ISSN 1948-9366 (online)

World Journal of

Gastrointestinal Surgery

World J Gastrointest Surg 2024 October 27; 16(10): 3074-3380

Published by Baishideng Publishing Group Inc

World Journal of Gastrointestinal Surgery

Contents

Monthly Volume 16 Number 10 October 27, 2024

EDITORIAL

ORIGINAL ARTICLE

Case Control Study

Retrospective Cohort Study

Contents

World Journal of Gastrointestinal Surgery

Monthly Volume 16 Number 10 October 27, 2024

Retrospective Study

Observational Study

World Journal of Gastrointestinal Surgery

Monthly Volume 16 Number 10 October 27, 2024

Contents

LETTER TO THE EDITOR

World Journal of Gastrointestinal Surgery

Monthly Volume 16 Number 10 October 27, 2024

Contents

ABOUT COVER

Editorial Board Member of *World Journal of Gastrointestinal Surgery*, Michele Ammendola, MD, Research Associate, Surgical Oncologist, Science of Health Department, Digestive Surgery Unit, University of "Magna Graecia" Medical School, Catanzaro 88100, Italy. michele.ammendola@unicz.it

AIMS AND SCOPE

The primary aim of *World Journal of Gastrointestinal Surgery* (*WJGS*, *World J Gastrointest Surg*) is to provide scholars and readers from various fields of gastrointestinal surgery with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJGS mainly publishes articles reporting research results and findings obtained in the field of gastrointestinal surgery and covering a wide range of topics including biliary tract surgical procedures, biliopancreatic diversion, colectomy, esophagectomy, esophagostomy, pancreas transplantation, and pancreatectomy, *etc*.

INDEXING/ABSTRACTING

The *WJGS* is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Current Contents/Clinical Medicine, Journal Citation Reports/Science Edition, PubMed, PubMed Central, Reference Citation Analysis, China Science and Technology Journal Database, and Superstar Journals Database. The 2024 Edition of Journal Citation Reports® cites the 2023 journal impact factor (JIF) for *WJGS* as 1.8; JIF without journal self cites: 1.7; 5-year JIF: 1.9; JIF Rank: 126/292 in surgery; JIF Quartile: Q2; and 5-year JIF Quartile: Q3.

RESPONSIBLE EDITORS FOR THIS ISSUE

NAME OF JOURNAL
World Journal of Gastrointestinal Surgery

ISSN
ISSN 1948-9366 (online)

LAUNCH DATE
November 30, 2009

FREQUENCY
Monthly

EDITORS-IN-CHIEF
Peter Schemmer

EDITORIAL BOARD MEMBERS
https://www.wjgnet.com/1948-9366/editorialboard.htm

PUBLICATION DATE
October 27, 2024

INSTRUCTIONS TO AUTHORS
https://www.wjgnet.com/bpg/gerinfo/204

GUIDELINES FOR ETHICS DOCUMENTS
https://www.wjgnet.com/bpg/GerInfo/287

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH
https://www.wjgnet.com/bpg/gerinfo/240

PUBLICATION ETHICS
https://www.wjgnet.com/bpg/GerInfo/288

PUBLICATION MISCONDUCT
https://www.wjgnet.com/bpg/gerinfo/208

ARTICLE PROCESSING CHARGE
https://www.wjgnet.com/bpg/gerinfo/242

STEPS FOR SUBMITTING MANUSCRIPTS
https://www.wjgnet.com/bpg/GerInfo/239

ONLINE SUBMISSION
https://www.f6publishing.com

World Journal of Gastrointestinal Surgery

Submit a Manuscript: https://www.f6publishing.com

World J Gastrointest Surg 2024 October 27; 16(10): 3343-3349

DOI: 10.4240/wjgs.v16.i10.3343

ISSN 1948-9366 (online)

CASE REPORT

Leukopenia-a rare complication secondary to invasive liver abscess syndrome in a patient with diabetes mellitus: A case report

Chun-Yan Niu, Bang-Tao Yao, Hua-Yi Tao, Xin-Gui Peng, Qing-Hua Zhang, Yue Chen, Lu Liu

Specialty type: Gastroenterology and hepatology

Provenance and peer review: Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade C, Grade C

