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## Contents

Monthly Volume 15 Number 12 December 27, 2023

## REVIEW

- 2674 Antimicrobial approach of abdominal post-surgical infections  
*Fiore M, Corrente A, Di Franco S, Alfieri A, Pace MC, Martora F, Petrou S, Mauriello C, Leone S*

## MINIREVIEWS

- 2693 Indocyanine green fluorescence in gastrointestinal surgery: Appraisal of current evidence  
*Kalayarasan R, Chandrasekar M, Sai Krishna P, Shanmugam D*
- 2709 Post-cholecystectomy iatrogenic bile duct injuries: Emerging role for endoscopic management  
*Emara MH, Ahmed MH, Radwan MI, Emara EH, Basheer M, Ali A, Elfert AA*

## ORIGINAL ARTICLE

## Case Control Study

- 2719 Multidisciplinary diagnosis and treatment nutritional support intervention for gastrointestinal tumor radiotherapy: Impact on nutrition and quality of life  
*Hui L, Zhang YY, Hu XD*

## Retrospective Cohort Study

- 2727 Association between the early high level of serum tacrolimus and recurrence of hepatocellular carcinoma in ABO-incompatible liver transplantation  
*Han JW, Choi JY, Jung ES, Kim JH, Cho HS, Yoo JS, Sung PS, Jang JW, Yoon SK, Choi HJ, You YK*
- 2739 Determining the need for a thoracoscopic approach to treat a giant hiatal hernia when abdominal access is poor  
*Pérez Lara FJ, Zubizarreta Jimenez R, Prieto-Puga Arjona T, Gutierrez Delgado P, Hernández Carmona JM, Hernández Gonzalez JM, Pitarch Martinez M*
- 2747 Predictive value of Hajibandeh index in determining peritoneal contamination in acute abdomen: A cohort study and meta-analysis  
*Hajibandeh S, Hajibandeh S, Evans L, Miller B, Waterman J, Ahmad SJ, Hale J, Higgi A, Johnson B, Pearce D, Helmy AH, Naguib N, Maw A*

## Retrospective Study

- 2757 Efficacy of pantoprazole plus perforation repair for peptic ulcer and its effect on the stress response  
*Leng ZY, Wang JH, Gao L, Shi K, Hua HB*
- 2765 Application of electroacupuncture in the prevention of low anterior resection syndrome after rectal cancer surgery  
*Xu LL, Xiang NJ, Cheng TC, Li YX, Chen P, Jiang ZW, Liu XX*

- 2774** Effects of remifentanyl combined with propofol on hemodynamics and oxidative stress in patients undergoing resection of rectal carcinoma  
*Huang J, Tian WJ*
- 2783** Percutaneous microwave ablation and transcatheter arterial chemoembolization for serum tumor markers and prognostics of middle-late primary hepatic carcinoma  
*Lin ZP, Huang DB, Zou XG, Chen Y, Li XQ, Zhang J*
- 2792** Novel invagination procedure for pancreaticojejunostomy using double purse string sutures: A technical note  
*Li J, Niu HY, Meng XK*
- 2799** Laparoscopic resection and endoscopic submucosal dissection for treating gastric ectopic pancreas  
*Zheng HD, Huang QY, Hu YH, Ye K, Xu JH*
- 2809** Prediction of the lymphatic, microvascular, and perineural invasion of pancreatic neuroendocrine tumors using preoperative magnetic resonance imaging  
*Liu YL, Zhu HB, Chen ML, Sun W, Li XT, Sun YS*
- 2820** Impact of hepatectomy and postoperative adjuvant transarterial chemoembolization on serum tumor markers and prognosis in intermediate-stage hepatocellular carcinoma  
*Hu YD, Zhang H, Tan W, Li ZK*

**Observational Study**

- 2831** Analysis of nutritional risk, skeletal muscle depletion, and lipid metabolism phenotype in acute radiation enteritis  
*Ma CY, Zhao J, Qian KY, Xu Z, Xu XT, Zhou JY*

