SUDDEN DEATH FROM PRIMARY LUNG ABSCESS IN MIDDLE-AGED DIABETIC MEN — TWO CASE REPORTS

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SYNOPSIS

We describe the course of illness in two middle-aged diabetic men who died suddenly from primary lung abscesses. They were in apparent good health and presented with fever, cough and small lung abscess [< 5 cm] on chest radiographs. While receiving standard antimicrobial therapy in hospital, they experienced sudden respiratory arrest from aspiration of purulent material presumably following the drainage of lung abscess contents. This is a very unusual complication and the similar clinical features between these two cases prompted this report.

Keywords: Lung abscess, aspiration, sudden death.

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INTRODUCTION

Primary lung abscess is a serious pulmonary infection associated with significant morbidity and mortality. The etiologic agents which may be responsible for abscess formation in the lungs have been identified using modern aerobic and anaerobic culture techniques on lower respiratory tract material obtained from transtracheal aspiration. The anaerobic commensals which inhabit the normal upper respiratory tract form the major group of pathogens (1). The route of infection is by either occult or gross aspiration of oropharyngeal secretions.

While treatment with appropriate antimicrobial agents will produce resolution in the majority of cases, surgical intervention may be life saving in a small number of patients (2, 3, 4, 5). The elderly and immunologically impaired hosts run a higher risk of encountering complications and death. Also at risk are chronically debilitated patients with poor cough and gag reflexes who might even die from underlying diseases while the pulmonary sepsis is under effective control. Other important prognostic factors include the virulence of infective organisms, size of the abscess and the ease of communication between the abscess and the bronchial tree.

The therapeutic recommendation for primary lung abscess in current standard text books is to rely primarily on medical, i.e. antimicrobial therapy and this coupled with the impression that young patients without serious underlying illnesses generally have very favorable prognoses may result in complacency during the management of this particular group of patients (3, 4, 5). To highlight this point, we describe the clinical course of two middle aged men who died suddenly from fulminant pulmonary sepsis during the medical treatment of lung abscess.

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CASE REPORTS

PATIENT 1 [admitted 6th. Nov. 1984, died 15th Nov. 1984]:

C.K.F. was a 44-year-old Chinese man. He had diabetes mellitus of over four years well controlled by an oral hypoglycemic drug. He was admitted with a history of fever for eight days and a dry cough. He had received outpatient treatment with oral erythromycin for a week without improvement. The physical examination was unremarkable. His blood counts showed a leukocytosis of 14,600 x 10 /L. with 89% neutrophils which exhibited toxic granules. His chest radiograph showed dense consolidation of the left mid-zone [3 cm diameter] with a visible air-fluid level. After appropriate specimens were collected for microbiology, intravenous penicillin and cloxacillin was started. On 14th Nov. [1 week after admission], because he remained febrile, intravenous metronidazole and gentamycin were added. The next morning, he suddenly became acutely breathless, cyanosed and collapsed with bradycardia and severe bronchospasm. He was intubated immediately and purulent material was sucked from the airway. He died later that same day in the intensive care. Post mortem examination revealed confluent bronchopneumonia in both lungs with an abscess cavity in the left upper lode measuring 2 cm in diameter. No culpable organisms were isolated from antemortem culture specimens.

PATIENT 2 [admitted 11th Oct. 1986, died 21th Oct. 1986]:

E.M.N. was a 44-year-old Malay man. He had diabetes mellitus of over 8 years duration treated with oral hypoglycemic drugs. He gave a 3 day history of cough and fever but denied chills or purulent sputum. The physical examination revealed inspiratory crackles over his right chest and skin changes consistent with diabetic dermopathy. He had a leukocytosis of 30,600 x 10 /L with 92% neutrophils. The chest radiograph showed right middle lobe consolidation with a 4 cm abscess cavity. He had moderate renal impairment [blood urea 111 mg/dl; serum creatinine 3.2 mg/dl.] and normochromic normocytic anemia of 10 g/dL. presumably in association with diabetic nephropathy. Intravenous penicillin and gentamicin were started and there was a partial response after 72 hours with decline in the temperature. On the morning of 15th. Nov. however, he suddenly collapsed with cyanosis,

tachypnoea, shock and bradycardia. Chest examination subsequently revealed diffuse coarse crackles and chest radiographs showed loss of definition of the abscess with surrounding patchy air-space consolidation. He died from hypoxic brain damage and nosocomial sepsis a week later. Blood specimens taken in the intensive care grew multiple gram negative enterobacteriacea of presumed nosocomial origin.

