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Contents

Monthly Volume 16 Number 8 August 28, 2024

MINIREVIEWS

ORIGINAL ARTICLE

Retrospective Cohort Study

Retrospective Study

CASE REPORT

LETTER TO THE EDITOR

World Journal of Radiology

Monthly Volume 16 Number 8 August 28, 2024

Contents

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CASE REPORT

Pulmonary abscess caused by *Streptococcus pseudopneumoniae* in a child: A case report and review of literature

Ran Ma, Yan-Mei Wang, Hua Guan, Li Zhang, Wei Zhang, Ling-Cai Chen

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Abstract

BACKGROUND

Lung abscess found on chest X-ray and computed tomography examinations is rare in infants and young children. Several pathogens can cause lung abscesses, with the most common pathogens being anaerobes, *Streptococci* and *Staphylococcus aureus*. *Streptococcus pseudopneumoniae* (*S. pseudopneumoniae*) is a member of the *Streptococcaceae* family, and is mainly isolated from respiratory tract specimens. There are currently no cases of lung abscess caused by *S. pseudopneumoniae* in the literature.

CASE SUMMARY

A 2-year-old boy was admitted to hospital due to persistent cough and fever. Lung computed tomography examination suggested the formation of a lung abscess. His diagnosis was not confirmed by testing for serum respiratory pathogens (6 items), respiratory pathogen nucleic acid (27 items), and laboratory culture. Finally, metagenomic next-generation sequencing of bronchoalveolar lavage fluid revealed the presence of *S. pseudopneumoniae*, confirming its role in causing the lung abscess. After receiving antibiotic treatment, reexamination with lung computed tomography showed that the abscess was resorbed and the patient's outcome was good.

CONCLUSION

This is the first report of a lung abscess in a child caused by *S. pseudopneumoniae*

infection. Metagenomic next-generation sequencing of bronchoalveolar lavage fluid is helpful in achieving rapid and accurate pathogen identification.

Key Words: *Streptococcus pseudopneumoniae*; Lung abscess; Children; Bronchoalveolar lavage fluid; Metagenomic next-generation sequencing; Case report

Core Tip: This report describes a 2-year-old boy presenting with a lung abscess attributed to *Streptococcus pseudopneumoniae* (*S. pseudopneumoniae*) infection. Pulmonary abscess is uncommon in pediatric respiratory diseases, and can be caused by a variety of pathogens. Despite multiple etiological tests conducted upon admission, no pathogen was identified. Eventually, metagenomic next-generation sequencing (mNGS) of bronchoalveolar lavage fluid was employed and confirmed the presence of *S. pseudopneumoniae*. There are currently no reports of pulmonary abscess caused by *S. pseudopneumoniae* infection in the international literature. This case highlights the significance of mNGS in pulmonary infectious diseases, which is regarded as a complementary approach to conventional respiratory pathogen diagnostic techniques.

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INTRODUCTION

Lung abscess is a rare disease in pediatrics and occurs when pathogens such as viruses, bacteria, fungi or parasitic pathogens invade the lung parenchyma, leading to lung tissue necrosis and the subsequent formation of a purulent cavity [1]. Clinically, lung abscess can be diagnosed by chest X-ray and computed tomography (CT) examinations[2,3]. A variety of pathogenic infections can trigger lung abscesses, with *Streptococcus*, *Staphylococcus aureus,* and anaerobes being the most common[2-4]. We searched the databases, including China National Knowledge Infrastructure, Cqvip Database, Wanfang Database, PubMed, Uptodate, and Web of Science, without language restrictions from their inception to January 28, 2024. We used the following keywords in the search: *Streptococcus pseudopneumoniae* (*S. pseudopneumoniae*) [Title/ Abstract] AND Pulmonary Abscess [Title/ Abstract] OR Lung Abscess [Title/ Abstract], and did not find any cases of lung abscess caused by *S. pseudopneumoniae* infection. *S. pseudopneumoniae* and *Streptococcus pneumoniae* (*S. pneumoniae*) have high genetic similarity, and are difficult to identify only by phenotype and biomarkers. Studies have shown that *S. pneumoniae* and *S. pseudopneumoniae* can be distinguished by publicly-available genome sequences[5]. Patients with a lung abscess usually choose conservative treatment with intravenous antibiotics[3,6]. Several cases have shown that *S. pseudopneumoniae,* usually collected from respiratory tract samples, was resistant to penicillin, cephalosporin, macrolide, fluoroquinolone, and cotrimoxazole[7-10], and relying on empirical medication poses a significant risk of delayed diagnosis, potentially leading to life-threatening consequences.

