Case Reports in Gastroenterology

Case Rep Gastroenterol 2024;18:144–152
DOI: 10.1159/000536619
Received: March 3, 2023
Accepted: January 29, 2024
Published online: March 18, 2024

Single Case

Gastrointestinal Variant of Lemierre Syndrome due to *Fusobacterium nucleatum*: A Case Report

Reshad Salam[a] Abhiroop Verma[a] Michael Noeske[b] Lynna Alnimer[a]
Eric M. Sieloff[c] Marc S. Piper[c]

[a]Department of Internal Medicine, Ascension Providence Hospital, Michigan State University College of Human Medicine, Southfield, MI, USA; [b]Division of Radiology, Metrohealth Medical Center, Case Western Reserve University, Cleveland, OH, USA; [c]Division of Gastroenterology, Department of Internal Medicine, Ascension Providence Hospital, Michigan State University College of Human Medicine, Southfield, MI, USA

Keywords
Liver · Abscess · Liver abscess · Pyogenic liver abcess · *Fusobacterium*

Abstract
Introduction: Pyogenic liver abscess is a noteworthy health concern in North America, characterized by a mortality rate ranging from 2 to 12%. This condition is often polymicrobial, with *Streptococcus* species and *Escherichia coli* as the predominant causal pathogens in Western countries. *Fusobacterium* species, typically commensals of gastrointestinal, genital, and oral flora, have been implicated in the rare formation of tonsillar abscesses and Lemierre syndrome, including its gastrointestinal variant known as pylephlebitis. ***Case Presentation:*** We present the case of an immunocompetent male with a 2-week history of abdominal distention and pain. Abdominal magnetic resonance imaging revealed multiseptated cystic hepatic masses and portal vein thrombosis. A subsequent liver biopsy confirmed *Fusobacterium nucleatum* etiology. The patient was initiated on intravenous cefepime and oral metronidazole antibiotics. Unfortunately, the patient succumbed to cardiac arrest before a final diagnosis could be established. ***Conclusion:*** *Fusobacterium* species-associated liver abscess, coupled with the rare gastrointestinal variant of Lemierre syndrome (pylephlebitis), poses a significant mortality risk. This case underscores the rarity and clinical challenges associated with these conditions. Increased awareness among clinicians is crucial for early diagnosis and prompt intervention, potentially improving outcomes in such cases.

Correspondence to:
Reshad Salam, reshad.salam@ascension.org

Case Rep Gastroenterol 2024;18:144–152
DOI: 10.1159/000536619

Introduction

The etiology of pyogenic liver abscess (PLA) exhibits geographical variation, with *Escherichia coli* being the predominant organism in North America, *Staphylococcus* and *Streptococcus* species in Europe, and *Klebsiella* spp. in Asia [1]. PLA represents 13% of all intra-abdominal abscesses, with a propensity to occur in the right liver lobe due to its larger size and rich blood supply as compared to other liver segments [2–4]. The pathogenesis of PLA is postulated to stem from the introduction of bacteria to the liver during intra-abdominal infection, namely, those involving the biliary tract [3–5]. A less common source of infection involves spread through the portal vein or hematogenous seeding from other sources [3, 5–7]. PLA is a significant health concern and has an estimated incidence of approximately 2.3 and 3.6 per 100,000 population in the USA and carries a mortality risk between 2 and 12% [7, 8].

