J Microbiol Immunol Infect
2003;36:141-144

Chromobacterium violaceum bacteremia: a case report

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Received: February 22, 2002 Revised: August 10, 2002 Accepted: September 20, 2002

Chromobacterium violaceum is confined in tropical and subtropical regions, which can cause life-threatening disease. It is the only *Chromobacterium* species that is pathogenic to humans. Because of its rarity, clinicians often do not appreciate its importance when it is isolated. We report a fulminate fatal case *of C. violaceum bacteremia* in a 20-year-old male Taiwanese. The clinical manifestations were fever and abdominal pain, followed by shock and pulmonary septic embolism. Emergent laparotomy identified acute appendicitis with rupture. Flomoxef sodium was administered immediately. However, his condition deteriorated rapidly and he died within 48 h after the onset of illness. Two sets of blood culture yielded *C. violaceum*. Physicians should be aware of the occurrence for this infection to develop in the summer season.

Key words: Bacteremia, *Chromobacterium violaceum*

Chromobacterium violaceum is a gram-negative rod that produces the purple pigment violaceum [1]. Wooley [2] first described the pathogenic potential of this organism in 1905. Since then, several reports described human infection with this organism. This organism typically invades the host via skin lesion, wound, cervical abscess, or the pharynx, and can lead to septicemia [3]. *C. violaceum* is confined to tropical and subtropical areas. The organism is unable to survive below 4°C, and thrives at a temperature range of 20°C to 37°C. It is found in the soil and water. Approximately 50 cases of *C. violaceum* infection have been reported in the literature. It is also very rare in Taiwan, a subtropical area, only a few cases have been previously reported [5,6]. We report the case of a patient with *C. violaceum* bacteremia with a fulminate clinical course.

Case Report

A 20-year-old male factory worker, visited the emergency room on August 5, 1999 due to fever and abdominal pain. He lived in an area with no mud flows or landslides and did not report using the groundwater or contaminated water. He denied a history of travel, trauma, sexual exposure, or exposure history. White cell count was 9000 /mm^3, hemoglobin 12 mg/dL, and platelet 200 000 /mm^3. Because no emergent condition was identified, the patient returned home but came back on the next day due to abdominal pain and shortness of breath. Blood pressure was 70/32 mm Hg, heart rate 100/min, respiration rate 40/min, and oral temperature 36.6°C. Pale face and cyanosis developed afterwards. Physical examination revealed diffuse abdominal tenderness with rebounding pain. The breathing sound was coarse bilaterally. Laboratory values were peripheral white blood cell count 21 000 /mm^3 with 29% neutrophils, 29% band form. Liver function tests revealed aspartate aminotransferase of 98 IU/L (normal range, 0-40 IU/L), alanine transaminase 96 IU/L (normal range, 0-40 IU/L), total bilirubin 2.92 mg/dL (normal range, <1.2 mg/dL), direct bilirubin 1.83 mg/dL, total protein 3.58 g/dL, and albumin 1.7 g/dL (normal range, 3.5-5.5 g/dL). Renal function tests showed serum urea nitrogen 48.4 mg/dL (normal range, 8-23 mg/dL), creatinine 4.8 mg/dL (normal range, 0.7-1.5 mg/dL), and glucose 136 mg /dL. Electrolytes were sodium 134 meq/L and potassium 4.1 meq/L. Arterial blood gas showed pH 7.316 mmol/L, pCO_2 27.5 mm Hg, pO_2 77.7 mm Hg,HCO_3 13.7 mmol/L, and O_2 saturation 93.4%. Chest roentgenogram showed bilateral reticulonodular le-sions (Fig. 1). Abdominal radiograph showed slightly dilated bowel loops. Computed tomography of the abdo-men with contrast enhancement revealed focal wall thickening of the distal ileum near the ileocecal valve and distended wall thickening of the appendix with increased mesenteric stranding in the right lower quadrant extending to the pelvic cavity. These findings were consistent with acute appendicitis (Fig. 2). No other visceral involvement was noted. Emergent lap-arotomy was performed on August

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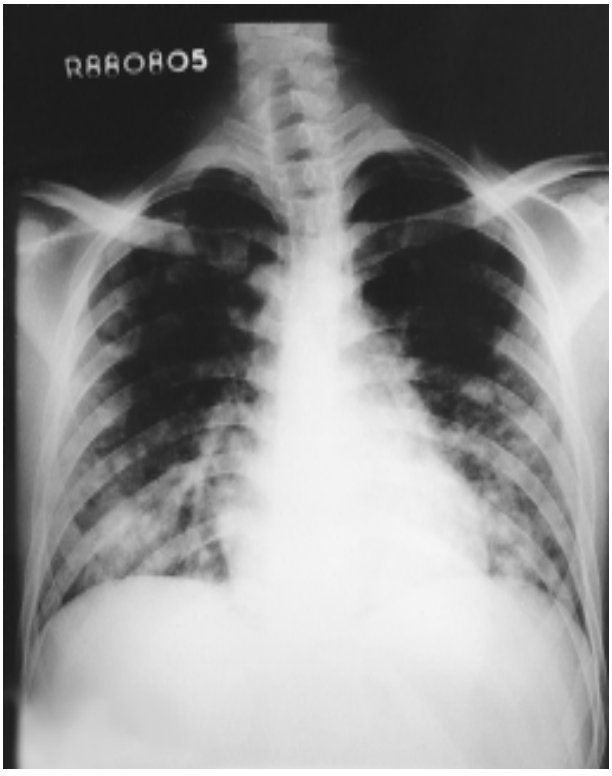

