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Placenta accreta spectrum disorder: An updated literature review

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Literature Review

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ABSTRACT

Placenta accreta spectrum disorder (PASD) refers to a range of pathological placental adhesions to the uterine wall, previously classified into three subtypes: placenta accreta, placenta increta, and placenta percreta, based on the invasiveness of the villous tissue. This article provides an updated review of the literature on PASD with new insights into the etiopathology of PASD. Recent evidence suggests that extravillous trophoblasts are not overly invasive and that accreta placentation is more likely due to decidualisation failure resulting from blastocyst implantation within a caesarean scar defect (CSD). Previous caesarean delivery has been the most well-known risk factor of PASD, with the increased occurrence of PASD along with the increased number of previous caesarean deliveries. Antenatal identification of PASD is strongly recommended to improve outcomes before the onset of labour or bleeding, so that placental abruption can be avoided. Ultrasonography can identify PASD in the first trimester with good sensitivity and specificity. A standardised approach with a comprehensive multidisciplinary care team is required to manage PASD effectively. The Royal College of Obstetricians and Gynaecologists (RCOG), along with The American College of Obstetricians and Gynaecologists (ACOG), have published guidelines for the best clinical management of PASD. Future research should concentrate on gathering prospective data on the diagnosis and management of PASD in order to assess the association between prenatal imaging, clinical grading, and histology findings. This will lead to more accurate PASD screening, reliable diagnostic criteria, and alternatives to prenatal treatment.

Spektrum plasenta akreta (SPA) mengacu pada perlekatan patologis plasenta ke dinding rahim, yang sebelumnya dibagi menjadi tiga subtipe: plasenta akreta, plasenta inkreta, dan plasenta perkreta berdasarkan dalamnya invasi jaringan vili. Artikel ini menyajikan tinjauan literatur terbaru mengenai SPA, dengan ilmu baru tentang etiopatologi PASD. Temuan terbaru menunjukkan bahwa trofoblas ekstravili tidak memiliki sifat invasif berlebihan, dan plasentasi akreta lebih mungkin terjadi karena kegagalan desidualisasi akibat implantasi blastokista di dalam bekas luka sesar. Riwayat persalinan sesar merupakan faktor risiko SPA yang paling diketahui, dengan peningkatan kejadian SPA seiring dengan peningkatan jumlah persalinan sesar sebelumnya. Diagnosis antenatal SPA sangat dianjurkan untuk meningkatkan luaran sebelum persalinan atau onset perdarahan sehingga gangguan plasenta dapat dihindari. Ultrasonografi dapat mengidentifikasi SPA pada trimester pertama dengan sensitivitas dan spesifisitas yang baik. Pendekatan standar dengan tim perawatan multidisiplin yang komprehensif diperlukan untuk manajemen spektrum plasenta akreta yang efektif. Royal College of Obstetricians and Gynaecologists bersama dengan ACOG telah menerbitkan pedoman untuk manajemen klinis

terbaik SPA. Penelitian selanjutnya sebaiknya berfokus pada pengumpulan data prospektif pada diagnosis dan tatalaksana SPA untuk menilai hubungan antara pencitraan prenatal, penilaian klinis, dan temuan histologi. Dengan demikian, dapat dihasilkan metode skrining yang lebih akurat, kriteria diagnosis yang lebih dapat diandalkan, serta alternatif tatalaksana prenatal SPA.

INTRODUCTION

Placenta accreta spectrum disorder is characterised as an aberrant trophoblast adherence of part or all of the placentae into the uterine myometrium.[1] The term "placenta accreta" was initially defined in 1937 at the Boston Lying-In Hospital by obstetrician Frederick C. Irving and pathologist Arthur T. Herting.[2] Complete or partial decidua basalis loss was utilised as a histological criterion for diagnosing placenta accreta.[3] All Irving and Herting's cases are defined as "vera" or "adherent," indicating that the villi were not penetrating the myometrium's surface but only attached to it. They explored the likelihood of deeper villi penetration but found no cases with histologic evidence of invasion of placental tissue into the myometrium.[2] Previous theory describes that an invasive placenta can proliferate and invade local organs. Recent findings have suggested that the extravillous trophoblast (EVT) in PASD is not excessively invasive, and the accreta placentation is more likely because of decidualisation failure due to blastocyst implantation inside a CSD.[4] This review aims to provide an updated literature review of placenta accreta with new insights into its etiopathology.

