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Sir,

Aspergillus iris granuloma in an immunocompetent individual

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Ocular *Aspergillosis* is usually associated with posterior segment involvement in immunocompromized hosts.[1–6] One case of ocular *Aspergillosis* is reported in an immunocompetent individual.[7] *Aspergillus* iris granuloma is very rare[2,3] and to the best of our knowledge it has not been reported in an immunocompetent individual. We describe a healthy young individual who presented with an *Aspergillus* iris granuloma. Histopathological and microbiological evaluation of the lesion assisted us in planning the appropriate management.

Case report

A 34-year-old male presented with a white opacity over the iris in his left eye for one month. He was diagnosed elsewhere as having inflammatory granuloma and had been put on systemic corticosteroids. General physical examination and systemic work-up were normal. The patient was apyrexial. Systemic investigations for specific foci including blood, urine, cardiological and bronchoscopic evaluation was negative. His corrected visual acuity was 20/20 in his right eye and 20/200 in the left eye. The right eye examination was normal. The left eye showed deep anterior chamber (AC) with 2 + flare and cells and 1 mm of hypopyon, and an exudative granuloma was observed over the iris surface at 6 o'clock (Figure 1a). It was not possible to get the details of the posterior segment, however vitreous was found to be echo-free on ultrasonography. Diagnostic AC tap performed on day 1 was found to be negative. Over the next two days AC exudation had increased and vision was light perception. Excision biopsy of the granuloma was performed on day 3 through a corneal incision. Part of the iris measuring 2 mm was abscised along with the granuloma and was subjected to histopathological and microbiological evaluation.[8] Five μg of intracameral amphoptericin B in 0.1 ml was given, due to high suspicion of fungus. Initial AC fluid and the biopsy grew *Aspergillus fumigatus* in 3 days on Sabouraud's dextrose agar and potato dextrose agar (at 27°C). Gomori methenamine silver (GMS) stain of the AC fluid revealed fungal hyphae (Figure 2a). Histopathology of the excision biopsy revealed thickened iris stroma, congested vessels and inflammatory cells (Figure 2b). Exudative membrane was observed on either surface of the iris. GMS stain revealed abundant septate branching fungal hyphae on the surface and within the iris stroma (Figure 2c). Five μg of intracameral amphotericin B in 0.1 ml was repeated on day 6. The patient was started on oral fluconazole 400 mg initial dose follwed by 200 mg daily, topical 5% natamycin eye drops every hour, 1% atropine sulphate eye drops three times a day and 0.03% flurbiprofen sodium eye drops two hourly. For the next one-month period the exudates showed regression and organization (Figure 1b). Parsplana vitrectomy was done to remove the fibrous tissue and vitreous. Posterior approach was adapted, in view of the iris and lens involvement with the fibrous tissue and anterior vitreous opacities on ultrasonography. Vitreous and fibrous tissue was negative for fungus. Systemic and topical antifungals were stopped at one-month post-vitrectomy period. At the sixth-month follow-up the visual acuity was 20/60, the eye was quiet (Figure 1c).

Comment

We suspected fungal etiology, due to the feathery nature of the granuloma (Figure 1a). An iris foreign

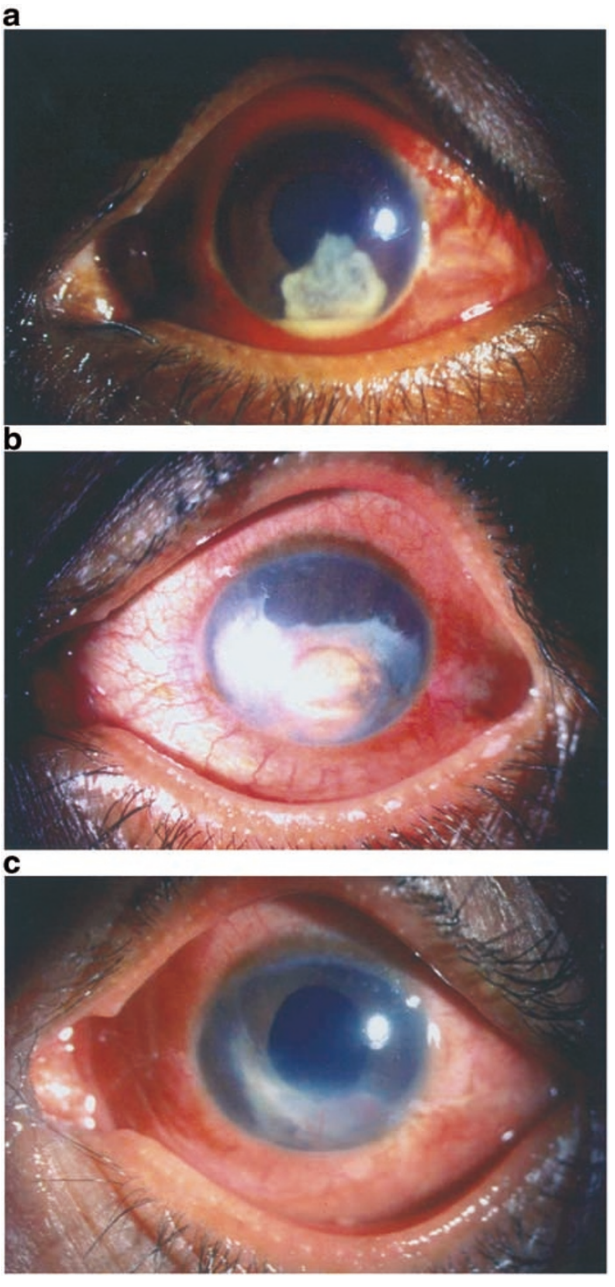

Figure 1 (a) Iris granuloma at presentation. (b) Residual fibrosis involving the pupillary area, anterior chamber and inferior cornea after control of infection with antifungal medication. (c) Clear anterior chamber at one-month post-vitrectomy follow-up.

