Magnetic resonance imaging of placenta accreta

Binoj Varghese, Navdeep Singh1, Regi A.N George1, Sareena Gilvaz2

Department of Radiodiagnosis, Amala Institute of Medical sciences and Mediscan, Thrissur, 1Departments of Radiodiagnosis, and 2Gynaecology and Obstetrics, Jubilee Mission Medical College and Research Institute, Thrissur, Kerala, India

Correspondence: Dr. Binoj Varghese, Department of Radiodiagnosis, Amala Institute of Medical Sciences (AIMS), Thrissur, Kerala - 680 055, India. E-mail: drbinojv@yahoo.co.uk

Abstract

Placenta accreta (PA) is a severe pregnancy complication which occurs when the chorionic villi (CV) invade the myometrium abnormally. Optimal management requires accurate prenatal diagnosis. Ultrasonography (USG) and magnetic resonance imaging (MRI) are the modalities for prenatal diagnosis of PA, although USG remains the primary investigation of choice. MRI is a complementary technique and reserved for further characterization when USG is inconclusive or incomplete. Breath-hold T2-weighted half-Fourier rapid acquisition with relaxation enhancement (RARE) and balanced steady-state free precession imaging in the three orthogonal planes is the key MRI technique. Markedly heterogeneous placenta, thick intraplacental dark bands on half-Fourier acquisition single-shot turbo spin-echo (HASTE), and disorganized abnormal intraplacental vascularity are the cardinal MRI features of PA. MRI is less reliable in differentiating between different degrees of placental invasion, especially between accreta vera and increta.

Key words: Abnormal placental vascularity; dark intraplacental bands; magnetic resonance imaging; placenta accreta

Introduction

Placenta accreta (PA) occurs when the chorionic villi (CV) invade the myometrium abnormally due to defect in the decidua basalis.[1] PA is used as an umbrella term for invasive placentation and is classified on the basis of the degree of myometrial invasion [Figure 1] as shown in Table 1.

Prevalence of PA is approximately 1 in 1000 deliveries, with a reported range from 0.04 to 0.9%. Differences in the study population and definition account for this wide range.[1] Major risk factors for PA are placenta previa and previous cesarean section. The risk of developing PA is 3% in women with only placenta previa and increases to 24% in those with placenta previa and one prior cesarean delivery.[2] The risk increases with the number of previous cesarean deliveries.[3] Other risk factors are increasing maternal age and history of uterine surgery.[4]

Placenta previa refers to abnormal low position of the placenta, near to or overlying the internal cervical os. The occurrence is 1 in 200 pregnancies at the time of delivery. The presence of placenta previa in at-risk pregnancies warrants detailed evaluation to exclude the PA. Normally, the lower margin of placenta is seen at least 2 cm away from the margin of the internal cervical os. The placenta previa is divided into four types depending on the relationship and distance to the internal os as follows:
- low-lying placenta – lower placental edge is within 2 cm from the internal os
- marginal previa – placental edge extends to the margin of the internal os, but does not cover it
- complete previa – placenta completely covers the internal os

Table 1: Classification of placenta accreta

<table>
<thead>
<tr>
<th>Placenta accreta vera</th>
<th>Chorionic villi (CV) in contact with myometrium, but not invading it</th>
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<tbody>
<tr>
<td>Placenta increta</td>
<td>CV partially invading the myometrium</td>
</tr>
<tr>
<td>Placenta percreta</td>
<td>CV penetrating through the entire myometrial thickness or beyond serosa</td>
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Varghese, et al.: MRI of placenta accreta

The consequence of PA is massive hemorrhage at the time of placental separation and complications of blood loss. Hysterectomy is usually required, leading to complications like adjacent organ injuries and serious co-morbidities. Placenta percreta can also lead to disruption of adjacent organs, most often the urinary bladder.

Accurate prenatal diagnosis is the most important factor affecting outcome, as underdiagnosing or overdiagnosing PA can create major problems. It gives opportunity for, timing and site of delivery. It also enables the obstetrician to ensure the availability of blood products, and the presence of a multidisciplinary surgical team. Definitive therapy is an elective cesarean hysterectomy at 34-36 weeks, although hysterectomy is not required or desirable in every case.