This literature study is conducted using secondary research data from various national and international books and articles. These sources were accessed through prominent databases, including PubMed, Scopus, Cochrane, EBSCOhost, ProQuest, and Google Scholar. The author used the keywords placenta accreta spectrum, placenta accreta, epidemiology, risk factors, interpregnancy interval, caesarean section, aetiology, diagnosis, placenta accreta index score, and management, with boolean operators (AND, OR). This review used literature published in 2017-2023, which could be accessed in full text. However, several articles published before 2017 were still used if the article's content was important and no more recent articles provided the information within the appropriate timeframe. This literature review was synthesised using a narrative method by grouping similar extracted data according to the measured outcomes in order to address the study objectives.

Incidence

There is a rising trend in the incidence of PASD. Studies in 1970 and 1980 reported that the prevalence of PASD was 1 in 2,510 births to 1 in 4,017 births. Based on a 2016 study, the overall rate of PASD in the United States is 1 in 272 births. The mean incidence of PASD is 0.04% and then increases to more than 0.9%.[5] Study also reported a rise in placenta accreta incidence in Asia, including Indonesia, with a 2% increase in incidence causing about 6% of maternal bleeding.[6] Dr. Sutomo Hospital Surabaya, a referral centre for accreta cases in eastern Indonesia, reported a significant increase in PASD with a total of 163 cases from January 2014 to December 2018.[7] Based on strong epidemiologic data, the rise in the prevalence of PASD disorders has been associated with increased caesarean delivery rates.[8] Caesarean delivery rates have increased from 10% to 30% worldwide in the past 40 years, while most middle-income and high-income countries have seen a 10-fold increase in the incidence of PASD. It should be recognised that changes in the incidence of PASD caused by high caesarean delivery rates can take up to ten years.[9] Over 90% of women with at least one prior caesarean section will develop PASD.[10,11]

Risk factors

Several risk factors are associated with PASD, with previous caesarean delivery being the most well-known risk factor.[8] The rate of PASD increased from 0.3% in women with one history of caesarean section to 6.74% in multiple caesarean sections.[12] A study in 2000-2011 found a 30.8% increase in PASD rate in women with multiple caesarean sections, with a 2.13 times higher risk compared to single caesarean sections.[13] Incisions in caesarean sections can be transverse or vertical, with a low transverse incision being preferred due to reduced blood loss, better reparability, and less adhesion formation.[14] O'Connor and Berndl reported a PASD case at a higher placental invasion site compared to most PASD cases due to a classical caesarean incision.[15] Classical caesarean section caused a higher accreta rate (0.88%) than lower transverse caesarean section women (0.19%), but no significant difference was found

after gestational age adjustment.[16] Continuous sutures along the inner side of the uterine wall increase the risk of placenta accreta in subsequent pregnancies compared to interrupted sutures.[17] Hysteroscopic correction and diverticulectomy are common surgical interventions for managing CSD to achieve anatomical correction, alleviate symptoms, restore fertility, increase myometrium thickness, and reduce the risk of PASD.[18]

Besides caesarean section, major uterine surgery associated with PASD is myomectomy. Currently, laparoscopic myomectomy (LM) is preferred over laparotomy for its minimal invasiveness. In cases of LM for submucosal myoma, uterine endometrium disruption can lead to uterine cavity breaches and PASD in subsequent pregnancies.[19] Procedures that cause minimal damage to uterine integrity, including curettage, manual placenta, endometritis, and uterine artery embolisation, also have been correlated to PASD in subsequent pregnancies.[20] Placenta previa is also a risk factor. The risk of developing placenta accreta in women with placenta previa is 3%, 11%, 40%, 61%, and 67% for first to fifth caesarean sections, respectively.[21] Short interpregnancy intervals of less than 18 months increase uterine rupture risk in the trial of labour following caesarean delivery, leading to suboptimal healing of the endometrial-myometrial interface, inadequate decidualisation, scar formation, and increased risk of PASD.[22]

Other risk factors for PASD are advanced maternal age, multiparity, as well as Asherman's syndrome.[23] The Nordic Obstetric Surveillance Study reported that pregnant women over 35 are 4.5 times more likely to develop PASD.[12] However, the relationship between maternal age and the incidence of PASD was confounded by other factors such as multiparity, placenta previa, or a history of previous surgery. The studies evaluating PASD in Asherman's syndrome are scarce. Tavcar et al. reported that 23.7% of patients treated for Asherman's syndrome have PASD.[24]

Sometimes, the embedding of villous tissue into the myometrium is not a result of major uterine surgeries. Instead, it can be due to abnormalities in the uterus itself. These abnormalities may include conditions such as a bicornuate uterus, myotonic dystrophy, adenomyosis, and submucous fibroids. This might explain the sporadic PASD cases documented before the 20th century. However, a recent study suggests that non-scarring ectopic placentation, either in the upper part of the fallopian tube or in the underdeveloped section of a bicornuate uterus, often results in uterine rupture during the early second trimester. This condition is commonly mistaken for placenta percreta. The confusion arises not due to an excessive invasion of villous tissue, but rather due to the inherent structural abnormality of the underlying myometrium.[4] The PASD has also been documented in women who have never given birth and have no previous uterine pathology, but the exact cause of this condition remains unknown. Because most of these case reports were published before the introduction of imaging technologies, it is difficult to determine the associated etiology of the abnormal placental attachment.[9]

