

## Lessons Learnt from Two Women with Morbidly Adherent Placentas and a Review of Literature

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### Abstract

**Introduction:** Pathologically adherent placentas occur when there is a defect of the decidua basalis, typically arising from previous caesarean section, resulting in abnormally invasive implantation of the placenta. The depth of placental invasion varies from the superficial (accreta), to transmural and possibly beyond (percreta). **Clinical Picture:** We report on 2 cases, one treated “conservatively”, the other with a caesarean hysterectomy, both of which led to a safe outcome for both mother and baby. **Conclusions:** Management relies on accurate early diagnosis with appropriate perioperative multidisciplinary planning to anticipate and avoid massive obstetric haemorrhage at delivery.

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**Key words:** Management, Obstetric haemorrhage, Placenta accreta, Placenta percreta, Placenta increta

### Introduction

Pathologically adherent placenta occurs when there is a defect of the decidua basalis, resulting in abnormally invasive implantation of the placenta into the substance of the uterus. As a result, there is no clear plane between the placenta and the underlying uterus to which it is implanted. The extent of adherence and invasion of the placenta varies from the superficial (accreta), into the myometrium (increta) and right through the myometrium to breach the serosa or beyond (percreta), involving adjacent structures such as the bladder. The principal risk for this condition is massive obstetric haemorrhage at delivery, particularly when attempts are made to separate the placenta in unrecognised cases.

Previously thought to be extremely rare, the incidence appears to be on the rise in more recent medical literature. This is largely due to the ubiquitous rise in caesarean section rates. Caesarean section causes a breach in the decidua basalis which forms the nidus for abnormal implantation of the placenta in subsequent pregnancies. Previous myomectomies and uterine curettage (usually in the context of pregnancy terminations) could also predispose to this, but by far, caesarean section is the leading cause. We report on 2 cases of pathologically adherent placentas, one treated “conservatively”, while the other had a caesarean

hysterectomy, both of which led to a safe outcome for both mother and baby.

### Case Reports

#### Case 1

A 29-year-old Chinese lady, gravida 3 para 1, with a previous caesarean section and an evacuation of uterus for molar pregnancy was referred to our department at 35 weeks' gestation for placenta praevia and placenta accreta. Antenatally, she was booked for antenatal care with a private obstetrician. A routine anatomy screening scan done at 20 weeks was reported as normal. At 32 weeks, however, a routine growth scan revealed placenta praevia major, with a strong suggestion of placenta accreta, due to the observation of numerous aberrant vessels between the placenta and the bladder. Antenatally she had been well otherwise, with no reported episodes of antepartum haemorrhage. A repeat ultrasound scan at our unit showed that the placenta was in the lower left lateral position, covering the cervical os completely, and had invaded the entire myometrium. As a result, the diagnosis was revised to placenta percreta. The left anterior base of the placenta lay adjacent to the postero-lateral wall of the bladder, with large vessels seen between the 2 structures. Invasion into the bladder could not be precluded. This was followed up

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with a magnetic resonance imaging (MRI) scan which confirmed placenta praevia with features compatible with placenta percreta at the left anterior and right posterior aspects. At the anterior aspect, there appeared to be invasion of the adjacent urinary bladder wall.

After discussion with an anaesthetist, urologist and interventional radiologist, we decided upon a conservative treatment. Caesarean section took place at 36+6 weeks under general anaesthesia. Blood products including fresh frozen plasma, cryoprecipitate and platelets were available when needed. Prior to incision, the urologist performed a cystoscopy, which showed tortuous and dilated veins with areas of telangiectasia over the posterior wall of the bladder. Ureteric stents were inserted. Subsequently, both internal iliac arteries were cannulated and balloon occlusion catheters positioned by the interventional radiologist. These balloons could be inflated if required to provide temporary occlusion of the internal iliac artery and its distal branches to aid in haemostasis. A vascular surgeon was also on standby. A midline incision was made and the fetus was delivered via a vertical fundal hysterotomy. Placenta percreta was confirmed on visual inspection of the uterus. A decision was made to leave the placenta in situ. The balloon catheters were not inflated because of minimal bleeding. After closure of the abdomen, embolisation of the placenta was performed.

