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CASE REPORT

Pseudomonas aeruginosa sepsis with ecthyma gangrenosum and pseudomembranous pharyngolaryngitis in a 5-month-old boy

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Pseudomonas aeruginosa infection that induced pseudomembranous laryngopharyngitis and ecthyma gangrenosum simultaneously in a healthy infant is rare. We reported on a previously healthy 5-month-old boy with initial presentation of fever and diarrhea followed by stridor and progressive respiratory distress. *P. aeruginosa* sepsis was suspected because ecthyma gangrenosum over the right leg was found at the emergency department, and the diagnosis was confirmed by the blood culture. Fiberscope revealed bacterial pharyngolaryngitis without involvement of the trachea. Because of early recognition and adequate treatment, including antimicrobial therapy, noninvasive ventilation, incision, and drainage, he recovered completely without any complications.

Introduction

Pseudomonas aeruginosa bacteremia usually occurs in immunocompromised groups, malignancy, chronic disease, burns, or preterm infants.[1] Community-acquired *P. aeruginosa* sepsis in previously healthy infants is not often seen. Ecthyma gangrenosum is a characteristic skin manifestation of *Pseudomonas* sepsis. The concomitant airway involvement in *P. aeruginosa* sepsis is rare. We report on a previously healthy 5-month-old boy with *P. aeruginosa* sepsis, ecthyma gangrenosum, and pseudomembranous pharyngolaryngitis, all of which initially presented as stridor and respiratory distress.

Case report

A previously healthy 5-month-old boy was brought to the emergency department with a 5-day history of fever and

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cough and 2 days of watery diarrhea, vomiting, poor appetite, and progressive dyspnea. He was lethargic, pale, and had barking cough, stridor, and respiratory distress with suprasternal and intercostal retractions. His body weight was 7.57 kg (50 percentile). Vital signs revealed blood pressure at 90/60 mm Hg, pulse rate at 171 beats/minute, respiratory rate at 50 breaths/minute, and temperature at 38.8°C (rectal). Oxygen saturation was 88 % under room air. He had dry lips and an injected pharynx. There were diffuse and bilateral crackles on auscultation. He had decreased bowel sounds and a soft abdomen. Liver and spleen were impalpable. The capillary refill time was 5 seconds. There were two lesions over his right leg with central ecchymotic and reddish indurations surrounded by erythematous halos, which showed the characteristics of ecthyma gangrenosum (Fig. 1). Initial management included intravenous normal saline challenge for shock, oxygen supplement, and epinephrine nebulization for respiratory distress. Then he was admitted to the pediatric intensive care unit.

He had no specific family, travel, or allergy history. However, a dog scratched him about 1 week previously, and he received an appropriate vaccination. Laboratory tests reflected leukopenia (white blood cell count, 2,600/μL,; neutrophil, 52%) with left shift (band form, 11%), elevated C-reactive protein (CRP, 14.82 mg/dL), hyponatremia (serum sodium, 126 mEq/L), positive stool occult blood test, metabolic acidosis, and disseminated intravascular coagulation (prothrombin time, 12.1 seconds; partial thromboplastin time, 117 seconds; fibrinogen degradation products >8 μg/mL; D-dimer, 2258 ng/mL; fibrinogen 269 mg/dL). Chest radiograph revealed bilateral diffuse infiltration. On the second day of hospitalization, ecthyma gangrenosum over his right leg became progressively larger and increased in number, some with central darker plaques.

Flexible bronchoscope found whitish exudates (pseudomembrane) coating on pharynx and tonsils without involvement of trachea (Fig. 2). Pathologic examination of the tissue specimen obtained from the pharynx when bronchoscope examination revealed numerous bacteria. No pathogen was identified from the throat virus culture, and the throat bacterial culture found normal flora. Blood culture yielded *P. aeruginosa* that was susceptible to ceftazidime and amikacin. Stool culture grew *Salmonella* group C2, which was susceptible to ceftazidime, and *Aeromonas* species. Empiric antibiotics including vancomycin (40 mg/kg/day), ceftazidime (150 mg/kg/day) and amikacin (20 mg/kg/day) were prescribed initially, but were changed to ceftazidime with amikacin according to the culture reports. Noninvasive ventilation was used for 3 days and fever subsided on the sixth day of hospitalization.

