A simple guide to ultrasound screening for placenta accreta spectrum for improving detection and optimizing management in resource limited settings

the International Society of Placenta Accreta SPECTRUM (IS-PAS) Low- and Middle-Income Countries Working Group

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REVIEW ARTICLE

Obstetrics

A simple guide to ultrasound screening for placenta accreta spectrum for improving detection and optimizing management in resource limited settings

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Placenta accreta spectrum is a pregnancy complication associated with severe morbidity and maternal mortality especially when not suspected antenatally and appropriate management instigated. Women in resource-limited settings are more likely to face adverse outcomes due to logistic, technical, and resource inadequacies. Accurate prenatal imaging is an important step in ensuring good outcomes because it allows adequate preparation and an appropriate management approach. This article provides a simple three-step approach aimed at guiding clinicians and sonographers with minimal experience in placental accreta spectrum through risk stratification and basic prenatal screening for this condition both with and without Doppler ultrasound.

KEYWORDS

cesarean, increta, invasive placenta, maternal morbidity, morbid adherence, Percreta, placenta accreta, placenta previa, prenatal diagnosis, ultrasound, uterine scar

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1 | INTRODUCTION

1.1 | Background and pathophysiology

Placenta accreta spectrum (PAS) is a rare pregnancy complication where the placenta fails to separate spontaneously after delivery and cannot be forcibly separated without causing catastrophic obstetric hemorrhage.[1] It is caused by abnormal placental implantation over a myometrial scar, and results in extrusion of placental tissue beyond the usual confines of the intrauterine cavity with fibrinoid deposition, and massive neovascularity.[2] PAS is typically graded according to the extent of placenta involvement ranging from abnormal adherence to the myometrium (accreta), to deep myometrial implantation (increta), and percreta, which breaches the serosal surface and may involve other surrounding structures. The degree of morbidity is highly dependent on the degree of extension, amount of neovascularity and involvement of other pelvic structures. In placenta accreta, the placenta can sometimes be detached because it is only adherent, as long as there is sufficient myometrium underlying the placenta to enable adequate uterine contraction to prevent catastrophic hemorrhage. Any attempt to manually remove the placenta in the more serious phenotypes (increta and percreta) can cause a uterine rupture and significant bleeding. Even if it does not, there is insufficient myometrium to contract and provide the "living ligature" of the terminal arteries. The normal physiologic changes of pregnancy result in placental perfusion approaching 800 mL/min at term.[3] Hence, rupture of the uterus or inadequate myometrial contraction results in torrential hemorrhage. The mainstay of management for PAS is not to disturb the placental bed.[4] The involvement of other pelvic structures in percreta requires a multidisciplinary team with experience of PAS to ensure safe dissection of surrounding structures at hysterectomy.[5] PAS is associated with a very high risk of maternal mortality, especially if the surgeon is caught unaware. In high-income countries with an abundance of experienced surgeons and readily available life-saving resources the maternal mortality rate has been reported to be 7% at the severe end of the spectrum.[6] Although the exact comparative figure remains unknown, it is likely that women with PAS in resource-limited settings have a much greater risk of death due to technical, logistic, and resourcing inadequacies.

1.2 | Establishing the need for the review

In 1994, the three-delay model was developed to offer health programs options to prevent maternal death with an emphasis on strategies to mobilize and adapt existing resources. The focus was to understand barriers in the interval between onset of obstetric complications and their outcome, and identify the point at which healthcare delivery could be optimized[7]: (1) delay in the decision to seek care; (2) delay in arrival at a health facility; and (3) delay in the provision of adequate care. We have used the philosophy of the three-delay model to propose an "IS-PAS 4-A strategy" to ensure optimal outcomes of PAS namely; (1) **A**wareness of the risk factors of the condition, (2) **A**ccurate prenatal diagnosis, (3) **A**dequate preparation, and (4) **A**ppropriate management. In this paper we aim to concentrate on the first two parts of the IS-PAS 4-A strategy. Our second article will address preparation and management in low- and middle-income countries.

Although the incidence of PAS is still relatively rare, the numbers are rising as the result of mounting cesarean section rates. An increased awareness leading to appropriate screening is important in improving maternal outcomes. Accurate prenatal diagnosis is often challenging because the published literature on the sonographic signs of PAS can be contradictory and potentially confusing to sonographers with limited experience with the condition. Even though the diagnosis can be readily made with B-mode (gray-scale) ultrasound, recent reports focus on Doppler technology, which is often not present on ultrasound equipment used in limited-resource settings.

The role of ultrasound in PAS screening also needs to move beyond making a binary diagnosis of presence or absence of PAS. Rather, there is the need to consider the anatomical findings that each sign represents and how that could influence planning for the surgery and the potential need for referral to a more experienced team, which can be extremely challenging in low-resource settings. This article aims to provide a simplified three-step approach for fetal imaging personnel to improve their understanding of the process for the basic screening for PAS, with and without Doppler ultrasound.

2 | STEP 1: ASSESS THE PRE-TEST PROBABILITY OF PAS

Defining the pre-test probability is a standard procedure in medical practice, which refers to the likelihood of a condition being present before a diagnostic test is performed. The greater the underlying risk of the condition, the higher the diagnostic accuracy most medical tests will have. The assessment of pre-test probability is essential in reducing false-positive diagnoses, especially as a recent study demonstrated that two or more imaging signs of PAS were present in the second trimester for 98% of low-risk pregnancies

with normal placentation.[8] Previous damage to the uterus predisposes to developing PAS in subsequent pregnancies. However, the depth of the original myometrial injury determines the likely severity of PAS as well as the morbidity associated with it.

