

Fatal Necrotizing Fasciitis Caused by *Pseudomonas aeruginosa* in Infants

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Two cases of Necrotizing fasciitis caused by *Pseudomonas aeruginosa* in infants are presented. One is female 4 days old; the other is an 8 months old male. Both infants died despite aggressive treatment and intensive resuscitation. Both infants exhibited dark and bluish discoloration of some region of their skin. *Pseudomonas aeruginosa* was recovered from the blood culture in both infants.

The aim of this communication is to report the occurrence of rare cases of necrotizing fasciitis caused by *Pseudomonas aeruginosa* in infants.

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Necrotizing fasciitis is a rare; it is rapidly progressing and potentially fatal infection of the superficial fascia and subcutaneous cellular tissue. Clinically, it is characterized by massive destruction of tissue; it is usually accompanied by systemic signs of toxicity; the condition has high rate of mortality and morbidity¹⁻³.

Necrotizing fasciitis is frequently polymicrobial and a combination of aerobic (streptococci and staphylococci) and anaerobic bacteria often lead to quick progression of the disease. Early diagnosis and aggressive surgical intervention are critical to improve the poor outcome of this infection^{1,4,5}. *Pseudomonas aeruginosa* may cause soft tissue infections but it is rarely associated with necrotizing fasciitis and few reports document such association⁶⁻⁸.

CASE ONE

Four days old Saudi female patient brought to emergency room; she was irritable and lethargic for 2 days. The patient was looking sick, pale, jaundiced, but she was not dehydrated, not in respiratory distress and not dysmorphic. She was a full term baby, the maternal parameters are shown in Table 1. The baby was admitted to neonatal intensive care unit (NICU). She was given the appropriate IV antibiotics as guided by culture. On the first day, she was treated with phototherapy for jaundice. After 48 hours, she was discharged against medical advice. After one day outside the hospital, the baby was re-brought to the hospital, the occasion at which was found to be lethargic, irritable, crying excessively and had poor oral intake. She had no history of fever or convulsion. She had no history of respiratory or other gastrointestinal symptoms and no history of skin rashes. The family lives in good housing and had an average income.

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The patient systemic examination was unremarkable except for a dark discoloration on the left forearm extending to the upper arm near the shoulder joint with bullae formation. A similar lesion was present on the right upper limb but with less intense color. The patient was admitted to the NICU. She was diagnosed as a fulminant sepsis; blood and urine were obtained for culture. The patient was resuscitated with fluids, Inotropes and started on Vancomycin and Meropenem. The laboratory results are summarized in Table 1.

Table 1: Clinical and Laboratory Findings of the Two Cases

Parameter	Case One	Case Two	Reference Values
Sex	Female	Male	--
Age	4 days	8 months	--
Birth weight	2,150 gm	Unknown	2,812- 4,173 gm
Apgar score	7 (at 1 min) and 9 (at 5 min)	Unknown	Score of 7-10 is normal
Temperature	37°C	37.3°C	36.1–37.8 °C
Heart rate	110/min	120/min	60-100/ min
Respiratory rate	45/min	40/min	15 to 20 / min
Blood pressure	60/35 mm Hg	102/58 mm Hg	120/80 mm Hg
Oxygen saturation (arterial)	92%	Not detected	96 - 100%
WBC	3.2 x 10 ³ cells/ uL	1.0 x 10 ³ cells/ uL	3.8 - 10.8 x 10 ³ cells/ μL
Hemoglobin	15.1gm/ dL	7.2 gm/dL	Male: 13 - 18 gm/dL Female: 12 - 16 gm/dL
Platelet count	210 x 10 ³ /μL	620 x 10 ³ /μL	130 - 400 x10 ³ /μL
Urea	96 mg/dL	54 mg/dL	7 - 18 mg/dL
Creatinine	1.8	1.0	0.6 - 1.2 mg/dL
Aspartate aminotransferase (AST)	242	Not detected	7 - 27 units/L
Alanine transaminase (ALT)	9	Not detected	1 - 21 units/L
pCO ₂	17 mmHg	16.3 mmHg	35.0-45.0 mmHg
HCO ₃	4.5 mm Hg	7.0 mm Hg	24-28 mm Hg
Prothrombin time (PT)	≥ 120 (upto13)	Not detected	25 - 41 sec

