Case Report

ANNALS OF MEDICINE & SURGERY

OPEN

Emerging liver infection of *Veillonella parvula* associated with acute respiratory distress syndrome: a case report

Aldin Malkoc, MD[a,*], Lana Mamoun, BS[b], Danielle Cremat, MD[a], Amira Barmanwalla, MD[a], Alexander Phan, MD[a], Amanda Daoud, MD[a], Kevin Perez, MD[a], Brandon Woodward, MD[a]

Introduction: *Veillonella parvula* is a bacteria that can be found in normal oral and gastrointestinal flora. *Veillonella* infection is rare in immunocompetent patients but is known to cause periorbital cellulitis, endocarditis, osteomyelitis and bacteremia; however, its association with acute respiratory distress syndrome (ARDS) has not been previously documented.

Case presentation: A 36-year-old female with no known history who presented with right-sided chest, flank and upper abdominal pain after a motor vehicle accident. Computed tomography showed multiple right rib fractures, small right pneumothorax, and a grade 4 liver laceration with active extravasation of the posterior aspect of the right liver lobe. Over the hospital course, the patient developed ARDS and was intubated for hypoxemia. A right posterior liver abscess was percutaneously drained, with a copious amount of air and ~30 ml turbid fluid aspirated. Cultures from the liver abscess grew *Veillonella parvula*. She was treated with Micafungin, Levofloxacin, and Metronidazole for the hepatic abscess, and was discharged home with outpatient follow-up.

Discussion: The authors present one of the first reported cases of a *V. parvula* infected liver abscess associated with ARDS in an immunocompetent patient.

Conclusion: These clinical findings are unique due to the nature of our patient's ARDS onset and the dearth of similar cases in the literature. The favorable outcome of our patient was due to a multidisciplinary and early identification of a *V. parvula* hepatic abscess. The authors' findings contribute to the future management of *V. parvula* and a greater understanding of its disseminating effects and presentation in immunocompetent patients.

Keywords: acute respiratory distress syndrome, liver abscess, Veillonella parvula

Introduction

Infections related to *Veillonella* species are rare in immunocompromised patients and reports of infection in the immunocompetent are even rarer[1]. *Veillonella parvula* is an anaerobic gram-negative coccus that can be found in the normal flora of the oral cavity, gastrointestinal, and genitourinary tracts[2]. *Veillonella's* virulent capacity is a result of lipopolysaccharide-induced Toll-like receptor 4 (TLR-4) pathway activation[2]. The *Veillonella* species' growth and biofilm formation depends on their symbiotic relationship with the *Streptococcus* species for the production of lactate[3]. Their oral biofilm production contributes to their antibiotic resistance and the pathogenesis of oral infections. It can persist in the oral cavity, disseminate, and cause infections in multiple organ systems[3,4].

HIGHLIGHTS

- *V. parvula* is a rare infection and is becoming more prevalent in immunocompetent patients who require critical care.
- Unique infections leading to acute respiratory distress syndrome need to have a low threshold for broad-spectrum treatment.
- Treatment of intra-abdominal *V. parvula* abscess infection will long term antibiotic administration.

Immunocompetent patients diagnosed with spondylodiscitis were unexpectedly found to have blood and tissue cultures positive for *Veillonella*[1,5]. Along with the extremely rare nature of *Veillonella* spinal infections, disseminated *Veillonella* has caused periorbital cellulitis, endocarditis, epidural abscesses, meningitis, septic arthritis, osteomyelitis, and bacteremia[6–12]. Additionally, *Veillonella* infections have been associated with inflammatory and autoimmune liver diseases[13,14]. It has been suggested that imbalances in the gut microbiome can lead to liver disease through the portal circulation and contribute to primary sclerosing cholangitis (PSC) and inflammatory bowel disease (IBD)[15,16]. However, there are no reports on liver abscesses secondary to *Veillonella Parvula* infections.

