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Review

Placenta accreta spectrum: imaging and diagnosis

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Key content

- Antenatal imaging is a screening tool integral to ensuring women are cared for in the most appropriate setting.
- The pretest probability of PAS should be considered before any imaging is performed.
- PAS is an iatrogenic disease. Any process which disrupts the endometrium increases the risk. Caesarean birth is the most common cause.
- Specialist placental assessment in asymptomatic women should be offered between 24 and 28 weeks.
- Ultrasound and MRI have similar diagnostic value, and MRI should only be used as an adjunct following ultrasound assessment by a specialist in placental imaging.

Learning objectives

- To understand the pathophysiology of PAS.
- To understand the distinction between screening for and diagnosis of PAS.
- To increase confidence in the interpretation of sonographic features of PAS.
- To provide a rationale for PAS care being provided in centres of excellence.

Ethical issues

- If women increasingly prefer caesarean section over vaginal birth, it is likely that this trend will result in increasing numbers of complex caesarean deliveries and PAS cases.
- Many studies have shown decreased morbidity and mortality if PAS cases are managed by an experienced multi-disciplinary team (MDT) in a PAS centre of excellence; therefore, accurate screening and timely referral are vital to improve patient care for women with risk factors.

Keywords: antenatal care / fetal medicine / pathology / perinatal diagnosis-ultrasound

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Introduction

Placenta accreta spectrum (PAS) disorder describes failure of placental separation at birth, with forcible removal risking life-threatening haemorrhage. It is a spectrum disorder ranging from abnormal adherence to deeply invasive placental tissue.[1] In 2012, the UK incidence was reported as 1.7/10 000 maternities.[2] As the incidence of PAS correlates with the rate of caesarean delivery, this has significantly increased.[3] It has been predicted that a rise in caesarean rate in the United States from 33% to 56% will be associated with 4504 additional cases of PAS and 130 maternal deaths a year.[4]

Antenatal detection and expert multi-disciplinary team (MDT) management reduce the incidence of complications including ureteric injury, blood transfusion and surgery within 7 days for bleeding complications.[5,6] Caesarean scar pregnancy is likely to be a precursor of PAS[7]; this review, however, focuses on PAS detection later in pregnancy. Particular attention is given to the sonographic signs and their role in optimising the care of women.[8] We also highlight challenges around clinicopathological and histopathological diagnosis of PAS peri- and post-natally.

Normal placental anatomy

The main blood supply to the uterus are the uterine arteries which give rise to the arcuate arteries and radial arteries. At the level of the junctional zone, the radial arteries branch laterally to form the basal arteries that supply the myometrium and basalis layer of the endometrium. These continue as spiral arteries which supply the endometrium. During pregnancy, the spiral arteries are remodelled to become a low-resistance network of dilated arteries.[9] The placental bed circulation is 'buffered' by arteriovenous

anastomoses in the myometrium which enables the blood to be shunted back to the uterine veins at third stage when the spiral arteries are closed by myometrial contraction.[10] It is vital that these placental AV anastomoses are considered when any embolisation is employed as material can pass through them causing iatrogenic pulmonary emboli.[11]

Placentation is inherently invasive and regulated by the decidua, as evidenced by tubal ectopic pregnancies.[12,13] After implantation, the trophoblast stem cells differentiate into cytotrophoblast and syncytiotrophoblast which form the outer layers of the chorionic villi. The cytotrophoblastic cells at the tips of these villi differentiate further to become extravillous trophoblast (EVT) which invades the decidual stroma and provokes remodelling of the uterine vessels.[14]

The EVT migrates through the decidua basalis, facilitated by the secretion of matrix metalloproteinases. The confluence of freely communicating trophoblastic spaces, lined by the syncitiotrophoblast, forms the intervillous space while condensed remains of the stratum compactum (SC) form the basal plate. Placental invasion continues through the upper third of the myometrium, as far as the junctional zone. Here, the smooth muscle cells of the media of the spiral arteries are replaced by amorphous fibrinoid material and the endothelium is replaced by endothelial EVT subtypes[14] so that there is 5- to 10-fold dilation at the vessel mouth.[9] Between the ends of the anchoring villi and the uterine decidual cells, a thin continuous fibrinoid layer called Nitabuch's membrane is laid down.[15]

Pathophysiology of PAS

PAS is an iatrogenic disease[16]; any process that disrupts the endometrium increases the risk. Endometrial ablation and curettage, including surgical termination of pregnancy, probably mostly cause abnormal adherence (accreta: FIGO Grade 1), whereas surgery which breaches the cavity is more likely to be associated with abnormal invasion (increta or percreta: FIGO Grades 2 and 3). Other causes of PAS have been suggested including uterine pathology such as bicornuate uterus, adenomyosis, submucous fibroids and myotonic dystrophy, but there is limited evidence to support the concept of microscopic defects in the endometrium[13] and these cases may be due to undisclosed surgery or incorrect diagnosis. Advanced maternal age,[2] assisted reproduction (especially with cryopreserved embryos),[3,17] Asherman's syndrome[16] and uterine artery embolisation[18] have also been linked to PAS.

Historically PAS was theorised to be a defect of trophoblast biology which resulted in excessive invasion of the myometrium. Unlike the trophoblastic defects identified in placental insufficiency and hydatidiform molar pregnancies, there has been no conclusive evidence to suggest this is the case.[13] The most widely accepted hypothesis is that a defect at the endometrial–myometrial interface leads to failure of normal decidualisation and abnormally deep penetration of EVT and anchoring villi.[13]

Surgery, particularly caesarean birth, is the most common cause of a uterine scar. Large and deep scars have been associated with an absence of reepithelialisation[19] and impaired local circulation in the affected area.[20] Without a normal epithelium, it is not possible for decidualisation to occur, so its regulatory influence is lost and deeper EVT penetration occurs.