Postoperatively, she was monitored closely for postpartum haemorrhage and infection. She had an uneventful postpartum stay in the hospital and was discharged on the 10th postoperative day. She was then followed up at the outpatient clinic with serial ultrasound scans of the placenta and serum beta human chorionic gonadotrophin ( $\beta$ HCG) levels. The  $\beta$ HCG was 194 U/L at 8 weeks postpartum and 85 U/L at 10 weeks postpartum. It dropped to <2 U/L at 13 weeks postpartum. The placenta showed a significant reduction in size from 9 cm on the 5th postoperative day to 5 cm at the 10th week postpartum on serial ultrasound. The patient passed out some of the placenta tissue and the rest of the tissue underwent involution as evidenced by a repeat ultrasound scan 5 months postpartum, which showed the uterus had returned to normal.

### Case 2

A 35-year-old Malay lady, gravida 2 para 1, with a previous caesarean section for placenta praevia major, was booked at our antenatal clinic at 10 weeks' gestation. She had an early dating scan at 10 weeks. A routine fetal anomaly scan showed no fetal anomalies at 21 weeks, but noted that the amniotic fluid was slightly reduced. A second scan was repeated at 22 weeks, which showed major placenta praevia covering the os and oligohydramnios. It was also noted that there was no distinct plane seen

between the placenta and the myometrium, prompting the suspicion of placenta accreta. Subsequently serial scans at 24 and 27 weeks showed the same features of placenta praevia, placenta accreta and oligohydramnios.

She was admitted at 28 weeks for antepartum haemorrhage and the decision was then for an emergency caesarean section. In anticipation of potentially severe obstetric haemorrhage, the interventional radiologist was called in to insert occlusion balloons into the internal iliac arteries. A senior obstetric consultant and a senior anaesthetic consultant also attended the caesarean section. A midline incision was made and the baby was delivered via a classical uterine incision. The occlusion balloons were inflated once the baby was delivered. In view of the massive blood loss and that the placenta only invaded the muscle layer and not to the bladder, the decision was made for a caesarean hysterectomy. The estimated blood loss was about 3 litres. She was transfused with 7 units of packed cells, 1.6 litres of fresh frozen plasma, 1 unit of cell-separated platelets (CSP), 1 litre of colloids and 1.5 litres of crystalloids. Occlusion balloons were deflated after the hysterectomy once haemostasis was secured and were subsequently removed post surgery. The patient was transferred to the intensive therapy unit (ITU) for monitoring. She was well postoperatively and was discharged on the 4th postoperative day.

A baby boy was delivered with a birth weight of 1700 g and Apgar scores of 5 at 1 minute and 7 at 5 minutes. He was transferred to the neonatal intensive care unit (NICU) for subsequent care. He stayed in the NICU for 19 days and was subsequently managed in the special care unit for another 19 days before he was discharged on day 39 of life.

Histopathological examination of the uterus confirmed invasion of the deep myometrium sparing the serosal layer (placenta increta).

### Discussion

The incidence of placenta accreta has increased 10-fold in the past 50 years and now occurs with a frequency of 1 per 2500 deliveries.<sup>1</sup> The incidence varies from 1:540 in Thailand to 1:93,000 in the United States. The high incidence reported in Thailand may be related to the increase prevalence of trophoblastic disease in Asia.<sup>2</sup> It has been suggested that the rarest form, placenta percreta, represent 5% to 7% of all abnormal placentations.<sup>3</sup> The 3 variants of placenta accreta are classified by the extent of myometrial villus infiltration. Placenta accreta is typified by chorionic villi on the myometrial surface, placenta increta by villus infiltration into the myometrium, and percreta by infiltration through the entire myometrium to breach the serosa and beyond.

The aetiology of placenta percreta is unknown. It has

been postulated to be related to the damage of the decidua basalis, which allows placental invasion into the myometrium. The barrier function of decidua is absent and the invasive trophoblasts may invade the myometrium.<sup>3</sup> There are several risk factors for placenta accreta and they include placenta praevia with or without previous uterine surgery, previous myomectomy, previous caesarean section, Asherman's syndrome, submucosal fibroids, maternal age more than 35 years<sup>1</sup> and previous trophoblastic disease.<sup>4</sup> Clark et al<sup>5</sup> observed an increased incidence of placenta praevia after caesarean section from 0.26% in women with a normal uterus to 0.65% after 1 and up to 10% after 4 or more caesarean sections. Some studies reported that the risk of placenta accreta increased to 39% to 40% for those who had had 2 caesarean sections.<sup>1,6</sup> About 75% of placenta percreta cases are associated with placenta praevia. In the presence of these risk factors, the obstetrician must have a high index of suspicion for placenta accreta and hence take appropriate precautions.

