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CASE REPORT

A Rare Case of Yersinia Pseudotuberculosis Infection with Septic Shock and Splenic Infarction

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Abstract: *Yersinia pseudotuberculosis* is a Gram-negative bacterium of the family *Yersiniaceae*, primarily transmitted via the gastrointestinal tract. Progression to sepsis is uncommon, and the combination of septic shock and splenic infarction is exceedingly rare. We report a 40-year-old male who initially presented with fever, abdominal pain, and distension, which rapidly progressed to sepsis and multi-organ dysfunction. Definitive diagnosis of *Y. pseudotuberculosis* infection was established by blood culture and metagenomic next-generation sequencing, supported by imaging evidence of splenic infarction. The patient was managed with stepwise antimicrobial regimens (including piperacillin–tazobactam, meropenem, levofloxacin, linezolid, and daptomycin), plasma exchange, continuous renal replacement therapy, and organ function support. Following comprehensive treatment, the patient recovered and was discharged in stable condition. This case highlights the importance of considering *Y. pseudotuberculosis* in atypical sepsis presentations and demonstrates that timely diagnosis and multidisciplinary management are crucial to improving outcomes in such rare and life-threatening infections.

Keywords: *yersinia pseudotuberculosis*, septic shock, splenic infarction, case report, nursing

Introduction

Yersinia pseudotuberculosis is a foodborne pathogen belonging to the Gram-negative genus *Yersinia* and is closely related to *Yersinia enterocolitica*.[1,2] This bacterium is an emerging zoonotic pathogen, primarily hosted by wild or domesticated animals, and human infections are mostly transmitted through contaminated food or water.[3] Clinically, most patients present with mild or self-limiting intestinal infections, with typical symptoms including fever, abdominal pain, diarrhea, and mesenteric lymphadenitis, which can often mimic acute appendicitis.[4,5] The overall incidence of human *Y. pseudotuberculosis* infection is low, with sporadic outbreaks reported in Europe and Asia, and progression to sepsis occurs in fewer than 5% of cases. Severe complications such as septic shock, multiple organ dysfunction, and splenic infarction are extremely rare, with only isolated cases documented worldwide,[6,7] Host comorbidities (eg, diabetes mellitus, immunosuppression) and bacterial virulence factors such as Yersinia outer proteins (Yops) have been implicated in severe progression.[8,9] In this report, we present a case of *Y. pseudotuberculosis* infection complicated by septic shock and splenic infarction, aiming to supplement the limited clinical data on these rare complications and to improve diagnostic awareness for timely recognition and multidisciplinary management.

Case Presentation

The patient is a 40-year-old male farmer who developed fever on May 28, of unknown etiology, predominantly occurring at night with a maximum temperature of approximately 38.5°C. He self-medicated with oral antipyretics (details unknown). On June 1, the patient reported a significant worsening of the fever, accompanied by persistent abdominal pain and distension. The pain was generalized across the abdomen, predominantly in the upper abdomen, and was associated with nausea, without vomiting, diarrhea, or melena. On June 2, the patient sought treatment at the local health

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center, where abdominal CT showed uneven liver density, splenomegaly, and mesenteric lymphadenitis. The blood routine showed 14.43×10^9/L leukocytes; 13.02×10^9/L neutrophils; 90.2% centrocytes; 67g/L hemoglobin; 111×10^9/L platelets; total bilirubin 31.42umo1/L; direct bilirubin 15.35umo1/L; glutamic oxaloacetic aminotransferase 159.5U/L; glutamic cetyl aminotransferase 139.1U/L; and liver function tests showed the following, aminotransferase 139.1 U/L; glucose 25.68 mmo1/L; CRP 262.7 mg/L. The patient presented with herpes zoster on the lower back 15 days ago, and was treated with antiviral therapy at a local clinic (details unknown), and the herpes has now crusted over. There was a past history of diabetes mellitus, and during clinical data collection it was found that the patient had consumed raw cucumber stored in the refrigerator, which may have been the source of Yersinia pseudotuberculosis infection.