Novelty: Grade B, Grade B

Creativity or Innovation: Grade B, Grade B

Scientific Significance: Grade B, Grade C

P-Reviewer: Moldogazieva NT

Received: June 7, 2024

Revised: September 6, 2024

Accepted: September 14, 2024

Published online: October 27, 2024

Processing time: 113 Days and 3 Hours

Chun-Yan Niu, Hua-Yi Tao, Yue Chen, Lu Liu, Department of Gastroenterology, Nanjing Lishui People's Hospital, Zhongda Hospital Lishui Branch, Southeast University, Nanjing 211200, Jiangsu Province, China

Bang-Tao Yao, Department of Ophthalmology, Nanjing Lishui People's Hospital, Nanjing 211200, Jiangsu Province, China

Xin-Gui Peng, Qing-Hua Zhang, Department of Medical Imaging, Nanjing Lishui People's Hospital, Zhongda Hospital Lishui Branch, Southeast University, Nanjing 210009, Jiangsu Province, China

Co-first authors: Chun-Yan Niu and Bang-Tao Yao.

Co-corresponding authors: Yue Chen and Lu Liu.

Corresponding author: Yue Chen, Master, Chief Doctor, Department of Gastroenterology, Nanjing Lishui People's Hospital, Zhongda Hospital Lishui Branch, Southeast University, No. 86 Chongwen Road, Lishui District, Nanjing 211200, Jiangsu Province, China.
sanyudouzheng@126.com

Abstract

BACKGROUND

Thrombocytopenia is a common complication of invasive liver abscess syndrome (ILAS) by *Klebsiella pneumoniae* (*K. pneumoniae*) infection, which indicates severe infection and a poor prognosis. However, the presence of leukopenia is rare. There are rare reports on leukopenia and its clinical significance for ILAS, and there is currently no recognized treatment plan. Early and broad-spectrum antimicrobial therapy may be an effective therapy for treating ILAS and improving its prognosis.

CASE SUMMARY

A 55-year-old male patient who developed fever, chills, and abdominal distension without an obvious cause presented to the hospital for treatment. Laboratory tests revealed thrombocytopenia, leukopenia, and multiple organ dysfunction. Imaging examinations revealed an abscess in the right lobe of the liver and thrombophlebitis, and *K. pneumoniae* was detected in the blood cultures. Since the patient was diabetic and had multi-system involvement, he was diagnosed with ILAS accompanied by leukopenia and thrombocytopenia. After antibiotic treatment and systemic supportive therapy, the symptoms disappeared, and the patient's condition

almost completely resolved.

CONCLUSION
Leukopenia is a rare complication of ILAS, which serves as an indicator of adverse prognostic outcomes and the severity of infection.

Key Words: Invasive liver abscess syndrome; *Klebsiella pneumoniae*; Leukopenia; Thrombocytopenia; Treatment; Prognosis; Case report

Core Tip: Thrombocytopenia is a common complication of invasive liver abscess syndrome (ILAS). However, there have been no reports of concurrent leukopenia with ILAS so far. This patient presented with a sustained high fever, abdominal distension, and pain after hospitalization. He subsequently developed multiple organ and system dysfunction, as well as an atypical leukopenia coexisting with thrombocytopenia. These features are often used as indicators of the severity of the disease. *Klebsiella pneumoniae* was detected on blood cultures, and antibiotics were adjusted timely based on drug sensitivity test results. After systemic support and blood glucose management treatment, the patient's condition resolved. Early antimicrobial therapy is an effective measure to control infection and improve prognosis.

Citation: Niu CY, Yao BT, Tao HY, Peng XG, Zhang QH, Chen Y, Liu L. Leukopenia-a rare complication secondary to invasive liver abscess syndrome in a patient with diabetes mellitus: A case report. *World J Gastrointest Surg* 2024; 16(10): 3343-3349
URL: https://www.wjgnet.com/1948-9366/full/v16/i10/3343.htm
DOI: https://dx.doi.org/10.4240/wjgs.v16.i10.3343

INTRODUCTION

Invasive liver abscess syndrome (ILAS) comprises a liver abscess caused by hypervirulent *Klebsiella pneumoniae* (hvKp) and metastatic infections in multiple extrahepatic organs such as the brain, lungs, and eyes. ILAS is a severe clinical condition with a global mortality rate of approximately 2%-19%[1]. Clinical symptoms include fever and chills, abdominal pain, and abdominal distension; however, metastatic infections can manifest as meningitis, brain abscesses, lung abscesses, endophthalmitis, and pneumonia[2,3]. The incidence of ILAS has increased significantly over the past two decades in southeast Asian countries[4]. Risk factors for ILAS include male sex, diabetes, and immunosuppressive conditions, such as malignancies[5]. However, early detection and appropriate treatment of ILAS remain challenging.

