CASE REPORT

A Fatal Case of Pulmonary Chromobacterium Violaceum Infection in an Adult

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SUMMARY

Chromobacterium violaceum **is a gram negative, facultative anaerobic coccobacillus. Human infections are rare and usually occur after exposure to contaminated soil or water. Infections can present with fulminant septicemia, multiple abscesses and rapidly spreading soft tissue infections. Here we present a fatal case of pulmonary *Chromobacterium violaceum* infection following aspiration of drain water. Treatment with fluroquinolones in combination with either co-trimoxazole or amikacin has been described in successfully treated cases.**

KEY WORDS:
Chromobacterium violaceum, aspiration, pulmonary

INTRODUCTION

Chromobacterium violaceum is a gram negative, facultative anaerobic coccobacillus. It is usually found in stagnant water and soil in the tropical and sub-tropical regions. Human infections are rare and only isolated, sporadic cases have been reported. Cases which have been described tend to run a rapidly progressive course with high mortality rates.

CASE REPORT

A 45 year old Indian gentleman presented to us with an acute onset of fever associated with cough, progressive shortness of breath and pleuritic chest pain for one day. He gave a history of falling into a drain two days earlier after losing control of his motorbike during a thunderstorm. His face was submerged in the muddy drain water and he claimed to have swallowed some of it.

The patient was previously under regular follow-up for chronic alcoholic liver disease and chronic venous ulcer over his left leg. He did not have Diabetes Mellitus and never had any contact with tuberculosis patients before. He was a chronic smoker and worked as a supervisor in an oil palm estate.

On admission, the patient was conscious and alert. He was noted to be tachypnoeic and tachycardic, with a respiratory rate of 26 breaths per minute and pulse rate of 100 beats per minute. His blood pressure was 128/78 mmHg and oxygen saturation on room air was only 91%. His lungs had bilateral generalized coarse crepitations. Chest X-ray showed bilateral patchy opacities over the middle and lower zones. The patient also had marked leukocytosis (WCC 21,900/ul) with mild anemia (10.8 g/dl) and thrombocytopenia (114,000/ul). Our initial diagnosis was aspiration pneumonia and he was started on IV Ceftazidime 2 g tds and IV Metronidazole 500 mg tds.

Table I: Antibiotic sensitivity for Chromobacterium violaceum cultured from patient's endotracheal secretions

Antibiotic	Sensitive	Resistant
Co-trimoxazole	X	
Gentamicin	X	
Amikacin	X	
Tetracycline	X	
Imipenem/Meropenem	X	
Superazon	X	
Ciprofloxacin	X	
Cefepime	X	
Tazosin	X	
Ceftazidime	X	
Chloramphenicol	X	
Cefuroxime		X
Ceftriaxone		X
Cefotaxime		X
Augmentin		X

He deteriorated rapidly and was transferred to the Intensive Care Unit (ICU) on the same day. He was electively intubated and ventilated as he was becoming increasingly drowsy and his arterial blood gas (ABG) showed type 1 respiratory failure with metabolic acidosis (pH 7.174, pCO_2 30.5 mmHg, pO_2 71.3 mmHg, HCO_3 11.0 mmol/l, BE -17.5, O_2 sat 90.3%). His initial renal profile showed hyponatremia and mildly elevated serum creatinine (urea 5.5 mmol/l, Na 125 mmol/l, K 4.8 mmol/l, creatinine 123 umol/l). Liver function test was consistent with his underlying chronic liver disease (serum albumin 29 g/l, totall bilirubin 87.7 umol/l, ALP 154 iu/l, AST 116 iu/l, AST 38 iu/l).

The patient failed to provide any sputum samples before he was intubated. Secretions from the endotracheal tube were obtained for bacterial culture and sensitivity (C&S) and Acid Fast Bacilli (AFB) direct smear in the ICU. The first endotracheal secretion C&S grew *Chromobacterium violaceum* and *Enterobacter aerogenes*. Direct smears for AFB were negative. The antibiotic sensitivity for *Chromobacterium violaceum* is given in Table I.

The patient's fever failed to settle after six days of IV Ceftazidime despite the organism being sensitive to it and IV Ciprofloxacin 200 mg bd was started. *Chromobacterium*

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violaceum was again isolated from the endotracheal secretion 8 days later. The sensitivity pattern was similar to the initial culture.

The patient's condition deteriorated progressively with worsening of chest radiograph appearance. He went on to develop septicemic shock requiring inotropic support, acute renal failure and disseminated intravascular coagulation. His conscious level failed to improve and he was noted to have bilateral chemosis. CT brain revealed no evidence of cerebral abscess or other lesion. The patient passed away 15 days later. Repeated blood cultures were negative.

DISCUSSION

Chromobacterium violaceum infections in humans are not common and they usually occur following contaminated traumatic soft tissue injuries or aspiration after near drowning episodes. Many practicing physicians may not be aware of its clinical significance when the organism is isolated from a patient's specimen. Being part of the normal soil flora, the culture results may initially be dismissed as 'contamination'.

Chromobacterium violaceum can be easily cultured on nutrient agar and it produces colonies with characteristic dark violet metallic sheen. Both pigmented and non-pigmented strains have been reported[1]. *Burkholderia pseudomallei* colonies may be mistakenly identified as *Chromobacterium violaceum* by certain identification methods. In fact, the clinical presentation of *meliodosis* is very similar to that of *Chromobacterium violaceum*. Both can present with fulminant life-threatening septicemia, multiple abscess formation in various organs e.g. lungs, liver and spleen and rapidly spreading soft tissue infections. While most cases of *meliodosis* affect those with diabetes mellitus and other underlying immune compromised states, reported cases of *Chromobacterium violaceum* infections were usually in previously healthy children[2] or adults[1] with the risk factor of exposure to contaminated soil or water. Both diseases are more prevalent in the tropics.

There are no standard guidelines for the treatment of *Chromobacterium violaceum* infections. The use of fluoroquinolones e.g. ciprofloxacin or perfloxacin in combination with either co-trimoxazole[3] or amikacin[4] have been frequently described in successfully treated cases. Response to penicillins and cephalosporins has been dismal and strains which are resistant to broad spectrum cephalosporins have been reported[1]. Prompt surgical debridement of infected tissue is also imperative. Despite appropriate therapy, *Chromobacterium violaceum* infection remains a potentially fatal disease with mortality rates in excess of 60%[5].

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