Chromobacterium violaceum Cellulitis and Sepsis Following Cutaneous Marine Trauma

Elliot Carter, MD; Kressida Cain, MD; Beth Rutland, MD

Chromobacterium violaceum is a gram-negative bacillary organism that characteristically produces the purple pigment violacein. Documented as the cause of clinically relevant human infections in only 35 cases in the United States, C violaceum is particularly seen in patients with a history of cutaneous injury or trauma. We report the case of an 18-year-old woman who was struck by a propeller in a boating accident and sustained multiple deep lacerations of her right lower extremity. Shortly after admission to the hospital, the patient became frebrile and developed leukocytosis. Bacterial cultures revealed C violaceum, which demonstrated a characteristic purple pigment production on blood agar. Antimicrobial therapy was initiated, but 2 days after admission, the skin and subcutaneous tissue surrounding the patient's wounds became necrotic, necessitating an above-the-knee amputation of the right lower extremity (transfemoral amputation). The patient's condition improved after continued antimicrobial therapy and she was subsequently discharged in good health. This case represents a successful outcome of a rare but frequently fatal infection due to a morphologically and geographically distinct human pathogen.

Cutis. 2008;81:269-272.

A common saprophyte of soil and water in tropical and subtropical geographic regions, *Chromobacterium violaceum* is a rare but often fatal cause of human disease worldwide, with

Accepted for publication June 12, 2007.

From the Department of Pathology, University of South Alabama, Mobile.

The authors report no conflict of interest.

mortality estimates of approximately 50%.¹ Only 35 clinically relevant cases of *C violaceum* infection have been documented in the United States since the organism was first described in 1881.^{1.30} These cases have predominantly occurred in the south-eastern United States, and associations with chronic granulomatous disease and exposure to stagnant and slow-flowing water have been established.

Case Report

An 18-year-old woman with no relevant prior medical or surgical history was involved in a boating accident off the Gulf Coast of Mobile, Alabama, in July 2004. She was ejected from her boat into the water and sustained multiple deep lacerations to the distal portion of her right lower extremity when she was struck by the boat's propeller (Figure 1). She was transported to the emergency department at the University of South Alabama Medical Center where her condition was stabilized and aggressive transfusion therapy was initiated to correct severe blood loss. Examination of her right lower extremity revealed that in addition to skin and soft tissue lacerations, she also had suffered a fractured femur and a lacerated femoral artery. Amputation of the right lower extremity was recommended by the trauma service, but the patient's family refused amputation at that time. She was transferred to the surgical intensive care unit. The next day, she had a fever (temperature, 38.7°C) and her white blood cell count was elevated $(19,000/\mu L;$ reference range, 4500–11,000/ μL). Bacterial cultures of the patient's right lower extremity wounds yielded gram-negative rods that were glucose positive, catalase positive, oxidase positive, and urea negative, and they produced small round purple colonies on blood agar (Figure 2). The identification of C violaceum was confirmed using automated culture methodology. Antimicrobial therapy consisting of doxycycline was initiated, but the patient developed cellulitis and necrosis of her right lower extremity

Correspondence: Elliot Carter, MD, University of South Alabama, Department of Pathology, 2451 Fillingim St, Mobile, AL 36617 (ecarter@usouthal.edu).



Figure 1. Amputated right lower extremity showing pattern injury from boat propeller.



Figure 2. Blood agar plate showing colonies of *Chromobacterium violaceum*.

and subsequently underwent an above-the-knee amputation of the right lower extremity (transfemoral amputation). The patient's condition improved after continued antimicrobial therapy. She was discharged with follow-up scheduled in a rehabilitation clinic.

Comment

Initially described by Bergonzini³¹ in 1881, *C violaceum* was first documented as a pathogen of mammals by Wooley³² and years later a pathogen of humans by Lesslar.³³ Only 35 clinically relevant cases of human infection with *C violaceum*

have been documented in the United States,¹⁻³⁰ with almost all cases acquired in the southeastern United States. The organism is found somewhat ubiquitously in the soil and water in tropical and subtropical geographic regions. While classically recognized because of its production of the purple pigment violacein, nonpigmented strains of *C violaceum* have been documented and may result from serial passage of pigmented strains in the clinical laboratory.²⁰ Interestingly, the monobactam aztreonam was first described as a natural metabolic product of this bacterium.³⁴

The rarity of human infections with C violaceum is somewhat surprising given the reported ease with which the bacterium is recovered from environmental sources in tropical and subtropical climates worldwide.²⁰ The bacterium appears to have a relatively narrow geographic distribution, with the vast majority of reported infections acquired between latitudes 35°N and 35°S.17 This natural distribution reflects the growth preference of C violaceum for temperatures between 20°C and 37°C and provides an explanation for the fact that most documented infections in the United States have occurred during the summer months.¹⁴ Moist soil and stagnant or slow-flowing water have been the most commonly reported sources of infection, particularly in patients who have had cutaneous injury or trauma, which presumably provides a portal of entry for this opportunistic pathogen.

