an ultrasound examination may confirm the suspected diagnosis by featuring hyperechoic mass in the cervix or vagina, with a hypoechoic cavity in the centre [5].

Successful management is dependent upon quick recognition of the pathology. Once the UI is diagnosed, uterotonic drugs must be discontinued immediately. The initial approach should include the so-called Johnson manoeuvre, which represents a manual correction of the inverted uterus to its correct position. The correction is performed by placing a hand inside the vagina and pushing the fundus along the vaginal long axis towards the umbilicus. Delayed manoeuvre increases a blood loss and worsens the chances of a full resolving. Tocolytic drugs can facilitate the restoration of the fundus to its position [7]. Another method for correction of UI is hydrostatic reduction, wherein the correction is achieved by the pressure of fluids infused into the vagina [8]. If the conservative procedures fail, surgical management is indicated. There are two procedures...
Huntington's operation consists of locating the cup formed by UI, clamping both round ligaments entering the cup, and pulling the clamps, provoking upward traction of the inverted fundus. Haultain procedure is based on incising the posterior surface of the uterus in order to bisect the constriction ring of the myometrium that is preventing reduction of the inversion [9]. If the placenta is still adherent to the uterus, the inversion should be resolved before removing of the placenta to limit further haemorrhage. Moreover, supportive therapy is crucial for the prevention of shock development.

Neurogenic and subsequently haemorrhagic shock are the main factors of accompanying morbidity and mortality. The essential conditions for a successful outcome of UI are prompt diagnosis and immediate treatment [10].

**CASE REPORT**

A 25-year-old, primigravida, white race at 39 weeks and 1 day of gestation presented with regular uterine contractions on May 5, 2019. Her medical history was burdened only by psoriasis. The patient also reported uterine myoma, however this was not in the documentation founded. The course of pregnancy was uneventful, her only medication was a daily prenatal vitamin and final body mass index (BMI) was 26.93 kg/m². She was admitted to the hospital at 12.10 pm with 4 cm cervix dilatation. Uncomplicated first stage of the labour reached full cervix dilatation at 2.25 pm. The patient did not cooperate adequately during the second stage of labour. Due to this fact, Kristeller manoeuvre, consisting of fundal pressure was used to complete the spontaneous labour at 2.35 pm. Alive fetus, female, had a birth weight 3500g, 51 cm and Apgar score 8, 9, 10. The fundal pressure as well as controlled cord traction was not in placental stage applied. The delivery of the placenta occurred spontaneously at 2.50 pm, however amniotic sack remained firmly attached to the uterine wall. Manual examination revealed a solid mass in the uterine cavity where the amniotic sack was attached. Partial UI was the first point of differential diagnosis but due to anamnesis a suspicion of myoma has been expressed and manual removal of amniotic sack under anaesthesia was indicated. The bleeding under the effect of uterotonic drugs was not profuse and blood loss was counted to 300-400 ml. The anaesthesia was induced at 3.10 pm and manual removal of firmly attached amniotic sack was performed. The solid mass was constantly palpable behind the cervix and the bleeding continued. Due to the misdiagnostic (myoma) the oxytocin was applied intravenously (10 IU) and prostaglandins intracervically (Prostin 15M) to control the bleeding. Transabdominal ultrasonography revealed 9 cm mass located presumably on the posterior wall of the uterus supporting the diagnosis of submucosal leiomyoma. However, the bleeding continued and the signs of neurogenic shock, mainly hypotension and bradycardia occurred. The diagnosis of partial UI was concluded. The application of oxytocin was stopped. Due to the accompanied symptomatology with blood loss approximately 1500 ml the surgery was indicated. Suprapubic laparotomy was performed at 3.30 pm and partially inverted uterus was found. The deep of fundal eversion was approximately 7-8 cm (Figure 1).

Huntington's operation was successfully performed but subsequently uterine atony has complicated the situation. The effect of uterotonic and uterokinetics was not seen and the blood loss reached 2500 ml. Thus, a radical surgery was indicated. Supracervical hysterectomy with bilateral salpingectomy was performed. The patient received during the operation a supportive therapy also including vasopressor support, two blood transfusions and two plasmas. She was transferred to anaeesthesiology department immediately after the operation. Another 5 blood transfusions were given during the first 24 hours after the operation. Day 3 after the operation was patient given back to gynecology department and normal postoperative period was seen.