Fusobacterium is a facultative anaerobic Gram-negative bacilli and is considered a member of the normal microbial flora of the gastrointestinal (GI), genital, and oral mucosa. The two main species of the group are *Fusobacterium nucleatum* and *Fucobacterium necrophorum* [7, 9]. *Fusobacterium* species are an extremely rare cause of PLA and are rarely isolated in the clinical setting. There are only 15 reported cases of PLA caused by *F. nucleatum* in immunocompetent individuals and merely 22 cases of *Fusobacterium*-related pylephlebitis reported in literature [4, 10]. The majority of intra-abdominal and pelvic *Fusobacterium* infections are caused by *F. nucleatum*, affecting mostly immunodeficient hosts and older individuals with chronic medical conditions and/or malignancies [8, 11, 12]. Lemierre syndrome (LS) is a dreaded complication of *Fusobacterium* species-associated tonsillar abscess with subsequent progression to internal jugular vein septic thrombosis. The most common causal pathogen linked to LS is *F. necrophorum* [7, 10]. Interestingly, *Fusobacterium* spp. can cause a unique GI variant of LS presenting with an intra-abdominal infection and associated septic thrombophlebitis of the portal venous system known as pylephlebitis [7, 10, 12, 13]. We present the case of a 50-year-old immunocompetent male who was found to have PLA and septic pylephlebitis secondary to *F. nucleatum.* To the best of our knowledge, this is the second reported case of cryptogenic *F. nucleatum*-associated liver abscess and septic thrombophlebitis in an immunocompetent patient with no concomitant oropharyngeal disease or GI infection.

Case Presentation

A 51-year-old Gulf War veteran with a medical history of diabetes mellitus type II, alcohol use disorder, and chronic pancreatitis presented to the emergency department with a 2-week history of abdominal pain, abdominal distension, nausea, and intermittent chills. Pain was described as a constant pressure, rated 10 out of 10 in intensity and reported to feel as if his abdomen is "about to burst." At times, changing positions by leaning forward or crouching into a ball temporarily improved his symptoms. Eating meals made the pain worse. Patient tried alleviating his symptoms with acetaminophen, which did not help.

Regarding his alcohol use disorder, he had quit alcohol use 9 years prior to presentation. He was a former smoker, admitting to smoking 1 pack of cigarettes per day for 20 years. He denied recent travel, had 2 dogs at home, but denied any exposure to livestock. His medication list included semaglutide, omeprazole, and acetaminophen. His last colonoscopy was 4 years prior to admission.

Vital signs upon presentation revealed tachycardia with a heart rate of 134 beats per minute, temperature of 100.4 F, blood pressure 130/96 mm Hg, and 100% oxygen saturation while on room air. On examination, he had a firm and distended abdomen with tenderness to palpation over the right upper and lower quadrants and palpable hepatomegaly.

Case Rep Gastroenterol 2024;18:144–152
DOI: 10.1159/000536619

Laboratory evaluation revealed a white blood cell count of 13,360 cells/mm^3 with a neutrophilic predominance, thrombocytosis with 529 platelets per µL, and hemoglobin 8.0 g/dL. A complete metabolic panel showed alanine aminotransferase (ALT) of 73 IU/L, aspartate transaminase (AST) of 171 IU/L, alkaline phosphatase (ALP) 625 IU/L, total bilirubin (TB) of 1.9 mg/dL, and direct bilirubin (DB) 1.3 mg/dL. Moreover, acute kidney injury was evident as, BUN 50 mg/dL, creatinine 1.2 mg/dL, he had lactic acidosis with an elevated lactic acid of 5.2 mmol/L. An initial computed tomography (CT) scan of the abdomen and pelvis with contrast showed multiple masses throughout the liver and an associated partial splenic vein and portal vein thrombosis (PVT) (shown in Fig. 1). A CT scan from 6 months prior showed no liver pathology (shown in Fig. 1). Magnetic resonance imaging of the abdomen with and without contrast did not disclose any biliary obstruction, however revealed evidence of hepatomegaly with the craniocaudal liver span in the mid clavicular line measuring up to 26 cm and several multiseptated cystic hepatic masses with an associated PVT (shown in Fig. 2). The largest of these cystic lesions was measured at 9.0 cm × 5.4 cm.