We here report a case of a 2-year-old boy with lung abscess caused by *S. pseudopneumoniae* infection. On admission, pulmonary CT showed lung abscess, and general bacterial culture and identification, antibody detection, qPCR, tuberculosis smear of bronchoalveolar lavage fluid (BALF), and *Mycoplasma pneumoniae*-DNA in BALF did not identify the pathogen. Chinese clinical guidelines recommend that BAL by bronchoscopy is helpful in the diagnosis and treatment of pulmonary infectious diseases[11]. We finally obtained BALF and identified the pathogen by metagenomic next-generation sequencing (mNGS). Therefore, children with respiratory diseases whose pathogens cannot be identified by routine clinical testing should undergo BALF mNGS, which is helpful in achieving rapid and accurate pathogen identification[12].

CASE PRESENTATION

Chief complaints

A 2-year-old boy was admitted to The First Affiliated Hospital of Shihezi University due to a persistent cough for seven days and fever for five days.

History of present illness

The boy had symptoms of cough and fever, did not received any anti-infectious treatment outside the hospital.

History of past illness

The family denied any abnormal past illness history.

Personal and family history

The family denied any history of tuberculosis exposure or foreign body inhalation, and had normal birth history, feeding history, growth history and family history.

Physical examination

On admission, the patient's body temperature was 40 °C, heart rate was 120 bpm, respiratory rate was 31 breaths/min, and blood pressure was 80/55 mmHg. Physical examination revealed hyperemia of the pharynx, bilateral tonsil enlargement (I-degree), and the absence of purulent secretions. Upon auscultation, there were thick breathing sounds in both lungs, but no dry-wet rales.

Laboratory examinations

Following admission, the patient's laboratory tests indicators not improved (Table 1), routine blood testing showed the dominance of neutrophils, and the index of inflammation had increased significantly. Thus, we considered that the child had a bacterial infection. General bacterial culture, respiratory pathogen antigens (6 respiratory pathogens, IgM Antibody Detection Kit, Autobio, Zhengzhou, China), respiratory tract pathogen PCR (27 nucleic acid test kits for respiratory pathogens, Yingweipu, Zhejiang, China) all yielded normal results.

On the third day of hospitalization, with informed consent from his family, the child underwent BAL to identity the pathogen, and the tracheoscope (Figure 1) revealed congestion and edema of the bronchial mucosa in the upper lobe of the right lung with a little viscous secretion. Phagocytes are dominant in normal BALF, and the proportion of neutrophils increases during bacterial infection. Pathological smears of the collected BALF (Figure 2) indicated the presence of markedly elevated neutrophils. *Mycoplasma pneumoniae* DNA in BALF was lower than the detected minimum value (Instructions for *Mycoplasma pneumoniae* nucleic acid detection kit, Daan Gene, Guangzhou, China) (Table 1). The collected pulmonary alveolar lavage fluid samples were subjected to Q-mNGS™ quantitative metagenomics (Dian Medical Laboratory, Zhejiang, China), and the bioinformatics process included: (1) Quality filtering; (2) Elimination of replicate reads; (3) Removal of low-complexity reads matching the human genome sequence; and (4) Classification of reads by simultaneously aligning to the NCBI Database. The mNGS of BALF revealed *S. pseudopneumoniae*, by comparing the sequence similarity between the sequencing fragment and the known drug resistance gene in the Comprehensive Antibiotic Resistance Database (Table 1).

Imaging examinations

After admission, pulmonary CT revealed a lung abscess, and a follow-up lung CT scan revealed that the right upper lobe posterior lung abscess had discharged. A lung CT scan after hospital discharge revealed that the lung abscess had been absorbed (Figure 3).

