(18/90) that screen for visual impairment use an acuity chart, specified as a Snellen chart in 12 practices and as 'the usual eye chart' in 6 practices.

Practices were asked what happened if someone was found to have a visual problem. Twenty-four per cent (42/172) said they refer the patient to a community optometrist, with 29 of these 42 (17%) stating the patient is also advised to see the general practitioner. Forty-five per cent (77/172) gave no answer to this question.

Comment

Only 52% of practices in our survey are screening for visual impairment, the majority of which are using questions about visual problems as a screening tool. The sensitivity of such questions compared with formal visual acuity testing in the general population has been found to be around 30%.[1] Of those practices giving an answer, referral to an optometrist is the single most likely action to be taken if a patient is found to have a visual problem. Despite the re-introduction of free sight tests, the cost of spectacles may still deter people from attending the optometrist or from obtaining glasses.[1] What happens to those people who do attend (particularly those people with ophthalmological disease rather than uncorrected refractive error) is unclear.

Twenty per cent of general practices are not offering regular screening assessments to patients aged 75 years and over – a similar proportion to the 15% found in 1992.[4]

The use of questions about visual problems as a screening tool and the lack of clear plans of intervention for those people found to have a visual problem were proposed as explanations for the lack of effectiveness of screening for visual impairment found in a systematic review of randomised controlled trials.[5] These two factors, along with the low proportion of practices carrying out screening, suggest that elderly people are not being adequately screened for visual impairment. The over-75 checks policy is currently under review. Visual impairment in elderly people is common, disabling and frequently treatable. If general practice is to continue to be given responsibility for screening then adequate resources, training in visual acuity testing, and clear plans of intervention which acknowledge the role of optometrists will be needed.

L.S. is funded by a Health Services Research Fellowship from Thames Health Authority Research and Development Directorate

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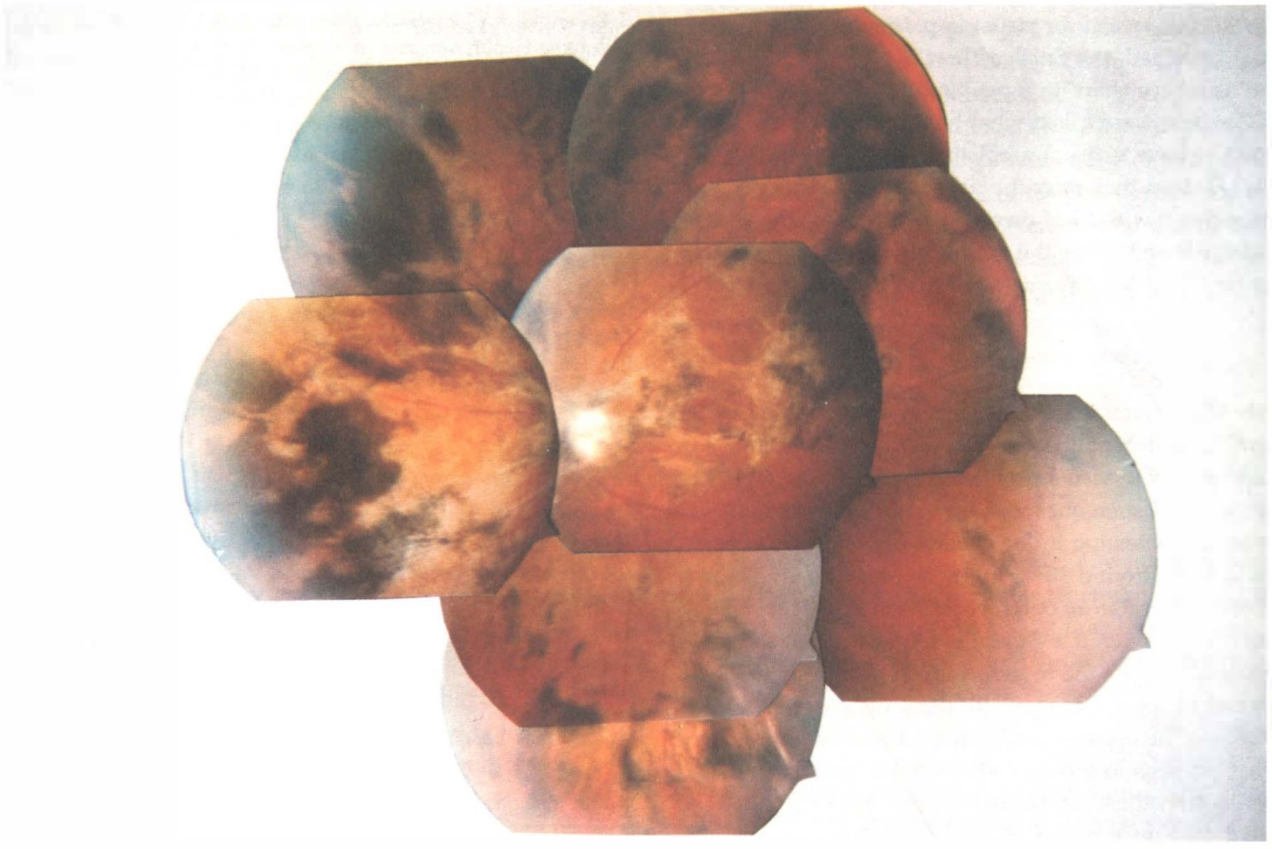
Sir,