**Randomized Controlled Trial**

- 2844** Holistic conditions after colon cancer: A randomized controlled trial of systematic holistic care *vs* primary care  
*Wang J, Qiao JH*

**Basic Study**

- 2855** Mutational separation and clinical outcomes of *TP53* and *CDH1* in gastric cancer  
*Liu HL, Peng H, Huang CH, Zhou HY, Ge J*
- 2866** Hepatic vagotomy blunts liver regeneration after hepatectomy by downregulating the expression of interleukin-22  
*Zhou H, Xu JL, Huang SX, He Y, He XW, Lu S, Yao B*

**META-ANALYSIS**

- 2879** Recent evidence for subcutaneous drains to prevent surgical site infections after abdominal surgery: A systematic review and meta-analysis  
*Ishinuki T, Shinkawa H, Kouzu K, Shinji S, Goda E, Ohyanagi T, Kobayashi M, Kobayashi M, Suzuki K, Kitagawa Y, Yamashita C, Mohri Y, Shimizu J, Uchino M, Haji S, Yoshida M, Ohge H, Mayumi T, Mizuguchi T*

- 2890** Prognostic role of serum carcinoembryonic antigen in patients receiving liver resection for colorectal cancer liver metastasis: A meta-analysis

*Tang F, Huang CW, Tang ZH, Lu SL, Bai T, Huang Q, Li XZ, Zhang B, Wu FX*

- 2907** Significance of carcinoembryonic antigen detection in the early diagnosis of colorectal cancer: A systematic review and meta-analysis

*Wang R, Wang Q, Li P*

### CASE REPORT

- 2919** Primary repair of esophageal atresia gross type C *via* thoracoscopic magnetic compression anastomosis: A case report

*Zhang HK, Li XQ, Song HX, Liu SQ, Wang FH, Wen J, Xiao M, Yang AP, Duan XF, Gao ZZ, Hu KL, Zhang W, Lv Y, Zhou XH, Cao ZJ*

- 2926** Portal vein embolization for closure of marked arteriportal shunt of hepatocellular carcinoma to enable radioembolization: A case report

*Wang XD, Ge NJ, Yang YF*

- 2932** Removal of a large rectal polyp with endoscopic submucosal dissection-trans-anal rectoscopic assisted minimally invasive surgery hybrid technique: A case report

*Polese L*

- 2938** Successful treatment of invasive liver abscess syndrome caused by *Klebsiella variicola* with intracranial infection and septic shock: A case report

*Zhang PJ, Lu ZH, Cao LJ, Chen H, Sun Y*

- 2945** Duodenojejunostomy treatment of groove pancreatitis-induced stenosis and obstruction of the horizontal duodenum: A case report

*Zhang Y, Cheng HH, Fan WJ*

- 2954** Awake robotic liver surgery: A case report

*Delvecchio A, Pavone G, Conticchio M, Piacente C, Varvara M, Ferraro V, Stasi M, Casella A, Filippo R, Tedeschi M, Pullano C, Inchingolo R, Delmonte V, Memeo R*

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## Successful treatment of invasive liver abscess syndrome caused by *Klebsiella variicola* with intracranial infection and septic shock: A case report

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### Abstract

#### BACKGROUND

*Klebsiella variicola* (*K. variicola*) is a member of the *Klebsiella* genus and is often misidentified as *Klebsiella pneumoniae*. In this report, we present a rare case of invasive liver abscess caused by *K. variicola*.

#### CASE SUMMARY

We report a rare case of liver abscess due to *K. variicola*. A 57-year-old female patient presented with back pain for a month. She developed a high-grade fever associated with chills, and went into a coma and developed shock. The clinical examinations and tests after admission confirmed a diagnosis of primary liver abscess caused by *K. variicola* complicated by intracranial infection and septic shock. The patient successfully recovered following early percutaneous drainage of the abscess, prompt appropriate antibiotic administration, and timely open surgical drainage.

#### CONCLUSION

This is a case of successful treatment of invasive liver abscess syndrome caused by *K. variicola*, which has rarely been reported. The findings of this report point to the need for further study of this disease.