The 'hypervascularity' observed on imaging PAS cases mostly results from abnormal (neo-) vascularity that develops in/on the serosa[13] and can be clearly seen during delivery. Remodelling of the deeper radial and arcuate arteries[21] combined with a lack of sufficient myometrium to contract providing the 'living ligature' results in the torrential haemorrhage seen when forcible placental removal is attempted.[22]

Numerous cell expression changes have also been observed in the trophoblast, but are more likely secondary to the altered myometrial environment.[13] This may be due to alterations in the uteroplacental oxygen gradient impacting cytotrophoblast differentiation.[9,23] As the enzymes that break down the extracellular matrix between decidual cells, also break down scar tissue, both trophoblastic invasion beyond the junctional zone and secondary uterine scar dehiscence (UD) can occur.[13]

Nitabuch's membrane facilitates placental separation when the myometrium contracts after the third stage. The absence of this layer has been noted in PAS; however, there may also be excessive fibrinoid deposition, which has been hypothesised as the cause of the abnormal attachment.[24]

While the underlying pathogenesis of PAS remains unclear, it must be remembered that EVT invasion is complete by around 22-week gestation.[25] The placenta then continues to mature and increase in size until about 32–34 weeks.[25] This maturation may make imaging signs more obvious, but the idea that early delivery can prevent an FIGO Grade 2 (increta) from becoming a Grade 3 (percreta) is incorrect. Also, there are no rapidly dividing cells in the syncitiotrophoblast of a mature placenta, so the use of methotrexate for treatment of retained placental tissue in the third trimester is fundamentally flawed. There is no evidence to support the use of methotrexate for PAS. Methotrexate can induce potentially life-threatening pancytopenia and nephrotoxicity, and one case series reported a maternal mortality directly attributed to methotrexate toxicity.[26]

Diagnosis and classification of PAS

Irving and Hertig originally described placenta accreta as an abnormal adherence of the placenta, in whole or in part, to the myometrium, with '*complete or partial absence of the*

decidua basalis'.[27] The grading of placental invasion by pathologists in the 1960s built on this description and still underpins the classification used today. Luke et al. used placenta accreta as an umbrella term for all abnormally adherent/invasive placentae but believed there was a need for further classification according to the depth of villous invasion.[28] They defined placenta creta as when villi are in contact with, but do not penetrate, the myometrium; placenta increta when they penetrate into the myometrium; and placenta percreta when full thickness invasion is noted.[28] It is the latter two categories, in particular, that can result in significant complications at birth depending on the extent of neovascularisation and involvement of surrounding pelvic organs.

Over the years, numerous terms have been used to describe this condition including morbidly adherent placenta, abnormally adherent or invasive placenta and PAS. The changing terminology and a lack of uniformity in pathological and clinical diagnosis have significantly contributed to confusion surrounding diagnosis and management[29,30] such as a surprisingly high regional incidence of 3.4%.[31] PAS should now be used as the terminology of choice to encompass the full spectrum of abnormality.[1]

The lack of accurate estimation of prevalence has contributed towards challenges in identifying diagnostic signs that are specific for the condition, monitoring outcomes of different management strategies and performing reviews and meta-analyses. To standardise the reporting of PAS, an expert panel developed the FIGO classification for the clinical diagnosis of placenta accreta spectrum.[32] They acknowledged that while histopathology by experienced perinatal pathologists is the gold standard diagnosis, this is not universally possible, such as in cases of an adherent rather than invasive placenta and those where the placenta is left in situ. Furthermore, retrospective histopathological grading has minimal clinical relevance as it does not provide guidance for intraoperative management.[33] Thus, a clinical classification for use at delivery was also developed.

Table 1 shows the clinical criteria recommended for universal use by FIGO.[32] Figure 1 shows some of the clinical features of placenta increta and Figure 2 a case of UD. It must be noted that findings may be non-uniform across the placental bed and that all forms of PAS, as well as UD, may be found in the same specimen.[34–36]

One proposed method for surgeons to rule out PAS is if the placenta can be fully detached digitally during delivery or from the fresh hysterectomy specimen.[33,37] In practice, the distinction between a difficult manual removal and an FIGO Grade 1 abnormally adherent placenta can be difficult. Manual removal of the placenta with an accompanying PPH is often labelled PAS, but it is important to recognise that the large blood loss may be due to other factors such as a delay in transfer to theatre with the bleeding occurring before the placenta was removed. To be diagnosed as FIGO Grade 1, the bleeding must come from the placental bed after 'forcible' removal of the adherent tissue and not stop without significant mechanical or surgical intervention such as oversewing or excising the bleeding.

Diagnostic controversies

In more recent years, the validity of the FIGO criteria, particularly for placenta percreta, have been called in to question.[35,36,38] It may be that many cases of UD and adhesions between the bladder and uterine serosae are misinterpreted as PAS, resulting in overly aggressive management.[35,36,39] Experienced clinicians often disagree about surgical findings as shown by one study in which 11 cases of 36 with UD were reported as placenta percreta.[36] Certainly, the incidence of percreta is likely rarer than first thought, with several studies reporting no cases at all despite initial ultrasound predictions.[35,36]

Clinicopathological diagnoses often grade PAS as more severe than the corresponding histopathological diagnosis.[34,36] The Luke cohort contained no cases of percreta[28] and the lack of cases where transmural villous invasion is seen into, and beyond, the uterine serosa has led some to question whether it is ever present.[35–39] The historical histopathological descriptors of PAS have also been challenged due to a lack of detailed confirmation of diagnosis in many series, possibly due to distortion of specimens at surgery[36,38] and loss of vascular changes prior to sampling.[33] The addition of perinatal pathology has been proposed to improve diagnostic accuracy,[33] but if an unnecessary hysterectomy has already been performed, this is of little use to the woman. Accurate intrapartum diagnosis with appropriate surgical management must be employed to prevent unnecessarily aggressive management of antenatally suspected PAS cases. The European Working Group on Abnormally Invasive Placenta (EW-AIP, now the International Society for PAS or IS-PAS) suggested a stepwise strategy to improve intrapartum diagnosis of PAS.[40] More recently, several articles have been published to aid in clinical differentiation between UD and PAS both on antenatal imaging[41] and at laparotomy.[42] Most experts agree that very large areas of UD with placenta praevia can still carry a high risk of morbidity.[39,42,43] However, the number of unnecessary hysterectomies from failure to appropriately diagnose PAS at delivery appears to be rising.