The patient was started on intravenous (IV) cefepime and oral metronidazole for empiric coverage of abdominal microorganisms in addition to therapeutic IV heparin given the intra-abdominal thrombosis. Given the acute onset of symptoms and radiologic findings, there was a concern for parasitic or bacterial infectious etiology of multiple hepatic cystic lesions. Bacterial and fungal blood cultures obtained on admission did not reveal growth of any organism and remained negative throughout hospital stay. Laboratory testing was negative for hepatitis A IgM, hepatitis B core antibody IgM, hepatitis B surface antigen, hepatitis C antibody, HIV antibody 1 and 2, *Entamoeba histolytica* IgG, and *Echinococcus* IgG antibody. The patient underwent a CT-guided liver biopsy with aspiration of abscess material. The H&E stain from the aspirate showed inflamed and degenerating liver parenchyma with scant identifiable portal tracts and abundant fibrinopurulent inflammation; however, the resultant cultures showed no growth (shown in Fig. 3). The antibiotics were changed from IV cefepime and oral metronidazole to IV ampicillin-sulbactam and oral trimethoprim-sulfamethoxazole; however, the patient showed no clinical improvement. A 16S PCR sent for next-generation sequencing from the aspirate returned positive for *F. nucleatum.* Unfortunately, the patient passed away due to cardiac arrest before the etiology of the liver lesions could be established.

Discussion

Infectious liver abscesses are separated into two classes based on the type of pathogen involved: pyogenic and amoebic. Amoebic liver abscesses are primarily caused by infection with *Entamoeba histolytica*, which is a parasite commonly present in tropical areas and spread via mature cysts in contaminated food or water [14]. Most PLAs are polymicrobial and result from infection with mixed enteric and anaerobic species [7, 8]. *Streptococcus* species and *Escherichia coli* are the most common causal pathogens of PLA in Western countries, but *Klebsiella pneumonia* is the predominant cause in Asian nations [4]. The incidence of PLA in the USA is reportedly increasing, with the rise being attributed to the increase in hepatobiliary interventions and multidrug-resistant organisms [15].

F. nucleatum is a rare cause of PLAs with only 20 cases reported in literature, of which 15 patients are described as immunocompetent [4]. General risk factors for liver abscess include male gender, malignancy, biliary tract procedures, alcohol-use disorder, diabetes mellitus, cirrhosis, renal failure, immunosuppression, liver transplantation, dialysis, advanced age [5, 9, 11]. Forty-three percent of patients with PLA have underlying biliary disease and individuals with diabetes have 3.6 times greater risk [4]. However, these risk factors occur at a lower frequency in those with liver abscesses associated with *Fusobacterium* species and instead

