ANTENATAL AND EARLY POSTNATAL ETIOLOGICAL VERIFICATION OF RELEVANT CONGENITAL VIRAL INFECTIOUS DISEASES

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Nonspecificity of clinical, laboratory and instrumental manifestations of congenital infectious diseases, including viral infections, and the diversity of methods for etiological verification of pathogens define both the need to choose the optimal approaches to the diagnosis of this pathology, and the feasibility of testing for a broad range of etiologic agents in case of suspected congenital viral infection. The analysis of current guidelines, international consensus documents issued by specialists, and published results of some studies has shown that identification of the genetic material of the pathogen with the use of amniocentesis/cordocentesis (for cytomegalovirus and parvovirus infections) or in the birth canal (for herpes simplex infection) is the key method for antenatal etiological verification of the widespread viral infections. During the postnatal period, molecular genetic testing is combined with serological diagnosis involving determining specific immunoglobulins M and G, as well as their avidity index.

Keywords: infections, children, pregnancy, cytomegalovirus, parvovirus, herpes viruses

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АНТЕНАТАЛЬНАЯ И РАННЯЯ ПОСТНАТАЛЬНАЯ ЭТИОЛОГИЧЕСКАЯ ВЕРИФИКАЦИЯ АКТУАЛЬНЫХ ВРОЖДЕННЫХ ВИРУСНЫХ ИНФЕКЦИЙ

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Неспецифичность клинико-лабораторных и инструментальных проявлений врожденных инфекционных заболеваний, в том числе вирусной природы, многообразие методов этиологической верификации возбудителей определяют как необходимость выбора оптимальных подходов к диагностике этой патологии, так и целесообразность обследования на широкий спектр этиологических агентов при подозрении на врожденные вирусные инфекции. На основании анализа действующих рекомендаций профессиональных сообществ, международных консенсусов специалистов, результатов отдельных опубликованных исследований показано, что ключевым способом этиологической верификации широко распространенных вирусных инфекций в антенатальном периоде является выявление генетического материала возбудителей при амнио-, кордоцентезе (для цитомегаловирусной и парвовирусной инфекций), в родовых путях (для герпетической инфекции). В постнатальном периоде наряду с молекулярно-генетическим исследованием проводят серологическую диагностику с определением специфических иммуноглобулинов классов М и G и индекса их авидности.

Ключевые слова: инфекции, дети, беременность, цитомегаловирус, парвовирус, герпесвирусы

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Congenital viral diseases (CVDs) are the disorders associated with high mortality and often with disability, which cause substantial socio-economic damage to society [1–4].

According to ICD-10, CVDs belong to class XVI, "Certain conditions originating in the perinatal period", P35 [5].

Historically, among CVDs, rubella is the best known, the classical manifestations of which have been described by N. Gregg, the australian ophthalmologist [6]. The development of the effective vaccine, and making it accessible have led to the diagnosis of congenital rubella (congenital rubella syndrome) currently being extremely rare and practically unknown in the developed countries. According to the State reports "On the state of sanitary and epidemiological well-being of the population in the Russian Federation", a total of seven congenital rubella syndrome cases were registered in the Russian Federation in 2008–2020 [7].

Over the past decades, CVDs caused by other viruses, especially by the viruses of the herpesvirus family, have been playing an increasingly important role. It is known that representatives of almost all types can be transmitted from mother to fetus. However, the rate of such transmission and the impact on the fetus are quite different, and, with respect to a number of types, poorly understood. It is now believed that among CVDs, cytomegalovirus infection is the most common [8]. The existing data on the rate of fetal damage, caused by other viruses of this family, often vary considerably, which is probably due to the use of different research methods and heterogenous groups of the examined individuals [9–15].

Implementation of modern methods for the diagnosis of infections (enzyme immunoassay, chemiluminescence immunoassay, immunocytochemistry, immunohistochemistry, and molecular genetic methods) had naturally led to the conclusion that, in addition to viral infections included in the original TORCH complex (rubella, cytomegalovirus, herpes simplex virus types 1 and 2), fetal damage may be caused by other viruses, such as parvovirus B19, enteroviruses, etc. [16].