Histopathology finding showed placenta accreta and multifocal direct adherence of amniotic sac into the uterine myometrium with the complete absence of decidua (Figure 2).

**LITERATURE REVIEW**

Presented narrative literature review was performed according to PRISMA guidelines. After determining search
strategy, PubMed and Web of Science online databases were used to identify relevant articles. The electronic search strategy was performed on 27th of December 2021, using phrase: (“acute” OR “puerperal” OR “postpartum”) AND (“uterine” OR “uterus”) AND “inversion”. A total of 898 articles were identified upon the first search. PRISMA flowchart of the screening process is presented in Figure 3. The exclusion criteria were as follows: articles in language other than English, non-puerperal uterine inversion, no full-text available for analysis and articles that were not a case report.

Following the revision of all the articles included in the review, we focused on parameters such as: the age of patients, number of pregnancies, mode of delivery, previous medical history, clinical features, possible causes of the uterine inversion, treatment, the need for hysterectomy and the outcome.

Initially, 898 papers were identified and after eliminating the duplicates a total of 531 articles were obtained. Moreover, 270 documents were excluded due to irrelevancy of either the title or the abstract. After second revision of 261 papers, we excluded 175 studies not meeting our criteria. Another revision of obtained articles left a total of 67 studies with 98 patients to be analysed. They were published from 1872 to 2020. All the articles were independently reviewed by two gynaecologists. A conflict of interest was ruled out for both, as neither was the author or co-author of any work.

5.1. Age

The age of the patients in whom acute puerperal UI was reported varied from 17 to 42 years. The age of two patients was unknown. The mean age of patients was 27.94 years.

Number of Pregnancies

Out of 98 patients, 41 were primipara (n=41), 46 were pregnant twice or three times (n=46) and 8 women were pregnant 4 or more times (n=8). Two patients were labelled as “multipara” (n=2). The status of one patient was not mentioned in the text (n=1) (Graph 1).

Mode of Delivery

The delivery of most patients was spontaneous vaginal (n=81). Thirteen patients had operative vaginal delivery, including forceps delivery (n=9) or vacuum extraction delivery (n=3), and one patient had both vacuum and forceps used (n=1). Four out of 98 patients had Caesarean Section performed (n=4) (Graph 1).

Previous Medical History

Out of all patients, two women had a history of uterine inversion (n=2). Five patients had previous placental retention (n=5). In one patient previous delivery was complicated by haemorrhage (n=1). One patient had undergone a sterility treatment before conceiving (n=1) and one woman had a history of endometrial polypectomy performed by transcervical resectoscope (n=1). One patient suffered from systemic lupus erythematosus (n=1).

Clinical Features

The haemorrhage was the main sign of UI presented in studied patients. It was described in 93 out of 98 patients. Severe haemorrhage leading to haemorrhagic shock was in 57 patients seen. The main described manifestations were as follows:
tachycardia, weak pulse, hypotension, paleness, restlessness, tachypnoea, clammy skin, or prolonged capillary refill. Lower abdominal pain was reported in 12 patients. The clinical manifestation in one patient was not reported (Graph 2).

Possible Causes

For the most common possible cause of UI an umbilical cord traction was reported (n=38). In 35 out of 98 patients the possible reason of the inversion was described as “unknown” (n=35). The fundal pressure was reported to be the possible cause of UI in 18 patients (n=18). The placental pathology was as the reason of UI identified in 14 cases (n=14). Namely, those pathologies were defined as follows: placenta accrete or placenta adherent (n=7), manual placental extraction (n=3), succenturiate placenta (n=2), placental retention (n=1), placenta increta (n=1). Among other possible causes, the authors enumerated also short umbilical cord (n=2) and prolonged methoxyflurane-oxygen anaesthesia (n=1). In some patients more than one possible cause was indicated (Graph 3).

Treatment

One patient (n=1) had successful vaginal uterine reposition done using ring-forceps.

Laparotomic reposition was used as the first-line treatment in 7 patients (n=7). In two of them due to insufficient effect the hysterectomy was unavoidable (n=2). In one patient after unsuccessful laparotomic reposition a balloon catheter was inserted instead (n=1). In 4 cases laparotomic reposition was without a lapse performed (n=4).

