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Case Report

A complex case of necrotizing pneumonia and parapneumonic effusion in a healthy 20-month-old child: Successful management with video-assisted thoracoscopic surgery and chest tube placement ☆

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ABSTRACT

Necrotizing pneumonia (NP) is characterized by destruction of pulmonary tissue, resulting in multiple thin-walled cavities. There are limited reports on NP and parapneumonic effusion cases in children associated with *Pseudomonas aeruginosa*. Currently, there is no consensus regarding the optimal timing for video-assisted thoracoscopic surgery (VATS) following failure of chest tube placement and antibiotic treatment. A healthy 20-month-old child was hospitalized with symptoms of community-acquired pneumonia, progressing to severe NP and parapneumonic effusion. Despite receiving broad-spectrum antibiotics and chest tube placement on the third day of treatment, the condition continued to deteriorate, prompting VATS intervention on the sixth day. The presence of a "split pleural sign" and extensive lung necrosis on chest computed tomography contributed to initial treatment failure. Multidrug resistance *P. aeruginosa* was identified through nasal trachea aspiration specimens on the eighth day of treatment, leading to an adjustment in antibiotic therapy to high-dose meropenem and amikacin. Subsequently, the patient became afebrile, showed clinical im-

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provement, and was discharged after 35 days of treatment. Through this case, we aim to emphasize an unusual pathogenic bacteria in the context of NP and the need for standardized surgical interventions in pediatric patients with NP.

Introduction

Necrotizing pneumonia (NP) is characterized by destruction of pulmonary tissue, resulting in multiple thin-walled cavities [1]. This condition predominantly affects children aged between 2 and 5 years and is an infrequent complication of community-acquired pneumonia (CAP), accounting for 3.7% of CAP cases [2]. The complication rate of pneumonia, including parapneumonic pleural effusion (PPE), empyema, NP, and lung abscess, is reported at 8.9% based on a retrospective study spanning nearly 2 decades by Masarweh et al. [3]. However, approximately 40% of pneumonia cases develop complications during hospitalization [3]. NP increases the risk of respiratory failure, PPE, empyema, pneumothorax, bronchopleural fistula, and septic shock. NP is also associated with increased mortality rates, prolonged febrile episodes, and extended hospitalization [2,4].

Antibiotics and surgical intervention are the 2 primary treatment modalities for cases of NP associated with PPE. A retrospective study involving 746 pediatric NP cases revealed 46.6% requirement for chest tube placement and 6.1% necessity for video-assisted thoracoscopic surgery (VATS) [2]. Although there is no consensus on the criteria for chest tube placement and VATS, studies indicated that the rate of surgical intervention for complicated CAP ranges from 38% to 77% [2,3,5]. The relatively high rate of surgical management, which includes chest tube placement, VATS, and lobular resection, emphasizes the necessity of early disease detection and prompt treatment.

There is a lack of large-sample studies on NP in pediatrics, as NP remains an uncommon complication of CAP. Many aspects of NP remain unclear, including risk factors, methods for early screening and diagnosis of NP in outpatients with CAP, the role of lung ultrasound, and the microbiological characteristics of NP-causing agents. Furthermore, there are no randomized controlled trials to inform guidelines on antibiotic therapy or the timing of surgical intervention following antibiotic treatment failure. We present a complex case of NP and PPE associated with the unusual pathogen *P. aeruginosa* in a healthy 20-month-old child. This case highlights the success of combined antibiotic therapy, chest tube placement, and VATS.

Case report

A 20-month-old female patient was admitted to Children's Hospital 2 due to fever and respiratory distress. The illness persisted for 7 days before hospitalization, initially presenting with upper respiratory infection symptoms such as coughing and rhinorrhea during the first 2 days. Subsequently, the patient developed a fever of 39°C and a worsening productive cough that did not respond to oral antibiotics (amoxicillin/clavulanic acid at 45 mg/kg/day, divided into 3 doses) over the next 3 days. The patient was diagnosed with pneumonia and treated with ceftriaxone (100 mg/kg/day, divided into 2 doses) at a lower-level hospital for 2 days. However, the symptoms did not improve, leading to the patient's transfer to Children's Hospital 2. No reported perinatal or psychomotor development disorders were reported, and no significant medical history was noted. The patient had received the complete series of the 5-in-one vaccine but had not received vaccinations against influenza and pneumococcus.

