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Ultrasound Features of Fetal Toxoplasmosis : Diagnostic and Prognostic Contributions in Prenatal Management

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Abstract

Background: Congenital toxoplasmosis (CT) is a parasitic infection that can cause severe fetal damage, particularly when maternal infection occurs early in pregnancy. Although biological methods remain the cornerstone of prenatal diagnosis, ultrasound plays a key role in detecting suggestive lesions and guiding antenatal care decisions.

Methods: This retrospective and descriptive study included 10 pregnant women diagnosed with confirmed Toxoplasma gondii seroconversion between 2018 and 2022 at the Department of Gynecology and Obstetrics of Ibn El Jazzar Hospital, Kairouan. Data were collected in collaboration with the Neonatology Unit and included clinical, biological, and ultrasound findings, as well as neonatal outcomes.

Results: Among the 10 cases, amniocentesis was performed in 6 patients, yielding 3 positive and 3 negative PCR results for Toxoplasma DNA. Ultrasound surveillance revealed no major fetal lesions except one case of mild ventriculomegaly in a newborn with multiple congenital anomalies not attributed to toxoplasmosis. All infected infants received in utero and postnatal treatment and demonstrated normal psychomotor development over one year of follow-up.

Conclusion: Ultrasound remains an essential tool in the prenatal surveillance of maternal toxoplasma seroconversion, contributing both to the detection of potential fetal damage and to the avoidance of unnecessary terminations of pregnancy. When integrated with serological and molecular diagnostics, it strengthens clinical decision-making and supports effective, conservative management.

Keywords: Congenital toxoplasmosis; ultrasound; seroconversion; amniocentesis; prenatal diagnosis.

INTRODUCTION

Toxoplasma gondii is one of the most widespread parasites on Earth. This anthropozoonosis is caused by T. gondii, which exists in three forms: the vegetative form (tachyzoite), the cystic form, and oocysts [1]. Transmission typically occurs via contaminated food—through ingestion of tissue cysts in undercooked meat (from intermediate hosts) or oocysts excreted by felids (definitive hosts), usually domestic cats in France [1]. These oocysts are commonly found in the environment and can contaminate water and raw vegetables.

In immunocompetent individuals, infection is often asymptomatic and only detectable via seroconversion. The disease is generally mild, presenting in its lymphatic form with fatigue, but it can be severe in

immunocompromised patients or in fetuses in the event of congenital transmission. Diagnostic tools have significantly improved thanks to immunological and molecular biology techniques; however, therapeutic progress has been limited, with only a few effective medications available [2–3].

Congenital Toxoplasmosis Context

Maternal-fetal transmission occurs when a previously non-immune pregnant woman acquires a T. gondii infection. Tachyzoites, the rapidly replicating form of the parasite, may cross the placenta during maternal parasitemia, leading to fetal infection [1].

Rare cases of congenital toxoplasmosis following pre-conceptional contamination have been reported, especially in immunocompromised individuals [4].

Congenital toxoplasmosis (CT) can result in a spectrum of clinical manifestations, from fetal death and encephalomyelitis to hydrocephalus, visceral involvement, or isolated ocular lesions (e.g., chorioretinitis). The severity of fetal damage is strongly associated with the timing of maternal infection [2–6]. The earlier in pregnancy the maternal infection occurs, the higher the risk and severity of fetal damage. The likelihood of vertical transmission increases with gestational age: approximately 1% during the periconceptional period, 15% in the first trimester, 44% in the second trimester, and 71% by the third trimester [3].

Prenatal screening for maternal seroconversion and CT enables early management to reduce both the transmission rate and the risk of fetal complications [6].

In Tunisia, the seroprevalence of toxoplasmosis among women of reproductive age ranges between 39.3% and 66.9% [7]. Prevention strategies include hygienic and dietary measures, as well as early detection and treatment of primary maternal infections during pregnancy.

Antenatal diagnosis of CT relies on serological and molecular tests. Ultrasound, as a monitoring tool, can reveal suggestive signs of congenital toxoplasmosis such as cerebral ventricular dilatation, intracranial calcifications, hepatosplenomegaly, and fetal ascites.

Ultrasound plays a major role by providing objective data that guide decisions regarding pregnancy continuation, help avoid unnecessary terminations, and allow early treatment of infected fetuses using the pyrimethamine-sulfadiazine combination—a regimen whose efficacy has been demonstrated by Couvreur and colleagues [12].

The aim of our study is to assess the **contribution of ultrasound in cases of maternal toxoplasma seroconversion**, and to discuss its **prognostic value** in antenatal care.

