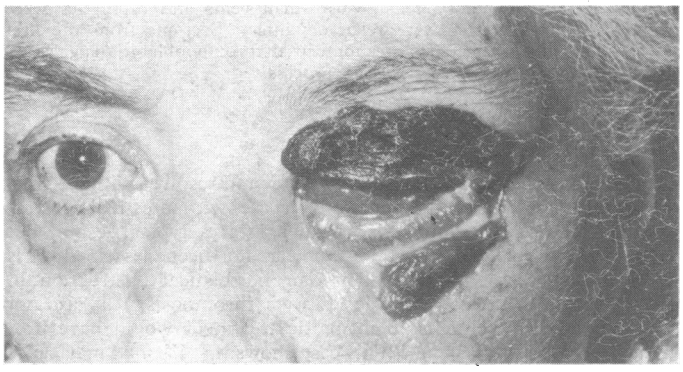
and discharging, but deep necrosis did not occur. There was much oedema in the right cheek extending to the neck. A large black scab formed over the upper lid while the lower lid became ulcerated and severely damaged, needing reconstruction.

(2) A 71-year-old woman, previously well, was admitted with severe cellulitis of the left face and left eyelids. A pure, heavy growth of *S pyogenes* was obtained from an eye swab and was present five days later. She developed severe left orbital cellulitis despite benzylpenicillin 20 megaunits daily. A pansystolic murmur of mitral incompetence occurred over the next week, despite an absence of growth in blood cultures, and she was considered to have acute streptococcal endocarditis. She developed a similar black scab on the upper lid with destruction of both upper and lower lid tissues and cicatrical ectropion (figure) requiring reconstruction.



Left orbital necrotising fasciitis due to *Streptococcus pyogenes*.

(3) A 41-year-old lorry driver was admitted febrile with an inflamed left elbow. He had no history of injury. He was treated with cephradine intravenously but within three days had developed blistering cellulitis, spreading up his arm, from which no bacteria were grown. He was treated additionally with erythromycin intravenously, but remained febrile. Eighteen days after admission a 2-in (5-cm) incision was made over his elbow to release 200 ml of pus. This yielded no bacterial growth. The wound was irrigated with Savlon (cetrimide and chlorhexidine). He remained febrile with necrotic material discharging from his wound. At a second operation after 16 days a vast loculated cavity was found, in which four corrugated drains were placed. Ten days later split skin grafts were placed over a large area of granulation tissue, which healed satisfactorily.

Comment

Necrotising fasciitis was described by Meleney in 1933 as a separate entity from erysipelas and synergistic gangrene.[1] The legs are usually affected and periorbital tissues[2] and elbows[3] are affected uncommonly. Surgical intervention with removal of all necrotic tissue is the treatment of choice.[1] Despite high doses of antibiotics our three patients still developed blistering and necrosis of the affected tissues. Debridement might have shortened the course of the disease but the decision is difficult when important tissues such as eyelids are affected.

All three patients had low antistreptolysin O titres (80-400) but high anti-desoxyribonuclease B titres (1200->9600), which is usual in skin infection with *S pyogenes*. All three had normal (<10 to 40) antibody titres to M-associated protein (MAP) type I.[4] High titres (>80) are observed in patients with rheumatic fever and uncomplicated infection due to certain opacity factor-negative serotypes associated with throat infection. There was a small rise in titre both to MAP II[4] (<10 to 20) and to the opacity factor (OF)[5] of M-type 75 (5 to 10) in the third patient, and a similar titre to M-type 75 OF was present in the second patient. M-type 75 streptococcus is often found in skin lesions in tropical countries but is uncommon in the UK, while anti-OF to M-type 75 occurs with low frequency (<3%) in the British population. These results confirm recent streptococcal infection in the third case and suggest that the patients had been infected with a "skin" strain. M-type 52 streptococcus, a "skin" strain, has been reported as the cause of one case of necrotising fasciitis.[3] It needs to be established whether this condition is due to "skin" rather than "throat" strains of *S pyogenes*.

We thank Dr J Bamforth, Mr R K Jackson, and Mr I H Chisholm for their help and permission to report on their patients, and Miss C A M Fraser for performing the anti-opacity factor tests.

