



Concurrent *Klebsiella pneumoniae* liver abscess and infective endocarditis: A rare case report and literature review

Yuanwen Chen^a, Yisheng Zhou^b, Zhibin Xu^{c,*}

^a Department of Burn and Plastic Surgery, The People's Hospital of Baoan Shenzhen, Shenzhen, Guangdong 518101, China

^b Department of Cardiology, Guangzhou Development District Hospital (Guangzhou Huangpu District People's Hospital), Guangdong 510730, China

^c Department of Organ Transplantation, The First Affiliated Hospital of Guangzhou Medical University, Guangzhou, Guangdong 510120, China

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ABSTRACT

Background: Investigating the clinical characteristics and treatment strategies of pyogenic liver abscess (PLA) complicated by infective endocarditis (IE), this study draws on a successfully treated case of PLA caused by *Klebsiella pneumoniae*, alongside a literature review of similar cases.

Case Summary: We report a 50-year-old male with type 2 diabetes who presented with acute fever, chills, and a liver abscess. The patient was initially treated with intravenous ceftriaxone (2 g daily). Due to the onset of septic shock, the antibiotic regimen was escalated to piperacillin-tazobactam (4.5 g every 8 h) and levofloxacin (0.5 g daily). Ultrasound-guided percutaneous drainage of the liver abscess was performed, and blood cultures confirmed *Klebsiella pneumoniae*. Upon the development of infective endocarditis, the treatment was adjusted to a combination of ceftriaxone and amikacin for one week, followed by six weeks of ceftriaxone monotherapy, resulting in full recovery.

Conclusion: This case report illustrates the rare association of *Klebsiella pneumoniae*-induced PLA with IE in a diabetic patient. It emphasizes the importance of individualized treatment strategies, with insights drawn from this case contributing to the understanding of managing such complex infections. While the successful outcome of this case provides valuable clinical insights, it highlights the need for careful consideration in treatment approaches. The findings from this single case should guide clinicians in similar scenarios but should not be generalized without further evidence.

Introduction

Liver abscesses are localized purulent infections in the liver, typically caused by pathogens entering via the biliary tract, hepatic artery, portal vein, or through direct spread[1]. Pyogenic liver abscesses (PLAs), which are predominantly bacterial, account for approximately 80 % of all liver abscess cases. While *Escherichia coli* and streptococci are common causative agents in Europe and North America, *Klebsiella pneumoniae* is the leading cause in China and the broader Asia-Pacific region[2], particularly due to its gastrointestinal colonization, especially the K1/K2 serotypes[3,4].

Klebsiella pneumoniae-induced PLAs can lead to severe complications such as endophthalmitis, metastatic infections, and necrotizing fasciitis [5,6]. However, the occurrence of infective endocarditis (IE) in

conjunction with PLA is extremely rare. This report presents a unique case of *Klebsiella pneumoniae*-induced PLA complicated by IE. The written consent was obtained from the local Ethics Committee of The First Affiliated Hospital of Guangzhou Medical University. The verbal consent was obtained from the patient. Through an exhaustive literature review and analysis of this case, we aim to delineate the distinctive clinical characteristics of PLA when complicated by IE, thus enhancing the understanding of its diagnosis and management.

Case presentation

Patient information

A 50-year-old man with a body weight of 70 kg was admitted to the

Abbreviations: PLA, pyogenic liver abscess; IE, infective endocarditis; CT, computed tomography; CRP, C-reactive protein; COPD, chronic obstructive pulmonary disease; CRC, colorectal cancer; ESBL, Extended-spectrum β -lactamase; ESC, European Society of Cardiology; AHA, American Heart Association.

* Correspondence to: No.28 Qiaozhong Zhong Road, Liwan District, Guangzhou City, Guangdong Province 510120, China.

E-mail addresses: 25551786@qq.com (Y. Chen), 269946815@qq.com (Y. Zhou), unignorable_xuzhibin@outlook.com (Z. Xu).

