Toxoplasmosis in Pregnancy

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14.1 Introduction

Toxoplasma gondii, an organism causing toxoplasmosis, is an intracellular protozoan commonly infecting mammals and other homoeothermic animals. It belongs to the class *coccidian* and consists of only one species. It causes fatal food-borne disease in the USA and is the third most common cause for it [1]. It is the disease of immune-compromised, involving patients affected with HIV AIDS, infants, patients suffering from chronic illnesses, cancers, or patients on immunosuppressant but rarely causes symptomatic infection in healthy individuals. One-third of healthy people infected with Toxoplasma remain asymptomatic while others may suffer from flu-like symptoms like headache, myalgia and rarely become carriers resulting in the spread of the disease without getting the illness for their entire life [2].

It is one of the most common infections in humans. Worldwide, the seroprevalence for Toxoplasmosis varies from 1 to 100% depending upon the environmental and the socio-economic conditions, food products, harvesting practices, hygiene, and human practices [3]. The incidence is higher in warm and humid areas due to higher transmission rates [4]. An American study demonstrated that 11% of women belonging to reproductive age (15–44 years) had IgG antibodies to *T. gondii* in their serum if born in America but in women born outside the US, the prevalence increased to 28.1% [5].

Toxoplasmosis in pregnant women presents clinically in a similar way as others but because of its high prevalence and possible affection on the fetus, it needs exclusive consideration. Congenital toxoplasmosis, one of the 'T'ORCH infections, is a morbid condition with an incidence of about 400–4000 per year [6]. The seroprevalence of toxoplasmosis in India varies from 4.7 to 51.8%. The acquisition of toxoplasmosis in the antenatal period, as indicated by maternal seroconversion rates varies from 0.2 to 1% [7].

14.2 *Toxoplasma gondii*: Life Cycle and Clinical Manifestations

Toxoplasma is an obligate intracellular protozoan existing in three forms.

Tachyzoite is the proliferative stage of the organism, present in a non-immune host in the bloodstream and lymphatics during which they travel to different tissues and nucleated cells. After invading the tissue and cells, the tachyzoite multiplies rapidly, causing lysis of the cells and

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thus releasing further tachyzoites in the neighboring cells, leading to necrosis of the tissue. This causes the manifestations of signs and symptoms of acute toxoplasmosis.

Bradyzoites are the cyst form (Tissue cysts) of the protozoa which are formed after the acute infection is over. These are the slowly multiplying forms present in the host cells particularly in the brain, eye, skeletal muscle, and cardiac muscle. These cysts may remain dormant for years or throughout life before getting reactivated in states of any immunosuppression. The cysts may contain thousands of bradyzoites.

Oocysts are resistant forms. They are found in the feces of infected cats. They are the main reservoir of this organism in nature and are the cause for the spread of infection in environment [8, 9].

14.2.1 Life Cycle

Life cycle of the parasite includes a definitive host in which the sexual multiplication occurs and one intermediate host consisting of the asexual cycle. Felines represent the only definitive host and rest all animals are the intermediate host. Human beings represent the dead-end hosts, i.e., they are incapable of transmitting the infection to other animals or humans.

The cats become infected by ingesting the tissue cysts or oocysts excreted from birds or mice. These ingested cysts are released into the environment, multiply with sexual reproduction, and the unsporulated cysts are released in feces in millions. The resistant oocysts sporulate in the soil and can remain there for more than a year. Vectors like earthworms transport the oocyst from the deposit site to raw eatables.

The risk factors of acquiring the infection include accidental ingestion of these oocysts from undercooked raw meat, pork, lamb, and beef containing tissue cysts [8], or unwashed fruits and vegetables containing these cysts on their surface. Children might get infected while playing with cats or playing with dirt containing oocysts via hands [10]. Blood transfusion, organ transplantation, or inhalation of sporulated cysts are some of the rare modes of transmission. Latest evidence also suggests water as the source of transmission [11]. The proliferative forms invade the bloodstream via intestine and spread throughout body via blood vessels and lymph fluid in different organs.

14.2.2 Clinical Manifestations

Most infections are asymptomatic. After an incubation period of about 10–23 days, flu-like symptoms may appear including headache, malaise, myalgia with fever and chills. Posterior cervical lymph node enlargement may be evident including other lymph nodes. Some features mimic infectious mononucleosis-like presence of atypical lymphocytosis on peripheral blood pictures. In severe cases, usually, in immunocompromised states, acute disseminated toxoplasmosis may occur causing encephalitis, myocarditis, hepatitis, pneumonitis, and ocular toxoplasmosis. Ocular toxoplasmosis usually involves the posterior pole of the eye manifesting with cystoid macular edema, band keratopathy, chronic iridocyclitis, cataract formation, secondary glaucoma, and retinal detachment [12]. These clinical manifestations can also occur after reactivation of the latent phase of the infection acquired earlier in life in immunocompromised conditions like AIDS.

