CASE REPORTS

HUMAN *CHROMOBACTERIUM VIOLACEUM* INFECTION IN SOUTHEAST ASIA: CASE REPORTS AND LITERATURE REVIEW

Anupop Jitmuang

Internal Medicine Unit, Nan Hospital, Nan, Thailand

Abstract. *Chromobacterium violaceum* infection in humans is a rare tropical and subtropical disease. The awareness of this organism is limited in spite its ubiquitous distribution. Several cases have been reported from Southeast Asia. A localized infection followed by an overwhelming septicemia and metastatic lesions is the usual pattern of this illness. Optimal antimicrobial treatment and duration are unknown. Consequently, the outcome is usually fatal. The study reported two patients who suffered from fulminant *Chromobacterium violaceum* sepsis with disseminated infection, and reviews the literature for cases reported from Southeast Asia.

INTRODUCTION

Chromobacterium violaceum is a gram-negative bacilli that can be isolated from natural aquatic habitats in tropical and subtropical regions. *Chromobacterium violaceum* is the exclusive species of this genus that causes human disease (Steinberg and Rio, 2005). In spite of ubiquitous distribution, human infection with this organism is rare, and awareness of the disease is limited (Chattopadhyay *et al*, 2002). The pattern of illness usually presents with a contaminated inoculation site, localized disease, regional lymphadenopathy, then hematogenous spread to visceral organs (Fisher *et al*, 2004). The author reports two cases of *Chromobacterium violaceum* infection, both with fatal outcomes.

CASE REPORTS

Case 1

A 6-year-old boy living in Nan Province, northern Thailand, presented with fever and right otitis externa for two days. He was previously well except for a history of perianal abscess at age 3 years. There was no history of exposure to soil or water via recreational activity or accident. An otolaryngologist prescribed him with oral cloxacillin and chloramphenicol ear drops. The infection progressed to a posterior auricular abscess with swelling of the right mastoid area, plenty of pus persistently drained from his right external auditory meatus. The same otolaryngologist performed an incision and drainage of the posterior auricular abscess after 7 days of ambulatory treatment. The patient was also admitted to Nan Hospital on 11 November 2002.

On admission, he was febrile at 39ºC, the respiratory rate was 22 breaths/minute, the heart rate was 100 beats/minute and his blood pressure was 110/70 mmHg. His conjunctiva were pale without icterus. Examination of the right ear showed a large amount of pus, but the site of discharge and the tympanic membrane could not be seen. Multiple pustules were identified on his face and trunk, approximately 1 mm in size. The cardiopulmonary examination was unremarkable. Abdominal dis-

Correspondence: Dr Anupop Jitmuang, Internal Medicine Unit, Nan Hospital, 1 Voravichai Road, Tambon Nai Weing, Amphoe Mueang, Nan 55000, Thailand.
Tel: 66 (054) 710138; Fax: 66 (054) 710977
E-mail: anupopmix@yahoo.co.th

tension and generalized tenderness were noted, with no hepatosplenomegaly or lymphadenopathy. Laboratory investigations revealed a leukocyte count of 40,000/mm^3 with 93% neutrophils and 7% lymphocytes, a hemoglobin of 8.8 g/dl, and a platelet count of 155,000/mm^3. A Gram's stain from the pustular lesion demonstrated numerous neutrophils and a few gram-negative bacilli. Renal and liver function tests were not recorded. Staphylococcal septicemia was the provisional diagnosis.

Intravenous cefazolin and gentamicin combined with chloramphenicol ear drops were commenced. One day after admission, he developed septic shock, acute renal failure, and metabolic acidosis. The pediatrician suspected peritonitis but the consultant surgeon did not think he had a surgical abdomen, but instead a paralytic ileus secondary to severe infection. Forty-eight hours after admission, purulent discharge still flowed from his right ear. A computed tomography scan of the temporal bone showed a complicated right mastoiditis. The patient's status deteriorated, necessitating mechanical ventilation and inotropic agents. He was sent to the operating room for emergency simple mastoidectomy. Circulatory collapse and cardiac arrest ensued, and he succumbed intraoperatively.

