

Placenta Percreta with Bladder Invasion: MRI Features of a Rare Case at FIGO Stage 3 Placenta Accreta Spectrum

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ABSTRACT

Placenta Accreta Spectrum (PAS) encompasses a continuum of abnormal placental attachment disorders, including placenta accreta, increta, and percreta. These conditions arise from a defective decidual interface that allows excessive trophoblastic invasion into the uterine wall. Among them, placenta percreta represents the most severe manifestation, characterized by complete penetration of the myometrium and uterine serosa, often extending into adjacent pelvic organs such as the urinary bladder. The incidence of PAS has markedly increased over recent decades, largely in parallel with the global rise in cesarean delivery rates and prior uterine surgeries. Other recognized risk factors include placenta previa, advanced maternal age, multiparity, and previous uterine instrumentation such as curettage. Clinically, placenta percreta is associated with catastrophic hemorrhage, massive transfusion requirements, adjacent organ injury, and high maternal morbidity and mortality. Early and accurate prenatal diagnosis is therefore crucial for improving maternal outcomes. Advances in ultrasonography and magnetic resonance imaging (MRI) have enabled more reliable detection of abnormal placental invasion and mapping of its extent, facilitating multidisciplinary surgical planning. Despite these improvements, management of placenta percreta remains complex and controversial, balancing maternal safety, fertility preservation, and surgical risks.

KEYWORDS: Placenta Accreta Spectrum (PAS), Magnetic Resonance Imaging (MRI).

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INTRODUCTION

Placenta Accreta Spectrum (PAS) is a condition characterized by abnormal trophoblastic invasion of the placenta into the myometrium of the uterine wall, representing one of the most severe complications in modern obstetrics (8). The spectrum encompasses three grades of severity based on the depth of invasion: *placenta accreta* (Grade 1), where chorionic villi adhere directly to the myometrium without an intervening decidua basalis; *placenta increta* (Grade 2), where the villi invade into the myometrium; and *placenta percreta* (Grade 3), the most severe form, where the villi penetrate the full thickness of the myometrium, breach the uterine serosa, and can invade adjacent pelvic organs (7).

The pathophysiology of PAS is predominantly explained by the “secondary defect” hypothesis, which posits that damage to the endometrium–myometrium interface from prior uterine instrumentation or surgery creates a site for abnormal placentation. Procedures such as cesarean delivery, myomectomy, or uterine curettage can result in scarring and deficient decidualization. In a subsequent pregnancy, if implantation occurs over this scar, the absence of a normal decidual layer allows extravillous trophoblasts to invade uncontrollably into the myometrial tissue and its deep vasculature (8). This iatrogenic etiology is underscored by the dramatic rise in PAS incidence, which has increased more than tenfold in the past several decades, mirroring the global increase in cesarean delivery rates (9).

Placenta percreta with invasion into the urinary bladder is a rare but particularly formidable manifestation of PAS, with an estimated incidence of approximately 1 in 10,000 births (11). This complication carries a substantial risk of life-threatening hemorrhage, urologic injury, and maternal mortality. Although hematuria is a logical presenting symptom, it is reported in only about 25–40% of cases with bladder invasion, making the clinical diagnosis challenging and frequently delayed (17, 18).

CASE REPORT

Case Presentation

A 27-year-old woman, gravida 5 para 2 abortus 2 (G5P2A2), at approximately 31 weeks of gestation, presented with a chief complaint of hematuria for two weeks prior to admission. She also reported lower abdominal pain that began one day before hospitalization and intermittent fever for three days. There was no history of vaginal bleeding during the current pregnancy.

Her obstetric history included two previous miscarriages, both managed with uterine curettage, followed by one cesarean section for her third pregnancy and one spontaneous vaginal delivery for her fourth pregnancy.

Obstetric ultrasonography revealed an anteriorly implanted placenta extending downward to cover the internal cervical os. The retroplacental clear zone was absent, with placental lacunae grade II–III, presence of bridging vessels, uterine wall bulging, and marked subplacental hypervascularity—findings highly suggestive of placenta accreta spectrum disorder.

Diagnostic Evaluation

The diagnostic workup included both ultrasonography (USG) and MRI to confirm the diagnosis and delineate the extent of invasion for surgical planning.

