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From Ultrasound To MRI: Imaging Modalities in the Early Diagnosis of Congenital Cerebral Toxoplasmosis

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Abstract **Case Report**

Congenital toxoplasmosis is a parasitic infection caused by Toxoplasma gondii, a protozoan parasite that can be transmitted from an infected mother to her fetus during pregnancy. While it often goes unnoticed in many individuals due to mild or absent symptoms, when contracted in utero, it can have severe consequences, particularly affecting the neurological development of the newborn. Neurological congenital toxoplasmosis is one of the most serious outcomes of this infection and is associated with a range of complications that can have long-term effects on a child's development and quality of life. Therefore, it is essential to incorporate screening methods for all pregnant women as part of routine prenatal care. We report the case of new born presenting a cerebral toxoplasmosis. *Key words:* Newborn, toxoplasmosis, TORSH, calcifications, MRI.

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INTRODUCTION

Toxoplasma gondii (T. gondii) is a protozoan parasite that infects up to 30% of the global population, though its prevalence varies widely across different regions, ranging from 10% to 80%. Infection is typically acquired through the consumption of contaminated water and food, particularly undercooked meat, contact with domestic or wild feline feces, and transplacental transmission during pregnancy [12]. The risk of mother-to-child transmission depends on the term of pregnancy at the time of maternal infection; it is less than 5% in the first trimester but can reach 90% in the last days of pregnancy. However, fetal disease tends to be more severe if contamination occurs earlier in pregnancy. [3] In immunocompetent individuals, the infection is often asymptomatic and self-limited. [2]

Congenital toxoplasmosis is a systemic infection that can lead to asymptomatic intracranial calcifications or severe neurological complications. Symptoms may include hepatosplenomegaly, skin rashes, pneumonia, anemia, thrombocytopenia, chorioretinitis, developmental delays, and seizures. Skull radiographs may reveal characteristic periventricular and cortical calcifications, which, though suggestive of congenital toxoplasmosis, can also be seen in other conditions like cytomegalovirus infection, herpes simplex infection, or congenital rubella. CT scans are useful for assessing suspected congenital CNS toxoplasmosis, as they help evaluate features such as location, pattern, and size of calcifications, as well as ventricular size and cerebral atrophy [4-5]

The prognosis for infants with neurological congenital toxoplasmosis varies depending on the severity of the infection and the timely initiation of treatment. Early diagnosis and appropriate therapy can significantly improve outcomes, yet some children may still experience long-term neurological deficits, including developmental delays, cognitive impairments, and visual or hearing problems. [2]

CASE REPORT

We report the case of a male newborn, 4 days of life, premature at 33 weeks gestation, born from a poorly monitored pregnancy. Serologies performed at the 7th month of pregnancy revealed a maternal toxoplasmosis infection (IgM+ and IgG+) one week before delivery. The mother was immediately placed on Rovamycin for one week. No history of fenugreek use, drug or toxic substance use, maternal hypertension or gestational diabetes was noted. The patient was admitted on day 1 of life to the neonatal intensive care unit for fetal-placental anasarca diagnosed prenatally. [figure1]

