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Genetic Predisposition in Placenta Accreta Spectrum: A Case Report Highlighting Familial Risk and Diagnostic Implications

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Abstract

Background: Placenta accreta spectrum (PAS) represents a continuum of abnormal placental adherence resulting from defective decidualisation and excessive trophoblastic invasion. While rising caesarean delivery rates explain most cases, emerging data suggest that genetic predisposition may also contribute.

Case: A 37-year-old multiparous woman presented at 36 + 5 weeks' gestation with preterm rupture of membranes and vaginal bleeding with a known, low-lying placenta. Emergency lower-segment caesarean section revealed focal adherence of the placenta to the anterior uterine wall, leading to postpartum haemorrhage of 4.4 L. Haemostasis was achieved using sequential uterotonic therapy, tranexamic acid, and intrauterine balloon tamponade (Bakri). Histopathology confirmed placenta increta. Subsequent counselling revealed a strong family history of abnormal placental implantation in first-degree relatives.

Discussion: The case exemplifies the challenges of diagnosing PAS antenatally and highlights potential familial clustering suggestive of heritable susceptibility. Emerging molecular data implicate dysregulation of extracellular matrix remodelling, matrix metalloproteinases (MMPs), and angiogenic pathways (TGF-β, VEGF, PGF, HIF-1α) as possible mechanisms that bridge genetic and acquired risk factors.

Conclusion: Recognising family history as a potential independent risk factor may refine future antenatal screening strategies. Integration of genetic history with imaging surveillance could enable earlier detection, reduce morbidity, and guide multidisciplinary planning for delivery in appropriately equipped centres, ultimately improving maternal outcomes.

Keywords: Placenta Accreta Spectrum; Placenta Increta; Genetic Predisposition; Extracellular Matrix; Angiogenesis; Intrauterine Balloon Tamponade; Massive Haemorrhage Protocol

Abbreviations: PAS: Placenta Accreta Spectrum; MMPs: Matrix Metalloproteinases; SROM: Spontaneous Rupture of Membranes; CTG: Cardiotocography; GBS: Group B Streptococcus; LUSCS: Lower-Segment Caesarean Section; MHP: Massive Haemorrhage Protocol; ECM: Extracellular Matrix; BM: Basement Membrane; ECM: Enzymes that Mediate; MRI: Magnetic Resonance Imaging; AFP: Alpha-Fetoprotein; HCG: Human Chorionic Gonadotropin; VEGF: Vascular Endothelial Growth Factor; PlGF: Placental Growth Factor; PGF: Placental Growth Factor; MDT: Multidisciplinary Team; PPH: Postpartum Haemorrhage; IUBT: Intrauterine Balloon Tamponade

Introduction

Placenta accreta spectrum (PAS) represents a profound failure of normal placental implantation, characterised by the invasion of placental villi beyond the decidua basalis and into or through the myometrium [1]. This spectrum includes placenta accreta (villi adhering to the myometrium), placenta increta (villi invading the myometrium), and placenta percreta (villi penetrating the uterine serosa, sometimes involving adjacent organs such as the bladder) [2]. It remains one of the most serious complications in modern obstetrics.

The incidence of PAS has increased markedly from approximately 1 in 30,000 deliveries in the 1960s to as high as 1 in 200 in recent surveillance studies, reflecting an almost unprecedented rise in obstetric morbidity [3]. This trend correlates closely with the global escalation in caesarean delivery

rates, as each successive procedure further compromises the endometrial–myometrial interface [4]. The risk of placenta accreta rises cumulatively, reaching up to 61 % after three or more caesarean sections. Other established risk factors include placenta praevia, advanced maternal age, and prior uterine surgery (e.g., myomectomy) [5]. However, emerging translational research points to a more complex aetiology, suggesting that genetic predisposition and familial clustering may contribute in some cases [6]. This implies that PAS may not always be an acquired, iatrogenic condition, but could also reflect an inherited genetic predisposition of implantation biology.

The central anatomical defect in PAS is the absence or attenuation of the decidual layer and its basement membrane, which normally act as barriers against excessive trophoblastic invasion [7]. Diagnosis is often first suspected during routine antenatal ultrasound surveillance, though up to 50 % of cases remain undetected before delivery. This case report describes an instance of unsuspected PAS presenting as severe postpartum haemorrhage. It emphasises the continuing need to refine diagnostic criteria and broaden our understanding of risk factors to improve antenatal detection and management.

