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Anaesthetic management of patients with placenta accreta

The management of a 28-year-old primigravida with placenta accreta diagnosed during Caesarean section is described. A hysterectomy was required to control massive haemorrhage, and the patient made a full recovery. The increased incidence of placenta accreta over the last three decades is thought to be associated with the concomitant increased frequency of Caesarean section, resulting in an increased incidence of placenta praevia (1.9 per cent to 3.9 per cent). Patients with placenta praevia who have had a previous Caesarean section have a remarkably increased risk of placenta accreta. Management of placenta accreta is primarily by control of haemorrhage on delivery of the placenta. Control can be assisted by infrarenal cross-clamping of the aorta and/or intra-myometrial injection of prostaglandin $F_{2\alpha}$ which produces myometrial and vascular contraction. Identification of patients at increased risk, preparation for treatment and effective treatment of placenta accreta will minimize maternal morbidity and mortality.

The abnormally adherent placenta has long been recognised as a cause of obstetrical haemorrhage. At times blood loss can be catastrophic, leading to difficult management problems, both for obstetricians and anaesthetists. Since the classic paper by Irving and Hertig¹ in 1937, a number of papers have been published on epidemiology, obstetrical management and outcome of placenta accreta.^{2,3} The present paper addresses the patients at risk from placenta accreta and their subsequent anaesthetic management.

In three recent cases at Grace Hospital, Vancouver, hysterectomy was required to control bleeding as a life-saving step. Attempts to preserve the uterus, in the face of massive bleeding, usually prolong the procedure

and increase the risk of serious complications. The increased risk of uterine rupture in subsequent pregnancies should be considered. Over a three-month period (September 1985 to November 1985, 1,933 deliveries) we had six cases of placenta accreta in our institution, clinically diagnosed at the time of delivery. Five of the cases were confirmed by histopathology.

Although the following case report illustrates the problems faced in the management of these patients it does not demonstrate any of the recognized risk factors (see Discussion), but four of our cases of placenta accreta were typical in that they each had three or four risk factors. Four were elderly (mean age 33 years) and three were multiparous. Three out of four had previous dilatation and curettage following a spontaneous abortion or retained placenta. Antepartum haemorrhage and preterm labour were seen in three of the patients and two were having a repeat Caesarean section in the presence of placenta praevia or anterior low lying placenta. Of the remaining two cases without prior Caesarean section, one had a placenta praevia, with a history of dilatation and curettage and the other a history of retained placenta, chorioamnionitis and postpartum haemorrhage with each of her previous vaginal births. Intramyometrial prostaglandin $F_{2\alpha}$ (Dinoprost Tromethamine®) was used in three of our cases and in two of these hysterectomy was not required.

Prostaglandin $F_{2\alpha}$, 0.1 per cent solution, was used in divided doses to a maximum of 10 mg. Rapid uterine contraction and cessation of haemorrhage was observed within minutes of these peri-incisional intra-myometrial injections.

Case report

A 28-year-old primigravida was admitted at 42 weeks gestation for induction of labour. She had no history of previous gynaecological procedures and had experienced an uneventful pregnancy. Her initial haemoglobin was 11.4 g·dl⁻¹ with a normal platelet count. Lumbar epidural analgesia using intermittent doses of 8 ml of 0.25 per cent bupivacaine was begun when the cervix was dilated to 6 cm. After 12 hours with no further progress, she was prepared for a Caesarean section. The patient's blood pressure and ECG were monitored and she received O₂ by

Key words

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FIGURE Classification of placenta accreta based on degree of penetration of myometrium (see text).

mask. Carbonated lidocaine (1.73 per cent), 16 ml with epinephrine (1:400,000) in divided doses, was administered into the epidural space. A healthy male infant was delivered, but massive haemorrhage began during attempted removal of the placenta, and a diagnosis of placenta accreta was made. Bilateral hypogastric artery ligation was performed and a hysterectomy was done under general anaesthesia using ketamine 40 mg IV, succinylcholine 100 mg IV and rapid-sequence tracheal intubation. Anaesthesia was maintained with N_2O/O_2 (50%/50%), pancuronium and IV fentanyl. Central venous pressure and direct arterial pressures were monitored. During the eight-hour procedure, the patient received 28 units of packed red blood cells, two units of whole blood, 12 units of platelets, 12 units of fresh frozen plasma, one unit of stored plasma, 15 units of cryoprecipitate and 13 litres of crystalloid to replace a measured blood loss of 15 litres. Postoperatively (with stable vital signs and no evidence of a coagulopathy) she was transferred to the Intensive Care Unit. The haemoglobin was $12.4 \text{ g} \cdot \text{dl}^{-1}$, PT and PTT were normal and platelets were $71,000/\text{ml}$. The patient made a full recovery.

