AN UPDATE ON MANAGEMENT OF MASSIVE POSTPARTUM HAEMORRHAGE

MEHR UN NISA OBAID
Northampton General Hospital, Northampton, West Midlands, U.K.

Some postpartum bleeding is inevitable but not all cases need intervention. It is when postpartum haemorrhage is excessive and does not stop that active management becomes mandatory. When blood loss is in excess of 500 ml, it is taken as significant and between 1000 and 1500 ml is termed massive.[1] The causes of excessive postpartum bleeding are many fold but abnormal placentation because of defect in the materno-trophoblastic interaction is common to most cases.[2] It should be remembered that blood loss is always underestimated.

Postpartum haemorrhage (PPH) remains an important cause of maternal mortality although maternal mortality as such has declined generally. Because of lack of expert obstetric help, women in the underdeveloped countries fall prey to ill management of the third stage of labour. Home deliveries by untrained traditional midwives, delay in reaching the hospital and all this on top of pre-existing anaemia and malnutrition push the mortality curve further up. An 8-year study of maternal mortality in eastern Himalayan region showed a maternal mortality of 55 per 10,000 and postpartum haemorrhage accounted for more than one fifth of deaths.[3] Active management of the third stage of labour with administration of oxytocic drugs significantly reduces blood loss and brings down the incidence of postpartum haemorrhage from 10% to 6%.[1] Pierre and colleagues studied the effects of oxytocic drug in the third stage of labour in 1000 patients and made the same observation.[4]

The patient may have delivered at home or in the hospital; in either case a full appraisal of the history is essential. Antenatal record is of utmost significance. However, in the set up of a third world country this might not be possible.

History

Certainly history about the parity is important. In one study, primipara were found more prone to obstetric haemorrhage than multipara.[5] Past-obstetric history throws light on any recurrent problem. Uterine rupture is more common amongparous women and those who had previous caesarean sections.[6] The incidence further increases if such patients are given oxytocin and epidural anaesthesia.[7] Any complication during the current pregnancy might have a contribution to the present problem like a continuing antepartum haemorrhage. The incidence of PPH increases with induction of labour especially if it proceeds sluggishly.[6]

Details of medications are taken as they do affect coagulation and vascular response to haemorrhage. For example, prophylactic use of oxytocin rather than

syntometrine prolongs bleeding.[5] Knowledge of second stage helps to exclude causes such as dystocia and bleeding from episiotomy wound or other trauma to the cervix and vagina. It should be ascertained whether the placenta has been delivered. Retention of placental cotyledons can cause serious obstetric haemorrhage. Uterine rupture gives rise to severe degree of shock with agonising pain. Uterine atony is another important cause of PPH which responds to intravenous ergometrine or/and oxytocin. If bleeding does not slow down with these drugs, uterine atony should be reviewed and another cause for severe bleeding must be sought. Any past history of bleeding disorder should alert one to the possibility of coagulation defect.

If the medical record is available, particular note should be made of her blood group and last haemoglobin. Various allergies and use of any drugs should also be noted. An important question from anesthetic point of view would be about the time of last eating and drinking.

Clinical Examination

An accurate assessment of the amount of blood loss should be made. It should be remembered that even experienced obstetricians often underestimate the loss. By the time the patient is examined, she might not be bleeding. This is because she is in shock and all the vessels are collapsed[8] As this assessment is crucial to accurate management, it should be carried out in good light and sufficient space not only to find the source of bleeding but also its rate and to continue effective resuscitation at the same time.[6]

The general appearance of shock is usually evident from the first look of the patient. Gross pallor, listlessness, extreme thirst etc are good hints to the seriousness. Tachycardia as a compensatory mechanism of the cardiovascular system does not always work. At times there is paradoxical vagally stimulated bradycardia. In a series of 273 patients in haemorrhagic shock, 7% had bradycardia with unrecordable systolic blood pressure.[9] It was more evident in patients with more severe and rapid blood loss.[1] Similarly blood pressure is usually maintained by extreme vasoconstriction till very late. Respiratory efforts are evidence of lack of oxygenation.

