Premature placental separation (PPS) is a major cause of bleeding in pregnancy and responsible for a large increase in maternal and fetal morbidity and mortality. The diagnosis is mainly clinical; however, laboratory and imaging findings can be used to support the clinical diagnosis. PPS represents a challenge in Obstetrics because it promotes serious consequences to mother-child and have incomplete defined etiology. The objective of this study was to discuss the most relevant aspects of PPS emphasizing the importance of a complete clinical examination associated with the obstetric ultrasound method as a diagnostic aid.

Key words: Abruptio Placentae/diagnosis; Abruptio Placentae/classification; Abruptio Placentae/prevention & control; Uterine Hemorrhage; Hypertension.

INTRODUCTION

Premature placental detachment (DPP) is characterized by bleeding in the deciduous-placental interface, which promotes partial or complete placenta detachment before birth. The diagnosis is usually reserved for beyond 20 weeks of gestation. Most placental detachments are related to the chronic vascular disease process. However, some are acute and associated with trauma, or systemic vasocstriction and elevated blood pressure. The instant cause of placenta detachment is breakage of maternal blood vessels in the decidua basalis; the diagnosis is clinical and based on the abrupt onset of abdominal pain (increased uterine tonus) associated with mild to moderate vaginal bleeding, together with uterine contractions and alterations in fetal heart rate (BCF). With the process evolution,
intense myometrial blood infiltration and disruption of muscle cytoarchitecture in the myometrium are also observed, which determines the transformation of hypertension into stasis/hypotonia.5

DPP is a major cause of maternal and perinatal morbidity. The perinatal mortality rate is about 10%, and the increased risk of death is associated with preterm delivery by about 30% of cases.1,3

CASE REPORT

MAS, 34 years old, primigravida with a gestation of 32:5 weeks and usual prenatal risk was referred to the maternity ward due to increase in systemic blood pressure. The patient was asymptomatic, with preserved tendon reflexes, and positive tape proteinuria. Obstetric ultrasound showed fetal bradycardia (BCF = 86 bpm), PBF = 0/8, and umbilical artery Doppler with zero diastole. Once referred to the obstetric center, she reported vision loss associated with severe abdominal pain and increased uterine tonus. Pregnancy interruption was performed with an emergency cesarean section and a single fetus extraction with absent heartbeat and being readily assisted in the delivery room; presented Apgar 0/0 and returned heart rate after 15 minutes of birth. Birth weight was 1,570 g. After placental expulsion, a large number of clots and detached areas were evidenced, corresponding to approximately 50% of the placental surface. The uterus was hypotonic, and when exteriorized, hematomas and bleeding suffusions were evident across its surface (Couvelaire uterus); hypotonia was controlled with intravenous oxytocin and rectal administration of misoprostol (Figure 1).

She progressed satisfactorily in the immediate postpartum period and was discharged on the fifth postoperative day. The newborn required mechanical ventilation and CPAP for ten days, besides having developed sepsis likely due to a focus on the central nervous system; he was discharged on the fifty-sixth day of life. He is under medical supervision in the pediatric neurology clinic of the Odete Valadares Maternity.

Figure 1 - Visualization of uterus and placenta after fetal extraction, complete placental delivery and hysterorrhaphy. A) Uterus in anterior view; B) Uterus in right lateral view; C) Uterus in posterior view; D) Placenta – maternal side presenting areas with clots corresponding to premature detachment in about 50% of the total area.
DISCUSSION

DPP complicates 0.4 to 1% of gestations. The incidence appears to be increasing, possibly due to increased prevalence of risk factors for the disease and/or changes in the investigation of cases. The immediate cause of premature placenta detachment is rupture of maternal vessels in the decidua basalis, which interact with the anchoring villi in the placenta. Bleeding rarely originates from fetal placental vessels. The accumulated blood divides the decidua, separating the placenta from the uterus. The bleeding can be reduced and self-limited and can continue to dissect from the placental decidual-interface leading to complete or nearly complete placenta separation. The detached part of the placenta is unable to promote exchange of gasses and nutrients; fetus and placenta are unable to compensate for this loss of function, which seriously compromises the fetus.

The etiology of bleeding in the decidua basalis remains speculative in most cases despite extensive clinical and epidemiological research. In a small percentage of cases, the detachment is related to sudden mechanical events such as blunt abdominal trauma or rapid uterine decompression. Most DPPs appear to be related to some placental chronic disease. In these cases, abnormalities in the early development of spiral arteries can lead to decidual necrosis, placenta inflammation culminating in infarction, and, ultimately, vascular disorders, and hemorrhage. Arterial bleeding due to high pressure in the placenta central area takes to the rapid development of clinical manifestations of detachment (e.g., major vaginal bleeding, maternal disseminated intravascular coagulation, abnormal fetal heart rate) that are potentially fatal. The clinical manifestations that occur over time are more commonly the outcomes (e.g., intermittent vaginal bleeding, oligohydramnios, fetal growth restriction) when venous bleeding occurs due to low pressure, generally in the placenta periphery (marginal detachment).

