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INTERNATIONAL ARCHIVES OF MEDICINE
SECTION: INFECTIOUS DISEASES
ISSN: 1755-7682

2015
Vol. 8 No. 92
doi: 10.3823/1691

Chromobacterium Violaceum Sepsis in Minas Gerais

CASE REPORT

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Abstract

Infection by *Chromobacterium violaceum*, known as chromobacteriosis, is a rare cause of severe sepsis with rapid spread and high mortality. In February 2009, a 14-year-old male patient was admitted to Santa Casa of Misericórdia of Juiz de Fora Hospital, and he deteriorated rapidly with severe sepsis and death by chromobacteriosis. In this article we discuss the main clinical findings, sensitivity profile, and antimicrobial therapy of this case.

Keywords
Chromobacterium violaceum, sepsis.

Introduction

The *Chromobacterium violaceum* is a bacillus or coco-bacillus-shaped bacterium which is Gram-negative, facultative anaerobic, and usually lives in water or soil in tropical and subtropical regions [1-5] The most striking feature of this type of bacteria is the production of a pigment called violacein, colouring the colonies violet. However, 9% of the isolated strains do not have pigments [1].

The first description of *Chromobacterium violaceum* occurred in the Philippines in 1905 [3, 6-8], and the first report of human infection occurred in 1927 in Malaysia [1, 3, 7] and the Southeast Asian region has the highest number of cases reported [2, 3]. In Brazil the first case was reported in 1984 and occurred in the Rio Negro region, in the Amazon [3]. There have been an estimated 150 cases worldwide and 19 cases in Brazil [1] in total. The infection caused by this agent rarely affects humans, can occur in healthy or immunocompromised individuals [1, 4, 5], and is known as chromobacteriosis. It is characterized by rapid spread, with severe sepsis and high mortality [2, 3, 9]. Transmission usually occurs through contact of open wounds with contaminated water [1, 3, 8].

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This article describes the occurrence of chromobacteriosis in a 14-year-old male patient, from the municipality of Simão Pereira, state of Minas Gerais, Brazil. The patient was transferred in to Santa Casa of Misericórdia of Juiz de Fora Hospital, a regional reference hospital, where he was seen by the Infectious Diseases service. The clinical course and identification of the infectious agent also took place at this hospital.

Clinical Case

Male patient, 14 years old, mixed race, student, born in the city of Juiz de Fora and resident of Simão Pereira, Minas Gerais, was referred to Santa Casa of Misericórdia of Juiz de Fora Hospital. He was admitted in February 2009, 1:00 am, with reports of having suffered blunt trauma injury in the anterior region of the right leg two weeks before. The lesion developed signs of inflammation associated with a fever of 39°C and the appearance of large-volume tumor on the right inguinal region, without hyperemia, increased temperature, or local pain. With this condition he was hospitalized in his hometown and received intravenous antibiotic treatment with ceftriaxone. Still unresolved a few days later, he was admitted by our service. He denied previous diseases or allergies. He reported exposure to flood waters, during which the said injury occurred on his right leg.

The referral documents included only a complete blood count with leukocytosis (predominance of neutrophils without deviation for juvenile forms) and urinalysis with pyuria of 10 pus cells per field, without other changes. No other report or testing was included.