Screening: antenatal imaging

It must be remembered that imaging is a screening tool to identify women at high risk of PAS and guide intrapartum planning, rather than truly a diagnostic tool. The first description of PAS with ultrasound was made in 1982 by

Table 1. FIGO clinicopathological criteria for diagnosis of PAS[32]

Grading	Clinical criteria
Grade 1—Abnormally adherent placenta (adherenta, [accreta] or creta)	
	At vaginal delivery • No separation with synthetic oxytocin or gentle cord traction • Attempts at manual removal result in heavy bleeding from placental site requiring mechanical or surgical procedures At laparotomy/caesarean • As above • No obvious distension over the placental bed (no 'bulge'), no neo vascularity and no placental tissue seen invading through placental surface
Grade 2—Abnormally invasive placenta (increta)	
	At laparotomy/caesarean • Abnormal macroscopic findings over placental bed: blueish/purple colouring, distension ('bulge') • Significant amounts of hypervascularity (dense tangled bed of vessels running parallel craniocaudially within the serosa) • No placental tissue seen invading through placental surface • 'Dimple sign' (gentle cord traction results in the uterus being pulled inwards without separation of the placenta)
Grade 3—Abnormally invasive placenta (percreta)	
3a: limited to the uterine serosa	At laparotomy/caesarean • Abnormal macroscopic findings on uterine serosal surface (as above) and placental tissue seen invading through the surface of the uterus • No invasion into any other organ—clear surgical plane identifiable between the bladder and uterus
3b: urinary bladder invasion	At laparotomy/caesarean • Placental villi are seen to be invading into the bladder but no other organs • Clear surgical plane cannot be identified between the bladder and uterus
3c: invasion of other pelvic tissue/organs	At laparotomy/caesarean • Placental villi are seen to be invading into the broad ligament, vaginal wall, pelvic sidewall or any other pelvic organ (± invasion of the bladder)

Of note the term uterus refers to both the uterine body and uterine cervix in this classification.

Tabsh et al.[44] Since then, imaging technology and interpretation of findings have developed significantly so that now, both 2D and 3D ultrasound, and MRI are used for prenatal assessment of risk of PAS.

The distinction between diagnosis and screening is more than pedantic; it impacts how women are counselled, how multidisciplinary teams plan management and can prevent inappropriate hysterectomy based purely on antenatal suspicion. It is also a crucial part of accurate reporting for research and audit. Unfortunately the lack of correlation between ultrasound findings and grading of PAS[45] and the heterogeneity in terminology and classification has limited the ability to compare studies and assess the diagnostic power of different imaging modalities and features.[8,38,45]

Pretest probability

The pretest probability of PAS must be considered when any imaging is performed. The higher the background risk, the greater a diagnostic test's accuracy tends to be. This is particularly important when screening for PAS because at least two ultrasound features of PAS have been found in 98% of low-risk pregnancies with normal placentation.[46] The features most frequently reported in normal placentas are lacunae, sub-placental hypervascularity and an irregular bladder wall.[46] While placental bulge, bridging vessels, a myometrium <1 mm thick and the loss of the retroplacental clear zone are rarely seen unless there is PAS or UD.

Most severe PAS cases occur in women with an anterior low-lying (<2 cm from the internal os) or praevia placenta, and a history of one or more caesarean deliveries. They may

Figure 1. FIGO Grade 2 (increta) at laparotomy. Prominent neovascularity seen over the serosa.

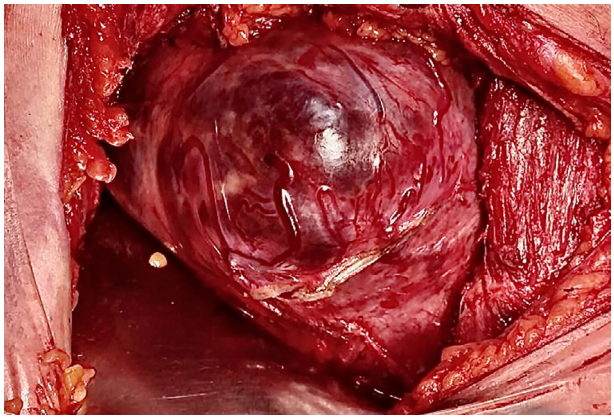

Figure 2. Uterine dehiscence at laparotomy. Some prominent vessels can be visualised under the serosa.

have already been diagnosed with a caesarean scar pregnancy. All such women should be screened by an expert in PAS imaging.[1] The risk of PAS in a woman with a placenta praevia and prior caesarean delivery is 3%, 11%, 40%, 61% and 67% for one, two, three, four and five or more caesarean births.[3] Good practice is also to refer all women with a history of previous PAS and those with ultrasound features raising suspicion of PAS for screening by an expert in PAS imaging.

Before scanning, a clear history should be taken to identify risk factors for PAS and to assess the woman's general health and current obstetric risks. It is also helpful to consider the dilatation at which previous caesareans were performed as a guide for where the previous scar is most likely to be. If the previous caesarean delivery occurred at ≤2 cm dilatation, the scar will usually be found in the uterine lower segment well above the internal os.[47] However, intrapartum caesarean deliveries at ≥8 cm result in the scar being close to the internal os if not on the cervix itself.[47] An understanding of where the scar is likely to be found can aid in assessment of whether the placenta may be implanted over it. If the centre of the placenta lies directly over the presumed site of the previous scar, the risk that the conceptus implanted within a niche resulting from the previous surgery is increased.

