

SURGERY

ELIZABETHKINGIA SPECIES AS PATHOGENS OF ACUTE BACTERIAL LUNG DESTRUCTION IN IMMUNOSUPPRESSED PATIENTS (CASE REPORTS)

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RESUME

Background. *Elizabethkingia* spp. is still poorly explored pathogens with an intrinsic phenotype of multidrug resistance to several classes of antibiotics. In immunosuppressed and immunocompromised individuals, it causes acute bacterial destruction of the lung. Patients who have been taking antibiotics for a long time are more likely to develop nosocomial infections associated with the *Elizabethkingia* genus.

Case report. We report two cases of effective surgical treatment of bacterial lung destruction caused by *Elizabethkingia* species in immunosuppressed patients, and one of them also developed a nosocomial infection. *Elizabethkingia* spp. demonstrated polyresistance, which required a combination of antibacterial drugs and bacteriophage.

Conclusion. Bacteria of the genus *Elizabethkingia* belong to opportunistic human pathogens and can cause severe, mainly nosocomial infections in immunocompromised patients. The treatment presents a certain challenge due to their high natural resistance to most antimicrobial drugs traditionally used to manage infections caused by Gram-negative bacteria.

Keywords: *Elizabethkingia*, lung destruction, immunosuppressive patients, antibiotic resistance, nosocomial infection, antimicrobial susceptibility

Received: 05.04.2025

Accepted: 03.12.2025

Published: 25.12.2025

For citation: Ayushinova N.I., Fadeeva T.V., Chernychenko V.V., Vetokhina A.V., Grigoryev E.G. *Elizabethkingia* species as pathogens of acute bacterial lung destruction in immunosuppressed patients (case reports). *Acta biomedica scientifica*. 2025; 10(6): 199-207. doi: 10.29413/ABS.2025-10.6.21

БАКТЕРИИ РОДА *ELIZABETHKINGIA* – ВОЗБУДИТЕЛИ ОСТРОЙ БАКТЕРИАЛЬНОЙ ДЕСТРУКЦИИ ЛЕГКОГО У ИММУНОСУПРЕССИВНЫХ ПАЦИЕНТОВ (КЛИНИЧЕСКИЕ НАБЛЮДЕНИЯ)

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РЕЗЮМЕ

Обоснование. *Elizabethkingia* spp. до сих пор является малоизученным патогеном с врождённым фенотипом множественной лекарственной устойчивости к нескольким классам антибиотиков, возбудителем острой бактериальной деструкции легкого у иммуносупрессивных и иммунокомпрометированных пациентов. У больных, принимавших антибиотики в течение длительного времени, чаще развиваются внутрибольничные инфекции, связанные с родом *Elizabethkingia*.

Клинические наблюдения. Представлены два наблюдения эффективного хирургического лечения бактериальной деструкции легкого, вызванной этими бактериями у иммуносупрессивных пациентов, причем у одного из них развилась внутрибольничная инфекция. *Elizabethkingia* spp. продемонстрировали полирезистентность, что потребовало комбинации антибактериальных препаратов и применения бактериофага.

Заключение. Бактерии рода *Elizabethkingia* относятся к условно-патогенным инфекциям человека, но могут быть этиологическим фактором тяжелых инфекций, нередко внутрибольничных инвазий у иммуносупрессивных пациентов. Лечение их – непростая проблема по причине высокой природной устойчивости *Elizabethkingia* к большинству антимикробных препаратов, которые применяют для лечения инфекций, вызванных грамотрицательными бактериями.

Ключевые слова: *Elizabethkingia*, деструкция легкого, иммуносупрессивные пациенты, антибиотикорезистентность, нозокомиальная инфекция, чувствительность к противомикробным препаратам

Статья поступила: 05.04.2025
Статья принята: 03.12.2025
Статья опубликована: 25.12.2025

Для цитирования: Аюшинова Н.И., Фадеева Т.В., Черныченко В.В., Ветохина А.В., Григорьев Е.Г. Бактерии рода *Elizabethkingia* – возбудители острой бактериальной деструкции легкого у иммуносупрессивных пациентов (клинические наблюдения). *Acta biomedica scientifica*. 2025; 10(6): 199-207. doi: 10.29413/ABS.2025-10.6.21

INTRODUCTION

Elizabethkingia is a genus of gram-negative bacteria named in honor of Elizabeth Osborne King, an American bacteriologist and researcher who worked at the U.S. Centers for Disease Control and Prevention (1912–1966) [1, 2].

