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Case Report

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Histopathic Uterine Laceration, Cervix, Vaginal and Bladder Ruptures in a Multiparous Woman During Impetuous Labor

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BACKGROUND Uterine lacerations are rare but one of the hardest and most dangerous obstetric aftereffects for life of a woman and a baby. Uterine sidewall altered by a scar after caesarean section or laparoscopic myomectomy is recognized as the leading cause of uterine rupture. In turn, histopathic laceration of the uterus during labor, associated with rupture of adjacent organs in a woman with a history of normal childbirth, seems casuistic and deserves discussion.

THE AIM OF THE STUDY To present a clinical case of non-operated uterine laceration of a multiparous woman in impetuous labor.

MATERIAL AND METHODS A multiparous woman, 35 years old, with histopathic uterine laceration, ruptures of cervix, vagina, and bladder with conization of the cervix in anamnesis in impetuous labor.

RESULTS The article describes a clinical observation of histopathic uterine laceration in labor.

CONCLUSION A favorable outcome with an organ-preserving surgery in case of histopathic uterine laceration is possible only if the diagnosis of the complication is done timely, and high-quality surgical treatment is carried out within a short space of time. The uterus after the history of cervical conization must be treated as carefully – due to its possible rupture – as the uterus altered by a scar after surgical interventions. Manual examination immediately after impetuous labor with a third-degree cervical rupture would allow for timely diagnosis of uterine laceration, prevention of blood loss and the performance of organ-preserving surgical intervention.

Keywords: non-operated uterine laceration, histopathic uterine rupture, impetuous labor, cervical laceration, bladder rupture
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BP — blood pressure
ECD — electrocoagulation diathermy
RL — rapid labor
UR — uterine rupture
WHO — World Health Organization

INTRODUCTION

Uterine rupture (UR) is a rare, but one of the most severe and always life-threatening complications for women and children in obstetric practice, often accompanied by massive blood loss, leading to hysterectomy. UR is classified as a severe maternal complication according to the criteria of "near miss" ("almost lost" or "almost dead"), and occupies the third position in the structure of obstetric complications associated with bleeding [1–3].

According to the World Health Organization (WHO), UR occurs in 5 to 7 cases per 10 thousand births, while its incidence varies significantly: from isolated cases in Australia, Canada, Ireland, and the USA to 50–263 per 10,000 births in Nigeria, Ethiopia, and Yemen [4]. In Russia, the incidence of UR averages 1.6 per 10 thousand births [1, 5]. The uterine wall altered by a scar after a cesarean section or laparoscopic myomectomy is recognized as the leading cause of uterine rupture [6, 7]. In turn, cases of ruptures of the non-operated uterus are rare, and are recorded from 0.3 to 0.7 cases per 10 thousand births, which is no more than 13% of all uterine ruptures [8].

The author of the first classification of UR in this country is L.S. Persianinov (1964), who identified two pathogenetic variants: spontaneous and forced. In turn, spontaneous URs are divided into mechanical, histopathic and mechanical-histopathic [9]. The causes of rupture of the non-operated uterus may be morphological changes in the myometrium due to inflammatory processes, dystrophic changes, trophoblast invasion, and a number of other factors [8–10].

Uterine ruptures can occur both during pregnancy and during childbirth. Undoubtedly, URs occur more often in women with a history of childbirth, and their number is directly proportional to the risk of this complication. At the same time, the literature describes cases of uterine rupture in women who are pregnant for the first time, in women who do not have scars on the uterus, and even in women who are not pregnant [6, 7].

Risk factors for rupture of the non-operated uterus include curettage, cervical conization, a large number of births, multiple pregnancies, induction of labor, use of prostaglandins, and other causes [6, 8]. Ruptures of the previously non-operated uterus are always unexpected, and often associated with the risk of asphyxia, fetal death, and maternal mortality [6, 8]. A rupture of the uterus without a scar, associated with rupture of adjacent organs in a woman with a history of normal childbirth, seems casuistic and deserves discussion.