At Children's Hospital 2, the patient showed chest retractions, a heart rate of 150 beats per minute, a respiratory rate of 50 breaths per minute, weighing 11 kilograms, measuring 83 centimeters in height, and a SpO_2 level of 95% with supplemental oxygen delivered via a cannula 3 liter/minute. Lung auscultation revealed diminished breath sounds in the left lung and right-sided crackles. Examination of other organs did not detect any abnormalities.

The clinical progression and antibiotic treatment are illustrated in Figs. 1 and 2. During the first 3 days of antibiotic therapy, which included vancomycin, ceftriaxone, amikacin, and clarithromycin, the patient remained febrile, respiratory distress (requiring oxygen supplementation via nasal continuous positive airway pressure), and a chest X-ray revealed worsening pulmonary consolidation and pleural effusion (Fig. 3). The total vancomycin dose per day was modified based on the peak and trough levels of vancomycin (Table 1). The bedside lung ultrasound revealed consolidation and collapse of the left lung, with multiple hypoechoic lesions suggestive of NP. Additionally, there was a moderate amount of pleural effusion, with septations inside and pleural thickening (Fig. 4).

On the third day, the patient underwent chest tube placement, leaking turbid yellow pleural fluid. The pleural fluid analysis is presented in Table 1, with elevated LDH levels (3131 U/L) and reduced glucose levels (2.5 mmol/L), consistent with complicated PPE. Other laboratory tests are presented in Table 1.

From day 3 to day 6 post-treatment (following the chest tube placement), the patient's fever persisted without improvement despite a reduction in CRP levels. Approximately 100 mL of turbid yellow pleural fluid through the chest tube per day was collected. The patient received additional intravenous clindamycin and an increased dose of vancomycin guided by AUC24. Blood cultures yielded negative results for bacterial growth. Transthoracic doppler echocardiography revealed no abnormalities. The contrast-enhanced chest computed tomography (CT) showed NP, PPE, and "split pleural sign" (Fig. 5).

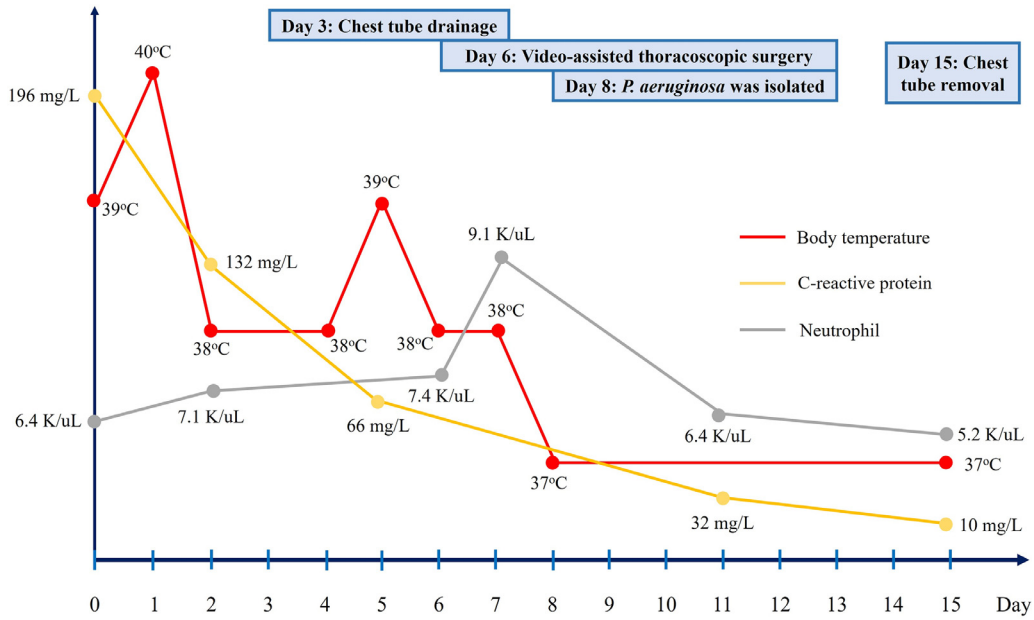

Fig. 1 – The progression of body temperature, C-reactive protein level, and neutrophil count from day 1 to day 15 after hospitalization.