In 2005, it was determined that four microbial representatives belong to the genus *Elizabethkingia*: *E. meningoseptica*, *E. miricola*, *E. anopheles*, and *E. endophytica*. Of these, *E. meningoseptica*, *E. anophelis*, and *E. miricola* are the most significant from a medical perspective. *Elizabethkingia* is a pathogen of acute bacterial lung destruction in immunosuppressed and immunocompromised patients [3].

Elizabethkingia miricola was first discovered in water condensate from the Russian space research station "Mir" in 2003. It is an aerobic, non-fermenting, gram-negative rod-shaped bacterium that causes lung infections manifesting as the formation of pulmonary nodules, subsequent abscesses, pleural effusions, and sepsis. Most pathogenic bacteria are isolated from blood or sputum and respiratory tract samples. Infections associated with *Elizabethkingia miricola* typically have a high mortality rate (18.2–41 %) [4, 5].

Patients who have been taking antibiotics for an extended period are more likely to develop nosocomial infections caused by *Elizabethkingia* [2, 6].

Elizabethkingia spp. remains a poorly understood pathogen that exhibits an intrinsic resistance phenotype to multiple classes of antibiotics. *Elizabethkingia miricola* has natural resistance to cephalosporin and carbapenem antibiotics. *Elizabethkingia* isolates are resistant to nearly all β -lactam, aminoglycoside, colistin, macrolide, tetracycline, and vancomycin antibiotics. They demonstrate variable susceptibility to piperacillin/tazobactam, fluoroquinolones, minocycline, tigecycline, rifampicin, levofloxacin, and trimethoprim/sulfamethoxazole [7–11].

Antibiotic resistance in the genus *Elizabethkingia* is associated with the production of various types of β -lactamase enzymes, including extended-spectrum class A (ESBL), which confer resistance to β -lactam antibiotics such as cefotaxime, ceftazidime, cefepime, and carbapenem-hydrolyzing class B metallo- β -lactamases (MBL), associated with resistance to aztreonam and carbapenems, including imipenem [10, 11, 12]. Consequently, inadequate etiotropic therapy and empirical treatment for *Elizabethkingia* infections are ineffective. Timely administration of appropriate antibiotics becomes crucial in order to improve treatment outcomes and reduce mortality rates.

Delftia acidovorans is an aerobic, non-fermenting, gram-negative bacillus isolated in bacteremia, corneal ulcers, otitis media, and pleural empyema. Rare and quite serious infections caused by *D. acidovorans* mainly affect elderly patients with multiple comorbidities, including malignancies. It can cause infections in both immunocompetent and immunocompromised

patients. In polymicrobial combinations, *D. acidovorans* is most often found together with other common pathogens such as *P. aeruginosa*, *E. coli*, and *S. maltophilia*. Antibiotic susceptibility testing has shown resistance to aminoglycosides such as gentamicin and amikacin, colistin, and susceptibility to imipenem, meropenem, ceftazidime, piperacillin/tazobactam, glycopeptides, and fluoroquinolones. Carbapenems, cephalosporins, and piperacillin/tazobactam with antipseudomonal activity are considered for antibacterial therapy [3, 11].

In adult patients, bacterial lung destruction caused by *Elizabethkingia* species is rare in clinical practice [2]. We have not found descriptions of lung gangrene and abscess in HIV-infected patients and patients with acute leukemia, which served as motivation for this study.

AIM

To illustrate the efficacy of comprehensive targeted treatment of immunosuppressed and immunocompromised patients with lung destruction caused by *Elizabethkingia* spp. – a pathogen with an intrinsic phenotype of multidrug resistance to several classes of antibiotics.

In 2023–2024, bacterial lung destruction caused by *Elizabethkingia* spp. was diagnosed in two patients at the Department of Purulent Surgery of the Irkutsk Regional Clinical Hospital. This clinical case was performed in accordance with the recommendations and guidelines of the SCARE group [13]. The study was conducted in compliance with the ethical standards outlined in the Helsinki Declaration and Order No. 200n of the Ministry of Health of the Russian Federation, dated April 1, 2016 "On Approval of Rules of Clinical Practice". Patients provided voluntary informed consent for the publication of their clinical data in the public domain.

