A case series of interventional radiology in postpartum haemorrhage

N. Agarwal¹, O. Deinde², F. Willmott¹, H. Bojahr³, P. MacCallum⁴, I. Renfrew⁵ & S. Beski¹

Departments of ¹Obstetrics and Gynaecology, ²Barts and The London School of Medicine, London, UK, ³Anaesthetics, ⁴Haematology and ⁵Interventional Radiology, Royal London Hospital

Postpartum haemorrhage (PPH) remains a significant cause of maternal morbidity and mortality in both developed and developing countries. In some instances, PPH can be anticipated and recent improvements in obstetric imaging techniques allow earlier and more reliable diagnosis of abnormalities associated with haemorrhage such as morbid placenta. However, the majority of PPH is unpredicted. Good practice notes published by the Royal College of Obstetricians and Gynaecologists state that interventional radiology should be used as emergency intervention in PPH and should be considered when primary management has failed allowing arteries to be embolised to achieve haemostasis. Through collaboration between interventional radiology and maternity, appropriate guidelines need to be developed, on both emergency and elective of interventional radiology in the prevention and management of PPH. As there is mapping for neonatal services, in the future there should be consideration to develop obstetric trauma units. Maternity units which lack facilities for interventional radiology would be able to refer their cases (like placenta accreta) for safe management in units with 24 h interventional radiology services.

Keywords: Embolisation, postpartum haemorrhage, radiology, uterine arterial balloon, uterine atony

Introduction

Postpartum haemorrhage (PPH) remains a significant cause of maternal morbidity and mortality in both developed and developing countries (RCOG 2007, 2009). PPH is divided into immediate (primary) and delayed (secondary) categories (WHO 2009). In some instances, PPH can be anticipated and recent improvements in obstetric imaging techniques allow earlier and more reliable diagnosis of abnormalities associated with haemorrhage such as morbid placenta (Fuller et al. 2006). However, the majority of PPH is unpredicted (RCOG 2007).

The severity of PPH can be divided into minor (500–1,000 ml) and major (>1,000 ml) haemorrhages, with further division of major haemorrhage into moderate (1,000–2,000 ml) or severe (>2,000 ml) haemorrhage. As a result of excessive blood loss, many women suffer severe morbidity, some requiring blood transfusions, intensive care facilities and even hysterectomy. Globally, it has been estimated that severe PPH occurs in about 11% of women who give birth (Fawcus 2007). In the 2003–2005 report of the UK Confidential Enquiries into Maternal Deaths, haemorrhage was the third most common direct cause of maternal death (6.6 deaths/ million maternities) (RCOG 2004, 2009). It is estimated that about 14 million women suffer severe blood loss postpartum, and that 1% of these die as a result (Fawcus 2007).

According to the good practice notes published by the Royal College of Obstetricians and Gynaecologists (RCOG 2007), interventional radiology should be used as emergency intervention in PPH and should be considered when primary management has failed. Percutaneous transcatheter arterial embolisation of the uterine artery has been reported as being advantageous in the management of PPH (RCOG 2007). When compared with other options, this technique offers a high success rate and preserves fertility (RCOG 2007). The procedure is performed under fluoroscopic guidance in which angiographic catheters are used to catheterise the anterior division of the internal iliac arteries; these arteries can then be embolised to achieve haemostasis.

We present a case series of 10 patients compiled over 2 years, at two London teaching hospitals.

Materials and methods

This was a retrospective study of women who underwent uterine artery embolisation (UAE) in a tertiary centre between April 2007 and September 2009. Medical records were obtained and reviewed for the following clinical and haematological data: age; ethnicity; smoking; parity; gravidity; previous caesarean section; previous PPH (including other relevant obstetric history); gestation; spontaneous or induced delivery; mode of delivery; weight of infant; duration of labour; time of delivery; cause of PPH; estimated blood loss; blood transfusions if any (number of units of red cells, frozen fresh plasma, cryoprecipitate and platelets); use of the Bakri balloon; hysterectomy; B-Lynch sutures; time between delivery and intervention and the outcome (complications and days to discharge).

Results

A total of 14 patients that had undergone UAE were identified. Of these, 10 medical records were available for review and the data were extracted, while four records could not be retrieved (see Appendix 1). In seven cases, UAE was used to control PPH when primary management had failed. In three cases, uterine arterial balloons were positioned before delivery in anticipation of massive obstetric haemorrhage, so that embolisation could be performed immediately after delivery. All UAs were conducted...
in the interventional radiology suite. Three cases were transferred from a local hospital where interventional radiology services were not available.

Five women had previously undergone caesarean sections, the median number of caesarean sections being 1 (range 0–3). None of the women had previously experienced PPH but one woman had had a previous ectopic pregnancy.

