found in seven individuals.[3] Sarcoidosis may also complicate or mimic the clinical features of CVID.[4] However, we think it unlikely that sarcoidosis is present in this patient in view of the thoracic CT scan results, near normal serum angiotensin converting enzyme levels, and normal lymphocyte subset numbers.

It is probable that disturbed auto-immunity links CVID and MCP in this patient, and such an association has been suggested in a previous case report of a patient presenting with bilateral granulomatous anterior uveitis, optic disc swelling and multifocal areas of choroidal pallor.[5] Investigations led to the diagnosis of CVID, and the anterior uveitis responded to topical steroids. It is, however, not clear what happened to the optic disc and choroidal abnormalities. A further case report has linked CVID with uveitis following campylobacter enteritis,[6] though in this case the uveitis was not associated with peripheral chorio-retinal scars. Also, the uveitis was acute at onset, suggesting that it was predominantly anterior in location (iritis), and possibly secondary to an abnormal immune response following the enteritis itself (Reiters syndrome).[7]

Retinal vasculitis has also been linked with CVID in a case series of three children, all of whom had CVID and bilateral retinal vasculitis with optic nerve and macular oedema.[8] In two of these patients there was evidence of posterior uveitis as well. The authors point out that CD4+ cells of patients with CVID can produce normal amounts of interferon and may therefore be able to perpetuate an auto-immune response once initiated. In contrast to the above reports, in which the ophthalmic involvement occurred shortly after or even before the diagnosis of CVID, our patient's uveitis developed some 17 years after diagnosis, and throughout that time he had been receiving immunoglobulin treatment.

Patients with CVID are predisposed to a wide variety of illnesses that are thought to have an auto-immune aetiology. On the basis of this case report and others we suggest that MCP might be one such disease, and we would advocate prompt ophthalmic referral for any CVID patient experiencing blurred vision.

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R Gray, J Vodden and M Gompels

Taunton and Somerset NHS Trust
Musgrove Park
Taunton
Somerset TA1 5DA
UK

Correspondence: R Gray
Tel: +44 1823 342944
Fax: +44 1823 342943
E-mail: roger.gray@tst.nhs.uk

Sir,

Mucocoele of the maxillary sinus and the eye

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Mucocoeles are common, expansile cyst-like lesions affecting the paranasal sinuses.[1] The majority occur in the frontal sinus, followed by ethmoid sinus, maxillary sinus, and sphenoid sinus. They grow gradually, expand in size, and rarely produce bony destruction of the sinus walls,[2] leading to orbital and ocular involvement. We report such a rare case.

Case report

A 69-year-old woman with 1-month history of right-sided facial numbness and constant aching was referred by her GP to see a neurologist. While waiting for the appointment, she developed transient double vision and mild right ptosis. She did not sustain any facial injury affecting the sinuses in the past, but gave the history of chronic right maxillary sinusitis and Caldwell–Luc

operation 21 years previously. She received treatment for bronchial asthma, systemic hypertension, and Type II diabetes mellitus.

The neurologist who saw her 5 months later made an initial diagnosis of trigeminal neuralgia, and suspected that she probably sustained a transient ischaemic attack of the brain stem. Several investigations, including haematology, blood biochemistry, CT scan of the brain, and examination of the CSF, were within normal limits.

Several months later, there was recurrence of double vision and worsening of her symptoms. This time the neurologist noticed right ptosis, proptosis, and limited ocular movement, but found no other neurological abnormalities. She was admitted for an urgent CT scan of the orbit and paranasal sinuses. This showed an extensive mucocoele of the right maxillary sinus extending into the orbit with optic nerve displacement. At this point, the patient was referred to see an ENT surgeon. An urgent ophthalmologic consultation was also sought.

