Bleeding conditions in obstetrics

The severity of the bleeding condition in pregnancy or peripartum depends on the nature, intensity and extent of the bleeding. The most common causes are:

1. **Before birth**:
   - placenta praevia;
   - placental abruption;
   - extrauterine pregnancy.
2. **During childbirth**:
   - uterine rupture;
   - premature recanalization of hemostatic coagulum (increased pressure, increased plasmin activity);
   - failure of hemostatic procedures (ligature).
3. **After birth**:
   - physiological vascular closure disorder;
   - placental residue;
   - hypotonic uterus;
   - afibrinogenemia;
   - transboundary injured birth canal.

According to the pathophysiological process leading to bleeding, we divide the causes of bleeding conditions into:

1. coagulopathy;
2. bleeding due to mechanical reasons.

Coagulation failure can occur on the basis of a hereditary defect (hereditary coagulopathy - hemophilia and other hereditary defects in the plasma-coagulation system) or it is a congenital coagulopathy (autoimmune disease, thrombocytopenia, liver failure, drugs - heparin , oral anticoagulants, antiaggregants, dilution of plasma and platelets after bleeding and volume replacement, DIC). The mechanical causes of bleeding include transboundary tissue damage, placental residues and uterine hypotonia.

Disseminated intravascular coagulation

Disseminated intravascular coagulation is a coagulopathy , which in obstetrics is one of the most common causes of maternal death (after thromboembolic complications). DIC is one of the most serious syndromes in which coagulopathy manifests.

Pathogenesis

There are three key moments in the pathogenesis of disseminated intravascular coagulation:

1. disruption of hemocoagulation balance ;
2. excessive thrombin activity;
3. dysregulation of plasmin activity.

In the fully developed state of DIC, there is uncontrolled activation of thrombin caused by the release of tissue factor into the circulation. Tissue factor is released from traumatized as well as non-traumatized tissues. In traumatized tissues, it is released from hematomas, exposed tissue, endothelium and leukocytes. In non-traumatized tissues, tissue factor is released from cells into the circulation under the influence of cytokines or endotoxin . In this case, DIC is part of SIRS , in sepsis . Systemic intravasal coagulation and numerous microthrombi occur. Subsequently, thrombolysis is activated (high D-dimers), microthrombi damage platelets and they are absorbed in the spleen. Thrombocytopenia occurs. Hemorrhagic diathesis and MODS occur .

Classification of DIC in obstetrics

1. **Acute DIC :**
   - the prothrombotic stage is short, goes unnoticed;
   - nausea, shortness of breath , cyanosis ;
   - usually manifests itself in unstoppable bleeding ;
   - if we do not intervene immediately, there will be a breakdown of the endothelium and uncontrollable bleeding into the mucous membranes and skin.
2. **Chronic DIC :**
   - it can take place covertly, we can find it in the laboratory − ↓ platelets, ↓ fibrinogen, ↓ antithrombin , ↑ aPTT , ↑ D-dimers, ↑ FDP;
   - may arise as part of SIRS or manifest as MODS;
   - is the risk of thromboembolic complications;
   - bleeding starts more slowly;
   - if the cause persists (abscess , infection), decompensation soon occurs and then acute DIC develops.

Risk factors

Risk factors mainly include:

- eclampsia , thrombophlebitis in history;
- HELLP syndrome ;
- coagulation disorders, hemolytic states;
- retention and stillbirth ;
- repeated revisions of the uterine cavity;
- septic birth (miscarriage), placenta accreta, amniotic fluid embolism;
- mole hydatidosa ;
- obesity.

Diagnostics

As a guide, we perform laboratory tests in the delivery room (Lee White, thrombin test) - if positive, we do not wait for the laboratory and deal with it.

- Lee White test: roughly indicative, at the bedside – a coagulum forms in the test tube within 1−2 minutes.
- Thrombin time: evidence of fibrinogen, it can also be done at the bedside - add 2 ml of blood to a tube with lyophilized thrombin, if there is fibrinogen in it, it will clot within 1 minute, if it is not, the blood will not clot.
- Laboratory tests: INR , aPTT, antithrombin III, fibrinogen, platelets, FDP, D-dimers.

Differential diagnosis

- Bleeding from a birth injury;
- various types of thrombocytopenia;
- von Willebrand's disease ;
- coagulopathy in HELLP syndrome.