Uterine abnormalities, smoking, and cocaine use are less common causes of DPP. Uterine abnormalities (e.g., bicornuate uterus) and leiomyoma are mechanically and biologically unstable areas for placental implantation. The pathophysiological effects of cocaine on the genesis of detachment are unknown. However, it may be related to the vasoconstriction induced by the drug, leading to ischemia, reflex vasodilation, and disruption of vascular integrity. About 10% of cocaine users may present DPP in the third trimester. The mechanism underlying the relationship between smoking and DPP is not well established, it is a hypothesis in which the vasoconstricting effects from smoking can cause placental hypoperfusion, which can result in decidual ischemia, necrosis, and hemorrhage, and subsequent premature placenta separation.

Vaginal bleeding is a major DPP signal and may range from mild and clinically insignificant to serious and life-threatening. However, the loss of blood may be underestimated if the bleeding is retained behind the placenta. There is no direct correlation between the amount of vaginal bleeding and degree of placenta separation. Therefore, the bleeding does not serve as a useful marker for preventing fetal or maternal risks. Conversely, maternal hypotension and FCF abnormalities suggest placenta separation as clinically significant and may result in fetal death and severe maternal morbidity. Acute disseminated intravascular coagulation and fetal death occur commonly when the placental separation area is greater than 50%.

About 10 to 20% of placental detachment is presented as preterm labors, with little or no vaginal bleeding. In these cases, generally termed “hidden detachment”, all the released blood or a great part of it is retained between the fetal membranes and decidua rather than escaping through the cervix and vagina. Therefore, pregnant women with abdominal pain and uterine contractions, even with a small amount of vaginal bleeding, must be properly evaluated for DPP by monitoring maternal and fetal well-being. In some minor cases, a small detachment may remain hidden and asymptomatic, being only incidentally recognized through ultrasonography. The identification of retroplacental hematoma through ultrasonography is a classic finding of DPP. The appearance of retroplacental hematomas is variable, looking solid, hypo, hyper, or isoechoic compared to the placenta. The absence of retroplacental hematoma does not exclude serious detachment, although worse outcomes may occur with sonographic evidence of retroplacental hematoma.

DPP can present the uterus with myometrial wall infiltrated by blood and classic ecchymotic uterus appearance, dark in color, and as serious consequences. As the result of this infiltration of blood into the myometrium, the organ loses its contractile force becoming static and possibly increasing bleeding, characterizing the Couvelaire uterus. The exact etiology of the Couvelaire uterus is still unknown. However, it has been widely associated with diseases such as DPP, placenta previa, coagulopathy, preeclampsia,
uterine rupture, and amniotic fluid embolism. It is believed that its pathophysiology was due to a toxin produced by the placenta during detachment, resulting in the penetration of uterine wall through the blood. However, it is currently considered the result of the myometrial invasion of blood from the retroplacental bleeding, which separates the muscle bundles and extends the bleeding to the serosal surface, giving the appearance of ecchymotic spots on the uterine surface. For decades, hysterectomy was the standard procedure for Couvelaire uterus. Hysterectomy is currently and generally not necessary because the condition resolves spontaneously with the addition of uterotonics such as oxytocin, prostaglandin (misoprostol), and Ergot derivatives.

The consequences of DPP are essentially related to the severity of placenta separation. For the pregnant women, there may be excessive bleeding and hypovolemic shock requiring blood and blood products transfusion, as well as kidney failure, adult respiratory distress syndrome, multi-organ failure, peripartum hysterectomy, and even death. The risks for the fetus are related to both the severity of detachment and gestational age at which delivery occurs. When the placental detachment is mild, there can be no significant adverse effects on the fetus. With an increasing degree of placenta separation, the risks increase, and situations like hypoxia, asphyxia, intrauterine growth restriction, low birth weight, and prematurity are responsible for high perinatal morbidity and mortality rates. In patients with classic symptoms, FCF alterations or intrauterine fetal death strongly suggest the clinical diagnosis of extensive placenta separation.

CONCLUSION

DPP is a very important entity in the obstetric clinic and characterized by bleeding in the deciduous-placental interface with partial or total placenta separation before birth. The diagnosis is particularly clinical. However, laboratory and imaging findings may be used to confirm the clinical diagnosis. Acute DPP usually presents with vaginal bleeding, initially abrupt, from mild to moderate, associated with abdominal or lower back pain and accompanied by uterine contractions with increased tonus. FCF alterations or intrauterine fetal death contribute to the clinical diagnosis of extensive placenta separation as occurred in the previously reported case. DPP has its complexity supported in its severity and unpredictability, calling attention to the fact that its prevention and proper control of risk factors still represent the best approach. This study aimed to demonstrate the importance of the clinical examination associated with obstetric ultrasonography in helping the diagnosis of DPP, which must be quick to allow the fast establishment of therapy in order to achieve the best maternal and fetal prognosis.

REFERENCES

Couvelaire uterus: a case report


