

# Viral Pneumonias in Adults: Radiologic and Pathologic Findings<sup>1</sup>

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## LEARNING OBJECTIVES

*After reading this article and taking the test, the reader will be able to:*

- Recognize the radiographic and CT findings of various viral pneumonias in adults.
- Describe the correlations between the radiologic findings and the histopathologic findings.
- Discuss differentiation between various viral pneumonias on the basis of radiologic and clinical features.

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Numerous viruses, including influenza virus, measles virus, Hantavirus, adenovirus, herpesviruses, varicella-zoster virus, cytomegalovirus, and Epstein-Barr virus, can cause lower respiratory tract infection in adults. Viral pneumonia in adults can be classified into two clinical groups: so-called atypical pneumonia in otherwise healthy hosts and viral pneumonia in immunocompromised hosts. Influenza virus types A and B cause most cases of viral pneumonia in immunocompetent adults. Immunocompromised hosts are susceptible to pneumonias caused by cytomegalovirus, herpesviruses, measles virus, and adenovirus. The radiographic findings, which consist mainly of patchy or diffuse ground-glass opacity with or without consolidation and reticular areas of increased opacity, are variable and overlapping. Computed tomographic findings, which are also overlapping, consist of poorly defined centrilobular nodules, ground-glass attenuation with a lobular distribution, segmental consolidation, or diffuse ground-glass attenuation with thickened interlobular septa. The radiologic findings reflect the variable extents of the histopathologic features: diffuse alveolar damage (intraalveolar edema, fibrin, and variable cellular infiltrates with a hyaline membrane), intraalveolar hemorrhage, and interstitial (intrapulmonary or airway) inflammatory cell infiltration. Clinical information such as patient age, immune status, community outbreaks, symptom onset and duration, and presence of a rash remain important aids in diagnosis of viral causes.

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**Abbreviation:** AIDS = acquired immunodeficiency syndrome

**Index terms:** Lung, infection, 60.206 • Pneumonia, 60.21 • Viruses, 60.206, 60.2062, 60.2063, 60.2064, 60.2065, 60.2066, 60.2069

**RadioGraphics 2002;** 22:S137–S149

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## Introduction

Numerous viruses may cause lower respiratory tract infection in adults. The viruses include influenza virus, adenovirus, measles virus, Hantavirus, varicella-zoster virus, and cytomegalovirus (1).

Clinically, viral pneumonia in adults can be divided into two groups: so-called atypical pneumonia in otherwise normal hosts and viral pneumonia in immunocompromised hosts (2). Influenza virus types A and B account for the majority of viral pneumonias in immunocompetent adults. Immunocompromised hosts are susceptible to pneumonias caused by cytomegalovirus and herpesviruses, as well as measles virus and adenovirus (Table 1). In specific populations (recent military recruits and some immunocompromised patients), the prevalence and severity of these pneumonias are high. Adenovirus pneumonia was common in military recruits prior to the development of a live attenuated adenovirus vaccine (3). Therefore, recognition of the various radiologic manifestations of viral pneumonias in conjunction with patients' clinical history is imperative for narrowing the differential diagnosis and determining appropriate management.

In this pictorial review, we present the radiologic and pathologic findings of viral pneumonias in adults. Specific topics discussed are common pathologic findings, common radiologic findings, viruses, and various viral pneumonias.

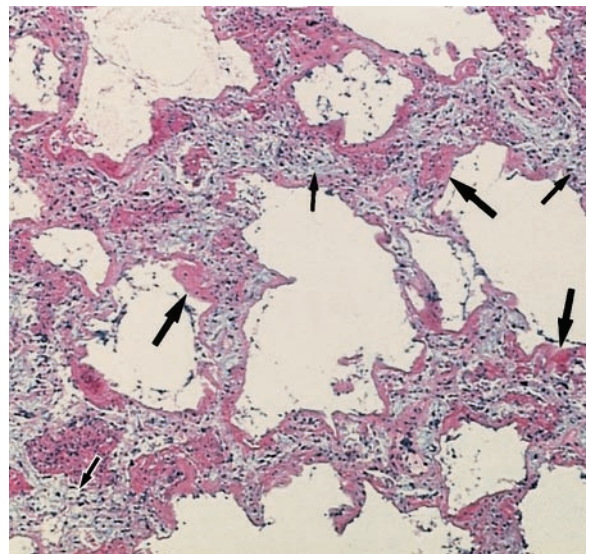
## Common Pathologic Findings

Viruses can result in several pathologic forms of lower respiratory tract infection including tracheobronchitis, bronchiolitis, and pneumonia. Because the organisms replicate within tissue cells, the most prominent histologic changes are seen in the epithelium and adjacent interstitial tissue. In tracheobronchitis, airway walls are congested and the lumen contains mononuclear cell infiltrates. Degeneration and desquamation of the epithelial cells are seen. Bronchiolitis, which is particularly important in children, appears with epithelial necrosis, neutrophilic exudate in the airway lumen, and predominantly mononuclear infiltrates in its wall (4,5).

Parenchymal involvement (pneumonia) initially involves the lung adjacent to the terminal

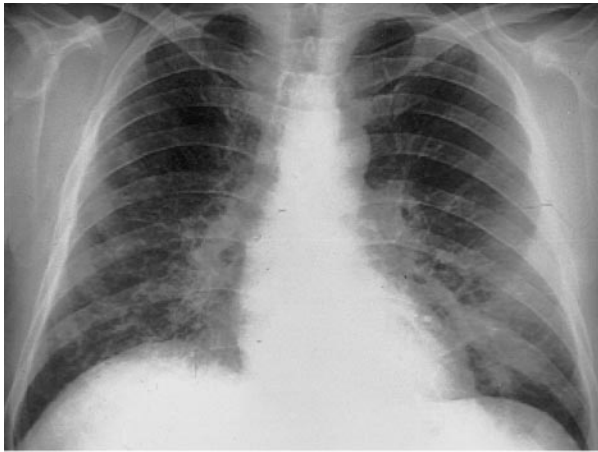
**Table 1**  
**Common Viral Infections in Immunocompetent and Immunocompromised Patients**

Immunocompetent patients
Influenza viruses
Hantaviruses
Epstein-Barr virus
Adenoviruses
Immunocompromised patients
Herpes simplex viruses
Varicella-zoster virus
Cytomegaloviruses
Measles virus
Adenoviruses



**Figure 1.** Photomicrograph (original magnification,  $\times 100$ ; hematoxylin-eosin stain) of a lung biopsy specimen from a 36-year-old man with pneumonia due to herpes simplex virus type 1 shows a fibrous exudate (large arrows) along the alveolar walls. Note the interstitial thickening due to fibroblastic proliferation (small arrows).

and respiratory bronchioles; however, extension throughout the lobule may occur. Rapidly progressive pneumonia may be seen particularly in the elderly and in immunocompromised patients (6). In these circumstances, the lungs histologically show diffuse alveolar damage comprising interstitial lymphocyte infiltration, air-space hemorrhage, edema and fibrin, type 2 cell hyperplasia, and hyaline membrane formation (7,8) (Fig 1).



a.



b.



c.

