

Review on *Chromobacterium Violaceum*, a Rare but Fatal Bacteria Needs Special Clinical Attention

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ABSTRACT

Chromobacterium violaceum is isolated from soil and water in tropical and subtropical areas. This Gram negative, capsulated, motile bacillus is considered as a saprophyte but occasionally it can act as an opportunistic pathogen for animals and human. It causes skin lesion with liver and lung abscesses, pneumonia, gastrointestinal tract infections, urinary tract infections, osteomyelitis, meningitis, peritonitis, endocarditis, respiratory distress syndrome and septic shock. Increasing reported cases with *Chromobacterium violaceum* infection has been noticed in recent decades. It should be considered for its difficult-to-treat entity characterized by a high frequency of sepsis, distant metastasis, multidrug-resistance and relapse. High mortality rate associated with this infection necessitate prompt diagnosis and appropriate antimicrobial therapy.

Key Words: *Chromobacterium Violaceum*, saprophyte, opportunistic, multidrug-resistance.

Introduction

Chromobacterium violaceum belongs to the family Neisseriaceae of β -Proteobacteria and was first described by Bergonzini in 1880.¹ It is a Gram negative, heterotrophic, flagellated bacilli which lives in a variety of ecosystems in tropical and subtropical regions.² *C. violaceum* is a facultative anaerobe which is oxidase and catalase positive. It grows optimally at 30-35°C. It is a saprophyte found mainly in soil and water. It grows readily on different bacteriological culture media including nutrient agar, blood agar, MacConkey agar and Muller-Hinton agar at 35-37°C and produce smooth low convex colonies with a dark violet metallic sheen.^{3,4} Few details regarding morphology, physiology, biochemical characteristics of *C. violaceum* are presented in Table.^{15, 6}

One particular characteristic of this genus is the production of a water insoluble secondary metabolite violacein, a purple pigment for which the synthesis is regulated by quorum sensing.⁷ and is responsible for the violet colonies in the media.

Although non-pigmented strains have also been reported.⁸ Though not essential for growth and survival, violacein has been suggested to be a respiratory pigment, having antiparasitic, antibiotic, antiviral, immunomodulatory, analgesic, antipyretic and anticancer effects. It has no association with pathogenesis.^{9, 10}

Although *C. violaceum* has been recognized as the single species of the *Chromobacterium* genus for a long time, nine novel species have been proposed since 2007: *C. subtsugae*,¹¹ *C. aquaticum*,¹² *C. haemolyticum*,¹³ *C. piscinae*,¹⁴ *C. pseudoviolaceum*,¹⁴ *C. vaccinii*,¹⁵ *C. amazonense*,¹⁶ *C. alkanivorans*,¹⁷ and *C. rhizoryzae*.¹⁸ Most of the *Chromobacterium* species were isolated from environmental samples (mainly from water, soil, and rhizosphere) and have not yet been associated with human infections. Exceptions include *C. violaceum*, isolated from both environmental and clinical samples and associated with several cases of fatal infections.¹⁹ and *C. haemolyticum*, isolated from a patient's sputum

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culture.¹³ and associated with a human case of bacteremia.²⁰ The non-purple species of this genus are *C. aquaticum*, *C. haemolyticum*, *C. alkanivorans* and *C. rhizoryzae*.

Table 1: Microbiological properties of *C. violaceum*.^{5, 6}

Properties	Description
1. Size	(0.6-0.9 × 1.5-3.0) mm
2. Shape	Rods with rounded end
3. Capsule	Always capsulated
4. Motility	Motile by means of a single polar flagellum
5. Growth at 40 C	-
6. Growth at 370 C	+
7. Growth at pH 4	+
8. Growth at pH 3	-
9. Growth in 2% NaCl	+
10. Growth in 4% NaCl	-
11. Anaerobic growth	+
12. Fermentation of glucose	+
13. Cyanide production	+
14. Lecithinase production (Turbid zone on egg yolk agar)	+
15. Acid production from	
L-arabinose	-
Trehalose	+
D-galactose	-
Gluconate	+
D-maltose	-
N-acetyl glucosamine	+
16. Lactate utilization	+
17. Casein hydrolysis	+
18. Esculin hydrolysis	-

