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CASE REPORT

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A case of severe psittacosis pneumonia complicated by splenic infarction

Zhao Yu[1†], Yuanyuan Qian[2†], Lan Lv[1] and Wenqing Hu[1*]

Abstract

Clinical data Chlamydia psittaci pneumonia is a community-acquired pneumonia caused by Chlamydia psittaci. While severe cases may lead to critical conditions such as respiratory failure, splenic infarction is relatively uncommon. A severe patient with Chlamydia psittaci pneumonia admitted to our hospital experienced a splenic infarction during treatment. Fortunately, the patient's situation was improved after careful treatment. Now, the patient has been discharged. Further exploration of the mechanism of concurrent splenic infarction is required.

Backgroud Psittacosis pneumonia, a zoonotic infectious disease transmitted from birds to humans, is caused by Chlamydia psittaci and represents a type of chlamydial pneumonia [1]. Insome instances, the disease may progress to severe pneumonia and respiratory failure, necessitating intensive support measures, including mechanical ventilation. The advent of technologies such as Metagenomic Next-Generation Sequencing (mNGS) for the etiological diagnosis of infectious diseases [2] has improved the diagnostic and treatment success rates for Psittacosis. Instances of severe chlamydial pneumonia with complications such as splenic infarction are uncommon. A patient with severe Psittacosis pneumonia complicated by splenic infarction was admitted to the Emergency Intensive Care Unit (EICU) of Haining People's Hospital and subsequently improved following effective anti-infective and anticoagulant therapy. This report is provided herein.

Keywords Psittacosis, Splenic infarction, Critical pneumonia

Clinical data

The patient, a 76-year-old female, was admitted to the hospital on June 4, 2023, where she presented with a three-day history of fever and cough, which escalated to somnolence on the day prior to admission. Initially, she presented a fever peaking at 39 °C and a paroxysmal dry cough without diurnal variation. Her condition deteriorated the following day, as indicated by altered consciousness and hypotension. Her medical history included hypertension, and she denied any history of diabetes, atrial fibrillation, or cardiac valvular disease. Her occupational background in agriculture involved prolonged exposure to domestic poultry, and she reported no smoking or alcohol consumption. Physical examination findings included: respiratory rate of 22 breaths/min, heart rate of 81 beats/min, and blood pressure of 70/46 mmHg. Coarse breath sounds and scattered wet rales were noted in both lungs, while cardiac examination revealed a regular rhythm without significant arrhythmias or murmurs. The abdomen was distended without tenderness or rebound pain, and no obvious hepatosplenomegaly was palpable.

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Postadmission assistant examination (DAY1), arterial blood gas analysis revealed a pH of 7.37, PCO_2 of 34.0 mmHg, PO_2 of 78.0 mmHg under mask oxygenation(oxygen flow rate of 5 L/min) and an oxygen saturation of 95%.The complete blood count and inflammatory markers results a white blood cell count of 12.4×10^9/L, with a neutrophil percentage of 95.9% and a lymphocyte percentage of 2.6%,a platelet count of 220×10^9/L, and a high-sensitivity C-reactive protein (CRP) level of 370.0 mg/L. The prothrombin time was 9.3 s, the plasma fibrinogen concentration was 9.22 g/L, and the D-dimer concentration was 2.278 μg/mL. Procalcitonin (PCT) was 9.58 ng/mL, and interleukin-6 (IL-6) was 1391.1 pg/ml, while the nucleic acid test for the novel coronavirus was negative.Mycoplasma pneumoniae antibody test: Negative.Detection of 13 pathogens and subtypes of respiratory viruses: Chlamydia positive.Chest CT scans taken on the same day revealed large areas of increased density in the upper and lower lobes of the right lung, with some consolidation (Fig. 1).