2.1 | Superficial uterine cavity injury

Women who have sustained damage to the uterine cavity from curettage, radiation, endometritis, endometrial ablation, or endometrial resection, are at increased risk of some degree of abnormal adherence of the placenta to the superficial myometrium (placenta accreta). In such instances, the myometrial tissue deep to the area of abnormal attachment is often thick enough to provide adequate uterine contraction after placental separation. It should be noted that no imaging modality, neither ultrasound nor MRI, can rule out abnormal adherence because imaging signs may be very subtle or even absent. Therefore, if risk factors are present, the most important ultrasound measurement is the myometrial thickness under the placenta, as this will be the most important feature should the placenta not separate spontaneously. A myometrium of 5 mm or more is likely to be able to contract adequately after delivery to provide a significant "living ligature" or at least facilitate the performance of local hemostatic sutures to control uncontrolled bleeding due to ineffective uterine contraction (expert opinion).

Instances where women have a "sticky" placenta or one entrapped behind a closed cervix, which are managed with manual removal of placenta and uterotonics only, are not considered to be PAS.[9] These should not be recorded or reported as PAS, but rather as a "retained placenta".

2.2 | Full thickness uterine wall injury

This is where the entire uterine wall has been disrupted from cavity to serosal surface and is usually a result of a surgical incision but may be from a perforation or previous uterine rupture. The surge in cesarean delivery rate in recent years has increased the number of women with a full thickness scar on their uterus, thereby increasing the risk of the more severe phenotypes of PAS.[10] The risk of PAS rises with increasing amounts of scar tissue, as shown by the dose relationship seen between risk of PAS and number of previous cesarean deliveries. When the placenta implants in the lower segment over the scar of a single cesarean delivery the risk of PAS is 4%, this increases to 11% with two previous cesarean deliveries and 40% with three.[9] Hence, thorough assessment and screening for PAS should always be carried out when anterior low-lying placenta/previa is seen in a woman with previous cesarean delivery.

It should be emphasized that placenta previa without previous damage to the lower segment of the uterus is very unlikely to be associated with a clinically significant PAS. Similarly, the presence of placenta overlying an area of full thickness scar from myomectomy, a previous rupture, or iatrogenic uterine perforation, also have a 4% risk of PAS. However, a myomectomy involving subserosal myomas that did not breach the cavity do not increase the woman's risk of clinically significant PAS.

2.3 | Previous cesarean delivery with placenta previa or low-lying placenta: a challenging combination

Although abnormal invasion can occur anywhere in the uterus where there is a full thickness scar, the combination of an anterior low-lying placenta (≤2 cm from the internal cervical os) or a previa with a history of previous cesarean remains the most dangerous type of PAS for multiple reasons; a previa increases the risk of vaginal bleeding and the potential for emergency delivery; the poor contractility of the lower segment means a previa is more likely to bleed more heavily even with normal placentation; a lower segment incision runs the risk of transecting the placental bed, which will result in significant hemorrhage; the placental bed is in close proximity to other structures including the bladder and ureters, making collateral damage at hysterectomy a greater risk; and the blood supply to the placental bed may come from not only the uterine arteries but also other arteries lower in the pelvis, making vascular control much harder in the event of hemorrhage.[11] Finally, in the lower uterine segment and pericervical tissue, the ureters cross in close proximity to where the uterine arteries enter the uterus and lie within the narrowest portion of the bony pelvis. Hence, lateral placental bulging in this region makes surgical dissection even more technically difficult and risks ureteric injury.

2.4 | Other risk factors

There have been reported cases of PAS after in vitro fertilization with embryo transfer without previous endometrial injury.[12,13] The risk of PAS in twin pregnancies is approximately four-fold higher than in singletons.[14] The exact reason is unclear; however, clinicians should be mindful of this risk and consider thorough placental ultrasound evaluation.

2.5 | Key take home message

If, on ultrasound scan, a woman is seen to have a previa or anterior low-lying placenta (≤2 cm from internal os,) the operator should ask if she has had a previous cesarean delivery. If she has, this must prompt thorough examination of the placental bed for signs of PAS by the person with the most experience available (consider tertiary referral or telemedicine if possible). Any concerns should prompt a request for a second opinion from a sonographer with expertise in diagnosing PAS.

3 | STEP 2: REMEMBER PAS IS A SPECTRUM, NOT A BINARY CONDITION

As the name suggests, PAS is not a binary condition, it ranges from abnormal adherence to involvement of surrounding pelvic structures. Varying grades of PAS often co-exist in the same placental bed[15] as well as some areas of scar dehiscence without abnormal adherence.[16] Due to these differences, the intraoperative appearance and architecture of the uteroplacental interface varies significantly from case to case. This is reflected in the prenatal sonographic findings. In view of this, there can never be a single sonographic sign that diagnoses PAS. Accurate ultrasound diagnosis therefore requires consideration of each of the imaging signs, such as a placental bulge or the lacunae, and what they represent. This will also provide vital information that should influence the management approach.

4 | STEP 3: CONSIDER THE CLINICAL FEATURE THAT EACH SONOGRAPHIC SIGN REPRESENTS

4.1 | Technical points

When screening for PAS, in a woman with previous cesarean delivery with low-lying placenta previa, the urinary bladder must be filled to the extent that the entire lower uterine segment and the uterovesical interface can be well visualized.[17] This may be uncomfortable for some women, but it is necessary for proper evaluation. Most women will report the urge to urinate well before they have a sufficiently filled bladder, but they should be encouraged to wait as incontinence is unlikely to occur. The choice of the scanning approach should be based on the experience of the operator, clinical and cultural context. Both transabdominal and transvaginal approaches give excellent results depending on the skill of the operator and quality of the gray-scale resolution of the ultrasound equipment. In patients with high body mass index (calculated as weight in kilograms divided by the square of height in meters), scanning difficulty with transabdominal approach can be overcome by asking the woman to lift her pannus and scanning beneath it or by using the transvaginal approach. The ideal time for thorough PAS screening is above 28 weeks to reduce false-positive diagnosis because two or more PAS imaging signs are seen in 98% of normal placentation in the second trimester.[8] Also, most signs of PAS become more prominent in the third trimester.

