EXCESSIVE PRIMARY POSTPARTUM HEMORRHAGE

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EXCESSIVE PRIMARY POSTPARTUM HEMORRHAGE (Abstract). The diagnosis and treatment of excessive bleeding within the first 24 hours after delivery and its main causes, namely uterine atony, retained placental fragments, vascular lesions by local tissue rupture, and blood clotting disorder are presented. Of great interest is the iconography, which is suggestive and original. Keywords: EXCESSIVE, OBSTETRIC, HEMORRHAGE, IMMEDIATE POSTPARTUM.

Excessive primary postpartum hemorrhage (after vaginal or C-section delivery) is often accompanied by an abnormal prenatal coagulogram (specific for obstetrics), which can be a warning sign. It has four major causes: uterine atony (in the case of malformed uterus, fibromyomatous uterus, over-distended uterus due to multiple pregnancy, polyhydramnios or fetal macrosomia, uterine muscle exhaustion after prolonged labor or uterus of an obese mother); retained placental or membrane fragments; uterine rupture, tears of the cervix, vagina, vulva, pelvic floor; coagulation disorders, most commonly disseminated intravascular coagulation (DIC) and secondary fibrinolysis in: uteroplacental apoplexy, dead fetus in utero, amniotic fluid embolism, chorioamnionitis, septic shock, even a severe acute hemorrhage, hemorrhagic shock, placenta previa, placenta accreta, preeclampsia-eclampsia etc. Bleeding can be external, internal or mixed.

Clinical causal assessment:
- uterus greater than 20 cm/10 cm; low uterine tone – examination of placenta and membranes (evidence of missing placental fragments, membranes);
- coagulability or non-coagulability of blood in the renal pelvis;
- examination of the birth canal: manual exploration of the uterine cavity (for the presence or absence of retained placental fragments and uterine rupture);
- speculum exam for cervical tears; inspection of the external genitalia and perineum.

Simple tests that can quickly pinpoint the diagnosis (1):
- Low platelet count: 15-20,000 indicate a DIC;
- Collection of blood in a 3.5 ml test tube without anticoagulant:
  - a small clot with a red blood cells deposit formed at bottom of the tube suggests DIC;
  - a clotting time (CT) exceeding 10 minutes means a drop of fibrinogen level below 100 mg%;
  - clot formation and its resolution within 30 minutes denotes secondary fibrinolysis;
- the total non-coagulability of blood is indicative of primary fibrinolysis. These tests require laboratory confirmation (1).
Management of uterine atonia:

- compression of abdominal aorta for 5-15 minutes, uterine massage;
- in the already installed IV infusion set introduce 10-15 U oxytocin and/or ergometrine or methergin 0.2 mg or intramuscularly 15-methyl PGF₂α; bleeding persists or recurs.

The uterine cavity is explored manually (for removing the retained placental fragments and checking for uterine tears, especially after manual detachment and removal of placenta or other obstetrical procedure or maneuver. If the uterus is intact, introduce your right hand into the uterine cavity, clench your fist and use the other hand to massage the uterus through the abdominal wall. If the bleeding persists inject ergomet or prostaglandins PGE₂ or PGF₂ into the cervix (1).

Prostaglandins PGE₂, PGF₂, and PG₂α can be injected most effectively intramyometrially, into the cervix or intravenously and prostaglandin analogue sulprostone (15-methyl PG₂α) intramuscularly (1).

It is preferable that intramyometrial (and into the cervix) or intravenous injections to be made after the manual inspection of the uterine cavity. Bleeding persists. Grasp the cervix with forceps (with six claws) into the four cardinal points and use them to strongly pull it down (thus an angle bend of the uterine artery that diminishes the bleeding is obtained). In modern obstetrics, intrauterine massage is no longer indicated (2).

In addition to these procedures, Luca (1) mentions the placement of hemostatic threads in "X" on the cervicoisthmic junctions or Henckel forceps on commissures.

In parallel the following are done: correction of volemia, the necessary volemic expansion with blood transfusions (blood collected within the last 6 hours), fresh frozen plasma (thawed), macromolecular solutions, isotonic NaCl or glucose solutions, and correction of coagulability.

Inspection of the uterine cavity is also indicated in case of tears of cervical commissures extending to the vaginal fornix in order to detect a possible commissural tear on the lateral aspect of the segment. If the uterus is not intact, a median sub umbilical laparotomy is performed.

Recent linear segmental cervical ruptures, without infiltration of the myometrial walls, with vaginal wall disinsertion and without broad ligament hematoma are managed by segmentoraphy plus suture of the intravaginal part of the cervix, followed by reinsertion of the detached portion of the vagina and abdominal drainage (plastic tube).

Uterine rupture accompanied by ileal pelvic hematoma increasing in size requires the identification of ureteral tract, external iliac artery and hypogastric artery with ligation of the latter to control the bleeding vessel responsible for causing hematoma. Identification of this vessel when pelvic anatomy is distorted by disease is very challenging, very bloody and very dangerous for ureter. For these reasons bilateral hypogastric artery ligation (BHAL) should be performed (3).

In extensive repairs, after protective hypogastric artery ligation, in case of an anfractuous rupture with wide myometrial serohematic infiltration or signs of infection, degenerative lesions (apoplectic), placenta previa (frequently accreta) or placenta accreta, total hemostatic hysterectomy is performed followed by adnexectomy on hematoma side only in case of massive hemorrhagic infiltration of adnexa. The next step is hematoma removal and hemostatic drainage of hematoma area and pelvis with externalization of the surgical dressing through vaginal section with visceral peritonization (4).