Upon admission, the patient's evaluations included: a Sequential Organ Failure Assessment (SOFA score) of 8 points; a CURB-65 score for community-acquired pneumonia of 3 points; a pneumonia severity index (PSI) of 149 points, classified as class V, with a predicted mortality rate of 27%; and a Venous Thromboembolism (VTE) score - Padua Prediction Score of 6 points, indicating a high risk.

The patient was diagnosed at admission with severe community-acquired pneumonia 、 septic shock.

Treatment: On the day of admission, on the basis of severe infection and shock symptoms, 10 mg of noradrenaline was immediately administered at a dose of 0.37 μg/kg/min to increase blood pressure, and 1 L of compound sodium chloride was injected for fluid resuscitation. The patient's oxygen saturation under the mask decreased to approximately 80%, and tracheal intubation and ventilator support were immediately provided (the inhaled oxygen concentration was 50%, and the positive end-expiratory pressure was 6 mmHg). Imipenem/cilastatin 0.5 g every 8 h combined with tigecycline 50 mg every 12 h was administered as anti-infection treatment, along with enoxaparin 0.2 mL subcutaneously every 12 h for anticoagulation, and treatments for expectoration and asthma relief. On admission Day 3, bronchoscopy was performed, and the lavage fluid was analyzed via mNGS at Zhejiang Shengting Medical Laboratory Co., Ltd., identifying a Chlamydia psittaci infection with 5,434 sequence and *Candida tropicalis* with 18 sequences (considering colonization), were identified, with no viral RNA sequences detected at admission Day 4. Considering the patient's imaging findings and laboratory tests, *Chlamydia psittaci* was identified as the main pathogenic bacterium.Treatment was adjusted to moxifloxacin sodium chloride injection (0.4 g daily) with 50 mg tigecycline every 12 h for anti-infection treatment, complemented by intermittent prone positioning ventilation and bronchoscopy suction among other supportive care measures. Subsequent treatment led to improvements in pulmonary function, increased oxygenation, and reduced inflammatory markers such as PCT and CRP, and no other positive pathogenic bacteria were found in the sputum culture. On admission Day 8, the patient reported concealed pain in the upper left abdomen with palpation tenderness but no muscle rigidity. Blood tests revealed a hemoglobin level of 74 g/L, a platelet count of 60×10^9/L, a fibrinogen level of 0.64 g/L, and a D-dimer level of 35.682 μg/mL; the Chinese disseminated intravascular coagulation scoring system (CDSS) score was 6 points. Echocardiogram+lower limb vascular color Doppler showed mild

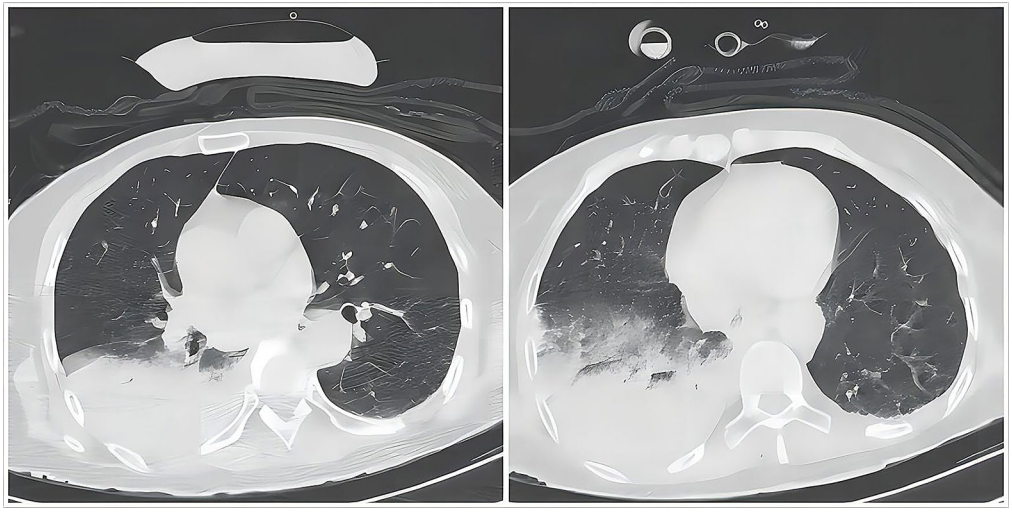

Fig. 1 Chest CT upon admission on June 4, 2023: large areas of increased density are seen in the upper and lower lobes of the right lung, with some consolidation