The aim of the study was to present a clinical observation of spontaneous histopathic rupture of the non-operated uterus during labor.

Objectives: Retrospective analysis of clinical observation of histopathic UR with determination of underestimated risk factors for the complication.

MATERIAL AND METHODS

A 35-year-old female patient with histopathic rupture of the uterus, cervix, vagina and bladder during rapid labor.

RESULT

We present a clinical observation of spontaneous histopathic UR in a 35-year-old woman admitted to the Obstetric Hospital No. 2 of the Novokuznetsk Perinatal Center on 24.07.20 at 18:00 with complaints of contractions for 1.5 hours (from 16:30), and leakage of amniotic fluid 30 minutes before hospitalization.

From the anamnesis: Second marriage, current pregnancy was desired, 3rd: in 2008, urgent delivery (3100 g); in 2013, medical, instrumental abortion at 9 weeks, without complications.

The pregnancy was accompanied by anemia (hemoglobin 98–104 g/l), threat of miscarriage, and chorionic detachment at 7 weeks. At 35 weeks, according to ultrasound examination, placental insufficiency and grade 1 fetal growth restriction

syndrome were diagnosed. She was regularly observed at the antenatal clinic. Of the gynecological diseases, she notes: chronic adnexitis, endometritis; in 2011, electrocoagulation diathermy (ECD) was performed due to "cervical erosion". The somatic history was complicated by varicose veins of the lower extremities, viral hepatitis C (2018).

On admission, the woman's condition was satisfactory, skin was flesh-colored, blood pressure (BP) was 120/80 mm Hg, pulse was 80 beats per minute. The uterus corresponded to 39 weeks of pregnancy, of ovoid shape; contractions with pushing for 40-50 seconds every 2 minutes; contractions were strong; outside of contractions the uterus was in normotonicity, painless on palpation. The cervical os was fully dilated, the presenting part, the head, was on the pelvic floor. The fetal heartbeat was muffled, up to 100 beats per minute, the water contained meconium.

Five minutes after admission at 18:05, a live, full-term boy was born in the first position, anterior view, occipital presentation, weighing 3450 g, 52 cm long, without malformations, without umbilical cord entanglement, in asphyxia, an Apgar score of 5/7. Ninety seconds after birth, the umbilical cord was clamped, the amniotic fluid was meconium-containing. Ten minutes later, the placenta separated on its own, the afterbirth was released, and the uterus contracted. Upon examination, the afterbirth was intact, the membranes were flabby and green in color, the placenta had a moderate amount of petrification, and the umbilical cord was 60 cm long. After another 5 minutes, the birth canal was examined, a 3rd degree cervical rupture was found at 3 o'clock, as well as a 1st degree rupture of the lateral vaginal wall on the right, and a 1st degree perineal rupture. The ruptures were sutured with separate synthetic sutures. Total blood loss was 200 ml. Blood pressure was 110/70 mm Hg, pulse was up to 90 beats per minute.

25 minutes after the birth of the child, severe pain appeared throughout the abdomen, increasing with breathing; and signs of peritoneal irritation in all parts of the abdomen; a decrease in blood pressure to 90/70 mm Hg, tachycardia to 93 beats per minute. At the same time, the fundus of the uterus was determined at the level of the navel; the uterus was dense; the discharge from the genital tract was bloody, moderate.

An ultrasound of the pelvic organs and abdominal cavity was urgently (at 18:35) performed: the uterus was enlarged to 18 conventional weeks of pregnancy, with clear, smooth contours; the uterine cavity was expanded to 30 mm, with blood clots in the cavity. There was a large amount of free fluid behind the uterus and in the abdominal cavity. In order to determine the integrity of the uterine walls, a manual examination of the uterine cavity walls was performed under intravenous anesthesia at 18:45. A complete rupture of the uterus along the left rib up to 10 cm in length was established. During catheterization of the urinary bladder, dark blood in the amount of 50 ml was obtained. Total blood loss was 900 ml. Indications for laparotomy were given.

