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Case Report

Gemella haemolysans Bacteraemia in a Patient with Solitary Liver Abscess

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We present a case of a 42-year-old man with a solitary liver abscess and *Gemella haemolysans* bacteraemia. No other focus of infection was identified. The patient did not have any predisposing factors. He was treated with antibiotics for 6 weeks and the abscess was drained. He made a complete recovery.

KEYWORDS: *Gemella haemolysans*, liver abscess

Introduction

Solitary or multiple liver abscesses arise as a result of haematogenous spread of bacteria from a distant site, or from local spread from contiguous sites. Associated disease of the biliary tract is the most common source of infection. Pyogenic liver abscesses are often polymicrobial in origin.[1] Early diagnosis followed by appropriate antibiotic therapy with or without percutaneous drainage is essential to reduce morbidity and mortality. Coliforms such as *Escherichia coli* and *Klebsiella* are the most common bacterial isolates from liver abscesses. To the best of our knowledge, we report the first case of *Gemella haemolysans* bacteraemia in a patient with a liver abscess.

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Case Report

A 42-year-old man developed fever, night sweats, rigors and generalized muscle ache for 1 week. Ten days later he started to feel nauseated, vomited a few times and developed a dry cough. He also noticed that he was passing "dark urine".

At presentation, he appeared very unwell, septic, pyrexial and icteric. There was evidence of right basal consolidation and moderate hepatomegaly. Investigations revealed a neutrophilic leukocytosis (total white blood cell count = 47.4×10^9/L; neutrophils = 44.5×10^9/L), raised C-reactive protein level (307 mg/L), abnormal liver function tests (aspartate aminotransferase = 219 U/L; alanine aminotransferase = 185 U/L; alkaline phosphatase = 277 U/L), and mild hyponatremia (129 mmol/L). Renal function and plasma glucose were normal. Markers for viral hepatitis (hepatitis B surface antigen, hepatitis A IgM, and hepatitis C antibody) and human immunodeficiency virus antibody were all negative. Blood culture taken on admission grew of *G. haemolysans*. *Gemella* was identified with the help of API 20 Strep system (Biomerieux, March l'Etoile, France). It was identified as *G. haemolysins* because it was 2-naphthyl phosphate positive, VP positive, and L-leucine Beta-naphthylamide negative. It was susceptible to penicillin, ceftriaxone, erythromycin, tetracycline and vancomycin.

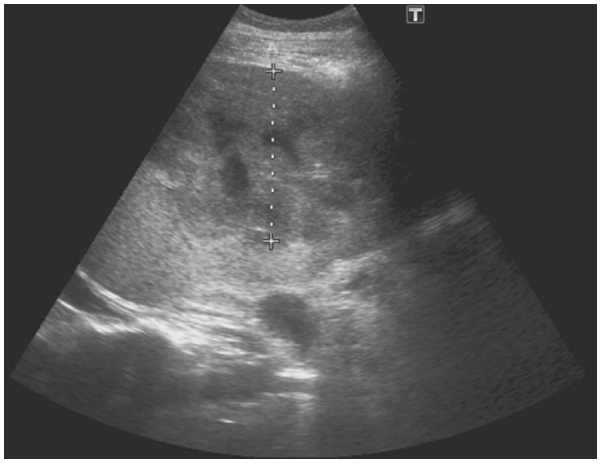

Figure 1. Ultrasonography of liver shows echo poor area with central areas of breakdown with peripheral enhancement suggestive of liver abscess.

Echocardiogram showed no evidence of infective endocarditis and colonoscopy was normal.

Ultrasound of abdomen confirmed enlargement of the liver with the presence of an echo-poor lesion with enhancing margins in the right lobe of the liver (Figure 1). Doppler flow of the lesion was negative. Diagnosis of liver abscess was confirmed by abdomen computed tomography (CT) scan. The scan showed a 12.5 × 10.0 × 7.5 cm lesion in the right lobe of the liver, which had a relatively subtle largely peripheral solid component on both arterial and venous phase imaging and a large multifocal central area of fluid density suggestive of central necrosis (Figure 2). There was also evidence of bibasal collapse consolidation (more marked on right side) with a small pleural effusion on the right side. The patient was treated with intravenous ceftriaxone and initial percutaneous aspiration, but he failed to improve despite 10 days of antibiotic therapy. A decision was taken to drain the abscess again, under ultrasound guidance, using the Seldinger technique. Straw colored turbid fluid was drained from the abscess cavity using a 6.5 French locking pig-tail catheter. Gram-stain of pus revealed no organisms. Culture of the pus did not grow any bacteria, most likely due to prior antibiotics treatment. No anaerobe was grown either. The source of bacteremia was unknown.