It should be noted that available official data on the incidence of infectious diseases in the Russian Federation [7] contain no records of neonatal varicella, as well as of congenital parvovirus and enterovirus infection.

Regarding the congenital infections, the timely etiological diagnosis is crucial, since it defines both the pregnancy management tactics and the possibilities of etiotropic therapy. Meanwhile, antenatal diagnosis is the most topical, since neonatal diagnosis results in the uncontrolled infectious process development in the fetus, and worsens the outcome of congenital disease [15–18].

The Federal Law No. 323-FZ of November 21, 2011 "On the Basics of Health Protection of the Citizens in the Russian Federation" [19] stipulates that from January 1, 2022 the guidelines, developed and approved in accordance with the normative document of the Ministry of Health [20], become the documents, which largely determine the range and the procedure for diagnosis and treatment interventions in Russia. In the light of the current intensive work on preparation and approval of the guidelines for a significant number of nosological forms of the diseases, including some congenital infections, the authors have considered it necessary to provide the review of the modern approaches to antenatal and neonatal etiological verification of some relevant CVDs (herpes simplex types 1 and 2, cytomegalovirus infection, parvovirus B19V infection).

METHODS

The review includes the current foreign guidelines and the international consensus documents issued by professional communities, as well as the basic (according to the authors) review papers, systematic reviews and meta-analyses. The search for information was performed in the databases in English and Russian (MEDLINE, PubMed, Scopus, Web of science, Cochrane Library, eLIBRARY, etc.), the search depth was 15 years (the references to older studies of fundamental importance are provided).

The review does not include any guidelines and consensus documents, related to perinatal aspects of HIV infection, viral hepatitis, and other viral infections, the diagnostic approach to which is reported in the SanPin Sanitary Rules and Regulations 3.3686-21 "Sanitary and Epidemiological Requirements for Communicable Diseases Prevention" [20]; the papers, related to perinatal aspects of CVDs, the guidelines on which are temporary or non-legislative (COVID-19 and Zika virus disease), and the papers, substantiating the choice of the methods and instruments, included in the guidelines (this has been done by the developers of the guidelines and consensus documents).

Common approaches to antenatal etiological diagnosis of CVDs

Antenatal etiological diagnosis can be roughly divided into two stages. The first stage ("screening" stage) involves identifying the signs of probable congenital infection by non-invasive methods (in this context, by methods, preserving the integrity of the amniotic membrane). At this stage, laboratory testing of body fluids (mostly blood, urine, saliva) of the pregnant woman is used in order to detect the genetic material or the antigens of microorganisms, or to reveal the markers of immune response (specific antibodies, antibody avidity determination), together with the imaging methods (most frequently various modifications of fetal ultrasound). Virological method (culturing viruses in a cell culture with subsequent identification) is seldom applied in practice due to its labour-intensity and time-consuming nature [21]. At this stage, one can form a well-informed opinion whether mother has some infectious disease, and assess the condition of the fetus, but he/she is unable to answer the question concerning the presence of congenital infectious disease in the fetus.

The second stage of the antenatal diagnosis ("expert" stage) is to prove or disprove the presence of certain infectious disease in the fetus, i.e. to establish a nosological diagnosis, specifying the etiology, which is decisive for the further pregnancy and childbirth management tactics. At this stage, the invasive procedures are performed aimed at obtaining the fetal biological samples (amniocentesis, cordocentesis) and assessing the samples in order to detect the genetic material of the microorganisms, isolate the pathogen, etc. (this paper does not address the issue of the chorionic villus sampling value for the diagnosis of CVDs).

Cordocentesis and amniocentesis are limited not only by the gestational age, but also by the age of infectious process in the pregnant woman, which reduces their diagnostic value in terms of the fetal disease duration (in case the pathogen has been transmitted). Therefore, in the modern antenatal diagnosis, great importance is attached to the screening stage and to the continuous improvement of screening.

Diagnosis of congenital infection caused by herpes simplex virus types 1 and 2

Genital herpes is one of the most common sexually transmitted infections, most often caused by the herpes simplex virus type 2 (up to 85%) [9, 11, 22]. Fetal infection occurs mainly after the neonate contacts with the virus when passing through the birth canal [23–30].

