CASE REPORT

A FATAL CASE OF *CHROMOBACTERIUM VIOLACEUM* SEPTICEMIA IN HONG KONG

Siu-keung Edmond Ma[1], Shuk-kwan Chuang[1], Tze-leung Danny Cheung[2], Kai-man Kam[2] and Ho-fai Thomas Tsang[1]

[1]Surveillance and Epidemiology Branch, [2]Public Health Laboratory Services Branch, Center for Health Protection, Department of Health, Hong Kong Special Administrative Region

Abstract. *Chromobacterium violaceum* causes a rare infection in human, usually in tropical or subtropical areas. We report a fatal case of *C. violaceum* infection affecting a 40-year-old previously healthy man in Hong Kong. He presented with a wound infection and lymphadenitis. Despite multiple antibiotic treatment, including ciprofloxacin, he succumbed shortly after admission to the hospital. We report the epidemiological investigation findings and discuss the possible sources of infection. Physicians should be alert to this rare but fatal infection. Injury prevention and proper wound care should be emphasized to the public.

INTRODUCTION

Chromobacterium violaceum is a gram-negative rod with both pigmented and non-pigmented colony types. The pigmented strains produce a violet pigment known as violacein, hence the name. The bacterium is commonly found in soil and water. Woolley (1905) first reported a fatal infection of *C. violaceum* in buffaloes in 1904. Human infection has been rare after it was first documented in 1927 in Malaysia (Sneath *et al*, 1953). There have been only about 150 cases reported worldwide, mainly in tropical and subtropical areas, including Thailand (Sirinavin *et al*, 2005), Singapore (Chong and Lam, 1997), Korea (Lee *et al*, 1999), South America (Macher *et al*, 1982), Brazil (Siqueira *et al*, 2005), India (Chattopadhyay et al, 2002; Ray, 2004) and Australia (Huffam *et al*, 1998).

Correspondence: Dr Edmond Siu-keung, Field Epidemiology Training Programme, Room 232, 2/F, Surveillance and Epidemiology Branch, Center for Health Protection, Department of Health, 147C Argyle Street, Kowloon, Hong Kong Special Administrative Region.
Tel: 852-2768-9708; Fax: 852-2711-4847
E-mail: mo_fetp1@dh.gov.hk or edmondmask@yahoo.com.hk

CASE REPORT

On May 28 2005, the Center for Health Protection of the Department of Health, Hong Kong received a report of a fatal case of *C. violaceum* septicemia involving a 40-year-old previously healtyman. He initially presented with malaise and headache with a right arm wound of about 1 cm in diameter on May 24. On the same day he was treated by a general practitioner with amoxillicin, neomycin cream and symptomatic medications, including paracetamol, chlorphenamine, mefenamic acid and antacid. However, his symptoms worsened and he was referred to a regional hospital after consulting another general practitioner. On admission (1 day after onset of symptoms), he was afebrile and physical examination showed a wound abscess of approximately 2 cm in diameter on his right arm. Treatment with intravenous ampicillin and cloxacillin (1 g q 6 hours) was commenced, along with incision and

drainage of the wound abscess. However, his condition deteriorated rapidly with disseminated intravascular coagulation (thrombocytopenia 9 x 10^9/l, reference: 140 - 380 x10^9/l; neutropenia 0.09 x10^9/l), deranged liver function (ALT 350 IU/l, reference: <58 IU/l) and renal function (urea 16.0 mmol/l, reference: 3.4-6.9 mmol/l; creatinine 323 µmol/l, reference: 62-106 µmol/l). Intravenous broad spectrum antibiotics were added, including ciprofloxacin, metronidazole, netromycin and a third generation cephalosporin and he was resuscitated in the intensive care unit. His condition continued to deteriorate and he succumbed 22 hours after admission.

Both his right arm abscess tissue and blood culture grew a pigmented strain of *C. violaceum* (Fig 1). The bacterium was identified by biochemical tests using API 20E and Vitek GNI+ (bioMerieux China Ltd) and confirmed by 16S rDNA sequencing. The isolate was sensitive to ciprofloxacin, gentamicin, sulphamethoxazole-trimethoprim, imipenem, piperacillin/tazobactam but resistant to amoxicillin/clavulanate, ampicillin, cefotaxime and cefuroxime. Investigations for other infections were all negative, including stool culture for *Salmonella*, *Shigella*, *Campylobacter*, vibrios, and *E. coli* O157, enzyme immunoassay for IgM antibody against *Leptospira*, enzyme immunoassay for *Clostridium difficile* enterotoxin A, Widal's agglutination titer, Weil Felix titer, and polymerase chain reaction for rickettsiae.

