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# Atypical Form of Ocular Toxoplasmosis

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## ABSTRACT

We present the case of a girl, aged 7 years, from rural area, admitted to our clinic with frontal and occipital headache and intermittent ocular pain, which started three weeks before admission; no pathologic antecedents.
When admitted, weight=51 kg, height=124 cm (BMI=36.16), no fever but with modified general state, excessive subcutaneous cellular tissue, normal cardio-pulmonary and digestive state, without meningeal symptoms. BP= 90/62 mmHg, HR= 80 b/min.; eye fundus examination at admission: both eyes - papillae with a faded, prominent contour/lineament, multiple hemorrhages with a peripapillary location and several soft exudates (RE > LE), maculae with preserved reflex; diagnosis: papillary edema. Head computed tomography, normal cerebral and cervical spine NMR, and normal aspect in hypophysis NMR; hemogram, renal and hepatic investigations, ionogram, glycaemia – normal values; the TORCH test pointed out increased values for IgM Toxoplasma 1.24 UI/ml (N= 0-0.8) and for IgG Toxoplasma 36.31 UI/ml (n=0-10).
We excluded: cerebral edema, malformations, hypophysis or optical chiasma tumors, Arnold-Chiari malformation.
We decided for the ocular toxoplasmosis diagnosis and the patient was sent to the infectious disease physician for an antiparasitic treatment. Before starting it, the patient no longer complained of headaches and the eye fundus exam was normal, most likely because of seroconversion, with a decrease of IgM and an increase of IgG Toxoplasmosis, as shown by the TORCH test.
The patient remained in our clinic's evidence, returning for regular ophthalmologic examination, and with a good evolution.

**Keywords:** toxoplasmosis, child, diagnosis, ocular pain, antiparasitic treatment

**Abbreviations:**
NMR – nuclear magnetic resonance
TORCH – Toxoplasmosis, Other agents, Rubella, Cytomegalovirus, Herpes simplex

## INTRODUCTION

Toxoplasmosis is a parasitic zoonosis, spread all over the world and caused by the protozoan parasite Toxoplasma gondii [1].

Specialists appreciate that approximately 50% of the world's population is already infected [1]. The prevalence of toxoplasmosis is in correlation with the environment and eating habits. Although very spread, Toxoplasmosis rarely causes human disease [2].

Toxoplasmosis represents the most common cause of posterior uveitis [3,4].

## CASE PRESENTATION

A seven-year-old girl, from rural area, was admitted on February 19, 2016, accusing headaches. The onset of the disease was approximately three weeks before admission, with frontal and occipital headache and intermittent ocular pain. She was ex-

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amined by a general practitioner and received treatment consisting of Ibalgin, Algocalmin, Midocalm, without showing any sign of improvement; she was admitted to the Pediatric Emergency Unit. In the morning, before coming to the hospital, she had a vomiting episode, without pain.

Heredocolateral antecedents: mother – 40 years, healthy, education – 3 classes; father – 44 years, healthy, education – 7 classes; 2 sisters of 20 and 18 years, healthy. No chronic disease within family.

Physiological personal antecedents: the third child in the family, coming from a monitored pregnancy, natural, full-term birth, weight at birth = 4600 g, Apgar score 8, naturally fed for the first three months, afterwards diversified, weaned at 2 years and 10 months; the prophylaxis of the carential rickets was incorrectly done; vaccination according to the ministry scheme; a pupil in the first grade.

Pathological personal antecedents: upper respiratory tract infections – ambulatory treated

Her 2-room house is in the rural area, where she lives together with other 4 persons. They get the water from a well and there are cats and dogs in the yard.

Patient's state when admitted: no fever, with modified general state, Weight= 51 Kg, Height= 124 cm, IMC= 36.16 (over the percentile 95), low appetite, with frontal and occipital headache, excessive adiposity, mainly at the lower level of the abdomen, basin, and thighs, developed mammary region, pulmonary stetacustic normal, rhythmic heart beats, HR= 72b/min., BP= 90/62 mmHg, SaO2=99%, slender, elastic, no pain abdomen, normal stool, normal urination, no sign of meningeal rash.

