Bimanual Uterine Compression as a Major Technique in Controlling Severe Postpartum Hemorrhage from Uterine Atony

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Abstract

A 27-year old woman, primigravida, 33 weeks' gestation, presented with complaints of labor pain and absent fetal movement. A dead fetus in utero, abruptio placentae, and labor pain were diagnosed. Severe postpartum hemorrhage from uterine atony and disseminated intravascular cogulopathy was noted after spontaneous delivery of the baby and placenta. Bimanual uterine compression for 40 minutes was performed as a major procedure accompanied by uterotonic drugs, correction of hypovolemic shock and coagulopathy by crytalloid, blood, fresh frozen plasma. The patient had no complications when seen at 6 weeks' postpartum follow-up.

Uterine atony, the most common cause of postpartum hemorrhage,[1,2] remains a leading cause of maternal morbidity and mortality. Conservative treatments include uterine massage, bimanual uterine compression, oxytocics, prostanoids and methyl ergonovine. Surgical treatments, including hypogastric artery ligation and hysterectomy, are considered only after failed conservative treatment. We describe bimanual uterine compression for controlling postpartum hemorrhage from uterine atony and DIC in the case of dead fetus in utero and abruptio placentae.

CASE REPORT

A 27 year old primi gravida with a singleton gestation at 33 weeks' gestation, was admitted to a community hospital in Bangkok complaining of labor pain and absent fetal movement. She had no antepartum vaginal bleeding. Her prenatal course was unremarkable. Her prenatal hematocrit was 30 vol per cent. On admission, the patient looked pale and her BP was 90/60 mmHg. The fundal height was 31 cm, and her cervix was 5 cm dilated. A dead fetus in utero was diagnosed by absent fetal heart activity in ultrasonogram. The patient was administered Ringer lactated solution and referred to Rajavithi Hospital. On admission at 9.40 pm on October 28, 1994, her BP was 90/60 mmHg. There were tetanic uterine contractions and absent fetal heart sound. The cervix was fully dilated.

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The provisional diagnosis were primigravida 33 weeks' gestation, abruptio placentae, dead fetus in utero and labor pain. The hematocrit at admission was 18 vol per cent. Possible cause of anemia was abruptio placentae. Blood grouping and cross matching were carried out. The patient went on to deliver a dead female fresh stillborn weighing 2,000 g at 9.46 pm.

The placenta was delivered with 200 ml of adherent blood clot behind the maternal site about 80 per cent of the surface area and was found to be completed. Possible cause of fetal death was the massive fetal hemorrhage behind large maternal surface area of placenta from the process of abruptio placentae. Immediately after the 3rd stage, there was profuse bleeding of approximately 1,000 ml from the introitus. Her BP was 70/40 mmHg, pulse rate was 100 beats/min and consciousness was blurred. Examination of the birth passage revealed no injury. Only uterine atony was noted. The episiotomy was repaired. The uterine tone could not be restored despite admission of 20 units of oxytocin in 1 litre of Ringer lactate solution, 2 doses of methylergometrine maleate (0.2 mg) intravenously and sulprostone 1 mg in 500 ml of 5 per cent dextrose in water. Foley catheter was retained. The urine volume was 200 ml at that time and then hematocrit and coagulogram were carried out. Results showed the hematocrit was 5.5 vol per cent with prolongation of coagulation studies (PT 167.3 sec, PTT 240.4 sec, VCT > 40 min).

At 10.00 pm, active bleeding continued while uterine atony remained. Bimanual uterine compression was performed by massaging the posterior aspect of the uterus with the abdominal hand and massaging through the vagina of the anterior uterine aspect with the other fist, the knuckle of which contacted the uterine wall. During this time, venesection was done and CVP was 4 cm H_2O. Many kinds of tranfused fluid such as 500 ml of hemacceal, 20 units with oxytocin in 1 L of Ringer lactated solution, 1 dose of methylergometrine maleate (0.2 mg) intravenously, whole blood 2 units, fresh frozen plasma (FFP) 4 units were administered.

At 10.10 pm, ten minutes after bimanual uterine compression was performed, the patient lost consciousness and her BP was 40/0 mmHg, pulse rate was 120 beats per min. Endotracheal intubation was done. The same kind of transfused fluid was replaced quickly. Active bleeding continued through the introitus.

At 10.25 pm, the patient recovered consciousness and her BP was 90/60 mmHg, pulse rate was 100 beats/min. The uterine contraction was restored. The vaginal bleeding was diminished and some clotted blood was seen.

Then bimanual uterine compression was continued for fifteen minutes and abdominal uterine compression was performed instead. The hematocrit was 14.5 vol per cent. The total time for this procedure was 40 minutes.

At 11.50 pm, 2 hours and 10 minutes after admission the intake volume was hemaccel 1,500 ml, RLS 1,500 ml, 5 per cent D/W 500 ml whole blood 1,600 ml, FFP 1,000 ml total intake was 6,100 ml the output volume was urine 2,050 ml, blood loss 3,500 ml total output volume was 5,500 ml. Ampicillin and chloramphenicol were given for prophylaxis because of many procedures on her body. The patient was tranferred to the ICU. The endotracheal tube was removed by the patient after her consciousness was completely regained. Her postoperative course was complicated by minimal pulmonary edema, but she responded well to diuretic and fluid restriction.

