Ocular Toxoplasmosis (outline)

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Toxoplasma Gondii is an obligate intracellular pathogen that replicates within a parasitophorous vacuole. Infections are initiated by digestion of parasites deposited in cat faeces or in undercooked meat and probably many more infection routes (1). Parasites then disseminate to target tissues such as all neuronal tissues including the retina where they then develop into long-lived asymptomatic tissue cysts (2). Occasionally, cysts reactivate and growth of newly emerged parasites occurs when the control of cysts by the host's immune system is escaped (3).

There is a substantial variation between patients in the severity of intraocular inflammation associated with ocular toxoplasmosis, attributable to multiple host- and disease-related factors. Results suggest that disease characteristics also vary in different areas of the world tributary to different virulence of strains (4). The mechanisms that regulate the inflammatory process in recurrent TRC are poorly understood and also the dynamics of the humoral
response are still to be fully explained (5,6,7).

The immune response in TRC, triggered by the presence of newly emerged parasites that originate from the rupture of the cysts, is crucial for the control of the infection and is activated towards the causing agent and therefore high titres of Ig can be found in the active phase of recurrence (8).

**Clinical presentation, diagnosis and treatment of toxoplasmic retinochoroiditis**

Unlike written in textbooks, the great majority of intraocular toxoplasmic episodes (retinochoroiditis) is not occurring following congenital toxoplasmosis but is the result of acquired (often silent) toxoplastic infection. Therefore retinochoroiditis is presenting in the classical way of a retinal focus in the vicinity of a previous scar in less than 60% of cases.

**Symptoms**

Most often patients complain of floaters and blurred vision. Sometimes the brow is painful when a hypertensive uveitis is present.

**Clinical signs**

Clinical presentation consists usually of a **solitary focus** of a whitish-yellow retinitis next to a pre-existing scar or without a scar. The disease is usually unilateral. **Vitritis** is usually severe taking the aspect of the often described “headlight in the fog” feature. Sometimes an associated **granulomatous hypertensive anterior uveitis** can be present.

Sometimes the toxoplastic focus can be located just beside or adjacent to the optic disc and this is termed **juxtapapillary toxoplasmosis** or Jensen’s juxtapapillary chorioretinitis, or it can even involve the optic disc directly. In this situation therapeutic intervention is an emergency.
**Investigations**

**Fundus imaging** is necessary to be able to monitor the evolution of the lesion. If possible **dual fluorescein and indocyanine green angiography** should be performed, showing the extent of retinal vasculitis, the importance of associated papillitis and the extent of choroiditis. Optical coherence tomography (OCT) gives details of the retinitis and information on the macula indicating the presence or not of cystoid macular oedema (CMO). **Visual field testing** is indicating the effect of the retinitis focus on the nerve fibre layer in the area involved showing a sectorial scotoma. Visual field testing is also important to monitor evolution of the disease and response to treatment.

In immunocompromised patients the extension of the retinitis can be very large, there can be more than one focus and there can be bilateral involvement.

**Differential diagnosis**

Any granulomatous posterior uveitis has to be considered, including infectious etiologies such as acute retinal necrosis (necrotic herpetic retinopathies), cytomegalovirus retinitis (in immunocompromised patients), syphilitic chorioretinitis and tuberculous chorioretinitis. Non infectious causes such as sarcoidosis should also be considered being however less likely as a differential diagnosis.

**Treatment**

Retinal foci in the vicinity of the optic disc or the macula or within the vascular arcades should absolutely be treated. Large lesions with severe vitritis outside these areas should probably also be treated. The aim of the treatment is to limit the tissue damaging inflammatory reaction and for this corticosteroids should be given, usually systemically (50 to 70 mg to begin with). In order to be able to give corticosteroids and to avoid proliferation of the parasite
concomitant anti-toxoplasmic antibiotics should also be given systemically. Several antibiotics can be used such as sulfadiazine (4g) and pyrimethamine (25mg) per day together with folinic acid, or clindamycin (4X600 mg/day), both for 4-6 weeks, or azithromycin (250 mg / day for 5 weeks) [10] to cite only a few possibilities.

Role of serologic testing in toxoplasmic retinochoroiditis

So far, in the literature and in uveitis conferences the role for toxoplasmic serology in toxoplasmic retinochoroiditis has been downplayed and even considered as irrelevant. This comes from the fact that general serology laboratories where samples from ocular patients are sent, do not understand what is the exact purpose for asking a serology in a patient with ocular toxoplasmosis. They invariably respond that the serology shows an old toxoplasmic infection and is negative for an acute infection (no IgM). This is an adequate response for internal medicine patients where an acute infection is usually searched for and motivates the serology quest. In ocular patients where, in an overwhelming majority of cases, the disease is the result of a recurrence, we want to know whether a clinical picture compatible with ocular toxoplasmosis is actually due to Toxoplasma Gondii and we therefore only ask to serology whether the patient has once been in contact with the agent which is confirmed by the presence even of low levels of IgGs. In summary, serology is useful in all active cases of suspected toxoplasmic retinochoroiditis. Very high titres are specific for toxoplasmic disease, thus confirming the suspected diagnosis. This is very important, especially in atypical cases.

Aim of this lecture is to summarize the pathophysiology of Toxoplasmic retinochoroiditis and give a hint on latest treatment options.
References


