Infection and Drug Resistance

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CASE REPORT

# Misdiagnosis Diagnosis of Pneumocystis Pneumonia as Chemical Pneumonitis

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**Background:** Auxiliaries, a mixed chemicals, for printing and dyeing characterized by their diverse range and complex chemical compositions are commonly utilized in the textile industry. These chemicals can lead to environmental contamination and pose health risks to humans.

**Case Description:** A 29-year-old man who worked in a printing and dyeing factory in Suzhou, China, reported having tightness in his chest and coughing. Despite seeking medical treatment at several hospitals, the initial diagnosis remained elusive. High-resolution chest CT scans showed multifocal lesions in both lungs. The patient had no significant medical history, and the respiratory symptoms only surfaced after exposure to dyeing auxiliaries. Physicians initially suspected chemical pneumonitis due to occupational exposure. However, a subsequent evaluation at a hospital specializing in occupational diseases led to a diagnosis of AIDS and pneumocystis pneumonia.

**Conclusion:** This case underscores the importance of comprehensive clinical diagnosis to avoid biases and reduce the incidence of misdiagnosis.

**Keywords:** chemical pneumonitis, high-resolution CT, misdiagnosis, pneumocystis pneumonia, printing and dyeing auxiliaries

## Introduction

Pneumonia, characterized as lung inflammation, is a prevalent respiratory system condition with a multifaceted etiology, including bacterial, viral, mycobacterium tuberculosis, and fungal infections.[1] Chemical pneumonia is an inflammatory reaction in the lungs caused by inhaling harmful chemicals, such as humic substances and humic-like substances.[2] In occupational health scenarios, workers may be exposed to various chemicals, leading to the occurrence of chemical pneumonia. Meanwhile, Pneumocystis pneumonia (PCP) is an infectious lung disease caused by Pneumocystis. In some cases, chemical pneumonia and PCP may have similar clinical manifestations, which increases the complexity of accurate diagnosis in occupational health settings.

The severity of respiratory tract injury from chemicals hinges on the substance's physicochemical properties, exposure concentration, contact duration, and individual host status. Chemical inhalation can cause mild symptoms like sore throat, cough, sputum production, asthma, and chest tightness, or severe outcomes such as chemical pneumonitis, pulmonary edema, and acute respiratory distress syndrome (ARDS). Chemical-induced respiratory issues are frequently seen in populations with occupational exposures, with known causative agents including nitrogen oxides, hydrogen sulfide, chlorine, sulfur dioxide, and gasoline. In the textile industry, printing and dyeing auxiliaries serve as surfactants in fabric processing to enhance outcomes. The wide application of these chemicals in various sectors leads to environmental and health concerns. While existing reports focus predominantly on environmental pollution caused by these auxiliaries,[3,4] there is limited literature on their health impacts, particularly regarding respiratory system exposure in occupational settings.

Received: 17 January 2024
Accepted: 18 April 2024
Published: 7 May 2024

In our clinical practice, cases of respiratory diseases attributed to exposure to dyeing auxiliaries have been rare, with scanty related literature. In this paper, we present the case of a patient engaged in printing and dyeing work, treated at our hospital in 2022. He was initially misdiagnosed as chemical pneumonia. This case illustrates the complexities of diagnosis in occupational health scenarios related to chemical pneumonitis.

# Case Description

## Clinical Course

The patient, a 29-year-old man employed as a machine operator in a Suzhou printing and dyeing factory in Jiangsu Province, China, experienced respiratory symptoms between April and May 2022 due to exposure to alkali steam from a malfunctioning machine. During the approximately one-hour repair process, he wore a standard disposable mask while working in a confined space of approximately 10 $m^2$ filled with alkali steam. Subsequently, he developed symptoms including chest tightness and coughing. Antibacterial infective and steroid therapies were used as the first line of treatment at a nearby Suzhou hospital for a suspected pulmonary infection, but they only provided a temporary relief from his symptoms. By July 25, 2022, the patient had visited multiple hospitals in Suzhou due to recurrent fever, cough, tightness in the chest, and dyspnea. His history of exposure to printing and dyeing auxiliaries, combined with the lack of other significant medical history, led physicians to consider a diagnosis of chemical pneumonitis. He was directed to a hospital specializing in occupational diseases in Suzhou for further evaluation. On July 26, he consulted the outpatient clinic of occupational diseases at our hospital. Multifocal nodular opacities, patchy regions, and honeycombing were seen on the high-resolution chest CT (HRCT) scans, mostly in the upper lung lobes. These regions showed heterogeneous densities, some dense foci with lucencies, and bronchial images (as shown in Figure 1A–C). The outpatient physician recommended further diagnostic investigations and symptomatic treatment, which the patient initially declined. But on August 13, he showed up at our hospital's emergency room because his symptoms were getting worse, he felt more difficulty breathing and needed oxygen. He was admitted for a thorough examination and treatment.

