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 hepato-megaly 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 (aspart-ame 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.

**Acknowledgements**

This study was presented at the 5th Congresso Paulista de Infectologia, Campinas, São Paulo, Brazil.
References