MATERIALS AND METHODS

This was a **retrospective and descriptive study** involving 10 patients followed in the Department of Gynecology and Obstetrics of Kairouan, all of whom had a **confirmed toxoplasma seroconversion during pregnancy**, as established by serological testing.

1. Study Population

The study included patients who presented with toxoplasma seroconversion and whose pregnancy outcomes were documented over a **five-year period**, from 2018 to 2022. Complete medical records were available for 10 patients.

2. Inclusion Criteria

The diagnosis of maternal infection was confirmed by the detection of **specific anti-toxoplasmic IgM and IgG antibodies** in at least two consecutive serological tests. This had to be supported by the presence

of a previously negative serological test either before or during pregnancy and/or a **significant increase in IgG titers over 2–3 weeks**.

In uncertain cases, **IgG avidity testing** was used to help estimate the date of seroconversion. A high avidity index (>30%) was considered indicative of an infection older than 3–4 months.

3. Management of Maternal Infection

Following the confirmation of toxoplasma seroconversion, patients received **Spiramycin (Rovamycine®)** at a dose of 9 million IU/day.

Amniocentesis was proposed from **18 weeks of gestation**, with a minimum interval of four weeks after the estimated date of infection. The goal was to perform **PCR testing on amniotic fluid** for the detection of T. gondii DNA. The PCR technique has a sensitivity of 65–80% and specificity of 100% [6].

Prenatal management was adapted according to the PCR result:

- **Negative PCR**: Spiramycin was continued at 9 million IU/day until delivery. Monthly ultrasound monitoring was performed.
- **Positive PCR**: Treatment was changed to a combination of **Pyrimethamine (Malocide®) 50 mg/day** and **Sulfadiazine (Adiazine®) 3 g/day**, along with **Folinic Acid (Lederfoline®)**. Ultrasound surveillance was performed every two weeks.

4. Definition and Management of Congenital Toxoplasmosis

Prenatal diagnosis of CT was confirmed by **positive PCR on amniotic fluid**. Affected patients received **Sulfadiazine (3 g/day)** and **Pyrimethamine (50 mg/day)** until delivery, along with **Folinic Acid (50 mg/week)** and regular **hematological and renal monitoring**.

Postnatal management included **transfontanellar ultrasound (TFU)**, **fundoscopic examination**, and testing for **specific IgM and IgG antibodies** [17].

Follow-up was maintained every three months during the first year of life. The diagnosis of CT was excluded if antenatal tests were negative, there were no clinical signs at birth, and **maternal IgG antibodies disappeared within the first year**.

Infants with confirmed antenatal CT received **Sulfadiazine (100 mg/kg/day)** and **Pyrimethamine (3 mg/kg every 3 days)**, with **Folinic Acid (50 mg/week)** supplementation. Clinical and ophthalmological evaluations were conducted every 3 months until the **maternal IgG disappeared**.

5. Collection of Sociodemographic Data

A **data collection form** was completed for each mother. Information was extracted from the obstetric medical records and from neonatal follow-up records at the Kairouan Neonatology Department.

The following data were recorded:

- Name, age, gravidity, parity, delivery mode and term, number of live births, fetal losses (miscarriages or medical terminations)
- Place and number of prenatal consultations
- Serological status (immune, seroconversion, or active infection), gestational age at the time of seroconversion (and IgG avidity test if applicable)
- Ultrasound monitoring: number, timing, and results
- Amniocentesis: timing, delay from seroconversion, and PCR results
- Treatment initiated
- Neonatal outcomes: name, birth weight, length, head circumference, clinical findings at birth, serology, TFU, fundus exam, treatment, and medium-term follow-up

6. Data Analysis

A **descriptive analysis** of the population was performed. No statistical testing was conducted.

RESULTS

I. General characteristics of the study population

1. **Age**
   The age of the patients ranged from 21 to 37 years, with a mean age of 26 years.
2. **Parity**
   Most of the patients were primigravida and primiparous.
3. **Obstetric History**
   Only 2 patients had a history of spontaneous miscarriage during the first trimester, of unknown cause.
4. **Prenatal Consultations**
   The average number of prenatal visits was 4.
5. **Place of Pregnancy Follow-up**
   More than half of the patients were monitored in a public hospital. The remaining patients had private follow-ups and were referred to the hospital for delivery.
6. **Timing of Seroconversion**
   In 4 patients, serological testing prior to pregnancy or before 16 weeks of gestation confirmed seroconversion.
   IgG avidity testing was performed in 2 cases.
7. **Ultrasound Monitoring**
   Five patients underwent **monthly ultrasound monitoring** after toxoplasma seroconversion. One patient had **biweekly monitoring** following a **positive PCR result** from amniotic fluid.
8. **Amniocentesis**
   Amniocentesis was performed in **6 patients**. The remaining 4 patients declined the procedure. Among the amniocenteses performed, **3 were PCR-positive**, and **3 were negative**.