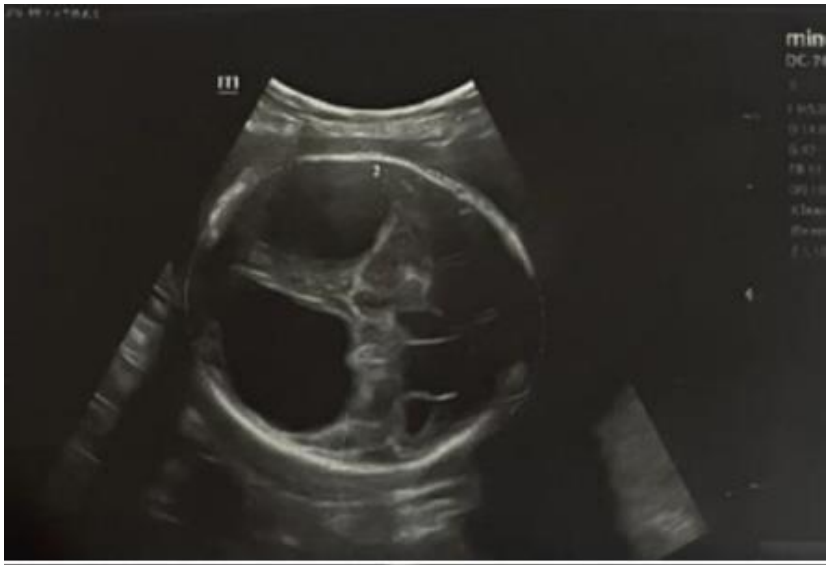

Figure1: Prenatal ultrasound showing major ventricular dilation

Clinically, the newborn appeared pink, reactive, moved spontaneously, and was stable hemodynamically and respiratorily. Neurologically, the anterior fontanelle was normally tense, with good axial and peripheral tone, a weak but present sucking reflex, and macrocephaly. [figure 2]

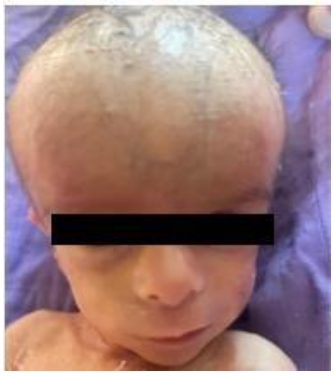

Figure 2: macrocephaly

Abdominal examination revealed abdominal distension with dullness on percussion. [figure3]

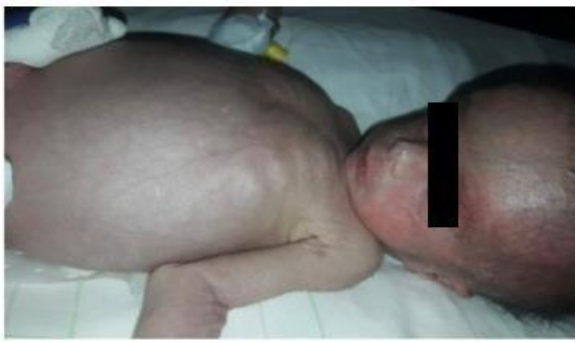

Figure 3: abdominal distention with macrocephaly

Biological tests showed leukocytosis with a white blood cell count of 17,900 and thrombocytopenia at 18,000 without bleeding signs. Toxoplasmosis serology was positive, while other TORCH serologies were negative. Additionally, the infant underwent an abdominal ultrasound revealing a moderate abdominal effusion, and a ventricular ultrasound (ETF) revealed significant triventricular dilation with echogenic material and mobile clots, along with multiple scattered cystic formations and an extraparenchymal collection in the left frontal region, which appeared hypoechoic and heterogeneous. It also showed hyper-echoic images corresponding to the basal ganglia, creating posterior

shadowing, possibly indicative of calcifications. [figure 4]

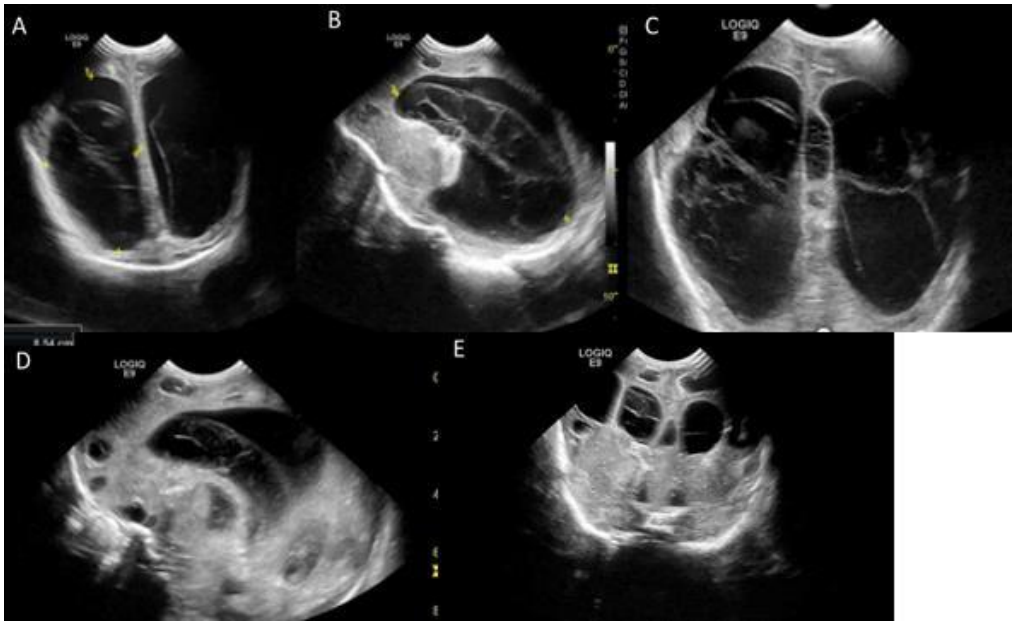

Figure 4: Postnatal Transfontanellar ultrasound in transverse and longitudinal sections showing major triventricular dilation with echogenic content associated with multiple septated intraparenchymal cystic formations of varying shapes and sizes