On the first postpartum day, the vital signs, consciousness and coagulogram were normal. The venesection and Foley catheter were removed from the patient on the second postpartum day and the hematocrit was 25.5 vol per cent. Her breasts' engorgement was noted on the fourth postpartum day. The patient was discharged on the sixth postpartum day. On follow-up after 6 weeks postpartum, she had had her menstruation two day before. The whole body and pelvic exammination were within normal limits.

DISCUSSION

The most common cause of postpartum hemorrhage is uterine atony[1-4]. It was one of the causes in our case. The first event in our case possibly was abruptio placentae with concealed hemorrhage and/or Couvelaire uterus because the blood did not escape externally. This type of abruptio placentae carries more complications because the likelihood of intense consumptive coagulopathy is increased[4]. It was also severe enough to kill the fetus. But consumptive coagulopathy in this case possibly was not caused by the

dead fetus because it rarely develops before 1 month after fetal death[4].

The causes of postpartum hemorrhage include poor myometrial contractility following infiltration with blood as in Couvelaire uterus, and the inhibitory effect of fibrin degradation products (FDPs) on myometrial contractility in disseminated intravascular coagulopathy (DIC)[5].

Severe postpartum hemorrhage provides a poorly profused myometrium from hypovolemic shock, causing the more severe uterine atony.

Uterine massage and manual compression are the first measures in the treatment of atony[6]. Adequate crystalloid and blood replacement should be given at a rate to maintain a urine output of 30 ml or more per hour[3]. There are two types of bimanual uterine compression[7].

a - the whole hand is introduced into the vagina, the fist is clenched and the whole uterus is squeezed between the fist and external hand.

b - the hand in the vagina grasps the cervix and the external hand grasps the fundus and doubles the body on the cervix.

In our case, we performed the first type of bimanual uterine contracton. Pressure, exerted against the posterior aspect of the uterus by the other hand through the abdomen with a rotatory motion, the uterus is compressed and massaged between the two hands. This provides twice the amount of uterine stimulation that can be achieved by abdominal massage alone[8].

Ian Donald commented that physicians could usually perform bimanual uterine compression for only a few minutes because of fatigue[9]. O'Conner TC and Cavanagh D suggested 5 minutes of bimanual uterine compression but should be continued for 30 minutes if there was no response in uterine contraction[10]. Queenan JT suggested that the response should occur within 5-15 min. If the uterus does not contract and bleeding dose not stop, institution to uterine packing and surgical therapy such as hypogastric artery liagtion and hysterectomy were performed[11].

We performed bimanual uterine compression for 40 minutes before the successful control of postpartum hemorrhage while correcting the volume depletion coagulopathy and uterine contraction promotion together.

Bimanual uterine compression promotes myometrial contraction effect mechanical compression of the uterine arteries, compression of the venous sinuses and the flow of blood reduced.

After performing bimanual uterine compression for ten minutes, we failed to control the bleeding. But we didn't do uterine packing because we had no experience and the uterus may dilate under the packing, with further concealed hemorrhage that may be fatal.

Surgical therapy was not chosen for the patient because she was a primigravidarum, so we tried conservative treatment in order to save her reproductive life.

Thirty minutes later, we successfully controlled the postpartum hemorrhage and continued this procedure for another fifteen minutes.

Compared with the longest duration of bimanual uterine compression (30 minutes) suggested by O'Conner TC and Cavanagh D,[10] our duration of the procedure was longer (40 minutes) for evaluation of the response of uterine contraction.

For our success, manpower was an important factor. Our team consisted of the obstetric staff, our residents, anesthetic resident, nurses and medical students.

Medication such as blood components, intravascular fluid, oxytocic drugs as well as bimanual uterine compression played an important role in stimulation of uterine contraction. Finally we avoided laparotomy in controlling the postpartum hemorrhage from uterine atony and disseminated intravascular coagulopathy.

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การใช้วิธีกดมดลูกด้วยมือเป็นวิธีสำคัญในการรักษาภาวะตกเลือดหลังคลอดที่รุนแรงจากภาวะมดลูกไม่หดรัดตัว

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ผู้ป่วยหญิงไทยคู่ อายุ 27 ปี ครรภ์แรก 33 สัปดาห์ มาโรงพยาบาลด้วยเรื่องทารกไม่ดิ้น 1 วัน ตรวจพบมีมดลูกหดรัดตัวแข็งตลอดเวลา และฟังเสียงหัวใจทารกไม่ได้ ได้รับการวินิจฉัยว่า ทารกเสียชีวิตในครรภ์ เจ็บครรภ์และมีภาวะรกลอกตัวก่อนกำหนด ได้รักษาโดยการให้คลอดปกติทางช่องคลอด หลังคลอดมีภาวะตกเลือดจนช็อคจากมดลูกไม่หดรัดตัวและภาวะเลือดแข็งตัวผิดปกติ ให้การรักษาหลักโดย bimanual uterine compression เป็นเวลา 40 นาที ร่วมกับการให้สารที่ช่วยทำให้มดลูกหดรัดตัว การแก้ภาวะช็อคโดยการให้สารน้ำ เลือด และพลาสมา ทดแทน หลังคลอด 6 สัปดาห์ ผู้ป่วยอาการปกติดี ไม่มีภาวะแทรกซ้อนใดๆ

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