Radiology Case Reports 19 (2024) 1642–1645

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Case Report

# Invasive pneumococcal disease caused by non-vaccine *Streptococcus pneumoniae* serotype 24B in an immunocompetent child ☆,☆☆

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ARTICLE INFO

*Article history:*
Received 13 December 2023
Revised 17 January 2024
Accepted 18 January 2024

ABSTRACT

Invasive pneumococcal disease typically occurs in immunocompromised patients, although some vaccine strains of *Streptococcus pneumoniae* have been reported to cause invasive pneumococcal disease in immunocompetent vaccine recipients. In this study, we presented a case of a 16-month-old immunocompetent patient with lung abscess and empyema caused by nonvaccine *S. pneumoniae* serotype 24B. A consolidation occupying the right upper lobe in the chest computed tomography results, as observed at presentation, changed to thick-walled cavitary lesions at the end of a month of intravenous antibiotics, and antibiotics were

*Abbreviations:* CT, Computed tomography; IPD, Invasive pneumococcal disease; PCV, Pneumococcal Conjugate Vaccine.

☆ Acknowledgments: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. We are grateful to Dr Kenjiro Kosaki, Center for Medical Genetics, Keio University School of Medicine, for his important contributions to genetic testing and interpretation. The authors would like to thank Kimiko Ubukata (6-1-1 Shinjuku, Shinjuku-ku, Tokyo 160-8402, Department of Microbiology, Tokyo Medical University, Japan) for her important assistance with determining the serotype of the pneumococcal isolate.

☆☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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https://doi.org/10.1016/j.radcr.2024.01.048

*Keywords:*
Pneumococcal pneumonia
*Streptococcus pneumoniae*
Serotype 24B
Case report
Invasive pneumococcal disease

continued for a total of two months. To the best of our knowledge this is the first report that focuses on the risk of invasive pneumococcal disease caused by *S. pneumoniae* serotype 24B in an immunocompetent child.



## Introduction

Following the introduction of pneumococcal vaccination, the incidence of invasive pneumococcal disease (IPD) caused by nonvaccine serotypes of *Streptococcus pneumoniae* (*S. pneumoniae*) due to serotype replacement has increased [1–3]. Although serogroup 24 is a common nonvaccine serotype that is associated with IPDs in Japanese children [4], an appropriate management of serotype 24B infection has not yet been established. Thus, this study aims to report a case of an immunocompetent child with IPD caused by nonvaccine *S. pneumoniae* serotype 24B and successfully treated with a suitable two-month antibiotic regimen.

## Case presentation

A 16-month-old Japanese patient that had been immunized with the 13-valent pneumococcal conjugate vaccine (PCV) was admitted to another hospital and presented with a five-day history of fever and cough. The guardians of the patient were determined to have no sick contacts, environmental exposures, or respiratory disease except for acute bronchitis caused by influenza virus at three months of age. The patient was diagnosed with pneumonia based on consolidation in the right upper lobe as confirmed via chest X-ray. The patient's respiratory failure progressed even after empirical treatment with intravenous sulbactam/ampicillin was started. The patient was transferred to our pediatric intensive care unit within 24 hours of admission since the patient's respiratory condition and level of consciousness deteriorated.

The patient's vital status at presentation was as follows: weight 10 kg; heart rate 150 beats per min; blood pressure 133/95 mm Hg; respiratory rate 30 breaths per min; body temperature 36.6°C; and percutaneous oxygen saturation 91% on room air. Decreased breath sound on the right lung field and consciousness disturbance were recorded upon physical examination. Blood cell count showed mild neutrophilia and anemia with erythrocyte fragmentation and severe thrombocytopenia. Blood chemical analysis showed a high creatinine count, procalcitonin count, interleukine-6 count of 24 pg/mL (reference range lower than 5 pg/mL), and normal serum level of C-reactive protein count of 0.06 mg/dL (reference range lower than 0.14 mg/dL) and complement component. The chest X-ray findings indicated consolidation occupying the right upper lobe with air bronchogram by right side unilateral pleural effusion (Fig. 1), with a normal thymus and spleen. The pleural effusion obtained by the diagnostic tap was purulent. The patient was diagnosed with a lung abscess with empyema and acute respiratory distress