Herpes simplex virus type 2 (HSV-2) is mainly transmitted through sexual intercourse, the infection is most often acquired at the age of 20–30 years. WHO estimates that 13% of the global population aged 15–49 have this infection [9]. Various studies and reviews estimate that the prevalence of HSV-2 among pregnant women is 20–30%. Furthermore, about 10% of women seronegative for HSV-2 live with seropositive partners and therefore are at risk of being infected with genital herpes during pregnancy [23, 24, 30, 31]. Among serodiscordant couples, women seronegative (having no antibodies) for herpes simplex virus type 1 (HSV-1) antigens have the probability of seroconversion of 3.7%; in women seropositive for antibodies to HSV-1 antigens, the risk of seroconversion for HSV-2 is estimated at 1.7% [23, 24, 30, 31].

The risk of congenital infection is defined by the time of primary maternal infection in relation to the conception, as well as by the fact of the infection reactivation during pregnancy. It is generally accepted that the risk of transmission in case of primary maternal infection in pregnancy is up to 50%, and in case of reactivation the risk is about 4% [9].

HSV transmission occurs in utero, intrapartum and postpartum. It is believed that in 75–85% of cases, fetal infection occurs just before labour after the rupture of membranes, or intrapartum when passing through the infected birth canal. The proportion of intrauterine infection is 5–8% of neonatal

Table. Ultrasound signs of congenital CMVI in the antenatal period [39, 40]

Signs	Rate, %
Intracranial calcification	0.6–17.4
Microcephaly	14.5
Hyperechoic bowel	4.5–13
Intrauterine growth restriction	1.9–13
Subependymal cysts	11.6
Ventriculomegaly	4.5–1.5
Ascites	8.7
Pericardial effusion	7.2
Hyperechoic kidneys	4.3
Enlarged liver	4.3
Thick placenta or placental calcification	4.3
Hepatic calcification	1.4
Hydrops fetalis	0.6

herpes cases [24–28, 30, 31]. The risk of neonatal infection varies between 30–50% for HSV infection occurring during late pregnancy (last trimester); infection in early pregnancy poses a risk of about 1% [11, 24, 25, 29].

Antenatal diagnosis of congenital infection caused by HSV type 1 and 2

Screening in pregnancy aimed to define antibodies to herpes simplex virus in the blood is not recommended, regardless of the history of HSV infection symptoms [25–28, 30].

Assessment of pregnant women is recommended in case of the primary HSV-2 infection acquired during the first two trimesters of pregnancy. In this case, it is necessary to assess the dynamic changes in the levels of IgM, IgG to HSV-2 with an interval of 2–4 weeks in order to reveal seroconversion, as well as to perform a series of molecular genetic tests (polymerase chain reaction, PCR) in order to detect HSV-2 DNA in the vaginal discharge of the pregnant woman, starting from the 32nd week of gestation, to define the tactics for delivery and to find the solution to the question of prescribing antiviral therapy. In case of primary HSV-2 infection development during the last 4–6 weeks of gestation, the risk of vertical transmission and infected neonate is high (41%). In such a case, it is recommended to consider the possibility of cesarean delivery and prescribing etiotropic therapy to both mother and neonate [26, 28, 29].

According to the authors of this article, obligatory etiotropic treatment with direct-acting antiviral agents (acyclovir) in patients with herpes simplex in late pregnancy makes it possible to avoid transabdominal invasive procedures aimed at diagnosis/exclusion of fetal damage, recommended in patients with some other infectious diseases (see below).

Neonatal diagnosis of congenital infection caused by HSV type 1 and 2

Clinical features of neonatal herpes are diverse and non-pathognomonic, which in a number of cases makes the diagnosis difficult. The main manifestations, suggesting the presence of herpes simplex in children below the age of 6 weeks are as follows: rashes in the form of mucocutaneous vesicles, sepsis-like illness, cerebrospinal fluid pleocytosis, seizures, focal neurologic signs, respiratory distress syndrome, episodes of apnea, progressive pneumonitis, thrombocytopenia, conjunctivitis, signs of hepatitis or liver failure, elevated transaminase levels, radiographic signs of brain damage [28, 29].

