**Prenatal Diagnosis and Perinatal Management of Placenta Previa Accreta: Past, Present and Future**

Min-Min Chou*

Division of Maternal-Fetal Medicine, Department of Obstetrics and Gynecology, Taichung Veterans General Hospital, Taichung, Taiwan.

**SUMMARY**

The reported incidence of placenta accreta varies widely in the English literature, from 1:540 to 1:93,000 deliveries. In Taiwan, the quoted incidence ranges from 1:625 to 1:1,652 deliveries in tertiary medical centers. Massive obstetric hemorrhage is still the leading cause of pregnancy-related death, and placenta previa accreta remains one of the major predisposing factors. With the increasing rates of cesarean delivery and uterine curettage for abortion, both placenta previa and accreta are steadily increasing in frequency. Therefore, more cases of placenta previa accreta can be expected in obstetric practice. In several recent series, placenta accreta has emerged as the major indication for peripartum hysterectomy, accounting for 40% to 60% of cases. It has, therefore, become a challenging problem of increasing clinical significance in obstetrics.

**Key Words:** color power Doppler ultrasound, gray-scale ultrasound, placenta previa accreta, three-dimensional color power Doppler ultrasound

**Introduction**

The reported incidence of placenta accreta varies widely in the English literature, from 1:540 to 1:93,000 deliveries [1]. In Taiwan, the quoted incidence ranges from 1:625 to 1:1,652 deliveries in tertiary medical centers [2–5]. Of patients with this abnormality, the reported frequency of placenta accreta is approximately 84%, of placenta increta is 13%, and of placenta percreta is 3% [6]. However, at Taichung Veterans General Hospital (TVGH), the incidence is 1:625 deliveries, and the frequency of placenta accreta is 53.2%, of placenta increta is 29.8%, and of placenta percreta is 17%. The higher proportion of placenta increta/percreta in TVGH compared with that reported in the literature reflects the hospital’s status as a high-risk referral tertiary medical center. Massive obstetric hemorrhage is still the leading cause of pregnancy-related death, and placenta previa accreta remains one of the major predisposing factors. With the increasing rates of cesarean delivery and uterine curettage for abortion, both placenta previa and accreta are steadily increasing in frequency. Therefore, more cases of placenta previa accreta can be expected in obstetric practice. In several recent series, placenta accreta has emerged as the major indication for peripartum hysterectomy, accounting for 40% to 60% of cases [7]. It has, therefore, become a challenging problem of increasing clinical significance in obstetrics.

**Placenta Previa Percreta: A Matter of Life and Death**

Placenta previa percreta is a rare and possibly catastrophic obstetric condition. It is the most dangerous form of invasive placentation because it is potentially life threatening and poses a serious risk for spontaneous
Anecdotal Case Reports in Taiwan

Liu et al reported undiagnosed placenta percreta causing uterine rupture with fatal hemoperitoneum at 22 weeks of gestation in an in vitro fertilization case (personal communication, 1986). Another patient with placenta previa percreta and bladder involvement survived after two major surgical procedures and massive blood transfusions at 39 weeks of gestation despite total estimated blood loss (EBL) of 47,000 mL. Lu et al reported a similar case with EBL of 25,000 mL at 28 weeks’ gestation [2]. Recently, Shih et al [9] and Chen et al [10] reported two multipara cases with placenta previa increta/percreta in which hysterectomy was performed at 15 weeks of gestation due to episodes of antepartum hemorrhage with massive blood loss of 12,000 mL and 5,450 mL, respectively, during surgery. These two case reports showed that even when hysterectomy was performed in early pregnancy, the surgical procedure was bloody and problematic. Surgical access to the lower uterine blood supply demands preliminary separation of the densely adherent bladder from the lower uterine segment, compromised by scarring from previous operations and friable and well-vascularized invading placental tissue. This shows that life-threatening bleeding may ensue from difficult surgery for placenta previa percreta, even though the diagnosis has been made in the first or early second trimesters and well-planned surgery is performed remote from term.

These case reports raised concern about the need to promote the importance of accurate early diagnosis of placenta previa accreta and the methods for achieving appropriate perinatal management because most obstetric practitioners lack the expertise to achieve a sufficient standard of care.

Prenatal Ultrasoundographic Diagnosis of Placenta Previa Accreta

Ultrasonography (US) is a useful tool in screening for placenta previa accreta. Previous reports have emphasized B-mode gray-scale US findings, such as loss of the normal subplacental anechoic zone, pulsatile flow into sonolucent placental vascular lakes, abnormal bladder–uterine wall interphase with thinning, and disruption and/or focal nodular projections of the linear hyperechoic boundary echo (Figure 1A) [7,11–14]. These features are adequate to evaluate most abnormally adherent placentas.

