

FATAL SEPSIS DUE TO *PSEUDOMONAS CEPACIA*

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ABSTRACT

Pseudomonas cepacia is known as an opportunistic pathogen in the patients with altered host defence. We report a case of hospital-acquired fatal sepsis due to *P. cepacia* in a child with no known defect in immune system. Virulence of the organism was indicated by the high magnitude of septicaemia. The organism was successfully eliminated by ceftriaxone but the patient died of renal failure.

Keywords: *Pseudomonas cepacia*, septicaemia, blood culture, anuria, renal failure.

SINGAPORE MED J 1994; Vol 35: 475-476

INTRODUCTION

Pseudomonas cepacia has recently been recognised as a respiratory pathogen in patients with cystic fibrosis⁽¹⁾. The organism has also been found to be occasionally associated with blood stream infection, septic arthritis, bacteraemia, meningitis, osteomyelitis, peritonitis, lung abscess and pneumonia in patients with altered host defence system⁽²⁾. Several reports indicate that it is virtually nonpathogenic in normal individuals, and if infection occurs, the course is usually benign even without antibiotic therapy^(3,4). We report here a case of *P. cepacia* septicaemia in a normal individual with fatal outcome.

CASE REPORT

A previously well but undernourished (weight/age 65% of 50th percentile of NCHS standard) 7-year-old boy presented with high fever, pain and swelling in the right hip joint with restriction of movement, swelling in the submandibular area and red macular eruptions throughout the body. The case was diagnosed as pyogenic arthritis of right hip joint with septicaemia. Haematological investigation showed a high total leucocyte (WBC) count (16,000/mm³) with neutrophilia (80%). Chest X-ray showed no abnormality. Blood culture yielded the growth of *Staphylococcus aureus* and the patient was given intravenous cloxacillin (100 mg/kg body wt) to

which the organism was sensitive. After 4 days of treatment the patient became afebrile, but his general condition remained unchanged, including the restriction of hip joint movement.

On the 7th day of admission, the temperature of the patient suddenly peaked to 104°F and his condition started to deteriorate further. At this point intravenous ceftriaxone (100 mg/kg body weight) was started. Blood culture on the same day (7th) showed the growth of *P. cepacia*, and the count indicated a high magnitude of septicaemia (Table I). The organism was sensitive to ceftriaxone and cotrimoxazole. However, the patient's condition continued to deteriorate and he developed oliguria on the 8th day leading to anuria on the 10th. Blood count and film showed a total WBC count of 6,500/mm³ with neutrophilia (82%) and thrombocytopenia with numerous crenated red blood cells (RBC). Culture of blood on the 9th and 10th day showed growth of the same organism (*P. cepacia*) but with gradual decrease in number (Table I). The last blood culture on the 12th day of admission did not yield any growth. Elevated blood urea and creatinine were detected on the 9th day, which increased further on the subsequent days. On the 13th day the clinical condition further deteriorated and he died of renal failure.

Table I – Results of bacteriological and biochemical investigations of the patient's blood at different time intervals.

Day(s) after admission	Blood Culture		Urea mmol/l	Creatinine umol/l
	Growth	cfu/ml		
1	<i>Staph. aureus</i>	4	–	–
7	<i>P. cepacia</i>	Numerous (>1000/ml)	–	–
9	<i>P. cepacia</i>	180	17.6	303.0
10	<i>P. cepacia</i>	4	27.0	501.0
12	No growth	0	32.0	656.0

Microbiology and Biochemistry

Blood cultures were done by Lysis-centrifugation method⁽⁵⁾. *P. cepacia* isolates were confirmed (score 0.999999; 0.59E-06) by the Public Health Laboratory Service (PHLS), England. Sensitivity of the isolates was tested by disc diffusion method⁽⁶⁾. The strains were sensitive to cotrimoxazole, and ceftriaxone and resistant to ampicillin, cephalixin, and gentamicin. Blood urea and creatinine level

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were determined by diacetyl monoxime and Jaffe-alkaline method respectively using a computerised analyser (4010, Boehringer) which showed a gradual increase since the patient was infected by *P. cepacia*.

DISCUSSION

To our knowledge through literature review and Med-line search this is the first report of fatal sepsis due to *P. cepacia* in a child with no clinical evidence of immunodeficiency.

Studies on *P. cepacia* suggest the nosocomial acquisition⁽³⁾ and person-to-person transmission⁽⁷⁾ of the organism. Isolation of the organism from the second blood culture of this patient indicates the hospital origin of the infection. We tried to isolate *P. cepacia* from different specimens like the floor and bed sheet swab, and water of the patient's ward using selective medium⁽⁸⁾, after getting the PHLS report, but all the cultures were negative.

Quantitative evaluation of septicaemia indicates the high magnitude of the disease caused by *P. cepacia* and successful elimination of the organism by ceftriaxone. Deterioration of the patient, even after successful elimination of the organism from blood, may be explained by the fact that the treatment of Gram negative sepsis may contribute to host injury through the increased release of endotoxin in blood^(9,10).

Recently, we have isolated several other strains of *P. cepacia* from the blood of septicaemic neonatal patients of our hospital which were subsequently confirmed by the PHLS laboratory. In most of these cases the infection were fatal. These observations argue against the fact that *P.*

cepacia is an organism of low pathogenicity. It will be interesting to study the virulence of these isolates in different animal models and molecular epidemiology by ribotyping.

ACKNOWLEDGEMENT

We thank Ms Maksuda Islam, research assistant of Dhaka Shishu Hospital, for technical assistance and Dr Anisur Rahaman, Assistant Professor of Bangladesh Medical College, for reviewing the manuscript.

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