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Technical note

Stripped fetal vessel sign: a novel pathological feature of abnormal fetal vasculature in placenta accreta spectrum disorders with MRI correlates

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ABSTRACT

Abnormal intraplacental hypervascularity is a well-known MRI feature of Placenta Accreta Spectrum (PAS), but the precise nature of these vessels has not yet been specified. Histopathological examination of eleven PAS-hysterectomy specimens and subsequent review of the corresponding MRIs, revealed the presence of large fetal vascular trunks extending deep towards the placental periphery and demonstrating deficient branching along their course ('stripped-fetal-vessel' sign). To our knowledge, this is the first report to describe the pattern of abnormal fetal vasculature in correlation with MRI in PAS.

1. Introduction

Identification of placenta accreta spectrum (PAS) disorders and estimation of the degree of placental invasiveness has become an important issue for clinicians, since this potentially fatal complication is likely to be encountered several times during a practicing lifetime [1]. In addition to Color Doppler sonography (CDS), Magnetic Resonance (MRI) has been recently introduced for the imaging evaluation of PAS disorders, exhibiting comparable diagnostic indexes to CDS [2]. MRI provides a larger field of view, and due to its excellent soft tissue contrast resolution, allows more thorough imaging of the placental parenchyma compared to sonography. Abnormal intraplacental hypervascularity is considered one of the most important MRI features for the identification of PAS disorders, with reported specificity values up to 80%, albeit, lower sensitivity values ranging from 42% to 69% [3,4]. However, there is lack of published data regarding the nature of these intraplacental vessels on MRI, with one study assuming a maternal origin [5].

This study aims to investigate the nature of the abnormal intraplacental vasculature seen in patients with PAS and correlate MR imaging with pathological findings.

2. Materials and methods

Institutional review board approved this prospective study and written informed consent was obtained from all patients. Between March 2016–June 2017, eleven patients (age: 29–45 years, mean: 36.1 years, gestational age: 31–37wks, mean:34.2wks) underwent cesarean hysterectomy due to intraoperative diagnosis of PAS. The surgical specimens were examined in detail by one expert perinatal pathologist (20 year of experience). All patients were at high risk for abnormal placentation due to the presence of placenta previa on routine second trimester sonography and preceding uterine instrumentation, including previous Cesarean section (n = 11), leiomyomatectomy (n = 7) or hysteroscopy (n = 5).

Surgical evidence of PAS in our study patients, was recorded by an experienced obstetrician (25 years of experience) in accordance to the FIGO general classification of PAS [6]; in particular, 1 case was classified as FIGO grade 3a (limited up to serosa), 8 cases as grade 3b (bladder involvement) and, 2 cases as grade 3c (bladder and parametrial involvement).

Histopathological examination confirmed placental invasiveness in all cases. Increta type of PAS was seen as focal or multifocal invasion of the myometrium, percreta diagnosis was based on the complete

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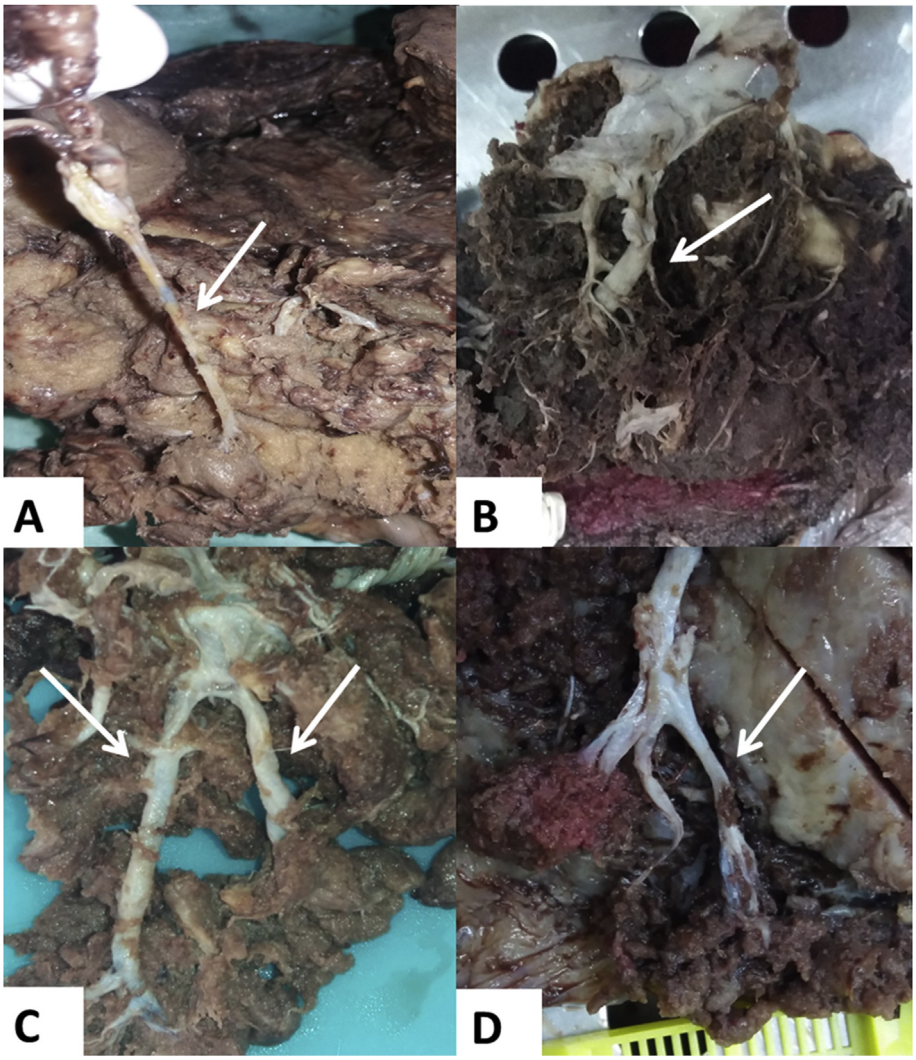