Findings:

Hb= 12.73 g/dl, L= 8740/mm$^3$, NS= 43%, Ly= 44%, M= 11%, E=1%, B=1%, T= 310,000 mm/$^3$, ESR=35 mm/1 h, PCR= 1.61 mg/dl, fibrinogen= 684.40 mg/dl, AST=17 U/l, ALT= 14 U/l, serum calcium = 9 mg/dl, cholesterol= 164 mg/dl, triglycerides= 109 mg/dl, serum ionogram: Na= 126 mEq/l, K=4.1 mEq/l, Cl= 107 mEq/l, sideremia= 47mg/dl, glycaemia= 80 mg/dl, urea= 23 mg/dl, uric acid= 4.37 mg/dl, creatinine= 0.53 mg/dl, negative uroculture, total proteins= 6.98 g%.

Pulmonary Rx. – no modification of pulmonary transparency.

Abdominal echography – normal aspect.

The eye fundus examination, which was rapidly performed when admitted, revealed: both eyes - papillae with faded lateral contour, being prominent, multiple hemorrhages, peripapillary disposed and several soft (RE>LE), maculae with preserved reflex.

The ophthalmologic examination revealed: visual acuity both eyes, BP both eyes=19 mmHg, normal ocular mobility, both eyes ophthalmometry value 16 mm, normal color. Diagnosis: both eyes bilateral

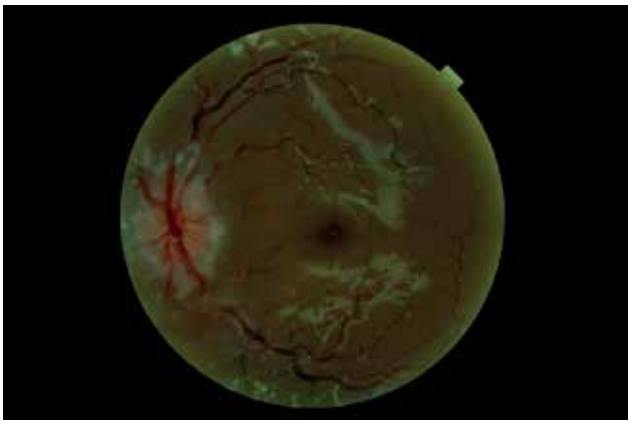

**FIGURE1.** Left eye - papillary edema

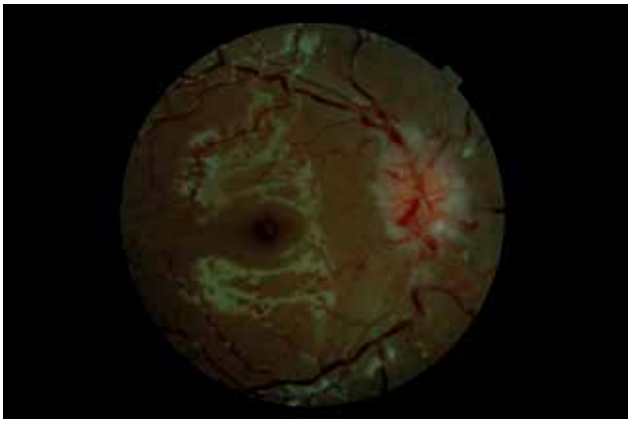

**FIGURE 2.** Right eye - papillary edema

papillary edema; a neurosurgical examination was recommended (Figure1, Figure2).

The emergency neurosurgical examination recommended a head CT with contrasting fluid or cerebral NMR.

The head CT with contrasting fluid performed the day she was admitted revealed a normal aspect.

The endocrinology examination recommended head NMR, TSH, prolactin, plasmatic cortisol, and thyroid echography: TSH=1.19 μUI/ml, prolactin= 344.9 μUI/ml, plasmatic cortisol= 436 mmol/l, normal aspect revealed by thyroid echography.