CLINICAL CASE 1

A 34-year-old patient with a history of approximately 10 years of exposure to human immunodeficiency virus (HIV) and who has been taking antiretroviral therapy intermittently was admitted to the hospital on March 29, 2023. One month prior to admission, the patient experienced the onset of symptoms such as cough and fever. The patient's condition was moderate in severity, with a saturation of 96 % and pale skin and pasty feet. The patient had a body temperature of up to 37.6°C. The complete blood count (CBC) revealed leukocytosis of $14 \times 10^9/\text{mL}$, with a left shift, and anemia with erythrocytes $2.7 \times 10^{12}/\text{mL}$ and Hb 71 g/L.

Multislice computed tomography (MSCT) of the lungs reveals: in the left pleural cavity, from the level of the first rib along the anterior surface, there is gas accumulation

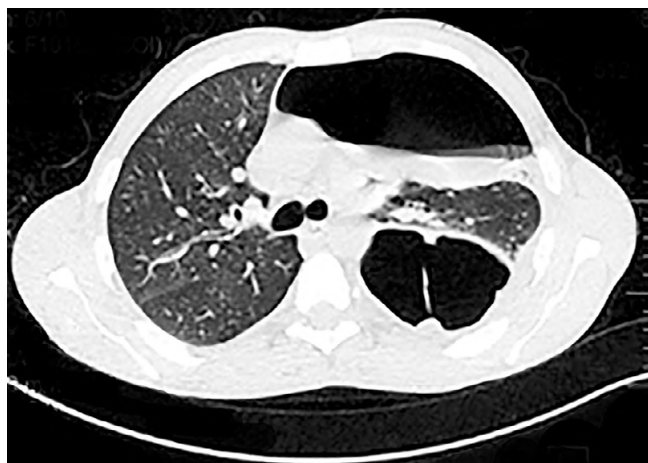
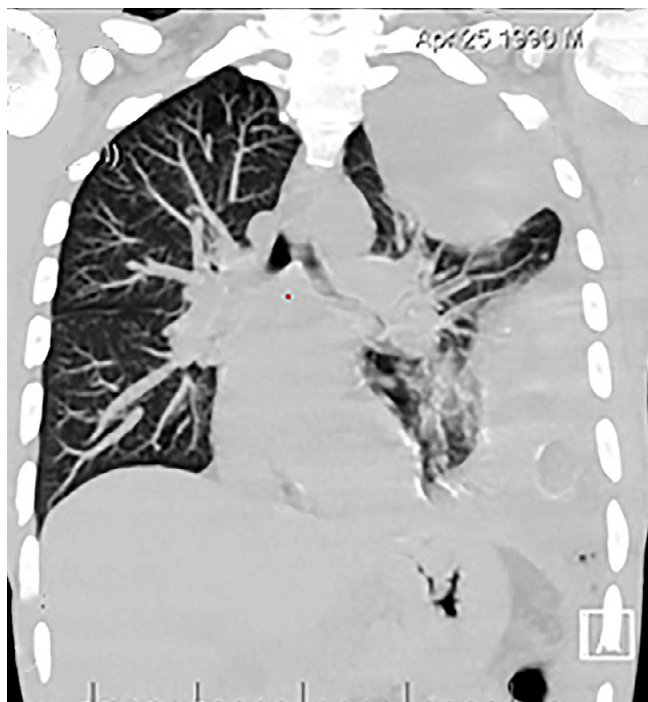


FIG. 1.
MSCT scan at admission to the clinic

with a fluid level measuring $142 \times 87 \times 250$ mm. From the 4th to 5th ribs along the posterior surface, a similar accumulation measuring $98 \times 73 \times 194$ mm is observed. The lower lobe of the left lung is hypopneumatic, with multiple cavitary lesions (Fig. 1).

The left pleural cavity was drained, resulting in the removal of 500 ml of pus and air discharge. *Elizabethkingia miricola* and *Delftia acidovorans* were isolated. The *E. miricola* isolate demonstrated multidrug resistance, including intrinsic resistance, to 13 out of 17 antimicrobial agents tested (Table 1). This included aminoglycosides (amikacin and tobramycin), fluoroquinolones (levofloxacin), cephalosporins (cefotaxime and cefepime), carbapenems (imipenem, meropenem, and ertapenem), and trimethoprim/sulfamethoxazole. In addition, resistance to inhibitor-protected penicillins (amoxicillin/clavulanic acid, piperacillin/tazobactam, ampicillin/sulbactam) and cephalosporins