Diagnosis before surgery: Early postpartum period after rapid labor. Complications: spontaneous complete rupture of the uterus, 3rd degree cervical rupture, 1st degree rupture of the right lateral wall of the vagina, and the perineum. Rupture of the urinary bladder. Intra-abdominal bleeding, decompensated hemorrhagic shock. Background: Chronic viral hepatitis C, varicose veins of the lower extremities, stage I anemia, rh-negative blood type.

At 19:00 (1 hour after admission), a lower midline laparotomy was performed. Up to 1000 ml of liquid blood were found in the abdominal cavity, a complete rupture of the uterus along the left rib to the attachment point of the left round ligament of the uterus, a hematoma of the broad ligament of the uterus and parametrium on the left, a hematoma of the mesosalpinx and fallopian tube on the left, linear rupture of the urinary bladder in the apex area with an extension to the back wall up to 5 cm long.

Considering the total blood loss of 1900 ml, clinical picture of hemorrhagic shock, massive trauma to the uterus and adjacent organs, it was decided to refrain from organ-preserving surgical intervention. Total hysterectomy with left fallopian tube, suturing of the bladder with a double-row suture, drainage of the abdominal cavity through the left iliac region were performed.

Pathomorphological examination revealed dissociated maturation of the chorionic villi, uterine rupture along the left rib (from the uterine angle with transition to the isthmic part and cervix). Microscopic specimen: multiple focal lymphoid infiltrates in the basal layer of the endometrium and myometrium (Fig. 1, 2). In the area of the uterine wall rupture, there were edema, myolysis, diffuse, weakly expressed leukocyte infiltration in all the layers. Mixed and leukocyte thrombi were found in the lumen of the myometrium veins (Fig. 3, 4). In the area of the cervical canal and the vaginal wall there were extensive hemorrhages with hemolysis of erythrocytes (Fig. 5, 6). The left fallopian tube was normal.

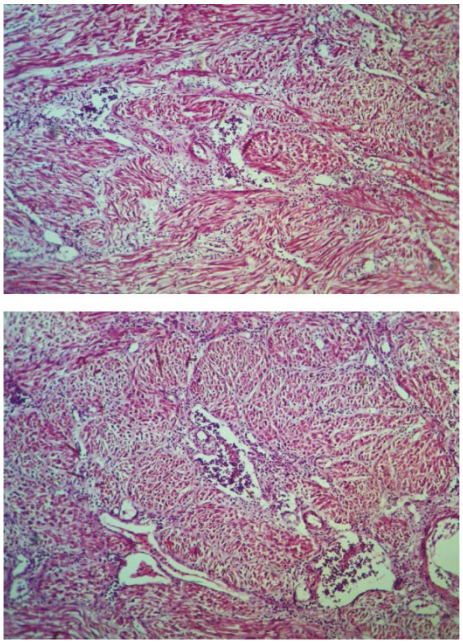

Fig. 1. Multiple focal lymphoid infiltrates in the subendometrial layer of the myometrium (hematoxylin and eosin stain, 100x magnification)

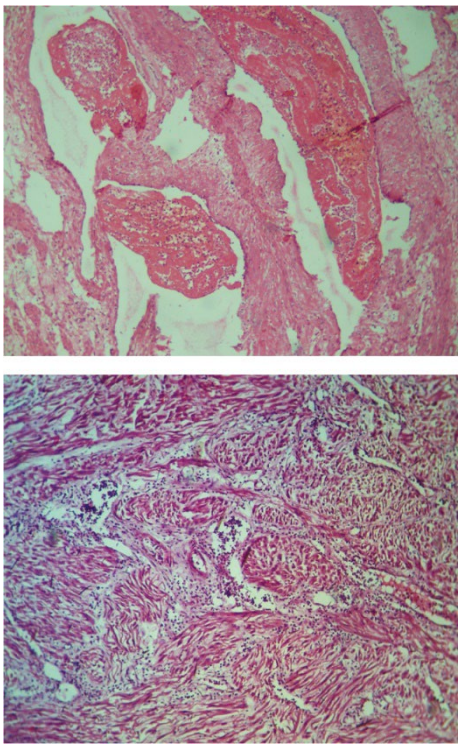

Fig. 2. Mixed and leukocyte thrombi in the lumen of the myometrial veins (hematoxylin and eosin stain, 100x magnification)