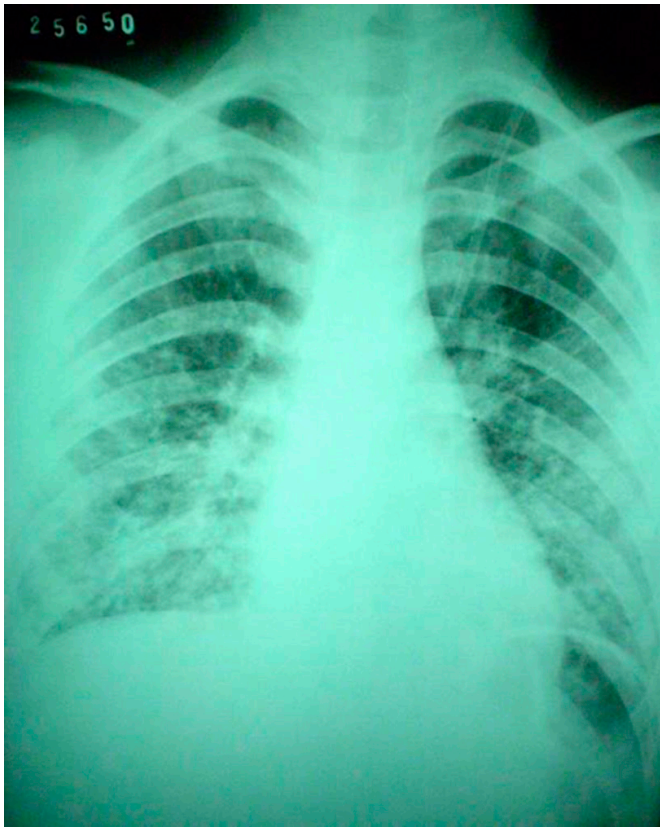
During the physical examination on admission, the patient had a Glasgow score of 15, feverish with axillary temperature of 38.5°C. Cardiovascular system with regular heart rhythm without murmurs on auscultation, blood pressure of 110 x 60 mmHg, heart rate of 96 beats per minute and no pathological jugular venous distension. Auscultation revealed no adventitious sounds, breath sounds were globally distributed, respiratory rate of 20 breaths per minute and absence of respiratory effort. The abdomen was normal to inspection and palpation, without visceral enlargement and with physiological bowel sounds. Neurological examination without abnormalities in consciousness level, balance, strength and muscle tone, sensitivity or motor function, with no signs of meningeal irritation. There was presence of inguinal lymphadenopathy without signs of inflammation. On the lower right leg there was a blunt injury on the anterior region with flushing, increased temperature and secretion of small amounts of pus.

At 6:00 am, the patient developed chest pain in precordium, beginning 20 minutes before, with an oppressive and ventilator-dependent aspect resistant to conventional analgesia. On this occasion he presented tachycardia with a heart rate of 120 beats per minute, slightly hypotensive with a blood pressure of 85 x 60 mmHg. Resting electrocardiogram was performed, without changes in addition to sinus tachycardia; respiratory rate of 20 breaths per minute. Volume expansion was started, with crystalloid, intravenous antibiotic therapy was prescribed (oxacillin and ceftriaxone), and further testing was requested.

Hours later, at 8:00 am, the patient developed dyspnea and cyanosis, with persisting hypotension (80 x 40 mmHg), tachycardia, and now with tachypnea (respiratory rate of 32 breaths per minute). He was then transferred to intensive care.

Initial testing showed pancytopenia with: hemoglobin 10.6 mg/dl, leukopenia 300 cells/mm^3 and platelets of 80,000/mm^3. Ureia, serum creatinine, sodium, and potassium were normal, d-dimer was high, and serum lactate was also very high at 15,9 mg/dl. Testing revealed metabolic acidosis and hypoxemia. Chest x-ray **(Figure 1)** with small scattered nodules in both pleuropulmonary fields without pleural effusion and with preserved cardiac area. Echocardiography without valvular or flow altera-

Figure 1: Chest X-ray with scattered small nodules in both pleuropulmonary fields without pleural effusion and with preserved cardiac area.



tions on Doppler, normal pericardium, left ventricular volumes within normal limits, and a reduced ejection fraction of 46.77%. Inguinal ultrasound showed the presence of enlarged lymph nodes (4 x 2 cm), with areas of liquefaction. Blood for culture was also collected.

The patient was submitted to tracheal intubation and mechanical ventilation, vasoactive drugs were started, maintaining the strong volume expansion with crystalloids; intravenous corticosteroids were started and antimicrobial treatment was altered, with cefepime added to oxacillin. He remained hypotensive, tachycardic and hypoxic despite the initial measures. He had a white blood cell count of 900 cells/mm^3 even after administration of neutrophil colonies stimulator. A biopsy of inguinal lymph nodes was performed.

At 5:00 pm he suffered his first cardiac arrest, with successful resuscitation, and at 7:00 pm the patient died.

A few days later the results of the lymph node biopsy and blood culture were made available. The biopsy showed no microscopic abnormalities and lymph node culture was negative. Blood culture grew *Chromobacterium violaceum*. The bacteria was sensitive to ciprofloxacin, levofloxacin and sulfamethoxazole-trimethoprim, had intermediate resistance to amikacin, gentamicin, tobramycin, and resistance to aztreonam, imipenem, ceftazidime, cefepime, ceftriaxone and piperacillin-tazobactam.

