Section: Paediatrics

Original Research Article

# SEVERE NECROTIZING PNEUMONIA IN CHILDREN: A CHALLENGE TO INTENSIVE CARE SPECIALIST

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ABSTRACT

**Background:** Necrotizing pneumonia (NP) is recently recognized as a complication of pneumonia. The data on NP are scant from developing world and we aimed to describe the characteristic features of NP in our children. Single centre retrospective cohort analysis. Institutional database of children treated for pneumonia between September 2024 and May 2025 was searched to identify children with NP.

**Materials and Methods:** The demographic characteristics, laboratory results, and clinical information were recorded for patients selected as NP and analysed.

**Results:** In total, 10 patients (3.7%) of NP were identified out of 272 patients with pneumonia. Median age was 3 years (range: 3 months to 12years). All cases had severe respiratory distress and 70% required mechanical ventilation and inotropic support. The causative pathogens were identified in 6/10 children (60%) with Staphylococcus aureus being most common (4/10). Pleural effusion and pneumothorax were seen in six cases. Four cases had bilateral pleural effusion and three had bilateral pneumothorax. Intercostal drainage (ICD) was placed in 70% and bilateral ICD was placed in 40% cases. Bronchopleural fistula (BPF) developed in two cases and one had bilateral BPF. Median [inter quartile range] ICD days and hospital stay were 9 (6–14) and 13.5 (7.5–18.5) days, respectively. Mean (6SD) total antibiotic (in hospital plus outpatient) days were 28.8 6 9.6 days. Four cases had airway hemorrhage and in three cases this was massive and fatal.

**Keywords:** Pneumonia, pleural effusion, staphylococcus aureus, airway haemorrhage

## INTRODUCTION

Pneumonia is the leading infectious cause of death among children under 5, killing over 2000 children a day. Pneumonia accounted for 15% of the 5.6 million under-5 deaths, killing around 880 000 children in 2017.[1] India accounts for majority of these deaths. Major complications of pneumonia include Paediatric acute respiratory distress syndrome (PARDS), pleural effusion, empyema, lung abscess and necrotizing pneumonia (NP). NP was first described in children in 1994,[2] Since then only a few retrospective series have been reported.[3–9] The finding in pneumonia is usually lobar pneumonia (diffuse consolidation involving the entire lobe of the lung) or bronchopneumonia/lobular pneumonia (suppurative inflammation localized in patches around bronchi which may or may not be localized to a single lobe of the lung).[10] Whereas NP represents a severe form of suppuration and histopathology is characterized by necrosis (coagulative and liquefactive necrosis) of lung parenchyma due to primarily a vascular process triggered by infection leading to vasculitis, activation of the coagulation system and thrombotic occlusion of intra-pulmonary vessels accompanied by cavity formation. This leads to one or more thin-walled cavities that can form pneumatoceles or evolve into pulmonary abscesses. Most cases of NP have prolonged hospital stay and require multiple interventions. Mortality varied from 0% to 56%.[3–9] Most of these studies are from the developed world, where mortality from pneumonia otherwise is very low compared with developing world. We undertook this, retrospective, observational study to review the cases of NP among children hospitalized at our centre and describe their

epidemiology, causative organisms, clinical characteristics, laboratory parameters, management strategies and outcomes.

## MATERIALS AND METHODS

**Case selection and definition:** Cases of NP among patients hospitalized at our hospital Paediatric Intensive Care Unit (PICU) and Paediatric High Dependency Unit from September 2024 to May 2025 were identified retrospectively using an electronic database of the hospital information system. A search for diagnosis of pneumonia, NP, pleural effusion, empyema and pneumothorax from discharge summary of hospitalized patient were done. Chest-X ray (CXR) reports of these patients were searched for term pleural effusion, blunting of costophrenic angle, pneumatoceles, breakdown areas or NP. Case records and CXRs of all these cases were analysed. Cases were identified as NP when the CXR revealed areas of parenchymal consolidation with lucencies within the area of consolidation or multiple thin-walled cavities or lung abscess (a cavity with air-fluid level).