Hydrostatic reposition was as the initial treatment used in nine cases (n=9). Out of these, one patient had vaginal packing used to prevent the recurrence of the inversion (n=1). In one patient the manual reposition followed an unsuccessful hydrostatic reposition (n=1). Laparotomy was performed in another 2 patients (n=2). In one of them the laparotomic reposition was performed, while in another one a hysterectomy represented a final therapy. Finally, the rest 5 patients profit from hydrostatic reposition (n=5).

Manual reposition was the treatment of choice in the great majority of patients (n=79). In 34 women, manual reduction only was sufficient, and no other procedures were required (n=34).

However, most of the women needed further measures to be taken in order to correct the UI. Fifteen patients had hydrostatic reposition done following the manual reposition (n=15). In one of these patients the uterine pack was applied to prevent further UI and in another 3 patients the hydrostatic reduction failed, and manual reposition was done once more, which was then successful.

In 18 cases, laparotomy was performed following the failed manual reposition (n=18). Out of these, the hysterectomy was performed in 4 women. The indication was persistent severe bleeding and perforation of uterine fundus with gangrene. One patient after hysterectomy died of cardiopulmonary arrest due to postpartum haemorrhage. Hysterectomy in one patient was performed straight after failed manual reposition. In eight cases successful reposition during laparotomy using a forceps was performed. Another one required further application of B-Lynch suture and bilateral hypogastric artery ligation to stop continuous blood loss. Additionally, one person had uterine artery embolization followed by hysterectomy and another had an intra-aortic balloon occlusion to achieve haemostasis followed by hysterectomy. Three patients had Hayman suture applied to the uterus during laparotomic intervention.

In seven cases, a successful manual reposition was followed by application of balloon catheter and in two patients by application of uterine pack to prevent a reinversion. Moreover, one patient, in whom manual reduction failed, had a balloon catheter applied to correct the UI. Rarely, in 2 patients a successful reposition of the uterus was performed under laparoscopic guidance, following failed manual reduction (Table 1).

The Need for Hysterectomy

In 9 out of 98 patients, the hysterectomy was required (n=9). In two of them, the hysterectomy was performed after unsuccessful laparotomic reposition. One patient had hysterectomy after failed hydrostatic reposition. Four patients underwent hysterectomy after unsuccessful manual reposition as the first line therapy. In the rest two patients the hysterectomy was performed after manual reposition with combination of uterine artery embolization or intra-aortic balloon occlusion.
Table 1: Management of the patients with acute puerperal UI (HYE = hysterectomy, LPT = laparotomy, LSK = laparoscopy).

<table>
<thead>
<tr>
<th>PROCEDURE No of CASES</th>
<th>SUCCESSFUL</th>
<th>UNSUCCESSFUL</th>
<th>FURTHER MANAGEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ring forceps 1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No attempts 2</td>
<td>0</td>
<td>2</td>
<td>Death</td>
</tr>
<tr>
<td>Laparotomy 7</td>
<td>4</td>
<td>3</td>
<td>HYE - 2</td>
</tr>
<tr>
<td>Hydrostatic reposition 9</td>
<td>5</td>
<td>4</td>
<td>Vaginal catheter – 1</td>
</tr>
<tr>
<td>Manual reposition 79</td>
<td>34</td>
<td>45</td>
<td>Hydrostatic reposition – 15</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>LPT – 10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Balloon catheter/uterine pack – 10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>LSK - 2</td>
</tr>
</tbody>
</table>

Outcome

Three out of 98 patients (n=3) died due to the complications of acute puerperal UI.

The first of them was published in 1931. The birth at home was complicated by UI and severe haemorrhage. The patient died approximately 60 minutes after delivery of the fetus due to the haemorrhagic shock. No intervention was performed because outdoor obstetric assistant arrived shortly before her death.

In the second case, published in 2014, manual removal of the placenta was reported as the possible cause of UI. No intervention was then performed and 42 years old tertipara died after the cardiopulmonary arrest due to the haemorrhagic shock.

Another patient was described 2014. In 19 year old women after manual reposition of the inverted uterus an atony with persistent haemorrhage occurred. During the emergency laparotomy a supracervical hysterectomy was completed under the constant external heart massage. An autopsy of the patient revealed massive air embolism as the complication of manual reposition.

The other 95 patients have fully recovered.