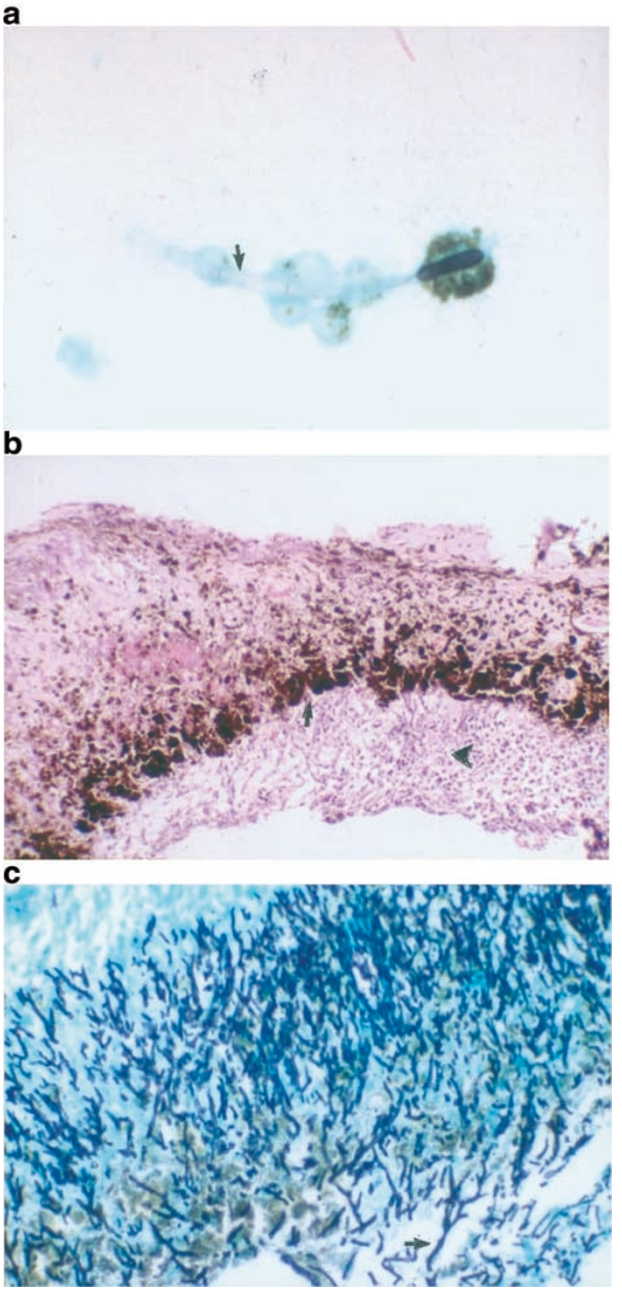

Figure 2 (a) GMS staining of the anterior chamber fluid showing single septate fungal hypha (black arrow) (× 500). (b) Histopathologic examination of the biopsy material showing iris tissue (black arrow) and adherent exudative membrane (arrowhead) (hematoxylin and eosin stain, × 125). (c) Gomori methenamine silver staining revealed abundant fungal hyphae (black arrow indicates a fungal hypha) (× 250).

body can be a differential diagnosis, which was ruled out by absence of trauma, wound of entry and foreign body on biopsy. Demonstration and isolation of the same fungus (*Aspergillus fumigatus*) from AC fluid and iris tissue indicates contiguous spread. The primary involvement of the iris is indicated by abundant fungal infiltration, while occasional hyphae were seen in the AC fluid (Figure 2). Two cases of iris involvement with *Aspergillus* were reported in the literature, both were observed in immunocompromized individuals.[2,3] Ours is the first report of *Aspergillus* iris granuloma in an immunocompetent individual.

The patient in our report was an immunocompetent, healthy adult individual who was tested negative for

possible endogenous sources. He was a farmer by profession, but there was no history of injury with vegetative matter. Although we found an endogenous embolus in the iris vasculature, we were not able to locate the primary source.

Our case highlights the need to consider fungal etiology as a differential diagnosis in an iris granuloma. In our case excision biopsy of the iris granuloma and an AC tap played a vital role in clinching the diagnosis. Institution of steroids without antifungal treatment could be disastrous in case of a misdiagnosis.

Our case is the first report of *Aspergillus* iris granuloma in an immunocompetent individual. A high degree of clinical suspicion, combined with microbiological and histopathological evaluation helped to arrive at an appropriate diagnosis and successful management.

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Sir,

E-Cadherin distribution in normal and dysplastic conjunctival epithelium

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Conjunctival and other mucosal associated lymphoid tissues share functional and morphological similarities including the presence of intraepithelial lymphocytes (IELs) in the basal epithelial layer.[1] The conjunctiva can induce immune tolerance to encountered antigen, wherein repeated challenge by an antigen at a mucosal surface causes suppression of the local immune response at the same and possibly other tissues, despite systemic immunogenicity to the same antigen.[2] It is believed that IELs of mucosal epithelia have a suppressor role in the induction of mucosal tolerance.[2,3] IELs express the integrin $\alpha E\beta 7$ (human mucosal lymphocyte antigen, HML-1), the ligand for which is E-cadherin, an epithelial intercellular adhesion molecule.[4] Normally E-cadherin is responsible for the induction and maintenance of cell polarity and differentiation in both foetal and adult epithelial