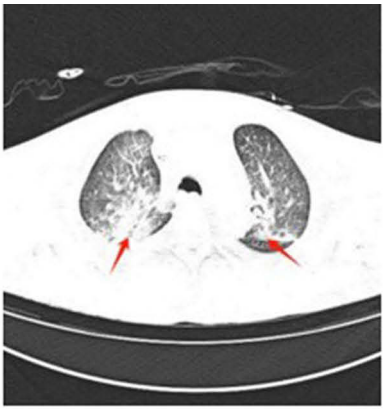
On June 3(The first day of admission), he was transferred to our hospital, and was admitted to the department for physical examination: clear consciousness, acute pain face, body temperature of 38.5°C, pulse of 111 beats/min, respiration of 30 breaths/min, blood pressure of 80/59mmHg; oxygen saturation level of 95%; no yellowing of the skin and sclera, and no rash; abdominal muscle tension of the whole abdomen, epigastric compression pain and rebound pain, abdominal mass, liver and spleen were not found in the subcostal area; the urine was pus-filled (urinalysis and urine culture were performed). Laboratory tests: leukocytes 16.34×10^9/L; erythrocytes 2.77×10^{12}/L; hemoglobin 61g/L; platelets 152×10^9/L; neutrophil percentage 87.7%; prealbumin 74mg/L; total bilirubin 29.2umo1/L; direct bilirubin 13.5umo1/L; creatinine 101umol/L; C-reactive protein 325.00 mg/L; interleukin663918pg/m1; prothrombin time 18.3 seconds; prothrombin activity 55.0%; activated partial thromboplastin time 60.7 seconds; PCT 9.6 mg/L; blood gas analysis showed PH 7.46; PaO2 174.98 mmHg; PaC02 32.04 mmHg; urine leukocytes (+)). Imaging studies suggested bilateral pulmonary exudates, bilateral pleural effusions, and liver abscesses (Figure 1).

Upon admission, the patient was diagnosed with sepsis complicated by septic shock, liver abscess, urinary tract infection, type 2 diabetes mellitus, acute renal failure, abnormal liver function, and pleural effusion. He received high-flow oxygen (35 L/min), vasoactive agents (norepinephrine 0.17 μg/kg/min and pituitrin 0.36 U/h) to maintain blood pressure, anti-infective therapy with piperacillin-tazobactam (4.5 g every 6 hours), and continuous renal replacement therapy (CRRT) to stabilize the internal environment, along with supportive treatments including acid suppression, gastric protection, hepatoprotective therapy, and temperature management.

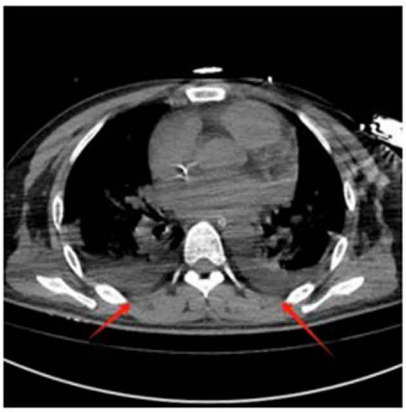
On June 5 (the third day of admission), the patient's temperature remained above 38.5°C (maximum 39.2°C). The dose of vasoactive drugs was increased compared to the previous day (norepinephrine 0.34 μg/kg/min, pituitrin 0.6 U/h). Relevant auxiliary examinations showed the following results: red blood cells 2.94×10^{12}/L, hemoglobin 63g/L, neutrophil count 7.99×10^9/L, neutrophil percentage 90.0%, interleukin 6>5000pg/mL, alanine aminotransferase 219U/L, aspartate aminotransferase 301U/L, total bilirubin 24.0μmol/L, direct bilirubin 14.9μmol/L, urea 9.38mmol/L, creatinine 118 μmol/L, C-reactive protein 362.23mg/L, urine leukocytes ++. Blood culture results suggested a Gram-negative bacillus infection, and liver and kidney functions worsened. Therefore, the antibiotic regimen was switched to meropenem (0.25g q8h) to continue anti-infection treatment, and plasma exchange was initiated. Hydrocortisone (0.1g q12h) and ustekinumab (20WU) were added for anti-inflammatory and symptomatic treatment. Additionally, a blood specimen was sent for next-generation sequencing (NGS) to further identify the infection source. On the same day, a repeat abdominal CT showed splenic infarction (Figure 2), though no specific intervention was performed.

