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CASE REPORT

Keratomycosis With Superadded Bacterial Infection Due To Corticosteroid Abuse-A Case Report

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ABSTRACT

We report a case of keratomycosis by *Aspergillus flavus* with superadded bacterial infection by *Pseudomonas aeruginosa*. An 83 years old male patient was referred to our institute with pain and copious mucopurulent discharge from the left eye of 15 days duration. Clinically, the case was diagnosed as keratomycosis. Gram stain showed Gram variable fungal elements and Gram negative bacteria, while the potassium hydroxide preparation showed branching septate hyphae.

Key Words: mycotic keratits, *Aspergillus flavus*, corticosteroids.

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Introduction

Keratomycosis is a clinical entity which is defined as the infection of the corneal stroma which is caused by a variety of fungal species. The majority of keratomycosis cases occur among the agricultural workers following corneal trauma with vegetative material contaminated with fungi. More than 70 genera of moulds and yeast have been associated with keratomycosis [1]. Hyaline moulds like aspergillus and fusarium are more frequently isolated as causative agents than the phaeoid (dematiaceous) mould [2],[4] .Fusarium and aspergillus dominate the list of the causative agents of keratomycosis. Concomitant occurrence of bacterial and fungal keratitis is not an uncommon occurrence. *Staphyloccous aureus* is the most common cause of bacterial keratitis. *Pseudomonas aeruginosa* is assuming predominance and is frequently reported as a cause of keratitis in association with daily or extended wear soft contact lenses, as a contaminant in the hospital environment, in fluoroscein solution, in cosmetics and in burns patients.

Case Report

An 83 year old male villager presented with pain and copious mucopurulent discharge from left eye since 15 days. He gave a history of cataract surgery by phacoemulsification 3 months ago, with insertion of a posterior capsule intra ocular lens (IOL). The patient was relatively asymptomatic till 2 months ago, while regularly attending follow-up. He was prescribed dexamethasone and chloramphenicol eye drops, one drop six times daily for one week, followed by one drop four times daily for four weeks, which were to be gradually tapered off. Initially, he complained of slight discomfort with foreign body sensation in his operated eye. The patient increased the frequency of the drops with the belief that it would bring relief. On the contrary, his condition worsened and he started

complaining of photophobia and profuse sticky discharge. By the time he presented in our institute, he had a severe form of the ulcerative disease.

On ocular examination, a central corneal ulcer, 10mm in diameter and whitish on the surface with characteristic feathery edges in the left eye was seen. There was a copious amount of mucopurulent yellowish green exudate that adhered tenaciously to the ulcer surface and covered the conjunctiva. The bulbar and palpebral conjunctivas were diffusely congested and vision was impaired in that eye. The right eye was opthalmologically normal. For microbiological examination, corneal scraping was collected under aseptic conditions and topical natamycin therapy was started.

A Potassium hydroxide (KOH) preparation revealed thick hyaline septate hyphae exhibiting acute angled branching. Gram stain revealed Gram variable hyphae with many pus cells and Gram negative bacteria. On blood agar, beta haemolytic colonies appeared after overnight incubation. The organism was oxidase positive, it produced a green pigment when it was subcultured on nutrient agar and it was identified as *Pseudomonas aeruginosa* by standard biochemical reactions. The isolate was sensitive to ceftazidime, ciprofloxacin, pefloxacin, amikacin and gentamicin. After five days of incubation at room temperature, sabourauds dextrose agar (SDA) showed a velvety yellow to green colony with a red brown reverse. On the basis of morphological features in slide culture, the isolate was identified as *Aspergillus flavus*. Since the patient had extensive ulceration, he was put on parenteral ofloxacin, oral fluconazole and topical natamycin and ciprofloxacin eye drops. The patient started responding from the third day onwards and was discharged after 15 days of intensive therapy.

Discussion

The most important defense barrier for the cornea is an intact corneal epithelial layer. The majority of corneal infections result from trauma to the corneal epithelium. Alteration of any local type (ectropion, entropion and lagophthalmos, tear deficiency diseases, etc) or systemic (diabetes, immunodeficiency states) defense mechanisms may predispose to corneal infection. Inappropriate use of antibiotics may eliminate the natural protection which is provided by the normal ocular flora and predispose the patient to opportunistic infection. The use of topical corticosteroids may cause localized immunosupression, predisposing the patient to bacterial keratitis. A study reporting bacterial keratitis in elderly patients found prior surgery as one of the leading risk factors [5].

