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Case Report ***Open Access***

Necrotizing Pneumonia in Children: A Case Report

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Abstract

Necrotizing pneumonia (NP) is an uncommon severe complication of pneumonia in children. It is characterized by destruction of the underlying lung parenchyma resulting in multiple small, thin-walled cavities and is often accompanied by empyema and bronchopleural fistulae. Early consultation with thoracic Surgeon can be life-saving. We report a case of a child with NP in whom no pathogen is documented in its aetiopathogenesis. Severe pneumonia, which shows slow response to recommended antibiotics treatment, should raise the suspicion of NP and possibly one of the polymicrobial origins. Even in resource constrained settings, prompt institution of antibiotics and supportive care can result in resolution of pulmonary lesions.

Keywords: Necrotizing pneumonia; Child; Community acquired pneumonia

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Introduction

Necrotizing pneumonia (NP) is quite uncommon in children compared with the adult age group, rare complication of pneumonia. It is characterized by progressive pneumonic illness in a previously healthy child despite appropriate antibiotic therapy and runs a protracted clinical course[1]. NP lies on a spectrum between pulmonary abscess and pulmonary gangrene and is accompanied frequently by empyema and bronchopleural fistulae (BPF). It is speculated that reduced blood flow from thrombosed vessels decreases antibiotic concentrations within the affected lung tissue, leading to persistent infection and further destruction of pulmonary tissue[2]. NP may be complicating 0.8-7% of all cases of community acquired pneumonia (CAP)[3].

There are some factors that lead to necrosis of the pulmonary parenchyma, such as the staining of Pantano-Valentine's leukocidin-producing *Staphylococcus* aureus, which have a greater virulence and therefore a greater effect on the tissue they infect. Similarly, there are other strains of high virulence demonstrated as the strain USA300, which has been associated with some cases of necrotizing pneumonia[4-6].

We are presenting this case because we hope this case will help to create awareness among clinicians about this clinical entity. Necrotising pneumonia should be considered when patient's condition not improving despite appropriate antibiotic. Early contrasted CT scan will aid in further assessment and diagnosis. The purpose of this report is Managing patients with necrotising pneumonia is challenging because there is no firm guidelines outlining management of necrotising pneumonia. The mainstay of treatment is supportive care with appropriate antibiotics. Surgical intervention may play a role when medical therapy fails.

Case Presentation

A 13-month-old baby boy was admitted through emergency of Evercare hospital as diagnosed case of Pneumonia with high grade intermittent fever of 2 days, cough for 7 days with noisy breathing & progressive

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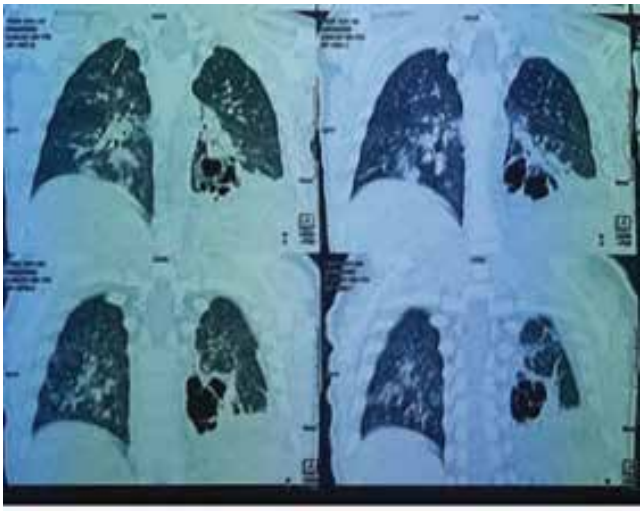
breathlessness of 2 days associated with abdominal distention for 2 days. There was no history of foreign body ingestion. He had no previous episode of difficulty with breathing. No H/O contact with TB patient. He had been growing well and had no history of previous hospitalizations. His vaccination was going on. His past medical history was not contributory. He was born term with ABW (3 kg) by LUCS in this hospital. Ayan was on exclusive breast feed up to 6 months then complimentary feeding was started. He was on usual family diet. His development of milestone was age appropriate. His parents were highly educated father was a service holder. Both parents and other two siblings were apparently well. He was admitted for 2 days in the course of the illness in a private hospital where he received some parenteral drugs with antibiotic. He was brought to our facility because of worsening of the symptoms. Examination revealed an acutely looking, pale, normal weight toddler (weight = 10 kg, height = 77 cm, WFH 0.5 standard deviation) who was tachypnoeic with a respiratory rate of 62/min and chest wall indrawing. His oxygen saturation was 91% in room air. There was no tracheal deviation, and chest excursions were equal on both sides. He had dull percussion notes with reduced air entry in the area of the middle & lower lobe of the left lung as well as coarse crepitations bilaterally. His pulse rate was 132/min and blood pressure of 95/60 mm/Hg. He had a hepatomegaly of 2 cm below the right costal margin with a span of 12 cm. An initial clinical diagnosis of severe bronchopneumonia, and he commenced empirically on intravenous antibiotics, oxygen therapy and other supportive care including a transfusion of 15 ml/kg of PRBC. Pediatric surgery consultation taken for abdominal distension and advice followed accordingly. Later antibiotics changed after 7 days as repeat investigations revealed no significant improvement and fever persisted. His full blood count showed haemoglobin 8.6 gm/dL, packed cell volume 28.2%, white blood cell 16.3X109/L, granulocytes 70%, lymphocytes 26.3% and platelets 295X109/L; CRP was 9.6 mg/dL; a chest X ray (Figure I) showed that patchy opacities were noted in perihilar and all zones of both lung fields was suggesting bronchopneumonia. Dense homogeneous opacity is seen in left lower zone obscuring left CP angle and extending along lateral chest wall-suggesting pleural effusion. His two blood cultures were negative, MT, Quantiferon TB gold, Gastric lavage MTB PCR all were negative for mycobacterium tuberculosis. Following persistent hypoxaemia, fever a repeat chest x-ray was done by the 10th day on admission (Figure II), showed Patchy opacities are noted in both perihilar and all zones of right lung field-suggesting bronchopneumonia. Dense homogeneous opacity is seen in left lower zone obscuring left CP angle and extending along lateral chest wall-suggesting pleural effusion. A CT chest was done on 12th day of admission (Figure III), showed necrotizing pneumonia in left lower lobe with mild pleural effusion. Right lower lobe pneumonitis.