The ophthalmologist found that her right visual acuity was 6/36 (Snellen's), with no clinical signs of optic nerve compression. She had right infraorbital numbness, complete upper lid ptosis, nonaxial proptosis, and severe restriction of ocular movement. There were no signs of uveitis and the intraocular pressure was normal. The right fundus showed inferior indentation, macular choroidal folds, and a normal optic disc. The left eye was normal with an unaided visual acuity of 6/9. An ultrasound B scan of the right eye showed a large orbital mass indenting the inferior aspect of the eyeball (Figure 1c).

MRI of the orbit and the sinuses revealed complete opacification of the right maxillary antrum with an expanding mass in all directions. A bulge in the orbital floor resulted in the elevation of the globe and proptosis (Figure 1a, b). These findings clearly suggested the diagnosis of maxillary mucocoele and it was decided to remove the lesion surgically.

The operation was performed immediately under general anaesthesia by the ENT surgeons who drained the fluid and removed the mucocoele completely through a wide inferior antrostomy. An immediate correction of the proptosis was noted at the end of the operation.
The right eye on the first day postoperatively showed that there was an improvement in the visual acuity (6/9), complete resolution of diplopia, full restoration of ocular movement and ptosis, and disappearance of the macular choroidal folds and inferior global indentation. The aspirated serous fluid was found to be sterile and the biopsy report confirmed benign cystic inflammatory lesion consistent with mucocoele of the maxillary sinus.

Comment

Here is a patient with right-sided maxillary mucocoele, which expanded into the orbit producing facial pain, restricted ocular movement, nonaxial proptosis, deformity of the eyeball, and reduced vision. Some common causes producing these features are considered and the management of mucocoele discussed.

The common causes of unilateral facial pain in the elderly include trigeminal neuralgia, atypical facial pain, postherpetic neuralgia, dental causes, sinusitis, ocular or orbital inflammation, or temporal arteritis. Trigeminal neuralgia is characterised by paroxysmal attacks of severe, short, sharp pain affecting one or more divisions of the trigeminal nerve. It is often superimposed by constant ache and the attacks precipitated by touching a 'trigger spot'. Atypical facial pain is dull and diffuse. Other causes include pathologies involving ears or even cerebellopontine angle tumours.[3] It is unlikely that this patient suffered a transient ischaemic attack of the brain stem. This condition is characterised by vertigo, vomiting, dysarthria, ataxia, diplopia, and hemisensory loss with full recovery in 24 h.[4] The causes of unilateral proptosis with oculomotility problems include thyroid eye disease, orbital inflammatory disease, or orbital apex syndrome.

Mucocoeles are common expansile cyst-like lesions affecting paranasal sinuses. The majority occur in the frontal sinus (60%), followed by ethmoidal sinuses (30%), maxillary sinuses (10%), and rarely the sphenoidal sinuses.[5] The most common cause of mucocoele of the maxillary sinus is trauma: accidental or iatrogenic (Caldwell–Luc procedure).[6] It can also occur in non-Hodgkin's lymphoma.[7]

The diagnosis of mucocoele is made on the basis of symptoms, imaging and surgical exploration and histological confirmation. The most consistent symptom is dull maxillary facial pain. Other symptoms include swelling and/or numbness of the cheek, poorly localised pain or tenderness, nasal obstruction, visual impairment, diplopia, and dental problems.[8] High-resolution CT scan will show homogenous lesions, which are isodense with brain and no contrast enhancement, unless infected. There are clearcut margins of bone erosions occurring in the sinus walls. In malignancy, the mass is likely to be irregular in shape, with erosion and destruction of sinus walls. In sinusitis or retention cyst, there is no bone destruction.[8]

The treatment of mucocoele is surgical removal by the ENT surgeons.[9] Some paranasal sinus mucocoeles can encroach the orbit and cause ocular compression. This case clearly demonstrates how relevant it is to refer all patients with ptosis and oculomotility problems to an ophthalmologist very early during their presentation.