II. Neonatal Outcomes

All pregnancies were carried to term. Only **one newborn**, with **positive prenatal CT diagnosis**, received postnatal treatment with:

- **Sulfadiazine (100 mg/kg/day)**
- **Pyrimethamine (3 mg/kg every 3 days)**
- **Folinic Acid (50 mg/week)**

Case No.	Seroconversion Timing (GW)	Amniocentesis	Treatment	Newborn Serology	TFU	Fundoscopy
1	19	Yes / PCR –	Rovamycine	–	–	–
2	16	No	No	–	–	–
3	10	Yes / PCR +	Rovamycine	Maternal IgG, IgM–	Normal	Normal

Case No.	Seroconversion Timing (GW)	Amniocentesis	Treatment	Newborn Serology	TFU	Fundoscopy
4	Periconceptional	No	Rovamycine	Maternal IgG, IgM–	Normal	Normal
5	8	Yes / PCR +	Rovamycine then Pyrimethamine + Sulfadiazine + Folinic Acid	Maternal IgG, IgM–	Normal	Normal
6	16	Yes / PCR –	Rovamycine	Maternal IgG, IgM–	Normal	Normal
7	8	No	Rovamycine	–	–	–
8	20	Yes / PCR –	Rovamycine	Maternal IgG, IgM–	Normal	Normal
9	36	No	Rovamycine	Maternal IgG, IgM–	Normal	Normal
10	24	Yes / PCR +	Rovamycine then Pyrimethamine + Sulfadiazine + Folinic Acid	Maternal IgG, IgM–	Mild ventriculomegaly	Normal

Only 7 neonatal records were available for review. These children were all followed **every three months** until maternal IgG antibodies **disappeared**, on average after one year. All had **normal psychomotor development**.

All newborns underwent **transfontanellar ultrasound (TFU)** and **fundoscopic examination**, both of which were normal **except in one case**, where prenatal CT was confirmed. This child showed **mild ventricular dilation** on TFU, along with other malformations including **cleft palate**, **microretrognathia**, **unilateral pyelectasis**, and a **ventricular septal defect (VSD)**.

III. Antenatal Diagnosis of Congenital Toxoplasmosis

Among the 10 patients included in our study, we retained the following **three clinical observations** with **positive amniocentesis results** indicating congenital toxoplasmosis (CT).

CASE 1 (Patient No. 3)

Mrs. S.M., aged 21, primigravida and primiparous. The pregnancy was monitored through **four prenatal consultations**.

Seroconversion was documented at **10 weeks of gestation (WG)**, confirmed by two positive serologies indicating an evolving toxoplasmic infection. The patient was started on **Spiramycin (Rovamycine®)**.

An amniocentesis was performed at **18 WG**, with proper timing relative to the seroconversion. The PCR result was **positive**.
However, the patient was **non-compliant** and did not adhere to any specific anti-toxoplasmic treatment.
Ultrasound follow-up was **irregular**, but the scans that were performed showed **no anomalies**, including the last scan done at admission for delivery, which showed **no cerebral lesions**, such as ventricular dilatation or intracranial calcifications. Amniotic fluid was reduced, and the placenta appeared calcified, suggesting **placental aging** unrelated to CT.
She delivered at **41 WG**. The newborn was **eutrophic** (birth weight: 3300 g, height: 51 cm, head circumference: 36 cm), and the clinical examination was normal. Treatment was initiated with **Sulfadiazine (Adiazine® 100 mg/kg/day)**, **Pyrimethamine (Malocide® 3 mg/kg every 3 days)**, and **Folinic acid (50 mg/week)**.
Serology revealed **maternal IgG positivity with negative IgM**.
The infant underwent **clinical and ophthalmological follow-up** every three months. Fundoscopy remained normal. Transfontanellar ultrasound (TFU) was also normal. Follow-up was discontinued at **1 year of age** following **complete disappearance of maternal IgG antibodies**.

