

Features of Transmission From Mother to Fetus During Pregnancy and Lactation

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Abstract: The article presents literature data on possible ways of infection of the fetus and newborn in the ante- and postnatal periods of development. It has been shown that the transplacental transmission of the pathogen and the infection of the fetus from the amniotic fluid can contribute not so much to the pathology of the maternal organism itself, but to the stressful situations to which the maternal organism is exposed. During the period of lactotrophic nutrition, stop breastfeeding the baby, in order to prevent infection of the baby, only for very urgent indications.

Key words: infection, placenta, amniotic fluid, breastfeeding.

Relevance. From the very beginning of our review, it should be pointed out that if the normal development of the offspring before conception depends on the state of the body of the mother and father, then after conception it is completely controlled by the mother's body during pregnancy by provisional organs and the placenta [3, 4, 5], then after childbirth in the period of lactation by the mammary gland of the mother [6, 7, 8, 9, 13, 14].

The metabolic and functional usefulness of the placenta largely determines the level of relationship between the mother and fetus throughout fetal development, in addition, amniotic fluid is one of the important components in the development of pregnancy progression. Currently, the placenta is considered as a "nutrition sensor" that coordinates the function of transport, in particular amino acids, although, in addition to the main nitrogen metabolism (through the placenta), the paraplacental transport of amino acids through the fetal membranes and amniotic fluid is well known, but this pathway of transfer is quantitatively insignificant [3, 4, 37]. But at the same time, with maternal pathology, both of these pathways can be the cause of infectious infection of the fetus.

Transmission of infection through the placental barrier.

It is believed that during a normal pregnancy, pathogens of various diseases circulating latently in the mother's blood do not cross the placental barrier and, thus, do not infect the embryo and fetus [3, 11, 12, 20]. At the same time, there is no doubt that pregnancy itself can provoke the transition of a latent disease into a clinically manifested form. In the studies of I. A. Arshavsky and his school [1], it was noted that when *Toxoplasma* is administered during a normal pregnancy to pre-immunized rabbits, the latter do not get sick and *Toxoplasma* does not cross the placental barrier. If, after the introduction of *toxoplasma*, an experimental neurosis is created in an immunized pregnant rabbit, that is, a situation of chronic emotional stress, then the mother becomes ill with toxoplasmosis, and the fetuses are invaded. Therefore, the placental

barrier cannot be absolutely reliable protection against pathogens for the fetus for all occasions, it can be damaged under the influence of adverse environmental factors.

Indeed, numerous papers are published in the pediatric literature every year that report the possibility of passing medicinal pharmacological substances, antigens, antibodies, microbes and viruses through the placenta [2, 3, 36, 37, 40].

Back in 1962, a translation of Heinz Flamm's book "Prenatal Human Infections" was published, which lists the infection and invasion of the embryo and fetus by numerous dermatotropic and neurotropic viruses, various bacteria, protozoa and helminths, leading to severe outcomes such as deformities, embryopathies, fetopathy with high degree of perinatal morbidity and mortality.

However, as noted above, all these phenomena take place under certain pathological conditions of the maternal organism or, as I.A. Arshavsky writes about this: "It is not difficult to assess how truly catastrophic it would be for humanity if in each case viruses, bacteria and protozoa, they crossed the placental barrier and thereby infected or invaded an antenatally developing organism" [1].

The available data indicate in which cases the latent carriage of various pathogens may or may not have negative consequences for the developing embryo and fetus. Thus, it has long been known [31, 32, 41] that syphilis in early pregnancy leads, in most cases, to the cessation of embryonic development. The disease in the middle third of pregnancy causes infection of the fetus due to the passage of spirochetes through the placental barrier. When the disease occurs at the end of pregnancy, the fetus is not infected, since spirochetes do not cross the placental barrier at the end of pregnancy. If syphilis occurs against the background of toxicosis at the end of pregnancy, then in this case the fetus may be infected. Note that this pattern is applicable to a wide variety of infections.

Thus, there is no doubt that the permeability or impermeability of the placental barrier in relation to various microorganisms, viruses and their toxins depends, in particular, on the period of pregnancy when the mother's body is exposed to a particular infection. It is known, for example, that the corresponding antibodies of Rh-negative mothers during pregnancy are detected in case of severe or moderate toxicosis of pregnancy [3, 32]. In addition, the possibility of the transition of macromolecular substances from mother to fetus also depends on the state of the last critical stage of its development [31, 37], since the body's ability to maintain a state of homeostasis, compared with an adult individual, is weakest expressed in the antenatal period.

Transmission of infection through the amniotic fluid.

At present, there is more and more convincing evidence that the digestive system in mammals, including humans, begins to function even in the antenatal period. At this time, the nutrition of the developing fetus is carried out in two ways. One way, as mentioned above, is parenteral, that is, hemotrophic, provided by the supply of nutrients and oxygen from maternal blood through the placenta to the blood of the fetus. Another way - enteral, that is, amniotrophic, is provided by substances contained in the amniotic fluid. It is assumed that amniotic fluid enters the stomach cavity of a developing organism due to the swallowing movements of the fetus.

Currently, a fairly large amount of data has been accumulated on the composition and properties of amniotic fluid, their possible role in prenatal nutrition and the transmission of various infections [15, 16, 17, 30].