Placenta percreta is associated with a maternal mortality reportedly as high as 10% and significant maternal morbidity, including massive haemorrhage, disseminated intravascular coagulation, hysterectomy, bladder and ureteric trauma, acute respiratory distress syndrome and acute tubular necrosis.<sup>7</sup>

Most cases present at the time of caesarean section. Other modes of presentation are acute abdomen and shock from ruptured uterus, antepartum haemorrhage, haematuria if the bladder is involved and as a complication at the third stage of labour.<sup>4</sup>

Placenta accreta complicating second-trimester pregnancy is also rare. Most case reports indicate that the mid-trimester presentation of placenta accreta could be in the form of uncontrollable vaginal bleeding or uterine rupture, causing intraperitoneal bleeding following second trimester miscarriage or dilatation and evacuation.<sup>6</sup>

Placenta percreta with bladder involvement appears to be associated with a particularly high morbidity, including massive haemorrhage and bladder resection. This made the prospect of conservative approach more appealing,<sup>7</sup> as in our first case. Despite bladder invasion, the most common presenting symptoms are premature onset of labour and vaginal bleeding. Gross haematuria is rare even when the bladder is invaded and, in one series, occurred in only 6 of the 27 reported cases of placenta percreta with bladder involvement.<sup>8</sup> In cases suspicious of placenta percreta, cystoscopy should be carefully performed and bladder biopsy should be avoided because it may precipitate severe haemorrhage. Placing ureteral stents during cystoscopy may aid in intraoperative identification and prevent ureteral injury.<sup>8</sup>

Abnormal levels of biochemical markers like alpha-feto protein (AFP) and creatine kinase (CK) have been linked with morbid adherence of the placenta. O'Brien et al<sup>9</sup> reported an association of elevated maternal alpha-feto protein with the extent of myometrial extrauterine invasion. In the absence of fetal anomalies, unexplained elevated maternal serum AFP may suggest the presence of placenta percreta. AFP may be more useful than CK in this respect, as there are reports of cases of placenta percreta in which the CK level was normal.<sup>10</sup> Moreover, AFP is more likely to be ordered than CK as it is part of second trimester screening.

Earlier case reports of placenta percreta reported that these were diagnosed in advanced pregnancy or indeed during delivery which often led to catastrophic outcomes for the patient. In view of the rising incidence of this complication, there is a need to diagnose it early in pregnancy. The imaging modalities of ultrasonography or MRI play an important role especially in patients who have the abovementioned risk factors. Ultrasound findings that suggest placenta accreta, increta or percreta are: (a) obliteration of the clear space, defined as the obliteration of any part of the echolucent area located between the uterus and the placenta; (b) visualisation of placental lacunae, defined as multiple linear, irregular vascular spaces within the placenta; (c) interruption of the posterior bladder wall-uterine interface such that the usual continuous echolucent line appears instead as a series of dashes.<sup>5</sup> The visualisation of placental lacunae had the highest sensitivity to detect placenta accreta (78.6%), followed by obliteration of clear space (57%) and the interruption of the posterior bladder-uterine wall interface (21.4%).<sup>11</sup> The use of colour Doppler does not enhance the accuracy over gray scale ultrasonography.<sup>11</sup> However, in our first case, we found colour Doppler ultrasound useful as the demonstration of placenta blood flow into the bladder interface confirmed bladder invasion. As can be seen, the predictive value of these 2 investigations is low,<sup>12</sup> and a high index of suspicion is essential for at-risk patients (Fig. 1).

Ultrasound may be limited in detecting the more severe implantation anomalies, specifically in its ability to evaluate the degree of extrauterine involvement in placenta percreta. The degree of invasiveness will alter the treatment plan and, MRI, due to its better inherent contrast, is able to exhibit this feature.<sup>13</sup> However, there are no studies to compare ultrasound with MRI. A point to note is the most common site for placenta accreta is anterior at the lower uterine segment. This allows a high frequency ultrasound transducer to evaluate this area with optimal resolution due to its superficial location. When the abnormality is located further away, as in posterior or fundal accreta or when the patient is obese, the resolution will be poor. MRI, thence, will be a better modality for antenatal diagnosis (Fig. 2).<sup>13</sup>

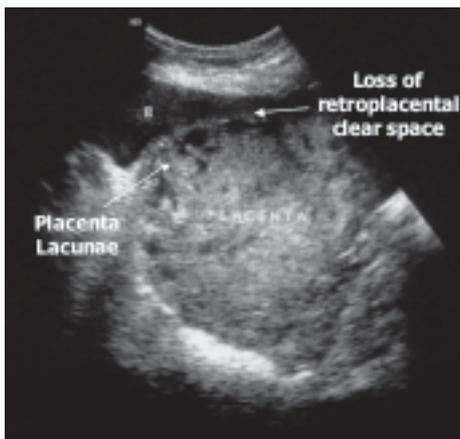


Fig. 1. Ultrasound features of placenta accreta.