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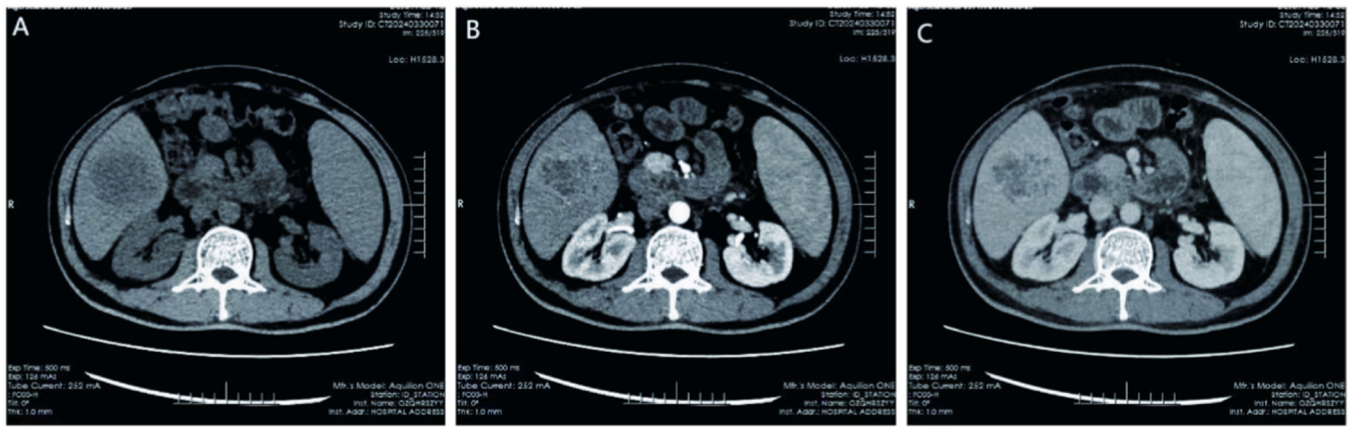


Fig. 1. CT findings indicative of a hepatic abscess. The right lobe of the liver was a circular low-density lesion with blurred edges. The peripheral enhancement and septal enhancement were observed in the arterial and venous phases of enhanced scanning, showing honeycomb changes.

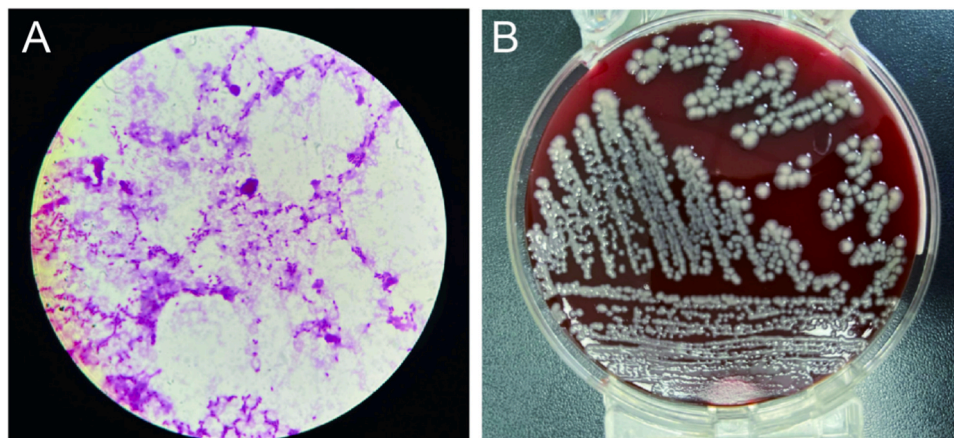


Fig. 2. *Klebsiella pneumoniae* was identified by laboratory. (a) Microscopic identification of *Klebsiella pneumoniae*, Gram staining, x1000. (b) *Klebsiella pneumoniae* colony, blood plate culture, 35 °C, 2 days.

hospital on March 10, 2024, following an episode of acute fever reaching up to 39.0 °C, associated with chills and rigors that began unexpectedly earlier that day. The patient reported no cough, sputum production, shortness of breath, nausea, vomiting, or diarrhea prior to onset. Initial evaluation at an external hospital, prompted by upper abdominal discomfort, revealed a liver abscess through computed tomography (CT) (Fig. 1). He was referred to our facility for further diagnostic and therapeutic management.

