PULMONARY ASPERGILLOSIS PRESENTING AS COR-PULMONALE IN AN IMMUNOCOMPETENT NIGERIAN MAN: A CASE REPORT

S. B. Sarko, C. N. Ekweani and I. O. Okpe

Department of Medicine, A. B. U. Teaching Hospital, Kaduna, Nigeria

ABSTRACT

Invasive pulmonary aspergillosis is a fulminating and highly lethal infection of severely immunocompromised patients. Risk factor for aspergillosis infection include granulocytopenia (from bone marrow infiltration), intensive cytotoxic chemotherapy, corticosteroid use, chronic obstructive airway disease, pneumonocnosis and malnutrition to mention a few. Rarely, pulmonary aspergillosis has been reported in immunocompetent patients. Reports of right ventricular dysfunction from pulmonary aspergillosis are not common. This is a report of pulmonary aspergillosis with cor pulmonale in an immunocompetent Nigerian man.

Key words: Invasive pulmonary aspergillosis, immunocompetent

INTRODUCTION

Aspergillus is a ubiquitous fungus that causes a variety of clinical syndromes in the lung, ranging from aspergillosis in patients with lung cavities to chronic necrotising aspergillosis in those who are mildly immunocompromised or have chronic lung disease. Invasive pulmonary aspergillosis (IPA) is a fulminating and highly lethal infection seen mostly in severely immunocompromised patients with a reported mortality rate of approximately 60%.

Risk factors for invasive pulmonary aspergillosis include, neutropenia, intensive cytotoxic chemotherapy for cancer, and in patients with a history of corticosteroid use. Severe aspergillosus infection had been reported in immunocompetent patients without underlying lung lesions. Invasive pulmonary aspergillosis is a major problem clinically because accurate diagnosis is difficult, the therapy is toxic and the mortality associated with this infection is high.

Cases of Aspergillus flavus endocarditis, *S. spondylitis* and pulmonary aspergillosis in immunocompetent patients have been reported recently. This is a report of chronic necrotising aspergillosis with cor-pulmonale from our centre.

Case report

A 35 yr old male, Nigerian, security man presented to our hospital with a 3- year history of recurrent productive cough.

Reprint requests to: Dr. Sani B. Sarko, Department of Medicine, A. B. U. Teaching Hospital, Kaduna, Nigeria. E-mail: asgsarko@hotmail.com
pleuritic chest pain and a 2-week history of symptoms of right heart failure. Cough was productive of frothy sputum, with fewer and night sweats. He denied history of weight loss. He had been admitted to a General hospital twice in the preceding 2 years for pneumonia. There was a history of significant alcohol ingestion.

Physical findings were tachypnoea (respiratory rate 34 cycles per minute), central cyanosis, bilateral pitting pedal and sacral edema, grade 3 finger clubbing, tachycardia (heart rate 118 per minute), normal blood pressure, raised jugular venous pressure (JVP) to the angle of the jaw, a right ventricular heave (R VH), normally placed apex beat, widespread coarse crepitations in upper and mid lung zones bilaterally, and a third heart sound S3 at the left lower sternal edge. The liver was tender and enlarged 10cm below the right costal margin. The working diagnosis was right heart failure from long standing lung disease (pulmonary).

Laboratory findings were normal complete blood count with haemoglobin of 13.0 g/dl, leukocyte count of 5.3 X 109/L (neutrophils 60%, lymphocytes 38%, Eosinophils 2%), normal renal function and blood sugar, and a normal erythrocyte sedimentation rate of 6mm fall per hour. Electrocardiography (ECG) showed sinus rhythm with rate of 120/min, right axis deviation of 120°, tall R waves in V1 and V2, and a positive deflection in the augmented right arm lead. Sputum microscopy and culture for bacteria (Acid fast bacilli inclusive) was negative in all the six samples analyzed. His serum was negative for human Immune deficiency Virus (HIV) antibodies and had normal CD4+ lymphocytes of 074 cells/mm3 (normal range is between 450 - 1570 CD4 cell per mm3). Sputum culture for fungi yielded Aspergillus Species. Chest X-Ray showed enlarged right ventricle, diffuse reticulo-nodular infiltrates in the upper and mid zones of the lung and bilateral shadows at the hila. The cardiothoracic ratio (CTR) is 0.61. (Picture). A diagnosis of copropulmonale from chronic necrotising aspergillosis was made.

