Case Report **ISSN 2835-6276**

American Journal of Medical and Clinical Research & Reviews

Streptococcus anginosus Bacteremia with Hepatic Abscess After Biliary Stent Exchange in a Patient with Relapsed Lymphoma: A Case Report

Kyaw Zaw Lin[1,2], Soraya Kernizan[3]

1. Emergency Department, Queens Hospital Center, New York City, United States.
2. Chang Family Medical Center, Brooklyn, New York, United States.
3. Emergency Department, Queens Hospital Center, New York City, United States.

Correspondence: Kyaw Zaw Lin

Received:30 Mar 2026; Accepted:05April 2026; Published: 15April 2026

Citation: Kyaw Zaw Lin. Streptococcus anginosus Bacteremia with Hepatic Abscess After Biliary Stent Exchange in a Patient with Relapsed Lymphoma: A Case Report. AJMCRR. 2026; 5(4): 1-8.

Abstract

A 46-year-old woman with a history of relapsed and transformed lymphoma, including marginal zone lymphoma (MZL), Hodgkin lymphoma, and subsequent transformation to diffuse large B-cell lymphoma (DLBCL) with hepatic involvement, presented with Streptococcus anginosus bacteremia and suspected hepatic abscesses following recent biliary instrumentation. She had previously undergone multiple lines of therapy, most recently chimeric antigen receptor T-cell (CAR-T) therapy with axicabtagene ciloleucel in July 2025, achieving an initial complete metabolic response.

Her clinical course was complicated by chronic obstructive cholestasis requiring biliary stent placement and serial endoscopic retrograde cholangiopancreatography (ERCP) procedures. Following a recent ERCP with stent exchange and lymph node biopsy in March 2026, she presented with fever and chills and was found to have S. anginosus bacteremia. Imaging demonstrated hepatic lesions consistent with abscesses in the setting of pneumobilia and biliary stenting.

She was treated with intravenous ceftriaxone, resulting in rapid clinical and laboratory improvement, including resolution of leukocytosis and clearance of blood cultures. Although interventional radiology–guided drainage was recommended, the patient declined invasive management and opted for prolonged intravenous antibiotic therapy. She was discharged with a plan for 4–6 weeks of ceftriaxone via peripherally inserted central catheter, along with antiviral and hepatoprotective prophylaxis.

This case underscores the risk of infectious complications, including bacteremia and hepatic abscess formation, in immunocompromised patients undergoing repeated biliary interventions, and highlights the role of conservative management in select patients.

Keywords: Streptococcus anginosus, Hepatic Abscess and Bacteremia.

Introduction

The Streptococcus anginosus group (SAG), historically referred to as the Streptococcus milleri group, constitutes part of the normal human microbiota of the oropharynx, gastrointestinal tract, and vagina [1]. These organisms, classified within the viridans streptococci, are gram positive, catalase negative, facultative anaerobic cocci that typically form small colonies on agar media [2]. Although SAG species frequently colonize mucosal surfaces without causing disease, they are also capable of producing invasive infections [3]. Several putative virulence factors have been identified—including fibronectin binding proteins, hyaluronidase, ribonucleases, deoxyribonucleases, and chondroitin sulfatase—although their precise roles in human disease remain unclear [4,5].

SAG organisms have been implicated in infections across a wide range of anatomic sites, including the skin and soft tissues, oropharynx, abdomen, brain, and respiratory tract [6]. Clinical presentations vary from minor odontogenic abscesses to severe systemic infections. Invasive disease is more common in individuals with trauma, diabetes, or malignancy [7,8]. SAG species are well recognized causes of dental abscesses, pharyngitis, tonsillitis, and peritonsillar abscesses, and may lead to complications such as parapharyngeal abscess, upper airway obstruction, necrotizing fasciitis, mediastinitis, and Lemierre syndrome [9]. Systemic complications, including sepsis and brain abscess, have also been reported. In children, SAG has been increasingly associated with severe otitis media or sinusitis complicated by epidural abscess, cerebritis, meningitis, or subdural empyema [10]. These organisms also contribute significantly to aspiration pneumonia [11]. Although community acquired pneumonia due to SAG is uncommon, cases may present with extensive pulmonary necrosis [12].

Mediastinitis is another severe thoracic manifestation of SAG infection, often arising from descending odontogenic or oropharyngeal infections or postoperative complications [13]. Patients may present with stridor, neck swelling, chest pain, dyspnea, and subcutaneous emphysema.

