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FIGO consensus guidelines on placenta accreta spectrum disorders: Epidemiology☆,★

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1 | INTRODUCTION

Placenta accreta was first described nearly 80 years ago as a clinico-pathological condition in which the placenta fails to separate partially or totally from the uterine wall.[1] Several concepts have been proposed to explain why and how it occurs. In the past, it was thought that a primary defect of the biological function of the trophoblast would lead to excessive invasion of the myometrium by placental tissue beyond the physiological decidual–myometrial junction zone.[2,3] The current prevailing hypothesis is that a defect of the endometrium–myometrial interface, typically at the site of a prior hysterotomy, leads to a failure of normal decidualization in the corresponding uterine area. This allows extravillous trophoblastic infiltration and villous tissue to develop deeply within the myometrium, including its circulation, and to sometimes reach the surrounding pelvic organs.[3] The cellular changes in the trophoblast observed in accreta placentation are probably secondary to the unusual myometrial biological environment in which it develops, and not to a primary defect of trophoblast biology leading to excessive invasion of the myometrium.[2,3]

Depending on the depth of trophoblast invasion into the myometrium, three subtypes have been differentiated by pathologists: (1) superficial placenta accreta (also called placenta creta, vera, or adherenta), where the villi attach directly to the surface of the myometrium without invading it; (2) placenta increta, where the villi penetrate deeply into the myometrium up to the external layer; and (3) placenta percreta, where the invasive villous tissue reaches and penetrates through the uterine serosa.[2,3] Placenta increta and percreta are often referred to as abnormally invasive placenta. More invasive placentation is not due to a further invasion of extravillous trophoblast in the uterine wall, but likely arises from an extended scar defect that allows the development of chorionic villi deep within the uterine wall, including within its peripheral circulation.[4] The striking rise in the incidence of abnormally adherent and invasive placentation in women with a prior cesarean delivery supports the latter concept.[3]

The challenge in writing this chapter on the epidemiology of accreta placentation was the heterogeneous definition of the condition. Nearly half of the cohort studies published over the last three decades do not provide evidence of correlation between prenatal ultrasound signs, clinical symptoms, and detailed pathologic findings at delivery.[5] In addition, the recent inclusion of both adherent and invasive forms of accreta placentation into one archaic category i.e. "morbidly adherent" makes the interpretation of clinical data more difficult. This could explain the wide variability in the prevalence of the different degree of accreta placentation, in the accuracy of prenatal diagnosis, and in differences in outcomes, as well as why prenatal detection rates remain low in recent population studies.[6–8] To facilitate the discussion, we use placenta accreta spectrum (PAS) disorders to include both adherent and invasive placental disorders.

Massive obstetric hemorrhage is one of the most severe morbidities of childbirth and one of the most important and potentially avoidable causes of maternal death. Retained placental tissue and secondary uterine atony remains one of the most common causes of

massive obstetric hemorrhage globally, and postpartum hemorrhage in particular.[9] Any attempt to manually remove a PAS disorder typically provokes heavy bleeding and is associated with high maternal morbidity and mortality.[10] The clinical symptoms of PAS disorders—in particular in cases of a partially adherent placenta—can be very similar to those of placental retention, and some authors have amalgamated the two conditions together.[11] However, a retained placenta, which is merely entrapped in the uterus after childbirth owing to constriction of the cervix, should not be included in the category of PAS disorders; nor should cases where a retained placenta is easily removed within 30 minutes after birth. This suggests that the prevalence of PAS disorders and in particular of invasive accreta placentation is likely to be lower than that reported by many previous clinical studies.

In many medical conditions, histopathologic findings are essential and often provide a gold standard for the definition of the condition. However, myometrial fibers can sometimes be found in the basal plate of normal placentas,[12] the decidua is not a continuous layer and it becomes thinner with advancing gestation,[3] and in many cases of placenta percreta the extended damage to the uterine wall, with no decidual and myometrial tissue left at the site of placentation, makes histopathologic examination impossible.[3] This leaves the clinical description as the most important criteria for definition and stratification of PAS disorders (Table 1). In the present chapter, we review the available epidemiologic evidence on PAS disorders and discuss their etiopathology.