Chorioretinal alterations in mucormycosis

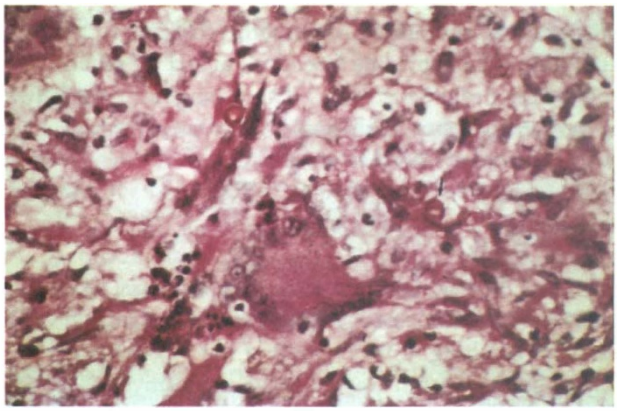
Mucormycosis is an acute infection caused by several fungi of the order Mucorales.[1] These fungi are normally saprophytic and non-pathogenic.[2] In mucormycosis several clinical syndromes are defined, rhino-orbito-cerebral being the most common.[2] Signs and symptoms of orbital mucormycosis include chemosis, periorbital cellulitis, ophthalmoplegia, proptosis, ptosis, abrupt visual loss, orbital pain and facial hypoesthesia. The fungi invade blood vessels, and cause necrotising vasculitis resulting in thrombosis of the vessel lumen.[3] Unless diagnosed and treated early, mucormycosis is often fatal, due to cerebral involvement.[2] We report a patient with rhino-orbital mucormycosis in whom choroidal ischaemia resulted in extensive chorioretinal pigmentary changes.

Case report

A 46-year-old woman reported that she had experienced left facial pain and had developed left proptosis and complete ophthalmoplegia with abrupt loss of vision. Coronal paranasal tomography had revealed left frontal, ethmoid and maxillary sinusitis with retro-orbital involvement on the left. Broad spectrum antibiotic therapy had not changed the condition. The patient was said by her ophthalmologist to have left optic atrophy. Medical history was negative for systemic diseases; however, fasting blood sugar was found to be 240 mg/dl. Biopsy taken by a local otorhinolaryngologist revealed necrotic material. The patient was then referred to our centre for further investigation.