Antibiotics were administered for 6 weeks via a peripherally inserted central catheter (PICC) line. The patient made a complete recovery. Follow-up CT scan of the liver was normal.

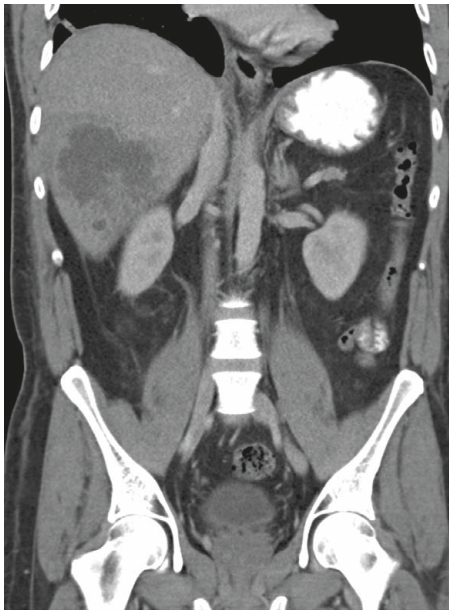

Figure 2. Computed tomography scan shows a 12.5 × 7.5 × 10 cm lesion in the right lobe of the liver, which had a relatively subtle largely peripheral solid component with a large multifocal central area of fluid density suggestive of central necrosis.

Discussion

Gemella species are facultative anaerobic Gram-positive *cocci*, which are commensal organisms of the human oral cavity, gastrointestinal tract, upper respiratory tract and genitourinary tract.[2] The members of this genus include *G. haemolysans*, *G. morbillorum*, *G. bergeri*, *G. sanguinis*, *G. palaticanis* and *G. cuniculi*. DNA hybridization and comparative 16s rRNA gene sequencing is used to classify the different members of this genus.[3] Infections due to *Gemella* closely resemble those caused by the Viridans *streptococci*.

G. haemolysans was first described in 1938.[4] It is catalase and oxidase negative and ferments carbohydrates.[5] It is easily decolorized in the Gram-stain and may appear as Gram-negative.[6] This led to misclassification of this organism in the genus *Neisseria* prior to the availability of nucleic acid hybridization techniques.[7]

Even though *G. haemolysans* is easily decolorized in Gram-stain, some cells however do not decolorize and remain Gram-positive. The microscopic film can therefore appear as Gram-variable cocci present in pairs or tetrads.

Generally human infection caused by *Gemella* species are associated with underlying conditions, including an immunocompromised state, cancer, heart disease, sinusitis

or poor dental hygiene as well as previous invasive procedures. However, our patient did not have any of these predispositions.

G. haemolysans has been reported to cause endocarditis, endophthalmitis, keratopathy, meningitis, brain abscess, pharyngeal abscess, pneumonia, empyema thoracis and vertebral osteomyelitis.[2,3,8–24]

Infections of the biliary tract (e.g. cholangitis, cholecystitis) are the most common identifiable source of liver abscess. Infection usually spreads to the liver from the bile duct along a penetrating vessel or from an adjacent septic focus (including pylephlebitis).[25] In 40% of the pyogenic liver abscess cases, no obvious source of infection can be identified. Most of the pyogenic liver abscesses are polymicrobial. The most frequently isolated organisms are *E. coli* and *Klebsiella*. However, other Gram-negative organisms, Gram-positive organisms and anaerobes also cause liver abscess. Fungal and mycobacterial liver abscesses are rare. There are rare reports of *G. morbillorum* from liver abscesses.[26–28] However, isolation of *G. haemolysans* from blood has not been previously associated with liver abscess.