On June 6 (the fourth day of admission) at 9 a.m., the patient presented with a heart rate of 48 beats/minute and an ECG showing partial S-T segment changes. Given the patient's medical history, pericarditis was suspected, though myocardial infarction could not be excluded. Meanwhile, metagenomic next-generation sequencing (mNGS) of a blood sample (No. UGPE4W5S), performed by Hangzhou Matrix Biotechnology Co., Ltd. on the Illumina sequencing platform with PCR-free library preparation, identified Yersinia pseudotuberculosis (7 sequence reads; relative abundance, 17.08%; quality score, 10,633.27), confirming the bloodstream infection. The patient was diagnosed with *Yersinia pseudotuberculosis* sepsis, complicated by septic shock, multiple organ dysfunction syndrome (MODS), and liver function impairment. After a multidisciplinary discussion, the patient was treated with levofloxacin 500mg qd and gentamicin 3mg/kg for anti-infection therapy, and continued treatment with plasma exchange, continuous renal replacement therapy (CRRT), ustekinumab, and corticosteroid-based anti-inflammatory therapy.

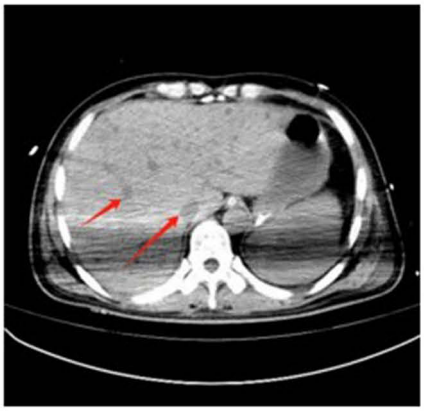
On June 7 (the fifth day of admission), the blood culture obtained on the day of hospitalization yielded a positive result using the automated BACT/ALERT 3D blood culture system (bioMérieux, France), and the isolated pathogen was identified as



(A)



(B)



(C)

Figure 1 (A) Lung CT images both lower lungs show a cloudy shadow with mildly increased density, suggesting an exudative lesion in both lower lungs (arrow); **(B)** CT shows a crescent-shaped low-density area with curved lines concave to the posterior medial side, with mild localized compression of the lung tissues, suggesting pleural effusion in both lungs (arrow); **(C)** Abdominal CT of the liver with multiple round or elliptical low-density areas (arrow).

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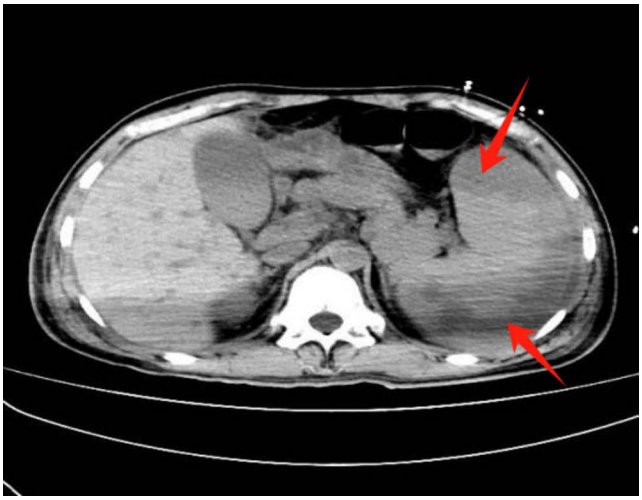

Figure 2 Abdominal CT suggests multiple low-density masses under the pericardium at the outer edge of the spleen with well-defined borders(arrow).

Yersinia pseudotuberculosis by an automated microbial mass spectrometry detection system (VITEK MS, Kratos Analytical Limited, UK). A review of relevant tests showed the following results: alanine aminotransferase 562 U/L, aspartate aminotransferase 717 U/L, total bilirubin 88.8 μmol/L, direct bilirubin 48.7 μmol/L, total protein 53.20 g/L, albumin 31.10 g/L, hydroxybutyrate dehydrogenase 734 IU/L, lactate dehydrogenase 977 IU/L, prothrombin time 22.0 seconds, plasminogen activity 41.0%, activated partial thromboplastin time 51.5 seconds, leukocytes 26.28×10^{9}/L, erythrocytes 3.53×10^{12}/L, hemoglobin 84 g/L, B-type natriuretic peptide 909.90 pg/mL, and calcitonin 27.36 ng/mL. The patient continued to receive anti-infective treatment (meropenem 0.25g q8h, levofloxacin 0.2g qd), anti-inflammatory therapy (ustekinumab 20WU q8h), continuous renal replacement therapy (CRRT) to maintain internal environment stability, acid suppression and gastric protection, hepatoprotection, nutritional support, and other treatments.