aortic regurgitation, mild mitral and tricuspid regurgitation, decreased diastolic function of the left ventricle, left ventricular FS of 40%, and EF of 70%. No vegetation was found in the cardiac cavity.No abnormalities in the arteries or veins of either lower limb were observed. Enhanced abdominal CT revealed a wedge-shaped low-density shadow within the spleen, with no significant enhancement. (Fig. 2).

Following discussions at a multidisciplinary treatment (MDT) meeting, the patient was diagnosed with severe psittacosis pneumonia complicated by splenic infarction. Given the absence of splenic abscess or rupture and the lack of need for surgical intervention, conservative medical management was employed. Enoxaparin injections were substituted with rivaroxaban 10 mg rivaroxaban administered nasogastrically once daily for anticoagulation, complemented by supportive treatments including transfusions of fibrinogen, platelets, red blood cells, plasma, and cryoprecipitate. Subsequent to these interventions, the patient's abdominal pain subsided, inflammatory marker levels decreased, and oxygenation levels increased.Since the gastrointestinal function was not affected, we replaced the intravenous tigecycline with doxycycline tablets on admission Day 9. Mechanical ventilation was ceased on admission Day 12, and the patient was transitioned to a regular ward with high-flow nasal oxygen. Compared with prior assessments, post-discharge imaging revealed (Fig. 3) substantial resolution of pulmonary inflammatory lesions and improvement in the low-density lesions in the spleen. The therapeutic approach during the hospital stay, evolution of the patient's condition, and discharge follow-up findings are illustrated in Fig. 4.

This study received approval from the Ethics Committee of Haining People's Hospital [Approval number: (2023) Ethics Review No. 118], and informed consent was duly obtained.

Discussion

Psittacosis, caused by Chlamydia psittaci of the Chlamydiaceae family, is an obligate intracellular parasite with 17 identified genotypes [3]. Transmission occurs through direct contact with infected birds, poultry, mammals, their feces, respiratory secretions, or contaminated aerosols. Chlamydia that accounts for is an atypical pathogen, pneumonia, accounting for approximately 1.03% (95% CI 0.79–1.30) of community-acquired pneumonia cases worldwide [1]. There are no comprehensive national epidemiological data on the epidemiological characteristics of China [4]. Clinically, symptoms range from asymptomatic to typical respiratory infection signs, with severe cases necessitating mechanical ventilation and intensive care support. Radiographic findings may include patchy consolidations or ground-glass opacities, bronchiectasis, and unilateral or bilateral lung involvement [5].Severe instances often exhibit bronchial dilation, which is correlated with disease severity. Although blood tests such as white blood cell count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and procalcitonin (PCT) can indicate anomalies, their lack of specificity contributes to frequent misdiagnoses or overlooked diagnoses with standard imaging and blood tests [6]. Serological testing is also a common testing method, but chlamydia parrot heat may cross-react with antibodies against other pathogens, thus affecting the accuracy of detection, which is not suitable for early diagnosis.Detection of Cps is the gold standard for diagnosis, but traditional cultural conditions are harsh and time-consuming, so it is generally not recommended as the preferred detection method [7]. In recent years, mNGS and other molecular