Intestinal hvKp, which is the main pathogen implicated in ILAS, invades the liver by passing through the intestinal barrier or *via* retrograde entry into the liver. It then enters the portal vein circulation[3], leading to bacteraemia and sepsis. Haematological findings of ILAS typically include an elevated white blood cell (WBC) count and decreased platelet count [6]. To the best of our knowledge, ILAS with concurrent leukopenia has not yet been reported.

CASE PRESENTATION

Chief complaints

Fever accompanied by abdominal distension for 3 days.

History of present illness

A 55-year-old man with a 3-day history of fever and abdominal distension was admitted to our hospital (No. 698690). He also experienced chills, headaches, and abdominal pain. He denied any history of systemic and ocular diseases. On admission, his fever was remittent and fluctuated between 37.5 °C and 40.5 °C. Conjunctival congestion and tenderness in the epigastrium and right upper quadrant were also detected.

History of past illness

He was previously healthy.

Personal and family history

Previously healthy with no history of special illnesses.

Physical examination

Temperature 37.7 °C, pulse 113 times/minute, respiratory rate 18 times/minute, blood pressure 107/74 mmHg, height

170 cm, weight 70 Kg. The patient was alert, awake, and oriented, with no signs of an altered level of consciousness. He had palpable enlargement of the superficial lymph nodes and coarse respiratory sounds in both lungs. His heart rate was 113 beats *per* minute, the rhythm was regular, no murmurs were heard on auscultation of the heart, the abdomen was flat and soft, mild tenderness was found in the upper and middle upper abdomen, there was no rebound pain or pain upon percussion of the liver area, and auscultation of bowel sounds revealed four bowel sounds per minute.

Laboratory examinations

Blood routine and inflammatory markers: Laboratory tests revealed significantly increased high-sensitivity C-reactive protein, interleukin-6, and procalcitonin levels, as well as reduced platelet counts of 70 × 10^9/L (reference range: 100 × 10^9/L to 300 × 10^9/L) and WBC counts of 3.76 × 10^9/L (reference range: 4 × 10^9/L to 10 × 10^9/L) (Table 1).

Urinalysis: Urine specific gravity 1.04 (1.003-1.030), urine protein + +, glucose ±, albumin 150 mg/L (0.0-23.8), urine protein/creatinine 0.15 (0.000–0.030).

Blood biochemistry testing: The fasting blood glucose, postprandial glucose, and HbA1c levels were 8.2 mmol/L, 13.8 mmol/L, and 6.2%, respectively. Additional laboratory test results revealed liver and kidney dysfunction, electrolyte imbalances, and an abnormal coagulation profile. The respective detection result values were as follows: Total bilirubin (TBIL) 45.6 μmol/L (3.4-20.5), direct bilirubin 20.8 μmol/L (0.0-6.8), alanine aminotransferase 57.2 U/L (0-40), aspartate transaminase 48.4 U/L (0-40), gamma glutamyl transpeptidase 247 U/L (8.0-58.0), creatinine 114.7 μmol/L (58.3-106.0), sodion (Na^+) 132.8 mmol/L (136.0-146.0), chloridion (Cl^-) 93.3 mmol/L (101.0-109.0), phosphorus 0.59 mmol/L (0.81-1.45), fibrinogen 5 g/L (2.0-4.0), antithrombin III 80.9% (80.0-120.0), D-dimer 3.67 μg/mL (0-0.55), and fibrinogen degradation products 12.3 μg/mL (0-5.0). These abnormal results indicate that multiple organ functions have been injured. Infectious disease screening yielded negative results for respiratory pathogen immunoglobulin M, hepatitis virus, anti-human immunodeficiency virus, anti-tuberculosis antibodies, and haemorrhagic fever virus antibodies. Blood cultures detected *Klebsiella pneumoniae* (*K. pneumoniae*).