Intra abdominal infections—including liver abscesses, intra abdominal abscesses, and cholangitis—are common due to the organisms' presence in the gastrointestinal tract. SAG species are among the most frequent etiologies of liver abscesses, often in conjunction with Enterobacteriaceae or anaerobes. Although fever and right upper quadrant pain are typical, these symptoms may be absent [14]. Risk factors include gastrointestinal pathology (e.g., diverticulitis, malignancy), biliary tract disease or instrumentation, immunosuppression, and diabetes mellitus. Unlike Klebsiella or Escherichia coli, SAG associated liver abscesses lack distinctive clinical predictors [15]. Notably, SAG associated liver abscesses without identifiable abdominal pathology may indicate occult gastrointestinal malignancy [16].

Case Report/Case Presentation

A 46-year-old woman with a complex history of lymphoma presented with bacteremia and suspected hepatic abscesses following recent biliary instrumentation. Her oncologic history was notable for marginal zone lymphoma (MZL) diagnosed in 2018, for which she achieved complete remission after treatment with bendamustine plus rituximab. In November 2022, she experienced relapse with Hodgkin lymphoma and was treated with brentuxi-

mab vedotin plus doxorubicin, vinblastine, and dacarbazine (BV-AVD), again achieving remission. In January 2024, she developed recurrent MZL and was treated with Zanubrutinib. Her disease subsequently transformed into diffuse large B-cell lymphoma (DLBCL) with systemic involvement, including hepatic disease.

Given disease progression, she underwent chimeric antigen receptor T-cell (CAR-T) therapy with axicabtagene ciloleucel on July 21, 2025, following fludarabine and cyclophosphamide lymphodepletion. Her course was complicated by grade 2 cytokine release syndrome requiring tocilizumab, without evidence of immune effector cell-associated neurotoxicity syndrome (ICANS). Post-treatment imaging initially demonstrated a complete metabolic response. However, Computed tomography (CT) performed on January 29, 2026, revealed findings concerning for possible relapse (Fig-1,2). Positron emission tomography showed a newly identified mildly fluorodeoxyglucose (FDG)-avid aortocaval/ periaortic lymph node and indeterminate uptake near the pancreas and inferior vena cava (Fig-3).

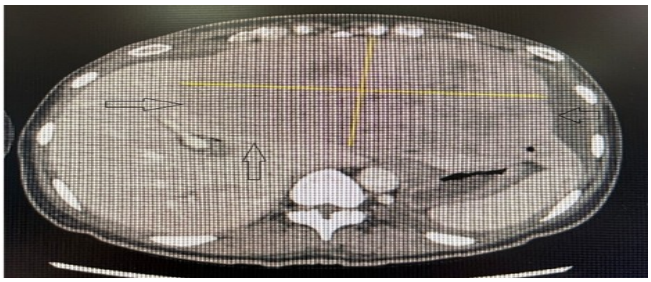

Fig:1

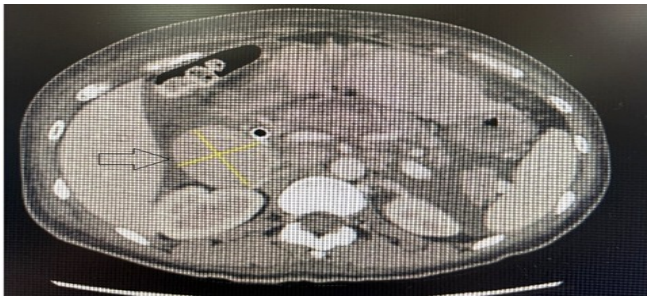

Fig:2

Fig-1 and 2: CT abdomen and pelvis with contrast: arrows showed recurrence/relapse of diffuse large B-cell lymphoma in liver and periaortic lymph node

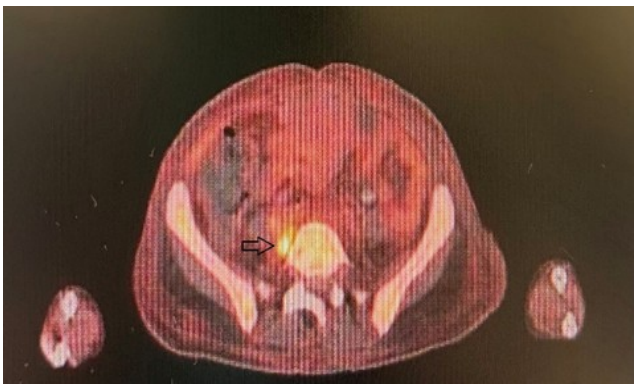

Fig:3

Fig-3: PET scan: arrow showed a newly identified mildly fluorodeoxyglucose (FDG)-avid aortocaval/ periaortic lymph node and indeterminate uptake near the pancreas and inferior vena cava.