Gemella is generally sensitive to β-lactam antibiotics.[29] Resistance has been reported to vancomycin, teicoplanin, erythromycin and tetracycline.[29–31] Cephalosporins are useful broad-spectrum therapeutic choices for treating pyogenic liver abscesses. In addition, metronidazole is often added to cover anaerobic organism. Antibiotic therapy should be administered intravenously for at least 2 weeks initially and then orally for up to 6 weeks.[32] The mortality rate for patients with hepatic abscesses treated with antibiotics and percutaneous drainage has improved dramatically in the last few decades but still remains at about 6%.[33] Worse outcome is associated with a delay in diagnosis, resistant organisms, incorrect choice of antibiotics, multiple abscesses, multiple organisms, shock, jaundice, underlying malignancy and immunocompromised state, multi-organ dysfunction and other medical co-morbidities.[6]

Gemella is a rare but important cause of pyogenic infections and if isolated from sterile sites, should never be dismissed as a contaminant. While bacteraemia due to streptococci and the related genera should always arouse the suspicion of infective endocarditis, investigations for the presence of other pyogenic foci should be carried out if no vegetations are found on cardiac ultrasound even in absence of predisposing factors. In particular, it is worth emphasizing that compared to typical Viridans *streptococci*, isolation of *Gemella* is less often associated with infective endocarditis. In Facklam's review, only 8/46 cases of Gemella infection could be attributed to endocarditis compared with *S. sanguis* (106/202) or *S. mutans* (64/152).

References

1. Alvarez JA, González JJ, Baldonedo RF, Sanz L, Carreño G, Jorge JI. Single and multiple pyogenic liver abscesses: etiology, clinical course, and outcome. *Dig Surg* 2001;18:283–8.
2. Khan R, Urban C, Rubin D, Segal-Maurer S. Subacute endocarditis caused by Gemella haemolysans and a review of the literature. *Scand J Infect Dis* 2004;36:885–8.
3. Woo PC, Lau SK, Fung AM, Chiu SK, Yung RW, Yuen KY. Gemella bacteraemia characterised by 16S ribosomal RNA gene sequencing. *J Clin Pathol* 2003;56:690–3.
4. Berger U, Pervanidis A. Differentiation of Gemella haemolysans (Thjøtta and Bøe 1938) Berger 1960, from Streptococcus morbillorum (Prevot 1933) Holdeman and Moore 1974. *Zentralbl Bakteriol Mikrobiol Hyg A* 1986;261:311–21.
5. Facklam R, Elliott JA. Identification, classification, and clinical relevance of catalase-negative, gram-positive cocci, excluding the streptococci and enterococci. *Clin Microbiol Rev* 1995;8:479–95.
6. Alvarez Pérez JA, González JJ, Baldonedo RF, Sanz L, Carreño G, Junco A, et al. Clinical course, treatment, and multivariate analysis of risk factors for pyogenic liver abscess. *Am J Surg* 2001;181:177–86.
7. Reyn A, Birch-Andersen A, Berger U. Fine structure and taxonomic position of Neisseria haemolysans (Thjotta and Boe 1938) or Gemella haemolysans (Berger 1960). *Acta Pathol Microbiol Scand B Microbiol Immunol* 1970;78:375–89.
8. Anil M, Ozkalay N, Helvaci M, Agus N, Guler O, Dikerler A, et al. Meningitis due to Gemella haemolysans in a pediatric case. *J Clin Microbiol* 2007;45:2337–9.
9. Díaz-Pedroche C, López-Medrano F, Arrese I, García-Martínez J. Cerebral abscess due to Gemella haemolysans. *Enferm Infecc Microbiol Clin* 2005;23:385–6. [In Spanish]
10. da Costa CT, Porter C, Parry K, Morris A, Quoraishi AH. Empyema thoracis and lung abscess due to Gemella morbillorum. *Eur J Clin Microbiol Infect Dis* 1996;15:75–7.
11. Eggelmeijer F, Petit P, Dijkmans BA. Total knee arthroplasty infection due to Gemella haemolysans. *Br J Rheumatol* 1992;31:67–9.
12. Eisenberger U, Brunkhorst R, Perharic L, Petersen R, Kliem V. Gemella morbillorum—spondylodiscitis in a patient with a renal graft. *Nephrol Dial Transplant* 1998;13:1565–7.