Discussion

The pathophysiologic basis of placenta accreta was reviewed by Meyers⁴ in 1955 and more recently by Hutton *et al.*⁵ The accepted definition of placenta accreta has been a histopathologic one, based on the absence or deficiency of the decidua basalis, between the villi and myometrium, causing abnormal adherence of the placenta

and preventing normal separation of placenta from myometrium.

Placenta accreta is also categorised by the degree of penetration found. "Placenta accreta vera" implies an adherence of the placental villi directly to the myometrium. "Placenta increta" involves actual invasion into the myometrium. In "placenta percreta," the villi erode through the myometrium to involve serosa, and even the surrounding structures² (Figure).

Aarberg and Reid⁶ classified placenta accreta by the amount of placental surface involved. "Total" accreta involves the whole placenta, while "focal" accreta involves a single cotyledon or part thereof. "Partial" accreta is intermediate between the two, involving more than one cotyledon, but not the whole placental surface.

From 1930 to 1950 the reported incidence was one per 30,739 deliveries, while in the 1950's this had risen to one per 19,012 deliveries.⁷ Review of cases reported by Read *et al.* from January 1975 to May 1979 revealed an incidence of clinically diagnosed placenta accreta of 1 per 2,652 deliveries and 1 per 4,027 deliveries with pathologically confirmed cases.⁷ This increased incidence is probably a reflection of better reporting, plus a true increase in the incidence of placenta accreta.

The cause of placenta accreta is often unclear. The common aetiological factors include uterine abnormalities (e.g., fibroids, bicornuate uterus), previous Caesarean section, manual removal of placenta, placenta praevia and uterine curettage, sepsis or surgery.⁸ Previous scarring of endometrium interferes with placental implantation in subsequent pregnancies.

Placenta praevia is found in 34–50 per cent of all cases of accreta.³ This is presumed to be on the basis of relatively poor blood supply in the lower segment and subsequent deficient development of decidua basalis. As there is a strong association between placenta praevia and placenta accreta, factors predisposing to placenta praevia also predispose to accreta. These factors include: maternal age (11-fold increase in the over 40 age group compared with those less than 20 years of age), parity (seven-fold increase in patients with parity of five or more) and previous Caesarean section (two-fold increase—3.9 vs. 1.9 per cent)^{9,10}

Recently, a very strong association has been observed between placenta praevia/accreta and prior Caesarean sections. Clark *et al.* demonstrated a 24 per cent risk of placenta accreta in the patient with placenta praevia and one prior Caesarean section. With two or more previous Caesarean sections, patients with placenta praevia run a risk of over 50 per cent of also having placenta accreta.¹⁰ This combination should alert both the obstetrician and the anaesthetist to the possibility of placenta accreta with its potential dangers. Caesarean section can then be

performed in a controlled setting, with blood available and appropriate personnel ready.

Despite these considerations, the majority of patients with placenta accreta have normal pregnancies. Those with co-existing placenta praevia may present with antepartum haemorrhage, but accreta, even if suspected, cannot be diagnosed until the third stage of labour.

Another association with placenta accreta is preterm labour and delivery, due to myometrial stimulation. In Fox's series of 622 patients, 162 (26 per cent) delivered before 37 weeks' gestation. Uterine rupture is not uncommon, occurring in 13 per cent of this large series. Two-thirds of the uterine ruptures occurred prior to the onset of labour, many of these during the second trimester. Only one in seven occurred during labour. The remaining 20 per cent occurred during the third stage of labour, exclusively as a result of overzealous manual removal of the placenta. The uncommon, but serious, complication of uterine inversion was seen in three per cent of Fox's series, almost all following vigorous attempts at manual removal.³

Placenta accreta should be suspected when retained placenta and excessive postpartum haemorrhage occur on attempted manual removal of the placenta. Further attempts at manual removal are contraindicated, as increased bleeding, uterine rupture and inversion are potential complications.