In cases of severe dissemination and septicemia, excessive activation of the inflammatory and coagulation pathways can

[a]Arrowhead Regional Medical Center and [b]California University of Science and Medicine, Colton, CA, USA

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

*Corresponding author. Address: Arrowhead Regional Medical Center, 400 N 51 Pepper Ave, Colton, CA 92324, USA. Tel.: +909 580 1000.
E-mail: Malkoca1@armc.sbcounty.gov (A. Malkoc).

Annals of Medicine & Surgery (2024) 86:4870–4874

Received 24 March 2024; Accepted 5 June 2024

Published online 14 June 2024

http://dx.doi.org/10.1097/MS9.0000000000002278

result in acute respiratory distress syndrome (ARDS), which is characterized by diffuse lung inflammation, alveolar edema, injury to lung endothelium and epithelium, and hyaline membrane deposition[17]. The Berlin Criteria for ARDS includes onset within one week of clinical insult, bilateral opacities not fully explained by effusions or nodules, and respiratory failure[18]. It was reported that ARDS-associated mortality rate is 38.5–58%; thus, it is crucial to raise awareness of disseminated *Veillonella* and its presentation[19,20]. To the best of our knowledge, there are no documented cases on a *Veillonella parvula* liver abscess that progressed to ARDS in an immunocompetent individual. This case report was prepared in accordance with SCARE guidelines[31].

Case presentation

A 36-year-old female with no reported medical history presented to our Level 1 trauma center following a motor vehicle accident. Initial vital signs included: blood pressure of 105/52 mmHg, heart rate of 94 beats per min, respiratory rate of 29 breaths per min, and oxygen saturation of 99% on room air with a temperature of 36.5°C. On physical exam, she had tenderness to palpation of the right flank and right anterior chest, with a soft and non-distended abdomen exhibiting generalized diffuse tenderness. Laboratory evaluation showed white blood cells 14×10^3/μl, hemoglobin 12.6 g/dl, and platelets 251×10^3/μl. Computed tomography (CT) of the chest, abdomen, and pelvis, showed fractures of right ribs 1, 2, 4, 5 and 6, small right pneumothorax, grade 4 laceration of the right lobe of the liver, and a small focus of active extravasation seen along the posterior aspect of the liver, Figure 1A and B.

She was admitted to the ICU for serial abdominal exams and hemodynamic monitoring. The interventional radiologist did not recommend intervention, as the liver laceration was likely from a small capsular branch without a clear connection to the hepatic artery. Following a 24-h period of stability in the ICU, she was downgraded to the telemetry unit.

The next day, she reported abdominal pain associated with emesis after an oral diet. Abdominal x-ray showed severe ileus. Chest X-ray showed hazy consolidations bilaterally with a dense consolidation at the right lung base. She was noted to be tachypneic and hypoxic. A right 28F chest tube was placed for concerns of a delayed hemopneumothorax. She was placed on a high-flow nasal cannula (HFNC) at 45 l per min (LPM) flow rate and 45% fraction of inspired oxygen (FiO_2). On hospital day 3, chest physiotherapy was initiated, and HFNC settings were increased to 70 LPM and 50% FiO_2 due to hypoxia. The arterial blood gas (ABG) at that time included a pH of 7.46, pCO_2 of 33, pO_2 of 88, HCO_3 of 22.9, and a *P*/F ratio of 176. Blood cultures were obtained multiple times but were consistently negative. Lactate levels and liver function tests were within normal limits.

On day 5, she was upgraded to the ICU due to worsening hypoxemia and hemodynamic instability. A CT angiography showed interval development of air within the right hepatic lobe hematoma (Fig. 2A), no evidence of pulmonary embolism, patchy multifocal groundglass consolidations (Fig. 2B), and a small right pneumothorax with chest tube in place. On day 7, she was intubated due to progressive worsening of hypoxemia and increased work of breathing. The ABG at this time showed a pH of 7.46, pCO_2 of 36, pO_2 of 49, HCO_3 of 25.1, and a *P*/Fc ratio of 108. Due to concern for ARDS, the patient was started on a low tidal volume ventilation strategy. A respiratory viral panel obtained was negative. Interventional radiology was consulted, and the right posterior liver abscess was drained, yielding a copious amount of air and ~30 ml of turbid fluid. A wound culture from the liver abscess grew *Veillonella parvula* on a sterile growth agar for anaerobic bacteria. Intravenous vancomycin (15 mg/kg) and piperacillin/tazobactam (4.5 g every 8 h) were initiated. Additionally, Micafungin was initiated for empiric fungal coverage.