Traditionally, the location of the abnormally adherent placenta determines which imaging modality is best suited for the most specific diagnosis. Most authors recommend a transabdominal approach to screen for placenta previa accreta through a semidistended bladder because anterior placentas are easier to evaluate than posterior placentas due to decreased penetration and acoustic shadowing from the fetus. In cases of posterior placenta previa, magnetic resonance imaging appears to provide a more confident diagnosis. Other authors have utilized a transvaginal technique with either an empty or semidistended bladder in the first or early second trimester. For patients with an anterior placenta previa and a suspicious area outside the range of the transvaginal probe, transabdominal ultrasound is the best method of diagnosis. Therefore, at TVGH, we use the transabdominal approach to diagnose placenta previa accreta in the second and third trimesters. Transvaginal scanning with the bladder semidistended to provide better delineation of the uterine serosa–bladder wall boundary zone is most suitable for the first trimester scan because abnormal trophoblastic development in placenta previa accreta with associated hypervascularity can occur very early after implantation (Figure 2).

Finberg and Williams found that gray-scale US had a sensitivity of 93% (14/15) and a specificity of 79% (15/19) in the diagnosis of placenta accreta [14]. Others have emphasized color Doppler imaging (CDI) and power Doppler US as very useful adjuncts in diagnosis [15–22]. The CDI criteria suggestive of placenta previa accreta include: diffuse lacunar flow pattern showing diffusely dilated vascular channels scattered throughout the whole placenta and the surrounding myometrial or cervical tissues, and high-velocity pulsatile venous-type flow in the sonolucent vascular spaces; focal lacunar flow pattern showing irregular sonolucent vascular lakes with turbulent lacunar flow distributed regionally or focally within the intraparenchymal placental area; interface hypervascularity with abnormal blood vessels...
linking the placenta to the bladder, with low-resistance arterial blood flow; markedly dilated peripheral subplacental vascular channels with pulsatile venous-type flow over the uterine cervix; and no subplacental vascular signal in the areas lacking the peripheral subplacental hypoechoic zone (Figure 1B) [21]. Characteristic CDI observations in patients who are not thought to have accreta include: discrete branching of surface chorionic arteries and intraplacental villous arteries clearly visualized within the homogeneous placental substance, with typical flow velocity waveforms; centrocotyledonary sonolucent avillous cavities containing a non-pulsatile low-velocity venous flow (<8 cm/s) pattern identified by real-time imaging; and normal subplacental venous complex with non-pulsatile low-velocity venous blood flow waveforms [21]. The reported sensitivity of two-dimensional (2D) CDI in diagnosing placenta previa accreta ranges between 82.4% and 100%, and the specificity ranges between 92% and 96.8% [19,21].

Therefore, gray-scale US and 2D CDI are the primary diagnostic screening tools. However, neither modality predicts the degree of bladder invasion in placenta percreta nor gives an accurate assessment of the scope of quantitative uteroplacental neovascularization. At TVGH, we have attempted to use three-dimensional (3D) color power Doppler imaging to assess quantitative uteroplacental neovascularization and predict the extent of bladder invasion in placenta previa increta/percreta [23,24]. The true value of 3D CDI and angiohistogram imaging lies in the ability to allow screening through the targeted region of interest and surrounding the spatial vascular network architecture, and to simultaneously analyze aberrant vasculature with different visual perspectives from sagittal, coronal, and axial scanning planes. Niche-mode display has been used to demonstrate the aberrant uteroplacental vessels over the anatomic area of interest. The stored volume is resliced in a number of parallel planes until the area of neovascularization is detected within the uterine serosa-bladder junctional zone. These viewing planes can be arbitrarily manipulated to better delineate the exact location and depth of bladder invasion by the aberrant

Figure 1. (A) Transabdominal gray-scale scan showing multiple large irregular sonolucent spaces traversing the entire placenta. (B) Conventional 2D color Doppler imaging showing abnormal lacunar flow pattern. (C) 3D color power Doppler imaging further defines the abnormal uteroplacental hypervascularity. B = bladder; H = head; P = placenta.
placental–myometrial vessels [23–25]. Since the mortality and morbidity associated with placenta previa accreta increase significantly with the degree of bladder invasion, correct prenatal diagnosis is crucial for appropriate management and surgical planning. Hence, 3D CDI may be a complementary tool to improve diagnostic confidence in characterizing the exact site, extent of involvement, and depth of aberrant vessel invasion throughout the object of interest by providing a more objective method; it is helpful for aiding clinical decision making and guiding appropriate management plans [23–25].

