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Intrapartal posterior cervicoisthmic uterine rupture

Case Report

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Abstract: A case is presented of complete intrapartal rupture of an intact, nonscarred uterus in the cervicoisthmic region in a quadripara without a history of cesarean section or other uterine operation, with dorsoposterior high longitudinal fetal head position.

Keywords: *Uterine rupture • Intrapartal • Cervicoisthmic*

1. Introduction

Intrapartal uterine rupture is a rare complication that occurs in 0.05%-2% of all pregnancies. It is an emergency situation associated with high maternal and perinatal mortality (50%:50%) and morbidity rates [1], and is characterized by disruption of the uterine tissue integrity [1-3].

Etiologically, uterine rupture can be spontaneous or traumatic (violent). On delivery, a very rare rupture of intact uterus not previously exposed to any procedures or diseases, and uterine rupture at predilection sites after previous operative procedures or diseases, representing *loci minoris resistentiae* for uterine rupture are distinguished. The risk of intrapartal uterine rupture is increased in the presence of local myometrial destruction and cicatricial tissue formed upon previous uterine procedures, malplacentation, and previous or current gestational trophoblastic neoplasia. Other risk factors include fetal macrosomia, malpresentation and malposition, fetopelvic disproportion, multiparity, uterine tumors, anomalies, locoregional destructive inflammatory pelvic diseases, cervical dystocia, prostaglandin and oxytocin labor induction, uncritical Kristeller's expression, and manual manipulations in the third labor stage [1-3].

A case is presented of atypical complete intrapartal rupture of an intact uterus in the cervicoisthmic region in a quadripara without a history of cesarean section, with dorsoposterior high longitudinal fetal head position.

2. Case report

A 42-year-old quadripara was admitted to the maternal ward with regular labor pains in the 40th week of gestation. Previous pregnancies and deliveries as well as the current course of gestation were normal, with normal birth weight of the previous children and without postpartum complication (endometritis, D&C). Her personal and family history was normal, without gynecological comorbidity (pelvic inflammatory diseases, endometriosis). Due to her age, fetal karyotyping was not performed on her own demand. Upon admission, vital functions were normal (BP, pulse, diuresis). Obstetric finding: the os dilated by 8 cm, the amnion intact, the fetal head balloting over the os; cardiotocography (CTG) showed normal fetal heart rate (FHR) and regular labor pains; estimated fetal weight 4200 g. In the delivery room on amniotomy, some 750 mL of milky amniotic fluid were obtained; the head was

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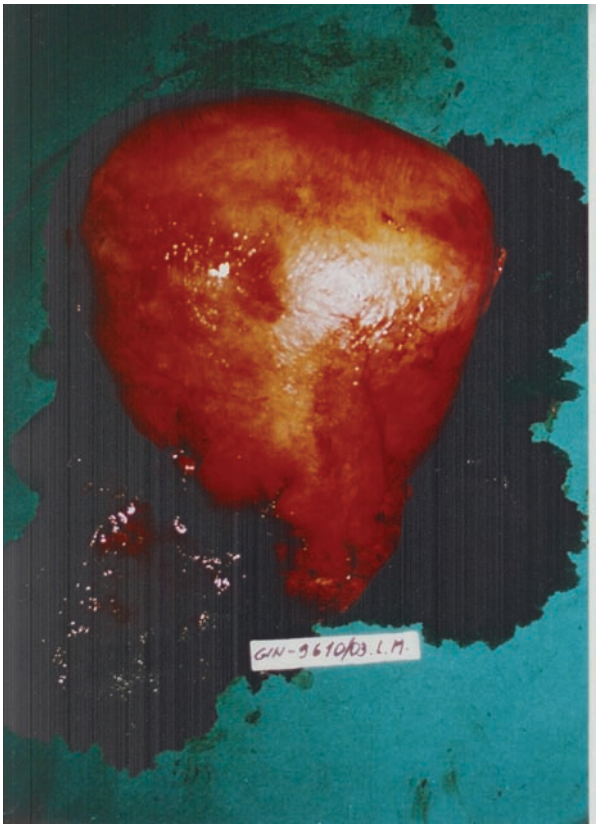
in -3 position, posterior fontanella and sagittal suture in oblique position II; CTG reactive. The patient was turned to lateral position and re-examined in 75 min to reveal secondary inert labor pains, fully open os, the head still in -3 position, dorsoposterior position I; an oxytocin (Syntocinon) infusion, 5 U/8 g b.w./min (4 mUI/min), was prescribed. The patient was turned on the left thigh and examined after 35 minutes of true labor pains; the head was still in dorsoposterior position, now with sagittal suture in longitudinal, -2 position, with *caput succedaneum,* CTG recording normal. Due to persisting dorsoposterior high longitudinal position, we decided to perform secondary cesarean section, upon consent obtained from the patient. Drip was disconnected.