Case Rep Gastroenterol 2024;18:144–152
DOI: 10.1159/000536619

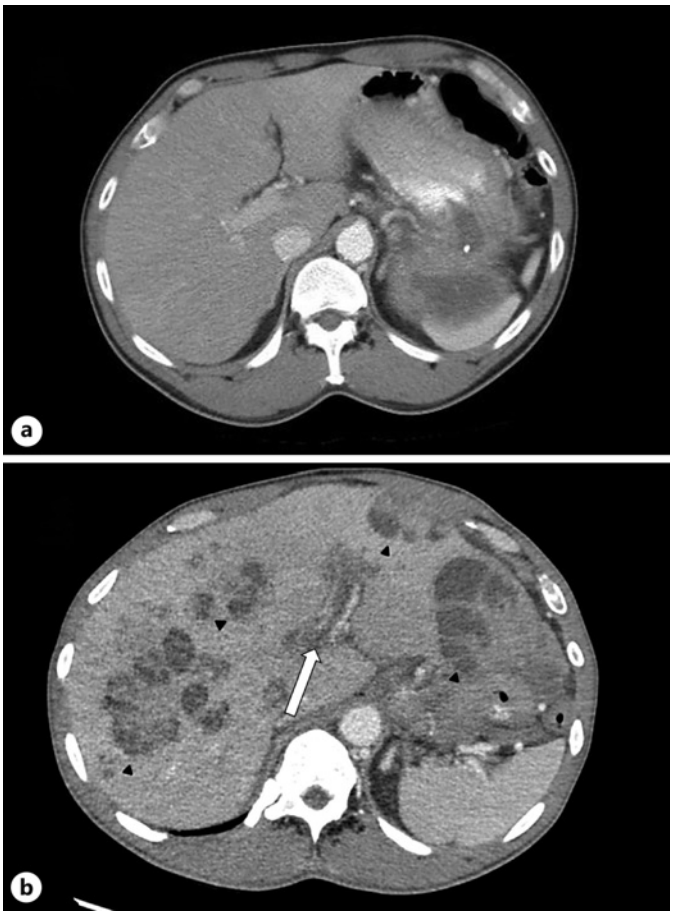

Fig. 1. a Transverse CT image of the abdomen taken 6 months prior to presentation. **b** Transverse CT image of the abdomen demonstrating multiple lesions throughout the grossly enlarged liver indicated by black arrows and hypoattenuation indicating thrombosis within the portal vein showcased by white arrow.

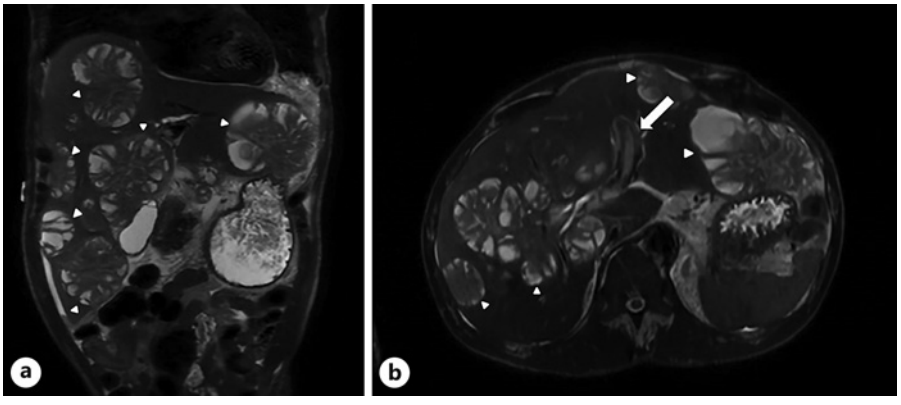

Fig. 2. a MRI of the abdomen, showing numerous lesions throughout the grossly enlarged liver by white arrowheads. **b** White arrows point to an area of increased attenuation within the portal vein indicative of thrombus.

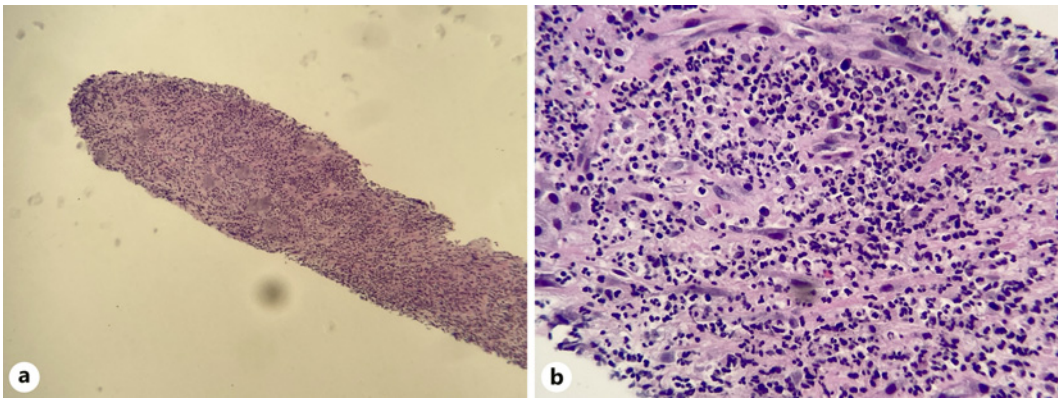

Fig. 3. Low (×40) H&E image of abscess material with granulation tissue (**a**) and high magnification (×200) H&E image of adjacent liver parenchyma with neutrophilic infiltrate (**b**).

other potential sources are thought to play a role in the development of *Fusobacterium*-associated abscesses, including the GI tract, cryptogenic, recent pharyngitis, and periodontal disease [9]. The patient described in the case had no underlying immunodeficiency or oropharyngeal disease. The initial working differential was broad and included disseminated hepatic malignancy, pyogenic or amoebic liver abscess, and *Echinococcus* hydatid cyst.

