Placenta accreta spectrum: pathophysiology and evidence-based anatomy for prenatal ultrasound imaging

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Placenta accreta spectrum is a complex obstetric complication associated with high maternal morbidity. It is a relatively new disorder of placentation, and is the consequence of damage to the endometrium-myometrial interface of the uterine wall. When first described 80 years ago, it mainly occurred after manual removal of the placenta, uterine curettage, or endometritis. Superficial damage leads primarily to an abnormally adherent placenta, and is diagnosed as the complete or partial absence of the decidua on histology. Today, the main cause of placenta accreta spectrum is uterine surgery and, in particular, uterine scar secondary to cesarean delivery. In the absence of endometrial reepithelialization of the scar area the trophoblast and villous tissue can invade deeply within the myometrium, including its circulation, and reach the surrounding pelvic organs. The cellular changes in the trophoblast observed in placenta accreta spectrum are probably secondary to the unusual myometrial environment in which it develops, and not a primary defect of trophoblast biology leading to excessive invasion of the myometrium. Placenta accreta spectrum was separated by pathologists into 3 categories: placenta creta when the villi simply adhere to the myometrium, placenta increta when the villi invade the myometrium, and placenta percreta where the villi invade the full thickness of the myometrium. Several prenatal ultrasound signs of placenta accreta spectrum were reported over the last 35 years, principally the disappearance of the normal uteroplacental interface (clear zone), extreme thinning of the underlying myometrium, and vascular changes within the placenta (lacunae) and placental bed (hypervascularity). The pathophysiological basis of these signs is due to permanent damage of the uterine wall as far as the serosa, with placental tissue reaching the deep uterine circulation. Adherent and invasive placentation may coexist in the same placental bed and evolve with advancing gestation. This may explain why no single, or set combination of, ultrasound sign(s) was found to be specific for the depth of abnormal placentation, and accurate for the differential diagnosis between adherent and invasive placentation. Correlation of pathological and clinical findings with prenatal imaging is essential to improve screening, diagnosis, and management of placenta accreta spectrum, and standardized protocols need to be developed.

Key words: cesarean delivery, increta, percreta, placenta accrete, prenatal diagnosis, ultrasound imaging

Introduction

The first case series of placenta accreta (PA) was published in 1937 by Irving and Hertig.[1] They reviewed 18 cases, which they described clinically as "the abnormal adherence of the afterbirth in whole or in parts to the underlying uterine wall" and histologically as "the complete or partial absence of the decidua basalis," signs that are still used today. They described all their cases as "vera" or "adherenta" where the villi were attached to the surface of the myometrium without invading it.

The grading classification of PA according to the depth of villous invasiveness inside the myometrium was introduced by modern pathologists in the 1960s.[2-4] They separated PA into 3 categories: placenta creta when the villi simply adhere to the myometrium, placenta increta (PI) when the villi invade the myometrium, and placenta percreta (PP) where the villi invade the full thickness of the myometrium [F1] (Figure 1). This terminology is still used by most pathologists. It is, however, often impossible to clinically differentiate between these categories, especially as they may coexist in the same placental bed (Figure 2), and confusion frequently occurs among clinicians regarding the difference between the terms "accrete" and "creta." Given the lack of international consensus on nomenclature, for the purposes of this review, we refer to it as the PA spectrum (PAS), which includes both abnormal adherence and abnormal invasion. We then use placenta creta, PI, and PP for specific examples where the histology is known.

In PAS, the lack of a plane of cleavage between the placental basal plate and the [F2] uterine wall leads to major hemorrhage if an attempt is made to forcibly remove villous tissue embedded within the myometrium.[5-8] The severity of the complications varies according to the depth of villous invasion. In PP, not only is there potential villous invasion of surrounding pelvic organs, but excessive neovascularity is often present making any surgical procedure technically