The patient had slow improvement over the following days and was extubated on hospital day 14. The ABG on the day of extubation revealed a pH of 7.43, pCO_2 of 40, pO_2 of 86, HCO_3 of 26.0, and a *P*/F ratio of 286. After consultation with our infectious disease specialist, the patient was discharged with the

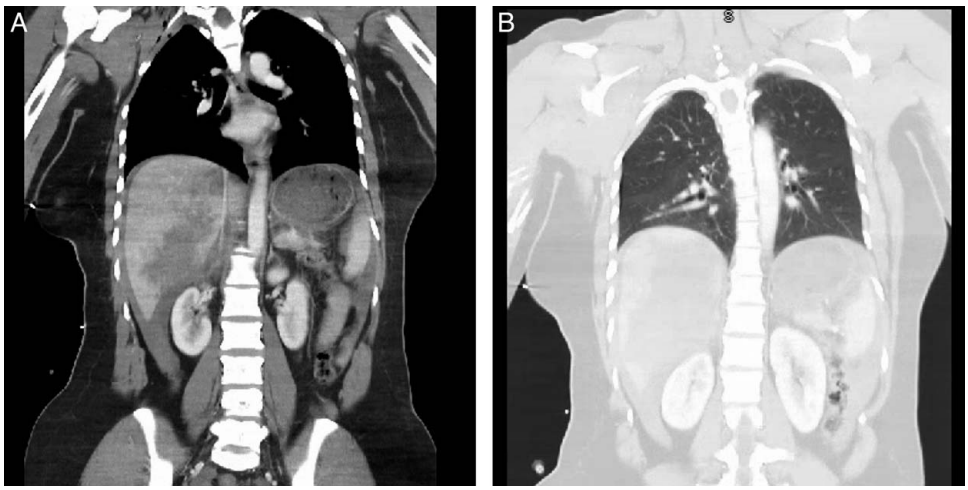

Figure 1. (A) Coronal section of a computed tomography (CT) of the chest, abdomen, and pelvis showing a grade 4 laceration of the posterior aspect of the right lobe of the liver with active extravasation seen along the posterior aspect of the liver. There is free perihepatic fluid, free perisplenic fluid, and moderate fluid in the pelvis. (B) Coronal section of a CT of the chest, abdomen, and pelvis showing a small right pneumothorax and fractures of the right first, second, fourth, fifth and sixth ribs.

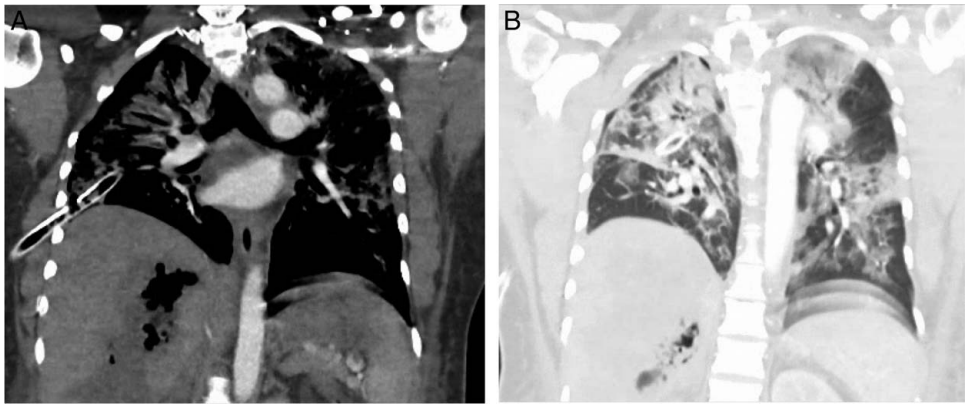

Figure 2. (A) Right hepatic lobe laceration noted with presumed hematoma in the right hepatic lobe posteriorly with multiple scattered foci of air present within the hematoma. (B) Patchy multifocal groundglass consolidation bilaterally with dense central bronchovascular consolidation.