**Key Words:** *Klebsiella variicola*; Invasive liver abscess syndrome; Intracranial infection; Case report

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**Core Tip:** We report a rare case of liver abscess caused by *Klebsiella variicola* (*K. variicola*) complicated by intracranial infection and septic shock. Invasive liver abscess syndrome was mainly caused by *Klebsiella pneumoniae* in previous reports. The patient successfully recovered following early percutaneous drainage of the abscess, prompt appropriate antibiotic administration, and timely open surgical drainage. Regarding the information in the case, we consider that more attention should be given to *K. variicola* in clinical practice.

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## INTRODUCTION

*Klebsiella variicola* (*K. variicola*) was initially believed to be a plant-associated distant lineage of *Klebsiella pneumoniae* (*K. pneumoniae*)[1]. Currently, *K. variicola* is gaining recognition as a cause of several human infections. Nevertheless, its virulence profile has not been fully characterized. The clinical significance of *K. variicola* infection is obscure. *K. variicola* is very difficult to differentiate from *K. pneumoniae* by imprecise detection methods, which have underestimated its real prevalence. In fact, approximately 20% of the human isolates assumed to be *K. pneumoniae* were in fact *K. variicola* or *Klebsiella quasipneumoniae*[2]. During the last two decades, invasive liver abscess syndrome due to *K. pneumoniae* has been increasingly reported worldwide, especially in the Asia Pacific region, and it is associated with high morbidity and mortality[3]. However, invasive liver abscess syndrome caused by *K. variicola* is still rarely described. Intriguingly, several methods (such as molecular, genomic, and proteomic methods) have been developed to correctly identify this species. In this paper, we present a case of invasive liver abscess syndrome due to *K. variicola* complicated by intracranial infection and septic shock. There should be increasing awareness among clinicians about this emerging invasive syndrome due to *K. variicola*.

## CASE PRESENTATION

### Chief complaints

A 57-year-old female patient was referred to our hospital with a high-grade fever associated with chills for 3 d.

### History of present illness

The patient presented with a high fever associated with chills for 3 d and then developed shock and went into a coma.

### History of past illness

One month prior, the patient complained of right-sided back pain and was diagnosed with liver abscess based on magnetic resonance imaging (MRI) of the liver (Figure 1A). She was treated with antibiotics (third generation cephalosporins) for 2 wk in a local hospital and then was discharged home.

### Personal and family history

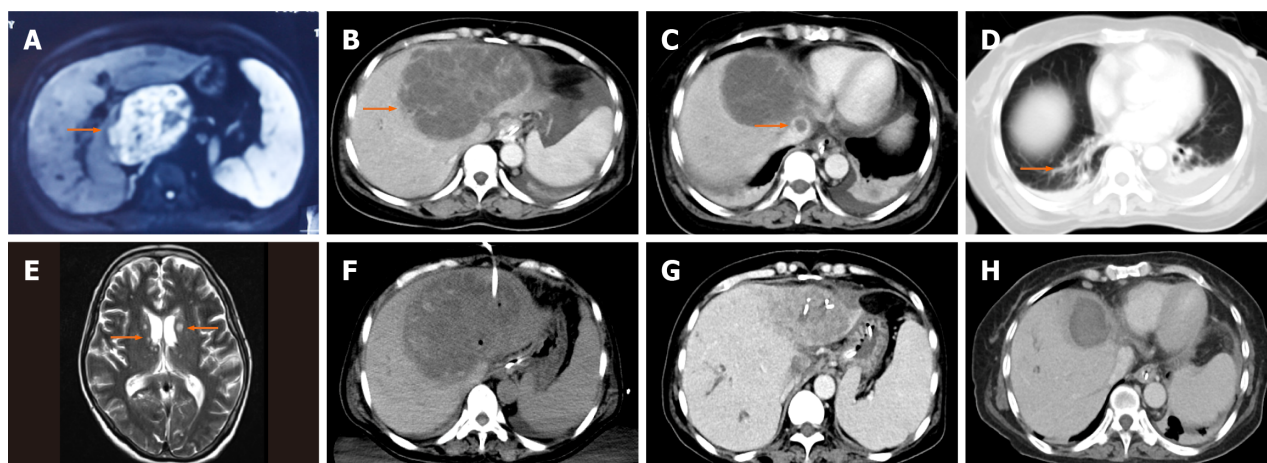
The patient had no family history of cancer. She had no history of diabetes mellitus or immunodeficiency and was neither a smoker nor a drinker.