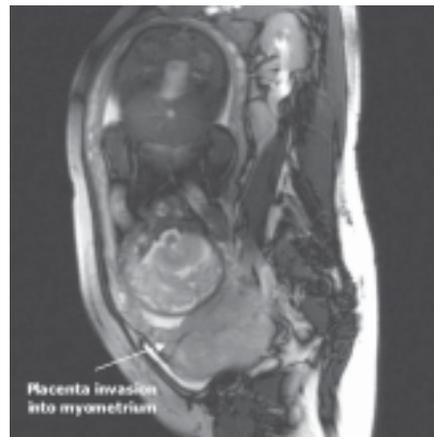


Fig. 2. Magnetic resonance imaging of placenta accreta.

In recognition of the high morbidity and mortality associated with placenta accreta, a multidisciplinary approach is recommended. The interventional radiologist, the anaesthetist, the haematologist, the neonatologist and an experienced consultant obstetrician play crucial roles. Particular consideration should be given to the anticipation and management of massive haemorrhage, including availability of packed cells, platelets, fresh frozen plasma, cryoprecipitate, whole blood and a cell saver.<sup>14</sup> It is important to recognise that it is the early replacement of blood and blood products to prevent disseminated intravascular coagulation that improves patient outcome.<sup>15</sup>

Various modifications of the uterine incision have been reported. It is optimal to avoid incising a placenta accreta because of the possibility of catastrophic haemorrhage. A classical incision, high transverse incision or a fundal incision can be used to avoid incising the placenta. Two case reports used transverse fundal uterine incision to deliver the fetus.<sup>16,17</sup> In our first case, we used a vertical fundal incision to deliver the fetus instead. In cases of bladder involvement, care must be taken not to attempt to dissect the bladder of the lower uterine segment which results in torrential bleeding. Anterior bladder wall cystotomy is particularly helpful for defining dissection planes and determining whether posterior bladder wall resection is required.<sup>8</sup>

Traditional management is by caesarean hysterectomy, which may be associated with significant maternal morbidity such as massive bleeding, bowel and urological injuries. The procedure is a daunting one, fraught with technical difficulties.<sup>15</sup> Postoperative complications include deep vein thrombosis, sepsis, bleeding and adult respiratory distress syndrome. The resultant loss of fertility is devastating if the patient is a young primigravida.<sup>15</sup> However, prompt hysterectomy has led to a reduction of maternal mortality to less than 2%.<sup>4</sup> “Conservative” management

involves caesarean delivery and leaving the placenta in situ. This may be complemented by bilateral embolisation of the uterine arteries, parenteral methotrexate or both.

Balloon occlusion devices can be placed in both internal iliac arteries prior to surgery by an interventional radiologist. The balloon occlusion devices can be inflated after the baby is delivered to minimise blood loss. The balloon can also be retained for 24 hours postoperatively to prevent postpartum haemorrhage.

The conservative approach was first described by Arulkumaran and colleagues in 1986.<sup>18</sup> Systemic methotrexate 50 mg as an intravenous infusion was administered on alternate days and the placental mass was expelled on day 11 postnatally. Subsequently similar cases have been reported. Systemic administration of methotrexate has been used in the majority of cases, although the route of administration, treatment schedule and total doses prescribed vary considerably.<sup>19</sup>

Conservative management of placenta accreta or increta with methotrexate is now an acceptable and reliable alternative to radical surgery, especially when future fertility is to be preserved. Kayem et al<sup>20</sup> described a case of a pregnancy with normal delivery without recurrence of placenta accreta after conservative treatment in which the placenta is left in the uterine cavity.<sup>20</sup> In our first case, methotrexate was not used as serial  $\beta$ HCG monitoring showed a steady downward trend. We therefore favoured allowing the placenta to involute spontaneously.

Uterine artery embolisation (UAE) has been a beneficial alternative procedure for the treatment of postpartum haemorrhage refractory to conservative non-surgical treatment. Prophylactic catheterisation before delivery and, if necessary, selective embolisation in the operating room are effective ways to stop bleeding. Serious complications of UAE, although rare, have been reported and this includes ovarian failure and sepsis.

