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Late-onset Neonatal Sepsis Due to Hafnia alvei

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Hafnia alvei infections are uncommon, and usually occur in patients with underlying illnesses, mainly adults. The authors describe a rare case of late-onset community-acquired neonatal sepsis in an infant without any underlying disease. The infant was successfully treated with cefotaxime.

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INTRODUCTION

Bacterial sepsis in the neonate is a clinical syndrome characterized by systemic signs of infection and accompanied by bacteraemia in the first month of life (1, 2). Late-onset neonatal sepsis occurs as a multisystemic illness after the first 3 d of life (2). It is much less likely to be associated with a history of obstetric complications. Bacteria responsible for late-onset disease include those acquired from the maternal genital tract, organisms acquired after birth from human contacts, and those from contaminated equipment. Neonates who have been discharged from hospital can also become infected with community-acquired pathogens.

Hafnia alvei is a rare cause of bacteraemia in adults (3). This report describes a rare case of late-onset neonatal sepsis caused by H. alvei. To our knowledge, this is the first report of disease caused by H. alvei in an infant without any underlying disease.

CASE REPORT

An 8-d-old male infant presented to the Emergency Department for evaluation of fever, grunting and poor appetite of 8 h duration. The infant had an uncomplicated vaginal delivery after 38 weeks gestation, and was born 6 h after rupture of membranes. The pregnancy had been uneventful and cultures performed in search of group B streptococci were negative. Birth weight was 3390 g. No prenatal, peripartum or postpartum complications were reported for either patient or mother. On the second day of life he was discharged from the hospital.

On presentation the parents stated that he had felt warm for 8 h and now was irritable and feeding poorly. He had no history of vomiting, diarrhoea, rashes, respiratory distress, or decreased urine output. His body weight was 3120 g. Initial physical examination

was notable for a body temperature of 38.8°C and irritability, but there were no obvious foci for infection.

Laboratory investigations revealed a white blood cell (WBC) count of 14.2×10^3/ml, with 71.5% neutrophils, 20.7% lymphocytes and 7.2% monocytes. The CRP level was 68 mg/l. Red blood cell count, platelet count, serum electrolyte concentrations, capillary blood gas and urinalysis were normal. Lumbar puncture yielded normal cerebrospinal fluid (CSF) with 1 WBC/ml, normal glucose and protein levels and no organisms on Gram-stain. Latex agglutination antigen studies on CSF were negative for group B Streptococcus, Haemophilus influenzae type B, Streptococcus pneumoniae and Neisseria meningitidis groups A, B, C, Y and W135. Urine, CSF and stool cultures were negative. The blood grew H. alvei in a pure culture, which was identified by the WIDER MIC-ID/GN (Soria Melguizo, Madrid, Spain) and RapID 32E systems (bioMerieux Vitek, Hazelwood, MO). The organism was resistant to ampicillin, amoxicillin/clavulanic acid, cefuroxime, cephazolin and cephoxitin but susceptible to aminoglycosides, cefotaxime, ceftazidime, ciprofloxacin, trimethoprim-sulfamethoxazole, imipenen and ticarcillin. Empirical antibiotic therapy was started with i.v. cefotaxime (50 mg/kg every 8 h) and ampicillin (50 mg/kg every 6 h). When the culture results were available, the ampicillin was stopped, and the patient received a 10-d course of cefotaxime. A follow-up blood culture performed after 48 h of antibiotic therapy was negative. The infant′s clinical status improved during the first 24 h. An immunological evaluation (which included serum IgG, IgA, IgE and IgM levels, IgG subclasses, leukocyte immunophenotyping, C3, C4, CH50 and AH50) were within normal range. HIV DNA polymerase chain reaction and HIV enzyme-linked immunosorbent assay antibody test were negative. The child remains well after 17 months of follow-up.

DISCUSSION

H. alvei is a motile, facultatively anaerobic, Gram-negative bacillus belonging to the Enterobacteriaceae family. This organism is part of the human gastrointestinal flora and of environmental habitats such as surface water and food. These bacteria are rarely isolated from human specimens and they are thought to be rarely pathogenic (3). Albert et al. (4) identified this organism as an enteric pathogen in 1991. Since that time, several cases of diarrhoea due to H. alvei have occurred, mainly in children.

Several cases had been reported in which H. alvei was associated with extra-intestinal invasive infections. Cases of septicaemia, endocarditis, meningitis, pneumonia, abscesses and surgical wound infections had been reported (3–7). Extra-intestinal infections are usually nosocomial, although they also may be community-acquired (3, 7). Infections mainly occur in patients with severe underlying illnesses such as neoplasms, acquired immunodeficiency syndrome and organ transplantation (3–5, 7–9). It also occurs in adults with chronic diseases such as diabetes, chronic obstructive pulmonary disease or chronic renal failure. There are other iatrogenic factors associated with bacteraemia and H. alvei, including abdominal surgery, the presence of central venous catheters, endotracheal tubes, urethral catheters and/or the previous administration of antibiotics (3, 7, 9).

The pathogenesis of the invasive disease remains unclear, but some authors have considered the possibility of access to the blood stream through the gastrointestinal tract. It is still unclear whether the low pathogenicity of H. alvei with respect to extra-intestinal infection is caused by the low expression frequency of virulence factors (3, 10).

