## Sepsis by Chromobacterium violaceum: First Case Report from Colombia

Julio Alexander Díaz Pérez<sup>1</sup>, Jorge García<sup>1</sup> and Laura Andrea Rodriguez Villamizar<sup>2</sup>

<sup>1</sup>Militar Hospital Nororiental, Bucaramanga; <sup>2</sup>Center of Public Health of Santander; Bucaramanga, Santander, Colombia

*Chromobacterium violaceum* is found in tropical and subtropical regions; it is the only *Chromobacterium* species pathogenic for humans. Due to its rare presentation, physicians often ignore the importance of this pathogen. We report a fulminant fatal case of bacteremia in a 38-year-old Colombian man. The clinical manifestations were fever, thoracic pain, respiratory failure and death. His condition, from the beginning of clinical diagnosis, went into continuous deterioration, till his death, within a few days after the symptoms began. Two hemocultures isolated *C. violaceum*. We conclude that doctors should consider this differential diagnosis in patients with systemic inflammatory response syndrome, with continuous deterioration. Kev-Words: *Chromobacterium violaceum*, bacteremia.

Chromobacterium violaceum is a Gram-negative bacillus that produces a purple pigment [1]. It was first described by Wooley in water buffalos, as a potential pathogen in the Philippines in 1905 [2], and in humans by J.E. Lesslar in 1927. Some reports have described human infection by this agent, though this is not frequent. This pathogen typically invades its host through the oropharynx or through skin lesions; it may develop into septicemia [2]. Chromobacterium violaceum is found in tropical and subtropical regions; it survives in temperatures over 4°C, but its ideal temperature is between 20 and 37°C. This bacterium survives wet and dry conditions. The incubation time may vary from 3 to 14 days, depending on the type of exposition. Approximately 60 cases of infection by C. violaceum have been reported worldwide in the literature [4-6]. This pathogen had never been described in Colombia. We report a case of a patient with bacteremia by C. violaceum with a fulminant clinical course.

## **Case Report**

The case was a 38 year-old male, an active member of the Colombian army, who consulted at his dispensary unit on May 16, 2006, reporting two days of general discomfort and fever, without vomiting or diarrhea. The laboratory tests reported: Hemogram (10,400leucocytes, 86% neutrophils, platelet count 163,800/mm<sup>3</sup>, 13% lymphocytes, hematocrit 45%); blood smear for Malaria negative; and urinary test for acetones (5 mg/dL). Treatment was initiated with 1 g quarter bid acetaminophen, 1 vial dipyrone, and 500 mg ascorbic acid three times a day, intravenous crystalloids and abundant oral liquids. On May 17<sup>th</sup> the symptoms persisted and consequently new laboratory tests were made, with no relevant changes in values. The patient was transferred to a reference hospital, with a diagnosis of Classic Dengue fever *versus* Typhoid fever. As an important antecedent, he reported eight Malaria

episodes; at physical examination CR 82, BP 80/60, afebrile, congestive oropharynx, hypoventilation in the left hemi thorax, with stertors, no acute abdomen, no meningeal signs, and no petequiae. Laboratory tests and chest X-rays were made, and management with intravenous crystalloids and analgesics was initiated.

Afterwards, management with 200 mg of Ciprofloxacin, IV every 12 hours, was prescribed, along with 500 mg acetaminophen every four hours, and MNB (SSN+Berodual) every six hours. Subsequently, new laboratory tests reported 3.900 leucocytes with 74% neutrophils, 3% bands, and 121,000 platelets. Chest X-rays showed mixed infiltrations in both lungs, with some tendency to consolidation in the upper left side, without pleural effusion. A few hours later, the patient began respiratory distress, with a respiratory rate of 40/min, tachycardia and low oxygen saturation. Continuous respiratory deterioration followed, even though additional management was initiated with venturi oxygen at 20% 10 Lt, and MNB every four hours. Pulmonary-focus sepsis was considered, along with acute respiratory distress and leftside pneumonia. Inotropic management was started with dopamine (4mcg/Kg/min), ceftriaxone (2g), and oxacillin (2g), and transfer to an intensive care unit was ordered. The patient entered the ICU in a very bad general condition, in septic shock, with severe respiratory insufficiency. An airway was maintained by orotraqueal intubation. A negative HIV test was reported, and hemocultivation of peripheral blood informed C. violaceum, with antibiograms showing sensibility to imipenem, meropenem, and tazobactam-piperacillin. Resistance was found to gentamicin, aztreonam, amikacin, ceftriaxone, cefepime, cefotaxime, ceftacidime, ciprofloxacin and piperacillin.

## Discussion

*Chromobacterium violaceum* is confined to tropical and subtropical areas. In Colombia, no cases had been reported in the main databases (Medline, Latindex and Scielo). The Chromobacteria are mobile, facultative anaerobes, Gramnegatives, and catalase positive. These organisms produce an alcohol-soluble purple pigment, non soluble in water, and they grow in ordinary culture media, including McConkey

Received on 18 March 2007; revised 06 July 2007.