Fig. 1. Long course of stripped fetal vessels (arrows) in placenta percreta, seen in four different (formalin fixed) hysterectomy specimens (A–D). In case 1C, the placenta is shaggy and fragmented, due to attempted manual removal previous to hysterectomy.

elimination of the myometrium up to the serosa. Creta, increta and percreta types of invasiveness were often seen in various parts of the same specimen; the highest degree of invasion was used for classification. All cases in the sample were histologically classified as percreta, regardless of the extent and proportion between increta and percreta areas.

All 11 patients had undergone prenatal MRI (gestational age: 24–36wks, mean: 32.4wks) with a dedicated protocol for placental evaluation in 3T magnet unit. After delivery, all MRIs were reviewed by two radiologists experienced in genitourinary imaging (20 and 10 years of experience, respectively), in an attempt to associate the histopathological findings with the imaging features on MRIs.

3. Results/discussion

Pathological examination of all specimens showed a distortion of the major fetal vasculature, with a multifocal increase in large subchorionic vessels and a loss of the usual branching pattern seen in normal placentas; specifically, macroscopic and microscopic examination revealed an enhanced fetal vasculature with large (d = 3–5 mm) and elongated (L = 2–8 cm) subchorionic and stem vascular trunks (Fig. 1, Fig. 2A). These appeared to originate from subchorionic lacunar spaces running in parallel to the fetal chorionic plate, subsequently dividing into diagonal or perpendicular stem vessels. In contrast to normal fetal stem villous vessels, these appeared naked, seen on microscopy to be minimally surrounded by chorionic tissue (Fig. 2B and C). Along their whole course towards the more distal parts of the placenta, the abnormal fetal vessels showed a deficiency in major branching, and were partly surrounded by sparse and easily detached chorionic villi. In the more severely invasive placenta cases, the abnormal fetal vasculature was more prominent, the gross appearance being that of “intercalated strings” (Fig. 1B).

Other histopathological findings included extensive basal thrombi, chronic basal and myometrial inflammation and changes of maternal vascular malperfusion, as previously described in PAS specimens [7].

Retrospective review of all corresponding MRIs showed at least one long (> 2 cm) flow void (vascular) structure of variable size (range: 1.8-10.2mm, mean: 4.7mm) emerging from the chorionic plate, crossing the parenchyma and getting close to the maternal surface of the placenta. Due to the lack of visible branches coming off this abnormal vessel throughout its placental course, we named it the ‘stripped fetal vessel’ sign (Fig. 2). In accordance to the pathological findings, this abnormal vascular pattern was more conspicuous on MRI in cases of extensive placental invasiveness.

Abnormal intraplacental vasculature on MRI in patients with PAS, first described by Dermal and colleagues, is defined as the presence of tortuous disorganized vessels of large (≥ 6 mm) caliber located within the placenta [8]. This intraplacental vascular pattern is absent on MRIs