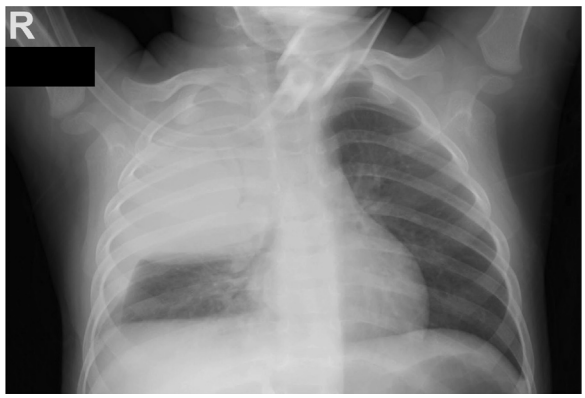


**Fig. 1 – Chest X-ray showing an opacity in the right upper field and pleural effusion in the right thoracic cavity at Day 1 of hospitalization.**

syndrome, which required 5 days of intubation. We started intravenous cefotaxime, vancomycin, azithromycin, and sulbactam/ampicillin upon admission to our pediatric intensive care unit. The intravenous antibiotics were replaced by panipenem due to the observation of renal impairment on day 3 of hospitalization. Moreover, we administered prednisolone (2 mg/kg/d) for acute respiratory distress syndrome from day 1 to 6 of hospitalization until extubation. We suspected hemolytic uremic syndrome, given the low platelet levels and decreased renal function, which did not require specific treatment, such as dialysis, and improved within 2 weeks. Enterohemorrhagic *Escherichia coli* was not detected by stool culture during the patient's treatment course. Furthermore, the patient's blood pressure normalized after appropriate antihypertensive treatment.

On day 2 of hospitalization, the real-time polymerase chain reaction analysis indicated *S. pneumoniae* genetic material in the patient's pleural effusions and sputum, while *S. pneumoniae* serotype 24B was grown from sputum cultures. It was found that two sets of blood and pleural fluid cultures were negative for *S. pneumoniae*. The serum C-reactive protein level decreased and remained below 0.01 mg/dL after day 3 of hospitalization. We replaced the treatment with ampicillin only according to antimicrobial susceptibility testing, which indicated susceptibility to ampicillin at day 15, and continued the intravenous antibiotic therapy for 30 days. Antimicrobial therapy was switched to the administration of oral amoxicillin (90 mg/kg/d), which was continued for an additional 30 days. The patient was discharged after switching to oral antimicrobials. After completing treatment, prophylactic doses of amoxicillin (20 mg/kg/d) and sulfamethoxazole and trimethoprim combination treatment (4 mg/kg/d) were continued for 18 and 30

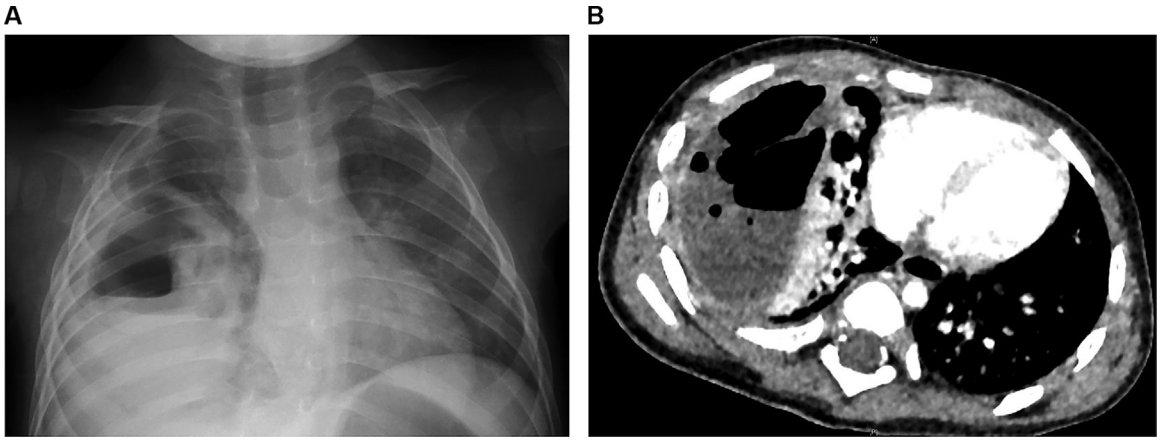


**Fig. 2 – Chest images on Day 30 of treatment. (A) shows the cavitary lesions with an air-fluid level in the right middle field on the chest X-ray; (B) shows an opacity in the right upper lobe and cavitary lesions with an air-fluid level in the right upper lobe on the chest computed tomography.**

