and nifedipine will not reduce after-load. Nevertheless, the negative inotropic effect may precipitate pulmonary oedema if the left ventricular end diastolic pressure is raised. Nifedipine should be avoided in the presence of a fixed obstruction to left ventricular ejection and raised ventricular diastolic pressures.

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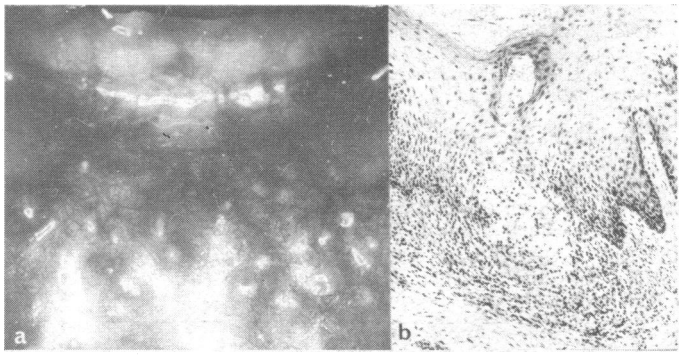
Department of Cardiology, Wentworth Hospital, P B Jacobs, Natal 4026, South Africa
DAVID J GILLMER, MRCP, physician and lecturer
PETER KARK, MB, registrar

Major and minor salivary gland swelling in Mycoplasma pneumoniae infection

Mycoplasma pneumoniae is a common respiratory pathogen predominantly associated with pneumonia or an influenza-like illness.[1] *M pneumoniae* may also cause a range of disorders including exanthemata[2] and mucocutaneous lesions, especially erythema multiforme.[3] Salivary gland swelling, however, has not been reported in association with *M pneumoniae* infection. We report a case of *M pneumoniae* infection in which the parotid, submandibular, sublingual, labial, and palatal salivary glands were all affected.

Case report

A 19-year-old woman was admitted to hospital in July 1979 with a history of a productive cough for 10 days unresponsive to ampicillin and enlargement of her parotid, submandibular, and sublingual salivary glands for two days. She had pulmonary consolidation in the left lower zone but no fever. The labial mucosa was ulcerated and many of the minor glands of both upper and lower lips and the soft palate were enlarged (fig (*a*)). There



Mycoplasma pneumoniae infection: (*a*) enlargement of multiple labial minor salivary glands; (*b*) section of labial salivary gland showing mucus extravasation cyst (stained with haematoxylin and eosin; original magnification ×200).

was also bilateral enlargement of all major salivary glands. Her total white cell count was 10×10^9/l (10 000/mm^3) with a normal differential count. The complement-fixing antibody titre to *M pneumoniae* rose from 1:16 to 1:1024 during her admission. The pneumonia and major salivary gland swellings settled with erythromycin treatment over nine days but she developed erythema nodosum on her thighs and legs. The minor salivary gland swellings persisted for a further two months before resolving. Labial biopsy showed a prominent periductal lymphocytic infiltrate with evidence of damage to the ductal epithelium, and considerable disruption of acinar architecture with mucus spillage into the connective tissue (fig (*b*)).

Comment

The nature of the infection in this patient, suggested by the clinical features of persistent cough and erythema nodosum, was confirmed by finding a rise of over fourfold in the serum complement fixation titre to *M pneumoniae*. The initial low antibody titre indicated that an anamnestic response was unlikely. A most unusual feature was the involvement of multiple salivary glands. The swelling of multiple intraoral (minor) salivary glands is unique and appeared to be due to the escape of salivary mucus from the duct system to form multiple small mucus extravasation cysts, or mucoceles. The swellings of the major salivary glands were clinically indistinguishable from mumps, which may be due to a number of different organisms[4] but has not been reported in mycoplasma infection. Although mycoplasmas are found in the normal oropharynx they have not been implicated in the more common oral diseases such as recurrent ulceration[5] or in any salivary gland disorder.

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University Department of Oral Medicine and Pathology, Glasgow Dental Hospital and School, Glasgow G2 3JZ
W WRAY, MB, FDS, lecturer
C SCULLY, PHD, FDS, lecturer
J RENNIE, PHD, FDS, lecturer
D K MASON, MD, FDS, professor

Department of Infectious Diseases, Belvidere Hospital, Glasgow G31 4PG
W C LOVE, MB, FRCP, honorary clinical lecturer

Hyperglycaemic hyperosmolar non-ketotic diabetic coma presenting as severe dysphagia

Dysfunction of oesophageal motility and of the lower oesophageal sphincter has been reported in patients with diabetes.[1 2] These abnormalities are presumed to be due to autonomic neuropathy and may cause minor symptoms such as heartburn. I have recently seen two patients, previously without gastrointestinal symptoms, whose presenting complaint was severe dysphagia, and both were suffering from non-ketotic diabetic coma.

Case reports

The first patient was a 58-year-old woman with maturity-onset diabetes of 11 years' duration, known ischaemic heart disease, and dependent ankle oedema. Her treatment was chlorpropamide 375 mg daily with a 150-g carbohydrate diet and oxprenolol 160 mg, perhexiline 100 mg, and amiloride 5 mg-hydrochlorothiazide 50 mg twice daily. For two weeks before admission she complained of progressive dysphagia for solids (she had had no previous gastrointestinal problems) associated with a weight loss of 6 kg. Before her emergency admission, precipitated by drowsiness, she could hardly swallow liquids. On admission she was extremely dehydrated but normotensive. Her blood urea concentration was 105 mmol/l (630 mg/100 ml) and her plasma electrolytes were sodium 108 mmol (mEq)/l, potassium 5·3 mmol (mEq)/l, chloride 60 mmol (mEq)/l, bicarbonate 22 mmol (mEq)/l. Her blood glucose was 64 mmol/l (1152 mg/100 ml) and her urine (Clinitest) contained over 2% sugar but no ketones. The calculated plasma osmolality[3] was 385 mmol/l. After treatment with intravenous fluids and insulin her blood urea and