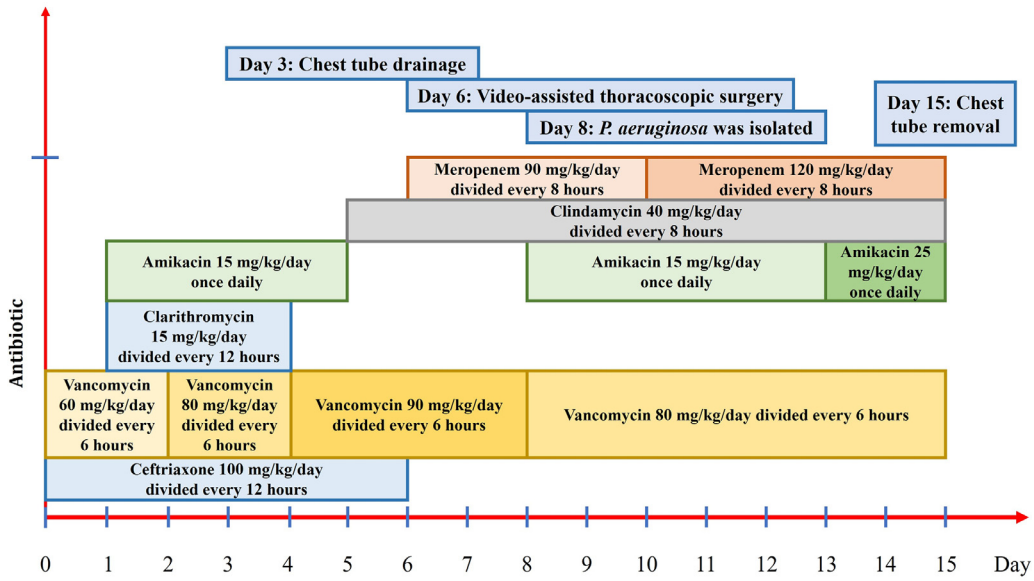

Fig. 2 – Summary of antibiotic types and duration of use by the patient.

The patient received meropenem supplementation based on the multiplex PCR results of nasal tracheal aspiration (NTA) presented in Table 3, indicating a high concentration of *Stenotrophomonas maltophilia*, *P. aeruginosa*, and *Streptococcus pneumonia*. During the VATS procedure on day sixth, nearly complete necrosis of the left upper lobe parenchyma with numerous pseudomembranes was observed. The pleural membranes of both upper and lower lobes were decorticated, pseudomembranes were removed, and adherent lung parenchyma was dissected. Multidrug-resistant *P. aeruginosa* (Table 2) was isolated from nasotracheal aspiration specimens (grade of Bartlett's criteria was 2 points), which were collected on the third day.

From the eighth day of treatment, the patient received additional amikacin, continued meropenem, vancomycin, and clindamycin. The patient's condition improved with the resolution of fever, reduced CRP levels, and decreased neutrophil count. Chest X-rays showed improvement from the sixth day to the 18th day of treatment (Fig. 6). The patient was discharged on the 35th day of treatment.

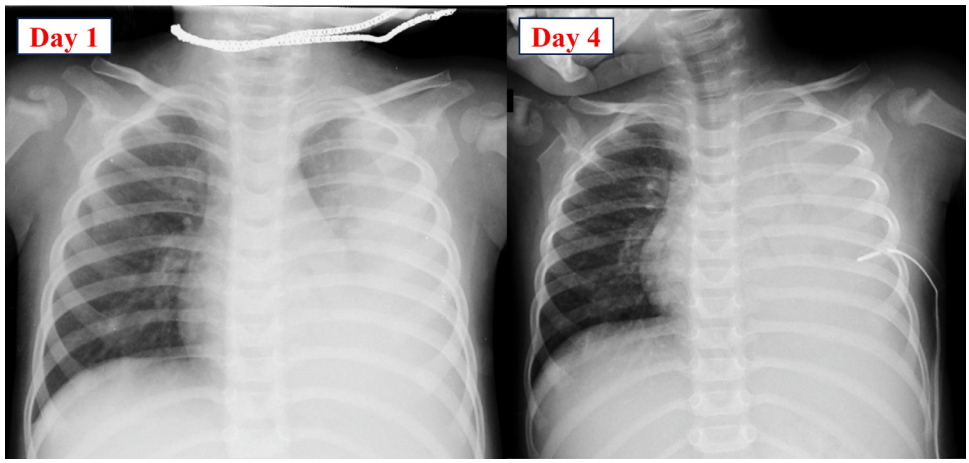

Fig. 3 – The chest X-ray from day 1 to day 4 post-hospitalization revealed progressing left lung consolidation and pleural effusion despite antibiotic treatment.

Table 1 – The patient's laboratory test results.

