CURRENT APPROACH TO TOXOPLASMOsis IN PRENATAL CARE

Bruno José Santos Lima
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/6158584238563073

Luíza Brito Nogueira
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/477921907499876

Victor Araujo de Oliveira Polycarpo
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/1309407063049802

José Genivaldo Santos Andrade
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/4578526655821585

Paulo Henrique Sources de Macedo
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/5045379082206975

Hortência Garcia Nogueira
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/1648872942075382

Ramilly Guimarães Andrade Santos
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/5509896112600945

Ana Mover Vieira de Jesus
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/2822030363557017

Bianca Mendonça Andrade
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/5400225363192464

All content in this magazine is licensed under a Creative Commons Attribution License. Attribution-Non-Commercial-Non-Derivatives 4.0 International (CC BY-NC-ND 4.0).
Abstract: Introduction: Toxoplasmosis is a parasitic disease caused by *Toxoplasma gondii*, an obligate intracellular parasite that exists in 3 forms: oocyst, tachyzoites and bradyzoites. In the case of pregnancy, this infection makes up one of the TORCHS – an acronym used to designate infectious diseases with important fetal repercussions. In fetal infection, resulting from transplacental passage after primary maternal infection during parasitaemia, complications may be relatively common depending on gestational age. With this in mind, this article aims to convene a current approach to toxoplasmosis in prenatal care in order to emphasize its importance, facilitate its procedure and update the scientific literature on the subject. Methodology: This research included national and international scientific publications published between 2006 and 2022. The methodology adopted was a literature review. After this research, the content was sequentially discussed and presented in the order of the prenatal approach, in addition to having created a table and flowchart to organize the summarized ideas. Results: The first contact with the pregnant woman about toxoplasmosis during prenatal care is done through the clinical history and guidelines. Prenatal screening must be performed by detecting IgM and IgG antibodies in the 1st prenatal consultation, since the diagnosis is eminently laboratory. If the patient is susceptible, these antibodies must be repeated every 2-3 months for surveillance. The referral of pregnant women with acute toxoplasmosis infection will allow follow-up for the diagnosis of fetal infection. In the face of an exclusively maternal infection, spiramycin must be promptly started. In the event of occurrence or suspicion (via ultrasound) of fetal infection, triple therapy must be recommended until the end of pregnancy. Conclusions: Although toxoplasmosis represents a common asymptomatic infection in the
adult population, its repercussions during pregnancy are significant and, therefore, must not be overlooked during prenatal care. **Keywords:** Prenatal care, Toxoplasmosis, Tracking.

**INTRODUCTION**

Toxoplasmosis is a parasitic disease caused by Toxoplasma gondii, an obligate intracellular parasite that exists in 3 forms: oocyst (shed only in cat feces), tachyzoites (infectious form of the acute phase of infection) and bradyzoites (slow-growing form observed in cats), tissue cysts).

In primary infection, the cat may shed thousands of oocysts daily for a period of 1-3 weeks. These oocysts can remain infective for periods longer than 1 year in hot and humid environments. Due to this survival rate, it is estimated that 40-80% of the adult population has already been infected by T. gondii, while the incidence of acute toxoplasmosis during pregnancy reaches 1%.

In the case of pregnancy, this infection makes up one of the TORCHS – an acronym used to designate infectious diseases with important fetal repercussions. The mother is usually exposed to the parasite through the ingestion of oocysts present in the environment or the ingestion of bradyzoites or tachyzoites present in meat or meat products. In addition, transmission can also occur via inhalation of dust contaminated by oocysts, poorly washed hands after handling gardens or a litter box with cat feces, virtually via blood transfusion or organ transplantation, among others.

In fetal infection, resulting from transplacental passage after primary maternal infection during parasitaemia, complications may be relatively common depending on gestational age. The later the pregnancy, the greater the probability of transplacental passage, but with less impact on fetal severity. The earlier, the more common are the conditions, such as miscarriage, cataract, strabismus, jaundice, diffuse intracranial calcifications, microcephaly, microphthalmia, restricted intrauterine growth, hydrops fetalis, deafness and hepatosplenomegaly. The classic triad is even described by chorioretinitis, hydrocephalus and intracranial calcifications. It is important to note that women infected prior to conception do not transmit the infection to the fetus, with the rare exception of immunocompromised pregnant women.