FINAL DIAGNOSIS

The mNGS suggested two types of viruses, but the number of detected sequences was low and the disease course was long; thus, *S. pseudopneumoniae* infection was considered. Taking into account the clinical manifestations, the mNGS test, CT findings, and the therapeutic effect of antibiotics, the patient was diagnosed with pulmonary abscess caused by *S. pseudopneumoniae* infection.

TREATMENT

Treatment was initiated by administering empirical parenteral antibiotics, which was ceftazidime. His temperature returned to normal after 3 days of treatment, but his cough was still serious. Following BAL, the patient's cough was significantly relieved. Under ceftazidime treatment, the child's clinical symptoms improved, and *S. pseudopneumoniae* was considered sensitive to ceftazidime.

OUTCOME AND FOLLOW-UP

Reexamination of the lung by CT showed that the abscess had been resorbed and the patient's outcome was good. Table 2 indicated the timeline of the patient's course of illness.

Table 1 Laboratory tests conducted after admission

Laboratory test	Results	Reference value
WBC (L)	10.6×10^9	$5.1\text{-}14.1 \times 10^9$
Neutrophil count (L)	4.88×10^9 (46%)	$0.8\text{-}5.8 \times 10^9$
Lymphocyte count (L)	4.1×10^9 (38.7%)	$2.4\text{-}8.7 \times 10^9$
Monocyte count (L)	1.4×10^9 (12.8%)	$0.18\text{-}1.13 \times 10^9$
Hemoglobin (g/L)	106 g/L	107-141
Platelet count (L)	354×10^9	$190\text{-}524 \times 10^9$
C-reactive protein (mg/L)	65.45	< 10
Interleukin- 6 (pg/mL)	24.82	< 7.0
Procalcitonin (ng/L)	0.85	< 0.05
ESR (mm/h)	65	0-15
Blood coagulation function test	Normal	Normal
Immunoglobulin testing (5 items)	Normal	Normal
Liver function	Normal	Normal
Renal function	Normal	Normal
Myocardial enzyme	Normal	Normal
T-SPOT.TB, PPD, Tuberculosis smear (BALF)	Negative	Negative
Pharynx swab, Blood, BALF culture	Negative	Negative
Serum respiratory pathogen antibody (6 items)[1]	Negative	Negative
Respiratory tract pathogen PCR (27 items)[2]	Negative	Negative
Mycoplasma pneumoniae DNA (BALF)	$< 1.0 \times 10^4$	$< 1.0 \times 10^4$
mNGS (BALF)	Pathogens: *Streptococcus pseudopneumoniae*: 36449 sequences. *Human herpesvirus 5*: 926 sequences. *Human parainfluenza virus* type *3*: 2089 sequences	
	Antibiotic resistance genes: No antibiotic resistance genes were detected	
	Genes specific to *Streptococcus pseudopneumoniae: Pseudo_232, 901, 231, 902, 899, 228, 1764, 641, 1933 (5)*	
	PLY gene	

[1]The words in bold are obvious outliers. The six serum respiratory pathogens included *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Legionella pneumophila*, *enterovirus*, *respiratory syncytial virus* and *adenovirus*.
[2]The 27 respiratory tract pathogens included *Group B Streptococcus*, *H1N1 influenza A virus*, *H3N2 influenza A virus*, *H3 influenza A virus*, *H5 influenza A virus*, *H7 influenza A virus*, *pertussis bacterium*, *rhinovirus*, *Boca virus*, *Enterovirus*, *Streptococcus pneumoniae*, *Chlamydia pneumoniae*, *Mycoplasma pneumoniae*, *parainfluenza virus type 1*, *parainfluenza virus type 2*, *parainfluenza disease type 3*, *parainfluenza virus type 4*, *coronavirus*, *respiratory syncytial virus*, *Streptococcus pyogenes*, *influenza A virus*, *Moraxella catarrata*, *Haemophilus influenzae*, *human metapneumovirus*, *Legionella pneumophila*, *adenovirus*, and *influenza B virus*.
WBC: White blood cell; ESR: Erythrocyte sedimentation rate; BALF: Bronchoalveolar lavage fluid; T-SPOT.TB: Tuberculosis-specific enzyme-linked immunospot assay; PPD: Purified protein derivative; PCR: Polymerase chain reaction.

