Placenta percreta with bladder involvement and profound haemorrhage: a report of two cases and short review of the literature

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ABSTRACT

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Placenta percreta is a rare complication of pregnancy with pathologic invasion of the full thickness of the uterine wall, causing life-threatening haemorrhage.

We present two cases of pregnant women with placenta percreta invading the bladder, along with a short review of the literature. The two women sustained early and late major haemorrhagic complications requiring massive transfusion (up to 142 PRBC and 353 blood product units) and urgent reoperation. They were both treated in the ICU and discharged in good state.

Massive haemorrhage due to Placenta percreta may be a fatal complication especially when the placenta invades the bladder. The anaesthetist and the intensivist may be challenged by the severity and extend of perioperative complications demanding rapid decision-making. A multidisciplinary approach for perioperative surgical and anaesthetic management is necessary for maternal outcome optimization. Post-operative care in the ICU is a prerequisite with high index of suspicion to follow transfusion requirements.

Introduction

Placenta percreta is a rare complication of pregnancy with pathologic invasion of the full thickness of the uterine wall, causing life-threatening haemorrhage. Abnormal placental attachment, due to inadequate decidua formation, between trophoblast and myometrium is the anatomical background of the situation. This abnormal implantation impedes complete separation of the placenta following delivery (vaginal or with Caesarean section), resulting in severe postpartum haemorrhage, along with uterine atony.

Three forms of abnormal surface formation between the placenta and the uterus are distinguished: placenta accreta, placenta increta and placenta percreta. In cases of placenta accreta the chorionic villi grow into the basal desidua, in placenta increta the chorionic villi penetrate into the musculature and in placenta percreta the villi pass through the myometrium and infiltrate not only the serosa but also the surrounding organs, such as the urinary bladder and the bowel, leading to serious complications.

This is a potentially fatal condition resulting in 7 percent of maternal and 9 percent of perinatal death with a further increase of 9.5 percent and 24 percent respectively, when complicated with bladder invasion[1]. Since preoperative diagnosis is not always possible, intraoperative diagnosis demands rapid decision-making in a fa-

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cility with personnel experienced in the management of intraoperative pelvic haemorrhage and available blood and blood product resources in order to prevent a fatal outcome[2,3]. Postoperative care in the ICU is a prerequisite with high index of suspicion to follow blood and clotting factors transfusion requirements.

We report two cases of Placenta percreta with bladder invasion, leading to massive haemorrhage, treated postoperatively in the ICU.

Figure 1: Case 1 antepartum sonography with findings indicative of placental invasion of the uterine and urine bladder wall (arrows)

Case 1

A 37-year-old, gravida-1, multipara woman with a history of a previous caesarean section and sonographic evidence of placenta previa (figure 1) was admitted for elective caesarean delivery at the 36th gestational week. No history of haematuria was reported.

General anaesthesia was induced with intravenous thiopental and succinylcholine. Anaesthesia was maintained with inhaled 1% sevoflurane up to 0.75 MAC in fresh gas flow of 4 $l.min^{-1}$ O_2/air (FIO_2 50%) and after a live neonate delivery, anesthesia was maintained with intravenous fentanyl, cis-atracurium and up to 0.75 MAC inhaled sevoflurane.

Placenta percreta with dilated vessels arising from the uterus and extending to the bladder and surrounding pelvic tissues, was diagnosed (figure 2,3). A decision was made to perform a caesarean hysterectomy and after a live infant delivery, total hysterectomy was completed, leaving a part of the anterior lower uterine segment adherent to the posterior bladder wall together with the invading portion of the placenta, avoiding any further dissection for the fear of bladder rupture.

Figure 2: Case 1. Macroscopic appearance of hysterectomy specimen: Placenta invading the myometrium (arrows: a, umbilical cord, b, placenta, c, area of placental invasion, d, myometrium)