14.3 Toxoplasmosis in Pregnancy and Congenital Toxoplasmosis

Infection during pregnancy is not more severe than in non-pregnant women and similarly, only a few cases are symptomatic [8]. Congenital toxoplasmosis is essentially the only fear of maternal toxoplasmosis which spreads by the trans-placental route [13]. There is no evidence that toxoplasmosis can transmit through breastfeeding [14]. The vertical transmission occurs in about 40% of pregnancies if the mother is infected for the first time during the pregnancy out of which 90% are asymptomatic mothers; this is by the crossing of placenta by tachyzoites via

blood [15, 16]. Mothers who have been infected before conception rarely transmit the infection to the fetus, by reactivation of the disease in the states of immunosuppression [5, 17]. There have been rare instances where an immunocompetent mother who acquired infection before conception has been found to cause congenital toxoplasmosis [18].

The prevalence of congenital toxoplasmosis varies from 0.1 to 0.3 per 1000 live births. The overall risk of transmission is 30%. Children affected with congenital toxoplasmosis are mostly normal developmentally [19]. As gestational age advances, the risk increases, i.e., at 13 weeks less than 15%, at 26 weeks 44% risks, and at 36 weeks almost 71% [20] (Fig. 14.1).

The congenitally infected newborns, although being asymptomatic, develop adverse sequelae by second or third decade of their life in almost all the cases [22]. But only 4% of infants experience permanent neurological damage, bilateral visual impairment, or die [23]. Congenital toxoplasmosis presents most commonly with neurological and ocular changes. Multifocal and diffuse parenchymal necrosis occurs which later transforms into calcifications and microglial nodules. Obstruction in the Foramen of Monro and aqueduct of Sylvius via the sloughed off necrotic tissues may lead to hydrocephalus. Later on, atrophy of brain tissues and thereon microcephaly occurs [20]. Ocular manifestations of congenital toxoplasmosis are due to choroidoretinitis, the most frequent ocular pathology, due to inflammation and necrosis of choroid and retinal tissues. This occurs due to the rupture of the cyst in the ocular tissues. One risk factor for choroidoretinitis is the presence of cerebral calcifications [21].

Although the transmission of infection is more with the increase in the period of gestation, the severity of affection to the fetus is less with the advancement in gestational age. Infections that take place in third trimester usually are subclinical in the early neonatal period [21]. Early gestational infections within 8 weeks lead to termination of pregnancy rather than fetal affection, but later on usually results in severe disseminated fetal infection, which can also lead to fetal demise in utero.

A study done in Europe found that the brain lesions were 20% after maternal infection at 10 weeks of gestation but 15% at 15 weeks of gestation and even lesser when infection was expected to be transmitted in third trimester [21]. Eye lesions were still high at more than 15% in late third trimester (Fig. 14.2).

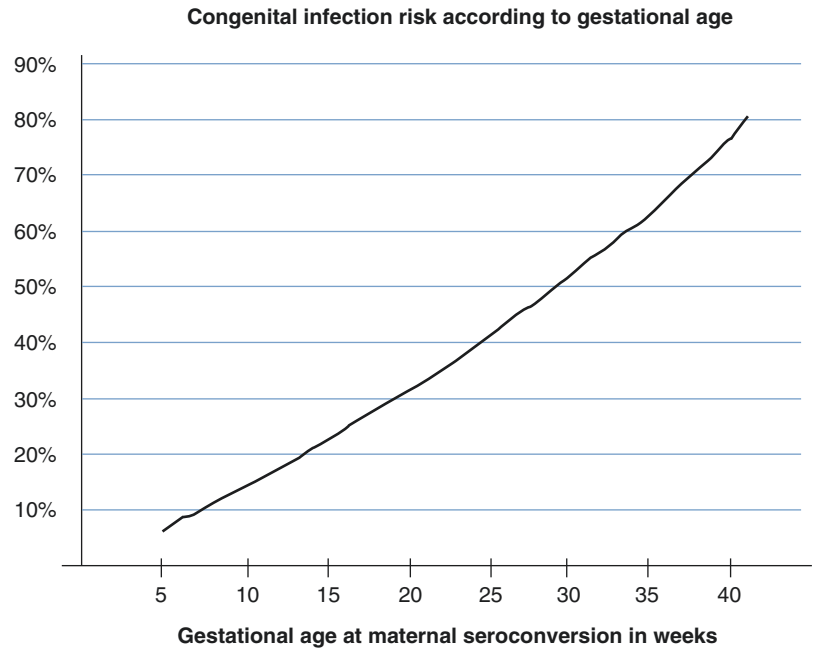

Fig. 14.1 Risk of congenital infection with an increase in gestation. Adapted from SYROCOT [21]