After 14 days' therapy with ceftazidime and amikacin, three skin lesions on the right leg progressed to central darker color with pus. Wound incision and drainage were performed (Fig. 3). Pus cultures of two skin lesions on Days 15 and 21 still yielded *P. aeruginosa*. Antibiotic therapy with ceftazidime alone was given from Day 15 until Day 28. The boy was discharged without any complications. In addition, the immunologic survey showed within normal limits the following: immunoglobulin (Ig) G 475 mg/dL (172—1069), IgA 80 mg/dL (4.4—84), IgM 62 mg/dL (33—126), IgE 49 IU/mL (0—170), CH50 67.8 CAE units (63.0—145.0), CD3 625 % (48.0—75.0), CD4 32.2% (33.0—58.0), CD8 27.2% (11.0—25.0), CD19 27.6% (14.0—39.0), CD16+56(NK) 7.5% (5.6—31.0), and active T cell 13.0% (8.0—15.0).

Discussion

The annual detection rate of *P. aeruginosa* bacteremia in children less than 18 years old was 3.8/1000.[1] *P. aeruginosa* sepsis rarely occurred among immunocompetent infants and children. Chusid and Hillmann postulated that viral infections could either directly weaken the mucosal barrier

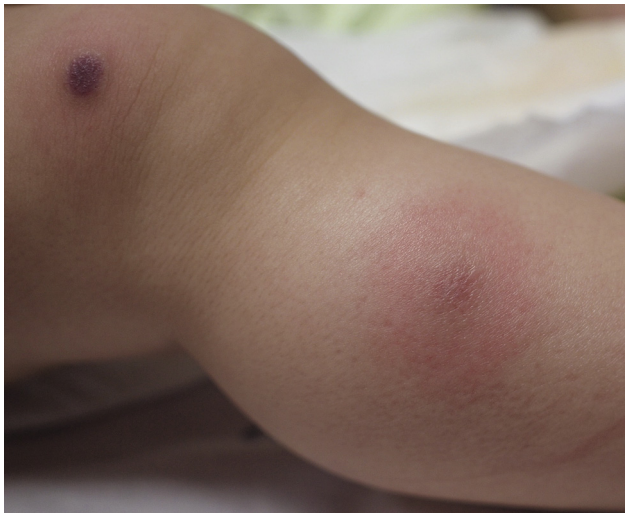

Figure 1. The upper skin lesion was over the right thigh (1 × 1 cm), with a central ecchymotic induration. The lower skin lesion was over the right lower leg (4 × 4 cm) with a central reddish plaque. Both were surrounded by erythematous halos.

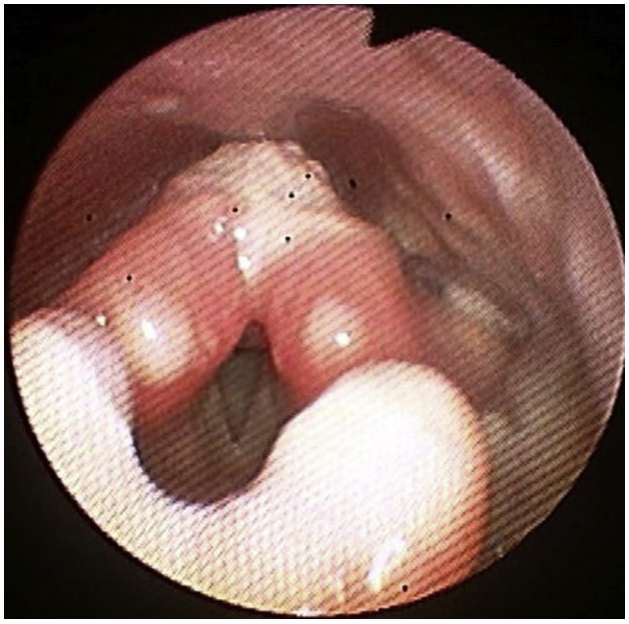

Figure 2. Whitish exudate (pseudomembrane) coating over the pharynx and tonsil.