DISCUSSION

The inversion of the uterus usually represents an emergent situation in a medicine. A typical feature is the sharp deterioration of the condition when the intervention is postponed. Relatively wide incidence, 1:2000 to 1:23000 childbirths, varies depending on geographical region with one of the highest incidences in India and lowest in the Europe [1]. There are no multi-centre studies and the literature consist of only case reports, small single-centre studies, and a single nationwide study from the Netherland [11]. The decrease in the maternal mortality is in the last years observed. The lowest mortality is reported in high-resource countries, however in low-income countries postpartum deaths of women are still reported [12].

The introduction of active management of the third stage of labour decreased the UI incidence by 4 time [1]. The active management consists of a group of interventions, including prophylactic uterotonie, cord clamping and cutting, controlled cord traction and uterine massage. Which of the processes has the highest impact for decreasing of UI incidence is discussed. The main negative influence can be in controlled cord traction and uterine massage seen. Controlled cord traction should be applied after the uterus has contracted sufficiently and with application of effective fundal counterpressure. It requires practical training to achieve an adequate level of skills to perform the procedures safely. Excessive fundal pressure or umbilical cord traction may result in increased risk of UI. Based on the presented analysis both conditions covered up to 52% of all aetiologies. Thus, if it is possible to omit controlled cord traction and uterine massage from active management of the third stage without losing efficacy, this would significantly decrease an incidence of UI. More transparent situation is seen in caesarean section. Cord traction under the visual control can be used for delivery of the placenta with an accepting risk of UI [13].

The level of fundal descent defines the degree of uterine inversion (first to fourth degree) [2]. This clear anatomical classification may be simplified in two terms. Partial UI describing the fundal descent up to vagina (first three degrees of inversion) and complete UI covering fourth degree of inversion. Similar anatomical classification defines the UI into three degrees. Incomplete UI when fundal inversion does not herniate through the level of the internal cervical os. If the internal lining of the uterine fundus passes through the cervical os with no palpable fundus abdominally, the criteria for complete UI are met. Finally, prolapsed UI is defined as prolaps of the uterus with fundus passing out of the introitus [1]. The diagnosis of this most severe degree of UI is not difficult. However, the diagnosis of degree I to III can be confusing, especially when inverted fundus does not reach cervical os.

In 1951 Jones classified inversion of the uterus into two types: puerperal or obstetric and non-puerperal or gynaecological [14]. Puerperal UI is seen following labour or miscarriage and may be acute or subacute. Non-puerperal, or chronic UI is mostly related to benign or malignant processes originating from uterine corpus. Generally, most of the cases of UI are puerperal, occurring immediately after delivery as acute UI. Non-puerperal UI is less common and usually presents as chronic case, although sudden onset was also reported [15]. Subacute puerperal UI is extremely rare, and the incidence reaches less than 3% [16]. The clinical severity of the situation usually accelerates as soon as the UI occurs after delivery. Here we reported a typical puerperal acute UI presenting as life-threatening emergency. All the 98 women from review met a criterion for acute puerperal UI and in all the cases a severe clinical symptomatology was seen.

The aetiology of uterine inversion, a certain risk factors may be defined, however in significant group of patients the aetiology is not recognized [17,18]. In reported review 35 out of 98 patients were not concluded with the reason of UI, which represents 35.7%. This can be explained by subjective perception
of umbilical cord traction or fundal pressure as not excessive. This supports a conclusion that avoiding of umbilical cord traction, even if controlled and uterine massage for delivery of the placenta may significantly decrease the incidence of UI. Iatrogenic risk factors include mainly excessive umbilical cord traction and rough Credé placental expression. Both, they covered altogether 51.85% of aetiologies in presented review. Even, in some patients as the reason of UI both conditions were mentioned. As the main endogenous risk factors, placental pathologies were mainly suggested. This covers pathological implantation of placenta, fundal localisation, retention of the placenta as well as manual removal of the placenta. Pathology of the placenta, mainly its implantation, as the cause of UI represented 12.96% of all reasons of UI in studied patients. This was also the aetiology of presented case report. Rare histology, placenta accreta and multifocal direct adherence of amniotic sac into the uterine myometrium with the complete absence of decidua was seen. There is no such a case of UI described in literature yet. Other risk factors include the usage of uterine-relaxing agents, uterine overdistension, fetal macrosomia, multiple pregnancy, polyhydramnios, nulliparity, coiling of the umbilical cord, excessively short umbilical cord, connective tissue disorders [Marfan syndrome and Ehlers-Danlos syndrome]; however, these causes are rare [1]. Wrong short thought about uterine myoma as the reason of neurogenic shock in presented patient was not correct. By reviewing the literature, the myoma was not as the cause of puerperal acute UI described. Thus, the uterine myoma may be removed from the differential diagnosis of postpartum neurogenic shock as well as from the cause of puerperal acute UI. However, an underestimation of the situation may cause that UI is misdiagnosed as a submucosal myoma.