Blood cultures and pus cultures from the skin pustule and right ear obtained on the day of admission all yielded *Chromobacterium violaceum*. All isolations were susceptible to ceftazidime, gentamicin, amikacin, and cotrimoxazole, but were resistant to ampicillin and cefazolin by disc diffusion method. Drug susceptibility to quinolones was not performed. The patient's parent did not consent to carry out an autopsy, the cause of death was not completely determined.

Case 2

A 54-year-old female farmer living in Tha Wang Pha, a local district of Nan Province, presented with an infected ulcer on her left forearm for three days after working in a paddy field. She had no underlying disease. She had undergone treatment with oral cloxacillin and used an herbal application for the ulcer. On 30 September 2007, she went to a district hospital due to worsening of the lesion. A primary physician found she was febrile and had an oval shaped, necrotic irregular border, pus-filled ulcer on the left forearm (Fig 1). Debridement was performed in addition to empiric intravenous cloxacillin and gentamicin therapy. After 3 days of hospitalization at the district hospital, her condition suddenly deteriorated. She became sepsis with a rising creatinine level (0.8 to 3.4 mg/dl). She was then transfered to Nan Hospital. At Nan Hospital, she appeared toxic, drowsy, and icteric. Her blood pressure was 90/50 mmHg, temperature was 37ºC, respiratory rate was 28 breaths/minute, and pulse rate was 100 beats/minute. The cardiopulmonary examination was unremarkable. The abdomen was distended with hepatomegaly. There was no splenomegaly or lymphadenopathy. A punched-out ulcer with minimal pus from the wound post-debridement was detected. A swab was sent for Gram's stain and culture. Two specimens of blood were drawn for culture. Laboratory investigations showed a hemoglobin of 13.3 g/dl, a leukocyte count of 9,000/mm^3 with 94% neutrophil, and a platelet count of 85,000/mm^3. The serum bilirubin was 3.25 mg/dl, alanine transaminase was 263 U/l, aspartate transaminase was 365 U/l and alkaline phosphatase was 184 U/l. The serum creatinine was 2.97 mg/dl. A chest radiography demonstrated bilateral interstitial infiltration. Intravenous cloxacillin and ceftriaxone were the initial antimicrobial therapy. Two hours after admission, the patient developed respiratory distress and metabolic acidosis necessitating intubation, volume replacement, vasopressors and mechanical ventilation. The laboratiory identified a few gram-negative bacilli from the pus swab, and ceftazidime was

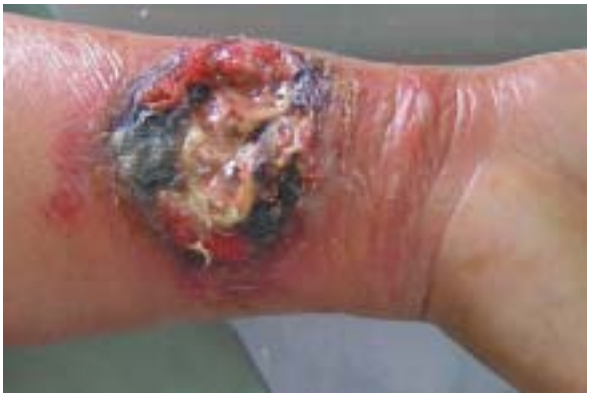

Fig 1–An oval, necrotic, irregular border, pus-filled ulcer on the left forearm of case 2 was partially treated prior to progression to fulminant septicemia.

Fig 2–The characteristic round, discrete, nondiffusible, dark-purple colonies identified on both pus and two sets of blood cultures from case 2.

substituted to ceftriaxone for cover pseudomonal infection and melioidosis. Soon after the intensive treatment, she went into cardiac arrest and failed to be resuscitated. She was dead five hours after admission. A post-mortem examination was refused by patient's relatives.

Three days after her death, the characteristic round, discrete, non-diffusible, dark-purple colonies of gram-negative bacilli were identified on both the pus culture and the two sets of blood cultures (Fig 2). These results with the subsequent biochemical tests were consistent with *Chromobacterium violaceum*. All isolates were susceptible to gentamicin, amikacin, norfloxacin, co-trimoxazole, chloramphenicol and tetracycline, but resistant to ampicillin, cefazolin, and ceftazidime by disc diffusion method.