liver drain in place and a 7-day course of oral levofloxacin 750 mg daily and oral metronidazole 500 mg every eight hours for hepatic abscess treatment. A CT of the abdomen with intravenous contrast was obtained on 4-week follow-up demonstrated near resolution of her liver abscess, Figure 3. Two-months after discharged, the pigtail drain was removed and at the 3-month follow-up appointment, the patient was doing well and returned to work.

Discussion

Our case focuses on a *V. parvula* infected liver abscess in an immunocompetent patient that progressed to ARDS. There are reports of co-infection with *Veillonella* in immunosuppressed patients with altered gut and oral microbiome, cystic fibrosis, pulmonary tuberculosis, and SARS-CoV-2, but the opportunistic presence of *Veillonella* is incredibly rare in a patient lacking predisposing factors[21–24]. The mechanism by which our patient acquired a *Veillonella* infection of the liver remains unclear. *Veillonella* infections have been associated with inflammatory and autoimmune liver diseases; however, our patient did not have any history of evidence of these co-morbid conditions[13,14].

To our knowledge, *V. parvula* was not previously reported to be associated with the development of ARDS in an immunocompetent individual. In the setting of ARDS, there is diffuse injury to lung endothelium and epithelium, and hyaline membrane deposition, due to the onset of inflammation and edema[17]. Patients developing ARDS have a dysregulation in the lung's immune defenses, which results in an imbalance of the normal lung microbiome, predominantly *Pseudomonas aeruginosa, Staphylococcus aureus, and Enterobacterales*[17,25,26,27]. While ARDS itself can be precipitated by a multitude of extrapulmonary and intrapulmonary and noninfectious causes, such as sepsis, oxygen toxicity, fat embolism, trauma, aspiration, pancreatitis, and blood transfusions[17], in our case we suspect the triggers were septicemia and trauma. Infections that are typically associated with immunocompromised individuals can occasionally occur in immunocompetent people for a variety of reasons including variability in immune response in immunocompetent individuals, environmental factors, and emerging or changing pathogens[28,29].

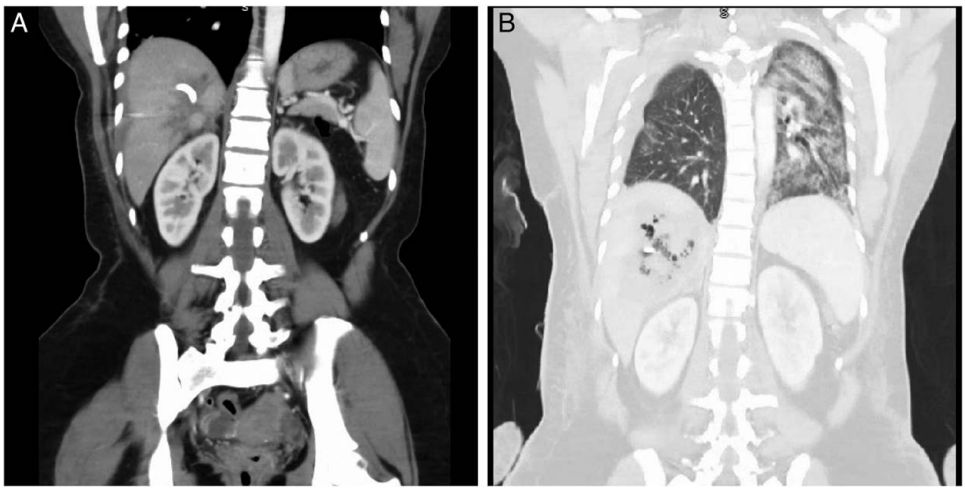

Figure 3. (A) Percutaneous drain within right posterior hepatic lobe with decrease in size of the air and fluid collection. Minimal residual surrounding fluid is seen. (B) Improved lung parenchymal fibrotic tissue.