Case Discussion

A 37-year-old G3P1 woman employed as a disability support worker presented to Mildura Base Public Hospital following preterm spontaneous rupture of membranes (SROM) at 36 + 5 weeks' gestation. On arrival, she had fresh vaginal bleeding but remained haemodynamically stable. Uterine tightenings occurred every two to three minutes, and the foetal heart rate tracing on cardiotocography (CTG) was normal. Abdominal examination confirmed a cephalic presentation with the head three-fifths palpable above the pelvic brim. A sterile speculum examination revealed a closed cervix and blood-stained liquor. This was her first episode of bleeding.

Antenatal records documented good engagement with clinical services. Ultrasound at 35 weeks had shown a grade II left-lateral placenta praevia, positioned 6 mm from the internal cervical os. There was no evidence of abnormal lacunae, increased vascularity, or loss of the hypoechoic zone, findings consistent with an uncomplicated praevia. The patient was Rhesus-positive. Her pregnancy was complicated by gestational diabetes mellitus diagnosed at 28 weeks, managed with diet, exercise, and metformin 1 g twice daily, maintaining good glycaemic control. At 34 weeks, she developed pregnancy-induced hypertension that did not require pharmacologic treatment. Her body mass index was under 30 kg/m^2. She was a light smoker, and her group B streptococcus (GBS) status was unknown. Her previous pregnancy had been an uncomplicated assisted vaginal delivery using forceps for a large-for-gestational-age infant. Before this presentation, she had been scheduled for an elective lower-segment caesarean section (LUSCS) with salpingectomy at 38 weeks.

At admission, intravenous fluids were commenced, and blood was sent for cross-matching and full blood count. Given the combination of uterine activity, vaginal bleeding, and SROM close to term, the multidisciplinary team-including paediatric and anaesthetic specialists-decided to proceed with emergency caesarean section. Spinal anaesthesia was performed without complication, and an indwelling catheter drained clear urine. A Pfannenstiel incision provided access to the lower uterine segment. The bladder was reflected, and a transverse uterine incision was made; no abnormal vessels were seen. The placenta did not obstruct entry. Amniotic fluid was clear and non-offensive. The infant was delivered easily using gentle fundal pressure and forceps assistance. Cord clamping occurred after 30 seconds, and cord gas samples were collected. Carbetocin 100 µg IV was administered for routine third-stage management.

Controlled cord traction and fundal massage allowed partial placental delivery-approximately 40–50 % separated and exteriorised-but the remainder remained firmly adherent to the anterior and lateral uterine walls. The anaesthetic team was alerted to the likelihood of abnormal placental adherence. Ergometrine 250 µg IV and 250 µg IM were given, together with tranexamic acid 1 g IV and misoprostol 400 µg buccally. An infusion of 40 U oxytocin in 1 L of normal saline was started at 250 mL/hour.

After ten minutes, piecemeal digital extraction and gentle uterine curettage were performed to remove the remaining tissue, but brisk bleeding persisted. The massive haemorrhage protocol (MHP) was activated. The patient received 4 units of packed red blood cells, 2 units of fresh-frozen plasma, 1 unit of platelets, and 10 units of cryoprecipitate. A Bakri balloon was inserted through the uterine incision and inflated gradually until minimal bleeding was observed. The uterus was closed in layers, and a pressure dressing was applied. Blood pressure remained stable, though tachycardia developed (heart rate up to 140 bpm). Urine output was minimal but clear. Estimated blood loss was 4.4 L.

Postoperatively, the patient was managed in the intensive care unit for 24 hours. Vaginal loss and drainage from the Bakri catheter remained minimal. She developed hypertension managed initially with prazosin, IV clonidine, and magnesium sulphate, then with labetalol 200 mg twice daily after transfer to a tertiary partner hospital. The Bakri balloon was removed on day 2; uterine tone was satisfactory, and vaginal loss remained within normal limits. An ultrasound at 3 weeks postpartum showed a bulky, anteverted uterus (470 cm^3) containing an echogenic mass measuring 60 × 56 × 45 mm, suggestive of retained placental tissue. Histopathological analysis confirmed placenta increta, characterised by chorionic villi directly adjacent to myometrial fibres without intervening decidua. During postpartum counselling, the patient reported a previously unsolicited family history of abnormal placentation-both her sister and mother had experienced complicated deliveries attributed to adherent placentae.