No predilection for a specific age group or gender has been shown in cases of C violaceum infection. Cases have been reported in males and females from the first few months of life to the seventh decade of life. Only one predisposing disease process has been established thus far-chronic granulomatous disease. Six of 35 patients with C violaceum infection reported in the United States have occurred in patients previously diagnosed with or subsequently found to have chronic granulomatous disease.^{10,14,15,25,29} Most bacterial infections affecting patients with chronic granulomatous disease are caused by catalaseproducing organisms,²⁵ and C violaceum is catalase positive. While patients with chronic granulomatous disease are undoubtedly at greater risk for infection with C violaceum, the mortality rate for patients with chronic granulomatous disease and C violaceum infection does not appear to be higher than for C violaceum infections in immunocompetent patients. The estimated mortality rate for C violaceum infections in the United States is approximately 50% and is greater than 60% worldwide^{1,28}; only 2 of 6 patients in the United States who had C violaceum infection associated with chronic granulomatous disease died from the infection.^{14,15}

Clinical manifestations of infection with *C violaceum* have ranged from minor skin and soft tissue infections to fatal sepsis with abscess formation in multiple organs. Less common manifestations, including osteomyelitis,²⁹ conjunctivitis,¹⁰ and pancreatitis,⁷ have been documented. With a relatively small number of reported cases of infections related to *C violaceum*, an analysis of factors affecting the severity of infection cannot be made with certainty. Other than the few cases reported in patients with chronic granulomatous disease, no strong association with chronic underlying illness has been established.

Chromobacterium violaceum can be recognized in the clinical laboratory by its biochemical characteristics and, in most cases, by its production of the purple pigment violacein on blood agar. The organism is catalase positive, nitrate positive, and produces acid from glucose; it is distinguished from *Aeromonas* sp by being lysine, maltose, and mannitol negative.³⁵ Pigment production is independent of virulence, as both pigmented and nonpigmented strains have been documented as causes of clinical disease.^{9,25}

Conclusion

Chromobacterium violaceum shows general resistance to β -lactam antimicrobial agents but is susceptible to ciprofloxacin hydrochloride, tetracycline, and imipenem.³⁵ Despite antimicrobial therapy, the mortality rate for patients diagnosed with *C violaceum* infection in the United States is approximately 50%. While growth temperature requirements make *C violaceum* a geographically limited cause of human disease, the high mortality rate associated with infections makes prompt diagnosis and appropriate antimicrobial therapy especially important.

REFERENCES

- 1. Midani S, Rathore M. Chromobacterium violaceum infection. South Med J. 1998;91:464-466.
- 2. Black ME, Shahan J. Bacillus violaceus infection in a human being. JAMA. 1938;110:1270.
- 3. Blereau RP. Septicemia and death caused by Chromobacterium violaceum. South Med J. 1980;73: 1093-1094.
- 4. Bilton BD, Johnson LW. Recurrent nonfatal *Chromobacterium violaceum* infection in a nonimmunocompromised patient. *Infect Med.* 2000;17:686-689.
- Centers for Disease Control (CDC). Chromobacteriosis— Florida. MMWR Morb Mortal Wkly Rep. 1981;29:613-615.
- Centers for Disease Control. Multiple abscesses and death due to Chromobacterium violaceum—Florida. MMWR Morb Mortal Wkly Rep. 1974;23:387.
- 7. Daguilh F, Kamakshi B. Pancreatitis, sepsis, and joint pain. *Infect Med.* 2004;21:16-17.
- Dauphinais RM, Robben GG. Fatal infection due to Chromobacterium violaceum. Am J Clin Pathol. 1968;50: 592-597.
- Desjardins M, Fenlon C, Madison D. Non-chromogenic Chromobacterium violaceum bacteremia. Clin Micro News. 1999;21:14-16.
- Feldman RB, Stern GA, Hood CI. Chromobacterium violaceum infections of the eye: a report of two cases. Arch Ophthalmol. 1984;102:711-713.
- 11. Grier DD, Qiu J, Rand K, et al. Pathologic quiz case: a 13-year-old boy with a 2-day history of fever, vomiting, and mental status changes. *Chromobacterium violaceum* bacteremia. *Arch Pathol Lab Med.* 2004;128:e131-e132.