Imaging examinations

Chest and abdominal computed tomography (plain scan + enhancement) revealed two pneumonia cases, bilateral pleural effusions, small intestinal fluid accumulation in the lower abdomen with slight dilation of the intestinal tract, and a possible abscess in the right lobe of the liver (Figure 1A-C).

Upper abdominal magnetic resonance cholangiopancreatography revealed a right lobe liver abscess, adjacent suppurative cholangitis, and bilateral pleural effusions accompanied by incomplete compression and expansion of adjacent lung tissue (Figure 1D).

MULTIDISCIPLINARY EXPERT CONSULTATION

Experts participating in the multidisciplinary consultation came from the following departments: Imaging Department, Respiratory Department, infectious diseases Department, Interventional Department, Laboratory Department and General Surgery Department.

FINAL DIAGNOSIS

Based on these findings, the patient was diagnosed with hvKp caused ILAS secondary to diabetes mellitus and leukopenia through multidisciplinary expert consultation.

TREATMENT

After the patient was admitted, he received the immediate empirical antibiotic piperacillin-tazobactam (4.5 g every 8 hours), intensive blood glucose control, and supportive treatment. On day 3 of admission, his condition worsened, and the WBC and platelet counts further decreased to 2.50 × 10^9/L and 65 × 10^9/L, respectively. After pathogen culture combined with drug sensitivity testing, the antibiotics were switched to meropenem (1 g every 8 hours) to which the *K. pneumoniae* was sensitive, which resulted in normalisation of the WBC and platelet counts by day 5 and day 8, respectively. The fever also resolved by day 13. After strict dietary and blood glucose management, his fasting blood glucose and HbA1c levels were 6.2 mmol/L and 6.1%, respectively by day 13. The follow-up blood culture performed on day 17 yielded negative results.

OUTCOME AND FOLLOW-UP

After 2 weeks of comprehensive treatment, the patient did not experience any further fever, his symptoms improved, and all examination findings had improved. At the 56-day follow-up, the patient did not experience any discomfort, and

Table 1 Blood routine and inflammatory markers

Date	WBC (4 × 10^9-10 × 10^9 /L)	N% (50%-70%)	PLT (100 × 10^9-300 × 10^9 /L)	HrCRP (0-10 mg/L)	IL-6 (< 7 pg/mL)	PCT-GN (< 0.5 ng/mL)
March, 04 2024	3.76	87.8	80	145.76	331.51	15.806
March, 06 2024	2.5	74	70	61.77	24.47	5.402
March, 09 2024	7.25	81.2	71	27.98	16.08	3.563
March, 12 2024	6.76	74.1	302	11.81	7.07	0.484
March, 15 2024	5.55	80.5	219	50.36	36.22	1.176
March, 18 2024	5.86	75	259	31.52	8.01	0.85
April, 29 2024	4.21	32.2	194	0.68	< 1	< 0.02

WBC: White blood cell; N: Neutrophil; PLT: Platelet; HrCRP: Hypersensitivity C-reactive protein; IL-6: Interleukin-6; PCT-GN: Procalcitonin.

physical examination revealed no obvious abnormalities. All laboratory tests were normal, including fasting blood glucose (5.6 mmol/L) and HbA1c (5.9%). Imaging results revealed complete resolution of thrombophlebitis and granulomatous changes following the liver abscess (Figure 1E and F). Thus, we concluded that we had successfully treated this patient, and he had a good prognosis.

DISCUSSION

Poor glycaemic control damages neutrophil phagocytic function and promotes the growth of pathogens in tissues, ultimately leading to systemic inflammatory response syndrome and multiorgan injury following hvKp infection. Laboratory test and imaging examination results of this patient indicated bacteraemia, conjunctivitis, pneumonia with pleural effusions, small intestinal fluid accumulation with dilatation, and multiorgan dysfunction. Uncommon findings included hepatic thrombophlebitis in the right liver lobe and leukopenia, which confirmed the diagnosis of severe ILAS. The above clinical manifestations and laboratory tests indicate that *K. pneumoniae* is a highly invasive bacterium that may lead to systemic dissemination and severe inflammation.

