Management of primary postpartum haemorrhage with arterial embolisation in Hong Kong public hospitals

Objective. To assess the utilisation, effectiveness, and safety of arterial (angiographic) embolisation for management of severe primary postpartum haemorrhage in Hong Kong public hospitals.

Design. Retrospective study.

Setting. All eight obstetrics and gynaecology units of the Hospital Authority in Hong Kong.

Patients. Women who underwent arterial embolisation for primary postpartum haemorrhage from July 1999 to June 2004 inclusive.

Main outcome measures. Cause of primary postpartum haemorrhage, estimated blood loss, patient condition before embolisation, and the intervals between the diagnosis of postpartum haemorrhage and the procedure.

Results. Primary postpartum haemorrhage occurred in 7200 (3.9%) cases of 183 700 deliveries; 90 (0.05%) underwent total hysterectomy, whilst 29 (0.016%) received angiographic embolisation. Arterial embolisation was 90% effective in treating medically uncontrollable primary postpartum haemorrhage, except in three patients who failed to respond and underwent a hysterectomy. All 29 patients survived, although due to severe haemorrhage one had a cardiac arrest, whilst another had transient right-leg claudication. Six patients developed mild fever.

Conclusions. In Hong Kong, arterial embolisation for severe primary postpartum haemorrhage is a safe and effective treatment modality but is under-utilised. If first-line medical treatment fails and patients are haemodynamically stable, the procedure should be considered an alternative management option. A prompt decision and early resort to arterial embolisation are advisable so as to reduce the morbidity and avoid resorting to open surgery.

Introduction

Primary postpartum haemorrhage (primary PPH) was identified as the most
important cause of maternal mortality and morbidity in Hong Kong public hospitals over the period 1999 to 2004. Uterine or internal iliac arterial embolisation was a new procedure introduced in the public hospitals for the management of severe PPH. The aim of the study was to assess the utilisation, effectiveness, and safety of this new intervention in the management of severe primary PPH.

Methods

This retrospective analysis was co-ordinated by the Quality Assurance Subcommittee of the COC. A survey form (Appendix) was designed and sent to all members of the Subcommittee in August 2004. They were asked to collect data on all cases of primary PPH treated with embolisation procedure over a 5-year period from July 1999 to June 2004.

The following information was retrieved: cause of primary PPH leading to embolisation, estimated blood loss before the decision to embolise, other procedures performed before embolisation, the patient’s condition before embolisation, and the intervals between the diagnosis of PPH and (i) the decision to embolise, and (ii) the start of the procedure. Information was also collected on the hospital location where the embolisation was performed, any further operations or procedures undertaken to stop bleeding, and the outcome (including complications) after embolisation.

Results

Over the 5-year period of this study, there were about 7200 patients with PPH (>500 mL) out of a total of 183 700 deliveries, giving a primary PPH rate of 3.9%. About 90 of these 7200 cases underwent postpartum hysterectomy, which approximates to 1 in 2000 or 0.05% of all deliveries, and 1.25% of patients with primary PPH. In all, 29 patients with primary PPH treated by embolisation were identified in the eight Hospital Authority (HA) hospitals with obstetric services, two of which reported no such procedure. Thus, the embolisation procedure was carried out for 1 (0.016%) in every 6300 deliveries or 0.4% of primary PPH patients (Table 1).

Among these 29 patients, uterine atony was the commonest of the recognised causes of PPH. The frequencies of such contributing causes are summarised in Table 2. Estimated postpartum blood loss at the time the embolisation decision was made varied from 1100 to 20 000 mL, with a median of 3500 mL.

Embolisation was the primary treatment used to control haemorrhage in 26 patients who failed medical therapy (uterine massage and oxytocic agents). In the two patients with vaginal tears, prior surgical repair was attempted but failed to control the bleeding. In one patient, the procedure was performed immediately after caesarean section for placenta percreta.

