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Research Article

PECULIARITIES OF VARICOSE VEIN DISEASE IN PREGNANCY

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ABSTRACT

The article under discussion depicts peculiarities of varicose vein disease in pregnancy. Venous disease in women often complicates pregnancy, childbirth and the postpartum period. Venous insufficiency complicates pregnancy, delivery and postpartum period and leads to increased maternal morbidity and mortality. The author of the article considers that correction of placental dysfunction, especially in the early stages, can significantly improve perinatal outcomes.

KEYWORDS

varicose vein disease, vascular system, pregnancy, anatomic, premature, amniotic fluid, extremities.

INTRODUCTION

Varicose vein disease (VD) is a disease of the vascular system of the body. In pregnant women the occurrence of varicose veins disease is associated with a slowing down of the rate of blood flow during pregnancy, as well as with the anatomic features of the venous system of the lower extremities.

DISCUSSION

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Increased venous pressure, decreased rate of blood flow leads to local metabolic disorders with a shift of pH to the acidic side and activation of the blood coagulation system. Venous disease in women often complicates pregnancy, childbirth and the postpartum period. The incidence of early and late gestosis (10%), chronic fetal hypoxia (10%), umbilical cord pathology (24-26%), untimely outflow of amniotic fluid (22-24%) is rather high due to varicose veins disease of the lower extremities, weak obstetrical activity (15%), premature detachment of the normally located placenta (2%), postpartum and early postpartum hemorrhages (18%), postpartum endometritis (7%) [1].

In physiological pregnancy there is stimulation and activation of the fetoplacental complex, which leads to an increase in the coagulant potential, almost doubling the content of all clotting factors against a decrease in fibrinolytic and anticoagulant activity. Beginning in the second trimester of pregnancy, endothelial production and secretion of Willebrand factor, thromboxane, endothelin-1, thrombomodulin, and fibronectin into the blood increased, suggesting the formation of an endothelial dysfunction syndrome in the feto-placental complex even during physiological pregnancy [8].

Hemostasis in pregnant women with varicose veins is a serious problem, and the effect of drugs improving microcirculation and regulating angioprotective effects on its parameters has been little studied. It is of great scientific and practical interest to study the effect of drugs affecting endothelial dysfunction being introduced into clinical practice. Pregnant women with varicose veins have a generalized impairment of all endothelial functions accompanied by the impaired regulation of vascular tone and permeability and increased procoagulant, proaggregant, antifibrinolytic and anti-inflammatory activity of the endothelial layer. Chronic venous insufficiency and VF are the most common group of extragenital cardiovascular pathology in pregnant and postpartum women, according to different authors - in 30-50% of women. Varicose veins first appear in 50-96% of women during pregnancy. Venous insufficiency complicates pregnancy, delivery and postpartum period and leads to increased maternal morbidity and mortality [9].

Pregnancy is often the triggering factor that manifests or causes symptoms of venous insufficiency and IBD. This association (venous insufficiency and pregnancy) is characterized by a rapid onset of symptoms and their partial regression after delivery. Treatment during pregnancy is aimed at elimination of symptoms and prevention of complications. The development of chronic venous insufficiency and VD during pregnancy is usually explained by the pressure of the pregnant uterus on the inferior vena cava and iliac vein. Persistent venous dysfunction in the weeks after delivery suggests that changes in venous function in pregnancy are influenced not only by venous compression by the pregnant uterus, but also by other factors. Some authors have studied the effects of sex hormones on the venous wall, but did not find a relationship between the development of chronic venous insufficiency and the plasma concentrations of estradiol, estriol or progesterone. However, 17% of women with normal pregnancy developed pathological venous insufficiency and VF. At the same time, outside of pregnancy we found almost no or very insignificant concentrations of estrogen receptors and significant concentrations of progesterone receptors in the great saphenous vein wall taken after venectomy for varicose veins. Consequently, sex hormones can directly affect the vein wall through the classical receptor pathway [7].

