Morbidly adherent placenta: The need for standardization

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Abstract

Morbidly adherent placenta (MAP) can be associated with major maternal morbidity, and is increasing in frequency. Determination of optimal management has not yet been satisfactory. We identify problems with lack of uniformity and the need for standardized nomenclature for the diagnosis, treatment and research of MAP. We suggest potential solutions and identify areas of future work.

Overview

Morbidly adherent placenta (MAP) occurs when the placenta fails to detach from the uterine wall due to abnormal implantation at the basal plate. This often leads to massive obstetric hemorrhage, and sequelae such as blood transfusion, multi-organ failure, need for morbid hysterectomy, and even death[1]. Owing to the relative rarity of the condition, few high quality data are available regarding the optimal management. However, it is increasing in frequency[2, 3] and has become one of the most morbid obstetric disorders in developed nations[2-7]. This is likely due to a dramatic and persistent increase in the rate of cesarean delivery[2-8]. Determination of optimal management has been further hampered by a lack of standard nomenclature. Some definitions are clinical, others based on imaging, and still others based on histopathology. However, there is considerable variation among the definitions used among countries, regions, hospitals and even clinicians. Cases of MAP managed conservatively (without hysterectomy) are hard to define since there is no specimen to evaluate histologically. Consequently, it is difficult to compare results of studies and to improve care for women with MAP.

This problem is highlighted by studies on the conservative management of MAP. Excellent outcomes have been reported after hysterotomy, leaving the placenta in-situ, partial removal of the placenta, or removal of the placenta with additional hemostatic suturing of the placental bed. However, none of these cases had confirmed placenta accreta spectrum based on histological examination. Thus, it is difficult to counsel women regarding the true risks of conservative management of MAP. Many of these women had fewer traditional risk factors for MAP (such as multiple prior cesareans) than in other series. Accordingly, their outcomes may not be applicable to women with multiple prior cesarean sections and placenta previa.

Accurate prenatal diagnosis is critical to reduce the risk of maternal morbidity and mortality. Indeed, several studies have reported that antenatal diagnosis of MAP leads to reduced blood loss and other complications[9-11]. In part, this is due to planned cesarean hysterectomy, delivery of the infant through a fundal hysterotomy and avoidance of the placenta. In addition, it allows for planned delivery under optimal circumstances in a center of excellence skilled in the delivery of women with MAP[12]. Unfortunately, the ability to antenatally diagnose MAP is imperfect, in part due to a

lack of uniform nomenclature and significant overlap of ultrasound markers of MAP with normally implanted placentas.

There are numerous reports on prenatal diagnosis of invasive placentation using ultrasound[13]. In fact, relatively high sensitivities and specificities have been described, albeit in high-risk cases and with full knowledge of clinical risk factors. Unfortunately, there is low consistency in the terminology used to describe the sonographic features of MAP. In turn, this makes it difficult to compare studies of sonographically diagnosed MAP. In addition, there are substantial clinical implications since failure to diagnose MAP may lead to avoidable morbidity while false positive diagnoses may result in unnecessary hysterectomies and loss of fertility. Clearly there is a need for standardized nomenclature when it comes to the diagnosis, treatment and research of MAP.

Issues with antenatal diagnosis of invasive placentation

Ultrasound features of invasive placentation have been described and can be divided into the following groups:

a. Direct visualization of placental tissue beyond the uterine cavity, such as a bulging mass in the urinary bladder. Although visualization of the placenta in the bladder cavity is strong evidence of abnormal placental invasion, this is a rare finding. Consequently, the sensitivity is low, but specificity is high. Overall, the prevalence of this sign in cases of confirmed invasive placentation is very low.
b. Abnormalities of the placental-uterine interface. Using gray scale ultrasound, loss of the normal hypo-echoic retro-placental space has been described as a sign of MAP. This feature is however, operator and angle dependent, varies between an anterior or posterior placenta and is associated with a high false positive rate.
c. Lower segment myometrial thickness. The myometrial thickness in the lower segment is measurable in millimeters. A study of 30 women with previous Caesarean delivery using both trans-abdominal and transvaginal ultrasound between 36 and 39 weeks of gestation, reported the thickness of the entire lower uterine segment to be 3.6 – 19.2 mm, and 1.0–9.7 mm, respectively. The

95% limits of agreement were 3.5 mm and 0.8 mm, respectively. This metric is poorly reproducible and subject to considerable variation[14]. The sign has been described in many cases without invasive placentation, and the observed specificity is low[15].