4.2 | Sonographic signs

Neovascularization

Abnormally invasive placentation (increta and percreta) will usually be accompanied by neovascularity, which occurs at the level of the serosal surface of the uterus (within the utero-vesical fold of the peritoneum in the case of a low-lying/previa placenta). This is seen at laparotomy as multiple, newly formed large vessels on the uterine surface at the area of abnormal invasion (Figure 1a).[9] The underlying pathophysiology remains unclear, however, recent histologic evidence indicates that PAS provokes rapid increased growth of originally much smaller vessels, which is why they have such immature vascular architecture including a poorly formed vessel wall.[18] This neovascularity has been described with a variety of different imaging signs, including uterovesical hypervascularity, sub-placental hypervascularity, bridging vessels, and bladder wall interruption.[19] The most useful of these is probably bridging vessels (Figure 1a,b), because these only occur when there is a significant amount of neovascularity at the serosal surface.

Ultrasound appearance

Depending on the number, size and course of the new vessels, this sign can manifest on gray-scale imaging in different ways at the uterine serosal surface (uterovesical interface in an anterior low/previa placenta). It can appear as pairs of hyperechoic lines (sometimes referred to as the "=" sign) running parallel to the uterine serosa and posterior bladder wall. This is generated by the ultrasound being reflected from both walls of the vessel producing an "=" sign

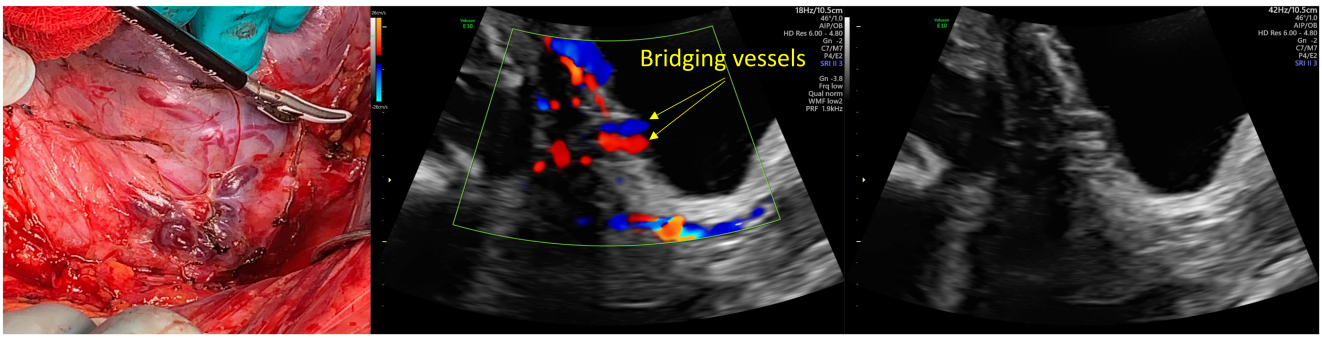


FIGURE 1 Demonstrating the same neovascularity seen at laparotomy (a), as bridging vessels with color Doppler ultrasound (b) and as bladder wall interruption (c). (a) Demonstrating the massive neovascularity seen at laparotomy between the anterior aspect of the uterus and the posterior bladder. (b) Demonstrating the same vessels as in (a) seen as 'bridging vessels' with color Doppler ultrasound. (c) Demonstrating the same vessels as in (a) seen as bladder wall interruption with gray-scale ultrasound.

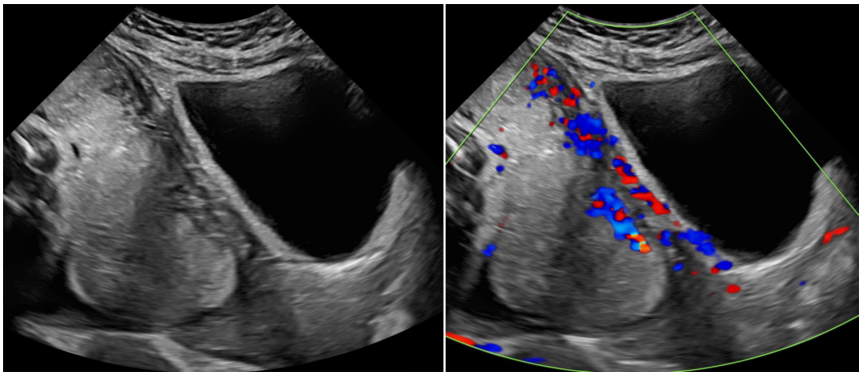
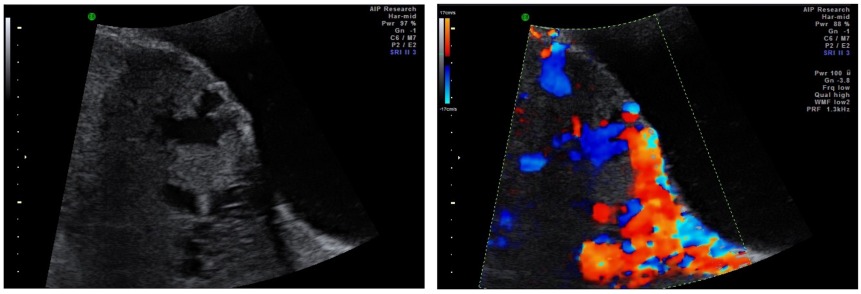


FIGURE 2 Demonstrating the thick, cystic vascular myometrium of a woman with proven adenomyosis at subsequent hysterectomy and a normal placenta—this should not be mistaken for sub-placental hypervascularity.