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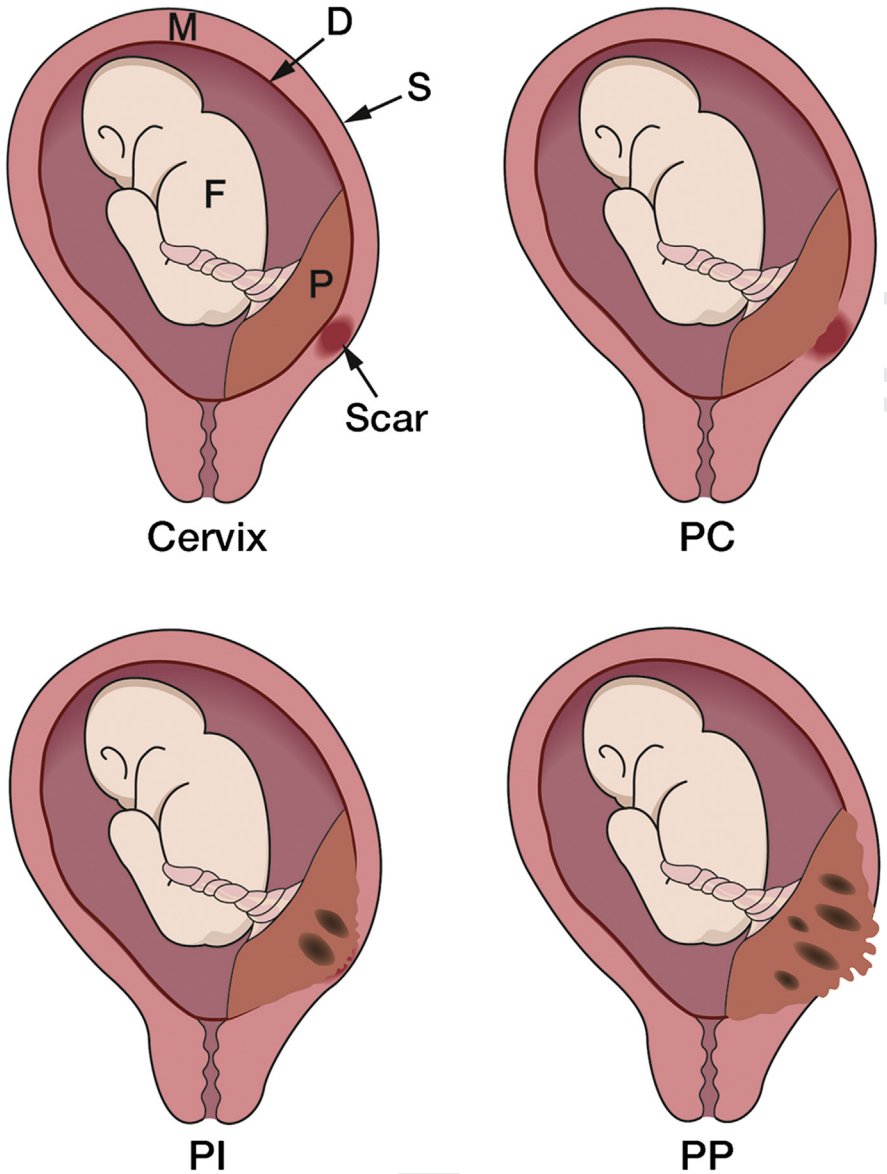
FIGURE 1



Anterior placenta (P) previa on cesarean scar and different grades of P previa accreta: creta (PC) where P villi adhere to myometrium (M); increta (PI) where villi invade M; and percreta (PP) where villi invade entire M and cross uterine serosa (S).

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difficult. Multidisciplinary teamwork and operator experience has been shown to reduce collateral damage, with several studies demonstrating that maternal morbidity is significantly reduced by delivery in a specialist center.[9-12] Thus, the prenatal diagnosis of PAS has become essential to its management and outcome. The first prenatal diagnosis of PAS was reported in 1967 by Sadovsky et al[13] using radioisotope placentography, and the first prenatal ultrasound description was made by Tabsh et al[14] in 1982.

Ultrasound imaging and magnetic resonance imaging (MRI) are now commonly used for the prenatal diagnosis of PAS.[15-18] However, recent population studies indicated that it remains undiagnosed before delivery in between half[19,20] and two thirds[21] of cases. The cost and limited access of MRI make it impractical as a screening tool, and ultrasound has therefore become essential in identifying women at high risk of PAS and tailoring their management. Although many ultrasound diagnostic signs have been described, wide heterogeneity in both study design and terminology used by different authors has made it difficult to define the ultrasound markers that enable the different grades of villous adhesion or invasion to be distinguished.[18] In this review, we evaluated the pathophysiology of different ultrasound signs associated with PAS to better understand their relevance to prenatal screening and diagnosis of accreta placentation.

Pathophysiology of the PAS

Several concepts have been proposed to explain why and how PAS occurs. The oldest concept is based on a theoretical primary defect of trophoblast biology leading to excessive invasion of the myometrium. The current prevailing hypothesis is that a secondary defect of the endometrium-myometrial interface leads to a failure of normal decidualization in the area of a uterine scar, allowing abnormally deep placental anchoring villi and trophoblast infiltration.[5] There is no doubt that the decidua normally regulates trophoblast invasion, as evidenced by the aggressive invasion of the muscular and serosal layers seen at sites of ectopic implantation in the fallopian tube[22] or in the abdomen.[23]

Scar implantation

During the secretory phase of the menstrual cycle, the endometrium transforms into a well-vascularized receptive tissue, which is characterized by the proliferation and differentiation of the stromal cells into decidual cells, the infiltration of maternal immune cells, and vascular remodeling of the endometrial vessels.[24,25] Decidualization of the endometrium stroma precedes blastocyst attachment and trophoblast infiltration. The process is complex and involves many local uterine components