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Endothelial dysfunction and changes in hemostasis, in particular, hypercoagulation, play a significant role in the development of chronic venous insufficiency. Endothelial dysfunction leads to changes in vascular reactivity, activation of intravascular coagulation cascade and disruption of vascular integrity. The main markers of endothelial dysfunction are decreased production of prostacyclin and No, a relative increase in thromboxane, and a significant increase in endothelin-1. Nitric oxide is the "face" of the endothelium, as it provides in the maintenance of vascular homeostasis: regulation of vascular tone; inhibition of adhesion, platelet aggregation and thrombosis; regulation of proliferation and apoptosis; regulation of oxidative processes; inhibition of leukocyte adhesion. Moreover, it regulates all these processes with "+" sign, i.e., it is one of the most demanded substances in pathological conditions. Under the influence of various damaging factors, the ability of endothelial cells to synthesize No decreases, while the formation of vasoconstrictors is preserved or increases, i.e., a condition defined as endothelial dysfunction (ED) is formed [2].

The above changes create a "favorable" environment for the formation of predominantly chronic placental dysfunction (PD). In 16-25% of patients with PD there are manifestations of CHD, chronic venous insufficiency and pelvic varicose veins. The treatment of this category of patients presents significant difficulties, as it can lead to the most severe complications in obstetrics, thrombosis and thromboembolism of the pulmonary artery [4].

Placental dysfunction is the most important problem of modern perinatology. The frequency of its detection varies from 3-4 to 45%, the perinatal morbidity reaches 70%. Despite the intensive use of the latest methods of diagnosis and treatment, insufficient placental function remains the leading cause of high morbidity and mortality of children not only in the perinatal period, but also at the stages of subsequent development. At the present stage PD is considered as a clinical syndrome caused by morphofunctional changes in the placenta and disorders of compensatory-adaptive mechanisms that ensure the functional fullness of the organ. It is the result of a complex response of the fetus and placenta to various pathological conditions of the maternal body and is manifested in a complex of disorders of transport, trophic, endocrine and metabolic functions of the placenta, underlying the pathology of the fetus and the newborn [5].

Primary and secondary PD are distinguished. Primary PD develops during the formation of the placenta (up to 16 weeks of gestation) and is most common in women suffering from habitual miscarriage, as well as in pregnant women with a history of infertility. Secondary PD usually occurs after the completion of placental formation processes and is caused by exogenous influences, primarily diseases suffered during pregnancy.

Chronic placental dysfunction (CPD) is a more frequent pathology, observed in about one in three pregnant women of the high risk perinatal pathology group. Perinatal mortality in this group reaches 60%. Chronic PD is a long-term placental dysfunction, often with compensatory increase in its weight, pathological immaturity of the villi, focal or diffuse sclerosis of their stroma, hemorrhages and extensive infarcts. Depending on the extent of the lesion, intrauterine hypotrophy develops or fetal death occurs. Changes occurring in the placenta in CPD, primarily due to thrombosis of the vessels of the villi and chorionic villi with subsequent ischemia, fibrin deposition and the development of infarction, which

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leads to a violation of placental perfusion and interaction of the trophoblast with the uterine vessels of the placental site, making them insensitive to most medications.

Preclinical diagnosis of fetal abnormalities is an important task. This is important because early detection of fetoplacental insufficiency in a number of cases allows timely corrective therapy. The development of methods for studying the feto-placental complex in dynamics allows timely diagnosis of the main clinical forms of fetal distress - delayed intrauterine development of the fetus and/or its chronic hypoxia. Prenatal diagnosis of these conditions traditionally includes ultrasound (ultrasound), ultrasound Dopplerometry, also conducted cardiotocography (CTG), the study of placental proteins and hormones. Less frequently, a cervical cytological examination with determination of the type of smear is performed. The study of the hemostasis system and markers of endothelial dysfunction is also relevant in PD.

The action of any pathological factors on the fetus is mediated through structural and functional disorders of the placenta, so of great interest is the study of placental and endometrial proteins, the so-called "pregnancy zone proteins" that act as hormones, enzymes, enzymes, receptors, growth factors and immunoregulatory agents. The study of placental proteins synthesized by different parts of the placenta (maternal and fetal) provides a new clinical perspective on their function and role in the development of pregnancy. Of the proteins produced by the placenta, of particular importance are placental hormones, in particular: progesterone (PG) and its analogue 17-hydroxyprogesterone (17-OP), chorionic gonadotropin (CG), placental lactogen (PL), estriol (E3), cortisol (C), (a)-fetoprotein (AFP).

