

Some aspects of diagnosis and therapy of genetically determined thrombophilia in obstetric-gynecological and general therapeutic practice

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Over the years, it does not decrease, but on the contrary, the relevance of diagnostics and therapy of thrombosis and predisposition to them increases. No less relevant is the issue of preventing such conditions.

to note that a lot has been done on this path, both by representatives of traditional and classical medicine and biology.

And representatives of such a direction as energy-informational medicine are simply obliged to use the entire arsenal of accumulated knowledge.

Since the authors work with obstetric and gynecological pathology, in particular, with the pathology of pregnancy, the material was prepared on the basis of and in relation to these aspects. However, the authors believe that in a similar way, the issues raised in the article can be considered in relation to other areas, such as cardiology, angiology, as well as surgical and general therapeutic practice.

As already mentioned, in recent years, medical science has accumulated a large amount of extremely relevant knowledge that can be successfully used in such areas as: homeopathy, bio- and multiresonance therapy, as well as ART and EPD according to R. Voll.

In particular, this concerns the issues of genetic predisposition to blood hypercoagulation, since a number of mechanisms of regulation of the blood coagulation system have been identified, and their genetic determination by certain genes of different chromosomes [8, 11]. In addition, it is extremely important to identify the variability of the genome and the expression of many genes, in particular, those regulating vascular processes and responsible for their pathological abnormalities {1}. In other words, it has been proven that in the course of human life, a number of genes can change the number of loci and, thereby, determine the state, in particular, of one or another link of homeostasis, for example, blood pressure and the state of blood vessels. On the other hand, there are a number of studies proving the possibility of a modifying effect of superweak stimuli on the synthesis of many biologically active substances and, consequently,

Adequate treatment implies adequate diagnosis. Targeted diagnostics are carried out using the ART method, which will be described in detail below. But the EPD according to R. Voll also does not lose its relevance, which allows quickly enough, using the analysis of a small amount of BAP, to assess the state of the vascular system and the type of lesion. And also to carry out control over the therapy process. For this purpose, according to the authors, use a number of BAPs: on the vascular meridian - KTI, the point of lymphatic microcirculation, which makes it possible to assess condition arterial bed. In addition, when detecting abnormalities of the CTI on the meridian of Allergy and inflammation, we can talk about the immune-allergic nature of the pathology. Identification of deviations at the points of allergy and autonomic regulation requires the search for specific allergens (infectious, toxic, etc.), so

how can it indicate the toxic-allergic nature of hemostasis disorders, for example, DIC in infectious lesions such as endometritis, septicemia, etc. More specifically, consideration of this issue is not included in the plans of this article. The identification of abnormalities at the point representing autoimmune processes allows us to speak of a violation of immune regulation with the development of autoimmune processes, in particular, the blood coagulation system, such as antiphospholipid syndrome, which can occur with decompensation of adrenogenital syndrome (AGS) (point of the gonads - adrenal glands on the Endocrine meridian), etc. The point of vascular sclerosis, based on the experience of the authors, can be considered as representing, in particular, the state of endothelial dysfunction. Since the process of endothelial dysfunction plays one of the leading roles in the development of vascular pathology, including atherosclerosis. Indirect data on the state of hemostasis can be obtained from the analysis of points of the aorta, veins and coronary vessels on the blood circulation meridian.

Diagnostics by the ART method makes it possible to assess the specific mechanism of hemostasis impairment. Since many genetic mechanisms of its regulation are already known.

So, in 1 chromosome there is a gene encoding antithrombin III and its deficiency blocks prothrombinase. In patients with thromboembolic complications, the incidence of AT III deficiency is 3–8%. Of the clinical manifestations, it is worth noting venous thrombosis, habitual loss pregnancy, stillbirth, thrombophilic complications after taking oral contraceptives (OC).

also in 1 chromosome is the gene responsible for the mutation V factor Leiden as a result of which resistance to protein C, which will be discussed below.

Gene protein C located in 2 chromosome. Protein C - synthesized in the liver, vitamin K dependent anticoagulant. Its deficiency is inherited autosomal dominantly. Clinical manifestations:

- habitual loss of pregnancy, stillbirth, fetal loss (up to 27.9%);
- venous thrombosis and thromboembolism at a young age (up to 10%);
- necrosis of the skin and subcutaneous tissue (especially during therapy with indirect anticoagulants);
- venous thrombosis while taking OK.

Same 2 chromosome the cytokine gene is encoded IL1, an inflammatory mediator. Which plays an important role in the regulation of blood coagulation, destabilizing antioxidant system, and is an mediator endothelial dysfunction.