2 | UTERINE SCAR AND ACCRETA PLACENTATION

Theoretically, any primary uterine anomaly or secondary damage to the uterine wall structure can lead to PAS disorders, including the invasive forms.[2,3,13] PAS disorders have been reported in primigravid women with no obvious uterine disorders.[13] However, these cases are extremely rare and past surgical history, in particular regarding pregnancy termination, may not always be accurate.[14]

2.1 | Cesarean scar

The increase in prevalence of PAS disorders has been directly linked to the increase in cesarean delivery rates in most middle- and high-income countries, and is supported by strong epidemiologic data.[6,8,15–26] There are currently no epidemiological data on the prevalence of PAS disorders in low-income countries.

In their original study published in 1937, Irving and Hertig[1] estimated the incidence of placenta accreta to be 1 in 30 000 deliveries in the USA. Their cohort study of 18 cases included only one woman with a prior cesarean delivery. By contrast, a matched case–control study published in 2005, including 111 cases of PAS disorders identified using strong clinical criteria or histopathologic examination, found the incidence of PAS disorders to be 1 in 533 births.[15] This incidence corresponds with an 8-fold and a 5-fold increase compared with the 1970s and 1980s, respectively, and is linked with cesarean delivery rates in the USA increasing from 12.5% in 1982 to 23.5% in 2002.

The increase in cesarean delivery rates in Europe occurred about a decade later than in the USA (Table 2). An Irish institutional cohort study of 157 162 multiparous women delivered over a 36-year period found that the cesarean delivery rates increased from 4.1% in 1975 to 20.7% in 2010, and that the incidence of PAS disorders increased from 1.65 per 1000 women to 2.37 per 1000 women with prior cesarean delivery between 2003 and 2010 (OR 2.2, 95% CI 1.05–5.1).[20] An Italian cohort study of cases of PAS disorders diagnosed at birth over four decades found that the incidence increased from 0.12% during the 1970s to 0.31% during the 2000s.[18] During the same period, cesarean delivery rates increased from 17% to 64%. Prior cesarean delivery was the only risk factor, showing a significant concomitant rise. A recent study from Hong Kong found that the prevalence of PAS disorders increased from 0.17 per 1000 births in the period 1999–2003 to 0.79 per 1000 births in the period 2009–2013.[24] None of the above studies provide data on the depth of placental invasion. In addition, the rate of PAS disorders increased in women with previous cesarean deliveries and those with an unscarred uterus,[24] which suggests that the authors

TABLE 1 A clinical grading system to assess and categorize placental adherence or invasion at delivery.[a]

Grade	Definition
1	*At cesarean or vaginal delivery*: Complete placental separation at third stage. Normal adherence of placenta
2	*(A) Cesarean/laparotomy*: No placental tissue seen invading through the surface of the uterus. Incomplete separation with uterotonics and gentle cord traction, and manual removal of placenta required for remaining tissue and parts of placenta thought to be abnormally adherent *(B) Vaginal delivery*: Manual removal of placenta required and parts of placenta thought to be abnormally adherent
3	*(A) Cesarean/laparotomy*: No placental tissue seen invading through the surface of the uterus. No separation with uterotonics and gentle cord traction with manual removal of placenta required and the whole placental bed thought to be abnormally adherent *(B) Vaginal delivery*: Manual removal of placenta required and the whole placental bed thought to be abnormally adherent
4	*Cesarean/laparotomy*: Placental tissue seen to have invaded through the serosa of the uterus but a clear surgical plane can be identified between the bladder and uterus to allow nontraumatic reflection of the urinary bladder at surgery
5	*Cesarean/laparotomy*: Placental tissue seen to have invaded through the serosa of the uterus and a clear surgical plane cannot be identified between the bladder and uterus to allow nontraumatic reflection of the urinary bladder at surgery
6	*Cesarean/laparotomy*: Placental tissue seen to have invaded through the serosa of the uterus and infiltrating the parametrium or any organ other than the urinary bladder

[a]Modified from Collins et al.[71]

TABLE 2 Changes in cesarean delivery rate and placenta accreta spectrum (PAS) disorder prevalence over time.