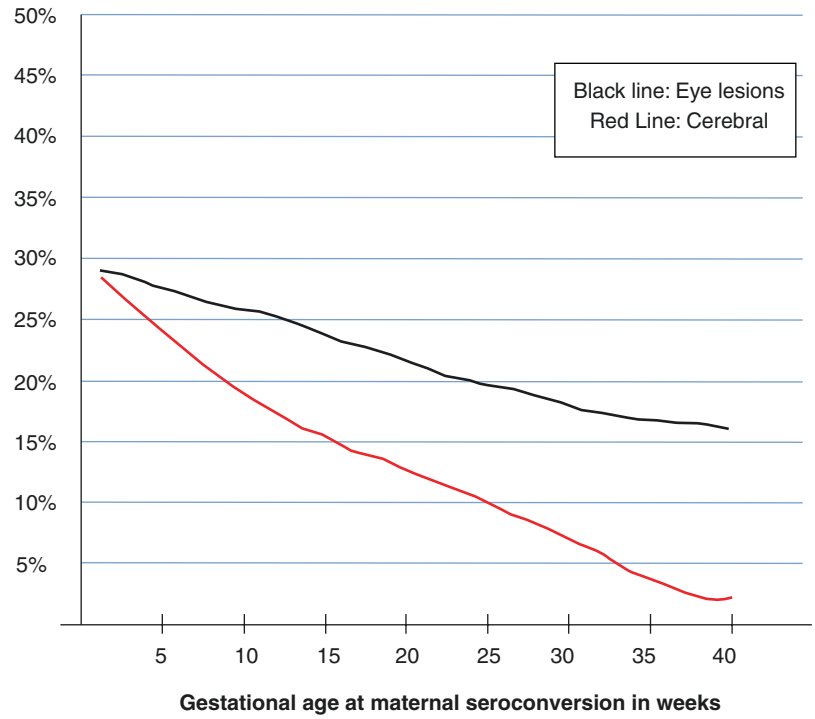

Fig. 14.2 Risk of cerebral and eye lesions with gestational age. Adapted from SYROCOT [21]

Most common features of congenital toxoplasmosis (CT) are hydrocephalus, chorioretinitis, and intracranial calcifications. Other features of CT include encephalomyelitis, convulsions, mental retardation, hepatomegaly, rash, anemia, erythroblastosis, strabismus, hearing and visual impairments, nystagmus and, growth and developmental delays [5]. Microphthalmia, psychomotor retardation, hypotonia, microcephaly, prematurity, and dysmaturity can also be the presenting features. About 80% of children asymptomatic at birth may develop neurological manifestations if not treated [24]. Therefore, every infection acquired in pregnancy must have a prompt diagnosis and treatment.

Antenatally, two-thirds of the scans show absolutely normal features. The common features of intrauterine infection on ultrasound include the presence of intracranial calcification, microcephaly, hydrocephalus, symmetrical cerebral ventricular dilatation while ascites, hepatosplenomegaly, pericardial effusions, echogenic focus in bowel, or severe intrauterine growth restriction may also be seen sometimes.

14.4 Diagnosis and Management of Toxoplasmosis

As toxoplasma infection is asymptomatic, few countries (e.g., Austria and France) with high prevalence rates adopt for universal and repeat screening tests by serological assays for diagnosis of acute toxoplasma infection. Universal screening is also opted in women suffering from immunocompromised states like HIV AIDS, transplant patients on chronic immunosuppression, etc. The earlier the diagnosis, the earlier the treatment can be provided and lesser is the time available with the parasite to cause the tissue destruction thus improving the overall neonatal outcome. Screening tests are otherwise administered if toxoplasma infection is suspected, for example in cases of antenatal ultrasound features suggestive of the disease or previous pregnancy affected with toxoplasmosis (ACOG Practice Bulletin 2003), or mother showing signs and symptoms of an acute toxoplasma infection. The screening algorithm is shown in Fig. 14.3. Sometimes, the diagnosis is made postnatally when the newborn

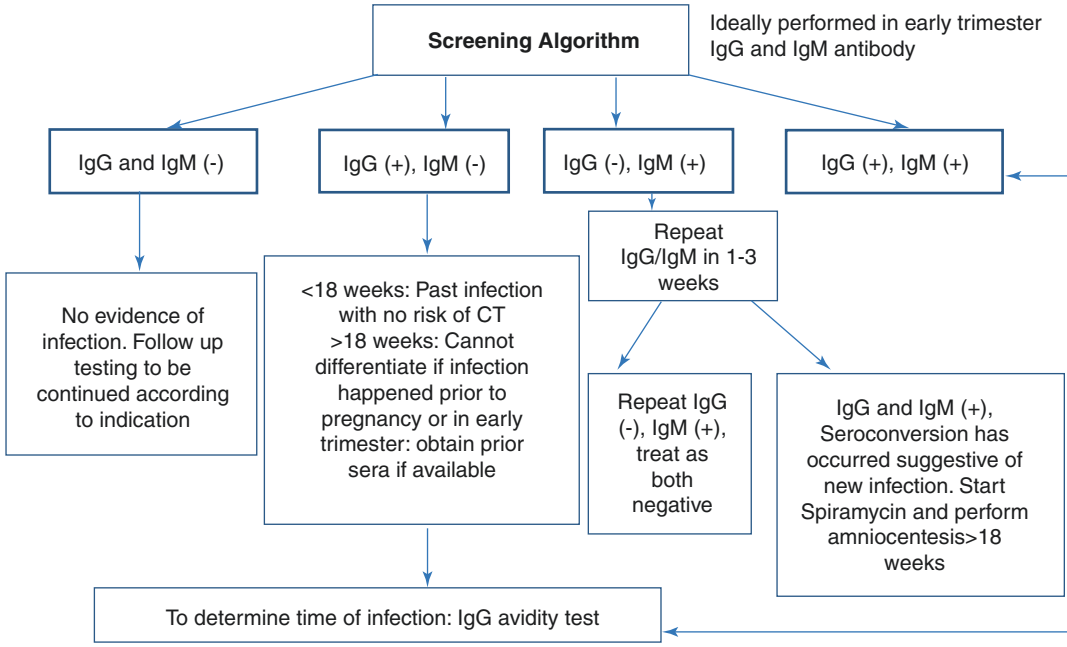

Fig. 14.3 Screening algorithm for toxoplasmosis infection in pregnancy

shows signs of congenital toxoplasmosis [9]. The testing is done in order to provide early prenatal treatment, thus preventing the infection to the fetus or reduce the severity of the infection.