Recently, at TVGH, we have studied the clinical application of 3D imaging for virtual and augmented reality in placenta previa accreta to the planning and guidance of perinatal management. The focus was on the introduction and validation of new maternal–fetal medical image analysis and image-guided interventional methodology into obstetric practice. Modeling and simulation of 3D in uteroplacental–fetal and maternal bladder spatial anatomic atlases based on tomographic images play an increasingly important role in appropriate planning of interventions and simulation of surgical procedures using 3D models as surgical simulators. 3D US imaging provides accurate and quick detection of abnormal uteroplacental neovascularization associated with instructive visual imaging effect. At TVGH, parents with placenta accreta are provided with clear “photographic” images of uteroplacental hypervascularity disorders so that the obstetrician can evaluate the angioarchitecture at different angles, giving a clear “plastic” impression of the severity of the placenta previa accreta to the parents (Figure 1C).

Finally, it must be emphasized that the three US screening techniques for antenatal diagnosis of invasive placenta (gray-scale, 2D CDI and 3D color power Doppler) appear to be complementary rather than competitive, and are not mutually exclusive.

**Diagnostic Pitfalls**

Bladder varices, possibly due to neovascularized vessels from previous cesarean deliveries, are easily mistaken for abnormal bladder–uterine serosa interface hypervascularity assumed to be placenta accreta [21]. Therefore, in patients with findings suggestive of interface hypervascularity, CDI must clearly demonstrate that there are abnormal blood vessels linking the placenta to the bladder. Posterior and lateral uterine wall invasion are difficult to evaluate with US, particularly in twin pregnancies. CDI diagnostic errors are often due to shadowing by fetal parts and separate placenta location.

Therefore, Levine and colleagues suggest that in patients with posteriorly implanted placenta at-risk for accreta, magnetic resonance imaging should be used when US cannot rule out the presence of accreta [19]. Other diagnostic pitfalls include aberration of venous drainage from the placenta, which is a rare condition. On grayscale imaging, blood is observed moving in the markedly dilated subplacental vascular channels in a sluggish manner. Power Doppler reveals spiral arteries crossing the lakes to the fetal cotyledons. Failure of the draining process may have been brought about by local venous obstruction or perhaps varicosities (Figure 3).

The placenta in abdominal pregnancy and rudimentary horn pregnancy should also cause concern [21]. Abdominal pregnancy is considered a form of placenta accreta because the placenta does not deliver spontaneously. According to Benirschke [26], the placental floor lacks decidua and a large vascular supply supports the intervillous circulation. CDI studies of one patient with term abdominal pregnancy show placental tissue impinging on the urinary bladder simulating placenta previa; placental hypervascularization was detected by transabdominal color Doppler US [21]. These sonographic features confirm that extraordinary local vascular development of placental circulation occurs in abdominal pregnancy. Therefore, the sonographer should carefully search for a displaced uterus elsewhere if placenta previa accreta is suspected.

Placenta accreta in a rudimentary horn pregnancy was reported for the first time by Heinonen in 1983 [27]. In a MEDLINE review by Basbug et al [28], of the...

---

**Figure 3.** A rare condition, aberrant venous drainage from the uncomplicated placenta previa, can be mistaken for placenta previa accreta. Color Doppler imaging shows blood moving in markedly dilated subplacental vascular channels in a sluggish manner and spiral arteries crossing the vascular lakes to the fetal cotyledons. Failure of the draining process may have been brought about by local venous obstruction or perhaps varicosities. B = bladder; CX = cervix; P = placenta.
51 cases with rudimentary horn pregnancies, seven were associated with placenta accreta (13.7%), suggesting that rudimentary horn pregnancy is more likely to be associated with placenta accreta than intrauterine pregnancy. In a study at TVGH, we encountered one case of term pregnancy in the rudimentary horn of an unicornuate uterus. The characteristic CDI features were multiple areas of placenta implantation with a diffuse-type lacunar flow pattern. [21,29].