**Figure 2.** Pneumonia due to influenza virus (type C) in a 46-year-old man with dyspnea. **(a)** Initial chest radiograph shows diffuse reticulonodular areas of increased opacity in both lungs. **(b)** Follow-up chest radiograph obtained 15 days after **a** shows progression of disease with diffuse consolidation throughout both lungs. **(c)** Thin-section (1-mm collimation) computed tomographic (CT) scan obtained 16 days after **a** at the level of the aortic arch shows diffuse ground-glass attenuation with some irregular linear areas of increased attenuation in both lungs. (Case courtesy of Dr Jung Hwa Hwang, Soonchunhyang University Seoul Hospital, Korea.)

### Common Radiologic Findings

Tracheobronchitis seldom results in any radiologic abnormalities in the acute stage; however, mucosal injury may manifest many years later as bronchiectasis. With bronchiolitis, the airway obstruction is usually partial and results in hyperinflation and poorly defined nodular opacities radiologically.

Viral pneumonia manifests radiologically as poorly defined nodules (air-space nodules of 4–10 mm in diameter) and patchy areas of peribronchial ground-glass opacity and air-space consolidation. Because of the associated bronchiolitis, hyperinflation is commonly present (4,5). The progressive form of pneumonia shows the rapid confluence of consolidation leading to diffuse alveolar damage, consisting of homogeneous or patchy unilateral or bilateral air-space consolidation and ground-glass opacity or poorly defined centrilobular nodules (9) (Fig 2).

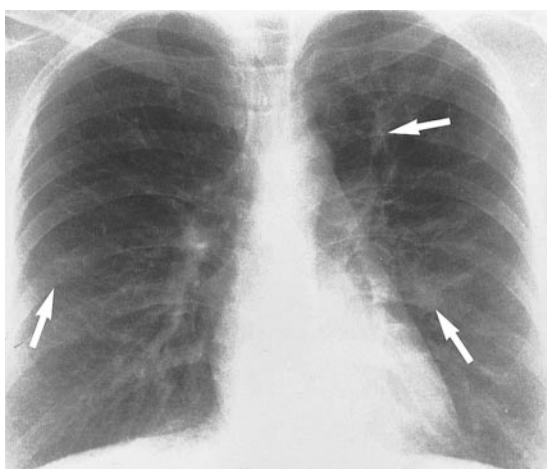
## Viruses

The respiratory viruses can be divided into two large groups according to the type of nucleic acid they contain: (a) the RNA group, which includes myxoviruses (influenza viruses and measles virus) and Hantaviruses; and (b) the DNA group, which includes adenoviruses and herpesviruses (herpes simplex type 1, varicella-zoster virus, cytomegaloviruses, Epstein-Barr virus).

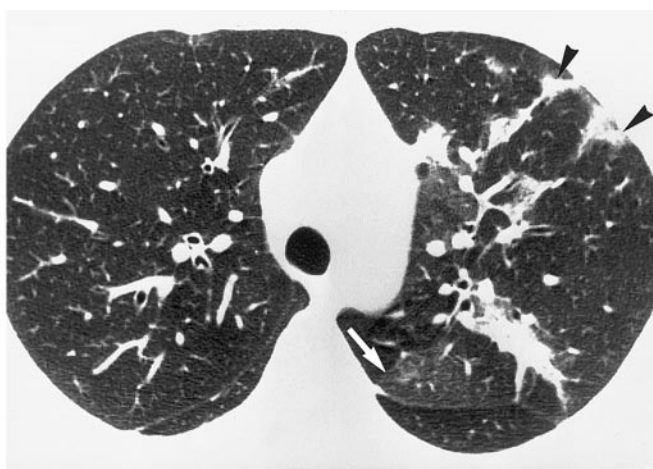
## Various Viral Pneumonias

### Influenza Virus Pneumonia

Influenza virus infection usually involves the upper respiratory tract including the trachea and major bronchi in children and young adults. However, elderly and immunocompromised persons are at increased risk for development of fulminant pneumonia. Influenza viruses are divided into three groups (A, B, and C) according to internal membrane and nucleoprotein antigens. Of these three groups, type A and occasionally type

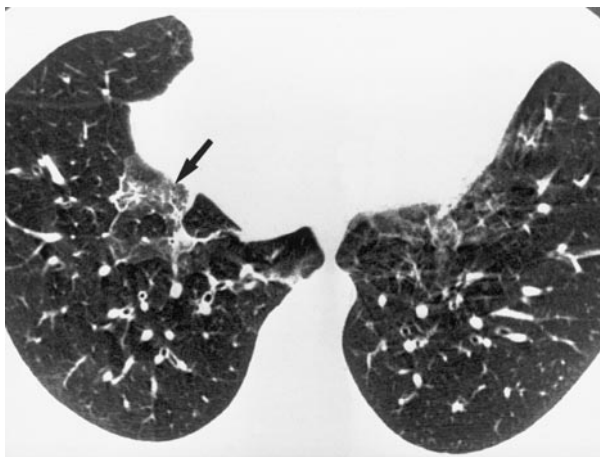


a.



b.