On chromosome 3 there is a gene protein S, which is a cofactor protein C. Also synthesized in the liver. Inheritance is also autosomal dominant. Clinical manifestations are similar to protein C deficiency: venous thrombosis and thromboembolism at a young age, often provoked (pregnancy, OC, infections, etc.). The pregnancy loss rate is 16.5%, more often stillbirths. It is worth considering separately 6 chromosome.

Firstly, because it is in it, in the immediate vicinity of the genes encoding HLA-ABC, HLA-DR

there is a gene encoding 21-hydroxylase, the defect of which forms AGS. It determines the state of the glucocorticoid system and, accordingly, the control of allergic and autoimmune conditions.

Secondly, communication data available HLA-DR and HLA-DQ with antiphospholipid syndrome (APS), which is the most common cause of thrombophilic complications during pregnancy. In this syndrome, the presence of antibodies to many phospholipids, in particular, cardiolipin, as well as some proteins, in particular prothrombin, is noted.

In addition to thrombosis, APS is associated with recurrent miscarriage and spontaneous miscarriage for up to 10 weeks, intrauterine fetal death over 10 weeks, placental insufficiency, placental abruption, as well as severe eclampsia, HELLP syndrome.

Third, 6 chromosome activity is encoded TNF- α (tumor necrosis factor), one of the most aggressive pro-inflammatory cytokines, which is involved in damage to a wide variety of cells and tissues. In particular, it causes hypercoagulation by damaging vascular endothelial cells, causing endothelial dysfunction. TNF- α promotes the activation of free radical processes.

The gene located in 7 chromosome, determines activity PAI-1, an inhibitor of plasminogen activator, with a dysfunction of which is associated, respectively, a violation of fibrinolysis, an increased risk of thrombosis and, as a result, cardiovascular pathology, as well as obstetric complications (preeclampsia, placental abruption, stillbirth).

also in Chromosome 7 contains the eNOS gene, whose polymorphism determines the synthesis of nitric oxide (NO), a vascular relaxation factor.

On chromosome 8 there is a gene PLAT tissue plasminogen activator, the deficiency of which, accordingly, causes hypercoagulability, as well as endothelial dysfunction.

Gene polymorphism GSP1, located in 11 chromosome, determines the activity of glutathione, which plays a key role in the resistance of cells to oxidation products, in particular, peroxide, as well as to free radicals, in preventing DNA breakdown.

On chromosome 17 there is a gene ACE, determining the activity of angiotensin-converting enzyme. except the ACE inactivating bradykinin, which is a stimulant of NO synthesis by endothelium, that is, it plays role in the progression of endothelial dysfunction.

On chromosome 21 there is a gene CBS, polymorphism which defines the presence of hyperhomocysteinemia. Well known developmental defects central nervous system with hyperhomocysteinemia, as well as the usual early pregnancy loss, early onset of gestosis, placental abruption, intrauterine growth retardation. These disorders are associated with the early development of thrombophilia, the mechanism of which involves violations of redox processes, an increase in the level of free radicals, a decrease in the level of nitric oxide, which leads to dysfunction of the vascular endothelium, as well as due to the effect on the activation of coagulation factors and their inhibitors.

In addition, it is necessary to take into account the activity of such factors as: PAF - platelet activating factor (7 chromosome) and PDGF BB - growth factor

megakaryocytes and platelets (5 chromosome). Both of these factors can play a significant role in the process of thrombus formation.

Diagnostics of the violation of the indicated links of hemostasis by the ART method does not present great difficulties. Testing is best done using the blood nosode information preparation available in the selector and, in particular, in VRT (additional pointers) as a test-indicator. You can also use blood autosode recording. Further, we can try to test specific chromosomes involved in disorders of the hemostasis process, the information copies of which are also at our disposal. With respect to the tested chromosomes, a number of the above biologically active substances are further tested, the electronic copies of which are available in the selector. This applies to potentiated drugs from OTI: IL1, anti HLA-ABC, HLA-DR, HLA-DQ, TNF- α , PAF, PDGF II [5], as well as,

potentiated antibodies to NO. You can also immediately test these drugs against a blood product of the immediately indicated drugs, but their set is not complete at the moment.

Also, according to the authors, it is necessary to carry out diagnostic testing on this problem not only in the case of already developed hypercoagulation, but also to try to predict a possible situation by preliminary testing against the background of the model of pregnancy presented to the patient's body [2].

