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ACQUIRED COAGULOPATHIES

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Abstract: Coagulopathy is a disruption of the blood coagulation system (hemostasis system) that occurs due to exposure to certain pathological factors. Depending on the type of disorder, the patient may experience excessive bleeding or, conversely, the formation of a large number of blood clots. In some cases, opposite processes occur in different parts of the body. Treatment of coagulopathies is complex and depends on the initial cause of the disease and the type of disorder.

Key words: Coagulopathy , bleeding, types of coagulopathies , diagnosis, treatment

The problem of bleeding is extremely relevant for all medical specialties. The greatest number of bleedings is associated with a violation of the integrity of blood vessels - mechanical damage, erosion, etc. But some of the bleeding is due to hemorrhagic syndrome associated with one or another coagulopathy . In emergency medicine, there are not very many reasons for the development of hemorrhagic syndrome. These are either congenital or acquired coagulopathies . Of greatest interest, in terms of differential diagnosis, are acquired coagulopathies , which doctors of almost all specialties often encounter.

List of purchased coagulopathies , which may result in hemorrhagic syndrome, are as follows:

1. disorders of vascular-platelet hemostasis;
2. deficiency of coagulation factors, developing for several reasons;
3. direct negative effect of blood substitutes on blood clotting (HES, dextrans, perftoran);
4. hypothermic coagulopathy ;
5. fibrinolytic coagulopathy - DIC Sh-GU;
6. hypocalcemia .

For successful treatment of hemorrhagic syndrome, the doctor must identify the cause of its development, since treatment tactics for various coagulopathies are fundamentally different. The still widely practiced universal treatment of hemorrhagic syndrome in the form of FFP transfusion does not justify itself and sometimes worsens the situation. Very often, several coagulopathies occur simultaneously, which increases blood loss and complicates treatment.

Disturbances of vascular-platelet hemostasis.

Acquired disorders of vascular platelet hemostasis are usually associated with a decrease in the number of circulating platelets, thrombocytopathy and toxic damage to the vascular endothelium. Thrombocytopenia most often develops with bleeding, “warm” shocks, and fat embolism. Thrombocytopathies may be associated with the use of drugs that inhibit their functional activity - colloids, antiplatelet agents , anticoagulants.

Diagnostics - counting platelets, determining their functional activity. Treatment is transfusion of donor platelet concentrate.

Deficiency of coagulation factors.

It develops for several reasons. Most often, against the background of blood loss and its replacement without the use of FFP - the so- called dilution coagulopathy .

Another reason is functional liver failure, sometimes isolated, but more often in combination with vitamin K deficiency. This is either liver damage (hepatitis, cirrhosis), when even in the presence of vitamin K, the synthesis of proteins of the coagulation system does not occur, or the synthesis of vitamin K is impaired due to cholestasis , intestinal dysfunction or due to vitamin K antagonists (indirect anticoagulants - warfarin , rat poisons, chemicals).

A variety of this coagulopathy can be considered DVSP - consumption coagulopathy without activation of fibrinolysis . This situation can develop with hypercoagulation and rapid consumption of factors, or with insufficient liver function, when the consumption of procoagulants does not have time to be replenished by synthesis in the liver. In itself, such coagulopathy does not lead to hemorrhages, but with any bleeding, the volume of blood loss is greater and hemorrhagic syndrome can develop faster.

Diagnosis is based on coagulogram data , taking into account the clinical situation and assessment of liver function. Treatment is transfusion of FFP under the control of a coagulogram .

Direct negative effect of blood substitutes (HES, dextrans, perftoran) on blood clotting.

Reckless and untimely use of synthetic colloids can lead to inhibition of both primary and secondary coagulation hemostasis. It has been revealed that colloids with the highest molecular weight have the greatest inhibitory effect, and this “side” effect is often used for therapeutic purposes, but with hypercoagulation and hemoconcentration . But doctors forget about this side effect when they treat blood loss with the same colloids - they replenish the blood volume with them. It is also not taken into account that for different colloids the mechanism of inhibition of all parts of hemostasis is the same and the use of small doses of dextrans and different HES gives a cumulative hemorrhagic effect. Coagulogram data, platelet count, determination of their activity, and analysis of previous infusion therapy.