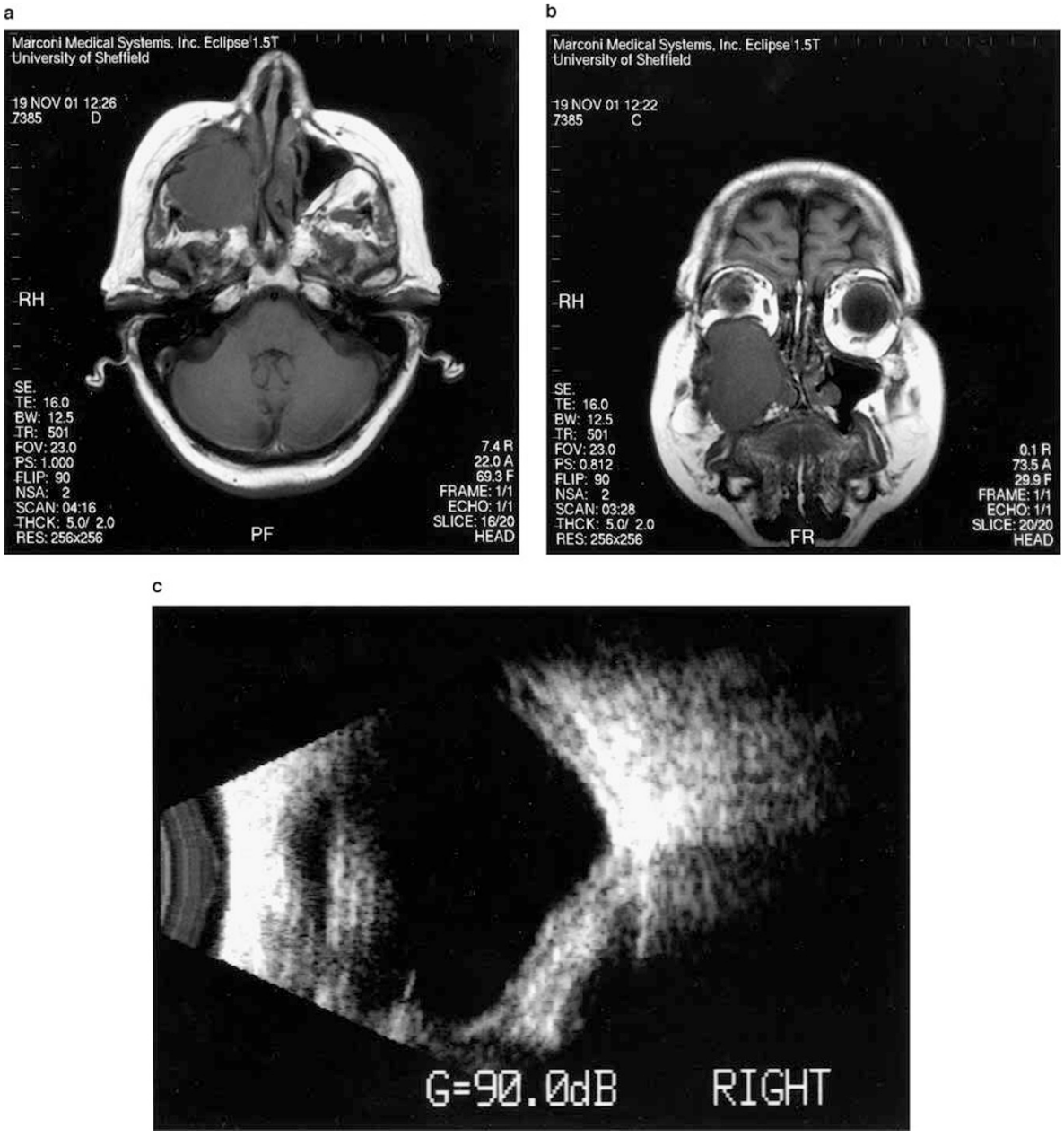

Figure 1 (a) and (b): MRI show complete opacification of right maxillary sinus with expanding mass in all directions. (c) Ultrasound B scan with large orbital mass indenting the inferior aspect of the eyeball.