Her clinical course was further complicated by chronic obstructive cholestasis attributed to lymphoma, previously associated with hyperbilirubinemia and severe pruritus. She underwent biliary stent placement and exchange in November 2025, followed by serial endoscopic retrograde cholangiopancreatography (ERCP) procedures approximately every three months. She was admitted from March 12 to March 13, 2026, for expedited gastrointestinal evaluation. During that admission, she underwent endoscopic ultrasound (EUS) with esophagogastroduodenoscopy (EGD) and biopsy of aortocaval lymph nodes, as well as ERCP with biliary stent exchange. EUS findings included a previously placed metal biliary stent, biliary and gallbladder sludge, and pneumobilia; the examination was otherwise limited but unremarkable.

On March 18, 2026, she presented to the emergency department with one day of fever and chills and was discharged on oral metronidazole (500 mg

twice daily) and cefpodoxime (200mg twice daily). Subsequently, she was recalled after one anaerobic blood culture grew Streptococcus anginosus, with polymerase chain reaction confirming Streptococcus species (excluding group A, group B, and Streptococcus pneumoniae). Upon return to the hospital on March 25, 2026, she reported adherence to metronidazole but had not initiated cefpodoxime. At that time, she denied fever, chills, night sweats, abdominal pain, gastrointestinal symptoms, or cardiopulmonary complaints.

Repeat evaluation demonstrated leukocytosis (white blood cell count 16.34 × 10⁹/L) with neutrophilic predominance and mild metabolic abnormalities. Liver function tests revealed alkaline phosphatase of 101 U/L, aspartate aminotransferase (AST) of 33 U/L, normal bilirubin, and albumin of 3.6 g/dL. Computed tomography (CT) of the abdomen and pelvis with contrast identified hepatic lesions measuring 3.2 × 1.2 cm and 3.0 × 4.8 cm in the left hepatic lobe, along with pneumobilia, mild intrahepatic biliary dilation, and a common bile duct stent in situ (Fig:4,5,6). CT of the chest and chest radiography showed no acute abnormalities. Testing for coronavirus disease 2019 (COVID-19) and influenza A and B was negative.

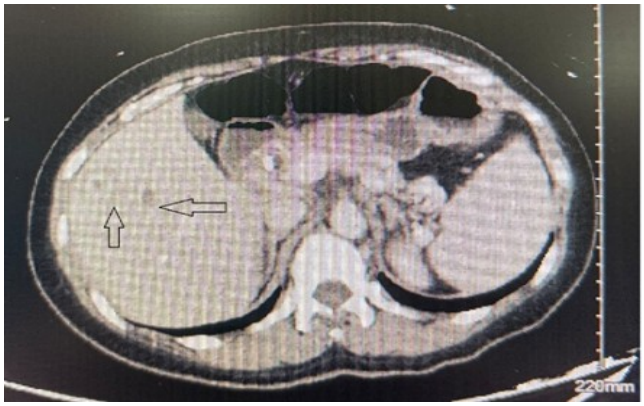

Fig:4

Fig-4: CT abdomen and pelvic with contrast: arrows showing hepatic lesions measuring 3.2 × 1.2 cm and 3.0 × 4.8 cm in the left hepatic lobe.

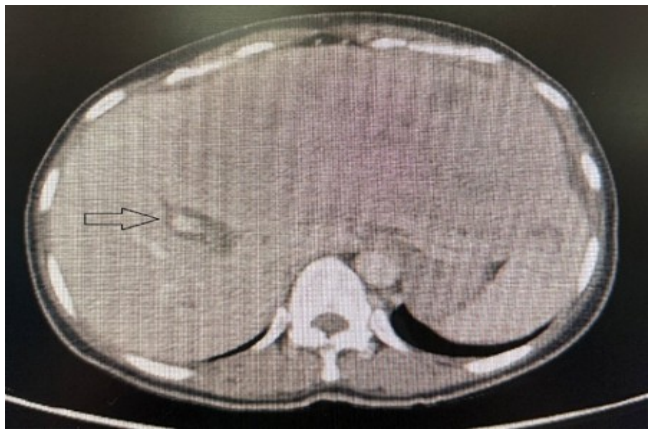

Fig:5

Fig- 5: CT abdomen and pelvic with contrast: arrows showing a hepatic3.0 × 4.8 cm abscess in the left hepatic lobe.