**Data collection:** The information recorded consisted of demographic characteristics, laboratory results, microbiological culture data and clinical information originally obtained at the time of admission, during the admission, ventilation need, inotropic need, hospital stay and whether the patient died (in-hospital death). For this study, a febrile day was defined as any 24-h period during which the patient had a recorded temperature of 38C. Hypoxia was defined as any recorded oxygen saturation < 90% by pulse oximetry, measured on room air. The study was approved by the hospital's Institutional Research Council.

**Statistical analysis:** Demographic, clinical and laboratory variables were summarized by standard descriptive statistics. Categorical variables were compared using the v2 test or Fisher's exact test, as appropriate. Continuous variables were compared either using the t-test or non-parametric Mann–Whitney test. The p-values of < 0.05 were considered statistically significant.

**Radiology:** CXR of every case showed necrotizing changes (areas of parenchymal consolidation with lucencies within the area of consolidation or multiple thinwalled cavities/multiple cystic lucencies; Fig. 2). Pleural effusion and pneumothorax were seen in six cases; four cases had bilateral effusions and three had bilateral pneumothorax, respectively. CXR in one case showed a cavity with air fluid level in right lower zone computed tomography (CT) chest was done in four cases. Necrotizing changes were seen in three cases and in one case, a cavity with air fluid level suggestive of lung abscess was seen.

**Pleural fluid:** Pleural fluid analysis done in six cases revealed exudative effusion in all. Most common organism was Methicillin sensitive Staphylococcus aureus (50%; Table 1).

**Antibiotic therapy:** Ceftriaxone, Cloxacillin and Clindamycin were used in three cases. Ceftriaxone and vancomycin were started in four cases. In four cases, Clindamycin/ vancomycin was upgraded to Linezolid. Meropenem and vancomycin was continued in three cases and in one case Colistin was added as per sensitivity.

**Severity and support:** All 10 cases had severe respiratory distress; 70% of them had cardiorespiratory failure requiring mechanical ventilation and inotropic support (Table 2). All seven cases had severe hypoxemia with a high oxygenation index (Table 2). Empyema and pneumothorax were seen in six cases. Intercostal drainage (ICD) was placed in 70% and bilateral ICD was placed in 40% cases. Intrapleural fibrinolysis was done in three cases with alteplase.

**Complications course and outcome:** Broncho pleural fistula (BPF) developed in two cases, one had bilateral BPF. Four cases had airway hemorrhage and in three cases this was massive and fatal. The hemorrhage was in form of fresh blood in three children and in child with lung abscess it was along with pus. Local incision and drainage of abscess were done in two cases and one of them also required laparotomy for intra-abdominal pus collection. Median (IQR) ICD days and hospital stay were 9 (6–14) and 13.5 (7.5–18.5) days, respectively. Mean 6 (SD) total antibiotic (in hospital plus outpatient) days were 28.8 6 9.6 days. Three children died and all of them had massive airway hemorrhage. Comparing the survivors with non-survivors only.

## RESULTS

In total, 10 patients (3.7%) of NP were identified out of 272 patients as shown in flow diagram (Fig. 1). Nine patients had NP and one had lung abscess. Comorbidities or underlying diseases (e.g. congenital heart diseases, tuberculosis, HIV etc.) were not seen in any patients. Median age at presentation was 3 years [interquartile range (IQR) 1.1– 3.8 years; Table 1]. Two were infants and 60% were 2–3 years old. Two children were underweight (weight below 2 SD) and rest all had normal weight for age. Majority of children (70%) had prior hospitalization with antibiotics started before presenting to us. Two children had local site abscess; one had right leg, and another had right pinna abscess. All patients had fever and severe respiratory distress at presentation. Hypoxia (SpO2 < 90%) was there in 70% cases. Hypotensive shock was seen in 60% cases. Seven children had hemoglobin below 10 g/dl and three had below 7 g/dl. Leucocytosis was seen in 70% of children at presentation and all had leucocytosis during stay. Hypoalbuminemia was seen in 70% of children. Statistically significant factor was the presence of airway hemorrhage (p-value 0.03).