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Public Health Laboratory, General Hospital, Southampton SO9 4XY
D SEAL, MB, MRCPATH, consultant microbiologist

Department of Medicine, University of Southampton, Southampton SO9 5NH
B LEPPARD, MRCP, senior lecturer in dermatology

Central Public Health Laboratory, Division of Hospital Infection, Colindale, London NW9 5EQ
J WIDDOWSON, BPHARM, PHD, top grade scientific officer

Southampton Eye Hospital, Southampton SO9 4XW
J McGILL, FRCS, consultant ophthalmic surgeon
P TORMEY, FRCS, senior registrar

Pulmonary oedema precipitated by nifedipine

Nifedipine (Adalat), a calcium antagonist, is useful in the treatment of angina pectoris, particularly when beta-blockers are contraindicated, and in suspected cases of coronary artery spasm. The drug has negative inotropic effects and theoretically may precipitate cardiac failure, although this has not been reported.[1 2] We describe a case of acute pulmonary oedema precipitated by nifedipine.

Case report

A 71-year-old man with symptoms of severe angina pectoris and a clinical diagnosis of moderate aortic valve stenosis and probable coronary artery disease was admitted for cardiac catheterisation. He had no signs of congestive cardiac failure. His electrocardiogram showed controlled atrial fibrillation. His chest radiograph showed a cardiothoracic ratio of 55% and no pulmonary venous congestion. His medication on admission was digoxin 0·25 mg daily, Dyazide (hydrochlorothiazide and triamterene) 1 tablet daily, and perhexiline maleate 100 mg twice daily. Cardiac catheterisation showed severe aortic valve stenosis (peak systolic gradient = 124 mm Hg and aortic valve area = 0·4 cm^2). The mean pulmonary capillary wedge pressure was 32 mm Hg and there was no mitral valve disease. Selective coronary arteriography showed diffuse triple vessel disease. He was not accepted for cardiac surgery and an attempt was made to control his symptoms medically. He was discharged taking the same medication but isosorbide dinitrate replaced the perhexiline and the dose of Dyazide was doubled.

When seen a month later he complained of increasing angina. There were no alterations in his physical signs and his electrocardiogram and chest radiograph were unchanged. His raised left ventricular end diastolic pressure contraindicated beta-blockers and we thought nifedipine might benefit him. He took the first 10 mg capsule that evening. Half an hour later he had extreme dyspnoea, which lasted several hours. The next morning he took a second capsule, which precipitated acute pulmonary oedema. He was admitted to hospital in extremis with most florid clinical and radiographic features of pulmonary oedema and no alteration in his electrocardiogram. He was treated conventionally with oxygen, morphine, and frusemide. Intravenous calcium chloride was also given to counteract the calcium-antagonist effect of nifedipine. He made a steady recovery and was symptomatically improved on discharge several days later taking digoxin, frusemide, and isosorbide dinitrate.

Comment

The negative inotropic effect of nifedipine does not usually reduce cardiac output or provoke cardiac failure, because of its potent vasodilatory effect on resistance vessels.[3] The drug has been recommended in the treatment of acute pulmonary oedema in cases in which left ventricular after-load reduction is desirable.[4] As this case shows, in aortic stenosis impedance to left ventricular ejection is fixed

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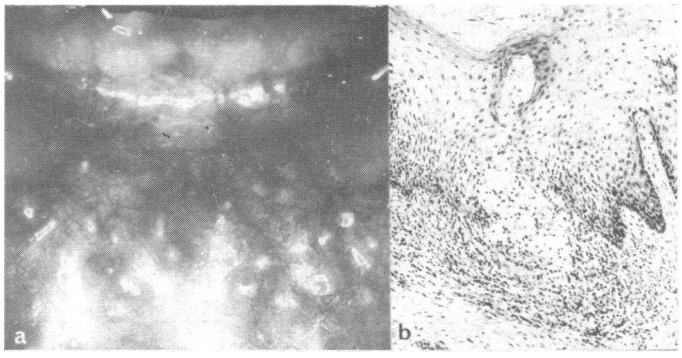
Department of Cardiology, Wentworth Hospital, P B Jacobs, Natal 4026, South Africa
DAVID J GILLMER, MRCP, physician and lecturer
PETER KARK, MB, registrar

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