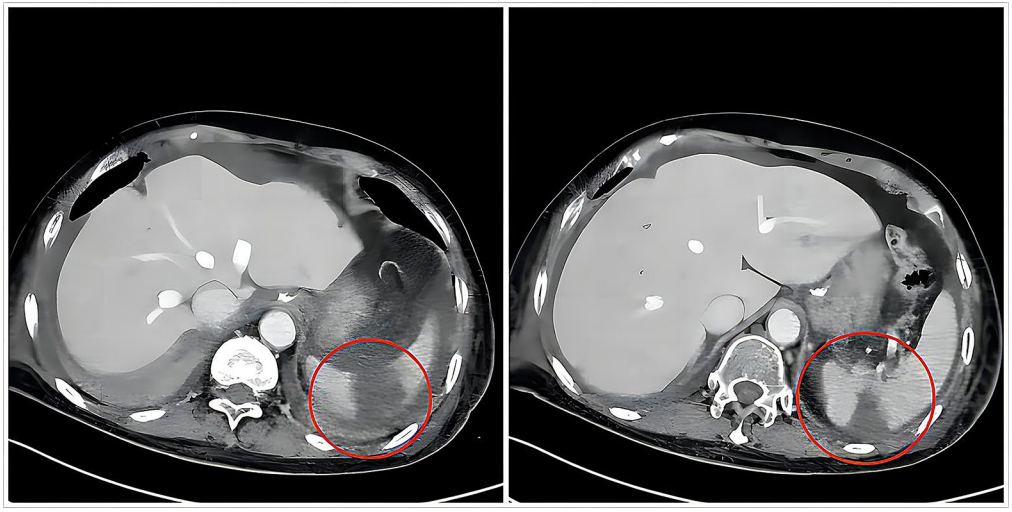

Fig. 2 Enhanced abdominal CT on June 12, 2023: A wedge-shaped low-density shadow is seen within the spleen, with no significant enhancement on enhancement