**Fig. 1.** Flow diagram showing selection of NP cases.

| | |
|---|---|
| Days of pre-admission antibiotics | 4 (2–12.25) |
| Prior hospitalization | 7 (70%) |
| Days of prior hospitalization | 2 (1–12) |
| Clinical features | |
| Fever | 10 (100%) |
| Height of fever (temperature ºC) | 39.3 6 0.6 |
| Cough | 9 (90%) |
| Chest pain | 3 (30%) |
| Vomiting | 3 (30%) |
| Shortness of breadth | 10 (100%) |
| Abdominal pain | 2 (20%) |
| Tachycardia for age | 9 (90%) |
| Tachypnoea for age | 10 (100%) |
| Severe retractions | 10 (100%) |
| Hypoxaemia ($SpO_2$ <90%) 7 (70%) | |
| Hypotension for age | 6 (60%) |
| Tracheo-mediatstinum shift 3 (30%) | |
| Dull chest percussion note | 6 (60%) |
| Decreased breadth sounds | 9 (90%) |
| Bronchial breadth sounds | 3 (30%) |
| Crepitations Laboratory values | 8 (80%) |
| Hemoglobin (g/dl) | 8.46 6 2.14 |
| Total leucocyte count (cells/ml) | 17 400 (5925–30 850) |
| Highest total leucocyte count (cells/ml) | 25 700 (17 450–42 850) |
| C-reactive protein (mg/l) | 10 (7.25-22.7) |
| Albumin (g/dl) | 2.87 6 0.78 |
| Pleural fluid analysis (n5 6) | |

**Fig. 2**. Representative CXR image of 2-year-old female hospitalized with NP. (A) CXR AP view show bilateral pneumothorax with bilateral chest tubes in situ. Bilateral lung parenchyma shows multiple cystic lucencies and reticular pattern. Endotracheal and Ryle tubes are also seen. (B) Repeat CXR of same patient after 6 weeks shows significant resolution of pneumothorax and parenchymal changes. Well-defined cysts are seen in right midzone and left lower zone 2. Representative CT image of 4-year-old male hospitalized with NP. The mediastinum (C) and lung (D) windows are show left hydro-pneumothorax and right pleural effusion. Bilateral lung consolidation with multiple air-filled cavities within the lung parenchyma seen here are consistent with NP.

