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Case Report

A rare case of pleural effusion due to *Acinetobacter junii* in a known patient of pulmonary tuberculosis

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ABSTRACT

The genus *Acinetobacter* comprises a heterogenous group of bacterias that are mostly pandrug resistant and implicated in variety of nosocomial infections. *Acinetobacter junii* is a rare human pathogen and is mainly associated with blood stream infections in paediatric oncology patients. We report a rare case of pleural effusion caused by *A. junii* in a known pulmonary tuberculosis patient who was on cat-1 antitubercular treatment.

Keywords: *Acinetobacter junii*, Pleural effusion, blood stream infection

INTRODUCTION

The genus *Acinetobacter* comprises a heterogenous group of non fermentative Gram negative bacteria. These organisms are frequently pandrug resistant and capable of causing significant morbidity and mortality in patients with severe underlying disease and risk factors (e.g. prior broad spectrum antibiotic therapy, malignancy, central venous catheter, mechanical ventilation).[1,2]

The main pathogenic species is *A. baumannii* complex which is implicated in variety of nosocomial infections like ventilator associated pneumonia, bacteremia and urinary tract infections.[2] *A. junii* is a rare human pathogen and is mainly associated with blood stream infections in paediatric oncology patients.[3-5] In this report we present a rare case of pleural effusion caused by *A. junii* in a known tuberculosis patient.

CASE REPORT

A 81 years old normotensive and non-diabetic female was admitted in ward 16 of TB and Chest Department with complaints of fever, dry cough and left sided chest pain for 12 days duration. She was a known case of sputum positive pulmonary tuberculosis and had been on treatment with ATT cat-1 under DOTS strategy since last 6 months.

Lab investigations

Her complete hemogram was Hb=10 gm/dl, WBC count 8300/µl (Neutrophil 72%, Lymphocyte 16%); platelet 4 lacs. The liver and renal parameters were normal. Widal test and Malarial parasite were negative. ECG and echocardiography were normal. Chest Xray and USG thorax showed moderate left sided pleural effusion with linear atelectasis in left lower lobe and patchy area of consolidation in left lingular and right middle lobe suggestive of infective etiology (Figure 1). Pleural tapping was done under proper aseptic precaution and it was tested for Adenosine deaminase(ADA), lactate dehydrogenase (LDH), amylase, triglycerides, protein, sugar, cytology, CBNAAT and microbiological investigations. ADA was 5 IU/ml, low sugar, high protein and cell count with neutrophilic predominance and malignant cell was absent. Cytological and biochemical analysis of pleural fluid was suggestive of non

tuberculous exudative pleural effusion. Mycobacterium tuberculosis was not detected on CBNAAT. Patient was negative for HIV and Hepatitis B.

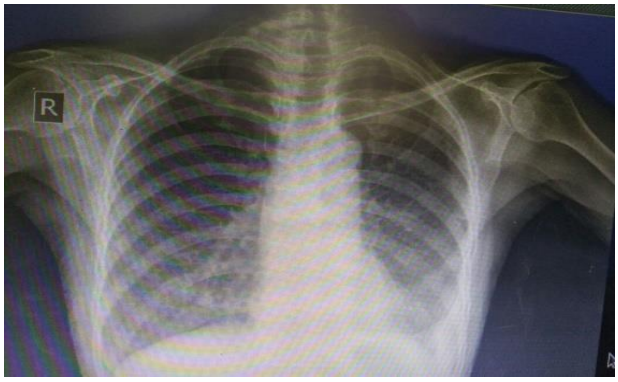

Figure 1: CXR PA view showing moderate left sided pleural effusion.

Microbiological investigations

Primary smear of pleural fluid showed presence of gram negative coccobacilli and pus cells. After overnight incubation at 37^0 C colonies were found on blood agar, MacConkeys agar which was 1.5-2 mm in diameter, non lactose fermenting and oxidase negative (Figure 2).

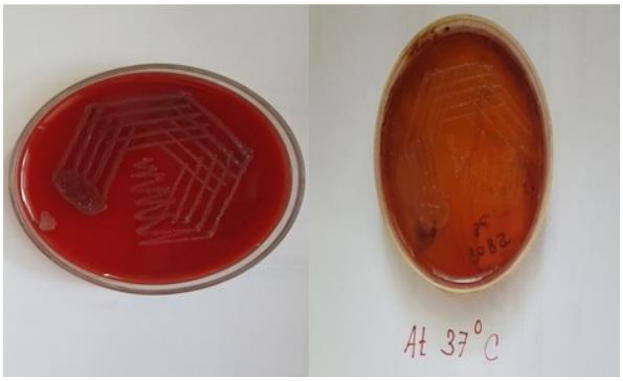

Figure 2: Non lactose fermenting colonies of *A. junii* on Blood and MacConkeys agar.