Figure I: Posteroanterior view of chest radiograph at presentation showing Patchy opacities are noted in perihilar and all zones of both lung fields- suggesting bronchopneumonia. Dense homogeneous opacity is seen in left lower zone obscuring left CP angle and extending along lateral chest wall- suggesting pleural effusion.

Figure II: Radiograph at 10th day of admission showing Patchy opacities are noted in both perihilar and all zones of right lung field-suggesting bronchopneumonia. Dense homogeneous opacity is seen in left lower zone obscuring left CP angle and extending along lateral chest wall– suggesting pleural effusion.



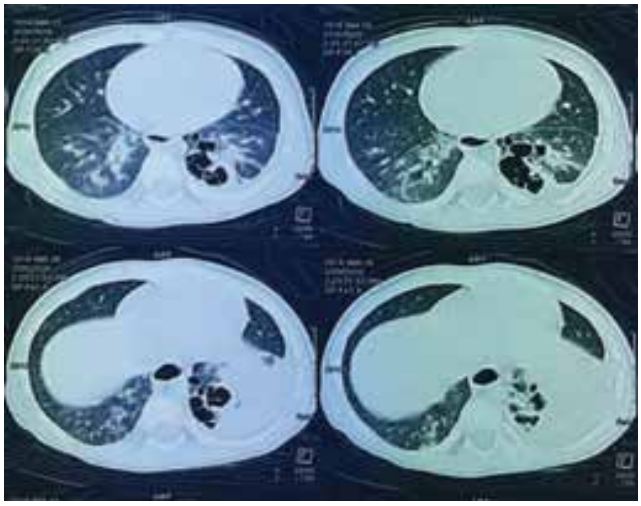


Figure III a, b: CT chest showing (a-coronal & b axial view) Multiloculated cystic area with destruction of the pulmonary architecture is noted in basal segment of left lower lobe. Dense band like opacity is seen adjacent to cystic area. There are diffuse ground glass opacities with pulmonary vascular congestion noted in right lower lobe. Mild left sided pleural effusion is noted. Necrotizing pneumonia in left lower lobe with mild pleural effusion. Right lower lobe pneumonitis.

He became afebrile for 72 hours on 17th day of admission then again became febrile. His diagnosis was reviewed to that of a NP, and antibiotics cover was broadened accordingly. A cardiothoracic surgical review suggested a non- operative approach (to avoid the surgical risk of development of a bronchopleural fistula), with continued conservative management. He remained pyrexic, but there was no oxygen dependency by the 24th day on, with gradual stabilization of his vital signs, he was planned for discharge on 25th day of admission. The chest X ray done at the point of discharge (Figure IV) showed right sided pneumothorax with lung collapse. He was discharged on the 24th day on admission, with oral antibiotics and other supportive care.