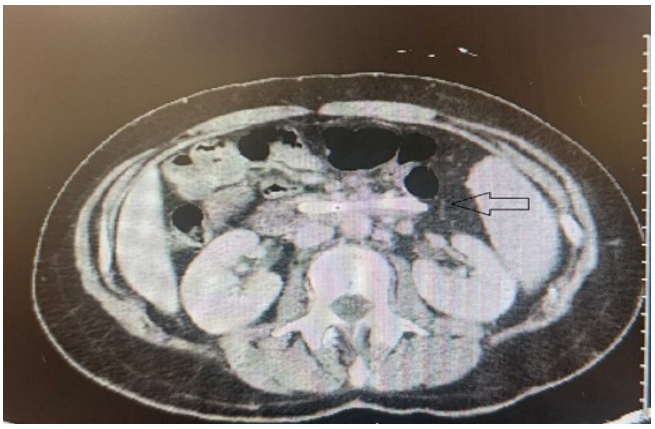

Fig:6

Fig- 6: CT abdomen and pelvic with contrast: arrow showing a common bile duct stent in situ

The infectious diseases service recommended repeat blood cultures and initiation of intravenous ceftriaxone (1 g daily), with echocardiography reserved for persistent bacteremia. The gastroenterology team attributed the hepatic lesions to likely abscess formation secondary to recent ERCP and biliary instrumentation and recommended maintaining the planned ERCP schedule. Interventional radiology was consulted, and percutaneous drainage of the hepatic abscesses was scheduled.

After three days of ceftriaxone therapy, the patient's leukocytosis improved (white blood cell

count decreased to 6.63 × 10⁹/L) without a left shift, and repeat blood cultures (two sets) demonstrated no growth. Liver enzymes on March 27, 2026, showed improvement (AST 14 U/L, alanine aminotransferase [ALT] 17 U/L, alkaline phosphatase 132 U/L, down trending). Despite recommendations, the patient declined interventional radiology–guided drainage of the hepatic abscesses. She elected to proceed with prolonged intravenous antibiotic therapy consisting of ceftriaxone (1 g daily for 4–6 weeks) via a peripherally inserted central catheter (PICC) line. She was also continued on valacyclovir for antiviral prophylaxis and ursodeoxycholic acid for Veno-occlusive disease prophylaxis.

Discussion

SAG species isolated from blood cultures rarely represent contamination [17]. Among the group, S. anginosus appears more likely to cause bacteremia than S. constellatus or S. intermedius [18]. SAG bacteremia is frequently associated with a focal infection; isolated bacteremia occurs in only 9–16% of cases [19]. Reported mortality rates range from 10% to 16% [20].

Echocardiography is recommended for patients with prosthetic valves, persistent bacteremia (>48 hours despite appropriate therapy), or those meeting Duke criteria for possible endocarditis. If transthoracic echocardiography is nondiagnostic, transesophageal echocardiography should be performed. S. anginosus appears more likely to cause endocarditis than S. intermedius or S. constellatus [21].

Isolation of SAG species from blood, percutaneous abscess aspirates, or other sterile sites generally indicates true infection [22]. In contrast, isolation from urine, biliary drains, or decubitus ulcers may represent colonization in the absence of clinical signs of infection [23].

Because SAG infections frequently involve abscess formation, evaluation for both local and distant abscesses is essential. All abscesses should be drained when feasible. A comprehensive diagnostic workup—including detailed history, physical examination, and targeted imaging—is required to identify intra abdominal abscesses, deep head and neck infections, brain abscesses, and endocarditis. Given the high prevalence of abdominal pathology in SAG bacteremia, abdominal imaging (CT or MRI) should be obtained in all patients, with chest and brain imaging considered when no clear source is identified [24].