DISCUSSION

Lung abscess in children is a very rare condition that significantly reduces their quality of life. Children with lung abscesses may present with fever, fatigue, cough, shortness of breath, and chest pain[13]. In the case of the child described here, the focus was confirmed by transpulmonary CT. Empirical antibiotic treatment is commonly the initial therapeutic option for lung abscesses. However, in some cases, the specific pathogen is never identified, resulting in poor treatment outcome. Lung abscesses can be attributed to a diverse range of infectious pathogens, with *Streptococcus* and anaerobes being the most frequently encountered in clinical settings. However, there are numerous cases where the specific pathogen responsible for the infection remains unclear.

In the present case, the pathogen could not be identified by general bacterial culture and identification, indicating that the positive rate of this strain was low; as qPCR has the limitation of only detecting targeted pathogens, 27 common respiratory pathogens were found negative after admission, and no pathogens were isolated from blood or BALF culture. These findings further supported the crucial role of mNGS in confirming *S. pseudopneumoniae* infection. This highlights the potential oversight and misdiagnosis of infections caused by pathogens such as *S. pseudopneumoniae*. *S. pseudopneumoniae*, which was first reported in 2004, is closely related to the main human pathogen *S. pneumoniae*. It belongs to the

Table 2 Timeline of patient' s course of illness

Date	Symptoms onset	Imaging examination	Treatments administered	Follow-up outcomes
August 30, 2023	Persistent cough for seven days and fever for five days	None	Ceftazidime was given for anti-infective treatment	Temperature returned to normal
September 2, 2023	Temperature was normal, but cough was still serious	Pulmonary CT	Continue anti-infective treatment	Inflammation of the posterior upper lobe of the right lung with abscess formation
September 4, 2023	The frequency of coughing slightly decreased	None	Bronchoalveolar lavage	The frequency of coughing decreased.
September 8, 2023	The frequency of coughing obviously disappeared	Pulmonary CT	Continue anti-infective treatment	
September 14, 2023	None	None	Cefdinil was given for anti-infective treatment	Hospital discharge
October 9, 2023	None	Pulmonary CT	None	The abscess was resorbed and the patient's outcome was good

CT: Computed tomography.

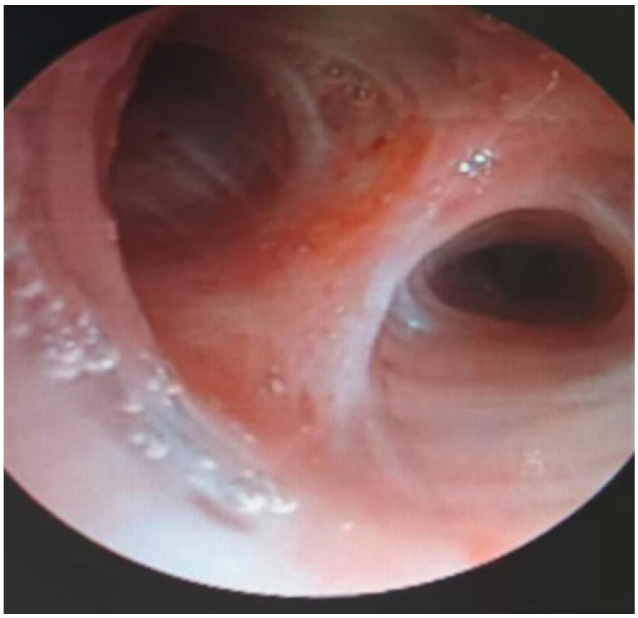

Figure 1 Bronchoscopic findings. The tracheal mucosa of the upper lobe of the right lung is congested and edematous with a little sticky secretion.

