

Case Study

<https://doi.org/10.20546/ijcmas.2017.605.192>**Rare Cause of Sepsis - *Chromobacterium violaceum* - A Case Report****B. Subitha^{1*}, T. Jeyamurugan², S. Nithya Gomatheswari¹ and G. Hariprasad³**

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Chromobacterium violaceum is a rare pathogen that causes potentially fatal infections in human beings. It is a normal inhabitant of soil and stagnant water of the tropical and subtropical areas. *Chromobacterium violaceum* infection usually occurs after broken skin exposed to contaminated soil or water. The organism is mostly resistant to pencillins and cephalosporins and sensitive to fluoroquinolones and aminoglycosides. Rapid progression to sepsis with metastatic abscesses is the striking feature of *C. violaceum* infection. High mortality rates associated with these infections necessitates prompt diagnosis and appropriate antimicrobial therapy. Here the present study reports a case of *C. violaceum* septicemia in a 4 ½ years old male child.

Introduction

Chromobacterium violaceum is a gram negative, motile, facultatively anaerobic, oxidase and catalase positive bacillus that is temperature sensitive and widely distributed in natural aquatic environments (Rajiv Karthik *et al.*, 2012; Lee *et al.*, 1999). Although first identified in 1881, its pathogenic potential was illustrated only in 1905 by Woolley from a fatal case of a buffalo in Philippines. Human case of infection caused by this pathogen was first established by Lessler from Malaysia in 1927 (Pallab Ray *et al.*, 2004; Christopher *et al.*, 2001). It grows easily on ordinary media like blood agar, MacConkey agar, and nutrient agar (Shamshul Ansari *et al.*, 2015). Both pigmented and non-pigmented strains exist, though the non-pigmented strains are rare.

The pigmented strains produce a violet non-diffusible pigment known as violacein, which is soluble in ethanol and insoluble in water and chloroform (Harapriya Kar *et al.*, 2013; Julio Alexander *et al.*, 2007). More than 150 cases have been reported worldwide from Vietnam, Taiwan, Japan, United States, Brazil, Argentina, Australia, Senegal, Sri Lanka and India (Rajiv Karthik *et al.*, 2012; Pallab Ray *et al.*, 2004;). In most of the cases, the predominant portal of entry appears to be broken skin exposed to the organism through contaminated soil and water (Shamshul Ansari *et al.*, 2015; James Campbell *et al.*, 2013). Main clinical features include sepsis, multiple liver abscesses and diffuse pustular dermatitis, and the possibility of relapse (Kaufman *et al.*, 1986). The microorganism,

previously thought to be confined to the geographic area between latitudes 35°N and 35°S, may be expanding its habitat beyond this range due to the effects of global warming (Ching-Huei Yang *et al.*, 2011).

Case report

A 4½ years old male child was referred to our hospital with complaints of 5 days duration of high grade fever, abdominal pain and respiratory distress. On examination, patient was febrile, and toxic. Examination of respiratory system revealed tachypnoea and bilateral crepitations. Per abdominal examination showed tenderness and mild hepatomegaly and CNS was within normal limits. Blood examination showed 9.9 g/dl haemoglobin, total leucocyte count of 2700/mm³ with 75 per cent polymorphs and platelet count 83000 /mm³. Biochemical parameters revealed normal renal functions and elevated liver enzymes. Serological tests for Hepatitis B, C, Dengue, Leptospira, Scrub typhus and HIV were negative. Peripheral smear study was negative for malaria. Chest X-ray showed bilateral infiltrates. Ultrasonography of abdomen confirmed hepatomegaly. A blood culture was sent at admission in brain heart infusion broth which was incubated at 37°C. Subcultures were done on blood agar, on MacConkey agar, and on nutrient agar. Culture plates were incubated at 37°C aerobically. After 24 hours, fine, violet colonies were observed. The organism was a motile, Gram negative rod. It was catalase and oxidase positive and reduced nitrate. It fermented glucose and trehalose but did not ferment lactose or mannitol. Triple sugar iron medium showed an alkaline slant and acid butt without gas and H₂S production. Citrate was not utilized. Arginine was decarboxylated but not lysine or ornithine. The organism was identified as *Chromobacterium violaceum* based on biochemical characteristics and pigment production. Antibiotic susceptibility of the organism was