In 26 patients, their haemodynamic condition was stable; blood pressure and pulse were normal or near normal when the procedure was performed. In the three remaining patients, before the procedure the blood pressure was below 90/50 mm Hg and the pulse rate exceeded 100 beats/min. One of these patients sustained a cardiac arrest during the embolisation procedure and was successfully resuscitated.

After excluding the patient with planned embolisation, details regarding the intervals between (i) delivery of the baby and initiation of the embolisation procedure, (ii) diagnosis of PPH and embolisation, and (iii) the decision to embolise and the start of the procedure are summarised in Table 3. Embolisation was undertaken within 30 minutes of the decision in 10 patients and within 60 minutes in 10 more; nine had the procedure more than 60 minutes later.

Except for one, all embolisation procedures were performed in the department of radiology on an emergency basis because of severe PPH. In one patient the embolisation was pre-planned before the delivery. She was diagnosed to have placenta percreta on ultrasonography and was scheduled for caesarean section. An embolisation catheter

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Table 1. Obstetric data from July 1999 to June 2004

<table>
<thead>
<tr>
<th>Obstetric variable</th>
<th>No. of patients</th>
<th>% of total deliveries</th>
<th>% of primary postpartum haemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deliveries</td>
<td>183 700</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary postpartum haemorrhage</td>
<td>7200</td>
<td>3.9</td>
<td></td>
</tr>
<tr>
<td>Postpartum hysterectomy</td>
<td>90</td>
<td>0.05</td>
<td>1.25</td>
</tr>
<tr>
<td>Arterial embolisation</td>
<td>29</td>
<td>0.016</td>
<td>0.4</td>
</tr>
</tbody>
</table>

Table 2. Causes of postpartum haemorrhage in patients treated by embolisation

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. of patients*</th>
<th>% of total*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uterine atony</td>
<td>18</td>
<td>62</td>
</tr>
<tr>
<td>Abnormal placentation</td>
<td>-</td>
<td>34</td>
</tr>
<tr>
<td>Placenta previa</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td>Placenta acreta</td>
<td>3</td>
<td>-</td>
</tr>
<tr>
<td>Placenta percreta</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Coagulation problem</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Vaginal tear</td>
<td>2</td>
<td>7</td>
</tr>
</tbody>
</table>

* Because three patients had more than one cause, the numbers of causes do not total 29 and percentages do not total 100.

Table 3. Time intervals to start embolisation

<table>
<thead>
<tr>
<th>Time interval from</th>
<th>Range</th>
<th>Mean</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delivery</td>
<td>2-12 h</td>
<td>5 h</td>
<td></td>
</tr>
<tr>
<td>Diagnosis of primary postpartum haemorrhage</td>
<td>1.5-12 h</td>
<td>-</td>
<td>3.5 h</td>
</tr>
<tr>
<td>Decision to embolise</td>
<td>15-120 min</td>
<td>56 min</td>
<td>-</td>
</tr>
</tbody>
</table>
was inserted in the department of radiology before the caesarean section, but the procedure was performed in the operating theatre when uncontrolled bleeding was encountered after delivery of the baby.

Three (10%) of the 29 patients required further treatment after embolisation and were considered failures. The first had uterine atony after caesarean section. Before embolisation her blood pressure was only 80/40 mm Hg and the pulse rate was 110 beats/min. She had further heavy bleeding and sustained a cardiac arrest during the procedure. After a successful resuscitation, hysterectomy was immediately performed. The bleeding was eventually controlled and she recovered without further complications. The second patient also had shock and uterine atony after caesarean section; her pre-embolisation blood pressure was 68/32 mm Hg. Bleeding continued after the procedure and only stopped after uterine packing. The third patient had the planned embolisation for placenta percreta. Although haemodynamically stable before embolisation, bleeding continued after the procedure and a hysterectomy was performed. All 29 patients survived their procedure. However, one patient had a cardiac arrest, another developed transient right-leg claudication and six experienced transient mild fever.

Discussion

As in previous reports, in our series arterial embolisation was safe and effective in controlling severe PPH, irrespective of the underlying cause; the overall success rate was 90%. Abnormal placentation typically accounts for over half of the failures, in which success rates of between 62 and 71% have been described. In the 10 patients with abnormal placentation in our series, we encountered only one failure, which occurred in association with the most severe form of morbidly adherent placenta.