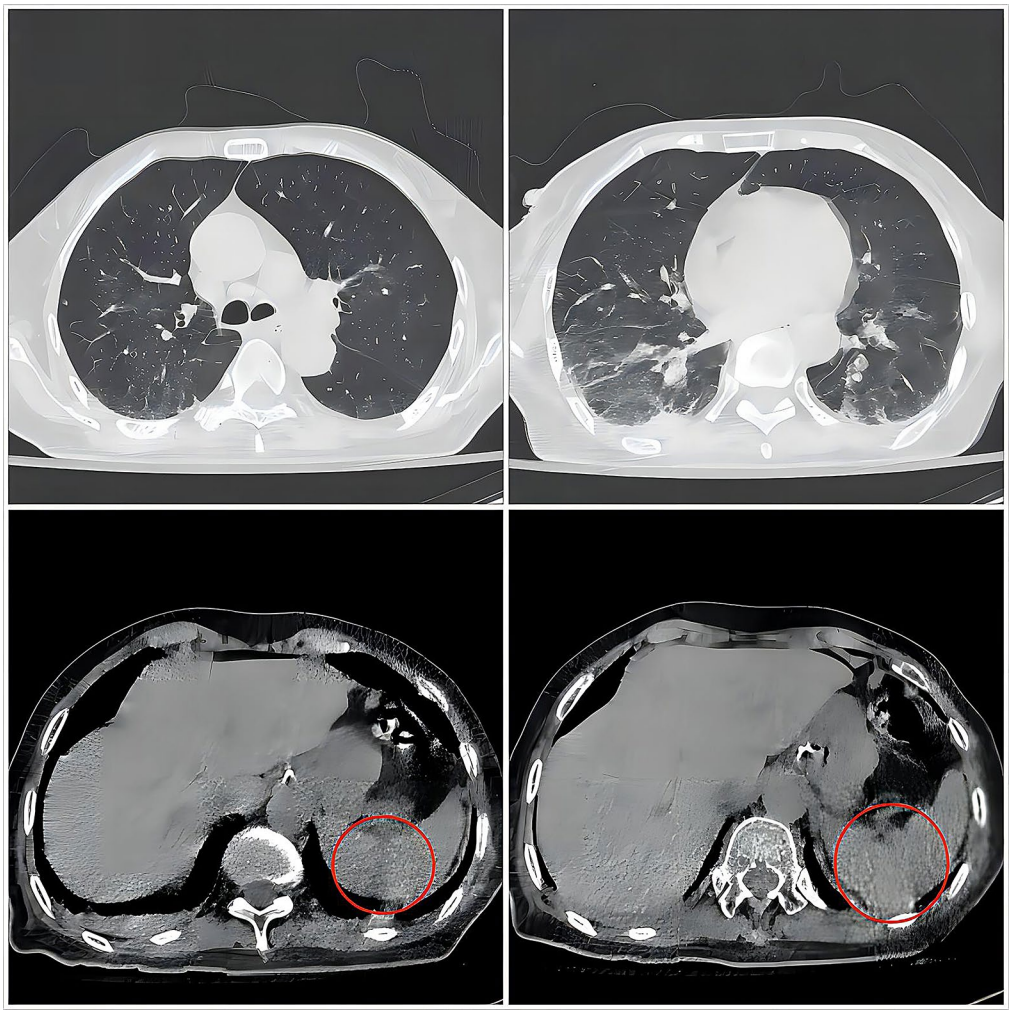

Fig. 3 Chest CT on July 26, 2023: Both lungs saw multiple patchy shadows, large sheet of increased density and ground glass shadows, with blurred edges slightly absorbed compared to the previous image (July 14, 2023). Mild enlargement of mediastinal lymph nodes, coronary calcification, with noted: low-density lesions in the spleen

biological detection techniques have been widely used in the diagnosis of infectious diseases, with completion of non-targeted sample detection in a relatively short time (minimum 24 h) [2, 8].We can adjust the anti-infection plan according to the test results in time, and improve the success rate of treatment. Tetracyclines are typically the first-line treatment, with macrolides or fluoroquinolones as alternatives if tetracyclines are unsuitable [9]. Current research indicates that the psittacosis pathogen is sensitive to both quinolones and tetracyclines, with a high cure rate [10].However, with the increasing utilization rate of quinolones, the resistance rate also tends to increase [11]. Some scholars have proposed the combination of anti-infectives in severe patients to address the above changes [12]. Recently, Chinese experts have also recommended the combination of anti-infection regimens. The gastrointestinal function of critically ill patients is impaired, the effectiveness of oral antibiotics is poor, and doxycycline injection cannot be obtained in our hospital. Based on these factors, we chose tigecycline injection combined with moxifloxacin injection for anti-infection treatment and found that it yielded positive results.

Splenic infarction (SI) is characterized by ischemic necrosis of the spleen due to obstruction of the splenic artery or its branches, potentially leading to serious complications such as splenic abscess, secondary infection, and significant hemorrhagic splenic rupture, which pose a critical risk to patient survival. Clinically, it manifests as upper left abdominal pain, fever, chills, and nausea, although asymptomatic presentations are possible, increasing the risk of misdiagnosis or delayed diagnosis. In some instances, a definitive diagnosis is only achieved postmortem via autopsy [13]. Several factors can cause splenic infarction: (1) vascular diseases of the spleen due to the end-arterial nature of splenic