Since the patient still accused headaches, sometimes ocular pain, a new ophthalmologic examination was performed: both eyes prominent edematous optical papillae, with about 4D, multiple hemorrhages, peripapillary soft exudates. In left eye, retinal hemorrhage, higher in the papilla with a petechial aspect. Diagnosis – bilateral papillary edema.

A new neurosurgical examination was performed – in observation for the hypophysis tumour of optical chiasma, Arnold-Chiari malformation. Hypophysis and head NMR were recommended.

The NMR examination for hypophysis, head and spine did not reveal any pathologic aspects (Figure 3).

Although the cerebral, spine and hypophysis NMR examinations, as well as the head CT, did not

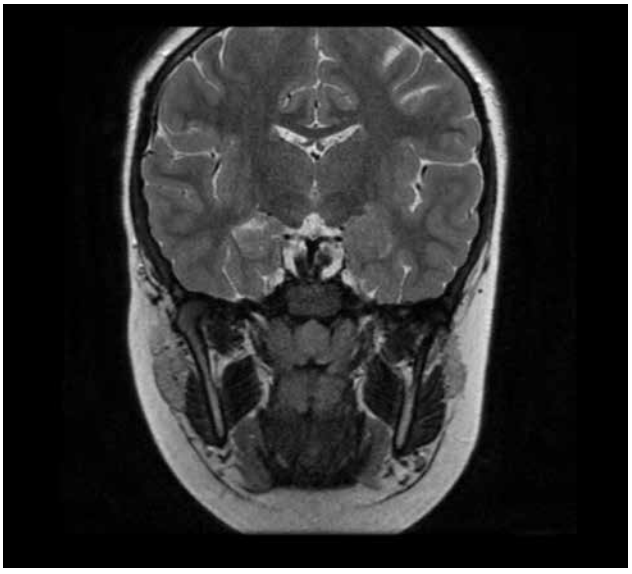

**FIGURE 3.** NMR examination

show modifications, the patient kept on complaining of splitting headaches; repeated eye fundus examinations emphasized the presence of bilateral papillary edema; we went on with the investigation requiring a neuro-pediatric examination, which recommended a TORCH profile, a test for the Lyme disease, for Toxocara, Methemoglobinemia, IDR PPD. There were normal values for the Lyme and Toxocara tests. Methemoglobinemia =0.6%. IDR 2UPPD 0 mm at 72 hours.

TORCH profile: IgG CMV= 162.7 UI/ml (0-0.5), IgM CMV= 0.24 (0-0.7 COI), IgG Herpes simplex tip1= 0.4 (0-0.6 COI), IgG Herpes simplex tip2= 0.06 (0-0.5 COI), IgG Rubella= 0.51 UI/ml (0-10), IgM Rubella= 0.20 (0-0.8 COI), IgG Toxoplasma= 36.31 UI/ml (0-1), IgM Toxoplasma= 1.24 (0-0.8 COI).

The day after she had the blood tests recommended by the neuro-pediatrician, the patient's general state improved and there were no more headaches; the eye fundus repeated examination was normal. The patient received only symptomatic treatment.

Since, after having the TORCH test, the values of the IgM and IgG for Toxoplasmosis gondii were increased, and taking into consideration the eye fundus aspect, we decided for the ocular toxoplasmosis diagnosis.

The patient was discharged, and she was recommended to go to a specialist in infectious diseases, to get an antiparasitic treatment.

The patient regularly returned to our clinic for control, and the ophthalmologic examination was normal and there were no more headaches.

## DISCUSSIONS

The life cycle of Toxoplasma gondii implies three forms [4]:

- Trophozoite: the invasive form which is responsible for the acute manifestations of the infection;
- Cyst: responsible for the latent and persistent infection;
- Oocyst: does not exist in humans, but only in cats (obligate host), with a role in the evolution cycle of Toxoplasma and in the infection transmission.

The parasite can infect many animals and birds, human beings too, but the complete evolution cycle occurs only in the family Felidae (the cat belongs to it), where the parasite reproduces and is eliminated through the faeces in the form of oocysts [4].