(a) (b)

FIGURE 3 Demonstrating the irregular large lacunae continuous with the placental bed and "fed" by large, high-velocity vessels. (a) Placenta accrete spectrum (PAS) lacunae; (b) PAS lacunae with color Doppler demonstrating high-velocity blood flow into the lacunae.

or a scalloped appearance. This is the sign often referred to as "bladder wall interruption" (Figure 1b) and is seen as bridging vessels with color Doppler imaging (Figure 1c). It must be emphasized that the appearance of "bridging vessels" is an ultrasound artefact caused by the cross-sectional two-dimensional imaging of the contorted neovascularity as it curves around a three-dimensional structure (the front of the uterus). It does not represent blood vessels actually "connecting" the placental bed with the urinary bladder.[20]

Subjective hypervascularity

The placenta is a highly vascular organ, and some signs such as "hypervascularity" are entirely subjective and must therefore be interpreted with caution depending on operator experience. It must be emphasized that the surgically challenging neovascularity associated with PAS occurs at the serosal surface (Figure 1c) and not within the myometrium. Care must be taken not to confuse conditions, such as adenomyosis, which manifest as thick cystic and vascular myometrium (Figure 2) from the neovascularization of PAS, which is associated with an abnormally thin myometrium. Also, the Doppler gain and pulse repetitive frequency must be adjusted to reduce blooming and motion artefacts as well as avoiding exaggerating normal sub-placental vascularity, which may give a false representation of hypervascularity. A pulse repetitive frequency setting of above 15 cm/s satisfies these requirements in most cases.

4.3 | Myometrial invasion

Abnormal lacunae

The presence of abnormal lacunae is the most common ultrasound sign of PAS present in literature.[21,22] Lacunae are multiple, large, irregular anechoic areas noted within the placenta, which give the placenta a "moth-eaten" appearance (Figure 3a).[20] In severe PAS, placental tissue is found more deeply within the myometrium than it should be, often passing the level of the spiral arteries and reaching the radial and arcuate vessels. This causes excessive dilatation of these higher pressure arteries[23] and a massively increased velocity of flow into the delicate intervillous space (Figure 3b).[24] This powerful flow distorts the architecture of one or more cotyledons and its corresponding interlobar septa, resulting in lacunae formation.[20] Although these lacunae can sometimes be seen throughout the whole placenta because of the underlying pathophysiology, on careful inspection they should be seen to be adjacent to the basal plate of the placenta. If the "placental holes" seen are only on the fetal side of the placenta or just at the edges, it must be carefully considered as to whether they actually are PAS-related lacunae.

PAS-related lacunae (Figure 3a) must not be confused with placental lakes (Figure 4a) or echogenic cystic lesions (Figure 4b). Placental lakes are cystic spaces (greater than 10 mm) usually centrally located within the cotyledon or lobule surrounded by placental tissue of normal echogenicity.[25] Placental lakes are frequently confused

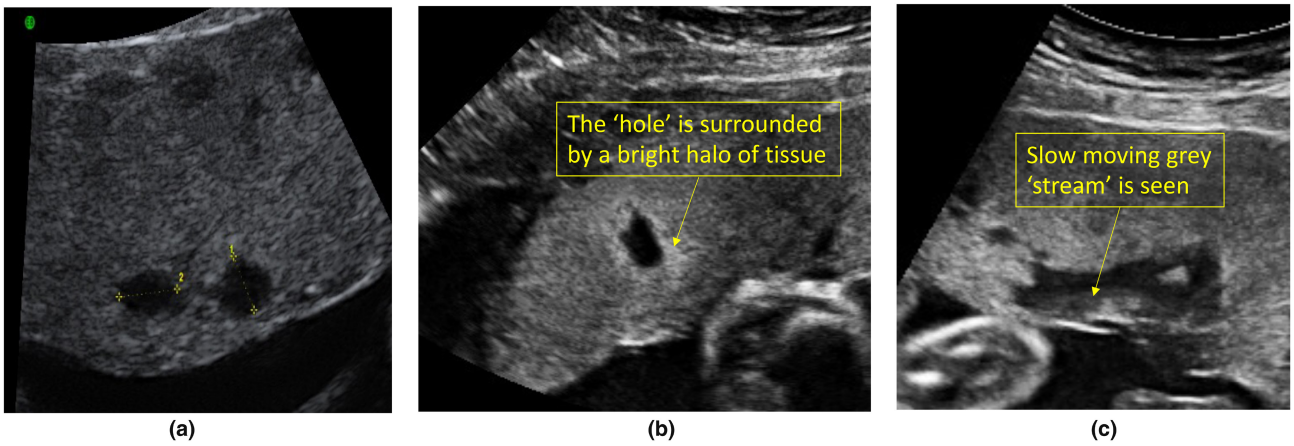


FIGURE 4 (a) Placental lake, (b) echogenic cystic lesion, (c) lake with low-velocity flow visible on gray-scale (B mode) imaging. (a) Simple placental lakes – note there is no increased brightness in the surrounding tissue. (b) Echogenic cystic lesion (resulting from a placental infarct). (c) Placental lake with slow flow visible in gray-scale—note the position away from the placental basal plate.

