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Short Communication

Congenital Infections by Protozoan Parasites in Humans

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Abstract

Maternal parasitic infections pose a potential risk of transmission to the fetus, with dire consequences for the health of both. For this reason, this article on congenital parasitic infections in humans is presented. It is the result of an analysis of current information on this field of knowledge. It reaffirms the fundamental conclusion that some protozoa among parasites can be congenitally transmitted through blood or uterine mucosa to the embryo or fetus. This knowledge is key to establishing adequate prenatal care based on the detection of multiple pathologies and a comprehensive health approach.

Key words: congenital infections; protozoa; fetus; pregnancy; transmission

Introduction

Congenital infections (erroneously called connatal, diaplacental, transplacental, prenatal, and intrauterine infections due to their broad definition, "the intrauterine passage of any etiological agent") have been described since ancient times and are of great importance in newborn morbidity. This fact has been revealed in recent years by the work of researchers, epidemiologists, and clinicians (primarily pediatricians and obstetricians) who have attempted to fully or partially elucidate the pathophysiology of these intrauterine infections, especially parasitic ones. Their importance, due to their frequency and severity, is such that specialized centers and institutes for the diagnosis and treatment of such complex infections and diseases have been created, and will surely continue to be created, throughout the world [1, 2].

This article analyzes important research on the congenital transmission of parasites, specifically unicellular protozoa, with special emphasis on pathogenesis, clinical manifestations, and treatment, in an attempt to concisely uncover what has been reported to date.

Factors involved in congenital transmission

In relation to the etiologic agent, those derived from the host, those related to the timing of infection during pregnancy, and placental lesions. Virulence (parasitic load) and tropism are of great importance. In the former, high parasitemia in acute infections increases the likelihood of congenital transmission, and in the latter, histiotropism favors transmission in chronic infections. The genetic makeup of the parasite also plays a role; for example, the TcV genotype of Trypanosoma cruzi is reported more frequently than the TcII genotype as the cause of congenital trypanosomiasis [2, 3].

Among the host-dependent factors involved in congenital transmission are individual and race susceptibility, which is closely related to genetic makeup and even age, as well as actively acquired immunity (pre-immunity, incomplete immunity for a strain, and immunity to superinfection for homologous strains) and passive immunity (specifically through the passage of antibodies through breast milk and the placenta, primarily the latter). The antibodies provided by the mother to the fetus or newborn last up to four months in the infant, long enough for the infant to produce its own antibodies, generally after the second week of extrauterine life [2].

In relation to the time or moment of pregnancy in which the infection occurs, it is noted that if it occurs before the sixth month the product of conception may not be viable, and if it occurs before the sixth week anomalies may occur in it, for example, in toxoplasmosis if the parasite passes from the mother to the product of conception during the first two months it can cause embryopathies, and after this period generate fetopathies. Placental lesions (of the chorionic ectomesoderm) morphological or by solutions of continuity, generally observed in the third trimester, determine the passage of protozoa to the product of conception [2].

Congenital Parasitic Transmission Routes

Parasites present in maternal blood reach the placental intervillous spaces (maternal-fetal interface) and from there pass into the mesenchymal tissue (a mechanism used by Toxoplasma gondii and T. cruzi) or into the placental spaces without trophoblasts, generally located on the periphery of the placenta [4, 5]. Congenital parasitic transmission can also occur in late pregnancy due to aging of the placenta or due to placental injury caused by the mechanical action of uterine contractions during labor, a mechanism used

by Plasmodium falciparum, Leishmania (Leishmania) donovani, and L. (L.) infantum [6, 7]. By any of these routes, the protozoa reach the amniotic membrane and fluid, and ultimately the conceptus [8].

Maternal immunity at the interface with the conceptus.

The mechanism of action of the maternal immune system in its relationship with the embryo and fetus in parasitic infections and infections in general is not fully understood. However, it is known that the maternal immune system is functional and receptive to the process of tolerance of the conceptus and the fight against pathogenic infections. In this regard, various immune cells (natural killer cells, macrophages, dendritic cells, and lymphocytes) and antimicrobial compounds (defensins [antimicrobial peptides], Toll-like receptors, and nucleotide-binding oligomerization domain proteins) have been observed in the placental endometrial decidua [9, 10].

Protozoa involved in congenital transmission

Chagas disease or American trypanosomiasis is estimated to have an incidence of 10,000 cases per year, ranging from 5% to 22.5%. The most affected countries, in order of frequency, are Bolivia, Chile, Paraguay, Mexico, and Argentina [11, 12]. Twenty cases of congenital transmission of African trypanosomiasis or sleeping sickness have been described, and it is strongly believed that underreporting exists [13]. Congenital toxoplasmosis is described in one case for every 3,000 to 6,000 births. The prevalence of congenital malaria ranges from 0.5% to 33%, primarily due to P. falciparum [14, 15]. Visceral leishmaniasis is the only type of infection capable of causing congenital infection, although the proportions are unknown [16, 17].

Conclusions

Because of the significant impact on the health of the fetus of congenital parasitic protozoan infections, research is being conducted and specialized institutes are being established for their diagnosis and treatment. The pathogenesis or origin of congenital protozoan infections, as well as those of other microbial agents, involves factors specific to the pathogenic organisms, the host, the timing of the infection during pregnancy, and factors attributed to the placenta. Regardless of the congenital route of parasitic transmission, protozoa reach the amniotic membrane and fluid, and ultimately the fetus. It is clearly established that the maternal immune system tolerates the fetus of conception and fights infections. Although congenital protozoan infections have a low prevalence, they should be considered a major public health problem due to the serious alterations they produce in the newborn.

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