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Chromobacterium violaceum Septicemia in a Paediatric Patient: Diagnostic and Therapeutic Challenges

Ann Mary Babu [1], Rugma R [1], Ramani Bai J T [1], Rekha SR [2]*

1 Department of Microbiology, Sree Gokulam Medical College, India.

2 Department of Paediatrics, Sree Gokulam Medical College, India.

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*Corresponding Authors: Ann Mary Babu: Department of Microbiology, Sree Gokulam Medical College, India.
Tel: +91-725-9828596, *E-mail:* annmaryia.amv@gmail.com

ABSTRACT

Background: *Chromobacterium violaceum*, a rare Gram-negative bacillus, is found in tropical and subtropical soils and stagnant water. Human infections, though uncommon, are often severe and rapidly progress to septicaemia with high mortality. Diagnosing *C. violaceum* infections in paediatric cases is challenging due to their clinical overlap with other bacterial and viral infections.

Methods: Blood and tissue cultures were analysed using automated VITEK 2 and conventional biochemical tests, which identified *C. violaceum* and guided the antimicrobial therapy.

Results: Persistent fever and necrotic lesions suggested bacterial sepsis rather than a viral cause. Blood and tissue cultures revealed *C. violaceum*, sensitive to aztreonam, gentamicin, imipenem, meropenem, chloramphenicol, ciprofloxacin, and cotrimoxazole. The patient was successfully treated with meropenem, cotrimoxazole, and gentamicin over three weeks, resulting in clinical improvement and discharge.

Conclusion: This case highlights the diagnostic challenges in distinguishing *C. violaceum* infections from other pathogens. Early identification through automated VITEK 2 and sensitivity testing facilitated effective treatment, underscoring the importance of considering *C. violaceum* in septic cases with necrotic skin lesions after water exposure in endemic areas. Prompt diagnosis and targeted therapy are essential for managing this life-threatening infection.

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Introduction

Chromobacterium violaceum is a saprophytic, gram-negative bacillus found in soil and stagnant water in tropical and subtropical regions.1 Although it is a rare opportunistic pathogen in humans, it can lead to severe, life-threatening sepsis.2 The first reported human infection was in Malaysia in 1927, and until recently, only about 150 cases have been described (3-5).

Case report

A two-year-old boy presented to the emergency department with a complaint of fever for two days, loose stools and vomiting for one day. His past medical history was unremarkable, and he was up to date with his immunizations. The child had a history of a fall while playing, resulting in abrasions on his leg, which had come into contact with dirty water while playing in rainwater.

On admission, a red macular rash measuring 0.3 cm x 0.3 cm was observed on the patient's cheek, initially suspected to be an insect bite. The patient's temperature was recorded at 101.1°F, and the systemic examination was otherwise unremarkable.

Initial blood tests revealed a total leukocyte count of 36,200 cells/mm³, a platelet count of 335,000 cells/mm³, and elevated inflammatory markers, including a C-reactive protein (CRP) level of 91.9 mg/L and an erythrocyte sedimentation rate (ESR) of 67 mm/hr. Though the clinical picture suggested a viral exanthem, in view of the high-grade fever, neutrophil predominant leukocytosis, and elevated inflammatory markers, antibiotic therapy with Cefotaxime was initiated after collecting blood and urine samples for culture and sensitivity. Considering the possibility of atypical varicella, oral acyclovir was started.

On the third day of admission, the rash progressed to vesicular lesions, with additional lesions appearing on the extremities (Fig 1). A lesion on the dorsolateral aspect of the right ankle (measuring 3 cm x 3 cm) became pustular, surrounded by erythema and oedema (Fig 2).

A vesiculopapular lesion subsequently developed into a necrotic patch (Fig 3). A Tzanck smear was negative, and serum and swab samples from the lesions for RT-PCR testing of poxviruses, varicella, and HSV were also negative (IAV, Trivandrum). In view of these results, oral acyclovir was discontinued.