In case of rupture of the uterine cervix, vagina, vulva and pelvic perineal-floor a hemostatic suture is performed.
Blood clotting disorders

The risk of excessive postpartum bleeding is higher in mothers with preexisting clotting disorders (such as idiopathic thrombocytopenic purpura, Willebrand disease, prothrombin complex deficiency) or most commonly in those presenting a disease at risk of hemorrhagic events by disseminated intravascular coagulation and secondary fibrinolysis, such as: uteroplacental apoplexy, fetal death and retention of dead fetus in utero, chorioamnionitis, septic shock, placenta previa or accreta, amniotic embolism, preeclampsia, eclampsia or even the associated severe bleeding, hemorrhagic shock, etc.

Thus, if after delivery mother’s blood hemostasis is within normal range, then prompt surgical hemostasis (as soon as possible after the occurrence of bleeding) is the essential therapeutic factor and must be associated with volemic restoration (expansion) with crystalloid solutions, macromolecular solutions, fresh blood (preferably collected within the last 6 hours), fresh frozen plasma (thawed), cryoprecipitate (containing fibrinogen, factor VIII, accelerators), platelet concentrates etc. Thus, hemorrhagic shock can be prevented or reversed (5).

Abundant bleeding with clotting disorders is approached in the same manner. The essential part of therapy is prompt surgical hemostasis (“save blood”) concomitant with the replacement of the lost circulating blood with fresh blood, fresh frozen plasma (thawed) in a higher proportion to the volume lost; fresh blood and fresh frozen plasma (thawed) provide coagulation factors (fibrinogen, factors VIII, V, etc.), macromolecular solutions, cryoprecipitate (administered if fibrinogen level is less than 100 mg%), platelet concentrates (administered if platelet count is below 60,000/mm). These are the primary and basic therapy of the hemorrhage syndrome.

In addition to these, in excessive secondary fibrinolysis (strongly confirmed by very short ECLT-euglobulin clot lysis time), Merger et al. (6) recommend the administration antifibriyonolitic drugs, such as Iniprol or Trasylol (50-100,000 U). Arias (7) suggested that “there is no place for the use of heparin in obstetric hemorrhages”. Luca (2000) showed that in trans catheter embolization of hypogastric arteries, the procedure time of selective catheterization is 1-2 hours (to be well done) and this time is often fatal. EACA (Epsilon-aminocaproic acid) is contraindicated (1).

In severe cases when even on correct treatment bleeding persists, bilateral uterine artery ligation (on the lateral margins of uterus), the bilateral hypogastric artery ligation, total hysterectomy and the pelvic excavation packed tightly with gauze should be performed (8).

Statistics show that of hysterectomies, the subtotal one is obviously most frequently performed (especially for uterine atonia). However, total hysterectomy is performed in cases of uteroplacental apoplexy, placenta previa, placenta accreta, irregular uterine rupture propagated towards the cervix, signs of infection and wide areas of serohematic infiltration within the myometrium.

BHAL results in a decrease in blood pressure in the hypogastric artery branches. During an operation for cervical cancer and pregnancy, in which we performed a preventive hypogastric artery ligation, we found that there was blood flow through the sectioned uterine artery, but pressure was much lower.

Figure 1 shows the trajectory of parietal peritoneal incision (dotted) for the surgical approach of hypogastric artery (uterovesical trunk, peritoneal incision, hypogastric artery external iliac artery, ureter, suspensory ligament of ovary).
Excessive primary postpartum hemorrhage

Figure 1. Trajectory of parietal peritoneal incision (dotted) for the surgical approach of hypogastric artery

Figure 2 is illustrative for median subumbilical laparotomy. The uterus is inverted over symphysis pubis and compresses it and the hands of the surgeon assistant simultaneously compress the lateral margins of the uterus (inferior side) thus blocking the flow in the ascending branches of the uterine arteries and reducing uterine bleeding.

Fig. 2. Hypogastric artery ligation
This way, a provisional uterine hemostasis necessary for performing BHAL is achieved and the pelvis is released (to make BHAL possible). The surgeon will check for surgical hemostasis and clotting at normal blood pressure at the end of surgery.

It should be recalled that in case of a clotting disorder, hemostatic hysterectomy produces new and multiple dangerous hemorrhagic foci (6). That is why it is imperative the clotting disorder to be corrected before hysterectomy.

If hemostasis is uncertain and patient's condition allows it BHAL can be performed before closing the abdominal wall to ensure hemostasis in the postoperative period and avoid reoperation (when BHAL is the only option) (9).

By decreasing blood pressure in the arteries, a clot forming at the level of a vascular continuity solution or in small vessels, or even a platelet plug cannot be grabbed and removed.

CONCLUSIONS

Bilateral hypogastric artery ligation is sometimes effective in achieving hemostasis when performed earlier, reducing intraoperative bleeding and making hemostatic hysterectomy unnecessary. But when the mother is at immediate vital risk, hemostasis can be achieved only by hysterectomy, while by BHAL hemostasis it can be achieved only sometimes.

BHAL reduces bleeding during hemostatic hysterectomy. Thus, BHAL is performed earlier so that in case of failure to have time to perform a hysterectomy

REFERENCES