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Perinatal Outcomes in Primary Toxoplasmosis during Pregnancy: Treatment Efficacy and Analysis of Neonatal Complications

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Abstract

This article presents the results of a retrospective cohort study conducted in perinatal centers of Stavropol and Vladikavkaz between 2023 and 2025. The study aimed to compare pregnancy, delivery, and neonatal outcomes during the first three months of life in women with primary toxoplasmosis based on whether they received specific therapy. The study included 110 women who received treatment, 40 women who declined therapy, and 48 non-infected pregnant women as a control group. In the treatment group, the rate of vertical transmission was 8.2%, no symptomatic forms of congenital toxoplasmosis were observed, and pregnancy complications occurred at the same frequency as in the control group. In the group that declined treatment, the risk of vertical transmission reached 50.0%, symptomatic disease developed in 35% of infected children, and among those infected during the first trimester, severe central nervous system and ocular lesions were observed in 80% of newborns. Half of the children with congenital infection required hospitalization during the first three months of life. Multivariate analysis confirmed that the absence of therapy increased the risk of vertical transmission by 12.4-fold. Timely administration of spiramycin or combination therapy reduces the risk of fetal infection by more than sixfold and completely prevents the development of symptomatic forms of congenital toxoplasmosis. These findings justify the need for mandatory toxoplasmosis screening during pregnancy, immediate initiation of therapy upon detection of primary infection, and long-term follow-up of children infected *in utero*.

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Introduction

Toxoplasmosis is a parasitic disease caused by the protozoan *Toxoplasma gondii*. The pathogen is an intracellular parasite capable of affecting various organs and tissues in humans (Matta *et al*., 2021; Grant & Wallace, 2024; Beltrame *et al*., 2025). The greatest danger of this infection lies in pregnant women due to the possibility of vertical transmission from mother to fetus (Mandelbrot *et al*., 2021; Trotta *et al*., 2021; Deganich *et al*., 2022; Kunie *et al*., 2025).

In immunocompetent adults, primary toxoplasmosis is mostly asymptomatic or presents with nonspecific manifestations such as low-grade fever, lymphadenopathy, and mild malaise (Milne *et al*., 2023; Osluf *et al*., 2024; Cerisola *et al*., 2025). However, for the fetus, especially during early gestation, infection can have severe consequences (Zhang *et al*., 2024; Morgan & Foster, 2025).

Congenital toxoplasmosis is characterized by a wide spectrum of clinical manifestations. Severe cases may result in antenatal fetal death or spontaneous miscarriage (McCluskey & Sato, 2024; Mazzilli *et al*., 2025). Infants who survive intrauterine infection may develop structural lesions of the central nervous system (Bollani *et al*., 2022; Uneno *et al*., 2024; Shen & Bao, 2025). The classic triad of congenital toxoplasmosis includes hydrocephalus, intracranial calcifications, and chorioretinitis (Dubey *et al*., 2021; Giorgione *et al*., 2022; Kota & Shabbir, 2023; Lindstrom *et al*., 2025). These conditions often lead to disability from the first months of life.

Beyond manifest forms, congenital infection can also be asymptomatic. Up to 70% of infants infected *in utero* are born without clinical signs. However, a significant proportion of these patients develop delayed complications in subsequent months and years, including chorioretinitis, sensorineural hearing loss, and psychomotor and intellectual developmental delays (Fortin & Mulkey, 2023; Lashch *et al*., 2023; Tran *et al*., 2023; Anunziata & Cussa, 2024; Gundeslioglu *et al*., 2024).

In the Russian Federation, toxoplasmosis screening is mandatory for pregnant women. Upon registration for pregnancy, levels of specific antibodies of classes G and M to *Toxoplasma gondii* are determined. Detection of class G immunoglobulins indicates past infection and protective immunity. Absence of both antibody classes indicates a seronegative status, requiring dynamic monitoring with repeated testing each trimester (Rodrigues & Junior, 2022; Csep *et al*., 2024).

Seronegative women constitute the risk group for primary infection during pregnancy. Across different regions of the Russian Federation, such patients account for 60% to 80% of all pregnant women. Primary toxoplasmosis during gestation is registered within this group (Szécsényi *et al*., 2024; Clark & Foster, 2025).