Upon admission, the patient was alert with mild jaundice and slight scleral icterus. Vital signs included a temperature of 36.1 °C, heart rate of 87 beats/min, respiratory rate of 21 breaths/min, and blood pressure of 127/72 mmHg. Lung auscultation was clear, and cardiac examination was normal. Laboratory tests revealed a leukocyte count of $3.79 \times 10^9/L$, with 94.4 % neutrophils, significantly elevated inflammatory markers (CRP 248.93 mg/L, procalcitonin 93.99 µg/L), and elevated liver enzymes. A chest CT scan showed a bulla in the right upper lung and minor inflammatory changes in the left lung, along with a hepatic lesion suggestive of an abscess.

Therapeutic intervention and timeline

After admission, the patient was started on empirical intravenous ceftriaxone (2 g daily) due to high fever (39.8 °C), chills, nausea, and poor appetite. The next day, hypotension was noted with a blood pressure of 71/51 mmHg, prompting escalation to piperacillin-tazobactam (4.5 g every 8 h) due to signs of septic shock. Ultrasound indicated a

potential liver abscess, and supportive care including insulin, dopamine, and gastric acid suppression was provided.

By March 12, the patient remained febrile with a peak temperature of 38.6 °C. Blood cultures returned positive for *Klebsiella pneumoniae* (Fig. 2). The susceptibility test showed intermediate sensitivity to ciprofloxacin and susceptibility to several other antibiotics. An ultrasound-guided percutaneous liver abscess drainage was performed, extracting brown purulent bile sent for further culturing.

On March 13, critical lab results indicated a platelet count of $24 \times 10^9/L$, a white blood cell count of $14.62 \times 10^9/L$ with 89.9 % neutrophils, and a CRP level of 190.31 mg/L. Imaging studies via head, chest, and abdominal CT scans revealed no significant brain abnormalities, bilateral pulmonary inflammation, pleural effusions with partial lung collapse, and post-drainage changes in the liver, demonstrating the infection is not well controlled and the possibility of the pulmonary infection. Given the increased susceptibility of diabetic patients to infections, including those caused by extended-spectrum beta-lactamase (ESBL)-producing organisms[7], levofloxacin at 0.5 g daily was added to the regimen in combination with piperacillin-tazobactam. This decision was guided by the recommendations outlined in the “2023 Update on Sepsis and Septic Shock in Adult Patients”[8] which supports the use of combination therapy in cases with a high risk of multi-drug resistant infections. Supportive care, including fluid resuscitation and vasopressors, continued to manage the septic shock.

By March 15, the patient was alert and afebrile. The liver abscess drainage tube remained in place with minimal output. Blood tests

