

# Urinary Tract Infection By *Chromobacterium violaceum*

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## ABSTRACT

*Chromobacterium violaceum*, a facultative anaerobic proteobacterium, is particularly isolated from water and soil in tropical areas and has been implicated in few infections like septicemia, visceral abscesses, skin and soft tissue infections, meningitis and diarrhea. But urinary tract infection caused by it is very rare. Limited awareness about this pathogen and inappropriate antibiotic therapy contribute to a high mortality rate. Here, we describe an unusual case of urinary tract infection by *Chromobacterium violaceum* in a young immuno-competent male which was managed aggressively with proper antibiotics as per the culture sensitivity report.

**Keywords:** *Chromobacterium violaceum*, Proteobacterium, Urinary tract infection

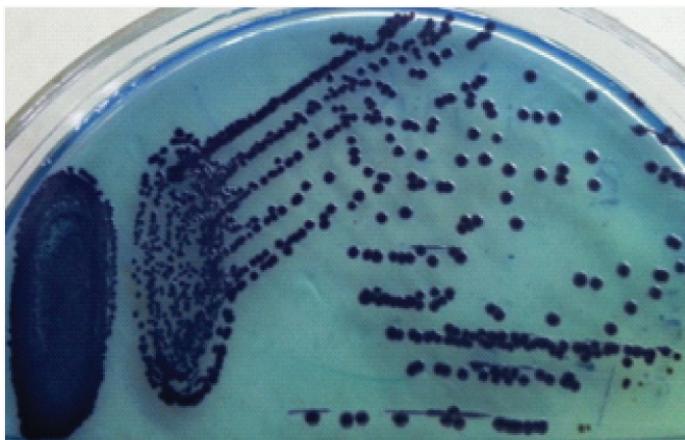
## CASE REPORT

A 19-year-old male attended the outpatient department of IMS and SUM hospital, Bhubaneswar, India in September, 2013 with chief complaints of burning micturition, fever with chills since seven days and pain in lower abdomen for three days. He had no past history of similar symptoms or any significant ailments. Abdominal examination revealed mild supra pubic tenderness. He was empirically treated with amoxicillin-clavulanic acid for urinary tract infection after advising necessary investigations. Laboratory findings showed a raised total leucocyte count (21,000/cmm) with relative neutrophilia (86%). Routine examination of urine showed albuminuria along with 8-10 pus cells per high power field with bacteriuria and occasional granular cast. Clean catch midstream urine sample plated on CLED medium revealed significant ( $>10^5$  CFU/ml) growth of single type of colonies of 2-3 mm diameter producing violet non-diffusible pigment [Table/Fig-1] after overnight aerobic incubation at 37°C. These organisms were gram negative, motile bacilli, catalase and oxidase positive, fermented glucose with production of acid without any gas. The bacterium was indole negative, non lactose fermenter on TSI media, utilized citrate, reduced nitrate, fermented mannitol and decarboxylated arginine. When incubated overnight anaerobically; there was no pigment formation which appeared after placing the plate for few hours under aerobic conditions. With these biochemical characters and pigment production it was identified as *Chromobacterium violaceum*. This was further confirmed by Vitek-2 system (BioMerieux, France) using GN card. Antibiogram

was done by Kirby Bauer's Disk Diffusion Susceptibility testing technique [Table/Fig-2]. The organism was found to be susceptible to ciprofloxacin, cotrimoxazole, imipenem, nitrofurantoin and cefotaxime and resistant to amoxicillin-clavulanic acid. The initial antibiotic was changed to ciprofloxacin as per the sensitivity report and continued for one week. Due to propensity of the organism to develop septicemia, blood culture was done which did not yield any growth. Other tests like that for HIV, HBsAg, diabetes screening were negative. After seven days of antibiotic treatment there was a marked clinical improvement and repeated urine examination did not show any significant finding.

## DISCUSSION

*Chromobacterium violaceum* is a facultative anaerobic, motile, oxidase positive gram negative bacillus [1]. In spite of ubiquitous distribution, human infections caused by *Chromobacterium violaceum* are uncommon with only a few cases reported in the literature [1-3], the first case being described by JE Lesslar in 1927 in Malaya [4]. Since then it has been associated with pneumonia, gastrointestinal infection, localized cutaneous lesions, localized or metastatic abscesses, osteomyelitis, meningitis, peritonitis, brain abscess, endocarditis, hemophagocytic syndrome, respiratory distress syndrome and fulminant sepsis [5]. Rapid progression to sepsis and multi organ dysfunction is a characteristic feature of *Chromobacterium* infection. But, urinary tract infection by this bacterium has only been occasionally reported as in 81-year-old male chronic kidney disease patient [6].



[Table/Fig-1]: Pigmented colonies on CLED agar



[Table/Fig-2]: Plate showing antibiogram

Human infections generally occur in subtropical and tropical areas particularly in summer months. This is attributed to the temperature sensitivity of the organism. *C. violaceum* infection may have become emergent due to global climatic change. It is considered as a bacterium of low virulence causing localized lesions and progressing to fatal septicemia in immuno-compromised individuals or in situations of inappropriate antimicrobial therapy. Patients with chronic granulomatous disease (CGD) have been considered to be vulnerable to *C. violaceum* infection [7]. Our patient was otherwise healthy and had no history of diabetes mellitus, HIV, steroid therapy or any other compromising illnesses, which must have contributed to his recovery without any complication.