Human infection with *Toxoplasma gondii* occurs primarily through the alimentary route. The main transmission factor is consumption of undercooked meat, predominantly pork and lamb, accounting for approximately 50% of cases. Additional routes of infection include consumption of unwashed vegetables, fruits, and herbs contaminated with soil, as well as unpasteurized goat milk. Contact with feces of infected cats, which excrete parasite oocysts, also represents a significant risk factor, particularly when cleaning cat litter boxes or working with soil without personal protective equipment (Ducrocq *et al*., 2021; Wehbe *et al*., 2022; Ganea *et al*., 2024; Cardona-López *et al*., 2025; Nasiri *et al*., 2025).

The incidence of primary toxoplasmosis during pregnancy in the Russian Federation ranges from 1 to 8 cases per 1000 pregnant women. The rate of congenital toxoplasmosis reaches 1.5 cases per 1000 newborns. For a city with a population of 500,000 people, where approximately 4000 births occur annually, this corresponds to 6–7 children with congenital infection per year. Symptomatic disease develops in 20–25% of infected newborns (Bulgakova & Chuelov, 2025; Markin *et al*., 2025; Raza *et al*., 2025).

This study aimed to compare pregnancy, delivery, and neonatal outcomes during the first three months of life based on whether women with primary toxoplasmosis received specific therapy. To achieve this goal, three observation groups were formed: women who received treatment, women who declined treatment, and a control group of non-infected pregnant women. This design allowed assessment of the clinical effectiveness of therapy in real-world practice and identification of differences in perinatal outcomes depending on patient choice.

Materials and Methods

The study was conducted at the perinatal centers of Stavropol and Vladikavkaz from January 2023 to December 2025. The study design was a retrospective cohort, comparative, with formation of observation and control groups.

The study included women diagnosed with primary *Toxoplasma gondii* infection during pregnancy. Inclusion criteria were documented seroconversion, defined as the appearance of specific immunoglobulin G in a previously seronegative patient, or detection of low-avidity immunoglobulin G in combination with positive immunoglobulin M (Ota *et al*., 2024; Maslyaninova *et al*., 2025; Ming *et al*., 2025). All included women were followed up at antenatal clinics in their place of residence and delivered at perinatal centers.

Exclusion criteria were multiple pregnancy, severe decompensated somatic pathology, including HIV infection and oncological diseases, as well as fetal congenital malformations unrelated to toxoplasmosis. Patients with incomplete data on the pregnancy course or neonatal outcomes were also excluded from analysis.

The total sample size was 198 patients. Women were distributed into groups based on whether they received specific toxoplasmosis therapy. The first group consisted of 110 women with primary toxoplasmosis who completed a full course of treatment according to clinical guidelines. The second group comprised 40 women with primary toxoplasmosis who declined therapy. Reasons for refusal were recorded based on medical documentation. The third group consisted of 48 women with no evidence of toxoplasmosis infection throughout pregnancy, confirmed by negative serological results at registration and during each trimester. The groups were comparable in age, parity, and presence of concomitant somatic pathology.

Diagnosis of primary toxoplasmosis was performed using serological methods. Immunoglobulins M and G were determined by enzyme-linked immunosorbent assay. When positive immunoglobulins M and G were detected, the immunoglobulin G avidity index was measured (Ribeiro *et al*., 2024; Teimouri *et al*., 2024). Values below 30% were considered low-avidity, confirming infection within the preceding 3–5 months. Seroconversion was established when immunoglobulin G appeared in patients who had previously tested negative for these antibodies.

Prenatal diagnosis of fetal infection included amniocentesis. The procedure was performed no earlier than 18 weeks of gestation and no earlier than 4 weeks after the estimated date of maternal infection. Amniotic fluid samples were tested by polymerase chain reaction for *Toxoplasma gondii* DNA (Grose *et al*., 1989).

Therapy for primary toxoplasmosis was administered according to current clinical protocols. Women with confirmed diagnosis received spiramycin at a dose of 3 million international units twice daily (De Santis *et al*., 2024; Cuenca-Martínez *et al*., 2025). The duration of a single course was 3–4 weeks, repeated as needed. Patients infected after 18 weeks of gestation or with evidence of possible fetal infection received combination therapy including pyrimethamine, sulfadiazine, and folinic acid.