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Discussion

Placenta accreta spectrum (PAS) is a serious pregnancy complication encompassing placenta accreta, increta, and percreta. In a normal pregnancy, the placenta remains confined within the decidua basalis, a specialised endometrial layer that regulates trophoblastic invasion. The decidua's extracellular matrix (ECM) provides a controlled environment for implantation 97). The basement membrane (BM), a dense ECM structure composed primarily of collagen IV, laminin, and perlecan, separates the foetal trophoblast from the maternal decidua and myometrium [8]. Acting as a biological "gatekeeper," it restricts trophoblastic invasion to the appropriate depth during early pregnancy [9]. In PAS, disruption of this decidual–myometrial barrier-most commonly from uterine scarring after caesarean delivery-allows direct contact between invasive extravillous trophoblasts and the myometrium [10]. The result is abnormal adherence and a high risk of massive postpartum haemorrhage with significant maternal morbidity and mortality [11].

Dysregulation of matrix metalloproteinases (MMPs)-enzymes that mediate ECM remodelling and normal trophoblast migration-has been implicated in both insufficient invasion (as in pre-eclampsia) and excessive invasion seen in PAS [12]. Altered expression or polymorphisms in MMP genes and angiogenic factors may predispose certain women to abnormal placental attachment, bridging the gap between acquired (scar-related) and inherent biological risk [13]. Timely antenatal diagnosis remains central to reducing morbidity, yet up to 50% of PAS cases remain unsuspected before delivery [14]. Ultrasound remains the first-line screening tool, with characteristic findings including loss of the hypoechoic zone between placenta and myometrium [15], the presence of irregular vascular lacunae [16], and abnormal serosal hypervascularity on Doppler [17]. However, diagnostic accuracy is highly operator-dependent, and features may be subtle or absent in focal or posterior cases [18].

Magnetic resonance imaging (MRI) is reserved for equivocal ultrasound results, posterior placenta praevia, or suspected invasion of adjacent organs such as the bladder [19]. MRI provides superior soft-tissue contrast, enabling better assessment of invasion depth, although its cost and limited availability limit widespread use [20]. Given these challenges, attention has turned to biochemical and molecular biomarkers to supplement imaging. Several analytes, including alpha-fetoprotein (AFP), human chorionic gonadotropin (hCG), vascular endothelial growth factor (VEGF), and placental growth factor (PlGF), show modest associations with PAS, reflecting underlying placental dysfunction [21]. However, no single biomarker or panel has yet achieved adequate sensitivity and specificity for population-level screening [22].

Evidence increasingly suggests that genetic factors may contribute to PAS susceptibility [6]. Familial clustering-where affected women report first-degree relatives with similar complications-supports an inherited predisposition [23]. The family history in this case, involving both the patient's mother and sister, aligns with this observation [24]. Genetic susceptibility is thought to involve subtle variations or dysregulation in genes controlling trophoblast invasion, ECM remodelling, and angiogenesis. MMPs, particularly MMP-7 and MMP-9, facilitate controlled trophoblast migration, while their inhibitors (TIMPs) prevent excessive invasion [25]. Aberrations in pathways such as transforming growth factor-beta (TGF-β) may compromise decidual integrity and increase PAS risk [26].

Healthy placental vascularisation is regulated by genes controlling angiogenesis, such as placental growth factor (PGF) and VEGF [27]. Polymorphisms in these or in transcriptional regulators, such as hypoxia-inducible factor-1 alpha (HIF-1α), can create a hyperangiogenic environment, promoting deep trophoblast invasion rather than normal vascular development [28]. Recent advances, including single-cell RNA sequencing and spatial transcriptomics, have identified distinct gene expression profiles in PAS placental tissue where susceptibility to abnormal growth may be related to dysregulation [29]. Despite these discoveries, however, no definitive genetic marker has been validated for clinical use.