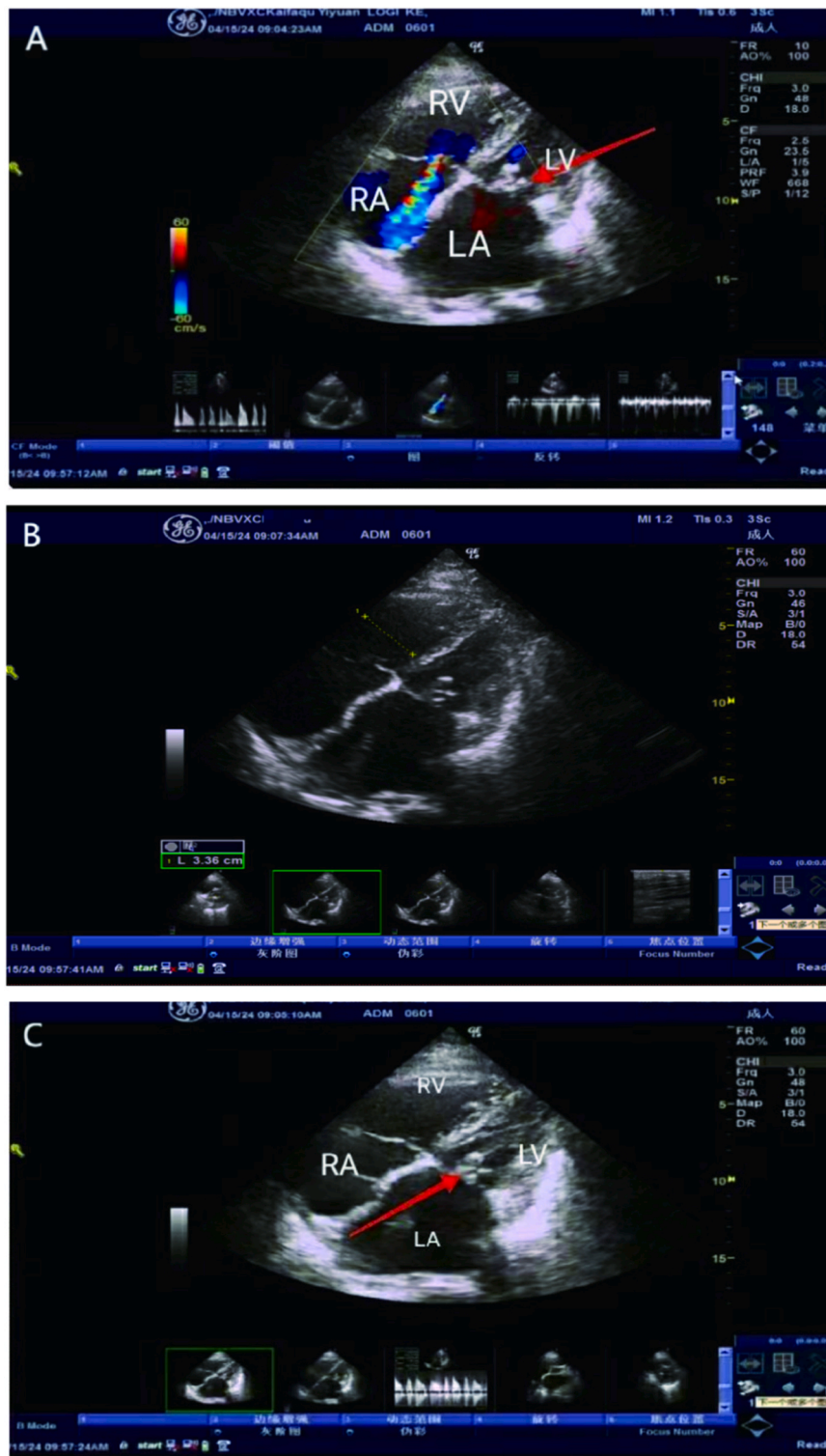


Fig. 3. Ultrasound imaging evidence supports infective endocarditis. (a) Color Doppler imaging shows: Mitral valve coaptation is adequate with no significant valve prolapse or regurgitation observed. (b) The anterior mitral valve tip was thickened, roughened and accompanied by growth, suggesting infective endocarditis. (c) Apical four-chamber view: Attachments of vegetation on the tips of the anterior and posterior mitral valve leaflets. The vegetation measured approximately 5–10 mm in width and 15–25 mm in length.

showed improvement: WBC at $7.69 \times 10^9/L$, neutrophils at 79.0 %, platelets at $52 \times 10^9/L$, CRP at 46.06 mg/L, and procalcitonin at 27.52 $\mu\text{g/L}$. A TTE was obtained due to persistent bacteremia, and this showed a thickened roughened tip of the anterior mitral valve with signs of growth, consistent with infective endocarditis. (Fig. 3). These findings confirmed the involvement of the cardiac endocardium, aligning with

the DUKE-ISCVID criteria[9] for infective endocarditis diagnosis. The antimicrobial regimen was adjusted to include ceftriaxone 2 g daily and amikacin 1 g daily.

By March 17, the patient reported improvement in general well-being with no fever, less fatigue, and better appetite. Blood cultures from March 16 onwards were negative. Laboratory tests showed WBC

Table 1
Clinical Characteristics of 16 Cases with Pyogenic Liver Abscess and Infective Endocarditis.