The child continued to have high-grade fever spikes, and repeat blood investigations showed increasing inflammatory markers. The antibiotic regimen was modified to Piperacillintazobactam, Linezolid, and Clindamycin. Persistent fever spikes prompted the collection of repeat blood cultures and tissue samples from the necrotic lesion. The antibiotic regimen was subsequently updated to Meropenem, with Linezolid continued and Doxycycline added.

In the microbiology laboratory, blood samples were loaded into the BACTAlert system for automated culture, while tissue samples were processed conventionally. The Gram stain of the tissue sample revealed scanty pus cells and Gram-negative bacilli. The sample was inoculated onto Blood Agar (BA), MacConkey Agar (MA), and Thioglycollate Broth and incubated.

Table 1. Physiochemical characters of the case isdentified as *Chromobacterium violaceum*.

Test	Result
Motility	Motile
Catalase	Positive
Oxidase	Positive (Dhar and Johnson method)
Indole Test	Negative
Citrate Utilization	Positive
Triple Sugar Iron Agar (TSI)	Alkaline slant, acidic butt, no gas, no H_2S
Mannitol Motility	Non-fermenter, motile
Nitrate Reduction	Reduced
Urea Hydrolysis	Positive
Methyl Red Test	Negative
Voges-Proskauer Test	Negative
Arginine Dihydrolase	Positive
Glucose Fermentation	Fermented with gas production

The following morning, the blood sample flagged positive in the BACTAlert system. It was subcultured onto Blood Agar and MacConkey Agar for further identification. Direct Gram staining of the blood sample revealed Gram-negative rods. Subsequent growth was observed on both Blood Agar and MacConkey Agar plates from both the blood and tissue samples. On Blood Agar, 2-3 mm round, convex, deep violet colonies with beta-haemolysis were observed (Fig 4). On MacConkey Agar, 2-3 mm round, violet-coloured colonies were noted. The Gram stain from the colony smear revealed Gram-negative rods with rounded ends.

Growth from the blood and tissue samples was processed for automated identification and sensitivity testing using the VITEK 2 system. Conventional biochemical tests and sensitivity testing by the Kirby-Bauer disk diffusion method as per CLSI guidelines were also performed.

The organism was identified as motile and tested positive for both catalase and oxidase. The oxidase reaction was evaluated using the method described by Dhar and Johnson. The following biochemical tests were performed (Table 1).

The organism was identified as *Chromobacterium violaceum* and antimicrobial susceptibility testing using Kirby-Bauer disc diffusion method test indicated that the organism was sensitive to Aztreonam, Gentamicin, Imipenem, Meropenem, Chloramphenicol, Ciprofloxacin, and Cotrimoxazole. Identification and sensitivity were confirmed by the VITEK 2 system (BioMérieux) using the Gram-negative ID and AST cards.

The patient was discharged on request due to financial constraints and to SAT Hospital, Trivandrum, where antibiotics Meropenem, Cotrimoxazole, and Gentamicin were administered for three weeks. The patient showed symptomatic improvement and was discharged in a stable condition.

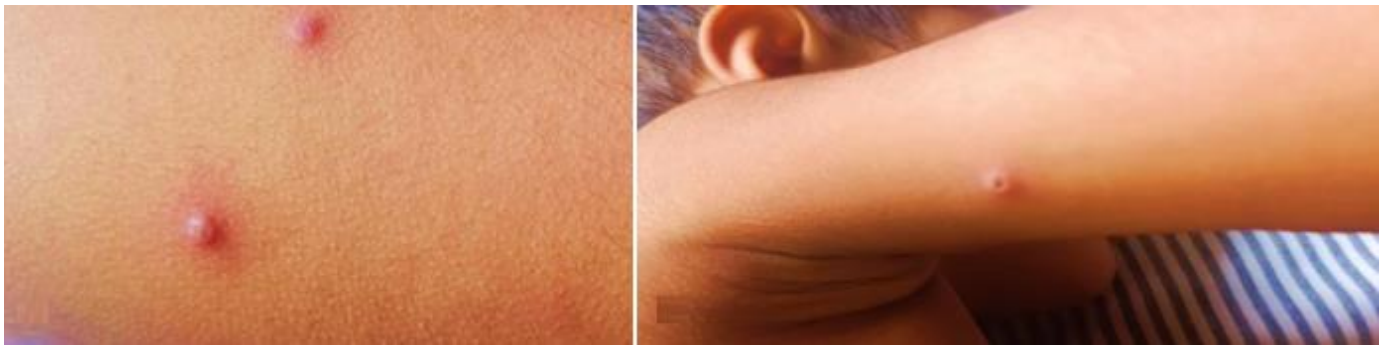

Fig 1. Vesicular lesions on extremities.