With this in mind, this article aims to convene a current approach to toxoplasmosis in prenatal care in order to emphasize its importance, facilitate its procedure and update the scientific literature on the subject.

**METHODOLOGY**

This research included national and international scientific publications published between 2006 and 2022. The methodology adopted for this accomplishment was a bibliographic review through searches in books, articles, monographs and journals published in scientific publications via Scielo, Pubmed and Google Scholar, to survey and analyze what has already been produced on toxoplasmosis in Brazilian prenatal care. The keywords used in the search were: toxoplasmosis, prenatal care, screening.

After this research, the content was sequentially discussed and presented in the order of the prenatal approach, in addition to creating a table and flowchart to organize the summarized ideas.

**RESULT**

**CLINICAL HISTORY**

The first contact with the pregnant woman about toxoplasmosis during prenatal care is done through the clinical history and guidelines. From the initial consultations, the doctor or nurse must already explain about the disease, myths and truths, as well as the
forms of prevention. They must also be aware of the possibility of clinical manifestations of the infection at any time, although most are asymptomatic.

Although the clinical picture is nonspecific and relatively uncommon, it is always valid to count on an assistance service that maintains the suspicion for the infection. About 10 to 20% of pregnant women manifest signs and symptoms such as skin rash, fever, myalgia and adenomegaly, as well as various viral infections. This spectrum can compose differential diagnoses for mononucleosis, rubella, syphilis, cytomegalovirus, tuberculosis and parvovirus, but without ever failing to exclude toxoplasmosis. Due to this imprecision, it is recommended that the hypothesis be raised in all febrile or adenomegalic processes of the pregnant woman, especially if there is a history of contact with felines, handling the earth or raw meat (without glove protection).

**Prenatal Screening**

Prenatal screening must be performed by detecting IgM and IgG antibodies in the 1st prenatal consultation, since the diagnosis is eminently laboratory. If the patient is susceptible, these antibodies must be repeated every 2-3 months for surveillance.

**Serological Interpretation**

Although it is an eminently laboratory diagnosis, its interpretation regarding serological tests is also broad. Results range from an immune patient to chronically infected with no likely fetal repercussions. This list of diagnoses is represented in the following table (Table 1).

IgG antibodies appear about 2 weeks after infection, peak at 6-8 weeks, and decline over the next two years. However, they remain detectable for life. IgM antibodies can appear as early as the first week of infection and usually decline within a few months. However, they sometimes persist for years after the initial infection, so the presence of IgM does not confirm an acute infection (although it does suggest it). IgA antibodies are not usually measured, but they can be detected after 7 days of infection and disappear about 4 months after it. May aid in the diagnosis of acute infection.

It is important to emphasize that cases of acute toxoplasmosis during pregnancy must be reported to epidemiological surveillance in sentinel units, in addition to representing a risk factor that may indicate prenatal care in a high-risk unit, to which they must be referred.

**Diagnosis of Fetal Infection**

The referral of pregnant women with acute toxoplasmosis infection will allow joint follow-up at a referral service for the diagnosis of fetal infection. In theory, this would have an indication to occur via PCR in amniotic fluid from 16 weeks of gestation (gold standard) or identification of IgM antibodies via cordocentesis. However, this is not common in practice due to the low availability of these tests, in addition to the risks.

The most common is to perform monthly ultrasound in patients with acute toxoplasmosis in search of fetal complications (figure 1), such as hydrocephalus, cerebral calcifications, fetal ascites, and changes in hepatic and splenic echotexture. The risk is that normal ultrasound does not rule out congenital infection.

**Treatment**

In the face of an exclusively maternal infection, spiramycin (1g, orally, 8/8h) must be promptly started, even before confirmation of fetal infection, in order to reduce the risk of placental transmission of the parasite. If maternal infection is greater than the 30th week of gestation, triple therapy must be applied: Sulfadiazine (1,500 mg orally,
IgG and IgM -

Susceptible patient.