Case	Gender/ Age	Comorbidity	Symptom	Valve involved	Drainage
1	Male/82	T2DM	Fever, chills	Mitral valve	Yes
2	Male/40	T2DM	Fever, fatigue, cough	NA*	Yes
3	Male/39	None	Fever, chills, cough	Mitral valve	Yes
4	Male/74	Chronic lymphocytic leukemia, psoriatic arthritis	Fever, chills, confusion	Mitral valve	Yes
5	Male/49	None	Fever, cough	Aortic valve	Yes
6	Male/68	Hypertension, COPD and splenic artery aneurysm	Fever, confusion	Aortic valve	Yes
7	Female/ 51	None	Fever, chills	Mitral valve	No
8	Male/25	None	Fever, hypotension	Aortic valve	Yes
9	Male/48	Hypertension	Fever, chills, diarrhea, abdominal pain	Aortic valve	Yes
10	Male/63	Pancreatic cancer	Fever, chills, hypotension	Aortic valve	No
11	Female/ 52	Cirrhosis	Abdominal pain, abdominal distension, anorexia	Aortic valve	No
12	Female/ 40	None	Fever, chills, lower back pain, headache	Aortic valve	No
13	Female/ 81	Hypertension, gastric adenocarcinoma	Fever, chills, nausea	Mitral valve	Yes
14	Male/49	None	Fever, cough, abdominal pain, nausea, and vomiting	Aortic valve	Yes
15	Male/39	T2DM	Fever, chills	Mitral valve	Yes
16 *	Male/50	T2DM	Fever, chills	Mitral valve	Yes

Note: T2DM, type 2 diabetes mellitus; NA, not available; COPD, chronic obstructive pulmonary disease;

▲"NA" (Not Available) is used in cases where specific information on valve involvement was not provided in the original case reports. For these patients, no valve-related data were available, and thus valve involvement could not be determined.

* Represents this case

$8.47 \times 10^9/L$, neutrophils 76.4 %, and platelets $112 \times 10^9/L$. CRP was 21.48 mg/L, and procalcitonin was 12.91 $\mu\text{g/L}$. A follow-up chest CT on March 20 showed improvement in pulmonary inflammation and a reduction in the liver abscess size.

By March 22, with stable vital signs and further lab improvement, amikacin was discontinued, and ceftriaxone was continued. Ophthalmology ruled out endophthalmitis and diabetic retinopathy. On March 24, the patient was discharged in stable condition with instructions to continue ceftriaxone therapy at a local hospital to complete the 6-week course.

Discussion

Literature review

A systematic review was conducted using the CNKI and PubMed databases with the search terms "liver abscess" and "infective endocarditis," covering the entire period until April 2024. The objective was to identify peer-reviewed clinical reports that provided comprehensive patient data on pyogenic liver abscesses (PLA) with concurrent infective endocarditis (IE). Exclusion criteria included review articles, reports with incomplete clinical data, and publications representing duplicate data. The search was mainly focused on the etiological distribution, emphasizing cases involving *Klebsiella pneumoniae*.

The search yielded fifteen pertinent articles that described fifteen individual cases, with two reports published in Chinese detailing two cases and thirteen reports in English documenting thirteen cases. Combined with data from our institution, the dataset encompassed sixteen patients: four females and twelve males, with a mean age of 54.1 ± 15.8 years. Comorbid conditions were present in 10 patients (62.5 %), including four with T2DM, three with malignancies (specifically chronic lymphocytic leukemia, pancreatic cancer, and gastric adenocarcinoma) and three with hypertension, chronic obstructive pulmonary disease (COPD), and hepatic cirrhosis. (Table 1).

Clinically, fever was reported in fourteen of the cases, with rigors observed in nine. Secondary infections at sites other than the liver were reported in seven patients: two instances of endophthalmitis both attributed to *Klebsiella pneumoniae*, two splenic abscesses, and three cases involving vertebral, cranial, and pulmonary infections, respectively.

