caused by human respiratory syncytial virus and are often mistaken for influenza. The first human MPV infection occurs during early childhood, but reinfections are common throughout life, especially in older subjects. Diagnostic methods include PCR tests for viral RNA and direct immunofluorescence. Ribavirin is the only antiviral treatment that is currently available for human MPV infections; it is used mostly in immunocompromised patients with severe disease. Although work is ongoing, a clinically effective and safe vaccine has yet to be developed.

Severe Acute Respiratory Syndrome

Severe acute respiratory syndrome (SARS) first appeared in November 2002 in the Guangdong Province of China and subsequently spread to Hong Kong, Taiwan, Singapore, Vietnam, and Toronto, where large outbreaks also occurred. The ease of travel between continents clearly contributed to this initial rapid spread. The epidemic went no further, however, perhaps in part because of public health measures, and the last cases of SARS were laboratory-associated infections reported in April 2004. The cause of SARS was a new coronavirus. Many upper respiratory infections are caused by coronaviruses, but the SARS virus differed from other coronaviruses in that it infected the lower respiratory tract and spread throughout the body. SARS is a cardinal example of sudden emergence of a new infectious agent (Chapter 8), but since 2004 the virus has completely disappeared as mysteriously as its original debut. It is unknown if or when it will appear again.

MORPHOLOGY

All viral infections produce similar morphologic changes. Upper respiratory infections are marked by mucosal hyperemia and swelling, lymphomonocytic and plasmacytic infiltration of the submucosa, and overproduction of mucus secretions. The swollen mucosa and viscous exudate may plug the nasal channels, sinuses or the Eustachian tubes, leading to suppurative secondary bacterial infection. Virus-induced tonsillitis causing hyperplasia of the lymphoid tissue within the Waldeyer ring is frequent in children.

In viral **laryngotracheobronchitis** and **bronchiolitis** there is vocal cord swelling and abundant mucus production. Impairment of bronchociliary function invites bacterial superinfection with more marked suppuration. Plugging of small airways may give rise to focal lung atelectasis. With more severe bronchiolar involvement, widespread plugging of secondary and terminal airways by cell debris, fibrin, and inflammatory exudate may, if prolonged, lead to organization and fibrosis, resulting in obliterative bronchiolitis and permanent lung damage.

Lung involvement may be quite patchy or may involve whole lobes bilaterally or unilaterally. The affected areas are red-blue and congested. Pleuritis or pleural effusions are infrequent. The histologic pattern depends on the severity of the disease. **Predominant is an interstitial inflammatory reaction involving the walls of the alveoli.** The alveolar septa are widened and edematous and usually have a mononuclear inflammatory infiltrate of lymphocytes, macrophages, and occasionally plasma cells. In acute cases neutrophils may also be present. The alveoli may be free of exudate, but in many patients there is intra-alveolar proteinaceous material and a cellular exudate. When complicated by ARDS, pink hyaline membranes line the alveolar walls (Fig. 15-4). Eradication of the infection is followed by reconstitution of the normal lung architecture.

Superimposed bacterial infection modifies this picture by causing ulcerative bronchitis, bronchiolitis, and bacterial pneumonia. Some viruses, such as herpes simplex, varicella, and adenovirus, may be associated with necrosis of bronchial and alveolar epithelium and acute inflammation. Characteristic viral cytopathic changes are described in Chapter 8.

Clinical Course. The clinical course of viral infections is extremely varied. Many cases masquerade as severe upper respiratory tract infections or as *chest colds*. Even individuals with well-developed atypical pneumonia have few localizing symptoms. Cough may be absent, and the major manifestations may consist only of fever, headache, muscle aches, and pains in the legs. The edema and exudation are both strategically located to cause mismatching of ventilation and blood flow and thus evoke symptoms out of proportion to the scanty physical findings.

Viral infectons are usually mild and resolve spontaneously without any lasting sequelae. However, interstitial viral pneumonias may assume epidemic proportions, and in such instances even a low rate of complications can lead to significant morbidity and mortality, as is typically true of influenza epidemics.

Health Care-Associated Pneumonia

Health-care associated pneumonia was recently described as a distinct clinical entity associated with significant risk factors. These are: hospitalization of at least 2 days within the recent past; presentation from a nursing home or long-term care facility; attending a hospital or hemodialysis clinic; and recent intravenous antibiotic therapy, chemotherapy or wound care. The most common organisms isolated are methicillin-resistant *Staphylococcus aureus* and *P. aeruginosa.* These patients have a higher mortality than those with community-acquired pneumonia.

Hospital-Acquired Pneumonia

Hospital-acquired pneumonias are defined as pulmonary infections acquired in the course of a hospital stay. They are common in patients with severe underlying disease, immunosuppression, prolonged antibiotic therapy, or invasive access devices such as intravascular catheters. Patients on mechanical ventilation are at particularly high risk. Superimposed on an underlying disease (that caused hospitalization), hospital-acquired infections are serious and often life-threatening complications. Gram-positive cocci (mainly *S. aureus* and *S. pneumonia)* and gram-negative rods (Enterobacteriaceae and *Pseudomonas* species) are the most common isolates. There are similar organisms isolated in ventilator associated pneumonia but gram-negative bacilli are more common.

KEY CONCEPTS

Acute Pneumonia

- *S. pneumoniae* (the pneumococcus) is the most common cause of community-acquired acute pneumonia; the distribution of inflammation is usually lobar.

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