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REVIEW/RESEARCH ARTICLE/SHORT COMMUNICATION

THERAPEUTIC AND DIAGNOSTIC TACTICS FOR THROMBOSIS OF THE LOWER LIMBS VINES IN PREGNANT WOMEN AND PUERPERAS

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Abstract

This article is devoted to the venous thromboembolic complications (VTEC) prevention during pregnancy, childbirth and the postpartum period, risk factors and pathogenesis, venous thrombosis management tactics of the lower extremities in pregnant women and puerperal period.

Keywords: varicose veins, venous thrombosis, post-thrombotic complications, thromboembolism, pregnancy, postpartum period.

Introduction

VTEC- venous thromboembolic complications;(VV) - varicose veins; (DVT)- deep vein thrombosis; (PE) - pulmonary embolism; LMWH – low molecular weight heparin; UFH – unfractionated (standard) heparin; (INA) - international normalized attitude; (VTE) - venous thromboembolism; (MHT) - menopausal hormone therapy; (APS) - antiphospholipid syndrome; (APA) - antiphospholipid antibodies.

The thrombosis prevalence and venous genesis thromboembolism, according to various estimates, is within 13-17% among the adult population. In 1-2% of cases, they end in death, in 20% cases, the post-thrombotic syndrome development. It has been found that about 70% all thrombotic complications thrombosis is hereditary thrombosis.

Intensive study of thrombotic complications during pregnancy revealed that their development is inextricably linked with certain hereditary anomalies. In pregnant women with clinical symptoms of thrombosis, the gene mutations frequency F-V-L, F-II-20210, MTHFR and other hereditary anomalies is much higher than in healthy pregnant women. However, these mutations carriers have not sufficiently studied important clinical and laboratory aspects of post-thrombotic complications of pregnancy.

Venous thromboembolic complications (VTEC) include varicose veins (VV) and thrombophlebitis, deep vein thrombosis (DVT), and pulmonary embolism (PE).

Thromboembolic complications in obstetrics - occurs in about 1 in 1000 pregnant women. It can develop at any stage of pregnancy, but the highest risk is noted during the first 6 weeks after birth, when it increases 20 times. Risk factors for VTEC include a history of VTEC, thrombophilia, obesity, elderly maternal age, inactivity, long distance travel, hospitalization during pregnancy, cesserean section, and other comorbidities, including bowel disease and preeclampsia.

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The deep vein thrombosis (DVT) risk and PE associated with pregnancy is about 5-10 times higher than outside pregnancy, but this risk is greatest in the postpartum period, when thromboembolism occurs 4-5 times more often. So, if the prevalence of DVT during pregnancy is 3: 1000, then after childbirth - 12-15: 1000.

PE is the most severe complication of VV and DVT, the pulmonary artery blockage or its branches with blood clots, which form more often in large veins of the lower extremities or pelvis (embolism). The blood clots source in PE is more often the veins of the lower extremities (thrombosis of the veins of the lower extremities), much less often - the veins of the upper extremities and the right heart.

Clinical variants of thrombophlebitis.

Type I. Thrombosis of the distal saphenous or small saphenous veins or their inflows. The thrombotic process is localized distal to the knee joint in the great saphenous vein lesion and below the saphenopliteal anastomosis in the lesser saphenous vein lesion. There is no pulmonary embolism threat. Subsequently, thrombophlebitis either subsides or goes into one of the following types.

Type II. The thrombosis spreads to the pre-sapheno-femoral or sapheno-popliteal anastomosis, without passing to the femoral/popliteal vein. There is no immediate threat of pulmonary embolism yet, but it becomes real in the near future with proximal spread of thrombosis.

Type III. Thrombosis, through the saphenous vein mouth, passes to the deep venous system. The tip of such a thrombus, as a rule, is non-occlusive and floats (floats) in the bloodstream of the femoral or popliteal vein. The thrombus is fixed to the venous wall only in the proximal saphenous vein. The threat of pulmonary embolism, including fatal, is very high. Subsequently, such a floating thrombus either turns into an embolus, or becomes a parietal iliocclusive thrombus of the main deep vein. Further spread of thrombosis along the deep venous system in the proximal and distal directions will lead to the development of an extended occlusion of the femoral-iliac segment.