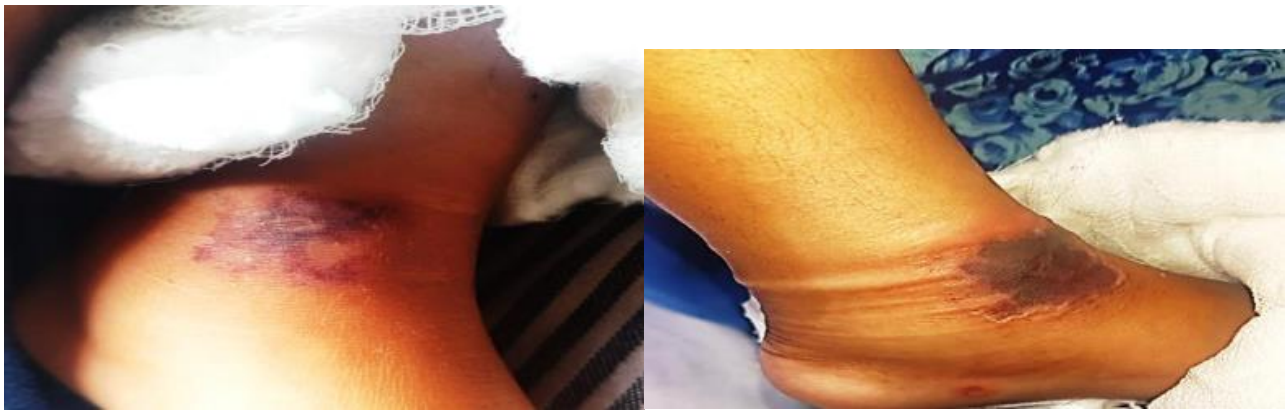

Fig 2. Lesion on the dorsolateral aspect of the right ankle.

Discussion

Chromobacterium violaceum is a saprophytic, gram-negative bacillus found in soil and stagnant water in tropical and subtropical regions.1 Although it is a rare opportunistic pathogen in humans, it can lead to severe, life-threatening sepsis (2). The first reported human infection was in Malaysia in 1927, and until recently, only about 150 cases have been described (3-5).

In majority of patients with *C. violaceum* infection, the skin is the portal of entry for organisms found in contaminated soil and water. It is even possible to be infected via the oral route (6). The patient's exposure to contaminated water after a fall likely introduced the pathogen into the wound, which subsequently led to systemic infection in this case. The differential diagnosis includes Gram-negative bacteria, such as *Pseudomonas* and *Aeromonas* species, which are well-known for causing severe soft tissue infections, particularly following trauma and exposure to contaminated water sources. The necrotizing skin lesion in this case raised the suspicion of an aggressive bacterial infection, possibly by a *Pseudomonas* species, often referred to as ecthyma gangrenosum.

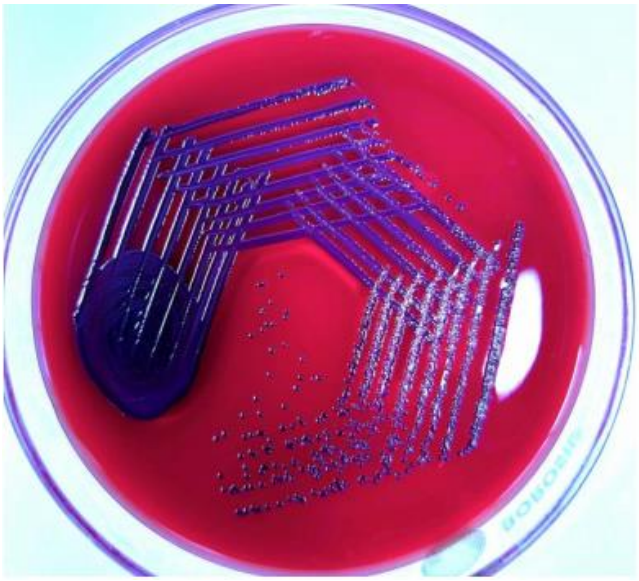

Fig 3. Violet-coloured colonies on Blood agar.