Thrombophlebitis and liver abscesses are distinctive imaging features of ILAS. Potential mechanisms include hvKp endotoxin-induced hypercoagulability and blood flow stasis from venous drainage of the abscess, direct inflammation-induced thrombosis, and coagulopathy with an elevated D-dimer level[7,8]. Thrombophlebitis is a marker of haematogenous dissemination that involves multiple organs and increases clinical complexity and mortality risk.

The haematological findings of ILAS include leucocytosis and thrombocytopenia[9]. Thrombocytopenia is associated with infection and exacerbated severity[8] and is an independent risk factor for invasive syndromes in patients with thrombophlebitis, *K. pneumoniae* infection, and liver abscesses[8-10]. Platelets are considered an extension of the cellular immune system. In an inflammatory environment, platelets are activated and release multiple different factors, including many pro-inflammatory factors. The activation of this inflammatory function leads to thrombosis, which may worsen inflammatory diseases[11]. Patients with ILAS and thrombocytopenia have impaired myeloid platelet production. Additionally, platelet activation-related thrombosis promotes platelet consumption[9].

Our patient experienced 4 days of leukopenia, which has not been previously reported in the literature. The following possible mechanisms were suggested for the patient's prolonged leukopenia: Severe gram-negative *K. pneumoniae* infection causing leukocyte consumption; inflammatory and pro-inflammatory factors leading to immune-mediated leukocyte destruction; Bacterial toxins and metabolic by-products that impair bone marrow granulocyte production; And compromised immune function in patients with diabetes[12]. Leukopenia and thrombocytopenia in ILAS are indicators of severe infection and poor prognosis. There is currently no report on diabetes accompanied by leukopenia. We consider that it may be related to the low immune mechanisms of diabetes, increased consumption after infection, and suppressed hematopoietic function of the bone marrow.

Compared with the general population, individuals with diabetes are more prone to infection, the infection process is more complex, and the infection is more likely to become severe[13]. The main mechanisms causing this result include: (1) High blood glucose causing inhibition of immune cell activity [such as cluster of differentiation (CD) 4, CD8, and natural killer cells] and cytokine production, leading to immune dysfunction; (2) High blood glucose causing inhibition of the adhesion, chemotaxis, and phagocytic function of neutrophils and WBCs, and damage to the antioxidant system involved in bactericidal activity ultimately, leading to an infection that is difficult to control; and (3) Elevated blood glucose leads to peripheral vascular disease, increased permeability of the vascular wall, and decreased blood oxygen