Author	Type of study	Country of origin	Cesarean delivery rate period A (years)	Cesarean delivery rate period B (years)	PAS disorders period A (years)	PAS disorders period B (years)
Wu et al.[15] (2005)[a]	Matched case–control study	USA	12.5% (1982)	23.5% (2002)	0.38 per 1000 births (1982)	1.88 per 1000 births (2002)
Higgins et al.[20] (2013)[b]	Cohort study	Ireland	4.1% (1975)	20.7% (2010)	1.65 per 1000 births after prior cesarean (2003)	2.37 per 1000 births after prior cesarean (2010)
Morlando et al.[18] (2013)[c]	Cohort study	Italy	17% (1970s)	64% (2000s)	1.20 per 1000 births after prior cesarean (1976–1978)	3.11 per 1000 births after prior cesarean (2000s)
Cheng and Lee[24] (2015)[d]	Cohort study	Hong Kong	19.5% (1999–2003)	27.1% (2009–2013)	0.17 per 1000 births after prior cesarean (1999–2003)	0.79 per 1000 births after prior cesarean (2009–2013)

[a]Total prevalence 0.19% (121 cases of PAS disorders out of 64 359 deliveries during the study period).
[b]Total prevalence 0.01% (36 cases of PAS disorders out of 275 121 deliveries during the study period).
[c]Total prevalence 0.16% (50 cases of PAS disorders out of 30 491 deliveries during the study period).
[d]Total prevalence 0.05% (39 cases of PAS disorders out of 81 497 deliveries during the study period).

included abnormally adherent and invasive placenta cases as well as cases of difficult removal of a retained placenta in their data.

The Nordic Obstetric Surveillance Study (NOSS) using direct clinical reports validated by national registers found that the rate of PAS disorders at cesarean delivery or laparotomy was 3.4 per 10 000 deliveries. When vaginal deliveries with difficult removal of the placenta and blood transfusion were included the rate was 4.6 per 10 000 deliveries.[8,27] A recent meta-analysis of five cohorts and 11 case–control studies reported a summary OR of 1.96 (95% CI 1.41–2.74) for PAS disorders after a cesarean delivery.[26] The corresponding data were not stratified for the number of prior cesarean deliveries. When stratified by the number of previous cesarean deliveries, the ORs for PAS disorders in a subsequent pregnancy increased from 8.6 (95% CI 3.5–21.1) after one prior cesarean to 17.4 (95% CI 9.0–31.4) after two previous cesareans, and to 55.9 (95% CI 25.0–110.3) after three or more prior cesarean deliveries.[8,9,15] A multicenter study of 30 132 women who underwent elective cesarean delivery (without prior labor) in 19 academic hospitals in the USA between 1999 and 2002 found that 143 had PAS disorders and that the risk increased from 0.24% after one prior cesarean to 6.74% after six or more previous cesarean deliveries.[16]

A decision-analytic model built using data on national birthing order trends after cesarean delivery in the USA between 1995 and 2005 estimated that if the number of primary and secondary cesareans continues to rise, by 2020 the cesarean delivery rate will be 56.2% and that, as a consequence, there will be an additional 6236 cases of placenta previa, 4504 PAS disorder cases, and 130 maternal deaths annually.[28] The study also calculated that the rise in these complications will lag behind the rise in cesarean deliveries by around 6 years. Poisson regression models were recently used to assess the relative incidence of morbidity among repeat versus primary cesarean delivery patients in the 2000–2011 US Nationwide Inpatient Sample dataset.[23] Overall, the study found that the unadjusted rate of PAS disorders increased by 30.8% among women with a repeat cesarean delivery. Compared with women with a primary cesarean delivery, women who underwent a repeat cesarean were 2.13 times more likely to have PAS disorders (95% CI 1.98–2.29).