The treatment protocol for a patient with ARDS followed the standard approach of low tidal volume ventilation strategy and treatment of underlying issues, which we employed for our patient successfully. At the time of this patient's hospitalization, studies supporting the administration of systemic corticosteroids in ARDS were inconsistent; as such, we did not utilize such measures. Previously reported cases of *Veillonella* infections were treated with penicillin, ceftriaxone, and metronidazole[30]. We administered broad-spectrum antibiotics once imaging revealed a liver abscess, and chest imaging showed concern for infection. Following results of cultures and consultation with our infectious disease specialists, we opted to discharge the patient on oral levofloxacin and metronidazole.

Conclusion

This is the first reported case of an immunocompetent individual with a *Veillonella parvula* infected liver abscess associated with ARDS. These clinical findings are unique due to the nature of our patient's ARDS onset and the dearth of similar cases in the literature. The favorable outcome of our patient was due to a multidisciplinary approach. We recommend consistent monitoring and follow-up imaging for the early identification of a *V. parvula* hepatic abscess. We suggest conservative measures and a rapid introduction of ARDS protocol in similar cases. Our findings contribute to future clinicians' management of *V. parvula* and a greater understanding of its disseminating effects and presentation in immunocompetent patients.

Ethical approval

Ethical approval was obtained from our institutional review board (IRB #23-24).

Consent

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

Source of funding

The research presented in this manuscript had no specific funding from any agency in the public, commercial or not-for-profit sectors.

Author contribution

A.M., L.M., B.W.: conceptualization, methodology, writing—original draft. D.C., A.B., A.P., A.D., K.P.: writing—review and editing. All authors read and approved the final manuscript.

Conflicts of interest disclosure

The authors declare there is no conflict of interest.

Research registration unique identifying number (UIN)

Not applicable.

Guarantor

Brandon Woodward.

Data availability statement

Not applicable.

Provenance and peer review

Not commissioned, externally peer-reviewed.