Humans get infected through [4,5]:

- Oocyst absorption; it is an indirect contamination and occurs following the ingestion of unwashed raw fruits and vegetables or the consumption of infected water, because of unwashed hands after gardening or tending to animals;
- Cyst ingestion – after eating smoked or insufficiently cooked meat;
- The transmission through tachyzoites, the frailest form of the parasite, which is destroyed within the environment and by gastric acid. The transmission of this form is possible transplacentally, being responsible for the congenital toxoplasmosis.

There were no cases of inter-human transmission, apart from the vertical transmission from the pregnant woman to her fetus. (Congenital toxoplasmosis).

A rarer means of infection transmission is represented by blood transfusions from people who are in the parasitemy level and by laboratory infections [4,5].

In the case of our patient, the cause of the infection was most probably the cat, but the environment she lives in must not be neglected.

Receptivity towards infection is general. There is insufficient information about the level and period of immunity [5]. It is a well-known fact that the specific antibodies IgG persist all life [1]. According to some authors, the infection persists in some cases, while in other cases immunity appears, the body being sterilized against the parasite [6]. The immunocompromised patients and the ones who underwent an organ transplant are in danger and they can develop serious infections, cerebral toxoplasmosis, and septicemia [6,7].

Toxoplasmosis is the most common cause of posterior uveitis [4]. 1 out of 4 posterior uveitis is estimated to be of toxoplasmosis etiology, 16 to 35% of the chorioretinitis represent a result of Toxoplasma gondii, reaching 43% in Western Africa [1]. The most common cases of chorioretinitis are the acquired ones [7].

The risk for this disease to reoccur, a year after the infection was acquired, varies between 0.3% and 3% [1].

For us, the etiologic diagnosis problems started when all cerebral imagistic examinations were normal, with headache persistence and modifications, pointed out by the eye fundus examination. It was also most surprising that, at a certain moment, the patient had no more headaches and the eye fundus examination was normal, the girl receiving only symptomatic treatment. An explanation for this development of the infection could be related to the fact that the TORCH test revealed lower values of the IgM, but higher values of IgG for toxoplasma, which could be interpreted as seroconversion from the acute to the chronic form.

Many adults have anti-toxoplasma IgG antibodies. Nevertheless, the simple presence of IgM antibodies shows a recent infection. Anti-toxoplasma IgM antibodies occur approximately two weeks after the moment of infection, developing a maximum concentration after four weeks, followed by a decrease. IgM antibodies can persist for more than one year [no more than 18 months) after the acute infection. The anti-toxoplasma IgG antibodies reach their maximum concentration 1-2 months after infection and they remain detectable for an indefinite period of time [1,5].

The ophthalmologic examination revealed bilateral papillary edema of the optical nerve; an area of vascular peripapillary turgescence was present but without typical lesions involving ocular toxoplasmosis and rare punctiform hemorrhages. There was no evidence of uveitis or chorioretinal lesions.

The ocular manifestations, in the present case, are atypical, rather suggesting an expansive intracranial process, for which all cerebral imagistic investigations were performed. Another feature is represented by the bilateral aspect of the papillary edema, since in toxoplasmosis, the typical lesions do not appear at a bilateral level.

- Recommendations to prevent toxoplasmosis gondii infection [8,9]:
- Washing hands;
- Washing the fruit and vegetables which are to be eaten;
- Avoiding the use of cow or goat raw milk;
- Cooking meat at high temperature;
- Using gloves and washing hands after changing the cat's litter box;
- No cat inside the house with a baby or a pregnant woman;
- Using gloves when gardening and washing hands afterwards.

## CONCLUSIONS

One reason which motivates the acquired toxoplasmosis, in the case of our patient, is represented by the lack of retinal foci and scars. Symptomatology was atypical, the patient denying the presence of miodesopsias and metamorphopsias. All these reasons showed that we were dealing with an atypical form of ocular toxoplasmosis.

**Conflict of interest**

The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

*Conflict of interest:* none declared
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