**Management of Placenta Previa Accreta: Operative and Conservative Strategies**

What perinatal management strategies should be adopted when prenatal diagnosis has been made? Ideally, delivery should be elective at the gestational age of proven lung maturity. Often, however, bleeding will prompt earlier surgical intervention. Therefore, the timing of cesarean hysterectomy should be decided according to hemodynamic morphologic features found on CDI. It may be appropriate to consider elective cesarean hysterectomy at 34 weeks’ gestation or even earlier, irrespective of fetal lung maturity, in patients with diffuse lacunar flow pattern, particularly associated with strong evidence of extensive placenta increta/percreta and episodes of antepartum hemorrhage, in order to decrease the risk of maternal morbidity and mortality associated with emergency surgery [21]. Surgical strategies for managing placenta percreta in late pregnancy have been plagued by massive bleeding, massive transfusion, and immediate or delayed hysterectomy. However, for patients with a focal lacunar flow pattern, uneventful cesarean hysterectomy can usually be performed at more advanced gestation (35–37 weeks’ gestation). Further studies are necessary to validate these suggestions.

If CDI shows a diffuse lacunar flow pattern and bladder–uterine serosa interface hypervascularity, urologic assessment, including cystoscopy and pre- or intraoperative placement of a ureteral stent, should be considered. Cesarean hysterectomy as described by Catanzarite et al [7] and Pelosi et al [30] should be used. If large engorged blood vessels are seen within the visceral peritoneum covering the lower uterine segment, extending to the top of the bladder, a high fundal classical incision should be performed at least 2 cm above the upper margin of adherent placenta to avoid injuring the hypervascular region in the friable lower uterine segment. At TVGH, we attempted to remove the abnormally adherent placenta in one case because of lack of clinical experience; this caused massive vaginal bleeding at more than 800 mL/minute. The uterine blood invisibly drained away from the vagina, down the operating table, and the delivery floor was flooded with blood, causing a life-threatening hemorrhagic complication [21]. This painful experience showed that if the combination of CDI findings and clinical uterine hypervascularity appearance during surgery is highly suggestive of placenta previa accreta, the placenta should not be removed because, if the involved cotyledon, particularly if located in the friable lower segment, is either pulled off the myometrium with perhaps somewhat excessive bleeding from that part of the implantation site or is torn from the placenta, bleeding will increase immediately or later [21]. During hysterectomy, if indicated, we sometimes use intentional cystotomy at TVGH and place two fingers downward to guide the dissection of the adherent bladder wall and apply the Penrose drain tourniquet technique to compress the lower uterine segment in an attempt to reduce blood flow to the uterus. Furthermore, a ureteral stent passage is often made preoperatively (via cystoscopy) or intraoperatively (via cystotomy) to facilitate recognition of the ureters, potentially preventing injury to the ureter and the trigone of the bladder.

Prophylactic perioperative balloon occlusion and embolization of the internal iliac arteries can be used to attempt to reduce intraoperative blood losses and facilitate surgery in patients with an ultrasonographic diagnosis of placenta previa percreta associated with extensive neovascularization in late viable gestation. It is essential to establish a stable occluding catheter position before entering the operating room. The balloons are only inflated after classical cesarean delivery, and it is mandatory to perform the embolization before deflating the balloon, to obtain better control of postpartum hemorrhage caused by extensive pelvic collateral circulation [31,32]. Another technique used to reduce intraoperative blood loss is two-stage conservative management (hysterectomy at 2–4 weeks postpartum) [33].

From the clinical standpoint, early antepartum recognition of placenta percreta with bladder involvement is extremely important because patients can receive appropriate counseling regarding the potential risk of uterine rupture with fatal hemoperitoneum or life-threatening hemorrhage in late pregnancy. Timely pregnancy termination before fetal viability in well-selected severe cases with no further desire for childbearing is appropriate because of decreased morbidity and mortality.

Recently, transarterial embolization (TAE) has been chosen at TVGH rather than balloon occlusion catheterization, to attempt to decrease intraoperative blood loss and facilitate surgery in patients with ultrasono-
graphic diagnosis of placenta previa percreta in early gestation. TAE helps to localize the site of hyper-vascularity. Embolization can also be performed as selectively as possible in the uterine arteries and other supplying anastomotic new blood vessels when necessary (Figure 4). Among six patients with placenta increta/percreta undergoing hysterectomy with preoperative prophylactic internal iliac artery embolization (IIAE), the procedure was associated with an apparent reduction in mean EBL (1,767 ± 1,213 mL) compared with two non-embolized patients (4,950 ± 1,061 mL; p = 0.0442). Patients undergoing embolization required fewer transfusions (4.33 ± 4.08 vs 16.50 ± 4.95; p = 0.043) [6]. This limited experience is encouraging because of the apparent reduction in operative blood loss after prophylactic IIAE. These preliminary findings are based on a small number of patients and, therefore, further investigation is needed to determine the effectiveness of this procedure.