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S Raman

University Department of Ophthalmology and Orthoptics
Level A Royal Hallamshire Hospital
Sheffield S10 2RJ, UK

Correspondence: S Raman
69 The Cornfields
Hebburn NE31 1YJ, UK
Tel: +44 191 428 1083
Fax: +41 1723 342 477
E-mail: somanraman@doctors.org.uk

Sir,

Aeromonas sobria corneal ulcer

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A case of perforated corneal ulcer secondary to *Aeromonas sobria* in a man with no pre-existing ocular pathology is presented.

Case report

A 58-year-old retired man presented to the ophthalmic casualty complaining of a sore right eye for the past week. He had no significant past medical or ocular history. On examination, he had a best corrected visual acuity of hand movements close to face with accurate projection in the right eye and 6/6 in his left eye. The left eye was normal. The right eye had circumcorneal injection with a 3.5×3.5 mm corneal ulceration. There was marked oedema and infiltration of the surrounding cornea. A 2.4 mm hypopyon was also recorded. A provisional diagnosis of a bacterial corneal ulcer was made and a corneal scrape performed for Gram's staining, bacterial/fungal culture and sensitivity. Antibiotic therapy using a combination of topical gentamycin forte (14 mg/ml), cefuroxime (50 mg/ml) and atropine (1%) eye drops was initiated. Gram negative rods were detected on Gram's staining and the topical treatment was reported to be adequate. Daily review of the patient over the next 5 days showed a gradual reduction of the hypopyon. The corneal ulcer, however, progressed creating a deep crater and a resultant decemetocele, which subsequently perforated on day 6. The perforation was sealed using histoacryl glue (Figure 1). A bandage lens was placed on the cornea following glue application. Culture and sensitivity along with biochemical testing demonstrated the organism to be an oxidase positive Gram negative rod, which was identified as *Aeromonas sobria* using API zone, bioMercurix sa and profile number 1050004. The organism was sensitive to cefatazidime, ciprofloxacillin, gentamycin and piperacillin. Subsequent follow-up for over 4 weeks revealed a gradual improvement in the corneal ulcer. The medication was gradually tapered off over that period. The glue, however, remained *in situ* for 2 months and fell off spontaneously leaving a vascularised cornea with an anterior synechiae and a complicated cataract. The patient is currently awaiting a triple procedure.

Discussion

Aeromonas sobria is a motile, polarly flagellated and oxidase positive Gram negative bacteria of family Pseudomonaceae.[1] Since its recognition as a human pathogen, it has been implicated in a number of infective pathologies including intestinal infections, peritonitis, bacteraemia, sepsis, osteomyelitis and wound infections[1] including corneal ulcers[2,3] and endophthalmitis.[4] Keratitis resulting from *Aeromonas sobria* infection is usually posttraumatic with a history of contamination with water or soil.[2–4] Although clinical presentation is non-specific, a hyperaemic purulent exudation and corneal ulceration associated with hypopyon is the usual presentation.[2] The extent of the lesion depends on the virulence of the species.[2] Frequent topical antibiotics are

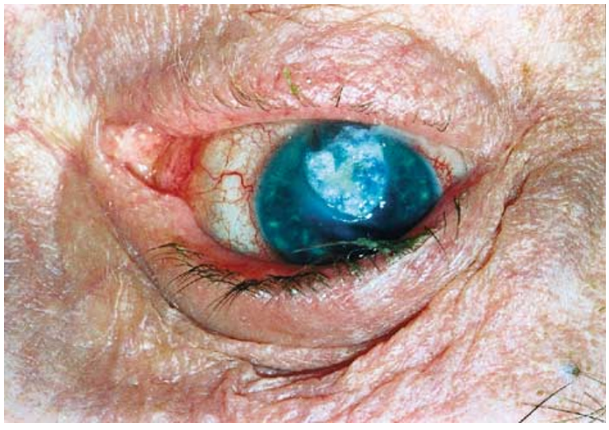

Figure 1 Anterior segment photograph showing the perforated ulcer being managed with tissue glue.