'-' and '+' respectively indicates absence and presence

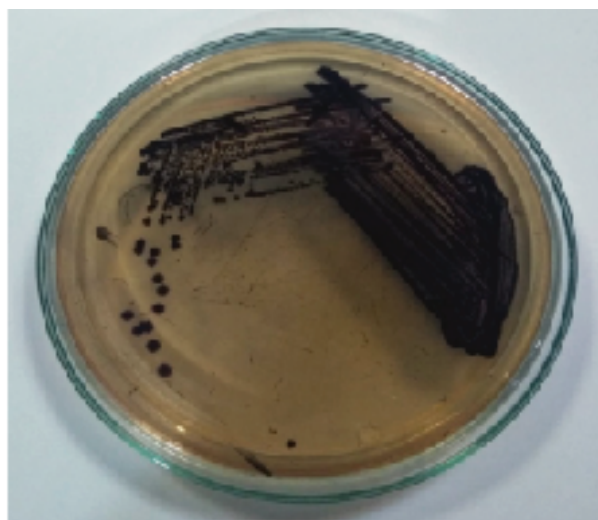


Figure 1: violet-pigmented colonies on the MacConkey agar media after 24 hours of incubation, typical of *C. violaceum* isolated in Microbiology lab of Anwer Khan Modern Medical College.

Clinical Reports

Though *C. violaceum* is primarily considered to be an environmental organism, not regularly associated with human infections, sporadic cases on its involvement in human infections have been reported. Its pathogenic potential was first described by Woolley in 1905, when he isolated it from a fatal infection in a buffalo.²¹ and the first case in humans was reported from Malaysia in 1927.²² Cases of *C. violaceum* infection in humans have been reported from Australia, India, Nepal, USA, Brazil, and Congo.²³⁻³¹

There have been about 150 cases reported worldwide.⁴ and mortality rate in such cases is found to be high (53%).¹⁹ Recently the first case of *C. violaceum* infection has been reported from Bangladesh.³² Also recently, the first case of *C. violaceum* infection in Cambodia was reported in a child who recovered after antibiotic treatment³³ and cases of infection by this bacterium were also reported in Taiwan,³⁴ Malaysia,³⁵ and Vietnam.³⁶ Recently, the first case of fatal adult bacteremia caused by *C. violaceum* was reported in Africa.⁹

C. violaceum infection is reported to be fatal, whenever it occurs in human subjects, and it is being

considered as an emergent pathogen.^{37, 38, 39} Age of patients in this review ranges from 11 months to 73 years. Specimens from these patients used for diagnosis include blood, urine, pus sample, and swab from the oropharynx. *C. violaceum* can also cause infections in animals and there are reported cases in pigs, monkeys, sheep and dogs.^{40, 41, 42, 43}

Pathogenesis and Pathology

The route of entry of *C. violaceum* still remains unclear. Most patients have a history of trauma or wounds which have been contaminated with soil or stagnant water. In some cases the injury may be so mild that it may not be recalled by the patient.⁴⁴ Infection via an oral route is suspected in patients with diarrhea. Unusual routes of exposure include scuba diving or near drowning, swimming, wading in water, falling or playing in muddy water and following surgical procedures.^{45,46,47} Despite low infectivity, *C. violaceum* is considered an opportunistic pathogen and its infection has a tremendous public health impact due to the mortality rate in humans and animals^{48,49} which justifies the study of its pathogenicity. But its pathogenesis is not yet clear.

In 1988, Miller and colleagues conducted a study that compared virulent and avirulent strains of *C. violaceum*, in which virulent strains had elevated levels of superoxide dismutase and catalase that may protect the microorganism from phagocytic attack in humans, possibly leading to its extreme virulence.⁵⁰ The genome sequence of *C. violaceum* ATCC 12472 demonstrated the presence of different pathogenic factors.⁵¹ Miki et al demonstrated that a type III secretion system (T3SS) encoded by genes present in the *Chromobacterium* pathogenicity islands 1 and 1a (Cpi-1/-1a) is involved with cytotoxicity that causes damage to hepatocytes and promotes the invasion of nonphagocytic cell. The Cila is a regulator of the T3SS and the effector protein CopE has been characterized as a guanine nucleotide exchange factor (GEFs) involved in epithelial cell invasion.⁵² Two recent works studied the protein secreted by *C. violaceum* into the culture medium, using mass spectrometry (MS).^{53, 54} These works identified proteins potentially associated with virulence such as hemolysin, outer membrane

proteins, collagenase, flagellar protein, metallopeptidases and type VI secretion system (T4SS) effector protein. The role of these proteins in pathogenicity remains to be determined.