2.2 | Other etiologies of accreta placentation

PAS disorders are not exclusively a consequence of cesarean delivery.[29] Procedures causing less surgical damage to the integrity of the uterine lining, such as uterine curettage, manual delivery of the placenta, postpartum endometritis and, more recently, hysteroscopic surgery, endometrial ablation, and uterine artery embolization have all been associated with PAS disorders in subsequent pregnancies.[2,3,12]

Development of PAS disorders has also been reported in women with no prior uterine surgery, but with uterine pathology such as bicornuate uterus, adenomyosis, submucous fibroids, and myotonic dystrophy (Table 3). These rare cases suggest that intramyometrial implantation of villous tissue is not always secondary to major uterine surgery and may explain the sporadic cases of PAS disorders observed before the 20th century. The prevalence of these uterine conditions in the general population, in particular fibroids and adenomyosis, and the lack of clear evidence of their association with invasive placentation suggest that they are probably not a major risk factor for PAS disorders. PAS disorders have been exceptionally reported in women with no previous pregnancies and no obvious uterine pathologies[13] but the etiology in these cases is impossible to evaluate. Overall, with the rapid increase in cesarean delivery rates worldwide, most of these other risk factors are now responsible for a relatively small proportion of PAS disorders.

The Nordic Obstetric Surveillance Study, which investigated severe obstetric complications between 2009 and 2012,[8] found that maternal age greater than 35 years increases the odds of PAS disorders by 4.5 (absolute risk: 7.5 per 10 000), confirming the results of a previous case–control cohort study[15] and retrospective cohort study.[21] This association is most likely due to confounding factors such as multiparity, risk of previa, and the risks of prior uterine surgery rather than advanced maternal age itself.

TABLE 3 Primary and secondary uterine pathologies reported to be associated with placenta accreta spectrum (PAS) disorders.[a]

Classification	Type of uterine pathologies
Direct surgical scar	Cesarean delivery
	Surgical termination of pregnancy
	Dilatation and curettage
	Myomectomy
	Endometrial resection
	Asherman's syndrome
Nonsurgical scar	IVF procedures
	Uterine artery embolization
	Chemotherapy and radiation
	Endometritis
	Intra-uterine device
	Manual removal of placenta
	Previous accreta
Uterine anomalies	Bicornuate uterus
	Adenomyosis
	Submucous fibroids
	Myotonic dystrophy

[a]Source: Irving and Hertig,[1] Jauniaux and Jurkovic,[2] Jauniaux et al.,[3] Parra-Herran and Djordjevic,[4] Jauniaux E, et al.,[14] Wu et al.[15]

The NOSS also found an OR of 3.1 for PAS disorders (absolute risk: 8.2 per 10 000) in pregnancies resulting from in-vitro fertilization (IVF).[8] The UK national case–control study using the UK Obstetric Surveillance System (UKOSS) found an adjusted OR (aOR) for PAS disorders of 32.1 (95% CI 2.0–509) for IVF pregnancies.[17] These data were confirmed by a recent case–control study of 1571 pregnancies resulting from IVF and/or intracytoplasmic sperm injection with autologous or donor oocytes, undergoing fresh or cryopreserved transfer (CET).[30] The multivariate analysis indicated an association between CET and PAS disorders (aOR 3.2, 95% CI 1.1–9.0). A case–control study of deliveries in a single tertiary care center also found a rate of PAS disorders of 1.6% after IVF compared with 0.12% in spontaneous pregnancies (OR 13.2, 95% CI 6.7–25.8) but parity, rate of cesarean delivery in the index pregnancy, and birth weight differed significantly suggesting an impact of confounding factors in the analysis.[31] A Japanese nationwide registry of assisted reproductive technology (ART) including 277 042 single embryo transfer cycles between 2008 and 2010 reported an OR 3.16 for PAS disorders.[32] A recent meta-analysis of cohort studies including 161 370 pregnancies resulting from ART compared with 2 280 241 spontaneous singleton pregnancies found no difference in the relative risk (RR) for PAS disorders.[33] Thus, more data are required to determine the impact of different ART on PAS disorders and other placental and cord anomalies.