Diagnosis of Maternal toxoplasmosis is based on the following methods:

- Tissue cultures from placenta, blood, or body fluids of mother– isolating *T. gondii*, but this method is rarely used as it requires a long time and high-grade laboratories.
- Tissue sections or body fluid smears- direct visualization of tachyzoites.
- Serological tests: Sabin Fieldman Dye test, Immunofluorescence antibody test, ELISA, Agglutination tests, IgG Avidity test [22].
- Polymerase chain Reaction in amniotic fluid sample (100% specific).

14.4.1 Diagnosis of Congenital Infection in Pregnant Woman

The serological test in mothers is done to diagnose that the infection exists, and to differentiate a recent infection from the old one. Congenital toxoplasmosis is caused by acute infection in mothers and hence differentiating acute from chronic is important.

Two antibodies, IgG and IgM specific for *T. gondii* are used in diagnosing infection. IgM antibody forms early after an acute infection from the 5th day and reaches a maximum level at around 1.5–2 months, after that it falls rapidly [8]. Whereas, IgG antibodies are formed after a week or 2, attain the highest level from 3 to 6 months, and are present thereafter. Although IgM antibodies fall after acute infection subsides, they may sometimes persist for years (15–18 months average) and both IgG and IgM may show false-positive results [25].

Interpretation of these antibodies is crucial in making a correct diagnosis and treatment of the infection. If both IgG and IgM antibodies are absent before or early in pregnancy, it is indicative of absence of previous infection [26]. If IgG antibodies are present with negative IgM antibodies, it suggests a chronic infection. If both IgG and IgM are positive, it can be suggestive of an acute infection, but the possibility of low titers of IgM antibodies from the previous infection cannot be ruled out. For a suspected recent infection,

perform a repeat test within 2–3 weeks, if similar results are there; perform further tests [8].

To help in the diagnosis of acute infection, new tests like IgG avidity test have come up [27]. It measures the strength of IgG antibody binding to *T. gondii*. If the infection is recent, the avidity of binding of IgG antibodies is low, and it takes about 5–6 months for the avidity to become high. Thus, patients with recent/acute infection will have a low avidity whereas patients who had acquired infection previously will show a high IgG avidity index. The sensitivity of IgG avidity can be up to 100% [28] (Table 14.1). Further management based on avidity testing is discussed in Fig. 14.4.

If IgG avidity test is suggestive of acute infection, further steps for prenatal diagnosis of congenital toxoplasmosis in the fetus include detection of *T. gondii* DNA by real-time polymerase chain reaction analysis of the amniotic fluid. It is done as low IgG avidity can persist for months [29]. The common target is the 35-multicopy B1 gene [14]. It is recommended to be performed after 18 weeks of gestation and after 4 weeks of an acute infection [20]. It is advantageous as it is associated with a low risk of fetal demise compared with previous tests like cordocentesis. Amniocentesis is performed only if there are ambiguous serological test results or ultrasound findings are suggestive of congenital toxoplasmosis [30]. *T. gondii* DNA PCR tests have also been tried on umbilical cord samples (cordocentesis) but it has a higher risk of intrauterine deaths and is less sensitive [31]. We can also extrapolate the severity of infection by the parasite load in amniotic fluid. A higher parasite load reflects high possibility of fetal severity and affection. A Japanese study compared the accuracy of the IgG avidity test and found that about 56% of women who had low IgG avidity had a positive PCR testing from an amniotic fluid sample [31]. Sensitivity of amniotic fluid PCR is highest if done at 17–21 weeks period of gestation. Negative results on PCR may still not rule out congenital infection. Ultrasound should be done monthly for women diagnosed with toxoplasmosis to look for fetal affection.

14.4.2 Diagnosis of Congenital Infection in the Neonate

Detection of IgM antibodies has the greatest importance for the diagnosis of congenital toxoplasmosis in neonate. IgM antibodies are large molecules and hence they do not cross the placenta and IgM antibodies are produced by the fetus. Hence, the presence of IgM antibodies

Table 14.1 Interpretation of serological testing for *Toxoplasma gondii* infection

IgG	IgM	Interpretation
Negative	Negative	No serological evidence whether acute or chronic infection but the pregnant woman is at risk of infection
Negative	Equivocal	Repeat testing after 2–3 weeks as IgM may have false-positive results, If repeat is same, treat as negative
Negative	Positive	Acute infection likely, confirm the infection as high false-positive rates and persistence of IgM antibodies
Equivocal	Equivocal/negative	Indeterminate, Obtain another sample
Equivocal	Positive	Repeat test after 2–3 weeks, if similar result, perform confirmatory test
Positive	Negative	Previous infection >6 months back, no intervention
Positive	Positive	Infection within 12–18 months, perform confirmatory test at higher laboratory

