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Case report/ Kazuistyka

Is *Pneumocystis jiroveci* infection still dangerous for infants? – A case report

Czy Pneumocystis jiroveci *jest nadal zagrożeniem dla niemowląt? – opis przypadku*

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ABSTRACT

Pneumocystis jiroveci is still a dangerous pathogen. The child with extreme immaturity has gone through severe circulatory and respiratory failure after birth, a few episodes of infection including the central nervous system, and has gone through a lot of invasive procedures like intubation, long-term central catheters and parenteral nutrition. Inclusion of trimethoprim and sulfamethoxazole targeted therapy resulted in clinical improvement and gradual resolution of inflammatory changes.

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Introduction

Pneumocystis jiroveci pneumonia (PJP) is a type of interstitial pneumonia caused by a cosmopolitan species of fungus *Pneumocystis jiroveci* (P.j.). It is a microbe formerly known as a bacteria and protozoan, which is transmitted by droplets [1]. Immunocompetent people undergo P.j. infection asymptomatically. Patients with immunodeficiency present symptoms commonly associated with the respiratory system, but less frequently certain symptoms can be observed within lymphatic nodes, bone marrow, spleen, liver, urogenital system, small intestine, eyes or ears [2].

This pathogen was firstly defined as a factor inducing pneumonia in 1940. In 1980 first clinical case reports were described among patients suffering from malnutrition [3]. Contemporary data consider PJP treatment or prevention among transplant, oncological or HIV positive patients [4–6].

Preterm newborns who were long hospitalized in NICU, mechanically ventilated, antibiotic and steroid treated, malnutrition and who were put under invasive therapeutic and diagnostic procedures, as well as orphans are in an infection risk group [7]. There are no literary findings about PJP infections in that age group. HIV patients with PJP constituent 50% [1].

Early symptoms of PJP are nonspecific – dry cough, dyspnea, fever, reluctance to eat and weakness. Furthermore we may observe augmentation of secreted mucus, pain in the thoracic cavity and chills. Foamy mucus emanating from the oral cavity should be interpreted as a symptom that may suggest pneumocystic etiology of the pneumonia.

Observation of glass opacities in the diagnostic imaging, hypoxia, leukopenia, eosinophilia, hypoalbuminemia and other disorders related with the immune system should be considered indicating the possibility of PJP [5, 6].

Infection can be confirmed by showing the presence of P.j. in a specimen stained using Grocott-Gomori, Gram-Weigert or Giemsa method in immunofluorescence examination with antibodies against trophozoites and cysts (higher sensitivity) or using polymerase chain reaction (PCR). Diagnosis can be based on the sputum examination or trachea and stomach aspirate (sensitivity 20–40%). Bronchoscopy with bronchoalveolar lavage (BAL) is the diagnostic method of choice for intubated infants and children (sensitivity 55–97%, even after 72 h from the initiation of pharmacotherapy). Bronchoscopy with transbronchial biopsy, transdermal pulmonic biopsy, and open lung biopsy are characterized by high specificity and sensitivity, but not used because of their invasiveness. Histopathological examination shows alveolar spaces filled with foamic, amorphous material resembling rich in proteins exudative fluid. Exudative fluid is accompanied by moderate interstitial reaction sometimes forming glassy membranes. Pathogen P.j. (diameter 6 μm), attaches to pneumocytes, but does not penetrate them. It is visible in electronic microscopy and using special staining (silver plating, toluidine blue) [5, 6].

Serological examinations are the most common in clinical practice. Presence of IgG and IgM antibodies against P.j. antigen during the primary weeks of pneumonia, good response to treatment and the maintenance of IgG during convalescence may be useful in the confirmation of etiology of the disease [1].

Drug therapy includes sulfamethoxazole/trimethoprim, alternatively pentamidine or atovaquone [8]. We present pneumonia in extremely premature newborn who was long-term treated in NICU and had many prematurity complications. The course of infection was nonspecific but extremely severe. Etiological diagnosis and proper treatment introduction led to clearance of inflammatory lesions in lungs.

A case report

A child from first pregnancy, born naturally outside our center, with extreme immaturity (GA 24 w), tangled threatening miscarriage and preterm birth, with full steroids prenatal therapy. The general condition was serious (according to Apgar score in 1′ of life – 1 point, 5′ – 6 points) with a body weight 680 g. After the birth baby was intubated, and given Curosurf intratracheally. Since first day of life the newborn was in a very serious clinical condition, with symptoms of severe respiratory and circulatory insufficiency, features serious infection, which was diagnosed based on clinical symptoms, white blood cell counts – 44 000/1 mm^3 in the first day of life, 29 000/1 mm^3 in the second day of life, and a high percentage of immature forms of neutrophils (I/T ratio 0.4). The parameters of conventional ventilation were high: peak inspiratory pressure (PIP) 20 cm H_2O, positive-end expiratory pressure (PEEP) + 5 cm H_2O, fraction of inspired oxygen (FiO_2) 0.3–0.24 and frequency of breaths 40/min. The first ultrasound of central nervous system revealed intraventricular hemorrhage second degree on both sides and the cavity of periventricular leukomalacia in the right hemisphere.

Due to severe respiratory failure we continued mechanical ventilation, supported temporarily with high frequency ventilation.

In the 5th day of life we introduced successful pharmacological ligation of hemodynamically significant patent ductus arteriosus. Control echocardiography in the 18th day of life presented recanalization of the ductus arteriosus. Surgical ligation was performed on the 24th day of life. Because of anemia 5-fold concentrate of red blood cells was transfused.

Despite many attempts to modify the ventilation parameters to 28-day of life the patient could be not extubated. Diagnosed with bronchopulmonary dysplasia, the patients received a course of systemic steroids (hydrocortisone). In the further course of the trial there was twice more severe generalized infection, including meningitis, requiring long-term antibiotic therapy. In its course there was necrotizing enterocolitis, operated in the Children's Memorial Health Institute with emergence of ileostomy in 33 day of life. In the following weeks of life hypothyroidism, cytomegalovirus infection, retinopathy of prematurity, anemia and cholestasis were diagnosed. Assisted ventilation was continued until 90 day of life, levothyroxine, ganciclovir, blocker of VEGF – A (Lucentis), hematopoietic – NeoRecormon, iron and hematopoietic vitamins, and bile drugs – Ursopol were used in the treatment.

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