Follow-up of newborns was conducted during the first 3 months of life. Diagnosis of congenital toxoplasmosis was established by detection of *Toxoplasma gondii* DNA by polymerase chain reaction in blood, urine, or cerebrospinal fluid within the first 10 days of life, as well as by serological testing for specific immunoglobulins M and A. All infants underwent neurosonography to assess brain structures, ophthalmologic examination to rule out chorioretinitis, audiological screening to detect sensorineural hearing loss, as well as complete blood count and biochemical analysis to assess liver function and detect thrombocytopenia and anemia.

The study design scheme is shown in **Figure 1**.

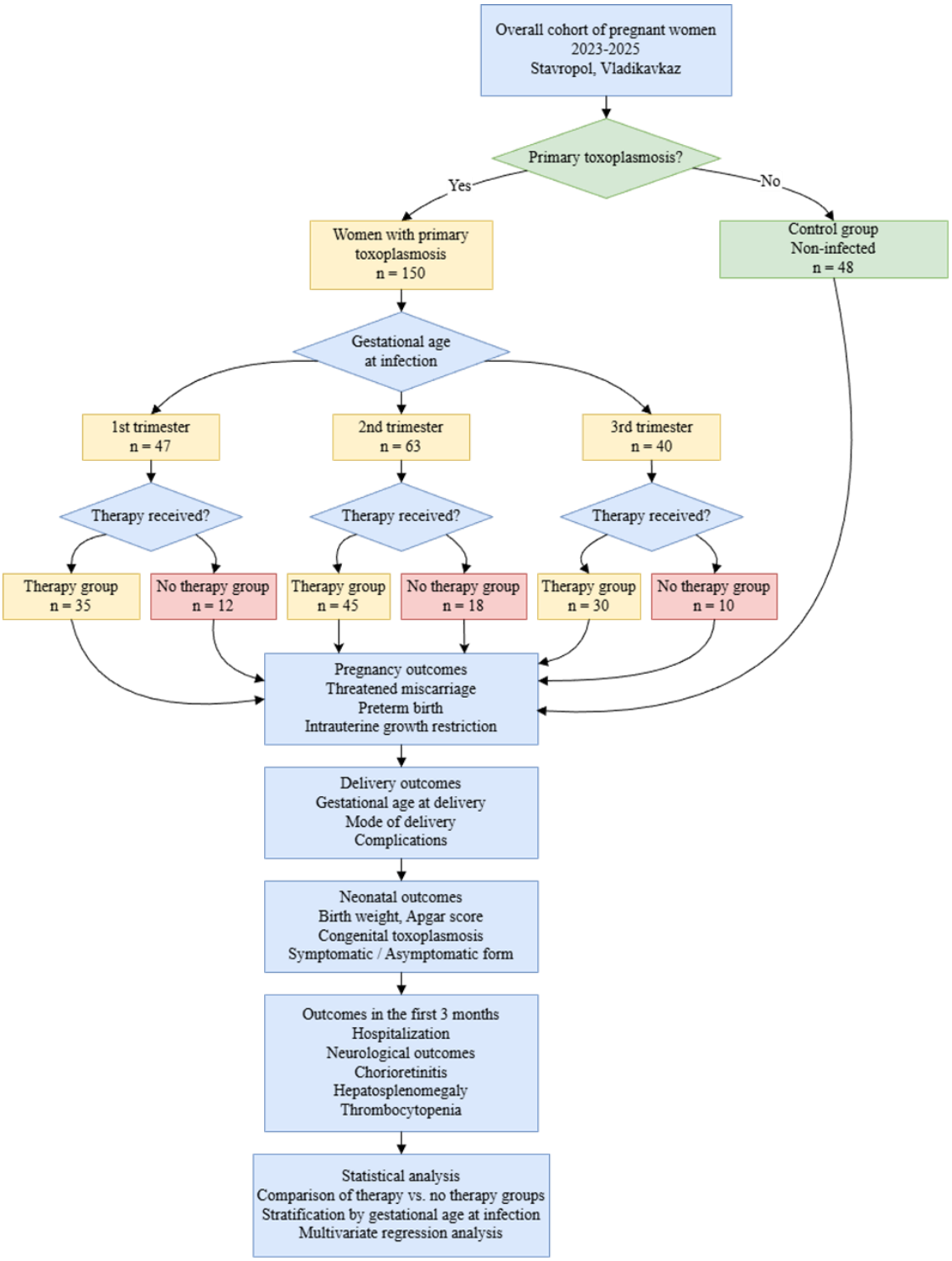

Figure 1. Study design: formation of observation groups with stratification by gestational age at infection and stages of perinatal outcome analysis.