Confirmatory test : IgG avidity and amniocentesis
Adapted from Montoya and Liesenfeld [14]

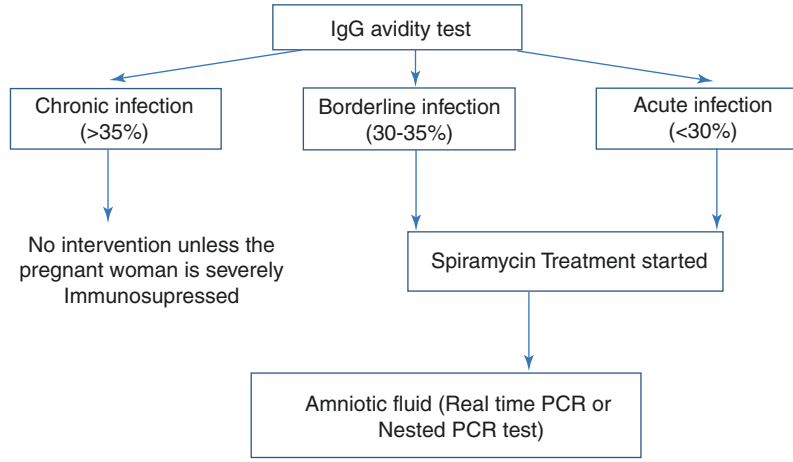

Fig. 14.4 Management of toxoplasmosis infection based on avidity testing

suggests infection in-utero. Similar serological methods are applied for the diagnosis as mother's IgA and IgG are also applied simultaneously. The presence of antibodies indicates infection but has no correlation with the severity [32]. Rarely, samples are taken from placenta or fetal tissues to visualize *T. gondii* cysts and ascertain the occurrence of vertical transmission via giemsa stain or immunoperoxidase staining [33].

14.5 Management of Toxoplasmosis

14.5.1 Treatment of Acute Infection Diagnosed in Mothers with No Fetal Affection

Maternal toxoplasmosis is rarely harmful to immunocompetent mothers, who may not require treatment at all, but the purpose of treating the infection is to prevent congenital toxoplasmosis. Spiramycin is a macrolide antibiotic used in the treatment when there is absence of fetal affection confirmed by a negative amniotic fluid PCR. It is given at a dose of 1 g every 8 h/day (1 g contains 3 million units) as soon as the maternal infection is confirmed and until delivery [14, 34, 35]. Earlier the treatment given, lesser are the chances of vertical transmission, and later the in-utero infection occurs, less severe are the clinical manifestations in the newborn. It is not used for the treatment once fetal infection is confirmed as it does not cross placenta. The incidence of infected infants is 50% less in each trimester if spiramycin treatment is used compared to the preceding years when there was no treatment [36]. Maternal ocular toxoplasmosis, if developed, requires an expert opinion whether antibiotics are needed or not as the course is unpredictable and may the condition may recover without any treatment as well. Most clinicians would still provide treatment for ocular toxoplasmosis, using a combination of Pyrimethamine, Sulfadoxine and systemic or intravitreal corticosteroids in varying regimens [12]. Alternative treatments are cotrimoxazole combination with corticosteroids, rarely usage of intravitreal clindamycin for severe cases [37]. Some cases may even require surgery if complications of ocular toxoplasmosis occurs.

Management of immunocompromised seropositive pregnant women is important as there are risks of disseminated toxoplasmosis, and reactivation of latent infection, both of which can have a dreadful effect on the mother and the baby (transmission of infection in early gestation). CD4 cell count is a useful method to start a prophylactic treatment for the mother. A woman having CD4 cell count of less than 200 cells/ml should receive cotrimoxazole (having a combination of 80 mg trimethoprim and 400 mg sulfamethoxazole in a single-strength tablet) once a day. In women having immunosuppression because of causes other than HIV or if CD4 cell count is more than or equal to 1200 cells/ml, spiramycin treatment is suggested for the duration of the pregnancy.

Although these strategies are not backed by many studies they are still applied by clinicians [14].

14.5.2 Antenatal Management If Fetal Affection Has Been Confirmed

Antenatally, treatment via antibiotics is given to limit fetal damage after transmission. The aim is to limit the damage as *T. gondii* cannot be eradicated. Pyrimethamine and sulfadiazine are used as a "gold standard" combination which is eight times more effective than either pyrimethamine or sulfadiazine used alone [38]. Other combinations of drugs have been studied in animal models that have shown anti-toxoplasma activity including Trimethoprim/Sulfamethoxazole and Clindamycin/ Sulfamethoxazole but are not clinically used [35].