Type IV. The thrombosis does not spread to the incompetent perforating veins of the lower leg or thigh and passes to the deep venous system. The presence or absence of a pulmonary embolism threat in these group patients depends primarily on the nature of the thrombus (floating, parietal, or occlusive) in the deep venous line.

Type V. Any of the above options with isolated simultaneous deep venous thrombosis of both the affected and the contralateral limb.

With DVT, occlusive and non-occlusive (floating, parietal) thrombosis is distinguished. A floating thrombus is considered an embolism hazard, since it has a single fixation point in the distal region, and its proximal part is located in the free flow of blood, it can turn into an embolus and cause PE.

Clinically, PE is classified into the following types:

• massive - more than 50% of the volume of the vascular bed of the lungs is affected (pulmonary embolism is manifested by shock and/or systemic hypotension;

• submassive - 30-50% of vascular bed volume of the lungs is affected (embolism of several lobar or many segmental pulmonary arteries) and is manifested by right ventricular failure symptoms;

• non-massive - less than 30% of the volume of the vascular bed of the lungs is affected (embolism of small distal pulmonary arteries), manifestations are absent or minimal (pulmonary infarction).

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By etiology, congenital pathology (EC), primary (idiopathic) disease with an unknown cause (Ep) and a secondary one with a known cause (Es) are distinguished.

Venous pathology of the lower extremities can affect superficial (As), deep (Ad) and perforating (Ap) veins.

Pathophysiological disease can occur with reflux (Pr) and obstruction (Po).

The list of basic and additional diagnostic measures:

1. Diagnostic measures carried out at the stage of an ambulance emergency:

- ECG

- pulse oximetry.

2. Basic (mandatory) diagnostic examinations carried out at the inpatient level during emergency hospitalization:

- CBC, BCT;

- GUA;

- D-dimer blood test;

- coagulogram;

- Duplex veins of the lower extremities;

- ECG;

- Ultrasound;

3. Additional diagnostic examinations carried out at the inpatient level during emergency hospitalization:

- BE;

- blood troponins T and I at PE;

- blood gases - PaO2, PaCO2;

- blood atrial sodium uretic peptide (BNP) in PE;

- echocardiography to assess right ventricular function in PE;

- pulmonary angiography with PE;

- ventilation perfusion scanning for PE;

- computed tomography for PE;

- chest x-ray for differential diagnosis.

General tactics for the prevention and treatment of VTEC in obstetrics:

• low molecular weight heparin is recommended for pregnant women to prevent VTEC instead of unfractionated heparin;

•Women receiving anticoagulation for VTEC who are pregnant are advised to take LMWH instead of vitamin "K" antagonists during the first trimester, during the second and third trimesters, and in late pregnancy just before childbirth;

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• it is recommended to limit the use of fondaparinux and a parenteral direct thrombin inhibitor for those pregnant women who have severe allergic reactions to heparin (eg, heparin-induced thrombocytopenia) and who cannot receive danaparoid;

• pregnant women are advised to avoid the use of oral direct thrombin (eg, dabigatran) and ingibotorvanti-Xa (eg, rivaroxaban, apixaban);

• For lactating women who are using warfarin, acenocoumarol, or UFH and wish to breastfeed, it is recommended to continue taking warfarin, acenocoumarol, or UFH;

• lactating women who are taking LMWH, danaparoid or p-hirudin and wish to breastfeed are advised to continue taking LMWH, danaparoid, or p-hirudin;

• lactating women are offered alternative anticoagulants instead of the Parinux sodium foundation;

• breastfeeding women are advised of alternative anticoagulants, and non-oral direct thrombin (eg, dabigatran) and factor Kha inhibitors (eg, rivaroxaban, ariksaban);

• during lactation, women who are taking low doses of aspirin for vascular indications who wish to breastfeed are encouraged to continue taking aspirin;

• for women at increased risk of VTEC after cesarean section due to one or at least two minor risk factors, pharmacological thromboprophylaxis (prophylactic LMWH) is recommended;

• for women who have contraindications to anticoagulants, mechanical prevention is recommended during labor (elastic stockings or intermittent pneumatic compression);

•For women undergoing caesarean section who are at very high risk of VTEC / have several additional factors Risk of atromboembolism that persists in the postpartum period, a combination of prophylactic LMWH with elastic stockings and / or intermittent pneumatic compression is recommended.;