Table 1. Uterine pathology associated with PASD

Classification	Uterine pathologies
Surgical scar	Dilatation and curettage, endometrial resection, myomectomy, surgical termination of pregnancy, caesarean section, Asherman's syndrome.
Nonsurgical scar	History of PASD, endometritis, manual placenta, intrauterine device, uterine artery embolisation, IVF procedures, chaemotherapy, and radiation.
Uterine anatomical anomalies	Bicornuate uterus, submucous fibroids, adenomyosis, myotonic dystrophy.

IVF: in vitro fertilisation, PASD: placenta accreta spectrum disorder. Modified with permission from Jauniaux et al.[9]

Etiopathology

The development of PASD involves a complex mechanism. The endometrial stroma undergoes decidualisation before blastocyst attachment and trophoblast infiltration, which is needed for normal implantation and placental development.[25] The epithelial-to-mesenchymal transition (EMT) is a biological process in which epithelial cells transform into migratory mesenchymal cells, which is crucial for the proper invasion and attachment of the placenta to the myometrium during the first trimester. In PASD, excessively active EMT persists during pregnancy and influences the migratory behaviour of EVT in pregnancy-associated spiral

artery remodelling.[26,27] Matrix metalloproteinase (MMP), particularly MMP-2 and MMP-9, aids trophoblast cells in destroying and penetrating extracellular matrix. Higher expression of MMP-2 and MMP-9 is observed in PASD patients.[26,28]

A normal placenta does not extend beyond the deep myometrium due to strict spatial and temporal regulation. The previous theory describes that invasive placenta in PASD can proliferate and invade local organs.[26] These features have been used for over 50 years and serve as the foundation for diagnosing and classifying accreta placentation.[25] However, the current theory demonstrated that the endometrial-myometrial junction defect at the site of a previous hysterotomy can lead to decidualisation failure, causing extensive EVT infiltration into the myometrium, including blood vessels and adjacent pelvic organs. The trophoblast alterations seen in PASD are most likely secondary to these physical changes rather than a primary defect leading to excessive myometrium invasion.[25]

Major surgical procedures such as caesarean section will cause scar tissue to form in the myometrial smooth muscle layer. Collagen and other "foreign" substances are formed. Myofiber disarray, tissue oedema, and tissue inflammation also occur in the wound-healing process following surgery. Multiple caesarean delivery (MCD) scars are frequently linked to a visible loss of myometrium that allows a direct connection between the endometrial cavity and the visceral serosa.[14,18,29] The decidual defect caused by artificial scar formation in myometrium has a negative impact on implantation by allowing the blastocyst to attach to scar tissue preferentially. The lower uterine segment (LUS) is more susceptible to CSD and accreta placentation due to its lower myofiber count and higher elastic connective tissue content compared to the upper segment.[4] The uterine circulation in women with previous caesarean delivery is poorly vascularised, with higher blood flow resistance. This results in impaired endometrial reepithelialisation affected by a large scar area resulting from MCD and scar dehiscence. The overlying area, poor decidualisation, and lack of myometrium structure underneath lead to the presence of anchoring villi deep into the uterine wall, allowing EVT to invade deep myometrium and beyond.[29] These microscopic characteristics are critical to the widespread misconception that accreta placental villous tissue is unusually invasive, which has resulted in substantial clinical data misinterpretation and heterogeneity. There is no substantiated proof indicating the EVT's aberrant invasiveness or villous tissue's ability to penetrate the uterine serosa and enter the pelvis during accreta placentation. The dimensions of the scar defect, the extent of placental tissue infiltration within the scar, and the remaining thickness of the uterine muscle in the scar region all impact the distance between the placental basal plate and the uterine serosa and determine the likelihood of accreta placentation.[4,18]

