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CASE REPORT

A rare nonfatal presentation of disseminated *Chromobacterium violaceum* sepsis

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We present a case of disseminated *Chromobacterium violaceum* sepsis with multiple liver and splenic abscesses presenting with skin lesions and cardiogenic shock, and later diagnosed to have chronic granulomatous disease. The patient was treated with prolonged antimicrobial therapy, after which she recovered and remained asymptomatic on follow-up.

Introduction

Chromobacterium violaceum, a common inhabitant of soil and water, is a facultative anaerobic Gram-negative saprophytic bacillus, found mainly in tropical and subtropical climates.[1,2] Only five childhood cases of invasive chromobacterial infection have been reported from the Indian subcontinent, with three of them showing complete recovery.[3,4] Disseminated *C. violaceum* sepsis is associated with a high mortality rate.[5,6] Inability to recognize this infective organism early because of varied presentation, its peculiar antibiotic sensitivity pattern, and high probability of relapse due to administration of a routine short course (7—14 days) of antibiotics, have been the bane of management of this infection. Here we report an unusual and aggressive, yet nonfatal presentation of *C. violaceum* sepsis in a child, resulting in a new diagnosis of chronic granulomatous disease (CGD).

Case report

An 11-year-old girl presented with fever for 4 days and excruciating limb and abdominal pain for 1 day. There was no history of any septic focus. The child had suffered an injury to the dorsum of the left foot 7 days earlier and she had waded through stagnant water. On examination, she

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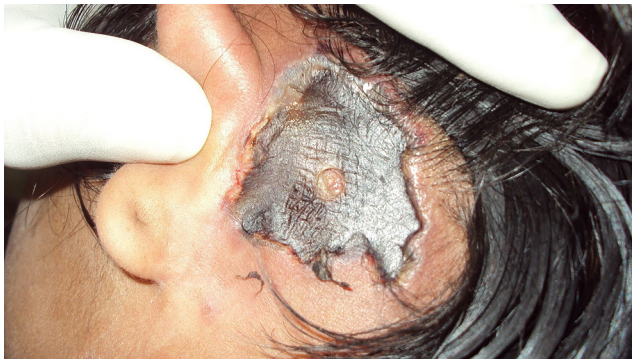

Figure 1. A 4 cm × 5 cm tender violaceous and necrotic lesion over the left postauricular area with formation of an eschar, suggestive of ecthyma gangrenosum.

was conscious, in immense abdominal pain, had gallop rhythm (heart rate 160/min), normal systemic oxygenation (on room air) and a blood pressure of 90/70 mmHg. She had multiple ecchymotic lesions with violaceous hue over the arms, trunk and back; several vesicular lesions over the arms and back, a 3 cm × 3 cm violaceous lesion over the dorsum of the left foot, which appeared after admission, eventually evolving into a necrotic patch, and a 4 cm × 5 cm tender violaceous and necrotic lesion over the left postauricular area with formation of an eschar, suggestive of ecthyma gangrenosum (Fig. 1). She had guarding and tenderness over the abdomen, with a soft, tender liver palpable 6 cm below the right costal margin (span 11 cm). The spleen was just palpable.

