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CASE REPORT

FATAL SEPTICEMIA CAUSED BY *Chromobacterium violaceum* IN A CHILD FROM COLOMBIA

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SUMMARY

A 4-year old child living in Colombia presented with a history of fever and severe abdominal pain for four days. The patient developed pneumonia, septic shock, multiple organ failure and died on the fifth day of hospitalization. *Chromobacterium violaceum* was isolated from admission blood cultures and was resistant to ampicillin, cephalosporins, carbapenems and aminoglycosides.

KEYWORDS: *Chromobacterium violaceum;* Pneumonia; Fatal septicemia.

INTRODUCTION

Chromobacterium violaceum is a saprophytic bacterium found mainly in tropical and subtropical climates. Despite its ubiquitous distribution, human infection with this organism is rare. Since the first human case was described in Malaysia in 1927, there have been fewer than 150 human cases reported worldwide, mainly in Asia, the United States, Australia, and Africa[9,12]. Only four cases have been reported in South America, one in Argentina[4] and three in Brazil[7,10,13]. Sepsis with this organism is often found with concomitant immune deficiency disorders, most frequently chronic granulomatous disease (CGD)[5,14].

Human infection with this organism can result in severe, systemic disease with a high fatality rate. *C. violaceum* infection may rapidly progress to sepsis with multiple organ abscesses, predominantly in lungs, liver, and spleen[9,12]. We report here, to the best of our knowledge, the first Colombian case of this infection.

CASE REPORT

A 4-year old girl from rural area east Cordoba, Colombia was admitted at the Clinica Monteria on September 1, 2006, presenting with a history of fever, anorexia and abdominal pain for four days. The pain was intense, continuous and localized in the upper part of the abdomen. The patient denied nausea, vomiting or diarrhea. Physical examination revealed palor, toxemia and the following vital signs: heart rate 126 bpm, respiratory rate 40/minute, blood pressure 83/57 mmHg, and temperature 37.2 ºC. Chest examination revealed the presence of wheezing. The abdomen was tense and distended, with pain in the epigastrium and right flank upon palpation. No enlarged organs were detected.

Laboratory investigation upon admission showed: hemoglobin 7.0 g/dL, leukocyte count 5,400/mm^3, with 10% band neutrophils, 62% segmented neutrophils, 24% lymphocytes, and 4% eosinophils, platelet count 79,800/mm^3, serum creatinine 1.4 mg/dL, blood urea 98.0 mg/dL, and arterial gases: pH 6.92, pO_2 56.2 mmHg, pCO_2 54 mmHg, HCO_3 15 meq/L and O_2 saturation 100%. Blood sodium, potassium, calcium and amylase levels were normal.

A chest radiograph revealed bilateral lobar pneumonia. Abdominal ultrasound was normal without hypoechoic areas in the liver.

The patient was treated empirically with ceftriaxone and amikacin, and intravenous administration of electrolytes and fluids. During the subsequent hours the patient became comatose, with cyanosis of the extremities and circulatory shock that did not respond to vasoactive drugs or mechanical ventilation. The patient died on the fifth day after admission; autopsy was not performed.

A gram-negative bacillus that formed smooth, violaceous colonies on blood agar was isolated from two blood culture collected on admission (Fig. 1). The bacillus was identified as *C. violaceum* by the Microscan system (Dade-Behring). The isolated strain was susceptible to chloramphenicol, tetracycline, trimethoprim-sulfamethoxazole, and ciprofloxacin, and resistant to ampicillin, cephalothin, cefoxitin, ceftriaxone, cefotaxime, ceftazidime, aztreonam, imipenem, amikacin, gentamicin and tobramicin. Therapy was changed to ciprofloxacin when identification and susceptibility became available on the fourth hospital day. However, despite the use of ciprofloxacin, clinical improvement was not observed and the patient died on the fifth hospital day.