USG and MRI are the modalities for prenatal diagnosis of PA, although limitations exist for each technique. As it is relatively inexpensive and easily available, USG remains the primary diagnostic tool for the diagnosis of abnormal placentation. In addition, routine USG examination at 18-20 weeks gestation affords an ideal opportunity to screen for the disorder. Placental lacunae with turbulent flow and abnormal areas of hypervascularity with dilated blood vessels at the placenta–myometrial interphase are the most useful USG markers for PA.

In recent years, there has been an increased interest in the use of MRI for evaluation of suspected PA. MRI should be reserved primarily for equivocal USG findings of abnormal placentation or posterior placenta with risk factors. It also has a complementary role in specifically delineating the extent of an USG-diagnosed placenta percreta.

The reported accuracies for diagnosis of PA by USG (including color Doppler) and MRI showed no statistically significant difference. In high-risk patients, a normal ultrasound at 18-20 weeks of gestational age does not completely exclude PA, and these patients should be reevaluated in the third trimester. MRI is typically not indicated after a negative screening ultrasound due to relatively high negative predictive value of USG.

The purpose of this article is to describe the MRI technique and features of normal placentation and accreta.

**Technique of MRI Scanning**

**Key guidelines**
1. Supine or left lateral decubitus position depending on patient tolerability
2. Use of multichannel surface coil and oxygen supplementation
3. Urinary bladder should be moderately distended
4. T2-weighted half-Fourier RARE and balanced steady-state free precession sequences in the axial, sagittal, and coronal planes
5. Breath-hold T1-weighted gradient-echo sequence in one plane.

The optimal timing of MRI is not established and usually follows an incomplete or inconclusive USG. Usually second trimester patients can tolerate supine positioning. Left lateral decubitus positioning is preferred for third trimester patients, as it decreases the risk of impaired venous return from caval compression by the gravid uterus. A phased-array surface coil is used whenever possible to maximize signal. Oxygen via a nasal cannula should be given routinely to patients to reduce fetal motion. Urinary bladder should be only moderately full to avoid patient discomfort and for better assessment of potential bladder invasion. A radiologist should be present at the time of the examination and should guide the technologist when repeat sequences or oblique images are needed.

MRI was performed with a 1.5-T scanner (Magnetom Vision, Siemens Medical Systems, Erlangen, Germany). T2-weighted

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**Figure 1 (A-D): Diagrammatic illustration showing different degrees of placental invasion (P, placenta; M, myometrium). Arrow shows stratum basalis of endometrium. (A) Low-lying placenta showing normal stratum basalis of endometrium (arrow). (B) Placenta accreta vera showing invasion of stratum basalis of endometrium and in contact with myometrium. (C) Placenta increta showing partial invasion of myometrium. (D) Placenta percreta showing invasion of myometrium and extension beyond serosa**

- central previa – midportion of the placenta (not the edge) completely covering the internal os.
from the basal plate into the intervillous spaces. The placental septi are projections limited at the fetal surface by the chorionic plate and amniotic

The placenta has two surfaces – fetal and maternal. It is limited at the fetal surface by the chorionic plate and amniotic membrane. The maternal surface is limited by the basal plate. Also, in between these two surfaces lies the villi, intervillous spaces, and placental septi. The placental septi are projections from the basal plate into the intervillous spaces.

MRI appearance of normal placentation accreta

Knowledge about the normal placental anatomy is essential in understanding the imaging appearances of the normal and invasive placentation.

Anatomy

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Umbilical cord is attached to the fetal surface (chorionic plate). From the cord insertion, branches of the umbilical vessels (chorionic vessels) radiate beneath the amnion. Chorionic vessels send their branches in a perpendicular direction deep into the placenta and form vascular network of villous trees. The maternal spiral arteries open into the intervillous spaces by piercing the basal plate. Uterine veins drain these spaces also by piercing the basal plate and run parallel to the decidua. This venous network forms the retroplacental echolucent zone described in USG.