DISCUSSION

Chromobacterium violaceum, formerly named *Bacillus violaceum manilae*, was first discovered in 1881 by Bergonzini. It was capable of causing fatal epidemic septicemia in water buffaloes in the Philippines in 1904 described by Wooley (Macher *et al*, 1982). Lesslar JE reported a man with fatal septicemia and liver abscess caused by *Chromobacterium violaceum* which was the first case report in humans (Sneath *et al*, 1953). Optimal growth for the organism is at 20ºC to 37ºC and thus is restricted geographically between latitudes 35 degrees North and 35 degrees South (Way *et al*, 2007). As a mesophilic bacterium *Chromobacterium violaceum* is unable to survive at 4ºC (Macher *et al*, 1982; Dromigny *et al*, 2002). Most cases have been reported from Southeast Asia (Sneath *et al*, 1953; Johnson *et al*, 1971; Hassan *et al*, 1993; Ti *et al*, 1993; Sagin *et al*, 1994a,b; Chong and Lam, 1997; Roberts *et al*, 1997; Sirinavin *et al*, 2005), India (Chattopadhyay *et al*, 2002; Ray *et al*, 2004), and southeastern United States, especially in Florida (Black and Shahan, 1938; Macher *et al*, 1982; Feldman *et al*, 1984; Simo *et al*, 1984). This study reported two cases of fatal *Chromobacterium violaceum* infection and reviewed the reported cases from Southeast Asia as shown in Table1.

In Southeast Asia, the infection was mostly recognized in Malaysia, where the first human infection was discovered. Patient ages ranged from 10 months to 54 years, however,

an incomplete record of patient's ages in the literature was noted. Most of the case reports worldwide were in young people (Macher *et al*, 1982; Ponte and Jenkins, 1992; Shao *et al*, 2002; Ray *et al*, 2004). Males were more affected than females, with concordance between series (Johnson *et al*, 1971; Macher *et al*, 1982; Sirinavin *et al*, 2005; Chang *et al*, 2007). Although the infection often occurs after exposure to contaminated water or soil via non-intact skin, five patients (25%) had a history of accidental skin injury during work or other activity. The injury may have been so minor that it was not recalled by the patient (Hassan *et al*, 1993). Two patients in the review had only diarrhea, leading the authors to postulate that the gastrointestinal tract is another portal of infection. Additionally, a few cases of *Chromobacterium violaceum* sepsis were associated with near-drowning (Ender and Dolan, 1997).

A skin lesion or localized adenitis followed by overwhelming septicemia with necrotizing metastatic lesions is usually the manifestation of this infection (Victorica *et al*, 1974). Case report No.1, a pediatric patient, presented with uncommon features. To our knowledge, this is the first case report of acute purulent otitis externa with complicating mastoiditis and fulminant septicemia caused by *Chromobacterium violaceum* in Southeast Asia. Case report No. 2, an adult patient, had a local skin infection, then rapidly developed severe sepsis which is a typical presentation. Rarely, meningitis (Macher *et al*, 1982; Ray *et al*, 2004), eye infection (Feldman *et al*, 1984; Simo *et al*, 1984), arthritis (Macher *et al*, 1982), urinary tract infection (Sneath *et al*, 1953; Johnson *et al*, 1971), and osteomyelitis (Tucker *et al*, 1979) have also been reported. Table 1 shows that fourteen cases (70%) had disseminated infection. The common sites of dissemination were liver, lung, spleen, and skin. Brain abscess, a rare complication, was found only in one case. Neither case had radiological investigations or a post-mortem evaluation, thus evidence for abscesses elsewhere could not be determined. Seven cases (35%), including the presented case, became septic following a local skin lesion. Cutaneous involvement is common, especially pustular lesion with surrounding erythema, and may progress to ulceration (Brown *et al*, 2006). Ecthyma gangrenosum was also repoted (Brown *et al*, 2006). Six cases (30%) suffered from localized disease: urinary tract infection in 2 cases, diarrhea in 2 cases, and local skin infection in 2 cases. An underlying immunocompromised state was identified in two pediatric cases. One had a low T-cell CD_4 number which was undiagnosed previously (Chong and Lam, 1997), and another was diagnosed with chronic granulomatous disease (Sirinavin *et al*, 2005). Some papers reported that *Chromobacterium violaceum* caused infection in pediatric patients with an immunodeficient state, such as chronic granulomatous disease, neutrophil dysfunction or severe polymorphonuclear G-6-PD deficiency (Macher *et al*, 1982; Mamlok *et al*, 1987; Sirinavin *et al*, 2005). In persons with the aforementioned syndromes, polymorphonuclear leukocytes and monocytes lack the ability to produce the oxygen metabolites required to kill phagocytised bacteria (Ponte and Jenkins, 1992). Consequently, the patient is susceptible to severe infection and dissemination to multiple organs caused by catalase-positive organisms, such as *Staphylococcus aureus*, gram-negative Enterobacteriaceae, yeasts, *Nocardia* sp, and *Chromobacterium violaceum*. Moreover, virulent strains of *Chromobacterium violaceum* produce an endotoxin and can withstand attack from phagocytic cells (Brown *et al*, 2006). Anyone infected with *Chromobacterium violaceum* should be evaluated for immunocompromised status, although there is no strong evidence to support an immunodeficiency as a risk factor for the infection (Teoh *et al*, 2006). However, most patients in