The migration of the trophoblast is stimulated by growth factors and cytokines, i.e., vascular endothelial growth factor (VEGF), epidermal growth factor (EGF), tumour necrosis factor (TNF)-α, interleukin (IL)-1ß, the hyperglycosylated form of human chorionic gonadotropin (hCG), and low oxygen concentrations.[25,29] The inhibitors of trophoblast migration and the extracellular matrix composition are also crucial in placentation process regulation.[30] In most PASD cases, there is extensive neovascularisation due to upregulated angiogenic growth factors, i.e. angiopoietin-2 (Ang-2) and VEGF. Antiangiogenic proteins, i.e., endothelial cell tyrosine kinase receptor (TIE-2), soluble fms-like tyrosine kinase 1 (sFLT-1), and VEGF receptor-2 (VEGFR-2) were also shown to be less expressed in syncytiotrophoblastic cells of PASD patients. These findings indicate that aberrant villous adherence results from abnormal expression of factors related to trophoblast growth, invasion, and angiogenesis.[26] When EVT migrates deeper than normal, the arterial vasculature is also altered along the full depth of the myometrium.[25] The uteroplacental vascular alterations caused by neovascularisation and increased deep uterine vessel recruitment by EVT along with chorionic villi beyond the junctional zone, led to the loss of the typical cleavage plane and prevented placental separation after delivery.[26] The increased recruitment of major arteries in the uterine wall during early pregnancy also leads to rapid maternal blood flow into the intervillous space and placental lacunae formation.[4,18]

Classification

Pathologists previously distinguished three subtypes of placenta accreta based on the depth of trophoblast penetration into the myometrium layer: adherent placenta accreta in which the villi are

only attached to the myometrium; placenta increta where villi invade the myometrium to the external layer; and placenta percreta when the villi invade all layers of the myometrium, including the serosa of the uterus and occasionally the surrounding pelvic organs. Abnormally invasive placenta refers to placenta increta and percreta.[1,25,29] However, there has been much confusion about the degree of accreta placentation because the adhesion or invasion of the villi is not uniform across the placental layers, restricting the microscopic diagnosis when the entire uteroplacental surface cannot be examined. Therefore, depending on the number of placental cotyledons involved, variations in the lateral extension of myometrial invasion additionally divide PASD into focal, partial, or total categories.[9] These terms offer standardised terminology that includes the depth of villous penetration, lateral extension of the placentation, and different penetration depths in the same PASD case.[31] However, recent findings have suggested that the EVT is not excessively invasive, and the accreta placentation is more likely because of decidualisation failure due to blastocyst implantation inside a CSD. The depth of adherence depends on residual myometrial thickness after the cesarean delivery or any other uterine surgery. Therefore, the classification of placenta accreta based on the depth of trophoblast penetration is no longer relevant.[4]

Histopathologic examinations are crucial and frequently provide a gold standard for diagnosing many medical conditions. The International Federation of Gynaecology and Obstetrics (FIGO) has recently established a clinical classification of PASD based on the placental detachment at delivery and the gross visualisation of invasion during surgery (Table 2).[9]

Table 2. Grading system of placental adherence at delivery

Grade	Definition
1	Full separation of the placenta during the third stage of labour. The placenta adheres normally.
2	(A) Caesarean section: No signs of placental tissue invading the surface of the uterus were found. For parts of the placenta that were inappropriately attached, the use of uterotonics and moderate cord traction was not enough, and it was necessary to manually remove the placenta. (B) Vaginal birth: Manual extraction of the placenta was needed, as there are indications of aberrant adhesion of certain placental parts.
3	(A) Caesarean section: No signs of placental tissue invading the surface of the uterus were found. The entire placental bed were inappropriately attached, the use of uterotonics and moderate cord traction was not enough, and it was necessary to manually remove the placenta. (B) Vaginal birth: Manual extraction of the placenta was needed, as there are indications of aberrant adhesion of the entire placental bed.
4	Caesarean section: Placental tissue was observed to have penetrated the serosa of the uterus, although a distinct boundary was distinguished between the bladder and uterus, enabling the safe separation of the urinary bladder during surgery.
5	Caesarean section: Placental tissue was observed to have penetrated the serosa of the uterus, and no distinct boundary was distinguished between the bladder and uterus.
6	Caesarean section: Placental tissue was observed to have penetrated the serosa of the uterus and infiltrated the parametrium or any organ except for the urinary bladder.

Reproduced with permission from Jauniaux et al.[9]

Diagnosis

In order to avoid placental rupture, patients with PASD should deliver at a level III or IV maternal care facility. Therefore, antenatal identification of PASD is strongly encouraged to improve outcomes. Obstetric ultrasonography is the most often used modality.[8] According to a comprehensive review and meta-analysis of 3707 women by Nouri et al., the overall performance of ultrasonography is remarkable, with a sensitivity of 90.72%, specificity of 96.94%, and odds ratio of 98.[59.32] Colour Doppler ultrasonography can increase imaging sensitivity to 90%, with 95-98% negative predictive values.[33] The most prevalent finding of PASD on colour Doppler imaging is turbulent lacunar blood flow with pulsatile venous-type flow and dilated peripheral sub-placental vascular channels, unlike the normal non-pulsatile sub-placental venous complex and low-velocity intravenous blood flow waveforms.[34]

