CASE REPORT

An infant with severe acute respiratory distress syndrome and an opportunistic polymicrobial pulmonary infection: a case report

Rasha Assiri

Division of Microbiology, Department of Pathology, College of Medicine, King Abdullah Hospital, Princess Nourah Bint Abdulrahman University, Riyadh, Saudi Arabia

ABSTRACT

A 7-month-old male baby presented to the pediatric hematology unit for investigations of recurrent episodes of neutropenia associated with fever and respiratory distress. He developed a stormy course with severe respiratory distress syndrome associated with opportunistic polymicrobial pulmonary infections. Extensive radiological, microbiological, and immunological investigations suggest immunodeficiency with opportunistic polymicrobial pulmonary infections.

KEYWORDS:
Immunodeficiency; Opportunistic infection; Polymicrobial.

INTRODUCTION

Opportunistic infections usually do not cause disease in a healthy immune-competent person but can seriously affect people with a poorly functioning or suppressed immune system. If untreated, it can be severe and even fatal. It is difficult to diagnose infections in immunocompromised patients [1]. Persistent infections during neutropenia have a very high mortality rate of up to 100% [2]. Moreover, it is established that localization of the focus of infection and determination of the underlying microorganism are the most essential step for appropriate therapy [3].

Empiric antimicrobial treatment is usually started in suspected infection but it is highly important to identify the localization and etiology of the infection. Clinical examination, chest radiography, and routine cultures (e.g., blood, sputum, feces, urine, etc.) could help in the diagnosis of the case. If chest radiographs show an opacity, broncho-alveolar lavage is usually performed. Escalation of the antimicrobial therapy is usually followed whenever fever is persistent and still no focus is identified. In such situations, the search for the focus of infection may be repeated and/or expanded. As the upper airways are normally colonized by physiologic microorganisms, the

Correspondence to:
Rasha Assiri
Division of Microbiology, Department of Pathology, College of Medicine, King Abdullah Hospital, Princess Nourah Bint Abdulrahman University, Riyadh, Saudi Arabia.
Email: dr.rasha.2008@hotmail.com

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lungs are not only the main entry site for infection but also the most frequently involved organ [4].

Here, we describe an infant with severe acute respiratory distress syndrome complicated by an opportunistic polymicrobial pulmonary infection suggestive of immunodeficiency.

**CASE REPORT**

A 7-month-old Saudi male infant was admitted to the pediatric hematology unit for investigations of recurrent episodes of neutropenia associated with fever and respiratory distress for 3 days. His fever was intermittent, spiking up to 40°C, subsiding with antipyretics (paracetamol) with no special pattern. This was associated with a cough that was mild, irritative, not spasmodic, not related to feed, and slowly increasing in intensity and frequency. He had also a mild respiratory difficulty, especially during feeding. There was no history of cyanosis, eye puffiness, or excessive sweating. No history of vomiting or diarrhea. Other medical history was unremarkable. He had a history of previous repeated admissions to the hospital because of similar episodes of neutropenia and fever. His neonatal history was unremarkable except for low birth weight (birth weight was 1.440 kg). His umbilical cord stump was sloughed by the 7th day of life. He received his vaccinations including Bacillus Calmette–Guérin with no serious side effects or complications. There were neither similar conditions nor chronic illnesses in his family. His growth parameters were acceptable for age and birth weight. His physical examination upon hospital admission showed relatively sick looking, a lethargic baby with mild pallor, but no jaundice or cyanosis. His vital signs were the temperature of 38.5°C, heart rate (HR) of 156 beats/minute, blood pressure (BP) of 102/56 mmHg, respiratory rate of 46 breaths/minute, and oxygen saturation of 94% with 2 l/minute oxygen via nasal cannula. Otherwise, the rest of his physical examination was normal.

The initial complete blood count revealed white blood count of 1,700/ml with absolute neutrophil of 1,326. Liver and renal functions were within the normal range. Throat swab culture showed fungal growth, while blood culture was negative. His chest X-ray showed haziness of the lung parenchyma involving upper parts of both right and left upper lobes with a normal cardiac shadow (Figure 1). Abdominal and renal ultrasonic examination were normal. By the 3rd day of his hospitalization, he became lethargic, more tachypnoeic, and distressed. He was transferred to the Paediatric Intensive Care Unit (PICU) where his physical examination showed HR: 168 beats/minute, BP: 96/58 mmHg with substernal, intercostal, and suprasternal retractions, working ala nasai with bilaterally audible breath sounds on both sides of the chest and bilateral fine crepitations. There were no audible murmurs over the heart. The right lobe of the liver was palpable about 5 cm below the costal margin in the midclavicular line, soft to firm in consistency and non-tender. The rest of his physical examination was unremarkable. Chest radiograph showed a mild increase in the haziness of his right and left upper lung lobes (Figure 2). Nasal continuous positive airway pressure was applied and he started to maintain his oxygen saturation >92%. Ongoing antibiotic ceftazidime was replaced with piperacillin/tazobactam (Tazocin). Vancomycin and cotrimoxazole (Septrin) were added along

**Figure 1** - Chest X-ray of the patient showing haziness of lung parenchyma involving upper parts of both right and left upper lung lobes with normal cardiac shadow.
with amphotericin-B. Two of his chest X-rays throughout his PICU course are shown in Figure 3, while Figure 4 shows chest X-ray on the day of discharge from the PICU.