PLAs are difficult to diagnose as history and physical examination often yield non-specific results. The main presenting symptoms in most patients include fever (90%), abdominal pain (50–75%), and chills (69%) with other less frequent complaints being nausea, vomiting, headache, anorexia, weight loss, and diarrhea [3]. Early identification and treatment of PLA is crucial given the high mortality rate in untreated cases. Risk factors associated with increased risk of mortality are history of congestive heart failure, liver disease, abscess greater than 5 cm in size, presence of anaerobic infection, polymicrobial bacteremia, failure to drain abscess, increasing total bilirubin, and need for open surgical drainage [3, 8, 16]. Laboratory abnormalities most frequently observed in PLA include hypoalbuminemia, elevated levels of CRP, gamma-glutamyl transferase or alkaline phosphatase, leukocytosis, and elevation in liver tests [5, 11]. Clinical diagnosis of pylephlebitis is challenging, yet a common thread among all reported cases is the presence of a triad of symptoms: dull pain in the right upper quadrant, fever, and leukocytosis [17].

Clinically, amoebic and pyogenic liver abscesses are difficult to tell apart with overlapping symptoms and no clear difference in laboratory findings. Abdominal imaging such as ultrasonography and CT scan are initially used to assess for evidence of a liver abscess but are unable to distinguish between ALA and PLA. The gold standard for diagnosis of PLA is fine-needle aspiration and subsequent culture; however, aspirate cultures are positive in only 70–80% of cases [18]. Blood cultures are positive in merely 30–60% of PLA cases [3, 18]. Additionally, while positive blood cultures provide definitive proof of infection, they are not always essential for diagnosis. Many patients may have infected portal system thrombus without positive blood cultures as blood cultures are positive in up to 62% of cases [19]. It is crucial to recognize that negative blood cultures should not exclude the diagnosis of PLA or pylephlebitis, particularly when signs, symptoms, and imaging support it. Therefore, it is essential to initiate empiric broad spectrum antibiotic therapy while awaiting final cultures and sensitivities. In our case, an aspirate sample was sent to the microbiology department of another hospital to be analyzed using *16s rRNA* PCR analysis. This is a ribosomal RNA (rRNA) gene polymerase chain reaction (PCR) used for detection and identification of bacterial pathogens. 16s RNA gene polymerase is present in all bacteria and is amplified through this

Case Rep Gastroenterol 2024;18:144–152
DOI: 10.1159/000536619

method for identification. While isolation of an organism on culture media requires viable growth of the bacteria from the clinical specimen, rRNA PCR can detect non-viable bacterial DNA after the initiation of antibiotics from sterile sites [20]. This technique has become an important clinical tool for the detection and identification of bacterial pathogens in those patients with high clinical suspicion of infection but negative bacterial cultures and has been repeatedly proposed as an important technique especially in cases of suspected septic thrombophlebitis [21, 22]. In a study conducted by Miller et al. [23], the use of PCR with *16s rRNA* testing on all heart valve tissues exhibited notable diagnostic performance in identifying infectious endocarditis (IE). The sensitivity, specificity, negative predictive value, and positive predictive value of PCR were reported as 92%, 77.8%, 77.8%, and 92%, respectively, contrasting with culture's values of 44%, 100%, 39.1%, and 100% for the diagnosis of definite IE [23]. PCR demonstrated a superior ability to identify the microbiologic agent in 83% of cases characterized by definitive IE and negative blood cultures. For prosthetic heart valve tissue, 16S rDNA PCR displayed a sensitivity of 93% and specificity of 83%, surpassing the values of 35% and 100% observed with culture [23]. In our patient, the negative aspirate culture was most likely a result of early antibiotic administration; however, the 16s PCR analysis allowed for accurate pathogen identification.