While ultrasound has the capability to identify PASD symptoms during the initial trimester, most women receive a diagnosis in the late trimesters. The ultrasound findings include numerous intraplacental lacunae, absence of the usual hypoechoic area between the placenta and the myometrium, reduced retroplacental myometrial thickness, uterine serosae-bladder interface abnormalities, and placenta infiltration into the myometrium, serosa, or bladder.[34] Transvaginal sonography can be used to grade intraplacental lacunae according to Finberg's criteria: Grade 0 for none was seen, Grade 1 for one to three typically tiny lacunae, Grade 2 for four to six bigger or more irregular lacunae, and Grade 3 for numerous throughout the placenta, some of which seemed large and irregular in shape (Figure 1C).[35] The placenta accreta index (PAI) score, developed by Rac et al., is a 9-point scoring system incorporating ultrasound findings and clinical risk factors for PASD, including ≥2 caesarean delivery (3 points), lacunae grade 3 (3.5 points) or grade 2 (1 point), sagittal smallest myometrial thickness ≤1 mm (1 point) or ≤3 mm (0.5 point) or ≤5 mm (0.25 point), anterior placenta previa (1 point), and bridging vessels (0.5 point). Probability of PASD at different cut-off points of PAI score: >0 (5%), >1 (10%), >2 (19%), >3 (33%), >4 (51%), >5 (69%), >6 (83%), >7 (91%), >8 (96%).[36] According to research by Happe et al., with a threshold of more than 4, PAI offers an 87% sensitivity, 77% specificity, 72 % positive predictive value, and 90 % negative predictive value for PASD diagnosis.[37]

Tovbin et al. generated a scoring system to predict morbidly adherent placenta (MAP) in women at risk of MAP based on ultrasonography and clinical features. Patients were grouped into one of three groups based on this proposed scoring system: 5 points (low), 6-7 points (moderate), or 8-12 points (high) likelihood for MAP (Table 3, Figure 2).[39] Agarwal et al. also described another MAP score derived from the PAI score. The PAI values were categorised into two broad categories, with <5 being unfavourable for invasion (MAP score 0) and ≥5 being favourable (MAP score 1).[40]

Clinical risk factors are just as crucial for predicting PASD as ultrasound results.[8] The PASD is associated with various risk factors, i.e., maternal age, multiparity, assisted reproductive procedures, prior uterine surgery (including curettage), and caesarian section delivery.[41] A prior caesarean delivery, as well as placenta previa, are the most prevalent risk factors. As a result, emphasising PASD screening in this population yields superior diagnostic outcomes.[9]

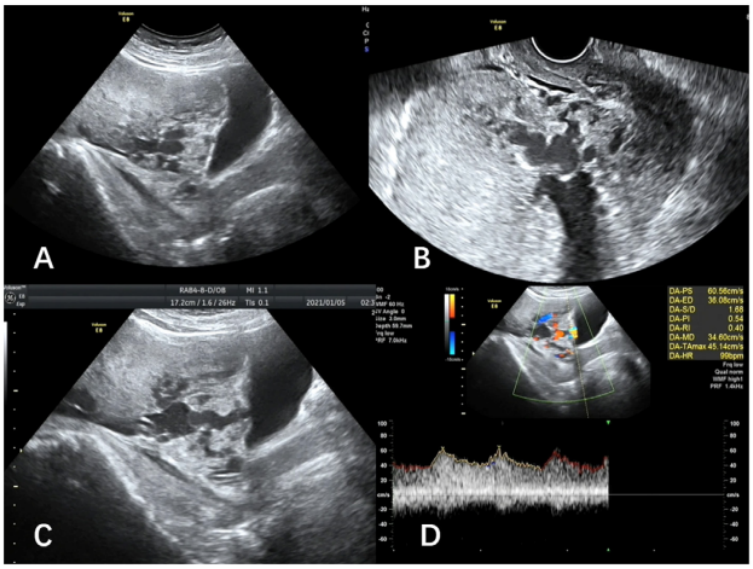

Figure 1. Sonography findings of Placenta Accreta Index parameter: (A) anterior placenta previa, (B) smallest myometrial thickness, (C) Grade 3 of intraplacental lacunae, (D) bridging vessels. Reproduced with permission from Zhang et al.[38]

Table 3. Morbidly adherent placenta score

Parameter	Score
"Number of previous caesarean deliveries"	
1	1
≥ 2	2
"Lacuna maximum dimension"	
≤ 2 cm	1
> 2 cm	2
"Number of lacunae"	
≤ 2	1
> 2	2
"Obliteration of uteroplacental demarcation"	2
"Location of placenta"	
Anterior	1
Placenta previa	2
"Doppler assessment"	
Blood flow in placental lacunae	1
Placenta–bladder and/or uteroplacental interface hypervascularity	2

