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View from Within: Radiology in Focus

Pneumoparotitis

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Abstract
Pneumoparotitis is a rare cause of parotid enlargement. It is due to a reflux of air through Stensen's duct into the acini of the parotid gland with subsequent dilatation. We report a case which followed a long history of autoinflation of the middle ears by the Valsalva manoeuvre. The plain radiographic, sialographic and ultrasound findings are presented.

Introduction

Pneumoparotitis describes the presence of air within the duct system and/or parenchyma of the parotid gland. The only clear aetiological factor is that of a raised intraoral pressure forcing air into Stensen's duct; in particular blowing forcefully against resistance with full cheeks rather than contracted cheeks. It occurs in glass-blowers (Wizgall, 1932), trumpet players, and balloon blowers. It may also occur in teenagers with behaviour problems who deliberately self-induce it, and in this context has even been used as a means of controlling angry parents! (Rupp, 1963).

Case report

A 36-year-old man presented to the ENT Department with bilateral parotid swelling which had been occurring intermittently for nine years. This had previously been diagnosed on several occasions as mumps. There was no history of occupational predisposition to raised intraoral pressure but he did admit to frequently performing a Valsalva manoeuvre with an open glottis and nose occluded in an attempt to 'clear the ears'. He hoped that this would improve his hearing which he felt had deteriorated since working in a mobile disco several years previously. Examination revealed bilateral parotid swellings with crepitus. Bilateral sialograms and an ultrasound of parotids were requested. The plain films showed air within the parotid glands (Fig. 1). At sialography, air was expressed from the ducts prior to cannulation. Following contrast injection, sialectatic air-containing ducts were demonstrated (Fig. 2). There was no evidence of any calculi. Ultrasound showed multiple small hyperechoic areas bilaterally (Fig. 3a) corresponding to air within the ducts; a normal parotid ultrasound is shown for comparison. (Fig. 3b).

A pure tone audiogram revealed a symmetrical sensorineural

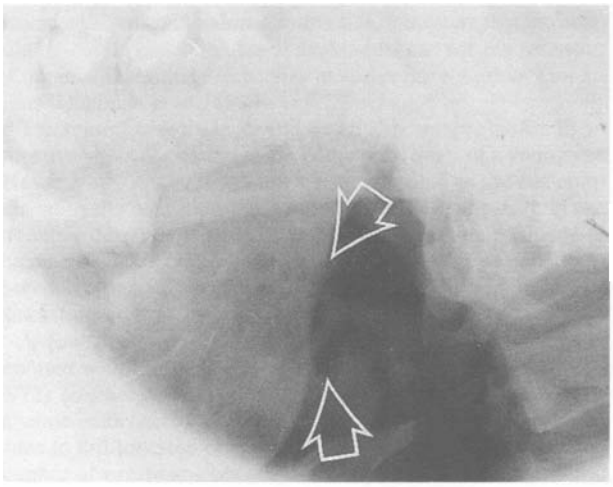

Fig. 1
Oblique radiograph of the right parotid gland showing air within dilated tubular structures (arrows).

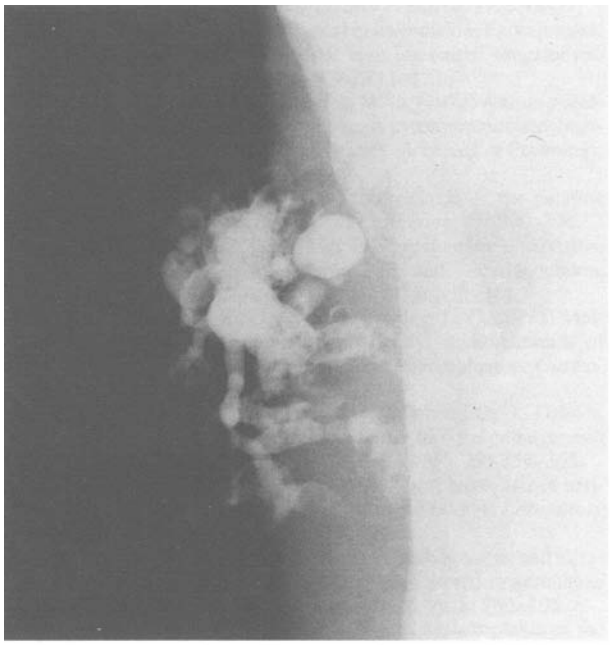

Fig. 2
Postero-anterior view of right parotid after injection of contrast. Contrast and air within multiple dilated ducts. The appearance on the left side was identical.