d. Color Doppler studies. Using color Doppler ultrasound, increased vascularity of the lower segment-urinary bladder interface has been described (Table 1). This is subjective since currently there are no quantitative indicators to measure such increased vascularity, rather there is the appearance of increased vascularity compared to normal controls. Location of the placenta in the lower uterine segment alone is enough to increase vascularity as compared to those with fundal placenta and with prior cesarean deliveries, increased vascularity and scarring is often seen in the lower uterine bladder interface in pregnancies without MAP. Indeed, 'increased' vascularity can be made to appear by changing the sensitivity of the ultrasound machine even in cases where placenta is not implanted in the lower uterine segment (Please see Figure 1). Almost all publications with placenta accreta examine only women with known risk factors such as low anterior placenta and previous Caesarean delivery. Therefore, selection bias may explain the 'increased' vascularity at the lower segment interface. It remains undetermined whether such 'increased' vascularity is a result of abnormal invasion versus low-anterior placental location and how this may be objectively measured. Collins et al[16] , using off-line analysis of volumes obtained with 3-D power Doppler ultrasound, showed significantly increased area of vascular confluence in the maternal-fetal interface of placenta in women with MAP as compared to controls. This could be helpful to remove subjectivity in reporting increased vascularity associated with MAP. However, this technique is not readily available to clinicians, and independent validation is needed before it can be used clinically

e. Abnormalities of the placental echo-structure: Presence of placental lacunae was an early described sign reported with MAP[17-19]. The pathophysiology is unclear but may result from placental tissue alterations resulting from long-term exposure to pulsatile flow. The content of the lacunae has been described variably as having low velocity flow[20], turbulent flow[21], diffuse or focal flow[22-24] or turbulent high velocity flow[25] (Table 2). 'Abnormal' and 'confluent' vessels have been described with invasive placentation, but no

definition is available of the normal range of appearances. Furthermore, location and size of placental lacunae have not been clearly defined in relation to the prediction of MAP.

f. Parametrial invasion: Many series describe abnormal placental invasion anteriorly, presumably through a previous uterine scar. Lateral (parametrial) invasion of placental tissue has been demonstrated on MRI[26]. Insufficient healing of the uterine incision extending into the lateral wall, and implantation with subsequent invasive placentation extending into the parametrium is a plausible explanation for this phenomenon. The frequency of parametrial invasion was 18% (62/342) of all cases of invasive placentation in one series[26].

Problems with ascertainment of invasive placentation

There are two main theoretical approaches to the definitive ascertainment of MAP. One is histopathological confirmation, and the other is based on surgical findings. Both are problematic for different reasons.

Placental Histological Examination

Most cases of MAP are due to placenta accreta spectrum, representing different grades of morbid placental attachment secondary to invasion of placental tissue deep into the myometrium beyond the normal utero-placental interface. Placenta accreta is histologically defined as placental attachment to the myometrium without intervening decidua. If there is deep myometrial invasion it is termed placenta increta and if invasion through myometrium into the serosa and beyond, even into surrounding structures such as the bladder, it is termed percreta. The term placenta accreta spectrum also is often used in cases of *clinically apparent* morbidly adherent placenta.

Placenta accreta is often simplistically defined as invasion of the myometrium with placental villi in contact with myometrial tissue. However, the main histological feature is the absence of normal decidua at the basal plate, and the detection of such findings is dependent on the mode of placental delivery and sampling issues. In one study microscopic foci of myometrial tissue adherent to the basal plate with deficient

intervening decidua, consistent with a mild or focal form of placenta accreta, were described in 36 placentas, of which only four had a clinical diagnosis of placenta accreta, none requiring hysterectomy[27]. It was concluded that mild cases of placenta accreta are frequently associated with previous uterine operations and multiparity, and are clinically suspected only infrequently. Similar findings were also reported in 44 of 457 (9.6%) preterm (birth < 32 weeks) placentas, with basal plate myometrial fibres present[28] and in another study of 90 consecutive singleton placentas basal plate myometrial fibers were seen in 27/90 (30%)[29]. Only 9 of these 27 had clinical features suggestive of placenta accreta, such as postpartum hemorrhage, delayed third stage of labor, retained placenta, or need for manual removal. The high frequency of myometrial fibers were attributed to extensive sampling and it was suggested that myometrial fibers in the placental basal plate can confirm, but do not necessarily indicate clinical placenta accreta.