During this quick assessment, abdomen is also examined. A tense tender abdomen with signs of peritonism may suggest ruptured uterus. Uterine atonia might be evident from abdominal palpation. Uterine size is documented. A quick examination of the vulva and vagina is also done to exclude any trauma there as the cause of the bleeding.

Immediate Management

After this initial quick assessment, two wide bored cannulae should be inserted in two different veins and at the same time blood samples should be taken for full blood count, urea and electrolytes, coagulation profile and 4-6 units of blood cross matched, depending on the estimate of blood loss. A volume expander like haemaccel should be set up immediately until cross matched blood is available. Very rarely unmatched O Rh Negative blood would have to be transfused. If the source of bleeding is thought to be non-contracting uterus or adherent placenta, intravenous oxytocin or IM ergometrine is considered. Use of intravenous prostaglandins may also be considered in such a situation.

It is important that as soon as the initial assessment is complete and the volume expander started, someone senior on the obstetric side is contacted and

further decisions are taken with mutual consultation. Similarly the anaesthetist is involved in the management right from the beginning.

Longer Term Management

Once the patient is stable and resuscitated, a detailed history is taken again and complete medical examination is done. A provisional diagnosis is made and a likely source of bleeding determined. A decision is made whether to proceed to theatre or continue with the conservative management. The seriousness of the situation usually leads to the former. Before shifting the patient to operation theatre, a fully informed consent is taken from the patient. Next of kin of the patient is also taken into confidence as the patient may not be in a position to understand.

The occurrence of coagulation defects including disseminated intravascular coagulation is well known in massive obstetric haemorrhage and accounts for large proportion of maternal deaths due to haemorrhage.[10] Massive transfusion itself can prolong prothrombin and partial thromboplastin time as well as reduce the platelet count. It might thus perpetuate bleeding. DIC can be recognised by prolonged thrombin time and reduction in fibrinogen level.[11] If continued haemorrhage is managed by transfusion of stored blood, coagulation factors are quickly depleted. Fresh blood and fresh frozen plasma (FFP) should be given to replace some of the factors and fibrinogen. Burke and Duignan recommend one unit of FFP for every four to five units of stored blood transfused.[6] Platelet transfusion still carries a risk of infection because the concentrate is obtained by pooling platelets from many sources.

If the patient is suffering from von Willebrands's disease or Christmas disease or is a carrier for haemophilia, cryoprecipitate will be needed. Immune Thrombocytopenic purpura (ITP) is encountered more in young women and the established treatment is high dose intravenous gamma globulin. It gives protection to both the mother and the fetus. Repeated doses may be needed if ITP is of severe form.[6]

Medical treatment of arresting postpartum haemorrhage is carried out if the cause is atonic uterus. Intravenous ergomerine or synthetic oxytocin are the most effective drugs in most cases. Prostaglandin F_2 analogues have also successfully been used for intractable uterine haemorrhage.[12] They can be used intramuscularly as well as directly into the myometrium.[13] However, prostaglandin should be used only with caution especially when the patient is volume deficient as these agents reduce peripheral resistance.

If bleeding is not arrested by these means, the diagnosis of uterine atony should be excluded or reconsidered. The patient should be quickly prepared for anaesthesia and shifted to the operation table. There is no role of uterine packing in the management of PPH today

Ultrasound examination of the uterue is the most efficient way of diagnosing retained placental tissue. The sonographic appearance of retained placenta is variable but detection of an echogenic mass strongly supports the diagnosis. However, ultrasound examination should be done before any instrumentation as iatrogenically introduced air can cause confusion.[14]

Once the patient is anaesthetised, a through examination is done when the patient is fully relaxed. A urinary catheter is inserted into the bladder to aid in the examination as well as monitor urinary output.