Within 2 hours of admission, the child's condition suddenly deteriorated with the advent of hypotensive shock (heart rate 170/min, respiratory rate 40/min, blood pressure 70/40 mmHg) with respiratory failure. Intravenous (i.v.) administration of normal saline, given as a part of fluid resuscitation resulted in an increase in the liver size and central venous pressure and persistent low mixed venous oxygen saturations (<60%) Chest radiograph demonstrated cardiomegaly (cardiothoracic ratio: 0.6) with bilateral pulmonary infiltrates. The arterial blood gas analysis demonstrated hypoxemia (PaO_2—50 mmHg) with normal hemoglobin saturation. Complete hemogram (Table 1) showed leukopenia with neutrophilic predominance, thrombocytopenia and anemia. In view of the features of cardiogenic shock,[7] along with extensive cutaneous manifestations, in a febrile child, a possible diagnosis of *Pseudomonas* sepsis was considered. She was mechanically ventilated and treated with cardiotonic drugs and decongestive measures (i.v. furosemide 2 mg/kg/day). Antimicrobial treatment, consisting of i.v. piperacillin—tazobactam (300 mg/kg/day) and gentamicin (5 mg/kg/day), was administered for 3 weeks. Blood culture and scrapings from the postauricular lesion along with the one over the right foot later grew *C. violaceum* that was sensitive to erythromycin, piperacillin—tazobactum, gentamicin, amikacin, ciprofloxacin, co-trimoxazole, meropenem, and imipenem. As the child had persistent fever spikes, i.v. ciprofloxacin (20 mg/kg/day) was added on Day 3. Computed tomography scan of the abdomen on the 11th day showed multiple nonenhancing hypodense lesions in the liver (largest 2 cm × 2 cm) and spleen (largest 1.6 cm × 1.2 cm), suggestive of abscesses (Fig. 2). Therapy with ciprofloxacin was continued for a total of 10 weeks (i.v. for 5 weeks followed by oral for 5 weeks), until complete resolution of abscesses. Levels of amylase, aspartate, and alanine transaminase in serum were normal, but those of C-reactive protein were elevated (Table 1). The Tzanck smear from the vesicular lesions did not show varicella inclusions.

On Day 2 (36 hours after admission), she developed supraventricular tachycardia not responding to multiple doses of i.v. adenosine (0.1 mg/kg/dose i.v.), but improved, with continued decongestive measures, over the next 12 hours (i.v. furosemide 2 mg/kg/day), further corroborating the diagnosis of cardiogenic shock.[8] The patient showed gradual improvement in her clinical status, and ventilatory and cardiotonic support were gradually withdrawn on 10th day after admission. Chest radiograph showed improvement over 15 days. Two-dimensional echocardiography, on Day 13, showed a structurally normal heart with normal ejection fraction. The child became afebrile from the 14th day of admission and the skin lesions healed with scab formation and scarification. The hepatic abscesses gradually responded with weekly ultrasound scans showing progressive reduction in size and numbers. On further work-up, the child was found to be suffering from chronic granulomatous disease, CGD (nitroblue tetrazolium test positive neutrophils 0%, reference value >90%; confirmed by dihydrorhodamine flow cytometry). ELISA for HIV antibodies was negative and G6PD levels were normal. Her elder sister was also diagnosed to have CGD on the basis of similar test results. Neither of the parents had CGD. Both siblings were

Table 1 Blood investigations

	Reference value	Admission	Day 11 after admission	Day 15 (discharge)
Hemoglobin (g/L)	110—180	96	108	110
Total WBC count (×10^9/L)	4.0—11.0	3.0	6.7	9.7
Platelet count (×10^9/L)	120—500	89	190	215
AST (U/L)	5—40	98	72	40
ALT (U/L)	5—40	83	66	34
Serum amylase (U/L)	30—100	50		
C-reactive protein (mg/L)	0—5	300	82	
Prothrombin Time (s)	11	12		

ALT = alanine transaminase; AST = aspartate transaminase; WBC = white blood cell.