Conversely, conservative surgery may leave residual adherent parts of the placenta *in-situ* and therefore histopathological confirmation will not be possible. Moreover, the pathologist is more likely to seek invasive placentation if extirpative surgery has been performed whereas features of MAP may be missed if the pathologist only assesses a small number of routine placental sections. Furthermore, practice and expertise among pathologists varies considerably and there is no "standard" approach for assessment and diagnosis of MAP. It is now generally agreed that histologically, the characteristic feature of invasive placentation is not only the close localization of villi and myometrium, but rather the histological lack of decidua between chorionic villi and myometrium, often with only fibrin intervening. We are not aware of any blinded studies or indeed any studies which have examined the effects of sampling on false negatives in true cases.

Clinical Diagnosis:

Placental tissue may be seen at the site of the surgical scar on direct visualization[25]. It may be argued that the picture resembles the fetal surface of the placenta, whereas one expects to see the maternal surface. It is not always easy to tell where the placenta ends and the myometrium begins. A vascular lower segment in a case of anterior placenta previa may well be indistinguishable from 'placenta in the scar'.

A diagnosis of MAP also is made when the placenta is adherent to the uterus and does not easily detach, however, again this is subjective, with no clear distinction between MAP and "retained placenta" in many cases. There are no objective criteria for the clinical diagnosis of MAP. The ultimate aim is prediction of major morbidity, whether or not invasive placentation is confirmed on histopathology or at surgery.

Future directions

Identification of at risk women: Several risk factors for invasive placentation have been described.

There is accumulating evidence to suggest that a Caesarean scar pregnancy detected in the first trimester is a precursor of MAP [30-33]. There is a lack of agreement regarding the most appropriate diagnostic criteria for Caesarean scar pregnancy[34]. However, a high proportion of Caesarean scar pregnancies result in morbidly adherent placenta in the absence of intervention [35-37]. This is not inevitable though, as uncomplicated pregnancy and delivery has been reported following conservative management of Caesarean scar pregnancy[34].

In the second half of pregnancy, the most common and clinically important risk factor is the combination of placenta previa and previous lower segment Caesarean delivery. Uterine surgery such as myomectomy, curettage or endometrial resection/ablation also increases the risk, but it is often difficult to know the exact site of the previous injury, although reports of prenatal diagnosis of fundal placenta accreta have been published. In such cases it is possible to deliver the infant without disturbing the placenta and deal with the placenta later. Hemostasis is likely to be easier, and parametrial structures are not directly at risk. In a series of 187 women with placenta previa and previous uterine surgery, 46 (24.6%) had previous myomectomy and 23 (12.3%) had prior curettage, but none had confirmed MAP[25]. The greatest yield of risk factors seems to be women with presence of placenta in the anterior lower segment with previous lower segment scar.

Agreement on standard operating procedure for evaluation of women: It is unclear if women should be examined with empty, partly filled or full urinary bladder, and if trans-abdominal or transvaginal scan should be used. Prospective studies should be directed to collect data by examining women abdominally as well as using TV

scan, and with and without bladder filling in all cases, also assessing possible parametrial extension on ultrasound. The most appropriate gestational age at which assessment should be performed is also unclear.

Use of MRI: The MRI literature for antenatal detection of MAP is biased, because MRI is not a screening method. Moreover, the technique can only be as good as the individuals interpreting the images. It has been suggested that MRI is particularly valuable for parametrial invasion[26]. Parametrial invasion is not commonly reported by other authors though. When the reported diagnostic performance of ultrasound so good with high sensitivity and specificity, it is debatable if MRI can add substantially to this. Use of safe contrasts may improve the diagnostic performance of MRI in future.

Agreement on how to ascertain clinically relevant MAP: Agreement of what constitutes clinically significant invasive placentation is required. The placenta should either be anterior and low lying (placental edge to internal os distance of 2 cm or less) or complete posterior previa with anterior extension. In those cases where a fundal incision is made, the placenta must not spontaneously detach after the delivery of the baby. Part of the placenta must remain attached to the uterine wall even if the rest of the placenta can be peeled off. Protocols for sampling have been published[38, 39], and include 2-4 full thickness 'random' sections, each of which should have a variable amount of basal / maternal material, including decidua and/or myometrium. As described above, presence of histopathological features without complications such as bleeding is of little clinical significance. Formal blinded assessment by histopathologists to ascertain the degree of inter-observer agreement of MAP is required, compared to an agreed clinico-pathological gold-standard for the diagnosis of clinically relevant MAP.

