available for the conditions being sought, resulting in overall benefit for the patient. While we accept that changes in the practices for preschool vision screening are obviously needed, the "knock on" effect of such changes on diagnostic services in terms of workload and cost will need to be taken into account. More importantly, the effects of early treatment of low vision, squint, refractive error, and amblyopia in very young children need to be clarified.

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Death rates and causes of death in 284 consecutive patients with giant cell arteritis confirmed by biopsy

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Although giant cell arteritis affects the arteries,[1] the statistical survival rate in several studies has been shown to be the same as that of the general population.[2,3] These series have been comparatively small, however, and possibly not large enough to detect minor differences in survival rates.

Patients, methods, and results

All temporal artery biopsies performed in Göteborg over 10 years (1 January 1977-31 December 1986) were identified, and 284 subjects were diagnosed as having histologically verified giant cell arteritis. A follow up investigation of all patients was performed in December 1987. The causes of death were obtained from death certificates and the National Bureau of Statistics. The calculations of the expected number of deaths and comparison with the observed number have been described.[2]

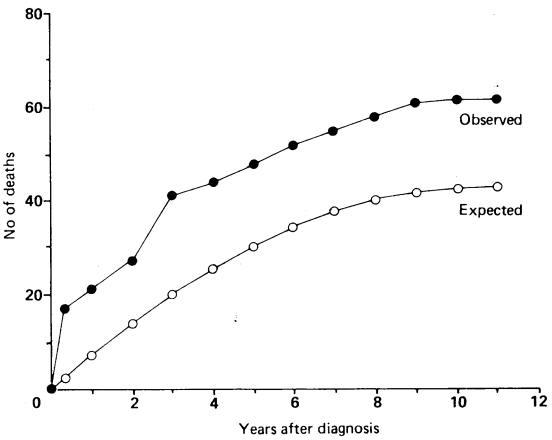
By December 1987, 82 of the 284 patients had died (64 women and 18 men). The expected number of deaths in an age and sex matched control group was 68·16, and the difference was not significant. The mean age at death was 79·5 years (59-90 years), and the mean time from diagnosis to death was 3·3 years (<0·5-108 months).

The observed numbers of deaths from vascular and malignant disorders at follow up were 62 and 9 respectively, compared with the expected numbers of 43 ($p<0·05$) and 12·9 (NS) (figure). One year after diagnosis the observed number of deaths from vascular disorder was 21 and the expected number 7·01 ($p<0·001$). Seventeen of these 21 subjects died within the first four months, eight of cerebrovascular disease, three from myocardial infarction, three from cardiac failure, two from rupture of a dissecting aneurysm of the aorta, and one from pulmonary embolism. From the second year after diagnosis to follow up, there was no significant difference between the observed and the expected number of deaths from vascular disorders.

Three of the 17 patients displayed symptoms typical of polymyalgia rheumatica, seven had only cranial symptoms (temporal arteritis), two suffered from both polymyalgia rheumatica and temporal arteritis, and five had only systemic symptoms. Their mean age at death was 76·1 years (61-89 years) and, in comparison with the total numbers of deaths, there was no sex difference.

All 17 patients had been treated with corticosteroids. In 13 of them, however, treatment had been insufficient to suppress disease activity as judged from clinical symptoms and the return of the erythrocyte sedimentation rate to normal. The cause was either too low a dose of corticosteroids, premature discontinuation of treatment, or simply too short a time (arbitrarily defined as less than one month) to suppress disease activity before death occurred.

Necropsy was performed in seven of the 17 patients.



Number of deaths from vascular disorders in patients with giant cell arteritis proved by biopsy compared with an age and sex matched control population

The microscopic findings showed widespread arteritic changes, affecting the coronary arteries in four patients, the aorta in five, and the cerebral arteries in six.

Comment

A fatal outcome in patients with giant cell arteritis has been poorly recognised, and previous reports have generally been case reports or necropsy studies.[4] The necropsy rate is low in many countries, and microscopic examinations of arteries in patients dying from vascular disorders are not routine, especially in elderly people. Hence, giant cell arteritis may be concealed among the cases currently diagnosed as ischaemic catastrophes due to arteriosclerosis.

There is a general consensus that all patients with giant cell arteritis should be treated with corticosteroids. As well as rapidly relieving clinical symptoms, such treatment may prevent ischaemic ophthalmic catastrophes.[5] Our findings indicate that corticosteroids may also prevent other ischaemic lesions.

