Introduction

Uterine rupture during childbirth is one of the most serious complications in obstetric practice, which can lead to both maternal and fetal death. According to the pathogenetic classification, in the Russian Federation, spontaneous uterine rupture can occur due to obstructed labor or a histopathological abnormality in the myometrium. The latter can be...
caused by a scar on the uterus after various surgical interventions, repeated intraterine interventions, impaired development of the genital organs, chronic endometritis, high birth parity (more than four in the anamnesis), multiple fetation, polyhydramnios, placenta increta, and destroying hydatidiform mole [1]. Despite significant progress in medical science, the frequency of uterine rupture over the past 13 years has almost not changed in the Russian Federation. Therefore, its frequency was 0.14 cases per 1,000 births in 2005 [2] and 0.16 cases per 1,000 births in 2018 [3]. However, coerced uterine ruptures and uterine ruptures due to mechanical causes are currently less common than those due to histopathological causes. This is due to a steady increase in the number of surgical interventions on the uterus, primarily cesarean section that had a frequency in 2018 of 300.5 cases per 1,000 births according to the Department of Monitoring, Analysis, and Strategic Development of Healthcare of the Ministry of Health of the Russian Federation [2]. In economically developed countries, uterine rupture was caused by a scar on the uterus in approximately 90% of cases [4]. The frequency of uterine rupture after myomectomy ranges from 0.3% to 0.5% [5], and after cesarean section, it is from 0.5% for cesarean section in the lower uterine segment to 4% for corporeal cesarean section [6].

Instrumental revisions of the uterine cavity and surgical abortions performed in the past also induce changes in the myometrium structure, which predispose to uterine rupture during pregnancy and labor [7]. However, the rupture rate of an unoperated uterus is far less than the operated one, and according to the World Health Organization, the frequencies are 0.6 per 10,000 and 100 per 10,000 deliveries, respectively [8].

According to the Verbov theory [1], as a result of pathophysiological processes, the elasticity of the myometrium decreases, while its fragility increases. As pregnancy progresses or during childbirth, the fibers of the pathologically altered myometrium break, the uterine wall becomes thinner until a defect is formed. In this case, uterine rupture may not be accompanied by a pronounced clinical presentation or it may be asymptomatic. This is due to the fact that this process is not always accompanied by severe hemorrhage or deterioration of the condition of the mother or fetus [1]. In some cases, the development of primary or secondary uterine inertia or parodina can serve as signs of a threatening uterine rupture [9]. However, due to the high frequency of adverse outcomes of this pathological process, it is currently necessary to continue the search for an algorithm for the effective diagnosis of uterine rupture both during pregnancy, childbirth, and the early postpartum period [10].

Clinical case description

Patient D., 28 years old, was followed-up during pregnancy in a maternity welfare center in the city of Chita (Trans-Baikal Territory). The chronic diseases of the patient included chronic tonsillitis and cardiac type neurocirculatory dystonia. In childhood, the patient was well developed for her age, and she was not physically or mentally retarded compared to her peers. This was her third pregnancy and it was desired. The first pregnancy was in 2010, and ended with an operative delivery due to pelvic presentation of the fetus at a term of 38 weeks of gestation. A full-term girl was born with a weight of 3,250 g and a length of 50 cm. The postoperative period was uneventful. In 2012, the patient underwent a surgical abortion at a gestational age of 8 weeks, and she reported having no complications. In 2017, a true pregnancy occurred. She had no history of allergies or blood transfusion. During this pregnancy, chronic cervicitis of ureaplasma, mycoplasma, and chlamydial etiologies were detected, and she was treated at 19–20 weeks of gestation with Josamycin in a dose of 500 mg 1 tablet 3 times a day for 10 days. The treatment effectiveness was not evaluated. The course of this pregnancy was complicated by exacerbation of chronic tonsillitis without fever at a gestational age of 14 weeks; the patient did not receive any specific drug therapy. Starting from week 34, she had bilateral edema of the legs and proteinuria up to 0.48 g/l in a daily portion of urine; blood pressure did not increase. The diagnosis of edema and proteinuria of pregnancy was established.

At 39–40 weeks of pregnancy, the patient was admitted to the obstetric department of the Regional Clinical Hospital of Chita with complaints of cramping pains in the lower abdomen since 1 h 40 min. The initial examination revealed no changes in the organs and systems, and edema was
not detected. Obstetric examination showed that the uterus became tonic for 20 s every 6–7 min; palpation in the lower segment was painless. Vaginal examination revealed that the cervix was along the axis pelvis plane, softened, shortened to 1.0 cm, passable for 3.0 cm; the fetal bladder was intact; the fetal head was pressed to the pelvic inlet. According to laboratory tests at the time of admission, leukocytosis (22.9 · 10⁹/l) was found in the general blood test. In a biochemical blood test, a coagulogram and general urinalysis showed that the indicators were within normal limits. Ultrasound examination of the fetus showed that the size of the fetus corresponded to 40 weeks of gestation; the estimated weight of the fetus was 3,890 g, the thickness of the lower segment of the uterus was 5 mm, and the lower segment was homogeneous. The diagnosis of early labor at a gestational age of 39–40 weeks and a scar on the uterus was established. Considering the woman’s intention to deliver by spontaneous vaginal delivery, the estimated fetal weight of less than 4,000 g, the spontaneous development of labor, it was decided at this stage to conduct the delivery through the natural birth canal.