CASE 2 (Patient No. 5)
Mrs. R.M., aged 27, primigravida and primiparous. The pregnancy was monitored through **10 prenatal consultations** in both the **private sector and hospital setting**.
Seroconversion occurred at **8 WG**, and Spiramycin (Rovamycine®) was initiated. Amniocentesis performed at **24 WG** returned **positive**. Treatment was then switched to **Sulfadiazine, Pyrimethamine (Malocide®)**, and **Folinic acid (Lederfoline®)**.
Serial **ultrasound monitoring** showed no signs suggestive of CT. In particular, the last scan did **not show any hydrocephalus, intracranial calcifications, hepatosplenomegaly, or ascites**.
She delivered at **40 WG + 5 days**. The newborn was **eutrophic** (birth weight: 3450 g, height: 50 cm, head circumference: 36 cm), and clinical examination was normal. Treatment included **Adiazine®, Malocide®, and Folinic acid**.
Serology showed **maternal IgG antibodies** and **negative IgM**.
The child was monitored clinically and ophthalmologically every three months. TFU and fundoscopy remained normal throughout follow-up. The child had **normal psychomotor development**, and follow-up was discontinued at **1 year of age** after complete disappearance of maternal IgG.

CASE 3 (Patient No. 10)
Mrs. C.A., aged 23, second gravida. The pregnancy was monitored in a **public hospital**, with **eight prenatal consultations**. Seroconversion occurred at **24 WG**, and treatment with **Spiramycin (Rovamycine®)** was initiated.
Multiple ultrasound scans were performed during follow-up. At **27 WG + 6 days**, prior to amniocentesis, ultrasound revealed **moderate unilateral ventriculomegaly without intracranial calcifications**, associated with **polyhydramnios**.
The **amniocentesis was positive**. Treatment was escalated to **Adiazine®, Malocide®, and Lederfoline®**.
Fetal karyotyping was normal. **Fetal MRI** showed only a **unilateral left pyelocaliceal dilatation**.

Delivery occurred at **38 WG + 6 days**. The newborn was **eutrophic** (birth weight: 2800 g, height: 46 cm, head circumference: 35.5 cm). Clinical examination revealed a **cleft palate**, **microretrognathia**, **glossoptosis**, and a **systolic murmur**. The infant was admitted to the **Neonatology Unit** for transient respiratory distress with spontaneous recovery.

Treatment included **Adiazine®, Malocide®, and Folinic acid**. Serologies showed **maternal IgG** and **negative IgM**.

TFU revealed **mild dilation** of the lateral ventricles and trigone (11 mm), with slight wall thickening. There were **no other intra- or extra-axial abnormalities**. Abdominal-pelvic ultrasound was normal. **Echocardiography** identified a **restrictive perimembranous ventricular septal defect (VSD)**.

The infant underwent **biweekly clinical follow-ups** for the first three months, then **quarterly ophthalmological evaluations**. Fundoscopy remained normal, with **no signs of toxoplasmic chorioretinitis**.

Follow-up was discontinued at **1 year**, after complete disappearance of maternal IgG antibodies.

DISCUSSION

The aim of our study was to describe the value of ultrasound in the detection and monitoring of fetal abnormalities following toxoplasma seroconversion during pregnancy.

The presence of ultrasound findings is not always associated with a poor prognosis. In France, where serological screening is routinely performed, ultrasound serves as a prognostic evaluation tool for infected fetuses rather than as a screening tool for toxoplasmosis.

In the literature, the rate of fetal involvement following maternal seroconversion varies widely. Ultrasound signs are present in more than 65% of cases when fetal infection occurs during the first trimester, and in about 20% of cases when infection takes place during the second trimester. Among all ultrasound-detectable abnormalities in congenital toxoplasmosis, none are considered pathognomonic [1].

Camille Codaccioni et al. [2] reported ultrasound anomalies in 58% of 88 fetuses infected following first-trimester maternal infection. A Chinese meta-analysis [3] showed that 68% of fetal malformations were detected when infection occurred in the first trimester. Variations in these results may be explained by differences in study populations and advances in ultrasound imaging.

In our study, ultrasound monitoring did not detect any abnormalities suggestive of congenital toxoplasmosis, except in one case where mild ventricular dilatation was later ruled out by fetal MRI. This anomaly had been initially detected through transfontanellar ultrasound (TFU).

Differences in patient recruitment, care practices, and the handling of loss to follow-up—as well as random sampling variations—likely account for these inconsistent findings.

Nonetheless, obstetric ultrasound remains a fundamental tool for monitoring toxoplasma seroconversion during pregnancy. It enables the detection of suggestive abnormalities and contributes to antenatal prognosis.

I. Seroprevalence of Toxoplasmosis

The global seroprevalence of toxoplasmosis in pregnant women is estimated at approximately 35%, ranging from 20% to 75%, and appears to be declining [4].

In France, national data estimate that between 1,000 and 1,300 pregnant women contract toxoplasmosis annually, corresponding to an incidence of 2.1 to 2.5 per 1,000 seronegative women at the start of pregnancy [5]. Current seroprevalence in pregnant women in France is around 31%, similar to the global average [6].