Prevention

1. **Primary prevention :**
    - ambulatory detection of all conditions where there is a coagulation disorder - especially AT III deficiency, proteins C and S , Leiden mutation of factor V, homozygous MTHFR 677TT defect, etc., also, for example, antiphospholipid syndrome .
2. **Secondary prevention :**
    - antenatal application of LMWH in pregnant women at higher risk (mainly in abortions and surgeries);
    - before sc, LMWH is routinely given to women in the following conditions: obesity, age over 30 years, hereditary thrombophilia, venous thrombosis in the anamnesis, preeclampsia, DM, previous abdominal surgery, placenta praevia, placental abruption, also during spontaneous stillbirth, maternal fever .

Treatment

If DIC is suspected, energetic intensive treatment is appropriate, if possible in a team (hematologist, anesthesiologist, internist, ...). The principle is to remove the provoking cause, regulate thrombin activity, maintain hemostasis.

Acute DIC

- Order frozen plasma and erysma immediately ;
- we will take blood for hemocoagulation examination;
- first measure – we administer AT III – a bolus of 1,000 units IV and then a continuous infusion of another 1,000 units;
- then we give heparin ;
- circulating plasma replacement (dextrans and plasma expanders are contraindicated - they interfere with platelets);
- we administer fibrinogen if its plasma level falls below 1 g/l.

Chronic DIC

- Combination of AT III with heparin;
- activated human protein C is newly used.

Dilutional coagulopathy

Dilutional coagulopathy occurs as a result of volume replacement during heavy bleeding, it is alleviated by the administration of fresh frozen plasma in the replacement. Disorders of hemostasis cause:

- dilution below 10-20 % activity of coagulation factors;
- dilution of platelets below 10-20 thousand/mm^3;
- inhibition of procoagulant activity caused by dilution (in the physiological state there is a relative

predominance of procoagulant activity);
- hypoxic-reperfusion syndrome and isolated fibrinogen supplementation predispose to a possible transition to DIC.

Rupture of ectopic pregnancy

Complications associated with ectopic pregnancy are the most frequently occurring abdominal emergency in gynecology. Ectopic pregnancy is most often located in the fallopian tube (95-97 %). If the trophoblast erodes the entire tubal wall, the fallopian tube ruptures. It has a stormy course. There is a risk of massive bleeding into the peritoneal cavity, as the blood practically does not clot (fibrinogen precipitates upon contact with the peritoneum). Symptoms of fallopian tube rupture include sudden pain in the lower abdomen, peritoneal irritation, collapse. Hemoperitoneum, cardiopulmonary decompensation, and shock state develop rapidly. Exceptionally, the blastocyst nidus and begins to develop in the cervix uteri, this pregnancy tends to have the most severe course (repeated, difficult-to-treat bleeding occurs, life-saving hysterectomy is often required).

🔎 *For more information see Ectopic Pregnancy.*

Pathological placement of the placenta

The cause of bleeding in the third trimester and during childbirth can be a pathologically located placenta. Placenta praevia threatens the life of both the mother and the fetus (mother with bleeding, fetus with hypoxia). The main symptoms of pathological placement of the placenta are:

1. **Bleeding**:
   - the main symptom that most often accompanies bed rest;
   - usually also as the first symptom at the end of the 1st trimester.
2. **Abortion'**:
   - can arise because the placenta does not find as much room to grow in the lower segment, the production of hCG decreases and the corpus luteum may disappear, but more often the miscarriage does not occur and the pregnancy continues;
   - if the pregnancy continues, in the second trimester, the lower segment begins to grow, which leads to partial separation of the bed and bleeding again;
   - repeated blood loss leads to anemization of the mother;
   - the closer to delivery, the more contractions (dilation of the throat), the more frequent and stronger the bleeding.
3. More frequent occurrence of **pathological fetal positions'**:
   - the placenta prevents the head from entering the pelvis;
   - if we find a pathological position towards the end of the pregnancy, we should rule out the en route bed.
4. Pathological placement of the placenta can sometimes be completely ``asymptomatic.

🔎 *For more information see Pathological placental retention.*

Links

Related Articles

- Bleeding in pregnancy

External links

- Peripertal life-threatening bleeding 2018— interactive algorithm + test (https://www.akutne.cz/algorithm/cs/327--/)

References

- CZECH, Eugene. *Obstetrics.* 2. edition. Prague : Grada, 2006. ISBN 80-247-1303-9.
- Quadruplets of developed questions based on the study materials of J. Beneš, L. Mikšík, e-learning and the book Gynecology and Obstetrics (Martius 2005).