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 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.

We report a case in which *Eikenella corrodens* was the sole causative agent of infection at an uncommon site: atraumatic suppurative mediastinitis and an associated purulent pericarditis 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 temperature of 39.9°C, with neck stiffness and tachycardia (120 be./min). The peripheral blood count showed a polymorph leucocytosis of 20.6 × 10⁹/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 pleuropapical 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

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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 decarbox-
ylase 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. 3 Pure culture of a single
organism, as in this case, is unusual and reported cases have
invariably been due to polymicrobial infection by bac-
terioi.de, 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 mor-
tality and poor response to antibiotics, which may be due to
inadequate penetration into devitalised tissue, make early
surgical exploration and drainage necessary for the man-
gement of such infections.

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