The UK national case–control study reported an aOR for PAS disorders of 3.4 (95% CI 1.3–8.9) after previous minor uterine surgery.[17] Surgical termination of pregnancy and uterine curettages are common procedures and have been associated with PAS disorders in subsequent pregnancies.[2,3,12,13] Fragments of myometrium are often found in the products of conception in around one-third of surgical terminations and uterine curettages for miscarriage.[34] Myometrial fibers have also been noted in the basal plate in placenta from previous deliveries in women presenting with PAS disorders and greater quantities of myometrial fibers in a delivered placenta have been associated with the development of PAS disorders in a subsequent pregnancy.[35] A small case–control study of 25 cases of PAS disorders found that 76% had myometrial fibers attached to the placental basal plate compared with 41% of controls (OR 4.8, 95% CI 1.8–13.0).[36] Overall, the trauma to the myometrium and the surface of the endometrium is often limited in a curettage procedure and should not be associated with the absence of re-epithelialization of the scar area by endometrial cells compared with the larger and deeper scars resulting from cesarean delivery. Thus, this small trauma to the uterine wall is less likely to lead to the development of extended invasive placentation such as placenta precreta.

3 | PLACENTA PREVIA ACCRETA AND CESAREAN SCAR PREGNANCY

The single most important risk factor, reported in around half of all cases of PAS disorders, is placenta previa.[8] The risk of previa increases with higher numbers of prior cesarean deliveries.[15,16,26–28,37–44] Overall, following a single cesarean, there is a 50% increase in risk of placenta previa, and after two cesareans there is a two-fold increase in risk compared with women with a history of two vaginal deliveries.[38] The risk of placenta previa in the USA is 40% higher in twin pregnancies and increases by age and parity in both singleton and twin pregnancies.[39] A retrospective cohort study of 399 674 women who gave birth to a singleton first and second baby between 2000 and 2009 in England found an OR for placenta previa after one cesarean delivery of 1.60 compared with vaginal birth (95% CI 1.44–1.76).[40] Their meta-analysis of 37 previously published studies from 21 countries showed an overall pooled random effect OR of 2.20 (95% CI 1.96–2.46) and an additional placenta previa in the next pregnancy for 259 cesarean deliveries at first birth.[40] These results were confirmed by two other systematic reviews.[26,41]

In 1997, a meta-analysis of the association between placenta previa and prior cesarean delivery found a "dose-response" pattern for the RR of previa.[37] The authors found a RR for previa of 4.5 (95% CI 3.6–5.5) for one, 7.4 (95% CI 7.1–7.7) for two, 6.5 (95% CI 3.6–11.6) for three, and 44.9 (95% CI 13.5–149.5) for four or more prior cesarean deliveries compared with vaginal delivery. A more recent systematic review of 22 studies (including over 2 million deliveries) reported that the incidence of placenta previa increased from 10 per 1000 deliveries with one previous cesarean to 28 per 1000 deliveries with three or more previous cesareans.[41] A large retrospective cohort study of 26 987 women comparing prior to onset of labor cesarean delivery and intrapartum cesarean delivery found that prior pre-labor cesarean is associated with a more than a two-fold increased risk of previa in the second delivery (aOR 2.62, 95% CI 1.24–5.56).[43] By contrast, the 20% increased risk of previa associated with prior intrapartum cesarean delivery is not significant (aOR 1.22, 95% CI 0.68–2.19).

The UKOSS study found that the incidence of PAS disorders including increta and percreta increases from 1.7 per 10 000 pregnancies overall to 577 per 10 000 pregnancies in women with both

a previous cesarean delivery and placenta previa.[17] The estimated ORs of PAS disorders in cases of placenta previa diagnosed prenatally range between 51.4 (95% CI 10.6–248)[15] and 614 (95% CI 372–844)[8] and aORs between 34.9 (95% CI 22.4–54.3)[42] and 65.0 (95% CI 16.6–255.0).[17] A large multicenter US cohort study[16] found that for women presenting with placenta previa and prior cesarean deliveries, the risk of accreta was 3%, 11%, 40%, 61%, and 67% for first, second, third, fourth, and fifth or more cesareans, respectively (Table 4). These risks are independent of other maternal characteristics, such as parity, body mass index, tobacco use, and coexisting hypertension or diabetes.[15,37,42] A systematic review and meta-analysis of observational studies found that compared with women with previa and no previous cesarean delivery, women presenting with a previa and three or more prior cesareans have a 15–20-fold increase (3.3%–4% vs 50%–67%) in their risk for PAS disorders.[41] A recent systematic review and meta-analysis of 3889 women with one or more prior cesarean deliveries presenting with placenta previa or low-lying placenta on ultrasound confirmed at delivery found that the incidence of placenta previa accreta was 4.1% in women with one previous cesarean and 13.3% in women with two or more previous cesareans.[44] In general, however, these estimates probably underestimate the risk of recurrence since the invasive forms of PAS disorders will often lead to hysterectomy, and thus prevent subsequent pregnancy.