Table 1 died before investigation of immunologic status.

In both presented cases, *Chromobacterium violaceum* was uncovered early by detection of gram-negative bacilli from pus specimens, though the morphology of this orgaism is difficult to differentiate from other enteric gram-negative bacilli. The physicians considered staphylococcal or other gram-negative infections as the likely cause of disease. Because of its rarity, *Chromobacterium violaceum* may be misinterpreted as a contaminant when cultured. Growing well on standard culture media, most reported cases in Table 1 were diagnosed by identification of deep violet colonies, named violacein, from blood samples, abscess fluids, or skin exudates. However, nonpigmented strains account for approximately 9% of *Chromobacterium violaceum* samples (Brown *et al*, 2006). Loss of pigmentation can result from long term serial passage in the laboratory (Ponte and Jenkins, 1992). A few case reports infected with the nonpigmented strain give evidence that pathogenicity does not appear to be related to pigment production (Desjardins *et al*, 1999; Lee *et al*, 1999). Nonpigmented strains may be easily confused with *Aeromonas* sp, *Pseudomonas* sp, or *Vibrio* sp (Feldman *et al*, 1984).

In addition to the violet, alcohol soluble, nondiffusible pigment, diagnosis also requires several biochemical tests. Most *Chromobacterium violaceum* isolates produce oxidase and catalase but are negative for Voges-Proskauer reaction and esculin (Lee *et al*, 1999). Fermentation of D-glucose, mannitol, maltose, and lysine decarboxylase and ornithine decarboxylase activities can differentiate *Chromobacterium violaceum* from *Vibrio* sp or *Aeromonas* sp (Lee *et al*, 1999). An innovative technique using multiplex PCR has been developed which allows the rapid differentiation of clinical isolates from *Burkholderia pseudomallei* that cause a similar clinical picture (Scholz *et al*, 2006). This is not practicable in most diagnostic laboratory settings and is not available worldwide.

One of the difficulties encountered in management is the choice of antibiotics for empiric therapy before the results of cultures are available (Ti *et al*, 1993). The first presented case was considered to be and treated as staphylococcal septicemia, even though gram-negative bacilli from the pus swab were detected. The second case was initially diagnosed as an infected wound, like the first patient, and was empirically treated as a staphylococcal wound infection. After identification of gram-negative bacilli, the second patient was deemed to have a severe gram - negative septicemia, and antibiotics were changed shortly thereafter. Human infections with *Staphylococcus aureus*, *Burkholderia* sp, or gram - negative Enterobacteriaceae are common in Southeast Asia, therefore *Chromobacterium violaceum* was not considered in the differential diagnosis. Four cases, including the presented case, as seen in the Table 1, had melioidosis as the suspected cause of septicemia initially (Ti *et al*, 1993; Chong and Lam, 1997; Sirinavin *et al*, 2005). There are several commonalities between *Chromobacteriun violaceum* infection and melioidosis, such as the area of endemicity, soil and water saprophytes, capability to cause acute septicemia associated with multiple abscesses, and a high mortality rate (Ti *et al*, 1993).