**Prior medical history:** Pre-admission symptomatic 7 (4.5–11) days

Antibiotic received prior to 7 (70%) admission

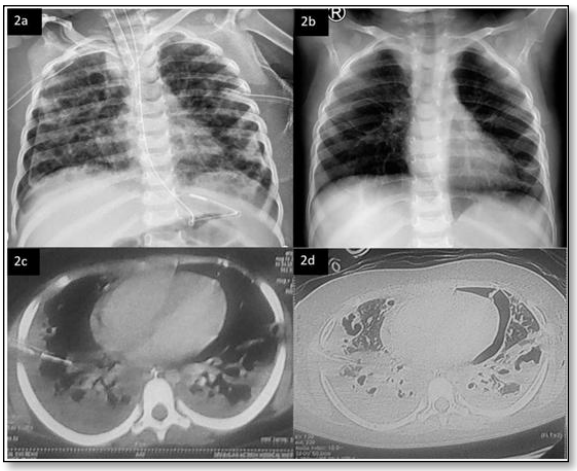


**Table 1: Demographic, clinical and laboratory parameters of patients with necrotizing pneumonia.**

| Parameters | Summary statistics |
|---|---|
| Age (years) | 3 (1.1–3.8) |
| | Male: female 1:01:00 AM |
| pH | 7.03 6 0.08 |
| Glucose (mg/dl) | 5 (4-43.5) |
| Protein (g/dl) | 3.45 6 0.69 |
| LDH (IU/l) | 2341 6 955 |
| Cell count (cells/ml) | 3550 (598.75–53 500) |
| Neutrophil % | 75.3 6 11.3 |
| Culture | 6 (60%) |
| Pleural fluid | |
| Blood | 1 (10%) |
| Organism MSSA | 3 |
| MRSA | 1 |
| Pseudomonas aeruginosa | 1 |
| Burkholderia cepacia | 1 |

Data are presented as mean 6 SD, median (IQR), n or n (%). LDH, lactate dehydrogenase; MSSA, methicillin sensitive S.aureus; MRSA methicillin resistant S.aureus.

**Table 2: Severity, support, course and outcome of patients admitted with NP.**

| Severity and support | |
|---|---|
| Prism III | 15 (7–17.75) |
| Pleural effusion/empyema | 6 (60%) |
| Bilateral pleural effusion | 4 (40%) |
| Pneumothorax | 6 (60%) |
| Bilateral pneumothorax | 3 (30%) |
| Broncho-pleural fistula | 2 (20%) |
| Airway haemorrhage | 4 (40%) |
| Intercostal chest tube drain | 7 (70%) |
| Bilateral intercostal chest tube drain | 4 (40%) |
| Intrapleural fibrinolysis | 3 (30%) |
| Mechanical ventilation | 7 (70%) |
| Maximum mean airway pressure | 18.5 6 2.7 |
| Maximum positive end expiratory pressure | 9.33 6 1.89 |
| $PaO_2/FiO_2$ | 112.83 629.74 |
| Oxygenation index | 20.61 69.81 |
| Inotrope support Surgery | 6 (60%) |
| Incision and drainage of local abscess | 2 (20%) |
| Laparotomy Course and outcome | 1 (10%) |
| Fever days | 5.5 6 2.94 |
| Intercoastal chest tube days | 9 (6-14) |
| Mechanical ventilation days | 5 (3-17) |
| Hospital stay | 13.5 (7.5-18.5) |
| Total antibiotic days | 28.8 6 9.6 |
| Deaths | 3 (30%) |

Data are presented as mean 6 SD, median (IQR), n or n (%). PaO2, partial pressure of oxygen in arterial blood; FiO2, fraction of inspired oxygen.

## DISCUSSION

This series identified 10 cases of NP in previously healthy children. The frequency of NP, prior hospitalization, antibiotics received, presence of pleural space disease, pleural fluid characteristics, interventional need and length of hospital stay are comparative to previous studies.[3–7] Streptococcus pneumoniae was reported as most common organism by Sawicki et al.[5] whereas Lemaitre et al like our series reported S.aureus as most common organism.[6] Mortality rate was higher in our study as compared with these studies.[5, 6] However, severity of illness, presence of shock and need for mechanical ventilation were not defined in these studies.[5, 6] Gillet et al.[8] and Taffarel et al.[9] have shown a mortality rate of 56% and 18% due to S.aureus NP. The severity of illness, presence of shock and need for mechanical ventilation in these studies are comparable to our series.[8, 9] All our NP cases were clinically severe pneumonia. More than two-third had cardiorespiratory failure at presentation and 70% cases required mechanical ventilation, ICD and inotropic support. Bilateral ICD were required in 40% cases and intrapleural fibrinolysis in 30%. All cases who required ventilation had hypoxia as per severe PARDS and required high ventilatory settings. However, they behaved differently than PARDS children and tolerated high peak/plateau pressures well. High plateau pressure was not associated with poor prognosis. The only factor associated with mortality was airway hemorrhage in univariate analysis. We could not do multivariate analysis as the numbers were small. One child died 24 h after extubation due to massive airway hemorrhage. Another child ventilated for 17 days had fatal airway hemorrhage prior to planned extubation day. Third child with lung abscess referred with massive airway hemorrhage and shock died within 10 h of stay. She had continuous pus emanating from endotracheal tube. Other studies also show airway hemorrhage as significant factor associated with mortality.[8, 9] However the risk factors for airway hemorrhage are not well known. The role of CT chest is also not well defined in predicting hemorrhage. Severe NP cases present significant challenge to intensivist. Apart from requiring ventilation, inotropic support and ICD placement in coagulopathic state; two peculiar problems need special attention. First, these children had persistent growth of organism from pleural pus despite using susceptible antibiotics especially in cases of staphylococcal NP. The strategies found beneficial for infection eradication were—(i) better pleural source control by using fibrinolysis in selected cases, (ii) addition of either clindamycin, linezolid or rifampicin to cloxacillin or vancomycin. Second peculiar problem was airway hemorrhage—this was mainly seen in third week of illness when majority of cases were otherwise recovering from sepsis and local infection. The only child who survived airway hemorrhage did not have torrential bleed and responded to increase in ventilation setting and blood transfusion. We thought hemorrhage may be due to erosion of bronchial vessels in three cases, whereas in one it may be because of rupture of abscess into the bronchial tree. The timing of fatal hemorrhage in our cases during relative recovery

