1200 mg magnesium carbonate, 600 mg sodium bicarbonate daily as well as 2280 mg deglycyrrhizinized liquorice. In addition, 3.5 pints of milk daily would provide him with 2380 mg calcium, 230 mg magnesium, but only 1.15 μg (46 iu) calciferol daily[1].

The traditional explanation for hypercalcaemia in the milk-alkali syndrome is that absorbable alkali causes a decrease in renal calcium excretion in the face of a high calcium intake. Secondly, calcium excess increases renal tubular bicarbonate reabsorption both directly and indirectly through salt and water depletion, thus maintaining the alkalosis[2].

Our data show that other factors may contribute to the metabolic state. There was hypermagnesaemia due to renal failure as well as excessive intake. Intact PTH was detectable in serum, evidence that the normal suppression of the parathyroid glands by a high circulating calcium concentration was impaired. Lastly, serum 25 OH vitamin D was elevated despite a normal dietary intake. It is not clear how mineral and alkali intake can influence vitamin D status except in calcium depletion, where calcium intake has been shown to regulate 25 OH vitamin D metabolism[3].

Milk-alkali syndrome is rare but should be considered when hypercalcaemia presents as an emergency in order to prevent unnecessary treatment with hypocalcaemic agents. It should be remembered that Caved-S contains both alkali and magnesium in absorbable forms.

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Acute angle closure glaucoma occurring after nebulized bronchodilator treatment with ipratropium bromide and salbutamol

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Keywords: acute close angle glaucoma; ipratropium bromide; salbutamol; nebulized bronchodilator

Two elderly patients without previous ophthalmic problems developed acute angle closure glaucoma whilst on treatment with nebulized ipratropium bromide (Atrovent) and salbutamol.

Case reports

Case 1

A 64-year-old woman with longstanding chronic obstructive airways disease developed severe dyspnoea secondary to basal pneumonia. She was treated with nebulized salbutamol (1 mg four times a day) and ipratropium (250 μg four times a day) via an Acorn face mask, as well as with systemic cefuroxime, theophyllin and prednisolone.

Three days later, when no longer dyspnoeic, she complained of blurred vision and a temporal headache. Apart from wearing a hypermetropic spectacle correction, she denied any previous ophthalmic problems. The visual acuities were reduced to counting fingers in the right eye and 6/36 in the left eye. The anterior chambers were shallow and the corneae oedematous. Her intraocular pressures were over 40 mmHg. Acute angle closure glaucoma was diagnosed and treated with topical pilocarpine and oral acetazolamide. Gonioscopy confirmed very narrow anterior chamber drainage angles. Pulsed neodymium YAG laser iridotomies were performed and hypotensive treatment discontinued. Since then the intraocular pressure has remained at 14 mmHg and the visual acuity at 6/9 in both eyes.

Case 2

A 70-year-old woman with a long history of asthma developed respiratory failure. This responded to a combination of oxygen, systemic amoxycillin and hydrocortisone as well as nebulized salbutamol and ipratropium via an Intertech Inspiron face mask.

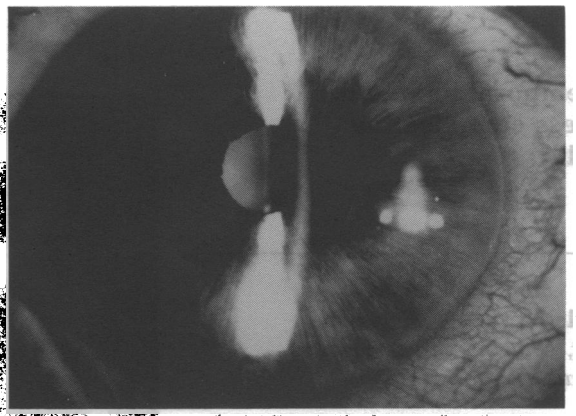

Figure 1. Slit lamp photograph of right eye of Case 2 showing a very shallow anterior chamber

Three days later she complained of right ocular pain and blurred vision. Her visual acuity was 6/6 in each eye with hypermetropic correction. The anterior chambers were shallow (Figure 1) and the intraocular pressures were 30 mmHg in the right eye and 12 mmHg in the left eye. Right angle closure glaucoma was diagnosed and treated with topical pilocarpine and acetazolamide. Gonioscopy confirmed the very narrow anterior chamber drainage angles. Bilateral pulsed neodymium YAG iridotomies were performed and the hypotensive treatment was then discontinued. The intraocular pressures have remained at 12 mmHg in both eyes since then.

Discussion

Acute angle closure glaucoma is an important cause of visual loss in the elderly. It is caused by closure of the anterior chamber drainage angles by contact between the peripheral iris and the posterior surface of the cornea. Eyes with narrow drainage angles are at risk of developing acute glaucoma, either spontaneously or after drug-induced mydriasis. Hypermetropic eyes tend to have narrow anterior chamber drainage angles and are, therefore, at increased risk[1].

