patients after cataract surgery.^{3–5} To the best of our knowledge, our patient is the first reported post-traumatic case and is also the youngest patient reported.

In 7 (88%) of the 8 reported cases, endophthalmitis developed within the first 2 weeks post-operatively. Five of the 8 cases (63%) developed recurrence after initial response to intravitreal antibiotics, as the strain was not sensitive or partially sensitive. Subsequent pars plana vitrectomy and repeated intravitreal antibiotics were required to control the endophthalmitis. In 2 of the 3 cases without recurrence, intraocular lens removal and vitrectomy were performed as the primary procedure to treat the endophthalmitis. Five of the 7 cases (71%) with post-cataract surgery endophthalmitis had a final visual acuity of 6/18 or better. The reasons for the poor vision in the other 2 cases were persistent cystoid macular oedema⁴ and tractional retinal detachment.⁵ In our patient, the delayed onset of endophthalmitis and good visual outcome is probably attributable to the low virulence of the bacteria, early pars plana vitrectomy with appropriate antibiotics, and the young healthy status of the patient.

A study on contamination of phacoemulsification and vitrectomy machines has shown that machines with internal vacuum control manifolds which were not sterilised routinely had bacterial contamination in the aspiration fluid samples. S. maltophilia was the most commonly isolated species in this study. Contamination of the aspiration fluid has the potential of causing endophthalmitis due to S. maltophilia, and as more cataract surgery is performed using automated phacoemulsification machines, one might expect the possibility of an increasing incidence of S. maltophilia endophthalmitis.

Endophthalmitis due to *S. maltophilia* is potentially difficult to eradicate due to multiple drug resistance.¹ Treatment of *S. maltophilia* may require either ceftazidime or amikacin since resistance to either one of the drugs has been reported in cases of endophthalmitis.⁴ However, when appropriate treatment is instituted, endophthalmitis due to *S. maltophilia* usually allows good visual outcome.

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Sir,

Maxillary antral adenoid cystic carcinoma: an unusual presentation

Maxillary adenoid cystic carcinoma is a rare and aggressive tumour, and approaches to treatment vary, partly because of its low prevalence. This tumour rarely presents to the ophthalmologist, but when this does occur the lesion is likely to be advanced, although the ophthalmic symptoms and signs may be subtle. A multidisciplinary approach to management is required, specifically involving ENT, radiotherapist, oculodental prosthetic technician, and the ophthalmologist.

Case report

A 67-year-old man presented to the eye casualty with a 4 week history of a red, watery left eye and 1 week of intermittent diplopia on downgaze. Friends had noticed that the eyes were asymmetrical. There were no symptoms of thyroid dysfunction and no pain or impaired vision. There was a history of anisometropic ambylopia in the left eye which had been treated with occlusion therapy in childhood but there was no history of squint. The patient was taking low-dose aspirin daily following a myocardial infarction in the 1970s.

On examination, visual acuities were 6/12 in the right eye and 6/18 in the left, improving on refraction to 6/6 and 6/12, respectively. There was 4 mm of left proptosis compared with the right, and the globe was displaced

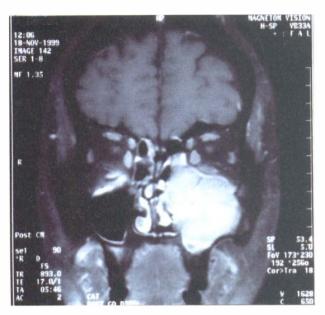


Fig. 1. Coronal MRI scan of the orbits and maxillary sinuses showing a mass filling the left maxillary sinus and eroding through the left orbital floor to displace the globe.

superiorly and temporally. A firm mass was palpable at the inferior orbital margin. Examination of extraocular movements showed limited depression of the left eye with diplopia on downgaze. There were no signs of compressive optic neuropathy (pupil reactions, colour vision, near vision and visual fields were normal). The anterior and posterior segments, as well as intraocular pressures, were normal.

Investigations included an MRI scan of the orbits, sinuses and brain. This revealed an erosive mass of the left maxillary antrum extending into the orbit (Fig. 1). A full blood count, urea and electrolytes and thyroid function tests were all normal. The patient was referred for an ENT opinion and an endonasal biopsy was taken. This showed an adenoid cystic carcinoma displaying a solid growth pattern with cribriform islands and a desmoplastic reaction. There was no perineural invasion (Fig. 2). A left maxillectomy was performed under frozen section guidance, and was followed by radiotherapy. An obturator was fitted to fill the post-operative cavity (Fig. 2), and to allow normal speech and ingestion of food and drink.

Following surgery the extraocular movements were normal in both eyes, there was no diplopia and visual acuities were 6/6 in the right eye and 6/9 in the left.

Comment

Adenoid cystic carcinoma of the maxillary antrum is uncommon, with tertiary referral centres seeing an average of one case per year. This tumour often presents late and has serious prognostic implications. Presentation is usually with a mass or epistaxis, ¹ nasopharyngeal obstruction, pain and rhinorrhoea. The authors have not encountered any reports of adenoid cystic carcinoma of the maxillary sinus presenting with ophthalmic symptoms, and we believe that this is the first such case to be reported.

