tions. In these regions predominantly filamentous fungi rather than yeasts cause fungal keratitis. More than 70 species belonging to 40 genera have been reported as causes of keratitis.[1] *Aspergillus* species were the most common in Saudi Arabia,[3] India,[4] and Bangladesh,[5] followed by *Fusarium* species, *Curvularia* species (6–8%) and *Candida* species. In Miami *Fusarium* species are the commonest.[6] There may be a history of agricultural trauma or washing in contaminated water.

In Northern climates fungal keratitis is less common and yeasts are more frequent than filamentous fungi. Often patients present who have already been treated unsuccessfully with topical antibacterial agents and corticosteroids. Clinical features of fungal ulcers include a dry, raised, necrotic or fluffy surface. They may be associated with endothelial rings.[5] Topical medication should be stopped for 24–48 hours prior to rescraping. Staining by Gram stain is highly sensitive[7] for hyphae and spores, and calcofluor white staining has also been used.[8] Direct inoculation of a Sabouraud plate is also reported as a method of isolation.[9]

Curvularia species are filamentous fungi, present as saprophytes in soil and air as well as on plants. Keratomycosis can be caused by *C. lunata*, which has been isolated from corneal ulcers in tropical and subtropical areas, where it accounts for 6% of mycotic ulcers.[1] In addition *C. geniculata*, *C. pallescens*, and *C. senegalensis* have been isolated from eye infections and there is one report of *C. brachyspora* as a cause of mycotic keratitis.[10]

Curvularia keratitis has been successfully treated with amphotericin B and Pimaricin 5% solution.[7] In this country no previous cases of *Curvularia* keratitis have been reported, although it is well recognised in subtropical and tropical areas. In this case there was a history of foreign travel and exposure to water in the Dead Sea. In the absence of trauma, the patient may have been predisposed to infection because of previous ptosis surgery. This case emphasises the importance of prompt diagnosis in recognising and identifying fungal infection, as antifungal agents will minimise visual morbidity. We would discourage the use of corticosteroids without an established microbiological diagnosis.

It is important to establish whether there is a history of foreign travel, as this may be indicative of fungal aetiology in corneal ulcer.

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Sir,

A Case of Central Retinal Artery Occlusion after Carotid Endoarterectomy

Occlusion of the central retinal artery is a well-documented entity that can be caused by trauma, thrombosis, vascular spasm or embolism.[1,2] In most cases resulting from emboli, these arise from ulcerated atheromatous plaques along the walls of the common or internal carotid arteries.[1,3]

This is the first report of central retinal artery occlusion following carotid endoarterectomy.

Case Report

A 65-year-old man was referred to the Division of Vascular Surgery of the University of Rome 'Tor

Vergata', Italy, with a 1 year history of left hemiparesis (face, arms and legs) and a single episode of amaurosis fugax of the right eye 5 months earlier. The patient was a smoker with high blood pressure. The lipid profile and blood chemistry parameters were within normal limits.

Doppler ultrasound and angiographic studies of the epiaortic vessels revealed an ulcerated, annular atheromatous plaque situated at the origin of the right internal carotid artery. The lumen of the latter was reduced by 90%. No changes were noted in the common or external carotids, the subclavian or the vertebral arteries. Ocular blood flow appeared to be within normal limits. The results of Holter monitoring, echocardiography and cranial computed tomography were unremarkable.

The patient underwent right carotid endoarterectomy under general anaesthesia. One minute before the artery was clamped, 1 cm^3 of heparin was administered intravenously. An intraluminal shunt was introduced after 7 minutes because of changes in the electroencephalogram. After surgery heparin calcium was administered subcutaneously.

On the morning of post-operative day 3, the patient reported that he had experienced a sudden loss of vision in the right eye around 12 p.m. the night before, but had not informed the nursing staff. Examination of the right eye revealed diffuse constriction of the retinal arterioles with multiple, refractile, yellowish emboli located at the vessel bifurcations (Fig. 1). The retina itself appeared uniformly pale, even at the fovea. The patient was able to detect hand movements at a distance of 30 cm with the right eye, and pupil reactivity was sluggish.

Occlusion of the right central retinal artery was diagnosed. Treatment (consisting of oral acetazolamide 250 mg, ocular massage, a rebreathing mask and anterior chamber paracentesis) was started immediately, but no improvement was noted. The patient was anticoagulated with heparin and started on aspirin 300 mg/day. The initial diagnosis was confirmed by fluorescein angiography performed 1 week after onset of symptoms. The follow-up examination at 1 year showed no sign of improvement.

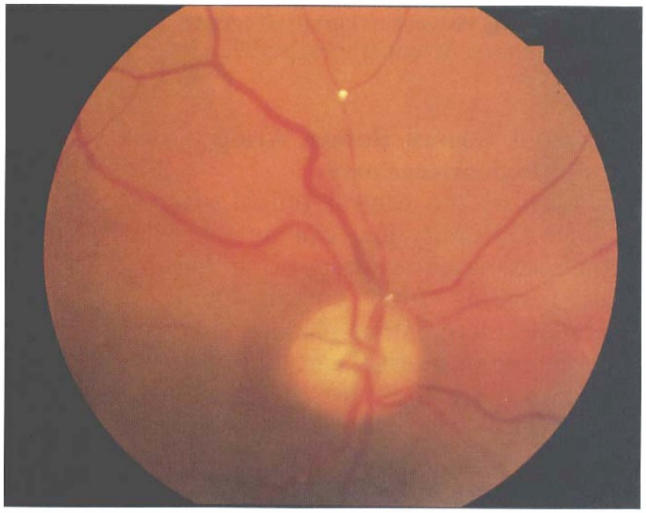

Fig. 1. *Fundus photograph showing multiple refractive emboli, located at the bifurcations of retinal arteries.*

