Brief Reports

Lobar pneumonia associated with adenovirus type 7

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A high proportion of acute respiratory tract infections are viral. However, adenoviruses account for less than 5% of these infections.[1-5] Most of the clinically significant adenovirus infections occur in children under 3 years of age[6-8] and in military populations.[9,10] The annual attack rate for civilian university students had been estimated to be as low as one adenovirus infection for every 5000 students.[1]

Adenovirus infection is usually a mild, febrile upper respiratory tract illness without distinguishing features.[4,5] Severe, sometimes fatal, lower respiratory tract infections, with bronchitis, bronchiolitis and bronchopneumonia, have been associated with adenovirus types 3, 4, 7 and 21.[6-8,11-14] Intestinal, myocardial, cerebral, renal, hepatic and other extrapulmonary manifestations may accompany adenovirus pneumonia in children and adults.[6,7,13,14] Bronchiectasis, persistent lobar collapse and residual lung fibrosis are common findings in follow-up studies after severe pneumonia. The incidence of these sequelae varies from 12% to 90%.[6-8]

In this report we present a case of lobar pneumonia in a young adult that radiologically mimicked bacterial lobar pneumonitis. The only pathogenic organism isolated was adenovirus type 7, and the serologic findings implicated this virus as the causative agent. To the best of our knowledge this is the first report of lobar pneumonia caused by an adenovirus.

Case report

Clinical course

An 18-year-old student presented to his family physician with a 3-day history of a sore throat associated with malaise, diffuse myalgia, a temperature spiking up to 38°C and profuse sweating. He had a cough but no difficulty breathing.

When there was no response to 3 days of oral penicillin therapy he was sent for consultation. The consultant found mild injection of the pharynx and signs of consolidation in the upper lobe of the right lung. Cyanosis and lymphadenopathy were absent.

Chest roentgenograms confirmed right upper lobe pneumonia suggestive of bacterial pneumonitis. The blood hemoglobin level was 14 g/dl, the leukocyte count 7.7 × 10^9/l (61% neutrophils, 31% lymphocytes, 6% monocytes and 2% eosinophils) and the erythrocyte sedimentation rate 20 mm/h. A tuberculin skin test with 5 U of purified protein derivative done at the initial visit was negative when read 48 hours later.

Pending further laboratory results the oral penicillin therapy was continued for 1 week. The patient was afebrile 5 days after the onset of symptoms and recovered uneventfully. A chest roentgenogram made 2 weeks after the onset of symptoms revealed complete resolution of the pneumonic consolidation.

Microbiologic studies

No pathogenic bacteria could be cultured from sputum samples obtained at the onset of the illness and on the day the patient presented for consultation.

Throat washings were also obtained from the patient on the day of his presentation to us. Electron microscopy did not reveal virus particles in these specimens. Aliquots were inoculated into HeLa, HEp-2, Rhesus monkey and human embryonic kidney (HEK) cell cultures. Cytopathologic changes characteristic of adenovirus infection appeared in the HEK cell cultures 9 days after inoculation. Particles with adenovirus features were seen in these cultures by electron microscopy, and the virus was identified as adenovirus type 7 by a neutralization test. The inoculated specimens did not yield virus in the remaining three cell lines.

A serum specimen was not obtained in the acute stage of the patient's illness because a viral illness was not suspected. The first such specimens were obtained 2 weeks after the onset of the illness, and additional specimens were obtained during the 2nd, 5th, 9th and 14th months after the onset. The titres of complement-fixing antibodies to adenovirus were 1:64 in the initial

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specimen, 1:128 in the specimens obtained during the 2nd and 5th months and 1:32 in the specimens taken during the 9th and 14th months. Complement-fixation tests for antibodies to influenza A virus revealed a titre of 1:64 in the first three serum specimens and a titre of 1:32 in the final two specimens. The titres of complement-fixing antibodies to other viruses and to *Mycoplasma pneumoniae* were unremarkable.

Discussion

Chest roentgenograms revealed that our patient was suffering from lobar pneumonia. Bacterial infection almost exclusively causes this condition. However, culture of several sputum specimens failed to reveal any pathogenic bacteria, the hematologic findings were not in accord with a bacterial origin for the illness, and the patient did not respond to antibiotic therapy. The only pathogenic organism isolated during the acute stage of the patient's illness was adenovirus type 7. A diagnostic increase in the titre of complement-fixing antibodies to adenovirus could not be demonstrated since no serum specimens were obtained during the acute stage of the illness. However, complement-fixation tests of serum specimens obtained during the second and fifth months after the onset of the illness revealed a high titre (1:128) of antibodies to adenovirus, and the titre declined thereafter. These observations indicate an adenovirus infection coincident with the patient's illness.

Adenoviruses are usually upper respiratory tract pathogens. The susceptibility of certain individuals to infections of the lower respiratory tract by adenoviruses is unexplained. Malnutrition is a strong predisposing factor[6,15,16] but did not play a role in our patient. The pathogenicity of adenoviruses may be increased when infection occurs in a respiratory tract that has been damaged by a preceding viral infection.[6,7,12] Complement-fixation tests of our patient's serum after his recovery also provided evidence of an infection with influenza A virus about the time of the adenovirus infection. This suggests that a preceding infection with influenza A virus may have predisposed him to a more severe clinical course of adenovirus infection.

The unusual feature of this case was the patient's presentation with lobar pneumonia rather than the interstitial pattern expected of adenovirus pneumonia. A poor response to antibiotic therapy, negative results of bacterial cultures and nonsupportive hematologic findings in cases of lobar pneumonia in young adults should make physicians consider a viral cause.

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