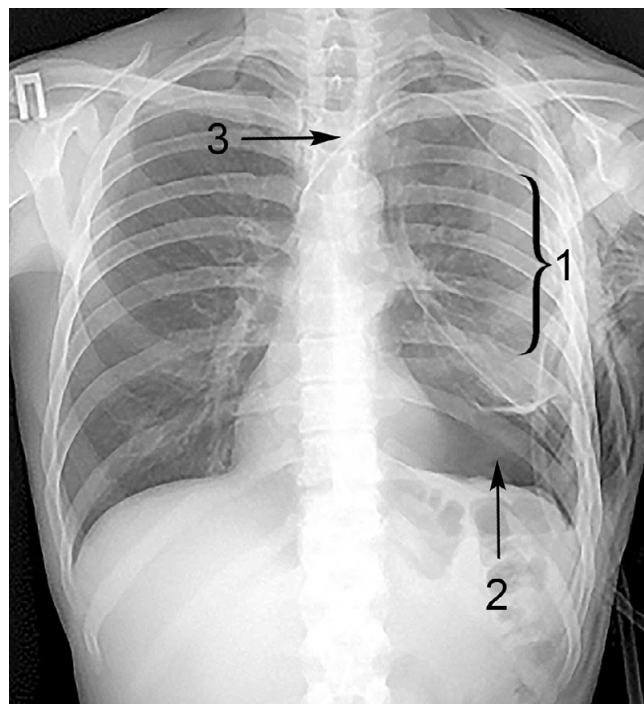


FIG. 2.
Survey radiograph on the 1st postoperation day. 1 – upper lobe; 2 – basal pneumothorax; 3 – catheter inserted via the subclavian vein

(ceftazidime/avibactam, ceftolozane/tazobactam) was detected. However, increased drug exposure resulted in intermediately susceptibility (I) for gentamicin, ciprofloxacin, and levofloxacin.

Delftia acidovorans showed susceptibility to increased exposure to imipenem, meropenem, and piperacillin/tazobactam. The organism was resistant to all other antibiotics tested, and no convincing evidence for the treatment efficacy with these drugs for this infection was obtained (Table 1).

Antibacterial therapy with levofloxacin 500 mg/day for 7 days, along with lavage of the pleural cavity using a 0.02 % chlorhexidine solution and blood transfusions, were administered. Due to the presence of hemoptysis, an endovascular occlusion procedure was performed on the branches of the 6th and 10th segments of the left pulmonary and bronchial arteries. This led to the cessation of bleeding. Following preoperative preparations, a lower lobectomy of the left lung was performed.

Pathohistological diagnosis: gangrenous abscess of the lower lobe of the left lung, with purulent-producing pleurisy.

On the 1st day after surgery, a chest X-ray was performed. The upper lobe of the removed lung appeared to be aerated, although it did not completely fill the half of the chest, with a persistent basal pneumothorax (Fig. 2).

On the 10th day of the postoperative period, *Pseudomonas aeruginosa* was isolated from the patient's pleural cavity at a concentration of 10^6 CFU/mL (Table 2).

TABLE 1

SUSCEPTIBILITY OF *ELIZABETHKINGIA MIRACOLA* AND *DELFTIA ACIDOVORANS* TO ANTIMICROBIAL AGENTS

Antimicrobial Agent	Isolated microorganisms, final result	
	<i>Elizabethkingia miricola</i> 105 CFU/ml	<i>Delftia acidovorans</i> 104 CFU/ml
Ceftazidime/Avibactam	R	R
Ciprofloxacin	I	R
Imipenem	R	R
Piperacillin/Tazobactam	R	I
Meropenem	R	I
Cefotaxime	R	I
Tobramycin	R	R
Amoxicillin/Clavulanic acid	R	R
Cefepime	R	R
Amikacin	R	I
Ertapenem	R	R
Ampicillin/Sulbactam	R	I
Gentamicin	I	R
Ceftolozane/Tazobactam	R	R
Levofloxacin	I	R
Trimethoprim/Sulfamethoxazole	R	R
Polymyxin E (Colistin)	R	R

Notes. S – susceptible, I – susceptible with increased drug exposure, R – resistant. *Elizabethkingia miricola* – intrinsic resistance to cephalosporins and carbapenems.