Relevant pathological anatomy of PA

Relevant pathological anatomy also needs attention at this context, because of its diagnostic value in PA – intraplacental lacunae (IPL) and placental or venous lakes (VL). IPL refer to intraparenchymal vascular spaces with ill-defined margins, irregular shape, and turbulent flow. They correspond to the increased volume of blood flow and high flow rate of the adherent placenta. Significant number of IPL at 15-20 weeks of gestation is the earliest reliable USG sign (sensitivity 79% and positive predictive value 92%) of PA in at-risk patients. Increase in the number and size of lacunae is associated with increased risk for PA.

VL refer to intraplacental sonolucent space. These appear usually rounded and show laminar flow. These include decidual septal cysts, intervillous thrombosis, and placental infarction (depending on chronicity). IPL sonologically differ from VL by their more indistinct margins and turbulent flow. Intervillous thrombi and placental infarctions are the potential causes of false positives in MRI diagnosis of accreta, due to their T2 hypointensity.

MR imaging appearance of normal placentation

Key imaging features of normal placentation

1. Homogeneous T2-intermediate signal intensity of placenta
2. Subtle thin, regularly spaced placental septi
3. Normal subplacental vascularity
4. Triple-layered sandwich appearance of myometrium
5. Pear-shape of normal gravid uterus with smooth contour.

The placenta shows homogeneous intermediate signal intensity on T2-weighted images and is usually clearly distinct from the underlying myometrium [Figure 2A-C]. Subtle thin, linear areas of decreased T2 signal intensity running through the placenta may be seen in high-Tesla MR images, which represent normal placental septi [Figures 2A, 3B and C, and 4A and B]. These normal septi are usually regularly spaced and uniformly thin. Normal subplacental vascularity is seen as numerous flow voids just under placenta [Figure 2B]. Few intraplacental flow voids can also be seen, usually in the region of umbilical cord insertion.

Placenta appears as a regular homogeneous structure at 19-23 weeks [Figure 3A]. Subsequently, placenta appears...
slightly lobulated at 24-31 weeks [Figure 3B] due to visualization of faint sporadic septi. This appearance progresses on advancing gestational age. Universal appearance of septi and stratification of placenta into lobules are seen after 36 weeks [Figure 3C].[13]

The myometrium shows variable thickness and thins as pregnancy progresses. It can be seen as triple layer of signal intensity [Figures 2C and D, 4A and B, 5A and B, and 6A and B]. The inner and outer layers are seen as thin bands of decreased T2 signal intensity. The middle layer is seen as thick intermediate signal intensity and frequently shows multiple vascular flow voids. Uterine contractions can cause transient focal T2-hypointense myometrial thickening [Figure 7]. The gravid uterus usually shows a smooth contour and a wider body and fundus than the lower segment.

However, the myometrium can normally appear thin and homogeneous beneath the placenta. This appearance is particularly common at sites where the gravid uterus is compressed by the maternal spine or aorta, just proximal to the bifurcation [Figure 8]. On nonenhanced T1-weighted images, both placenta and myometrium show homogeneous intermediate signal intensity and is not useful for assessment of abnormal placental invasion [Figure 9].

Imaging signs predictive of abnormal placentation
Various MR imaging features of PA with differing sensitivities and specificities are described in literature.
Earlier MR criteria, which focused on primary signs of direct invasion of placenta into myometrium were nonspecific. The most acceptable secondary signs\[4,14,15\] with good inter-rater reliability are listed below.

**Imaging findings of PA**

**Cardinal signs**

1. Dark intraplacental bands on T2-weighted images
2. Heterogeneity within the placenta
3. Abnormal disorganized placental vascularity
4. Uterine bulging
5. Focal interruptions of the myometrial wall (high specificity for increta and percreta)
6. Tenting of urinary bladder (highly specific for percreta)

Dark intraplacental bands [Figures 10A, 11, and 12] appear as nodular or linear areas of low signal intensity on T2-weighted images (HASTE and True FISP) and typically extend within the placenta from the placenta–myometrium interface. These bands are thicker than the normally fine placental septa and show a random distribution. They represent areas of fibrin deposition within the placenta.