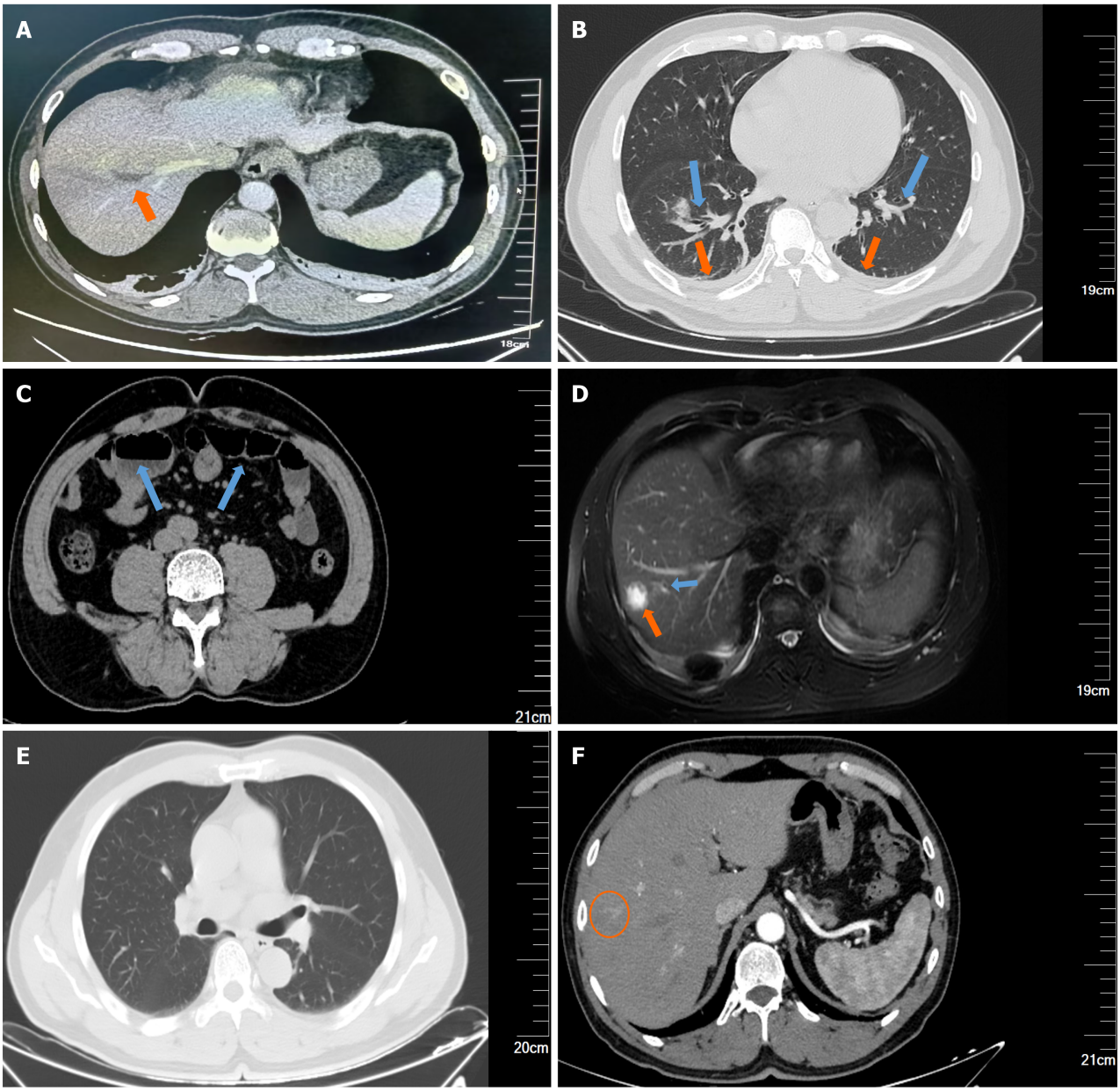

Figure 1 Imaging examination results of the patient. A: Hepatic enhanced computed tomography (CT) showed hepatic thrombophlebitis in the right liver lobe (orange arrow: Filling defect in the vein with irregular wall thickness); B: Abdominal CT showed bilateral lung inflammation (blue arrow) and bilateral pleural effusion (orange arrow); C: Intestinal CT showed fluid accumulation in the small intestine and bowel dilatation (blue arrow). During a 56-day follow-up; D: Magnetic resonance cholangiopancreatography showed a right liver lobe abscess and purulent cholangitis (orange arrow: 20 mm × 16 mm liver abscess; blue arrow: Purulent cholangitis with thickened duct walls); E: Abdominal CT showed absorbed pneumonia and pleural effusion; F: Hepatic enhanced CT revealed low-density nodule (approximately 6 mm) in the upper segment of the right lobe (S8) indicating granulomatous changes after liver abscess treatment (orange circle) and complete resolution of the hepatic vein thrombus.

content in the surrounding tissues. These factors provide favorable conditions for the growth and proliferation of bacteria. Therefore, individuals with diabetes who are infected with *K. pneumoniae* are more prone to hematogenous dissemination, which leads to multiple organ involvement and abscess formation[14]. Because this patient was diagnosed as diabetes for the first time and his blood glucose level was not very high, he had no obvious clinical manifestations of diabetes and related complications. This may be a beneficial factor for his good prognosis.

In terms of immunity, the function of polymorphonuclear leukocytes is inhibited, and the adhesion, chemotaxis, and phagocytosis of WBCs are reduced. Additionally, with a background of diabetes and metabolic disorders, the persistent low-grade inflammation, the imbalance in gut microbiota, and the impairment of intestinal barrier function may increase the risk of bacteria entering the blood from the intestinal tract[13].

