

To the Editors:

## ***Chromobacterium violaceum* infection in a provincial hospital in Sri Lanka**

*Chromobacterium violaceum* is an aerobic, gram-negative bacillus usually found as a saprophyte in soil and stagnant water in tropical and subtropical regions. It produces violacein, a pigment which imparts the violet black colour to the laboratory media [1,2]. *C. violaceum* is generally susceptible to fluoroquinolones, carbapenems, and gentamicin but resistance to cephalosporins is common [1].

It is a ubiquitous organism but a rare human pathogen, and can cause fatal sepsis with metastatic abscesses in lungs, liver, and spleen. Nearly 150 cases have been reported, mainly in Asia and South America [3]. Infection with this organism is usually acquired through trauma, and may begin with cellulitis, pustules, and gangrene [1-4]. Infection is more common in patients with chronic granulomatous disease (CGD) [1,2].

### **Case 1**

A 9-month old baby boy with fever, vomiting, multiple joints swelling, and generalised pustular rash was investigated for pyrexia of unknown origin. He had a past history of recurrent infections. On examination there was hepatosplenomegaly and pallor. The C-reactive protein level was raised (60mg/l) and other blood tests were normal. *Staphylococcus aureus* was isolated from blood cultures. He was treated with vancomycin but there was no improvement. Ultrasound scan of the brain and

abdomen, chest xray and bone marrow aspiration were normal. Since there was consanguinity and history of recurrent infections, he was investigated for possible immunodeficiency. He developed gangrene in two toes, and culture of pus from a pustule yielded purple coloured colonies (figure 1), identified as *Chromobacterium violaceum* by the biochemical profile (Analytical Profile Index). Antimicrobial sensitivity of the isolate showed resistance to ceftazidime, ceftriaxone, co-amoxycylav and sensitivity to ciprofloxacin, meropenem, gentamicin, amikacin and timentin. The patient died and the autopsy showed abscesses in the lungs.



Figure 1. ***Chromobacterium violaceum* colonies on nutrient agar.**

**Case 2**

A 2-month old baby boy developed high fever, vomiting and pustules over the legs and abdomen. The C-reactive protein was raised (72mg/l). The leucocyte count was 24 800 /  $\mu$ l with 70% neutrophils. The liver was enlarged and enzyme levels were raised. Empiric treatment with cefotaxime and gentamicin was initiated. After 3 days of treatment, serial blood cultures were positive for *Chromobacterium violaceum*, with the same sensitivity pattern as in case 1. Antimicrobial therapy was changed to meropenem and gentamicin. After 7 days symptoms and signs regressed and treatment was continued for 21 days. Immunodeficiency screening was negative. Samples from the child's water source grew *C violaceum*.

Systemic infection caused by *C violaceum* is rare but severe and is associated with a high fatality rate of over 60% [1,2,3]. *C violaceum* causes fever, skin lesions and hepatic and lung abscesses [3,4,5]. *C violaceum* infection is common in immunocompromised patients but may occur in apparently healthy people [2,3,5]. Even though recommended therapy for *C violaceum* infection is not established, carbapenems appear to be effective. Clinicians in tropical and subtropical countries should

consider infection with *C violaceum* in patients with skin lesions and rapidly progressing sepsis.

**References**

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