Management of SAG infections requires both antimicrobial therapy and source control through abscess drainage or surgical debridement [25]. Intravenous antibiotics are recommended for most patients, except in minor odontogenic infections where oral therapy combined with dental intervention may suffice. Ceftriaxone is the preferred agent due to its favorable tissue penetration and convenient dosing schedule. Alternatives include penicillin G or ampicillin sulbactam. For patients with penicillin allergy or resistant isolates, vancomycin or linezolid may be used. Because SAG infections are often polymicrobial, empiric anaerobic coverage with metronidazole should be considered.

Duration of therapy depends on the infection site and adequacy of source control. Treatment should continue until clinical and radiologic resolution, guided by infectious disease consultation. Agents such as daptomycin, fluoroquinolones, macrolides, sulfonamides, and tetracyclines are not recom-

mended for SAG infections [25].

Transition to oral therapy may be appropriate after clinical improvement, adequate source control, and a short course of intravenous therapy (median 4 days), as supported by observational data [26]. Suitable oral regimens include amoxicillin clavulanate, cefuroxime, or linezolid, often combined with anaerobic coverage such as metronidazole or clindamycin. However, prolonged intravenous therapy is required for central nervous system infections, endovascular infections, complicated bacteremia with metastatic foci, or bacteremia of unknown source. Prognosis is generally favorable in the absence of endocarditis [27].

Uncomplicated bacteremia typically requires two weeks of intravenous therapy. Longer courses are necessary for inadequately drained abscesses, endocarditis, or osteomyelitis. In cases involving multiple small abscesses that cannot be drained, extended antimicrobial therapy (6–8 weeks) with close monitoring is recommended [28,29].

Conclusion

This case highlights the complexity of managing immunocompromised patients with relapsed and transformed lymphoma, particularly in the context of biliary obstruction and repeated instrumentation, which may predispose to bacteremia and hepatic abscess formation.

Author Contributions

Kyaw Zaw Lin, PA-C: conceptualization, data curation, writing–original draft. writing–review and edition and Soraya Kernizan, DO: resources, supervision.

Acknowledgments

We would like to thank the patient for their consent to participate in this case report.

Funding

The authors received no financial support for the research, authorship, or publication of this article.

Ethics Statement

The authors have nothing to report.

Consent

Informed written consent was obtained from the patient for the publication of this case report and accompanying images.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data supporting the findings of this case report are available from the corresponding author upon reasonable request.

References

1. Jiang, S., Li, M., Fu, T., et al. (2020). Clinical characteristics of infections caused by Streptococcus anginosus group. Scientific Reports, 10, 9032.
2. Willcox, M. D., & Knox, K. W. (1990). Surface -associated properties of Streptococcus milleri group strains and their potential relation to pathogenesis. Journal of Medical Microbiology, 31, 259–264.
3. Scheithauer, B. K., Wos-Oxley, M. L., Ferslev, B., et al. (2009). Characterization of the complex bacterial communities colonizing biliary stents reveals a host-dependent diversity. ISME Journal, 3, 797–807.

