



Surgical conservation of the uterus in the management of third-trimester placenta percreta using tubal and uterine artery ligation and uterine packing

Placenta praevia is a clinical conundrum due to its associated increased risk of bleeding, both as an antepartum event and at the time of caesarean section (CS). This is particularly so when it is a high grade placenta praevia associated with a previous low uterine segment scar or myometrial invasion by the trophoblastic tissue, as in cases of percreta.¹

Dealing with young women who may wish to have more children, the 'generally recommended caesarean-hysterectomy without delay for placenta percreta management',² resulting in devastating emotional and/or cultural consequences, should be avoided wherever possible, especially in nulliparous patients. With this in mind, two cases of placenta percreta identified at the time of emergency CS were successfully managed with conservation of the uterus and ability to conceive.

Case 1

A 37-year-old patient, para 1, gravida 1, had a stillbirth by CS in 1995. She consulted in June 1999 for secondary infertility, having previously been treated for a year with 1 - 3 tablets of Clomid. The date of her last menstrual period was 25 May and a pelvic sonar examination showed a 7-week gestation, with a low-lying grade 1 placenta. Her due date was estimated to be 2 February 2000.

Prenatal visits went well until December 1999, when an emergency CS was performed for preterm rupture of membranes and antepartum haemorrhage.

On laparotomy, a placenta percreta was found to be partially covered by the bladder. A baby girl of 1 800 g was quickly delivered through a transverse incision of the serosal layer.

The infant had Apgar scores of 6, 7 and 10. The placenta was then totally removed piece by piece, followed by excision of the myometrial edges, partial cystectomy and torrential bleeding. Haemostasis was only achieved by means of bilateral ligation of the uterine and tubal arteries plus endo-uterine packing with a cotton gauze passed through a digitally dilated cervical canal into the vagina, for extraction 36 hours later. Strict intensive care follow-up was implemented, with a decision to resort to hysterectomy if necessary. The mother and her baby were discharged on days 6 and 25 post-CS respectively after uneventful recovery. At follow-up on 26 February 2000, the patient had heavy but painless normal menstrual flow. Diagnostic endometrial biopsy by suction using a Karman vacuum kit revealed no abnormalities. Natural contraceptive methods plus condoms were prescribed to

prevent any pregnancy for at least the following 24 months.

On 17 March 2001 the patient was presumed to have had a miscarriage since a beta-HCG test was positive, and the uterus was found to be empty on pelvic ultrasound examination (ectopic pregnancy and ovarian gestational tumour having been excluded).

Case 2

The second case was a 31-year-old woman, para 2, gravida 3, with a history of miscarriage plus two CSs, one for antepartum haemorrhage resulting in a stillbirth in 1992, and a repeat CS in 1995. She consulted for secondary infertility in November 1999 as she was wanting another baby with her new husband, aged 39 years.

On 18 January 2002 she started treatment for secondary infertility. On 17 April 2002 she was 7 weeks pregnant, with expected date of delivery 11 December 2002. Later, she was admitted at 28 weeks' gestation (September 2002) for premature rupture of membranes and conservative management, i.e. Rocephin, sterile hygienic pad checking, daily white blood cell count, erythrocyte sedimentation rate (ESR), C-reactive protein, urinalysis, cervical swab cultures and corticoid administration for fetal lung maturation.

Ten days later, an emergency CS was performed for chorioamnionitis. The patient had elevated C-reactive protein (41.1 mg/l) and elevated leucocytes ($14.6 \times 10^9/l$), while swab culture and urinalysis were negative.

Laparotomy was done adjoining the bladder reflection, and a blue tortuous trophoblastic tumour was noted through a thin serosal layer. A transverse incision was done quickly and a 1 125 g baby girl was delivered, through a placental tear. There was massive blood loss of at least 2 000 ml. Bilateral uterine and tubal artery ligation using Vicryl no. 1 was not sufficient to control the bleeding. Haemostasis was achieved after five additional sutures (Vicryl no. 2/0) of each active bleeder on the lower uterine segment, plus intra-uterine packing with cotton gauze reaching the vaginal cavity via a digitally dilated cervical canal. The entire placental tissue was removed by swab dissection. The incisional myometrial defect was carefully excised before suturing in two layers with Vicryl no. 1. Two packed red cell units of blood were given and 2 000 ml of colloid were infused intra-operatively. After 36 hours of strict intensive care follow-up, the packing was removed without complication. The patient was discharged on day 5, followed by her baby (weighing 2 160 g) 5 weeks later, after uneventful recovery.



Comment

Diagnosis

Apart from rare cases of placenta percreta recently described by LeMaire *et al.*³ appearing without any risk factor, most common predisposing conditions should help to make a correct diagnosis, such as: repeat CS, placenta praevia^{4,5} and multiparity with a history of hysterotomy, myomectomy, cornual resection, dilatation and curettage, manual removal of a retained placenta and endometritis.⁶

Placenta percreta can be easily identified in the second trimester using ultrasound with or without colour Doppler and/or magnetic resonance imaging (MRI).^{5,7-12} Using these methods it can also be identified later in pregnancy, and occasionally during laparotomy for CS as in our two cases. Ultrasound and MRI can also be used to identify acute abdomen with haemoperitoneum in pregnancy.^{1,6,13-15}

Complications

Silent^{9,16,17} or acute forms of uterine rupture^{6,13,14,15,18,19} should be kept in mind when one discusses differential diagnosis of antepartum haemorrhage or acute abdomen in the second trimester of pregnancy.

