250-mg doses for the subsequent 4 days. The patient’s condition improved by day 3 of therapy, although her symptoms relapsed 12 days later.

Similar improvement and relapse followed a second 5-day course of azithromycin treatment. Thereafter, 250 mg of azithromycin was given daily for a total of 30 days; this therapeutic course resulted in a marked decrease in her symptoms. She returned to full-time work as a manager at her company and maintained an energy level of 8–9 of 10 for the next 2 years. Follow-up antibody titers are shown in Table 1.

Most of our patients had symptoms referable to the upper or lower respiratory tracts, but radiographic studies of the sinuses and chest were unremarkable. Seven of the 10 patients had high levels of total antibody to C. pneumoniae 0.5 to 3 years following an episode of symptomatic respiratory infection. Three patients had low antibody titers of 1:128 and 1:256, but their symptoms did decrease with antibiotic therapy. Low or absent antibody response to C. pneumoniae was documented for patients with persistently positive respiratory cultures [8]. Comparatively, the mean titers ± SD for 90 controls were 32 ± 36 (range, <8 or 4 to 256). Only two of 19 fatigued patients with reciprocal titers between 32 to 64 responded to 1 month of azithromycin treatment (data not shown).

The spontaneous rise of titers for several patients correlated with an increased severity of fatigue and a concomitant increase in respiratory symptoms. This observation suggests that relapses of symptoms could be due to persistent infection with periodic reactivation rather than reinfection. All of the patients with relapses responded to additional azithromycin treatment.

C. pneumoniae is a common coinfection in patients with respiratory infection [9]. Symptoms of acute purulent sinusitis and mastoiditis in patients 7 and 8, respectively, did decrease after 2–3 weeks of ceftriaxone treatment, but severe fatigue persisted for the next 2 years; fatigue resolved only after 2 months of azithromycin treatment.

Recently, Falck et al. [10] found C. pneumoniae DNA in throat secretions from 10 of 11 patients with chronic rhinorrhea, fatigue, and throat biopsies positive for C. pneumoniae. Seventy percent of the patients had elevated titers of IgG (1:512) or IgA (1:128) antibody. All of their patients responded to prolonged courses of macrolide therapy, but symptoms frequently recurred.

Collectively, these results suggest that C. pneumoniae is an uncommon yet treatable cause of chronic fatigue. The sensitivity, specificity, and interlaboratory variability of the DNA test will need to be better defined. Although seemingly less sensitive and prone to interlaboratory variation, the widely available microimmunofluorescence test may be a practical screening test for this entity before throat biopsy is performed.

References


Acute Fever and Petechial Rash Associated with Influenza A Virus Infection

The child with acute fever and petechial exanthem is a challenge to the primary care physician, because several rapidly fatal diseases present in this manner [1]. Of patients with fever and petechiae, 8% to 20% have a serious bacterial infection, and 7% to 10% have meningococcal sepsis or meningitis [2]. Petechial rash illnesses, often with aseptic meningitis, are relatively common manifestations of infections due to several enteroviruses, and these infections usually occur in the summer or fall [1, 3, 4]. Viral exanths, which occur in the winter and spring, are not uncommon but are rarely petechial [4, 5].

A previously healthy 3-year-old boy was admitted to UCLA Children’s Hospital (Los Angeles) in mid-January because of the sudden development of a petechial rash. He had a 3-day history of fever (temperature to 39.7°C), cough, and rhinorrhea. One day before admission, he was treated with trimethoprim-sulfamethoxazole for left otitis media. He received a total of three doses. On the day of admission, his parents noted the petechial rash that was initially on the face but had spread to the trunk and back within hours. There was no history of recent travel or contact with ill persons. He attended day care.

Reprints or correspondence: Dr. James D. Cherry, UCLA School of Medicine, Department of Pediatrics, MDCC 22-442, 10833 Le Conte Avenue, Los Angeles, California 90024 (jcherry@pediatrics.medsch.ucla.edu).