An ophthalmologic examination was requested for screening associated lesions, revealing microphthalmia, bilateral congenital cataracts, and iridocyclitis (iris rubiosis). Imaging was further supplemented by a CT scan of the brain revealing major hydrocephalus with signs of ventriculitis, associated with intra- and extra-axial collections featuring peripheral and central calcifications, suggestive of a chronic infectious process. [Figure 5]

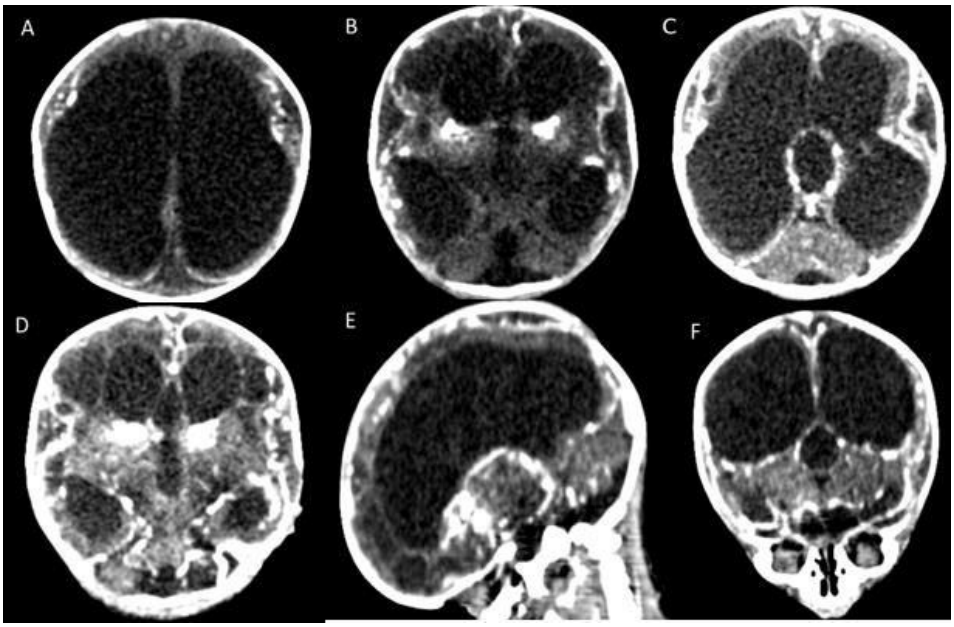

Figure 5: Brain CT scan in axial, coronal, and sagittal sections, without contrast agent injection (A, B) and after contrast agent injection (C, D, E, F): showing marked tetraventricular hydrocephalus with parenchymal compression, associated with subependymal parietal thickening and calcifications. Intra- and extra-axial hypodense collections are seen, showing peripheral enhancement and calcifications. Additional calcifications are noted in the basal ganglia, falx cerebri, and tentorium cerebelli, suggesting a chronic or congenital process

A brain MRI was performed as part of the diagnostic workup and demonstrated features suggestive of meningoencephalitis, consistent with the underlying disease process [Figure 6]

