Annals of Clinical and Medical Case Reports[R]

ISSN 2639-8109 | Volume 15

Case Report Open Access

A Complex Autoimmune Case Complicated by Cat Scratch Disease

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Received: 03 Dec 2025
Accepted: 12 Dec 2025
Published: 24 Dec 2025
J Short Name: ACMCR

Citation:

Jian Liu, A Complex Autoimmune Case Complicated by Cat Scratch Disease. Ann Clin Med Case Rep® 2025; V15(1): 1-5

1. Abstract

A 30-year-old female patient with a complex medical history, including autoimmune hemolytic anemia, systemic lupus erythematosus (SLE), and hyperthyroidism, has experienced recurrent blood cell decreases over the past 17 years. Recently, she was admitted to the hospital due to a 6-day history of fever, which was slightly alleviated by ibuprofen, but the fever recurred intermittently.

Upon admission, imaging studies of the chest and abdomen revealed enlargement or nodules in organs such as the liver, thyroid, and lungs. Broad-spectrum antibiotics were used for infection treatment, and the patient's temperature returned to normal, but fever still persisted. Tests for mycoplasma pneumonia, chlamydia pneumonia, and blood cultures were all negative. Initial questioning of the patient's medical history revealed no significant findings. Ultimately, high-throughput genomic sequencing (mNGS) identified a significant presence of Bartonella henselae in both whole blood and plasma. Upon further questioning, the patient reported being scratched by a pet cat seven months ago and was diagnosed with cat scratch disease (CSD).

Given her long-term use of immunosuppressive therapy, her immune system was compromised, making her more susceptible to severe infections after exposure. After a multidisciplinary team (MDT) discussion, the decision was made to treat the CSD infection with doxycycline and quinolone antibiotics. Following treatment, the patient showed significant clinical improvement, with resolution of the fever and a good therapeutic outcome.

This case highlights the challenges in managing infections in immunocompromised patients, particularly those with multiple overlapping autoimmune conditions. Patients with severe autoimmune impairment are more vulnerable to infections, such as pneumonia, and may present with complicated symptoms. Initially, the patient's recurrent fever was treated as a common infection without success, but the final diagnosis of cat scratch disease was made. The patient's long incubation period, complex course, and risk of misdiagnosis emphasize the importance of detailed management and a comprehensive approach to treating complex, immunocompromised patients.

2. Introduction

Cat Scratch Disease (CSD) is a zoonotic disease caused by Bartonella henselae, usually transmitted to humans through cat scratches or bites [1]. Typical symptoms include localized lymphadenopathy and mild fever, which is usually self-limiting in immunocompetent individuals. However, in immunocompromised patients, the clinical presentation of CSD can be more severe, with the infection potentially spreading rapidly and leading to a systemic infection or life-threatening conditions. The diagnosis of CSD primarily relies on medical history, clinical presentation, and laboratory tests (such as antibody detection or lymph node biopsy). In some immunocompromised patients, CSD can lead to more complex clinical manifestations, even progressing to a systemic disease, including liquefaction in the liver and intra-abdominal lymph nodes [2,3].

Autoimmune Hemolytic Anemia (AIHA) is a condition in which the immune system abnormally targets and destroys the body's own red blood cells, leading to anemia [4]. This condition is often associated with other autoimmune diseases, such as Systemic Lupus Erythematosus (SLE) [5]. The typical symptoms of AIHA include fatigue, pallor, jaundice, and splenomegaly. Diagnosis primarily relies on the Coombs test and haematological examination. AIHA results from a dysfunction of the immune system, which means that patients with compromised immune function are more susceptible to infections, and such infections may exacerbate the underlying disease [6]. SLE is a chronic, systemic autoimmune disease that typically affects multiple organs, including the skin, joints, kidneys, and heart. Common symptoms of SLE include a butterfly-shaped rash, joint pain, oral ulcers, and kidney damage. The pathogenesis of SLE involves the immune system mistakenly attacking the body's own tissues and organs. Diagnosis of SLE relies on clinical manifestations, laboratory tests (such as anti-double-stranded DNA antibodies, antinuclear antibodies, etc.), and other imaging studies [7,8].

Epilepsy is a neurological disorder characterized by recurrent seizures [9]. These seizures typically result from abnormal electrical activity in the brain's neurons. Various factors can trigger epileptic episodes, including medications, infections, and hypoxia. Epilepsy may become more difficult to control due to the presence of autoimmune diseases or other pathological conditions, necessitating long-term pharmacological treatment and regular monitoring [10].

