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Comunicações Curtas e Casos Clínicos

Retinal angiogenesis arrest in a full term infant with congenital toxoplasmosis

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RESUMO

Apresentamos um caso de toxoplasmose congénita, numa criança de termo, com área de retina avascular na vizinhança de lesões de retinocoroidite. Admitimos que um estímulo antiangiogénico *in utero* possa ter interrompido o desenvolvimento normal dos vasos retinianos.

Palavras-chave
Toxoplasmose congénita, retinocoroidite, angiogénese, neovascularização.

ABSTRACT

We report the finding of avascular retina in the vicinity of toxoplasmosis retinochoroiditis lesions in a full term infant. We postulate that an antiangiogenic stimulus in utero might have interrupted normal retinal vessel growth.

Keywords
Congenital toxoplasmosis, retinochoroiditis, angiogenesis, neovascularization.

INTRODUCTION

Congenital toxoplasmosis results from transplacental passage of the parasite *Toxoplasma gondii* from an infected mother to the foetus. These children are often assymptomatic at birth[1] but the majority of them will develop severe complications such as chorioretinitis, blindness, hydrocephalus, microcephaly, psychomotor or mental retardation, epilepsy or deafness months to years later[2].

CASE DESCRIPTION

A full term infant born from non-consanguinous parents was referred to our hospital with the diagnosis of severe congenital toxoplasmosis. Pre-natal care revealed maternal seroconversion for toxoplasmosis at 18 weeks gestational age (GA) (IgM > 40 IU/mL, IgG > 7000 IU/mL). Treatment was delayed, and spiramycin was started at 28 weeks GA. Fetal ultrasound revealed moderate bilateral ventriculomegalia and brain calcifications, confirmed by fetal brain-Magnetic Ressonance Imaging (MRI) at 30 weeks gestacional age.

Birth at 39 weeks GA from an uneventfull vaginal delivery, with birthweight of 2122g (percentile <5) and head circumference of 33 cm (percentile 10). Follow up was made in another center and anti-toxoplasma medication was not started at birth. The diagnosis of congenital toxoplasmosis was made at 10 months of age. He presented microcephaly (head circumference <5th percentile), inferior member hypertonia and left hemiparesis. Serology for toxoplasmosis IgG 240 IU/mL and negative IgM. Brain MRI showed multiple parenchymal nodular calcifications and mild ventricular dilation. No organomegalia on abdominal ultrasound. Specific toxoplasmosis treatment was started with pyrimethamine, sulfadiazine and folinic acid.

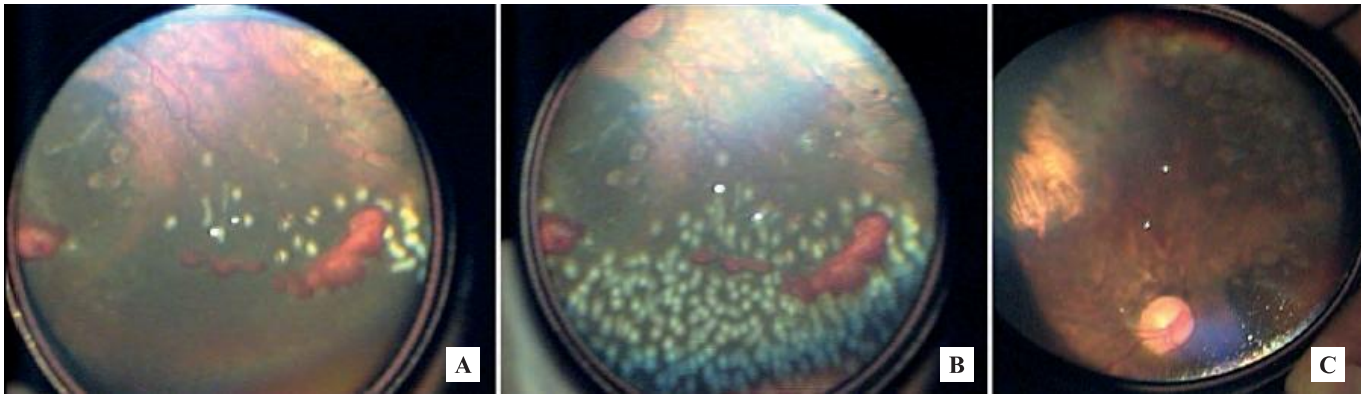

Fig. 1 | **A)** Tufts of active neovascularization beetween two chorioretinal scars in the transition vascular/avascular retina very similar to stage 1 retinopathy of prematurity. LASER was applied to verify areas of plane retinal detachment. **B)** Left eye immediately after LASER ablation of avascular retina. **C)** Chorioretinal scar and LASER spots 6 months after treatment.

Ophthalmological observation revealed a non fixating nystagmus. Biomicroscopy was unremarkable in both eyes. His left eye presented two superior chorioretinal scars. The peripheral superior retina near the scars was avascular and in the transition between vascular and avascular retina we found active neovascularization very similar to stage 1 retinopathy of prematurity (Figure 1A). All the other retinal quadrants presented normal vascularization until *pars plana*. His right eye presented a nasal retinochoroidal scar and vascularization was normal. Laser ablation of avascular retina in his left eye was performed (Figure 1B).

Two months later he kept a global psico-motor development delay but fixated and interacted. LASER scars were stable and there were no signs of disease activity (Figure 1C). Toxoplasmosis treatment was continued until one year of therapy was complete. At 4 years of age he presents no signs of systemic or ocular recurrence.

DISCUSSION

For many years, observation of solid tumor growth in both animals and humans has suggested that infection can cause tumours to shrink or even regress[3]. Different microorganisms have been shown to interfere in tumor growth[4] and specifically tumor angiogenesis inhibition by acute toxoplasmosis infection in mice has been demonstrated[5].

Retinal vascularization begins at the optic nerve at 16 weeks' gestation and proceeds centrifugally, reaching the edge of temporal retina at 40 weeks' gestation.

Retinochoroiditis is the most common ocular manifestation of congenital toxoplasmosis[6]. In this child, normal retinal vessel growth was interrupted in the vicinity of these lesions.

We postulate that an antiangiogenic stimulus in utero by toxoplasmosis acute infection might have interrupted normal retinal vessel growth. It is the first time, from our knowledge, that there is a report of disturbed retinal angiogenesis by an infectious local stimulus.

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