In the postoperative period, a course of antibiotic therapy with polymyxin E by inhalation was administered for 10 days. The follow-up radiograph showed that there were no signs of infiltrative changes in the resected lung; however, there was some opacification in the lateral sinus. The right lung appeared to be clear and without any evidence of sinuses being blocked (Fig. 3).

The patient was discharged from the hospital with a referral to a regional center for AIDS prevention and control, with a diagnosis of gangrene of the lower lobe of the left lung. Complications included parapneumonic empyema of the left pleural cavity, a grade 1A pulmonary hemorrhage, respiratory failure of grades 1–2, severe anemia, and left laryngeal paresis. The underlying condition was HIV disease, with manifestations of other bacterial infections and a stage 4B of secondary disease progression, without highly active antiretroviral treatment.

In 2024, we identified a second member of the *Elizabethkingia* genus, *Elizabethkingia meningoseptica*. This organism is typically a nosocomial pathogen that causes infections in patients with weakened immune systems [6, 10].

Elizabethkingia meningoseptica strains have intrinsic resistance to most antimicrobial agents used

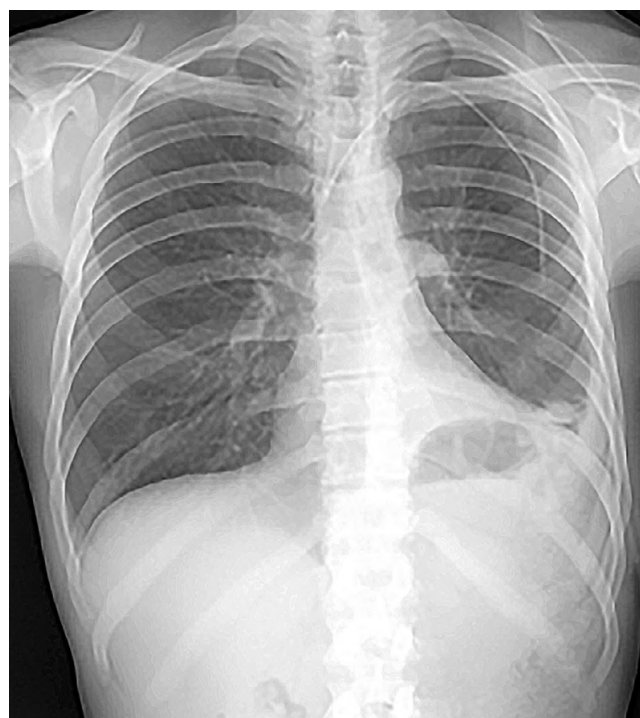


FIG. 3. Radiograph scan before discharge. The upper lobe is straightened and fills the hemithorax

TABLE 2

SUSCEPTIBILITY OF *PSEUDOMONAS AERUGINOSA* TO ANTIMICROBIAL AGENTS

Antimicrobial Agent	
Fosfomycin	I
Cefepime	I
Ceftazidime	I
Ciprofloxacin	R
Chloramphenicol	R
Tobramycin	R
Amikacin	S
Imipenem	R
Meropenem	I
Piperacillin/Tazobactam	R
Ceftazidime/Avibactam	S
Polymyxin E (Colistin)	S

Notes. S – susceptible, I – susceptible with increased drug exposure, R – resistant.

to treat infections caused by gram-negative bacteria, including polymyxins, aminoglycosides, chloramphenicol, and the majority of extended-spectrum β -lactam agents. The multidrug resistance is due to the production of three types of β -lactamases: two carbapenem-hydrolyzing class B metallo- β -lactamases (the only microorganisms with two chromosomal-encoded MBL genes), and a non-inducible, extended-spectrum class A β -lactamase (ESBL). *E. meningoseptica* frequently exhibits resistance to colistin, tetracycline, erythromycin, and linezolid, and it is also moderately susceptible to clindamycin and vancomycin [6, 8, 9, 14]. These factors significantly complicate the selection of appropriate antibiotic treatment regimens.

CLINICAL CASE 2

A 33-year-old patient was admitted to the hospital with a diagnosis of metapneumonic, right-sided empyema. The patient had a history of acute leukemia (T-cell lymphoblastic T-I variant, first episode on May 13, 2024) and neuroleukemia (treated on July 18, 2024, with remission achieved from July 16, 2024). There was also a history of thoracoscopy for lung decortication. The patient's condition was classified as moderately severe, with chest pain on the right side and a temperature 38–39°C.