At antenatal booking, three of the women were obese (BMI range 30–35). Antenatally one woman developed gestational diabetes and another obstetric cholestasis. Five women were diagnosed antenatally on ultrasound with placental abnormalities (two cases of placenta accreta, two cases of placenta previa and one case of placenta percreta). In three of these cases (two accreta, one percreta), the decision was made to place uterine arterial balloons before delivery in anticipation of massive obstetric haemorrhage. Two of the women underwent catheterisation of the uterine arteries and insertion of balloons, prior to delivery in order to aid uterine artery embolisation in the event of PPH. In one of these cases, embolisation of the internal iliac arteries was performed prior to delivery. In the third case, lines were fitted in the left and right femoral arteries and an aortic balloon was inserted. The two women with placenta accreta had both experienced multiple episodes of antepartum haemorrhage.

Of the other five women, one suffered multiple pulmonary emboli during the antenatal period and was started on a therapeutic dose of enoxaparin (1 mg/kg twice a day), while two women were treated with antibiotics for urinary tract infections. Three women were anaemic and were started on iron replacement. One woman was HIV-positive and another had epilepsy, and were already established on antiretroviral therapy and antiepileptics, respectively at booking (Table I).

Five women were admitted for elective caesarean section, four due to placental abnormalities. Two women were admitted for induction of labour, the first due to obstetric cholestasis and who had forceps delivery, and the other was for post-dates, who had an emergency caesarean section for fetal distress. Two women presented in spontaneous labour, one resulting in a normal vaginal delivery and the other in an emergency caesarean section due to fetal distress. One woman presented with antepartum haemorrhage at 20 weeks’ gestation with known placenta previa and accreta. The patient was admitted for monitoring, but further haemorrhage resulted in a hysterotomy delivery. This was followed by a massive PPH requiring hysterectomy and abdominal packing.

Of the infants born, seven needed no neonatal resuscitation. Two of the infants required resuscitation following delivery but subsequently did well and one infant, born at 20 weeks, was pronounced dead within minutes of birth (Table II).

Of the 10 cases, one woman suffered a minor postpartum haemorrhage (<1,000 ml blood loss), while the other nine cases developed massive obstetric haemorrhages (>2,500 ml blood loss). Two secondary obstetric haemorrhages occurred, one at 48 h and the other 4 weeks after delivery.

In seven cases, primary management failed to achieve haemostasis. This included manual and medical treatment of PPH, as well as surgical: Bakri balloons and B-Lynch sutures. Indeed PPH occurred in all 10 cases, even in the three where uterine arterial balloons were placed beforehand in anticipation of massive obstetric haemorrhage following delivery. In one of the three cases, the woman was transferred back to the interventional radiology theatre where embolisation was performed due to the failure of the uterine artery occlusion balloons to achieve haemostasis when inflated. In the second case in which embolisation had been performed prior to delivery, inflation of the occlusion balloons in the iliac artery reduced bleeding but failed to stop it. In the third case embolisation was not required.

In the other seven cases, emergency uterine artery embolisation was performed to achieve haemostasis. In seven cases where uterine artery balloons were placed as an emergency for PPH, the median interval time between delivery and embolisation was 12 h and 30 min. In the other three cases, where uterine arterial balloons were placed in advance, time to intervention was not found in one case and in the other case, where a secondary PPH occurred 8 weeks postnatally (the woman experienced four episodes of postpartum haemorrhage). It was at this point that embolisation occurred.

During the postpartum period, two women experienced pyrexia following intervention (controlled by antibiotics and antipyretics). One woman experienced postnatal depression, another developed a clot in her right femoral artery and underwent an open embolectomy. Further embolisation was performed on the internal iliac arteries 1 day post-intervention, and 27 days post-operation a haematoma was drained under ultrasound guidance. During her recovery she also experienced a urinary tract infection. One patient reported pain, which was controlled by analgesia. The other seven patients made unremarkable recoveries (Table III).

<table>
<thead>
<tr>
<th>Cause of PPH</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atonic uterus</td>
<td>2</td>
</tr>
<tr>
<td>Placenta previa</td>
<td>3 (2)</td>
</tr>
<tr>
<td>Placenta accreta/percreta (associated with previous CS)</td>
<td>1</td>
</tr>
<tr>
<td>RPOC/endometritis</td>
<td>1</td>
</tr>
<tr>
<td>Medical</td>
<td>1</td>
</tr>
</tbody>
</table>

Mean units transfused (distribution), number of patients

| Packed red cells          | 15 (4–50), 8 |
| Platelets                | 2.7 (2–4), 6 |
| Cryoprecipitate          | 3.5 (1–10), 6 |
| FFP                      | 8.6 (4–12), 71 |
| Factor VIIa              | None |
| Surgical treatment (number that underwent hysterectomy) | 5 (2) |
| None                    | 1 (0) |
| Bakri balloon            | 1 (0) |
| Bakri balloon + B-Lynch suture | 3 (1) |
| Discharge in days (distribution) | 13.1 (5–37) |
Discussion