**Figure 3.** Pneumonia due to influenza virus in a 21-year-old man with a cough. **(a)** Initial chest radiograph shows poorly defined nodules (arrows) and reticular areas of increased opacity in both lungs. **(b, c)** Thin-section (1-mm collimation) CT scans obtained at the levels of the aortic arch **(b)** and suprahepatic inferior vena cava **(c)** show multifocal peribronchovascular or subpleural consolidation and ground-glass attenuation in both lungs. Some lesions have a lobular distribution (arrows). Note the acinar nodules (arrowheads). (Case courtesy of Jin Mo Goo, MD, PhD, Seoul National University Hospital, Korea.)



c.

B organisms cause influenza virus pneumonia (10). Although the pneumonia is usually mild, it can be overwhelming and fatal within 24 hours. Predisposing conditions for the infection include mitral stenosis, pregnancy, diabetes, old age, and immunosuppression (6,11).

Histologically, airway walls are congested and lumina contain mononuclear cell infiltrates. Degeneration and desquamation of the epithelial cells are seen. Parenchymal change shows typical features of diffuse alveolar damage (7,12).

Serial radiographs show poorly defined, patchy areas of air-space consolidation, 1–2 cm in diameter, that rapidly become confluent (Fig 2). Diffuse or patchy areas of ground-glass attenuation mixed with consolidation are frequently seen at CT (Fig 2). Small centrilobular nodules representing alveolar hemorrhage may be associated (Fig 3). Pleural effusion is rare. The radiologic abnormalities usually resolve in approximately 3 weeks (9). In one study, influenza virus pneumonia showed air-space consolidation or ground-glass attenuation with a lobular distribution at high-resolution CT. Hyaline membrane formation in the alveolar parenchyma around the bron-

chiole probably accounts for the predominant CT findings of air-space consolidation or ground-glass attenuation with a lobular distribution (13).

### Measles Virus Pneumonia

Measles virus infection is a disease of small children. Even with active immunization, a significant number of older individuals develop the disease, probably due to combined causes of nonimmunization, vaccine failure, and exposure to the organism in later adulthood (14). Pulmonary disease from measles virus infection occurs mainly in two forms: one is primary measles virus pneumonia and secondary bacterial pneumonia and the other is atypical measles virus pneumonia. Although measles virus can cause pneumonia in 3%–4% of infected patients, the majority of affected patients with this organism have secondary bacterial infection. *Haemophilus influenzae* and *Neisseria meningitidis* are the most common organisms of the secondary bacterial infection (15).



**Figure 4.** Pneumonia due to measles virus in a 13-year-old boy with a fever, cough, and skin rash. The patient had an increased immunoglobulin M antibody titer to measles virus. Chest radiograph shows poorly defined nodules and patchy consolidation in the left middle and lower lung zones.

Atypical measles develops when children who have been immunized with inactivated measles viruses are exposed again to measles virus or to live measles virus vaccine. Although not proved, an immune mechanism is regarded as the underlying pathophysiology because clinical and radiologic findings change rapidly (16).

The prevalence of measles virus pneumonia is higher in pregnant women and patients who are immunocompromised due to hematologic malignancy (leukemia or lymphoma), acquired immunodeficiency syndrome (AIDS), or immunosuppressive therapy (17–19).

Measles virus pneumonia without bacterial superinfection appears with epithelial hyperplasia and diffuse alveolar damage. Epithelial hyperplasia associated with many foci of squamous metaplasia is seen in bronchioles and peribronchial alveoli. It is also seen in the tracheobronchial epithelium with cystic dilatation of mucous glands. Histologically, measles virus pneumonia is characterized by multinucleated giant cells containing up to 50 nuclei in the alveolar air spaces and within the bronchiolar and tracheobronchial epithelium (18).

Chest radiographic findings of primary measles virus pneumonia are mixed reticular opacities and air-space consolidation (15) (Fig 4). In children, lymph node enlargement in the hilum may be associated. CT findings include ground-glass attenuation, air-space consolidation, and small

centrilobular nodules (20). These findings represent the underlying pathologic mechanism of diffuse alveolar damage, producing both interstitial and air-space diseases. Atypical measles virus pneumonia appears with spherical or segmental consolidation, which clears rapidly. Hilar lymph node enlargement and pleural effusion are frequently associated (16).

### Hantavirus Pneumonia

Hantaviruses are lipid-enveloped, single-stranded RNA viruses. Several antigenically different viruses from around the world (Hantaan, Seoul, Puumala, Dobrava, Prospect Hill, and Sin Nombre) have been found to cause a typical symptom complex called *hemorrhagic fever with renal syndrome*. Infected patients experience clinically characteristic courses of fever, hypotension, and renal failure. The most recently identified sixth organism (Sin Nombre virus) is known to more frequently cause severe and fulminant pulmonary disease than other organisms (21,22).

The natural reservoir of all Hantaviruses is wild rodents and deer mice, the latter being the most important animal harboring the Sin Nombre variant in the United States (21,23). The organism is believed to be transmitted to humans by inhalation of dried rodent excreta associated with outdoor activities in rural areas, such as cleaning barns, plowing with hand tools, and harvesting rice.

Hantavirus pulmonary syndrome characteristically presents as respiratory distress from noncardiogenic edema. After an incubation period of 9–35 days, the syndrome begins to progress through its three stages. The initial stage is the prodromal phase, which is followed by the cardiopulmonary and convalescent phases.

Histologically, interstitial and air-space edema, mild to moderate interstitial infiltrates of lymphocytes, epithelial necrosis, vascular thrombosis, and hyaline membranes are seen (24). The lung disease in the Hantavirus syndrome has some distinct pathologic differences from diffuse alveolar damage due to other causes. They are extensive cellular debris, destruction of type I cells, prominence of type II cells, neutrophil infiltrates, and fibrosing alveolitis (24).

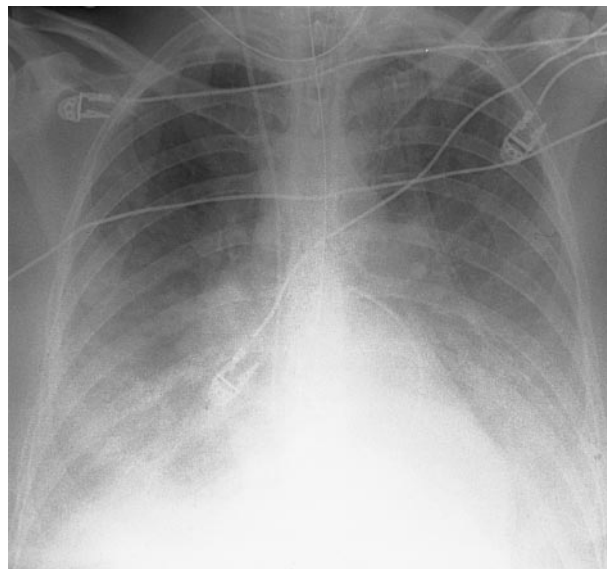
Radiographically, Hantavirus pulmonary syndrome presents as interstitial edema with or without rapid progression to air-space disease. The air-space disease shows a central or bibasilar distribution. Also, pleural effusion is a common



a.

b.