- **Ultrasonography (USG):** An initial USG performed at the referring hospital at a gestational age of 34–35 weeks identified a singleton fetus with appropriate biometry. It confirmed a complete anterior placenta previa and demonstrated multiple classic signs highly suggestive of PAS. These included a loss of the retroplacental clear zone, numerous large and irregular placental lacunae (Grade II–III), prominent bridging vessels at the utero-vesical interface, placental bulging into the bladder, and marked subplacental hypervascularity on color Doppler imaging. These sonographic markers are pathognomonic for an abnormally invasive placenta.
- **Magnetic Resonance Imaging (MRI):** A pelvic MRI was subsequently performed to provide superior anatomical detail and confirm the extent of organ invasion. The MRI confirmed a complete placenta previa (Grade IV) and the diagnosis of placenta percreta. The imaging crucially demonstrated transmural invasion of placental tissue through the anterior myometrium and uterine serosa. It revealed direct infiltration of the posteromedial wall of the urinary bladder through a defect measuring approximately 2.98 cm, with placental tissue extending into the bladder lumen. This finding is characteristic of the most severe form of PAS and necessitates complex surgical planning involving urological expertise.

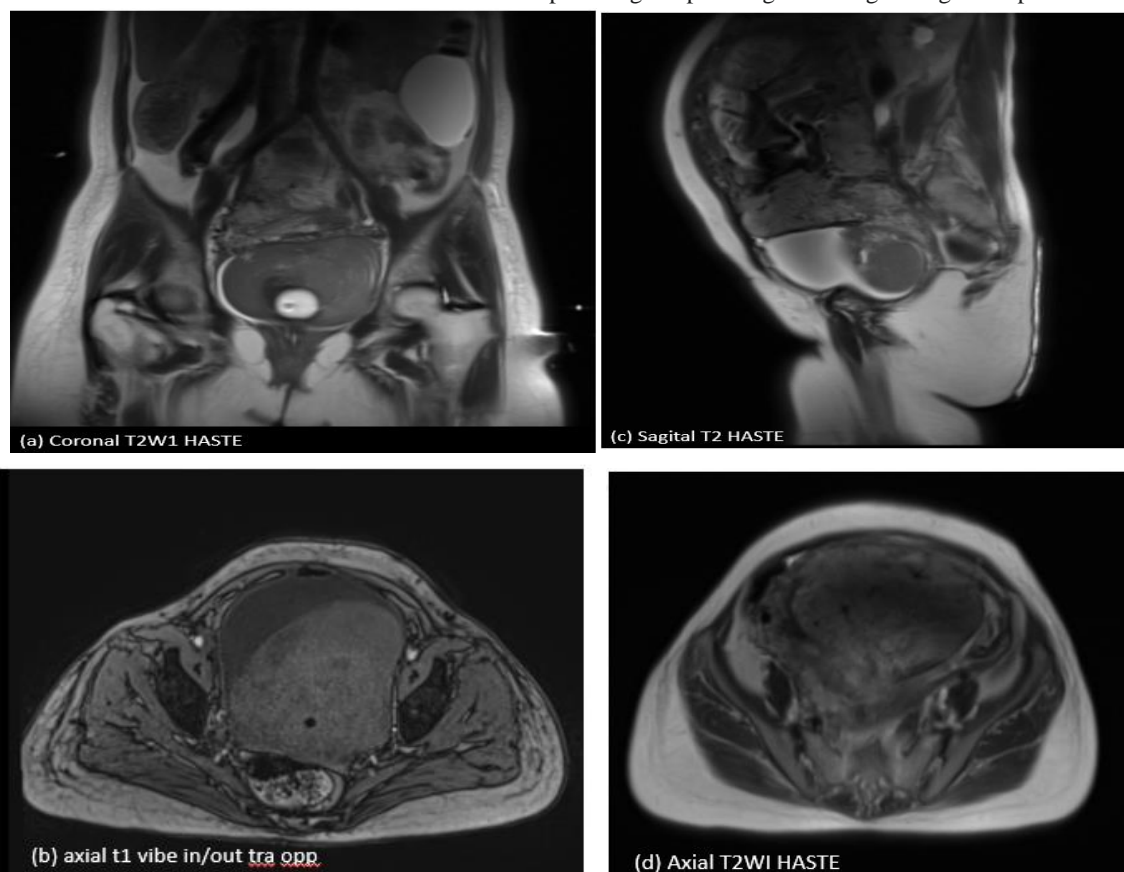


Figure 1. (a) coronal T2WI HASTE MRI image show direct placental infiltration of the posterolateral wall of the urinary bladder, accompanied by intravesical blood clot formation.