Reproduced with permission from Tovbin et al.[39]

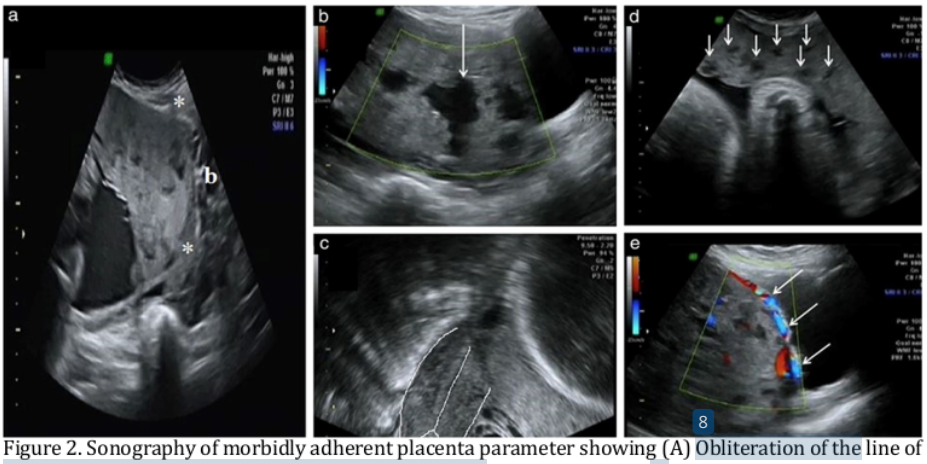

Figure 2. Sonography of morbidly adherent placenta parameter showing (A) Obliteration of the line of demarcation (*) between the uterus and the placenta. (B) Lacunar size of ≥ 2 cm, (C) Placenta previa, (D) > 2 placental lacunae, (E) hypervascularity between the placenta and the bladder. Reproduced with permission from Tovbin et al.[39]

Prenatal diagnosis of PASD disorders with magnetic resonance imaging (MRI) is becoming more prevalent, with overall diagnostic accuracy of 94%, specificity of 84%, and diagnostic odds ratio of 89%.[1] The most common MRI findings of placenta accreta include abnormal uterus bulging, black intraplacental bands on T2-weighted imaging, varying intensity within the placenta, disruption of the uteroplacental zone, and dysfunctional placental vasculature.[34] Also, MRI was suggested as beneficial for spotting parametrial infiltration by villous tissue in complicated cases with posterior placenta previa or determining the degree of infiltration in suspected percreta. MRI is more costly and less widely available than ultrasonography. It also needs to be interpreted by an expert. Therefore, MRI is not commonly suggested as a routine examination of suspected PASD.[42]

Numerous placental and foetal hormone levels have been shown to vary in the placenta previa accreta versus non-accreta previa serum. Women with PASD commonly had reduced levels of maternal β-hCG at 11–12 weeks of pregnancy but greater levels of pregnancy-associated plasma protein-A (PAPP-A). Women with placenta previa, however, are 31 times more likely to develop PASD if their blood β-hCG and alpha-fetoprotein (AFP) levels are greater than 2.5 times multiple of the median (MoM).[43]

Management

For efficient therapy of the placenta accreta spectrum, a systematic strategy with a multidisciplinary care team is essential. The RCOG, along with The ACOG, have established recommendations for the optimal clinical care of PASD.[8] Appropriate prenatal diagnosis, careful planning, and effective communication are all critical, as is the establishment of surgical teams to execute safe PASD therapy.[10] The implications of the delivery time for maternal and newborn risk and benefits must be evaluated. Unless there are extenuating circumstances in a patient, a gestational age of 340/7 – 356/7 weeks is regarded to be the best planned caesarean birth or hysterectomy.[44] Antenatal corticosteroids are required for lung maturation in prenatally diagnosed accreta and to predict birth before 370/7 weeks of gestation. Preoperative haemoglobin optimisation is essential because of the possibility of bleeding after delivery.[10] Caesarean section and hysterectomy, expectant management (retaining the placenta in situ indefinitely), and surgical techniques that preserve the uterus are the three main management choices for PASD.[45]