(a)

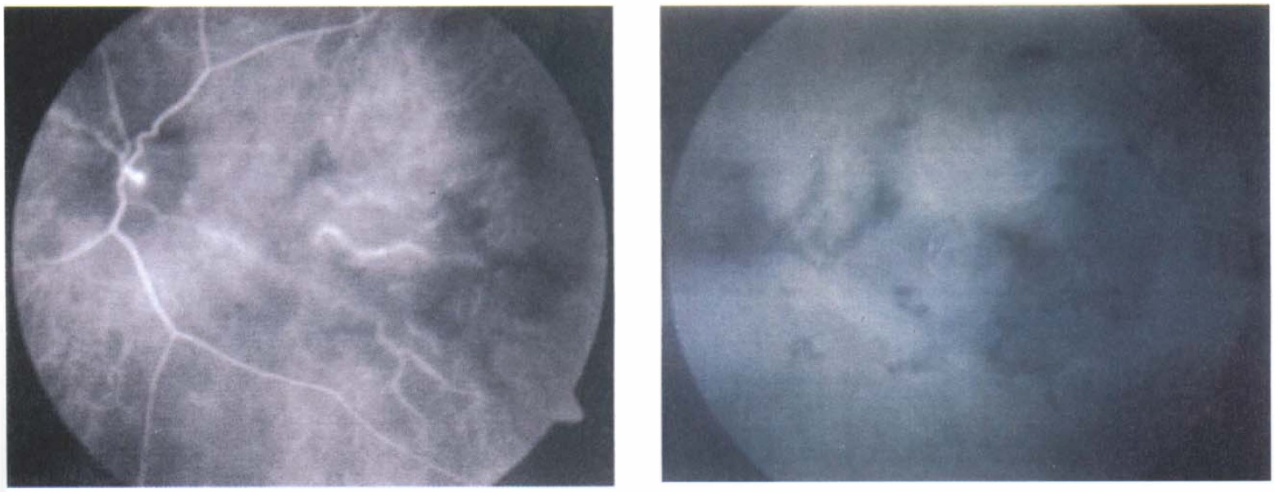


(b)

Fig. 1. *(a) Composite fundus photograph of the left eye revealing optic atrophy and wedge-shaped areas of retinal pigment epithelium hyperplasia. (b) Biopsy from left maxillary sinus, disclosing infiltration of mucosal tissues with granulomatous reaction. At the centre of granulations suppurative necrosis and fragmented non-septate hyphae (arrow) consistent with mucormycosis are seen.*

Three months after the start of the symptoms, ophthalmological examination at our centre revealed 20/20 vision in the right eye. The left eye had no light perception and an amaurotic pupillary response. Anterior segments were normal. The left eye was slightly proptotic with limited ocular movements. Ophthalmoscopy of the left fundus revealed widespread chorioretinal pigmentary changes emanating from a pale optic nerve head (Fig. 1a). Fluorescein angiography (FFA) revealed normal retinal vasculature with zones of hypo- and hyperfluorescence typical of choroidal infarction and secondary retinal pigment epithelium (RPE) scarring. Indocyanine green angiography (ICGA) demonstrated hyperfluorescence in all stages in the involved areas, due to vascular filling defect in the early phase and masking by the RPE hyperplasia in the late phase (Fig. 2a, b).

Diabetes mellitus was controlled by oral hypoglycaemic agents. The patient underwent transnasal endoscopic exploration of the left frontal, ethmoid and maxillary sinuses, and biopsy specimens disclosed fragmented non-septate hyphae leading to the diagnosis of mucormycosis (Fig. 1b). Therapy included intravenous amphotericin B (1.2 mg/kg per day) for 6 weeks, nasal irrigation with 20 cm^3 of amphotericin B solution in sterile water (100 $\mu g/cm^3$) twice daily for 3 weeks, and repeated endoscopic debridement of the left paranasal sinuses.



