**Chromobacterium violaceum Infection**

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**ABSTRACT:** Chromobacterium violaceum infection is confined to the tropical and subtropical areas, with almost all reported cases occurring in the Southeast. The most common feature of this infection is sepsis, followed by cutaneous involvement and liver abscesses. Treatment consists of surgical drainage of purulent collections and appropriate antimicrobial therapy, such as chloramphenicol, gentamicin, imipenem, trimethoprim-sulfamethoxazole, or ciprofloxacin. Although C violaceum infection is rare, it is potentially fatal and remains an important entity for clinicians to suspect and treat appropriately.

**Chromobacterium violaceum** is a saprophyte of soil and water and generally considered non-pathogenic. The first report of its pathogenicity was described by Wooley1 in 1905 when he reported this organism as the cause of fetal infection in water buffaloes in the Philippines. The first human case occurred in Malaya in 1930. Since then, several reports described human infection with this organism, mostly in the southeastern United States. We report the successful treatment of C violaceum infection in a pediatric patient and discuss the importance of this unusual organism as a human pathogen, including the possible role of leukocyte dysfunction as a host factor.

**CASE REPORT**

An 11-year-old previously healthy boy was admitted to the hospital in September 1994 with fever, vomiting, diarrhea, abdominal pain, and a 6 kg weight loss.

This adolescent had been in his usual state of health until 3 weeks before admission, when he had persistent high-grade fever and recurrent emesis not containing blood or bile. Abdominal pain was diffuse over the upper abdomen. The diarrhea was watery with a frequency of four to six stools a day. He lived in Panama City, Florida, which was affected by flooding several weeks before the onset of the symptoms.

At the time of initial evaluation, the patient was moderately ill. His oral temperature was 104°F; other vital signs were normal. Physical examination was unremarkable except for the abdomen, which was soft, but diffusely tender to palpation without guarding or rebound. The liver was slightly tender and palpable 2 cm below the right costal margin. The spleen was not palpable, and no masses or ascites were found.

Initial laboratory values were peripheral white blood cell count of 13,800/µL with 85% neutrophils, 4% band forms, 7% lymphocytes, and 4% monocytes; hemoglobin of 9.7 g/dL; hematocrit of 27%; and platelet count of 517,000/µL. The erythrocyte sedimentation rate was 109 mm/hr by the Westergren method. Electrolytes were normal, and results of liver function tests revealed total protein 7 g/dL, albumin 3 g/dL, total bilirubin 0.3 mg/dL, direct bilirubin 0.1 mg/dL, aspartate aminotransferase 33 U/L, and alanine transaminase 14 U/L. Chest roentgenogram was unremarkable, except for the presence of small, right-sided basilar pleural effusion. An abdominal radiograph showed slightly dilated bowel loops. Computed tomography (CT) of the abdomen with contrast enhancement revealed a large liver abscess involving the right lobe and more than 40 small abscesses (Figs 1 and 2). No other visceral involvement was noted.

Percutaneous drainage of the liver abscess was done. No organisms were seen on Gram’s stain of the purulent material. Aerobic and anaerobic cultures both grew Chromobacterium violaceum, sensitive to trimethoprim-sulfamethoxazole (TMP/SMX), imipenem, chloramphenicol, and ciprofloxacin. Blood, urine, and stool cultures were sterile.

This patient was initially treated with imipenem, chloramphenicol, and TMP/SMX pending the culture result. Dual intravenous antibiotic therapy with ciprofloxacin and
TMP/SMX was continued for a total of 7 weeks. Ciprofloxacin was discontinued because of concern about its effect on the growth of cartilage in pediatric patients, and oral TMP/SMX was continued to finish a total of 12 weeks. Repeat CT of the abdomen 4 weeks after starting therapy revealed regression of the liver abscesses (Fig 3). The patient has done well on follow-up 3 years later.

DISCUSSION

Chromobacteria are motile, facultatively anaerobic Gram negative rods, which are catalase positive (hence its increased incidence in patients with chronic granulomatous disease). This organism produces a violet, alcohol-soluble, water-insoluble pigment, and grows without difficulty on routine culture media, including sheep blood and MacConkey agar. Nonpigmented strains have been isolated and documented in one case of human infection, although the pathogenicity is not related to pigment production.

Chromobacterium violaceum is confined to tropical and subtropical areas. The organism is unable to survive at 4°C, and thrives at a temperature range of 20°C to 37°C. It is found in the soil and water of the southeastern United States. To our knowledge, 33 cases have been reported in the literature besides our case. Sixteen cases were reported from Florida, three from Louisiana, and one each from Georgia, Texas, New Jersey, and Ohio. The patient from Ohio had visited Florida before his illness. Remaining cases were reported from Malaya (nine), France (one), and West Africa (one).

All infections, as in our case, were acquired during the months of June through September. The organism enters the human body through a minor trauma to the skin or ingestion of contaminated water. Twenty-three cases have occurred in the United States. Nine were related to wading in pools of rain water, muddy ditches, or simply walking barefoot, four cases were after trauma, and one was after breast surgery. Three cases were related to scuba diving or swimming in fresh water, and two followed near drowning, while in four cases a predisposing factor was not identified.

The clinical manifestation of C violaceum infection includes sepsis and visceral abscesses involving the liver, kidneys, and lungs. Other presentations are cellulitis at the site of trauma, urinary tract infection, lymphadenitis, osteomyelitis, sinusitis, and orbital cellulitis and meningitis.

Sepsis is the most common feature of this infection (19/34 patients), followed by cutaneous lesions/lymphadenitis (15/34) and liver abscesses (14/34). Nine patients had pneumonia or lung abscesses, two patients had diarrhea, and two others had urinary tract infection. Half the patients with cutaneous manifestations had associated sepsis.

Chromobacterium violaceum infections are serious and mandate prompt medical and surgical intervention. This organism is usually sensitive to chloramphenicol, imipenem, gentamicin, TMP/SMX, and ciprofloxacin, but it is generally resistant to penicillins, cephalosporin, and aztreonam. Workup for immune deficiency especially chronic granulomatous disease (CGD) should be done; in our patient, this workup revealed no abnormalities.

The incidence of CGD in patients with C violaceum is estimated to be 21% (6 of 23 cases).

The prognosis of C violaceum infection is
guarded. Of the 34 reported cases, 11 patients have survived (5 with CGD), 20 died, and 3 had unknown outcome.

The case fatality rate of all reported cases with known outcome is 65%. Of 19 patients with sepsis, only 4 survived, showing the grave prognosis of this organism—blood dissemination leading to a case fatality rate of 80%.

Comparing the death rate of the cases occurring in the United States revealed a death rate of 9/11 (81%) in the period from 1937 to 1979 and 5/12 (41%) for the period 1980 to 1994, indicating improved medical management.

The interval between the onset of the illness and death ranges from 7 months to 15 months. Physicians, particularly in the Southeast, should be aware of this infection. A high index of suspicion is important when facing a pediatric patient with sepsis during June through September and in whom a history of exposure to stagnant water is elicited.

References
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