### Physical examination

When admitted, the patient's initial vital signs were: Body temperature, 37.3 °C; heart rate, 111 beats per minute; blood pressure, 136/85 mmHg; respiratory rate, 30 breaths per minute. She had an oxygen saturation of 98% on 3 L/min oxygen. She was in a condition of somnolence. Physical examination of the heart and lung did not reveal abnormalities. Tenderness could be elicited in the right upper quadrant of the abdomen. The neck was stiff, and Kernig's sign was positive.

### Laboratory examinations

Laboratory tests showed a white blood cell count of  $11.77 \times 10^9/L$  with an elevated neutrophil percentage of 90.4%. The concentration of high-sensitivity C-reactive protein was 214.6 mg/L. The results of the liver function test were as follows: Aspartate aminotransferase at 104 IU/L and alanine aminotransferase at 111 IU/L. There were no significant abnormalities in the coagulation panel. The random blood glucose was 16.34 mmol/L, and the glycosylated hemoglobin was 6.5%.



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**Figure 1** Magnetic resonance imaging or computed tomography images of the patient. A: A large heterogeneously cystic-solid mass was present in the left liver 1 mo prior to admission; B: An area of abnormal attenuation measuring 125 mm × 97 mm in the left lobe of the liver; C: Thrombus detected in the inferior vena cava by abdominal enhanced computed tomography (CT); D: Small patchy infiltrates identified in both lungs; E: Bilateral multiple T2 hyperintensities detected in the basal ganglia and subcortical white matter in the brain magnetic resonance imaging; F: Emergency ultrasound-guided percutaneous drainage of liver abscess was performed when the patient's condition deteriorated; G: The size of liver abscess markedly decreased in the reexamination of abdominal CT; H: Inferior vena cava thrombus completely disappeared.

### Imaging examinations

An abdominal computed tomography (CT) scan showed a single abscess in the left lobe of the liver (125 mm × 97 mm) (Figure 1B) and a thrombus in the inferior vena cava (Figure 1C). A chest CT scan showed focal small patchy infiltrates in both lungs (Figure 1D).

On the 2<sup>nd</sup> d after admission, she presented with a persistent high fever and was in a coma. Considering the possibility of intracranial infection, a cranial MRI examination was performed. There were multiple abnormally high signals on T2-weighted images in the whole brain (Figure 1E).

## FINAL DIAGNOSIS

Combined with the patient's medical history, the final diagnosis was invasive liver abscess syndrome caused by a *K. variicola* infection.