## Post-Admission Examination

His vital signs showed a temperature of 36.2 °C, pulse rate of 82 beats per min, respiratory rate of 21 breaths per min, blood pressure of 123/72 mmHg, and $SpO_2$ at 96%. The results from the laboratory tests showed that the patient had a white blood cell count of $1.11\times10^9$ /L, red blood cell count of $4.13\times10^{12}$ /L, hemoglobin level of 128 g/L, platelet count of $139\times10^9$ /L, direct lymphocyte count of $0.36\times10^9$ /L, albumin level of 27.1 g/L, Hypersensitive C-reactive protein (HsCRP) 25.9 mg/L. The arterial blood gas analysis showed a pH of 7.426, $PCO_2$ of 36.6 mmHg, $PO_2$ of 91.5 mmHg, lactate of 2.5 mmol/L, and a PF Index of 223.24 mmHg. The patient denied a history of tuberculosis and denied alcohol consumption or smoking. Initially, we considered that this was a common pulmonary bacterial infection and administered antibiotics for anti-infection treatment and other symptomatic treatment. We continued to complete the other further examinations. When the patient's HIV antibody screening results were positive on the third day, we asked a specialist for consultation and reexamined the blood results. The patient admitted to being homosexual, and his weight dropped at least 10 kg and and had recurrent fever in the past half year. The result of the consultation was to transfer the patient to the infection department for further treatment and to review some examinations. After the transfer, on the fourth day of hospitalization, the patient's examination results revealed a white blood cell count of $7.37\times10^9$ /L, red blood cell count of $4.14\times10^{12}$ /L, hemoglobin level of 130 g/L, platelet count of $141\times10^9$ /L, direct lymphocyte count of $0.30\times10^9$ /L, albumin level of 28.5 g/L, T-lymphocyte percentage of 57.8%, CD4+ T-cell percentage of 3.97%, CD8+ T-cell percentage of 51.1%, and a CD4+/CD8+ ratio of 0.08. The arterial blood gas analysis showed a pH of 7.427, $PCO_2$ of 38.2 mmHg, $PO_2$ of 87.0 mmHg, lactate of 3.0 mmol/L, and a PF Index of 142.70 mmHg. The results of the tests for anti-neutrophil cytoplasmic antibody (ANCA), autoimmune antibodies, and T-cell test for tuberculosis infection were negative, TORCH test showed negative for IgM antibody. The combination test for HIV antigen and antibody was positive. A repeat HRCT scan revealed uneven lung lucency, thickening of the interlobular septa, disorganized lung markings, multifocal patchy and nodular opacities of different sizes with cystic lucencies, and honeycomb-like changes in the upper lung lobes (as shown in Figure 2A–C).