Streptococcaceae family and is characterized by the absence of a capsule, insolubility in bile, and resistance to optochin under 50 mL/L CO_2 conditions[14,15]. The appearance of sputum Gram staining smears in patients with *S. pseudopneumoniae* infection is similar to that of those with *S. pneumoniae*. The historical challenge in the detection of *S. pseudopneumoniae* using classical microbiological methods such as the optochin sensitivity test and bile solubility test resulted in a low detection rate[16]. Dupont *et al*[17] preliminarily identified 20 strains of *S. pseudopneumoniae* using routine methods, among these, 7 strains were identified as *S. pneumoniae* by Matrix-Assisted Laser Desorption Ionization Time of Flight mass spectrometry (VITEK MS®, bioMérieux). Gonzales-Siles *et al*[5] analyzed the gene sequence of the *Streptococcaceae* family and found that 9 genes were specific to *S. pseudopneumoniae* and 10 genes were specific to *S. pneumoniae*, these specific genes can be considered as potential gene biomarkers of these species. Following mNGS, nine of the *S. pseudopneumoniae* gene markers were also observed in the present case. Therefore, based on the phenotypic characteristics of *S. pseudopneumoniae*, genotyping methods such as PCR and gene sequencing are helpful for accurate identification[15, 18]. In this study, the BALF sample was sequenced on the Nanopore Gridion platform, and the sequencing results were further verified using the NCBI database to distinguish between *S. pneumoniae* and *S. pseudopneumoniae* (Figure 4). There is a file on the methods used and information on the mNGS procedure in the attachment.

S. pseudopneumoniae belongs to the *Streptococcaceae* family and is invasive. A study demonstrated that *S. pseudopneumoniae* can cause peritonitis/septicemia in mice[19]. Upon invading the respiratory tract, the strain's surface protein PspK plays a pivotal role in promoting colonization within the respiratory epithelial cells, leading to initiation of the infection process[20]. Virulence gene *PLY* in *S. pseudopneumoniae* kills various types of cells through pore-forming cytolytic activity.

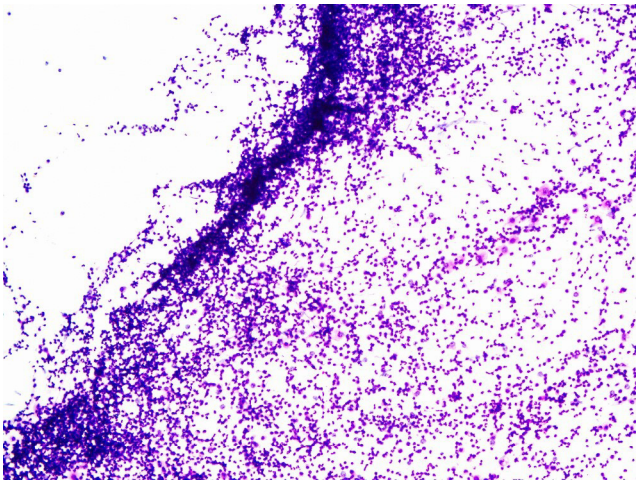

Figure 2 Bronchoalveolar lavage fluid pathological smear. Smear showing neutrophils (90%), mononuclear phagocytes (9%), and lymphocytes (1%).