13. Eisenhut M, Jones C, Hughes D, Herrington S, Kokai G. Acute renal failure associated with Gemella haemolysans pneumonia. *Pediatr Nephrol* 2004;19:448–50.
14. Frésard A, Michel VP, Rueda X, Aubert G, Dorche G, Lucht F. Gemella haemolysans endocarditis. *Clin Infect Dis* 1993;16:586–7.
15. Kailasanathan A, Anderson DF. Infectious crystalline keratopathy caused by Gemella haemolysans. *Cornea* 2007;26:643–4.
16. Lee MR, Lee SO, Kim SY, Yang SM, Seo YH, Cho YK. Brain abscess due to Gemella haemolysans. *J Clin Microbiol* 2004;42:2338–40.
17. Martha B, Duong M, Buisson M, Grappin M, Piroth L, Chavanet P, Portier H. Acute Gemella haemolysans spondylodiscitis in an immunocompetent patient. *Presse Med* 2003;32:1273–5. [In French]
18. May T, Amiel C, Lion C, Weber M, Gerard A, Canton P. Meningitis due to Gemella haemolysans. *Eur J Clin Microbiol Infect Dis* 1993;12:644–5.
19. Omran Y, Wood CA. Endovascular infection and septic arthritis caused by Gemella morbillorum. *Diagn Microbiol Infect Dis* 1993;16:131–4.
20. Pradeep R, Ali M, Encarnacion CF. Retropharyngeal abscess due to Gemella morbillorum. *Clin Infect Dis* 1997;24:284–5.
21. Ritterband D, Shah M, Kresloff M, Intal M, Shabto U, Seedor J. Gemella haemolysans keratitis and consecutive endophthalmitis. *Am J Ophthalmol* 2002;133:268–9.
22. Roche M, Smyth E. A case of septic arthritis due to infection with Gemella morbillorum. *J Infect* 2005;51:e187–9.
23. Valipour A, Koller H, Setinek U, Burghuber OC. Pleural empyema associated with Gemella morbillorum: report of a case and review of the literature. *Scand J Infect Dis* 2005;37:378–81.
24. Veziris N, Fuhrman C, Chouaid C, Marque E, Housset B, Lange J, et al. Empyema of the thorax due to Gemella haemolysans. *J Infect* 1999;39:245–6.
25. Brook I, Frazier EH. Microbiology of liver and spleen abscesses. *J Med Microbiol* 1998;47:1075–80.
26. Hsu CY, Su YC, Wang TL, Chong CF, Chen CC. Gemella morbillorum liver abscess. *Scand J Infect Dis* 2007;39:637–8.
27. Nam HJ, Yoon SJ, John BM, Jung SH, Kim A, Ko BS, et al. Liver abscess caused by Gemella morbillorum. *Korean J Gastroenterol* 2005;46:56–9. [In Korean]
28. Whitney AM, O'Connor SP. Phylogenetic relationship of Gemella morbillorum to Gemella haemolysans. *Int J Syst Bacteriol* 1993;43:832–8.
29. BERGER U. The sensitivity of Gemella haemolysans to some commonly used antibiotics. *Arch Hyg Bakteriol* 1960;144:12–6. [In German]
30. Buu-Hoï A, Sapoetra A, Branger C, Acar JF. Antimicrobial susceptibility of Gemella haemolysans isolated from patients with subacute endocarditis. *Eur J Clin Microbiol* 1982;1:102–6.
31. Reed C, Efstratiou A, Morrison D, Woodford N. Glycopeptide-resistant Gemella haemolysans from blood. *Lancet* 1993;342: 927–8.
32. Ng FH, Wong WM, Wong BC, Kng C, Wong SY, Lai KC, et al. Sequential intravenous/oral antibiotic vs. continuous intravenous antibiotic in the treatment of pyogenic liver abscess. *Aliment Pharmacol Ther* 2002;16:1083–90.
33. Lee KT, Wong SR, Sheen PC. Pyogenic liver abscess: an audit of 10 years' experience and analysis of risk factors. *Dig Surg* 2001;18:459–65; discussion 465–6.