Hyperthyroidism refers to the excessive secretion of thyroid hormones, leading to an accelerated metabolism. The most common cause is Graves' disease, an autoimmune disorder of the thyroid gland. Typical symptoms of hyperthyroidism include weight loss, palpitations, hand tremors, increased appetite, and anxiety. Diagnosis of hyperthyroidism is usually based on serum tests (elevated T3 and T4 levels, decreased TSH), and treatment options include medication, radioactive iodine therapy, and surgery.

These diseases reflect the diverse and complex responses of the immune system, particularly in immunocompromised states, where patients' conditions may become more intricate and challenging to manage. Immunosuppressive therapy can trigger or exacerbate the risk of certain immune-related diseases and may make patients more susceptible to infections such as cat scratch disease (CSD), thereby complicating disease treatment and management.

We present a complex case of a 30-year-old female patient with a history of autoimmune haemolytic anaemia, systemic lupus erythematosus (SLE), epilepsy, and hyperthyroidism, who also suffered from a severe infection of cat scratch disease (CSD). The patient's condition was further complicated by long-term immunosuppressive therapy and a 17-year history of recurrent cytopenia. She was admitted to the hospital due to a fever lasting six days, which temporarily subsided with ibuprofen but recurred. Rapid diagnosis of CSD was achieved through mNGS (metagenomic next-generation sequencing), and after multidisciplinary team (MDT) discussion, treatment with doxycycline and quinolone antibiotics was initiated, leading to significant clinical improvement. The combination of immunosuppressive therapy, long-term steroid use, and infection placed the patient at high risk for disease exacerbation, highlighting the critical importance of early diagnosis and timely intervention in this case.

3. Case Report

The patient was initially diagnosed with autoimmune haemolytic anaemia in 2007 but exhibited no obvious external symptoms. In 2008, she was hospitalized again due to seizures and was diagnosed with epilepsy. She was prescribed cyclosporine A (50 mg twice daily) and dexamethasone tablets (2.5 tablets once daily) for 10 years. In 2017, the patient was admitted again due to fatigue and was diagnosed with systemic lupus erythematosus (SLE) and hyperthyroidism in addition to autoimmune haemolytic anaemia. She was maintained on long-term treatment with methylprednisolone tablets (1 tablet daily for anti-inflammatory purposes), methimazole tablets (0.5 tablet daily to control hyperthyroidism), cyclosporine soft capsules (50 mg twice daily), and calcium supplements, which kept her condition stable. Throughout this period, the patient experienced recurrent episodes of cytopenia.

On October 8, 2024, the 30-year-old female patient was admitted to the hospital due to a persistent fever lasting six days. Self-administration of ibuprofen provided temporary relief but failed to resolve the fever. Upon admission, the medical team considered the possibilities of systemic lupus erythematosus and haemolytic anaemia relapse, as well as bloodstream infection. Systemic lupus erythematosus and haemolytic anaemia were reconfirmed, and abnormal results were noted in the complete blood count. Four blood cultures showed no evidence of bacteria or fungi, and tests for malaria parasites, Mycoplasma pneumoniae, and Chlamydia were negative. Bone marrow routine examination revealed an elevated NAP (neutrophil alkaline phosphatase) score. Bone marrow biopsy indicated that the granulocytic and erythroid series were predominantly composed of mid-to-late-stage immature cells, with scattered megakaryocytes and segmented nuclei cells being the majority. PAS (periodic acid-Schiff) staining was positive, as shown in Figure 1.

Sequential treatments were administered, including piperacillin-tazobactam combined with moxifloxacin (October 8, 2024 – October 12, 2024), and meropenem combined with vancomycin for anti-infective therapy (October 12, 2024 – October 17, 2024). Additionally, methylprednisolone was given at a dose of 1 vial (20 mg) once daily (QD) via intravenous infusion for anti-inflammatory purposes. The patient's temperature remained intermittent (a single intravenous infusion of dexamethasone 5 mg was administered in the early morning of October 15, 2024). The patient experienced chills and occasional rigors during fever episodes and complained of significant back pain.

Colour Doppler ultrasound revealed an enlarged left ventricle, mild mitral valve regurgitation, diffuse thyroid disease with enlargement, bilateral renal calculi, and altered liver echoes. Due to the persistent fever, chest and abdominal CT scans (Figure.2) were performed, which showed solid micro-nodules in the lower lobes of both lungs, diffuse thyroid enlargement, pleural effusion with segmental atelectasis in the lower lobes of both lungs, hepatomegaly, abdominal edema, and bilateral renal calculi. MRI of the spine ((Figure.3) showed morphological changes in multiple thoracolumbar vertebrae and subcutaneous soft tissue edema in the thoracolumbar region.