Recognition of familial risk depends on history and may offer a practical, cost-effective way to improve surveillance. Enhanced antenatal screening for these women could include earlier ultrasound assessment, serial imaging, and MRI when indicated [30]. When PAS is suspected, care should be coordinated through tertiary centres with access to multidisciplinary expertise and the ability to activate a massive haemorrhage protocol (MHP) [31]. Optimal management requires collaboration between maternal–fetal medicine specialists, anaesthetists, interventional radiologists, and surgical teams [32].

The optimal timing for delivery in confirmed or suspected placenta accreta spectrum (PAS) is generally between 34 + 0 and 35 + 6 weeks' gestation [33]. This interval balances foetal maturity against the rising maternal risk of spontaneous labour or catastrophic haemorrhage. The gold standard for definitive management is a planned peripartum Caesarean hysterectomy, in which the foetus and entire uteroplacental unit are delivered en bloc with the placenta left in situ, thereby minimising intraoperative blood loss and allowing controlled haemostasis under optimal surgical conditions [34].

Fertility-sparing approaches such as leaving the placenta in situ for gradual resorption are reserved for exceptional cases in haemodynamically stable patients desiring future fertility. However, such management carries substantial risks of delayed haemorrhage, sepsis, and the eventual need for interval hysterectomy, and should only be attempted in tertiary centres with close multidisciplinary follow-up [35]. Regardless of management strategy, the multidisciplinary team (MDT) must be prepared to activate a massive haemorrhage protocol (MHP)

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at any stage. This ensures the rapid provision of blood products, including packed red cells, plasma, platelets, and cryoprecipitate, consistent with contemporary damage-control resuscitation principles [36,37].

The index case exemplifies the unpredictable nature of unsuspected PAS, recognised intraoperatively following intractable postpartum haemorrhage (PPH). In such emergencies, maternal survival depends on swift, coordinated pharmacological, mechanical, and surgical intervention. Initial pharmacologic control begins with rapid, sequential administration of uterotonic agents-oxytocin, ergometrine, and misoprostol supported by the antifibrinolytic tranexamic acid to enhance clot stability [38]. When medical therapy fails and a firmly adherent placenta is encountered, mechanical measures such as piecemeal digital extraction or cautious uterine curettage may be required to remove residual tissue; however, these carry a significant risk of continued bleeding [37,39].

Intrauterine balloon tamponade (IUBT) is a valuable adjunct in such situations. As demonstrated in this case, insertion of a Bakri balloon provides direct hydrostatic compression on the placental bed, achieving rapid, temporary haemostasis. This permits physiological stabilisation and serves as a bridge to definitive surgical management or safe transfer to a tertiary centre [40]. If bleeding had persisted despite these measures, emergency hysterectomy would have been the definitive, life-saving intervention [41].

In this patient, successful stabilisation and uterine preservation demonstrate the effectiveness of structured, tiered haemorrhage control and highlight the pivotal role of IUBT in contemporary emergency management of PAS [42]. This outcome demonstrates how early activation of MHP protocols and coordinated multidisciplinary response can enable effective resuscitation even in high-risk cases. It also reinforces the importance of preparedness and systems-level coordination, particularly in regional or resource-limited settings, where timely adherence to evidence-based algorithms can be life-saving.

Conclusion

This case exemplifies the complexity and unpredictability of placenta accreta spectrum (PAS), particularly when up to half of affected women may escape antenatal diagnosis. Even with routine surveillance, PAS can first present as an obstetric emergency, demanding rapid recognition and coordinated multidisciplinary action. Beyond the immediate clinical challenge, the case raises the possibility of an inherited predisposition to abnormal placentation. Recognising family history as an independent risk factor may enhance early identification of at-risk women and support more individualised models of antenatal and birthing care.

Future research should focus on defining and validating specific genetic markers that confer susceptibility to PAS and evaluating their predictive accuracy alongside imaging and biochemical screening. Integrating genetic findings with established sonographic criteria may improve diagnostic precision, preparedness, and outcomes, ultimately reducing the significant maternal morbidity and mortality associated with this disorder. Finally, the case underscores the enduring importance of multidisciplinary collaboration and clearly defined referral networks. Ensuring that regional and rural hospitals maintain efficient pathways to tertiary services is fundamental to equitable, evidence-based management of PAS and to advancing maternal safety in modern obstetric practice.

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