(a) (b)

Fig. 2. *(a) Early-phase indocyanine green angiography (ICGA) of the left eye. There is hypofluorescence due to avascular filling defect at the posterior pole. (b) Late-phase ICGA. There is hypofluorescence due to the masking effect of the retinal pigment epithelium hyperplasia.*

The latest follow-up visit, 4 months after the exact diagnosis, revealed full ductions of the left eye without any proptosis. Ophthalmoscopic examination did not reveal any change.

Comment

Rhino-orbito-cerebral mucormycosis classically presents as a subacute (progressing over days) to fulminant (over hours) necrotising, invasive process.[4] It begins with the inhalation of spores into the nasal mucosa, where in immunocompetent hosts a phagocytic response is generated, killing the spores. In immunocompromised hosts, such as diabetics with poor glucose control or ketoacidosis, those with haematological malignancies and the immunosuppressed, the spores may germinate into hyphae within the nasal mucosa, which then invade the sinuses, orbits and the brain.[5] Rarely mucormycosis occurs in otherwise healthy individuals.[5] In our patient diabetes mellitus was diagnosed after the onset of sinusitis, and was controlled by oral hypoglycaemic agents.

Ocular signs, predominantly orbital cellulitis with ophthalmoplegia, are present in 80% of cases and are among the earliest clinical manifestations.[6] Nearly 50% of mucormycosis cases are associated with visual loss, in which ocular and optic nerve ischaemia from fungal infiltration of blood vessels resulting in thrombosis plays a significant role.[7] Our case presented with visual loss, due to the occlusion of posterior ciliary arteries resulting in optic disc and choroidal infarction in the form of triangular syndrome,[8] manifesting as optic atrophy and widespread reactive RPE hyperplasia respectively.

RPE reaction to choroidal ischaemia begins with intracellular oedema and cell necrosis in the area of damage. Over time, adjacent RPE cells migrate to repopulate this region. RPE plaque formation as a response to ischaemia was noted as early as 1 week after experimental posterior ciliary artery occlusion, especially in the areas with the most severe necrotic changes.[9] On ICGA, choroidal ischaemia manifests itself as hypofluorescence due to vascular filling defect.[10] In our case, though the images could not be taken at the acute stage of posterior ciliary artery occlusion, it was still possible to observe the vascular filling defects on early ICGA.

To the best of our knowledge, the evolution of fundus changes from oedema of the optic disc and peripapillary retina to optic atrophy and chorioretinal pigmentary changes has been reported in only one case with rhino-orbital mucormycosis.[11] Unique to our case is the demonstration of chorioretinal changes caused by choroidal ischaemia secondary to mucormycosis, both clinically and on ICGA.

Treatment of rhino-orbital mucormycosis includes early diagnosis, aggressive surgical debridement including exenteration, when necessary, adequate drainage of sinuses, intravenous amphotericin B and the control of the predisposing disease.[3] In our case, though the diagnosis was delayed, repeated surgical debridement with intravenous and local amphotericin B was successful.

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Sir,

Malignant solitary fibrous tumour of the orbit: report of a case with 8 years follow-up

Solitary fibrous tumour (SFT) is a rare spindle cell neoplasm of the pleura of adults. It has been recently described in the orbit[1] among other extrapleural sites.[2,3] A small percentage of this type of tumour has malignant histological features and may manifest aggressive clinical behaviour, including recurrence, invasion of adjacent tissues and distant metastases.[4] No cases of the malignant variant have been reported in the upper respiratory tract and orbit.[1] We report the first case of a malignant orbital SFT with several recurrences, and showing progressive histological dedifferentiation and local invasion.

Case report

Clinical findings. A 64-year-old man presented unilateral exophthalmos due to a palpable mass in his left upper orbit. The lesion had been resected twice in the past 3 years since presentation. The mass is now radiologically well defined, with a medium signal on T1, in the upper external orbit, and pushed slightly on the optic nerve (Fig. 1, left).