Discussion

Cerebral embolism caused by atheromatous plaques is one of the most common and devastating illnesses in developed countries. Carotid endoarterectomy, consisting in the removal of the plaque and reconstruction of the vessel wall, has been proposed to prevent these events.[4]

Data from the North American Symptomatic Carotid Endoarterectomy Trial[5] and the European Carotid Surgery Trial[6] clearly show that, in patients with carotid stenosis of 70% or more and symptoms of transient ischaemic attacks, a combination of surgery and medical treatment is superior to medical management alone.

Carotid endoarterectomy itself, however, is not without risk. Morbidity and mortality associated with this procedure vary greatly depending on the experience of the surgeon. In 606 patients operated on in our centre, minor strokes occurred postoperatively in 0.6% and major strokes in 0.6%; the mortality rate was 1%.[7]

To our knowledge, this is the first report of central retinal artery occlusion after carotid endoarterectomy. The appearance of the emboli noted during the fundus examination (Hollenhorst emboli) strongly suggests that they originated from atheromatous plaques in the carotid artery.[1,8] Since the preoperative investigations failed to reveal any other lesions in either of the carotid arteries, it is quite possible that the emboli represented residual fragments of the plaque that was removed. However, scrupulous cleaning of the surface of the artery after removal of the plaque is one of the most important steps of the endoarterectomy procedure, and we cannot exclude the possibility that other sources of emboli were missed on the pre-operative examinations.

The failure of treatment in this case is probably the result of the delay with which the event was reported by the patient. Successful treatment of occlusion of the central retinal artery must be initiated within 6 hours of onset.[2] In this case, the loss of vision occurred suddenly during the night, and perhaps because of the similar, but transient, episode that the patient had experienced 5 months previously, he was not sufficiently alarmed to notify the nursing staff. Treatment was therefore begun approximately 12 hours after the occlusion, when irreversible ischaemia had already occurred.

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Sir,

Severe Reversible Ocular Anterior Segment Ischaemia Following Topical Trifluorothymidine (F3T) Treatment for Herpes Simplex Keratouveitis

Anterior segment ischaemia can result from vascular disease, ophthalmic surgical procedures, hyperviscosity syndromes and disseminated intravascular coagulopathy.[1,2] An acute ischaemic reaction in the anterior segment following long-term topical trifluorothymidine (F3T) treatment has previously been reported in the literature with conjunctival neovascularisation, band keratopathy and iris atrophy.[3] The reversible nature of F3T-induced ischaemia has not previously been documented.

We report a case of severe anterior segment ischaemia in a patient with known sarcoidosis using topical F3T for recurrent herpes simplex keratouveitis which improved on withdrawal of the topical medication. Conjunctival biopsy showed non-specific inflammatory features with no evidence of vasculitis.

Case Report

A 43-year-old man with past history of sarcoidosis, recurrent episodes of anterior uveitis in the left eye, and a 16 year history of recurrent herpes simplex dendritic keratitis in the right eye developed injection and irritation of the right eye. The patient had no previous history of trauma or ocular surgery to the right eye. Initially visual acuity was 6/18 on the right and 6/5 on the left. Herpes simplex keratouveitis was diagnosed clinically. Due to prior hypersensitivity to acyclovir, treatment with F3T five times a day, betamethasone q.i.d. and atropine b.d. was initiated. The treatment regime was reduced to F3T t.i.d. after 1 month but increased again to the initial frequency due to worsening keratitis.

Over the ensuing 1 month the patient developed generalised corneal oedema with ischaemic changes starting in the tarsal conjunctiva and progressing to 360° of ischaemia of the right anterior bulbar conjunctiva and sclera (Fig. 1). The inferior tarsal conjunctiva also became ischaemic. The visual acuity in the right eye deteriorated to count fingers at 50 cm. There was a large central corneal epithelial defect extending beyond the inferior limbus onto the bulbar conjunctiva. The anterior chamber had a moderate flare. Intraocular pressure by Goldmann

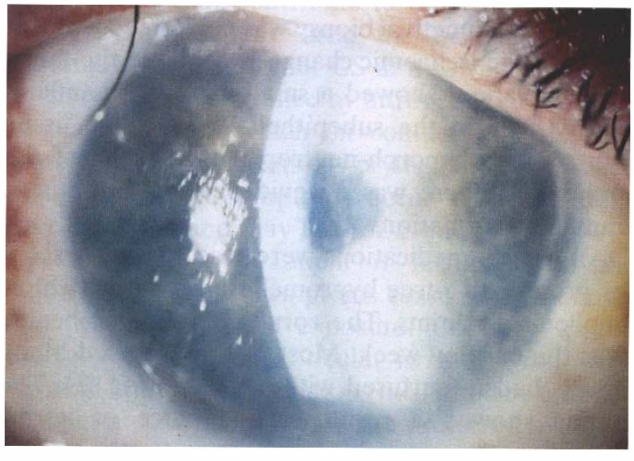

Fig. 1. *Generalised corneal oedema and limbal ischaemia.*