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Focal Placenta Accreta and Spontaneous Uterus Rupture in the Post-Partum

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Abstract

Introduction: Uterine rupture occurs when the integrity of the myometrial wall is compromised. It could interest the uterine body (prior to labour) or the lower segment (during labour). The main causes of uterine rupture are obstetric anamnestic factors and/or the administration of oxytocin.

Case presentation: The authors report the case of a 42 years old patient without risk factors for uterine rupture. After the expulsion of the placenta the mother had continuous blood loss, for this reason she was subjected to subtotal hysterectomy. Histological examination revealed the absence of decidua in the uterine fundus and thinning of the myometrium. Histological examination of the lesion at the site commented that there was infiltration of the chorionic villi into the endometrium and this is undoubtedly the features of placenta accreta.

Conclusion: The authors emphasize the importance of careful observation of the placenta after delivery for the absence of even small placental cotyledons in order to eliminate a rare risk factor for silent uterine rupture.

Keywords: Focal placenta accreta; Post-partum; Uterine rupture

Introduction

Uterine rupture is potentially life-threatening to both mother and baby. It occurs when the integrity of the myometrial wall is compromised. This usually occurs during the last weeks of pregnancy, labour or delivery. However, damage to the uterus prior to labour is usually in the uterine body while damage during labour is usually in the lower segment. One of the main predisposing factors for uterine rupture is the scarring of the uterus due to a previous surgery, namely caesarean sections [1]. A study conducted in 2005 by the World Health Organisation on the worldwide prevalence of uterine rupture resulted in a median of 0.053% [2]. At present, poor antenatal care, cephalopelvic disproportion, uterine scars and oxitocin administration are considered to be the most important etiopathogenetic factors [3]. Other risk factors to consider are congenital anomalies, age, multiparity and curettages (especially the ones complicated by perforation, fibrosis and/or adenomyosis). Very few cases of spontaneous uterine rupture in pregnant women without known risk factors have been reported in scientific literature [4] [Figure 1].

Case report

We report a case of a 42 years old woman from Romania, at the second pregnancy, with a previous abort and a previous spontaneous birth, who gave birth vaginally at the thirty-ninth weeks gestation. The baby weighed 2800 grams at birth. After spontaneous delivery, the patient began to bleed and was subjected to curettage. Continuing blood loss, it was decided to make a subtotal hysterectomy [5]. The piece removed showed a breach in the left margin of the uterus. Histological examination showed in the uterine fundus the absence



Figure 1: Mostra immagine a dimensione intera

of decidua, thinning of the myometrium, utero-placental arteries thrombosed. These data allowed a diagnosis of focal placenta accreta [6]. This was the starting point of the break which extended up into the uterine margin during the expulsion of the foetus. After subtotal hysterectomy the patient had a normal postoperative course and now is in excellent health.

Discussion

The peculiarity of this case report is that the uterine rupture was spontaneous and occurred in a patient with no underlying risk factors.

The main causes of uterine rupture in pregnancy are related to obstetric anamnesis and / or the administration of oxytocin. The main obstetric anamnestic factors are represented by previous gynaecological operations on the uterus, previous caesarean sections, scraping due to abortions and their complications. During the course of pregnancy there are some factors that can cause spontaneous rupture of the uterus and silent during labour: foetal macrosomia and abnormal foetal presentation [7].

The patient in question did not present any of these diseases. She gave birth at term a fetus of 2900 gr. vertex presentation in the left front. It was the histological examination of the uterus that revealed a possible cause: the absence of decidua in the site of placenta implantation and thinning of the myometrium, which probably represented the "locus minoris resistentiae" which started a silent and, at first, incomplete breaking of the uterus, which then led to a full thickness tear, due to myometrial hematoma that was forming in the meantime.

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In conclusion by this case report we can highlight the importance of the observation of the placenta after delivery for any absences even small placental cotyledons to eliminate a rare risk factor for silent uterine rupture.

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