Case Report

Correspondence

Rashid M. Rashid
rashidrashid.mdphd@yahoo.com

Received 14 June 2006
Accepted 14 October 2006

'*Streptococcus milleri*' aortic valve endocarditis and hepatic abscess

Rashid M. Rashid,¹ Wajeeh Salah² and Jorge P. Parada^{2,3}

¹MD PhD Program, Loyola University Chicago, Maywood, IL, USA

²Stritch School of Medicine, Loyola University Chicago, Maywood, IL, USA

³Hines VA Hospital, Hines, IL, USA

Although well-recognized animal pathogens, group C streptococci are relatively rare causes of human infection. The phenotypically small-colony group C '*Streptococcus milleri*' are typically associated with suppurative disease of soft tissue and organs, including liver abscesses, while bacteraemia and endocarditis are distinctly less common. Herein, a case of '*S. milleri*' causing both endocarditis and liver abscess in the same patient is reported.

Introduction

Members of the '*Streptococcus milleri*' group are part of the normal flora of human mucous membranes and are infrequent significant pathogens. Evidence for the pathogenicity of members of the '*S. milleri*' group is patchy and mainly circumstantial. Although they are clearly found in certain suppurative infections, many of these are found to be polymicrobial. Successful treatment of suppurative infections typically requires surgery and broad-spectrum antibiotics. Although case reports of both group C streptococcal endocarditis and liver abscess have appeared in the literature, '*S. milleri*' endocarditis is quite rare, and '*S. milleri*' endocarditis and a hepatic abscess in the same patient is distinctly uncommon.

Herein, we report such a case and review these infections, with a focus on '*S. milleri*' endocarditis. Our literature review was conducted via PubMed using the keywords *Streptococcus milleri*, group C streptococcus, endocarditis, liver abscess, hepatic abscess and streptococcal bacteraemia; findings were restricted to English language journals.

Case report

A 63-year-old male with a past medical history significant for pancreatic cancer, status post uncomplicated Whipple procedure (other than resulting diabetes mellitus) in August 2001, presented to the emergency department in April 2006 with complaints of fevers up to 104°, chills, rigors, weakness and dizziness worsening over 2 weeks. He also reported new onset dyspnoea on exertion, dry cough and dull right upper quadrant pain.

On examination, the patient was ill-appearing, unkempt, and noted to have poor dental health with persistent tooth drainage. He was febrile, tachycardic, hypotensive and had a syncopal episode. After admission to the medical intensive care unit with an initial diagnosis of septic shock, he

stabilized with fluid resuscitation, Levophed pressor support and empiric therapy with azithromycin and piperacillin/tazobactam.

Laboratory findings included marked leukocytosis with left shift (white blood cell count was $23.4 \times 10^9 \text{ l}^{-1}$ with 70 % neutrophils, 29 % bandemia, toxic granules and Döhle bodies present) suggesting sepsis, but not supporting ascending cholangitis as the source (alkaline phosphatase = 106 IU l^{-1} , total bilirubin = 0.6 mg dl^{-1} , alanine aminotransferase = 36 IU l^{-1} and aspartate aminotransferase = 62 IU l^{-1}). Blood cultures revealed a β -haemolytic group C '*S. milleri*' species. An initial 2D cardiac echocardiogram revealed no valvular vegetations, but a transoesophageal echocardiogram showed a small aortic valve vegetation (Fig. 1) and mild aortic insufficiency. A CT scan of the abdomen revealed a large liver abscess in the right hepatic lobe (Fig. 2). Antibiotics were then changed to gentamicin and ampicillin/sulbactam. A percutaneous hepatic drain was placed, and β -haemolytic group C '*S. milleri*' species, viridans streptococcus '*S. milleri*' and *Bacteroides fragilis* were cultured from the fluid. A CT scan of the head revealed no acute findings.

The patient improved, with resolution of fevers, sterilization of blood cultures, and a repeat abdominal CT scan that showed an interval decrease in the size of the patient's liver abscess. A repeat transoesophageal echocardiogram showed no progression of disease and the patient was discharged home on intravenous antibiotics (2 weeks gentamicin and 6 weeks of ampicillin/sulbactam). Post-therapeutic surveillance blood cultures remained negative.

Discussion

Group C streptococci are chain-forming Gram-positive cocci, facultative anaerobes, and are usually β -haemolytic on sheep blood agar, where they produce large (>0.5 mm) or

Fig. 1. Transoesophageal echocardiogram image of a trileaflet aortic valve with thickening at the valve commissures and a mobile echodensity (<7 mm in diameter) attached to the left ventricular outflow tract side of the aortic valve.

small (<0.5 mm) colonies (currently designated '*S. milleri*' or *Streptococcus anginosus* group).

Group C streptococci, especially large-colony strains, are well-recognized pathogens in a variety of animals and birds, but only rarely cause disease in humans. Small-colony group C streptococci are endogenous for humans and are well-recognized skin and mucosal colonizers (Hare, 1940; Rolston, 1986; Gossling, 1988), with the majority of infections in or near areas of known colonization (Gossling, 1988; Duma *et al.*, 1969). When pathogenic or causes of bacteraemia, members of the '*S. milleri*' group are

Fig. 2. CT scan of the abdomen showing a large hepatic abscess. The air-fluid level in the right lobe can be seen.

typically associated with suppurative disease of soft tissue and organs (Molina *et al.*, 1991; Kowlessar *et al.*, 2006). Bacteraemia and endocarditis caused by small-colony group C '*S. milleri*' are distinctly uncommon. One review of >130 cases of endocarditis over nearly a decade found that β -haemolytic streptococci accounted for fewer than 5% of cases (Sandre & Shafran, 1996). A second review of 31 β -haemolytic streptococci endocarditis cases found none were caused by '*S. milleri*' (Stein & Panwalker, 1985).