In the operating theater, just before general endotracheal anesthesia (tiobarbital, succinyl, N_2O/O_2, fentanyl) the FHR turned bradycardiac, up to 50/min. A male child, 4300 g/54 g, Apgar score 5/5, umbilical blood pH 7.20, was born by cesarean section. During the operation, before opening the uterus, a hematoma was observed on the left lateral segment of the uterus. Upon extraction of the child and fundal placenta, complete cervicoisthmic rupture of the posterior uterine wall, of about 10 cm in length (Figure 1) was detected, and total hysterectomy without adnexa was performed because of the patient's parity and local finding. The postoperative course was uneventful, and the patient and her newborn were discharged from the hospital with normal findings on postoperative day 8. The newborn's neonatal course was normal, for the time free from manifest neuromotor deficit. Pathomorphological diagnosis without pathohystological findings of the uterine tissue: *ruptura uteri regio isthmica et cervicalis* (Figure 1).

Figure 1. Cervicoisthmic posterior complete uterine rupture.



3. Discussion

In contrast to the uterus previously exposed to some procedures or diseases, rupture of an intact uterus is a very rare obstetric emergency. According to Dickinson [2], uterine rupture is divided to rupture of a pre-existing cicatrix, uterine rupture in a multipara as the result of oxytocin action, and uterine rupture as a direct trauma from obstetric procedures, especially forceps. Some 1%-3% of cicatrix ruptures are reported after previous cesarean section; however, Shipp *et al.* [4] found no increase in the incidence of uterine rupture after cesarean section with low vertical incision *versus* transverse isthmic hysterotomy.

Complete uterine rupture is disruption of all uterine layers including the endometrium, myometrium and perimetrium, whereas incomplete (partial) uterine rupture refers to disruption of the endometrium and myometrium while the perimetrium (peritoneum) remains intact. Uterine rupture usually occurs at cicatricial sites; in the case of intact uterus, it occurs along the margins or at the site of contraction (Müller) ring. In their 12-year clinical material, Miller *et al.* [5] recorded 13 cases of uterine rupture in women without previous cesarean section, yielding an incidence of 1:16,849 deliveries. The risk factors for uterine rupture were labor induction with oxytocin, prostaglandins, vacuum-extraction and forceps, multiparity, and fetal malpresentation. Owing to timely and appropriate intervention, there was no maternal or perinatal mortality, however, neonatal and maternal morbidity is not reported [5].

Fetal morbidity and mortality in uterine rupture are associated with sudden peracute anoxia. Depending on the rupture extension and uteroplacental integrity, the associated morbidity may range from mild cerebral hypoxic-ischemic lesions and cerebral palsy through fetal death, which is exclusively due to exsanguination or peracute anoxia. The complications of uterine rupture include maternal death (5%), neonatal death (14%), 5-min Apgar score <7 (52%), maternal blood transfusion (24%), neonatal hypoxic lesion (14%), hysterectomy (14%), and endometritis (10%) [1,4,5-13].

Uterine rupture is immediately preceded by tense

and sensitive lower uterine segment, uterine hypertonia, blood stained discharge, pronounced Bandl's ring, tense and painful round ligament, possible edema of the cervix, vagina and vulva, difficult and painful miction, maternal fear and anxiety, and CTG changes in the form of bradycardia or late decelerations. Uterine rupture is accompanied by abrupt abdominal pain, cessation of labor pains, the uterus turns soft and toneless (Hoehne's sign), vaginal bleeding, tachycardia and hypotension as the signs of hypovolemic obstetric shock with collapse [8], and acute abdomen syndrome due to haematoperitoneum, which requires emergency laparotomy. CTG indicates bradycardia and/or fetal cardiac arrest [8-11]. Ruptures at the sites of cicatrices and minor ruptures are treated by primary suture, whereas complete extensive uterine ruptures, especially complicated ones, usually require hysterectomy.

In our patient, there were no typical signs of the pending uterine rupture, whereas cesarean section was indicated by persistent high longitudinal dorsoposterior position of the fetal head. Most probably, the overextended uterus in our quadripara and fetal malpresentation were the risk factors that led to subclinical uterine rupture, where the lower uterine segment (cervicoisthmus region) with persistent dorsoposterior position represented the *locus minoris* for the occurrence of intrapartal rupture. The rupture occurred just before the cesarean section, when peracute fetal hypoxia with bradycardia was demonstrated; that is why typical signs of acute abdomen and hemorrhagic shock could not have developed in the patient. Hysterectomy was performed because of the patient's multiparity and extent of the complete uterine rupture.

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