CASE REPORT

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Conservative management in puerperal hemorrhage due to placenta accreta

Manejo conservador en hemorragia puerperal por placenta acreta

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ABSTRACT

The placenta accreta spectrum is on the rise due to multiple causes, especially the increase in cesarean sections. Diagnosis is challenging, as most are associated with hemorrhage after delivery. Prenatal morphologic ultrasonography should evaluate for signs of suspected placental accreta. We present the case of a patient diagnosed with placenta accreta after abundant puerperal hemorrhage 48 hours after delivery, which required two uterine curettages and the administration of intramuscular methotrexate. As for treatment, although previously radical management was chosen, the current trend is conservative management in order to preserve the patient's fertility.

Key words: Placenta accreta, Conservative treatment

RESUMEN

El espectro placenta acreta se encuentra en incremento debido a múltiples causas, especialmente por el aumento de las cesáreas. El diagnóstico supone un reto, pues la mayoría de casos se asocia a hemorragia tras el alumbramiento. La ecografía morfológica prenatal debe valorar signos que hagan sospechar acretismo placentario. Se presenta el caso de una paciente diagnosticada de placenta acreta luego de abundante hemorragia puerperal 48 horas después del alumbramiento que requirió dos legrados uterinos y la administración de metotrexato intramuscular. En cuanto al tratamiento, aunque anteriormente se optaba por el manejo radical, la tendencia actual es un manejo conservador con el fin de preservar la fertilidad de la paciente.

Palabras clave. Placenta accreta, Tratamiento conservador

Introduction

The placenta accreta spectrum is a rare pathology, but its incidence is increasing[(1)]. This abnormal placentation is classically classified into 3 types. Placenta accreta consists of invasion of the myometrium by trophoblastic tissue. Deep myometrial invasion is known as placenta increta. And in placenta percreta, penetration extends beyond the uterine serosa, invading even neighboring organs. Furthermore, according to the lateral extension, it is subdivided into focal, partial and total, depending on the number of cotyledons involved.

Penetration of the placenta into the myometrium prevents the placenta from separating from the uterus during the third stage of labor, resulting in severe postpartum hemorrhage[(2)]. Early diagnosis is a challenge to avoid massive hemorrhage at delivery in cases not diagnosed prior to delivery.

Case report

The patient was a 33-year-old primigravida with no medical or surgical history of interest, controlled gestation and no risk factors. In the ultrasounds performed during pregnancy, the placenta was located on the posterior and fundic side and no signs of placenta accreta spectrum were mentioned. She presented spontaneous labor at $39^{2/7}$ weeks of gestation which ended with instrumental delivery using a Kiwi type vacuum cup under epidural

anesthesia, due to prolonged expulsive period. The newborn was normal, male sex, weight 3,340 grams, Apgar 9/10 and umbilical artery pH 7.30. After delivery, the placenta was delivered with apparent normality and its revision showed that it was intact. On the second day prior to discharge, the uterine height was found to be increased to a finger's breadth above the umbilicus, so an ultrasound scan was performed which showed an image compatible with chorioamniotic remains, and it was decided to perform a puerperal curettage under ultrasound control. The uterine cavity was apparently empty by ultrasound and the material extracted was compatible with placental remains, confirmed by pathological anatomy.

One month after delivery, she began heavy uterine bleeding estimated at one liter. Ultrasound revealed a 20 mm hyperechogenic image compatible with placental debris and a hyperechogenic image of 37.9 x 34.9 x 24.6 mm on the posterior uterine surface at myometrial level, ill-defined and with a color Doppler pattern with medium-high intensity suggestive of a focus of placental accretism (Figure 1). For this reason, a second curettage was performed, uterine tamponade was placed and uterotonic drugs were prescribed.

The next day the uterine tamponade was removed and no bleeding was observed. A new ultrasound showed a retroverted uterus, 85 x 65 mm, puerperal and well involuted, endometrial line with hyper-refringent spots secondary to curettage. Intramiometrial echogenic image of 73 x 52 mm with color map in its interior compatible with placental accretism (Figure 2).

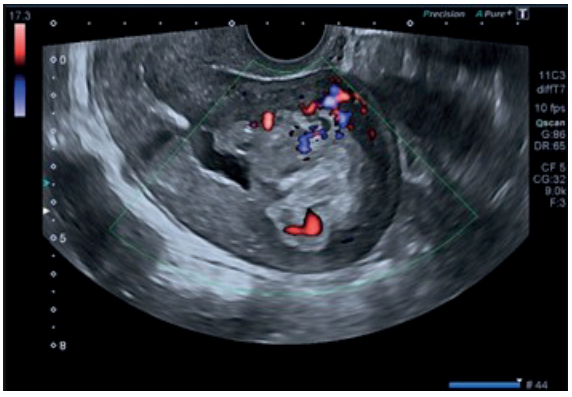
The patient was informed of the findings and the possibility of resorting to hysterectomy if severe bleeding occurred.