Incidence estimates for '*S. milleri*' infection are difficult, but have been reported as 0.14/100 000 (Laupland *et al.*, 2006). Typically, patients with group C infection are elderly, frail men (>2:1 male to female cases) with multiple medical comorbidities, poor dental hygiene (Laupland *et al.*, 2006), or are immunocompromised by malignancy, immunosuppressive medication, AIDS or surgery (Gossling, 1988; Vartian *et al.*, 1985; Skogberg *et al.*, 1988; Salata *et al.*, 1989), leading to the characterization of group C streptococci as the cause of opportunistic infections in compromised hosts.

Positive group C streptococcus cultures should prompt further investigation into the source of infection. New and unique '*S. milleri*' infections are sporadically reported, such as a case of '*S. milleri*' epidural abscess in pregnancy (Lampen & Bearman, 2005) and post-scorpion sting '*S. milleri*' endocarditis (Wheatley *et al.*, 2005), or infection as a complication of colonoscopic procedures (Paraskeva *et al.*, 2000). Furthermore, the capacity for rapid transformation into a life-threatening infection (Bala *et al.*, 2006) reinforces the importance of timely and appropriate management of group C streptococcal infections. Treatment of '*S. milleri*' infections can be complicated, with some infections requiring multiple drainage procedures, and producing high mortality rates (Jacobs *et al.*, 1994).

In conclusion, although cases of '*S. milleri*'-associated endocarditis and liver abscess have been previously reported separately, the occurrence of both entities in the same patient is exceptionally rare. This case report may serve as a useful reminder of the importance of timely diagnosis and treatment of sepsis in patients. In addition, our patient typifies sufferers of group C streptococcus infections in that he is an elderly male with multiple comorbid illnesses, perhaps immunocompromised by his prior Whipple procedure for pancreatic cancer, who developed an '*S. milleri*' polymicrobial liver abscess. Atypically, however, he not only also developed bacteraemia, but was found to have endocarditis involving a previously apparently normal native aortic valve. Management included drainage of the abscess and broad antibiotic coverage for his polymicrobial liver abscess, as well as use of synergistic antimicrobial therapy for '*S. milleri*' endocarditis.

References

- Bala, A., Saxena, P. & Konstantinov, I. E. (2006). Transthoracic drainage of large *Streptococcus milleri* liver abscess. *J Thorac Cardiovasc Surg* **131**, 744–745.

- Duma, R. J., Weinberg, A. N., Medrek, J. F. & Kunz, L. J. (1969). Streptococcal infections. *Medicine* **48**, 87–127.
- Gossling, J. (1988). Occurrence and pathogenicity of the *Streptococcus milleri* group. *Rev Infect Dis* **10**, 257–285.
- Hare, R. (1940). Sources of hemolytic streptococcal infections of wounds in war and in civil life. *Lancet* **1**, 109.
- Jacobs, J. A., Pietersen, H. G., Stobberingh, E. E. & Soeters, P. B. (1994). Bacteremia involving the “*Streptococcus milleri*” group: analysis of 19 cases. *Clin Infect Dis* **19**, 704–713.
- Kowlessar, P. I., Connell, N. H., Mitchell, R. D., Elliott, S. & Elliott, T. S. (2006). Management of patients with *Streptococcus milleri* brain abscesses. *J Infect* **52**, 443–450.
- Lampen, R. & Bearman, G. (2005). Epidural abscess caused by *Streptococcus milleri* in a pregnant woman. *BMC Infect Dis* **5**, 100.
- Laupland, K. B., Ross, T., Church, D. L. & Gregson, D. B. (2006). Population-based surveillance of invasive pyogenic streptococcal infection in a large Canadian region. *Clin Microbiol Infect* **12**, 224–230.
- Molina, J. M., Lepout, C., Bure, A., Wolff, M., Michon, C. & Vilde, J. L. (1991). Clinical and bacterial features of infections caused by *Streptococcus milleri*. *Scand J Infect Dis* **23**, 659–666.
- Paraskeva, K. D., Bury, R. W. & Isaacs, P. (2000). *Streptococcus milleri* liver abscesses: an unusual complication after colonoscopic removal of an impacted fish bone. *Gastrointest Endosc* **51**, 357–358.
- Rolston, K. V. I. (1986). Group G streptococcal infections. *Arch Intern Med* **146**, 857.
- Salata, R. A., Lerner, P. I., Shlaes, D. M., Gopalakrishna, K. V. & Wolinsky, E. (1989). Infections due to Lancefield group C streptococci. *Medicine* **68**, 225–239.
- Sandre, R. M. & Shafran, S. D. (1996). Infective endocarditis: review of 135 cases over 9 years. *Clin Infect Dis* **22**, 276–286.
- Skogberg, K., Simonen, H., Renkonen, O. V. & Valtonen, V. V. (1988). Beta-haemolytic group A, B, C and G streptococcal septicemia: a clinical study. *Scand J Infect Dis* **20**, 119–125.
- Stein, D. S. & Panwalker, A. P. (1985). Group C streptococcal endocarditis: case report and review of the literature. *Infection* **13**, 282–285.
- Vartian, C., Lerner, P. I., Shlaes, D. M. & Gopalakrishna, K. V. (1985). Infections due to Lancefield group G streptococci. *Medicine* **64**, 75–88.
- Wheatley, G. H., III, Wait, M. A. & Jessen, M. E. (2005). Infective endocarditis associated with a scorpion sting. *Ann Thorac Surg* **80**, 1489–1490.