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A case of intraamniotic hemorrhage with ruptured marginal cord insertion at 36 weeks of gestation

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Summary

Spontaneous hemorrhage into the amniotic cavity is a very rare complication of pregnancy. In the case described here, acute intra-amniotic hemorrhage occurred as the result of the rupture of the blood vessels in membranous marginal cord insertion in physiological week 36 of a multiparous pregnancy. Fast and accurate diagnosis afforded a satisfactory result. The aim in presenting this case report is to draw attention to important aspects of the diagnostic and therapeutic process in this difficult form of placental pathology.

Key words: Marginal cord insertion; Velamentous cord insertion; Intra-amniotic bleeding.

Introduction

Velamentous cord insertion is a situation in which the blood vessels of the umbilical cord propagate in the fetal membranes before they reach the placenta. This results in a reduction in the volume of the Wharton's jelly surrounding them and an increased risk of trauma. In marginal cord insertion, in turn, the umbilical blood vessels insert in the margin of the placenta, rather than in the center, severely restricting the connection with the placental tissues. The pathologies of the structure and function of the placenta described in this work constitute a major threat to the health and life of both mother and child.

Case Report

A 34-year-old pregnant woman in week 36 of pregnancy was admitted urgently to hospital on account of a diminished feeling of fetal movement that had lasted several hours. In the interview, it emerged that a first pregnancy had ended in an emergency cesarean section at term on account of signs of impending intrauterine fetal asphyxia (though there was no immediate evident risk to the fetus). The current pregnancy had so far been without complications. Physical examination showed a longitudinal cephalic fetal position and a lack of uterine muscle tone. An urgent ultrasound scan was carried out; it showed a single living fetus in the uterine cavity with a normal amount of amniotic fluid. The placenta was located posteriorly and on the left side of the uterus. There were no ultrasonographic signs of placental hematoma. A hyperechogenic fluctuating structure was detected around the umbilical cord of the fetus, presenting no Doppler flow (Figure 1A). Its extremity reached the area of the umbilical cord's insertion into the placenta, remaining in direct contact with the placenta. In the area of insertion of the umbilical cord, a detached hypoechogenic region with dimensions of 3.87×7.57 cm was visible between the amnion and the chorion (Figure 1A). Normal-resistance flows were also observed in the umbilical artery (average UmbA PI = 1.00) (Figure 1B). In view of the disturbing ultrasound image, a cardiotocographic observation was made, which showed a fetal heart rate of 130 per minute, a nonreactive strongly constricted oscillation, and a lack of contractile activity on the part of the uterine muscle (Figure 2A, B). Given the ultrasound results and the cardiotocographic (CTG) recording, a suspected diagnosis of acute intra-amniotic bleeding, with the threat of intrauterine fetal asphyxia, was made. It was decided to urgently perform a cesarean section. During the cesarean section, bloody amniotic fluid was found filling the amniotic cavity. A female fetus was extracted from the longitudinal cephalic position, with a mass of 3,100 grams and a length of 51 cm with average health (Apgar scores: 4/5/6/6). Examination revealed numerous blood clots, localized particularly around the umbilical cord, cord insertion, and the ruptured umbilical cord vessels responsible for the intra-amniotic hemorrhaging (Figure 3A). There were no macroscopic characteristics of placental hematoma. The placenta was submitted in its entirely to histopathological examination. The course of treatment and the postoperative period were uneventful.

The child's condition was described as average. Immediately after delivery, fresh blood was aspirated from the nasopharynx and stomach, tactile stimulation and positive pressure ventilation with oxygen mix were applied, and fluid replacement was performed. The neonate was then sent to intensive care for further observations. In view of the hypoglycemia, coagulation disorders, anemia, and pulmonary inflammation seen in the radiograph, empirical antibiotic therapy, antithrombin III, and concentrated red blood cells and platelets were administered. Temporary mechanical ventilation was also required, but from the fourth day this was no longer necessary, and on the fifth day, the infant was transferred to a cot. The postpartum period proceeded normally with proper involution of the uterus. The mother and child left the Clinic of Gynecology and Obstetrics 15 days after birth, due to the newborn's therapy.

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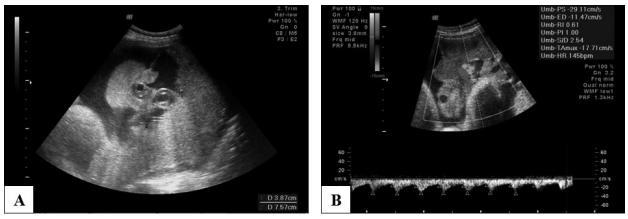

Figure 1. — Hyperechogenic structure of dimensions 3.87×7.57 cm localized around the fetal end of the umbilical cord and presenting no Doppler flow (A). Ultrasound examination: normal-resistance flows in the umbilical artery (UmbA PI = 1.00) (B).

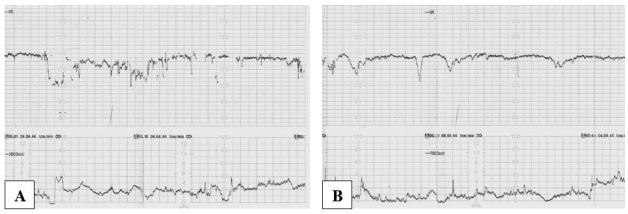

Figure 2. — Cardiotocographic (CTG) records (A, B). Presently non-reactive, strongly constricted oscillation with baseline fetal heart oscillation of 130 per minute. No contractile action of uterine muscle.