Treatment of PLA is mainly through needle aspiration or percutaneous catheter drainage alongside selective antimicrobial therapy [18, 24]. Criteria for percutaneous drainage include ongoing fever despite >48–72 h of appropriate medical therapy, abscess size >3 cm, and clinical or imaging features concerning for impending perforation [18, 24]. Surgical drainage is indicated in the event of abscess rupture, incomplete percutaneous drainage, inadequate clinical improvement after 4–7 days of percutaneous drainage, and multiloculated abscesses [24]. Parenteral antibiotics are generally employed for ≥2 weeks before transition to oral antibiotics for an additional 4–6 weeks of therapy [24]. In *Fusobacterium*-associated PLA, the optimal treatment and duration of therapy is still undefined. Blood cultures obtained during hospitalization did not reveal any growth for our patient; therefore, antibiotic susceptibility could not be ascertained. However, metronidazole, ampicillin-sulbactam, cephalosporins, and carbapenems generally demonstrate high susceptibility (95–100%) against *Fusobacterium* species, while resistance to penicillin within this group ranges from 5 to 17% [25–27]. Clindamycin resistance rates vary regionally, ranging from 6.7% to 25% [25, 27]. The patient was initially treated with IV cefepime and oral metronidazole but switched to IV ampicillin-sulbactam and oral trimethoprim-sulfamethoxazole for broader coverage. These antibiotics should have provided adequate coverage with great susceptibility against *Fusobacterium* species. The total duration of antibiotic therapy recommended is generally 4–6 weeks, depending on clinical and imaging findings [7, 12].

Fusobacterium species have been implicated in the formation of LS, which classically presents as septic thrombophlebitis of the internal jugular vein and oropharyngeal infection, most commonly abscesses [28]. Our patient was found to have portal vein and splenic vein thromboses in the setting of *F. nucleatum*-associated hepatic abscess for which therapeutic IV heparin was initiated. This unusual presentation is considered a GI variant of LS, which has rarely been reported in literature [7, 10]. Thus far, there has been only 1 case of *F. nucleatum*-associated hepatic abscess and associated thrombophlebitis in an immunocompetent host, as reported by Rahmati et al. [7] The thrombogenic nature of *Fusobacterium* species is explained by their ability to activate the intrinsic pathway of coagulation via the human Hageman factor (Factor XII), incite platelet aggregation, and display hemagglutination activity on human erythrocytes. The consistent association of *Fusobacterium* spp. and other bacteria, especially anaerobes, with forms of septic thrombophlebitis in different venous locations has led to the hypothesis that these

conditions may share similarities in pathogenesis, prognosis, and optimal management [28]. As in other forms of septic thrombophlebitis, the role of anticoagulation in pylephlebitis as well is controversial and hindered by insufficient data.

Some studies report similar mortality in patients who were treated with therapeutic anticoagulation therapy and antibiotics versus antibiotics only [29]. However, a recent retrospective study by Naymagon et al. [30] examined a cohort of 67 pylephlebitis patients, with those treated with anticoagulation demonstrating significantly higher rates of PVT resolution compared to non-anticoagulated patients (58% vs. 21%, $p = 0.0201$) [27]. This also led to lower rates of future chronic portal hypertensive symptoms in anticoagulated patients (11% vs. 47%, $p = 0.0034$). The study showed that treatment with IV heparin, followed by transition to enoxaparin, warfarin, or direct oral anticoagulants, resulted in similar PVT resolution rates, with mean and median times to resolution being 4.4 months and 3 months, respectively [30]. Repeat abdominal imaging to assess for resolution prior to discontinuation of anticoagulation might be warranted and PVTs, which do not resolve, would need a longer course of anticoagulation.