On June 8 (the sixth day of admission), the patient was reexamined, revealing platelets of 40×10^{9}/L, prothrombin activity of 45.0%, total bilirubin of 131.2 μmol/L, and direct bilirubin of 79 μmol/L. Given the worsening hepatic failure and jaundice, Ademetionine, Transmetil (1g qd) was added to the treatment regimen.

On June 11 (the ninth day of admission), blood culture results identified *Staphylococcus* species (Gram-positive cocci), which prompting the addition of Linezolid (600 mg q12h). Since meropenem had been administered for one week and the patient's infection markers had decreased compared to previous measurements, the anti-infective therapy was switched to piperacillin-tazobactam (4.5g q8h) in a step-down approach. Levofloxacin treatment was continued for *Yersinia pseudotuberculosis* infection.

On June 12 (the tenth day of admission), the patient's temperature returned to normal; however, on June 15 (The thirteenth day of admission), the patient developed a high fever again, with a maximum temperature of 38.3°C. As a result, linezolid was switched to daptomycin, and piperacillin-tazobactam was continued for anti-infective therapy. Levofloxacin, which had been used for 10 days, was discontinued to avoid potential side effects and prevent the development of drug-resistant bacteria. The patient's hemodynamic status gradually improved, and vasoactive drugs were sequentially discontinued. The patient underwent comprehensive treatment (Table 1), and their condition gradually improved. Vital signs, including temperature, heart rate, respiratory rate, and blood pressure, gradually returned to normal, and oxygen saturation was maintained within the normal range (Table 2). Respiratory function parameters, such as the oxygenation index (P/F ratio), significantly improved, and lactate levels decreased to normal (Table 3). Laboratory tests showed that white blood cell count, hemoglobin, platelets, and other indicators gradually returned to normal, while liver and kidney function markers (such as total bilirubin and creatinine) significantly improved (Table 4).By June 20 (The eighteenth day of admission), the patient was no longer in a life-threatening condition and was transferred to the general ward of the Department of Infectious Diseases for further treatment. The patient was discharged on July 5 and followed up 28 days after discharge, showing good health.

Table 1 Patient's Treatment in the ICU

Treatment	Jun 3	Jun 5	Jun 6	Jun 7	Jun 8	Jun 9	Jun 11	Jun 12	Jun 13	Jun 15	Jun 16	Jun 18	Jun 20
Norepinephrine (ug/kg/min)	0.17	+	+	+	+	+	+	+	+	stop			
Pituitrin (u/h)	0.36	+	+	+	+	stop							
Piperacillin tazobactam	4.5g/q6h	+	+	+	+	+	+	+	+	+	+	+	Continued after transfer 4.5g/q8h
CRRT	CVVHDF	+	+	+	+	+	+	+	stop				
Meropenem		0.25g/q8h	+	+	+	+	stop						
Hydrocortisone		0.1g/q12h	+	stop									
Ustekinumab		20WU/q8h	+	+	+	+	+	+	+	+	+	stop	
Levofloxacin			500mg/qd	+	+	+	+	+	+	stop			
Gentamicin			3mg/Kg	+	+	+	+	+	+	stop			
Ademetionine, transmetil				1g/qd	+	+	+	+	+	+	stop		
Linezolid							600mg/q12h	+	+	+	+	+	stop
Daptomycin								0.5g/qd	+	+	+	stop	
Plasma exchange		qd	+	+	+	+	+	+	stop				

Note: "+" indicates continuous use of the measure without change or adjustment. The patients' plasma exchange was performed with 2000 mL of fresh frozen plasma per session.

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Table 2 Patient's Vitals in ICU

Test	Jun 3	Jun 5	Jun 6	Jun 12	Jun 15	Jun 20
Max temperature (°C)	38.5	39.2	37.5	36.9	38.3	36.7
Heart rate (bpm)	111	128	48	85	127	101
Respiration (bpm)	30	30	18	28	29	29
Bloodpressure (mmHg)	80/59	97/64	113/63	151/87	149/71	141/71
SpO_2 (%)	95	99	93	100	99	100
Perfusion index	3	1.7	1.2	3.4	2.9	3.5

Abbreviation:SpO2, peripheral capillary oxygen saturation.

Table 3 Patient's Respiratory Function

Parameter	Jun 3	Jun 5	Jun 6	Jun 12	Jun 15	Jun 20
Respiratory pattern	High flow	High flow	High flow	High flow	High flow	High flow
FiO_2 (%)	45	45	40	35	35	35
P/F (mmHg)	389	309	316	480	262	568
Lac (mmo1/L)	1.94	4.38	9.73	1.50	2.63	1.86

Abbreviations: FiO_2, fraction of inspired oxygen; P/F, Lac, lactate.