Laboratory tests	Normal range	Value
Blood		
Vancomycin trough level before fourth dose	μg/mL	2.28
Vancomycin peak level on the third day	μg/mL	22.25
Vancomycin trough level on the third day	μg/mL	5.57
AUC24 for vancomycin on the third day	μg/mL x hr	**360**
AST	<45 (U/L)	**135**
ALT	<40 (U/L)	5
Urea	1.67-7.49 (mmol/L)	2.2
Creatinine	20.33-88.4 (μmol/L)	30
Hemoglobin	10.5-14 (g/dL)	**9.8**
Platelet	150-400 (K/uL)	383
Chlamydia pneumoniae IgM		Negative
Mycoplasma pneumoniae IgM		Negative
Pleural fluid		
Protein	g/L	31.5
Glucose	mmol/L	2.5
LDH	U/L	3131
ADA	U/L	55
Neutrophil cellular component	%	80%
Mycobacterium tuberculosis PCR		Negative
Culture for bacteria		Negative
Nasotracheal aspiration specimens		
Culture for bacteria	Multi-drug-resistant *P. aeruginosa*	
GeneXpert MTB/RIF	Negative	
Mycobacterium tuberculosis PCR	Negative	
AFB	Negative	

ADA, adenosine deaminase; AFB, acid-fast bacilli; ALT, alanine aminotransferase; AST, aspartate aminotransferase; AUC24, the area under the concentration-time curve from 0 to 24 hr; MTB, *Mycobacterium tuberculosis*; PCR, polymerase chain reaction; RIF, rifampicin. Abnormal values are highlighted in bold.

Discussion

Our case describes a complex scenario of NP and PPE associated with an uncommon pathogen, *P. aeruginosa*, in a healthy 20-month-old child. The patient underwent successful treatment involving a combination of broad-spectrum antibiotics, chest tube placement, and VATS. Through this case, we intend to highlight the complexities of managing NP and PPE associated with *P. aeruginosa* in children.

Epidemiological studies illustrate inconsistent findings regarding risk factors for NP in children with CAP. Hsieh et al. conducted a retrospective study involving 71 children with NP due to *S. pneumoniae* in Taiwan, revealing that NP often progresses rapidly in previously healthy children. Identified risk factors for NP included an elevated immature neutrophil

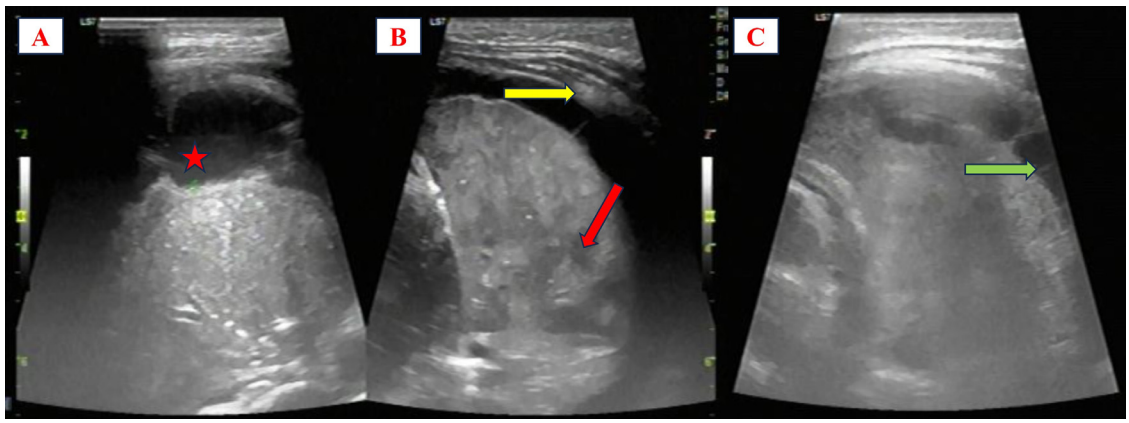

Fig. 4 – Bedside lung ultrasound on the third day. A shows echogenicity in the pleural fluid (red star). B shows multiple hypoechoic lesions within the left lung (red arrow) and pleural thickening (yellow arrow). C illustrates septation within the pleural cavity (green arrow).