Ultrasound

Despite the rising incidence of PAS, it remains relatively rare; consequently, the majority of clinicians may have little experience scanning PAS cases. Centralising imaging for, and subsequent management of, PAS increases exposure to the full spectrum of the condition thereby optimising patient care. Ultrasound imaging is highly accurate when performed by a skilled operator.[1] Women with a high pretest probability of PAS or a high suspicion of PAS from ultrasound signs should therefore be assessed in a designated PAS centre with experience in diagnosing and managing the condition.[1]

Sonographic features of PAS can be separated into anomalies of the uterine contour and uteroplacental interface and those of the uteroplacental circulation. The former are associated with a lower specificity of PAS[35] and are a result of uterine remodelling following scarring of the lower segment.[38] The latter can be highly subjective to levels of operator experience and machine settings,[48] but confer a higher probability of intraoperative complications and PAS.[39] Using a combination of ultrasound signs improves the specificity for detection of PAS[49] and accurate differentiation between PAS and simple UD.[41]

Preparation

Machine. A transabdominal approach is usually first line and is often sufficient even in women with significant abdominal adiposity who can be asked to lift their pannus to facilitate probe placement just above the symphysis pubis. A convex 3–5 MHz ultrasound probe can be used although a higher frequency probe may improve the quality of the image especially for an anterior low-lying placenta where less penetration is needed. Transvaginal probes are high frequency but significantly narrow the field of view. However, if abdominal views are not sufficiently clear, such as due to abdominal adiposity or scar tissue, a transvaginal approach can provide excellent assessment of the relationship of the placenta to the cervix and surrounding vasculature.

More modern US machines offer compound imaging such as CRI, CrossXBeam and sonoCT to reduce artefact but these 'smooth' the image and may blur the uteroplacental interface.

This should be turned off when examining greyscale features of PAS.[48] When using colour flow mapping, the correct velocity is crucial to visualise low flow but not to generate aliasing artefact; a pulse repetitive frequency setting above 15 cm/s should be adequate.[48,50,51] For power Doppler, the sub-noise gain setting should be used which is unique to each patient. This is found by increasing the gain until bloom artefact appears then gradually reducing it until the artefact just disappears.[52]

Gestation. The primary screening for PAS occurs at the mid-pregnancy anomaly scan where women with a low-lying or previa placenta should be asked if they have a history of previous caesarean birth. If they do, this should prompt referral for further imaging. Women with increased probability of PAS and vaginal bleeding or an increased risk of preterm birth should be reviewed by a specialist as soon as practicable in case of emergency birth. Asymptomatic women can usually wait for more detailed assessment until after 24 weeks when trophoblast invasion is complete.[48] Ideally, specialist review should have occurred by 28 weeks to give ample time for MDT planning.

Bladder filling. The bladder must be adequately filled with a minimum of 200–300 mL to adequately assess several important signs.[48,50,53] The measurements to take are: the uterovesical fold to the base of the bladder (sagittal plane); the anterior bladder wall to the posterior bladder wall at the midpoint of the bladder (sagittal plane); and the distance from side to side through the middle of the bladder (coronal plane).[53] Most inbuilt ultrasound machine calculators will calculate this using an ellipsoid equation which gives a lower volume than that of a cuboid or cylindrical volume, but helps to ensure adequate filling. Women should be warned that they may feel uncomfortable and operators should be mindful of this when scanning.

Practicalities. If too much probe pressure is applied, the retroplacental clear zone can be falsely obliterated and the myometrium can appear additionally thinned. Furthermore, significant 'drop-out' artefact can occur when the probe is not perpendicular to the placental bed.[48]

Ultrasound features

The EW-AIP first standardised the ultrasound features of PAS in 2016.[54] Only a few additional signs have been added by consensus since, with the separation sign[55] being published too late for inclusion into the most recent consensus update.[56] Table 2 shows a list of the ultrasound features recommended for assessment, their descriptors and proposed pathophysiology. There are a number of technical issues and important features of these signs that are worth noting.

Loss of the clear zone (Figure 3) is very susceptible to too much probe pressure. The appearance of the clear zone also varies with advancing gestation, bladder filling (Figure 4) and placenta location within the uterus (it is difficult to assess with a posterior placenta).[13] Visualisation of this space is best using a high level of zoom with appropriate focus level and adequate dynamic contrast. If seen, it is a valuable marker for ruling out PAS in that area of the placental bed.

The separation sign can be employed to more clearly assess loss of the clear zone and is a valuable 'rule out' sign.[55] It should be looked for throughout the entire placental bed and cannot be performed with posterior placentation. It involves using the probe to compress the placenta against the myometrium. This pressure is then quickly released to see the appearance, or enhancement, of the clear zone as the myometrium and placenta 'bounce' back at different velocities due to their differing elasticities. Videos of this sign are available[55] and https://www.placentaaccretasspectrum.com/ultrasound-imaging-of-pas/the-separation-sign.

Abnormal placental lacunae (Figure 5) have been described as giving a 'moth-eaten',[13] or 'swiss-cheese' appearance but should not be confused with simple placental lakes. The distinction between physiological lakes and abnormal lacunae can be challenging despite them resulting from different physiological processes.[59]

- Lakes are a common feature of normal placentas. They are homogeneous hypoechogenic lesions found anywhere within the placenta and may be up to 5 cm or more in size, especially if marginal.[59]
- When lakes have peripheral echogenicity, they should be called echogenic cystic lesions. These are associated with intervillous thrombosis and placental insufficiency and are not due to invasive placentation.[59]
- The abnormal lacunae of PAS are numerous, large and irregular hypoechoic areas extending from the basal plate where associated loss of the clear zone and myometrial thinning is seen.[24]
- Turbulent blood flow may be seen in both lakes and lacunae.[59] In PAS lacunae, high velocity flow (>10 cm/s) is often seen with colour Doppler (Figure 5), whereas the low velocity flow of placental lakes may only be visible on greyscale and not seen with Doppler imaging.