Pathogenesis of bacterial keratits initially requires the adhesion of bacteria to disrupted or normal corneal epithelium. A few bacteria such as *Nisseria gonorrhoea*, *Cornybacterium diphtheria*, *Shigella* spp. and *Listeria* spp. may directly penetrate the corneal epithelium [4]. In the present case, there were three predisposing factors, namely, old age, operative manipulation and the excess use of topical corticosteriods and antibiotics. The corneal epithelium may have been damaged during the operation, which must have led to the corneal ulcer. The aggravating factors like old age and the excess use of corticosteriods and antibiotics may have led to the flaring up of the infection.

Corticosteroids and antibiotics can also interfere with the normal chemotactic response. Corticosteroids may enhance the invasion of saprophytic organisms, but may also alter the clinical signs of the infection [12].
Corticosteroids and immunosupressive agents impair the host defenses by
-Inhibiting chemotaxis and ingestion by phagocytes.
-Blocking degranulation.
-Interfering with lysosomal levels.
-Reducing the production of phagocytes.

Topical antibiotics can suppress various inhibitory substances by suppressing the normal flora and leading to secondary infection by saprophytic organisms. Partial suppression of bacterial replication by sublethal antibiotics can:
-Diminish the suppurative process
-Mask the typical features of the infection
-Produce atypical features.

Concomitant infection by both bacteria and fungus is not uncommon, since they share the same predisposing factors. The relative

frequency of different bacteria as causative agents in keratitis may vary geographically. *Pseudomonas* species is reported to be the most commonly isolated organism [7],[8], especially in association with daily or extended wear contact lenses [8],[9]. Being widely distributed in nature, pseudomonas can easily contaminate ophthalmical preparations [9],cosmetics [10], etc. Pseudomonas produces perhaps the most distinctive bacterial corneal infection, but ulcers caused by other Gram negative bacteria lack such discriminating features. The bacterial corneal infection caused by Pseudomonas presents with a rapidly progressive central or paracentral broad shallow ulcer, with a copious mucopurulent, yellowish green exudate that adheres tenaciously to the grey ulcer surface and covers the conjunctiva. The remaining cornea has a ground glass appearance with loss of transparency or a diffuse graying of the epithelium away from the ulcer site [12]. The ulcer can progress rapidly (with or without treatment) to a stromal abscess that can spread concentrically and symmetrically to form a ring ulcer, which is accompanied by a large hypopyon. Perforation is a distinct threat. Several clinical types of pseudomonas keratitis are seen. The most common one is caused by a cornea virulent strain that has the potential for the rapid destruction of the stroma and early descematocele formation (liquefactive necrosis), with risk of perforation within a day. The virulence is attributed to the production of proteolytic enzymes.

The fungi are opportunistic organisms and colonize when the natural defenses of the eye are abrogated. Essentially, all are saprobic fungi and are not associated with infection in healthy individuals[11]. Injudicious use of corticosteriods and antibacterial agents for external ocular disease enhances the risk of keratomycosis Under normal conditions, saprophytic fungi are destroyed by humoral and cellular defense mechanisms and a large number of spores of aspergillus and fusarium generally produce only mild keratitis. However, when corticosteroids or immunosuppressive agents are given, the invasive ability of the fungus is enhanced.

Filamentous fungi are responsible for up to one third of all traumatic infections. Fungi are unable to penetrate the intact epithelium and hence, any trauma, particularly organic matter, facilitates the penetration of the fungal innoculum into the corneal stroma [11]. Filamentous fungal keratitis can occur after surgeries such as penetrating keratoplasty and radial keratotomy .In fungal keratitis, the inflammatory reaction results from
-Replicating and non replicating fungi
-Mycotoxin
-Proteolytic enzymes
-Soluble fungal agents

Our patient responded to topical natamycin and ciprofloxacin with systemic ofloxacin and fluconazole, thus indicating its use in the management of mixed infectious keratitis due to *Aspergillus flavus* and *Pseudomonas aeruginosa.*

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