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SHORT COMMUNICATION

Retinal fibrovascular proliferation associated with *Nocardia* subretinal abscess

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PURPOSE. *To report the development of extensive fibrovascular proliferation in association with* Nocardia *subretinal abscess.*
METHOD. *Case report.*
RESULTS. *Extensive retinal neovascularization with tractional retinal detachment developed soon after ocular involvement in a 61-year-old patient with systemic nocardiosis. Fundus fluorescein angiography showed extensive area of capillary nonperfusion and severe leakage from the neovascular complex. The* Nocardia *subretinal abscess responded to systemic antibiotics, and the retinal neovascularization and tractional retinal detachment stabilized after 3 months.*
CONCLUSIONS. *Retinal ischemia and severe retinal neovascularization may complicate intraocular nocardiosis. The authors propose secondary retinal vasculitis as a contributing factor towards the development of retinal ischemia in this setting.(Eur J Ophthalmol 2006; 16:641-3)*

KEY WORDS. *Intraocular nocardiosis, Neovascularization,* Nocardia *asteroides, Retinal detachment, Subretinal abscess*

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INTRODUCTION

Nocardia asteroides is a Gram-positive, filamentous, aerobic bacterium with some morphologic and staining properties in common with fungi. Nocardiosis generally occurs in immunosuppressed patients and primarily affects the lungs and the pleura. Hematogenous spread from the lungs to other organs occurs in approximately one third of patients. The systemic manifestations of nocardiosis include pneumonia and brain, kidney, adrenal gland, skin, and joint abscesses. Intraocular infection is an uncommon feature and typically presents with choroidal or subretinal abscess, chorioretinitis, and panuveitis (1). Other forms of ocular involvement include corneal ulcer, keratitis, scleritis, and periorbital infection (2). We report the case of a 61-year-old man, on systemic corticosteroid therapy for idiopathic thrombocytopenic purpura, who developed intraocular nocardiosis complicated by retinal neovascularization and tractional retinal detachment.

Case report

A 61-year-old man presented with a 2-week history of pain and loss of vision in the left eye. He had recently been hospitalized for persistent pneumonia, multiple cerebral abscesses, and skin lesions. Transbronchial and skin biopsies had demonstrated infection with Nocardia asteroides, and he was receiving treatment with trimethoprim sulfa, Biaxin, demeclocycline, and rifampicin. Past medical history was notable for idiopathic thrombocytopenic purpura treated by systemic corticosteroid therapy.

The best-corrected visual acuity was 20/20 in the right eye and counting fingers (CF) in the left. Biomicroscopy of the left eye was notable for anterior chamber inflamma-

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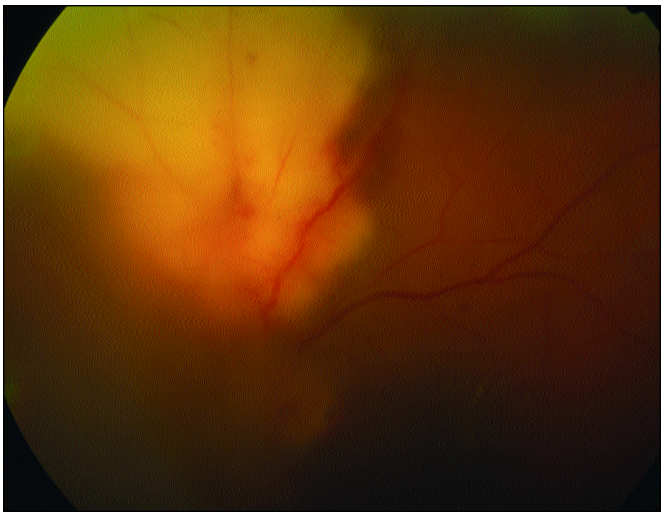

Fig. 1 - *Fundus photograph showing a large subretinal abscess, with surrounding serous elevation of the retina, and perivascular sheathing. The view of the nasal retina is obscured by dense vitreous opacities.*

tion, posterior synechiae, and moderate cataract. Funduscopy disclosed vitritis, mild swelling of the optic disc, a large subretinal abscess superior to the optic disc, and overlying retinal perivascular sheathing (Fig. 1). Examination of the right eye was normal. The patient declined surgical intervention and was treated with topical cycloplegics, steroids, and continuation of his systemic antibiotics.

