Morbidly adherent placenta describes a spectrum of pathological conditions in which the placental villi adhere directly to (placenta accreta) or invade into (placenta increta) the myometrium, or invade through the myometrium to the serosa (placenta percreta). Because of the lack of intervening decidual tissue, these placenta do not separate normally from the uterus after delivery, leading to catastrophic postpartum haemorrhage (PPH) and peripartum hysterectomy.

In the past, morbidly adherent placenta was uncommon, with an estimated average incidence of 1 in 7,000 deliveries before the 1970s. However, its incidence has steadily increased over recent decades and could be as high as 1 in 500 deliveries. The most important independent risk factors for this condition are the presence of placenta praevia, previous uterine scar (from caesarean section or other forms of uterine surgery) and advanced maternal age.1-3

Successful management of morbidly adherent placenta requires close collaboration between obstetricians, anaesthetists and radiologists. Research into the management of morbidly adherent placenta has focused mainly on two areas: better antenatal diagnosis and improved intraoperative care. The accurate diagnosis of morbidly adherent placenta enables elective delivery before antepartum haemorrhage occurs. At present, diagnosis mainly relies on ultrasonography (US) and magnetic resonance imaging (MRI).

In a normally implanted placenta, there is usually a hypoechoic layer between the placenta and myometrium. The typical US signs of morbidity adherent placenta (figure 1) are:

1. Disappearance of the echolucent area between the uterus and placenta.
2. Presence of multiple placental lacunae (Swiss-cheese appearance).
3. Extension of the placental vili into the myometrium, serosa or the bladder.

The absence of the hypoechoic retroplacental zone alone has a detection rate of 93% but a high false-positive rate of 21%. Presence of multiple lacunae is considered to be a more specific sign for morbidly adherent placenta, but only allows identification in 78-93% of cases.1 The observable extension of placental tissue to the serosa or into the bladder is the only pathognomonic sign, but is insensitive because it is only applicable to cases with placenta percreta. Colour Dopler US may provide additional information on increased vascularity or the presence of turbulent flow and improve accuracy of diagnosis.5

The MRI findings of invasive placentation (figure 2) are:

1. Thicken dark nodular contour of the placenta-uterine interface with extensions of the dark bands within the placenta.
2. Mass effect of the placenta on the uterus causing an outer bulge.
3. Heterogeneous placental signal with large placental lakes or vessels.

In addition, MRI facilitates placental mapping for correct uterine incision, especially in an anterior placenta.6

Using these imaging findings, in addition to placental site and the presence of a uterine scar, the sensitivity and specificity of antenatal diagnosis of invasive placentation have been reported to be 77% and 96% for US, and 88% and 100% for MRI. Ultrasound and MRI may be complementary in their roles in assessing for morbidly adherent placenta.6,7

From 2007, the UK Confidential Enquiries into Maternal and Child Health have recommended that interventional radiology (IR) should be considered in the management of morbidly adherent placenta. However, a recent UK survey showed that only 31% of units have experience of using the technique, 46% have considered it and 23% have no experience at all. Moreover, availability was found to be extremely variable, with only 29% of units having 24/7 access to IR, either on site or via formalised local networks.8

IR can be used reactively (in the event of massive haemorrhage in a patient with morbidly adherent placenta) or prophylactically (in women at risk).

There are no randomised controlled trials of the use of IR morbidly adherent placenta, either in a reactive or prophylactic context. Most current evidence comes from case series and the commonly measured outcomes are a reduction in the rate of hysterectomy, decreased blood loss and the need for transfusion of blood products.9,10 In evaluating the literature it is important to distinguish between the techniques of balloon occlusion and transcatheter embolisation.

• Balloon occlusion involves the inflation of a balloon proximally within a vessel supplying the uterus (figure 3). This does not result in complete cessation of flow as there are multiple collateral pathways to the uterus (eg the ovarian arteries) which continue to supply blood. The aim is to reduce pulse pressure (in a manner akin to surgical vessel ligation) slow bloodloss and improve the surgical field until definitive haemostasis is achieved.

• Embolisation involves the delivery of particulate agents which flow distally from the point of injection and produce distal (arteriolar) vessel occlusion. The level of occlusion is beyond the collateral pathways to the uterus and therefore embolisation is more effective than balloon occlusion in achieving haemostasis – usually achieving definitive haemostasis. It has the disadvantage of not being immediately reversible (unlike a balloon which can be deflated). Additionaly, high quality fluoroscopy is essential for the safe practice of embolisation.

The techniques can be combined, usually with balloon occlusion followed by embolisation.

The literature relating to balloon occlusion is entirely in the form of case series and small case-control studies. There is significant heterogeneity as to vessel occluded, size and type of balloon used and whether the balloon was inflated prophylactically or reactively. Some report IR to be beneficial, some have produced equivocal results, others have failed to demonstrate any benefit (table 1). Recently the use of aortic occlusion has been described as a salvage procedure in patients who are exsanguinating. The hetero-
genuity of techniques and results in the literature prevent any conclusion being made as to the utility of balloon occlusion in PPH.