Hysterectomy

For the treatment of PASD, the ACOG advises a planned premature caesarean surgery and hysterectomy with the placenta left in position.[21] Patients are placed in a dorsal lithotomy posture to provide for better surgical visibility of the pelvis as well as unobstructed access to the vagina and bladder.[8] A midline skin incision is usually required when the placenta is anterior and advancing towards the level of the umbilicus, allowing for high upper-segment transverse uterine incisions above the placenta's top limit.[10] Wide transverse incisions like the Maylard or Cherney incision are other alternatives. A comprehensive uterine examination is required to determine a particular placental location and the extent of placental invasion, which then will enable the most efficient approach to the uterine incision for delivery.[8] Devascularizing procedures on both lateral sides of the uterus and clamping the uterus at the lowest possible spot with a clamp just below the placenta border while maintaining the ureters can reduce maternal bleeding morbidity.[46] When extensive invasion of nearby tissues makes caesarean hysterectomy impossible, or when the PASD is discovered post-partum, a delayed hysterectomy may be required.[47]

Postoperative uterine artery embolisation, internal iliac artery or abdominal balloon blockage, and intraoperative internal iliac artery embolisation have all been recommended to avoid perioperative and post-partum bleeding in high-risk women. Arterial embolisation in PASD has a success rate of roughly 90%, with further peripartum hysterectomy necessary in 11.3 % of patients.[48] Although the use of prophylactic balloon catheter implantation in the iliac arteries in PASD is associated with a 70% success rate, it is still controversial due to the high risk of possible complications. Haemodynamic parameters must be constantly monitored in these circumstances. Constant monitoring of blood loss, haemoglobin, coagulation markers, electrolytes, as well as blood gas is necessary since it can provide objective replacement needs in real-time.[47] The use of tranexamic acid lowers bleeding complications and death in nonobstetric patients by preventing fibrin breakdown.[49] An intravenous dose of 1 gram should be given within 3 hours of delivery. If the bleeding continues, the second dosage could be administered 0.5-23.5 hours later.[50] Severe and refractory post-partum haemorrhage has been treated with recombinant activated factor VIIa.[51] More factors should be examined in the bleeding and placenta accreta spectrum. Many clotting factors are ineffective at degrees below 36° C. Thus, patients should be kept warm. Acidosis should also be avoided. Prophylactic antibiotics should be re-dosed if blood loss is substantial, which is commonly defined as 1,500 mL or more of expected blood loss.[52]

Patients with placenta accreta require thorough haemodynamic monitoring in the early postoperative period. Patients should return for a six to eight-week postoperative assessment,

including a pathology review and a discussion of ongoing gynaecologic care. Breastfeeding is advised and frequently effective, but due to continual estrogen production by decomposing placental tissues, the choice of non-removal of the placenta may be difficult.[42]

Conservative surgery

Conservative uterus-preserving treatment may be appropriate when the extent of the PASD zone is limited, can be fully observed, and accessible, i.e., complete posterior, fundal, or anterior without deep pelvic or cervical invasion.[48,53] Patients with localized placental adhesion can be managed with manual or surgical removal of the placenta, followed by repair of the resulting defect. There is evidence that en bloc excision of the whole uteroplacental defect followed by uterine closure minimises haemorrhage and is able to preserve potential fertility in individuals with a massive defect. Alternatively, in recent research by Pala et al., placental excision, followed by the implantation of a Bakri balloon, was proven successful in avoiding the need for hysterectomy in 84 % of patients.[54]

One-step conservative surgery (OSCS) is regarded as one of the most effective techniques for treating PASD after a hysterectomy. Compressive sutures, surgical or endovascular vascular occlusion, and targeted excision of the incorrectly infiltrated myometrium are used in this procedure. To establish whether a patient is a candidate for OSCS, three requirements must be met: bladder detachment from the uterus (with no vesicouterine fibrosis); above the cervix, there exists at least 2 cm of healthy myometrium; and the affected myometrium is < 50% of the uterus axial circumference. The first step in treating PASD is retrovesical dissection followed by vesicouterine pedicle ligation. Following that, a segmental hysterotomy is carried out right above the aberrant location, allowing the fetus to be safely extracted without more blood loss. Following the surgical cut into the myometrium, the obstetrician positions their palm on the upper end of the myometrium (artificial abruption) between the regular myometrium and the placenta until it reaches the oval-shaped membranes (Ward procedure). After childbirth, the uterus is everted to thoroughly check the posterior uterine wall. When placenta previa or placenta accreta is present, the internal pudendal artery branches expand and deliver a substantial volume of blood to the area where the aberrant placenta is attached. The colpouterine pedicles rise through the vaginal walls and join the uterine arteries on both sides. The pedicles are constantly located in the positions corresponding to 3 o'clock, 6 o'clock, and 9 o'clock. Based on these specific topographic parameters, lower haemostatic sutures are strategically positioned to disrupt the abnormal region's blood flow before its removal. The last step involves excising the anomalous myometrium and placenta, followed by uterine restoration. This technique has less frequency of transfusions and vascular interventions compared to hysterectomy with shorter operation time (164.4 minutes vs 216.5 minutes) and more to be applied in scenarios with limited resources.[45] Various surgical-vascular control approaches have also been reported to improve results. Vascular control with identification-ligation of the upper vesical, upper vaginal, and uterine arteries resulted in much less blood loss than uterine conservative-resective surgery with internal iliac artery ligation (IIAL).[55]