Data analysis was performed using descriptive statistics methods. Categorical variables are presented as absolute numbers and percentages. The chi-square test was used to compare the three groups; Fisher's exact test was applied when the number of observations was small. The Student's t-test was used for comparison of continuous variables. Multivariate logistic

regression analysis with calculation of odds ratios and 95% confidence intervals was performed to identify risk factors for adverse outcomes. Differences were considered statistically significant at $p < 0.05$.

Results and Discussion

The study included 150 women with primary toxoplasmosis diagnosed during pregnancy and 48 women in the control group. The distribution of patients by gestational age at infection and therapy administration is presented in **Table 1**.

Mean age was 28.4 years in the treatment group, 27.9 years in the no-therapy group, and 28.1 years in the control group. The groups showed no statistically significant differences in parity, presence of concomitant somatic pathology, or socioeconomic status. Among infection risk factors, consumption of undercooked meat was the most common, reported by 62% of patients. The presence of a cat with outdoor access was recorded in 41% of women. Work with soil without gloves was noted in 28% of patients.

Table 1. Distribution of patients by gestational age at infection and therapy

Gestational age at infection	Treatment group (n=110)	No-therapy group (n=40)	Total (n=150)
1st trimester (1–12 weeks)	35 (31.8%)	12 (30.0%)	47 (31.3%)
2nd trimester (13–26 weeks)	45 (40.9%)	18 (45.0%)	63 (42.0%)
3rd trimester (27–40 weeks)	30 (27.3%)	10 (25.0%)	40 (26.7%)

Distribution of gestational age at infection was comparable between the treatment and no-therapy groups (p=0.78).

Pregnancy outcomes differed significantly across the three groups. The most favorable outcomes were observed in the control group and the treatment group. In the no-therapy group, complication rates were significantly higher **(Table 2)**.

Threatened miscarriage in the first and second trimesters was nearly three times more frequent in the no-therapy group compared to the treatment group. Preterm birth occurred in one-fifth of women in the no-therapy group, compared to one in eighteen in the treatment group. Intrauterine growth restriction was detected in 25% of newborns in the no-therapy group, nine times higher than in the treatment group.

Table 2. Pregnancy complications by group

Complication	Treatment group (n=110)	No-therapy group (n=40)	Control group (n=48)	p (treatment vs no-therapy)
Threatened miscarriage	13 (11.8%)	14 (35.0%)	5 (10.4%)	<0.001
Preterm birth	6 (5.5%)	8 (20.0%)	2 (4.2%)	0.008
Intrauterine growth restriction	3 (2.7%)	10 (25.0%)	1 (2.1%)	<0.001
Placental insufficiency	8 (7.3%)	12 (30.0%)	3 (6.3%)	<0.001

Pregnancy complication rates in the treatment group did not differ from the control group, whereas all rates in the no-therapy group were significantly higher.

Prenatal diagnosis by amniocentesis was performed in 128 patients with primary toxoplasmosis. Positive polymerase chain reaction for *Toxoplasma gondii* DNA in amniotic fluid was obtained in 9 women from the treatment group (8.2%) and 20 women from the no-therapy group (50.0%). The difference between groups was statistically significant (p<0.001).

Vertical transmission rates by gestational age at infection are presented in **Table 3**.

In the treatment group, congenital toxoplasmosis was diagnosed in 9 newborns (8.2%). All cases were asymptomatic. None of the children in this group showed clinical manifestations at birth; all were born at term with normal birth weight and no signs of central nervous system or visceral involvement.

In the no-therapy group, congenital toxoplasmosis was confirmed in 20 newborns (50.0%). Among these, 7 children (35%) had symptomatic disease, and 13 children (65%) were asymptomatic.