Typical clinical feature of acute UI is rapid deterioration of symptoms if the intervention is postponed. The clinical symptoms are mentioned in the literature consistently. It includes a sudden onset of vaginal bleeding leading to haemodynamic instability and haemorrhagic shock [19]. However, the severity of shock usually does not correlate with the blood loss, especially immediately when the UI occurred. At the beginning, non-significant blood loss is accompanied by rapidly progressing shock. The reason is the share of neurogenic shock arising due to parasympathetic stimulation caused by the stretching of the inverted tissue. Neurogenic shock is defined by the haemodynamic triad which includes hypotension, bradycardia, and peripheral vasodilatation. Symptoms of haemorrhagic shock will appear usually later, after repositioning of the uterus or placental detachment. Other symptom represents abdominal pain, which is reported less often. The explanation may be due to severe shock with the overall alteration of the patients’ condition when the patient is not complaining the symptom [1]. In the presented patient a typical neurogenic triad was the initial clinical symptom. Brisk deterioration of neurogenic shock together with non-profuse bleeding was supported by uterotonic and uterokinetics given initially due to misdiagnosis by uterine myoma. Significant haemorrhage occurred after laparotomic reposition of the inverted uterine fundus. The patient did not complain to abdominal pain at all. The haemorrhage was the leading symptom described in the patients included in the review. Significant bleeding with haemorrhagic shock was reported in 58% of patients and abdominal pain only in 12%. Surprisingly the neurogenic shock was not as the clinical symptom mentioned.

The diagnosis is usually made clinically with a bimanual examination. Fourth degree of UI does not represent an issue in process of differential diagnosis. Whole inverted uterus and vagina is seen beyond the vaginal introitus. Over the time, a swelling of the tissue appears. Bimanual examination can reveal a palpable fundus in vagina when third degree of UI is present, or in cervix when second degree occurs. The equivocal diagnostic may occur mainly in case of the first degree of UI. The absence of the fundus revealed by transabdominal palpation expresses suspicion to UI. In incomplete UI the ultrasonography examination can also help with the correct diagnosis. Transabdominal approach can confirm two signs of UI, “target sign” and “pseudostripe sign”. “Target sign” can be obtained by transabdominal transverse sonogram and is defined as hyperechoic inverted fundus surrounded by hypoechoic fluid between the fundus and vaginal wall. By movement of the probe to sagittal view of the uterus a “pseudostripe sign” can be seen. It is defined as uterus with the endometrial pseudostripe represented by the two opposing serosal surfaces [20].