It has been established that during the last months of pregnancy, a developing human fetus swallows about 750 ml of amniotic fluid daily [18]. The amniotic fluid contains various nutrients, including carbohydrates and amino acids [19]. A normally developing fetus covers about 10% of its energy needs from amniotic fluid, which contains a high concentration of

nutrients. Based on this, some authors have proposed the use of transamniotic feeding of the fetus, in case of growth retardation [20].

In separate studies, special experiments were carried out to prove the importance of amniotic fluid in the nutrition of a developing fetus [33]. It turned out that ligation of the esophagus in rabbits in the third trimester of gestational development leads to growth retardation by 10%. The weight of the liver in these rabbits is significantly reduced compared to the control. Growth retardation is completely removed if amniotic fluid is injected into the stomach of a developing fetus through a special cannula. The trophic role of the amniotic fluid is manifested in the fact that it contains various nutrients, including proteins, glucose and lactose. Absorption of these substrates through fetal intestinal epithelial cells has been shown in various species of placental animals [34]. Retardation of intrauterine growth due to impaired swallowing of amniotic fluid by the fetus, which is one of the most informative indicators of intrauterine anomalies. It has been shown that newborns with atresia of the esophagus, mouth and nasal openings have significantly less weight than newborns with insufficient anorectal formation [1, 42]. It is interesting to note that an artificial reduction in the ingestion of amniotic fluid in rabbit fetuses leads to hypoplasia of the gastrointestinal tract. Which, it turned out, correlates with the suppression of the secretion of hydrochloric acid by the gastric glands. This phenomenon can be prevented by infusion of rabbit amniotic fluid into the fetal stomach. Therefore, the amniotic fluid contains a specific trophic factor that supports the growth of the gastrointestinal tract [35, 36] of the developing fetus. Amniotic fluid contains a number of biologically active peptides, among which, for example, epidermal growth factor and gastrin regulate the growth of the gastrointestinal mucosa [40, 41].

Currently, all researchers agree that transamniotic nutrition, along with transplacental nutrition, plays an important role in intrauterine development of the fetus. This fact itself suggests that amniotic fluid under certain conditions can play the role of an “open gate” in intrauterine infection of the fetus. Indeed, intrauterine infection of the fetus is possible only if the permeability of the barrier mechanisms of the placenta is impaired, as mentioned above [18, 19]. Such disorders occur as a result of dystrophic changes in the body of pregnant women, sometimes as a result of infection and involvement in the inflammatory process. In this case, the causative agent of a particular disease must overcome such obstacles as: a) integumentary syncytium of the villi; b) the main epithelial layer of the villi; c) villus stroma; d) vascular endothelium of the villi.

In the case of amniotic fluid, the mechanisms of transmission of infection will be completely different. Amniotic fluid in healthy women is sterile, however, they are easily infected. Bacteriological studies of amniotic fluid revealed the presence of pathogenic microorganisms in them even in women who considered themselves healthy. In 2/3 of the surveyed pregnant women, *Candida albicans* was found. Amniotic fluid is especially easily infected with premature rupture of the membranes. It has been shown that intact membranes also do not prevent infection of the amniotic fluid [30]. Viruses brought by the mother's bloodstream are able to adsorb on the amniotic membrane with subsequent infection of the amniotic fluid and the fetus. The infection enters the amniotic fluid from the mother through the chorionic syncytium [40, 41, 42]. Direct infection through the amniochorial plate is also allowed in case of its phlegmonous lesion [32, 43], for example, in case of listeriosis.

Thus, from this rather cursory description of the information available in the literature, we see that the developing fetus can become infected through the placenta and amniotic fluid.

Intrauterine infection depends on the functional state of the mother's body, the stage of fetal development, and a number of other circumstances [1, 32, 34, 40].

Before proceeding to the description of the period of breastfeeding, it should be pointed out that the mammary gland after childbirth is the only organ that connects the body of the mother and the newborn, participating not only in feeding the child with mother's milk, which is ideal in all respects for the baby [9, 21, 22, 23, 24, 38], as well as in the transfer of adoptive immunity to the infant [10, 25, 26, 27, 28, 29, 39]. However, it should be pointed out that during the period of milk feeding, infection of the developing organism can occur in a variety of ways, among which the most likely are the routes of infection through the gastrointestinal tract of the offspring, that is, through mother's milk.

Indeed, breastfeeding has a great advantage, as it prevents many diseases and reduces the risk of early death, but at the same time, it is one of the ways of infecting a child with various infections. Thus, according to the data given in the UNICEF report, infection in the period of late pre- and early postnatal development is about 14%. Let's compare the frequency of infection through breast milk with the dangerous consequences of artificial feeding. In conditions of poor sanitation, infant mortality from diarrhea in bottle-fed infants is 14 times higher than in breastfed infants. If an HIV-infected woman ignored breastfeeding and replaced it with formula-feeding, deaths from diarrhea and respiratory infections often outnumbered those from HIV.

Thus, we can conclude that, of course, HIV infection can be transmitted through mother's milk, but replacing milk with artificial nutrition does no less harm to a developing child. Therefore, the main attention of doctors should be directed to preserving the needs of nursing mothers infected with HIV or other topical infection with strict observance of sanitary and hygienic conditions.

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