**Figure 5.** Pneumonia due to Hantavirus (*Sin Nombre* species) in a 39-year-old American woman with dyspnea. (a, b) Initial posteroanterior (a) and lateral (b) chest radiographs show bilateral perihilar and basilar ground-glass opacity. Fissures are accentuated on the lateral radiograph (b). The heart is of normal size. (c) Follow-up anteroposterior chest radiograph obtained 4 days after a shows progression to extensive bilateral consolidation in the middle and lower lung zones.



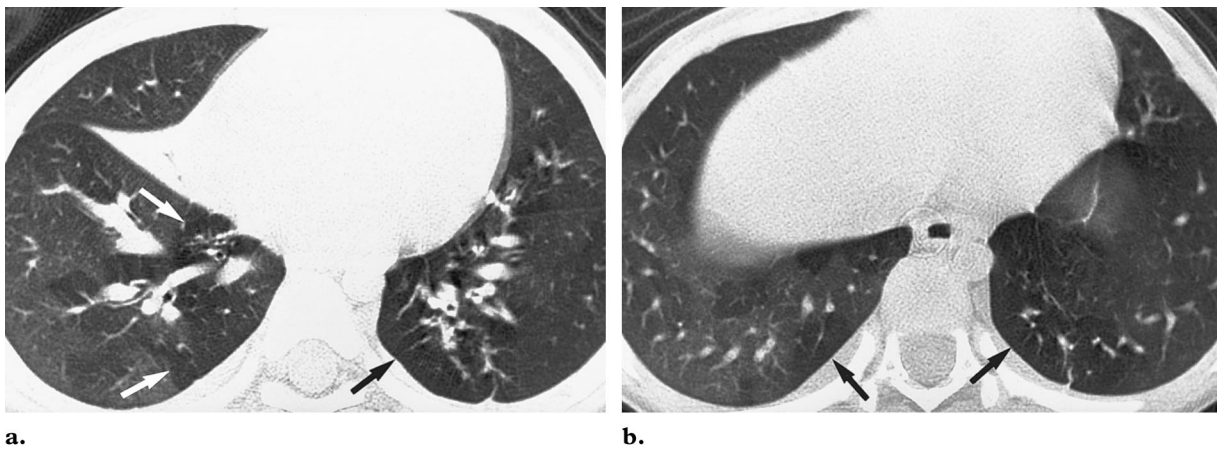
c.

finding. The radiographic findings in Hantavirus pulmonary syndrome are consistent with a pulmonary capillary leak (Fig 5). However, pulmonary manifestations may occasionally be secondary to renal failure. In these cases, pulmonary edema and cardiomegaly with or without pleuropericardial effusion are the predominant findings in the oliguric phase of renal failure (25). However, in infection of the *Sin Nombre* organism, more severe findings with diffuse alveolar damage are usually seen (Fig 5). Recently, Boroja et al (23) identified two broad categories of Hantavirus pulmonary syndrome clinically and radiographically: (a) a rapidly progressive, fulminant, and often fatal clinical form with radiographic features of rapidly progressive alveolar pulmonary edema, air-space consolidation, and pleural effusions;

and (b) a limited, less severe clinical form usually associated with mild interstitial edema and minimal air-space disease. All patients with the limited form of Hantavirus pulmonary syndrome survived the illness, whereas 46% of those with the fulminant form died.

### Adenovirus Pneumonia

Adenovirus infection occurs frequently and may represent 7% of viral respiratory tract infections (26). At present, 42 immunologically distinct adenoviral serotypes have been isolated from humans (4). The infection may manifest as pharyngitis, pharyngoconjunctivitis, laryngotracheobron-



**Figure 6.** Pneumonia due to adenovirus in a 15-year-old boy. Thin-section (1-mm collimation) CT scans obtained at the levels of the inferior pulmonary vein (**a**) and the dome of the liver (**b**) show complete atelectasis of the right middle lobe and multifocal air trapping (arrows), an appearance indicative of acute bronchiolitis.

chitis, bronchiolitis, or pneumonia. The adenovirus organism may also be involved in the pathogenesis of bronchiectasis (27). Adenovirus pneumonia in adults was commonly identified in military recruits prior to the development of a live attenuated adenovirus vaccine but now is uncommon. Although immunocompromised patients are more prone to develop adult respiratory distress syndrome with adenovirus pneumonia, the condition may also be a complication in immunocompetent patients (3).