Over the subsequent 3 months, the vision remained CF in the left eye, and the anterior chamber inflammation, vitritis, and the subretinal abscess resolved. Extensive fibrovascular proliferation developed over the area of the subretinal abscess, resulting in a large area of tractional retinal detachment involving the superior retina and part of the macula (Fig. 2A). The view of the fundus became limited by the development of cataract and posterior synechiae. Fundus fluorescein angiography revealed areas of capillary nonperfusion and retinal neovascularization with extensive leakage (Fig. 2B). The patient declined further treatment. At 5 months follow-up, the vision was hand motion, the fibrovascular complex and the tractional retinal detachment persisted.

DISCUSSION

The diagnosis of ocular nocardiosis may be made with culture of the organism from sputum or transtracheal aspirates along with a compatible ocular picture (3). In the current case, the diagnosis of intraocular nocardiosis was based on the pulmonary and skin infection with Nocardia asteroides and characteristic appearance of the subretinal abscess. Of interest in our patient is the progressive retinal vascular proliferation and tractional retinal detachment that developed soon after the subretinal abscess.Retinal vascular proliferation is a recognized sequel of severe uveitis. The mechanism is unclear and may involve production of angiogenic factors by cellular mediators of the inflammatory response.

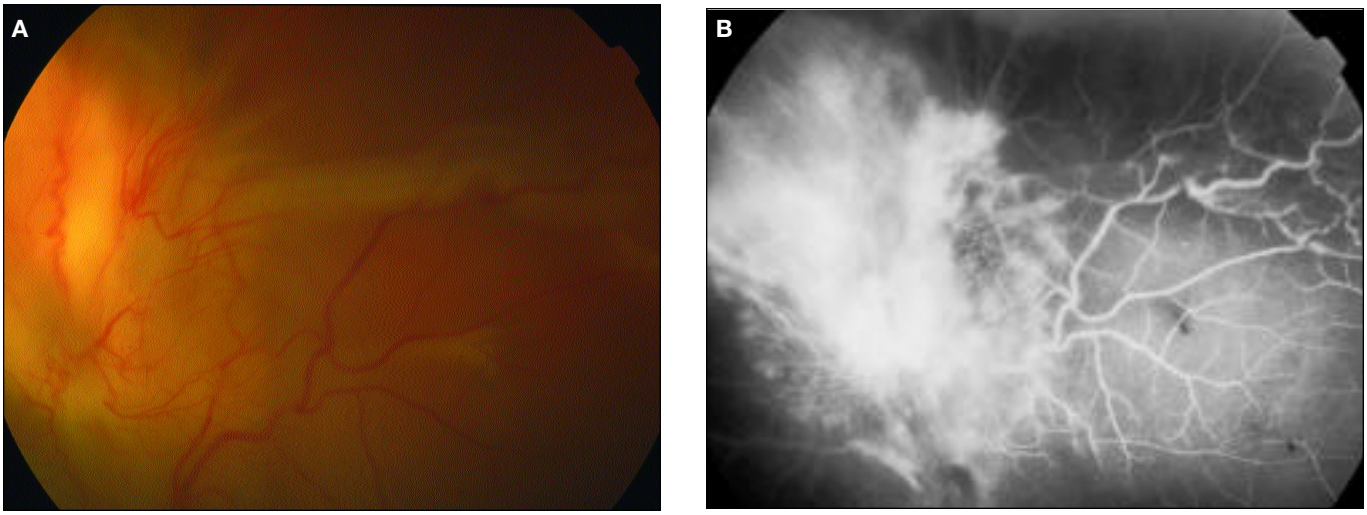

Fig. 2 - A) *Fundus photograph showing gross retinal neovascularization superior to the optic disc with extensive tractional retinal detachment.* **B)** *Fundus fluorescein angiogram showing retinal neovascularization with tractional retinal detachment. The fundus detail is partly obscured due to the development of cataract, posterior synechiae, and residual vitreous haze.*

A high concentration of bacterial toxins may damage the retinal microvasculature and promote intravascular thrombosis causing capillary occlusion. Vascular occlusion may also develop secondary to retinal vasculitis.

In our patient, the presence of perivascular sheathing suggested retinal vasculitis over the area of the abscess. Review of the fluorescein angiogram of a case of Nocardia choroidal abscess reported by Bozbeyoglu et al (4) appears to indicate retinal vasculitis over the involved area.

Fundus fluorescein angiography in our patient demonstrated a large area of retinal capillary nonperfusion corresponding to the subretinal abscess. We propose secondary retinal vasculitis as a contributing factor towards the development of retinal ischemia in this setting. It is likely that the resultant ischemia, together with severe inflammation, provoked an aggressive fibrovascular response.

The authors have no financial interests.

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