- 12. Hodge RA. Non-chromogenic Chromobacterium violaceum in a urinary tract infection. Clin Micro News. 2002;24:15.
- Johnson WM, DiSalvo AF, Steuer RR. Fatal Chromobacterium violaceum septicemia. Am J Clin Pathol. 1971;56:400-406.
- Macher AM, Casale TB, Fauci AS. Chronic granulomatous disease of childhood and *Chromobacterium violaceum* infections in the southeastern United States. *Ann Intern* Med. 1982;97:51-55.
- 15. Macher AM, Casale TB, Gallin JI, et al. *Chromobacterium violaceum* infectious and chronic granulomatous disease [letter]. *Ann Intern Med.* 1983;98:259.
- Mamlok RJ, Mamlok V, Mills GC, et al. Glucose-6phosphate dehydrogenase deficiency, neutrophil dysfunction and *Chromobacterium violaceum* sepsis. J Pediatr. 1987;111(6, pt 1):852-854.
- 17. Moore CC, Lane JE, Stephens JL. Successful treatment of an infant with Chromobacterium violaceum sepsis. Clin Infect Dis. 2001;32:E107-E110. Epub Mar 9, 2001.
- Myers J, Ragasa DA, Eisele C. Chromobacterium violaceum septicemia in New Jersey. J Med Soc N J. 1982;79:213-214.
- Nunnally RM, Dunlop WH. Fatal septicemia due to Chromobacterium janthinum. J La State Med Soc. 1968;120:278-280.
- Ponte R, Jenkins SG. Fatal Chromobacterium violaceum infections associated with exposure to stagnant waters. *Pediatr Infect Dis J.* 1992;11:583-586.
- 21. Schattenberg HJ, Harris WH. A definite and unique occurrence of rapidly fatal infection caused by *Bacillus violaceus manilae*. JAMA. 1941;24:2069-2070.
- Seabolt JP, Overman SB, Wilson HD. Microbiology problem: Chromobacterium violaceum infection. Am J Med Technol. 1979;45:894.
- Simo F, Reuman PD, Martinez FJ, et al. Chromobacterium violaceum as a cause of periorbital cellulitis. Pediatr Infect Dis. 1984;3:561-563.

- 24. Sneath PH, Whelan JP, Bhagwan Singh R, et al. Fatal infection by *Chromobacterium violaceum*. *Lancet*. 1953;265: 276-277.
- 25. Sorenson RU, Jacobs MR, Shurin SB. *Chromobacterium violaceum* adenitis acquired in the northern United States as a complication of chronic granulomatous disease. *Pediatr Infect Dis.* 1985;4:701-702.
- 26. Soule MH. A study of two strains of B. *violaceus* isolated from human beings. *Am J Pathol*. 1939;15:592-595.
- Starr AJ, Cribbett LS, Poklepovic J, et al. Chromobacterium violaceum presenting as a surgical emergency. South Med J. 1981;74:1137-1139.
- 28. Suarez AE, Wenokur B, Johnson JM, et al. Nonfatal chromobacterial sepsis. *South Med J*. 1986;79:1146-1148.
- 29. Tucker RE, Winter WG Jr, Wilson HD. Osteomyelitis associated with *Chromobacterium violaceum* sepsis. a case report. J Bone Joint Surg Am. 1979;61(6A):949-951.
- Victorica B, Baer H, Ayoub EM. Successful treatment of systemic Chromobacterium violaceum infection. JAMA. 1974;230:578-580.
- Bergonzini C. Sopra un nuovo bacterio colorato. Annular Soc Nat Modena. 1881;14:149-158.
- Wooley PG. Bacillus violaceus manilae (a pathogenic microorganism). Bull Johns Hopkins Hospital. 1905;16: 89-93.
- Lesslar JE. Rep Inst Med Res. 1927:28. Cited by: Sneath PHA, Whelan JPF, Bhagwan Singh R, et al. Fatal infection by Chromobacterium violaceum. Lancet. 1953;2:276-277.
- Duma RJ. Aztreonam, the first monobactam. Ann Intern Med. 1987;106:766-767.
- 35. von Graevenitz A, Zbinden R, Reinier M. Actinobacillus, Capnocytophaga, Eikenella, Kingella, Pasteurella, and other fastidious or rarely encountered gram-negative rods. In: Murray PR, Baron EJ, Jorgensen JH, et al, eds. *Manual of Clinical Microbiology*. 8th ed. Washington, DC: American Society for Microbiology Press; 2003: 609-622.