Table 3. Frequency of congenital toxoplasmosis by gestational age at infection and therapy

Gestational age at infection	Treatment group	No-therapy group
1st trimester	2 of 35 (5.7%)	5 of 12 (41.7%)
2nd trimester	4 of 45 (8.9%)	9 of 18 (50.0%)
3rd trimester	3 of 30 (10.0%)	6 of 10 (60.0%)

In the treatment group, vertical transmission rates did not depend on gestational age at infection (p=0.72). In contrast, in the no-therapy group, there was a trend toward increased risk at later stages.

Among children with symptomatic congenital toxoplasmosis (7 children), clinical manifestations varied and depended on the gestational age at maternal infection. All cases of severe symptomatic disease with central nervous system and ocular involvement were associated with infection in the first trimester. Data are presented in **Table 4**.

In the no-therapy group, four children infected in the first trimester developed the classic triad of congenital toxoplasmosis: hydrocephalus, intracranial calcifications, and chorioretinitis. One child in this subgroup was born with isolated hydrocephalus without pronounced calcifications. Among the three children infected in the second trimester, hepatosplenomegaly with jaundice and moderate thrombocytopenia predominated. Chorioretinitis at birth was detected in two children from this subgroup. In children infected in the third trimester, clinical manifestations at birth were absent or limited to mild hepatosplenomegaly.

Table 4. Structure of symptomatic congenital toxoplasmosis by gestational age at infection (no-therapy group)

Clinical manifestation	1st trimester (n=5)	2nd trimester (n=9)	3rd trimester (n=6)
Hydrocephalus	4 (80%)	2 (22.2%)	0 (0%)
Intracranial calcifications	3 (60%)	1 (11.1%)	0 (0%)
Chorioretinitis at birth	3 (60%)	2 (22.2%)	0 (0%)
Hepatosplenomegaly	4 (80%)	5 (55.6%)	1 (16.7%)
Jaundice	3 (60%)	4 (44.4%)	1 (16.7%)
Thrombocytopenia	3 (60%)	3 (33.3%)	0 (0%)

The classic triad of congenital toxoplasmosis (hydrocephalus, calcifications, chorioretinitis) was observed exclusively with first-trimester infection. In third-trimester infection, clinical manifestations were limited to moderate hepatosplenomegaly and jaundice.

Follow-up of children during the first three months of life revealed significant differences between groups **(Table 5)**. In the treatment group, all children with congenital toxoplasmosis remained asymptomatic throughout the observation period. None required hospitalization for infectious complications. Ophthalmologic examinations at 1 and 3 months of age revealed no signs of chorioretinitis. Dynamic neurosonography showed no new calcifications or progression of ventriculomegaly.

In the no-therapy group, 10 of 20 children with congenital toxoplasmosis (50%) required hospitalization in the neonatal pathology unit. Hospital stays ranged from 14 to 45 days. Seizures requiring anticonvulsant therapy were recorded in two children during the first month of life. One child was diagnosed with progressive hydrocephalus requiring ventriculoperitoneal shunting.

Among the three children in the no-therapy group who were born without clinical manifestations but had confirmed infection, delayed manifestations were identified during the first three months. Two were diagnosed with chorioretinitis in the second month of life. One child showed psychomotor developmental delay in the third month of life. These children were also hospitalized for anti-inflammatory and antiparasitic therapy.

In the control group, no cases of congenital toxoplasmosis were registered during the three-month follow-up. Hospitalizations of children in this group were due to causes unrelated to toxoplasmosis and did not exceed average population rates.

Table 5. Complications during the first three months of life in children with congenital toxoplasmosis

Complication	Treatment group (n=9)	No-therapy group (n=20)	p
Hospitalization in the neonatal pathology unit	0 (0%)	10 (50%)	0.007
Seizures	0 (0%)	2 (10%)	0.320
Progressive hydrocephalus	0 (0%)	1 (5%)	0.550
Chorioretinitis (newly diagnosed)	0 (0%)	2 (10%)	0.320
Psychomotor developmental delay	0 (0%)	1 (5%)	0.550

No complications were recorded in the treatment group during the first three months of life. In the no-therapy group, half of the children with congenital toxoplasmosis required hospitalization, and 10% developed neurological and ophthalmological complications.

Multivariate logistic regression analysis was performed to identify independent risk factors for congenital toxoplasmosis. The model included the following variables: absence of therapy, gestational age at infection (first trimester as reference category), maternal age, and parity.