On gram staining these isolates appeared as gram negative cocco- bacillary forms (Figure 3). Further identification and antibiotic sensitivity was done by conventional as well as automated method (VITEK 2 Compact Biomerieux, India Pvt, Ltd). Conventional methods done were – motility (non motile), no growth observed at 42^0 C and 44^0 C, Arginine was dihydrolysed, hugh leifson OF media showing non fermenting pattern (Figure 4), urease not produced. The isolate was identified as *A. junii* and it was sensitive to amikacin, ciprofloxacin, tigecycline, minocycline, aztreonam, cefoperazone-sulbactam, intermediate sensitive to piperacillin-tazobactam, imipenam, meropenam but resistant to cotrimoxazole, gentamicin, ceftazidime, cefepime, doripenam. There was no growth on LJ media and no acid fast bacillus was seen on ZN staining of the pleural fluid.

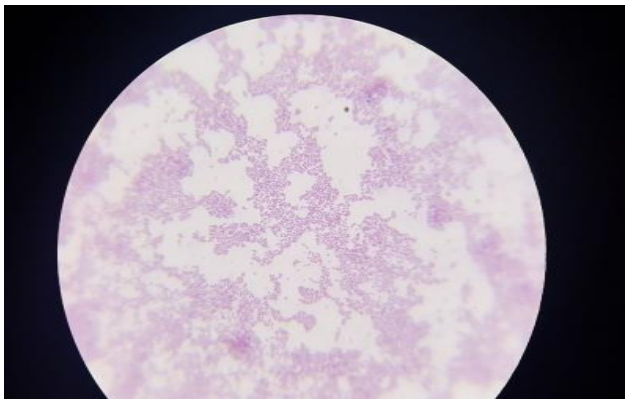

Figure 3: Gram negative coccobacillary forms on gram staining.

She was put on appropriate antibiotics and there was complete resolution of her signs and symptoms. Patient was discharged satisfactorily.

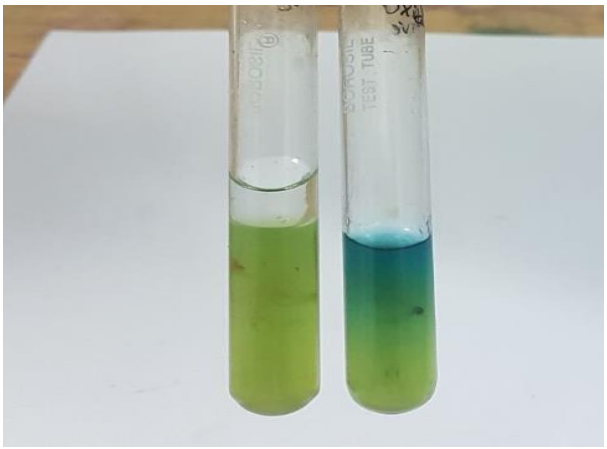

Figure 4: Hugh leifson OF media showing non fermenting pattern.

DISCUSSION

Acinetobacter junii is classified according to DNA-DNA hybridization studies as genomospecies 5 of the genus *Acinetobacter*, members of which have drawn significant attention as a cause of nosocomial infections.[6] It is a newly emerging pathogen that mainly affects patients with co-morbidities, malignancy or invasive procedures. It is associated with outbreaks of septicaemia in neonates and paediatric oncology patients.[3-5] Few cases of meningitis, peritonitis, ocular infection and septicaemia in an adult oncology patient caused by *A. junii* have also been reported.[7-9]

In larger analysis from Europe, strains of *A. junii* constitute 3.6-4.8% of all Acinetobacter isolates- 12/331 and 9/186 respectively.[10] Very few cases of septicaemia due to *A. junii* have been reported from India. The present

case represents a rare case of pleural effusion due to *A. junii* in a known tuberculosis patient who was on antitubercular treatment.

The course of infection by *A. junii* is generally benign and it exhibits more susceptibility to antibiotic therapy than *A. baumannii* which is reported in the present case. Other studies have reported similar findings.[9,11]

In conclusion, special attention should be given for the correct identification of *non baumannii Acinetobacter* species as they are the emerging pathogen in various nosocomial as well as community acquired infections which will contribute to the better knowledge regarding the epidemiology and the clinical impact of these species as human pathogens.

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