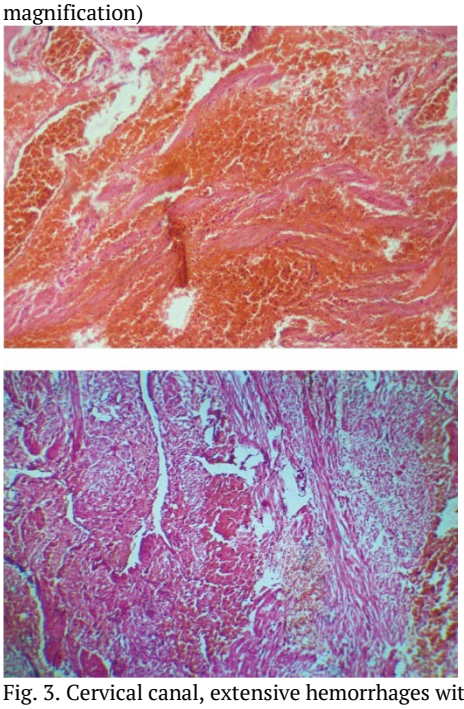

Fig. 3. Cervical canal, extensive hemorrhages with hemolysis of erythrocytes (hematoxylin and eosin stain, 100x magnification)

Pathological conclusion: complete rupture of the uterine wall, hemorrhages in the cervical canal,

vaginal wall. Thrombosis of the myometrial veins. Acute serous myometritis, persistent endometritis.

The early postoperative period proceeded favorably in the Resuscitation and Anesthesiology Department. The next day after surgery, the patient was transferred to the Gynecological Department, and then discharged with recovery on the 10th day.

DISCUSSION

The presented clinical observation demonstrates an unfavorable course of this pregnancy from the very beginning, which was most likely a consequence of a persistent inflammatory process in the uterus, confirmed by the data of histopathological examination. It seems to us, that the inflammatory process in the uterus, along with a history of intrauterine interventions (medical abortion, ECD), as well as non-specific connective tissue dysplasia (varicose veins) were the cause of pathological changes in the uterine wall as a substrate for histopathic UR, on the one hand, and predictive risk factors for the development of precipitate labor, on the other hand [6, 9–11]. In turn, rapid labor (RL) was a trigger for grade III cervical rupture extending to the body of the uterus. It is known, that histopathic ruptures most often occur at the height of the last push, which explains the birth of children in mild asphyxia or in a generally satisfactory condition [8]. According to the literature, RL in every third case is associated with maternal trauma, which is confirmed by our observation [12]. In case of grade III cervical rupture, manual revision of the uterine cavity walls is mandatory even without taking into account RL, and ECD in the anamnesis [4]. Manual examination of the uterine cavity walls immediately after delivery would probably have allowed for timely diagnosis of UR, prevented blood loss, and performed organ-preserving surgical intervention. However, in conditions of hemorrhagic shock, it was not possible to carry out adequate treatment of UR by suturing it while maintaining the quality of life and reproductive potential of the woman.

CONCLUSION

The presented observation demonstrates the need to be alert for uterine rupture after cervical electrocoagulation diathermy in the anamnesis, as in cases after surgical interventions on the uterus (caesarean section, myomectomy). The presence of a scar after electrocoagulation diathermy on the cervix should be considered a risk factor for its rupture with transition to the body of the uterus during rapid labor. Performing a manual examination of the walls of the uterine cavity immediately after childbirth in case of a 3rd degree cervical rupture is a mandatory measure, which allows for timely diagnosis of uterine rupture and immediate organ-preserving surgical treatment.

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