Kotwal *et al.*,²⁰ Abbas *et al.*²¹ and Washecka and Behling²² have reported many cases of urological complications due to placenta accreta,^{23,24} increta^{25,26} and percreta, namely haematuria (31%), bladder laceration (26%), urinary fistula (13%), and ureteral transection (6%).

Feto-maternal death is another severe complication, particularly in underdeveloped settings, where highly defined ultrasonography is not available for correct and early diagnosis. Placenta percreta is then a life-threatening condition resulting in fetal and maternal deaths (9% and 7% respectively), usually due to torrential intra-caesarean bleeding.¹³ A computerised search of the literature from 1991 to 1997 indicated that 90% of patients lose more than 3 000 ml blood intra-operatively and need a blood transfusion.¹⁰

Occult infiltrating trophoblastic tissue with internal iliac vessel invasion is reported as a very rare and hard to deal with complication.¹⁶

Management

The focus in most cases is on the difficulties in achieving haemostasis and identifying the proper therapeutic options available to the treating clinician.¹ It is generally accepted that caesarean hysterectomy should be performed without delay to lower the mortality rate.^{2,27-29} In case of surgical difficulty, cystotomy helps a great deal in assessing bladder and lower ureteric anatomy and integrity.⁷ Other options available include hypogastric artery ligation or embolisation. Reviewing both contemporary assessments and older studies on hypogastric

artery ligation, Jurcevic *et al.*¹ report varying success with this procedure from 40% to 100%.³⁰⁻³² Moreover, greater surgical skill and ability are needed for hypogastric artery ligation than with tubal and uterine ligation associated with 36-hour endo-uterine packing, oxytocic infusion and broad-spectrum antibiotics.

While arterial embolisation of the uterine vessels before surgery has been described in cases of placenta accreta,³³ and certainly represents a new and exciting treatment option that appears to be highly effective, Jurcevic *et al.*¹ suggests that consideration and use of alternative successful management should be implemented without delay, especially in those settings where more sophisticated options are not feasible.

Conclusion

Instead of the currently recommended caesarean hysterectomy — as in the management of our two cases — conservation of the uterus and ability to conceive can be achieved through a dual new surgical approach. This involves suturing the tubal/uterine arteries and the lower uterine segment spiral vessels, in combination with endo-uterine packing. The latter was previously reported by Shannon and Dodenhoff³⁴ in 1947 and Druzin³⁵ in 1989. Indeed, bilateral uterine/tubal artery ligation plus tamponading the lower uterine segment certainly constitutes an easy and accessible treatment option for all obstetricians, and for any general practitioner accomplished in CS delivery.

Although total or subtotal abdominal hysterectomy is suggested as definitive treatment, in two cases in the literature conservative surgical management of acute abdomen caused by placenta percreta in the second trimester produced successful results. In one case oversewing the myometrial defect prolonged the pregnancy for an additional 8 weeks,⁸ and in another case it achieved a viable fetus at 32 weeks' gestation.¹⁴ This supports our proposal of uterine conservation whenever possible.

The authors acknowledge Dr C Kumin and the staff of the neonatal ICU, Pretoria Gynaecological Hospital (A Badenhorst, E Britz, J Motshegare, P Celliers and M Vanrensburg) for their wonderful collaboration.

K C Tshibangu
M A de Jongh
M Y Mamabolo
V Peranovic

*Department of Obstetrics and Gynaecology
 Pretoria Gynaecological Hospital*

V M Sooboo

*Louis Pasteur Private Hospital
 Pretoria*



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IN BRIEF

Magnesium sulphate for neuroprotection in preterm births

The risks of mortality, cerebral palsy and other neurosensory impairments in very preterm infants are well known. Many of these risks can be reduced by administration of magnesium sulphate to the mother, but there have been no large randomised controlled trials in which magnesium sulphate was given solely for neuroprotection. Such a study was recently reported in *JAMA* (2003; **290**: 2669-2676).

The study involved women with fetuses younger than 30 weeks, for whom birth was planned or expected within 24 hours. The women were randomly assigned to receive a loading infusion of 8 ml of 4 g (16 mmol) 0.5 g/ml magnesium sulphate or isotonic sodium chloride solution for 20 minutes followed by a maintenance infusion of 2 ml/h for up to 24 hours. Data were analysed from 2-year survivors of preterm delivery.

Among 1 047 survivors, total paediatric mortality was lower and cerebral palsy prevalence and combined death or cerebral palsy were less frequent for infants exposed to magnesium sulphate, but none of the differences were statistically significant. Substantial gross motor dysfunction, and combined death and substantial gross motor dysfunction, were significantly reduced in the magnesium group. The authors were able to conclude that magnesium sulphate given to women immediately before very preterm labour may improve paediatric outcome. Only minor side-effects were noted, and were not severe enough to warrant discontinuation in more than a few women. The authors do not, however, recommend widespread use of magnesium sulphate unless confirmed by additional trials.