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Atraumatic suppurative mediastinitis and purulent pericarditis due to *Eikenella corrodens*

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ABSTRACT Atraumatic suppurative mediastinitis is an uncommon infection. A case with an associated purulent pericarditis caused by *Eikenella corrodens* is reported.

The infrequent isolation of *Eikenella corrodens* in pure culture from patients with subdural empyema, endocarditis, meningitis, osteomyelitis, septic arthritis, pneumonia, lung abscess, and empyema[1 2] indicates that this organism is probably a low grade but important pathogen in some circumstances. *Eikenella corrodens* has also been reported in mediastinitis but only as part of a mixed bacterial flora after oesophageal perforation.[3]

We report a case in which *Eikenella corrodens* was the sole causative agent of infection at an uncommon site: atraumatic suppurative mediastinitis[4 5] and an associated purulent pericarditis[6] occurred concurrently in a previously healthy young woman, whose primary infection was probably pneumonia.

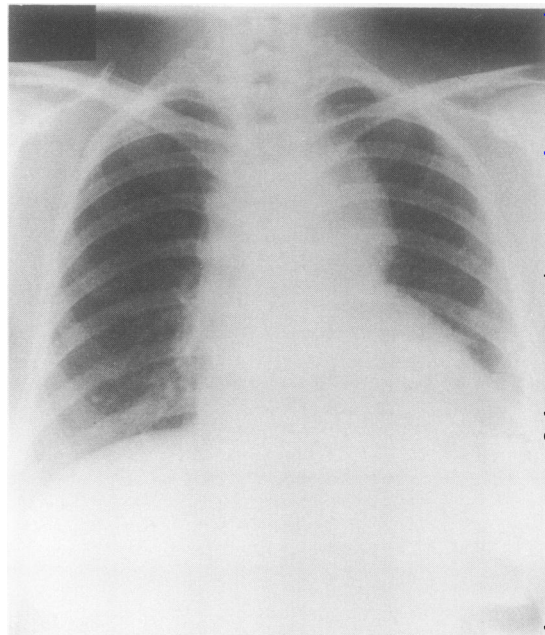
Case report

A 28 year old woman was admitted with a five day history of retrosternal chest pain, severe headache, vomiting, and sore throat, treated for two days by her general practitioner with oral erythromycin. Three weeks earlier, within two days of returning from Portugal, she had had an episode of non-bloody diarrhoea and abdominal pain lasting seven days. There was no recent history of aspiration, foreign body inhalation, or oral cavity or dental infection. Five years previously she had been investigated for recurrent haemoptysis and intermittent production of purulent sputum. Bronchography at that time suggested minimal bronchiectasis of the right middle lobe.

On admission she had a tempera re of 39·9°C, with neck stiffness and tachycardia (120 be: /min). The peripheral blood count showed a polymorph leucocytosis of $20{\cdot}6 \times 10^9$/l and an erythrocyte sedimentation rate of 110 mm in one hour. Lumbar puncture showed normal cerebrospinal fluid. Cultures of sputum, urine, blood, and throat swabs and serological tests for mycoplasma and legionella gave negative results. The electrocardiogram was normal. The chest radiograph (figure) showed considerable anterior mediastinal widening to the left of the trachea, suggesting adenopathy. The observation of an air bronchogram on a penetrated radiograph did, however, raise the possibility of collapse and consolidation of the left upper lobe. Mediastinal tomography failed to differentiate between lymphadenopathy and a pulmonary parenchymal lesion.

Parenteral treatment with cefuroxime, gentamicin, and erythromycin was started but produced no clinical improvement. A loud pleuropericardial friction rub became audible four days after admission, echocardiography showing a small pericardial effusion. Computed tomography of the chest showed a large mixed density mass in the upper mediastinum, suggesting malignant adenopathy.

After preliminary rigid bronchoscopy, which showed extrinsic compression of the left main bronchus, mediastinotomy disclosed a large fleshy "tumour," densely adherent to the left lung, with a macroscopic appearance suggesting lymphoma. Biopsy specimens, however, showed fibrosis, fibrin, and acute on chronic inflammation of lung tissue only, with no evidence of malignancy. Two days later, at thoracotomy, it was established that the "tumour mass" was



Posteroanterior chest radiograph on admission, showing upper mediastinal widening.

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in fact part of the left upper lobe, which was consolidated and adherent to the mediastinum; the pericardium was inflamed, and enclosed a straw coloured effusion. After mobilisation of an intensely inflamed mediastinum, necrotic purulent material was aspirated from the upper mediastinum; a pericardial window was fashioned; and biopsy specimens of pericardium, mediastinum, and left upper lobe were obtained for histological and microbiological examination. The mediastinum was irrigated with warm saline, and the chest closed over an apical and an upper mediastinal drain. Within 12 hours the patient's clinical condition had improved considerably and her temperature gradually fell over the following three days.

Histological examination showed a fibrinous pericarditis with oedema, pus cells in the pericardial fluid and fibrinous pleurisy with acute inflammation, oedema of the alveolar septae, and intra-alveolar histiocytes with a dense fibroblastic reaction, consistent with pneumonia. No organisms were detected in direct smears of the mediastinal pus with Gram and Ziehl-Neelsen stains, but after four days *Eikenella corrodens* was isolated in pure culture. Colonies on blood agar did not show characteristic "pitting" and growth on MacConkey agar was poor. The characterising features of this organism were that it was a Gram negative, non-motile rod, facultatively anaerobic on primary isolation, catalase negative, oxidase positive, lysine and ornithine decarboxylase positive, nitrate positive, and unable to ferment sugars. For aerobic growth it appeared to have an obligate requirement for haemin, but further testing showed it to be porphyrin positive. The isolated strain was shown to be sensitive to ampicillin, gentamicin, and cefuroxime, but resistant to clindamycin. Oral amoxycillin was given for three weeks, during which there was continued clinical improvement. When followed up at six months the patient remained well, and the chest radiograph was normal.

Discussion

To our knowledge, this is the first report in which *Eikenella corrodens* has been implicated as the sole pathogen in a case of atraumatic suppurative mediastinitis. The patient also had pericarditis and the primary source of the infection may well have been occult left upper lobe pneumonia with either direct extension or lymphatic spread into the mediastinum.[7]

Most infections occur in the posterior mediastinum,[7] and recognised primary sources in addition to pneumonia include oesophageal perforation, extension of a retropharyngeal abscess, orofacial cellulitis, dental abscess, empyema, and neck and mediastinal surgery.[5] Pure culture of a single organism, as in this case, is unusual and reported cases have invariably been due to polymicrobial infection by bacteroides, peptococci, clostridia, and anaerobic streptococci.[5]

There are no specific diagnostic radiological features of mediastinitis. The earliest and most frequent radiological sign is mediastinal widening.[8 9] Consequently, as in this case, the diagnosis may not be clinically obvious and may become evident only at thoracotomy. Furthermore, the high mortality and poor response to antibiotics, which may be due to inadequate penetration into devitalised tissue, make early surgical exploration and drainage necessary for the management of such infections.

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