An Australian case–control study, including 65 cases of PAS disorders and 102 controls matched for coexisting placenta previa, number of previous cesareans, and maternal age found that women with a primary elective cesarean delivery without labor are more likely to develop a PAS disorder in a subsequent pregnancy presenting with placenta previa compared with those undergoing primary emergency cesarean delivery with labor (OR 3.0, 95% CI 1.5–6.1).[22] This is in line with the results of the NOSS study that report an OR of 4.1 (95% CI 2.0–8.1) of having PAS disorders after a first elective cesarean delivery compared with a first emergency cesarean delivery.[45]

A multicenter observational study of 176 women with prior myomectomy and 455 women with prior classical cesarean delivery showed that no PAS disorders (0%, 95% CI 0%–1.98%) occurred in the prior myomectomy group whereas the incidence was 0.88% (95% CI 0.30%–2.19%) in the classical cesarean delivery group compared with 0.19% (95% CI 0.13%–0.27%) in a control group of 13 273 women with a prior low-segmentf transverse cesarean delivery.[46] For those with placenta previa, the OR for PAS disorders in the classical cesarean delivery group was 2.09 (95% CI 0.27–15.33) and when adjusted for maternal age and gestational age at delivery the OR was 0.82 (95% CI 0.10%–6.49%) when compared with the prior low-segment transverse cesarean group.

TABLE 4 Rates of placenta accreta spectrum (PAS) disorders, placenta previa, and hysterectomy by number of previous cesarean deliveries.[a]

No. of previous cesareans	No. of women	Incidence of PAS disorders	Rate of PAS disorders if placenta previa	No. of hysterectomies
0	6201	15 (0.24%)	3%	40 (0.65%)
1	15 808	49 (0.31%)	11%	67 (0.42%)
2	6324	36 (0.57%)	40%	57 (0.9%)
3	1452	31 (2.13%)	61%	35 (2.4%)
4	258	6 (2.33%)	67%	9 (3.49%)
5	89	6 (6.74%)	67%	9 (8.99%)

[a]Modified from Silver et al.[16]

These results suggest that elective cesarean deliveries may be associated with a higher risk of PAS disorders than emergent cesarean delivery and that a prior myomectomy is associated with a very low risk of PAS disorders in subsequent pregnancies. Possible confounding factors include the surgical techniques used for both the cesarean deliveries and myomectomies. In addition, in cases of myomectomy, entry into the uterine cavity during the procedure and size of the myometrial scar may influence the risk of PAS disorders in subsequent pregnancies.

Since the first publication by Ben-Nagi et al.[47] of a case of cesarean scar pregnancy diagnosed in the first-trimester, which subsequently developed into placenta previa accreta, there has been mounting evidence that this condition can be a precursor for PAS disorders. Epidemiologic evidence remains limited to a few retrospective cohort studies.[48–50] Not all scar pregnancies require major surgery or lifesaving hysterectomy at the time of delivery,[51] which suggests that in an undetermined number of cases the scar defect can be large enough to host an entire gestational sac without the villi of the definitive placenta invading into the remaining myometrium or the uterine serosa. As the cervical wall is essentially made up of connective tissue with only 10% of smooth muscle fibers,[12] a cervical scar pregnancy almost always presents with bleeding early in pregnancy and the symptoms of an accreta and non-accreta scar pregnancy are therefore very similar. The diagnosis of PAS disorders can only be confirmed by histopathology, and thus, in case of successful conservative management, it is difficult to be certain that a scar pregnancy is truly accreta.