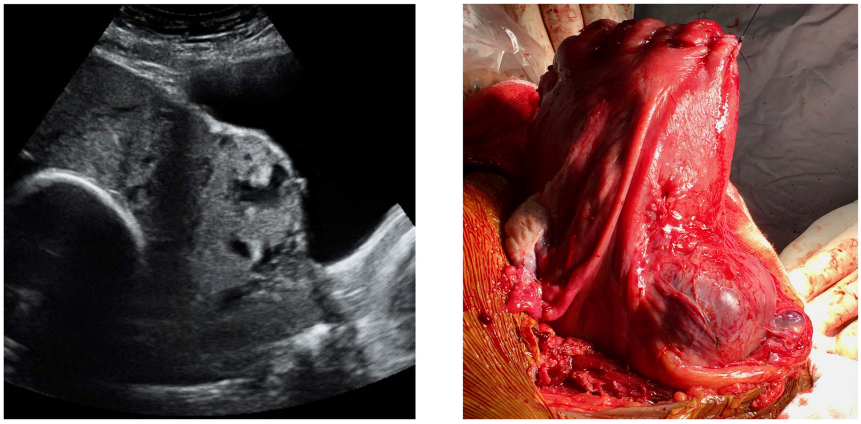


FIGURE 5 Examples of a placental bulge on ultrasound scan and after delivery—the "bulge" is caused by a loss of structural integrity in the muscle of the lower segment causing it to bulge outwards, it becomes more pronounced after delivery as the upper segment contracts.

with PAS-related lacunae and the terms "lake" and "lacunae" are often incorrectly used interchangeably. This is evidenced by studies that report the presence of lacunae in normal placentation in low-risk pregnancies.[8,26] On real time gray-scale imaging, lacunae and lakes appear as hypoechoic areas within the placenta. However, typical PAS-related lacunae are often irregularly ellipsoid in shape and extend from the placental bed where they receive blood supply from the feeder vessels (deep myometrial vessels, i.e. radial or arcuate arteries). Placental lakes often, but not always, contain slow-moving blood and are easily compressible with the ultrasound probe whereas typical PAS-related lacunae are not compressible. It must be noted that lacunae and lakes can be present in the same PAS placenta. However, they should be differentiated based on their size and location. Also, PAS-related lacunae are often numerous in the region of the abnormal placentation whereas lakes are usually few and widely distributed.

Placental infarcts result from interrupted maternal blood supply to the placenta and often present as hypoechoic regions with hyperechogenic rim (echogenic cystic lesion) or well-circumscribed lesions with mixed echogenicity and are often associated with pre-eclampsia and fetal growth restriction.[27] Placental infarcts can be differentiated from lakes and lacunae by the characteristic hyperechoic rim.

Color Doppler

PAS-related lacunae can usually be confirmed by demonstrating high-velocity flow (>10 cm/s) with feeder vessels on Doppler interrogation (Figure 3b), whereas lakes sometimes show very low flow velocity for which the signals are rarely detected on Doppler but can be seen in gray-scale imaging (Figure 4c). Echogenic cystic lesions resulting from placental infarcts show no flow on color Doppler interrogation but occasionally contain static or very slow moving, irregularly shaped contents.

Placental bulge

A placental bulge describes the outpouching of the uterus containing the placenta due to inadequate residual myometrium to maintain the structural integrity of the uterus (Figure 5).[20] This sign can be seen with both ultrasound and MRI and is highly predictive of increta

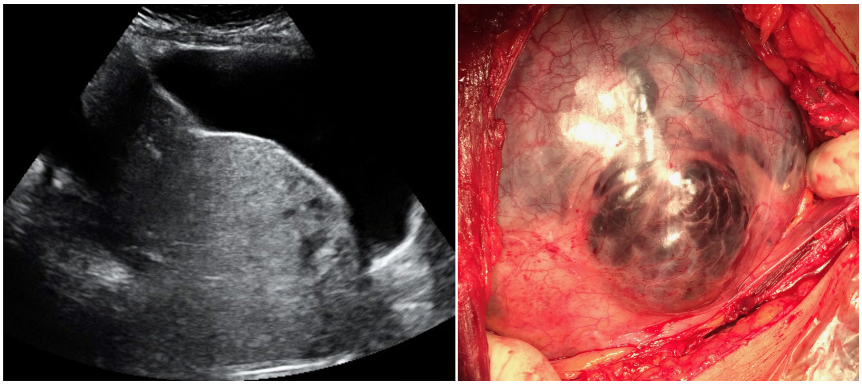


FIGURE 6 Example of a uterine dehiscence—the bladder is full at ultrasound but empty at laparotomy, hence it has collapsed down revealing the bulge of placenta, note the completely normal uterine tissue around the defect and the lack of signs of placenta accrete spectrum on the ultrasound.

or percreta when used in combination with other imaging signs of PAS.[28] This sign represents the absence of sufficient myometrial tissue to support the placenta and hence is extremely useful in both the prenatal diagnosis of PAS and the subsequent management plan. If there is a bulge, the clinician can be confident that there is not enough residual muscle to contract and provide the living ligature required to stop bleeding from the placental bed therefore, forced removal of the placenta should not be attempted because it will result in bleeding.

A placental bulge can also occur with a normal placenta as a result of progressive dehiscence of a uterine scar resulting in the underlying placenta bulging through it.[16] However, in these cases, the bulge is usually smaller, the placenta is fairly homogeneous with no evidence of placental lacunae, neovascularization, or other signs of PAS (Figure 6).

Loss of retroplacental "clear zone"

The retroplacental hypoechoic zone is the echolucent space between the placenta and endometrium. A clear understanding of what the retroplacental hypoechoic zone represents is yet to be determined; however, it has been linked to the presence of decidua glands and vascular plexus involving basal arteries and terminal branches of the spiral arteries.[20] The absence of this sign indicates a loss or deficiency of Nitabuch's layer and the subsequent "fusion" of placenta and myometrium. This is currently the only known direct marker for PAS. However, the specificity of this sign is an issue of controversy because of its susceptibility to false-positive results. The retroplacental hypoechoic space is influenced by external compressive force, usually from pressing hard with the ultrasound probe. Due to the susceptibility of the retroplacental hypoechoic zone to compressive effect, care must be taken to prevent/minimize the compressive effect of the probe when assessing the uteroplacental bed for the presence of the clear zone.[17] This sign becomes more prominent in advancing gestation because of myometrial thinning and prominent dilatation of the uteroplacental circulation.[29] To have a clear assessment on ultrasound, the dynamic range, chroma(tint) and focus must be used to improve contrast resolution and zoom feature, to enlarge the image for thorough assessment.

