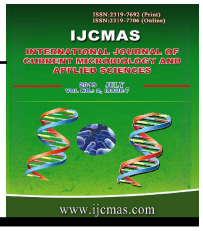
Int.J.Curr.Microbiol.App.Sci (2013) 2(7): 245-249

International Journal of Current Microbiology and Applied Sciences
ISSN: 2319-7706 **Volume 2 Number 7 (2013) pp. 245-249**
http://www.ijcmas.com



Original Research Article

A first case report in tertiary care hospital, Navi Mumbai, India - *Chromobacterium violaceum* septicaemia in a child

Harapriya Kar, Vijay Mane, A.D Urhekar, Samir Pachpute, Anahita Hodiwala, Sharawari Samant, Asha Kore, Maya Kale, Dipashri Khiste, Bhaskar Das and Nitin Goel Insan

Department of Microbiology, MGM Medical College Kamothe, Navi Mumbai, Maharashtra, India- 410209.
***Corresponding author e-mail:** vijayshreemane14@gmail.com*

Keywords

Proteo-bacterium; Systemic infection; *C.violaceum*; Septicemia.

A B S T R A C T

Chromobacterium violaceum is a proteobacterium found in soil and water in tropical regions. The organism rarely causes infection in humans, can cause a severe systemic infection by entering the bloodstream via an open wound. *Chromobacterium violaceum* is a rare human pathogen with a high rate of mortality. The first case was reported from Malaysia in 1927; about 150 cases have been reported till 2007 in the world literature. The present case study report of septicaemia was reported in male child.

Introduction

Chromobacterium violaceum is a gram-negative, facultative anaerobe, motile, oxidase-positive bacillus (James and Campbell, 2013). It is widely distributed in natural aquatic environments and is sensitive to temperature (Rekha Rai *et al*., 2011). Therefore, it has a predilection to the tropical and subtropical areas (Ponte and Jenkins,1992; Ching-Huei Yang, 2011). It is worth noting that the effects of global warming may increase, and the geographic distribution of this microorganism may change in the future. Infection due to *Chromobacterium violaceum* is rare, so most clinicians are not familiar with it. However, human infection is so rare but does occur. Rapid progress to sepsis with metastatic abscess is the striking feature in *Chromobacterium violaceum* infection (Sirinavin *et al*., 2005; Aldridge *et al.*, 1988). It can cause localized infections, septicemia and disseminated lesions, which may become fulminant leading to mortality(Zetti Zainol Rashid).

Case History

Male child about 10 years reported to a tertiary care hospital with complaints of acute abdominal pain, high grade fever, cough, increased frequency of loose motion and several episodes of vomiting for two days before admission. Clinical

examination revealed that the abdominal pain was centrally localised, non radiating and constant in nature. Patient had past history of Behçet disease .The child didn't have history of any medication or decrease in urine output. Any of the siblings didn't have history of Behçet disease. Birth and developmental milestone history of child was also normal. Patient had ptosis and pigeon chest. He had no other underlying diseases.CNS and CVS were within normal limits. On admission he had a pulse rate of 120 beats/minute, respiratory rate was 30/min, and SpO_2 was 100%. Systemic examination showed abdominal rigidity and guarding. Liver and spleen were not palpable. Ultrasonography of abdomen was normal. Patient was started on antimicrobial regime ceftriaxone (100mg/Kg), Amikacin (15mg/Kg) and metronidazole (30mg/Kg). Routine and bacteriological investigations were carried out on the same day of admission.

Result and Discussion

The next day patient showed fever, confusion, pain in abdomen, and hemodynamic changes, and died within 24 hrs of acute renal failure and cardiorespiratory failure. Blood culture taken 1 day before his death was positive for *Chromobacterium violaceum*. Haematology and biochemistry investigation result in Table 1,2 respectively.

Bacteriological study

Cultural characteristics

Chromobacterium violaceum was isolated from blood culture and stored in Microbiology laboratory. The organism showed better growth on the usual plating media, including Blood agar**,** MacConkey agar and Mueller-Hinton .Colonies on blood agar were about 1 mm in size, circular, convex, and smooth, beta haemolytic and produced dark purple pigment after 24 h incubation at 37°C. The organism was catalase and oxidase positive. It was difficult to interpret the latter because the violet pigment prevented the observation of result. This difficulty was overcome by incubating the agar plate anaerobically; there was no pigment formation. After a few hours under aerobic conditions, the colonies became violet again.