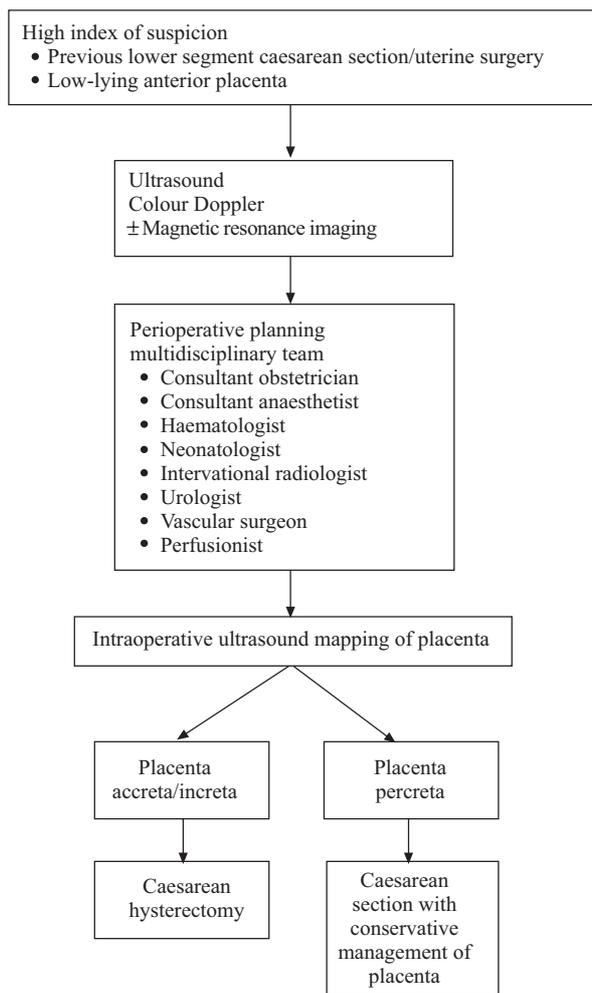


Fig. 3. Management for placenta accreta/percreta.

UAE failure for the treatment of abnormal placentation-induced postpartum haemorrhage appears to be independent of age. It is also associated with iatrogenic myometrial injury caused by vigorous digital separation of the placenta, severe disseminated intravascular coagulopathy after onset of massive blood loss, delayed or incomplete embolisation, and unrecognised bleeding from collateral circulation.<sup>21</sup>

The decision to perform a traditional caesarean hysterectomy or for “conservative” management should be decided on a case-by-case basis. We used a “conservative” approach for our first case because the placenta had invaded into the bladder, suggesting that any attempts to perform a hysterectomy would entail massive blood loss. Our approach resulted in minimal bleeding intraoperatively. In contrast, for the second case, the invasion of the placenta was confined to the myometrium of the uterus and a caesarean hysterectomy was performed relatively easily. In this case, a hysterectomy was deemed the safer option as it was a technically viable option and avoided the potential complications of spontaneous haemorrhage and infection

associated with a conservative approach. In both cases, an essential step is to ensure that the placenta is never incised or manipulated in any way, particularly upon entry into the uterus prior to delivery of the baby. Preoperative ultrasound mapping of the placenta is therefore important,<sup>22</sup> and we have also found that intraoperative scanning, prior to incising the uterus, has been helpful in delineating the boundaries of the placental edge. Although this has not been a routine practice in most centres, and indeed at ours, and may well incur further logistical arrangements, placental mapping may help to delineate the area of the uterus overlying the placenta to be avoided prior to the first uterine incision.

Often, placenta accreta is diagnosed intra-operatively, attempts are then made to separate the adherent placenta, causing massive and exsanguinating haemorrhage. Operative control may be challenging. Possible conservative methods available for haemostasis include using medical oxytocics, applying hot packs to the placental bed after delivery of the placenta, using under-running sutures on the placental bed, employing B-Lynch sutures and internal iliac artery ligation. However, most patients will require a hysterectomy, thus further emphasising the need for a high index of suspicion antenatally (Fig. 3).

## Conclusion

Placenta accreta and its variants, placenta percreta and placenta increta, are rare complications of human placentation that may threaten maternal life due principally to their potential for massive haemorrhage. Its incidence is rising due to the increasing caesarean rates worldwide. Our experience underscores the importance of making the diagnosis antenatally. Favourable outcome can only be achieved with preoperative multidisciplinary input and perioperative measures require adequate time in order to be instituted. In this, like so many other obstetric emergencies, to be forewarned is to be forearmed.