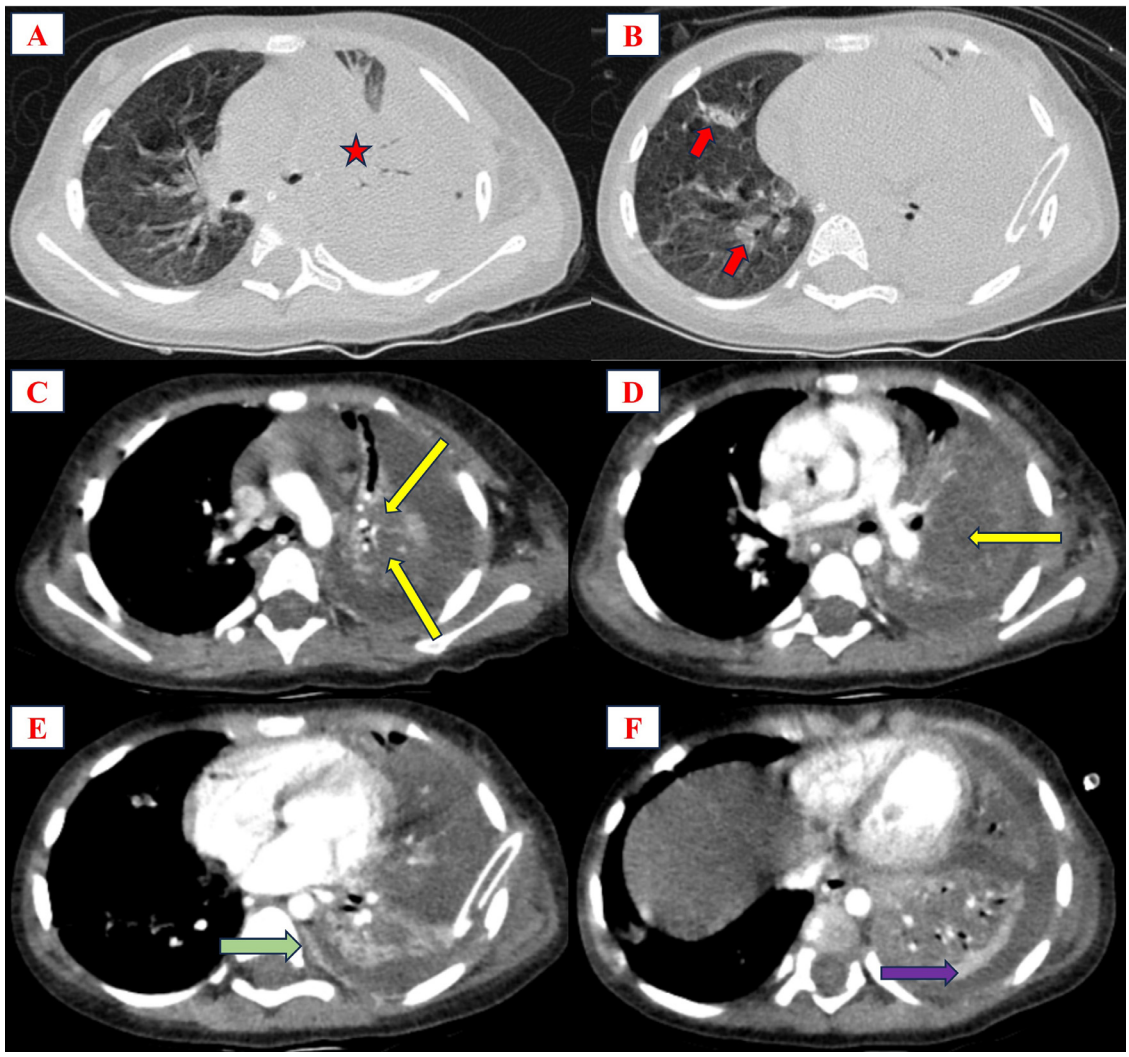

Fig. 5 – Chest CT on the fourth day. A and B showed consolidation in the left lung with an air bronchogram sign (red star) and scattered consolidations in the right lung (red arrow). C and D revealed low-attenuated areas inside the consolidation, suggesting NP (yellow arrow). E and F showed thickened parietal (green arrow) and visceral (pink arrow) pleura, referred to as "split pleural sign".

