

Chromobacterium violaceum infection

Introduction

Chromobacterium violaceum is a common facultative anaerobic, gram-negative rod found in the soil and water of tropical and subtropical climates. Its human infection is rare but fatal. The authors report a case of *C. violaceum* infection presenting with liver abscesses, skin pustules and pneumonia.

Discussion

C. violaceum grows well in all standard bacteriological media used in a clinical laboratory. After 18 hours of incubation, the colonies appear smooth, convex and violet in colour.

In this case, *C. violaceum* strains were isolated from blood cultures and from pus aspirated from the pustules. The organism grew well on blood and MacConkey's agars. Preliminary identification was made based on the morphology and purple colouration of the colonies besides catalase and nitrate tests. The isolates were subsequently confirmed with API 20 NE (BioMérieux, France) and BBL Crystal enteric/nonfermenter identification system (Becton Dickinson, USA). Both of

them gave a high index score of identification (more than 90%) for *C. violaceum*. The antibiotic susceptibility of the isolates is shown in Table 1.

The first reported human infection of *C. violaceum* was from Malaysia in 1927 (Sneath et al, 1953). Since then there have been sporadic cases from Singapore, Taiwan, Korea, Brazil, Argentina, Senegal

Table 1. Antibiotic susceptibilities of *Chromobacterium violaceum* isolates

Sensitive to	Resistant to
Amikacin	Ampicillin
Gentamicin	Ampicillin-sulbactam
Cefepime	Cefotaxime
Cefoperazone	Ceftazidime
Ciprofloxacin	Cephalexin
Imipenem	Ceftriazone
Netilmicin	Cefuroxime
Sulfamethoxazole-trimethoprim	

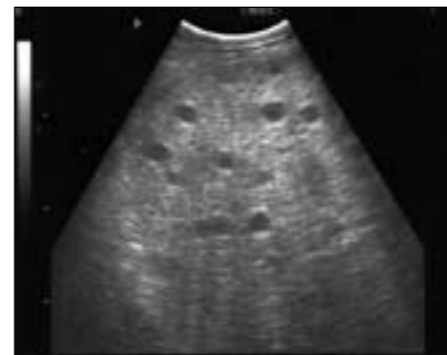
and the USA. Shao et al (2002) analysed 62 case reports worldwide and concluded that the mortality of blood dissemination of *C. violaceum* can be as high as 75%.

The most common route of entry is via non-intact skin and rarely followed near drowning. There was one report of infection via conjunctival exposure (Feldman et al, 1984) and one through an insect bite (Petrillo et al, 1984). *Chromobacterium* infection has been reported in individuals as young as 4 months old (Moore et al, 2001).

Figure 1. Multiple pustular lesions over right forearm.



Figure 2. Abdominal ultrasound: multiple hypochoic lesions in the liver.



Dr Hoi-Poh Tee is Clinical Specialist, Department of Internal Medicine, **Mr Alex L. Francis** is Senior Microbiologist, Department of Pathology, Hospital Tengku Ampuan Afzan, 25100 Kuantan, Pahang, Malaysia, **Dr Soon-Hin How** is Assistant Professor, Department of Internal Medicine, Kulliyah of Medicine, International Islamic University Malaysia, Malaysia

Correspondence to: Dr Hoi-Poh Tee

As with *Burkholderia pseudomallei* infection (melioidosis), immunocompromized individuals are more susceptible to *Chromobacterium* infection. Patients with chronic granulomatous disease are particularly vulnerable to *C. violaceum* infection (Macher et al, 1982). Glucose-6-phosphate dehydrogenase deficiency and leukocyte dysfunction were reported associations (Mamlok et al, 1987). Unfortunately, the patient in the current case died before being tested for possible immunodeficiency.

Symptoms of *Chromobacterium* infection vary, depending on the organs that are involved. Other than pneumonia, liver abscesses and skin pustules, as seen in this case, *C. violaceum* also causes local cellulites, lymphadenitis, nasopharyngeal abscess (Shao et al, 2002), exudative conjunctivitis, periorbital cellulites, meningitis and osteomyelitis.

Generally *Chromobacterium* demonstrates resistance to penicillins and cephalosporins, indeed the patient in this case did not respond to the initial antibiotics. Aldridge et al (1988) studied 25 antibiot-

ics against *C. violaceum* and concluded that ciprofloxacin was the most active antibiotic. The organism is generally susceptible to piperacillin, imipenem, chloramphenicol, doxycycline, trimethoprim-sulfamethoxazole and aztreonam.

It is important to differentiate *Chromobacterium* infection from the more common *B. pseudomallei* infection. Both organisms are tropical soil saprophytes and therefore common among agricultural workers. Other clinical similarities include formation of disseminated abscesses and higher prevalence among immunosuppressed persons. However, ceftazidime, which is recommended in melioidosis, is not effective against *C. violaceum*. Therefore delay in recognizing the less common *Chromobacterium* infection can prove fatal.

Conclusions

C. violaceum infection is a rare but highly fatal infection. The presentation mimics melioidosis and awareness of the disease can guide physicians in instituting early, appropriate antibiotics. **BJHM**

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Case Report

A 22-year-old Indonesian oil palm plantation worker presented with a 1-week history of fever associated with severe headache, pain in the right upper abdomen and seizure a day earlier. He had a cough with expectoration but no bowel symptom. There was no history of injury reported before the present illness.

The patient was febrile, dehydrated and drowsy on admission, with Glasgow coma score of 14/15. His blood pressure and pulse rate were 100/60 mmHg and 96/min respectively. Lung examination showed right-sided crackle, with no neck stiffness or any signs suggestive of immunocompromized state. Abdominal examination revealed tender hepatomegaly of 10 cm in length.

The initial chest radiograph showed opacity in the right middle lobe. Total white cells count was 11.1×10^9 /litre. Platelet count was 100×10^9 /litre with normal haemoglobin. Random blood glucose and renal profile were normal. Coagulation profiles were deranged with international normalization ratio of 4.5 and activated partial thromboplastin time ratio of 3.4. Liver function tests revealed alanine aminotransferase of 446 U/litre, aspartate aminotransferase of 253 U/litre and albumin of 30.5 g/litre.

The patient was given empirical intravenous ceftriaxone, metronidazole and acyclovir for possibility of meningo-encephalitis. Despite fluid resuscitation, he remained hypotensive and required inotropic support. On the second day, multiple pustular skin lesions (Figure 1) were noted on the patient's trunk, upper and lower limbs. Aspiration of the pustules revealed pus that contained gram-negative bacilli. Lumbar puncture was done and cerebrospinal fluid was normal. He required multiple platelet concentrates and plasma transfusions for disseminated intravascular coagulation. Abdomen ultrasound revealed multiple liver abscesses (Figure 2). He was intubated and ventilated for worsening respiratory failure. Samples of pus and blood cultures were positive for *Chromobacterium violaceum*. Intravenous imipenem 500 mg four times daily and ciprofloxacin 400 mg twice daily were administered. However, he died the same evening.