There are no specific guidelines or consensus to address ILAS treatment. Antibiotic therapy is the primary treatment for most ILAS cases[10]. After diagnosis and detection of infection metastasis, early identification of the pathogen, selection of antibiotics to which the organism is susceptible[15], and a comprehensive evaluation of extrahepatic abscesses are crucial. Untreated or delayed cases result in a poor prognosis.

Whether anticoagulation therapy is necessary for ILAS with thrombophlebitis is unclear. Molton *et al*[16] suggested that reperfusion of the affected veins is closely related to the complete resolution of abscesses, and anti-inflammatory drugs can spontaneously resolve most purulent hepatic venous thrombosis cases. Anticoagulant therapy was not administered to our patient because he had thrombosis, mild abnormalities in the coagulation function, thrombocytopenia, and bacteraemia. Thus, anticoagulant therapy would have had more disadvantages than benefits in this case. Moreover, thrombophlebitis improved gradually after active anti-inflammatory treatment alone. Because the patient was recovering well, he refused to undergo a liver abscess puncture to allow for further evaluation of the pathogenic bacteria, bacterial typing, and identification of the virulence gene.

CONCLUSION

ILAS involves an acute onset and rapid progression with a nonspecific clinical presentation and presents diagnostic and therapeutic challenges. These factors suggest a poor prognosis if treatment is not promptly initiated. Thrombocytopenia has been confirmed to be a poor prognostic predictor for ILAS, however, not only the existence of leukopenia but also the coexistence of leukopenia with thrombocytopenia in ILAS is extremely rare. In this case, the patient presented with both leukopenia and thrombocytopenia, which portend an evolution toward a more critical condition. However, we were able to promptly diagnose and successfully cure the patient in this case. Therefore, we considered leukopenia as another poor prognostic predictor apart from thrombocytopenia in ILAS. Early pathogen identification, precise anti-infective treatment, and multiorgan function protection are critical to controlling ILAS and improving clinical outcomes.

FOOTNOTES

Author contributions: Niu CY and Yao BT conceived the designing, acquiring and analyzing data and wrote the manuscript; Tao HY collected the data; Peng XG and Zhang QH analyzed the imaging and contributed to diagnosis; Chen Y and Liu L established the diagnosis and reviewed the manuscript; All authors read and approved the final manuscript; Niu CY and Yao BT contributed equally.

Supported by the 2022 Nanjing Health Science and Technology Development Special Fund Support Project, No. YKK22240.

Informed consent statement: Written informed consent was obtained from the patient.

Conflict-of-interest statement: The authors declare that they have no conflict of interest.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

Country of origin: China

ORCID number: Chun-Yan Niu 0000-0001-8226-8040; Bang-Tao Yao 0000-0003-4036-7440; Yue Chen 0009-0007-8339-2984.

S-Editor: Fan M
L-Editor: A
P-Editor: Zhao YQ

REFERENCES

1 **Chen YC**, Lin CH, Chang SN, Shi ZY. Epidemiology and clinical outcome of pyogenic liver abscess: an analysis from the National Health Insurance Research Database of Taiwan, 2000-2011. *J Microbiol Immunol Infect* 2016; **49**: 646-653 [PMID: 25442876 DOI: 10.1016/j.jmii.2014.08.028]

2 **Zhang CG**, Wang Y, Duan M, Zhang XY, Chen XY. Klebsiella pneumoniae invasion syndrome: a case of liver abscess combined with lung abscess, endophthalmitis, and brain abscess. *J Int Med Res* 2022; **50**: 3000605221084881 [PMID: 35345919 DOI: 10.1177/03000605221084881]

3 **Jin Q**, Zhang X, Yang H, Zhao B, Wang Y. Blindness in Right Eyes after Enema: A Case of Klebsiella pneumoniae-Related Invasive Liver Abscess Syndrome with Endophthalmitis-Caused Blindness as the First Symptom. *Case Rep Med* 2024; **2024**: 5573160 [PMID: 38380356 DOI: 10.1155/2024/5573160]

4 **Lee CR**, Lee JH, Park KS, Jeon JH, Kim YB, Cha CJ, Jeong BC, Lee SH. Antimicrobial Resistance of Hypervirulent Klebsiella pneumoniae: Epidemiology, Hypervirulence-Associated Determinants, and Resistance Mechanisms. *Front Cell Infect Microbiol* 2017; **7**: 483 [PMID: 29209595 DOI: 10.3389/fcimb.2017.00483]

