Fatal septicemia caused by *Chromobacterium violaceum*

Shalin1 Perera1, P M G Punchihewa1, M C G Karunanayake1 and Nelun de Silva1

(Index words: Clinical features, antibiotic sensitivity)

**Abstract**

We report a case of *Chromobacterium violaceum* septicemia in a 10-year old boy admitted to hospital in September 2001, two weeks after he suffered a crush injury of the left foot. In spite of surgical debridement and antibiotics his condition worsened and he developed multiple liver and lung abscesses. He died one week after admission and a blood culture grew *Chromobacterium violaceum*.

**Introduction**

*Chromobacterium violaceum*, was identified in 1881 (1) and first described as a human pathogen in 1927 (2). Although rarely isolated from clinical specimens *Chromobacterium violaceum*, is easily identified on conventional culture media by its striking deep violet pigment (1,3). *Chromobacterium violaceum* infections have been reported from several countries (1,2,3). We report the first recorded case of septicemia due to *Chromobacterium violaceum* in Sri Lanka.

**Case report**

In September 2001 a 10-year old boy, from a remote village in the Southern Province, was admitted to the surgical casualty ward of the Karapitiya Teaching Hospital with an ulcer on the left foot following a crush injury sustained two weeks before. There was bilateral inguinal lymph node enlargement. Wound debridement was done, and he was treated with cloxacillin and penicillin. He developed fever with chills and rigors on the third day after admission and the next day, severe abdominal pain, vomiting and dyspnoea. Thereafter his condition deteriorated rapidly. Chest examination revealed bilateral diffuse rales. The liver was tender and enlarged up to the right iliac fossa and the spleen was palpable 4 cm below the left costal margin. He had at this stage leucocyte count of 15 000/µl with 91% neutrophils and a normal platelet count. Chest xray showed multiple diffuse cavitatory lesions in both lung fields. Ultrasound scan of the abdomen showed a grossly enlarged liver with multiple 5 to 10 mm cystic lesions resembling abscesses.

His blood pressure was normal and there was no bleeding tendency. Intravenous gentamicin and cefuroxime were added to cloxacillin after blood was taken for culture. Two hours later he was transferred to an intensive care unit for ventilatory support. He succumbed on the same day and blood cultures subsequently grew *Chromobacterium violaceum*.

Blood culture yielded smooth shiny violet black colonies on blood (acobic culture) and MacConkey agar. On Gram stain the organisms were Gram negative, slightly curved bacilli which occurred singly or in pairs and showed the characteristic bipolar or barred appearance. The organism was identified as *Chromobacterium violaceum* by standard microbial identification procedures and confirmed by API 20 E. Susceptibility testing done by the NCCLS method revealed that the isolate was sensitive to ciprofloxacin, imipenem, meropenem, cotrimoxazole, tetracycline, gentamicin, netilmicin and aztreonam, and resistant to ampicillin, augmentin, cefuroxime, ceftriaxone and ceftazidime.

**Discussion**

*Chromobacterium violaceum* is a saprophyte present in soil and water in tropical and subtropical areas (1,2,3). Infection usually follows exposure of broken skin to contaminated water or soil (1,3). Symptoms such as fever, vomiting, pestules and lymphadenitis commonly precede evidence of systemic infection (3). Septic shock develops rapidly, as may pneumonia and visceral abscesses involving liver, spleen and lung.

*Chromobacterium violaceum* is generally considered to be of low virulence, but septicemia is usually fatal unless promptly treated (2,3), with a mortality rate of 60% for reported cases in the USA (3,6). Severe infection with a *Chromobacterium violaceum* is reported to be more common in patients with chronic granulomatous disease (4,5) even though serious sepsis due to this organism can occur in previously healthy people (3).

*Chromobacterium violaceum* sepsis should be suspected in the presence of cutaneous lesions with multiple liver and lung abscesses and lymphadenitis. Appropriate specimen such as blood, wound pus, ascitis fluid should be taken early in the course of the disease.

1Senior Registrar in Microbiology, 2Paediatrician, 3Registrar (Paediatrics), 4Head, Department of Microbiology, Teaching Hospital and Faculty of Medicine, University of Ruhuna, Karapitiya, Galle, Sri Lanka. (Correspondence: N de S. Received 12 November 2001, revised version accepted 14 October 2002).

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An unusual cause of melaena in a child

Sanath P Lamabadusuriya1 and Shehan Perera2

(Index words: Hookworm infection, endoscopic diagnosis)

Case report

A previously healthy and playful 2-year-old girl was admitted to Base Hospital, Chilaw with a history of melaena and lethargy for one week.

On admission she was lethargic and very pale with a hepatomegaly of 1 cm. Her haemoglobin was 3.5 g/dl and the stool examination and ultrasound scan of the abdomen were normal. In spite of two blood transfusions she continued to have melaena and remained pale. She was then transferred to Lady Ridgeway Hospital. She gave no history of drug ingestion, or features suggestive of gastritis. She was from a poor socio-economic background without toilet facilities. The entire family used the surrounding scrub jungle for defecation. The children used the same surroundings for defecation. The children used the same surroundings for defecation.

Results of investigations were as follows: Hb 7.65 g/dl, stools report – no ova; stool culture – no pathogens isolated; liver function tests – normal; white blood cell count – 13 600/ml (eosinophils 13%). She continued to have melaena and the haemoglobin dropped to 3.7 g/dl in spite of frequent transfusions. She was transfused 16 units of blood during a period of 35 days.

Ultrasound scan of the abdomen showed a normal portal vein with no evidence of chronic liver disease. Upper gastrointestinal endoscopy showed no varices and the first part of the duodenum including the stomach was normal; barium meal and follow through was normal up to the distal small intestine. 99 mTc Meckels scan showed no evidence of ectopic gastric mucosa, 99 mTc red blood cell scan showed no evidence of haemorrhage into the gastrointestinal tract and CT scan of abdomen showed a normal small intestine.

As all investigations were normal a repeat upper gastrointestinal endoscopy together with a colonoscopy was done at the Professorial Surgical Unit of the Colombo South Teaching Hospital. The upper gastrointestinal endoscopy showed a large number of worms attached to the second part of the duodenal mucosa. A specimen was taken and these were identified as hookworms (Necator americanus). A repeat stool examination at the Department of Parasitology, University of Colombo showed hookworm ova (150 eggs/g). A diagnosis of heavy hookworm infestation was made on the 42nd day of the illness and she was treated with a single dose of 400 mg albendazole. The child showed a dramatic improvement. The melaena ceased, haemoglobin levels remained stable and no further transfusions were required. The entire family was dewormed and the child was discharged on appropriate treatment. Two weeks later the haemoglobin was 8.6 g/dl.

Discussion

Melaena is due to upper gastrointestinal haemorrhage, usually caused by reflux oesophagitis, Mallory-Weiss syndrome, gastric or duodenal ulcers, oesophageal varices, gastric carcinoma or acute gastric erosions. Rare causes include hereditary telangiectasia, pseudoxanthomata elasticum and blood dyscrasias. Hookworm infestation was not listed as a cause of melaena other than a report on a few infants who developed overt gut bleeding and melaena after transplacental infection (1). A literature search on the medline (1990-2001) showed no reports of hookworms being associated with melaena.

In this child the worm load was so great that it caused a massive blood loss giving rise to melaena. Necator

1Senior Professor and 2Registrar, Professorial Paediatric Unit, Lady Ridgeway Hospital for Children, Colombo 8. (Correspondence: SPL, Telephone + 94 1 811486, e-mail: deanmed@hotmail.com. Received 2 November 2002, accepted 6 January 2003).