Keratitis due to *Cylindrocarpon lichenicola*

Sir,

Ulceration of the cornea due to fungi (mycotic keratitis) is an important ophthalmologic problem in the developing world, where agriculturalists are at risk for ocular trauma by fungus-laden plant material or soil particles. Keratitis caused by rare or emerging fungi may pose diagnostic challenges to the clinician under such circumstances. We describe in this paper, keratitis due to *Cylindrocarpon (Fusarium) lichenicola*, an emerging fungus, in a 56-year-old Indian male.

The patient, a farmer, presented with complaints of pain, redness and irritation of the left eye following ocular injury caused by hay 5 days earlier. He was not a contact lens-wearer and did not give a history of any prior ocular disease. Slit-
lamp examination of the affected eye [Figure 1] revealed a corneal ulcer (6x5 mm) with irregular margins, raised, with necrotic slough, infiltration involving 75% of the corneal thickness and a 1 mm central endothelial plaque. The anterior chamber exhibited aqueous flare and a 2 mm hypopyon, the lens was cataractous and visual acuity was restricted to “hand movements” only. The right eye was normal with a visual acuity of 6/60 (unaided). Since an infection was suspected, scrapings were taken from the base and edges of the corneal ulcer by a sterile blade. Microscopic examination of these scrapings, stained by lactophenol cotton blue [Figure 2a] and Gram stain, revealed numerous septate fungal hyphae. A presumptive diagnosis of filamentous fungal keratitis was made; treatment was initiated with topical natamycin (5%) and ciprofloxacin (0.3%) hourly and topical mydriatics twice daily. The ulcer initially remained quiescent. However after 5 days of therapy, the ulcer exhibited signs of worsening, necessitating surgical intervention (therapeutic penetrating keratoplasty [TPK]). Histopathological examination of the infected corneal button removed at surgery revealed numerous septate fungal hyphae [Figure 2b]. This graft became opaque, so TPK was repeated after two weeks. This second graft also underwent opacification. The patient refused further surgery and the eye eventually went in for phthisis bulbi.

Scrapings inoculated onto Sabouraud glucose-neopeptone agar (SDA) [Figure 3a] and sheep blood agar grew cottony, white-red fungal colonies within 48 hours. The reverse of the fungal colony on SDA exhibited brown pigmentation. The fungus was identified as Cylindrocarpon lichenicola since there were: branched, septate, hyaline hyphae; phialides on simple or sparsely-branched conidiophores; cylindrical to fusiform, smooth-walled macroconidia, each with 3 to 6 septa, a blunt rounded apex and distinctly truncate base [Figure 3b]; smooth-walled chlamydoconidia on short branches.[2] There was no bacterial growth in culture. Culture of the infected corneal button also grew C. lichenicola.

Cylindrocarpon species occur in the soil as saprophytic or weak pathogenic fungi on the roots of many herbaceous and woody plants in India and other tropical countries.[3] Cylindrocarpon thrives in the soil of southern India, where temperatures and humidity remain relatively high all year round[3] and is a common agent of post-harvest fruit invasion.[2] Thus, agriculturalists and outdoor workers may become accidentally infected with Cylindrocarpon while at work. Cylindrocarpon species may cause non-ocular infections (athlete’s foot, intertrigo, mycetoma and peritonitis) in humans.[4] Cylindrocarpon keratitis appears to occur very rarely,[5-7] the species involved have been. C. lichenicola (originally called Fusarium lichenicola and later Cylindrocarpon tonkinense) and Cylindrocarpon vaginata.

The taxonomic status of Cylindrocarpon species is currently unsettled. Some species of Cylindrocarpon and Fusarium share teleomorphs (sexual forms) in the genus Nectria. Also, sequencing of ribosomal DNA revealed that C. lichenicola is
C. lichenicola is sometimes misidentified as F. solani. However, C. lichenicola differs from F. solani in forming macroconidia which are predominantly straight rather than curved, by having apical cells that are rounded rather than tapering and basal cells with truncate and offset rather than attenuated pedicels (foot cells), by lacking microconidia, by having pigmented chlamydoconidia and by a brown color rather than cream-colour on reverse of SDA medium. Our fungal isolate exhibited these features of C. lichenicola [Figure 3b].

In our patient, the cornea probably became ulcerated and infected with Cylindrocarpon (Fusarium) lichenicola following ocular injury by soil-contaminated hay containing Cylindrocarpon spores (conidia). Due to inadequate treatment before presentation, the ulcer probably worsened, so that the patient presented to us with severe keratitis. After initiating hourly topical natamycin treatment, the ulcer remained quiescent for 5 days; however, a rapid deterioration necessitated TPK. Successful treatment of Cylindrocarpon tonkinensis keratitis by using topical natamycin 5% has been reported.[1] However, others have reported less favorable outcomes for Cylindrocarpon keratitis, even with the best available treatment.[2][3] These earlier reports and our present experience, indicate that Cylindrocarpon spp. may cause a severe form of keratitis that responds poorly to medical or surgical intervention. Hence, clinicians should suspect fungal infections in corneal ulcers that arise following injury by plant material or soil and institute suitable diagnostic and therapeutic measures as early as possible.

Relapsing encephalopathy secondary to non-hepatic hyper-ammonemia

Sir,

A 76-year old man, with a past history of atrial fibrillation, hypertension, anterior and posterior circulation “transient ischaemic attacks” (TIA) over 3 years, subdural hematoma while on warfarin and peptic ulcer disease, presented with upper gastrointestinal bleed. There was no history of prolonged alcohol use and no signs of liver disease or portal hypertension. He was resuscitated with fluids and blood and underwent an emergency endoscopy. This revealed evidence of Billroth II gastrectomy and a stomal ulcer with fresh blood and clot, that was injected. There were no varices to suggest portal-systemic shunting. Post endoscopy, he was unresponsive for 24 hours and subsequently improved to being drowsy with slurred speech. On the 3rd hospital day, he regained consciousness and returned to his pre-morbid state over one week. Investigations showed normal electrolytes, liver function tests and coagulation. No new changes were noted on a subsequent ultrasound. Haemoglobin concentration was 9.7 g/dl at presentation and returned to 11.6g/dl after transfusion. A computed tomography of the head was unremarkable except for evidence of previous surgery and generalised atrophy. A diagnosis of posterior circulation TIA was made. He returned to outpatient clinic in a month and was noted to be well.

One month later he presented with drowsiness and “tremor”. His wife reported “good” and “bad” days. He had asteincis but no focal neurological deficit. A complete work-up (including checking metabolic parameters, thyroid function tests, estimation of calcium, arterial blood gas (ABG) and serum ammonia) was performed. No new changes were noted on a repeat CT scan. By the next day, the patient improved and insisted on being discharged.

A month later he presented with confusion and encephalopathy. A diagnosis of relapsing encephalopathy secondary to hyperammonemia was made based on previous and current ammonia levels. His ABG showed respiratory alkalosis without hypoxemia. He was initiated on lactulose therapy and a protein-restricted diet. A work-up was undertaken to look for an underlying chronic liver disease. An ultrasound and CT scan of the abdomen showed normal liver architecture and no evidence of portal hypertension or portal-systemic shunting. Incidental small haemangiomas were noted in the

References


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