Absence of therapy was the most significant risk factor. Women who declined treatment had a 12.4-fold higher probability of transmitting infection to the fetus compared to those receiving therapy. First-trimester infection increased the risk of severe congenital toxoplasmosis by 8.7-fold compared to third-trimester infection. Maternal age and parity showed no statistically significant association with vertical transmission risk.

This study demonstrated substantial differences in perinatal outcomes between pregnant women with primary toxoplasmosis who received specific therapy and those who declined treatment. The findings not only allow assessment of the effectiveness of current therapeutic approaches but also identify key factors determining prognosis for the fetus and newborn.

A schematic representation of toxoplasmosis outcomes for pregnancy and the fetus, depending on therapy administration, is shown in **Figure 2**.

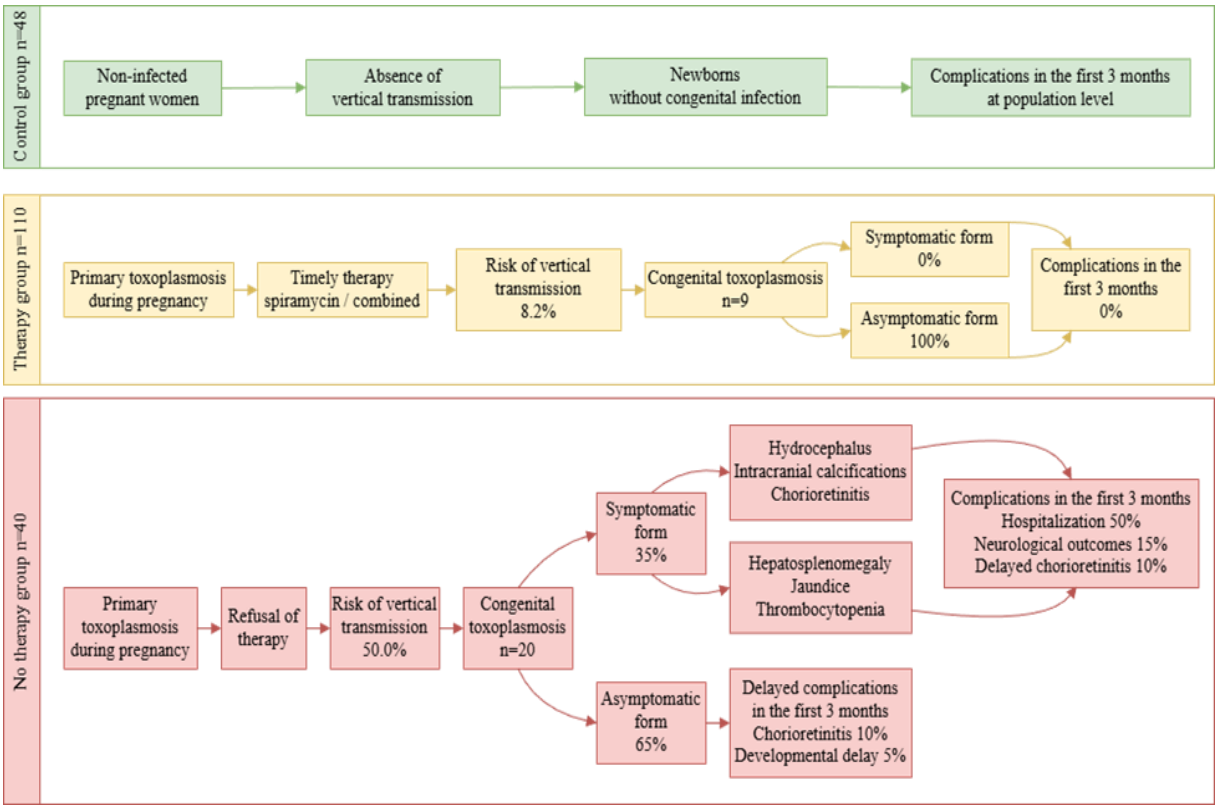

Figure 2. Comparative scheme of perinatal outcomes in primary toxoplasmosis depending on therapy administration.