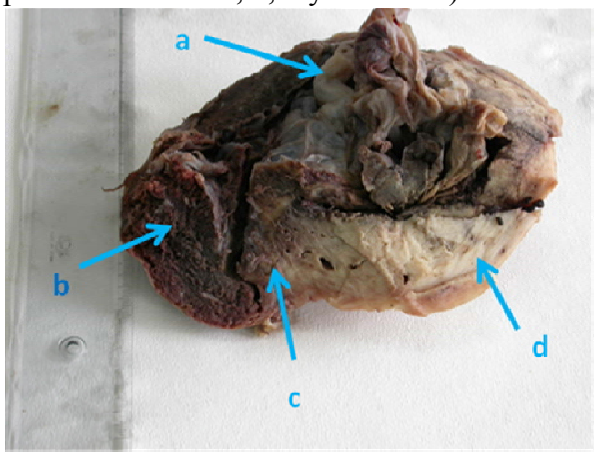
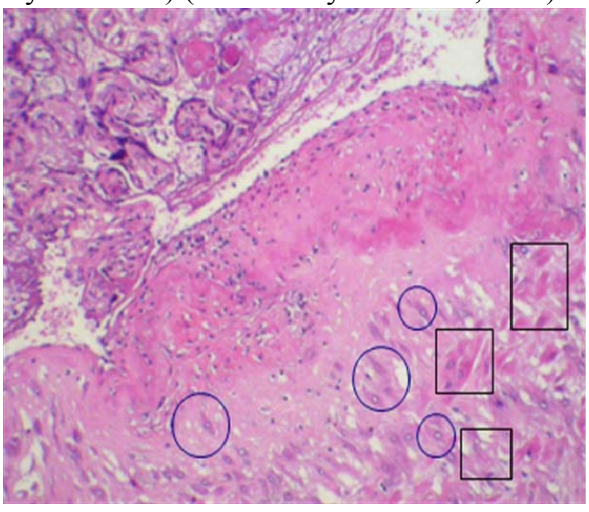


Figure 3: Case 1. Absence of decidua basalis (circles: trophoblast, squares: myometrium) (Haematoxylin-Eosine, x 40)



The situation during hysterectomy resulted in massive haemorrhage with haemoglobin 6.26 $g*dl^{-1}$ and platelets $41*10^3*microL^{-1}$ (from preoperative values of 11.89 $g*dl^{-1}$ and $135*103*microL^{-1}$ respectively) and INR of 3.1. These findings along with fibrinogen 112 $mg*dl^{-1}$ and d-dimers 500 $mcg*l^{-1}$ were

diagnostic of disseminated intravascular coagulation (DIC). She required transfusion of 16 units of PRBC, 23 units of FFP, 22 units of cryoprecipitate, and 12 units of platelets. Haemorrhagic shock required norepinephrine infusion up to 1.2 mcg*kg^{-1}*min^{-1} intraopera-tively and postoperatively in the ICU.

Twelve hours later, following massive transfusion and with values of Hb 8.6 g*dl^{-1}, platelets 7*10^{3}*microL^{-1} and INR 1.4, she was still suffering severe haematuria and persistent shock.

A reoperation was decided and bleeding from the excision area was partially controlled by abdominal packing with laparotomy pads.

Thirty hours later, although she was not in shock, but still in need of massive transfusion (Hb 5.25 g*dl^{-1}, platelets 19.9*10^{3}*microL^{-1}, INR 1.51), she was successfully reoperated on for ligation of the hypogastric arteries.

During the first 72 hours after hysterectomy, total transfusion amounted to 142 PRBC, 212 FFP, 92 platelets, and 49 cryoprecipitate units.

Although complicated at the tenth day of her ICU stay, with acute lung injury (ALI, PaO_2/FIO_2:286) and sepsis attributed to ventilator associated pneumonia, she was discharged to the ward in good general state, after a total 20-day ICU stay.

Case 2

A 45-year-old gravida-0, primipara woman, at the 29th week of an in vitro fertilization twin gestation, was admitted after spontaneous membranes rupture. Urgent caesarean delivery and hysterectomy were decided because of a previously MRI documented placenta percreta involving the bladder. The same anaesthetic scheme was used as in case 1.

After caesarean delivery of healthy twins, total hysterectomy was performed with no evidence of remaining vasculature between the uterus and the posterior bladder wall, so the bladder was preserved.

Eleven days later, severe haematuria required massive transfusion and urgent laparotomy. A remaining placental vessel penetrating the bladder wall was identified as the cause of the haemorrhage, which was successfully ligated.

Perioperatively, she sustained haemorrhagic shock with severe hypotension and bleeding, requiring 14 units of PRBC, 15 units of FFP and 6 units of cryoprecipitate transfusion. Norepinephrine infusion up to 0.15mcg*kg^{-1}*min^{-1} was necessary during the first 24 hours postoperatively. The bladder was again preserved.

After a 3-day ICU stay she was discharged to the ward, with no further complications.