In a study from our institution using prophylactic uterine artery (UA) balloon occlusion in 13 women, the maximum estimated blood loss was 800mL. Four women required transfusion of 1-4 units of packed red cells. No blood products were necessary. There were no hysterectomies or admissions to the intensive care unit. However, we encountered significant fetal bradycardia in two cases following the insertion of the balloon catheters, which we hypothesised was due to UA spasm. The babies that were subsequently delivered by emergency caesarean section were compromised with a low umbilical artery pH and poor Apgar scores, and one needed a brief period of ventilation in the neonatal unit. Management in our study was also complicated by the fact that the obstetric operating theatre was situated distant to the radiology suite and valuable time was lost in transferring the mother from the radiology suite to the operating theatre.

The literature relating to transcatheter embolisation is more mature. There are several sizeable case series which demonstrate its effectiveness as a sole technique or as an adjunct to surgery in the control of unexpected PPH of all causes. Clinical success is in excess of 95% in this patient cohort, though abnormal placentation is a recognised risk factor for failure, with clinical success rates of about 75%, especially if the placenta is left in-situ. The utility of embolisation in PPH is well established and is the basis of the recommendation from the UK Confidential Enquiries into Maternal and Child Health that IR be available for the management of these patients.

The question of whether the IR suite or the obstetric operating theatre is the best location for delivering patients with morbidity adherent placenta has been debated. Placement of balloons within branches of the internal iliac artery (IIA) and embolisation are techniques requiring high quality fluoroscopy available only in the angiography suite, though this environment is often not suitably equipped for the surgical and anaesthetic teams. The alternative (management entirely in the obstetric operating theatre with a mobile image intensifier) risks suboptimal imaging and adversely affects the success and complication rates of IR procedures.

The ideal is a fully equipped joint angiography-operating theatre, with high quality fixed C-arm image intensification and angiographic software packages, theatre grade air-adaptable anaesthetic equipment and space for the multiple teams involved in these complex cases. These facilities are now installed in many centres, driven mainly by the requirements of trauma and vascular-surgical interventions. It is difficult to defend the practice of insertion of IR catheters in the radiology suite followed by caesarean delivery in the obstetric theatre where such facilities exist, especially in the light of our experience with fetal compromise. Where such facilities are absent, compromises will be made. Multidisciplinary discussion on a case-by-case basis is vital.

Maternal complications associated with IR are not insignificant. Fever, thromboembolism of the pelvic arteries, lower limb ischaemia, rectal wall necrosis, cauda equina syndrome, sciatica, and puncture site complications such as haematoma, false aneurysms and dissection of femoral arteries have all been reported. The Royal College of Radiologists has published complication rates for IR in non-pregnant patients. As pregnancy is a prothrombotic state, a higher incidence of thrombotic complications should be expected.

Until now fetal compromise resulting from IR was regarded as a theoretical risk as insertion of balloon catheters in the narrow and tortuous UA could potentially cause arterial spasm and reduce placental perfusion. Following our reporting of fetal compromise, it is vital that continuous fetal monitoring be used during IR procedures. The fact that IR techniques are not innocuous means that patients should not be subjected to them unless a clear indication has been established. This applies especially in an elective (prophylactic) context.

There is still some way to go to establish the precise role of IR in the management of morbidly adherent placenta. The primary goal of research in this area must focus not only on prevention of maternal morbidity and mortality but also on the safety of the fetus. Given the strong recommendations from the confidential inquiries, it would appear unlikely that we will be able to conduct a randomised controlled multicentre trial to answer these questions. We therefore have to gather evidence from case series in a more structured manner, for example via a national registry. In the meantime, local discussion between obstetricians, anaesthetists, radiologists, neonatologists, and managers is essential to ensure protocols are drawn up for the management of these complex patients well in advance.

References


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TABLE 1

Outcomes following prophylactic pelvic artery balloon occlusion in the management of suspected placenta accreta.
FIGURE 1
An ultrasound image of placenta accreta. The placenta is seen low in the pelvis. There are echolucent lacunae within it (arrow) and thinning of the hypoechoic subplacental zone (arrowheads). These are signs associated with an increased likelihood of abnormally adherent placentation. Image courtesy of Dr M Weston, Leeds Teaching Hospitals NHS Trust.

FIGURE 2
T2w coronal pelvic MRI of placenta accreta. The placenta (arrowheads) is seen low in the uterus, abutting the bladder and bulging into it. Additionally there is a T2 dark band crossing the placental substance (long arrow). These are signs associated with an increased likelihood of abnormally adherent placentation. The hypointense inner myometrial layer is poorly visualised where the placenta abuts the bladder, though this is a less specific sign. Short arrow – fetal head. Image courtesy of Dr M Weston.

FIGURE 3
Intra-procedural screen-grab during the placement of pelvic arterial balloon catheters. Bilateral IIA balloon catheters (long arrows) have been placed from the contralateral sides across the aortic bifurcation. Contrast has been injected down the central (wire) lumen of the catheter in the left internal iliac artery, opacifying distal branches (arrowhead) and the placenta (short arrows). The fetal head is clearly visible. Double arrowhead – cardiotocograph monitor.

FIGURE 4
Antero-posterior maximum intensity projection magnetic resonance angiogram of the pelvis, demonstrating pelvic vascular anatomy. Ao - aorta; CIA - common iliac artery; EIA - external iliac artery; IIA - internal iliac artery; AD-IIA anterior division of the IIA; UA - uterine artery arising from the AD-IIA. The exact branching pattern of the AD-IIA is variable; PD-IIA - posterior division of the IIA; CFA - common femoral artery; SFA - superficial femoral artery; PFA - profunda femoris artery.