Examination of the vulva, vagina and cervix will quickly show any tear in

these structures and they can be repaired under good light. Examination of the uterus will reveal any retention of the placenta which can then be evacuated manually. Placental products can also be evacuated under epidural anaesthesia if the diagnosis is pretty certain.[15] Traditional manual evacuation is assisted by intravenous Oxytocic drugs. In an initial report Carroli suggests that direct injection of oxytocin into the umbilical vein reduces the need for manual evacuation.[16] Rupture in the uterus will be evident in which event a formal laparotomy is mandatory. A haematoma in the broad ligament can also be palpated in the fornices and needs evacuation by formal laparotomy.

Laparotomy

Pfannensteil incision is quite adequate for exploration of the pelvic organs. Haematoma in the adnexa and/or broad ligament can be evacuated by incising peritoneum over it. A suction drain should be left in the bed before closing the wound.

If the uterus is ruptured, attempts can be made at its repair if the tear is small and condition of the patient permits tedious surgery. Rupture of lower segment caesarean scar can usually be repaired. However, rupture of the classical caesarean scar or traumatic rupture frequently requires hysterectomy.[6] If the patient has completed her family and/or condition of the patient does not permit attempts at repair, a formal hysterectomy should be performed.

Hysterectomy is also the most effective method of controlling PPH due to persistent uterine atony and morbidly adherent placenta.[17] Subtotal hysterectomy is simpler and speedier to perform and is effective in most cases. Total hysterectomy is reserved for tears involving the lower segment or placenta accreta. In any case, the procedure should be conducted by senior medical personnel both on the anaesthetic and surgery side. Smith showed in his report that mortality of obstetric hysterectomy increases if performed or anaesthetised by lower than consultant level.[18]

Internal Iliac Artery Ligation

Ligation of both internal iliac vessels reduces pulse pressure distally rather than totally arresting haemorrhage because of extensive collateral circulation. The approach is transperitoneal which is incised lateral to the ureter and further dissection is done bluntly. The anterior division of the iliac artery is ligated with a non absorbable suture. Alternatively an absorbable suture may be used which will allow recanalisation of the vessel later on.[19] This procedure is most effective in cases of uterine atony and midline perforation.[20] Various rates of complications have been reported and efficacy of the procedure has been doubtful.[21]

Angiographic Control of Haemorrhage

Where radiology department is well equipped, the bleeding vessel can be visualised and embolised with gelatin sponge via femoral artery.[22] Aortography should be repeated to confirm that the haemorrhage has been arrested. This method of haemostasis is still in its initial stages and the number of patients benefited is slowly growing. This technique can also be applied if the bleeding does not stop after hysterectomy and internal iliac artery ligation.[23] Otherwise it is a great life and fertility saving procedure.[24]

Vasoconstrictive agents like Dopamine and Pitressin have also been used directly into the internal iliac

vessels for the control of obstetric haemorrhage.[25,26]

Post Operative Care

Postoperatively the patient should be closely monitored. If the anaesthetist or the surgeon is worried, the patient should be transferred to intensive care unit for the first day or so. If the urinary output and infused fluids do not tally, a CVP line should be considered. CVP line will also be helpful if pulmonary oedema is feared because of over infusion.

Because of the long operative time and extensive dissection as well as the presence of haematomas, it is advisable to start the patient on a broad spectrum antibiotic right from the start.

If the patient has any history of bleeding diathesis or any suggestion on coagulation profile, close liason with the haematologist should be achieved. The bleeding and coagulation time of the patient should be brought to as normal as possible with whatever factor is deficient.