If congenital infection in the fetus is confirmed by PCR testing after 18 weeks of period of gestation, treatment is done with Pyrimethamine: 50 mg twice a day for 2 days followed by 50 mg daily; sulfadiazine: 75 mg/kg initial dose, followed by 50 mg/kg twice a day (maximum, 4 g/day) along with folinic acid (leucovorin): 10–20 mg daily (throughout the treatment and 1 week after completion of pyrimethamine) [14]. Sulfadoxine has been recently used for its longer half-life and better compliance [39]. Pyrimethamine is not recommended in the first trimester due to increased risks of teratogenicity and bone marrow toxicity both to mother and the fetus and thus, spiramycin is given until a confirmatory amniocentesis report is available which is, later on, switched to Pyrimethamine [14]. Treatment is usually given for 4–6 weeks. No RCTs are there which can opine on the effectiveness of the treatment [40] (Fig. 14.5). But studies have shown that when treated with pyrimethamine and sulfadiazine, placental cultures had *T. gondii* only 50% of times whereas those who had not received any treatment in prior decades had 95% of times *T. gondii* in them [41]. There is evidence of decrease in the severity of disease such as meningitis and mortality in infants treated antenatally [42].

If the infection acquired is very early, use of Pyrimethamine is precluded. If the antenatal affection is severe with features like hydrocephalus or severe ventricular dilatation, then termination of pregnancy can also be offered after expert opinion and discussing with the pregnant woman; a confirmed diagnosis is mandatory from either amniotic fluid PCR or fetal blood sampling before the decision for termination is taken [43]. Mothers having toxoplasma chorioretinitis must also be treated as it is taken as a marker of acute infection [14].

14.5.3 Management of Congenital Toxoplasmosis

Diagnosis of congenital toxoplasmosis in neonates is also based on serological evidence, tissue culture, PCR and presence of signs and symptoms of disease at birth with history of maternal infection. If the maternal infection was proven to be acquired during pregnancy then the treatment is started whether or not the child shows any signs of infection. If the maternal infection was acquired but amniotic fluid PCR was negative or unavailable, then serological tests are done after 10 days of life. If diagnostic criteria are met, the treatment is started (Fig. 14.6).

There is a diagnostic dilemma if the child shows clinical signs of toxoplasmosis at birth but no testing was done during the antenatal period. In this case, simultaneous testing of mother and newborn should be done with IgG and IgM. If maternal serology is positive and acute infection is diagnosed, treatment for congenital toxoplasmosis is started. Along with this, neonatal IgM, IgG, and IgA antibody testing is done after 10 days of birth. If diagnostic criteria are met, treatment is started. If serology in neonate is negative, then repeat testing after 1 month and 2 monthly thereafter is repeated till 12 months of age (Fig. 14.7).

In congenitally affected newborns, daily oral pyrimethamine (1 mg/kg) for 2 months followed by 0.5 mg/kg for 10 months and sulfadiazine (100 mg/kg) with folinic acid (and not folic acid) 10 mg three times weekly is given for 12 months [40].

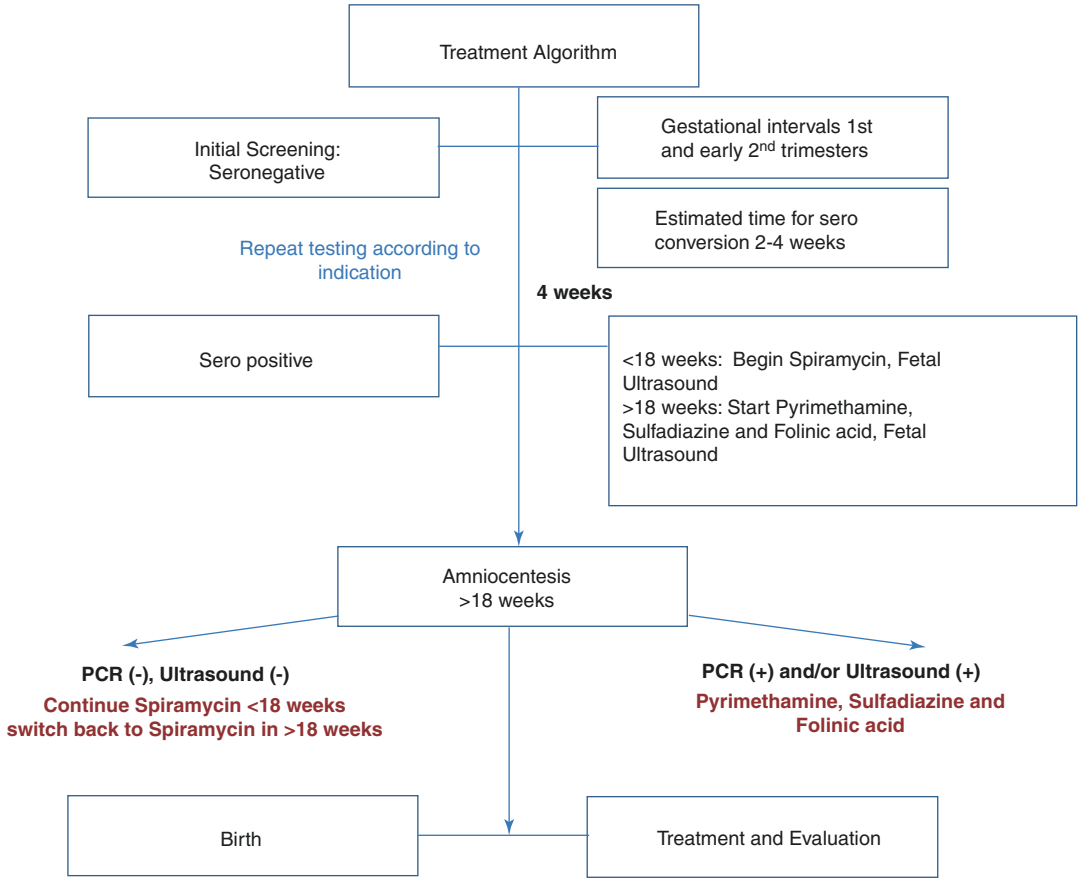

Fig. 14.5 Treatment algorithm. Adapted from Boyer et al. [43]