Given the complex condition and the unclear cause of the fever, with no abnormalities observed in the 8-week history, mNGS testing was conducted, which detected Bartonella species in both blood and plasma. The patient reported being scratched by a domestic cat seven months prior.

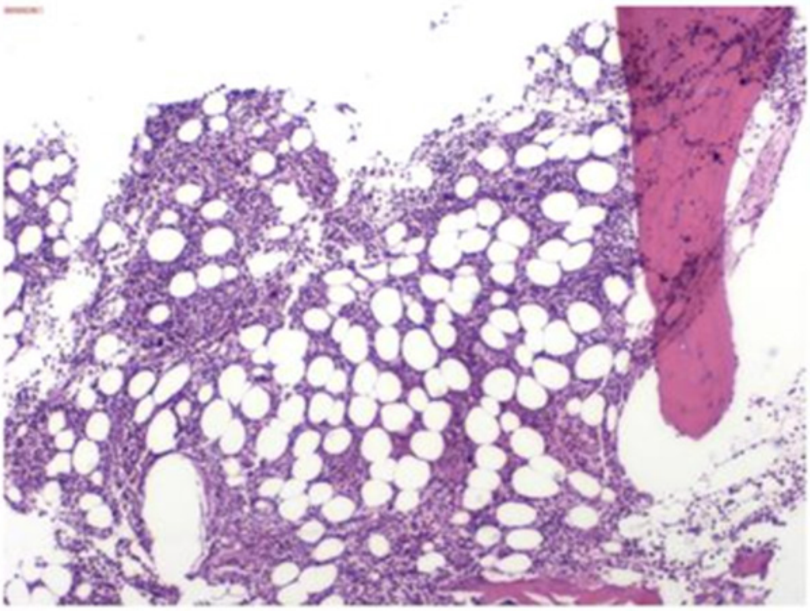

Figure 1: PAS Staining Image.

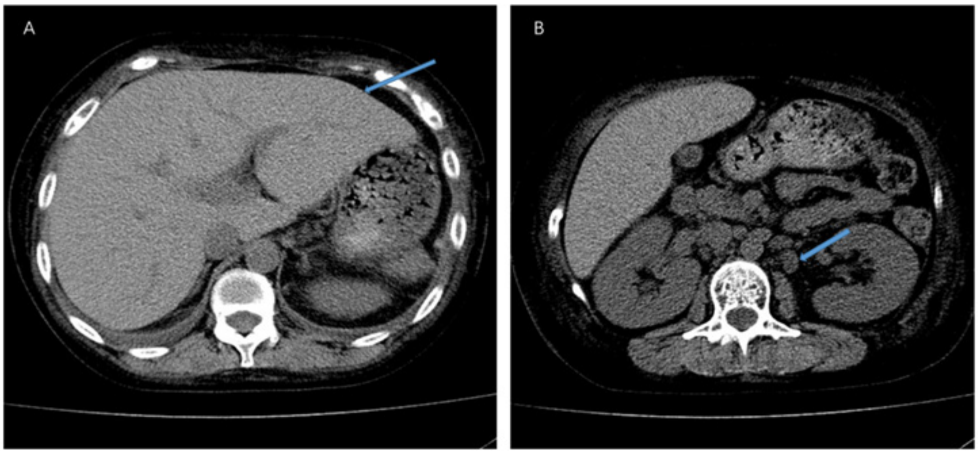

Figure 2: Chest and abdominal CT: (A) hepatomegaly (B) Retroperitoneal lymphadenopathy.

Figure 3: MRI of the spine: Multiple biconcave vertebral bodies in the thoracolumbar region.

The rapid diagnosis of cat-scratch disease via mNGS led to a multidisciplinary team (MDT) discussion, resulting in the decision to treat with tetracycline and quinolone antibiotics. The regimen included doxycycline tablets at 0.1 g orally twice daily (bid) combined with intravenous infusion of moxifloxacin at 400 mg once daily (qd). Due to high fever, a single intravenous injection of dexamethasone 5 mg was administered on October 14, 2024. Considering the possibility of haemolytic anaemia, given the patient's decreased haemoglobin levels, the dose of methylprednisolone was increased to 40 mg intravenous infusion once daily (qd). Subsequently, the patient's temperature gradually normalized without recurrent fever, and the steroid dose was tapered. The patient was discharged on October 27, 2024. During the follow-up visit one month later, the patient had not experienced fever again. The patient has been continuously monitored to date and remains fever-free.