CBC: leukocytes $5.94 \times 10^9/\text{mL}$, mild anemia (Hb 98 g/L), and thrombocytosis $567 \times 10^9/\text{mL}$. MSCT



FIG. 4.
MSCT scan of the chest

of the chest revealed an acute pulmonary-pleural abscess in the sixth through tenth segments of the patient's right lung (Fig. 4).

Under ultrasound guidance, a Malek 10 Fr chest tube was placed in the right 6th intercostal space, along the scapular line, in a seated position. A purulent discharge of approximately 150 mL was obtained from the pleural cavity. The exudate was found to contain 10^6 CFU/mL of *Elizabethkingia meningoseptica* (Table 3).

Considering the patient's medical history, which included multiple courses of antibiotic therapy, and the resistance of the isolated microorganism to various antibiotics, the following treatment was administered: the purulent cavity was cleaned with Sextophage (20 ml) for 1 hour, followed by cleaning with a 0.02 %

chlorhexidine solution. On the 20th day, the drain was removed. In the subsequent culture, no aerobic or facultative anaerobic microorganisms were detected. A chest X-ray revealed that the lungs had expanded.

The patient was discharged from the hospital for follow-up care by a hematologist. The diagnosis was "acute pulmonary-pleural abscess on the right". The underlying disease was acute leukemia, T-cell lymphoblastic variant (T-I), the first attack, and neuroleukemia at the onset. The patient is currently in remission.

One and a half years later, the patient was seen by the hematologist again and continued treatment (Fig. 5).

TABLE 3

SUSCEPTIBILITY OF *ELIZABETHKINGIA MENINGOSEPTICA* TO ANTIMICROBIAL AGENTS

Antimicrobial Agent	
Trimethoprim/Sulfamethoxazole	R
Tobramycin	R
Tigecycline	R
Meropenem	R
Piperacillin/Tazobactam	R
Amikacin	R
Cefepime	R
Gentamicin	R
Ciprofloxacin	R
Cefotaxime	R
Amoxicillin/Clavulanic acid	R
Imipenem	R
Ertapenem	R

Notes. S – susceptible, I – susceptible with increased drug exposure, R – resistant.

DISCUSSION

Bacteria of the *Elizabethkingia* genus are opportunistic pathogens that cause serious nosocomial infections in patients with weakened immune systems [3, 14, 15]. These bacteria are resistant to most antimicrobial treatments, making treatment difficult [2, 5, 9]. Clinicians and bacteriologists need to be aware of these microorganisms and identify them in order to develop appropriate treatment strategies. Compliance with infection control measures is essential to prevent the spread of *Elizabethkingia* within hospitals and the occurrence of outbreaks [2, 11]. The detection of these bacteria from significant sources of infection in patients can indicate an underlying immune deficiency, prompting further investigation. This information is valuable for pulmonologists, thoracic surgeons, and infectious disease specialists. It will allow for the prescription of appropriate treatment for these patients.

CONCLUSION

Based on the above considerations, in the submitted observations, patients with significant comorbid conditions successfully underwent targeted treatment for the discussed pulmonary infection.

Funding

The study had no financial support.

Conflicts of interest

The authors declare no conflicts of interest.

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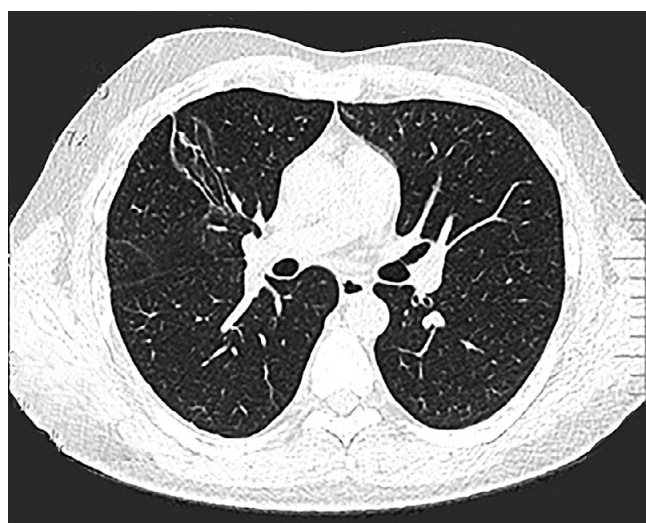


FIG. 5.
MSCT scan in 1.5 years later

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