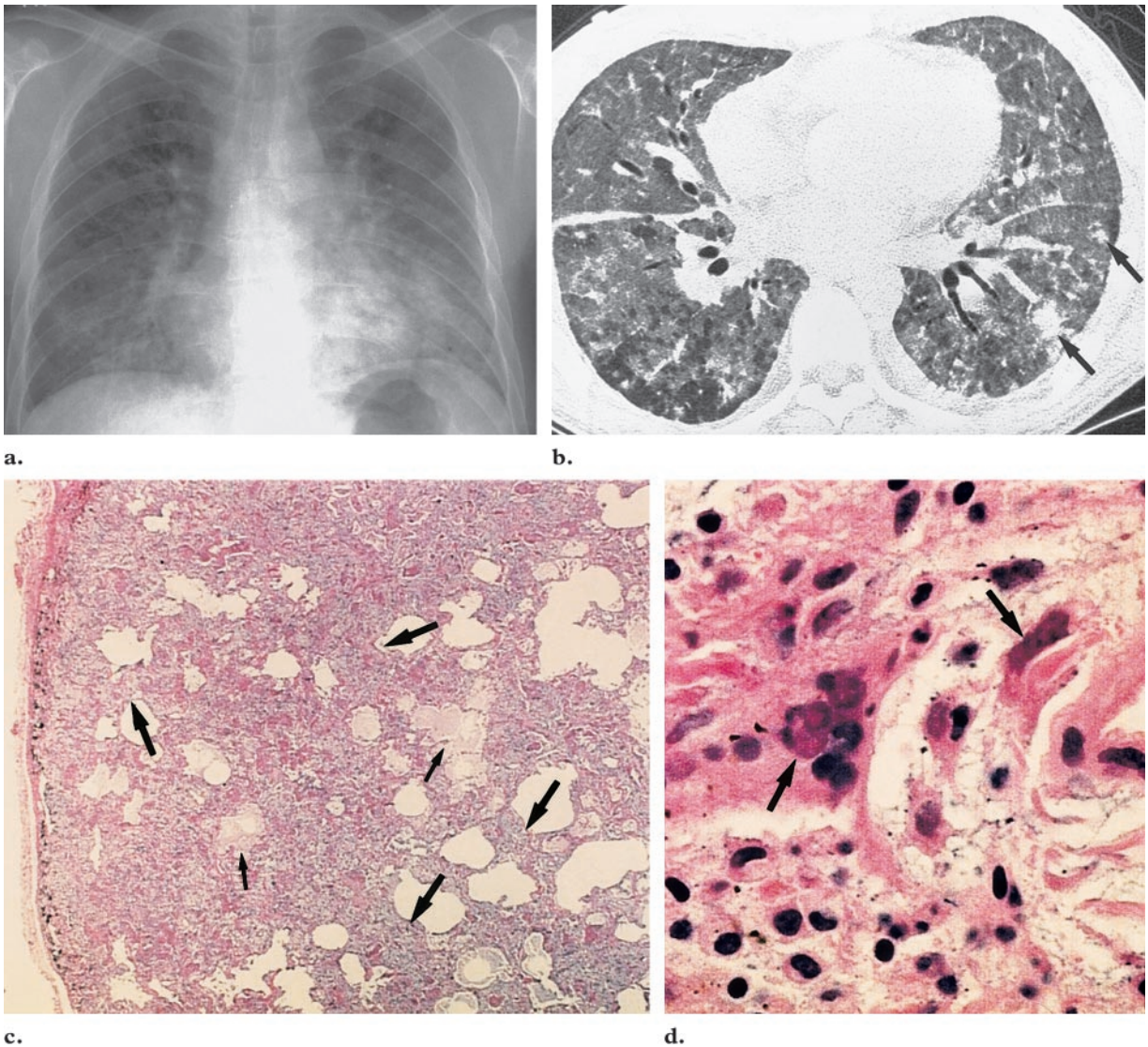
Pathologically, the lungs in adenovirus pneumonia are large and heavy and show patchy areas of hemorrhagic consolidation, mixed with areas of overinflation or atelectasis. In the airways, mucopurulent or hemorrhagic materials are seen along with wall thickening caused by congestion. Microscopically, the lung parenchyma contains necrotic changes with diffuse alveolar damage. In mild forms, findings of interstitial inflammatory cell infiltration may predominate (28,29). Nuclear inclusion bodies, most prominent in alveolar lining cells but also found in the airway epithelium, may be identified in infected cells. Their presence in the expectorated sputum may provide evidence supportive of the diagnosis.

The most frequent radiographic findings of adenovirus pneumonia, especially in children, consist of diffuse bilateral bronchopneumonia and severe overinflation. Lobar atelectasis is also a frequent finding (Fig 6). Right upper lobe atelectasis occurs most commonly in infants, while left lower lobe atelectasis is more common in older children. In patients with uncomplicated pneumonia, the disease resolves within 2 weeks

radiographically. Chronic disease is more common in children under 2 years of age at the time of the acute illness than in older patients (30). In a recent study by Han et al (4), adenovirus pneumonia in children mimicked bacterial pneumonia on chest radiographs in most patients. In their study, which included 21 pediatric patients with adenovirus pneumonia, chest radiographs showed typical lower respiratory infection with hyperaeration, bronchial wall thickening, and patchy areas of atelectasis in only two patients. In the remaining 19 patients, lobar or segmental air-space consolidation was seen. The consolidation was bilateral in 12 of 19 patients (63%). Pleural effusion was present in 13 of 21 patients (62%). The authors suggested that a normal or decreased white blood cell count associated with lymphocytosis and progression of disease despite extensive antibiotic therapy may help differentiate adenoviral from bacterial pneumonia.

### Herpes Simplex Virus Type 1 Pneumonia

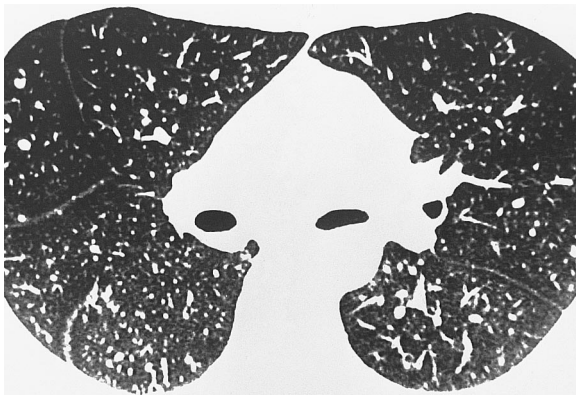
Herpes simplex virus type 1 pneumonia is an unusual infection that typically affects patients who are immunocompromised or whose airways have been traumatized from intubation, smoke inhalation, or chronic cigarette smoking (31). There are two possible routes for lower respiratory tract involvement: aspiration or extension of oropharyngeal infection into the lower respiratory system and hematogenous spread in patients with sepsis (32).



**Figure 7.** Pneumonia due to herpes simplex virus type 1 in a 36-year-old man with myelodysplastic syndrome. **(a)** Initial chest radiograph shows diffuse ground-glass opacity, poorly defined nodules, and patchy consolidation in both lungs. **(b)** Thin-section (1-mm collimation) CT scan obtained at the level of the inferior pulmonary vein shows diffuse ground-glass attenuation and some air-space nodules (arrows) in both lungs. Note the small bilateral pleural effusions. **(c)** Photomicrograph (original magnification,  $\times 12$ ; hematoxylin-eosin stain) shows diffuse alveolar wall thickening (large arrows) due to fibroblastic proliferation (diffuse alveolar damage, organizing stage). Note the intraalveolar exudate (small arrows). **(d)** Photomicrograph (original magnification,  $\times 200$ ; hematoxylin-eosin stain) shows eosinophilic inclusions (arrows) within large nuclei.