• Pregnant women with acute VTE are recommended therapy with a matched dose of LMWH given subcutaneously instead of a matched dose of UFH.;

• pregnant women of antenatal treatment with vitamin K antagonists;

• Pregnant women with acute venous thromboembolism are advised to continue taking anticoagulants for at least 6 weeks postpartum (for a total duration of therapy of at least 3 months) compared to shorter treatment periods.;

•Pregnant women receiving LMWH adjusted dose therapy and scheduled for labor are advised to suspend LMWH at least 24 hours prior to induction of labor / cesarean neurological anesthesia) instead of continuing the LMWH until labor itself.;

• all pregnant women with primary venous thromboembolism are advised to undergo postpartum prophylaxis for 6 weeks with a prophylactic / medium dose of LMWH / vitamin K antagonists targeting an international normalized ratio of 2 to 3..

• To ensure the safe use of LMWH and fondaparinux, it is necessary to determine creatinine clearance or azotemia before their use. With a decrease and excretory, the use of direct anticoagulants is associated with an increased risk of large bleeding.

• to ensure the effectiveness of heparin miilifond parinuks, physiological anticoagulants - Antithrombin III or plasma tolerance to heparin.

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With a decrease in the content of physiological anticoagulants, the effectiveness of heparins is significantly reduced. In these cases, it is recommended to maintain a sufficient level of physiological anticoagulants by transfusion of fresh frozen plasma or the introduction of AT III.

Treatment tactics at the delivery stage:

• before delivery, the therapeutic doses of LMWH and unfractionated heparin are canceled within 24 hours, and in the case when the timing of labor is unknown, only unfractionated heparin should be used;

• The optimal method of pain relief for operative delivery in the absence of severe respiratory failure is regional anesthesia (spinal, epidural). The safety of regional anesthesia and anticoagulant administration is ensured by careful observance of time intervals;

• with severe respiratory failure - general anesthesia in conditions of artificial ventilation.

• with operative delivery, for the prevention of complications such as an epidural hematoma, it is necessary to carefully observe the time intervals between the administration of anticoagulants and the implementation of regional anesthesia and removal of the catheter from the epidural space.

Prevention in the postpartum period:

• regardless of the method of delivery, the patient should be activated as early as possible - a few hours after childbirth or surgery;

• when planning long-term (months) thromboprophylaxis with quartarfarin begins from the first day after delivery and is combined with the use of LMWH for 4-5 days to achieve an international normalized ratio of 2.0-3.0. Post-episode DVT during pregnancy anticoagulant therapy after childbirth lasts continuously for at least 3 months (mainly with a selected dose of warfarin);

• does not recommend the use of direct thrombin inhibitors (dibigatran) and factor Xa inhibitors (rivaroxaban, apixaban) for lactating women.

Treatment tactics for PE during pregnancy:

• for the correction of arterial hypotension / shock vasopressors (norepinephrine 2-30 mcg / min, epinephrine, dompin) and inotropic drugs for right ventricular dysfunction (dobutamine levosimendan);

• massive infusion therapy to correct hemodynamic disturbances in PE is contraindicated.

• with PE and severe pain syndrome - narcotic analgesics:

• Promedol 2% -1.0 i/m or i/v, Morphine 1% -1.0 i/m or i/v.

Non-drug treatment:

With DVT:

• provide an elevated position of the lower limb (at the initial stage);

• provide elastic compression (stockings) of the lower extremities.

There is no evidence that early mobilization of patients with acute DVT increases the risk of PE; moreover, it is indicated that early mobilization leads to more rapid muregression of lower limb edema and pain.

Drug therapy:

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Initial anticoagulant therapy with a high clinical likelihood of acute DVT / with its verification or with PE is represented by three options for the use of heparins:

• intravenous administration of unframed heparin - 5000 IU bolus i/v and then continuous intravenous infusion 1000-2000 IU/h. Control of the activated partial thromboplastin time is performed 6 hours after the start of therapy and its values should increase in relation to the norm by 1.5-2.5 times. Subcutaneous administration of unfractionated heparin is an adequate alternative to intravenous administration. Loading dose -5000 IU intravenously, and then 15000-20000 IU every 12 hours.

• subcutaneous injection of low molecular weight heparin. Therapeutic dosages LMWH determination of the number of platelets is necessary 5-7 days after the start of treatment.