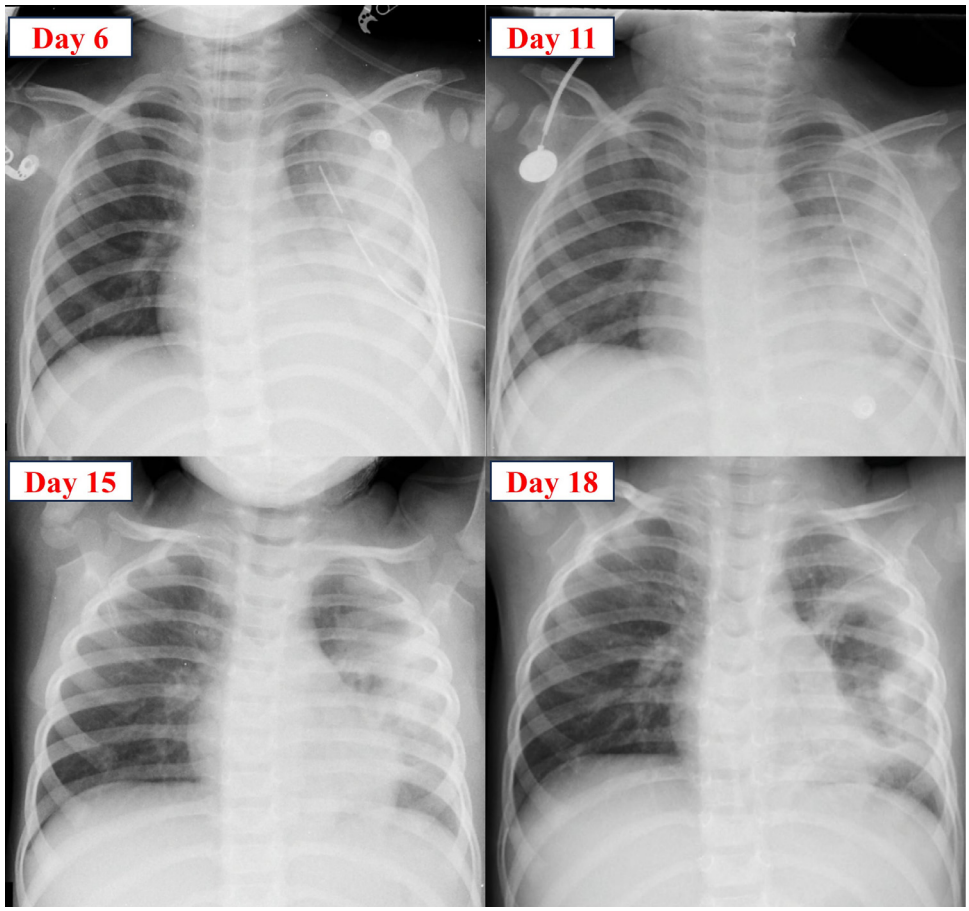

Fig. 6 – Progression of the patient's chest X-ray from the sixth day to the 18th day of treatment.

Table 2 – Antibiotic susceptibility of *Pseudomonas aeruginosa*.

Antibiotic	Result	MIC
Piperacillin-Tazobactam	Susceptible	16/4 μg/mL
Ceftazidime	Resistant	> 16 μg/mL
Cefepime	Susceptible	8 μg/mL
Imipenem	Resistant	> 8 μg/mL
Meropenem	Resistant	> 16 μg/mL
Gentamicin	Resistant	> 8 μg/mL
Amikacin	Susceptible	≤ 8 μg/mL
Ciprofloxacin	Intermediate	1 μg/mL
Ceftazidime-Avibactam	Resistant	> 8/4 μg/mL
Colistin	Intermediate	≤ 1 μg/mL

MIC, minimum inhibitory concentration.

count, C-reactive protein levels exceeding 12 mg/dL, and the absence of pre-existing medical conditions [6]. Conversely, another study involving 746 children with NP indicated that complex chronic conditions increased the risk of NP and mortality [2]. Other potential risk factors, such as asthma and prior NSAIDS usage, have yet to be confirmed [1]. Moreover, lower SaO_2 levels, higher fever, and elevated CRP levels in the complicated CAP group may predict the likelihood of requiring surgical intervention [3]. Although NP is a rare complication in children with CAP, clinical practitioners must remain alert to the potential rapid progression from CAP to NP in previously healthy pediatric patients.

P. aeruginosa is mainly recognized as a nosocomial infection and frequently causes diseases in immunocompromised, burned, or pediatric patients with cystic fibrosis [7]. However, *P. aeruginosa* is rarely reported as a pathogen in cases of CAP with NP in children. Common causative pathogens of pediatric NP include *S. pneumoniae, S. aureus, M. pneumoniae* [4], *Haemophilus influenzae*, and *Acinetobacter baumannii* [8]. A review of 197 bacterial or fungal isolates in children with NP revealed that 82% of NP cases were due to *S. pneumoniae* and *S. aureus*, with only 3 cases isolating *P. aeruginosa* (1.5%) [1]. The frequency of NP caused by *P. aeruginosa* is rare, as reported by Yonghan, accounting for only 1 out of 282 cases [8]. However, in children with complex chronic conditions, the rate of *P. aeruginosa* infection can increase up to 12.8%, compared to 4% in those without pre-existing medical conditions [2]. In adults, *P. aeruginosa* is recognized as a severe but rare causative agent of CAP, NP, or cavitation lung disease, often associated with a high mortality rate [9]. In our case, *P. aeruginosa*, resistant to ceftazidime and carbapenem, was isolated from NTA specimens. We cannot definitively confirm *P. aeruginosa* as the sole pathogen causing the disease; moreover, based on the multiplex PCR results, *S. pneumoniae* is also a potential coinfecting agent. We hypothesize that *P. aeruginosa*