DISCUSSION

There is little doubt that these two men died from asphyxiation following aspiration of purulent material from solitary lung abscesses and the subsequent disseminated pulmonary sepsis. The primary event was respiratory arrest and not cardiac arrest in both cases. This was suggested by the findings of respiratory distress [attempted gasping and coughing], cyanosis and bradycardia [often associated with hypoxemia]. One of the authors [TKL], who resuscitated Patient 1, obtained purulent material from the endotracheal tube and the post mortem examination revealed the expected findings of extensive aspiration pneumonia within 24 hours of collapse. In the case of Patient 2, following resuscitation, diffuse coarse crackles were heard, there was bronchopneumonia on the chest radiographs and no acute cardiac event could be documented to account for the sudden collapse.

The indolent course of the illness which immediately preceded the catastrophic respiratory event was most remarkable and this, together with the similarity of other clinical features, prompted us to report these two cases. Although they were febrile and had marked leukocytosis, both patients were freely ambulant and had stable pulse rate, blood pressure and respiratory rate on hospital charts. Diabetes mellitus was the underlying illness in both cases and asymptomatic nephropathy was evident in Patient 2. The diabetic state probably constituted a significant risk factor in the two patients.

While contamination of the airways with infected secretions was the major event leading to their demise, uncontrolled underlying sepsis might have contributed to the propensity for sudden rupture of the lung abscesses in these two patients. In this context, the possibility that either inappropriate or inadequate dosing of antimicrobial agents should be seriously considered. The failure to isolate putative infective organisms in both subjects will allow us only to speculate and not derive firm conclusions. Intra-venous penicillin is the antibiotic recommended for primary lung abscess by most authorities and was received by both patients (3, 4, 5). This regimen is active against most anaerobic organisms which are the major pathogens in lung abscess and empiric experience indicates that penicillin alone is usually adequate. Recent evidence from a large, prospective randomised study suggests that clindamycin is a superior agent and should be substituted for penicillin if the patient is seriously ill or if no prompt response to penicillin occurs (6). Although metronidazole exhibits a broad spectrum of in-vitro activity against anaerobic organisms its effectiveness in primary lung abscess has not been demonstrated in clinical trials and therefore it cannot be recommended as the first agent (7).

Because a wide variety of aerobic organisms may also cause cavitating pneumonia, penicillin alone will not be adequate therapy against these organisms which tend to be more virulent than anaerobes. The presence of long standing diabetes mellitus may predispose to infection by virulent aerobic organisms such as staphlococcus aureus or gram negative enteric bacteria which may cause rapidly necrotizing pneumonia (8). While both our patients received antibiotic combinations which provided broad spectrum cover for both gram positive and gram negative aerobic organisms, without definitive microbiology, we cannot be certain that the antimicrobial therapy was adequate in either. Nevertheless, the regimens used were in accordance with current consensus on the treatment of lung abscess reflected in authoritative texts (4, 5). The absence of grossly putrid sputum in both of the cases described in this report may be a clue that aerobic organisms were responsible. The alternative explaination for scanty sputum is poor communication between the abscess cavity and the bronchial tree. This may have predisposed the abscess to sudden rupture and widespread flooding of the airways.

Sudden drainage of a very large lung abscess and aspiration into the normal lung is a rare but well described complication (9). We have observed this complication occasionally and always in association with very large abscess cavities. The cavities in these two patient were below 5.0 cm in diameter and would generally not be considered at high risk. We suspect that the underlying diabetes mellitus produced a state of immunosupression which, in conjunction with a highly virulent organism and relatively poor drainage, resulted in a critical situation.

Although bronchoscopy was planned for both cases, we doubt that the fiberoptic instrument would have provided adequate drainage. Furthermore, the procedure itself could have precipitated rupture of the abscess. Needle aspiration is not recommended for the very reason that it might result in disseminated infection. Percutaneous tube drainage has been used successfully in selected patient with large [over 8 cm in diameter] and easily accessible lesions (10). The cavities in our patients were both small and deeply seated. Surgical resection is indicated only for large and slowly resolving abscesses which have failed to close after a trial of medical therapy up to 6 weeks in duration. Even in retrospect, surgery was certainly not indicated in our two unfortunate patients.

In conclusion, a primary lung abscess which does not respond promptly to medical therapy may cause sudden death from aspiration in patients with diabetes mellitus. The risk of sudden flooding of purulent material into the airways may be related to the virulence of the causative organisms rather than size of the abscess cavity. We therefore urge careful selection of antimicrobial agents based on bacteriological information and extreme vigilance in monitoring patients, even those who may appear relatively well.

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