Treatment is transfusion of FFP under the control of a coagulogram and platelet concentrate for thrombocytopenia or thrombocytopathy .

Hypothermic coagulopathy .

This type of coagulopathy is most often observed with acute massive blood loss and can be combined with

any other coagulopathy . It must be remembered that the hemostasis system significantly reduces activity at body temperatures below 35 ° C and practically does not work at body temperatures below 34 ° C. The introduction of FFP into a cold body is also ineffective - hemorrhagic syndrome will continue! In this case, the coagulogram indicators will be normal!

Diagnostics - measuring body temperature.

Treatment is only warming the body and injected solutions.

Fibrinolytic coagulopathy - DIC III - IV

Most often observed in obstetrics, because the uterus, especially a pregnant one, is “stuffed” with physiological and pathological fibrinolytics . The state of hemostasis becomes critical for several reasons and according to the following scheme - first there is always “mechanical” blood loss - placental abruption, blood loss during childbirth or caesarean section. Further, against the background of hyperactivation of the coagulation system, powerful fibrinolysis begins , the reason for which is the release into the bloodstream of tissue plasmin or substances with a similar effect from the tissues of the uterus, which is filled with them, especially during its pathological changes. The release of pathological fibrinolytics can also occur in other situations - during severe destructive processes, bacteremia.

The dynamics of the situation are very fast - the uterus was “bruised” during a caesarean section or during childbirth or already during amputation, toxins were released due to bacteremia,

etc. - and a huge amount of fibrinolytics or substances that provoke their own fibrinolysis entered the bloodstream .

Diagnosis is based on assessment of plasma fibrinolytic activity.

Treatment - initially it is necessary to “extinguish” fibrinolysis , since the introduction of FFP only intensifies the hemorrhagic syndrome - physiological anticoagulants and fibrinolytics are also administered along with plasma . Inhibition of fibrinolytic activity is achieved by introducing inhibitors of animal origin - contrical , trasylol , gordox . Fibrinolysis inhibitors are used strictly under the control of a coagulogram . Recently, the use of tranexamic acid drugs has been considered safer and no less effective. In extreme cases , the use of aminocaproic acid is permissible.

Only after antifibrinolytic therapy do FFP transfusions begin. Antifibrinolytics can be continued in parallel with FFP transfusion.

Quite effective in fibrinolytic coagulopathy the use of recombinant drugs - N ovoseven , Feiba , etc. Unlike FFP , they do not contain fibrinolytics and can be used in parallel with antifibrinolytics .

Hypocalcemia .

Hypocalcemia as a factor determining hypocoagulation with hemorrhagic syndrome is a rather rare situation that arises under a combination of circumstances. This situation occurs during acute massive blood loss, when the level of plasma calcium decreases during replacement and dilution. And this small amount of calcium is bound by a preservative that enters the bloodstream along with blood components - each hemocone contains a residue of active citrate. As a result, all plasma calcium necessary for hemostasis is bound, which determines or maintains hemorrhagic syndrome.

Treatment - monitoring and correction of calcium levels in the treatment of massive blood loss or hemorrhagic syndrome. Or follow the old rule - for every liter of transfusion it is necessary to administer 10 ml of calcium chloride or calcium gluconate .

Conclusions . Very often, doctors are faced with a situation where hemorrhagic syndrome or coagulopathic bleeding is determined by several reasons. Begin with mechanical blood loss , this critical situation is quickly layered with hemodilution and dilution of coagulation factors and platelets, the anticoagulant effect of colloids, which are usually thoughtlessly used to compensate for blood loss, as well as hypothermic cogagulopathy associated with blood loss, intraoperative cooling of the body and the introduction of cold (room temperature) solutions. All this is aggravated by metabolic disorders and hypocalcemia . Treatment usually boils down to FFP transfusion, the introduction of recombinant factors and the use of hemostatic agents . No one bothers to analyze the situation and make differential diagnoses coagulopathy . Accordingly, positive results are not always obtained, either at the cost of great costs or irreversible losses, because in a hurry, time is lost for rash actions.

If all of the listed causes of hemorrhagic syndrome are excluded or after they are eliminated and tests are normalized, if bleeding continues, then it should be classified as associated with a violation of the integrity of the vessels and treated surgically.

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