Various pathogens were reported from the sixteen patients with PLA and concurrent IE identified during our review process. Six cases were attributed to *Klebsiella pneumoniae*; four involved anaerobic bacteria, including three cases of *Clostridium perfringens* and one of *Fusobacterium nucleatum*; three cases were due to β -hemolytic streptococci, with two involving *Streptococcus pyogenes* and one *Streptococcus constellatus*. Three other cases involved *Eikenella corrodens*, *Chromobacterium violaceum*, and *Candida albicans*, respectively. All isolated pathogens were non-multidrug-resistant strains.

Geographically, *Klebsiella pneumoniae* cases originated from China, the Philippines, Switzerland, and India. Aortic valve infections were most common, with mitral valve involvement also observed. Treatment primarily involved β -lactam antibiotics combined with aminoglycosides, with most patients undergoing liver abscess drainage. Five patients recovered, while one succumbed to the infection.

Among non-*Klebsiella* infections, treatment varied but generally included combinations of β -lactam antibiotics with metronidazole or penicillin. Outcomes were mostly positive, except for one patient with *Streptococcus constellatus* who died from septic and cardiogenic shock. The patient with *Eikenella corrodens* infection was successfully treated with cefuroxime and ciprofloxacin. The *Chromobacterium violaceum* patient was initially treated with meropenem for six weeks, followed by six weeks of oral ciprofloxacin, and recovered. The patient with *Candida albicans* infection, treated with amphotericin B and caspofungin, did not undergo aortic valve replacement due to high surgical risk and eventually succumbed to hepatic encephalopathy. (Table 2).

Clinical management of *Klebsiella pneumoniae*-induced pyogenic liver abscess with concurrent infective endocarditis

The treatment of pyogenic hepatic abscesses typically involves antibiotics and percutaneous drainage when more significant than 5 cm, with surgical treatment rarely required[10]. Additionally, it is essential to differentiate between pyogenic and amoebic liver abscesses, as they have different clinical and pathological characteristics[11]. In addition, liver abscesses can be caused by rare pathogens such as *Gemella morbillorum*, which typically result in bacteremia or localized infections in

Table 2
Treatment and Prognosis of 16 Cases with Pyogenic Liver Abscess and Infective Endocarditis.

Case	Valve replacement	Concurrent infection	Pathogen	Multidrug resistant	Therapy	Outcome
1	No	Vertebrae	<i>Klebsiella pneumoniae</i>	No	IMP+AMK	Cured
2	No	Eye, pleura	<i>Klebsiella pneumoniae</i>	No	IMP+AMK+LVK	Cured
3	Yes	Eye	<i>Klebsiella pneumoniae</i>	No	TZP+GM	Cured
4	No	None	<i>Klebsiella pneumoniae</i>	No	CRO+GM	Cured
5	No	None	<i>Eikenella corrodens</i>	No	CIP+CXM	Cured
6	No	None	<i>S.anginosus</i>	No	CRO+MTZ	Cured
7	No	None	<i>F.necrophorum</i>	No	CRO+MTZ	Cured
8	Yes	Spleen	<i>F.necrophorum</i>	No	PG	Cured
9	No	Brain	<i>F.nucleatum</i>	No	PG+MTZ	Cured
10	No	None	<i>S.anginosus</i>	No	SAM+GM	Cured
11	No	Spleen	<i>Candida albicans</i>	No	AmB, CAS	Died
12	No	None	<i>Chromobacterium violaceum</i>	No	MEM, CIP	Cured
13	No	None	<i>S.constellatus</i>	No	CTX, AMP	Died
14	No	Lung	<i>F.necrophorum</i>	No	TZP, SAM	Cured
15	No	None	<i>Klebsiella pneumoniae</i>	No	CRO+GM	Died
16 *	No	None	<i>Klebsiella pneumoniae</i>	No	CRO+AMK	Cured

Note: IMP, imipenem; AMK, amikacin; LVX, levofloxacin; TZP, piperacilin-tazobactam; GM, gentamicin; CRO, ceftriaxone; CIP, ciprofloxacin; CXM, cefuroxime, MTZ, metronidazole; PG, penicillin; SAM, ampicillin-sulbactam; AmB, Amphotericin B; CAS, caspofungin; MEM, meropenem; CTX, cefotaxime; AMP, ampicillin. Multidrug resistant was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories.