The patient was intubated and mechanically ventilated. Peritoneal dialysis was performed. Four hours after admission, the rashes extended to involve both shoulders and upper chest. Her condition remained very critical and she became hypotensive, bradycardic and not responding to treatment. In spite of the aggressive resuscitation, she died. Lumbar puncture was not done as the patient was thrombocytopenic and had abnormal coagulation profile. *Pseudomonas aeruginosa* was recovered from a blood culture after 24 hours when incubated at 37°C, but urine culture revealed no bacterial growth.

CASE TWO

An 8-months-old Sudanese male infant presented with a history of vomiting, diarrhea and fever for 3 days. He was looking very sick, pale, dehydrated, lethargic and had a temperature of 37.3°C. He was a full term baby; the maternal parameters are shown in Table 1. His systemic review was unremarkable.

He was admitted previously for 7 days due to focal convulsion at the age of 7 months, his work up was negative with a normal brain CT; the Patient was discharged in good condition and no medications. He is on breast and bottle formula with adequate intake. He is vaccinated

up-to-date, with normal developmental history. He lives with his parents in good housing and they have an average income.

The laboratory result of his admission is summarized in Table 1. This patient was rehydrated with 3 boluses of normal saline then he was put on maintenance and deficit fluid replacement. The patient was started on Vancomycin and Ceftazidime according to the culture results. He was given packed red blood cell transfusion. CSF examination revealed RBC: 10^2 cell/mm³, WBC: nil with normal protein and sugar. CSF culture yields no growth. Nasal secretion culture showed *Pseudomonas aeruginosa* when incubated at 37°C for 24 hours. *Pseudomonas aeruginosa* was also recovered from a blood culture but urine culture revealed no bacterial growth.

After admission, the lesion over his genitalia started to become bluish in color with edema progressing to both thighs and lower abdomen. His condition deteriorated and he became hypothermic, hypotensive and desaturated. The patient was intubated and ventilated. Within few hours, the edema and discoloration extended to both thighs, the whole abdomen and chest. The patient continued to be very critical and subsequently arrested, and all the measures failed to revive him. His skin biopsy taken from his lower abdomen showed hyperkeratosis, mild acanthosis and dermal inflammatory infiltrates supporting the diagnosis of superficial perivascular dermatitis.

DISCUSSION

Pseudomonas aeruginosa can be a fatal cause of necrotizing fasciitis⁶⁻⁸. According to Fustes-Morales et al, *Pseudomonas aeruginosa* was the most frequently isolated bacteria which represented 85% of 39 patients with necrotizing fasciitis⁹.

In the present report, the two cases had no apparent injury or surgical wounds and both cases seem to be immunocompetent. The diagnosis was suspected from their clinical presentation, the rapid progression of the skin lesions and was supported by the laboratory findings. *Pseudomonas aeruginosa* was recovered from their blood cultures in contrast to the common believe which does not associate *Pseudomonas aeruginosa* with necrotizing fasciitis.

The rapid fatal course of these two cases did not allow us to perform surgical exploration or peritoneal dialysis in both cases. It is still unclear how these two patients contracted these invasive fatal infections. It will be very difficult to predict their response to surgical intervention and intravenous immunoglobulins if one or both were included in the management early in the course of their illness.

CONCLUSION

Two cases of fatal necrotizing fasciitis caused by *pseudomonas aeruginosa* in infants are presented.

The authors of this report, therefore, alert the medical professionals to the magnitude of this important condition and similar conditions which needs early prompt clinical intervention as well as submitting appropriate specimens for laboratory analysis.

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