During the case follow-up (within 4 h), the intensity of labor did not increase, and there was no positive dynamics in the birth canal. The diagnosis of primary uterine inertia was established. Delivery by cesarean section was proposed to the patient. After obtaining consent, antibiotic prophylaxis with cefazolin at a dose of 1.0 g was administered intravenously 30 min before surgery. A Pfannenstiel laparotomy was performed, followed by cesarean section in the lower uterine segment. A live full-term boy weighing 3,800 g was extracted with an Apgar score of 8 and 9 points. The placenta was located along the front wall of the uterus, 7 cm above the internal os, separated by moderate traction of the umbilical cord, and the retained products were 22 × 22 × 1.5 cm in size. The lower segment was represented by connective tissue, thinned to 0.2 cm, and was not deformed. When performing exteriorization, a complete rupture of the uterus of a rounded shape with a size of 5 × 4 cm was revealed in the uterine fundus (see Figure). A rupture in the uterine fundus after excision of the edges was sutured with a continuous three-row suture using a polyglycolide thread. Scar tissue of the lower segment was excised; the incision on the uterus was closed with a double-row continuous suture using a polyglycolide thread. Hemostasis was achieved. The abdominal cavity was drained using an active drainage in the left iliac region. The laparotomy incision was closed in layers. Blood loss during the surgery amounted to 800 ml. The postoperative diagnosis included surgical delivery at a term of 39–40 weeks; incompetent uterine scar; primary uterine inertia; complete uterine rupture; Pfannenstiel laparotomy; cesarean section in the lower uterine segment; suturing the rupture of the uterine fundus; abdominal drainage.

On the day 1 of the postoperative period, an increase in leukocytosis (28.4 · 10⁹/l), an increase in ESR (67 mm/h), and decrease in hemoglobin level (95 g/l) were revealed through the general clinical blood test, due to which antibacterial therapy was prescribed (cefazolin at a dose of 1.0 g intravenously 1 time per day), as well as antianemic (iron (III) hydroxide dextran at a dose of 0.1 g orally 2 times a day) and uterotonic therapy (solution of oxytocin 5 U intramuscularly 2 times a day).

On the day 2 postpartum, the patient’s condition deteriorated as she presented with general weakness, headache, and fever of approximately 38.0°C. The general clinical blood test revealed leukocytosis (27.8 · 10⁹/l), an increase in ESR (72 mm/h), and a decrease in hemoglobin (83 g/l) and hematocrit (23.4%). The infusion therapy was started. Antibacterial therapy was supplemented with a solution of metronidazole (100 ml
intravenously twice a day). The antibacterial drug was not changed. After that, the patient’s condition improved and her body temperature returned to normal.

On the day 4 of the postpartum period, the uterine sub-involution was noted (according to the vaginal examination, the uterus was increased to 14 weeks; but according to the ultrasound, the size of the uterus was 117 × 75 × 85 cm), as well as leukocytosis (18.4 × 10⁹/l), and moderate post-hemorrhagic anemia (84 g/l). The patient was transferred to the gynecological department of the Regional Clinical Hospital of Chita, where she continued antibacterial, uterotonic, and antianemic therapy. The patient was discharged from the hospital in satisfactory condition on the day 10 postpartum.

Discussion

It is very probable that the first stage of labor served as a trigger for the complete rupture of the uterine fundus, and the predictor was a uterine perforation in the course of the medical abortion, which was not diagnosed in 2012.

This case illustrates the complex clinical presentation and the difficulty in diagnosing a rupture in patients with a uterine scar. It is significant that with the timely detection and adequate treatment of the spontaneous uterine rupture (surgical delivery, suturing of uterine rupture), we were able to avoid a perinatal loss and to maintain the quality of life, including the reproductive potential of the woman.

The modern literature rarely presents clinical cases that describe the rupture of an unoperated uterus, caused by complications of intrauterine manipulations. Most often, such ruptures are explained by histopathologic changes in the myometrium [11–13].

Conflict of interest. The authors declare no conflict of interest.

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• Information about the authors (Информация об авторах)

Marina N. Mochalova — MD, PhD, Associate Professor, Head of the Department of Obstetrics and Gynecology, The Medical and Dental Faculties, Chita State Medical Academy, Chita, Russia. https://orcid.org/0000-0002-5941-0181. E-mail: marina.mochalova@gmail.com.

Lyubov A. Kuzmina — MD, Head of the Department of Pregnancy Pathology. Perinatal Center, Regional Clinical Hospital, Chita, Russia. https://orcid.org/0000-0003-2035-7966. E-mail: prostopochta1804@mail.ru.
Anastasia Yu. Mironenko — Assistant. The Department of Obstetrics and Gynecology, the Medical and Dental Faculties, Chita State Medical Academy, Chita, Russia. https://orcid.org/0000-0001-5061-8026. E-mail: mironenkoanastasia4@gmail.ru.

Igor B. Likhanov — MD. The Department of Pregnancy Pathology, Perinatal Center, Regional Clinical Hospital, Chita, Russia.

Viktor A. Mudrov — MD, PhD, Associate Professor. The Department of Obstetrics and Gynecology, the Medical and Dental Faculties, Chita State Medical Academy, Chita, Russia. https://orcid.org/0000-0002-5961-5400. E-mail: mudrov_viktor@mail.ru.