FATAL *CHROMOBACTERIUM VIOLACEUM* SEPTICAEMIA

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ABSTRACT

A 19-year-old Malay male succumbed to a septicaemia caused by Chromobacterium violaceum 11 days after onset of illness. The organism is a common soil saprophyte and may be considered as contaminant on culture. It is essential to recognise its clinical significance in purulent processes so that appropriate therapy can be instituted. We report the first fatal case of Chromobacterium violaceum infection in the University Hospital, Kuala Lumpur.

Keywords: *Chromobacterium violaceum, fatal septicaemia*

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INTRODUCTION

Chromobacterium violaceum rarely cause human infection. The first reported human case occurred in Malaysia in 1927 and since then, fewer than 30 cases have been reported worldwide. Most reported cases had fatal outcomes although there were 3 reported cases of survival from Florida where the patients had been vigorously treated[1-4]. We present the first fatal case of *Chromobacterium violaceum* infection at the University Hospital, Kuala Lumpur, of a young man following a trauma sustained at work.

CASE REPORT

A 19-year-old Malay male working on a highway project was admitted to the district hospital on 23 June 1990 with a crush injury of his right hand. He was noted to have a compound fracture of the proximal phalanx of the right index finger with a 3-cm laceration on the dorsum of the hand. The extensor tendons and the neurovascular bundle on the radial side were severed. There was also a 2-cm laceration on the dorsum of the right middle finger with cut extensor tendon. The neurovascular bundle on the ulnar side was also severed. He also sustained a 5-cm laceration on the palm adjacent to the distal palmar crease. He underwent surgery where the fractures were stabilised with K wires. The tendons were repaired. The neurovascular bundle was not reanastomosed. The wounds were cleaned, debrided and the skin was left open. The wound on the palm was sutured. He was started on intravenous (I/V) cloxacillin 500 mg qid. He was then transferred to the University Hospital, Kuala Lumpur for further management of his injury. On admission, his general condition was satisfactory. He was febrile with a temperature of 38°C. Examination revealed an oedematous right hand but the wounds on the fingers and the palm were found to be clean. Circulation to all the fingers was found to be intact. Laboratory studies showed a normal haemoglobin level with white cell count of 11,700/ul and platelets were adequate. The blood urea and electrolyte levels were also within normal limits. The liver function test was also normal. The wounds were cleaned twice daily with normal saline. The hand was elevated. Intravenous cloxacillin was continued and gentamicin 80 mg tds was added to cover the gram negative organisms as the patient was still febrile. Blood was sent for culture. He developed an abscess on the dorsum of the right hand adjacent to the wound of the index finger. The abscess was incised and drained on the 5th day and pus swabs were sent for culture. His white cell count was noted to have increased to 12,800/ul. The wound was dressed daily with eusol. His temperature however continued to fluctuate. He was continued on intravenous cloxacillin and gentamicin as the bacteriological results were not known. On the 8th day he was found to be very toxic and his temperature rose to 40°C. He was also restless, confused, disoriented and delirious. In his confused state, he ran amok in the ward and tried to jump out of the window. He sustained a 4-cm laceration on the extensor aspect of his left forearm when he broke a window pane. His laboratory studies at this point showed a dropping haemoglobin level of 10.5 g/100 ml and an increasing white blood cell count of 14,500/ul. Blood urea and electrolytes were within normal limits. He was diagnosed to have organic brain syndrome secondary to sepsis. He was sedated with 5 mg I/V haloperidol. The cloxacillin was discontinued and replaced with I/V ceftazidime 1 g tds. He underwent emergency toilet and debridement treatment on the left forearm. The right index finger was noted to be gangrenous and the decision to amputate it was made in an effort to control the overwhelming sepsis. On the 9th day, the organism *Chromobacterium violaceum* was reported to have been isolated from the pus swab and blood culture. The organism was sensitive to gentamicin but resistant to ceftazidime. The latter was hence discontinued. On the 10th day he became more toxic with generalised septic spots and basal crepitation in the right lower zone. His respiratory rate was 40 per minute and regular, BP was 100/160 mmHg. Urinary output was noted to be poor. White blood cell count increased further to 32,000/ul and platelets dropped to 23,000/ul. Chest X-ray revealed right lower lobe pneumonia. He was then transferred to the Intensive Care Unit for management of the pneumonia, acute renal failure and septicaemia. The gentamicin was stopped and replaced with I/V netilmicin 80 mg tds to which the organism was also sensitive. Intravenous metronidazole was also added. The right hand remained oedematous but there was no evidence of the presence of abscess. Abscesses elsewhere were sought. The liver was enlarged but ultrasound of the liver did not show any abscesses. Computerised tomography of the brain was also performed and this revealed no abscess. HIV test

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was also negative. Further bacteriological swabs from the wound isolated the same *Chromobacterium violaceum*. On the 11th day, in an effort to control the spreading infection, right mid forearm amputation was performed. Unfortunately, shortly after the operation the patient had a cardiac arrest. Resuscitation was unsuccessful and he succumbed to the infection on the 11th day post-trauma.

Histopathological findings

Post mortem was refused by the patient's relatives. The histology of the amputated index finger revealed extensive areas of necrosis and inflammation with microabscesses. The right hand and forearm showed extensive necrosis involving the bone with abscesses and colonies of gram negative bacilli. Needle biopsy of the liver revealed liver necrosis with acute hepatitis compatible with septicaemia.