If a certain type of pathology is identified with the participation of polymorphism of certain chromosomes and, accordingly, certain mediators, adequate therapy is required. For what can be used, according to the test results, electronic copies of the previously described drugs, that is, drugs of the company "OTI", in various potencies, chromosomes, as well as potentiated DNA. These drugs can be used both in the form of tested potencies independently, and by selection according to the KMH marker, as well as by adaptation along chiroglyphic lines [5]. In any case, it must be remembered that we are dealing with nosodes, moreover, directly carrying program information. Accordingly, the effect of a conflict of programs may arise, which is discussed in an article by one of the authors (Bobrov I.A.), published in this collection. This can be expressed in the development of symptoms against Hering's law (from top to bottom, from the inside out, from late symptoms to early ones), as well as the appearance of new symptoms, including mental ones [6]. Evaluation of such symptoms characterizes the peculiarities of the patient's constitution, including the one that prevents the normalization of hemostasis, which requires the selection of a constitutionally similar drug.

Along with the constitutional drug, in the treatment of the pathology under consideration, such homeopathic remedies as snake venoms, Ac. aminocapronicum [4], etc.

Also, in the case of identifying factors that can disrupt the process of free radical oxidation and peroxidation (chromosome 2 - IL1, chromosome 6 - TNF- α , chromosome 7 - NO, chromosome 11 - GSP1 gene, 17 chromosome - ACE, chromosome 21 - CBS gene, hyperhomocysteinemia), makes sense recommend antioxidants, including homeopathic ones, such as potentiated hydrogen peroxide or Ozone [3]. And in case of detection of abnormalities on chromosome 21 - the CBS gene, hyperhomocysteinemia, prescribe

folic acid.

This type of blood autonosode, as an inverted recording of the process of thrombus formation in the patient's blood, is very effective both in complex and in monotherapy.

The experience of treating fifty pregnant patients with thrombophilia has shown that complex therapy using the described method is able to optimize the therapy process. It became possible to either abandon allopathic therapy altogether, or to reduce it to a minimum.

Bibliography

1. Bebikhov D.V., Nikonenko T.A., Postnov A, Yu., Postnov Yu.V. Non-Mendelian inheritance of arterial hypertension: repetitive DNA sequences as candidates for the role of genomic determinants // *Cardiology*. - Volume 41 6. - 2001. - Ed. "Mediasphere".
2. Bobrov I.A., Pechnikova E.Yu. Functional diagnostics and therapy infertility and pathology of pregnancy using the model of pregnancy in EPD, ART and BRT // Abstracts and reports of the XIII International conference "Theoretical and clinical aspects of bioresonance and multiresonance therapy". Part II. M.: IMEDIS, 2007. - P. 103-110.
3. Bobrov I.A. Possibilities of minimizing diagnostic and therapeutic exposure through the use of nonspecific nosodes of metabolism // Abstracts and reports of the XII International conference "Theoretical and clinical aspects of bioresonance and multiresonance therapy". Part I. M.: IMEDIS, 2006. - pp. 225-229.
4. Bobrov I.A. Potentiated Aminocaproic Acid - New homeopathic anticoagulant and fibrinolytic for the treatment of disseminated intravascular coagulation and thrombophilia // Abstracts and reports of the XIII International conference "Theoretical and clinical aspects of bioresonance and multiresonance therapy". Part II. M.: IMEDIS, 2007. - P. 110-113.
5. Gotovsky Yu.V., Mkhitaryan K.N. Lectures on chronosemantics.- M.: IMEDIS, 2004.
6. Köller G. Basic principles of homeopathy. Direction of cure (Hering's law) // *Homeopathy*. - M.: Medicine, 2000. -- S. 139.
7. Samokhin A.V., Tomkevich M.S., Gotovsky Yu.V., Misiano F.G. Immunology, apoptosis and homeopathy. - M.: IMEDIS, 1998.
8. Sidelnikova V.M., Kiryushenkov P.A. Hemostasis and pregnancy.- M., 2004.
9. Epshtein O.I., Shtark M.B., Dygai A.M., Sergeeva S.A., Golberg E.D., Petrov V.I., Voronina T.A., Starostina M.V. Pharmacology of ultra-low doses of antibodies to endogenous regulators of functions. - M.: Ed. RAMS, 2005.
10. Epshtein OI Neurophysiological mechanisms of pharmacological effects of potentiated ("homeopathized") antibodies to brain-specific protein S-100. Abstract of thesis. diss. Cand. honey. sciences.- Tomsk, 1999. - 24s.
11. Endothelial dysfunction at preeclampsia. Pathogenesis, predisposition, diagnosis, and genetic prevention. Methodical recommendations of the Research Institute of Obstetrics and Gynecology named after V.I. BEFORE. Ott RAMS Saint Petersburg State Medical University named after I.I. Academician I.P.

Pavlova. - SPb: "Publishing house. N-L ", 2003.

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