The definition of the "biophysical profile" of the fetus, which includes a comprehensive assessment at ultrasound of 5 parameters: fetal respiratory movements, motor activity, fetal muscle tone, the amount of amniotic fluid, non-stress test at cardiotocography (CTG), has become widespread. Of great importance is the identification and interpretation of indirect criteria for placental insufficiency, as well as the search for prognostic markers of gestational homeostasis disorders, on the basis of which we could allocate risk groups for the development of PD and conduct preventive courses of therapy in these groups. Among them, there are markers of primary placental insufficiency - placenta previa and low location of the placenta, placenta surrounded by a ridge, double lobe or extra placenta, marginal or sheath attachment of the umbilical cord. Markers of secondary PD include a placental or membranous placenta, placental thickening (more than 5 cm) or thinning (less than 2 cm), dilation of the intervillous space, placental infarction, abundant or small fluid flow.

Another method of diagnosing PD, although largely historical, but no less informative, is the study of cervical smear types - cytology. Several pathological types of smears are distinguished. Cornificatory - hormonal disorders in pregnancy - threat of termination of pregnancy - mixed vaginal flora, epithelium of different layers, eosinophilic and karyopycnotic index (CI) > 50%. Estrogenic - PD, gestosis, rhesus conflict, threat of termination of pregnancy significant reduction in cellular elements, shift towards superficial cells, their isolated location, CI up to 30-40%, palmar cells reduced or absent. Type with predominance of deep intermediate cells with large nuclei - severe gestosis, AP in threatening, perinatitis - these cells are arranged in groups, the palatial cells are few or absent. Regressive - PD,

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perenaturation - isolated cells of all epithelial layers, the presence of basal and parabasal cells [10].

In recent years, various methods of treating placental dysfunction have been developed and continue to improve, but the problem is far from being resolved. Correction of placental dysfunction, especially in the early stages, can significantly improve perinatal outcomes. PD therapy gives a sufficiently positive effect only at the compensated and subcompensated stages of the process. PD treatment in the third trimester has no significant effect on fetal development, but can improve fetal condition, increase resistance to hypoxia and prepare the fetus for delivery. With signs of decompensated fetal PD, treatment is not considered and early delivery is considered. One of the indicators of critical fetal status is zero or diastolic blood flow recorded in the umbilical artery or fetal aorta by Doppler. The detection of critical blood flow, as well as changes in the cardiotocogram ("mute" curve type, areactive nonstress test) indicate the need for urgent delivery. Therapy should begin with the treatment of the underlying disease and the elimination of the influence of adverse factors. If placental insufficiency is confirmed and any infectious foci are identified, specific treatment according to generally accepted schemes to prevent intrauterine infection against a broken placental barrier is mandatory [11-15]. Therapy of the placental barrier must be comprehensive, i.e., it must include a set of treatment methods. These are physical methods of influence (uterine electrorelaxation, thermal procedures on the peri-renal area - diathermy, inductothermy), reflexively relaxing myometrium and expanding vessels; abdominal decompression, which improves uteroplacental blood flow; hyperbaric oxygenation, which provides preservation of respiratory enzyme activity; medicinal agents. The general directions of pharmacotherapy for PD are the correction of endothelial dysfunction, disorders of uteroplacental blood flow and microcirculation; normalization of gas exchange in the "mother-placenta-fetus" system; improvement of placental metabolism; restoration of impaired cell membrane function. Therapy should be prolonged, at least four weeks, of which 10-14 days are spent in hospital.

CONCLUSION

Delivery of pregnant women with IBD, regardless of the presence of PD, is carried out at no more than 40 weeks, and pregnancy management is carried out with timely prevention or treatment of PD. The course and outcomes of amniotomy-induced labor do not differ from those of self-induced labor. Pregnant women with IU should be included in the risk group for the development of PD and timely prevention of PD should be carried out. All pregnant women with VD, in addition to a general clinical examination and routine ultrasound and CTG, should be examined with fibronectin, placental proteins and homocysteine, as well as with the hemostasis system and IAS in AF to predict the development and course of VD.

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