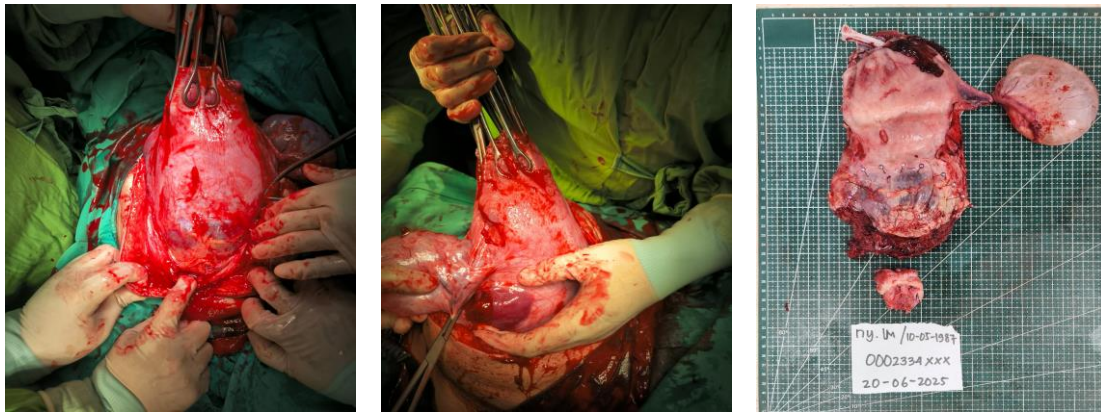
(b) axial T1 VIBE in/out tra opp MRI image shows inhomogeneous isointense to partially hyperintense signals and irregularity and demonstrated transmural invasion of placental tissue through the anterior myometrium and uterine serosa

(c) sagittal T2WI HASTE MRI image show direct placental infiltration of the posterolateral wall of the urinary bladder, accompanied by intravesical blood clot formation.

(d) axial T2WI HASTE MRI image show a lobulated placenta with irregular margins and complete loss of the retroplacental T2 dark zone at the anteroinferior and posteroinferior uterine segments

A planned cesarean hysterectomy was performed by a multidisciplinary team. Intraoperatively, extensive neovascularization and placental invasion into the bladder were confirmed, leading to an iatrogenic bladder rupture during dissection. The patient experienced massive intraoperative hemorrhage amounting to 17,000 cc. Through coordinated surgical efforts, including aortic

clamping and massive transfusion protocol, hemostasis was achieved, and both the mother and the 1890 g male infant survived.



DISCUSSION

This case serves as a powerful clinical exemplar of the established pathophysiology of Placenta Accreta Spectrum (PAS). The patient's obstetric history, featuring two uterine curettages followed by a cesarean section, represents a classic sequence of uterine insults that cumulatively increase the risk of abnormal placentation (8). Each procedure contributes to the formation of scar tissue and a defective endometrium–myometrial interface, creating a nidus for abnormal implantation. This “multiple-hit” model of uterine injury provides a compelling rationale for the severity of the disease observed. The initial, more diffuse endometrial damage from curettage, compounded by the deep, focal myometrial scar from the cesarean delivery, likely created a profoundly compromised implantation site. When the subsequent pregnancy implanted over this multiply-injured area, the trophoblastic tissue encountered a deficient decidua basalis, predisposing it not merely to abnormal adherence but to the most aggressive form of invasion—placenta percreta. This progression suggests that the risk and severity of PAS may be proportional to the cumulative burden of prior uterine trauma (9).