Table 3 – The multiplex polymerase chain reaction results of nasal tracheal aspiration specimens.

Organism	CT	Result	Organism	CT	Result
Community-acquired bacteria			**Fungal**		
Streptococcus pneumoniae	28.14	7.47×10^5	*Pan Aspergillus*	(-)	-
Haemophilus influenzae	33.26	2.14×10^4	*Aspergillus fumigatus*	(-)	-
Haemophilus influenzae type B	(-)	-	*Aspergillus flavus*	(-)	-
Moraxella catarrhalis	(-)	-	*Aspergillus niger*	(-)	-
Streptococcus pyogenes (GAS)	(-)	-	*Aspergillus terrus*	(-)	-
Streptococcus agalactiae (GBS)	(-)	-	*Candida albicans*	33.34	2.03×10^4
Streptococcus suis	(-)	-	*Candida kefyr*	(-)	-
Nosocomial bacteria			*Candida tropicalis*	(-)	-
Staphylococcus aureus (MRSA)	(-)	-	*Candida krusei*	(-)	-
Staphylococcus aureus (MSSA)	(-)	-	*Candida glabrata*	(-)	-
Staphylococcus epidermidis (MRSE)	(-)	-	*Cryptococcus neoformans*	(-)	-
Staphylococcus epidermidis (MSSE)	(-)	-	*Pneumocystis jirovecii*	(-)	-
Coagulase-negative staphylococcus	32.11	4.76×10^4	*Penicillium marneffei*	(-)	-
Panton Valentine Leukocidin (PVL)	(-)	-	*Histoplasma capsulatum*	(-)	-
Enterococcus faecalis	(-)	-	*Fusarium oxysporum*	(-)	-
Enterococcus faecium	(-)	-	*Fusarium verticillioides*	(-)	-
Escherichia coli	(-)	-	*Coccidioides immitis/ posadasii*	(-)	-
Enterobacter cloaceae	(-)	-	*Sporothrix globosa*	(-)	-
Enterobacter aerogenes	(-)	-	*Sporothrix schenckii/ brasiliensis*	(-)	-
Klebsiella pneumoniae	(-)	-	*Mucormycosis (Rhizopus oryzae)*	(-)	-
KPC	(-)	-	*Fusarium solani*	(-)	-
NDM-1	(-)	-	**Virus**		
Pseudomonas aeruginosa	27.78	9.59×10^5	*Influenzavirus A*	(-)	-
Burkhoideria cepacia	(-)	-	*Influenzavirus B*	(-)	-
Burkholderia pseudomallei	(-)	-	*Influenzavirus C*	(-)	-
Acinetobacter baumannii	36.44	2.36×10^3	*Parainfluenzavirus 1*	(-)	-
Stenotrophomonas maltophilia	25.65	4.20×10^6	*Parainfluenzavirus 2*	(-)	-
Morganella morganii	(-)	-	*Parainfluenzavirus 3*	(-)	-
Providencia sp.	(-)	-	*Rhinovirus*	(-)	-
Proteus mirabilis	(-)	-	*Respiratory syncytial virus* (RSV)	19.14	3.84×10^8
Citrobacter freundii	(-)	-	*Human metapneumovirus*	(-)	-
Elizabethkingia meningoseptica	(-)	-	*Measles virus*	(-)	-
Fusobacterium nucleatum	30.13	1.88×10^5	*Adenovirus*	(-)	-
Atypical bacteria			*Epstein-Barr Virus (EBV)*	26.81	1.88×10^6
Mycoplasma	(-)	-	*Cytomegalovirus* (CMV)	33.2	2.23×10^4
Mycoplasma pneumoniae	(-)	-	*Bocavirus*	(-)	-
Chlamydia pneumoniae	(-)	-	*Varicella-Zoster Virus (VZV)*	(-)	-
Chlamydia trachomatis	(-)	-	*Common-cold virus*	(-)	-
Chlamydia psittaci	(-)	-	*Rubella virus*	(-)	-
Legionella pneumophila	(-)	-	*SARS-CoV-2*	(-)	-
Bordetella pertussis	(-)	-	**Mycobacterium**		
Bordetella parapertussis	(-)	-	*Mycobacterium tuberculosis*	(-)	-
			Nocardia asteroides	(-)	-