Discussion

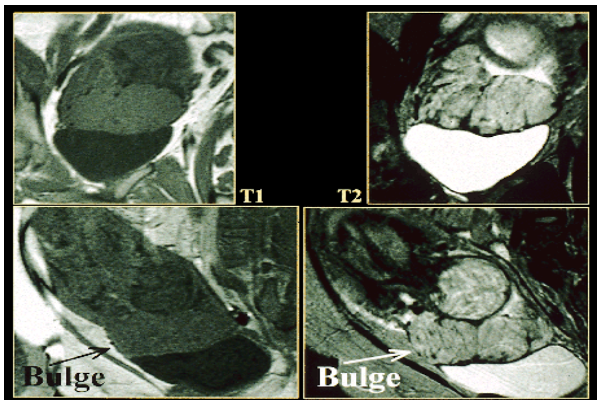
The incidence of abnormal placental formation occurring in 1:30000 deliveries in the 1950s [4,5], has risen over the last decades to 1:2500 deliveries, largely due to the marked increase of caesarean deliveries[6,7]. The more severe situation of placenta percreta invading the urinary bladder, remains a rare occurrence with less than 100 cases published in the literature[2,8-12]. Gross haematuria is rare and reported only in a small fraction of the these cases[11,13]. In our cases, haematuria became evident only postpartum and as a late sign in the second case.

The mechanism for this abnormal placentation is thought to be a thin, poorly formed decidua in the scarred area of the lower uterine segment, that cannot resist deep invasion by trophoblast, occuring most commonly after a prior caesarean delivery[7]. Other predisposing factors include prior cervical dilatations and curettages, endometritis and endometrial defects, maternal age greater than 35 years, increasing parity, submucous leiomyomata. Our first case had placenta previa and a history of previous caesarean delivery.

Placenta percreta is a potentially fatal condition resulting in up to 7 percent of maternal and 9 percent of perinatal death with a further increase to 9.5 percent and 24 percent respectively when complicated with bladder invasion[1,8,9]. Placenta percreta is a significant risk factor for early postpartum haemorrhage[14], while at least in the developing world, early postpartum haemorrhage still accounts for 11.7 percent of

direct maternal deaths[15]. Even where no maternal deaths are reported, major complications are prevalent, making invading placenta a leading cause of emergency postpartum hysterectomy[16,17]. Implementation of systematic approaches and multidisciplinary team work has contributed to improved outcomes[18].

Figure 4: Magnetic resonance image showing lack of demarcation between placenta and uterus suggestive of placenta accreta.



If placenta percreta is not diagnosed early enough, life-threatening bleeding may occur upon manual detachment of the placenta. In pregnant women presenting with shock, abdominal pain and haemoperitoneum, rupture of the uterus produced by placenta percreta should be considered[19,20].

Various imaging procedures can be used to detect the various forms of invading malplacentation. The most useful modalities for evaluating placental position and implantation are transabdominal and/or transvaginal ultrasonography with a sensitivity and specificity of at least 85 percent. In the normal situation, the interface between the placenta (which has a homogenous appearance) and bladder is characterized by a hypoechoic boundary that represents the myometrium and the normal retroplacental myometrial vasculature. Even when placenta accreta is present, this hypoechoic boundary is lost and the placenta appears contiguous with the bladder wall. Clear sonographic criteria are the absence of a normal, hypodense retroplacental myometric zone, a reduced or absent surface between uterine serosa and urinary bladder, and possibly the presence of focal exophytic tissue.

Color Doppler ultrasonography can also detect abnormal vascularization of the myometrium with a sensitivity of 82 percent and a specificity of 97 percent[21]. The findings include diffuse and focal intraparenchymal placental lacunar flow, bladder-uterine serosa hypervascularisation, prominent subplacental venous complex and loss of subplacental Doppler vascular signals.

In cases of uncertainty, magnetic resonance imaging is able to detect the level and topography of invasion assessing the extent of myometrial, parametrial, and bladder involvement, in 97,7 percent of the patients (figure 4) [10,22,23].

A further sign of an invading placenta may be an otherwise inexplicable elevated serum alpha-fetoprotein (AFP) concentration in maternal blood[24].

When haematuria is present, urinary bladder invasion should be considered but cystoscopy is not always helpful to make a preoperative diagnosis[11].

Antepartum planning may include[25,26]:

- Patient counseling for the suspected diagnosis and potential complications (e.g., haemorrhage, need for a caesarean delivery and hysterectomy)
- Delivery should be scheduled with optimal availability of necessary facilities and personnel experienced in the management of intraoperative pelvic haemorrhage.
- A preoperative anesthesia consultation should be obtained early enough before the date of delivery.
- Adequate blood and clotting factors should be available at the time of delivery.
- An intensive care unit should be available for postoperative care, as needed.