4 | DEPTH OF VILLOUS INVASION DISTRIBUTION IN PAS DISORDERS

Prenatal evaluation of the depth of placental invasion is essential for planning individual management of women diagnosed with PAS disorders.[52] Despite the fact that around 90% of women diagnosed prenatally with placenta previa accreta in the last 30 years have undergone an elective or emergent cesarean hysterectomy,[44] there are limited data on the depth of villous invasion in these cases. In a recent systematic review of 1078 cases of PAS disorders diagnosed prenatally, fewer than 40% of cohort and case–control prenatal ultrasound studies provide information on the depth of villous invasion.[5] This may be due to limited access to trained perinatal pathologists in most centers delivering women with PAS disorders and the confusion around simple placental retention reported by both clinicians[10] and pathologists[53] as mild forms of PAS disorders, and clinical descriptions of placental tissue appearing under the serosa of an old scar dehiscence at cesarean delivery[3] as abnormally adherent placenta.

Dannheim et al.[54] recently proposed methods of gross dissection, microscopic examination, and reporting of hysterectomy specimens

containing PAS disorders. Previous studies have indicated that PAS disorders can be focal or partial and heterogeneous, mixing adherent and invasive accreta villous tissue.[55–58] In addition, the histopathologic diagnosis of PAS disorders can be very difficult if the surgeon has attempted to remove the placenta during delivery or impossible in cases of conservative management with the placenta left in situ.

Table 5 presents the data from pathologic studies and prenatal diagnosis series of PAS disorders with detailed clinical and histopathologic data on depth of villous invasion.[55–68] In pathologic studies, the distribution of placenta creta, increta, and percreta is 69.5%, 23.7%, and 6.8%, respectively. In prenatal diagnosis series, the incidence of placenta creta is lower (50.7%) and placenta previa higher (25.1%). This observation may be due to the different populations studied, as well as changes in cesarean delivery rates between the 1970s and 2000s. Two studies[58,63] have provided detailed data on the relationship between the depth of villous invasion and the number of previous cesarean deliveries. They noted five placenta creta, one placenta increta, and two placenta previa after one cesarean delivery; seven placenta creta, seven placenta increta, and 11 placenta previa after two cesarean deliveries; and six placenta creta, three placenta increta, and eight placenta previa after more than two cesarean deliveries. A recent systematic review and meta-analysis of 23 cohort studies including a population of 350 939 women from mainland China found a prevalence of placenta creta and placenta increta of 0.48% and 0.23%, respectively. Surprisingly, no cases of placenta previa accreta were reported in that population which suggest issues around the clinical definition of PAS.[69] The prevalence of placenta increta increased with time from 0.3% in 1970–1979 to 0.48% in 2010–2016, and was lower in central geographic regions than in north and south regions and in women living inland compared with those living in coastal areas. The authors attributed these differences in prevalence to higher unplanned pregnancies and surgical termination of pregnancies in coastal cities than in central rural areas.[69] This could also be due to higher demand for elective cesarean deliveries and advanced maternal age in urban areas.[70]

A standardized clinical classification (Table 1) describing and categorizing the different forms of PAS disorders at delivery has recently been proposed.[71] It focuses mainly on the severe forms and it has not been tested prospectively but it provides a good starting point for further prospective epidemiologic studies. Ultrasound imaging is a promising screening tool for PAS disorders[44] and a combination of well-defined ultrasound features and standardized clinical criteria with detailed histopathologic correlation should also be used in future clinical research.[3,5,71]

5 | THE IMPACT OF SURGICAL TECHNIQUES

It has been suggested that surgical techniques used for entering and closing the uterus during cesarean delivery could play a role in the etiology of PAS disorders.[12] For example, single-layer uterine closure versus a multiple overlapping layer type of closure, or locked versus interrupted suturing, or different suture materials could influence the risk of developing PAS disorders in subsequent pregnancies. Overall, single-layer closure compared with double-layer closure of the uterine incision is associated with a reduction in mean blood loss and duration of operative procedure.[29] However, a systematic review[72] has indicated that single continuous locked suture of the cesarean incision may be associated with thinner residual myometrium thickness as evaluated by postoperative ultrasound. A recent systematic review and meta-analysis of nine randomized controlled trials including 3696 participants found a

TABLE 5 Distribution of the different grades of placenta accreta spectrum (PAS) disorders in older case series and more recent cohorts of women with a prenatal diagnosis.