The management of the acute UI requires an accelerated interventions as delay of treatment could lead to significant maternal morbidity and mortality. The reposition of the uterus and haemostasis are the main goals of the treatment and more options are available to achieve it. First step should be always the fundal reposition followed by haemostasis, as the first to be reversed is neurogenic shock. Therefore, uterotonics and uterokinetics must be stopped and tocolysis should be given, usually magnesium sulphate. Second large-bore cannula in opposite hand is placed for colloids to combat hypovolemia. If the placenta is still attached, it is usually not removed and reposition is performed with attached placenta. Manual reposition, so-called Johnson manoeuvre is the quickest way how to perform uterine correction. It does not require any instruments or anaesthesia and can be performed immediately after the oxytocin administration stopped. Successful rate is reported to be 43-88% [21]. However, limited data are available. Successful procedure covers not just the replacement of the uterine fundus into its correct position, but also when the reinversion will not occur. Majority of the patients from presented study had Johnson manoeuvre as the first line therapy, 79 out of 90 patients. The successful procedure was only in 43% of the patients seen. The rest of women, 57% were consequently treated by laparotomy or hydrostatic reduction or by using intrauterine balloon catheter. The acute puerperal UI represents a rare indication for laparoscopy. Haemodynamically stable circulation is inevitably needed to perform the procedure. Most of the cases of acute puerperal inversion are clinically defined as the pereacute, thus laparoscopy is contraindicated. This is in opposite to non-puerperal UI where haemodynamically stable patient is seen, and laparoscopy can be the approach of choice [22]. Another non-invasive procedure used to reposition of uterine fundus is hydrostatic reduction, so-called O’Sullivan
technique. The patient must be in Trendelenburg position under anaesthesia and under the effect of uterine relaxants. Inverted uterus with or without placenta is given into vagina. Ventouse cap is placed into vagina as a fluid retainer, and warm sterile saline localized 150 cm above the vagina is given to correct an inversion [23]. Successful rate is reported to be 80% [24]. The disadvantage of the procedure is the need of instruments, anaesthesia, and the skills of the obstetrician. Hydrostatic reposition as the first line therapy was used in 9.18% of UI patients from presented review. This is in concept of more difficult preparation needed for procedure. In 4 patients the manoeuvre failed and the rest, 5 patients profited from the procedure. The success of the technique also confirms another parameter. In one third of patients, where manual reposition as the first line therapy failed, the hydrostatic reduction was used to correct an inversion. Only 20% of them needed another management and 80% of patients did not need any other intervention. Thus, successful rate of O’Sullivan technique for UI correction reaches higher numbers comparing to Johnson manoeuvre. On the other hand, O’Sullivan technique needs more time to be performed.

The combination of procedures and postponing the final settlement might rapidly increase maternal mortality. The failure of manual or hydrostatic reposition indicates laparotomy. There are several techniques available to correct an inverted uterine fundus. The first choice is reported to be Huntington’s technique. If this procedure fails, the incision of the cervical ring along the vertical plane should be done. Anterior incision is called as the Ocejo technique and posterior incision is defined as Haultain technique [25]. Posterior incision is preferred due to lower risk of bladder injury [26]. By incision of the ring the additional space for manual reposition or Huntington’s technique will appear. The deepening of the bleeding due to uterine atony may be after the fundal reposition seen. When the treatment by uterokinetins and uterotonics fails the suture compression of the uterus can be done. There are more types of sutures described in the literature. However, Hayman suture or B-Lynch suture and Matsubara-Yano compression are preferred for prevention of reinversion [27]. If all the conservative techniques fail supracervical hysterectomy represents a radical, life-saving procedure.

Presented case report was successfully managed by Huntington’s technique. However, uterine atony with severe bleeding not reacting to medical therapy occurred. Supracervical hysterectomy with bilateral salpingectomy was finally performed as the salvage therapy.

The laparotomy as the primary treatment of UI in reviewed group of patients was performed in 7.14% of cases. Altogether 27.55% of the patients with acute puerperal UI had laparotomy in their management performed. Finally, in 9.18% of UI patients was the hysterectomy as definitive treatment used. The results support the need of sufficient erudition of obstetricians with the laparotomy treatment of UI.

Regarding the results of the review, the following can be defined as the most optimal management of the acute UI. As the first step to perform manual reposition after interruption of the uterotonics administration. If reinversion occurs and the patient does not show the signs of haemorrhagic shock hydrostatic reduction may be a following step. If both methods fail the laparotomy should be indicated. A conservative procedure followed by uterine suture can be method of choice. Insufficient success of the methods as well as haemodynamically instable patient represent an indication for radical surgery.

CONCLUSION

The acute puerperal inversion of the uterus represents an emergency affecting the maternal mortality mainly in developing countries. The omission of the controlled cord traction and uterine massage from the active management of the third stage of labour can markedly decrease the incidence of UI. Endogenous risk factors are focused to placental pathology. The neurogenic triad is the first sign of UI. Postpartum haemorrhage usually deepens after repositioning of the uterine fundus. Fast recognition of the pathology by bimanual examination and ultrasonography are the key points in the process of the management. Manual reposition is the quickest manoeuvre for treatment. Hydrostatic reduction has a high rate of success, however longer time and skills are needed for procedure. The laparotomy should not be delayed if conservative procedures fail.

DECLARATIONS

Ethics Approval and Consent Participate

The patients agree with the participation (confirmed in consent form).

Consent for Publication

The patient agrees with the publication of the data in presented form (confirmed in consent form).

REFERENCES