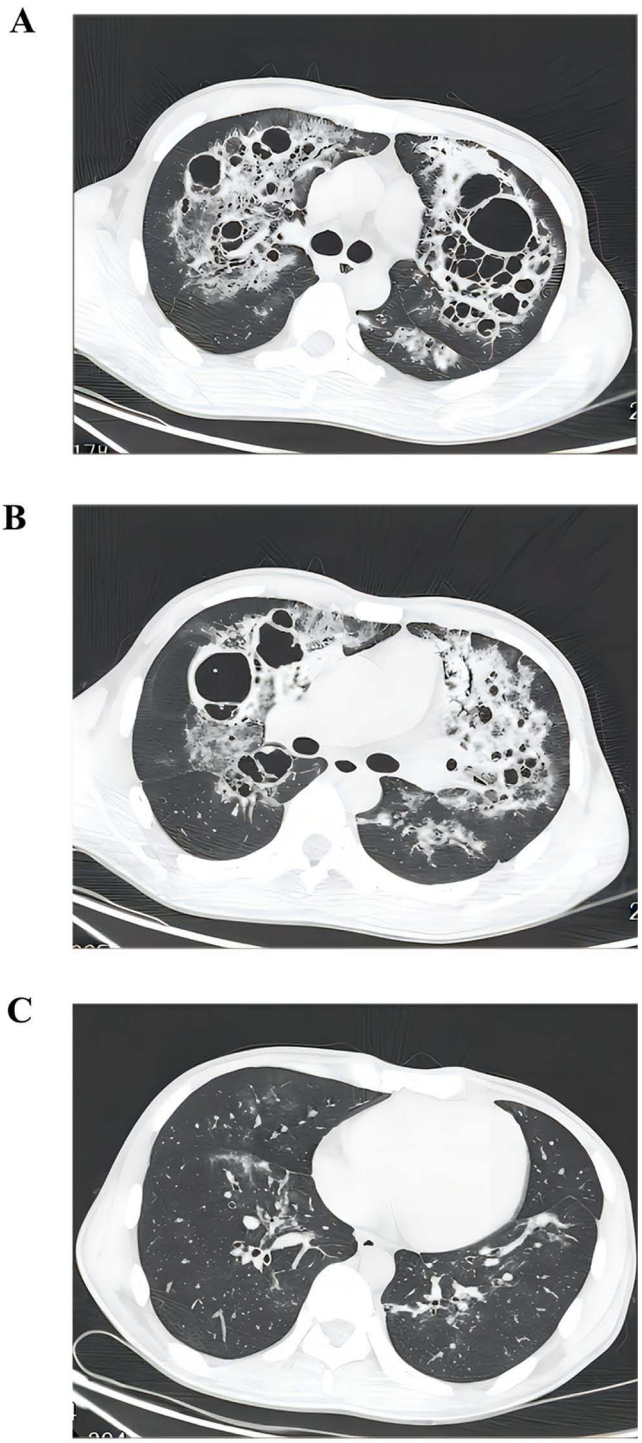


**Figure 1** The chest HRCT scan from July 26, 2022, taken at the time of initial diagnosis, shows the patient's lungs with multiple nodular opacities, patchy areas, and honeycombing, predominantly in the upper lobes, characterized by uneven densities and some areas showing lucencies and bronchial images. (**A**) Tracheal bifurcation level: honeycomb of different sizes and infiltrating patchy shadows can be seen in most areas, with the honeycomb as the main change, (**B**) The bronchial level of the right middle segment: septal thickening with nodular infiltrates and honeycomb of different sizes, more septal thickening with nodular infiltrates than honeycomb, (**C**) The bronchial level of the basal segment: The local infiltration shadow was visible in a small area, and the honeycomb was not obvious.