Pathologically, disease caused by herpes simplex virus type 1 manifests as focal or diffuse ulcers in the tracheobronchial epithelium with or without necrotizing bronchopneumonia (33,34). The airway lesion is characterized histologically by epithelial necrosis and ulceration. Pneumonia is usually characterized by alveolar necrosis and a proteinaceous exudate with a variable polymorphonuclear inflammatory response (35) (Fig 7).

Patchy segmental or subsegmental consolidation and ground-glass opacity are the most common findings on chest radiographs. CT demonstrates predominantly multifocal segmental or subsegmental areas of ground-glass attenuation and less dominant focal areas of consolidation. Pleural effusion is common (36) (Fig 7). Herpes simplex virus type 1 pneumonia is often a polymicrobial infection and is frequently associated with coexisting bacterial pneumonia.



**Figure 8.** Pneumonia due to varicella-zoster virus in a 30-year-old man with a fever and skin rash. Thin-section (1-mm collimation) CT scan obtained at the level of the bronchus intermedius shows multiple 1–2-mm-diameter nodules scattered throughout both lungs. (Courtesy of Dong Wook Sung, MD, Kyung Hee University Hospital, Seoul, Korea.)

### Varicella-Zoster Virus Pneumonia

Varicella-zoster virus pneumonia is the most serious complication of disseminated varicella-zoster virus infection with mortality rates of 9%–50% (37). Reported prevalences of varicella-zoster virus pneumonia have varied from less than 5% to up to 50% of all varicella infections in adults (38). Varicella-zoster virus most commonly causes self-limited benign disease (chickenpox) in children. However, in adults it tends to cause significant complications such as varicella-zoster virus pneumonia. More than 90% of cases of varicella-zoster virus pneumonia in adults occur in patients with lymphoma and immunocompromised patients (39).

Histologic features of varicella-zoster virus pneumonia are those of diffuse alveolar damage. With recovery from the initial disease, spherical nodules are seen, scattered randomly throughout the lung parenchyma. Histologically, the nodules are composed of an outer, often lamellated fibrous capsule frequently enclosing areas of hyalinized collagen or necrotic tissue. Calcification varies in intensity (40).

Chest radiographic findings of varicella-zoster virus pneumonia consist of multiple 5–10-mm ill-defined nodules that may be confluent and fleeting. Hilar lymphadenopathy and pleural effusion are unusual. The small, round nodules usually resolve within a week after the disappearance of the skin lesions but may persist for months. Usually resolving in 3–5 days in milder disease, the small nodules may persist for several weeks in widespread disease. The lesions calcify and can

persist as numerous, well-defined, randomly scattered, 2–3-mm dense calcifications (40,41).

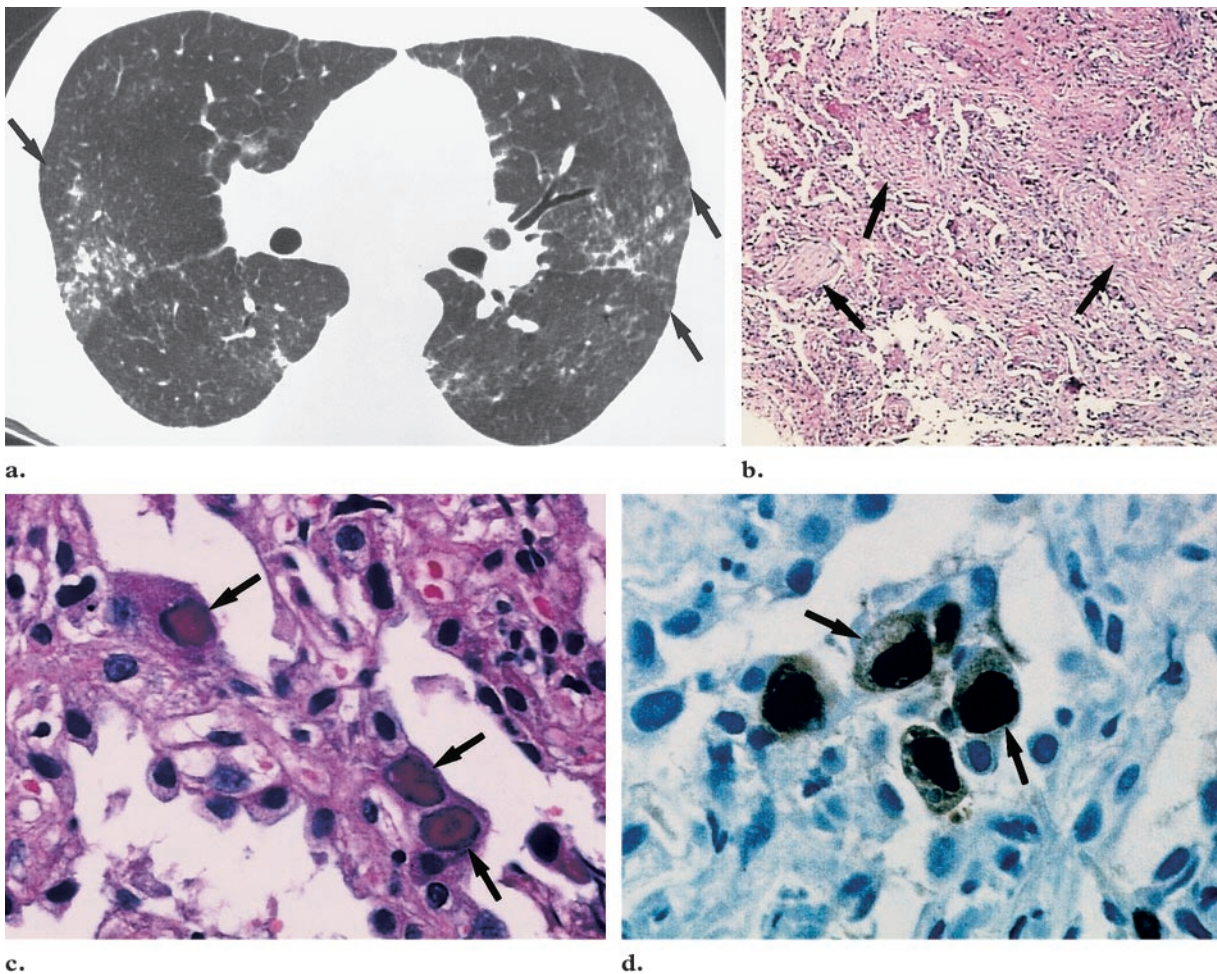
High-resolution CT usually shows 1–10-mm well-defined and ill-defined nodules diffusely throughout both lungs (Fig 8). Nodules with a surrounding halo of ground-glass opacity, patchy ground-glass opacity, and coalescence of nodules are also seen. These findings disappear concurrently with healing of skin lesions after antiviral chemotherapy (40).