Table 4 Patient's Laboratory Results

Parameter	Jun 3	Jun 5	Jun 6	Jun 12	Jun 15	Jun 20
WBC (10^9/L)	16.34	31.38	34.98	20.21	17.85	9.76
PLT (10^9/L)	152	109	89	54	110	105
NEU (%)	14.32	28.49	31.27	15.48	18.63	7.66
GR (%)	87.7	90.9	89.4	87.3	85.6	80.4
TBIL (umo1/L)	29.2	55	80.1	142.7	47.1	18.9
DBIL (umo1/L)	13.5	34.6	45.1	86.8	32.3	11.2
CRE (umol/L)	101	258	186	184	183	146
CRP (mg/L)	325	532.45	353.97	117.26	122.04	92.05
PT (s)	18.3	25.6	28.6	17.9	17.1	16.6
PA (%)	55	35	46	46	62	65
APTT (s)	60.7	111.3	80	43.7	47.8	51.2
PCT (mg/L)	9.6	85.23	34.43	6.53	5.21	2.93
BAm (ug/dl)	–	71	–	–	62	32
AST (u/L)	177	301	3169	47	24	28
ALT (u/L)	156	219	1112	49	17	18
BG (mmo1/L)	21.3	13.2	14	16.2	12.3	12.3

Abbreviations: WBC, white blood cell count; PLT, platelet count; NEU, neutrophils; GR, granulocyte; TBIL, total bilirubin; DBIL, direct bilirubin; CRE, creatinine; CRP, c-reactive protein; PT, prothrombin Time; PA, prothrombin activity; APTT, activated partial thromboplastin time; PCT, procalcitonin; BAm, blood ammonia; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BG, blood glucose.

Discussions

Recognition of Yersinia Pseudotuberculosis Infection

Yersinia pseudotuberculosis infection in humans primarily invades the mesenteric lymph nodes, causing inflammation of the ileocecal region.[10] The common clinical manifestations include fever, right lower abdominal pain, diarrhea, and hepatic abscesses.[11,12] Standard treatment relies on early pathogen detection and targeted antibiotic therapy, such as aminoglycosides, third-generation cephalosporins, or fluoroquinolones.[13] In this patient, the main clinical manifestation was persistent generalized abdominal pain, which was most severe in the upper abdomen, without vomiting or diarrhea.

These symptoms were inconsistent with the typical clinical presentation of *Y. pseudotuberculosis* infection. However, it has been reported that some patients may present only with abdominal pain, which may lead to misdiagnosis or missed diagnosis in the early stages of the disease.[6]

The primary sources of Y. pseudotuberculosis infection are carrier animals, and transmission occurs mainly via the fecal–oral route.[14] The patient reported no known relevant exposure history but did have a history of herpesvirus infection and had consumed raw cucumbers stored in a refrigerator one week prior to admission. Y. pseudotuberculosis is psychrotrophic, capable of surviving and multiplying in low-temperature environments, and is commonly found in raw meat, dairy products, and vegetables.[15,16] Thus, it was initially considered that the infection in this patient may have been triggered by consumption of contaminated food in the context of reduced immunity following herpesvirus infection. Herpesviruses are primarily transmitted through direct contact (eg, skin-to-skin or mucosa-to-mucosa contact). Although the transmission routes of herpesviruses and Y. pseudotuberculosis differ, herpesvirus infection may weaken mucosal barriers and alter host immune responses, thereby facilitating bacterial invasion and modifying clinical manifestations.