The most frequent cause of PPH is uterine atony, occurring in 1 of every 20 deliveries, with many patients presenting with no known risk factors (Shevell and Malone 2003). In our case series of women managed by interventional radiology, placental abnormalities accounted for the main cause of PPH (50%), followed by uterine atony (30%). Other causes were endometritis as a result of retained products of conception (10%) and the combination of diclofenac and enoxaparin (10%). In our obstetric population, 50% of the women had undergone previous caesarean sections, of these 40% had a previous history of placenta praevia. These are both risk factors for morbidity adherent placenta, which is associated with massive obstetric haemorrhage. Any patient who gives a history of prior PPH has up to a 10% risk of recurrence even without other identifiable risk factors (Shevell and Malone 2003), however in our case series of every 20 deliveries, with many patients presenting with no complications included haematomas at the puncture site, infection, fever and infection (Shevell and Malone 2003); other associated factors for morbidly adherent placenta, which is associated with 40% had a previous history of placenta praevia. These are both risk factors for controlling massive obstetric haemorrhage. This technique has recently been increasingly used as a method of controlling massive obstetric haemorrhage. This technique has been refined to target the internal iliac or uterine arteries, requires appropriately trained interventional radiologists and is only available in a small number of tertiary centres (Moore et al. 2008). Ideally, this technique would allow placement of arterial catheters before delivery and when needed, catheters can be advanced to a more distal location in the vessel than ligation would allow. Balloon catheters can first be used to tamponade the area, and if necessary subsequent embolisation of the vessel may follow, where bleeding is witnessed. The technique may also be used in the emergency setting, providing that access to the technique is available. Gelfoam pledges are used for embolisation, and when performed, angiography follows immediately to see if success has been achieved (Shevell and Malone 2003). The success rate of the technique was 100%, with nine out of nine case studies illustrating achieved haemostasis via embolisation, where blood flow was reported to decrease or cease. Our own figures match that of the literature, where the rate of embolisation success has been reported as being over 90% (Soncini et al. 2006). A 2002 review summarised case series totalling 100 women and reporting 97% success with selective arterial embolisation for obstetric haemorrhage (RCOG 2009). The advantages of embolisation include the preservation of fertility and decreased incidence of re-bleeding from collaterals due to more distal occlusion obtained with embolisation than with surgical ligation (Hong et al. 2004). This interventional radiological procedure represents an efficient solution if the PPH is in the corpus of the uterus and the woman is haemodynamically stable (Ornan et al. 2003).

Complications of pelvic embolisation are rare and include fever and infection (Shevell and Malone 2003); other associated complications include haematomas at the puncture site, infection, vascular injuries, allergic reactions, uterine infarction and necrosis, coagulopathy and acute intra-arterial thrombosis or ischaemia of the lower limb. The patients in the case series reported postoperative pain and fever (Shevell et al. 2003). There were no reports of limb ischaemia in any of the cases.

In 70% of our cases interventional radiology was used in an emergency setting, in 30% of the cases it was used electively. Some authors report the efficacy of embolisation being compromised by its use in emergency settings, reporting increased blood loss and coagulopathy in patients who underwent emergency procedures versus those who had elective arterial catheter placement (Shevell and Malone 2003). Our series was too small to compare elective and emergency embolisation, but organisation of the multidisciplinary team was easier in the elective setting. In 30% of our cases, women were transferred as emergencies from another centre, where intervention was not available. Haemorrhage was rapidly controlled by UAE in 100% of these cases. The technique seems most advantageous when placed in controlled settings.

Uterine artery embolisation is a safe and effective way of controlling PPH (Moore et al. 2008). With the increasing reported rates of caesarean section, more women will be at risk of PPH, due to placental abnormalities. This technique should become more widely available and considered in such women who are suspected to be at risk of PPH.

This case series has shown the role and efficacy of UAE in obstetrics. UAE should be used in both the elective setting and primary early intervention in women who are actively bleeding. This avoids the need for ligation or hysterectomy in some cases. UAE is a relatively quick technique in experienced hands, but our series did reveal a delay between PPH and intervention by UAE. Although this can be attributed to stabilising the patient, the fact that the patient has to be transferred between departments adds to the delay. There is a possibility that either the interventional radiology theatre could be equipped for laparotomies or that appropriate radiological equipment could be integrated into obstetric theatres. This would reduce transfer delays.

Appropriate departmental protocol and guidelines need to be developed through collaboration between both departments (interventional radiology and maternity). In addition to these guidelines, an appropriate protocol should be formulated for maternity units, which do not have onsite interventional radiology suites. The resulting guidelines should be available in all major obstetric units and interventional radiological suites. These guidelines should offer guidance on both emergency and elective use of interventional radiology in the prevention and management of PPH. The guidelines should highlight what to do in an emergency situation, including appropriate notification, obtaining senior help, how to and where to access imaging and when to make contact with the tertiary maternity centre and interventional radiologist. As there is mapping for neonatal services, in the future, there should be consideration to develop obstetric trauma units. Maternity units which lack facilities for interventional radiology would be able to refer their cases (like placenta accreta) for safe management. Units which have helicopter emergency medical service (HEMS), and 24 h interventional radiology services should step up to become tertiary units for maternity services.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References