CT, cycle threshold.

is the causative pathogen, given the higher copies per mL of *P. aeruginosa* compared to *S. pneumoniae* in the multiplex PCR results. Furthermore, the clinical progression lacks a distinct resolution phase, minimizing the suspicion of *P. aeruginosa* superinfection in the hospital. However, determining the actual causative agent remains challenging; hence, antibiotic therapy should still encompass common pathogens to address this uncertainty. NP associated with *P. aeruginosa* in a previously healthy child adds complexity to the treatment, as it is an unusual pathogen in this population. This poses a significant challenge in NP management since empirical antibiotics covering *P. aeruginosa* are not routinely used in NP cases.

Complicated CAP should be considered if symptoms do not respond to appropriate treatment within 48 to 72 hours [10]. Patients should undergo screening for PPE, NP, lung abscess, sepsis, or metastasis infection. Chest X-rays show lower sensitivity in diagnosing NP than CT scans [1]. Donnelly et al. highlighted that 50% of cases with fluid-filled cavities may be undetected on chest X-rays [11]. In the early stages of NP, the liquefied lung parenchyma not connected to the airway may be challenging to notice on X-rays. In cases of suspected NP, a chest CT scan should be performed. Several studies have used lung ultrasonography as a potential alternative to CT scans in diagnosing NP [12,13]. Lung ultrasonography offers the advantage of being noninvasive and avoiding radiation exposure, especially in the pediatric population. Moreover, impaired perfusion and hypoechoic lesions observed on ultrasonography can predict pneumatocele formation and correlate with necrosis

in CT scans [12]. Our case encountered a delay in diagnosing NP, despite the potential for earlier diagnosis through lung ultrasonography. The lung ultrasonography images from the case correlated with findings on CT scans, including pleural thickening, NP, and PPE.

The optimal timing for VATS intervention in cases of treatment failure with antibiotics and chest tube placement has yet to be confirmed. In our case, despite receiving broad-spectrum antibiotics and chest tube placement, the patient's condition did not improve. This could be attributed to the extensive pleural thickening and widespread lung parenchymal necrosis contributing to treatment failure. VATS may aid in removing fibrous walls within the pleural cavity, removing pleural peel to allow lung re-expansion, and eliminating pus from the pleural space under direct visualization. VATS is the first-line intervention when antibiotic treatment and chest tube placement fail, rather than an open thoracostomy [14]. Some authors define complicated NP as extensive necrosis or cavitation larger than 50% of the involved lung lobe [15,16]. Cases of complicated NP tend to require wedge resections or lobectomies, and postoperative complications, pneumothorax, and more extended hospital stays are frequently observed. The recommended timeframe for surgical intervention in cases of NP and PPE unresponsive to appropriate treatment is 7 days [14]. Other situations requiring surgical intervention include complex empyema with significant lung pathology (extensive pleural thickening, trapped lung), bronchopleural fistula, or secondary empyema [14]. Early VATS (e.g., within 24-48 hours of hospital admission) has been proposed by some authors due to its benefits in reducing hospital stay, complication rates, and the number of days with chest tube placement [17,18]. However, as VATS depends mainly on each center's and surgeon's experience, there is currently no consensus on the optimal timing for VATS procedures. Several studies have been conducted to predict extensive necrosis in NP using indicators such as CRP, serum albumin, IgM [19], lung ultrasonography [12], or IFN-γ [20]; however, there are still limited data and predictive factors for the likelihood of requiring surgery during hospitalization.

Conclusion

Our clinical case highlights the complexities of managing NP in children. Multidrug-resistant *P. aeruginosa* can contribute to severe community-acquired NP in a healthy pediatric patient. Further research is needed to predict the likelihood of *P. aeruginosa* infection in NP, as empiric antibiotic therapy does not initially cover *P. aeruginosa*. Pediatric NP inpatients require close monitoring of clinical progression, as there is no consensus on the optimal timing or method of surgical intervention. Successful treatment relies on broad-spectrum antibiotics, chest tube placement, and timely surgical intervention.

Patient consent

Written informed consent was obtained for the publication of this case report.

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