Thorax 1982;37:861-862

Short reports

Actinomycosis of the trachea affecting the right supraclavicular region

O MAIWAND, AR MAKEY, A KHAGANI

Colindale Hospital, London

Actinomycosis is a worldwide chronic infection caused by an anaerobic Gram-positive organism. The human disease is most commonly caused by *Actinomyces israelii,* described by Israel in 1878. The organism is a normal commensal of the mouth and is found especially in the tonsils and in carious teeth. This endogenous infection is characterised by granulation formation and by fibrous infiltration and may present as a pseudotumour mass.[1] Classically there are multiple small, coalescent abscesses with communication sinuses and "sulphur" granules, which when recognised are diagnostic. A normocytic normochromic anaemia is frequently seen.[2] *Actinomyces* is a difficult organism to isolate and the infection is difficult to treat. Treatment consists of surgical drainage of the infected abscesses and administration of antibiotics. Penicillin is the drug of choice, to be given for at least three months; in patients allergic to penicillin clindamycin may be the alternative choice. Intravenous treatment is not necessary.[2] To our knowledge, actinomycosis of the trachea and supraclavicular region has not been reported previously.

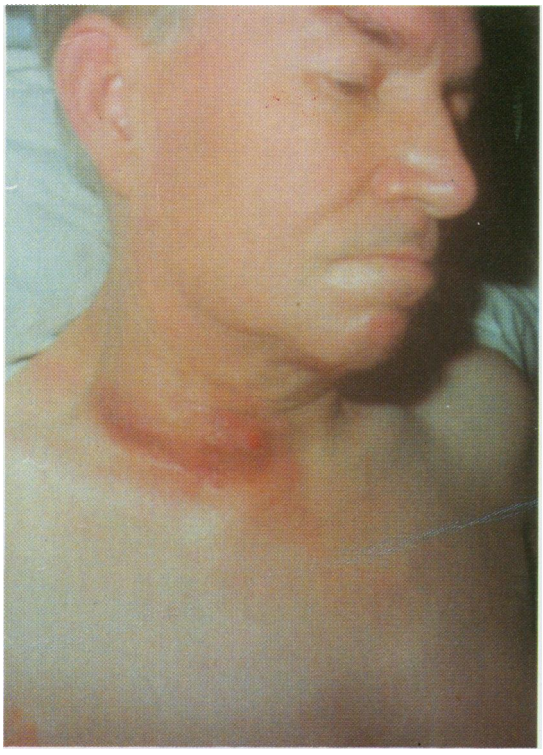

Fig 1 *Indurated mass at the root of the patient's neck with imminent sinus formation.*

Address for reprint requests: Mr O Maiwand, Colindale Hospital, Colindale Avenue, London NW9 5HG.

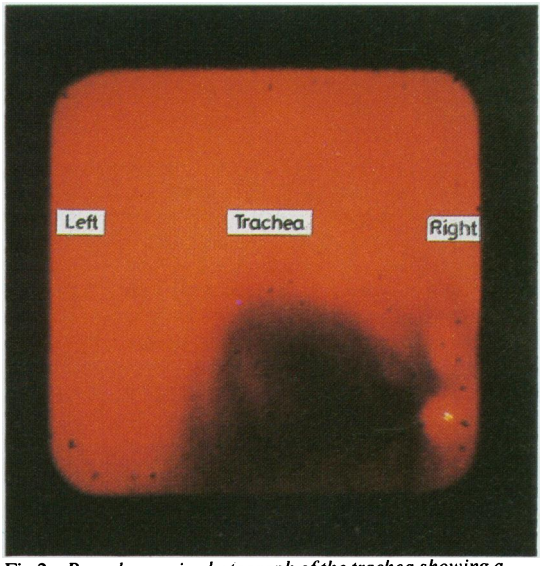

Fig 2 *Bronchoscopic photograph of the trachea showing a nodular mass on the right lateral tracheal wall.*

Case report

A 68-year-old Polish man who came to England 20 years ago had worked as a farmer and later as a handyman. Early in 1975 he developed a persistent cough and a swelling in the right supraclavicular region. There were no other chest symptoms. The swelling increased in size and he was referred to this unit.

General examination showed no systemic disease, though the presence of a few carious teeth was noted. The supraclavicular mass was covered with bluish-red adherent oedematous skin. It was fixed and hard and lay partially behind the sternomastoid muscle (fig 1). A chest radiograph showed no abnormality. Bronchoscopy showed the presence of a nodular mass, 1½ x 1 cm, on the right lateral wall of the trachea 5 cm below the cords (fig 2). Histological examination of biopsy specimens taken at the time showed diffuse acute and chronic inflammatory changes. One fragment showed a microabscess that contained colonies of actinomycetes. Histological

examination of a biopsy specimen taken from the supraclavicular swelling showed areas of granulation tissue and a colony of actinomycetes. Gram-film examination of pus from the supraclavicular region also showed actinomycetes. Anaerobic culture of the pus on blood agar media for 12 days failed to grow the organism, possibly because treatment had had to be started.

The patient was treated with benzylpenicillin 600 mg intramuscularly twice daily for four weeks, followed by phenoxymethylpenicillin 250 mg orally four times daily for six months. Bronchoscopic examinations later in 1975 showed that the tracheal disease had resolved completely and no supraclavicular mass could be detected. So far there has been no relapse.

Discussion

Actinomycosis is an uncommon but well-recognised condition, affecting principally the cervicofacial region. Less commonly it occurs in the right iliac fossa and least commonly in the lung, where it usually affects the pleura and chest wall, sometimes coexisting with bronchial carcinoma.[3]

Haematogenous spread of the disease is rare.[4] Lymphatic spread is believed not to occur owing to the large size of the organism. Most human infection is endogenous. The mouth is the site of primary infection, which usually originates from the gum, carious teeth, or tonsils. McQuarrie and Hall found dental sepsis in all of their nine cases of thoracic actinomycosis.[5] Males are affected more often than females and the usual age range is 10-30 years. Local trauma and tissue hypersensitivity to the organism may play a part in the initiation of the disease, which then spreads directly to the adjacent tissue. The site of entry of the infection in our patient was most likely the tracheal wall, in an area of mucosal abrasion. From there it spread to the surrounding soft tissue in the supraclavicular region. As his chest radiograph was normal the disease is most unlikely to have arisen peripherally in the upper lobe and secondarily affected supraclavicular region and thence the trachea.

We wish to thank Dr JC McAlpine, the department of bacteriology at Edgware General Hospital, and the staff at Colindale Hospital for their co-operation.

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