phase made it difficult to predict and plan accordingly.

One of the major factors in reducing under 5 mortality in countries like India is to identify severe cases of pneumonia and associated risk factor early. NP as severe complication must be a contributing factor to pneumonia mortality, despite the recent studies showing low case fatality.[3–6] The reason for this low mortality in these series may be due to (i) most studies are from developed world where mortality from pneumonia is low; (ii) case selection less than one-third had severe disease and those involving predominant pleural space disease were selected as NP. Although CT chest is more sensitive than CXR for diagnosing NP, its need and utility in clinically mild or non-severe disease is questionable. NP involves extensive tissue necrosis with destruction of normal lung architecture. Patchy necrosis can be seen even in bronchopneumonia cases and CT chest may show these mild cases as NP. In our opinion, the most significant prognostic factor in pneumonia is clinical severity followed radiological picture. Although there is overlap between alveolar space and pleural space disease (e.g. syn-pneumonic effusion in lobar pneumonia or lung infiltrates in empyema) it is the predominant involvement of alveolar or pleural space that determines the outcome. A simple flow diagram for pneumonia is presented in Fig. 3. It shows that in a clinically severe case, predominant alveolar space disease fulfilling PARDS criteria and mixed alveolar and pleural space disease with significant destruction of lung architecture (NP) carries the worst prognosis. Predominant pleural space disease has significant morbidity but has very low mortality.

Retrospective single centre design with small case numbers are the main limitations of this study. Still, this study provides useful information regarding severe NP from developing world. The use CXR instead of CT chest (gold standard) for categorizing NP and considering only PICU patients may have underdiagnosed the true number of NP cases. However, we feel severe NP cases can be reliably diagnosed with CXR. NP should be recognized as a complication of pneumonia distinct from PARDS, pleural effusion and empyema. It is important to identify the appropriate level of severity of pneumonia and its complications and refer these high-risk cases to specialized centre. Further prospective studies are required to better understand the clinical– pathophysiological correlation and better management strategies of NP.

## CONCLUSION

Necrotizing pneumonia (NP) is recently recognized as a complication of pneumonia. In our case series we studied demographic characteristics, laboratory results, and clinical information of NP and analyzed the findings. All cases had severe respiratory distress and most of the cases required mechanical ventilation and inotropic support. The most common causative pathogens were Staphylococcus aureus. Pleural effusion and pneumothorax were commonly associated. Prognosis is not good even in the best of the centre. Multiorgan failure, Bronchopleural fistula, massive airway hemorrhage etc were the most common cause of death.

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