Various blood conservation techniques can reduce exposure to allogeneic blood, thereby reducing risk and conserving the blood supply. These include intra-operative cell salvage, acute normovolaemic haemodilution and preoperative autologous donation[27].

Intra-operative cell salvage although controversial, seems effective and useful in obstetrics. Leukocyte depletion filtering of cell-salvaged blood section significantly reduces particulate contaminants to a concentration equivalent to maternal venous blood[27,28].

On the other hand, since there is some evidence, normovolemic haemodilution may be considered, although it may induce anaemia and cardiac failure and cannot be used in an emergency. It may have a limited role in combination with other techniques[25,27,29,30].

Intravenous iron and exogenous erythropoietin administration have been used for the treatment of postpartum anaemia[25,31]. However, there are insufficient data to recommend their use to reduce transfusion requirements after postpartum haemorrhage.

Autologous blood transfusion has been proposed as a safe option in patients with placenta previa, diagnosed by 32 weeks of gestation, who have a haemoglobin level of 11 $g*dl^{-1}$ or above[32,33]. Preoperative autologous donation may produce anaemia, does not eliminate transfusion risk, cannot be used in an emergency and is usually not acceptable to Jehovah's Witnesses. It should be reserved for exceptional circumstances (rare blood type or unusual antibodies)[27].

In our cases, massive transfusion was necessary, defined by the need to replace more than 50 percent of blood volume in three hours. Transfusion trigger was haemoglobin level every 1-2 hours, aiming at a level > 7 $g*dl^{-1}$. Coagulation was checked after every five to seven transfused units of PRBC. FFP, platelets and cryoprecipitate were transfused in order to keep INR below 1.5, platelet count above $50*10^{3}*microL^{-1}$ and fibrinogen above $100mg*dl^{-1}$. Relevant strategy is proposed in current transfusion guidelines [34,35].

The surgical therapy for placenta percreta includes either a more invasive strategy, with uterus and involved tissues removal or a conservative approach, with the placenta left “in situ” after delivery, in haemodynamically stable patients. The latter appears to be a safe choice. If surgical excision of the placenta is attempted or necessary, physicians experienced in pelvic dissection must be involved for the fear of maternal morbidity and mortality[8,17,36,37].

Successful surgical repair of the uterus after placenta percreta extrication has also been reported [12].

An open approach with hysterectomy, but without removal of involved tissues, was adopted in our first case, while in the second, the surgical removal of the uterus did not give any clues of the subsequent complication.

Bilateral ligation of the internal iliac arteries offers a possibility to reduce blood loss during the hysterectomy. The same may be considered for the hypogastric arteries[24], which was the case in the final reoperation for our first patient. There are controversial reports of management based on immediate ligation of both hypogastric arteries before hysterectomy[38].

Balloon occlusion of the aorta or internal iliac vessels has been utilized to prevent haemorrhage as well[39]. The baloons may be inflated intermittently, during the uteral dissection, thus markedly decreasing blood loss and keeping the operative field drier. Catheters may be left “in situ” for several hours postoperatively, and then utilized for selected prophylactic embolization of small pelvic vessels in the event of postoperative bleeding[25,40].

Bladder management is also an important issue. It ranges from simple closure of the bladder hole to ureteric diversion with reimplantation of the ureters, or partial cystectomy[9-11]. On the other hand, conservation of the bladder is possible in cases of placenta percreta[2], as in both of our cases.

Case reports have also described successful conservative treatment with methotrexate chemotherapy following delivery, to aid destruction of the trophoblastic tissue with placenta left in situ, but with inconsistent results[41,42].

Serious infection, delayed vaginal haemorrhage, and DIC may occur and further surgery is sometimes required[42]. In our first case, surgical complications became evident very soon; infection and sepsis were not attributed to perioperative issues.

Human chorionic gonadotropin concentrations may become negative before the placental tissue has completely resorbed. Subsequent pregnancy with normal delivery has also been reported [12,43].

Conclusion

As it became apparent in our cases, although clinical presentation and imaging techniques can raise suspicion for placenta previa percreta, this potential catastrophic condition may remain undiagnosed or its extent under appreciated. The decision to proceed with definitive surgery should be carefully considered. Even then late postoperative haemorrhagic complications, have to be faced as in our second case.

A multidisciplinary approach for preoperative, intraoperative, and postoperative management of placenta percreta is required for maternal outcome optimization.

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