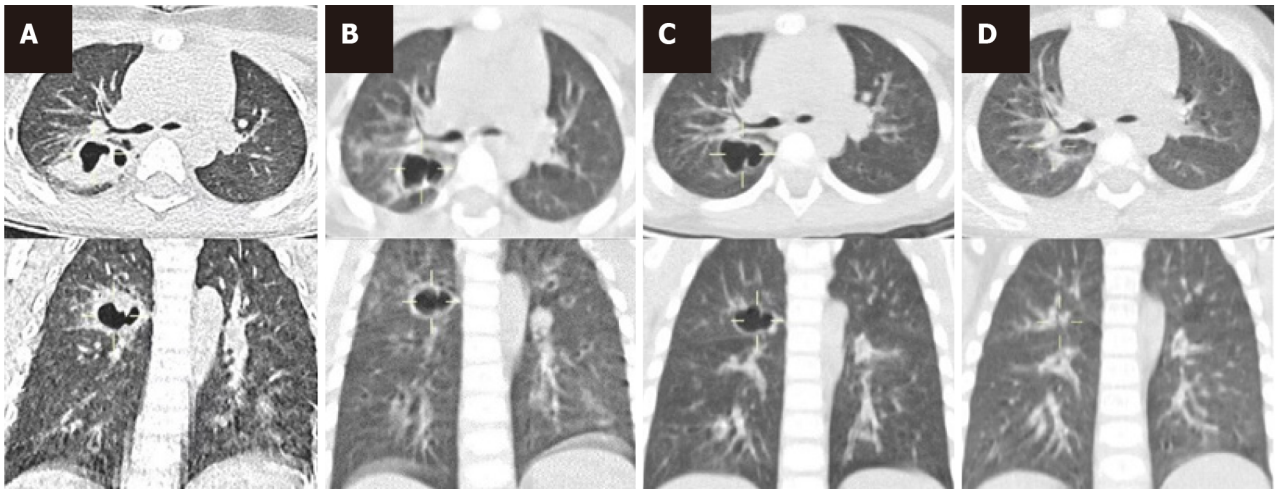

Figure 3 Computed tomography findings. A: Computed tomography (CT) scan showing inflammation of the posterior upper lobe of the right lung with abscess formation on September 2; B: CT scan showing an abscess in the posterior segment of the upper lobe of the right lung on September 8, which was more noticeable than that around the abscess on September 2; C: CT scan on September 22, showing the cystic cavity structure of the posterior segment of the upper lobe of the right lung, which was more absorbed than that around the film on September 8; D: CT scan showing focal pneumonitis in the posterior segment of the right upper lobe on October 9; Compared to the film on September 22, the thin-walled transparent focus of the posterior segment of the right upper lobe disappeared, and mild bronchitis was found in both lungs.

This activity not only triggers the activation of inflammatory responses but also exacerbates pulmonary inflammation[5,9,21-22]. In the context of the present case, the development of lung abscess attributed to *S. pseudopneumoniae* may be related to this mechanism. Some studies have reported that *S. pseudopneumoniae* infection leads to meningitis[23,24], and this association may be attributed to the role of *PLY* in promoting the transport of bacteria across the blood-brain barrier [25].

S. pseudopneumoniae, as an opportunistic pathogen, mainly causes respiratory infections. *S. pseudopneumoniae* can cause infection resulting in or aggravating chronic respiratory diseases. Ren *et al*[26] obtained BALF from patients with bronchiectasis and pulmonary infection for mNGS, and mNGS detected *S. pseudopneumoniae*, *S. pneumoniae*, and *Staphylococcus aureus*. Laurens *et al*[27] analyzed respiratory tract samples from 38 patients with pneumonia complicated by *S. pseudopneumoniae* infection, including 5 cases with single detection of *S. pseudopneumoniae* and 16 cases with detection of *S. pseudopneumoniae*, *Staphylococcus aureus*, and *Haemophilus influenzae*. In the present case, the diagnosis, confirmed by mNGS, showed lung abscess with *S. pseudopneumoniae*, *human parainfluenza virus* and *human herpes virus*, underscoring the occurrence of simultaneous detection involving *S. pseudopneumoniae*.

As a supplement to traditional methods, mNGS has a higher positive rate, higher sensitivity, and a wider pathogen spectrum[28], and has a diagnostic advantage in patients undergoing empirical treatment[29]. Therefore, it has obvious advantages in shortening the time to pathogen diagnosis and appropriate anti-infective treatment, and can greatly improve the prognosis of patients. However, the interpretation of results requires the help of experts, consolidation of clinical indicators, sample types, pathogen types and other factors, which makes the current application of mNGS in clinical detection controversial. This is why *S. pseudopneumoniae*, one of the three pathogens detected by mNGS, is considered the causative agent in this study.