5 **Wang H**, Xue X. Clinical manifestations, diagnosis, treatment, and outcome of pyogenic liver abscess: a retrospective study. *J Int Med Res*

2023; **51**: 3000605231180053 [PMID: 37345580 DOI: 10.1177/03000605231180053]

6 **Jun JB**. Klebsiella pneumoniae Liver Abscess. *Infect Chemother* 2018; **50**: 210-218 [PMID: 30270580 DOI: 10.3947/ic.2018.50.3.210]

7 **Iwadare T**, Kimura T, Sugiura A, Takei R, Kamakura M, Wakabayashi SI, Okumura T, Hara D, Nakamura A, Umemura T. Pyogenic liver abscess associated with Klebsiella oxytoca: Mimicking invasive liver abscess syndrome. *Heliyon* 2023; **9**: e21537 [PMID: 38027784 DOI: 10.1016/j.heliyon.2023.e21537]

8 **Feng C**, Di J, Jiang S, Li X, Hua F. Machine learning models for prediction of invasion Klebsiella pneumoniae liver abscess syndrome in diabetes mellitus: a singled centered retrospective study. *BMC Infect Dis* 2023; **23**: 284 [PMID: 37142976 DOI: 10.1186/s12879-023-08235-7]

9 **Chen L**, Wang H, Wang H, Guo Y, Chang Z. Thrombocytopenia in Klebsiella pneumoniae liver abscess: a retrospective study on its correlation with disease severity and potential causes. *Front Cell Infect Microbiol* 2024; **14**: 1351607 [PMID: 38562965 DOI: 10.3389/fcimb.2024.1351607]

10 **Chang Y**, Chen JH, Chen WL, Chung JY. Klebsiella pneumoniae invasive syndrome with liver abscess and purulent meningitis presenting as acute hemiplegia: a case report. *BMC Infect Dis* 2023; **23**: 397 [PMID: 37308846 DOI: 10.1186/s12879-023-08383-w]

11 **Repsold L**, Joubert AM. Platelet Function, Role in Thrombosis, Inflammation, and Consequences in Chronic Myeloproliferative Disorders. *Cells* 2021; **10** [PMID: 34831257 DOI: 10.3390/cells10113034]

12 **Akash MSH**, Rehman K, Fiayyaz F, Sabir S, Khurshid M. Diabetes-associated infections: development of antimicrobial resistance and possible treatment strategies. *Arch Microbiol* 2020; **202**: 953-965 [PMID: 32016521 DOI: 10.1007/s00203-020-01818-x]

13 **Holt RIG**, Cockram CS, Ma RCW, Luk AOY. Diabetes and infection: review of the epidemiology, mechanisms and principles of treatment. *Diabetologia* 2024; **67**: 1168-1180 [PMID: 38374451 DOI: 10.1007/s00125-024-06102-x]

14 **Luo Y**, Hu W, Wu L, Duan S, Zhong X. Klebsiella pneumoniae invasive syndrome with liver, lung, and brain abscesses complicated with pulmonary fungal infection: a case report and review of the literature. *Int J Emerg Med* 2023; **16**: 92 [PMID: 38129781 DOI: 10.1186/s12245-023-00574-1]

15 **Serban D**, Popa Cherecheanu A, Dascalu AM, Socea B, Vancea G, Stana D, Smarandache GC, Sabau AD, Costea DO. Hypervirulent Klebsiella pneumoniae Endogenous Endophthalmitis-A Global Emerging Disease. *Life (Basel)* 2021; **11** [PMID: 34357049 DOI: 10.3390/life11070676]

16 **Molton JS**, Chee YL, Hennedige TP, Venkatesh SK, Archuleta S. Impact of Regional Vein Thrombosis in Patients with Klebsiella pneumoniae Liver Abscess. *PLoS One* 2015; **10**: e0140129 [PMID: 26444702 DOI: 10.1371/journal.pone.0140129]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: office@baishideng.com
Help Desk: https://www.f6publishing.com/helpdesk
https://www.wjgnet.com