Review antibiotic susceptibilities in Southeast Asia revealed most isolates were susceptible to chloramphenicol, aminoglycosides, co-trimoxazole, tetracycline, quinolones, and carbapenems, as shown in Table 2. The specimens were generally resistant to ampicillin and cephalosporins. Ceftazidime, which is recommended for melioidosis, has equivocal benefit for the infection. Four patients, including the presented case, who were treated with ceftazidime, had poor outcomes (Hassan *et al*, 1993; Ti *et al*, 1993; Chong and Lam, 1997). Aminoglycosides were used in seven

Table 1

Clinical presentation of patients infected with *Chromobacterium violaceum* in Southeast Asia.

Ref	Age (years)	Sex	Country	Clinical presentation	Specimen	Treatment[1]	Outcome	Route of infection
13, 27	NA	NA	Malaysia	Pyema, liver abscess	NA	NA	Died	Unknown
13, 27	NA	NA	Malaysia	Pyema, liver abscess	NA	NA	Died	Unknown
13, 27	NA	NA	Malaysia	UTI	NA	NA	Died	Unknown
13, 27	NA	NA	Malaysia	Pyema, liver abscess	NA	NA	Died	Unknown
13, 27	NA	NA	Malaysia	Local skin abscess	NA	NA	No record	Unknown
13, 27	20	M	Malaysia	UTI	Urine	NA	No record	Unknown
13, 27	25	M	Malaysia	Ulcer of left thigh and regional adenitis, later pyema with liver and pleural abscesses	Pus (liver, pleura)	Ineffective antibiotics	Died	Unknown
13, 27	Adult[b]	M	Malaysia	Mild diarrhea	Stool	NA	Recovered	Unknown
13, 27	35	M	Malaysia	Diarrhea, rectal bleeding	Stool	NA	Recovered	Unknown
30	3	F	Singapore	Local papule, later extensive cellulitis of the left foot	Pus (skin)	Effective antibiotics, surgical drainage	Recovered	Unknown
30	24	M	Singapore	Severe sepsis, multiple abscesses (liver, lung, skin, brain)	Pus (skin), blood	Effective antibiotics	Died	Unknown
12	19	M	Malaysia	Crush injury, infected wound, later severe sepsis with pneumonia	Pus (skin), blood	Ineffective antibiotics, surgical drainage	Died	Con-taminated crushed hand
21	10 months	F	Malaysia	Local skin infection, then multiple septic foci (digits, nose, eye, joint)	Pus (skin), blood	Effective antibiotics, debridement	Recovery	Accidental cuts and grazes
21	9	M	Malaysia	Multiple ulcers on lower limbs, later sepsis	Blood	Ineffective antibiotics,	Died	Cut wound after fell on muddy gravel road
22	4	M	Malaysia	Chronic ulcer on foot, later pneumonia with severe sepsis	Blood	NA	Died	Unknown
5	11	F	Singapore	Severe sepsis, pneumonia, skin pustules	Blood	Ineffective antibiotics,	Died	Unknown
20	27	M	Thailand	Local infected wound,then exudative tonsillitis,later deep neck infection with septic emboli (liver,skin)	Pus (mastoid), tracheal aspirates	Two effective antibiotics, mastoidectomy, debridement and drainage	Recovered	Cut his leg on coral
26	3.3	M	Thailand	CGD,chronic relapsed illness with multiple abscesses (lung, liver, spleen)	Pus (liver, spleen), BAL	Four effective antibiotics,	Recovered	Unknown
Case No.1	6	M	Thailand	Otitis externa, mastoiditis, later severe sepsis with multiple abscesses (skin, post-auricular)	Pus (skin, mastoid), blood	Ineffective antibiotics, mastoidectomy	Died	Unknown
Case No.2	54	F	Thailand	Infected wound,later severe sepsis	Pus (skin) blood	Ineffective antibiotics, debridement	Died	A skin ulcer after culti-vation

Ref=Reference, NA=Not available, M=Male, F=Female, CGD=Chronic granulomatous disease, UTI=Urinary tract infection, BAL=Bronchoalveolar lavage, [a]Treatment comprised of antibiotics, surgical intervention, or both. Effective antibiotics defined as therapy with one or a combination of ciprofloxacin, carbapenems, co-trimoxazole, and chloramphenicol. Ineffective antibiotics defined as regimens not consisting of any effective drugs. [b]Age was not recorded.