Monitoring of IgG levels is done to see the response of the treatment but the treatment should not be discontinued if IgG levels are not detectable before 12 months as they tend to fall. After completion of treatment, a follow-up examination is done 3 monthly for 1 year and 6 monthly during the 3rd year and yearly thereafter for life long. It should be combined with regular eye examinations. If eye signs of active infection are present after the completion of the therapy then a repeat treatment for 3 months can be given [20].

The management must consist of multidisciplinary involvement including otorhinolaryngologists, ophthalmologists, and neurologists. The need for long-term follow-up is essential. As hearing disabilities may not be evident at birth therefore all children born to mothers suspected of having toxoplasmosis must be screened for hearing loss. Corticosteroids are added if active cerebral involvement or chorioretinitis is present. Overall, studies suggest a good prognosis of children born live [45]. Even after immediate treatment, 10% of neonates may still develop severe disease.

14.6 Prevention from Toxoplasmosis

As toxoplasmosis is an asymptomatic infection and routine screening is not cost-effective in areas of low prevalence, all the women of reproductive age group and pregnant women must be made aware of methods of prevention from contracting toxoplasmosis. Following are the methods to decrease the risk of getting infected:

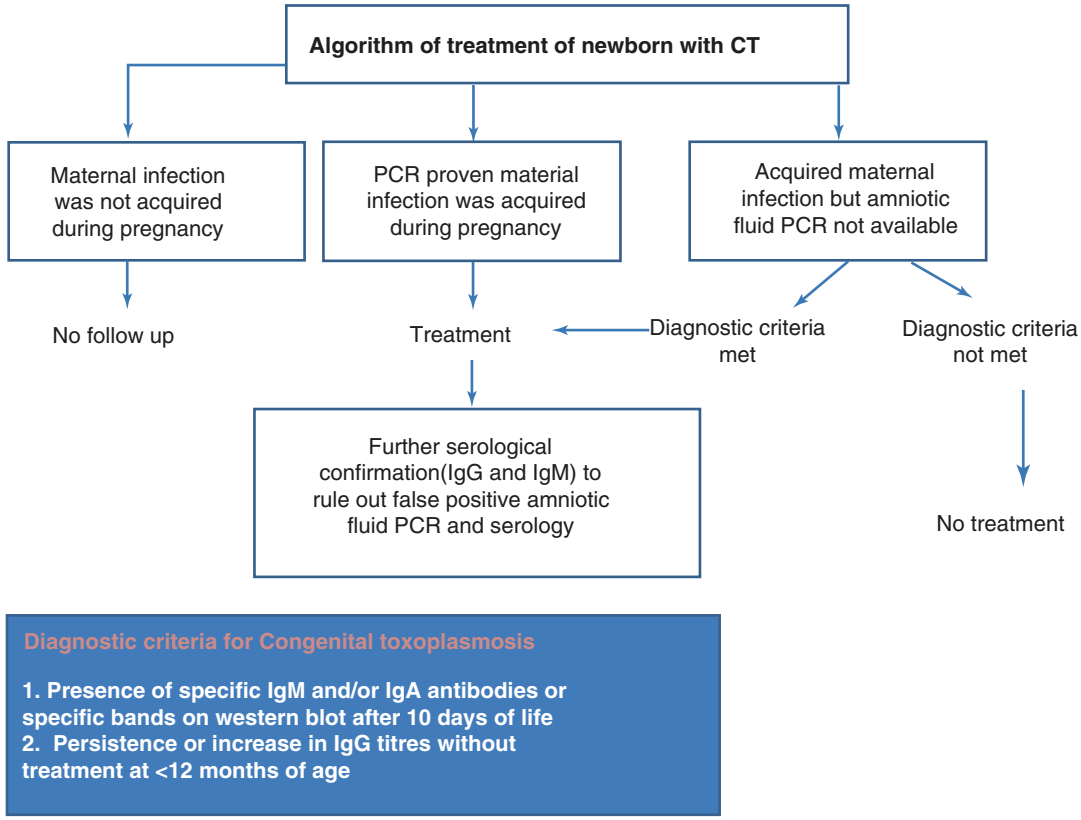

Fig. 14.6 Management of newborn with CT. Adapted from Pomares and Montoya [44]