Fig. 1. Chest roentgenogram shows bilateral reticulonodular lesions.

6, 1999. Ruptured appendicitis with intraabdominal abscess was found. One hundred millimeters turbid ascites was noted. Flomoxef sodium 2 g was administered intravenously every 12 h. However, his

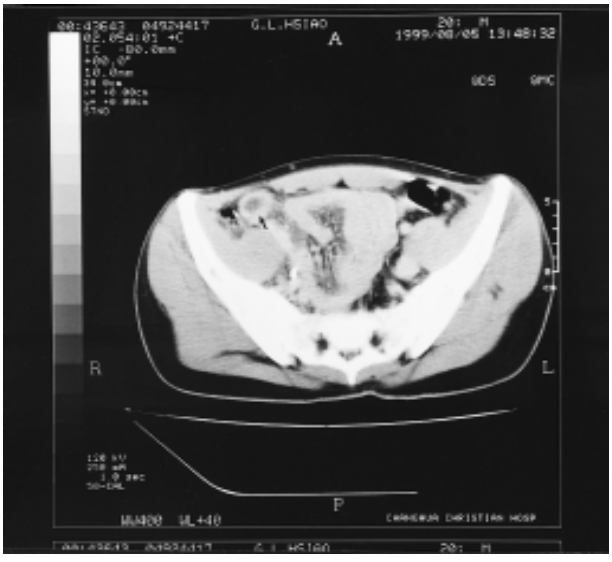

Fig. 2. Abdominal computed tomography shows acute appendicitis (arrow) with adjacent bowel wall thickening.

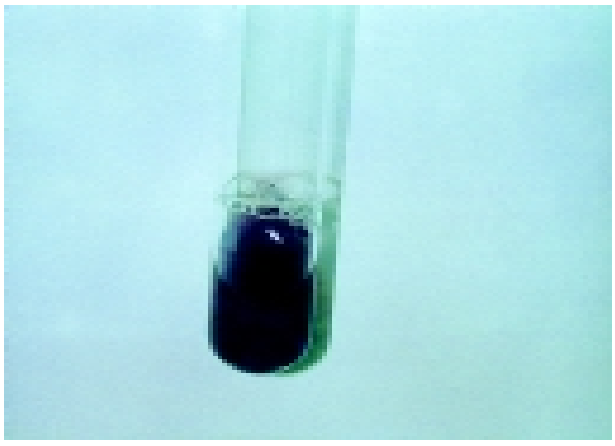

Fig. 3. *C. violaceum* produces a violet pigment in tryptic soy broth.

condition continued to deteriorate, and he died postoperatively.

Aerobic and anaerobic blood cultures (BACTEC NR-860 system, Beckton Dickinson Diagnostic Instrument Systems, Sparks, MD, US) grew gram-negative bacillus. The organism grew on routine culture media, including sheep blood and McConkey agar (Fig. 3), and produced a violet pigment in tryptic soy broth. Oxidase test was positive. Results using the conventional biochemical test method GFB-14E (Creative Microbiological, Taiwan) are shown as Table 1. According to the 5-digital computer-coding system (GFB-14E), the results were positive for *Vibrio parahaemolyticus*, *Vibrio damsela*, *Vibrio alginolyticus*, *Vibrio cincinnatiensis*, *C. violaceum*, *Vibrio vulnificus*, and *Vibrio fluvialis*. The API-20NE kit (BioMerieux Vitek, Hazelwood, MO, US) was used for further identification. The results of the biochemical reaction are shown in Table 1, and indicated that the organism was *C. violaceu*m. Antibiotic susceptibilities were tested using the disc diffusion method (BBL, Becton Dickinson, Cockeysville, MD, US) according to the guidelines of the National Committee for Clinical Laboratory Standards [4]. The results showed resistant to ampicillin, ampicillin-sulbactam, piperacillin, cefazolin, cefuroxime, cefmetazole, cefonicid, ceftazidime, cefixime, ceftizoxime, cefotaxime, and flomoxef; but susceptible to cefepime, imipenem-cilastatin, gentamicin, tobramycin, amikacin, ofloxacin, and trimethoprim-sulfamethoxazole.