### Cytomegalovirus Pneumonia

Cytomegalovirus is considered a member of the herpesviruses family and can cause severe symptomatic pulmonary disease in immunocompromised patients.

It has been suggested that cytomegalovirus pneumonitis in allogeneic transplant recipients is caused by immune mechanisms mediated by a T cell response to virally induced antigens expressed in the lungs. Severe necrotizing pneumonitis may occur despite suppression of virus replication during ganciclovir therapy. AIDS patients, with more profound immune deficiency than transplant recipients, may be unable to begin the immune response necessary to cause cytomegalovirus pneumonitis by this mechanism. In AIDS patients, the lung damage may be due directly to cytopathogenic effects of cytomegalovirus, related to the extent of active virus replication. These differences in pathogenic mechanisms of pulmonary cytomegalovirus infection result in different histopathologic features between AIDS patients and transplant recipients. In transplant recipients with cytomegalovirus pneumonia, necrotizing inflammation is predominant with relatively few cytomegalovirus-infected cells at histopathologic examination. In AIDS patients with cytomegalovirus pneumonia, a high density of cytomegalovirus inclusion bodies is present and they are responsible for severe, symptomatic pneumonitis. The cytopathogenic effect of cytomegalovirus probably causes diffuse alveolar damage more frequently in AIDS patients than in patients without AIDS.

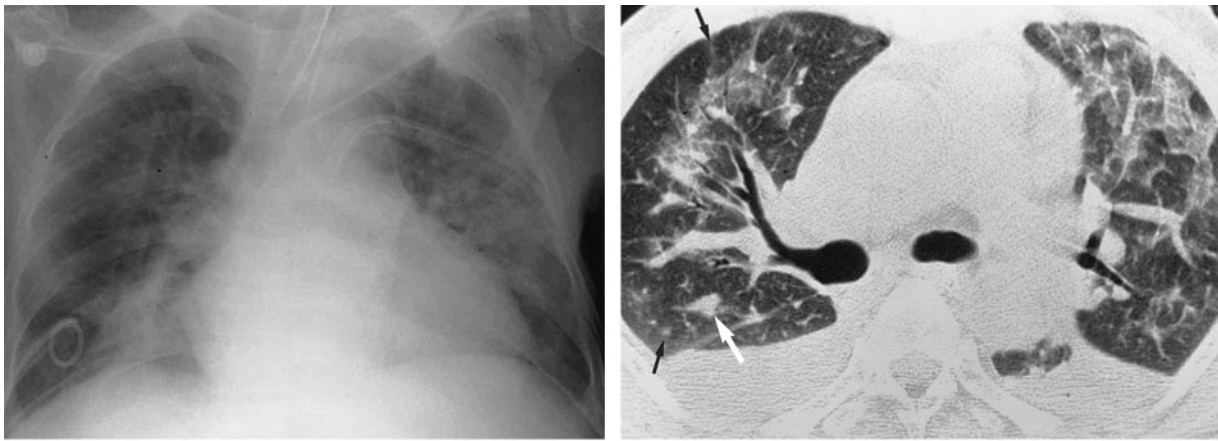
The CT findings of cytomegalovirus pneumonia are diverse and have been described without distinction between AIDS patients and patients without AIDS. Common findings are mixed alveolar-interstitial infiltrates such as ground-glass attenuation, consolidation, nodules, poorly defined small centrilobular nodules, bronchial dilatation, and thickened interlobular septa (42–45)



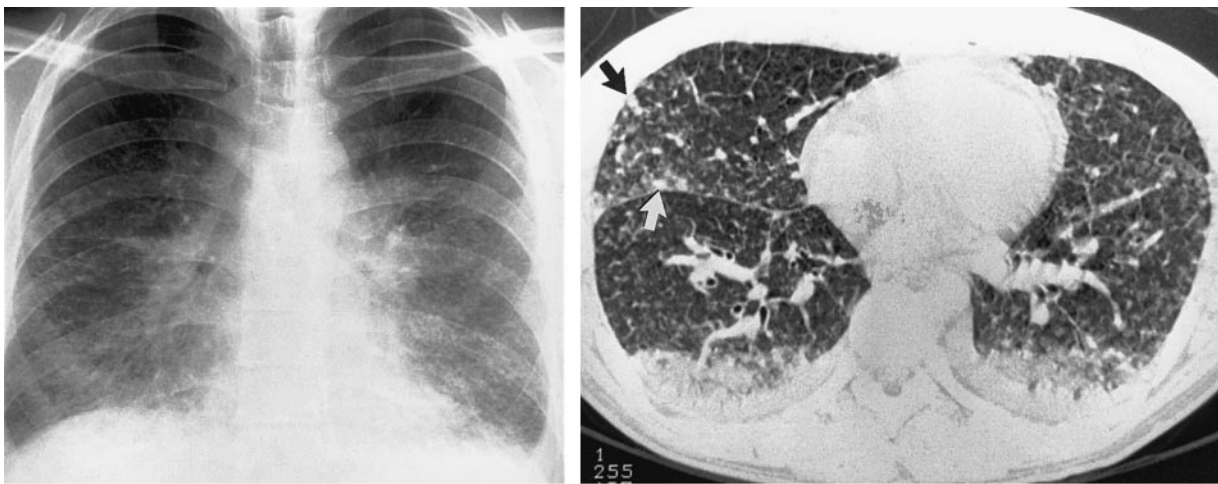
**Figure 9.** Pneumonia due to cytomegalovirus in a 28-year-old man with acute myeloid leukemia. **(a)** Thin-section (1-mm collimation) CT scan obtained at the level of the bronchus intermedius shows multifocal patchy ground-glass attenuation and poorly defined centrilobular nodules (arrows) in both lungs. **(b)** Photomicrograph (original magnification, ×40; hematoxylin-eosin stain) shows diffuse interstitial and intraalveolar fibroblastic proliferation (arrows) with some mononuclear cell infiltration (diffuse alveolar damage, organizing stage). **(c)** Photomicrograph (original magnification, ×400; hematoxylin-eosin stain) shows three large nuclei containing eosinophilic inclusion bodies (arrows) within hyperplastic pneumocytes. **(d)** Photomicrograph (original magnification, ×400; immunohistochemical marker for cytomegalovirus) shows positive intranuclear inclusion bodies (arrows).