The treatment of placenta accreta is primarily to control bleeding and restore haemodynamic stability. The amount of bleeding, the proportion of placental surface involved, the depth of invasion and the patients' desire for further pregnancies must all be taken into account in deciding therapy. In small focal accreta, curettage may certainly be adequate, with oversewing of the placental bed if bleeding persists. However, in densely adherent major accreta, as well as all incretas and percretas, hysterectomy is the treatment of choice. The mortality rate is reported to be 28.3 per cent, following vaginal delivery with conservative management, 6.6 per cent following vaginal delivery with immediate hysterectomy and 5.8 per cent following Caesarean section with immediate hysterectomy.³

Anaesthetic management

With changing obstetrical practice and particularly the increase in the Caesarean section rate, anaesthetists are more frequently faced with the problem of placenta accreta. The diagnosis cannot be made prior to delivery of the placenta, but it is possible, and certainly prudent, to identify patients at risk.

Placenta accreta may occur as an unexpected complication following vaginal delivery. The anaesthetist is then faced with a bleeding, hypovolaemic, unstable patient. First, an adequate airway must be established. Stabilize

the patient haemodynamically by elevating the legs to increase venous return, while starting a second large bore intravenous infusion and administering fluids rapidly. Blood for emergency group and match must be sent. Compression of the aorta should be attempted and may prove to be vital. However, compression through a lax anterior abdominal wall may not be as effective as at laparotomy. Intramyometrial injection of $F_2\alpha$ (trans-abdominally or transvaginally) may be required. MAST trousers may serve to autotransfuse and increase the afterload. Use of vasoactive drugs, such as dopamine or phenylephrine, may be used to maintain cardiovascular stability, and help stabilize the patient for transfer to a tertiary care unit for appropriate surgical management.

The identified high-risk Caesarean section patient, in our opinion, should be prepared for general anaesthesia. Two large bore intravenous cannulae should be inserted, and the patient's blood must be sent for emergency group and match. A blood warmer should be available, as large volumes of fluid may be infused in the event of intraoperative haemorrhage.

Diagnosis of placenta accreta is established when delivery of the placenta is attempted and unusual adherence is encountered. Heavy bleeding may start immediately, especially if the myometrium has been deeply penetrated by chorionic villi. Ironically, further attempts to remove placenta accreta at this point may cause catastrophic haemorrhage, as the raw myometrium is exposed.

At this time, manually cross-clamp the aorta by applying direct pressure infrarenally, thus compressing the aorta against the vertebral column. This simple and effective measure can be done quickly by the obstetrician or his assistant and requires no dissection. Cross-clamping the aorta will reduce the pulse-pressure and heavy bleeding at the surgical site. This will not only allow better visualisation of the operative site, but will also enable the anaesthetist to gain control of haemodynamic status.

Takagi,¹¹ in 1976, demonstrated that direct intramyometrial injection of 15-methyl $F_2\alpha$ (Carboprost®) transabdominally, or transvaginally, was superior to ergot preparations (intravenous or intramuscular) in causing immediate uterine and vascular contraction which lead to a reduced rate of bleeding and fewer systemic side effects. Such a manoeuvre during Caesarean section may increase uterine tone and lead to cessation of haemorrhage, thus obviating the need for hysterectomy.^{12,13} It should be pointed out that 15-methyl prostaglandin $F_2\alpha$ (Carboprost®) is a synthetic, more potent form of the drug, and should be used in a smaller total dose (0.25–1.0 mg).

In the studies mentioned, the side-effects from prostaglandin $F_2\alpha$ have been minimal, or non-existent, with intramyometrial injection, which reflects our experience.^{11,14} One might expect that if enough $F_2\alpha$ was to

enter the systemic circulation, bronchoconstriction and pulmonary hypertension could occur.¹⁴ Prostaglandin $F_{2\alpha}$ may also cause coronary artery constriction, nausea, vomiting and diarrhoea if administered intravenously or intramuscularly.

Haemodynamic instability may recur on removal of the aortic cross-clamp, thus it is prudent to maintain the cross-clamp until the patient is stable, and surgical haemostasis secured. If hysterectomy is undertaken, it may be necessary to maintain the aortic cross-clamp until the uterine arteries have been ligated. At the time of removal, the cross-clamp should be released slowly, while continually assessing the patient's haemodynamic status.

The role of hypogastric artery ligation in placenta accreta is controversial. O'Leary *et al.*¹⁵ have suggested it is of doubtful value, as it is a cumbersome, time-consuming procedure which is associated with complications, especially in inexperienced hands.