In conclusion, we urge clinicians to be mindful of *Fusobacterium* species-associated PLA and the GI variant of LS, which may accompany it. Patients often present with nonspecific symptoms and elevated infectious and/or inflammatory markers. Although the gold standard for PLA diagnosis is biopsy for aspirate culture, 16s RNA PCR testing can provide accurate diagnostic yield especially in culture-negative bacterial cultures possibly associated with septic thrombophlebitis. Clinicians should consider PCR and magnetic resonance imaging imaging in patients with unclear abdominal infection because it may affect treatment by revealing antibiotic sensitivities and by bringing the possibility of anticoagulation among the options. Treatment involves prompt administration of appropriate antimicrobial therapy and drainage of the abscess. Studies suggest there is a role of anticoagulation for management of septic thrombophlebitis in the GI variant of LS; however, uncertainties persist regarding the optimal anticoagulant and duration. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000536619).

Acknowledgment

Histopathology imaging provided by Dr. Jonathan C. Mowers (pathologist).

Statement of Ethics

Ethical approval is not required for this study in accordance with local or national guidelines. Written and informed consent was obtained from the patient prior to his death for publication of the details of their medical case and any accompanying images. Written and informed consent could not be obtained from the next of kin of the patient for the publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

We declare no sources of funding.

Author Contributions

Reshad Salam, MD: writing – original draft. Abhiroop Verma, MD, Michael Noeske, MD, Lynna Alnimer, MD, Eric M. Sieloff, MD, and Marc S. Piper, MD, MSc: writing – review and editing.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material files. Further inquiries can be directed to the corresponding author.

References

1 Svensson E, Jönsson A, Bläckberg A, Sunnerhagen T, Kiasat A, Ljungquist O. Increasing incidence of pyogenic liver abscess in Southern Sweden: a population-based study from 2011 to 2020. Infect Dis. 2023;55(6):375–83.
2 Serraino C, Elia C, Bracco C, Rinaldi G, Pomero F, Silvestri A, et al. Characteristics and management of pyogenic liver abscess: a European experience. Medicine. 2018;97(19):e0628.
3 Roediger R, Lisker-Melman M. Pyogenic and amebic infections of the liver. Gastroenterol Clin North Am. 2020;49(2):361–77.
4 Collins L, Diamond T. *Fusobacterium nucleatum* causing a pyogenic liver abscess: a rare complication of periodontal disease that occurred during the COVID-19 pandemic. BMJ Case Rep. 2021;14(1):e240080.
5 Wilson R, LeBlanc R, Hamour A. Cryptogenic pyogenic liver abscess due to Fusobacterium nucleatum: a case report. BCMJ. 2014;56(3):130–4.
6 Hammami MB, Noonan EM, Chhaparia A, Khatib FA, Bassuner J, Hachem C. Denture-associated oral microbiome and periodontal disease causing an anaerobic pyogenic liver abscess in an immunocompetent patient: a case report and review of the literature. Gastroenterol Res. 2018;11(3):241–6.
7 Hitchcock N, Nelson T, Taylor. Pyogenic liver abscess and gastrointestinal Lemierre syndrome due to *Fusobacterium nucleatum*. Am J Hosp Med. 2021;5(1):2021.
8 Nagpal SJ, Mukhija D, Patel P. Fusobacterium nucleatum: a rare cause of pyogenic liver abscess. Springerplus. 2015;4:283.
9 Jayasimhan D, Wu L, Huggan P. Fusobacterial liver abscess: a case report and review of the literature. BMC Infect Dis. 2017;17(1):440.
10 Lazar N, Sardarli K, Imam Z, Khasawneh M, Hader I. A rare twist of the forgotten disease: a case of Fusobacterium necrophorum sepsis with portomesenteric thrombosis and a review of the literature. Case Rep Gastrointest Med. 2021;2021:6699867.
11 Wijarnpreecha K, Yuklyaeva N, Sornprom S, Hyman C. Fusobacterium nucleatum: atypical organism of pyogenic liver abscess might be related to sigmoid diverticulitis. N Am J Med Sci. 2016;8(4):197–9.
12 Rahmati E, She RC, Kazmierski B, Geiseler PJ, Wong D. A case of liver abscess and Fusobacterium septicemia. IDCases. 2017;9:98–100.
13 Zheng L, Giri B. Gastrointestinal variant of Lemierre syndrome: Fusobacterium nucleatum bacteremia-associated hepatic vein thrombosis: a case report and literature review. Am J Ther. 2016 May-Jun;23(3):e933–6.
14 Zulfiqar H, Mathew G, Horrall S. Amebiasis. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2022.
15 Sharma A, Mukewar S, Mara K, Dierkhising R, Kamath P, Cummins N. Epidemiologic factors, clinical presentation, causes, and outcomes of liver abscess: a 35-year Olmsted County study. Mayo Clin Proc Innov Qual Outcomes. 2018;2(1):16–25.
16 Losie JA, Lam JC, Gregson DB, Parkins MD. Epidemiology and risk factors for pyogenic liver abscess in the Calgary Health Zone revisited: a population-based study. BMC Infect Dis. 2021;21(1):939.
17 Radovanovic N, Dumic I, Veselinovic M, Burger S, Milovanovic T, Nordstrom CW, et al. *Fusobacterium necrophorum* subsp. *necrophorum* liver abscess with pylephlebitis: an abdominal variant of lemierre's syndrome. Case Rep Infect Dis. 2020;2020:9237267.