In the current context, the main method of etiological verification used in such situations involves detecting the viral genetic material by PCR. Swab specimens from the mouth, nasopharynx, conjunctivae, and anus ("surface cultures"), cerebrospinal fluid samples in case of CNS involvement, additional blood samples in case of generalized forms of the disease, and print smears of the contents of vesicles should be obtained. Although the cytopathic effect of the herpes simplex virus becomes obvious during the first five days of culturing, and virological method enables typing of virus strains, such assessment is time-consuming, expensive and lacks practical applicability [29–32].

Diagnosis of congenital cytomegalovirus infection

The highest risk of fetal cytomegalovirus (CMV) infection and the development of severe forms of the disease are observed when the pregnant woman acquires primary cytomegalovirus infection (CMVI) (up to 30% of pregnant women are seronegative). The prevalence of primary CMVI in pregnant women reaches 1% [33, 34].

Antenatal diagnosis of cytomegalovirus infection

Primary maternal CMVI in pregnancy is difficult to suspect due to scarcity of symptomatic forms and nonspecific symptoms (acute respiratory illness with mildest catarrhal symptoms, multifocal lymphadenitis, hepatomegaly and splenomegaly). Ultrasound signs of possible fetal damage are also nonspecific (Table).

Currently, the routine testing of the specific IgM and IgG, the avidity of the latter (quantification in the blood serum), and the viral load (PCR) is not recommended [35–38]. These methods are feasible only in women from the risk groups: under 25 years of age, with signs of acute respiratory illness before week 20 of gestation, multiparous women having organized children, working in children's educational institutions (i.e., being at high risk of infection) [35–38].

Whenever the laboratory, clinical and instrumental signs of primary CMVI (latent viral reactivation, superinfection) are found, assessing the amniotic fluid obtained using amniocentesis (performed on or after the 6th week since the estimated time of the disease onset and not earlier than on the 20th week of gestation) by PCR is indicated. Cordocentesis is not recommended, because, according to the authors of the guidelines and consensus documents, this procedure has no

advantage over amniocentesis in the diagnosis of congenital CMVI [36–38, 41].

In the case that amniocentesis is impossible (or the informed refusal is obtained from the pregnant woman), or in the case of no signs of congenital CMVI detected during the first fetal ultrasound, the repeated screening fetal ultrasounds shall be performed every 2–3 weeks [36–38, 41].

The signs of congenital CMVI progression identified by fetal ultrasound can be considered the indication for terminating a pregnancy for medical reasons [35, 36, 38].

Postnatal diagnosis of cytomegalovirus infection

The indications for laboratory and instrumental examination, aimed to exclude/verify congenital CMVI in the newborn, are as follows: the baby showing clinical signs of congenital infection regardless of their possible etiology (including leukopenia, thrombocytopenia, elevated of hepatic transaminases and direct bilirubin); failure to pass the hearing screen; the documented primary maternal CMVI during pregnancy regardless of the presence or absence of clinical manifestations in the baby; subfebrile temperature, maternal influenza-like illness during the first 20 weeks of gestation; threatened preterm labour; preterm birth, intrauterine growth restriction; genetic material of the pathogen identified in the afterbirth by PCR; signs of intrauterine infection detected during the radiological examination [36, 38, 39].

Identification of the CMV DNA in saliva, urine, and blood during the first three weeks of life is the method of choice for etiological verification of the disease in case of suspected congenital CMVI in the neonate. Saliva (buccal swab can be collected) and urine testing are optimal, and blood testing is less optimal [36, 38, 41–44]. Cerebrospinal fluid is assessed only in case of CNS involvement and lumbar puncture performed with the use of molecular genetic method, involving identification of the CMV DNA. Simultaneous quantification of the CMV IgM and IgG levels in the blood serum, obtained from neonates, is a more affordable, but a less informative method: specific IgM in the first days of life should indicate the primary infection. However, these are not always found in babies with congenital CMVI; false positive results also occur [45]. The high-titer specific IgG antibodies are often found in neonates, however, the concentration of antibodies transferred across the placenta is reduced during the first three weeks of life [36, 38, 45, 46].