The patient was managed with diuretics, low dose Angiotensin converting enzyme inhibitors (ACE-I), low salt diet, and oral spironolactone. He improved remarkably and was discharged on oral spironolactone 200mg daily. He was well at one year of follow up.

**Figure 1:** Chest X-ray showing diffuse reticulo-nodular infiltrates in the upper, mid and lower zones bilaterally.

**DISCUSSION**

Chronic necrotising aspergillosis, also called semi-invasive aspergillosis, was first described in two reports in 1981 and 1982. Chronic necrotising aspergillosis (CNA) is an indolent, destructive process of the lung due to invasion by Aspergillus species (usually A. fumigatus). It progresses slowly over months to years, and there is no vascular invasion or dissemination to other organs. This chronic course probably explains the 3-year duration of symptoms in our patient and the right
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Heart failure that brought the patient to our hospital.

The presentation is usually with fever, cough, sputum production and weight loss of one to six month duration. A minority of patients may however be asymptomatic. Radiological features include infiltrates in the upper lobes or the superior segments of the lower lobes. A fungal ball may be seen in nearly one half of the cases. 1,2 Adjacent pleural thickening is a characteristic finding and may be an early depiction of a locally invasive process. 3 Chronic necrotizing pulmonary aspergillosis is usually seen in middle aged and elderly patients with documented or suspected underlying lung disease like chronic obstructive pulmonary disease (COPD), inactive tuberculosis, previous lung resection, radiation therapy, pneumothorax, cystic fibrosis, lung infection or rarely sarcoidosis. 4 It has also been described in patients with mild immunosuppression, including those with diabetes mellitus, poor nutrition, patients on low dose corticosteroids, therapy, alcoholism, chronic liver disease, and patients with connective tissue diseases such as rheumatoid arthritis and amyloidosis. Our patient takes significant amount of alcohol and since alcoholics are oftentimes poorly nourished, we thought this might have contributed to the infection in our patient. Alcohol could also have contributed to his cardiac failure since alcohol consumption has been strongly linked to heart failure in Nigerian hypertensive patients. 5 His negative HIV antibodies, and normal CO2 - lymphocytes supports the competence of his immune system as at the time of evaluation.

Even thought, pulmonary aspergillosis usually affects patients with specific risk factors, that have immune deficiency as a common denominator, invasive pulmonary aspergillosis has been reported in immunocompetent patients. 6,7 This calls for a heightened index of clinical suspicion especially for clinicians in the developing countries where this disease could easily be confused with pulmonary tuberculosis that has similar clinical features. With the prediction of Aspergillus to invade blood vessels, massive pulmonary aspergillosis commonly leads to areas of infarction and haemorrhage in the lungs. 8 This on the long term leads to pulmonary hypertension, increasing demand on the right heart with consequent failure as seen in our patient. Inability to do echocardiography may have missed associated endocarditis or myocarditis.

Therapy for right ventricular dysfunction resulting from diseases of the pulmonary vasculature, aortas or lung interstitium (constrictive), is directed at the underlying disease. 9 In our patient, the definitive treatment was therapy for pulmonary aspergillosis.

Treatment of pulmonary aspergillosis with antifungal medications is indicated once the diagnosis is made. The response to therapy with IV amphotericin B is generally favorable. 10 However, therapy with itraconazole has emerged as an effective alternative to the relatively toxic amphotericin B. 11,12 Our patient responded well to itraconazole even though only 39% partial or complete response has been reported in one study. 12

Other modalities of treatment include drug combination therapy and surgery. Surgical resection is generally reserved for healthy young patients with focal disease and good pulmonary reserve. Patients not tolerating antifungal therapy, and patients with residual localized, but active disease despite adequate antifungal treatment. The long-term prognosis for patients with chronic necrotising aspergillosis is not well documented. Patients have survived...
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for 1 – 2 years following therapy. 9

REFERENCE