Hypovolaemic shock with haemodynamic instability and rapid profuse bleeding appear to be important factors associated with failure of the embolisation. Two of our three patients had significant pre-procedure hypotension before undergoing further intervention to control the bleeding. One of them developed cardiac arrest during the procedure, but whether embolisation precipitated the event in such a compromised patient is unclear. Given the high risk of failure and potential for cardiac arrest, patients with haemodynamic instability and profuse bleeding should be managed surgically. Nevertheless, in keeping with other reports, our data suggest that arterial embolisation is a safe procedure.

Despite its high efficacy in controlling severe PPH, arterial embolisation appears to be underused in the HA hospitals. Two of eight obstetric service hospitals did not provide such a service and over the past 5 years, only 29 arterial embolisation procedures were performed. This accounts for 0.016% of all deliveries or 0.4% of patients with PPH; such proportions being much lower than the respective figures (0.08% and 2.19%) were detailed in a recent report. The likely reasons for these low rates locally are the unavailability of interventional radiologists in some hospitals or the lack of such a 24-hour service.

Postpartum haemorrhage is a potentially fatal obstetric emergency and carries serious morbidity. Although medical management and obstetric manoeuvres such as uterine massage are the first line of treatment, delay in controlling the bleeding may result in severe hypovolaemic shock, disseminated intravascular coagulation, and hepatorenal failure, all of which worsen the prognosis. If severe haemorrhagic shock is not treated and reversed within the first ‘golden hour’, the chance of survival diminishes. It is imperative to initiate resuscitation within the first 60 minutes of catastrophic haemorrhage and continue with appropriate treatment as needed. According to our findings, the mean period elapsing between the diagnosis of PPH and the procedure represents an excessive delay in the decision to embolise is another important consideration. Similar published data from elsewhere are not available for comparison. The clinician’s understanding and skill in various treatment options for PPH, interaction between junior and senior staff and other relevant disciplines, availability of adequate resources, and patient characteristics may all be factors that influence time taken to treat severe PPH.

Prophylactic (preoperative) catheterization of the uterine or internal iliac arteries before elective delivery in patients at high risk may help prevent primary PPH. Ironically our experience with only one case of prophylactic catheterization turned out to be unsuccessful, possibly because the patient had the most severe form of morbidly adherent placenta (placenta percreta). Prophylactic catheterization allows embolisation to be performed in the operating theatre immediately after delivery of the baby, thus facilitating early hysterectomy if the bleeding remains uncontrolled. Importantly, prophylactic catheterization before childbirth has been reported to be a promising tool for the control of bleeding and preservation of fertility.