Bacteriological findings

The organism *C. violaceum* was isolated from pus swabs, blood culture and tracheal aspirate taken from the patient. The organism grew easily on nutrient agar, blood agar and MacConkey agar. The colonies were smooth, round, convex and shiny measuring 1-2 mm in size after 24 hours incubation at 37°C. There was haemolysis on blood agar plates and deep purple pigment. The pigment was found to be soluble in alcohol but not in water or chloroform. The gram stained smear showed a pleomorphic gram negative rod. Biochemically, the organism gave a negative indole, methyl red, Voges- Proskauer and citrate reactions. It fermented glucose with production of acid but no gas while sucrose, mannitol, dulcitol and lactose were not fermented. Oxidase reaction was positive. The arginine dihydrolase reaction was positive while the ornithine and lysine decarboxylases were negative. Nitrate was reduced. Urease reaction was negative.

The antibiogram of the organism showed that it was sensitive to the aminoglycosides, gentamicin , netilmicin, piperacillin and amikacin but resistant to the cephalosporins, cefoperazone, ceftazidime and cefuroxime along with ampicillin.

DISCUSSION

The organism *Chromobacterium violaceum* has not been seriously regarded as a cause of fatal septicaemia. With the exception of sporadic case reports, little is known or heard of its potential pathogenic nature. The organism is typically a small, pleomorphic gram negative bacillus with polar and lateral flagella and it grows readily on ordinary culture media. The biochemical reactions of our isolate do not differ from those already described for the organism. The colonies were large and pigmented dark purple[5].

Infection with the organism is largely confined to the tropical and subtropical climates. Several cases reported in the USA were from the south eastern parts of the country. Other reported cases were from Malaysia, Australia, France, Africa and India. The organism is ubiquitous in nature, being a soil saprophyte and water inhabitant[6].

Wooley in 1905 first described its pathogenicity in fatal infections of water buffaloes in the Philippines. The organism has also been reported in several mammals especially the monkeys or gibbons, also in pigs and cattles. In 1927, the first human case was reported in Malaya. The infection of this organism in man is rare , but when its occurs, it may cause a highly fatal septicaemia with formation of internal abscesses in multiple organs. It may also cause localised abscess diarrhoea and urinary tract infection[7].

The route of infection is unclear. As in our patient, most other patients had a history of trauma or wound which had been contaminated with soil or water. In some, the injury may have been so minor that it is not recalled by the patient. In those cases presenting with diarrhoea, ingestion could be the mode of infection. In our patient the tracheal secretion gave a scanty growth of the organism and therefore was likely to be present as a transient coloniser rather than an infection following inhalation.

The clinical features shown by this patient were not unlike most other cases reported. The onset of symptoms in our patient was about 4 days post trauma. Considerable time may elapse between onset of symptoms and that of systemic involvement. In a review of human infections due to *Chromobacterium violaceum* by Johnson et al, they showed that the period of onset of illness to death varies between 5 days to 15 months[7]. Our patient showed signs and symptoms of septicaemia after 6 days in the ward. A history of injury is the most common obtained from infected patients. From the injury, an abscess develops which does not heal despite repeated treatment. Quite rapidly and suddenly, the patient will become septicaemic and the general condition progressively worsens. Characteristically, the patient will present with necrotic lesions on the body as were seen in our patient. In some patients, there are evidence of personality changes. Patients were found to be confused, disoriented and have alternating lucid intervals[6]. Our patient was diagnosed as suffering from an organic brain syndrome when he was found to be confused and disoriented on the 7th day of his illness.

The infectious process, when systemic, will ultimately form abscesses in multiple organs including lungs, liver, spleen and heart. Death generally follows the overwhelming septicaemia. Our patient showed an enlarged liver although no abscess was detected by ultrasound. The extent of our patient's illness could not be ascertained as autopsy was refused. Sepsis with liver abscess appears to be the characteristic feature in the fatal cases of C. *violaceum* infection. No patient with liver abscess has survived.

Little is known of any predisposing factors or immunodeficiency status that are associated with the fatal form of this infection. Macher et al in their review of 12 *C. violaceum* infections in the United States found that 3 of these patients had an underlying chronic granulomatous disease thus suggesting that patients with underlying neutrophil dysfunction syndrome are at special risk of developing the fulminant form of the infection[8]. Our patient had no known history of underlying disease or immune suppression.

Many of us are unfamiliar with the fulminant nature of the infection caused by this organism. Microbiologists may regard it as culture contaminant when isolated. The organism, particularly the non-pigmented form, will be dismissed as less virulent. Therefore few patients have received adequate, vigorous and definitive antibiotic treatment early in the infection. Like the strain isolated from this patient, the organism is often sensitive to tetracycline, chloramphenicol, erythromycin and gentamicin although resistant to the cephalosporins.

CONCLUSION

Chromobacterium violaceum infection in human is rare and cases are sporadically reported over the years. The potential pathogenic nature of this organism has clearly been demonstrated by the very fulminant nature of its infection and the many fatal cases it caused. When isolated from purulent processes, its presence must be regarded as clinically significant and thus must be treated early and aggressively if death is to be prevented.

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