Myometrial thinning

Myometrial thickness less than 1 mm, or an area of imperceptible myometrium behind the placenta, has been reported as a sign of PAS.[30] Myometrial thinning has been attributed to the progressive migration/invasion of the extravillous trophoblast through the abnormally healed myometrium resulting in minimal/absent myometrium to support the placental bed. In this case, any attempt to separate the placenta, could result in torrential hemorrhage. However, data obtained do not establish a clear association between myometrial thickness and the severity of PAS. Both myometrial thicknesses of more than 2 mm[31–33] and abnormally thin myometrium[29] have been reported in PAS. To be clear, thinning of the lower uterine segment can be a normal finding in the third trimester and may be related to fetal presentation. This may be further emphasized after a previous cesarean delivery due to significant scar thinning resulting from poor myometrial healing.[34]

Occasionally, the placenta may overlie an area of simple uterine scar dehiscence without any abnormal placentation, this phenomenon is known as a "uterine window" because the placenta can be seen through the lower segment at delivery[4] (Figure 6). Even though this is not PAS, there is a significant risk of uterine rupture so it must be approached with care. The presence or absence of additional sonographic and clinical markers of PAS will differentiate between the two pathologies.[16] Hence, when screening for PAS, myometrial thickness should not be used as the sole parameter for diagnosis but must be used in conjunction with other imaging signs.

4.4 | Involvement of the cervix

In rare cases, the placenta can involve the cervix (probably as a result of the previous cesarean scar being on the cervix as can occur with a fully dilated cesarean delivery). In such cases, the whole lower segment appears to be filled with bulging placenta and the cervix shows massive hypervascularity. Often the cervix itself is virtually invisible transabdominally. It is important to know about this from the imaging because

it should guide subsequent management. Any attempt at focal resection or sub-total hysterectomy in these situations risks massive hemorrhage.

5 | RULING OUT PAS

The signs described so far demonstrate the uterine/placental morphologic changes when there is abnormal placentation. However, lack of these signs does not definitely rule out all cases of PAS, as abnormal adherence has virtually no associated signs. However, to have clinically significant invasion there should be some myometrial compromise, so a thick myometrium with no placental bulge is very unlikely to be seen in cases of increta or percreta. Currently, the only direct markers described in literature are the loss of the retroplacental hypoechoic zone. This marker is valuable for ruling out PAS but not for ruling it in. In simple words, the presence of the retroplacental hypoechoic zone excludes the presence of PAS in that area of placental bed. However, care must be taken to develop sufficient experience to assess these signs and to examine the whole placenta bed before PAS is ruled out.

5.1 | Key take home message

PAS is not a binary condition; it is a spectrum from abnormal adherence to severe percreta; hence the ultrasound appearance will significantly differ from case to case. The sonographer must therefore consider the underlying pathophysiology of the known ultrasound markers of PAS when screening high-risk patients and report on the clinical implications of all the signs seen. The steps have been summarized in Table 1.

6 | SUSPICION OF PAS: WHAT TO DO NEXT

In a high pre-test probability for PAS, we recommend thorough screening of the entire placental bed by an expert in the prenatal diagnosis of PAS. Modern technology and the use of telemedicine have performed well in some low-resource settings as a novel way to bridge the gap of distance, for example when the closest referral center is hours away or in a neighboring country.[35] When available, use of telemedicine has the potential to obtain an expert second opinion without adding undue financial burdens or disruption to the daily life of the woman and her family, simply to repeat imaging elsewhere.

Placentation process is completed in the middle of the second trimester.[36] Beyond this point, further trophoblastic invasion does not take place. However, the need for follow up in diagnosed cases of PAS remains uncertain and is a topic of debate. Currently, only two published studies longitudinally assessed the progression of PAS from the first trimester till delivery.[37,38] Both studies revealed no significant changes in the ultrasound signs between the second and third trimesters. Changes seen within the placenta in the third trimester are only related to ageing of the placenta, making the signs easier for the sonographer to see, not the progression of invasion.[38]

TABLE 1 Simplified steps in PAS screening

Step 1: Awareness
- Assess risk factors for PAS (*pre-test probability*)
  - **Anterior low-lying (<2 cm from internal os) or placenta previa + previous cesarean delivery/ies**
  - History of uterine surgery or myometrial/endometrial damage

Step 2: Remember it is a spectrum
- Ultrasound signs represent different anatomical features, e.g. neovascularity
- Severity and intra-operative findings vary significantly
- **Each PAS will have different ultrasound signs representing anatomical features unique to that case**

Step 3: Consider the clinical relevance
- **Utero-placental bed**
  - Placental bulge = defect in uterine muscle (PAS or dehiscence) definitely insufficient muscle to contract
  - Myometrial thinning (<1 mm or undetectable) = probably insufficient muscle to contract
  - Loss of "clear zone" = loss of smooth placental surface with probable "fusion" of placenta to uterus
- **Abnormal lacunae**
  - Large, irregular, anechoic areas connecting with the myometrium = destruction of the placental tissue by high pressure "feeder" blood vessels from deep within the uterus (radial/arcuate arteries)
  - *If Doppler is available the feeder vessels can be seen (>10 cm/s)*
- **Neovascularization signs**
  - Bladder wall interruption = presence of tangled mat of new blood vessels between the anterior uterine wall and posterior bladder wall (ultrasound artifacts from vessel walls cause "=" appearance or "scalloping")
  - *If Doppler is available this is seen as "bridging vessels"*

Abbreviation: PAS, placenta accreta spectrum.