## TREATMENT

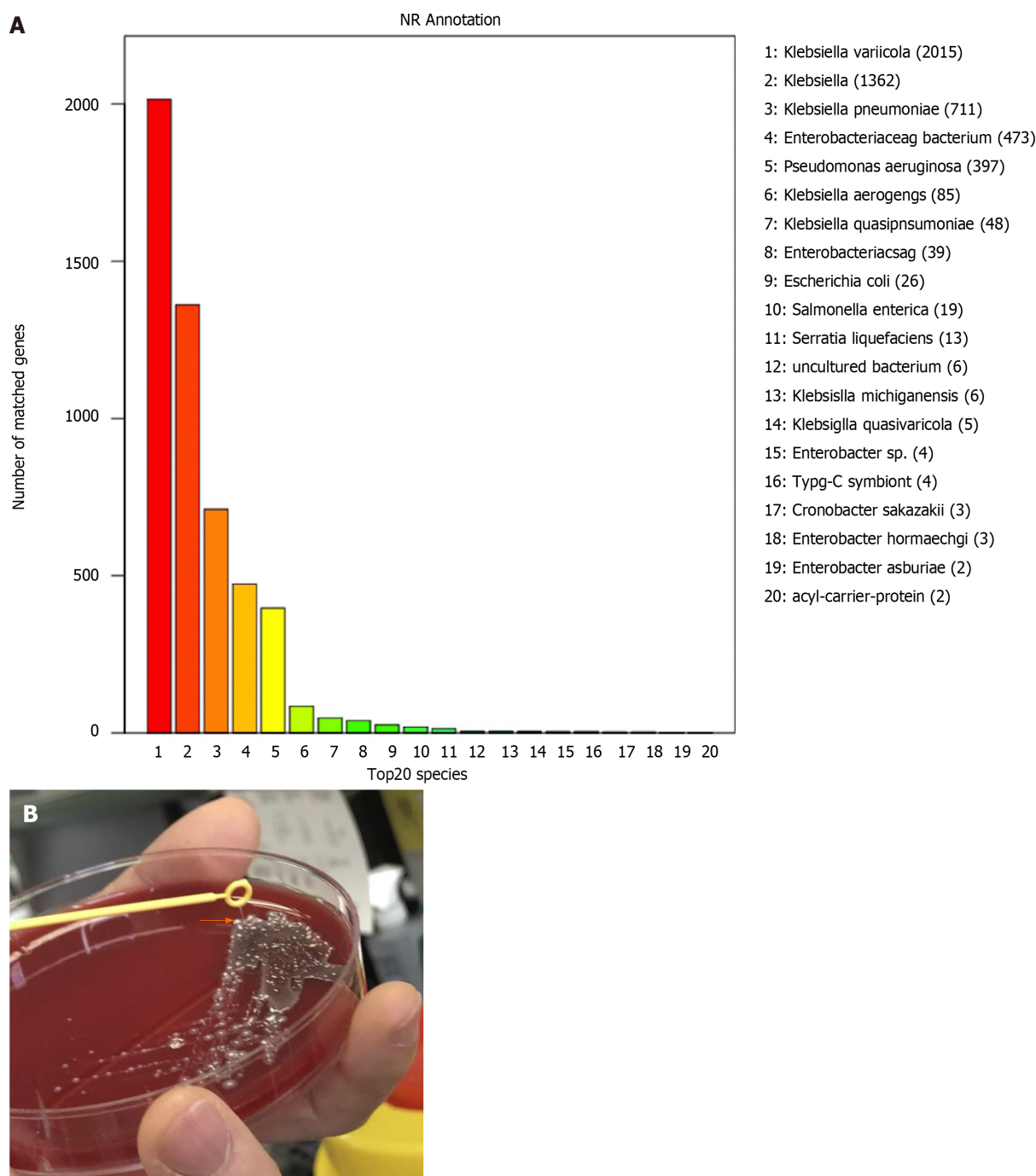
Considering the diagnosis of invasive liver abscess syndrome with septic shock and intracranial infection, empirical treatment with intravenous meropenem (2000 mg q8h) was immediately initiated. Furthermore, emergency ultrasound-guided percutaneous drainage (10F) of the liver abscess was performed (Figure 1F), which drained 900 mL of yellow pus over the first 24 h. The liver aspirate was submitted for bacterial culture.

On the 5<sup>th</sup> d after admission, *K. variicola* was isolated from two independent samples of blood and liver pus. These results were different from those of blood cultures from the local hospital. Furthermore, the isolate was confirmed by whole-genome sequencing (WGS) (Figure 2A). The strain showed a hypermucoviscous phenotype, which was investigated by the string test (Figure 2B). The isolate was resistant to ceftriaxone and cefoxitin but susceptible to amoxicillin-clavulanate, aminoglycosides, cotrimoxazole, and meropenem. Therefore, antibiotic treatment with meropenem was continued.

The drainage volume of the liver abscess gradually decreased, but the two hemogram parameters, the white blood cell count and neutrophil percentage, remained at considerably high levels. On the 10<sup>th</sup> d, surgical drainage of the liver abscess was performed. Following aspiration of approximately 800 mL of the purulent fluid, a dual drainage tube (28F) was inserted into the bottom of the abscess. The cavity of the abscess was rinsed with saline, and continuous negative pressure was used to drain fluid through the tube.