Magnetic resonance imaging (MRI) is widely regarded as the definitive imaging modality for assessing the depth of invasion in suspected PAS, offering superior soft-tissue contrast compared to ultrasound, especially in cases of posterior placentation or suspected extrauterine extension (4,12). The hallmark MRI features of placenta percreta include uterine bulging, heterogeneous placental signal with T2-dark intraplacental bands, and focal interruption of the hypointense myometrial and bladder walls (3,10). While MRI in this case correctly identified bladder wall disruption and intraluminal extension, the patient's presenting symptom of chronic and acute hematuria introduced a significant diagnostic confounder. The presence of an evolving hematoma within the bladder lumen can create considerable diagnostic ambiguity, potentially masking the true extent of placental invasion. The signal characteristics of blood products on MRI are highly dependent on their age. Placental tissue typically demonstrates an intermediate, heterogeneous signal on T2-weighted sequences (15). In contrast, an acute hematoma (less than 3 days old, containing deoxyhemoglobin) is characteristically T2-hypointense, whereas a late subacute hematoma (1–4 weeks old, containing extracellular methemoglobin) is T2-hyperintense (2,13).

This variability presents a formidable challenge for interpretation. The radiologist is confronted with an intravesical mass of mixed and overlapping signal intensities. The intermediate T2 signal of the invading placental tissue could be indistinguishable from the heterogeneous signal of a mixed-age clot. Furthermore, the T2-dark signal of an acute clot could be misinterpreted as an intact segment of the bladder wall, while the T2-bright signal of a subacute clot could be mistaken for surrounding edema or even urine. This makes defining the precise demarcation between invasive placenta, organized clot, and the native bladder wall exceptionally difficult. This inherent ambiguity underscores that while MRI is invaluable for surgical planning, the operative team must maintain a high index of suspicion and be prepared for intraoperative findings that are more extensive than what is depicted on imaging. The ultimate intraoperative diagnosis of a FIGO grade T4 complication (organ rupture) highlights this critical limitation of prenatal imaging in the setting of active hemorrhage (7,11).

The management of PAS in a specialized, high-volume center with a dedicated multidisciplinary team is now the undisputed standard of care and has been shown to significantly reduce maternal morbidity and mortality (5,16). This case provides a compelling testament to this principle. The coordinated, synergistic effort of the team was crucial at every stage of the patient's care:

Pre-operative Planning: The collaborative meeting of maternal-fetal medicine, anesthesiology, urology, and vascular surgery specialists allowed for the anticipation of massive hemorrhage and potential organ injury. This facilitated proactive measures such as securing adequate blood products, preparing for advanced hemodynamic monitoring, and ensuring the immediate availability of all necessary surgical expertise (8).

Intra-operative Execution: The surgical management was a masterclass in coordinated action. The maternal-fetal medicine and gynecologic oncology surgeons navigated the hostile surgical field to perform the hysterectomy. The vascular surgeon's timely and proficient placement of an infrarenal aortic clamp was a pivotal, life-saving maneuver that provided essential proximal vascular control, transforming an uncontrollable hemorrhage into a manageable surgical field (14). The urogynecology team's

expertise was then indispensable for performing a complex, multi-layered repair of the severely injured bladder (6). Simultaneously, the critical care anesthesiology team expertly managed the massive transfusion protocol, aggressively treating coagulopathy and preventing irreversible hypovolemic shock and cardiovascular collapse (16).

The seamless transitions between these highly specialized tasks—from delivery to hysterectomy, to aortic clamping, to bladder repair, and back to final hemostasis—demonstrate a level of system-level preparedness and a shared mental model that defines a true “Center of Excellence.” The successful outcome was not merely the result of having different specialists present in the operating room; it was the product of a pre-established, rehearsed protocol. This case strongly argues that tertiary centers managing PAS should invest in developing and drilling standardized protocols for these high-acuity, low-frequency events, as this system-level maturity is what ultimately ensures patient survival (1,16,17,18).

CONCLUSION

This case of placenta percreta with bladder invasion, ultimately classified as FIGO Stage 3, vividly illustrates the condition's potential for life-threatening maternal morbidity. It demonstrates how a classic confluence of historical risk factors can culminate in the most severe and invasive form of PAS. While advanced imaging modalities like MRI are indispensable for pre-operative planning, this case highlights that their diagnostic accuracy can be significantly confounded by clinical factors such as active hematuria, which may obscure the true extent of placental invasion. The successful maternal and neonatal outcome in this near-fatal case was unequivocally dependent on a pre-planned, highly coordinated surgical and anesthetic approach executed by a dedicated, protocol-driven multidisciplinary team operating within a tertiary referral center. This underscores the principle that in the management of severe PAS, system-level preparedness is paramount to survival.

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