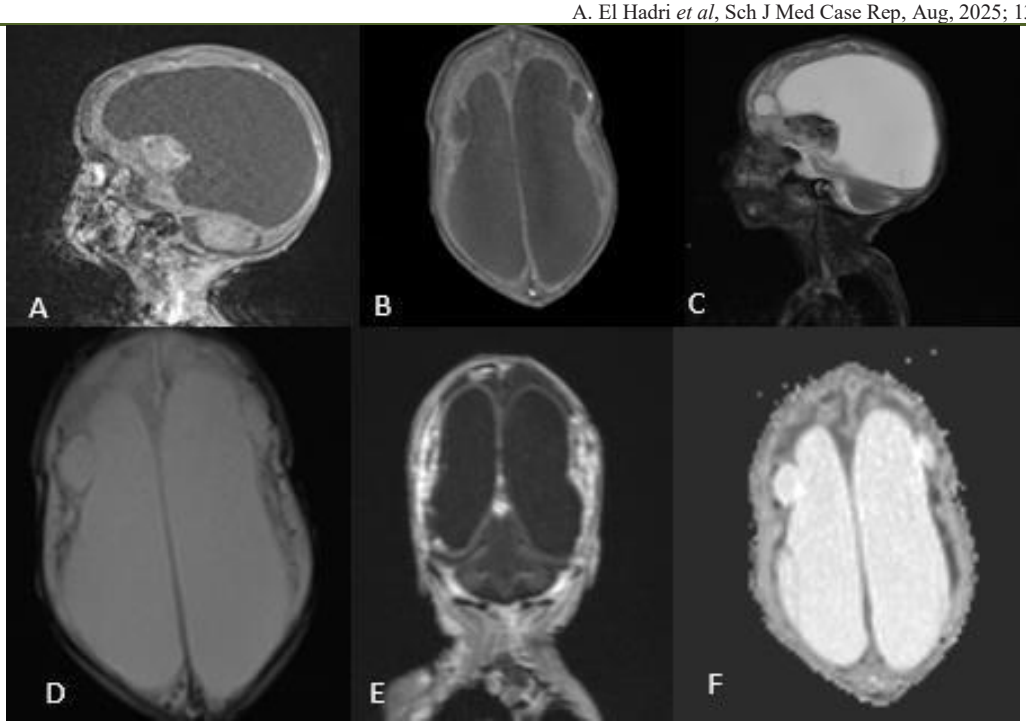

Figure 6: Brain MRI in T1 in sagittal (A) and axial (B) , T2 Sagittal (C) and axial (D), TOF coronal (E) and Diffusion (F): showing major dilatation of the lateral ventricles and moderate dilatation of V3 and V4, which appear septated, with thickening and enhancement of their walls. Persistent bilateral frontal periventricular fluid collections are noted, welldefined, hypointense on T1 and hyperintense on T2, showing peripheral enhancement after contrast administration. This is associated with pronounced pachymeningeal and leptomeningeal contrast enhancement

The patient was placed on trimethoprim-sulfamethoxazole (Cotrimoxazole).

DISCUSSION

Congenital toxoplasmosis (CT) results from transplacental infection by Toxoplasma gondii during pregnancy. The risk of fetal involvement increases when maternal seroconversion occurs late in pregnancy. However, the severity of fetal disease decreases in this case. Therefore, early maternal serological monitoring is essential. Once maternal infection is confirmed, prenatal diagnosis via Polymerase Chain Reaction (PCR) testing on amniotic fluid is recommended. Antenatal ultrasound often does not reveal abnormalities in about two-thirds of fetuses. The most common and specific lesions are symmetrical cerebral ventricular dilatation (which typically has an unfavorable prognosis) and cerebral calcifications. Less specific signs, such as hepatomegaly, ascites, pericardial effusion, hyperechogenic fetal bowel, and increased placental thickness, may also be present. Once the fetal infection is confirmed, the mother's treatment should be switched from spiramycin to a combination of pyrimethamine, sulfonamide, and folinic acid. In severe cases, pregnancy termination may be considered. [2-6-7] A congenital infection can manifest in various clinical forms at birth with the severe form being easily identifiable due to its neurological symptoms. In contrast, the asymptomatic form, which occurs in about 90% of cases during the neonatal period, may later develop symptoms such as psychomotor delays, hearing loss, and foci of chorioretinitis. [2-8] In immunocompetent hosts, Toxoplasma gondii infection is typically asymptomatic and self-limited. However, in individuals with compromised immune systems and fetuses infected during pregnancy, the infection can lead to more severe systemic involvement. This is particularly evident in the central nervous system and eyes, where the immune response is insufficient due to the protective barriers in these areas, resulting in more pronounced and severe manifestations, however the classic triad of chorioretinitis, hydrocephalus, and intracranial calcifications is relatively uncommon [9].