In the next few days, her critical condition improved, and she gradually recovered her consciousness. On the 23<sup>rd</sup> d, she was transferred to the general ward and continued receiving antibiotic therapy. At 18 d after the surgery, CT reexamination revealed a marked reduction in the size of the abscess (Figure 1G), and the inferior vena cava thrombus had completely disappeared (Figure 1H).





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**Figure 2 Identification of the pathogen.** A: Whole-genome sequencing results of pathogen identification; B: String test showing the hypermucoviscous phenotype (orange arrow) of the strain *Klebsiella variicola*. The strain was grown on agar and the culture was touched with a disposable loop; the formation of a mucoid string greater than 5 mm indicates the formation of the hypermucoviscous phenotype.

## OUTCOME AND FOLLOW-UP

The patient was discharged with a drainage tube (12F) due to ongoing bile leakage, while antibiotic therapy (cotrimoxazole) was continued in the outpatient clinic due to an intracranial infection. A telephone follow-up after 3 mo showed that she had recovered with no neurologic sequelae, and the antibiotic treatment was discontinued. The drainage volume gradually decreased, and the latest abdominal ultrasound showed no lesions in her liver.

## DISCUSSION

Bacterial liver abscess is a potentially life-threatening disease. It is caused by various organisms, including *Escherichia coli*,

*K. pneumoniae*, *Streptococcus anginosus*, and anaerobes such as *Bacteroides*. In recent years, the incidence rate of bacterial liver abscess has been rising, especially those caused by *K. pneumoniae*. Primary liver abscess (KLA) caused by *K. pneumoniae* emerged in East Asia but has been increasingly reported in other parts of the world[4]. Approximately 13% of patients with KLA have septic metastatic ocular or central nervous system lesions, which are associated with high morbidity and mortality[4]. *K. variicola* is a relatively newly discovered bacterium that was first described in 2004[1]. Due to its new discovery and close resemblance to *K. pneumoniae*, it is often misclassified as *K. pneumoniae*[5]. The clinical significance of *K. variicola* infection has been observed by imprecise detection methods, which underestimate its real prevalence.

*K. variicola*, as well as *K. pneumoniae*, is an opportunistic pathogen responsible for various infections, such as bloodstream infections, respiratory tract infections, and urinary tract infections[6,7]. In general, *K. variicola* isolates displayed lower antibiotic resistance rates than *K. pneumoniae*[8]. However, this fact was not associated with a better treatment response[9,10]. In fact, infections caused by *K. variicola* are more severe than those caused by *K. pneumoniae*[6].

Historically, *K. pneumoniae* was divided into three different phylogenetic groups based on sequencing of *gyrA* and *parC* genes. KpI was the largest cluster, which included *K. pneumoniae* subspecies; the KpII and KpIII clusters included *K. quasipneumoniae* and *K. variicola*, respectively[11]. Unfortunately, *K. variicola* was likely under-recognized because of its similar phylogenetic and biochemical properties to *K. pneumoniae*. Although biochemical techniques could be suggestive, these tests were not conclusive given that overlap might occur[11].

At present, the only definitive method to differentiate species is WGS or targeted sequencing[12]. Currently, matrix-assisted laser desorption/ionization-time-of-flight mass spectrometry (MALDI-TOF MS) has been widely used for the identification of bacterial species as it is a fast and economical technique. The most up-to-date versions of MALDI-TOF MS databases include reference spectra that allow to differentiate *K. variicola* from *K. pneumoniae*. In our case, *K. variicola* was identified at our hospital by using MALDI-TOF MS, Bruker library version 6.0.0.0 (Bruker Autoflex MALDI TOF-MS, Germany), but the results of the blood culture in the local hospital were *K. pneumoniae*. Considering that the results were inconsistent, the genomic DNA of the bacteria isolated from the pus culture was extracted using QIAamp DNA Micro Kit (QIAGEN, Hilden, Germany) following the manufacturer's instructions and the isolate was identified as *K. variicola* by WGS. WGS was performed on the Illumina NovaSeq PE150 (Illumina, San Diego, United States) and the draft genome sequence was annotated using the microbial genome database which contains a large collection of microbial genomes from NCBI. This Whole Genome Sequencing project has been deposited in National Genomics Data Center under the accession No. PRJCA020759 (<https://ngdc.cnbc.ac.cn>). The prevalence of *K. variicola* is unclear, mainly due to the difficulties in identifying this species or its misidentification[5]. Although the management of this case was not affected by misidentification, correct species identification carried important prognostic and epidemiologic implications. As it has been previously suggested that patients with *K. variicola* have a poor prognosis, hypermucoid strains often have the ability to spread from the site of infection to other areas, making their removal and treatment difficult[13].