**Intrapartum and Postpartum Management of Abnormally Residual Adherent Placenta**

As placenta accreta might not be diagnosed antepartum or during labor, especially when no risk factors are present, adequate preparations cannot be made. The intrapartum and postpartum management of adherent placenta remains controversial. Potential complications include hemorrhage, infection, and persistent placental tissue. Although hysterectomy remains the recommended treatment for most patients, uterine conservation is justified in certain well-selected, non-bleeding cases where future child-bearing is strongly desired. Such conservative management options may include adjuvant methotrexate (MTX) treatment, curettage, tamponade of the placental implantation site with inflated Foley catheter bags, local excision, and repair or oversewing of the implantation site.

If placenta accreta is diagnosed at the time of cesarean section, two-stage conservative management intrapartum, with the abnormally adherent placenta left in situ after delivery, particularly in conditions of invasive placentation with extension into the bladder or gastrointestinal tract and when the operating obstetrician lacks experience in pelvic dissection, is thought to be appropriate to avoid the risk of severe hemorrhage at the time of cesarean delivery. Hysterectomy, bladder wall resection, and reimplantation of ureters are made technically easier, less bloody, and are achieved with fewer complications when performed postpartum compared with the situation when the same surgical procedures are performed at cesarean section. However, elective hysterectomy ought to be considered earlier (2–4 weeks postpartum) than suggested in the literature, to avoid development of further complications, including coagulopathy and an increased risk of severe genital tract infection or hemorrhage [33].

Medical treatment of residual adherent placenta with MTX is still controversial. Some authors advocate leaving the placenta in situ in selected hemodynamically stable patients when blood loss is minimal and total hysterectomy is difficult and problematic [34–36]. MTX affects placental tissue by reducing its vascularity, leading to its necrosis, separation, and expulsion. Adjuvant MTX may help in expediting the absorption of the residual placenta, which should reduce the risk of severe complications of late postpartum hemorrhage. Even though MTX treatment seems to reduce blood flow within the placenta, transcervical removal of the firmly adherent placenta should not be attempted because of the risk of potentially profuse hemorrhage, unless necrotic fragments of the placenta are spontaneously expelled. Recently, a case was referred to TVGH from a local medical center, with residual adherent placenta at the time of cesarean section, and conservative intra-
partum management with a firmly adherent placenta (6 × 7 × 7 cm) left in situ (implanted in the lower segment of the uterus). After admission, intravenous cefazolin and gentamicin were given. MTX (1 mg/kg) was injected intramuscularly on the 25th and 39th postpartum days. Unfortunately, severe complications of sepsis developed due to leukopenia (white blood cell count dropped from 7.08 × 10^9/L to 0.2 × 10^9/L). Hysterectomy was performed 48 days after cesarean delivery. The patient has gradually recovered from the sequelae of sepsis 4 months after cesarean delivery.

The number of reported cases of residual adherent placentas successfully treated with MTX is small and the outcome is uncertain. Therefore, it is unclear whether this treatment is applicable in the expectant management of placenta previa percreta [37].

**Future Aspects**

Although the pathogenesis of placenta accreta is well characterized at the macroscopic (e.g. uteroplacental hypervascularity in the region of interest) and microscopic levels (e.g. decidual defect and intramyometrial infiltration of the placental villous tissue), the molecular basis of this disorder remains largely unexplored. The decidual defect in placenta accreta, a common histologic finding, is usually caused by previous cesarean section or uterine curettage. Due to the decreased production of tissue inhibitors of metalloprotease and transforming growth factor-beta resulting from decidual maldevelopment, the proliferation, migration, and invasiveness of trophoblasts might be changed significantly. From a preliminary molecular study at TVGH, we acknowledge that decidual defect is a major contributing factor to the formation of placenta accreta. However, epidermal growth factor receptor (EGFR) and c-erbB-2 expression in the syncytiotrophoblast may play an additive or synergistic role. Expression of EGFR and c-erbB-2 in the villous and extravillous cytotrophoblast is less likely to contribute to the development of placenta accreta [38].

**Toward the Goal, Press On**

Early antepartum identification of placenta accreta/increta/percreta offers the opportunity for termination of a life-threatening pregnancy or alteration in the surgical approach to achieve a planned delivery with the use of internal iliac artery balloon occlusion techniques in viable late pregnancy and/or IIAE for non-viable pregnancy in the first and early second trimesters, thus diminishing blood loss, morbidity, and mortality. First-trimester diagnosis should therefore be pursued aggressively in any woman with placenta previa and a history of previous uterine surgery [39].

**Acknowledgment**

This study was supported by the Medical Research Council (Grants No. 926404C and 936406C) of the Taichung Veterans General Hospital, Taiwan.

**References**