Diagnosis of congenital infection caused by human parvovirus B19

Human parvovirus B19 infection (HPV) in pregnancy increases the risk of fetal loss, spontaneous abortion and stillbirth. The risk of adverse pregnancy outcomes reaches 10%. The maternal infection acquired during pregnancy leads to fetal infection in 24–51% of cases [49, 50].

In the majority of cases, when parvovirus infection occurs during pregnancy, the fetus is not affected. However, infection may result in non-immune fetal hydrops due to severe anemia, congestive heart failure and myocarditis, which, with delayed diagnosis and treatment, leads to perinatal loss. The most sensitive period in terms of fetal exposure to human parvovirus B19 (B19V) is the period between 11 and 23 weeks of gestation. The fetal mortality rate in case of infection acquired before 20 weeks of gestation is about 17%, and in case of infection during the later stages this value is about 6%. However, infection acquired during the 3rd trimester can result in intrauterine fetal death without any signs of non-immune hydrops and anemia in 7.5% of cases [51–54].

Antenatal diagnosis of parvovirus B19 infection

About 40% women of childbearing age are seronegative for parvovirus and are therefore susceptible to infection. Approximately 50% of the infected pregnant women are asymptomatic, they develop no classic rash of erythema infectiosum, which is found in children. Atypical symptoms, such as arthralgia, are more frequent in adults than in children.

Testing for HPV is not regulated anywhere in the world and is merely advisory. It is worth noting that screening each and every pregnant woman both for HPV and CMVI is not recommended by professional communities. Identification of non-immune hydrops fetalis or fetal death is the only indication for testing the pregnant woman for parvovirus B19 [49, 51].

Enzyme-linked immunosorbent assay for assessment of specific IgM, IgG antibodies in blood serum of pregnant women is used as a screening laboratory test. While the existence of IgG confirms the past infection, any non-negative response to immunoglobulin M (positive or equivocal IgM test result) would require mandatory molecular genetic testing, involving identification of parvovirus B19 DNA in woman's blood, and the infectious diseases specialist consultation in the future [51–54].

In case of acute maternal HPV infection confirmed by laboratory tests (positive IgM antibody to parvovirus B19V, presence of parvovirus B19 DNA in blood), the constant fetal monitoring with the use of Doppler ultrasonographic assessment of the peak velocity of systolic blood flow in the middle cerebral artery for the diagnosis of fetal anemia and hydrops fetalis performed every 1–2 weeks for 12 weeks is indicated [55]. If ultrasound signs of non-immune hydrops fetalis are detected, the pregnant woman should be hospitalized at the specialized obstetric unit in order to perform amniocentesis or cordocentesis (after 18–20 weeks). If severe fetal anemia is suspected based on the Doppler ultrasound data, cordocentesis is preferred [51, 56].

Postnatal diagnosis of parvovirus B19 infection

The indications for laboratory and instrumental examination required to exclude/verify the congenital HPV infection in the neonate are almost the same as indications for testing for other CVDs (for example, CMVI). Diagnostic approaches are also similar: molecular genetic method (PCR) is the main method of etiological verification, the whole blood and its derivatives are used as a substrate [51, 56, 57]. Other clinical and laboratory tests are performed when clinically indicated.

CONCLUSION

The list of congenital infections goes well beyond the components of TORCH-complex. Currently, there are more than 50 microorganisms capable of causing fetal damage.

Antenatal etiological verification of infections (combined with neonatal verification) is the most important instrument to define the pregnancy and childbirth management tactics, and to assess the prospects for development of the fetus and the neonate. Today, the accurate verification of the pathogen in the fetus involves invasive interventions, which limits the feasibility of those to a certain extent.

Scientific research in the field of congenital infections is, among other things, following the path of studying the

potential of using both qualitative and quantitative methods for assessment of various infectious process markers, which is, for example, reflected in proposals to optimize testing the HIV-positive pregnant women for CMVI [58, 59]. Perhaps in the near future such approaches would become more extensively used, especially in the situations related to mixed infections.

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