Address for correspondence: Dr. Julio Alexander Díaz. Departamento de Patología, Facultad de Salud, Universidad Industrial de Santander, Cra. 32 № 29 – 31. E-mail: pat\_uis@yahoo.com.

The Brazilian Journal of Infectious Diseases2007;11(4):441-442.© 2007 by The Brazilian Journal of Infectious Diseases and ContextoPublishing. All rights reserved.

agar. Species that do not produce the purple pigment have also been found to be pathogenic in humans, and consequently there it has been established that pathogenicity does not depend on pigment production [7]. Colombia has extensive areas of tropical and subtropical forests, which offer good conditions for chromobacteriosis.

Most infections by C. violaceum occur from June to September [7]. This bacillus usually enters humans through a minor skin trauma or through the ingestion of contaminated water. Huffam et al. [8] reported four cases in tropical northern Australia. Two other cases of infection by C. violaceum were reported from a subtropical region of Korea [9]. Twenty-three cases have been reported from the United States, including nine cases related to ingestion of contaminated water [10], four cases developed after trauma [11-13], and one case developed after breast surgery [14]. Three cases were related to swimming in contaminated water [15,16], and the other cases were not explained [15]. Predisposing factors have not been identified [6-8]. Robert et al. [9] reported a case of a traveler who acquired a cervical invasive infection by C. violaceum through the ingestion of contaminated water or through a skin lesion.

The clinical symptoms of an infection by *C. violaceum* include sepsis, liver, kidney or lung abscesses, other presentations with cellulites at a trauma site, urinary tract infection, lymphadenitis, osteomyelitis, sinusitis and meningitis. Sepsis is the most common manifestation of this infection, followed by lymphadenitis and hepatic abscess. Nine of the reported cases had pneumonia or pulmonary abscesses, two had diarrhea and the other two had a urinary tract infection. All cases with cutaneous manifestations were associated with sepsis [9-15].

Chromobacterium violaceum infection is a serious medical condition that needs emergency medical attention and surgical treatment, and it should be considered in cases of Systemic Inflammatory Response Syndrome in patients that were previously healthy, but with progressive impairment [9-15]. This organism is usually sensitive to chloramphenicol, imipenem, gentamicin, and ciprofloxacin, but it is resistant to  $\beta$ -lactams. Prognosis after establishment of the infection is

severe, with a high mortality rate, over 65% in focalized cases and 80% in bacteremia cases [15]. Our patient died 76 hours after the beginning of symptoms, with a fast progressive and fatal infection.

## References

- Sneath P.H.A., Auckland F.E. The serology and pathogenicity of the genus *Chromobacterium*. J Gen Microbiol 1953;265:276-7.
- 2. Wooley P.G. *Bacillus Violaceous manilea* (a pathogenic organism). Bull Johns Hopkins Hosp **1905**;16:89.
- Johnsos W.M., Disalvo A.F., Steuer R.R. Fatal Chromobacterium violaceum septicemia. Am J Clin Pathol 1971;56:400-6.
- National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing: eighth information supplement. NCCLS document M100-S8. Villanova, PA: National Committee for Clinical Laboratory Standads; 1998.
- Chou Y.L., Yang P.Y., Huang C.C., et al. Fatal and non fatal chromobacterial septicemia: report of two cases. Chang Gung Med J 2000;23:492-7.
- Wu S.H. Fatal chomobacterium septicemia. Chinese J Microbiol Immunol 1986;19:65-70.
- Miller D.P., Blevins W.T., Steele D.B., Stowers M.D. A comparative study of virulent and avirulent strains of *Chomobacterium* violaceum. Can J Microbiol 1988;34:249-55.
- Huffam S.E., Nowotny M.J., Vurrie B.J. Chromobacterium violaceum in tropical northern Australia. Med J Australia 1998;168:335-7.
- Lee J., Kim J.S., Nahm C.H., et al. Two cases of *Chromobacterium* violaceum infection after injury in a subtropical region. J Clin Microbiol **1999**;37:2068-70.
- Black M.E., Shehan J. Bacillus violaceum infection in a human being. JAMA 1938;110:1270-1.
- Simo F., Reuman P.D., Martinez F.J., Ayuub E.M. *Chromobacterium violaceum* as a cause of periorbital cellulitis. Pediatr Infect Dis 1984;3:561-3.
- Schattenberg H.J., Harris W.H. A definition and unique occurrence of rapid fatal infection caused by bacillus violaceous manilae. JAMA **1941**;117:2069-72.
- Felman R.B., Stern G.A., Hood C.I. *Chromobacterium violaceum* infection of the eye: a report of two cases. Arch Ophthalmol 1984;102:7111-3.
- Victoria B., Baer H., Ayoub E.M. Successful treatment of systemic *Chromobacterium violaceum* infection. JAMA 1974;230:578-80.
- Starr A.J., Cribbert L.S., Poklepovic J., et al. *Chromobacterium* violaceum presenting as a surgical emergency. South Med J 1981;74:1137-9.