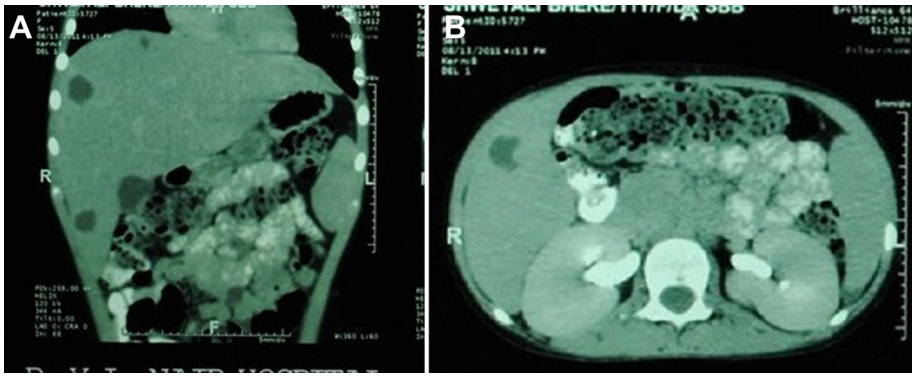

Figure 2. Computed tomography images. (A) Multiple nonenhancing subcentimetric and few enlarged hypodense lesions noted throughout the liver, largest 1.9 cm × 1.9 cm in segment eight of liver. (B) Spleen: 10.6 cm with a hypodense area of 1.6 cm × 1.2 cm at the lower pole.

advised co-trimoxazole (5 mg/kg/day) and itraconazole 100 mg twice daily (10 mg/kg/day), prophylaxis. The child remained asymptomatic at follow-up 3 months after initial presentation.

Discussion

The patient with *C. violaceum* sepsis presented with skin lesions, respiratory failure and multiple hepatic and splenic abscesses and was diagnosed to have an underlying immunodeficiency. Our case possibly had cardiogenic shock at presentation and we believe that *C. violaceum* bacteremia presenting primarily with cardiac symptoms has only rarely been reported.[7,8] Although the cause is not clear, we hypothesize it to be related either to infective endocarditis, which is known to occur in association with *C. violaceum* infection,[9,10] or due to myocardial dysfunction secondary to particular predilection of the organism for myocardiocytes.[11,12] However, in the absence of objective findings, this cannot be conclusively proved. As in our case, cutaneous involvement in the form of pustular lesions with surrounding erythema and ecthyma gangrenosum, is common.[13] As the child had suffered injury to the left foot prior to illness, while wading through muddy water (which is a known risk factor for *Chromobacterium* sepsis); we suspect that the skin injury served as the portal of entry for the organism. Our patient demonstrated ecchymotic macules distributed in time and place. Such peculiar lesions associated with systemic disseminated *C. violaceum* infection are attributed to cutaneous septic embolization.[6] The presence of skin lesions often leads to a misdiagnosis of disseminated varicella or pseudomonal infection.

Approximately 150 cases (including 8 from India) of *Chromobacterium* infection (systemic and localized) have been reported.[2,3] The infection, being rare, is hardly ever suspected on clinical grounds. Immunodeficiency predisposes to this infection, although many patients with *C. violaceum* infections have no underlying immunodeficiency. Patients with G6PD deficiency and CGD are prone to develop this infection.[14]

The usual initial manifestations could be nonspecific and include fever, cough, otitis, cellulitis, and adenitis or it could present with bacteremia, osteomyelitis, and multiple organ abscesses predominantly in the lungs, liver, and spleen.[6] Bacteremia or disseminated *C. violaceum* infections are associated with poor prognosis,[6,15] as can be made out from the fatal outcome in three of the six Indian cases with sepsis or meningitis.[4] The organism is usually susceptible to ciprofloxacin, aminoglycosides, chloramphenicol, carbapenems, aztreonam, tetracyclines, and cotrimoxazole.[4] The importance of initiating therapy with one or more of these antibiotics cannot be overemphasized as the infection is associated with 100% mortality in cases that do not receive at least one of these drugs.[6] Shorter courses of antibiotics (7—14 days) result in clinical improvement but have often been associated with fatal relapses.[6] Antimicrobial therapy (initial parenteral route followed by oral administration) with one or more of the above drugs, for 8—15 weeks, is considered essential for ensuring eradication of the infection, especially of deep seated abscesses.[6]

Human infections caused by *C. violaceum* are rare. It is, therefore, of vital importance that pediatricians are reminded about diagnosing this infection, by suspecting it early on clinical grounds, so that effective antibiotic therapy can be promptly initiated. It would also help reassure them that appropriate supportive care and prolonged anti-microbial therapy can be successful, despite disseminated infection. In addition, diagnosed cases should be investigated for immunodeficiency status, especially for CGD and G6PD deficiency.

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None.

Conflicts of interest

All contributing authors declare no conflict of interest.

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