Discussion

C. violaceum is confined to tropical and subtropical areas, but is very rare in Taiwan. Previously, only 3 cases have been reported [5,6]. An additional previous

Table 1. Biochemical characteristics of the isolated *C. violaceum*

Test	Result
Hemolysis (clear zone)	+
Triple sugar iron agar	Alkaline/acid
Gas from glucose	–
H_2S	–
Simmons citrate	–
Urease	–
Motility	+
Indole	–
Voges-Proskauer	–
Lysine decarboxylase	–
Ornithine decarboxylase	–
Arginine dihydrolase	–
Potassium nitrate test	+
Tryptophane test	–
Esculin hydrolysis	–
Gelatin hydrolysis	+
p-nitrophenly-β-D-galactopyranoside	–
n-acetyl-glucosamine test	+
Gluconate test	+
Caprate test	+
Adipate test	–
Malate test	+
Phenyl-acetate test	–
Catalase	+
Oxidase	+
Acid from	
glucose	+
fructose	+
lactose	–
maltose	–
mannitol	–
xylose	–
sucrose	–
adonitol	–
inositol	–
rhamnose	–
salicin	–
arabinose	–
trehalose	+
sorbitol	–
erythritol	–

unreported case of *C. violaceum* bacteremia in Taiwan, a 17-year-old man who sustained a traumatic injury with wound contamination by soil-water on farmland, was treated at Taipei Veterans General Hospital in 1985 (M. Y. Yen, personal communication). The patient developed mixed infection of *C. violaceum* and *Aeromonas hydrophila*. *C. violaceum* was isolated from both blood and wound. He was successfully treated with ceftazidime. Sporadic cases of *C. violaceum* bacteremia are probably underreported.

Chromobacteria are motile, facultatively anaerobic gram-negative rods, which are catalase positive. The organism produces a violet, alcohol-soluble, water-insoluble pigment, and grows on routine culture media, including sheep blood and McConkey agar. Nonpigmented strains have been isolated and were documented in one case of human infection, although the pathogenicity was not related to pigment production [7].

Most *C. violaceum* infections occur during the months of June through September [7]. The organism enters the human body through a minor trauma of the skin or ingestion of contaminated water. Huffam *et al* [8] reported 4 cases in tropical northern Australia. Two cases of *C. violaceum* infection after injury in a subtropical region were reported from Korea [9]. Twenty-three cases have been reported from the United States, including 9 cases which were related to wading in a pool of rain water, muddy ditches, or walking barefoot [10], 4 cases developed after trauma [11-13], and 1 case developed after breast surgery [14]. Three cases were related to scuba diving or swimming in fresh water [15,16], and 2 cases followed near drowning [15, 17]. The predisposing factors were not identified [16, 18]. Robert *et al* [19] reported one traveler who acquired *C. violaceum* deep neck infection. Johnson *et al* [3] described that *C. violaceum* invaded a host via skin lesions, wound, ingestion of contaminated water. Review of the literature indicated that the route of entry for the patient of this report was probably via the intestinal tract after ingestion of contaminated water.

The clinical manifestations of *C. violaceum* infection include sepsis and visceral abscess involving the liver, kidneys, and lung. Other presentations are cellulitis at the site of trauma, urinary tract infection, lymphadenitis, osteomyelitis, sinusitis, and meningitis. Sepsis is the most common feature of this infection followed by lymphadenitis and liver abscess. Nine of the reported cases had pneumonia or lung abscess, 2 had diarrhea, and 2 others had urinary tract infection. Half the reported cases with cutaneous manifestations had associated sepsis. This patient presented with diarrhea, abdominal pain, and dyspnea. Both the intestine and lung were involved. Physicians should beware of this infection. A high degree of suspicion is important to diagnose this condition in the pediatric or adolescent patient with sepsis. History of exposure to stagnant water during June through September provides an important clue.

C. violaceum infections are serious and mandate prompt medical and surgical intervention. This organism is usually sensitive to chloramphenicol, imipenem, gentamicin, co-trimoxazole, and ciprofloxacin, but it is generally resistant to penicillins, cephalosporins, and aztreonam. The prognosis of *C.*

violaceum infection is grave. Of the 34 reported cases, 11 patients survived, 20 died, and 3 had uncertain outcome. The case fatality rate of all reported cases with known outcome is 65%. Of the reported 19 cases with sepsis, only 4 survived, showing the grave prognosis of this organism. Blood dissemination of this organism led to a case fatality rate of 80%. This patient died within 48 h after onset of illness. *C. violaceum* disseminated rapidly, results in high mortality. The cause of death of this patient was likely due to delay of diagnosis and ineffective antibiotic therapy.

Acknowledgment

The authors would like to thank Dr. MY Yen (VGH-KS) for providing data on the additional case of *C. violaceum*.

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