Bladder wall interruption (Figure 5) is a frequently mis-interpreted sign as an irregular interface can result from bladder reflection during a previous caesarean delivery. True PAS bladder wall 'interruption' is usually seen as two parallel hyperechoic lines resembling an equals sign (=) crossing the myometrium–bladder interface. It may also appear to give the myometrium–bladder interface a 'scalloped' appearance. This sign is most likely an artefact produced by insonating the neovascularity lying between the anterior aspect of the uterus and the posterior bladder wall.[13] Identifying the presence of this abnormal vascularity is

Table 2. Descriptors of the ultrasound features of placenta accreta spectrum and their proposed pathophysiology

Ultrasound finding	Definition[54–56]	Proposed pathophysiology[13,38,55,57,58]
2D greyscale		
Loss of 'clear zone'	Loss or irregularity of the hypoechoic plane in the myometrium underneath the placental bed	Thinned decidual and myometrial layers causing loss of the hypoechoic signal due to remodelling of the uterine wall and lack of Nitabuch's layer
Abnormal placental lacunae	Presence of numerous large and irregular intraplacental hypoechoic spaces (Finberg Grade 3), often containing turbulent flow visible on greyscale imaging	Distortion of placental cotyledon due to chronically high-velocity maternal blood flow entering the intervillous space directly from a radial or arcuate artery
Bladder wall interruption	Loss or interruption of the bright bladder wall (hyperechoic band or 'line' between the uterine serosa and bladder lumen)	Artefact associated with the presence of neovascularity at the serosal surface
Myometrial thinning	Thinning of the myometrium overlying the placenta to <1 mm or undetectable	This occurs with a major scar defect, where the myometrium is thinner than normal or completely replaced by fibrous tissue. It becomes more pronounced with increasing gestation.
Placental bulge	Deviation of the uterine serosa away from the expected plane, caused by abnormal bulge of placental tissue into neighbouring organs, typically the bladder; the uterine serosa appears intact, but outline shape is distorted	Large placental tissue herniation through a weakened uterine wall following myometrial thinning or dehiscence
Focal exophytic mass	Placental tissue seen breaking through the uterine serosa and extending beyond it; most often seen inside the filled urinary bladder	Focal placental tissue herniating through a small defect of uterine wall; extremely rare
Separation sign	The appearance or enhancement of the retroperitoneal clear zone with probe pressure and then rapid release	Remodelling of the uterine wall removes the inherent difference in elasticity of the placenta and myometrium
Colour Doppler imaging		
Uterovesical hypervascularity	Striking amount of colour Doppler signal seen between the myometrium and posterior wall of the bladder; this sign probably indicates numerous, closely packed, tortuous vessels in that region (demonstrating multidirectional flow and aliasing artefact)	Excessive dilatation of deep uterine circulation (radial and arcuate) probably from abnormal proximity to extra-villous trophoblast
Subplacental hypervascularity	Striking amount of colour Doppler signal seen in the placental bed; this sign probably indicates numerous, closely packed, tortuous vessels in that region (demonstrating multidirectional flow and aliasing artefact)	Excessive dilatation of deep uterine circulation (radial and arcuate) probably from abnormal proximity to extra-villous trophoblast
Bridging vessels	Vessels appearing to extend from the placenta, across the myometrium and beyond the serosa into the bladder or other organs; often running perpendicular to the myometrium	Artefact arising from neovascularity within the peritoneum seen in 2D cross section. Neovascularity may be due to increased growth of originally smaller vessels which are often quite immature
Placental lacunae feeding vessels	Vessels with high velocity blood flow leading from the myometrium into placental lacunae, causing turbulence upon entry	High velocity blood flow arising from radial and arcuate arteries.
Intracervical lakes	Tortuous hypervascularised anechoic spaces within the cervix	Excessive dilatation of cervical vessels resulting from development of part of the definitive placenta in a scar defect

Table 2. (Continued)

Ultrasound finding	Definition[54–56]	Proposed pathophysiology[13,38,55,57,58]
3D US ± power Doppler ± volume rendering		
Intraplacental hypervascularity	Complex, irregular arrangement of numerous placental vessels, exhibiting tortuous courses and varying calibres	Excessive dilatation of deep uterine circulation (radial and arcuate) resulting from development of part of the definitive placenta in a scar defect
Obliteration of the retroplacental space 'tramline appearance'	'Partial obliteration' is defined as loss of some or part of the uterine-bladder interface; 'full obliteration' as complete obliteration of the uterine–bladder interface	Disruption of uterine/bladder serosa and the retroplacental clear zone with fusion of the placenta and myometrium

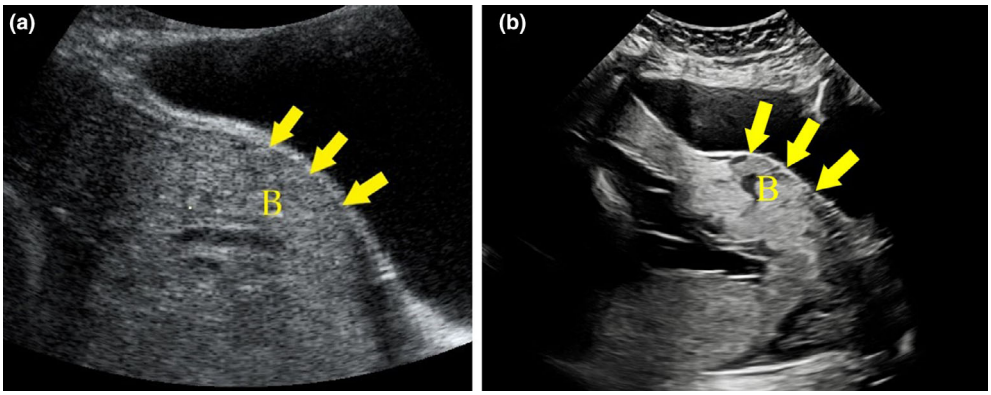

Figure 3. (a) Placenta praevia with uterine dehiscence. Placental bulge (B) with a well-filled bladder, vanishingly thin myometrium and loss of clear zone (arrows). (b) FIGO Grade 3b (percreta). Placental bulge, vanishingly thin myometrium, loss of clear zone and placental lacunae.