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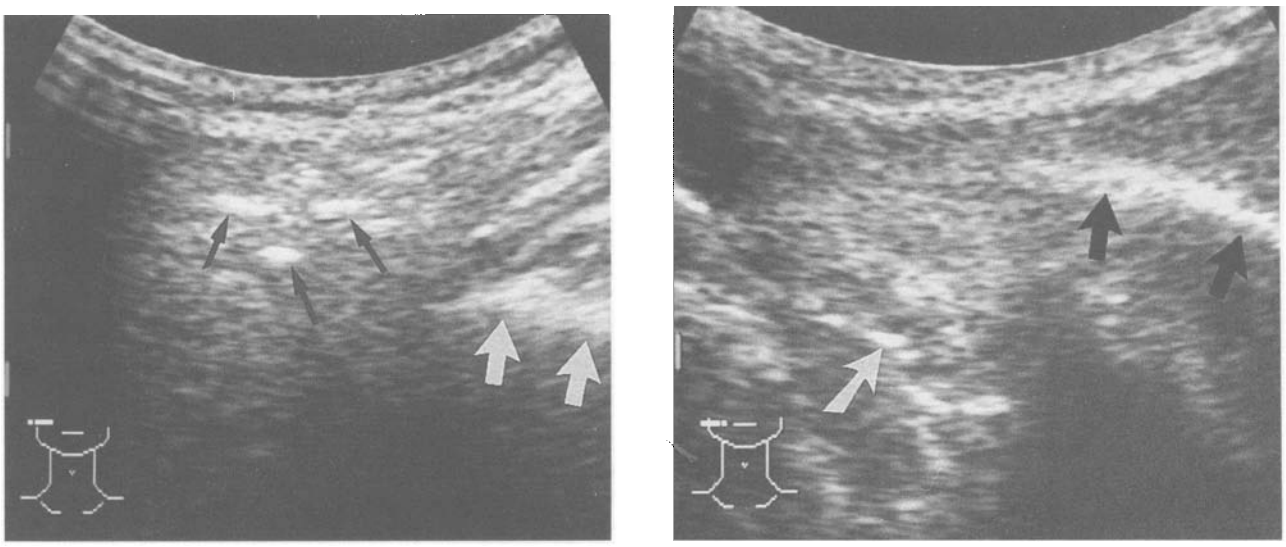

(a) (b)

FIG. 3

(a) Tranverse scan of right parotid. The black arrows point to echogenic areas within the substance of the gland. the white arrows point to the ramus of the mandible.

(b) Tranverse scan through a normal parotid for comparison. Black arrows point to the ramus of the mandible. The white arrow points to the internal carotid artery deep to the gland.

hearing loss above 2000 Hz., presumably the reason for his inappropriate habitual Eustachian tube auto-inflation.

The patient was advised to desist from forcefully puffing out his cheeks and subsequently his parotid swellings improved and the discomfort resolved.

Discussion

Raised intraoral pressure is essential for the production of pneumoparotitis. Normal respiratory intraoral pressure does not rise above 3 mmHg. In glass-blowers and trumpet players, however, it may rise as high as 150 mmHg (Rysenaer *et al.*, 1963). It is under these conditions that air may be insufflated to produce expansion and sometimes rupture of the parotid acini. Occasionally air may spread beyond the parotid into the subcutaneous tissues of the face, neck and mediastinum (Greisen, 1968; Calcaterra and Lowe, 1973).

The plain films and sialograms demonstrate the diagnostic features of pneumoparotitis. In a previous case report of a patient with pneumoparotitis (Telfer and Irvine, 1989), the ultrasound was said to be normal. Iko (1984) describes a case of Klebsiella infection of the parotid where C.T. demonstrated pockets of air within the gland and an ultrasound showed some hyperechoic areas which he considered might be caused by these. In the patient we describe, the multiple echobright areas are doubtless caused by intraductal air, an appearance analogous to that seen with air within the biliary tree.

Yoel *et al.* (1970) found that glass-blowers with pneumoparotitis improved markedly on changing occupation. If this fails to produce improvement there is the option of surgical treatment which Telfer and Irvine (1989) have employed to good effect in a patient with pneumoparotitis. This operation is modelled on the procedure orignally described for, and used successfully in, the treatment of drooling (Wilkie and Brodie, 1977). It consists of posterior transposition of the parotid duct using a buried mucosal flap based on a periductal pedicle so that the opening of Stensen's duct comes to lie at the anterior pillar of the fauces. Other treatments that have been attempted or postulated include excision of the duct and much of the gland, infiltration of the peri-arterial sympathetic nerves at the level of the superficial temporal artery with local anaesthetic, and even clamping of the duct (Greison, 1968).

This patient's habit of auto-inflating his Eustachian tubes in an attempt to improve his damaged hearing led to bilateral pneumoparotitis. The diagnosis was made by the presence of crepitus on palpation and elegantly confirmed on sialography and ultrasound. Explanation and counselling alone were all that was required to produce symptomatic improvement.

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