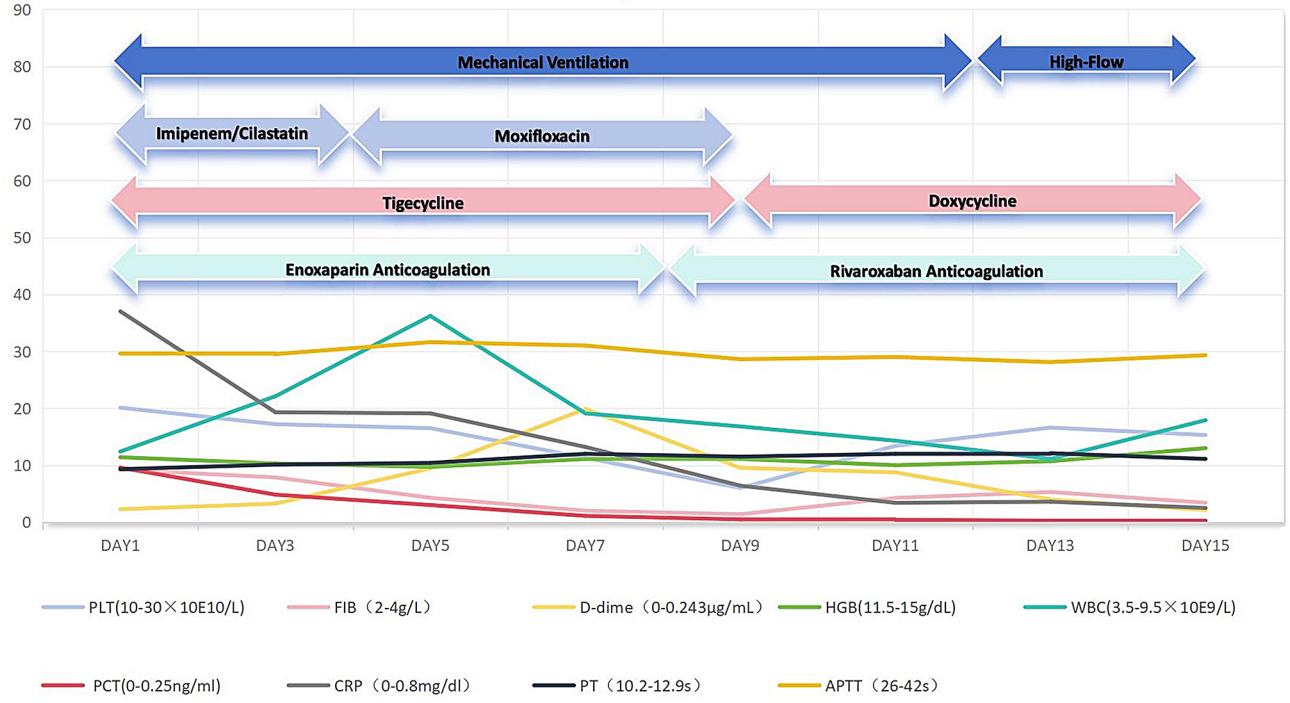

Fig. 4 Treatment, changes in medical condition, and post-discharge follow-up during the hospital stay from June 4, 2023, to July 26, 2023. Mechanical Ventilation Support(DAY 1-DAY 12); High-Flow Inhalation(DAY 12-DAY 15); Imipenem/Cilastatin 0.5 g q8h(DAY 1-DAY 4); Moxifloxacin 0.4 g qd(DAY 4-DAY 9); Tigecycline 50 mg q12h; (DAY 1- DAY 9); Doxycycline(DAY 9- DAY 15); Enoxaparin Anticoagulation(DAY 1- DAY 8); Rivaroxaban Anticoagulation(DAY 8- DAY 15). (PLT: Platelets; FIB: Fibrinogen; HGB: Hemoglobin; WBC: White blood cell; PCT: Procalcitonin; CRP: C-reactive protein; PT: Prothrombin time; APTT: Activated Partial Thromboplastin Time.)