Antimuscarinic drugs, such as ipratropium bromide are competitive antagonists of acetylcholine. In the eye they cause pupil dilation leading to closure of the anterior chamber drainage angles. Helprin and Clarke[2] reported

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two cases of fixed dilated pupils after nebulized ipratropium. There are at least two reports of angle closure glaucoma following treatment with nebulized ipratropium alone and one report of acute glaucoma after treatment with nebulized ipratropium and salbutamol[3,4].

The effects of salbutamol on the eye are an increase in aqueous humour production of about 35%[6,7]. Aqueous outflow is increased by an even higher amount. The net effect is to lower intraocular pressure. Kalra and Bone[8] showed that ipratropium bromide and salbutamol in aerosol form can be absorbed through the cornea after escaping from the face mask. Their cases did not develop angle closure glaucoma because the patients received only one dose. However, the drugs are normally used several times a day, so an accumulative effect is likely.

We suggest the following sequence of events. Ipratropium escapes from the face mask, diffuses through the cornea producing pupil dilatation and, in eyes with susceptible angles, angle closure. Salbutamol is absorbed in a similar way and increases aqueous humour production by its effect on the ciliary body, resulting in increased aqueous flow. Pushing the eyes forward could cause angle closure even without pupil dilatation[5].

Physicians and ophthalmologists should be aware that treatment with nebulized ipratropium and salbutamol can cause acute glaucoma. Patients may not complain when in severe respiratory distress. However, symptoms of blurred vision or ocular pain in patients on these drugs should, therefore, be investigated.

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Simultaneous temporal lobe and cerebellar abscess complicating acute otitis media

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Keywords: intracerebral abscess; bilateral; acute otitis media

Intracerebral abscesses are a recognized complication of otitis media, usually chronic suppurative otitis media with cholesteatoma[1]. An intracerebral abscess is a localized suppurative process occurring within the brain substance. It is a relatively rare condition, with an incidence in Great Britain of three or four cases per million per year[3]. When arising from a focal suppurative process such as an otitis media, it is invariably as a single abscess. Infection may spread through congenital or traumatic defects into the extradural space, or via structures such as the cochlea, labyrinth or via communicating veins. Multiple cerebral abscesses are usually haematogenous in origin, and are more common in the immunosuppressed patient[3].

We present a case which presented as an acute otitis media that was complicated by simultaneous cerebellar and temporal lobe abscesses. This is a rare occurrence, and raises some interesting points with respect to aetiology, presentation and management of these conditions.

Case report

A 21-year-old Caucasian woman presented to the ENT department at Charing Cross Hospital, London, with a one week history of left otalgia, variable hearing loss, and paroxysmal vertigo. A diagnosis of otitis media with superimposed otitis externa was made and topical antibiotic treatment was commended. The condition did not resolve spontaneously, and it was necessary to admit her for 3 days of intravenous antibiotic therapy. Following this, a reasonable improvement occurred, until she was seen again one week later with a left-sided headache, persistent nausea and unsteadiness of gait. On examination there was bulging of the left postero-superior meatal wall, with left-sided cerebellar signs.

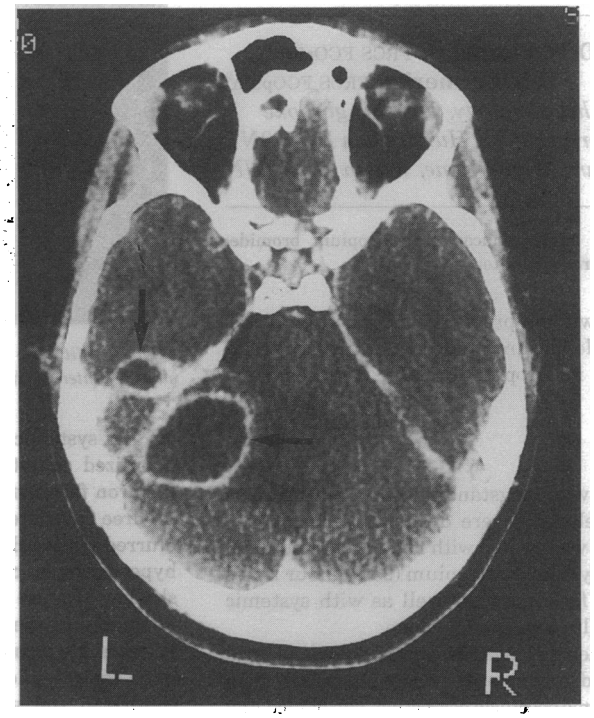

Figure 1. Both abscesses can be clearly seen as ring enhancing masses on the left side

Urgent computerized tomography (CT) scan of the head with contrast enhancement showed an abscess in the left lateral lobe of the cerebellum, and an abscess in the left temporal lobe (Figure 1). Following initial resuscitation she underwent a left lateral posterior fossa craniectomy, and 7 ml of grey pus was obtained on open drainage of the cerebellar abscess, with

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