REFERENCES

1. Von Dongen PW, et al. Oxytocics for prevention of postpartum haemorrhage. A review. Pharma-Weekbl. 1991; 13: 238.

2. Khong TY and Sawyer IH. The human placental bed in health and disease. Reprod-Fertil-Dev. 1991; 3: 373.

3. Ray A. Maternal mortality in a subdivisional hospital of eastern Himalayan region. J Indian Med Assoc. 1992; 90: 124.

4. Pierre F, et al. For a systemic policy of IV oxytocin inducted placenta deliveries in a unit where a fairly active management of third stage of labour is yet applied: results of a controlled trial. Eur J obstet Gynaecol Reprod Biol. 1992; 43: 131.

5. Gilbert L, Porter W and Brow V. A Postpartum haemorrhage a continuing problem. Br. J Obstet Gynaecol. 1987; 94: 67.

6. Burke G and Duignan NM. Massive obstetric haemorrhage. Progress in obstetrics and Gynaecology, Churchill Livingstone London, Vol 9. 1991; 111.

7. Molloy B, et al. Delivery after Caesarean section review of 2176 consecutive consecutive cases. Br Med J. 1987; 294: 1645.

8. Shweni PM, Bishop BB and Hensen JN, et al. Severe post-partum haemorrhage after Caesarean section. S Afr Med J. 1987; 72: 17.

9. Barriot P and Riou B. Haemorrhagic shock with paradoxical bradycardia. Intensive Care Med. 1987; 13: 203.

10. DoH. Report on confidential Enquiries into Maternal Deaths in England and Wales, 1982-84. 1989. Her Majesty's stationary office, London.

11. Letsky E. Haemostasis in pregnancy. Morgan BM Foundations of obstetric anaesthesia Ferran Press, London, 1987, Chap 12.

12. Hayashi RH, Castillo MS and Noah ML. Management of severe postpartum haemorrhage due to uterine atony using an analogue of prostaglandin F.2a Obstet. Gynaecol. 1981; 58: 426.

13. Bruce SL, et al. Control of postpartum uterine atony by intramyometrial prostaglandin. Obstet Gynaeco. 1982; 59: 47.

14. Hertzberg BS and Bowie JD. Ultrasound of the postpartum uterus. Prediction of retained placental tissue. J Ultrasound Med. 1991; 10: 451.

15. Whitfield CR. Complications of third stage of labour. Integrated Obstetrics and Gynaecology for Postgraduates Dewhurst Jed. 1981; Blackwell Oxford: 437.

16. Carroll G. Management of retained placenta by umbilical vein injection. Br J obstet Gynaecol. 1991; 98: 348.

17. Giwa-Osagie. Of mortality and morbidity of emergency obstetric hysterectomy. J obstet Gynaecol. 1983; 4: 94.

18. Smith AM. Emergency obstetric hysterectomy. J obstet Gynaecol. 1982; 2: 245.

19. Dubay ML, et al. Internal artery igation for obsetric haemorrhage recanalisation of vessels. Am J Obstet Gynaecol. 1980; 136: 689.

20. Evanss and McShane, The efficacy of internal iliac artery ligation in obstetric haemorrhage. Surg Gynaecol Obstet. 1985; 160: 250.

21. Clarke SL, et al. Hypogastric artery ligation for obstetric haemorrhage. Obstet Gynaecol. 1985; 66: 353.

22. Greenwood LH, et al. Obstetric and non malignant gynaecologic bleeding: treatment with angiographic embolisation, Radiology. 1987; 164: 155.

23. Duggan PM, et al. Intractable postpartum haemorrhage managed by angiographic embolisation case report and review. Aust NZ J Obstet Gynaecol. 1991; 31: 229.

24. Marpeau L, et al. The role of pelvic arterial embolisation in the treatment of severe

postpartum haemorrhage. Gynaecol obstet Biol Reprod. 1992; 21: 233.

25. Margrina JF, et al. Elective arterial infusion of Pitressin for the control of puerperal haemorrhage after hypogastric artery ligation. Obstet gynaecol. 1981; 58: 646.

26. Mud HJ, et al. Non surgical treatment of pelvic haemorrhage in obstetric and gynaecologic patients. Crit care Med. 1987; 15: 534.