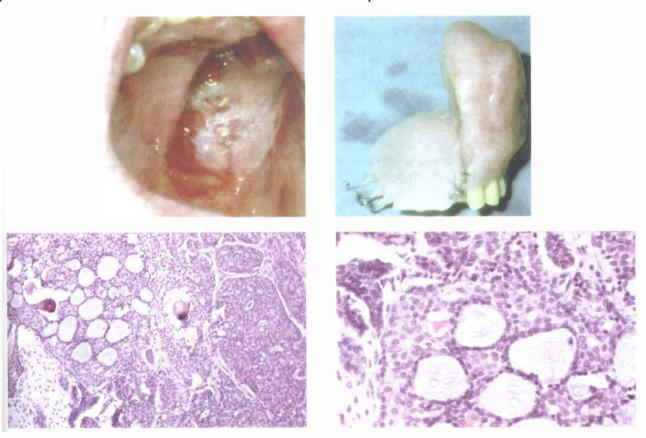


Fig. 2. The post-operative cavity is shown from the buccal view (above left); the obturator (above right) is designed to fill the cavity. Photomicrographs (below left, $\times 100$; below right, $\times 400$) of the biopsy specimen show adenoid cystic carcinoma having a solid growth pattern with cribriform islands and a desmoplastic reaction. No perineural invasion is evident.

The differential diagnosis in this case included a primary adenoid cystic carcinoma of the lacrimal gland as well as thyroid eye disease. Although thyroid eye disease can cause both axial and non-axial proptosis, the latter implies extraconal pathology and alerted the clinician to the possibility of a space-occupying lesion.

This type of carcinoma has a high recurrence rate and a poor prognosis for survival. The most significant prognostic factors are perineural invasion and initial mode of treatment.² Treatment involves surgery and adjunctive radiotherapy,²⁻⁴ but debate exists regarding how best to combine these modalities. Post-operative radiotherapy is reported to give a 10 year survival of 37.6% and 10 year cancer-free survival of 13.6%.² However, pre- and post-operative radiotherapy is claimed to reduce the occurrence of distant metastases and intracranial tumour extension, as well as offering a 10 year cancer-free survival of 59.3%.³ The number of patients with adenoid cystic carcinoma of the maxillary antrum is too low to allow statistical analysis of data on treatment outcomes.

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Sir,

Recurrent sixth nerve palsy following Measles Mumps Rubella vaccination

A 13-month-old girl had a spontaneous and rapid-onset left sixth nerve palsy following immunisation to measles, mumps and rubella. This resolved completely over 8 weeks and then recurred 15 weeks after initial

presentation. Resolution was again complete. All investigations were normal. Serum antibody titres to the measles and mumps virus demonstrated immunisation.

Case report

A 13-month-old month old girl presented with her parents to eye casualty with a 1 day history of left squint. She had mild left face turn, limitation of left abduction and a diagnosis of left sixth nerve palsy was made. Her ophthalmic and neurological examination were otherwise normal, as was refraction. One week previously she had been inoculated with the Measles, Mumps and Rubella (MMR) vaccination with no ill effect. Normal investigations included: an urgent CT scan, C-reactive protein, serum anti-nuclear and anti-mitochondrial antibodies, blood culture and serum rotavirus. Faeces were negative to parasites and *C. difficile*. Full blood count showed a mild lymphocytosis and there were positive serum titres for the measles and mumps virus.

The patient was followed up closely by the paediatric neurologist and ophthalmologist. The squint resolved completely and spontaneously over 8 weeks. Fifteen weeks after the initial presentation the same squint returned. Investigations were again normal and the palsy again resolved completely and spontaneously. MRI scan was normal showing no cause for the recurrent nerve palsy.

Comments

The MMR vaccination is made from live attenuated viruses and has been administered for over 25 years. Neurological complications are rare, approximately one child per million, and onset is usually within 15 days. Of most concern is encephalopathy and while a link between the immunisation and cranial neuropathies has been suggested, it has never been proven.¹

A sixth nerve palsy following the MMR vaccination has been reported once before by Werner *et al.*² with similar onset, course and resolution. It also recurred several months later, again with full resolution. Investigations were all normal. The same paper reported a sixth nerve palsy following diphtheria, pertussis and tetanus immunisation.² Chan *et al.*³ have reported a third nerve palsy following measles immunisation. It resolved spontaneously and all investigations were again normal.

Robertson *et al.*⁴ have stated that 49% of non-traumatic sixth nerve palsies in this age group are secondary to tumours. Werner *et al.*¹ commented that most of those cases have other signs such as papilloedema and the incidence of tumours with true isolated sixth nerve palsies is likely to be less. There is widespread agreement that these children should be followed up regularly and fully investigated to eliminate the other causes of this condition.