References

[1] Gouze H, Noussair L, Padovano I, *et al*. Veillonella parvula spondylodiscitis. Med Mal Infect 2019;49:54–8.
[2] Matera G, Muto V, Vinci M, *et al*. Receptor recognition of and immune intracellular pathways for Veillonella parvula lipopolysaccharide. Clin Vaccine Immunol 2009;16:1804–9.
[3] Mashima I, Nakazawa F. The interaction between Streptococcus spp. and Veillonella tobetsuensis in the early stages of oral biofilm formation. J Bacteriol 2015;197:2104–11.
[4] Abram AM, Szewczyk MM, Park SG, *et al*. A co-association of Streptococcus mutans and Veillonella parvula/dispar in root caries patients and in vitro biofilms. Infect Immun 2022;90:e0035522.
[5] Ziga M, Gianoli D, Waldeck F, *et al*. Spondylodiscitis due to anaerobic bacteria Veillonella parvula: case report and literature review. Surg Neurol Int 2021;12:496.
[6] Wellens L, Casteels I, Huygens M. Veillonella parvula periorbital cellulitis: an unusual pathogen causing a common clinical sign. GMS Ophthalmol Cases 2019;9:Doc17.
[7] Richards T, Stephen J, Lui CL. Severe disseminated Veillonella parvula infection including endocarditis, bilateral psoas abscess, discitis, and osteomyelitis but sparing spinal and hip prostheses: a case report. J Med Case Rep 2022;16:157.
[8] Bhatti MA, Frank MO. Veillonella parvula meningitis: case report and review of Veillonella infections. Clin Infect Dis 2000;31:839–40.
[9] Singh N, Yu VL. Osteomyelitis due to Veillonella parvula: case report and review. Clin Infect Dis 1992;14:361–3.
[10] Prod'homme M, Pfander G, Pavese P, *et al*. Acromioclavicular septic arthritis caused by Veillonella parvula. Case Rep Orthop 2019;2019:7106252; Published 2019 Nov 27.
[11] Fisher RG, Denison MR. Veillonella parvula bacteremia without an underlying source. J Clin Microbiol 1996;34:3235–6.
[12] Chen YC, Ko PH, Yang CJ, *et al*. Epidural abscess caused by Veillonella parvula: case report and review of the literature. J Microbiol Immunol Infect 2016;49:804–8.
[13] Loomba R, Ling L, Dinh DM, *et al*. The commensal microbe Veillonella as a marker for response to an FGF19 analog in NASH. Hepatology 2021;73:126–43.
[14] Wei Y, Li Y, Yan L, *et al*. Alterations of gut microbiome in autoimmune hepatitis. Gut 2020;69:569–77.
[15] Lichtman SN, Keku J, Clark RL, *et al*. Biliary tract disease in rats with experimental small bowel bacterial overgrowth. Hepatology 1991;13:766–72.
[16] Lichtman SN, Sartor RB, Keku J, *et al*. Hepatic inflammation in rats with experimental small intestinal bacterial overgrowth. Gastroenterology 1990;98:414–23.
[17] Bos LDJ, Ware LB. Acute respiratory distress syndrome: causes, pathophysiology, and phenotypes. Lancet 2022;400:1145–56.
[18] ARDS Definition Task ForceRanieri VM, Rubenfeld GD, *et al*. Acute respiratory distress syndrome: the Berlin Definition. JAMA 2012;307:2526–33.
[19] Rubenfeld GD, Caldwell E, Peabody E, *et al*. Incidence and outcomes of acute lung injury. N Engl J Med 2005;353:1685–93.
[20] Estenssoro E, Dubin A, Laffaire E, *et al*. Incidence, clinical course, and outcome in 217 patients with acute respiratory distress syndrome. Crit Care Med 2002;30:2450–6.
[21] Hu Y, Kang Y, Liu X, *et al*. Distinct lung microbial community states in patients with pulmonary tuberculosis. Sci China Life Sci 2020;63:1522–33.

[22] Vandeplassche E, Sass A, Ostyn L, *et al*. Antibiotic susceptibility of cystic fibrosis lung microbiome members in a multispecies biofilm. Biofilm 2020;2:100031.
[23] Hauser PM, Bernard T, Greub G, *et al*. Microbiota present in cystic fibrosis lungs as revealed by whole genome sequencing. PLoS One 2014; 9:e90934.
[24] Bao L, Zhang C, Dong J, *et al*. Oral microbiome and SARS-CoV-2: beware of lung co-infection. Front Microbiol 2020;11:1840.
[25] Isnard P, Larue M, Pitsch A, *et al*. A Simple Bacterium Links Heart Infection to Inflammatory Liver Disease. Hepatology 2021;74:3549–51.
[26] Matthay MA, Zemans RL, Zimmerman GA, *et al*. Acute respiratory distress syndrome. Nat Rev Dis Primers 2019;5:18.
[27] Luyt CE, Bouadma L, Morris AC, *et al*. Pulmonary infections complicating ARDS. Intensive Care Med 2020;46:2168–83.
[28] Rouientan H, Gilani A, Sarmadian R, *et al*. A rare case of an immunocompetent patient with isolated pulmonary mucormycosis. IDCases 2023;31:e01726.
[29] Goudarzi A, Sarmadian R, Yousefichaijan P, *et al*. Streptococcus Viridans meningitis in an immunocompetent child: a case report. Clin Case Rep 2023;11:e7058.
[30] Li J, Wang H, Li N, *et al*. Antibiotic susceptibility and biofilm-forming ability of Veillonella strains. Anaerobe 2022;78:102667.
[31] Agha RA, Franchi T, Sohrabi C, *et al*. The SCARE 2023 guideline: updating consensus surgical CAse REport (SCARE) guidelines. Int J Surg 2020;84:226–30.