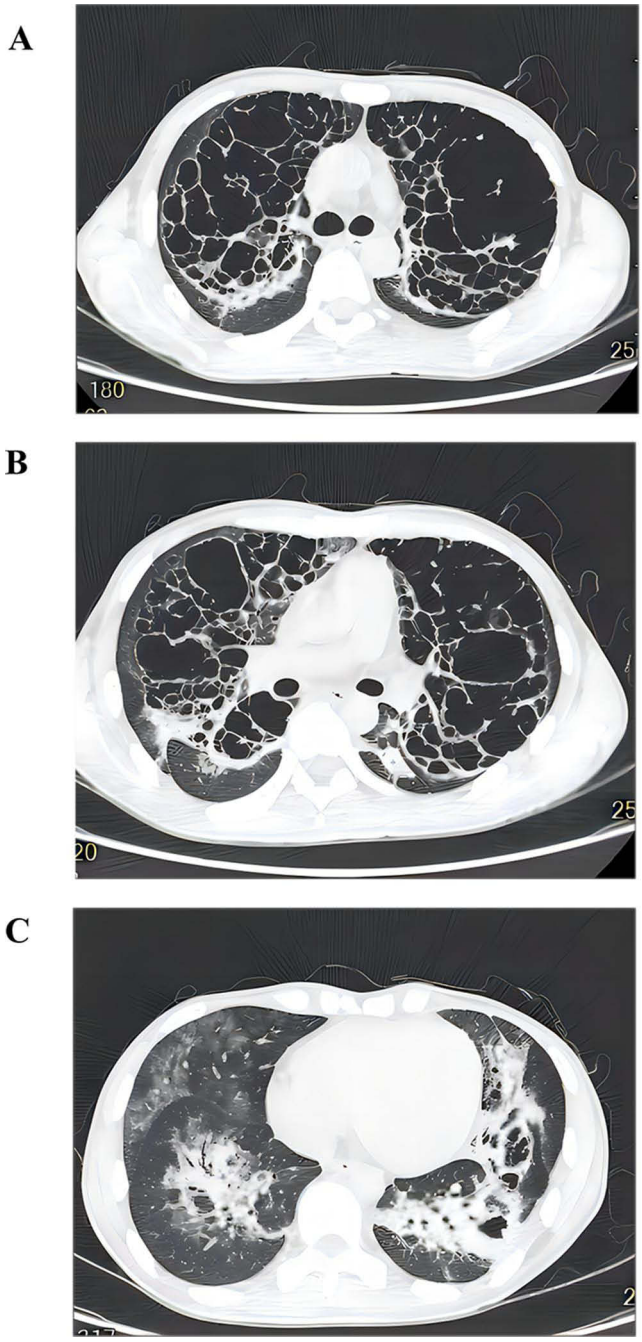


**Figure 2** A follow-up chest HRCT scan from August 16, 2022, during the patient's hospitalization, reveals increased and enlarged lucencies in the upper lobes of both the lungs and more extensive lesions in the lower lobes compared to the images from July 26, 2022. (**A**) Tracheal bifurcation level: It remained predominant honeycomb with significantly less shaded infiltrated patches, the lobular interval was thinner, (**B**) The bronchial level of the right middle segment: Like the changes in upper lung, the honeycomb range was expanded, the lobular septum became thinner, and the nodule infiltration was significantly reduced, (**C**) The bronchial level of the basal segment: The scope of nodular invasion expanded and honeycomb was added.

## Final Diagnosis

The final diagnosis was AIDS and pneumocystis pneumonia (PCP).

## Treatment

We used compound sulfamethoxazole for anti-PCP treatment, while considering the possibility of combined opportunistic infections, antibiotics and fluconazole were used to fight bacterial infections and antifungal infections.

## Prognosis

Unfortunately, the patient was discharged to home t the day after transfer for personal reasons, and we could not follow up his condition.

# Discussion

Chemical-induced respiratory injuries commonly manifest as bronchitis, chemical pneumonitis, pulmonary edema, and ARDS. The induction of inflammatory responses in the respiratory tract, direct irritative and corrosive effects on tissue cells, and damage from free radicals are the mechanisms by which chemical substances cause lung injury. Acute lung injury signs such as interstitial and alveolar pulmonary edema can be seen in severe cases, which involve chemical infiltration into the alveoli. Mild cases may only exhibit cough and sore throat, which are typical symptoms of bronchitis. In severe circumstances, this may develop into ARDS.[5] Due to the direct injury and indirect injury to the lungs by inhaled chemicals, the airway mucosa can be congested and edema, resulting in increased permeability of epithelial and endothelial cells and activation of inflammatory cells, causing the massive release of oxygen free radicals and deregulation of inflammatory mediators. If the lung inflammatory infection is combined, the white blood cell count and HsCRP are increased. Patients have mainly respiratory symptoms, rarely have fever, let alone show significant weight loss. In this report, the patient's leukemia count did not increase but decreased, and HsCRP was mildly increased, which is inconsistent with the examination results of chemical pneumonia.

Commonly encountered substances that can cause chemical pneumonia include concentrated hydrochloric acid, chlorine gas, sulfur dioxide, nitrogen dioxide, paraquat, metal fumes, and fire smoke. The severity and prognosis of chemical pneumonitis depend on factors like the toxin's concentration, inhaled dose, duration of exposure, individual's susceptibility, and the physicochemical properties of the toxin. A Study[6] has indicated that the size of the inhaled particles plays a crucial role in determining the site and nature of the respiratory tract injury. Particles larger than 10 μm typically settle on the mucous membranes of the nose and pharynx, those between 3–10 μm settle in the trachea and bronchi, particles 0.1–3 μm predominantly accumulate in the alveoli, and particles smaller than 0.1 μm are generally exhaled. The solubility and physicochemical properties of the chemicals that enter the airways and alveoli lead to chemical pneumonitis, which damages vascular endothelial cells, macrophages, and the epithelial and bronchiolar cells in the bronchi. This can lead to diffuse bronchiolar obstruction, as well as alveolar and interstitial edema, and hemorrhage.[7] Thus, if the injury is bronchial, the HRCT scan may show thickening of the bronchial wall; if it is bronchiolar and alveolar, the HRCT scan may display ground-glass opacities distributed along the bronchioles or extensive foggy changes in the lungs.[8]

In the printing and dyeing process, a variety of chemical substances, including auxiliaries and surfactants, are used. Alkali solutions, commonly used in printing and dyeing, have their permeability enhanced by surfactants. Commonly used surfactants include sodium hydroxide, sodium carbonate, sodium bicarbonate, ammonia solution, and sodium silicate. As the textile printing and dyeing industry uses several chemical raw materials, there is a risk of environmental pollution during the stages of processing when thousands of chemicals are produced. Any accidental mishap during production or processing puts the workers' health at risk. The printing and dyeing process involves a multitude of organic compounds, presenting complex and diverse health hazards.

