CASE REPORT

Choroidoretinal granuloma in a young female patient

Horace F Massa, Zisis Gatzoufas, Georgios Mangioris, Georgios D Panos

Department of Ophthalmology,
University Hospitals of Geneva,
Geneva, Switzerland

Correspondence to

Dr Georgios D Panos,
gdpanos@gmail.com

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SUMMARY

A 16-year-old Brazilian female patient presented with blurring of vision in the right eye. Corrected visual acuity was OD 2/20, OS 20/20. Afferent pupillary defect was absent and anterior segment examination revealed anterior uveitis. Fundus examination showed light vitritis and a raised grey-white granuloma located at posterior pole with focal serous retinal detachment on optical coherence. Indocyanine green angiography disclosed a complete mask effect in granuloma's area. Differential diagnoses were infectious (bacterial, viral, fungal and parasites) diseases, systemic inflammatory diseases, tumours. Blood serologies (HIV, toxoplasma, Borrelia, cytomegalovirus (CMV), herpes simplex virus (HSV), varicella-zoster virus (VZV), rubeola) showed positive results for IgM and IgG for toxoplasma, and anterior chamber tap (PCR for toxoplasma, CMV, HSV, VZV) revealed toxoplasma DNA. Anti-toxoplasma therapy, pyrimethamine, sulfadiazine and calcium folinate, was administered immediately. On follow-up granuloma regression was observed, with complete visual restoration. This case demonstrates a clinically challenging posterior pole granuloma.

BACKGROUND

Posterior pole chorioretinal granuloma can be the manifestation of various infectious, foreign body, systemic inflammatory diseases.^{1 2}

A thorough general and ocular history and physical examination are important for diagnosis and proper treatment.

In our report we present a young patient with a posterior pole granuloma caused by toxoplasmosis and discuss the other potential causes and new therapeutic trends.

CASE PRESENTATION

A 16-year-old Brazilian female patient presented with a history of gradual blurring of vision in the right eye for 10 days.

Her best-corrected visual acuity (BCVA) was 2/20 in the right eye (OD) and 20/20 in the left eye (OS). No relative afferent pupillary defect was present. Slit-lamp examination revealed right keratic precipitates but no cell and flare. Intraocular pressure was 18/12 mm Hg (OD/OS) with normal lens in both eyes. Fundus examination showed mild vitritis (grade 1+), vasculitis and a raised, grey-white lesion in the temporal posterior pole near macula typical of a granuloma with surrounding serous retinal detachment (figure 1). Optical coherence tomography (OCT) confirmed the presence of a retinal elevation (figure 2). Indocyanine green-

angiography disclosed complete early and late masking by the lesion evoking a full choroidal thickness granuloma. Fluorescein angiography showed leaking around the lesion into serous retinal detachment. Rapid progression of the disease suggested an infectious aetiology.

INVESTIGATIONS

Serological screening was positive for toxoplasmosis (IgM and IgG antibodies). Aqueous humour PCR was positive for toxoplasma DNA. HIV and tuberculosis (TB spot) were negative.

DIFFERENTIAL DIAGNOSIS

- ▶ Ocular toxoplasmosis
- ▶ Other infectious uveitides (candida, syphilis, tuberculosis, viral)
- ▶ Sarcoidosis
- ▶ Masquerade (lymphoma)

TREATMENT

Oral medication included pyrimethamine 25 mg twice a day, sulfadiazine 1 g four times a day, folinic acid 25 mg twice a day and methylprednisolone 80 mg tapered 10 mg/week. No pyrimethamine or sulfadiazine loading doses were administered.

OUTCOME AND FOLLOW-UP

The patient was seen in a week and then followed every 2 weeks; at 8 weeks right visual acuity was fully recovered (20/20) and the granuloma shrank into a fat pigmented scar.

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Figure 1 Colour photo of the retina. Toxoplasmic granuloma with surrounding serous retinal detachment.

Figure 2 Optical coherence tomography confirming retinal bulging surrounded by subretinal unreflective zone confirming the serous retinal detachment and macular oedema.

DISCUSSION

Chorioretinitis with chorioretinal granuloma is a clinical manifestation of a highly inflammatory process that can lead to retinal destruction and scarring.

Ocular examination in our patient suggested a panuveitis based on the corneal endothelial inflammatory precipitates and a choroidal granuloma of infectious aetiology. Bacterial infection by *Treponema pallidum*, *Bartonella henselae*,³ *Mycobacterium tuberculosis*⁴ and very rarely *Streptococcus pyogenes*⁵ or fungus such as *Sporothrix schenckii*⁶ and *Candida albicans*⁷ or parasite such as *Ascaris* especially in children⁸ may produce a granuloma.

Other aetiologies include systemic inflammatory diseases such as sarcoidosis⁹ or more rarely foreign body granuloma in intravenous drug users.¹⁰

Interestingly our patient had a grade 1 vitritis and no general symptoms; therefore, we ruled out an HIV-related immunodepression.

Viral infections do not cause granulomas but might look somewhat similar and be associated with a uveitis. A small choroidal tumour could look like a granuloma. Benign type 2 hamartoma¹¹ or malignant tumour (especially retinoblastoma during childhood) or metastatic tumour, one-third of which are the initial manifestation of malignancy (most commonly breast and lung cancer¹²), lymphoma¹³ and amelanotic melanoma could mimic a granuloma. The presence of uveitis and the hypofluorescence of the lesion were highly suggestive of a non-neoplastic lesion in this case. Uveal lymphoma¹⁴ with vitreous condensation and hypofluorescence at angiography is probably the most pernicious differential diagnosis.

In our case there was no history of dog or cat exposure, risky sexual activity, or intravenous drug use. Brazilian origin,¹⁵ posterior pole granuloma location, and appearance of retinochoroiditis with overlying vitritis (rather than only choroiditis) rather than only choroiditis oriented the diagnosis in favour of ocular toxoplasmosis. Therefore, oral antitoxoplasmic treatment was given at first intention. As IgG and IgM antibodies were positive for toxoplasmosis and aqueous PCR confirmed the presence DNA, we conclude that our patient represents a case of acquired ocular toxoplasmosis with retinochoroidal granuloma.

Classic therapy of ocular toxoplasmosis consists in an association of 2–4 g of sulfadiazine loading dose given over 24 h, followed by 1 g four times daily and 75–100 mg pyrimethamine loading dose over 24 h followed by 25–50 mg daily. Calcium folinate 2×25 mg is also given to avoid pyrimethamine-induced bone marrow depression.

Trimethoprim 80 mg/sulfamethoxazole 400 mg (2 tablets twice a day) or intravitreal clindamycin plus dexamethasone

might be acceptable alternatives.^{16 17} Other oral treatments including azithromycin, clindamycin, spiramycin, atovaquone need further assessment to determine their efficiency in comparison to classic therapy.

Steroid treatment was initiated after negative quantiferon TB spot. Systemic prednisone 1 mg/kg is prescribed to reduce chorioretinal destruction and may be offered in the presence of severe ocular inflammation or in the presence of macula infection; however, the risk of steroid-induced complications should be weighed, especially in a teenager, as there is no strong evidence of benefit.^{18 19}

In conclusion, our case report highlights the features of ocular toxoplasmosis presenting as a posterior pole granuloma.

Learning points

- Infectious diseases should be ruled out.
- Be aware of masquerade syndrome due to viruses and tumours.
- Sulfadiazine, pyrimethamine and calcium folinate form the classic treatment.
- Trimethoprim/sulfamethoxazole or intravitreal clindamycin plus dexamethasone might be acceptable alternatives to classic treatment.

Competing interests None.

Patient consent Obtained.

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