Fig 1–*Chromobacterium violaceum* on standard plate count agar plate.

Post-mortem findings showed a right arm wound measuring 5 x 2 cm in diameter, which had prior surgical debridement. Scanty necrotic tissue was noted in the underlying soft tissue. Numerous yellowish nodules were seen throughout the lungs, liver parenchyma and spleen. Histology showed multiple foci with minimal acute inflammatory cell reaction and focal microabscesses in these sites. Splenic swab revealed scanty growth of *C. violaceum.*

The patient lived with his family in a village and used stream water for daily activities, such as bathing and cleaning. He worked as a property manager and did not need to work outdoors. Three days before the onset of symptoms, he stayed at a campsite in the countryside where he served as a trainer for 2 days. He was engaged in wall climbing and crawling in the grass at the campsite. There was a pond within the camp. However, there was no history of direct contact with the pond water. His three family members and the 130 persons who stayed at the campsite were all asymptomatic.

Water samples taken from the pond on May 31 showed positive cultures for *C. violaceum*. Water samples collected from the patient's home on June 2 were negative for the bacterium. Compared with the clinical isolate, *C. violaceum* isolated from the water samples in the environment exhibited more diverse biochemical profiles. The antibiotic susceptibility pattern of the isolates from environmental samples was similar to that of the patient's isolate, except that most were sensitive to cefotaxime. Analysis of the patterns of *Spe*I-restricted genomic DNA fragments as resolved by pulsed-field gel electrophoresis (PFGE) showed that the strain of *C. violaceum*

found in the patient was different from those isolated in the pond water.

DISCUSSION

We reported a case of *C. violaceum* infection which is rarely seen in Hong Kong. This case occurred in late May, which is early summer in Hong Kong. This follows the same seasonal pattern of *C. violaceum* infection reported in the literature in tropical and subtropical areas, from May through September (Macher *et al*, 1982; Chong and Lam, 1997; Lee *et al*, 1999; Siqueira *et al*, 2005).

Our patient was a previously healthy adult, which contrasts with the observations of most previously reported cases being children (Macher *et al*, 1982; Chong and Lam, 1997; Lee *et al*, 1999; Chattopadhyay *et al*, 2002; Siqueira *et al*, 2005; Sirinavin *et al*, 2005). Predisposing medical conditions to *C. violaceum* infection may include chronic granulomatous disease (CGD) and certain genetic conditions, such as glucose-6-phosphate-dehydrogenase (G6PD) deficiency (Macher *et al*, 1982; Ray *et al*, 2004; Sirinavin *et al*, 2005) but these were absent in our patient.

Common manifestations of *C. violaceum* infection are diffuse pustular dermatitis, sepsis, and multiple liver abscesses. Other presentations may include osteomyelitis, pneumonia, urinary tract infection, meningitis, otitis media, and orbital and periorbital cellulitis. The overall case fatality rate is as high as 65% (Chen *et al*, 2003) and with up to 80% having bacteremia or disseminated infection (Sirinavin *et al*, 2005). In our patient, the clinical presentation was cutaneous abscess formation followed rapidly by septicemia and disseminated intravascular coagulation, culminating in death within 3 days of the onset of symptoms.

The bacterium is usually susceptible to ciprofloxacin, aminoglycosides, chloramphenicol, tetracycline, imipenem and sulphamethoxazole-trimethoprim but resistant to penicillins, cephalosporins and aztreonam (Chen *et al*, 2003). Ciprofloxacin had been shown to be the most active antibiotic against *C. violaceum in-vitro* (Aldridge *et al*, 1988) and the clinical isolate in our patient was also sensitive to this antibiotic. Unfortunately, administration of ciprofloxacin failed to reverse the course of illness once septicemia and disseminated intravascular coagulation developed in our patient. Upon seeing a patient with rapidly progressive wound infection and who reports a positive exposure history to stagnant water or muddy soil in tropical or subtropical areas, clinicians should consider *C. violaceum* in the differential diagnosis.

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