To complete the study, diagnostic hysteroscopy was requested 72 hours after the second curettage, which reported a uterine cavity occupied by deflufled material, mainly intramiometrial, reaching the cavity from the anterior aspect, compatible with placental debris. A sample was taken for biopsy which was reported as deciduocortical debris with marked degenerative changes and acute inflammation.

These data were reported to the patient, who expressed her desire to preserve the uterus and, if possible, to try conservative management. The

Figure 1. Ultrasound image showing the uterus with a hyper-refringent formation measuring 37.9 x 34.9 x 24.6 mm, corresponding to placental accreta spectrum.

Figure 2. Ultrasound image showing hyper-refringent formation with myometrial invasion; pulsed Doppler indicated abundant vascularization.

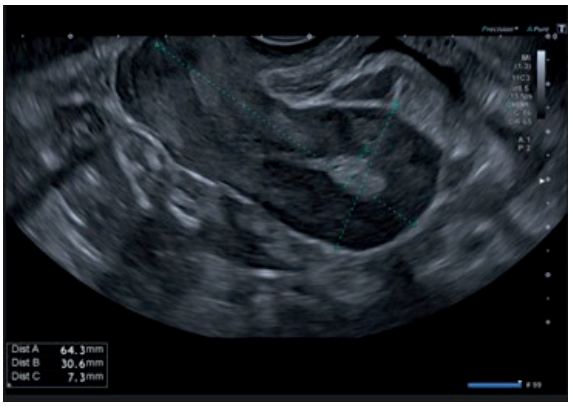


risk of infection and bleeding was pointed out. Despite this, the patient expressed her desire for conservative management, so it was decided to administer a single dose of methotrexate 79.5 mg IM (by Mosteller formula). In addition, the possibility of ovarian braking was offered, which the patient consented to, and a dose of a gonadotropin-releasing hormone analog -tryptorelin (*Decapeptyl*) 11.25 IM- and a gonadotropin-releasing hormone antagonist, orgalutran (*Ganirelix*) was administered at a dose of 0.25 mg daily for 5 days.

The patient was discharged asymptomatic. In the control ultrasound six months later, the intramiometrial image was still described with a size of 7 x 9 mm without Doppler uptake. That is, very significant improvement with respect to the initial picture (Figure 3). Due to the above, it was decided to use a gestagen (medroxyprogesterone acetate, *Progevera*) in a continuous regimen.

Currently the patient is in amenorrhea and without reproductive desire.

Figure 3. Ultrasound image showing a uterus of normal morphology and size. Hyper-refringent formation of 7.3 mm in the uterine fundus, considerable decrease in size with respect to previous ultrasounds.



Discussion

Placenta accreta spectrum is a pathology that is on the rise due, above all, to the increase in cesarean sections. It may also be due to multiparity, advanced reproductive age, endometriosis, uterine dilatation and curettage, conventional myomectomies and hysteroscopic resections of septa and myomas[3]. In addition, one of the main risk factors is the combination of previous cesarean section and placenta previa, which increases the risk exponentially. Other less frequent causes would be endometritis secondary to pelvic inflammatory disease due to sexually transmitted diseases, together with the increase in contraception with intrauterine devices.

Diagnosis may be suspected in patients who have previously undergone surgery to treat uterine conditions due to obstetric, gynecological or other causes[4]. And, although it could be diagnosed during morphologic ultrasound, it is very common for the diagnosis to occur in postpartum hemorrhage after delivery.

Ultrasound signs that could indicate the presence of abnormal placentation would be thinning of the uterus-placenta interface without clear boundaries, anechoic lacunae and turbulent blood flow. Therefore, during second trimester ultrasound, in addition to looking at fetal morphology, placental morphology and not only its uterine location should be evaluated. MRI with or without gadolinium could also be used to improve prenatal diagnosis, being complementary to ultrasound in isolated cases. If we compare ultrasound and MRI with gadolinium, we find high sensitivity (77% and 88%, respectively) and high specificity (96% and 100%, respectively) for both modalities[3].

The confirmatory diagnosis is given by pathological anatomy. The gold standard would be the absence of basal decidua, which is not always found due to the distortion that occurs at the moment of delivery, except in cases of hysterectomy[5].

Therefore, in more than 50% of the cases the diagnosis of anomalous placentation is made in the context of massive puerperal bleeding after delivery, which could even lead to hypovolemic shock, disseminated intravascular coagulation and death of the patient.