Serology is recommended every 2-3 months during pregnancy and at delivery. The patient must be instructed on primary prevention.

IgM + and IgG -

Acute infection or false positive.

An IgA test must be requested, which, if positive, confirms acute infection (< 4 months) and, if negative, suggests a false-positive IgM.

In practice, few services measure IgA, so an acute infection is considered to start treatment with Spiramycin 1g (3 million IU) every 8/8h, immediately, with repeat serology in 3 weeks to detect the appearance of IgG.

If IgG remains negative after this period, spiramycin is discontinued and a false-positive result is considered, repeating serology every 3 months.

IgG + and IgM -

Immune patient/chronic infection.

You may have a past infection and there is no risk of reactivation in immunocompetent patients.

IgM + and IgG -

Acute or chronic infection.

Differentiation must be performed using the IgG avidity test, which assesses the strength of antibody-antigen binding. The Ministry of Health reinforces that it must be done on the same serum sample in which positive IgM and IgG antibodies were found.

Low avidity represents acute infection (< 4 months) and high avidity, chronic infection (> 4 months). Thus, in patients with more than 16 weeks of positive serology for IgM and IgG, the avidity test will not guarantee that the infection occurred before pregnancy, justifying treatment in this situation. Some authors suggest repeating the serology in 3 weeks, which will be considered positive if there is a 4x increase in IgG titers.

| Table 1. Serological diagnosis of Toxoplasmosis. |

![Figure 1. Obstetric ultrasound of fetal skull, showing ventricular dilatation. Source: HIGA, Lourenço, et al., Scientia Medica (Porto Alegre), 2010.](image)
12/12h) associated with Pyrimethamine (25 mg, orally, 12/12h) and Folinic Acid (10 mg/day) until the end of pregnancy.

In the event of occurrence or suspicion (via ultrasound) of fetal infection, triple therapy must be recommended until the end of pregnancy, in the same dose schedule mentioned above. The care of this therapy, however, is due to the fact that pyrimethamine must be avoided before 20 weeks of gestation due to the teratogenic effect (class C). The use of Sulfadiazine in the 3rd trimester must be monitored due to the possibility of Kernicterus in the newborn. Thus, spiramycin is the medication to be continued at both ends of pregnancy (< 20 weeks and > 37 weeks).

If PCR of amniotic fluid is negative, maintenance of spiramycin is indicated until the end of pregnancy. In places where procedures for investigating fetal involvement are not available, the possibility of using the aforementioned therapeutic regimen for fetal treatment must be evaluated.

**CONCLUSIONS**

Although toxoplasmosis represents a common asymptomatic infection in the adult population, its repercussions during pregnancy are significant and, therefore, must not be overlooked during prenatal care.

Over the years, some studies have given greater robustness to the analysis of the impacts on early diagnosis and treatment of some TORCHS, with discordant effects between each disease. For example, while screening for herpes is not recommended in prenatal care, it is known that toxoplasmosis is preventable and has a well-defined treatment.

After new foundations, the current approach to toxoplasmosis in prenatal care is summarized in figure 2 and its practice must be well established in the training of the entire team responsible for the obstetric service of primary and specialized care. After all, the avoidable impacts on fetal morbidity and mortality can have repercussions for a lifetime.

---

**Figure 2. Current approach to toxoplasmosis in prenatal care.**

- **Toxoplasmosis in pregnancy**
  - Suspicious clinical history + positive serological test
  - Positive screening* (IgM+ and IgG-** or IgM+ and IgG+ with low avidity)
  - Maternal Diagnosis
  - Treatment with Spiramycin (or triple therapy if GA > 30 weeks) +
  - Referral to a referral service for fetal diagnosis
  - Positive fetal diagnosis***
  - Triple therapy****

*Antibodies repeated every 2-3 months if susceptible patient.
**If IgA+ or in the impossibility of measuring lgA.
***PCR+ in amniotic fluid, IgM+ in cordocentesis or presumptive USG.
****Sulfadiazine + Pyrimethamine + Folinic Acid.
REFERENCES