4. Kodama, Y., Ishikawa, T., Shimoyama, Y., et al. (2018). The fibronectin-binding protein homologue Fbp62 of Streptococcus anginosus is a potent virulence factor. Microbiology and Immunology, 62, 624–632.
5. Jacobs, J. A., & Stobberingh, E. E. (1995). Hydrolytic enzymes of Streptococcus anginosus, Streptococcus constellatus, and Streptococcus intermedius in relation to infection. European Journal of Clinical Microbiology & Infectious Diseases, 14, 818–825.
6. Whiley, R. A., Beighton, D., Winstanley, T. G., et al. (1992). Streptococcus intermedius, Streptococcus constellatus, and Streptococcus anginosus (the Streptococcus milleri group): Association with different body sites and clinical infections. Journal of Clinical Microbiology, 30, 243–247.
7. Jiang, S., Li, M., Fu, T., et al. (2020). Clinical characteristics of infections caused by Streptococcus anginosus group. Scientific Reports, 10, 9032.
8. Jiang, S., Li, M., Fu, T., et al. (2020). Clinical characteristics of infections caused by Streptococcus anginosus group. Scientific Reports, 10, 9032.
9. Santos, F. V., Pires, S. X., Pereira, C., et al. (2020). Deep neck space infection and Lemierre's syndrome caused by Streptococcus anginosus: A case report. IDCases, 19, e00669.
10. Otto, W. R., Paden, W. Z., Connors, M., et al. (2021). Suppurative intracranial complications of pediatric sinusitis: A single-center experience. Journal of the Pediatric Infectious Diseases Society, 10, 309–315.
11. Shinzato, T., & Saito, A. (1995). The Streptococcus milleri group as a cause of pulmonary infections. Clinical Infectious Diseases, 21 (Suppl 3), S238–S243.
12. Hannoodi, F., Ali, I., Sabbagh, H., & Kumar, S. (2016). Streptococcus intermedius causing necrotizing pneumonia in an immunocompetent female: A case report and literature review. Case Reports in Pulmonology, 2016, 7452161.
13. Porta, G., Rodríguez-Carballeira, M., Gómez, L., et al. (1998). Thoracic infection caused by Streptococcus milleri. European Respiratory Journal, 12, 357–362.
14. Pang, T. C., Fung, T., Samra, J., et al. (2011). Pyogenic liver abscess: An audit of 10 years' experience. World Journal of Gastroenterology, 17, 1622–1630.
15. Masood, U., Sharma, A., Lowe, D., et al. (2016). Colorectal cancer associated with Streptococcus anginosus bacteremia and liver abscesses. Case Reports in Gastroenterology, 10, 769–774.
16. Law, S. T., & Kong, L. M. K. (2013). Is there any difference in pyogenic liver abscess caused by Streptococcus milleri and Klebsiella spp.? Retrospective analysis over a 10-year period in a regional hospital. Journal of Microbiology, Immunology and Infection, 46, 11–18.
17. Casariego, E., Rodriguez, A., Corredoira, J. C., et al. (1996). Prospective study of Streptococcus milleri bacteremia. European Journal of Clinical Microbiology & Infectious Diseases, 15, 194–200.
18. Junckerstorff, R. K., Robinson, J. O., & Murray, R. J. (2014). Invasive Streptococcus anginosus group infection: Does the species predict the outcome? International Journal of Infectious Diseases, 18, 38–40.
19. Bert, F., Bariou-Lancelin, M., & Lambert-Zechovsky, N. (1998). Clinical significance of bacteremia involving the Streptococcus milleri group: 51 cases and review. Clinical Infectious Diseases, 27, 385–387.

20. Laupland, K. B., Pasquill, K., Parfitt, E. C., et al. (2018). Streptococcus anginosus group bloodstream infections in the western interior of British Columbia, Canada. Infectious Diseases, 50, 423–431.
21. Chamat-Hedemand, S., Dahl, A., Østergaard, L., et al. (2020). Prevalence of infective endocarditis in streptococcal bloodstream infections is dependent on streptococcal species. Circulation, 142, 720–730.
22. Belko, J., Goldmann, D. A., Macone, A., & Zaidi, A. K. (2002). Clinically significant infections with organisms of the Streptococcus milleri group. Pediatric Infectious Disease Journal, 21, 715–723.
23. Van der Auwera, P. (1985). Clinical significance of Streptococcus milleri. European Journal of Clinical Microbiology, 4, 386–390.
24. Furuichi, M., & Horikoshi, Y. (2018). Sites of infection associated with Streptococcus anginosus group among children. Journal of Infection and Chemotherapy, 24, 99–103.
25. Seeto, R. K., & Rockey, D. C. (1996). Pyogenic liver abscess: Changes in etiology, management, and outcome. Medicine, 75, 99–113.
26. Kowlessar, P. I., O'Connell, N. H., Mitchell, R. D., et al. (2006). Management of patients with Streptococcus milleri brain abscesses. Journal of Infection, 52, 443–450.
27. Suzuki, H., Hase, R., Otsuka, Y., & Hosokawa, N. (2016). Bloodstream infections caused by Streptococcus anginosus group bacteria: A retrospective analysis of 78 cases at a Japanese tertiary hospital. Journal of Infection and Chemotherapy, 22, 456–460.
28. Bert, F., Bariou-Lancelin, M., & Lambert-Zechovsky, N. (1998). Clinical significance of bacteremia involving the Streptococcus milleri group: 51 cases and review. Clinical Infectious Diseases, 27, 385–387.
29. Ripley, R. T., Cothren, C. C., Moore, E. E., et al. (2006). Streptococcus milleri infections of the pleural space: Operative management predominates. American Journal of Surgery, 192, 817–821.