The key finding of this study was confirmation of the high efficacy of specific therapy in preventing vertical transmission of toxoplasmosis. In women receiving treatment, the rate of fetal infection was 8.2%, compared to 50.0% among those who declined therapy. Thus, timely administration of spiramycin and, when indicated, combination therapy reduced the risk of transmission by more than sixfold. These findings align with results from large observational studies conducted in European prenatal diagnosis centers. In studies covering long-term experience with pregnant women with primary toxoplasmosis, vertical transmission rates with timely treatment ranged from 5% to 10% (Aksoy Sanay *et al*., 2024; Mickevičius *et al*., 2024; Cetinkaya Demir *et al*., 2025; Nakajo & Nishiura, 2025). In historical control groups where treatment was absent or delayed, fetal infection rates reached 40–60% depending on gestational age (Avelino *et al*., 2014; Buonsenso *et al*., 2022; Jabin & Guthrie, 2025; Silva Santos *et al*., 2025; Walana *et al*., 2026).

Of particular note is the observation that treatment efficacy was independent of gestational age at infection. In our treatment cohort, transmission rates were 5.7% for first-trimester infection, 8.9% for second-trimester, and 10.0% for third-trimester, with no statistically significant differences between these values. This indicates that timely initiated therapy neutralizes the established pattern whereby transmission risk increases with advancing gestation. Similar findings were reported in studies from the French national toxoplasmosis screening program, where treatment effectiveness remained consistently high across all trimesters when initiated early (Lopez *et al*., 2000; Picone *et al*., 2020; Hsiao *et al*., 2024; Rohilla *et al*., 2025; Wong *et al*., 2025).

The most dramatic differences between groups concerned the development of symptomatic congenital toxoplasmosis. In the treatment group, no cases of clinically manifest infection were recorded among newborns. All nine children with confirmed congenital toxoplasmosis were born without any disease manifestations. Conversely, in the no-therapy group, one-third of infected children (35%) had clinical manifestations at birth, with severity directly dependent on gestational age at maternal infection (Baquero-Artigao *et al*., 2013; Abedian *et al*., 2024; Alhossan *et al*., 2024; Elsayed *et al*., 2025).

The classic triad of congenital toxoplasmosis—hydrocephalus, intracranial calcifications, and chorioretinitis—was observed exclusively with first-trimester infection. Among five children infected during this period, four developed severe central nervous system and ocular lesions leading to disability from the first days of life. In second-trimester infection, clinical manifestations were less severe and predominantly limited to hepatosplenomegaly, jaundice, and hematologic abnormalities. In third-trimester infection, symptomatic disease at birth was absent.

These findings are fully consistent with pathophysiological concepts of congenital toxoplasmosis development (Moghaddami *et al*., 2024; Novak & Dvorak, 2025; Arshad *et al*., 2026). Infection during early gestation, when active differentiation of central nervous system tissues occurs, leads to gross structural

abnormalities. Infection in later stages, when major brain structures are already formed, more often results in asymptomatic disease or minimal visceral involvement. Similar patterns have been described in multicenter studies on the natural course of congenital toxoplasmosis in untreated patients (Borges *et al*., 2019; Prescott *et al*., 2023; Solmell *et al*., 2024).

Follow-up of children during the first three months of life revealed important differences between groups. In the treatment group, no delayed complications were recorded. All children with asymptomatic congenital infection remained clinically healthy throughout the observation period. Ophthalmologic examinations and neurosonography showed no progression of pathological changes.

In the no-therapy group, three children born without clinical manifestations developed delayed complications within the first three months. Two developed chorioretinitis, and one showed psychomotor developmental delay. Additionally, one child with symptomatic disease at birth developed progressive hydrocephalus requiring surgical intervention. These findings underscore the importance of long-term follow-up for children with intrauterine toxoplasmosis, even in the absence of clinical manifestations at birth. Publications on long-term outcomes of congenital toxoplasmosis indicate that chorioretinitis can present at any age. At the same time, sensorineural hearing loss and psychomotor developmental delay are often identified only in the second or third year of life (Garweg *et al*., 2022; Schneider & Krüger, 2025).

