Case Report

Streptococcus gallinaceus bacteraemia in an abattoir worker presenting with a febrile illness

Michelle N. D. Balm,^{1,2} Han T. Truong,¹ Anwar S. Choudhary,¹
Geoffrey M. Robinson¹ and Timothy K. Blackmore^{1,2}

Correspondence

Michelle N. D. Balm
michelle.balm@ccdhb.org.nz

Department of Internal Medicine¹ and Department of Microbiology², Capital and Coast District Health Board, Wellington Hospital, Private Bag 7902, 6003 Wellington South, New Zealand

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Streptococcus gallinaceus is a newly described species of viridans streptococci, previously only identified as causing disease in broiler chickens. This organism was recovered in pure culture from blood taken from a New Zealand abattoir worker presenting with a febrile illness. This first report of bacteraemia caused by *S. gallinaceus* in a human may help the understanding of the ecology of this recently described organism.

Introduction

The genus *Streptococcus* includes a wide range of Gram-positive chain-forming catalase-negative cocci. The classification of this genus has been both expanded and redefined over the last two decades, particularly within the viridans group. The introduction of molecular techniques of identification, including sequencing of the 16S rRNA gene, allows more precise identification and classification than methods that rely on phenotypic characteristics (Facklam, 2002). We describe in this report the first known human case of a bacteraemic illness caused by *Streptococcus gallinaceus*, a newly described viridans streptococcus.

Case report

A 60-year-old New Zealand Maori abattoir worker presented to hospital with a 5 day history of general malaise, fever, rigors and headache. There were no localizing symptoms. There was no travel or contact history and he was not on regular medications. The abattoir at which the patient worked processed sheep, cattle, and occasionally goats. His duties involved working with animal offal. His medical history was characterized by chronic asymptomatic hepatitis B infection, and he also had a febrile illness 9 years earlier characterized by persistent pyuria, haematuria and abdominal discomfort. No obvious cause was found at that time, despite urological investigation, but he recovered fully with empiric broad-spectrum antibiotic treatment.

On examination, the patient was febrile to 40.4 °C and he was in atrial fibrillation with a rate of 160–190 min⁻¹. There was no conjunctival suffusion or jaundice. Poor dental hygiene was noted, but no other abnormalities were detected on examination. Laboratory analysis revealed an elevated white blood cell count at $16.9 \times 10^9 \text{ l}^{-1}$ (normal $4.0\text{--}11.0 \times 10^9 \text{ l}^{-1}$) with 84 % neutrophils. The C-reactive protein was elevated (97 mg l⁻¹; normal <3 mg l⁻¹), and the

transaminases were raised to almost twice the upper limit of the normal range. Creatinine, electrolytes, haemoglobin and coagulation screen were all within normal limits. Chest X-ray showed an enlarged heart with no evidence of pneumonia. Abdominal ultrasound revealed multiple mobile gallstones only. Treatment was commenced with ceftriaxone (2 g intravenously once daily) and amoxycillin (1 g intravenously three times daily), because of clinical concerns of endocarditis. However, trans-thoracic and trans-oesophageal echocardiograms subsequently showed no evidence of vegetations. Only one blood culture set was obtained before commencing antibiotics. Despite improvement in his symptoms, the patient's fever did not resolve, and treatment was changed after 5 days to amoxycillin (4 g intravenously three times daily) and gentamicin (240 mg intravenously once daily) with subsequent resolution of his clinical illness. He received antibiotic treatment for a total of 2 weeks and remains well after 6 months of follow up.

Microbiological investigations

Within 24 h, growth was detected from the aerobic bottle of the only set of blood cultures taken prior to the commencement of antibiotics (BD Bactec 9000 system, Becton Dickinson). Gram stain revealed Gram-positive cocci, in pairs and short chains. Small, grey α -haemolytic colonies formed on sheep blood agar after overnight incubation at 37 °C in 5 % CO₂. Phenotypic testing of the isolate with the Rapid ID32 Strep API system (bioMérieux) gave inconsistent results, with unsatisfactory profiles suggesting the possibility of *Streptococcus mitis* (profile 72756441101) or *Streptococcus sanguis* (profile 72746041100). The Vitek 1 GPI card (bioMérieux) identified the isolate as *Streptococcus uberis* with 98 % correlation. This species is described as a viridans streptococcus which causes bovine mastitis, and has not been reported as causing infection in humans (Douglas *et al.*, 2000).