H. alvei is usually resistant to amoxicillin and first-generation cephalosporins because it has inducible and constitutive beta-lactamases and can develop resistance to second- and third-generation cephalosporins (11). The most active antimicrobials are usually netilmicin, ciprofloxacin, imipenen and aminoglycosides (3, 7, 8, 11), although in 1 study with 8 strains a high sensitivity for cefotaxime and ceftriaxone was noted (7).

Treatment of H. alvei infection on the basis of antimicrobial susceptibility testing results is effective. Treatment with imipenen or a third-generation cephalosporin in combination with an aminoglycoside is recommended for severe cases (3, 7). In this case, given that empirical therapy had started with cefotaxime, it was decide to maintain this as a monotherapy for its susceptibility in the antibiogram (MIC 1 mcg/ml) and for good clinical response.

In conclusion, H. alvei is a rare human pathogen that may be responsible for serious extra-intestinal nosocomial and community-acquired infections. These infections mainly occur in adult patients with underlying illnesses although the possibility of an extra-intestinal infection should be taken into account in infants.

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Prosthetic Valve Endocarditis Caused by Salmonella enteritidis

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Until now, only 12 cases of prosthetic valve endocarditis caused by Salmonella spp. have been reported in the English literature. High complication and mortality rates, a fulminant course and the requirement for early surgical intervention deserve special attention in this kind of infective endocarditis. A new case of Salmonella prosthetic valve endocarditis complicated by sepsis-induced cholestasis and a literature review are presented in this report.

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INTRODUCTION

Foodborne infections caused by non-typhoidal Salmonella continue to be an important problem in many areas of the world (1). During a gastrointestinal illness, bacteraemia occurs in 5% of immunocompetent patients (2). Because Salmonella spp. have a propensity to attach to damaged and atherosclerotic endothelium, 25% of bacteraemic patients over 50 y of age develop endovascular infections, mostly arteritis (3). Fortunately, infective endocarditis (IE) caused by Salmonella spp., with its malignant clinical course and high mortality rate, is rare in the spectrum of salmonella infections (4). Although there have been reports of native valve endocarditis, only 12 reports of prosthetic valve endocarditis caused by Salmonella spp. could be found in the English literature up to now (5–16). This case report, describes a new case of Salmonella prosthetic valve endocarditis in a patient complicated by sepsis-induced cholestasis and a concise review of the relevant literature.

CASE REPORT

A 51-y-old man, previously reported to be in good health, was admitted to the tertiary care centre with a 3 d history of fever, confusion and diarrhoea. He had had rheumatic fever when he was 15 y old and had undergone mitral and aortic valvular replacements with Key Shally and Björk–Shiley prostheses in 1970.

On admission to the hospital his temperature was 39.5°C, pulse 98 beats/min, respiratory rate 30/min and blood pressure 92/55 mmHg. Sclerae were icteric. Cardiac examination revealed an irregular rhythm, metallic clicks, but no murmurs. He was confused with no local neurological signs. Initial laboratory tests yielded the following results: haemoglobin 10.8 g/dl, leucocyte count 14,900/µl (92% polymorphonuclear leucocytes), platelet count 49,000/µl, blood urea nitrogen 54 mg/dl, creatinine 2.9 mg/dl, total bilirubin 8.8 mg/dl, direct bilirubin 5.8 mg/dl, alkaline phosphatase 127 U/l, albumin 3.1 g/dl, alanine aminotransferase 20 IU/l, lactate dehydrogenase 1370 U/l and C-reactive protein 12.9 mg/dl. Anti-human immunodeficiency virus (HIV) antibody was negative. The electrocardiogram showed atrial fibrillation and left ventricular hypertrophy voltage criteria. A chest radiograph showed cardiomegaly with clear lung fields. Computed tomography of the brain was unremarkable. Transthoracic echocardiography (TTE) revealed 10 mm calcified nodular vegetation on the aortic cusp and some degree of aortic insufficiency jet. A transoesophageal echocardiography (TEE) was carried out, which revealed thickening and irregularity on the mitral valve, not recorded by TTE. The APACHE II score was 26 on admission to the medical intensive care unit.

After taking blood cultures, antimicrobial therapy was commenced with intravenous penicillin G and gentamicin. The patient's clinical status was complicated by disseminated intravascular coagulation (DIC) and septic shock in the following days, and he required ventilatory support. Antimicrobial treatment was switched to sulbactam–ampicilline (SAM) and ciprofloxacin, because of clinical deterioration. On the 7th day, multiple blood cultures obtained on admission were reported to be positive for Salmonella enteritidis and SAM was discontinued. In the clinical course, serum bilirubin levels rose up to 50.99 mg/dl with a significant component of conjugated hyperbilirubinaemia (Fig. 1). Serum alanine and aspartate aminotransferase levels were always below 3 times the upper normal limit, and alkaline phosphatase was within the normal range. All viral (hepatitis A, B, C) and autoimmune serological markers were negative. Ultrasonographic examination did not reveal any gallstone or biliary tract dilatation. The clinical status of the patient improved and on the 10th day of hospitalization he was