## REFERENCES

1. Committee on Obstetric Practice. ACOG committee opinion. Placenta Accreta. Number 266, January 2002. American College of Obstetricians and Gynecologists. *Int J Gynaecol Obstet* 2002;77:77-8.
2. Cox SM, Carpenter RJ, Cotton DB. Placenta percreta: ultrasound diagnosis and conservative surgical management. *Obstet Gynaecol* 1988;71:454-56.
3. Morken NH, Henriksen H. Placenta percreta – two cases and review of the literature. *Eur J Obstet Gynecol Reprod Biol* 2001;100:112-5.
4. Khadra M, Obhrai M, Keriakos R, Johanson R. Placenta percreta revisited. *J Obstet Gynaecol* 2002;22:689.
5. Clark SL, Koonings RP, Phelan JP. Placenta previa/accreta and prior caesarean section. *Obstet Gynecol* 1985;66:89-92.
6. Khan AM, Dawlatly B, Khan D, Deol N. An unusual presentation of

- placenta accreta. *J Obstet Gynaecol* 2004;24:180-1.
7. Bennett MJ, Sen RC. 'Conservative' management of placenta praevia percreta: report of two cases and discussion of current management options. *Aust N Z J Obstet Gynaecol* 2003;43:249-51.
  8. Abbas F, Talati J, Wasti S, Akram S, Ghaffar S, Qureshi R. Placenta percreta with bladder invasion as a cause of life threatening hemorrhage. *J Urol* 2000;164:1270-4.
  9. O'Brien JM, Barton JR, Donaldson ES. The management of placenta percreta: conservative and operative strategies. *Am J Obstet Gynecol* 1996;175:1632-8.
  10. Singh M, Rane A, Green E. Placenta percreta: four cases in one month. *Aust N Z J Obstet Gynaecol* 2002;42:82-4.
  11. Comstock CH, Love JJ Jr, Bronsteen RA, Lee W, Vettraino IM, Hunag RR, et al. Sonographic detection of placenta accreta in the second and third trimesters of pregnancy. *Am J Obstet Gynecol* 2004;190:1135-40.
  12. Clement D, Kayem G, Cabrol D. Conservative treatment of placenta percreta: a safe alternative. *Eur J Obstet Gynecol Reprod Biol* 2004;114:108-9.
  13. Maldjian C, Adam R, Pelosi M, Pelosi M 3<sup>rd</sup>, Rudelli RD, Maldjian J. MRI appearance of placenta percreta and placenta accreta. *Magn Reson Imaging* 1999;17:965-71.
  14. Hudon L, Belfort MA, Broome DR. Diagnosis and management of placenta percreta: a review. *Obstet Gynecol Surv* 1998;53:509-17.
  15. Jurcevic P, Grover S, Henderson J. A reassessment of options for the management of placenta praevia percreta. *Aust N Z J Obstet Gynecol* 2002;42:84-8.
  16. Shukunami K, Hattori K, Nishijima K, Kotsuji F. Transverse fundal uterine incision in a patient with placenta increta. *J Matern Fetal Neonatal Med* 2004;16:355-6.
  17. Leaphart WL, Schapiro H, Broome J, Welander CE, Bernstein IM. Placenta previa percreta with bladder invasion. *Obstet Gynecol* 1997;89:834-5.
  18. Arulkumaran S, Ng CS, Ingemasson I, Ratnam SS. Medical treatment of placenta accreta with methotrexate. *Acta Obstet Gynecol Scand* 1986;65:285-6.
  19. Adair SR, Elamin D, Tharmaratnam S. Placenta increta; conservative management – a successful outcome. Case report and literature review. *J Matern Fetal Neonatal Med* 2004;15:275-8.
  20. Kayem G, Pannier E, Goffinet F, Grange G, Cabrol D. Fertility after conservative treatment of placenta accreta. *Fertil Steril* 2002;78:637-8.
  21. Chou YJ, Cheng YF, Shen CC, Hsu TY, Chang SY, Kung FT. Failure of uterine arterial embolization: placenta percreta with profuse postpartum hemorrhage. *Acta Obstet Gynecol Scand* 2004;83:688-90.
  22. Boehm FH, Fleischer AC, Barrett JM. Sonographic placental localization in the determination of the site of uterine incision for placenta praevia. *J Ultrasound Med* 1982;1:311-4.
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