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Editorials

Presumed ocular histoplasmosis syndrome in the Netherlands

The presumed ocular histoplasmosis syndrome (POHS) occurs in areas of the USA in which *Histoplasma capsulatum* is endemic. The ocular triad of POHS consists of peripheral punched out chorioretinal scars, peripapillary atrophy, and maculopathy. The maculopathy most frequently consists of a subretinal neovascular complex associated with a previous focal chorioretinal scar. Vitritis is not observed in POHS.

The characteristic ocular presentation was associated with infection with *H capsulatum* through epidemiological studies.^{1,2} However, only rarely has the *H capsulatum* antigen and organism been identified in an eye with POHS. Khalil enucleated an eye with choroidal melanoma and demonstrated chorioretinal foci with various stages of chronic inflammation in which both the antigen and organism were identified.³

A non-human primate model of experimental POHS has been developed and studied by Smith and colleagues.⁴⁻⁶ They demonstrated that following an acute systemic infection focal chorioretinal infiltrates developed in the eye, with the subsequent evolution to focal atrophic lesions. Thus, there is epidemiological, clinical, and experimental evidence to support an association between *H capsulatum* infection and the classic ocular triad of POHS.

In this issue of the *BJO* (p 7), Suttorp-Schulten *et al*'s report was conducted in the Netherlands to identify those patients with the diagnosis of POHS who presented with the classic ocular triad. We do not know the size of the total population reviewed, nor the size of the subpopulation who carried the diagnosis of POHS, but 81 patients were identified with the diagnosis and complete clinical data, including follow up and fluorescein angiography. Of the 81 patients, 51 had a clinical appearance compatible with POHS. The other 30 patients in this study lacked atrophic peripheral chorioretinal scars and should be dismissed. Although the latter may represent infection with *H capsulatum*, only patients who meet all of the criteria for the ocular disease should be included. However, the remaining 51 patients present strong evidence that an ocular syndrome identical to POHS exists in the Netherlands.

Although *H capsulatum* is a global pathogen, ubiquitous in the soil in many regions of the world, attempts to isolate the fungus in Europe have been unsuccessful. Indeed, none of the several dimorphic fungi known to cause

systemic disease in North America—including *Paracoccidioides*, *Coccidioides*, *Blastomyces*, and *Cryptococcus*—are endemic to northern Europe. However, the recent medical literature contains a host of case presentations of systemic mycoses in AIDS patients in Europe. The existence of histoplasmosis and other systemic mycoses in these patients suggests that Europeans may be exposed to non-endemic fungi at a higher rate than previously appreciated.⁷⁻⁹ Alternatively, it is possible that these patients contracted an asymptomatic systemic mycosis through travel in an endemic area. A detailed history of such travel is not provided in this report.

In summary, the interesting report by Suttorp-Schulten and colleagues provides further evidence in Europe for an ocular syndrome identical to that called POHS in the USA. It is most likely that exposure to *H capsulatum* or an analogous fungus in northern Europe is responsible for the ocular disease. The study of histopathological tissue with the polymerase chain reaction with the appropriate primers for *H capsulatum* and other mycoses may clarify this unresolved question. Such investigative studies are most important and should encourage physicians in northern Europe with patients with POHS to obtain consent for a postmortem study of their eyes.

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