

Case Report

An uncommon organism causing necrotizing pneumonia in a toddler

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ABSTRACT

Necrotizing pneumonia (NP) is an uncommon complication of bacterial pneumonia in children, which must be looked into if a severe pneumonia has poor response to recommended antibiotics. The present case is a toddler with NP in whom fever and cough persisted despite treatment with first-line antimicrobial therapy, computed tomography (CT) scan revealed consolidation with multiple cavities, *Pseudomonas aeruginosa* was the pathogen isolated from bronchoalveolar lavage, which a very uncommon organism is causing NP. Community acquired necrotizing pneumonia caused by *Pseudomonas* is not reported in paediatric population. Hence, we report this case.

Keywords: Necrotizing pneumonia, *Pseudomonas aeruginosa*, Bacterial pneumonia, Non-resolving pneumonia, Pulmonary infection child

INTRODUCTION

Necrotizing pneumonia (NP) is a severe complication of community acquired pneumonia, and has a progressive and prolonged clinical course in a previously healthy child despite antibiotic therapy.¹ It is characterised by liquefactive necrosis and cavity formation within the necrosed lung tissue.² NP can occur in any lobe, but lower lobe involvement is more common.^{3,4} NP was previously believed to be rare in children but there has been an increase in the number of reports in the last two decades.⁵ The most common organisms implemented are *Staphylococcus aureus* and *Streptococcus pneumoniae*.⁵ *Pseudomonas aeruginosa* causing NP is rare, and hence we report this case.

CASE REPORT

2-year-old boy was admitted with high-grade fever and cough for 7 days, and breathlessness for 2 days, with positive contact history for tuberculosis. On examination, he was sick looking, pale, under-weight, febrile, with the

heart rate 144/min, respiratory rate 60/min, blood pressure 92/62 mmHg, oxygen saturation 92% in room air. His respiratory examination showed dullness on percussion with reduced breath sounds and crepitations in the left lung. Other systemic examinations were normal. Chest X-ray showed left middle and lower zone consolidation (Figure 1). A diagnosis of left side pneumonia was made, and started on intravenous ceftriaxone, oxygen, and other supportive cares. His complete blood count showed hemoglobin – 8.5 g/dl, white blood cells (WBC) – 14600/cu.mm with 65% neutrophils, 30% lymphocytes, and platelet count – 565000/cu.mm, erythrocytic sedimentation rate (ESR) – 110/hour. His biochemical values were normal. Child continued to have high-grade fever spikes, repeat chest X-ray on 5th day showed consolidation and cavitation's in left middle zone with minimal pleural effusion. Hence, antibiotic was changed to vancomycin and meropenem. Tuberculosis workup and retro-viral was negative. Even after 5 days of antibiotics, fever spikes persisted, chest X-ray and computed tomography (CT) scan was done, which showed consolidation with multiple cystic areas within the upper

segment of left lower lobe, a large cavity of size 2.5×2.9×3.6 cm, suggestive of necrotizing pneumonia (Figure 2).

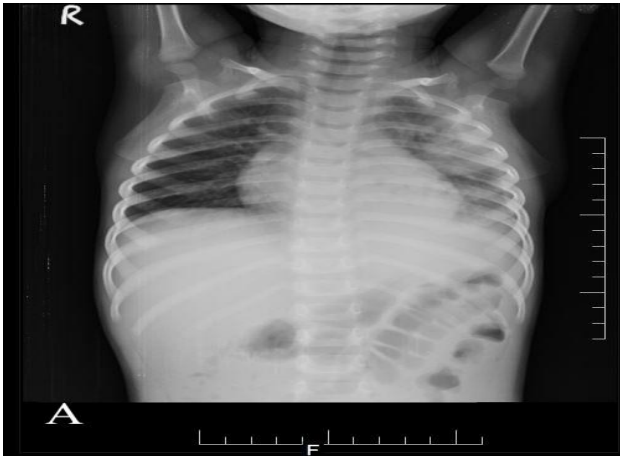


Figure 1: Chest radiography showing left middle and lower zone consolidation.