Improving outcomes in pregnancies complicated by PAS should not end with prenatal diagnosis, adequate preparation and appropriate management play a crucial role in improving the outcome. Our next article will focus on how these two very crucial factors can be achieved in a limited-resource setting.

7 | CONCLUSION

The incidence of PAS may be rare, but it is definitely rising with the increasing rate of cesarean deliveries worldwide. This article is intended to provide a simplified guide to aid the thought process of ultrasound operators when they need to undertake prenatal screening for PAS. It is vital that they do not try to just identify the signs but consider what each sign represents anatomically and how this correlates with subsequent surgical challenges. Then report this to their surgical colleagues in a way that they understand.

AUTHOR CONTRIBUTIONS

TAB and SC conceptualized this review and drafted the initial manuscript. MJ, AJN-C, VS, RAA, and KF reviewed the initial draft and contributed to finalizing the manuscript.

CONFLICT OF INTEREST

The authors have declared that there are no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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REFERENCES

1. Morlando M, Collins S. Placenta accreta spectrum disorders: challenges, risks, and management strategies. *Int J Women's Health*. 2020;12:1033-1045. doi:10.2147/IJWH.S224191
2. Jauniaux E, Hussein AM, Elbarmelgy RM, Elbarmelgy RA, Burton GJ. Failure of placental detachment in accreta placentation is associated with excessive fibrinoid deposition at the utero-placental interface. *Am J Obstet Gynecol*. 2022;226:243.e1-243.e10. doi:10.1016/j.ajog.2021.08.026
3. Battaglia FC, Meschia G. Review of studies in human pregnancy of uterine and umbilical blood flows. *Med Wieku Rozwoj*. 2013;17:287-292.
4. Collins SL, Alemdar B, van Beekhuizen HJ, et al. Evidence-based guidelines for the management of abnormally invasive placenta: recommendations from the International Society for Abnormally Invasive Placenta. *Am J Obstet Gynecol*. 2019;220:511-526. doi:10.1016/j.ajog.2019.02.054
5. Eller AG, Bennett MA, Sharshiner M, et al. Maternal morbidity in cases of placenta accreta managed by a multidisciplinary care team compared with standard obstetric care. *Obstet Gynecol*. 2011;117:331-337. doi:10.1097/aog.0b013e3182051db2
6. O'Brien JM, Barton JR, Donaldson ES. The management of placenta percreta: conservative and operative strategies. *Am J Obstet Gynecol*. 1996;175:1632-1638. doi:10.1016/s0002-9378(96)70117-5
7. Thaddeus S, Maine D. Too far to walk: maternal mortality in context. *Soc Sci Med*. 1994;38:1091-1110. doi:10.1016/0277-9536(94)90226-7
8. Philips J, Gurganus M, DeShields S, et al. Prevalence of sonographic markers of placenta accreta spectrum in low-risk pregnancies. *Am J Perinatol*. 2019;36:733-780. doi:10.1055/s-0038-1676488
9. Jauniaux E, Ayres-de-Campos D, Langhoff-Roos J, Fox KA, Collins S. FIGO classification for the clinical diagnosis of placenta accreta spectrum disorders. *Int J Gynecol Obstet*. 2019;146:20-24. doi:10.1002/ijgo.12761
10. Jauniaux E, Bhide A. Prenatal ultrasound diagnosis and outcome of placenta previa accreta after cesarean delivery: a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2017;217:27-36. doi:10.1016/j.ajog.2017.02.050
11. Jaraquemada JMP, Mónaco RG, Barbosa NE, Ferle L, Iriarte H, Conesa HA. Lower uterine blood supply: extrauterine anastomotic system and its application in surgical devascularization techniques. *Acta Obstet Gynecol Scand*. 2007;86:228-234. doi:10.1080/00016340601089875
12. Salmanian B, Fox KA, Arian SE, et al. In vitro fertilization as an independent risk factor for placenta accreta spectrum. *Am J Obstet Gynecol*. 2020;223:568.e1-568.e5. doi:10.1016/j.ajog.2020.04.026
13. Ito M, Oshita K, Tanaka K, Hara M, Hiraki T. Massive obstetric hemorrhage during cesarean section in a patient after conception by frozen-thawed embryo transfer: a case report. *JA Clin Rep*. 2020;6:2. doi:10.1186/s40981-019-0308-0
14. Miller HE, Leonard SA, Fox KA, Carusi DA, Lyell DJ. Placenta accreta spectrum among women with twin gestations. *Obstet Gynecol*. 2021;137:132-138. doi:10.1097/AOG.0000000000004204
15. Luke RK, Sharpe JW, Greene RR. Placenta accreta: the adherent or invasive placenta. *Am J Obstet Gynecol*. 1966;95:660-668. doi:10.1016/S0002-9378(16)34741-X
16. Adu-Bredu TK, Owusu-Bempah A, Collins S. Accurate prenatal discrimination of placenta accreta spectrum from uterine dehiscence is necessary to ensure optimal management. *BMJ Case Rep*. 2021;14:e244286. doi:10.1136/bcr-2021-244286
17. Chantraine F, Collins SL. Prenatal ultrasound imaging for placenta accreta spectrum (PAS): a practical guide. *Curr Obstet Gynecol Rep*. 2019;8:86-93. doi:10.1007/s13669-019-00267-8
18. Schwickert A, Henrich W, Vogel M, et al. Placenta percreta presents with neoangiogenesis of arteries with Von Willebrand factor-negative endothelium. *Reprod Sci*. 2021;29:1136-1144. doi:10.1007/s43032-021-00763-4
19. Collins SL, Ashcroft A, Braun T, et al. Proposal for standardized ultrasound descriptors of abnormally invasive placenta (AIP). *Ultrasound Obstet Gynecol*. 2016;47:271-275. doi:10.1002/uog.14952
20. Jauniaux E, Collins S, Burton GJ. Placenta accreta spectrum: pathophysiology and evidence-based anatomy for prenatal ultrasound imaging. *Am J Obstet Gynecol*. 2018;218:75-87. doi:10.1016/j.ajog.2017.05.067
21. Sentilhes L, Kayem G, Chandraharan E, Palacios-Jaraquemada J, Jauniaux E. FIGO placenta accreta diagnosis and management expert consensus panel. FIGO consensus guidelines on placenta accreta spectrum disorders: conservative management. *Int J Gynaecol Obstet*. 2018;140:291-298. doi:10.1002/ijgo.12410
22. Shawky M, AbouBieh E, Masood A. Gray scale and doppler ultrasound in placenta accreta: optimization of ultrasound signs. *Egypt J Radiol Nucl Med*. 2016;47:1111-1115. doi:10.1016/j.ejrnm.2016.04.010
23. Jauniaux E, Hussein AM, Zosmer N, et al. A new methodologic approach for clinico-pathologic correlations in invasive placenta previa accreta. *Am J Obstet Gynecol*. 2020;222:379.e1-379.e11. doi:10.1016/j.ajog.2019.11.1246
24. Cramer SF, Heller DS. Placenta accreta and placenta increta: an approach to pathogenesis based on the trophoblastic differentiation pathway. *Pediatr Dev Pathol*. 2016;19:320-333. doi:10.2350/15-05-1641-OA.1
25. Jauniaux E, Moscoso G, Campbell S, Gibb D, Driver M, Nicolaides KH. Correlation of ultrasound and pathologic findings of placental anomalies in pregnancies with elevated maternal serum alpha-fetoprotein. *Eur J Obstet Gynecol Reprod Biol*. 1990;37:219-230. doi:10.1016/0028-2243(90)90028-y
26. Hamada S, Hasegawa J, Nakamura M, et al. Ultrasonographic findings of placenta lacunae and a lack of a clear zone in cases with placenta previa and normal placenta. *Prenat Diagn*. 2011;31:1062-1065. doi:10.1002/pd.2833
27. Aurioles-Garibay A, Hernandez-Andrade E, Romero R, et al. Prenatal diagnosis of a placental infarction hematoma associated with fetal growth restriction, preeclampsia and fetal death: clinicopathological correlation. *Fetal Diagn Ther*. 2014;36:154-161. doi:10.1159/000357841
28. Jha P, Rabban J, Chen L-M, et al. Placenta accreta spectrum: value of placental bulge as a sign of myometrial invasion on MR imaging. *Abdom Radiol (NY)*. 2019;44:2572-2581. doi:10.1007/s00261-019-02008-0
29. Jauniaux E, Zosmer N, Subramanian D, Shaikh H, Burton GJ. Ultrasound-histopathologic features of the utero-placental