The patient reported primary exposure to sodium hydroxide, a chemical that has not been linked to many lung injuries. There have been reported cases of workers exposed to sodium hydroxide developing permanent obstructive pulmonary disease.[9] Chest X-rays in such instances indicated significant pulmonary emphysema, and arterial blood gas analyses showed hypoxemia without carbon dioxide retention.[10] Animal studies have shown that rats exposed to high concentrations of sodium hydroxide exhibit inflammatory changes in the bronchi, with bronchial epithelial cells presenting hyperplasia, wrinkling, ulceration, and necrosis. Also observed was the extreme hyperplasia of bronchial lymphoid tissue compressing the bronchial lumen.[11] In this patient, the HRCT scan of the lungs showed multiple nodular opacities, patchy areas, and honeycombing, predominantly in the upper lobes, with uneven densities and some dense areas showing lucencies and bronchial images. The findings were primarily indicative of interstitial lung changes. A follow-up HRCT scan after 20 days showed further progression of lung lesions, which were atypical for lung injury caused by inhalation of sodium hydroxide. In patients with AIDS who also have concurrent PCP, the presentations on the HRCT scan can vary significantly. Some patients may show

ground-glass opacities, while others exhibit consolidation, thin-walled cystic airspaces within ground-glass opacities, or more extensive reticular or reticulonodular patterns. Occasionally, a single patient may exhibit more than one of these symptoms. Typically, the most prevalent and distinctive HRCT manifestation of PCP is ground-glass opacity.[12] According to reports, about one-third of patients with AIDS may exhibit pneumothorax or pulmonary cysts, frequently accompanied by a distribution of ground-glass opacities resembling a map. This is especially true when pulmonary cysts are present at the same time, as these cases are highly suggestive of PCP.[13]

In general, the patient's exposure history, medical history, laboratory test results, and radiological findings are used to distinguish between chemical pneumonitis and PCP.

At present, the diagnosis of PCP depends on the detection of pathogens, the detection rate of respiratory secretions smear is very low, and the positive rate of bronchoalveolar lavage fluid (BALF) is significantly higher than that of sputum specimens.[14] BALF may still be the best specimen to detect PCP, but not everyone is willing to accept the test, and these samples can only be obtained when patients are safe and stable.

PCP is a major cause of morbidity and mortality in AIDS patients. In this report, the patient's sputum symptoms were not obvious, and he refused to undergo bronchoscopy, so his sputum or alveolar lavage could not be further examined. Previous studies have indicated that the incidence of PCP infection is $CD4^+$ T cell-count-dependent and that the prevalence of PCP infection in HIV/AIDS patients increases with decreasing $CD4^+$ T cell count.[15] At last the experts comprehensively analyzed the diagnosis based on his clinical manifestations, laboratory tests, HRCT, and local epidemiological characteristics.

But in this instance, the patient's focus on his work exposure and his deliberate concealment of other parts of his medical history had a major impact on the diagnosing physician's assessment. The results of this case study demonstrate how withholding or deleting medical history information can result in incorrect diagnoses in clinical settings.[16–20]

## Conclusion

In conclusion, for patients with atypical clinical features, it is crucial to adopt a comprehensive approach that includes detailed inquiries into both past medical and occupational history. This holistic approach is fundamental to minimize the risk of misdiagnosis, enabling the early detection and treatment of diseases. For clinical cases lacking laboratory confirmation, an initial diagnosis can only be made by clinical experience or effective by diagnostic treatment.

## Ethics Approval and Consent to Participate

This study was conducted with approval from the Ethics Committee of The Fifth People's Hospital of Suzhou. This study was conducted in accordance with the declaration of Helsinki. Written informed consent was obtained from all participants.

## Consent for Publication

Consent for the publication of the case was obtained from the patient.

## Acknowledgments

We would like to acknowledge the hard and dedicated work of all the staff that implemented the intervention and evaluation components of the study.

## Funding

The Occupational Health Research Project of Jiangsu (JSZJ20231103); The Science and Technology Development Project of Suzhou (SZYJTD201904, SZXK202115).

## Disclosure

The authors declare that they have no competing interests in this work.

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