**Chromobacterium violaceum** septicaemia from north India


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Though **Chromobacterium violaceum** is a common inhabitant of soil and water in tropical and sub-tropical regions, human infections are rare but when they do occur result in high mortality. Since the first case from Malaysia in 1927, about 150 cases have been reported in world literature. Till date 6 cases have been reported from southern and eastern parts of India. We report here a case of **C. violaceum** septicaemia, probably the first case from north India. The patient, a 6 and a half year old boy was admitted with high fever. The patient had anaemia, neutrophilic leucocytosis and bilateral chest infiltrates. Routine and bacteriological investigations were carried out to establish the aetiological diagnosis. **C. violaceum** was isolated in pure culture from blood and pus. The patient was successfully treated with ciprofloxacin and amikacin. This is probably the first documented case report of **C. violaceum** infection from north India and the only Indian case with septicaemia which survived.

**Key words** **Chromobacterium violaceum** - septicaemia

**Chromobacterium violaceum** is a common inhabitant of soil and water in tropical and sub-tropical regions¹. It was first identified in 1881 and its pathogenic potential was first described by P.G. Woolley in 1905, who isolated it from a fatal infection in buffalo in the Phillipines². J.E. Lessler first described it as a human pathogen in 1927 in Malaysia³. It is a Gram negative rod which is non-fastidious, facultatively anaerobic, fermentative and positive for oxidase and catalase reactions⁴. 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 chloroform⁵. More than 150 cases have been reported worldwide from Vietnam, Taiwan, Japan, United States, Brazil, Argentina, Australia, Senegal and Sri Lanka⁶. In most of the cases, the predominant portal of entry appears to be broken skin exposed to the organism through contaminated soil and water⁶. Main clinical features included sepsis, multiple liver abscesses and diffuse pustular dermatitis⁷, and most of them were fatal³⁸. Till date six cases have been reported from India (3 from Manipal, and 1 each from Mangalore, Vishakhapatnam and West Bengal)⁹¹⁴. We report here a case of **C. violaceum** septicaemia with multiple skin lesions in a child with glucose-6 phosphate dehydrogenase (G6PD) deficiency, from north India.

A 6 and a half year old boy was admitted to Nehru Hospital, Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh on August 15,
2002, with symptoms of high fever for five days and rapid breathing for two days. He developed a pustule over right upper back on 2nd day of fever, which ruptured spontaneously. On examination, patient was febrile, pale, with a pulse rate of 120/min, respiratory rate of 46/min, and blood pressure of 110/60 mm Hg. Examination of respiratory system revealed tachypnoea and bilateral crepitations. Per abdominal examination showed mild hepatosplenomegaly and CNS was within normal limits. Blood examination showed 6.6 g/dl haemoglobin and a total leucocyte count of 10000/mm³ with 75 per cent polymorphs. Chest X-ray showed bilateral infiltrates. Ultrasonography of abdomen confirmed hepatosplenomegaly.

Pus collected from the skin pustule was inoculated on sheep blood agar (SBA, Hi-Media Laboratories, Mumbai) and McConkey agar plates (MA, Hi-Media Laboratories, Mumbai) and in a tube of Robertson’s cooked meat medium. Blood was cultured by conventional method in two bottles containing 45ml each of tryptone soy broth (Hi-Media Laboratories, Mumbai, India) and bile broth (Hi-Media, Mumbai) and subcultures made on SBA and MA plates. SBA plates were incubated aerobically with 7 per cent CO₂ at 35°C in a CO₂ incubator. All other media were incubated at 35°C aerobically. C. violaceum grew on SBA and MA plates from blood and pus. After aerobic incubation for 24 h at 37°C on blood agar, 1.0-1.5 mm violet pigmented colonies developed with a narrow zone of clear haemolysis and a smell of hydrogen cyanide (HCN). On MA plates, 1.0-1.5 mm violet pigmented colonies appeared. The organism was a facultatively anaerobic (non-pigmented anaerobically), motile, Gram negative rod. It was catalase and oxidase positive and reduced nitrate. It fermented glucose (acid and gas) 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.

Antibiotic susceptibility of the organism was tested by disk diffusion method only, following the modified Stoke’s method. It was sensitive to chloramphenicol, amikacin, erythromycin, ciprofloxacin, and gentamicin and resistant to penicillin, cefotaxime and ceftriaxone. The strain was negative for β-lactamase tested by nitrocefin (Cefinase®, BBL®, Maryland, USA) disc.

Patient, at admission, was suspected to have staphylococcal septicaemia and empirically started on cefotaxime and vancomycin for three days. When the culture and sensitivity reports were available, antibiotics were changed to ciprofloxacin (10 mg/kg iv twice daily for seven days followed by orally in the same dose) and amikacin (7.5 mg/kg im in two divided doses). After 14 days of therapy patient recovered and was discharged.

Human infections caused by C. violaceum are rare. Majority of the cases have been reported from tropical and sub-tropical countries including India (Table). The non-pigmented strains have pathogenic potential comparable to pigmented ones but are difficult to identify as they are confused with Pseudomonas spp. or Vibrionaceae because of similarity in their biochemical properties. The organism is usually resistant to penicillins and cephalosporins.