In Tunisia, 66.9% of women of reproductive age have protective residual immunity to toxoplasmosis, reducing their risk of developing active infection [7]. An earlier study by Fayala H. [8] in Tunisia reported 8 cases of congenital toxoplasmosis among 58 amniocenteses following maternal seroconversion. Between 2004 and 2007, 11 confirmed cases of congenital toxoplasmosis were diagnosed at the Parasitology-Mycology Department of the Pasteur Institute of Tunis [9].

In our series, we identified 3 cases of congenital toxoplasmosis out of 6 amniocenteses performed following maternal seroconversion.

II. Antenatal Screening and Management of Congenital Toxoplasmosis

1. Antenatal Screening

Maternal-fetal transmission of toxoplasmosis occurs during parasitemia, often early in infection when the mother is asymptomatic. Passage from the placenta to the fetus can be delayed and unpredictable, as the placenta acts as a filter [1]. The risk of transmission increases with gestational age, rising from 1% in the periconceptional period to 15% in the first trimester, 44% in the second trimester, and 71% by the end of the third trimester [10].

Other studies suggest transmission rates range from 6% in the first trimester to as high as 60-81% in the third trimester [11].

Maternal primary infection before 16 weeks of gestation poses the highest risk for severe fetal involvement. Fetal transmission risk is estimated at 5% in treated mothers and 15% in untreated mothers [12]. After 16 weeks, the severity of fetal impact decreases as pregnancy progresses, but transmission risk increases: 20% between 16 and 20 weeks and over 90% at term [12].

In our study, 6 maternal seroconversions occurred before 16 WG, and IgG avidity testing was used in some cases to estimate the timing of infection. One case was identified as periconceptional.

This underscores the importance of **early serological testing** (before the end of the first trimester) and monthly follow-up for seronegative patients.

Serological follow-up during pregnancy is essential for detecting maternal seroconversion and evaluating fetal risk. This allows for early and appropriate therapeutic interventions, reducing the likelihood of transmission and improving neuro-ophthalmological outcomes. Importantly, subclinical congenital toxoplasmosis currently represents up to 75% of cases [1,2].

2. Diagnosis

Detection of Toxoplasma gondii DNA by **polymerase chain reaction (PCR)** has significantly improved diagnosis, with a sensitivity of 70–80% and specificity of 94–100% [13].

Amniocentesis is typically performed **at least 4 weeks** after the estimated infection date to minimize false-negative results. The recommended timing is **around 18 WG** [14].

3. Treatment

If PCR is **negative**, Spiramycin is continued until delivery. If **positive**, the combination of **pyrimethamine and sulfadiazine**, along with **oral folinic acid**, is indicated [1].

4. Management Approach

Ultrasound anomalies often appear **several weeks after infection**, so **monthly ultrasound** is recommended. **Quarterly scans** are sufficient in cases of preconceptional maternal infection.

A negative PCR result does **not exclude fetal infection**, so monthly ultrasound monitoring must continue. If new abnormalities are detected, **a second amniocentesis** may be warranted.

In confirmed fetal infection, **biweekly ultrasounds** are advised to detect potentially severe and delayed lesions [1].

The combination of maternal serology and imaging findings contributes to screening, diagnosis, and prognosis of fetal infection.

III. Role of Ultrasound in Monitoring Congenital Toxoplasmosis

No ultrasound finding is pathognomonic for congenital infections. However, certain signs should prompt investigation of potential fetal infections, particularly intrauterine growth restriction (IUGR), intracranial or intra-abdominal calcifications, hydrocephalus, anasarca, oligohydramnios, and hepatosplenomegaly [15].

In confirmed CT, ultrasound serves to both detect suggestive anomalies and assess their severity, which plays a key role in decisions regarding the pregnancy's outcome.

Ultrasound surveillance must be **iterative and monthly**, even in first-trimester infections, as lesions may present **very late** [15]. The severity of fetal involvement is greatest with **early maternal infection**, occurring in over 65% of cases in the first trimester and 20% in the second trimester [16].

T. gondii has a particular tropism for **neurons and myocytes**, hence the predominance of neurological lesions, particularly in the **brain and retina**, due to their shared embryological origin [1].

Common lesions include:

- **Neurological anomalies**
  - **Ventricular dilatation**: the most frequent finding [17], resulting from aqueductal obstruction due to periventricular inflammation and ependymal destruction. It typically begins in the posterior horns and may extend bilaterally. Severe cases can lead to full hydrocephalus.(fig1). 48% of cerebral findings correspond to ventricular dilatation [2] (Fig2).