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At admission, the child was in no acute distress, but his temperature was 40.1°C. The heart rate was 137, the respiratory rate was 30, and the blood pressure was 121/75 mm Hg. He had clear rhinorrhea and flat scattered petecchiae on his face, chest, upper back, and right arm. The lesions on the face and trunk were very small (1–2 mm in size), while the ones on the right hand had a purpuric appearance due to apparent coalescence.

Laboratory studies disclosed the following: WBC count, 8,400/mm³ (with 77% neutrophils, 15% lymphocytes, 6% monocytes, and 2% basophils); hemoglobin level, 14.3 g/dL; platelet count, 119,000/mm³; and erythrocyte sedimentation rate, 16 mm/h. Urinalysis was unremarkable, and blood culture for bacteria revealed no growth. Nasal washing was performed, and a specimen was submitted for virus culture and direct viral antigen detection. Influenza A virus was isolated on day 3 in rhesus monkey kidney cells by shell vial culture.

The child was treated with cefotaxime. In the hospital, no new petechial lesions occurred, and the temperature returned to normal in 48 hours; at this time, the child was discharged to home with the rash resolving.

The classic illness due to influenza virus infection consists of the sudden onset of fever, chills, headache, myalgia, arthralgia, cough, sore throat, and rhinorrhea [6, 7]. In younger children, the illness may present with different patterns including classic disease, a nonspecific febrile illness, or other respiratory tract manifestations such as croup, bronchitis, bronchiolitis, and pneumonia [6, 7]. In addition, this age group may have gastrointestinal symptoms [7], and other findings such as seizures and rashes can be seen in a small percentage of patients [5, 6].

The rash in influenza virus infection is usually macular or maculopapular but has been so florid that it has been mistaken for measles [8]. In an extensive evaluation in a general practice in England, Hope-Simpson and Higgins [5] noted that about 8% and 2% of influenza B and influenza A virus infections, respectively, were associated with rash. The rashes were not described however. Ryan-Poirier [6] reported that macular, maculopapular, and petechial rashes occur in a small percentage of children with influenza virus infections, but no specific cases were presented.

The petechial rash in this child could have been due to the administration of trimethoprim-sulfamethoxazole rather than the viral infection [9]. However, the lack of a maculopapular component to the rash and its short duration favor a viral etiology. In the absence of thrombocytopenia, there are no reports of isolated petechial rashes associated with trimethoprim-sulfamethoxazole therapy.

An additional explanation for this presentation could be concurrent meningococcal infection. The association of influenza A virus infection and meningococcal disease is well known [10]. Treatment with trimethoprim-sulfamethoxazole could have aborted the infection and sterilized the blood culture. However, the normal erythrocyte sedimentation rate and WBC count argue against the possibility of bacteremia.

In summary, we describe a child with an acute illness suggestive of bacteremia (fever and petechial rash) that was associated with influenza A virus infection. As in the summer and fall when fever and petechial rash illnesses due to enterovirus infections are common, children with this syndrome in other seasons should be treated for meningococcemia even though a viral etiology may be likely.

Monica E. Silva, James D. Cherry, Richard J. Wilton, Negar M. Ghafouri, David A. Bruckner, and Marjorie J. Miller
Departments of Pediatrics and Pathology and Laboratory Medicine, UCLA School of Medicine and UCLA Children’s Hospital, Los Angeles, California

References

Ecthyma Secondary to Herpes Simplex Virus Infection

Ecthyma is a cutaneous infection generally caused by bacterial organisms. We report the first case of ecthyma caused by herpes simplex virus (HSV).

A 73-year-old male with a 2-year history of IgA κ subtype multiple myeloma presented with erythematous papules and plaques with central ulceration and necrosis. The multiple myeloma had been previously treated with a course of cyclophosphamide and prednisone followed by a course of cyclophosphamide and chlorambucil. Because the most recent bone marrow biopsy revealed >75% plasma cells and the patient was developing progressive renal insufficiency, he was treated with methylprednisolone (2 g intravenously three times per week) during the 2 weeks before presentation.

Physical examination was significant for several erythematous, edematous plaques (~2–3 cm in diameter) with prominent central necrotic eschars that were present on the patient’s midback (figure 1). One of the lesions had two 2-mm vesicles