Medical treatment provided at the stage of emergency emergency care:

• with PE and severe pain syndrome - narcotic analgesics: trimeperidine hydrochloride 2% -1.0 i/m or i/v, morphine 1% -1.0 i/m or i/v.

• if necessary - carrying out resuscitation measures.

Surgical intervention:

Inpatient surgery:

• Crossctomy

• Ligation of the mouth of the small saphenous vein

• Ligation of Superficial femoral vein

• thrombectomy

• installation of "Kava-filter".

Preventive actions:

Assessment of the risk of venous thromboembolic complications in obstetrics should be carried out at the following stages:

Before pregnancy:

• identification of constantly taking anticoagulants / antiplatelet agents (prosthetic heart valves, vascular prostheses, after suffering arterial or venous thrombosis);

• detection of thrombosis in relatives of the first and second generations - to a depth of up to 60 years.

• with previously transferred episodevenous thromboembolic complications and not associated with estrogen intake, the patient should be tested for the diagnosis of thrombophilia.

During pregnancy planning / early pregnancy, all women should be advised to:

• documented risk assessment for VTEC;

• reassessment of risk factors in case of hospitalization of a woman for any reason or development of other intercurrent diseases;

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• prophylactic use of LMWH in the antenatal period in the presence of previously transferred VTEC and / or hereditary or acquired thrombophilia high risk and / or three or more other risk factors according to the appendix.

At moderate and high risk of venous thromboembolic complications and the presence of contraindications, thromboprophylaxis methods are used.

Further management:

Long-term prophylaxis (up to 6 weeks postpartum) after hospital discharge is recommended for high-risk patients who have significant risk factors after delivery..

Indicators of the effectiveness of treatment and the safety of diagnostic and treatment methods:

• PE warning at DVT;

• relief and elimination of PE.

Conclusion: Arterial and venous thrombosis, as well as thromboembolic complications, are considered one of the most life-threatening complications of various diseases. Diagnosis of thrombosis should include not only characteristic clinical symptoms, but also the results of laboratory and instrumental studies for the appointment of pathogenetically based treatment. Thus, the detection of antiphospholipid antibodies in a patient with strombosis indicates the need for long-term anticoagulant therapy using LMWH and acetylsalicylic acid (ASA) and refusal from the use of warfarin and glucocorticoids. Hyperhomocysteinemia, both genetically determined and acquired, is effectively corrected with the help of folic acid and B vitamins, and sticky platelet syndrome - with the help of ASA.

The leading role in the occurrence of thromboembolic complications during pregnancy is played by genetic thrombophilia and antiphospholipid syndrome. In conditions of hypercoagulation, which is observed during pregnancy, decompensation of the hemostatic system occurs, and thrombophilia is manifested by the clinical development of thromboembolic and obstetric complications. The most unfavorable in relation to thromboembolic complications in pregnant women is a combination of multigenic forms of thrombophilia and antiphospholipid syndrome and the presence of homozygous forms of thrombophilia.

The detection of multiple fibrinolysis defects in the majority of patients with venous thromboembolism during pregnancy indicates their leading role in the occurrence of thromboembolic complications. The occurrence of thrombosis may be due to the decompensation of the hemostasis system under conditions of inhibition of fibrinolysis inherent in physiological pregnancy, and in conditions of fibrinolysis disorders associated with the circulation of antiphospholipid antibodies. To these factors are added defects of several factors at once, which are responsible for the functioning of the fibrinolytic system. All patients with thrombosis and obstetric complications during pregnancy or with a history (both personal and family) should be screened for hereditary forms of thrombophilia. This allows us to determine the further tactics of the patient's treatment, its duration, the choice of the drug, it allows to prevent both thromboembolic and obstetric complications when planning a subsequent pregnancy, to give the patient recommendations on anticoagulant therapy in the event of external risk factors for thrombosis (trauma, surgery), to carry out if necessary a study on thrombophilia in the patient's relatives. In addition, oral contraception and menopausal hormone therapy are absolutely contraindicated in patients with thrombophilia.

The discovery of the genetic forms of thrombophilia and antiphospholipid syndrome, the study of their role in the pathogenesis of thrombosis, the development of effective therapy and prevention of venous

thromboembolism allows us to say that thromboembolic complications are preventable causes of maternal mortality.

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