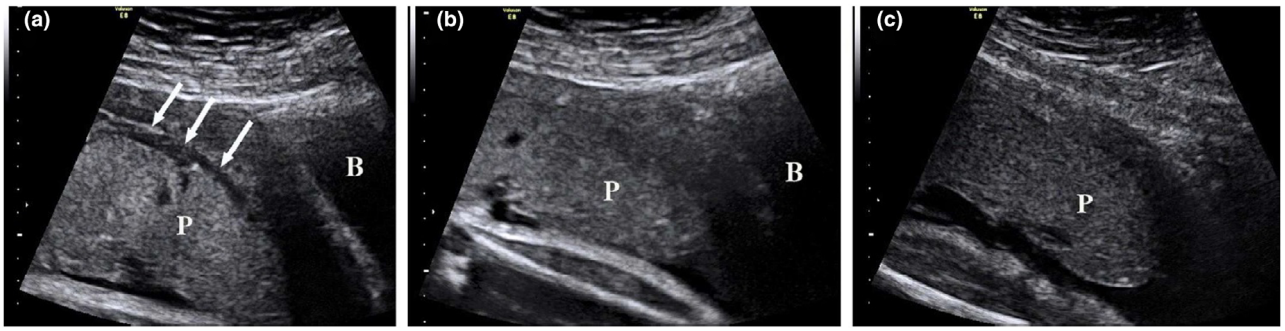

Figure 4. Demonstration of changes to the clear zone (arrows) in the same low-lying placenta at 32-week gestation between a (well-filled bladder [B] and minimal probe pressure), b (increased probe pressure) and C (empty bladder and minimal probe pressure).[13]

important for differentiating between PAS and simple UD,[41] as well as for subsequent management as the vessels may not become apparent until after the surgeon has started the bladder reflection.

Myometrial thinning (Figure 3) may be a sign of UD or PAS. Thinning naturally occurs with advancing GA but is most pronounced from 32 to 34 weeks when the lower segment is stretched by a combination of fetal presentation and Braxton Hicks uterine contractions.[13] Assessment of myometrial thinning provides a useful gauge as to how successful tamponade and uterotonics might be if manual removal is attempted and prompts counselling about local

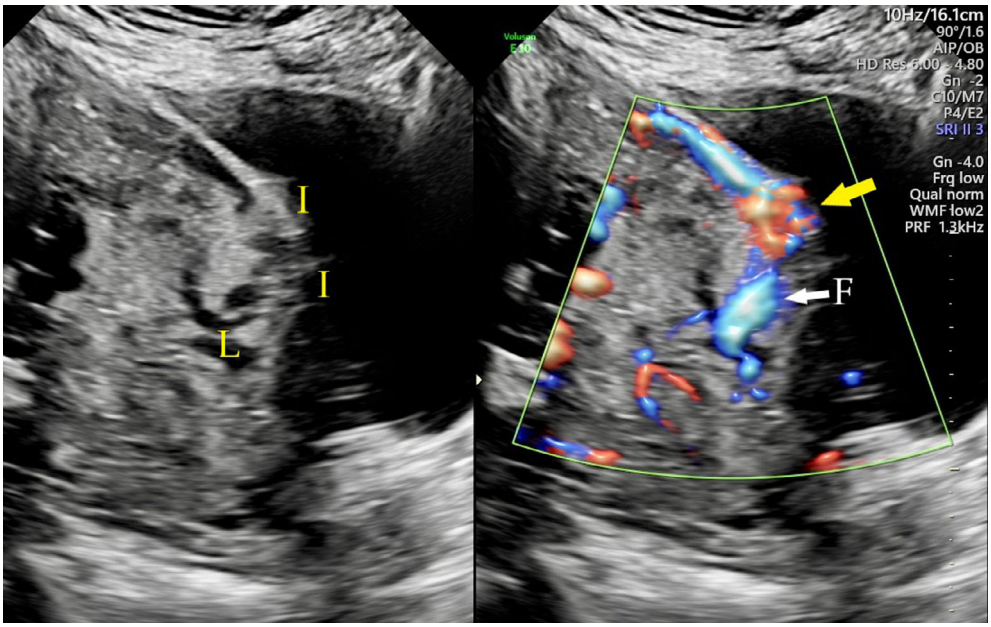

Figure 5. Bladder wall interruption 'equals sign' (I), bridging vessels when colour Doppler applied (yellow arrow), lacunae (L) and feeder vessels (thin arrow & F) in a case of FIGO Grade 3a (percreta). A fuller bladder would make the placental bulge and loss of clear zone more pronounced.

excision. The degree and location of this should be reported.[60] This sign may be influenced by probe pressure and bladder filling.

Placental bulge (Figure 3) is thought to result from loss of structural integrity of the myometrium due to deep EVT invasion[13] and has been described at laparotomy as the 'snowman' sign.[61] A large UD can also result in a placental bulge due to absent myometrium over the defect, but it should not have any other associated ultrasound features of PAS.

An assessment of the proximity of bulge to other structures is crucial for surgical planning and can raise suspicion of parametrial involvement, which may warrant a second opinion or MRI assessment. A bulge into the broad ligament or close to structures such as the ureters may prompt consideration of other pre-operative strategies such as ureteric stenting.

Focal exophytic mass refers to placental tissue seen extending beyond the serosa, usually protruding into the bladder. It is exceptionally rare, and many experts question its validity for the diagnosis of PAS.

Uterovesical and subplacental hypervascularity (Figure 6) both result from excessive dilatation of the radial and arcuate arteries vessels deep within the myometrium and excessive neovascularisation of the serosa.[13] The settings used will need to be adjusted according to the tissue attenuation of the woman. The settings appropriate for interrogation of the uterine arteries should be high enough to avoid aliasing and being too low, which gives false positives. What constitutes

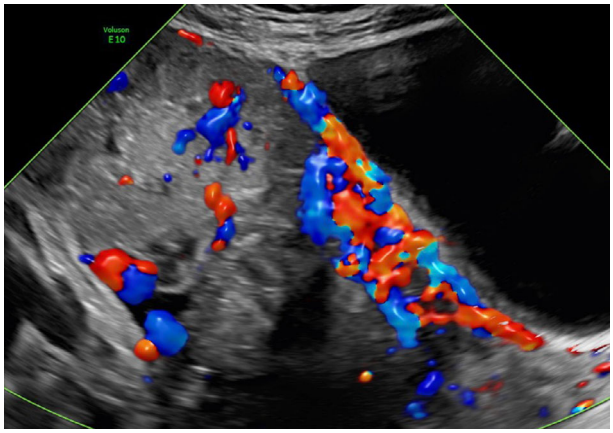

Figure 6. Hypervascularity at the uterovesical interface in a case of FIGO Grade 2 (increta).

hypervascularity is, however, entirely subjective, with this sign being frequently reported in normal placentas,[46] as such it is a distinctly unreliable marker of PAS. However, in cases of PAS, increased vascularity in the cervix and upper third of the vagina indicate possible varicosities that may bleed heavily at colpotomy. Awareness of this can facilitate management plans to optimise the stability of the woman, such as returning all cell salvaged blood before embarking on colpotomy.