Heterogeneous signal intensity in the placenta [Figures 10A, 11, and 12] depends primarily on the presence or absence of abnormal T2 dark bands. It may also represent areas of hemorrhage in the placenta or increased vascularity. Homogeneous placenta can exclude abnormal placenta with high levels of confidence.

**Figure 6 (A-C):** Partial placenta previa in a 28-year-old woman at 34 weeks of gestation. (A, B) Sagittal True FISP and T2 HASTE MR images show a homogeneous placenta (+) with smooth distinct placenta–myometrium interphase (arrows) and triple-layered appearance of normal myometrium. The clarity and sharpness of the interphase is superior in the TrueFISP images. (C) Coronal T2 HASTE MR image shows a homogeneous placenta (+) with multiple normal chorionic vessels subamniotically at fetal surface (arrowheads) and increased subplacental vascularity (arrows).

**Figure 7 (A, B):** Axial T2 HASTE and True FISP MR images show focal transient hypointense myometrial thickening (white arrow) at the regions of uterine contraction. Note the normal trilaminar myometrial architecture (black arrow) and myometrial thickening at different locations in the images, denoting the transient nature of contractions.

**Figure 8:** Axial T2 HASTE MR image shows loss of normal trilaminar myometrial architecture and mild focal thinning of placenta at the site of compression of myometrium in front of aorta (black arrow), just proximal to the bifurcation. Normal trilaminar signal pattern of myometrium can be seen at other regions (white arrows).

**Figure 9 (A, B):** T1 WI showing normal placenta posteriorly at the upper segment of uterus. Both placenta and myometrium show homogeneous intermediate signal intensity (P, placenta; arrow, myometrium): (A) spin echo image (SE); (B) FLASH 2D image.
Abnormal disorganized placental vascularity is described as hypertrophied, tortuous disorganized vessels deep within the placenta, located in some of the areas of dark bands. These areas of signal void on T2 HASTE images show hyperintensity on flow-sensitive True FISP images. This corresponds with IPL described earlier.

A focal outward contour bulge [Figure 10A-D] or disruption of the normal pear shape of the uterus, with the lower uterine segment being wider than the fundus, can be seen in PA. Focal interruptions of the myometrial wall [Figure 10C and D] or extension through the myometrium [Figure 10E] with occasional invasion of adjacent structures can also be seen. Placenta directly invading or tenting the urinary bladder is highly specific for placenta percreta. MRI is particularly useful in showing parametrial extension which is not apparent on USG.

Focal thinning and indistinctness of the myometrium and loss of thin T2 dark uteroplacental interface [Figure 13] are unreliable signs of PA. MRI is less reliable in differentiating between different degrees of placental invasion, especially between accreta vera and increta.

Common pitfalls in the diagnosis of PA

Dark intraplacental bands are also seen in placental infarction and intervillous thrombus. Placental infarcts are common in term placentas and in patients with risk factors for placental insufficiency. These confounding factors should be kept in mind while diagnosing PA.

Heterogeneity of the placenta is a subjective finding, and the spectrum of normal appearance varies with gestational age. Hence, a good degree of clinical experience is needed for appropriate interpretation of placental heterogeneity. Even though abnormal intraplacental vascularity denotes placental invasion, the increased pelvic vascularity is not a reliable indicator of adherent placenta.

The lower segment widening of gravid uterus may result in more of an hourglass shape to uterus. Even though it is described as a reliable sign of PA, this appearance is seen occasionally in healthy gravid uterus. Imaging in oblique planes, where uterus has rounded contours at uteroplacental
interphase, may give rise to pseudo impressions regarding the signs. So, additional imaging perpendicular to the suspicious interphase and localization of abnormality in at least two orthogonal planes are suggested.

**Conclusion**

MRI is a complementary diagnostic modality in patients with high risk for PA and should be considered when USG is inconclusive or incomplete. Familiarity with MRI technique to assess the placenta and experience with imaging appearances of normal and invasive placentation will help the radiologist in contributing to an optimal outcome.

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