Discussion

The clinical condition presented by this patient was characterized by the quick evolution of a febrile syndrome into severe sepsis and death. Upon admission at Santa Casa of Misericórdia of Juiz de Fora Hospital, the patient was initially suspected of having leptospirosis due to exposure to flood waters, which is also the vehicle of infection for chromobacteriosis [1, 3]. After initial laboratory testing, additional diagnostic hypotheses were staphylococcal sepsis evolving with severe neutropenia and lymphoproliferative syndrome with the presence of lymph node enlargement, presenting with leukopenia, which would be a characteristic of a serious and sudden sepsis in the context of neutropenia. These diagnoses were assessed through Doppler echocardiography, blood culture and biopsy of inguinal lymph nodes, and general laboratory testing and chest X-ray. The etiologic diagnosis was established only after the isolation of *Chromobacterium violaceum* in blood culture days after the patient's death.

Chromobacterium violaceum is not a demanding bacteria type, easily growing in standard culture media (1:10). Its violet coloration is due to the presence of the violacein pigment [1, 3, 7] which has commercial and industrial importance due to its an-

tibacterial, anti-parasitic, and anti-tumor properties, and its use in the manufacture of biosurfactants [1, 11].

The clinical manifestations of chromobacteriosis begin after the incubation period ranging from 3 to 14 days, according to the form of exposure to the agent [1, 8]. It includes inflammatory signs at the entry site, associated with fever, nausea, vomiting, abdominal pain [1, 3, 4] with rapid evolution to sepsis, rapid spread and high mortality [2, 3, 12, 13]. There is formation of multiple abscesses in the lung, liver, spleen, brain (more rarely), as well as cellulite, lymphadenitis and osteomyelitis [1-3, 6, 7, 12, 14]. Other symptoms include conjunctivitis, diarrhea, urinary tract infections, sinusitis and meningitis [5, 8, 10].

Some patients may have persistent micro abscesses and septic foci after treatment, specially those who did not follow adequate treatment [4].

Laboratory findings are nonspecific, and there are changes in the blood count, such as leukocytosis, leukopenia and left shift. Definitive diagnosis of infection is done by culture and subsequent biochemical testing [4, 10]. There are no serological tests for this indication [4].

Antimicrobial therapy against chromobacteriosis may be difficult to select, since *Chromobacterium violaceum* is resistant to most agents used in the medical practice, such as penicillins and cephalosporins [1].

The mechanisms involved in bacterial resistance to beta-lactam agents are: presence of beta-lactamases and absence of penicillins receptors [12]. The presence of efflux pumps is a mechanism present in multidrug-resistant strains of bacteria, and are likely responsible for resistance to tetracycline, erythromycin and sulfadiazine [12].

Chromobacterium violaceum is usually sensitive in vitro to fluoroquinolones, specifically to ciprofloxacin, carbapenems, aminoglycosides, piperacillin, trimethoprim-sulfamethoxazole, ceftazidime and cefepime [1, 9, 10, 12, 15-17]. Resistance to cephalosporins is variable, and it is common to identify strains resistant to ceftazidime and cefepime [1, 15, 17], such as the one isolated in our case report. Also as a general rule, the microorganism is resistant to ampicillin, amoxicillin, ceftriaxone, cephalexin, rifampin, tetracycline, azithromycin and erythromycin [1, 7, 12, 15-18], and may have intermediate resistance to norfloxacin, polymyxin B and cefoxitin [15]. The strain isolated in our case was resistant or intermediately resistant to antibiotics that are usually effective against *Chromobacterium violaceum*, such as imipenem, piperacillin, aminoglycosides, ceftazidime and cefepim, as seen in other reports [18].

The use of ciprofloxacin, a fluoroquinolone against which no resistance has been observed, seems to be a good therapeutic option, and it is also an antibiotic of widespread use and accepteable cost. [4, 5, 10].

Conclusion

Chromobacteriosis is an unusual disease in which infection occurs through contact of an open wound with contaminated water. It has high mortality and rapid spread, and an evolution of severe sepsis. There is presence of multiple abscesses in various sites such as the lung and liver, and lymphatic dissemination and phlogosis at the inoculation site.

Evaluating the antimicrobial susceptibility profile of *Chromobacterium violaceum* in several case reports, the authors observed that there is a need to better define the most appropriate treatment, since the different isolated strains show different profiles, however, as a general rule, ciprofloxacin seems to be an efficacious choice.

Conflict of interest

The authors declare that they have no conflict of interest.

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