artery branches, predisposing them to thrombosis from atherosclerosis, bending, or reduced blood flow with age; (2) hypercoagulable states, such as those associated with pancreatic cancer or malignant lymphoma, which can induce thrombosis and subsequent infarction [14]; (3) cardiac diseases such as infective endocarditis, atrial fibrillation, and rheumatic heart disease, which can lead to cardiac valvular vegetation [15]; and (4) iatrogenic causes during vascular interventional procedures, which can lead to inadvertent entry of embolic into the splenic artery, causing ectopic embolisms [16]. In addition, severe infection can easily lead to entry of pathogenic bacteria into the blood and their proliferation in the liver, spleen, and macrophage system to form bacterial emboli [7].Bacterial bolts spread through the bloodstream to the splenic artery or cause embolism in situ within the splenic artery. The literature has identified leukocytosis, lymphopenia, prolonged activated partial thromboplastin time, decreased fibrinogen, liver dysfunction, elevated LDH, and increased blood urea nitrogen as positive indicators of splenic infarction, serving as predictive factors for patient prognosis [17].Patients with severe chlamydia pneumonia may have complications, including endocarditis, meningitis, acute respiratory distress syndrome, rhabdomyolysis, and diffuse intravascular coagulation (disseminated intravascular coagulation, DIC) [16], among which endocarditis and DIC can easily lead to embolic events. In this case, despite anticoagulation prophylaxis during the treatment of confirmed psittacosis pneumonia, a splenic infarction still occurred. The patient had no tumors, cardiac disease, or underlying hematological conditions, and ultrasound did not reveal portal vein system thrombosis or cardiac valvular vegetation, suggesting none of the aforementioned causes were present.The CDSS daily score never exceeded 7 (7 points or greater for DIC) [18], which helped to exclude splenic infarction from endocarditis and DIC. During the course of the disease, laboratory tests revealed dynamic coagulation function and platelet changes (Fig. 4), so it can be inferred that the patient was more likely to have splenic infarction with in situ thrombosis caused by chlamydia parrot fever infection.

In recent years, cases of splenic infarction in situ caused by thrombosis have been reported in the both domestic and foreign literature, However, due to the sporadic nature of these cases, the underlying mechanism was not very clear. Childers, Hahn, Yin Wenwen, et al. reported rare cases of splenic infarction associated with coronavirus disease (COVID-19), Mycoplasma pneumoniae, and Brucella infection, respectively [19–21]. The analysis may include the following features:1.Infections causes endothelial dysfunction of vascular cells,

triggering fibrin formation, platelet adhesion, and aggregation, which can eventually lead to the formation of splenic artery embolism [22]0.2. Reducing blood flow and hypercoagulability in septic shock promotes thrombosis and blocks the splenic artery.3. The interaction between the coagulation pathways and the complement cascade in patients infected with atypical pathogens such as Mycoplasma pneumoniae may also be the mechanism underlying thrombosis [20]. Some reports have indicated a correlation between chlamydia fever infection and peripheral vascular thromboembolic events [23–25]. Although there have been no reports of splenic infarction caused by Chlamydia psittaci, further research is needed to determine whether this correlation suggests a similar coagulation mechanism or a hypercoagulable state between Chlamydia psittaci and atypical pathogens such as Mycoplasma pneumoniae.

The present case is an inaugural report of severe psittacosis pneumonia with concurrent splenic infarction. A review highlights the need to be vigilant for rare complications such as splenic infarction, in severe cases of psittacosis pneumonia. Among patients on mechanical ventilation, especially those who are sedated states, changes in abdominal signs and vital signs should not be overlooked.Detection of decreased fibrinogen and platelet levels, elevated D-dimer levels, fluctuations in inflammatory markers, coagulation functions, and hematologic laboratory parameters should heighten the suspicion of potential thrombotic or hemorrhagic events. The causative link between severe psittacosis pneumonia and subsequent splenic infarction merits deeper investigative scrutiny.

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Author contributions
ZY is responsible for collecting medical records and writing papers；YYQ is responsible for collecting medical record data and processing images and tables；LL is responsible for literature search and organization；WQH is responsible for literature screening and guiding paper writing.†Zhao Yu and Yuanyuan Qian contributed equally to this work.

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Data availability
All data generated or analysed during this study are included in this article.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Committee of Haining People's Hospital.

Consent for publication
Written informed consent was obtained from the individual for the publication of any potentially identifiable images or data included in this article.

Clinical trial
Clinical trial number: not applicable.

Competing interests
The authors declare no competing interests.

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