The diagnosis of congenital cerebral toxoplasmosis is based on a combination of clinical findings, serological tests, imaging studies, and histopathological examination. In rare cases, the clinical signs of congenital toxoplasmosis may indicate severe fetal pathology, such as hepatomegaly, splenomegaly, lymphadenopathy, jaundice, and neurological abnormalities. More commonly, the symptoms are dissociated, presenting as isolated visual problems, evolving hydrocephalus, seizures, and psychomotor delay. In some cases, signs may be absent during the neonatal period.

[10] Neuropathological lesions typically include meningeal inflammation and areas of necrosis in both the meninges and brain, predominantly affecting the basal ganglia, adjacent white matter, and the region of the aqueduct of Sylvius. Histological examination reveals macrophages in the necrotic areas, accompanied by lymphocytes, epithelioid cells, and plasma cells.

Detecting the parasite, which appears only in its encysted form within the inflammatory nodules, is challenging. [6-10].

A positive biological diagnosis of congenital toxoplasmosis is based on the detection of specific IgM and/or IgA antibodies, with IgG being the only antibody that crosses the placenta. The sensitivity of serological tests varies depending on the timing of the infection, increasing from 40% in the first trimester to 70% in the third trimester. The specificity of these tests reaches 100% when performed on peripheral blood rather than cord blood. [6] Neuroimaging techniques, such as ultrasound, CT scans, and magnetic resonance imaging (MRI), typically reveal findings like intracranial calcifications, ventriculomegaly, and hydrocephalus. Studies comparing cranial CT, MRI, and ultrasound in infants with congenital toxoplasmosis generally conclude that CT is the most sensitive modality for detecting neuroradiological changes, especially calcifications and cortical atrophy with ventricular dilatation. [11]

The CT appearance of cerebral lesions in congenital toxoplasmosis correlates with the timing of maternal infection, which in turn influences the severity of neurological symptoms. Early infection, occurring before 20 weeks of pregnancy, is typically associated with severe neurological manifestations such as microcephaly, hydrocephalus, tetraplegia or diplegia, seizures, mental retardation, and blindness. On CT scan, this stage often reveals ventricular dilatation, porencephalic cysts, and extensive calcifications, particularly in the basal ganglia. Hydranencephaly and multicystic encephalomalacia may also be observed. Infection between 20 and 30 weeks of pregnancy can cause severe neurological disturbances similar to early infection, but in some cases, neurological signs may be absent. CT scans in this period typically show multiple periventricular calcifications and ventricular dilatation, though these may be absent in rare cases. Late infection, occurring after the 30th week of pregnancy, is less likely to result in severe neurological disease, though chorioretinitis remains common. On CT, small periventricular and intracerebral calcifications are still frequent, but ventricular dilatation is rare [10].

All patients with congenital toxoplasmosis should receive pharmacological treatment based on a combination of pyrimethamine, sulfadiazine, and folinic acid to target Toxoplasma gondii and reduce the risk of complications. Those with hydrocephalus have traditionally been treated with CSF shunting. However, some reports suggest that endoscopic third ventriculostomy can be effective in selected cases, with a success rate of at least 50%. Proper management of hydrocephalus is associated with improved intellectual outcomes. Haut du formulaire

Early intervention is critical to improving the chances of a favorable outcome and preventing longterm neurological and ocular damage. Regular monitoring and supportive care are essential components of managing the disease and ensuring the infant's optimal recovery and development [8]. Bas du formulaireBas du formulaire

The existence of severe cerebral lesions on CT scans and neurological disease, despite preventive maternal treatment, highlight the ineffectiveness of such treatment in providing real protection, neurological disturbances may be even more common. Prevention remains crucial for avoiding the severe consequences of congenital infection and protecting overall health. By following simple preventive measures, the risk of transmission can be minimized, contributing to healthier pregnancies and reducing the burden of this disease [7].

CONCLUSION

Congenital cerebral toxoplasmosis remains a significant cause of neurological impairment in newborns, with potential for severe, lifelong consequences. Early diagnosis and treatment are crucial to managing the disease and minimizing long-term impairments, such as developmental delays, seizures, and visual issues. Although the prognosis depends on the timing and severity of the infection, prompt initiation of appropriate antimicrobial therapy can significantly improve outcomes.

Preventative measures, including proper hygiene and prenatal screening, play a vital role in reducing the risk of congenital transmission. Continued efforts in research and clinical care are essential for improving both the prevention and management of this potentially devastating condition.

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