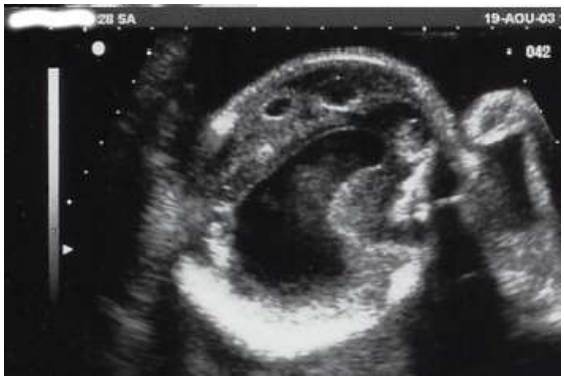

Figure 1 [11]: Fetal brain ultrasound at 28 weeks of gestation, transverse section.

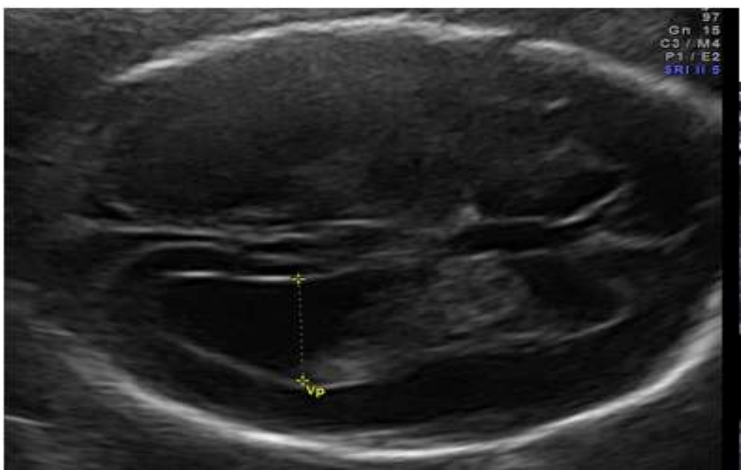

Figure 2 [11]: Ventricular dilatation.

- **Intracranial calcifications**: result from necrotic areas; typically periventricular, may be continuous or scattered [2].

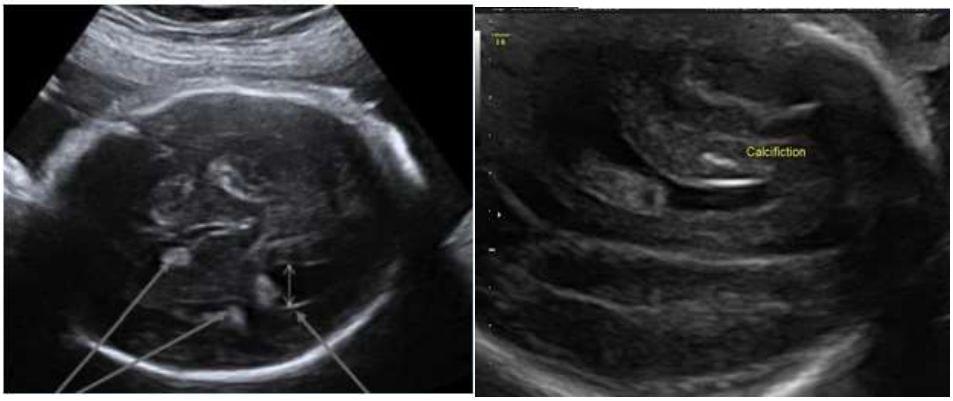

Figure 3 [2]**:Intracranial calcifications secondary to toxoplasmose infection**

- **Other cerebral anomalies**: such as porencephaly, multicystic encephalomalacia, and hydranencephaly [18].

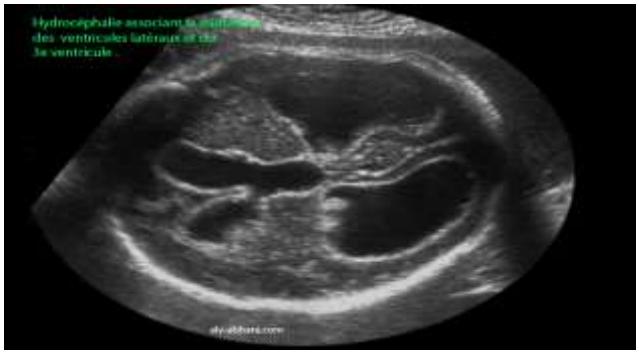

Figure No. 4 [2]: Hydrocephalus and Porencephaly Fetus at 30 weeks of gestational age

- **Ocular signs**: fetal microphthalmia and cataract [19].