Beyond direct effects on the fetus, primary toxoplasmosis significantly impacted the pregnancy course. In the no-therapy group, rates of threatened miscarriage, preterm birth, intrauterine growth restriction, and placental insufficiency were significantly higher than in the treatment and control groups, with differences reaching threefold to ninefold. Our findings on higher rates of preterm birth and intrauterine growth restriction in untreated patients align with retrospective studies reporting increased risk of obstetric complications in the absence of timely therapy (Losa *et al*., 2024; Miciak & Jurkiewicz, 2024; Salomè *et al*., 2024).

The mechanisms underlying these complications are likely related to inflammatory reactions in the placenta triggered by pathogen invasion. Spiramycin therapy, which accumulates in placental tissue at high concentrations, not only prevents vertical transmission but also suppresses local inflammation, thereby reducing the risk of placental insufficiency and associated complications (Kalantari *et al*., 2021; Rani & Gehrke, 2025).

Multivariate regression analysis identified two independent risk factors for adverse outcomes: absence of therapy and early gestational age at infection. Absence of treatment increased the probability of vertical transmission 12.4-fold compared to those receiving therapy. First-trimester infection increased the risk of severe congenital toxoplasmosis 8.7-fold compared to third-trimester infection. Maternal age and parity showed no statistically significant association with vertical transmission risk, consistent with epidemiological studies indicating that sociodemographic factors primarily influence infection acquisition rather than transmission probability after maternal infection has occurred (Iriti *et al*., 2024; Jaafar & Rahman, 2024; Moafa *et al*., 2024; Woldegerima *et al*., 2024; Alnabulsi *et al*., 2025).

Several limitations should be considered when interpreting these results. First, the retrospective design depends on the completeness and accuracy of medical records; some complications or comorbidities may not have been fully documented. Second, treatment allocation and no-therapy groups were non-randomized, as patients themselves made treatment decisions. Reasons for refusal may relate to personality traits, education level, or adherence to medical follow-up, introducing potential selection bias that cannot be fully eliminated in retrospective analysis. Third, follow-up of children was limited to the first three months of life, whereas delayed complications such as chorioretinitis, sensorineural hearing loss, and intellectual developmental delay may manifest at later stages.

The findings of this study have direct practical implications. They convincingly demonstrate that timely therapy for primary toxoplasmosis during pregnancy reduces the risk of fetal infection by sixfold and completely prevents symptomatic forms of congenital infection. For patients making treatment decisions, this information should serve as a key argument. Furthermore, these results support strict adherence to screening protocols. Identification of seronegative women in early pregnancy and their dynamic follow-up enables diagnosis of primary infection at the earliest possible stage when therapy is most effective.

Conclusion

This study of 150 patients with primary toxoplasmosis and 48 control women demonstrated high efficacy of specific therapy in preventing vertical transmission and severe complications in the fetus and newborn.

In the absence of treatment, the risk of fetal infection was 50.0%, symptomatic congenital toxoplasmosis developed in 35% of infected children, and with first-trimester infection, severe central nervous system and ocular lesions reached 80%. Half of all children with congenital infection required hospitalization during the first three months of life, and 10% of children born without symptoms developed delayed complications.

Timely therapy with spiramycin or combination regimens reduced vertical transmission risk to 8.2%, more than sixfold lower than in the no-therapy group. Symptomatic congenital toxoplasmosis was completely absent in the treatment group. Pregnancy complications (threatened miscarriage, preterm birth, fetal growth restriction) occurred at the same frequency as in non-infected women. During the first three months of life, children infected *in utero* whose mothers received therapy showed no clinical manifestations or delayed complications.

Multivariate analysis confirmed that the absence of therapy increased vertical transmission risk 12.4-fold, while first-trimester infection increased the risk of severe symptomatic disease 8.7-fold compared to third-trimester infection.

Thus, primary toxoplasmosis during pregnancy is a manageable infection. Current screening and therapeutic approaches can

prevent severe consequences for the fetus and newborn. Refusal of therapy carries an unacceptably high risk of delivering a child with severe disabling pathology.

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Conflict of interest: None

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Ethics statement: This study was conducted in accordance with the ethical standards of the institutional research committee and with the principles of the Declaration of Helsinki (2013 revision). The study represents a retrospective analysis of clinical cases, and all patient data were processed in compliance with applicable data protection regulations. Patient confidentiality was maintained throughout the study by anonymizing all clinical data, with removal of all personal identifiers prior to analysis and publication.

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