Differential diagnosis from other intestinal infections is also crucial. Typhoid fever often presents with high fever and watery diarrhea;[17] bacillary dysentery typically manifests as left lower abdominal pain with mucoid bloody stools;[18] intestinal *Escherichia coli* infection is mainly characterized by watery or hemorrhagic diarrhea.[19] None of these features were consistent with the present case, suggesting that *Y. pseudotuberculosis* infection should be considered even in atypical cases. At present, stool or blood pathogen testing remains the gold standard for confirming *Y. pseudotuberculosis* infection.[20] To better understand the particularity of this case, we compared the three major pathogenic *Yersinia* species, which exhibit significant differences in transmission, clinical manifestations, and severity. *Y. pestis* is the causative agent of plague, primarily transmitted via flea bites or respiratory routes, leading to septicemic or pneumonic plague, often without preceding gastrointestinal symptoms.[21] *Y. enterocolitica* is a typical enteric pathogen, commonly causing self-limiting gastroenteritis with watery diarrhea as the main symptom.[22] *Y. pseudotuberculosis*, though less common, is usually acquired through ingestion of contaminated food, causes mesenteric lymphadenitis, and may progress via the enteric route to sepsis.[23] Growing evidence indicates that, unlike *Y. pestis*, which rapidly disseminates systemically and suppresses early host inflammatory responses via the type III secretion system (T3SS) and modified LPS structures, leading to fulminant septicemia,[5,8] *Y. pseudotuberculosis* sepsis primarily proceeds through intestinal invasion, initially presenting as localized mesenteric lymphadenitis, before hematogenous dissemination to organs such as the liver and spleen via intestinal lymphatics or the portal circulation—a fundamentally different mechanism of sepsis.[13,24] In addition, subspecies-level identification is of great significance, as differences in virulence may explain variations in disease severity and prognosis.

Treatment and Care of Septic Shock

Septic shock is a leading cause of mortality in critically ill patients, and early recognition and timely management are crucial for treatment.[25,26] Upon admission, the patient met the diagnostic criteria for sepsis, presenting with fever, hypotension (<70 mmHg), elevated lactate, and signs of an acute abdomen. Fluid resuscitation and hemodynamic support were performed according to goal-directed protocols. Lactate served as the main indicator of tissue perfusion, with resuscitation targets including mean arterial pressure (MAP) ≥65 mmHg, urine output ≥0.5 mL/(kg·h), and central venous oxygen saturation ($ScvO_2$) ≥70%. Initial resuscitation included administration of 20 g albumin, 1000 mL colloid solution, and 1000 mL crystalloid solution. When lactate levels continued to rise and urine output declined, vasoactive agents were adjusted, and continuous renal replacement therapy (CRRT) and plasma exchange were implemented.[27,28] The peripheral perfusion index (PPI) was used as a supplementary measure to lactate monitoring.[29] A PPI <2 was associated with elevated lactate levels, whereas lactate decreased after fluid resuscitation when PPI reached ≥2.5, indicating that bedside noninvasive monitoring can effectively reflect tissue perfusion.[30] Antimicrobial therapy was directed against enteric and Gram-negative bacteria, with initial use of fluoroquinolones, β-lactams, carbapenems, or aminoglycosides.[14,31] When infection markers rose again, linezolid and daptomycin were added, successfully controlling fever and the inflammatory response. Organ support included high-flow oxygen to improve tissue oxygen delivery, CRRT for acute kidney injury, plasma exchange for suspected liver injury, and intravenous insulin for blood glucose control to reduce lactate production associated with hyperglycemia. Following these interventions, lactate levels normalized on day 5 after admission, urine output improved, and organ function stabilized.

Care of Patients with Splenic Infarction

The primary cause of splenic infarction in patients is considered to be a reduction in blood perfusion to the spleen following shock, which slows blood flow within the splenic artery and its branches. This, in turn, leads to tissue hypoxia, causing vasospasm and vascular occlusion, ultimately triggering splenic infarction.[6] The most serious complication of splenic infarction is hemorrhage following splenic rupture, which is primarily manifested by a sudden increase in left upper abdominal pain, abdominal distension, hypotension, and the presence of a fluid collection around the spleen observed on ultrasound. Therefore, early detection of relevant clinical symptoms and changes in the patient's condition is a key nursing measure for patients with splenic infarction. No splenic rupture occurred during the hospitalization of this patient.

Conclusion

This rare case of septic shock and splenic infarction caused by *Y. pseudotuberculosis* infection highlights the diagnostic challenges of atypical clinical presentations. Early recognition, targeted antimicrobial therapy, multidisciplinary organ support, and close monitoring of tissue perfusion and splenic complications are essential for favorable outcomes. Clinicians should maintain a high index of suspicion for atypical presentations to improve early diagnosis, therapeutic efficacy, and patient recovery.

Data Sharing Statement

No datasets were generated or analysed during the current study.

Ethics Approval and Consent to Participate

The present case report was approved by the Ethics Committee of the First Clinical Medical College of China Three Gorges University (NO.2025-018-01). Written informed consent was obtained from the patient and the patient's family for the publication of this clinical case report.

Consent for Publication

Written informed consent was obtained from the patient and the patient's family for publication of this case report and any accompanying images.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare no conflicts of interest in this work.

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