Biochemical features

The biochemical characteristics of the isolated strain are shown in Table 3. In triple sugar iron agar, *Chromobacterium violaceum* resulted an alkaline slant and acid butt without gas or hydrogen sulfide. It didn't grow in Simmons citrate agar. The arginine was dehydrolased.Lysine and ornithine was not decarboxylated. Gelatine was hydrolysed

Antibiotic susceptibility tests

Disk Diffusion Susceptibility tests (Kirby-Bauer's technique) showed the organism susceptible to Pefloxacin, Oflaxacin, Gentamicin, Netilin, Amikacin, Ceftazidime, Ciprofloxacin, Lomiflaxacin, Cefaperazone and Cefotaxime. It was resistant to Augmentin and Cefuroxime (Figure 3).

Chromobacterium violaceum exhibits both pigmented/ non-pigmented strain, though the non-pigmented strains are rare. The pigmented strains produce violet non diffusible pigment known as violacein. It acts as antioxidant pigment associated with remarkable purple colour (James and

Table.1 Hematology results over *Chromobacterium violaceum* **infection**

Tests	Results	Normal range
TLC	14.6	4400-11300/mm^3
DLC		
Neutrophils	91	45-70%
Eosinophils	01	00-06%
Lymhocytes	07	20-40%
Monocytes	01	00-40%
Basophils	00	00-07%
PCV	30.1	41.5-50.4%
RBC	3.85	4.5-5.9 million/mm
MCV	78.2	80-96.1%
MCH	24.9	27.5-33.2%
MCHC	31.9	33.4-35.5%
Haemoglobin	9.6	13.5-17.5gm
Platelets	1.88	1.5-4.0 lacs/cu.mm

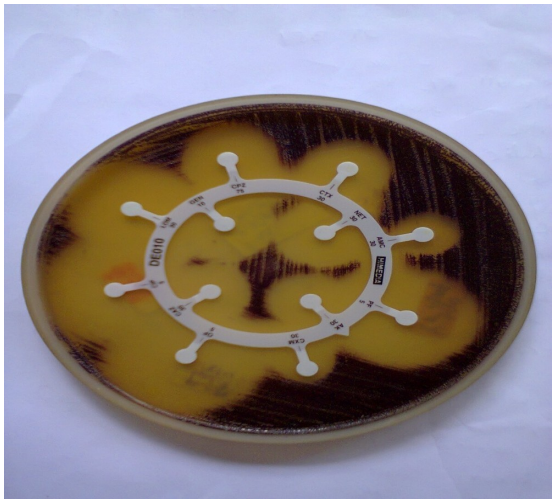
Table.2 Blood chemistry results of a *Chromobacterium violaceum* infection on admission

Tests	**Results**	**Normal range**
Creatinine	1.5	0.7–1.3mg/dl
Total Bilirubin	0.92	0.3-1.0 mg/dl
D. Bilirubin	0.71	0.0-0.2mg/dl
I. Bilirubin	0.21	--
SGOT	60.4	Up to 37U/L
SGPT	56.0	Up to 42U/L
Alkaline phosphatase	155.6	Up to 640 U/L
Na+	142.6	133–148 meq/l
K+	2.76	3.8-5.2meq/l
Protein	6.4	6.0-8.3gm/l
Albumin	3.8	3.2-5.0gm/l
Globulin	2.6	--

Table.3 Biochemical characteristics of *Chromobacterium violaceum*

Biochemical tests	Results
Haemolysis	B-haemolysis
Triple sugar iron agar	Alkaline/Acid
Gas from glucose	-
Simmon's citrate	-
Urease	-
Motility	Motile
Indole	-
Lysine decarboxylase	-
Ornithine decarboxylase	-
Arginine dihydrolase	+
Esculin hydrolysis	-
Gelatin hydrolysis	+
DNase	+
Catalase	+
Oxidase	+
Acid from Glucose	+
Sucrose	-
Lactose	-
Mannitol	-
Xylose	-
Fructose	+
Trehalose	+

Figure.1 Antimicrobial susceptibility testing of *Chromobacterium violaceum*



Campbell, 2013; Rekha Rai *et al*., 2011; Ponte and Jenkins, 1992; Ching-Huei Yang, 2011).

Human infections caused by *Chromobacterium violaceum* are uncommon. Only a few cases have been reported in the literature (James and Campbell, 2013; Rekha Rai *et al*., 2011; Ponte and Jenkins,1992). The pathogenic potential of this organism was first described in 1905 by P. G. Woolley, who isolated it from a fatal infection in buffalo in the Philippines. The first human infection was described by J. E. Lesslarin 1927 in Malaya. In 1938, Black and Shahan reported the first case in the United States. Ognibene and Thomas reported two fatal cases with this organism in Vietnam(Ching-Huei Yang, 2011; Sirinavin *et al*., 2005; Aldridge *et al.*, 1988; Zetti Zainol Rashid; Kaufman, 1986). Almost all of the cases reported have come from tropical and subtropical areas, mainly from Southeast Asia and the south eastern United States. In 1984, Petrillo described the first case in South America, in Brazil (Petrillo *et al*., 1984). The present case study is the first in Navi Mumbai. Quick diagnosis, accurate bacterial identification, and specific treatment are very important, because *Chromobacterium violaceum* may cause serious infection in healthy and young people (Ching-Huei Yang, 2011).