Diagnosis of *Chromobacterium violaceum* infection is based on culture. They grow on ordinary culture media. The organism is Gram negative, motile, facultative anaerobic, catalase and oxidase positive bacillus and produces an alcohol soluble, water insoluble, non diffusible purple pigment- violacein. Its diagnosis is challenging in cases of 9% of strains which do not produce pigment. It has been established that pathogenicity does not depend on pigment production. Sivendra and Tan have stressed the importance of not using violet pigmentation as the sole criterion for separating *C. violaceum* from other Gram negative bacteria, in particular *Aeromonas* spp and *Vibrio* spp to avoid misdiagnosis and confusion [8].

The bacillus usually enters the body through minor skin trauma or through ingestion of contaminated water and seafood. Unusual routes of exposure include infection after scuba-diving or near drowning [9]. After breast surgery [10] and appendicectomy [11]. In our case, no similar history of the source of infection could be elucidated.

Antimicrobial susceptibility data on *C.violaceum* is very limited due to rarity of its isolation. It is found to be extremely resistant to penicillins and cephalosporins while sensitive to carbapenems and flouroquinolones. Increased beta-lactamase activity in *C.violaceum* was reported in the study by Farrar [12]. Ciprofloxacin is considered as the most active drug that can be used for treatment of this infection [13]. In our case it was sensitive to ciprofloxacin and imipenem while resistant to amoxillin-clavulanic acid.

Importance of early diagnosis and proper antimicrobial therapy can never be neglected in this case to avoid progression to sepsis.

Prolonged antimicrobial treatment for six weeks is recommended in *Chromobacterium* infection, as relapse of the disease has been documented and postulated to be due to the presence of internal organ abscesses [14]. Our patient did not develop any complications due to institution of early and proper antibiotic regimen.

## CONCLUSION

*C. violaceum* is considered as an emergent pathogen in view of the recent climatic changes. It has a propensity to develop into fatal septicemia unless appropriately treated. Thus it is required that the clinician must be aware of the sensitivity pattern and duration of treatment required for this infection. UTI by *Chromobacterium* is uncommon, nevertheless can occur and requires prompt management in order to avoid a fatal outcome.

## REFERENCES

- [1] James I, Campbell A. Successful antimicrobial regime for *Chromobacterium violaceum* induced Bacteraemia. *BMC Infect Dis*. 2013;13:4.
- [2] Rai R, Karnaker VK, Shetty V, Krishna Prasad MS. *Chromobacterium Violaceum* septicaemia-A Case Report. *Al Ameen J Med Sci*. 2011; 4:201-03.
- [3] Ponte R, Jenkins SG. Fatal *Chromobacterium violaceum* infections associated with exposure to stagnant water. *Pediatr Infect Dis J*. 1992;11:583-86.
- [4] Sneath PHA, Whelan JP, Singh RB, Edwards D. Fatal infection by *Chromobacterium violaceum*. *Lancet*. 1953;2: 276-77.
- [5] Yang CH, Li YH. *Chromobacterium violaceum* infection: A clinical review of an important but neglected infection. *J Chin Med Assoc*. 2011;74: 435-41.
- [6] Ma T, Shi W, Cheng J, Zhang JK, Hu LF, Ye Y, et al. *Chromobacterium violaceum* infection in China: Three case reports and literature reviews. *African Journal of Microbiology Research*. 2011;5(20): 3096-102.
- [7] Winkelstein JA, Marino MC, Johnston RB, Boyle J, Curnutte J, Gallin JL, et al. Chronic granulomatous disease: report on a National Registry of 368 patients. *Medicine*. 2000;79:155-69.
- [8] Sivendra R, Tan SH. Pathogenicity of nonpigmented cultures of *Chromobacterium violaceum*. *J Clin Microbiol*. 1977;5:514-16.
- [9] Starr AJ, Cribbitt LS, Poklepovic J, Friedman H, Ruffolo EH. *Chromobacterium violaceum* presenting as a surgical emergency. *South Med J*. 1981;74:1137-39.
- [10] Victorica B, Baer H, Ayoub EM. Successful treatment of systemic *Chromobacterium violaceum* infection. *JAMA*. 1974; 230:578-80.
- [11] Chen CH, Lin LC, Liu CE, Young TG. *Chromobacterium violaceum* bacteraemia: a case report. *J Microbiol Immunol Infect*. 2003;36:141-44.
- [12] Farrar WE, O'Dell NM. β-lactamase activity in *Chromobacterium violaceum*. *J Infect Dis*. 1976; 134: 290-93.
- [13] Aldridge KE, Valaninis GT, Saners CV. Comparison of the in vitro activity of ciprofloxacin and 24 other antimicrobial agents against clinical strains of *Chromobacterium violaceum*. *Diagn Microbiol Infect Dis*. 1988;10:31-9.
- [14] Sirinavin S, Techasaensiri C, Benjaponpitak S, Pornkul R, Vorachit M. Invasive *Chromobacterium violaceum* infection in children:case report and review. *Pediatr Infect Dis J*. 2005; 24:559-61.

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