Bridging vessels (Figure 5) seem to be a more useful predictive feature than hypervascularity as they only occur

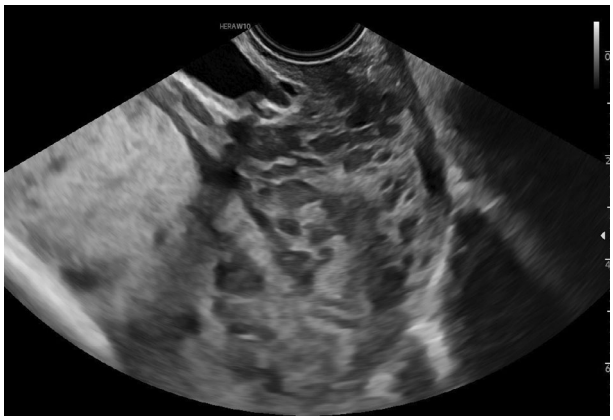

Figure 7. Intracervical lakes in a case of FIGO Grade 3c (percreta).

when there is a significant amount of neovascularity at the serosal surface.[51] The presence of bridging vessels should alert the surgeon to the presence of large, friable vessels, which increase the risk of bleeding when reflecting the bladder. The lack of neovascularity on the serosal surface is an important feature for intrapartum differentiation between PAS and UD (Figures 1 and 2).

Intracervical lakes (Figure 7), not to be confused with the uterine or cervical arteries, are tortuous anechoic spaces within the cervical tissue itself, which appeared to be hypervascular on colour Doppler. They are indicative of deep EVT invasion and have been linked to increased blood loss and requirement for hysterectomy.[62]

3D volume rendering has been able to detect the retroplacental clear zone (Figure 8) in cases of UD when 2D ultrasound alone could not.[34] The tramline disruption sign and distortion of the bladder wall, and obliterated vesicouterine space are other signs that can be identified in PAS[34,63] (Figure 8B). The potential to discriminate between UD with a non-adherent placenta and PAS may help avoid unnecessary hysterectomy and anxiety for both clinicians and women, but the utility of this newer technology has yet to be confirmed.[56]

Differentiating PAS and uterine dehiscence (UD). As with differentiating between PAS and UD at surgery, there are controversies regarding the antenatal features seen with ultrasound. An additional challenge is the increasingly presented theory that PAS and UD coexist in the same placental bed, and hence, features of both can be visualised.[34–36] With many shared ultrasound signs, risk scoring systems, such as the Placenta Accreta Index, over-predict cases of UD as high risk of abnormal invasion.[41] There are some ultrasound signs which can help to make the distinction, which is important because confirmation bias can result in more radical surgery than is necessary, which carries greater risk of morbidity.

Table 3 summarises the differences between PAS and UD. Myometrial thinning and a placental bulge are common features of both PAS and UD, but a bulge may only be seen in very large areas of UD.[39] A very thin or imperceptible myometrium will mean the clear zone cannot be visualised with 2D US either. In UD, the placenta is also more likely to have a more homogenous appearance, but the key distinction can be found in the vasculature.[41] Where placenta lacunae are suspected, a regular shape is more likely in UD while numerous, large, irregular lesions connecting with the basal plate are more associated with PAS. A hyperechoic ring

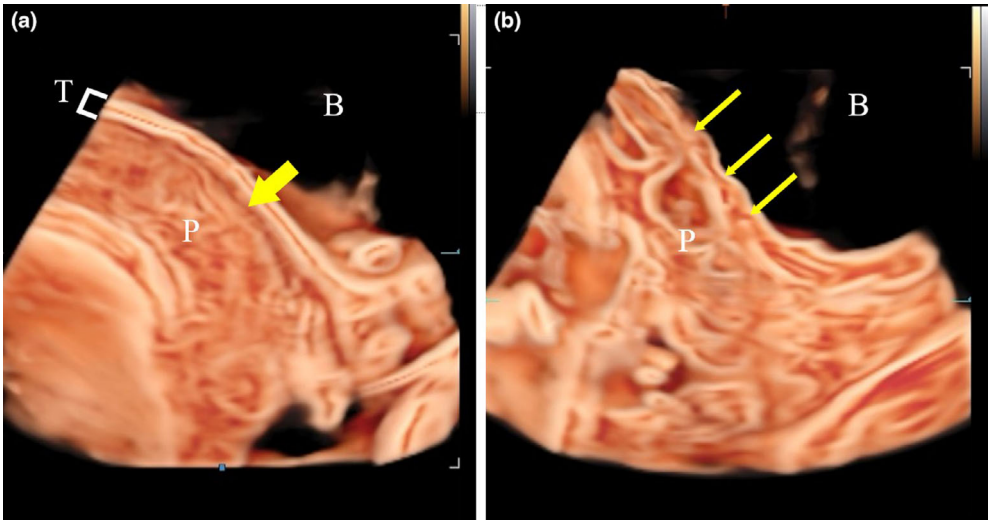

Figure 8. (a) Retroplacental clear zone (yellow arrow) and tramline sign visualised in placenta praevia. (b) Tramline disruption sign distortion of the bladder wall and loss of retroplacental clear zone in a case of FIGO 3B PAS. Placenta (P) and bladder (B).