There is no clinical evidence that predisposing factors or pre-existing disease make the individual more susceptible to infection with this organism (Dauphinais and Robben.1968), although some cases have been described in patients with chronic granulomatous disease, and it was suggested that this might be a predisposing factor (Macher *et al*., 1982). The main features in most of the cases with a fatal outcome seem to be sepsis,

multiple liver abscesses, and diffuse pustular dermatitis (Lee et al.,). A case with pulmonary involvement was previously described (Lee and Wright. 1981). Though human infections with *Chromobacterium violaceum* are rare it can cause human infections like septicaemia, liver abscess, lung abscess, skin lesions, dental infections, urinary tract infections and diarrhoea. It is associated with high mortality rate. Infection process when systemic will ultimately form abscess in multiple organs. Death generally follows overwhelming septicaemia (Shenoy *et al*., 2002). The site of entry of the organism is frequently a skin lesion or injury, and days or even months may elapse before systemic disease is manifested. Since the patient had symptoms of abdominal pain, vomiting, frequency of motion, the organism could have gained entry through the gastrointestinal tract, which later on manifested with septicaemia. The clinical and bacteriologic remission in our case proved to be insufficient as a parameter of cure, needs to be another source of parameter to reveal and cure such type of infection.

References

Aldridge, K.E., G.T. Valaninis and Saners, C.V. 1988. Comparison of the *in vitro* activity of ciprofloxacin and 24 other antimicrobial agents against clinical strains of *Chromobacterium violaceum.* Diagn. Microbiol. Infect. Dis. 10

Ching-Huei Yang., 2011. *Chromobacterium violaceum* infection: A clinical review of an important but neglected infection. J. Chinese Med. Associa. 74.

Dauphinais, R.M., and Robben, C.G. 1968.Fatalinfection due to *Chromobacterium violaceum*. Am. J. Clin. Pathol. 50: 592-597.

James, I., and Campbell, A. 2013. successful antimicrobial regime for *Chromobacterium violaceum* induced Bacteraemia. BMC Infect. Dis. 13:4

Kaufman,S.C. 1986. First case report from Argentina of fatal septicaemia caused by *Chromobacterium Violaceum.* J. Clin. Microbiol. 23(5):956.

Lee, J., J. S. Kim, C. H. Nahm, J. W. Choi, J. Kim, S. H. Pai, K. H. Moon, K. Lee, and Chong, Y. Two Cases of *Chromobacterium violaceum* Infection after Injury in a Subtropical Region. Ann. Intern. Med. 97:51-55.

Lee, T. S., and Wright, B.D. 1981. Fulminating chromobacterial septicaemia presenting as respiratory distress syndrome. Thorax .36:557-559.

Macher, A. M., B. T. Casale and Fauci, A.S. 1982. Chronic granulomatous disease of childhood and *Chromobacterium violaceum* infection in the south eastern United States. Ann. Intern. Med. 97:51-55.

Petrillo, V. F., V. Severo, M. M. Santos and Edelweiss, E.L. 1984. Recurrent infection with Chromobacterium *violaceum*: first case report from South America. J. Infect. 9:167-169

Ponte, R., and Jenkins, S.G. 1992. Fatal *Chromobacterium violaceum* infections associated with exposure to stagnant Pediatfer. Inct. Dis. J. 11:583

Rekha Rai, Vimal Kumar Karnaker, Veena Shetty and Krishna Prasad, M.S.2011. *Chromobacterium Violaceum* Septicaemia-A Case Report. Al *Ame. en J. Med. Sci.*4 (2):201 -203

Shenoy, S., S. Baliga, G. Wilson and Kamath, N.2002. *Chromobacterium violaceum* septicaemia. Indian. J. Pediatr. 69(4):363 -364.

Sirinavin, S., C. Techasaensiri, S. Benjaponpitak, R. Pornkul and Vorachit, M. 2005. Invasive *chromobacterium violaceum* infection in children: case report and review. Pediatr. Infect. Dis. 24:559- 61.

Zetti Zainol Rashid. *Chromobacterium Violaceum* Infections; A Series Of Case Reports In A Malaysian Tertiary Hospital. The Inter. J. Infect. Dis. ISSN: 1528-8366.