Most of the patients had a short incubation time. The most common symptoms of *C. violaceum* infections were fever, pain over the infected site associated with various skin lesions, abdominal pain, abscesses in various organs like liver, skin, lungs, lymph nodes and brain and rapid progression to sepsis.^{31,47,55} It is also worth noting that the clinical symptoms of *C. violaceum* infection may not occur immediately after specific exposure to water or soil; instead, they may occur 60 days after exposure. The clinical spectrum of *C. violaceum* infection includes urinary tract infection, pneumonia, gastrointestinal infection, localized cutaneous lesions, localized or metastatic abscesses, osteomyelitis, meningitis, peritonitis, brain abscess, endocarditis, hemophagocytic syndrome, respiratory distress syndrome and fulminant sepsis.

Infection is reported to be severe in malnourished and immunocompromised Patients.⁵⁶ Infection occurs fast and thus, a rapid diagnosis and antibiotic susceptibility profile determination are of paramount importance.³⁶ There have been reports that *C. violaceum* septicemia occurs more commonly in patients with chronic granulomatous disease, neutrophil dysfunction, and severe polymorphonuclear G6PD deficiency.^{57,58} In these conditions, polymorphonuclear leucocytes and monocytes lack the ability to produce oxygen metabolites required to kill phagocytised bacteria⁵⁹ which renders the patient susceptible to develop septicemia and dissemination of infection to multiple organs. Moreover, virulent strains of *C. violaceum* can produce an endotoxin that withstands attack from phagocytic cells. The rapid progression of this infection and the high fatality rate prevents evaluation for these underlying immunodeficiencies in most cases. It is possible that occult microabscess or hidden septic focus may persist in a patient's internal organs despite adequate treatment. Therefore, it is necessary to treat patients with *C. violaceum* infection for an extended period and maintain close follow-up procedures.

The isolation of *C. violaceum* from water sampled in a hospital and the description of hospital-acquired

infections indicated the potential of this bacterium to cause nosocomial infections.^{60,61} One study for neonatal sepsis conducted by Anah and others in 2008 had demonstrated isolation of *C. violaceum* from 10 neonates (6.3%).⁶² These data indicate that this microorganism might have existed in hospital settings and this warrants attention. Rapid progression to life-threatening sepsis associated with metastatic abscess in *C. violaceum* infection is the most striking feature.

Laboratory data are nonspecific in *C. violaceum* infection; however, an abnormal hemogram with leukocytosis, leukopenia or left shift may occur.¹⁹ Diagnosing *C. violaceum* infection is currently based on a culture of clinical specimens followed by subsequent biochemical identification. There are no available serological tests. In 2006, Scholz and colleagues developed a method for detecting *C. violaceum* by multiplex polymerase chain reaction but it has not been widely accepted yet.⁶³

Antimicrobial Susceptibility

Data on antimicrobial susceptibility of *C. violaceum* remain very limited because this pathogen is rarely isolated from clinical specimens and results of susceptibility testing vary in different clinical settings. The Clinical and Laboratory Standards Institute has not yet established minimum inhibitory concentration (MIC) associated with *C. violaceum*. It is found to be extremely resistant to penicillins and cephalosporins.⁶⁴ In 1988, Aldridge and others conducted another study to compare the in vitro activity of ciprofloxacin and other antimicrobial agents against clinical strains of *C. violaceum*. The effective agents of *C. violaceum* including ticarcillin, carbenicillin, and cefoxitin, which were reported in 1976.⁶⁵ Before 1990, the usual treatment for *C. violaceum* infection was chloramphenicol, trimethoprim-sulfamethoxazole, tetracycline or aminoglycosides. However, several new potent antimicrobials that were introduced after 1990 include fluoroquinolone and carbapenem as penicillins and cephalosporins are extremely resistant due to increased beta-lactamase activity in *C. violaceum*. According to study by Aldridge, ciprofloxacin was the most active drug to combat *C. violaceum*. Actually combination therapy with a regimen of chloramphenicol, beta-lactams, trimethoprim-sulfamethoxazole or fluoroquinolones

along with one of the aminoglycosides is the drug of choice. An extensive treatment period of parenteral antimicrobials for 2 to 4 weeks for *C. violaceum* infection followed by maintenance therapy with an oral agent such as trimethoprim-sulfamethoxazole, tetracycline, or fluoroquinolone for 2 to 3 months to prevent relapse is recommended.^{57,66,67,68} Therefore, it is important for physicians to be aware of *C. violaceum* infection and its appropriate antimicrobial treatment regimen.

In conclusion, *C. violaceum* infection may become an emergent infection with fatal outcome after further global climate change. It represents a difficult-to-treat entity, so special attention is required. Very prompt diagnosis, optimal antimicrobial therapy and adequate therapeutic duration for *C. violaceum* infection are the keys for successful outcome.

Conflict of interest: None.

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