Agreement on terminology of description: Consensus is required regarding the definition of clinical and imaging features such as 'echo poor areas'; and lacunae, number and location of lacunae, as distinguished from other ultrasonographic features such as 'lakes' or 'fall-out areas', Doppler interrogation of the contents of lacunae and documentation of the type of flow and storage of 3-D volumes for blinded off-line assessment is required. The lower segment-bladder boundary examined with grey

scale and colour flow mapping should be assessed. All features should be assessed blindly. Significant inter-observer variability in the diagnosis of invasive placentation has been reported when examiners were blinded from clinical data[40].

Towards these ends, the European group on invasive placentation is sharing experiences of women with suspected MAP as a first step towards developing consensus for the diagnosis and management of this condition. Recently, standardized ultrasound descriptors[41] and reporting[42] for abnormally invasive placenta have been suggested based on consensus amongst experts, with a focus on unambiguous definition in a move towards universally agreed terminology. However, subjectivity still remains with several features such as 'increased' vascularity and 'irregularity' of the bladder interface, which remain otherwise undefined. Assessment of the predictive accuracy of these signs will remain problematic without agreement on what constitutes a clinically relevant MAP. We suggest a consistent clinical definition of abnormal placental invasion, imaging terminology and pathological criteria to this end (Table 3). The terminology deliberately avoids the term 'accreta'. As already mentioned, the aim is to identify disorders associated with major maternal morbidity/mortality.

Whilst definitive antenatal diagnosis may not be possible, by developing scoring systems and assigning weight to individual signs depending on their positive likelihood ratios, improved detection and management should be possible. Such future developments should be on the basis of robust data rather than logic, reasoning or consensus of 'experts'.

Acknowledgements: The authors report no conflict of interest.

Legends to figures

Figure 1 – Colour flow mapping in a pregnancy where the placental attachment is in the posterior uterine wall in the upper uterine segment. Alteration of the equipment setting can result in a subjective appearance of increased vascularity.

Table 1 Description of increased vascularity

Author	Year	Description
Calì[25]	2013	Hypervascularity/Abnormal vascularity of serosa–bladder interface, Hypervascularity of uterine serosa-bladder interface (coronal view), Irregular intraplacental vascularization with tortuous confluent vessels across placental width (lateral view)
Peker[43]	2013	Hypervascularity/Abnormal vascularity of serosa–bladder interface
Mansour[44]	2011	Hypervascularity/Abnormal vascularity of serosa–bladder interface
El Beherry[21]	2010	Vessels crossing the interface disruption site
Shih[22]	2009	Hypervascularity/Abnormal vascularity of serosa–bladder interface, Numerous coherent vessels involving the whole uterine serosa–bladder junction (basal view), Hypervascularity (lateral view), Inseparable cotyledonal and intervillous circulations (lateral view)
Miura[22]	2008	Hypervascularity/Abnormal vascularity of serosa–bladder interface
Wong[45]	2008	Vessels extending from the placenta to the bladder, Vessels crossing the interface disruption site, Vessels extending from the placenta to the bladder, Increased sub-placental vascularity, Vessels bridging the placenta and the uterine margin
Japaraj[46]	2007	Hypervascularity/Abnormal vascularity of serosa–bladder interface, Dilated peripheral subplacental vascular channels with pulsatile venous type flow over the cervix,

Table 2. Description of placental lacunae

Author	Year	Large linear lacunae with low velocities CD flow	Diffuse or focal lacunar flow pattern	Vascular lakes with turbulent flow	Vascular lak turbulent flow velocity (PSV
Calì[25]	2013				X
Chalubinski[47]	2013			X	
Peker[43]	2013				X
El Behery[43]	2010			X	
Shih[22]	2009		X		X
Wong[45]	2008		X		
Japaraj[46]	2007				
Chou[23]	2000		X		X
Twickler[20]	2000	X			

Table 3 **Suggested terminology of description**

Imaging definition	
2 D gray scale	Presence of bulk of the placenta in the lower uterine se Presence of placental lacunae (multiple) Bladder wall interruption Interruption of the bladder wall Placental bulge/exophytic mass
Colour Doppler/Power angio	Bridging vessels Placental lacunae with/without flow
3-D ultrasound	Signs as in 2-D ultrasound
Clinical definition	Placenta that does not separate at all, or separates or baby. Attempt at removal of the placenta leads to brisk hemo Placenta seen to be permeating the full thickness of urinary bladder or parametrium.
Pathology definition/criteria	Histological lack of decidua between chorionic villi a intervening. Close localisation of villi and myometrium can be an a

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