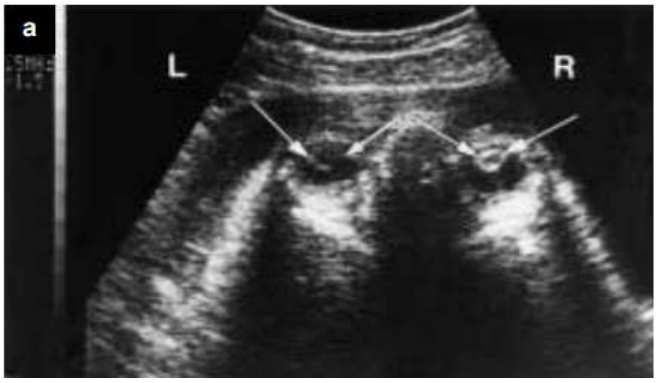

Figure No. 4 [2]: Hydrocephalus and Porencephaly Fetus at 30 weeks of gestational age

- **IUGR**: though rare, microcephaly may occur [20].
- **Abdominal signs**

- **Hepatosplenomegaly and parenchymal calcifications**: common anatomical findings, often unrecognized on prenatal imaging [2].

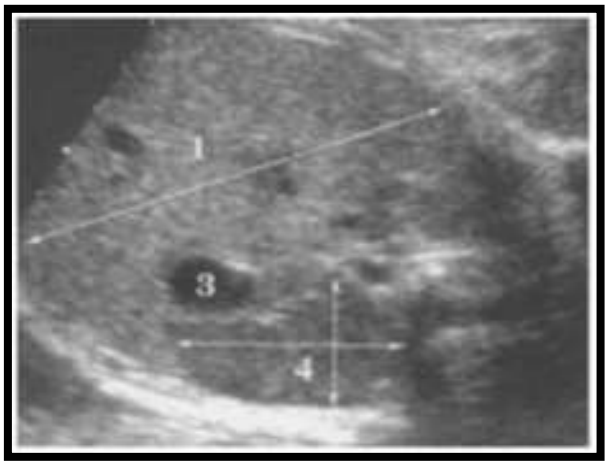

Figure No. 6 [2]: Hepatosplenomegaly

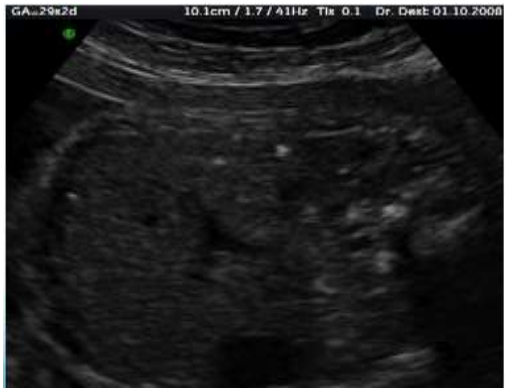

Figure No. 6 [2]: Hepatosplenomegaly

- **Hyperechogenic intestines**: typically transient and benign.

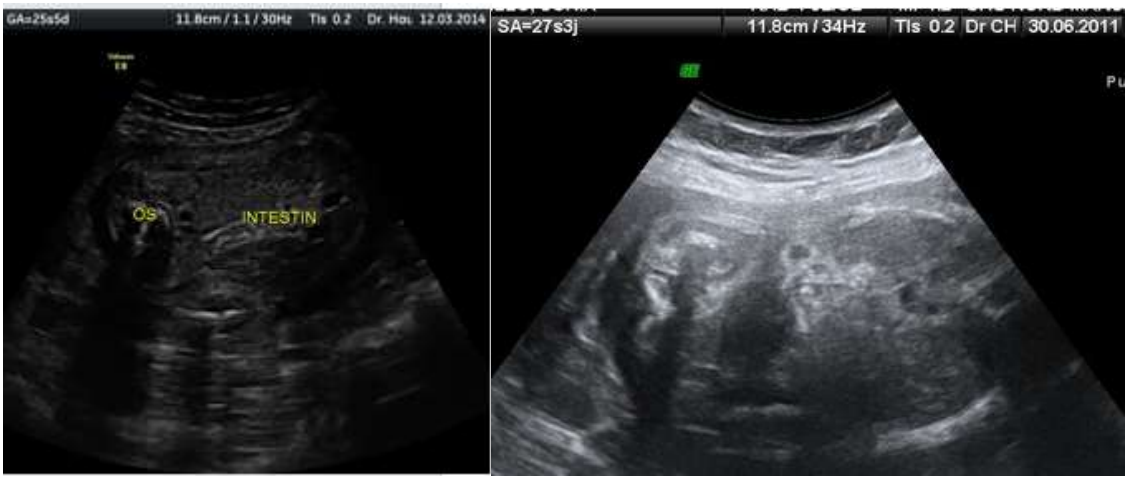

Figure No. 8 [2]: Intra-abdominal Calcifications

- **Serous effusions**
  - **Ascites**: due to polyserositis, visible around the liver, spleen, umbilical vein, and falciform ligament.