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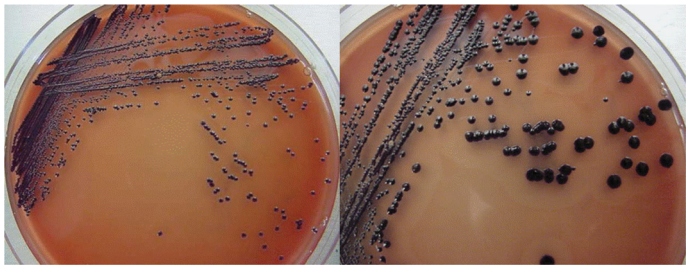

Fig. 1 - Violaceus colonies of *Chromobacterium violaceum* on a blood agar plate.

DISCUSSION

C. violaceum, a motile and facultatively anaerobic gram-negative bacillus, is a common inhabitant of soil and water in tropical and subtropical areas of the world. It rarely causes invasive disease in humans. Localized infection with regional lymphadenopathy occurs after contamination of damaged skin exposed to soil or environmental water. Systemic infection can occur following the aspiration or ingestion of contaminated water[2]. When cultured, the organism produces large, smooth, convex colonies with a violet-black color, although nonpigmented strains do occur[1,2].

Most cases of infection by *C. violaceum* have been reported from Australia, Asia, India, Argentina, Brazil and the United States. Systemic infection caused by *C. violaceum* is rare but severe and is associated with fatality rates ≥ 60%[3,8,12,13]. Previous reports of *C. violaceum* sepsis have noted fever, hepatic abscesses, skin lesions, facial cellulitis and otitis[1]. Report of brain abscesses secondary to *C. violaceum* sepsis have also been reported[9,15].

Predisposition to *C. violaceum* infection occurs in patients with underlying defects in host defenses, particularly CGD[5,14] and G6PD deficiency[6,11]. The patient reported here had no history of previous manifestations of abnormality in leukocyte function or immunodeficiency disease and apparently was previously healthy. It was not possible obtain a family history of abnormalities in leukocyte function or immunodeficiency diseases.

The patient was in daily contact with the rural environment (river, stagnant water) but did not present with skin lesions when admitted to the Clinica. However, bilateral lobar pneumonia was present on admission, suggesting inhalation as the point of entry of the infection, with progression to septicemia. Her initial symptoms were fever and abdominal pain, with the presence of toxemia, circulatory shock and respiratory insufficiency. The fulminant septicemia was probably a consequence of the lipopolysaccharide endotoxin of the organism.

The virulence of *C. violaceum* may have a significant role in the development of infection, and it is attributed partly to endotoxin and partly to inadequate host defenses[9]. If *C. violaceum* infection is identified, therapy with trimethoprim-sulfamethoxazole and a fluoroquinolone should be administered. Long-term treatment is needed to fully eradicate the organism and resolve potentially fatal abscesses in the liver or brain. Our patient died after being treated with an antibiotic regimen that initially included ceftriaxone and amikacin, and later ciprofloxacin, when susceptibility became available. Furthermore, the use of carbapenems or aminoglycosides would not have been useful in this patient due to resistance of this species to these agents.

As in other studies, the *C. violaceum* isolated from our patient was resistant to ampicillin, cephalosporins and aztreonam. It was susceptible to chloramphenicol, tetracycline, ciprofloxacin and trimethoprim-sulfamethoxazole; the latter two antibiotics have been recommended for the treatment of patients affected by *C. violaceum*. This report documents the first case of *C. violaceum* in Colombia, and the possibility of this species should be considered in patients presenting with septicemia in Colombia, with empiric therapy broadened to include this pathogen.

RESUMEN

Septicemia mortal causada por *Chromobacterium violaceum* en una paciente pediátrica de Colombia

Una niña de 4 años que vivía en Colombia presentó historia de fiebre y dolor abdominal severo por cuatro días. La paciente desarrollo neumonía, shock séptico, múltiple falla de órganos y muerte el quinto día de hospitalización. *Chromobacterium violaceum* fue aislado de cultivos de sangre y mostró resistencia a ampicilina, cefalosporinas, carbapenems y aminoglicosidos.

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