16S rRNA gene sequencing was employed because of the difficulty in identifying what was considered a significant blood culture isolate. DNA was extracted from bacterial cells grown on blood agar using the High Pure Template Preparation kit (Roche). DNA encoding 16S rRNA was amplified by PCR using the universal primers F27 and R1492. Sequencing of the 16S rDNA was then carried out using the BigDye terminator version 3.1 cycle sequencing kit on an ABI 3100 DNA sequencer (Applied Biosystems). The resulting sequence was compared with those available in the GenBank, EMBL and DJB databases using the BLAST program through the National Centre for Biotechnology Information server. The 5' end sequence using primer F27 in the alignments was 99.65 % identical (575/577 nucleotide homology) to the database sequence of *Streptococcus gallinaceus* (accession no. AJ307888.1). The MICs to penicillin and ceftriaxone were 0.016 and 0.064 µg ml⁻¹, respectively, using Etest (AB Biodisk). Using viridans streptococci breakpoints, the organism was susceptible to vancomycin and high level gentamicin with Clinical and Laboratory Standards Institute-approved methods.

Given his risk of occupational exposure, leptospirosis serology was conducted on paired sera by the microscopic agglutination technique (MAT). Initial testing showed an antibody titre of 1 : 1600 for *Leptospira hardjo*. Two convalescent samples at 2 and 5 weeks showed stable levels of 1 : 800. The patient was not tested for Q fever or brucellosis, because these diseases are not known to occur in New Zealand livestock.

Discussion

We consider that infection with *S. gallinaceus* was the likely cause for the patient's febrile illness. This organism was isolated in pure culture from blood prior to antibiotics. It has not previously been reported to our knowledge from humans. Nor has it been identified before in New Zealand; thus it would be an unusual environmental contaminant. It is possible, but unlikely, that the patient's illness was caused by leptospirosis. We postulate that the high and persistently elevated titres are consistent with prior and ongoing occupational exposure to leptospirosis, and not suggestive of acute seroconversion. The patient's occupation as an abattoir worker brought him into contact with cattle and deer offal, recognized sources of *L. hardjo* in New Zealand and Australia. It is possible that the patient's unexplained febrile illness with 'sterile' pyuria 9 years earlier may have been due to leptospirosis. Serological surveys of New Zealand abattoir workers have demonstrated that 1.7–28.7 % have positive leptospirosis serology, and that there is no association between single raised titres and clinical signs of infection. Leptospirosis titres remain elevated for many months or years after exposure (Blackmore & Schollum, 1982; Blackmore *et al.*, 1984).

S. gallinaceus was first described in 2002, when an unknown streptococcal-like organism was isolated in blood taken from

adult broiler chickens with septicaemia. Gross pathology findings in these chickens included hepatosplenomegaly, renomegaly, multiple areas of hepatic and splenic infarction, and valvular endocarditis. *S. gallinaceus* has similarities to other viridans streptococcal species, including *Streptococcus acidominimus*, *Streptococcus ovis* and *Streptococcus suis* (Collins *et al.*, 2002), and phenotypically to species in the *Streptococcus anginosus* group (Facklam, 2002). However, 16S rRNA gene divergence values of greater than 3 % warrant the organism being regarded as a distinct species (Stackebrandt & Goebel, 1994).

A second report in 2004 of a longitudinal study of mortality in flocks of broiler chickens in Denmark found a 29 % prevalence of endocarditis and septicaemia in one flock, of which 85 % was attributable to *S. gallinaceus*. The authors postulated that *S. gallinaceus* represented a commensal organism of the digestive tract that is afforded entry into the circulatory system during periods of concurrent illness (Chadfield *et al.*, 2004). A further study by the same group demonstrated that intravenous inoculation of *S. gallinaceus* into 4-week-old chickens results in septicaemia and endocarditis in the majority of birds (Chadfield *et al.*, 2005). At present, the New Zealand Poultry Board, responsible for screening chickens reared for commercial use in New Zealand, does not screen for *S. gallinaceus*, and this organism has not been identified within New Zealand.

We have shown that *S. gallinaceus* is not correctly identified with API and Vitek biochemical test systems. It is therefore possible that *S. gallinaceus* isolates may have been dismissed as viridans streptococci in the past. This first reported case of human infection occurring in an abattoir worker suggests that the infection may have been acquired from animal contact. The fact that there was no described contact with live poultry raises the possibility that *S. gallinaceus* is present in cattle or sheep in New Zealand. It is possible that *S. gallinaceus* colonizes these species, but unless it were to cause significant animal disease, it is likely to go undetected. Microbial testing of food-producing animals concentrates on recognized zoonotic pathogens.

Finally, this case highlights the usefulness of molecular typing to back up conventional phenotypic testing methods to help identify unusual or atypical isolates. This first report of bacteraemia in a human caused by *S. gallinaceus* may help the understanding of the ecology of this recently described organism.

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