Of the seven cases reported from India including the present one, 2 were neonates, 4 were children below 7 yr, and one a young adult. Most of the cases reported worldwide were young. Eleven out of 12 patients reported from the United States had a mean age of 15 yr. Fever was the presenting symptom in four cases from India. Skin lesion or superficial abscess was a feature at presentation or developed during the course of infection in five out the seven cases. Septicaemia was bacteriologically proven in four cases while two had meningitis. Only one case presented with diarrhoea without any evidence of dissemination. C. violaceum is generally considered to be of low virulence, but septicaemia is usually fatal if not promptly treated. One report from the United States mentioned the case fatality rate as high as 57 per cent. The cases with fatal outcome showed features like sepsis, multiple liver abscesses, meningitis and pulmonary complications. Four out of the five Indian cases with septicaemia or meningitis had a fatal outcome.
### Table. Salient features of the seven published Indian cases of *C. violaceum* infection

<table>
<thead>
<tr>
<th>Sl. no.</th>
<th>Year</th>
<th>Age</th>
<th>Sex</th>
<th>State &amp; city</th>
<th>History</th>
<th>Clinical presentation</th>
<th>Specimen</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Reference no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1979</td>
<td>4 yr</td>
<td>M</td>
<td>Andhra Pradesh, Vishakhapatnam</td>
<td>Fever 10 day</td>
<td>Septicaemia Meningitis Ulcers</td>
<td>Blood Skin lesions</td>
<td>Gentamicin Kanamycin Cortisone</td>
<td>Died</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>1987</td>
<td>NB</td>
<td>M</td>
<td>Karnataka, Manipal</td>
<td>Fever 4 day</td>
<td>Meningitis</td>
<td>CSF</td>
<td>Gentamicin Co-trimoxazole</td>
<td>Died</td>
<td>9</td>
</tr>
<tr>
<td>3</td>
<td>2000</td>
<td>2 yr 10 months</td>
<td>F</td>
<td>Karnataka, Manipal</td>
<td>Diarrhoea</td>
<td>Diarrhoea</td>
<td>Stool</td>
<td>Gentamicin Ampicillin</td>
<td>Recovered</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>2002</td>
<td>2 month</td>
<td>F</td>
<td>Karnataka, Manipal</td>
<td>Preauricular swelling, discharge 20 days</td>
<td>Pustules Ear discharge Septicaemia Meningitis</td>
<td>Blood Liver pus Sinus discharge Urine</td>
<td>Gentamicin Ceftazidime Amoxycillin-clavulanic acid</td>
<td>Died</td>
<td>11</td>
</tr>
<tr>
<td>5</td>
<td>2002</td>
<td>8 days</td>
<td></td>
<td>Karnataka, Mangalore</td>
<td>Fever, ear discharge 4 days</td>
<td>Pustules Septicaemia Meningitis</td>
<td>Blood Skin</td>
<td>Gentamicin Cefotaxime Ceftriaxone</td>
<td>Died</td>
<td>12</td>
</tr>
<tr>
<td>6</td>
<td>2003</td>
<td>24 yr</td>
<td>M</td>
<td>West Bengal</td>
<td>Wound abscess 2 wk</td>
<td>Abscess right Leg Pus</td>
<td></td>
<td>Ciprofloxacin</td>
<td>Recovered</td>
<td>14</td>
</tr>
<tr>
<td>7</td>
<td>2002</td>
<td>6.5 yr</td>
<td>M</td>
<td>Union Territory Chandigarh</td>
<td>Fever 2 days</td>
<td>Septicaemia Pustules</td>
<td>Blood Skin biopsy</td>
<td>Amikacin Ciprofloxacin</td>
<td>Recovered</td>
<td>Present study</td>
</tr>
</tbody>
</table>


were susceptible to mezlocilin, piperacillin, imipenem and aztreonam and except one all were sensitive to ticarcillin. Most strains were resistant to penicillins, including ampicillin, and addition of clavulanic acid did not increase susceptibility indicating thereby that the resistance was not mediated by β-lactamases. The same study reported that cephalosporins were ineffective except cefotetan\(^8\). In general, the isolates were sensitive to aminoglycosides, gentamicin being reported to be the most effective. Most studies have also reported good in vitro efficacy of chloramphenicol, doxycycline and cotrimoxazole. The present isolate was sensitive to erythromycin, amikacin, chloramphenicol, ciprofloxacin and gentamicin and resistant to penicillin, cefotaxime and ceftriaxone.

Though human infections caused by \textit{C. violaceum} are rare, the increasing incidence suggests this to be an emerging pathogen. High index of suspicion and appropriate antimicrobial therapy is required for this potentially fatal infection. Even when the infection appears to be localized, because of its propensity to disseminate, especially in children, the prognosis should be guarded.

References


2. Woolley PG. \textit{Bacillus violaceus manilae} (a pathogenic organism). \textit{Bull John Hopkins Hospital} 1905; 16 : 89-93.


