Case Report

Chryseobacterium meningosepticum as a cause of cellulitis and sepsis in an immunocompetent patient

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Chryseobacterium meningosepticum is a Gram-negative rod with a worldwide distribution. A case of C. meningosepticum cellulitis with severe sepsis and hepatitis in a 36-year-old male patient is described.

Case report

Chryseobacterium meningosepticum is a Gram-negative rod widely distributed throughout the world. This organism is known to cause outbreaks in neonatal intensive care and haemodialysis units in underdeveloped countries (Chiu et al., 2000; Gungor et al., 2003; Arvanitidou et al., 2003). It is a very rare cause of cellulitis, which occurs in 3% of all C. meningosepticum infections described and usually in immunocompromised patients (Bloch et al., 1997). We describe a case of C. meningosepticum cellulitis with severe sepsis and hepatitis. A 36-year-old male patient was admitted with a history of 7 days of myalgia, fever, nausea and vomiting. A right shoulder cellulitis, tachycardia, tachypnoea and hepaticomegaly were the clinical findings on physical examination. Laboratory tests showed leukocytosis, renal failure (creatinine = 2.7 mg dl$^{-1}$ and urea = 103 mg dl$^{-1}$), hepatitis (aspartate aminotransferase = 122 U l$^{-1}$, alanine aminotransferase = 150 U l$^{-1}$, direct bilirubin = 2.8 mg dl$^{-1}$ and alkaline phosphatase = 223 U l$^{-1}$) and metabolic acidosis. A human immunodeficiency virus test was negative. Ceftriaxone was started empirically, and after 48 h Sphingobacterium was identified in two separated blood samples, collected from a peripheral venous site, by an automated bacterial identification system (Vitek 2). Then, ceftriaxone was changed to ciprofloxacin and the clinical condition improved within 72 h. The strain was later identified as C. meningosepticum by a semi-automated system – Walkway (Dade-Behring) – which gave 99.9% certainty. Antibiotic susceptibilities were determined (Microscan): amikacin >32 mg l$^{-1}$ (resistant); ampicillin/sulbactam >16/8 mg l$^{-1}$ (resistant); ciprofloxacin ≤ 1 mg l$^{-1}$ (sensitive); piperacillin/tazobactam = 8/1 mg l$^{-1}$ (sensitive); trimethoprim/sulfamethoxazole ≤ 2/38 mg l$^{-1}$ (sensitive); imipenem ≥ 8 mg l$^{-1}$ (resistant); ceftriaxone >32 mg l$^{-1}$ (resistant).

This case is believed to be the first report of C. meningosepticum with significant hepatic involvement, although 11 cases of soft tissue infections secondary to C. meningosepticum infection have been reported. Cellulitis in this patient was probably caused by continuous traumatic lesion on the skin, since he worked in a free market carrying boxes on his shoulders. Half of the soft tissue infections reported were acquired in the community, and only one had increased liver enzymes, though in this case following necrotizing fasciitis (Lee et al., 2006).

The result of an automated bacterial identification system should be observed with caution, especially when the patient does not improve with broad-spectrum antibiotic therapy, because several bacteria, including Aeromonas salmonicida and Sphingobacterium spp., may be confused with C. meningosepticum (Chiu et al., 2000). The change in the therapy guided by the susceptibility test was fundamental in the management of this patient. A blind change of antibiotic would be to a carbapenem, but this organism is usually multiresistant to antibiotics typically prescribed for treating Gram-negative bacterial infections, including extended-spectrum β-lactam agents, aminoglycosides and imipenem (Kirby et al., 2004). This case also raises the possibility of the acquisition of the infection as a result of occupational exposure, which is a cause for concern.

We would like to conclude by drawing attention to the risk of this organism as a cause in cases of cellulitis, sepsis and other kinds of infection, because while a large number of drugs have no efficacy against this organism, appropriate therapy showed excellent results.

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References