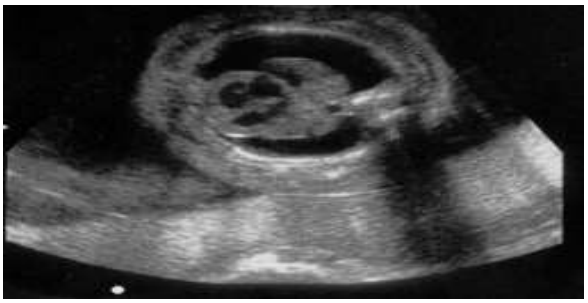

Figure No. 9 [2]: Fetal Anasarca

- **Pleural or pericardial effusion**: may indicate parasitic myocarditis and anasarca [2].
- **Placental anomalies**: include increased placental thickness or heterogeneous appearance with calcifications [11].

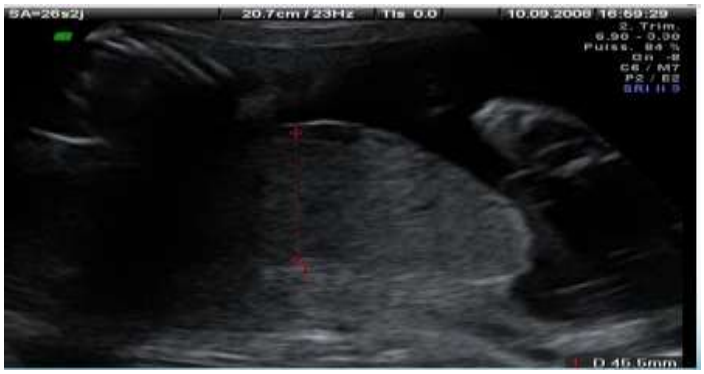

Figure No. 10 [2]: Placental Hypertrophy

A normal ultrasound does **not rule out evolving CT**, especially in cases of isolated chorioretinitis, which cannot be detected by ultrasound [19].

The severity of fetal infection—and decisions regarding medical termination—depend mainly on **ultrasound and, where necessary, fetal MRI**. MRI is valuable for confirming or detailing cerebral lesions, especially around **32 WG**.

IV. Neonatal Outcomes

At birth, clinical and neurological assessments are essential. TFU should be performed to detect any undiagnosed intracranial calcifications. If anomalies are found, **CT or MRI** may help refine the diagnosis. Fundoscopy is performed within the first month to detect retinal anomalies.

In the absence of infection markers, ophthalmic follow-up may be optional. However, in confirmed infections, fundus exams are repeated 2–3 times in the first year, then twice yearly in childhood, and annually through adolescence [23].

Cord blood serology is crucial for detecting IgG, IgM, and IgA. As IgM and IgA do not cross the placenta, their presence indicates congenital infection [22].

Serology is repeated at 1 month and then every 2–3 months to monitor maternal IgG decline. Persistence of IgG beyond 1 year confirms autonomous antibody production by the infant, indicating **late confirmation of congenital infection** [19].

In our series, after 1 year of follow-up, all 3 children with confirmed CT had **normal psychomotor development**, and **none showed signs of toxoplasmic chorioretinitis**.

V. Study Limitations

Several biases affect this study, including:

- **Selection bias**: Only patients from the Ibn Jazzar Kairouan maternity unit were included.
- **Small sample size**: Only 3 cases of confirmed congenital toxoplasmosis were identified.

This study may serve as a foundation for a **larger multicentric study** to improve maternal care and help prevent congenital toxoplasmosis more effectively.

Conclusion

Ultrasound plays a pivotal role in the antenatal management of maternal toxoplasma infection. Although it cannot definitively diagnose congenital toxoplasmosis, it provides critical insights into fetal condition and guides medical decisions, including the consideration of invasive procedures and potential pregnancy termination.

Our findings reaffirm that no ultrasound sign is pathognomonic, and a normal scan does not exclude fetal infection. Nonetheless, regular and targeted ultrasound surveillance remains indispensable for early identification of complications.

The integration of serological monitoring, molecular diagnostics, and fetal imaging enables early intervention and improves the prognosis of congenital toxoplasmosis.

Given the variability of fetal manifestations and the silent nature of most cases, early serological screening and patient compliance with follow-up are essential to optimize maternal-fetal outcomes.

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