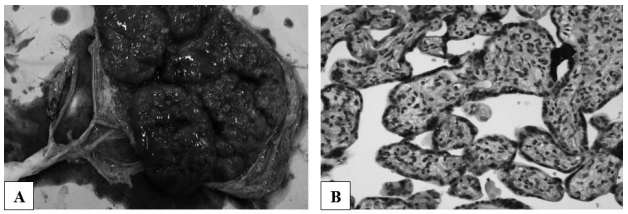

Figure 3. — Macroscopic view of the placenta with visible torn marginal insertion of a three-vessel umbilical cord (A). Microscopic histological specimen colored with hematoxylin-eosin method showing the proper structure of the placental villi (magnification ×200) (B).

The results of the histopathological examination were as follows: macroscopically, the placental dimensions were 14.5×3 cm and the umbilical cord was 21 cm long. The umbilical cord had marginal insertion and three vessels, with one end wider (Figure 3A). Microscopically, the placenta was from the third trimester and showed only minor signs of aging. The umbilical cord and fetal membranes lacked lesions. Apart from the marginal insertion of the umbilical cord, no deviations from normality were observed (Figure 3B).

Discussion

Numerous structural and functional abnormalities of the afterbirth are known. They range from those without clinical significance, such as false knots or the presence of additional blood vessels, to those that pose a direct threat to the child's life, such as tightening knots, ruptured vessels of velamentous cord insertion, or umbilical cord hematoma after extravasation of umbilical vein varix into the Wharton's jelly [1]. Velamentous cord insertion occurs in about 1.5% of singleton pregnancies and about 6% of twin pregnancies, while marginal cord insertion is found in about 6.3% of singleton and 10.9% of twin pregnancies [2]. The predominant risk factors for these two pathological forms of umbilical cord insertion are twin pregnancy and conception using assisted reproductive technologies. Next in significance are bleeding during pregnancy, advanced age of the mother, chronic illness of the mother, female sex of the fetus, and a history of pathological cord insertion [2]. The presence of velamentous or marginal cord insertion is associated with an increased risk of obstetric complications, such as placenta previa, placental abruption, premature birth, and preeclampsia [3]. Other neonatal complications are also associated with this pathology, such as abnormal fetal heart rate, low birth weight, low Apgar results at one and 5 minutes, and death [3]. Research shows that 99% of the cases of abnormal umbilical insertion can be reliably diagnosed using clinical ultrasound between weeks 11–14 of pregnancy [4, 5].

Extravasation of blood into the amniotic cavity is most commonly caused by iatrogenic damage during amniocentesis, in particular when performed on patients with anteriorly located placentas (100% of patients tested immediately after transplacental amniocentesis showed bleeding into the amniotic cavity [6]). Such bleeding usually remains asymptomatic, ceases spontaneously, and has no effect on the outcome of pregnancy. Primary bleeding into the amniotic sac is, however, very rare and, to date, only a few cases have been described in the literature [6–8]. Among other aspects, inflammation of the membranes may be associated with the risk of hemorrhage into the amniotic cavity. According to Witter *et al.* [8], maternal blood can perforate from potential spaces between the chorion and decidua towards the amniotic cavity. The location of perforation may undergo encrustation. The same probable cause of intra-amniotic hemorrhage is described by Kurata *et al.* [9] who, in laboratory studies of pregnant woman, found a significant degree of anemia (Hb 7.3 mg/dl; Ht 22.9%) and a lack of HbF when studying amniotic fluid obtained by amniocentesis. There is no evidence that chorioamnionitis causes intra-amniotic bleeding, but it may nonetheless initiate pathological processes that lead to such bleeding. Intra-amniotic bleeding may also be caused by damage to blood vessels as a result of sudden movements of the fetus, injuries, and increased pressure [10]. Sijanovic *et al.* describe the case of a pregnant woman at term showing symptoms of hemorrhagic shock (Hb 9.8 mg/dl; Ht 28.5%; RR 90/60, HR 112/min) and a 12×8 cm blood clot identified by ultrasound. Histopathological examination showed no signs of infection or pathology of the afterbirth. In addition, pla-

cental marginal sinuses are a likely source of intra-amniotic bleeding described in the literature. On ultrasound images, especially from the third trimester of pregnancy, these present as hypoechogenic areas on the edge of the placenta; they are formed of placental veins resembling a hematoma [11]. The literature also describes a case of intra-amniotic hemorrhage during pregnancy with circumvallate placenta. The cause of the hemorrhage was a tear at a fibrous part of the placental margin that penetrated into the intrachorionic space on the maternal side of the placenta, forming a passage for blood flow into the amniotic cavity. This type of bleeding is most often painless, and does not impair the functioning of the placenta or fetus since it does not separate the placenta from the wall of the uterus [12].

In the case of marginal cord insertion, the vessels enter the fetal membranes before reaching the edge of the placenta. The membranous part lacks Wharton's jelly, which exposes them to the possibility of injury in the event of the rupture of the membranes or their impairment [2]. It is likely that, in the present case described here, the umbilical vessels were torn by the fetus itself, as the patient detected the problem by observing the subsequent weakening of fetal movements.

Intra-amniotic bleeding, regardless of its cause, presents an immediate danger to the fetus and the mother. Ultrasound diagnosis allows a probable diagnosis. The treatment of choice is a cesarean section, and this is what was urgently implemented in the case described. In some cases, however, blood clots form a mass that can mimic a tumor, a vanishing twin, gastroschisis, meningocele, or fetal meconium. Asymptomatic mothers pose particular difficulties to diagnostic. In doubtful cases, particularly in preterm pregnancies, the mother should be closely watched for dark brownish or greenish fluids containing blood-degradation products, using ultrasound diagnosis and amniocentesis [1]. Ambiguous masses in the ultrasound imaging of the amniotic cavity should, in any case, arouse vigilance, as only a quick and accurate diagnosis can result in a successful maternity.

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