Table 2
Antimicrobial susceptibilities of *Chromobacterium violaceum* isolates from patients' specimens in Southeast Asia.

Studies	Antimicrobial agents and susceptibilities[a]									
	Ampicillin	Cephalosporins[b]	Ceftazidime	Piperacillin	Aztreonam	Imepenem, Meropenem	Amino-glycosides	Quinolones	Chloram-phenicol	Co-trimoxazole	Tetra-cycline
Ti *et al*, 1993											
case 1	R	R	R	S			S		S	S	S
case 2	R	R	R	S			S	S	S	S	
Hassan *et al*, 1993	R	R	R	S			S				
Sagin *et al*, 1994a,b	R	R	S				S	S		S	S
Chong *et al*, 1997					S		S	S	S		
Roberts *et al*, 1997	R	R	R	R		S	S	S	S	S	S
Sirinavin *et al*, 2005	R	R	R			S	S	S		S	
Present study											
Case 1	R	R	S				S			S	
Case 2	R	R	R				S	S	S	S	S

[a]By disc diffusion method, R = Resistant, S = Susceptible
[b]First and second generation cephalosporins

cases, including both the presented cases, but the outcomes were poor: one patient was handicapped and the others died (Hassan *et al*, 1993; Ti *et al*, 1993; Sagin *et al*, 1994; Chong and Lam, 1997). This brings into doubt the usefulness of aminoglycosides for empiric therapy (Desjardins *et al*, 1999). *Chromobacterium violaceum* consists of a large number of gene products associated with drug resistance, for instance, penicillin binding proteins, beta-lactamase precursors (cephalosporinase), and mutidrug resistance proteins (drug efflux) (Fantinatti-Garboggini *et al*, 2004). Hence, MICs should be performed in patients infected with *Chromobacterium violaceum*. The appropriate antibiotics and duration of treatment are still unknown. As can be seen in Table 2, one or more of the following in combination are likely to be effective in treatment: co-trimoxazole, chloramphenicol, quinolones, or carbapenems. The detection of an internal organ abscess may also require surgical drainage. Long-term treatment is needed to fully eradicate the organism and resolve potentially fatal abscesses (Moore *et al*, 2001). In the three cases that survived invasive infection as seen in Table 1, the duration of therapy was at least 3 months (Sagin *et al*, 1994; Roberts *et al*, 1997; Sirinavin *et al*, 2005). Because of frequent relapse, some literature suggested an additional 4 weeks to 3 months with oral antibiotics after a period of parenteral therapy (Ponte *et al*, 1992; Shao *et al*, 2002).

Twelve of twenty patients died.

The overall mortality rate was 60%, two cases had missing data. All of the fatalities manifested as an invasive infection with disseminated disease. Early recognition of the organism, appropriate treatment and duration, and adequate surgical drainage are contributory factors leading to improved outcomes.

In conclusion, *Chromobacterium violaceum* infection is a tropical/subtropical diseases. Whenever a patient presents with fulminant sepsis complicated by multiple internal organ abscesses, physicians should consider this infection as a part of the differential diagnosis. Because of its rarity, the initial isolation of *Chromobacterium violaceum* is frequently considered a saprophytic contaminant. A high index of suspicion and appropriate antimicrobial therapy are necessary to treat this potentially fatal disease. Special radiological investigations and surgical drainage should be considered. Specific antibiotics and the duration of treatment have not been well established. Because of frequent relapse, long term antimicrobial treatment and closed follow-up are important.

ACKNOWLEDGEMENTS

The author would like to thank Dr Nantana Prompapat and Dr Patchareeporn Suksikkhakosol for their considerable assistance in providing information regarding the patients. The author is also grateful to the other professional staff at Nan Hospital, and the National Institute of Health involved in managing the study.

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