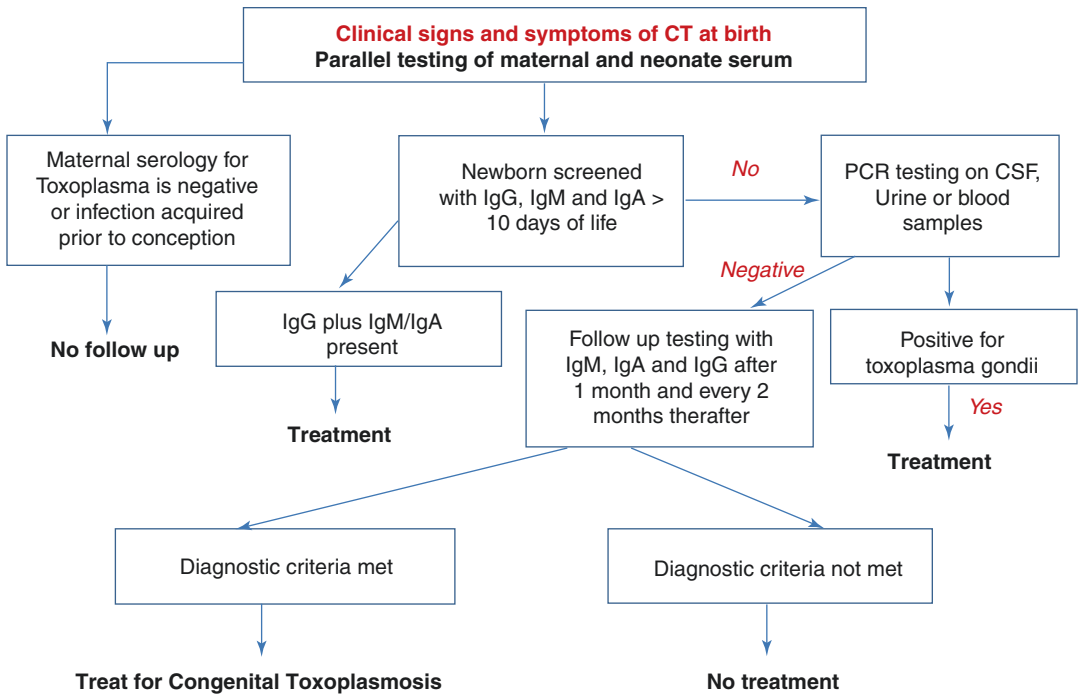

Fig. 14.7 Detection of congenital toxoplasmosis in the newborn. Adapted from Pomares and Montoya [44]

Primary prevention
1. Consumption of well-cooked meat heated to temperatures of 165–170 °F [16]. Freezing to at least 20 °C for 24 h and thawing also kills *T. gondii* cysts [13]
2. Always wash fruits and vegetables before consumption
3. Avoid contact with items that are potentially contaminated with cat faeces, prevent children from playing near sand dust and cats, avoid playing with stray cats
4. Wash hands thoroughly after handling raw meat or vegetables soiled by earth, after gardening, changing cat litter (wearing gloves, hand washing with soap and water)
5. Thoroughly wash all utensils that are in contact with undercooked meat
6. Health education to be provided to all the pregnant women to prevent toxoplasma infection [43]

Secondary prevention
1. Universal screening programme for early detection of antibodies specific to *T. gondii* in pregnant women, starting early treatment and preventing fetal affection
2. Universal screening on pregnant mothers can only be applied in states of high prevalence keeping in mind the cost-effectiveness ratios
3. Neonatal universal screening programme (detection of toxoplasma-specific IgM antibodies): identification of 70 and 80% of cases of congenital toxoplasmosis [46]
4. All tests require confirmation before starting treatments as false-positive rates are high

14.7 Conclusion

Toxoplasma infection in a pregnant woman may results in congenital toxoplasmosis (CT) of the neonate; worldwide, 400–4000 children are born with congenital toxoplasmosis every year. 90% of the infected woman are asymptomatic but the rate of vertical transmission is 40% which increases with advancing gestational age. Most infected newborns develop adverse neurological and ocular sequelae by second or third decade of their life. Most common features of congenital toxoplasmosis include hydrocephalus, chorioretinitis, and intracranial calcifications. Spiramycin, a macrolide antibiotic is used in the absence of fetal affection to prevent vertical transmission, while combination of Pyrimethamine and sulfadiazine is the "gold standard" used in the presence of fetal affection. Prevention of maternal toxoplasma by health education and routine screening of women living in areas with high prevalence is of paramount importance.

Key Points

- The risk of congenital sequelae and complications in fetus infected in early pregnancy is 85%.
- Diagnosis in mother is by serological tests for specific antibodies.
- If maternal infection occurs, a detailed ultrasound examination to identify markers, with consideration to parasitological culture of amniotic fluid and molecular studies PCR on amniotic fluid should be done.
- Prevention by health education is of paramount importance. If a woman has acquired an infection, it is recommended to conceive after 6 months of interval [23].
- Prenatal education regarding methods of prevention of the disease in areas of high prevalence must be offered and mothers must be educated about the consequences of congenital toxoplasmosis.
- Antiparasitic therapy to prevent congenital transmission or limit fetal damage after transmission should be encouraged.
- Termination of pregnancy may be an option if early affection has been confirmed.
- As toxoplasmosis is an asymptomatic infection and routine screening is not cost-effective in areas of low prevalence, all the women of reproductive age group and pregnant women must be made aware of methods of prevention from contracting toxoplasmosis.

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