Figure IV: Posteroanterior view of chest radiograph at presentation showing Patchy opacities are noted in both perihilar

Discussion

The present case is of relevance as tropid respons to intravenous antibiotic. The major pathogens are *Streptococcus pneumoniae* and *Staphylococcus aureus* and the diagnosis should be considered when, despite appropriate antibiotics, the child remains febrile and unwell with persistent signs of respiratory distress and pneumonia. Other respiratory bacterial and fungal pathogens reported occasionally in studies included in the present review are *Streptococcus pyogenes*, *Streptococcus anginosus* group, Haemophilus influenza, *Pseudomonas aeruginosa*, Stenotrophomonas maltophilia and anaerobic organism. Viruses are rarely the sole cause of NP. The diagnosis of NP should always be considered in a child with pneumonia who remains unwell, despite at least 72 hours of appropriate antibiotics. Occasionally children with NP can deteriorate rapidly following their initial presentation with features of severe sepsis, including septic shock, multi-organ failure, and hypoxic respiratory failure[7].

Contrast-enhanced chest CT scans are more sensitive than chest radiographs and have become the standard imaging procedure for making the diagnosis of NP, evaluating the lung parenchyma changes not visible on plain chest radiographs, and determining whether any underlying congenital lung malformations exist[8]. The key diagnostic features are poor or absent vascularity, loss of pulmonary architecture, and finally cavity formation.

Histopathological findings in autopsy and surgical lung specimens from adults and children with NP are characterized by necrosis of lung parenchyma, which

was thought primarily to be a vascular process triggered by infection leading to vasculitis, activation of the coagulation system and thrombotic occlusion of intrapulmonary vessels accompanied by cavity formation[2,8-9]. The mixture of coagulation and liquefactive lung necrosis leads to one or more thin-walled cavities that can form pneumatocoels from the one-way passage of gas[10], or evolve into pulmonary abscesse[8].

A multi-disciplinary team of paediatric respiratory physicians, paediatric or thoracic surgeons, and infectious diseases experts is often required. The overarching aims are to control and ultimately reverse the patho-biologic changes associated with NP. These include providing supplemental oxygen to relieve hypoxia, ensuring adequate analgesia to reduce pleuritic pain and improve ventilation, administering prolonged antibiotic therapy, and decreasing any mass effect or intrathoracic pressure by draining gas and/or intra-pleural fluid[8-9,11].

A prolonged course of IV antibiotics is the cornerstone of therapy. The initial choice of antibiotics in otherwise healthy, fully immunized children should be the same as for empyema and cover gram positive organisms, especially pneumococci, Staphylococcus aureus and Streptococcus pyogenes[12] taking into account local epidemiologic and microbiologic data. Treatment can then be tailored according to microbiological results, although these may only be positive in 8.0% to 55.0% of cases. The optimal duration for antibiotic treatment of NP is unknown. The median length of antibiotic courses ranges from 13 to 42 days, with 3 of the 5 studies providing these data reporting a median antibiotic course duration of 28 days[3].

Switching from intravenous to oral antibiotics is appropriate once the child is afebrile for at least 24 hours and no longer showing signs of sepsis, their respiratory distress is resolving, enteral feeds are being tolerated, and inflammatory markers are declining[13]. At this point antibiotics are continued for at least another 10 to 14 days, a recommendation that aligns with consensus guidelines for paediatric CAP complications. The use of clindamycin has been shown to ameliorate the effect of PVL by binding to the 50s subunit of the microbial ribosome, thereby diminishing toxin production. Other drugs with similar effect include linezolid, rifampicin and fusidic acid[14].

Surgical intervention with either video-assisted thoracoscopic surgery or minithoracotomy to debride pyogenic material around the lung (decortication), breakdown loculations, and remove pus may be required when fever, signs of sepsis and/ or respiratory distress continue, despite chest tube insertion with or without fibrinolytic therapy and repeat imaging shows persistent or increasing intra-pleural collections[15].

Conclusion

NP is a serious complication of invasive pneumonia. This possibility should be considered when patient with complicated pneumonia fails to respond to optimal antibiotic therapy and closed chest tube drainage. CT scan is the standard diagnostic tool for NP and should be obtained without delay once this diagnosis is suspected. CT scan also plays an important role in the decision making for surgery because of the clearly definable findings. About half of the patients with NP can be successfully treated with medical therapy while the remaining patients require surgical intervention. Early diagnosis of this condition is crucial since it will influence the length and modalities of treatment.

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Conflict Of Interest
The authors have no conflicts of interest to disclose

Authors' contributions
Ferdous J were involved in the conceptualization of the study, data collection, and preparing the initial manuscript draft. Hossain MI was involved in the literature review, revising, and preparing a final manuscript draft. Ferdous J were involved in the literature review, revising, and editing of the manuscript. Both authors accepted and approved the final version of the manuscript.

Data Availability
The data used to support the findings of this study are included within the article.

Ethics Approval and Consent to Participate
Written informed consent for publication of the patient's clinical details and images was obtained from the patient's legal gurdians prior to submission. A copy of the signed consent form is available for review by the editor of the journal.

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