In *C. violaceum* infection there is often a preceding occurrence of local cellulitis, pustules, ulcers with necrotic bases, or lymphadenitis, before systemic infection symptoms emerge (2). This presentation may be mistaken for septicemic melioidosis, which is more prevalent in Southeast Asia, atypical varicella-zoster due to vesicular

skin eruptions (rashes), or even monkeypox because of the associated swollen lymph nodes seen in few cases (2). *C. violaceum* infections present with a wide and varied clinical spectrum, including conditions such as urinary tract infections, pneumonia, gastrointestinal infections, localized skin lesions, localized or metastatic abscesses, osteomyelitis, meningitis, peritonitis, brain abscesses, endocarditis, hemophagocytic syndrome, and respiratory distress syndrome. This case presents a diagnostic challenge due to the overlapping clinical features of viral and bacterial infections. The initial presentation of fever with vesicular rash suggested a viral etiology, such as varicella, leading to initiation of antiviral therapy with acyclovir. However, the persistence of fever and the progressive necrotic lesion pointed towards a more complex underlying bacterial infection.

Chronic granulomatous disease is a recognized risk factor for infections caused by *C. violaceum* (7). Patients with this condition are particularly vulnerable to infections by catalase-producing bacteria like *C. violaceum* due to their impaired oxidative metabolism during phagocytosis (8).

Many strains of *C. violaceum* produce violacein, an insoluble pigment that gives the colonies a violet-black color on solid media under aerobic conditions. This pigment, which is responsible for the species' name, also plays an important role in pathogenesis (9). About 9% of *C. violaceum* strains are reported to be nonpigmented. Non-pigmented, oxidase-positive strains may initially resemble *Vibrio* or *Aeromonas*, but can be differentiated by their growth in nutrient broth without NaCl, their ability to ferment D-glucose, mannitol, and maltose, and their lysine and ornithine decarboxylase activities (10).

The scarcity of human cases, despite significant exposure in wet tropical areas, could be due to an unexpected mechanism of pathogenesis (11).

Infections caused by *C. violaceum* are linked to high morbidity and mortality. Case reports indicate mortality rates between 53% and 80%, with greater risk of death observed in patients experiencing disseminated disease (12).

Treating *C. violaceum* infections can be challenging because the bacterium shows resistance to multiple antibiotics, including penicillins and cephalosporins (13). The mortality rate is considered high, and effective treatment typically requires the use of multiple antibiotics (9). Patients who survived and recovered were primarily treated with aminoglycosides, fluoroquinolones, and carbapenems, based on culture and susceptibility-guided antibiotic regimens. Although less common, some strains have also been reported to be resistant to imipenem and aminoglycosides. Despite an initial cure, relapse occurring more than two weeks after the end of treatment is likely due to the presence of a lingering infection. To prevent relapse, oral medications such as trimethoprim sulfa-methoxazole, doxycycline, or ciprofloxacin are often prescribed for several weeks to months following 2 to 4 weeks of intravenous antibiotic therapy (2).

Conclusion

This case underscores the importance of considering *C. violaceum* in the differential diagnosis of patients presenting with soft tissue infections following water exposure, especially in tropical or subtropical regions. Given the organism's propensity for rapid progression to septicemia and high mortality rates, early identification and appropriate antimicrobial therapy are critical for successful outcomes. The use of automated systems, such as VITEK 2, alongside conventional culture methods, played a crucial role in the timely identification of the pathogen and initiation of effective treatment.

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Ethics approval and consent to participate

Not needed.

Conflict of interest

None declared.

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