4. Discussion

Systemic lupus erythematosus (SLE) is an autoimmune disease in which the immune system is abnormally activated, attacking the body's own tissues and organs, leading to systemic multi-organ involvement, such as autoimmune haemolytic anaemia and lupus nephritis. Long-term use of corticosteroids and immunosuppressants is required to control the disease. The patient in this case had a complex medical history, initially diagnosed with autoimmune haemolytic anaemia that was refractory to treatment, which later progressed to systemic lupus erythematosus, along with comorbidities including epilepsy and hyperthyroidism. The patient had been on corticosteroids and immunosuppressants for 17 years.

Cat scratch disease (CSD) is a self-limiting disease characterized by localized lymphadenopathy. The causative agent, Bartonella ensilage, is a polymorphic, Gram-negative, silver-staining bacterium that primarily affects adolescents. After a scratch, the disease typically has a latent period of 2 weeks to several months, with lymph node enlargement often occurring in drainage areas such as the elbow, axilla, and neck, while inguinal involvement is rare. Some patients experience fever, malaise, and muscle or joint pain. Most patients recover spontaneously within 2 to 3 months, but some may present with atypical clinical manifestations such as facial nerve palsy, gastroenteritis, myelitis, and retinal lesions.

The standard diagnosis of CSD is based on a typical history, exclusion of other causes, and characteristic histological and serological findings. J.A. Vargas-Hitos and colleagues reported the first case of CSD in a patient with SLE who owned a cat and was occasionally scratched, making the diagnosis of cat scratch disease more straightforward [11]. A literature review by Rošić et al [12]. indicated that persistent fever after contact with a cat is the most common symptom of Bartonella ensilage infection in transplant patients [12-15]. However, in this case, the patient did not own a cat but was scratched by another person's cat 7 months prior. There were no symptoms in the 6 months following the scratch, and the patient did not mention the cat scratch history when admitted. It was only after high-throughput sequencing (mNGS) detected Bartonella ensilage that a detailed history of cat contact was elicited.

This case had a long incubation period, which is easily overlooked. We speculate that this may be related to the complexity of the patient's autoimmune disease. This suggests that for such complex cases, the conventional retrospective history period should be appropriately extended. Furthermore, during the fever phase, the patient exhibited a significant increase in white blood cell count, which raised concerns about excluding steroid use and infection stimulation, which complicated the diagnosis.

Bartonella ensilage is sensitive or highly sensitive to many antibiotics and their derivatives, such as aminoglycosides, revamping, ciprofloxacin, and azithromycin, in vitro. However, the correlation between the minimum effective inhibitory concentration of antibiotics and the clinical efficacy is relatively poor. This discrepancy may be due to the ability of Bartonella ensilage to parasitize red blood cells or endothelial cells in the human host, allowing the bacteria to persist in the host's bloodstream. Clinical treatment generally involves combination therapy with antibiotics, with recommendations for aminoglycosides, azithromycin, or quinolones, as they are more effective, and treatment generally lasts for more than 2 weeks.

In this case, due to the patient's long-term immunosuppressant use and compromised immune function, the risk of disease exacerbation following infection was higher. After a rapid diagnosis of CSD via mNGS, a multidisciplinary team (MDT) discussion led to the recommendation of doxycycline combined with quinolone antibiotics for treatment, ultimately resulting in significant clinical improvement for the patient.

5. Conclusion

Cat-scratch disease (CSD), which typically has a relatively benign clinical presentation and prognosis, may manifest as a severe and life-threatening illness in immunocompromised hosts. In such cases, almost any organ system can be affected, and the outcomes are unpredictable. This requires a deep understanding of the patient's immune processes and necessitates early diagnosis to implement appropriate and effective treatment. According to this study, although the relevant data are limited, reintroducing immunosuppressive therapy after successful treatment of CSD does not appear to increase the risk of disease reactivation.

Regarding the treatment of CSD, the disease is often self-limiting, and most cases do not require medication. However, in immunocompromised hosts, such as patients with systemic lupus erythematosus (SLE) who are on long-term immunosuppressive therapy, as seen in this case, the immune system is weakened. When such patients develop CSD, the condition may worsen significantly. If not treated promptly and thoroughly, complications such as bacillary angiomatosis may arise, potentially leading to encephalopathy, osteomyelitis, chronic and severe organ damage, and even death.

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