Genital tract arterial embolisation was originally described in 1979 for the treatment of persistent bleeding after postpartum hysterectomy for severe primary PPH and was recommended by the Society of Obstetricians and Gynaecologists of Canada for management of such cases. It has been used for more than 20 years to control PPH and should be offered irrespective of the underlying cause. The current management options for severe PPH are legion, and new modalities have recently become available. In a
haemodynamically stable patient not responding to medical treatment with uterotonics (including oxytocics, ergot alkaloids, or prostaglandins), early consideration and implementation of arterial embolisation has several advantages, compared to immediate surgical interventions such as internal iliac artery ligation or hysterectomy under general anaesthesia. First, embolisation allows the uterus to be preserved thereby enabling the possibility of another pregnancy. Second, angiographic catheter placement under fluoroscopy allows precise localisation of the bleeding and collateral vessels, facilitating targeted embolisation to prevent bleeding (including that via the collateral circulation), which may explain its higher success rates (>90%) compared with hypogastric artery ligation (40-75%). Third, it avoids the risks associated with laparotomy and major surgery and general anaesthesia. Finally, if embolisation fails, hysterectomy can still be performed. However, it is not safe for unstable patients with heavy bleeding who need to be transferred to a distant radiology suite for angiographic embolisation. The ideal set-up for embolisation is to have a delivery suite or operation room equipped for angiography and a dedicated radiology team on standby. Other useful treatment modalities not addressed in our survey include tamponade using a urologic (eg Rusch) balloon catheter, a Sengstaken-Blakemore oesophageal catheter or uterine packing. These may have an important role, especially in haemodynamically unstable patients with severe PPH in the presence of coagulopathy. Such interventions are relatively straightforward and can be implemented rapidly, buying time to correct any coagulopathy and/or blood loss in preparation for definitive surgery. Tamponade or packing may also arrest bleeding, thus avoiding the need for surgery altogether. More recently, recombinant activated factor VIIa (Novo seven) has become available for the management of intractable intra-abdominal haemorrhage, which may also have a role in the non-surgical treatment of severe PPH. In patients who have severe PPH during a caesarean section, other efficacious surgical manoeuvres may be offered. These include: stepwise arterial ligation (of the uterine-ovarian then hypogastric arteries), undersuturing of the placental bed site, or brace (eg B-Lynch) suturing. If such methods fail then a hysterectomy should be performed without delay. Angiographic embolisation has a definite role in patients after vaginal delivery, provided significant bleeding from retained products has been excluded since this intervention allows treatment without resort to laparotomy. Arterial embolisation can also be performed in postoperative patients who continue to bleed, but may be impossible if internal iliac artery ligation has been performed. Angiographic embolisation could have a role in placenta previa patients with suspected morbid placental adherence, who already have an angiographic catheter in-situ preoperatively.

Angiographic embolisation is an effective though underutilised treatment modality for severe intractable PPH, but it is by no means the only modality. Its use should be limited only to haemodynamically stable patients for which it can lead to a favourable outcome. Thus, it is important that medical staff understand its indications and limitations. Awareness of the safety and effectiveness of this procedure is likely to increase the popularity of this treatment option, but will depend on an on-call interventional radiology service. Such a service should be available in all large obstetric units.

Conclusions

Uterine or internal iliac arterial embolisation is an effective and safe procedure for treatment of severe primary PPH in haemodynamically stable patients. It allows preservation of the uterus and allows patients a chance to have further pregnancies. In Hong Kong, the procedure is underutilised for the treatment of primary PPH. In selected patients, prompt decisions and early resort to arterial embolisation are advisable in order to reduce the morbidity associated with severe PPH and avoid open surgery. The procedure requires an interventional radiologist and is mostly performed in the department of radiology. Ideally, all major hospitals with obstetric units should consider installing angiographic equipment and radiolucent operating tables in the operating suite. The patient’s condition must be stabilised before carrying out the procedure. In addition to pelvic angiographic embolisation, medical staff should be aware of the range of different available treatment options for PPH. In unstable patients with heavy intractable bleeding, the latter should be considered. Obstetric units should create and regularly update their own set of protocols or guidelines on the management of PPH, in order to keep up with emerging new technologies and resources in their institution. Prophylactic insertion of an embolisation catheter in the internal iliac or uterine artery may be considered in high-risk cases, but requires further study on its clinical effectiveness and feasibility.

References

Arterial embolisation for primary postpartum haemorrhage

Appendix

Survey on the management of primary postpartum haemorrhage (PPH) with the uterine artery/internal iliac artery embolisation procedure

1. What was the amount of blood loss before the embolisation procedure was decided?
2. What was the cause of the PPH?
3. What other procedures and/or operations were undertaken before the embolisation (in order of sequence)?
4. What was the condition of the patient immediately before the embolisation?
5. What was the interval between the completed delivery and the embolisation?
6. What was the interval between the diagnosis of PPH and the embolisation procedure?
7. What was the interval between the decision to embolise and the start of the procedure?
8. Where was the embolisation procedure undertaken: X-ray department, operating theatre, labour ward, others (please specify)?
9. Were further operations or procedures undertaken to stop bleeding after the embolisation?
10. What was the outcome of the patient: alive or dead? If alive, were there any residual problems? Were there any complications about the embolisation procedure?