tested by disk diffusion method. The results were interpreted as per the Clinical and Laboratory Standards Institute (CLSI) guidelines for other non-Enterobacteriaceae. The isolate was resistant to Ampicillin, Cefotaxime, Ceftriaxone and Imepenam. The organism was sensitive to Ciprofloxacin and Amikacin. The patient progressed to severe respiratory distress, hypotension then shock and finally expired within 48 hrs of admission before the results of antibiotic sensitivity test came through.

Results and Discussion

Human infections caused by *C. violaceum* are rare. Although *C. violaceum* is considered nonpathogenic, this bacterium causes systemic infection in previously healthy individuals. Most patients have no underlying disease, but Macher and Mamlok *et al.*, (1997) have found *C. violaceum* to be more common in patients with Chronic Granulomatous Disease and polymorphonuclear leukocyte glucose 6 phosphate dehydrogenase deficiency. Our patients had neither of those conditions.

The dominant route of infection for this pathogen is through exposure of injured skin to contaminated water or soil, with effects ranging from cutaneous lesions and visceral abscesses to severe sepsis, which progresses rapidly to death (Shamshul Ansari *et al.*, 2015 Pallab Ray *et al.*, 2004). Leucopenia as severe as detected in the present patient has been reported in pediatric cases. This is probably due the consequences of the lipopolysaccharide and other toxins produced by the large number of bacilli infecting the tissues. Virulent strains have elevated levels of superoxide dismutase and catalase that may protect the microorganism from phagocytic attack in humans, possibly leading to its extreme virulence (Ching-Huei Yang *et al.*, 2011).

Fig.1 Violet color pigmented colonies of *Chromobacterium violaceum* in Nutrient agar

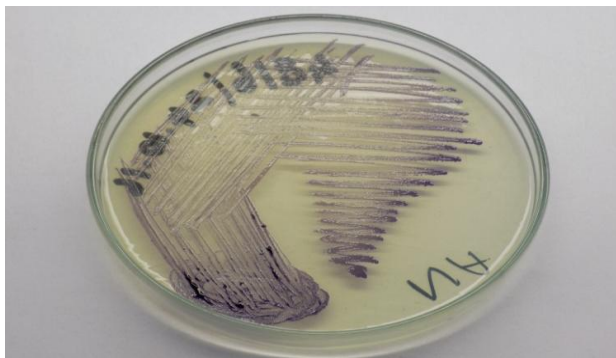


Fig.2 Antibiotic sensitivity pattern of *Chromobacterium violaceum*



Diagnosing *C. violaceum* infection is currently based on a culture of clinical specimens followed by subsequent biochemical identification. There is no serological test available for diagnosis. In 2006, Scholz and colleagues developed a method for detecting *C. violaceum* by multiplex polymerase chain reaction, but it has not been widely accepted to date (Vishnu Kaniyarakkal *et al.*, 2016). Data on antimicrobial susceptibility patterns of *C. violaceum* is very limited owing to the rarity of isolation from clinical specimens (Narayan Prasad *et al.*, 2016). Most strains show resistance to penicillins and other beta-lactam antibiotics. According to study by Aldridge, Ciprofloxacin is the most effective antibiotic in vitro (Aldridge *et al.*, 1988). Therefore, it is important for physicians to be aware of *C. violaceum* infection and its appropriate antimicrobial treatment regimen.

In conclusion, though human infections caused by *C. violaceum* are rare, the increasing

incidence suggests this to be an emerging pathogen. Thus, from this case study we recommend rapid diagnosis and prompt treatment of infections caused by *Chromobacterium violaceum*.

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