* Represents this case

humans[12]. The spread of infection leading to PLA can occur through portal circulation, biliary infections, or arterial hematogenous seeding in the setting of systemic infections[13]. PLA risk factors include hepatobiliary infection, pancreatic disease, diabetes, and prior liver transplant[14].

Diagnostics rely heavily on imaging techniques such as ultrasound and CT scans, complemented by aspirational biopsy for pathogen confirmation[15]. The management of such cases involves percutaneous or surgical drainage to control the source of infection and the use of targeted antimicrobial therapy. It is recommended that antimicrobial treatment be initiated after blood cultures are obtained but before drainage procedures are performed to reduce the risk of systemic infection spread[16].

Extended-spectrum β -lactamase (ESBL)-producing *Klebsiella pneumoniae* strains are rarely detected in PLA, positioning cephalosporins as the treatment of choice. Third-generation cephalosporins, in particular, are more effective than first-generation options[17]. Sensitivity to cefotaxime in *Klebsiella pneumoniae* isolates from patients without underlying biliary pathology is exceptionally high at 99.2 % (361/364), with a notable susceptibility of 91.7 % (11/12) in those with biliary conditions[18].

A systematic review focusing on *Klebsiella*-induced infective endocarditis (IE) included 67 patients, 45 of whom had infections caused by *Klebsiella pneumoniae*. The aortic valve was the most common site of infection, followed by the mitral valve[19]. Cephalosporins and aminoglycosides were the most commonly used antimicrobials. The overall mortality rate was 19.4 %, with aortic valve infections significantly correlating with an increased risk of death. Current guidelines from the European Society of Cardiology (ESC) and the American Heart Association (AHA) recommend early surgical intervention and prolonged antimicrobial treatment of 6 weeks for non-HACEK Gram-negative bacillus-induced IE, typically involving a combination of β -lactam and aminoglycoside antibiotics[20]. However, there is a shortage of prospective, large-scale research to determine the best antimicrobial treatment strategy.

In the current analysis, six cases of mitral valve IE secondary to *Klebsiella pneumoniae*-induced PLA were identified. Only one patient underwent valve replacement surgery, but all were treated with β -lactam and aminoglycoside therapy following abscess drainage, resulting in a resolution of the infection. The treatment regimen initiated with a combination of ceftriaxone and amikacin, with a subsequent switch to ceftriaxone monotherapy to complete the 6-week course. Despite all isolates showing susceptibility to third-generation cephalosporins, a review of treatment approaches identified potential overtreatment, with

two cases receiving imipenem, one of which involved a combination with amikacin and levofloxacin for triple antimicrobial therapy, and one case treated with piperacillin-tazobactam.

Diabetes is a significant risk factor for disseminating *Klebsiella pneumoniae* beyond the liver, suggesting that this pathogen may be a potential source for IE. Empirical therapy combining third-generation cephalosporins with aminoglycosides may be considered a practical initial approach for managing *Klebsiella pneumoniae*-induced PLA complicated by IE.

Conclusion

This case highlights the rare presentation of *Klebsiella pneumoniae*-induced pyogenic liver abscess complicated by infective endocarditis in a diabetic patient. The outcome, achieved through timely percutaneous drainage and an adjusted antibiotic regimen, suggests that early intervention may contribute to favorable results. While this case provides some insights into the potential treatment strategies for such complex infections, further studies and larger case series are needed to draw more definitive conclusions. Clinicians should remain vigilant in managing similar cases, with a focus on individualized care based on the patient's unique clinical presentation.

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CRedit authorship contribution statement

Zhibin Xu: Writing – original draft, Investigation. **Yisheng Zhou:** Formal analysis, Data curation. **Yuanwen Chen:** Writing – review & editing, Supervision.

Declaration of competing Interest

The authors declare that they have no known competing interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data Availability

All materials, data, and protocols are present in the manuscript or are available upon request.

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