18 Longworth S, Han J. Pyogenic liver abscess. Clin Liver Dis. 2015;6(2):51–4.
19 Jevtic D, Gavrancic T, Pantic I, Nordin T, Nordstrom CW, Antic M, et al. Suppurative thrombosis of the portal vein (pylephlebits): a systematic review of literature. J Clin Med. 2022;11(17):4992.
20 Church DL, Cerutti L, Gürtler A, Griener T, Zelazny A, Emler S. Performance and application of 16S rRNA gene cycle sequencing for routine identification of bacteria in the clinical microbiology laboratory. Clin Microbiol Rev. 2020;33(4):e00053.
21 Bourgeaud J, Delabays B, Van den Bogaart L, Ratano D. Complex Lemierre syndrome with multisystemic abscesses. BMJ Case Rep. 2023;16(4):e254638.
22 Patel M. 16S rDNA PCR in diagnosis of Lemierre's syndrome. Lancet Infect Dis. 2013;13(3):197.
23 Miller RJ, Chow B, Pillai D, Church D. Development and evaluation of a novel fast broad-range 16S ribosomal DNA PCR and sequencing assay for diagnosis of bacterial infective endocarditis: multi-year experience in a large Canadian healthcare zone and a literature review. BMC Infect Dis. 2016;16:146.
24 Khim G, Em S, Mo S, Townell N. Liver abscess: diagnostic and management issues found in the low resource setting. Br Med Bull. 2019;132(1):45–52.
25 CLSI. Performance standards for antimicrobial susceptibility testing. Clinical and Laboratory Standards Institute; 2023.
26 Kim M, Yun SY, Lee Y, Lee H, Yong D, Lee K. Clinical differences in patients infected with Fusobacterium and antimicrobial susceptibility of Fusobacterium isolates recovered at a tertiary-care hospital in Korea. Ann Lab Med. 2022;42(2):188–95.
27 Reissier S, Penven M, Guérin F, Cattoir V. Recent trends in antimicrobial resistance among anaerobic clinical isolates. Microorganisms. 2023;11(6):1474.
28 Valerio L, Riva N. Head, neck, and abdominopelvic septic thrombophlebitis: current evidence and challenges in diagnosis and treatment. Hamostaseologie. 2020;40(3):301–10.
29 Nygren D, Elf J, Torisson G, Holm K. Jugular vein thrombosis and anticoagulation therapy in Lemierre's syndrome: a post hoc observational and population-based study of 82 patients. Open Forum Infect Dis. 2021;8(1):ofaa585.
30 Naymagon L, Tremblay D, Schiano T, Mascarenhas J. The role of anticoagulation in pylephlebitis: a retrospective examination of characteristics and outcomes. J Thromb Thrombolysis. 2020;49(2):325–31.