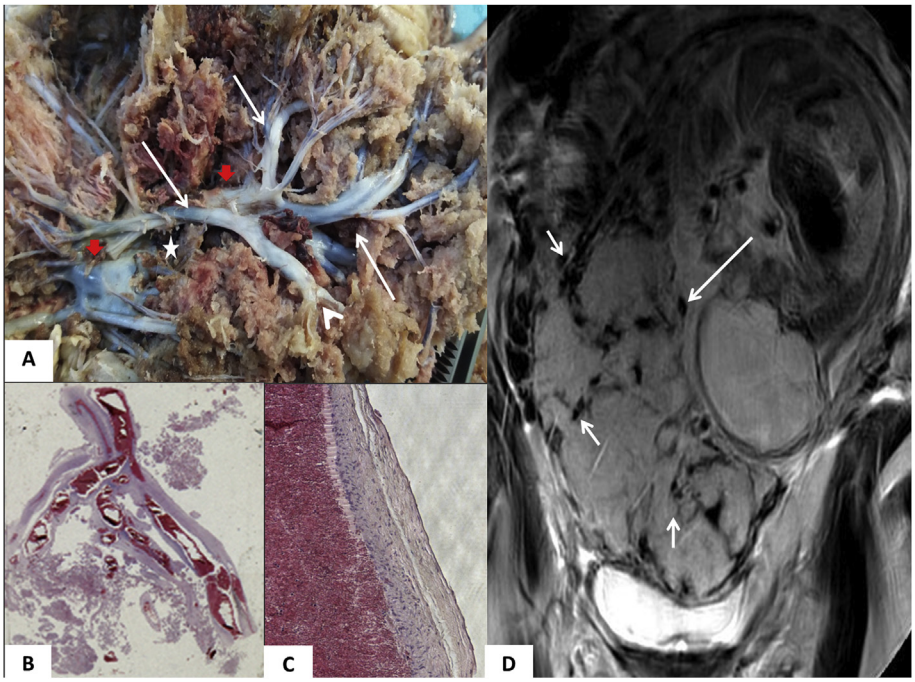

Fig. 2. Placenta percreta at gestational week 32. (A) Macroscopic examination of the placenta lying on the fetal surface (close up view). There are numerous large fetal vascular trunks (white arrows) arising from subchorionic lacunar spaces (red arrows), surrounded by sparse placental parenchyma and showing deficient branching (asterisk shows small residual vessels in areas where the surrounding parenchyma was easily trimmed off). The abnormal fetal vessels extend to the periphery until they disappear by branching directly into distal chorionic villi (white arrowhead) (B) Histological picture of subchorionic and stem villous vessels (H-E X1) (C) The fetal vascular wall is surrounded by a thin rim of chorionic tissue (H-E X50) (D) Coronal T2 MR image of the same patient performed at gestational week 28 demonstrates multiple flow void (vascular) structures extending from the chorionic plate (long white arrow) to the distal placental parenchyma (small white arrows). Emergency hysterectomy was performed in this case due to massive bleeding.

of non-invasive placentas and is considered as a highly indicative sign of abnormal placentation [3,8]. Ueno and colleagues suggested that these dilated vascular structures are of maternal origin, arising from the underlying uterine myometrium at the site of invasion, and subsequently infiltrating the placenta [5]. Our findings, conversely, support a fetal origin of the prominent intraplacental vessels seen on MRI of patients affected with PAS. Our assumption is that the stripped-fetal-vessel sign may suggest a defective vasculogenesis in the fetal vascular tree, resulting in deficient formation of chorionic villi. This combination is reflected in the long stripped vessels surrounded only by sparse chorionic villous parenchyma. Enhanced maternal vasculature was also observed in our PAS cases; such vascular spaces, though, were seen only close to the basal plate and the myometrium, and did not correlate with the abnormal intraplacental vessels typically seen on MRI. Knowledge and understanding of this characteristic abnormal vascular course (from the umbilical cord deep to the distal placenta) is important, as this feature may help radiologists distinguish the abnormal intraplacental vessels from low signal intraplacental bands on T2 images. Interestingly, in our study the mean estimated diameter of the abnormal intraplacental vessels on MRI was lower (4.7 mm), compared to the cut-off of 6 mm proposed in previous studies [8]. In accordance to previous reports, the extent of the aberrant intraplacental vasculature on both pathological examination and MR imaging was proportionate to the extent of placental invasiveness [8]. Therefore, identification of stripped fetal vessels on prenatal MRI may be used as a potential prognosticator for placental invasiveness and patients' clinical outcome; however, larger prospective studies are needed to investigate the clinical implementation of this finding.

In conclusion, this is, to our knowledge, the first report describing a specific pattern of abnormal fetal vasculature in placentas with PAS, in correlation with MRI findings, elucidating an important sign for identifying PAS, facilitating prenatal imaging diagnosis and prognosis, and contributing to better understanding of the pathophysiology of abnormal placentation.

Authors' contribution and conflict of interest

Anastasia Evangelia Konstantinidou: I declare that I participated in the study's concept and design, data collection, manuscript writing and editing, and that I have seen and approved the final version. I am the guarantor of the study. I declare no conflicts of interest.

Charis Bourgioti: I declare that I participated in the study's concept and design, data collection, manuscript writing and editing, and that I have seen and approved the final version. I declare no conflicts of interest.

Stavros Fotopoulos: I declare that I participated in the study's concept and design and clinical data collection, and that I have seen and approved the final version. I declare no conflicts of interest.

Efi Souka: I declare that I participated in the study's data collection and literature research, and that I have seen and approved the final version. I declare no conflicts of interest.

Maria Evangelia Nikolaidou: I declare that I participated in the study's concept and design and clinical data collection, and that I have seen and approved the final version. I declare no conflicts of interest.

Konstantina Zafeiropoulou: I declare that I participated in the study's data collection and literature research, and that I have seen and approved the final version. I declare no conflicts of interest.

Lia Angela Moulopoulos: I declare that I participated in the study's concept and design, data collection and manuscript editing and that I have seen and approved the final version. I declare no conflicts of interest.

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