The baby developed severe acute respiratory distress syndrome (ARDS) with an air leak. All blood cultures were negative. Cultures from lung biopsy specimen grew *Pneumocystis jirovecii* and *Mycobacterium tuberculosis*. Serum immunoglobulin levels were normal. Lymphocyte subset studies result was consistent with absolute lymphopenia; (T CD3 14; 1%), which was very low. This suggests that the diagnosis of immunodeficiency superimposed with mixed pulmonary infection composed of *Candida albicans*, *Pneumocystis jirovecii*, Cytomegalovirus (CMV), and *M. tuberculosis*. Molecular study was not available to confirm the clinical diagnosis of immunodeficiency.

**DISCUSSION**

Opportunistic infection is the most serious concern in people with a poorly functioning or suppressed immune system. If untreated, it can have severe or even fatal consequence. The most prevalent opportunistic pathogens in immunocompromised subjects include CMV, *C. albicans*, *Pneumocystis jirovecii*, and *Mycobacterium*. CMV is a common infection in immunocompromised children. Both humoral and cellular immune mechanisms are important in protection against CMV. CMV infection involves almost all organ systems of the body, but pulmonary involvement is a major cause of serious and often fatal, pneumonia in children with congenital immunodeficiency, AIDS, organ or marrow transplantation, or malignancy [1]. *Candida* species are recognized as a leading contributor to morbidity and mortality in patients with primary immunodeficiencies, hematologic malignancies, HIV infection, prolonged neutropenia, and corticosteroid administration. Neutropenic children colonized with *Candida tropicalis* are at higher risk for dissemination compared with those colonized with *C. albicans*.
According to the guidelines of the American society of Infectious Diseases, the first line treatment of candidiasis includes amphotericin B and fluconazole, which are approved for use in pediatrics [6]. In the present case report, amphotericin B showed good efficacy, and it can also be used for other species of Candida while awaiting culture results [7]. Pneumocystis jiroveci, earlier known as Pneumocystis carinii, causes infection in infants with severe malnutrition and children with primary immunodeficiencies, including combined immune deficiency and hyper-IgM syndrome, hematologic malignancies, or bone marrow transplants. Patients with AIDS typically have a longer duration of symptoms and a more insidious presentation in comparison to non-HIV immunocompromised patients. In children with and without AIDS, physical examination at the time of initial evaluation may reveal tachypnoea, nasal flaring, and intercostal, subcostal, or supracostal retraction.

Pneumocystis disease can progress quickly, and the success of treatment depends largely on the stage of disease at the time of treatment initiation. Therefore, prompt diagnosis and treatment are essential. The first-line treatment is trimethoprim-sulfamethoxazole (TMP-SMZ) given either intravenously or orally. The second-line treatment includes oral Dapsone with oral or IV TMP-SMZ, oral Atovaquone, or IV Pentamidine isethionate. Treatment duration is 21 days. In the present case report, the patient condition was improved with IV Sulfamethoxazole (TMP-SMZ) along with piperacillin/tazobactam injections. For patients who present with moderate-to-severe disease (PO$_2$ <70 mm Hg or PAO$_2$-to-PAO$_2$ gradient >35 mm Hg), early corticosteroid administration is also indicated.

Mycobacterial infections are frequent, opportunistic pathogens associated with AIDS. With the onset of AIDS epidemic, disease due to both M. tuberculosis and atypical strains such as Mycobacterium avium-intracellular have been increasingly recognized in both AIDS and non-AIDS immunodeficient populations. For patients with suspected disseminated disease, mycobacterial blood cultures can be obtained for diagnosis. Positive cultures for TB should be evaluated for drug sensitivity testing [8]. In the present case report, the patient developed severe ARDS. It occurs in 1%–4% of all admissions to PICU. Patients with ARDS may benefit from the use of methylprednisolone [9]. Our patient was commenced on methylprednisolone which was tapered over several weeks.

In conclusion, we here presented an infant who developed acute severe respiratory distress syndrome associated with polymicrobial opportunistic infection suggestive of immunodeficiency. Extensive radiological, microbiological, and immunological investigations suggest immunodeficiency; however, the molecular study was not available to pinpoint the underline cause of immunodeficiency.

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