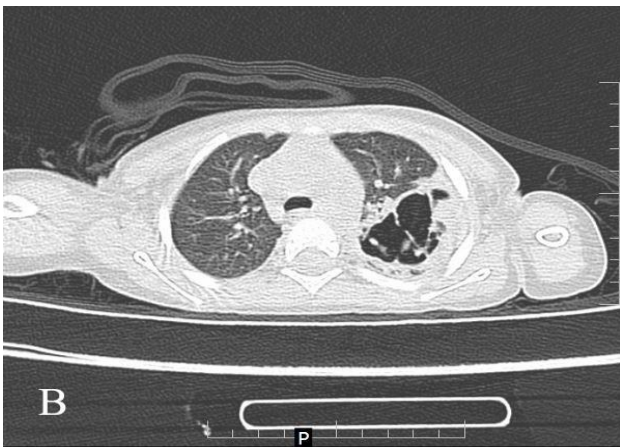


Figure 2: CT thorax showing consolidation with a large cavity in posterior segment of left upper lobe with multiple septations.



Figure 3: Chest radiography at 3-months follow-up showing complete resolution.

Pulmonologist opinion was sought for bronchoscopy, which showed muco-purulent discharge in left main bronchus, Bronchoalveolar lavage (BAL) for cartridge based nucleic acid amplification test (CBNAAT) was negative but showed heavy growth of multi-drug resistant strain of *Pseudomonas aeruginosa*, with intermediate sensitivity to ciprofloxacin. Hence, he was started on ciprofloxacin and within 48 hours child became afebrile, showed gradual stabilization of vitals, and he was weaned off from oxygen. Child was discharged with oral antibiotics for 7 days. Follow-up chest X-ray taken after 3 months showed complete resolution (Figure 3).

DISCUSSION

NP is an uncommon, but severe complication of pneumonia, “characterized by destruction of the underlying lung parenchyma resulting in multiple, small, thin walled cavities and is often accompanied by empyema and BPF”.^{5,6} It is a continuum of spectrum between pulmonary abscess on one side, and pulmonary gangrene on the other.⁶ The major pathogens are *Streptococcus pneumoniae* and *Staphylococcus aureus*, uncommon organism includes *Streptococcus pyogenes*, *Klebsiella pneumoniae*, *Hemophilus influenzae*, *Mycoplasma* and *Pseudomonas*.⁶ The clinical features of NP are similar to those of pneumonia which includes fever, cough, the shortness of breathing, localising chest signs such as dullness on percussion, decreased breath sounds, bronchial breathing on auscultation. The diagnosis is made by chest imaging studies; CT scan is the gold standard in diagnosis however, an intervention with bronchoscopy will help in identification of organism and institution of appropriate antibiotics.⁵ Complications of NP include para-pneumonic effusion, pleural empyema, broncho-pleural fistula, pneumothorax.⁵ Management includes either medical, surgical intervention (in form of segmentectomy, lobectomy, decortication and chest drainage tube placement) or both. The main stay of treatment is intravenous antibiotic therapy, rarely surgical intervention is warranted.⁷ NP has a protracted hospital course, median duration of stay is 12–30 days, or even longer in those with complications.⁷ Unlike adults, children have low fatality rate, and deaths are uncommon despite the fact that they may require intensive care management.⁸ Prevention of NP depends on reduction of incidence of community acquired pneumonia through improved nutritional status of the child⁹ and by vaccinations.¹⁰

In our case, the *Pseudomonas* was the organism isolated from BAL, which very uncommon organism is causing NP. *Pseudomonas* causing NP is predominantly hospital acquired, community acquired *Pseudomonas* is not reported in paediatric population. *Pseudomonas* NP in a child should raise the suspicion of immunodeficiency or cystic fibrosis. The proposed pathogenesis of NP in *Pseudomonas* infection is due to invasive nature of the organism and resultant thrombotic endarteritis.¹ In our child this is the first hospital admission for pneumonia, and cystic fibrosis workup (sweat chloride test) was negative

on follow-up. So, we could not find any identifiable risk factor for this child developing *Pseudomonas* NP other than malnutrition. Multidisciplinary approach with microbiologist and pulmonologist helped in expediting the management. This case was discussed due to the rare organism causing of NP and to the best of our knowledge no such case has been reported in the past decade on *Pseudomonas* causing NP in toddlers.²

CONCLUSION

NP is a serious complication of invasive pneumonia. NP should be considered when, despite appropriate antibiotics, the child remains febrile and unwell with persistent signs of respiratory distress. Timely intervention with bronchoscopy may help in isolation of the disease pathogen so that appropriate antibiotics can be instituted for better outcome.

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