Surgical management by means of total abdominal hysterectomy has been the classic treatment choice. However, in recent years a conservative trend has emerged that avoids hysterectomy and its complications and seeks to preserve the future fertility of patients[5]. Conservative treatments include embolization of the uterine arteries or hysteroscopic resection of the affected area of the uterus[3], although these treatments may subsequently interfere with fertility. Another less invasive measure would be the administration of methotrexate which accelerates placental resorption, implemented in our case with good evolution of the patient.

It can be seen that the treatment of abnormal placental implantation is varied and there is still no consensus on the guideline to be followed. It would be decided on a case-by-case basis, depending on the stability of the patient and the amount of bleeding. The most radical measure would be hysterectomy in cases of abundant bleeding and instability of the patient. A more conservative measure will be tried when the patient is primigravid.

References

1. Tantbirojn P, Saleemuddin A, Sirois K, Crum CP, Boyd TK, Tworoger S, Parast MM. Gross abnormalities of the umbilical cord: related placental histology and clinical significance. Placenta. 2009;30(12):1083-8. DOI: 10.1016/.placenta.2009.09.005
2. Stanek J. Association of coexisting morphological umbilical cord abnormality and clinical cord compromise with hypoxic and thrombotic placental histology. Virchows Arch. 2016;468(6):723-32. DOI: 10.1007/s00428-016-1921-1
3. Bergman P, Lundin P, Malmstrom T. Mucoid degeneration of Wharton's jelly: An umbilical cord anomaly threatening

fetal life. Acta Obstet Gynecol Scand. 1961;40:372-8. DOI: 10.3109/00016346109159935

4. Thomson LL, Hoo JJ. Linear disruption of umbilical cord: a rare anomaly of the cord associated with acute fetal distress and perinatal death/profound psychomotor retardation. Am J Med Genetics. 1996;62:348–9. DOI: 10.1002/(SICI)1096-8628(19960424)62:4<348::AID-AJMG5>3.0.CO;2-N

5. Labarrere C, Sebastiani M, Siminovich M, Torassa E, Althabe 0. Absence of Wharton's jelly around the umbilical arteries: An unusual cause of perinatal mortality. Placenta. 1985:555-9. DOI: 10.1016/s0143-4004(85)80010-2

6. Trivedi S, Ratanoo L, Purohit S, Rastogi P. Absence of Wharton's jelly: an association with feto-maternal morbidity. Int J Reprod Contracept Obstet Gynecol. 2020 Mar;9(3):1318-20. DOI: https://doi.org/10.18203/2320-1770.ijrcog20200926

7. Benirschke K, Kauffmann P, Baergen RA. Pathology of Human Placenta. Fifth edition. 2006: pag. 381. Springer New York, NY. DOI: https://doi.org/10.1007/b137920

8. Nanaev AK, Kohnen G, Milovanov AP, Domogatsky SP, Kaufmann P. Stromal differentiation and architecture of the human umbilical cord. Placenta. 1997;18(1):53-64. DOI: 10.1016/s0143-4004(97)90071-0

9. Takechi K, Kuwabane Y, Mizuna M. Ultrastructural and Immunohistochemical Studies of Wharton's Jelly Umbilical Cord Cells. Placenta. 1993;14:235-45.

10. Cole J, Israfil-Bayli F. Wharton's jelly: The significance of absence. J Obstet Gynaecol. 2016 May;36(4):500-1. DOI: 10.3109/01443615.2015.1094041

11. Kulkarni M, Matadh P, Achok C, Pradeep N, Avinash T, Kulkarni A. Absence of Wharton's Jelly around the umbilical arteries. Indian J Pediatr. 2007;74(8):787-9. DOI: 10.1007/s12098-007-0142-7

12. Murphy SJ, Deegan N, O'Leary BD, McParland P. absence of Wharton's jelly. BMJ Case Rep 2020;13:e237222. doi:10.1136/bcr-2020- 237222

13. Oliveira CC, Dufloth RM, Coelho KR. Absence of Wharton's jelly: case report. J Bras Patol Med Lab. 2014;50(6):452-5.

14. Botezatu R.; Raduteanu S, Ciobanu AM, Gica N, Peltecu G, Panaitescu AM. Absence of Wharton's Jelly at the Abdominal Site of the Umbilical Cord Insertion. Rare Case Report and Review of the Literature. Medicina. 2021;57:1268. https://doi.org/10.3390/ medicina57111268

15. Wade M, Gueye M, Mbodji A, Ndiaye MD. Absence of Wharton's jelly around an umbilical artery. Int J Reprod Contracept Obstet Gynecol. 2022;11:259-61. DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20215115

16. Damasceno EB, de Lima PP. Wharton's jelly absence: a possible cause of stillbirth. Autops Case Rep. 2013 Dec 31;3(4):43-7. doi: 10.4322/acr.2013.038. PMID: 28584806; PMCID: PMC5453660.