Invasive liver abscess syndrome is associated with both host and virulence factors. A striking finding of this study is the hypermucoviscous phenotype of *K. variicola*. Emerging evidence has demonstrated an association between the hypermucoviscous phenotype and invasive isolates of *K. pneumoniae*. It has been increasingly reported, including in young and immunocompetent patients, that the hypermucoviscous phenotype is significantly associated with the capacity of *K. pneumoniae* to cause serious infections, such as pyogenic liver abscess syndrome and metastatic dissemination to other parts of the body, including the eyes, central nervous system, and lungs[14-16]. The hypermucoviscous phenotype was present in the encapsulated strains of *K. pneumoniae* (mostly K1 and to a lesser extent K2), which produced vast amounts of extracapsular polysaccharide constituting a mucoviscous web that protected these strains from phagocytosis by neutrophils and from killing by serum complement[17,18]. To the best of our knowledge, there have been few reports concerning *K. variicola* with the hypermucoviscous phenotype causing invasive liver abscess syndrome[19]. Apart from virulence factors, KLA has been significantly associated with underlying diabetes mellitus compared to non-*K. pneumoniae* primary liver abscess, especially in those who developed metastatic infection[20]. *K. variicola* has also been associated with infections in immunocompromised individuals. Some comorbidities, such as systemic lupus erythematosus, cancer, diabetes mellitus, hepatobiliary diseases, solid organ transplantation, and alcoholism, have been reported in several studies[9,10]. In this case, our patient was a healthy middle-aged woman with no history of diabetes mellitus or immunodeficiency. However, the laboratory investigations after admission showed that the patient's random blood glucose level was as high as 16.34 mmol/L, and her glycosylated hemoglobin was mildly higher (6.5%). It is generally accepted that a high blood glucose level can reduce phagocyte chemotaxis, phagocytosis, and bactericidal activity and can contribute to bacterial growth and a compromised host defense system[21]. Our case suggested that *K. variicola* infections could also occur in immunocompetent patients.

There are currently no definite guidelines for managing invasive liver abscesses. The basic consensus is the combination of early percutaneous drainage or open (laparoscopic) surgical drainage of the abscess and the prompt administration of appropriate antibiotics. As interventional radiology advances, percutaneous drainage has become more widespread. However, aggressive hepatic resection has been found to be more beneficial to patients with an acute physiology and chronic health evaluation II score of 15 or greater. Our patient benefited from the empirically and sufficiently intravenous use of meropenem and early percutaneous drainage of abscesses. However, the patient's condition did not improve in the following days. Surgical drainage was performed in a timely manner considering that the liver abscess was relatively large (larger than 10 cm in diameter). Fortunately, the surgery was successful, and the patient was cured.

## CONCLUSION

We have presented a rare case to raise clinician awareness of this worldwide emerging invasive syndrome. There is no doubt that invasive liver abscess syndrome caused by *K. variicola* is a devastating disease that can progress rapidly. In addition, urgent diagnosis and treatment are very important. Regarding the information raised in this case, we suggest that *K. variicola*, as an underappreciated pathogen, should be given more attention in clinical practice.

## FOOTNOTES

**Author contributions:** Zhang PJ, Lu ZH, Cao LJ, Chen H, and Sun Y conceived the idea of the treatment in this case; Zhang PJ drafted the original manuscript; and all authors reviewed the manuscript draft and revised it critically.

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