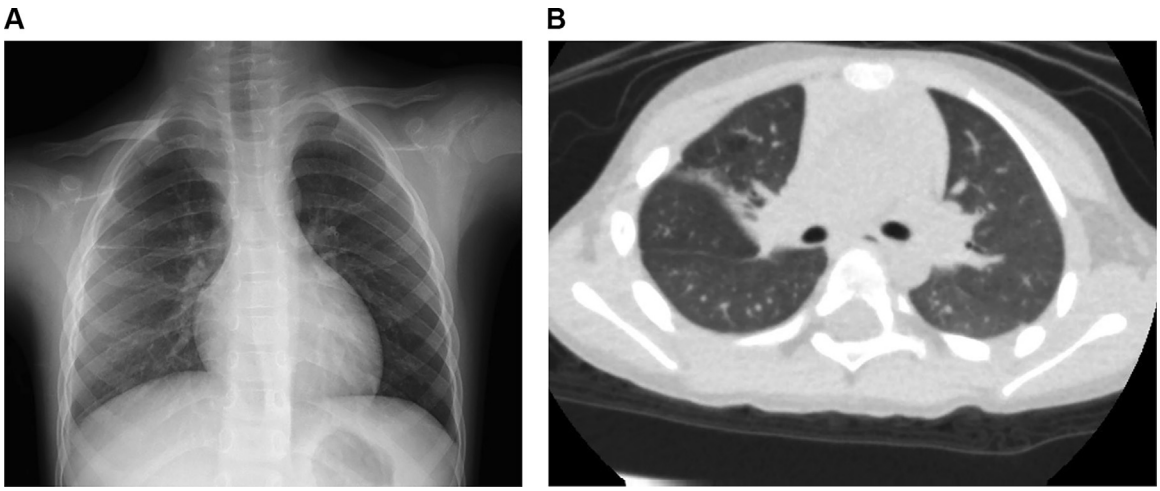


**Fig. 3 – Chest images showing linear shadows in the right upper lung field after treatment. (A) is a chest X-ray taken 4 years after treatment. (B) is a chest computed tomography taken 2 years after treatment.**

months until after receiving 23-valent pneumococcal polysaccharide vaccine at two years and meningococcus vaccine at three years of age.

The chest X-ray and computed tomography (CT) findings on Day 30 indicated a development of cavitary lesions with fluid inside and a residual pleural effusion (Figs. 2A and B). The chest imaging results indicated only a slight abnormality in the right upper lung field on CT at two years and on chest X-ray four years after treatment (Fig. 3A and B).

The patient did not develop any invasive bacterial disease during the period of 4 years following discharge. The patient had no laboratory findings indicating immunodeficiency, including targeted gene panel sequencing, which detected no variations in primary immunodeficiency-associated genes. However, transient mild hypocomplementemia was observed, which was restored to normal levels at 2 years of age.

## Discussion

The incidence of IPD caused by *S. pneumoniae* serotype 24B may continue to increase in the post-PCV era. After introducing pneumococcal vaccination, the incidence of IPD caused by nonvaccine serotypes has increased worldwide [2,3], especially in Japan, because of serotype replacement. Serotype 24B is not covered by either the current 13-valent PCV and 23-valent pneumococcal polysaccharide vaccine or the recently approved 15-valent [5] and 20-valent [6] PCVs. Although serotype 24B is not currently a common causative serotype of IPD, the incidence of IPD caused by serotype 24B is speculated to become more prevalent after global immunization with the current PCVs. Furthermore, reports are scarce on IPD caused by serotype 24B in immunocompetent children.

Serotype 24B has a high IPD complication rate [7]. In our case, the duration required to improve chest imaging was similar to that indicated in previous reports [8,9]. We considered that the complication of hemolytic uremic syndrome after the start of treatment was characteristic. Since the incidence of hemolytic uremic syndrome decreased after pneumococcal vaccination was introduced and cases were more likely to be associated with serotypes not covered in the 13-valent PCV [10], it is suggested that pediatricians determine whether serotype replacement due to vaccination also affects the type and severity of complications.

The long course of antimicrobial management (two months) was effective in treating serotype 24B infection in our patient. The duration of antibiotic therapy for lung abscesses has not yet been determined and depends on the patient's clinical and radiographic response. Antibiotic therapy should continue until fever subsides, putrid sputum improves, and abscess fluid clears (typically 5-21 days if antibiotics are administered intravenously), followed by oral antibiotics for 28 to 48 days [11]. Experts recommend continuing antibiotic therapy until the chest radiograph indicates that the lung abscess has resolved or a small, stable lesion has been observed [12]. Based on the findings of this report, to avoid the risk of relapse, we prolonged the treatment for two months, which was longer than usual. The improvement in imaging findings indicated satisfactory clinical and radiographic outcomes.

In severe lung abscesses, such as our case, it is necessary to evaluate the patient for congenital lung diseases, such as congenital pulmonary airway malformation [13] or immunodeficiency. Our patient presented no congenital lung diseases, as indicated by the absence of an abnormal shadow on the chest X-ray findings at 3 months of age. Immunodeficiency was suspected because the patient's serum C-reactive protein and interleukine-6 counts were low, despite the patient's severe condition at the beginning of treatment. However, genetic testing and general blood tests, including white cell count with differential and flow cytometry for cell populations, neutrophil function, and antibody level and function, did not confirm the diagnosis of primary immunodeficiency.

## Conclusion

This study reported a case of an immunocompetent child with complicated lung abscess and empyema due to *S. pneumoniae* serotype 24B. Although the patient was severe enough to cause disturbance of consciousness and hemolytic uremic syndrome, the patient improved clinically and radiologically with long-course antimicrobial management. This case suggests that clinicians should consider the possibility of IPD even in immunocompetent patients, and that extended antibiotic treatment may be appropriate in these cases.

## Patient consent

The guardians of the patient consented to this publication.

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