(Figs 9, 10). Kang et al (42) reported CT findings of cytomegalovirus pneumonia in 10 transplant recipients. Abnormal findings were seen in nine patients including nodules ( $n = 6$ ), consolidation ( $n = 4$ ), ground-glass attenuation ( $n = 4$ ), and irregular linear attenuation ( $n = 1$ ). Moon et al (43) reported that poorly defined centrilobular small nodules (90%) and areas of consolidation (70%) are also common. In the study by Kim and Lee (44) of high-resolution CT findings in 11 immunocompromised patients, ground-glass attenuation ( $n = 11$ ), irregular linear attenuation ( $n = 10$ ), consolidation ( $n = 7$ ), multiple small nodules or a mass ( $n = 6$ ), and bronchial dilatation or wall thickening ( $n = 5$ ) were seen.

At pathologic examination of cytomegalovirus pneumonia, cytomegalic cells are recognized within the areas of alveolar damage (Fig 9). The alveolar-filling process is caused by hemorrhage, neutrophilic and fibrinous exudates, and hyaline membrane formation. These areas may correspond to the areas of ground-glass attenuation and/or consolidation on high-resolution CT scans (Fig 9). Interstitial infiltrates are composed mainly of lymphocytes and result in alveolar wall or interlobular septal thickening. The interstitial infiltrates may appear at CT as ground-glass attenuation. Nodules correspond histopathologically to the areas of inflammatory or hemorrhagic nodules or organizing pneumonia. Poorly defined centrilobular nodules represent the areas of intraalveolar collections of macrophages, red blood cells, and fibrin (hemorrhagic and inflammatory nodule) (42,43,45).



**Figure 10.** Pneumonia due to cytomegalovirus in a 45-year-old man who underwent liver transplantation. **(a)** Chest radiograph obtained 4 weeks after liver transplantation shows patchy air-space consolidation in both lungs. An endotracheal intubation tube, a pigtail drainage catheter in the right pleural space, a chest tube in the left pleural space, and a central venous catheter are seen. **(b)** Thin-section (1-mm collimation) CT scan obtained at the level of the right upper lobe bronchus 2 days before **a** shows multifocal patchy ground-glass attenuation in both lungs. Note the consolidation (white arrow) and the small, poorly defined nodules (black arrows). There are associated bilateral pleural effusions.



**Figure 11.** Pneumonia due to Epstein-Barr virus in a 25-year-old man with a fever, chills, and palpable lymph nodes. **(a)** Chest radiograph shows ground-glass opacity and multiple small nodules in both lungs, especially in the middle and lower lung zones, along with small bilateral pleural effusions. **(b)** Thin-section (1-mm collimation) CT scan obtained at the level of the left inferior pulmonary vein shows numerous small nodules (arrows) and diffuse ground-glass attenuation.

### Epstein-Barr Virus Pneumonia

Pulmonary involvement associated with Epstein-Barr virus infection is a rare but potential complication of infectious mononucleosis (46).

Infectious mononucleosis usually occurs in young adults aged 15–30 years. Infectious mononucleosis caused by Epstein-Barr virus usually resolves over a period of weeks or months without sequelae but may occasionally be complicated by a wide variety of neurologic, hematologic, hepatic, respiratory, and psychologic complications.

Pathologically, mononuclear inflammatory cells are apparent along bronchovascular bundles

and interlobular septa in interstitial pulmonary infiltrates. These mononuclear cells are also present, in lesser numbers, in the alveolar exudates (47).

Intrathoracic involvement by infectious mononucleosis is uncommon. The most common radiologic abnormalities are mediastinal lymphadenopathy and rarely interstitial infiltrates (Fig 11). Pulmonary consolidation in infectious mononucleosis associated with interstitial pulmonary

**Table 2**  
**Summary of CT Findings in Viral Pneumonias**

Cause of Pneumonia	Centrilobular Nodules	Ground-Glass Attenuation with Lobular Distribution	Segmental Consolidation	Thickened Interlobular Septa	Diffuse Ground-Glass Attenuation
Influenza virus	+++	+++	+	...	+
Measles virus	++	+	+	...	+
Hantavirus	...	...	++	+	+++
Adenovirus	++	+	+++	...	...
Herpes simplex virus	+	+++	+++	...	+
Varicella-zoster virus	+++	+	...	...	...
Cytomegalovirus	++	++	+	+	++
Epstein-Barr virus	+	+	+	...	+

Note.—Plus signs indicate the relative frequency of the findings from lowest (+) to highest (+++).

infiltrates is very rare (48). Splenomegaly is common and may be seen in up to 47% of chest radiographic examinations (49).

A rapidly progressive respiratory illness in infectious mononucleosis has rarely been reported (50).

### Conclusions

The radiologic findings of adult viral pneumonias are variable and overlapping (Table 2). Specific-organism diagnosis of a viral pneumonia cannot be made on the basis of imaging features alone. Clinical features such as patient age; immune status; time of year; illness in other family members; community outbreaks; onset, severity, and duration of symptoms; and the presence of a rash remain important aids in diagnosing viral causes of both atypical pneumonia and pneumonia in immunocompromised patients.

Therefore, recognition of the radiologic findings will help narrow the differential diagnosis and the combination of clinical features can significantly improve the accuracy of diagnosis in viral pneumonias.

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