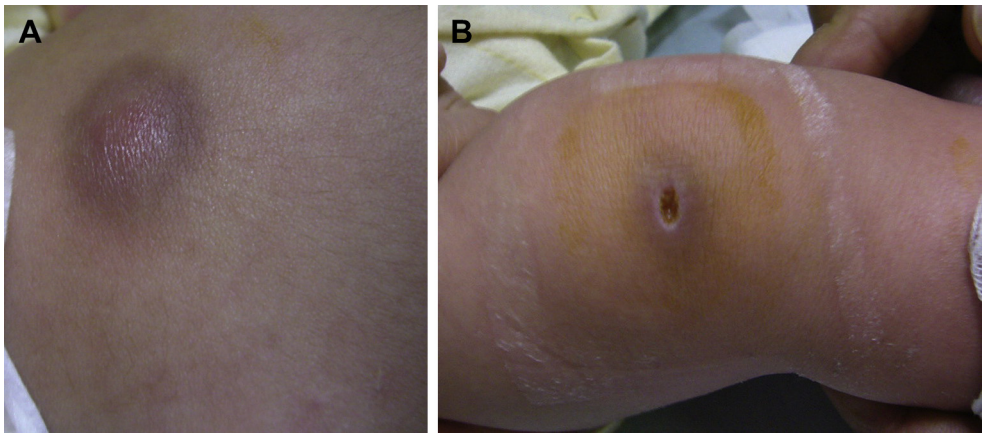

Figure 3. Ecthyma gangrenosum on Day 21 of hospitalization. (A) Ecthyma gangrenosum over the right thigh with central ecchymotic induration and pus; (B) ecthyma gangrenosum over the right knee after wound incision and drainage.

of the gastrointestinal tract or temporarily disrupt host defenses.[2] Our patient is an immunocompetent infant. He had respiratory and gastrointestinal tract infection before *P. aeruginosa* bacteremia. Those antecedent acute illnesses may be the inducers of transient impaired immunity in our patient and cause *P. aeruginosa* sepsis later.

One study reported 43 episodes of community-acquired *P. aeruginosa* sepsis in previously healthy infants and children. Fever (91%) and diarrhea (72%) were the two most common initial symptoms.[3] Preceding conditions of community-acquired *P. aeruginosa* sepsis in infants include ecthyma-like skin lesions and virus-associated transient neutropenia.[4] Other symptoms and signs, as in our patient, include vomiting, activity decrease, poor feeding, tachypnea, tachycardia, skin lesions, and hypotension.[3] *P. aeruginosa* bacteremia involving airway is rare and stridor was hardly found. But we did have experience with pseudomonas tracheitis associated with a more severe course including tracheal stenosis.[5] *P. aeruginosa* bacteremia could be presented by oral ulcers, pseudomembranous necrotizing pharyngitis, and epiglottitis.[6–8] Our patient had barking cough, stridor and progressive dyspnea with pseudomembrane coating over pharynx and tonsils. Fortunately, intubation or progression to tracheitis was avoided; early appropriate antimicrobial therapy may be the key point.

Skin lesions associated with *Pseudomonas* sepsis include ecthyma gangrenosum (21%—64%), subcutaneous nodules, gangrenous cellulitis, hemorrhagic vesicles, and bullae, papules, macules, petechiae, and purpura.[4,6,9] Ecthyma gangrenosum may be the first sign of *Pseudomonas* infection or develop in the later course.[9] It usually appears before the results of the blood culture and help clinicians to choose appropriate antibiotics.[6] Nonetheless, ecthyma gangrenosum could be caused by other pathogens, including group A *Streptococcus, Staphylococcus aureus, Aeromonas hydrophila, Serratia marcescens, Escherichia coli*, *Aspergillus* spp., *Mucor* spp., and *Candida*.[2,9,10]

Considering the antimicrobial therapy for *Pseudomonas* infection, dual therapy is often suggested for a synergistic effect in immunocompromised people, signs of septic shock, or when the susceptibility of the organism is in doubt. One study mentioned that incision and drainage for larger lesions and subcutaneous nodules would prompt rapid healing.[6] Repeated cultures of wound may be necessary for evaluating the possibility of resistance or the continuation of current antibiotic therapy.[9,11]

The overall mortality rate associated with *P. aeruginosa* bacteremia in children was 20%. The fatality rate was higher among the young infants (age 0 to 1 year; 36%) than among older children (age >5 years; 31%).[1] However, higher fatality rates have also been reported, ranging from 38% to 50%.[12,13] The overall cure rate was 62%; it was 67% for patients receiving appropriate antibiotics but only 14% for those receiving inappropriate antibiotics. One- to 2-day delays in appropriate antibiotic administrations reduced the cure rate from 74% to 46%.[13] One study of 133 episodes of *P. aeruginosa* bacteremia defined some variables as independent influencing factors of the outcome: development of septic shock, inappropriate antibiotic therapy, and the development of septic metastasis.[14] Compared with our patient, a 5-month-old boy with *P. aeruginosa* sepsis presented by ecthyma gangrenosum and pseudomembranous pharyngolaryngitis had no complications finally.

In conclusion, *P. aeruginosa* infection is rare in healthy children, but could occur in patients with croup syndrome and sepsis. Careful physical examination is important. Ecthyma gangrenosum may appear before finding out the pathogen. Early recognition and prompt treatment with antipseudomonal antibiotics is vital to reduce morbidity and potential mortality.

Conflicts of interest

All authors declare that they have no conflicts of interest related to the material discussed in this article.

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