interface in placenta accreta spectrum. *Placenta*. 2020;97:58-64. doi:10.1016/j.placenta.2020.05.011
30. Jauniaux E, Collins SL, Jurkovic D, Burton GJ. Accreta placentation: a systematic review of prenatal ultrasound imaging and grading of villous invasiveness. *Am J Obstet Gynecol*. 2016;215:712-721. doi:10.1016/j.ajog.2016.07.044
31. Al-Serehi A, Mhoyan A, Brown M, Benirschke K, Hull A, Pretorius DH. Placenta accreta: an association with fibroids and Asherman syndrome. *J Ultrasound Med*. 2008;27:1623-1628. doi:10.7863/jum.2008.27.11.1623
32. Chikazawa K, Imai K, Liangcheng W, et al. Detection of Asherman's syndrome after conservative management of placenta accreta: a case report. *J Med Case Rep*. 2018;12:344. doi:10.1186/s13256-018-1869-7
33. Engelbrechtsen L, Langhoff-Roos J, Kjer JJ, Istre O. Placenta accreta: adherent placenta due to Asherman syndrome. *Clin Case Rep*. 2015;3:175-178. doi:10.1002/ccr3.194
34. Swift BE, Shah PS, Farine D. Sonographic lower uterine segment thickness after prior cesarean section to predict uterine rupture: a systematic review and meta-analysis. *Acta Obstet Gynecol Scand*. 2019;98:830-841. doi:10.1111/aogs.13585
35. Nieto-Calvache AJ, Nieto-Calvache AS, Aguilera LR. Telemedicine as a strategy to facilitate placenta accreta spectrum treatment. *J Matern Fetal Neonatal Med*. 2021;0:1-2. doi:10.1080/14767058.2021.1971646
36. Lyall F. Mechanisms regulating cytotrophoblast invasion in normal pregnancy and pre-eclampsia. *Aust N Z J Obstet Gynaecol*. 2006;46:266-273. doi:10.1111/j.1479-828X.2006.00589.x
37. Calì G, Timor-Trisch IE, Palacios-Jaraquemada J, et al. Changes in ultrasonography indicators of abnormally invasive placenta during pregnancy. *Int J Gynecol Obstet*. 2018;140:319-325. doi:10.1002/ijgo.12413
38. Gorczyca ME, Springer S, Pateisky P, Ott J, Ulm B, Chalubinski K. Comparison of ultrasound descriptors of abnormally invasive placenta (AIP) over the course of the second and third trimester—is an increase verifiable? *J Clin Med*. 2021;10:4960. doi:10.3390/jcm10214960

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