Our results show that patients with giant cell arteritis run an increased risk of dying from vascular disorders in the initial phase of the disease. After four months, following the start of corticosteroid treatment, the risk equalled that of the general population.

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Treating malignant otitis with oral ciprofloxacin

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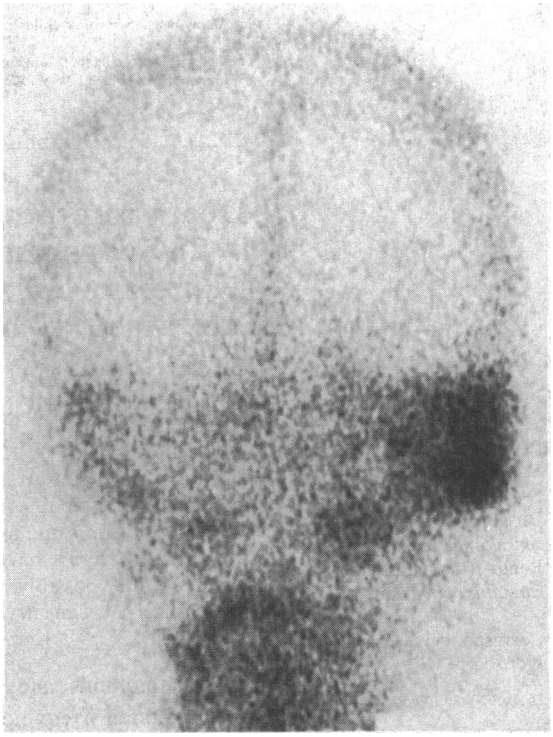
Malignant external otitis is caused by *Pseudomonas aeruginosa*.[1] It occurs mainly in poorly controlled diabetics[2] but also in people who are immunosuppressed, anaemic, or suffering from malnutrition. Meatal cellulitis and osteitis with extensive granulations spread to affect the base of the skull, the mastoid process, and the petrous apex. Infection of soft tissue causes progressive cranial nerve palsies. Secondary intracranial sepsis and contralateral disease occur occasionally. Traditionally, treatment consists of an aminoglycoside and a penicillin that is effective against pseudomonas given systemically for up to three months.[3] Poor control in patients who require insulin is common. Aggressive surgery (extensive mastoidectomy and decompression of the facial nerve) was recommended initially,[1,2] but a more conservative approach is now practised, operations being confined to meatal debridement and draining subperiosteal collections.[4]

Traditional treatment entails patients staying in hospital long term, which is costly, and repeated assay of drug concentrations to avoid toxicity in patients who are likely already to have impaired renal function. We report on two patients with malignant external otitis who were treated with oral ciprofloxacin.

Case reports

Case 1—A 61 year old man with diabetes controlled by diet was referred with a 24 day history of a painful, discharging right ear. Previous treatment with amoxycillin, flucloxacillin, and gentamicin given systemically had proved ineffective. Examination showed profuse meatal granulation. His facial nerve function was intact. A technetium-99m radioisotope scan (figure) showed increased uptake in the right mastoid. A computed tomogram showed soft tissue filling the external meatus, middle ear, and mastoid process and bony sequestration. A meatal swab grew *P aeruginosa* that was sensitive to all antibiotics that are effective against pseudomonas, including ciprofloxacin. Treatment was started with glibenclamide, co-proxamol, oral ciprofloxacin (750 mg twice daily), and meatal debridement. His diabetes became controlled by diet within two days. After seven days he was discharged taking ciprofloxacin and co-proxamol. After 21 days he was free of pain and had no meatal granulations, and a meatal swab yielded no growth. Treatment was stopped after nine weeks, and after five months he remained free of disease.



^{99m}Tc radioisotope scan (case 1) showing increased uptake in right mastoid process

Case 2—A 72 year old insulin dependent diabetic was referred with a 10 week history of otorrhoea of the left ear and a two week history of facial swelling on the left side. Oral amoxycillin had produced no improvement. Examination showed a fluctuant preauricular swelling, purulent otorrhoea, and extensive meatal granulations. His facial nerve function was intact. A ^{99m}Tc radioisotope scan showed increased uptake in the mastoid and preauricular region. A computed tomogram showed soft tissue filling the meatus and the air cells of the mastoid, a low attenuation mass deep to the masseter and temporalis muscles, and obliteration of the fat planes of the infratemporal fossa. A meatal swab grew *P aeruginosa*. The meatus was debrided and the preauricular swelling aspirated. Treatment was started