A 3.3 cm macroscopically well-circumscribed mass was excised. It showed micoscopic involvement of the margins by the neoplasm. Therefore, an exenteration was performed. There was no evidence of residual tumour by macroscopic or microscopic examination of the specimen. Thirty-eight months later the patient had a recurrence of the tumour on the base of the orbit. It extended through the lateral wall, temporal muscle fibres and subcutaneous tissue. A new resection of various neoplastic nodules measuring < 3 cm in total was performed. However, the lesions recurred again 13 months later. A mass of 5 cm was found invading through the lateral wall of the orbit, temporal muscle and the maxillary region (Fig. 1, right). The patient died with a cervical abscess shortly after the surgical resection and 8 years after the onset of the orbital symptoms.

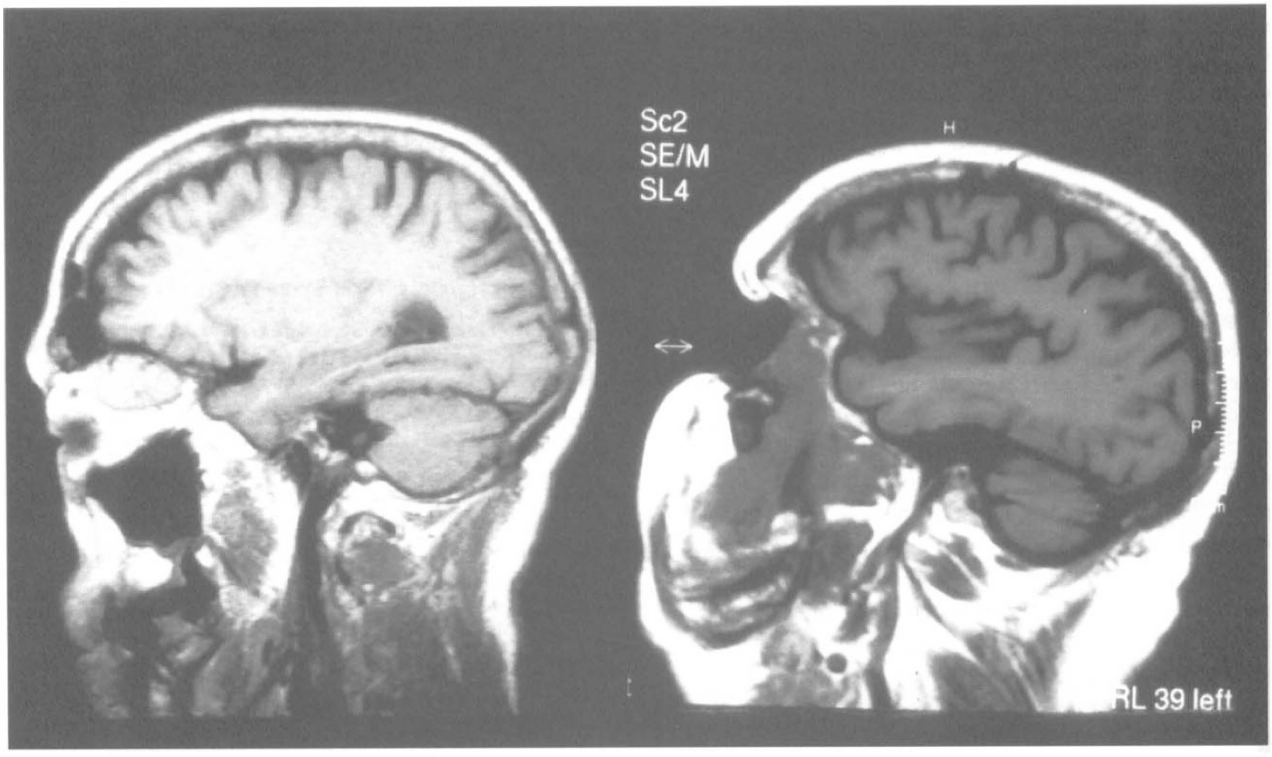

Fig. 1. *CT scan performed before exenteration (left) and extension of the tumour 38 months later (right).*