Table 3. Distinguishing features of placenta accreta spectrum and uterine dehiscence

	PAS	UD
Myometrial thinning	√	√
Loss of clear zone 2D	√	√
Separation sign	Likely diffusely absent	Only absent where myometrium is vanishingly thin
Placental bulge	√	√
Lakes/lacunae	Numerous, irregular, large and connecting with placental basal plate	Are more regular. May have a hypoechogenic rim
Lacunae blood flow	High flow with colour Doppler (>10 cm/s)	Low, or no flow, seen with colour Doppler
Bladder wall interruption	√	x
Bridging vessels	√	x
Focal exophytic mass	√(extremely rare)	x
Uterovesical and subplacental vascularity	√	x
Intracervical lakes	√	x
3D volume rendering	Loss or retroperitoneal clear zone Tramline disruption sign Distortion of the bladder wall Obliterated vesicouterine space	Retroplacental clear zone visible *More research needed*

increases the probability that they are echogenic cystic lesions not related to PAS, and colour Doppler should be used to identify the presence of high velocity flow. An absence of high velocity flow (>10 cm/s) with colour Doppler increases the probability that the lesions are lakes and UD might be more likely than PAS.[41] Experienced fetal medicine specialists have often had little training in assessing normal vasculature of the placental bed and hypervascularity is easy to overdiagnose. True hypervascularity of the placental bed, upper vagina and cervix, bridging vessels and the corresponding bladder interruption are features of increased vasculature due to PAS.[39,41]

We support proposals to treat all cases suggestive of PAS as a general problem that should be managed according to surgical findings rather than with a fixed antenatal plan. Good bladder reflection off the uterus and identifying sufficient healthy myometrium are important when considering less radical surgical options.[42] Care must be taken when interpreting histopathology results from UD as villi may be seen adjacent to the serosal surface due to the lack of myometrium where the uterus has dehisced.

Writing the report. Experts now recommend an emphasis on describing the anatomy over merely assigning presumed pathologic labels.[60] The real value of any imaging report is when it enables the surgical team to accurately predict what they will encounter during surgery. A proposed reporting pro forma has been suggested by expert consensus[64] which ensures consideration of the pretest probability and important ultrasound features of PAS but used on its own does not provide a topographical description that would be of benefit to a surgical team. The fetal growth and presentation should be reported in addition to the placental and uterine features.

The role of magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) of the placenta is performed using ultra-fast techniques with a semi-filled bladder. No contrast is required,[65] slices should be no thinner than 5 mm to avoid artefact and false positives and the sequences should be aligned to the uterine axis.[60] The main features of PAS on MRI are an abnormal uterine bulge, dark intraplacental bands on T2-weighted imaging, tenting of the bladder, heterogenous signal intensity within the placenta, disorganised vasculature of the placenta and focal interruption of the myometrium.[1,65] Several other features have also been included in a list of unified descriptors by EW-AIP.[66] As with ultrasound, MRI features of PAS are not unique to PAS. Both intraplacental bands and placental heterogeneity have been demonstrated in normal placentas[67] and a uterine bulge will likely be seen in UD[38] and may be noted with an extremely thin myometrium.[68]

Some experts think that MRI is more useful than ultrasound for topographical characterisation of placenta invasion into the parametrium and other organs, to better facilitate surgical mapping, whilst others believe that the dynamic nature of ultrasound is more beneficial for this.[60] MRI likely does have advantages over ultrasound for assessment of a posterior placenta,[1,60] but this cohort is relatively small because PAS is most commonly identified in women with both an anterior low-lying placenta and previous caesarean birth. It may also be beneficial in women with a significantly raised BMI in whom ultrasound may be difficult. Another advantage is that images can be re-evaluated anywhere, at any time, and so, although expert

evaluation of MRI is not widely available, opinion can be sought from other places. Significant disadvantages are that MRI remains a much more expensive imaging tool and is not universally accessible, especially in low- and middle-income countries.

In expert hands, both ultrasound and MRI have similar diagnostic value.[1] There is no evidence that MRI is superior to ultrasound and its true diagnostic accuracy is difficult to ascertain because there is usually high pretest probability from prior ultrasound.[65] Concern about its use has also been expressed as in some cases where MRI has been used, a change of diagnosis has occurred incorrectly that altered management potentially causing physical and psychological harm to mothers and their babies.[69]

Current guidance recommends that MRI should not be a routine adjunct to ultrasound, but rather remain a complementary modality after ultrasound has been performed by an expert in PAS if ambiguity remains about the diagnosis or extent of invasion.[1] Many believe that MRI requires still greater standardisation and optimisation, and proof of reproducibility and benefit in the diagnosis of PAS.[60]

Conclusion

Optimising birth for women with PAS is a case of ensuring the right team are present, in the right place, at the right time. Emergency deliveries cannot be avoided, but high-quality antenatal care and MDT planning will improve the experience of both the woman and the team caring for her. Care in designated PAS centres also reduces the incidence and gravity of emergencies.[6,70]

UK national guidance states that women with suspected PAS should be cared for by an MDT in a specialist centre with expertise in diagnosing and managing invasive placentation.[1] Despite this, many women with PAS are still managed in centres where they encounter only one or fewer cases every year.[71] There is no place for improvised care and ad hoc management outside of true emergencies,[60] which may mean closing the abdomen before the hysterotomy and transferring a woman with clinically suspected PAS to another hospital.

The global rise in the rates of caesarean birth and associated incidence of PAS highlights a need for developing training programmes to improve detection of women likely to have a complex birth.[72] We join calls for a network of regional designated PAS centres to ensure women are scanned by experts who regularly image PAS, and who also have the experience in complex pelvic surgery needed to reduce maternal morbidity and mortality.[1,73] These centres would also create the optimal environment for trainees to develop specialist skills in diagnosis and management of PAS, which are currently lacking from most training programmes.

Useful websites

- https://www.placentaaccretasspectrum.com/
- https://is-pas.org/
- https://www.isuog.org/resource/uog-videoclip-placental-lakes-vs-lacunae-spot-the-differences.html

Author contributions

AS and SLC both conceptualised this review. AS drafted the article with help from AC and SLC, who both assisted with revisions. AS, AC and SLC all approved the final version and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy and integrity of any part of the work are appropriately investigated and resolved.

Conflict of interest statement

AS and AC have no interests to declare. SLC co-authored the 2018 green-top guideline on diagnosis and management of placenta praevia and placenta accreta[1] and the FIGO consensus guidelines on Prenatal diagnosis and screening for placenta accreta spectrum disorder.[50] She is also the current Chair of the International Society for Placenta Accreta Spectrum.

Ethics approval

Written permission was granted by women whose placental images have been included in this review.

Supporting Information

Additional supporting information may be found in the online version of this article at http://wileyonlinelibrary.com/journal/tog

Data S1.

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