Author (year)	Total no. of cases	No. of placenta creta	No. of placenta increta	No. of placenta percreta
Luke et al.[55] (1966)	21	14	7	0
Weekes et al.[56] (1972)	7	6	0	1
Breen et al.[57] (1977)	40	31	7	2
Morison et al.[58] (1978)	50	31	14	5
Total case series (%)	118	82 (69.5%)	28 (23.7%)	8 (6.8%)
Twickler et al.[59] (2000)	9	3	2	4
Comstock et al.[60] (2004)	15	8	3	4
Woodring et al.[61] (2011)	10	8	1	1
Lim et al.[62] (2011)	9	5	3	1
Cali et al.[63] (2013)	41	15	9	17
Maher et al.[64] (2013)	42	28	13	1
Riteau et al.[65] (2014)	26	16	0	10
Algebally et al.[66] (2014)	32	16	12	4
Satija et al.[67] (2015)	10	3	4	3
Kumar et al.[68] (2016)	9	1	2	6
Total cohorts with a prenatal diagnosis (%)	203	103 (50.7%)	49 (24.2%)	51 (25.1%)

TABLE 6 Recommendations for the evaluation of epidemiological data on placenta accreta spectrum (PAS) disorders.

Recommendations	Resource settings	Quality of evidence and strength of recommendation
The recent increase in the incidence and prevalence of PAS disorders is a consequence of the rise in cesarean deliveries over the last two decades	All	High and Strong
A cesarean delivery scar increases the risk of placenta previa in subsequent pregnancies	All	High and Strong
A myomectomy scar increases the risk of PAS disorders in subsequent pregnancies	High	Low and Weak
Minor surgical procedures such as uterine curettage can lead to PAS disorders in subsequent pregnancies	All	Low and Weak
Women with a previous history of cesarean delivery presenting with a low-lying placenta or placenta previa in the second trimester of pregnancy have become the largest group of women with the highest risk of PAS disorders	All	High and Strong
Women should be informed that their risk of PAS disorders increases with each cesarean delivery	All	High and Strong
Women who request a pre-labor elective cesarean delivery should be informed that their risk of developing PAS disorders is higher than after emergency/emergent cesarean delivery	High	Low and Weak
Women presenting with cesarean scar pregnancy in the first trimester of pregnancy should be informed of the high risk of invasive placentation and/or major placenta previa later in pregnancy and should be offered the option of terminating the pregnancy	High	Moderate and Strong
The use of standardized protocol and terminology for both the clinical diagnosis and histopathological confirmation of PAS disorders is essential to obtaining new and more accurate epidemiological data	All	High and Strong

similar incidence of uterine scar defects in women who had a single-layer compared with double-layer closure (RR 0.77, 95% CI 0.36–1.64).[73] Outcomes were considered inaccurate because the studies reviewed had included relatively few patients and events (five trials with 350 participants). Nonetheless, these data suggest that type of uterine closure has little influence on uterine scar healing and thus less impact on PAS disorders than emergent versus elective cesarean delivery.[22]

A case–control study of 98 women with one or more prior cesarean deliveries presenting with placenta previa including 38 PAS disorders found no difference in single-layer versus double-layer closure in the incidence of PAS disorders.[74] Multivariate logistic regression analysis showed that continuous suture was associated with a higher risk of PAS disorders than interrupted sutures (aOR 6.0, 95% CI 1.4–25.2). A retrospective case–control study of 53 cases and 157 controls also found that the use of monofilament suture for hysterotomy closure in prior cesarean delivery reduces the risks of having placenta previa (aOR 0.26, 95% CI 0.08–0.80) and thus PAS disorders in future pregnancies.[75] More prospective multicenter studies are required to evaluate the impact of surgical techniques used during cesarean delivery on the risks of PAS disorders in subsequent pregnancies.

6 | RECOMMENDATIONS

Recommendations for the evaluation of epidemiological data on PAS disorders are given in Table 6.

CONSENSUS PANEL

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CONFLICTS OF INTEREST

The authors have no conflicts of interest.

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