Clay-Burchell,¹⁶ in a study on non-parturients, demonstrated a rich collateral circulation, leading to continued pelvic bleeding, after hypogastric ligation. Since these collaterals are enlarged and engorged in parturients, this increases bleeding problems. Hypogastric artery ligation serves to reduce distal pulse pressure, but does not stop collateral bleeding, while aortic cross-clamping reduces pulse pressure and decreases collateral bleeding. In our experience, hypogastric artery ligation has only delayed the decision to proceed to hysterectomy and has resulted in additional blood loss and morbidity.

In these patients blood loss may be massive. Read *et al.*⁷ reported an antepartum blood loss averaging 477 ml. The average delivery and immediate postpartum blood loss was 3846 ml and 4716 ml, respectively, in those with concurrent praevias, and 2639 in the non-praevia group. Average blood transfusion was 7.9 units, with a range from 0–39 units. Clark *et al.*¹⁰ reported an average blood loss of 3.5 L in patients having Caesarean section plus hysterectomy.

In the face of hypovolaemic shock, the cardinal principle is rapid restoration of effective intravascular volume and adequate oxygen carrying capacity to ensure tissue oxygenation. If the blood loss is over 20 per cent of estimated intravascular volume, or if haemorrhage is on-going, blood transfusion should be instituted. The vast majority of patients can be controlled with packed red blood cells and normal saline. When large quantities of stored blood are administered, potential problems such as coagulopathy, hypothermia, leftward shift in the oxyhaemoglobin dissociation curve, acid-base imbalance, electrolyte abnormalities and infection must be borne in mind.¹⁷

Dilutional coagulopathy frequently occurs when effec-

tive platelets or clotting factors are diluted by massive transfusion with stored blood and crystalloid. The platelet count should be checked after each five units of transfusion. Platelet concentrates should be available after 10 to 15 units of transfusion. Each unit of platelets can be expected to raise the platelet count by 10,000 in the 70 kg adult and, in the face of continued bleeding, it is best to err on the side of safety. A haematology consultation may be helpful in planning complete blood constituent replacement and supervising the postoperative course.

The prothrombin and partial thromboplastin times are primary screening tests for clotting factors and should be done after each ten units. The administration of one unit of fresh frozen plasma for every four units of stored blood transfused will replenish clotting factors.

In the event of coagulopathy, a differential diagnosis of dilutional coagulopathy, DIC, and primary fibrinolysis must be considered. To differentiate these, further laboratory studies, including fibrinogen level, thrombin time and fibrinogen breakdown products will be necessary.

These patients, subjected to massive transfusion and prolonged surgery, with extensive tissue manipulation, are at risk for developing coagulopathy, ARDS and infection. It is prudent to monitor these critically ill patients in an intensive care setting until they are stable.

Conclusion

Identification of patients at high risk for placenta accreta will facilitate the planning and care of this potentially serious condition. The highest risk is seen in those patients with previous Caesarean section and concurrent confirmed placenta praevia.

Intraoperative cross-clamping of the aorta will reduce blood loss. When attempts to control massive haemorrhage are unsuccessful, a prompt hysterectomy may be life saving. The use of $PGF_{2\alpha}$, combined with aortic cross clamping, may enhance uterine contraction and reduce further blood loss. The role of hypogastric artery ligation is limited, especially in inexperienced hands.

Ultrasound examination between 32 and 37 weeks of gestation in patients with prior Caesarean section is a useful way of identifying placenta praevia in this high risk population.

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Résumé

On décrit la conduite thérapeutique chez une femme primigeste de 28 ans ayant un placenta accreta diagnostiqué durant une césarienne. On a dû pratiquer une hystérectomie afin de contrôler l'hémorragie massive et la patiente s'est rétablie complètement. On croit que l'augmentation de l'incidence de placenta accreta au cours des trente dernières années est associée à l'augmentation concomitante de la fréquence de césarienne, ayant pour résultat une incidence accrue de placenta praevia (1.9 à 3.9 pour cent). Les patients avec un placenta praevia, qui ont déjà subi une césarienne, sont à risque accru de placenta accreta. Le traitement du placenta accreta est d'abord le contrôle de l'hémorragie au moment où le placenta est libéré. On peut aider à contrôler cette hémorragie par le clampage infrarénal de l'aorte et/ou par l'injection, dans la paroi utérine, de prostaglandine $F_{2\alpha}$ qui produit des contractions myométriques et vasculaires. L'identification des patientes à risque accru, la préparation pour le traitement et un traitement efficace pour le placenta accreta minimiseront la morbidité et la mortalité maternelle.