No randomised controlled trials comparing the various conservative treatment methods. A comprehensive review of observational studies compares the failure rates (secondary hysterectomy or maternal mortality) and failure rates (subsequent menstruation or pregnancy) of uterus-preserving treatment. The cumulative success rate for artery embolisation was 89.8%, with subsequent menstruation in 87.1% and subsequent pregnancy in 30% of the patients. The success rate of the artery occlusion balloon was 78.6%. Uterus-preserving surgery resulted in a 63.2% success rate, 81.1% following menstruation, and 77.8% subsequent pregnancy. Following arterial embolisation, artery occlusion balloon, and uterus-preserving surgery, 11.3%, 19%, and 30% of patients required a secondary hysterectomy, respectively. Maternal mortality was found in 3.7% of uterus-preserving surgery patients. Surgical methods with the uterus-preserving technique are linked with lower (16%) unintentional damage of the urinary tract, compared to standard hysterectomy (57%) due to the use of ureteric stents.[56] According to a large number of authors, uterine preservation surgery for PASD has a high success rate, occasionally

exceeding 100%.[57] Nevertheless, the significance of these discoveries is greatly limited by the retrospective nature of this research, the small sample size, the shortage of control groups, the absence of histological verification, and the lack of standardised photographic or clinical confirmation of PASD at birth.[58]

Expectant management

The phrase "expectant management" refers to the practice of leaving the placenta in situ, either partially or entirely.[59,60] In individuals with a more widespread placenta accreta spectrum, the use of expectant management is being considered as a treatment option. The most common treatment is to ligate the cord around the placenta and leave the complete placenta in situ or to remove the only placenta that separates spontaneously before the uterus closes.[48]

Multidisciplinary team (MDT) approach

Women who received conventional obstetric care without a specialised protocol are at a higher risk of receiving emergency surgery, major blood transfusions, and reoperation for bleeding within seven days after birth compared to those who were diagnosed with PASD in the prenatal period and treated by a multidisciplinary team (MDT) centre. Anesthesiologists, maternal-fetal medicine specialists, neonatologists, as well as gynaeco-oncology or reconstructive surgeons, will collaborate on a preoperative approach to ensure the best possible outcome. As that same group gains progressive experience, the MDT can potentially enhance maternal outcomes and improve the quality of care over time.[61]

CONCLUSION

The PASD is a serious condition with high morbidity and mortality rates. Previous theory describes that an invasive placenta proliferates and can invade local organs. The current theory is that the presence of a CSD in the endometrial-myometrial junction can lead to decidualisation failure, causing extensive EVT infiltration into the myometrium. Clinical risk factors are equally as crucial as ultrasound findings in predicting PASD. Future research should concentrate on gathering prospective data on the prenatal diagnosis and risk management of PASD.

CONFLICT OF INTEREST

All authors declare that there is no conflict of interest.

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AUTHOR CONTRIBUTION

The authors confirm their contribution to the paper as follows: study conception and design: PM, PML, KM, IAL; methodology: PM, HA, CK, BS; draft manuscript preparation: PM, HA, BS; visualisation: BS: validation: PML, KM, IAL, CK; project administration: PM. All authors reviewed the results and approved the final version of the manuscript.

LIST OF ABBREVIATION

PASD: placenta accreta spectrum disorder; CSD: caesarean scar defect; LM: laparoscopic myomectomy; IVF: in vitro fertilization; EMT: epithelial-to-mesenchymal transition; MMP: matrix metalloproteinase; EVT: extravillous trophoblast; MCD: Multiple caesarean delivery; LUS: lower uterine segment; VEGF: vascular endothelial growth factor; EGF: epidermal growth factor; TNF: tumor necrosis factor; IL: interleukin; hCG: human chorionic gonadotropin; Ang-2: angiopoietin-2; Tie-2: endothelial cell tyrosine kinase receptor; sFLT-1: soluble fms-like tyrosine kinase; VEGFR-2: VEGF receptor-2; PAI: Placenta Accreta Index; MAP: morbidly adherent placenta; MRI: magnetic resonance imaging; PAPP-A: pregnancy-associated plasma protein-A; AFP: alpha-fetoprotein; MoM: multiple of the median; RCOG: Royal College of Obstetricians and Gynaecologists; ACOG: American College of Obstetricians and Gynaecologists; OSCS: one-step conservative surgery; IIAL: internal iliac artery ligation; MDT: multidisciplinary team

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