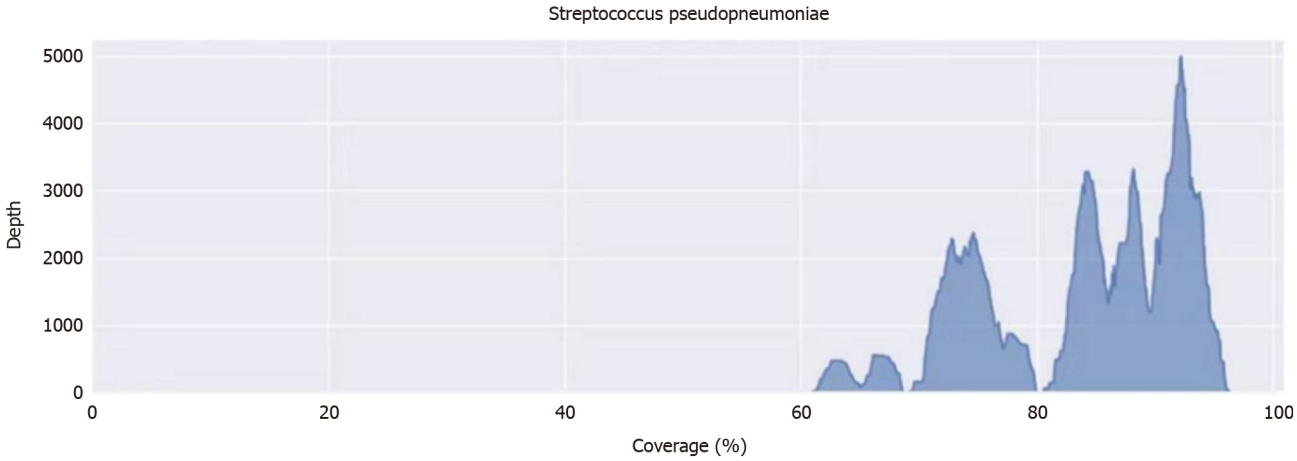

Figure 4 Information of metagenomic next-generation sequencing. The abscissa is the expansion subregion, and the ordinate is the sequence number multiplied by the sequencing length 50. Gene number of *Streptococcus pseudopneumoniae*: CP002925.1, information on the metagenomic next-generation sequencing is shown in the supplementary file.

In addition to intrapulmonary diseases, some cases of extrapulmonary diseases caused by *S. pseudopneumoniae* have been reported, including myocarditis and meningitis in newborns infected with *S. pseudopneumoniae*[23], recurrent tonsillitis caused by *S. pseudopneumoniae* infection in adolescents[30], and delayed blister-associated endophthalmitis in middle-aged patients with *S. pseudopneumoniae* infection[31]. Fuursted *et al*[32] noted that *S. pseudopneumoniae* infection in elderly patients can be secondary to sepsis associated with liver or bile-duct infections. Some middle-aged and elderly people may suffer from meningitis after *S. pseudopneumoniae* infection[24]. This case study revealed that *S. pseudopneumoniae* infection can lead to lung abscesses in young children. Thus, *S. pseudopneumoniae* infection can occur in all age groups.

It is reported that tetracycline and macrolide resistance are the two most common types of drug resistance[7]. Ghandi *et al*[23] reported that a neonate with meningitis and myocarditis caused by *S. pseudopneumoniae* was sensitive to erythromycin and azithromycin. In the present case, the comprehensive antibiotic resistance gene test did not detect the existence of any drug resistance genes, and the child was treated with ceftazidime on admission, and the therapeutic response was excellent, indicating that *S. pseudopneumoniae* was sensitive to ceftazidime. Therefore, children with *S. pseudopneumoniae* infection can be treated with macrolides and cephalosporins to observe the efficacy of these drugs.

CONCLUSION

S. pseudopneumoniae infection can cause lung abscesses. Clinically, in addition to symptomatic treatment, the causative microorganisms need to be identified promptly in patients with lung abscesses. In cases where routine laboratory examinations fail to identify the pathogen, advanced techniques such as serological antibody testing, PCR, and mNGS techniques could be employed. For instance, where pathogens have been identified, and targeted antibiotic treatments have not yielded satisfactory results, it is advisable to expedite the examination of drug-resistant genes. This allows for the selection of medications to which the infecting pathogen is not resistant, avoiding prolongation of the disease and an unfavorable prognosis. The use of mNGS has guiding significance in the discovery of atypical pathogens and the implementation of appropriate treatment.

FOOTNOTES

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