Viral Pulmonary Disorders in Animals: Neoplastic and Nonneoplastic

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24.1 Brief Introduction

Respiratory infections in animal species are as ubiquitous as they are in humans. Species that may be affected include mammals, birds, and reptiles. In these animal species some viruses primarily infect the respiratory tract, while other viruses infect non-respiratory organs. Viruses are generally classified according to the type of their nucleic acid, their protein structure, and whether or not they have a lipid-containing envelope surrounding the viral particle. In general, most viruses gain entry into the lungs via the conducting airways. In nonprimate mammalians these infections are most prominent in the cranioventral lung lobes because of their horizontal position. Table 24.1 lists some of the major viruses that cause pneumonia and other lung diseases in animals.

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24.2 Poxviridae

Poxviruses are DNA viruses that belong to the *Poxviridae* family. These viruses are capable of infecting a variety of species including human and some mammalians, including monkeys, rabbits, and squirrels, as well as reptiles and birds. They are regarded as oncogenic since they lead to proliferation of epidermal and occasionally mesenchymal tissues. Poxviridae are highly epitheliotropic and cause cutaneous and/or systemic disease. Infections in the lung of squirrels (Bangari et al. 2009) may manifest as tumor-like nodules (Fig. 24.1). Histologically, a papillary growth pattern of cuboidal cells that might be type II pneumocytes may be seen (Fig. 24.2). These cells often contain intracytoplasmic eosinophilic inclusion bodies (Fig. 24.3).

24.3 Herpesviridae

Herpesviridae is the name of a family of enveloped, double-stranded DNA viruses with relatively large complex genomes. They replicate in the nucleus of a wide range of vertebrate hosts, including horses, cattle, mice, pigs, chickens, turtles, lizards, and fish, and even some invertebrates, such as oysters. In many species there are strains of the virus that specifically target the respiratory system including dogs, cats, cattle, horses, and chickens. In these species, the virus can target the upper but also the lower respiratory tract, predisposing the

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Table 24.1 Viral infections in the lung in animal species

Virus family	Viral agent	Affected species
DNA viruses		
Poxviridae	Pox	Mice, monkeys, rats, sheep
Herpesviridae	Herpes	Dogs, cows, cats, horses, elephants
	Cytomegalovirus	Cats, cattle, ground squirrels, guinea pigs, horses, mice, monkeys, sheep, swine
Adenoviridae	Adenovirus	Type 2 dogs, type 3 and 5 cows, type 5 and 6 horses, monkeys, pigs, rabbits
Caliciviridae	Calicivirus	Cats
RNA viruses		
Orthomyxoviridae	Influenza	Avian, horses, seals, pigs
Paramyxoviridae	Parainfluenza	Guinea pigs, hamsters, type 2 dogs, type 3 cows, goats, horses, sheep, type 2–9 avian
	Parainfluenza 1	Mice, rats, hamsters (Sendai virus)
	Measles	Monkeys
	Respiratory syncytial virus	Cows, sheep
	Distemper	Dogs, coyotes, dolphins, minks, raccoons, seals, wolves
	Rinderpest	Cow, sheep
	Nipah virus	Pigs
	Hendra virus	Horses
Coronaviridae	Feline coronavirus	Cats
Retroviridae	Lentivirus	Sheep
	Type D oncovirus	Sheep

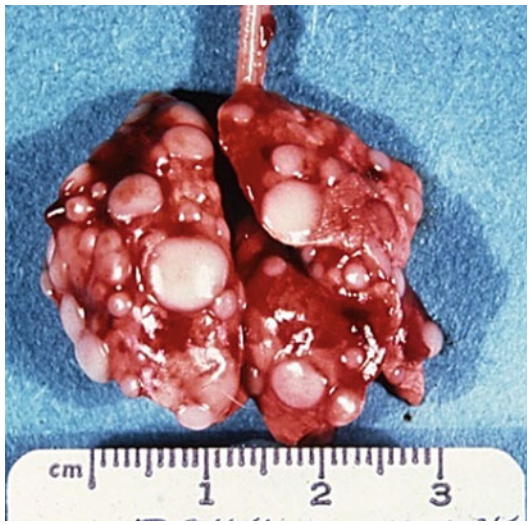


Fig. 24.1 Squirrel lung. Note multiple, firm nodules indicative of poxvirus infection

host to secondary bacterial infection through impairment of lung defenses.

Equine multinodular pulmonary fibrosis is caused by the equine herpesvirus 5 (EHV-5), a γ-herpes virus. Infected lungs manifest two distinct gross patterns of disease. A pattern of numerous coalescing nodules that are pale white and moderately firm (Fig. 24.4) is the most common. In some cases, the growth pattern is that of multiple discrete nodules (Fig. 24.5). Histologically, the lungs show severe interstitial fibrosis, accompanied by a mixed inflammatory infiltrate. The majority of the inflammatory cells are lymphocytes, as well as macrophages, neutrophils, and occasionally eosinophils. The alveolar spaces contain moderate numbers of neutrophils and macrophages. Macrophages with eosinophilic cytoplasm and large eosinophilic intranuclear inclusion bodies (Williams et al. 2007) can be seen. Equine herpesvirus 1 (EHV-1) may cause vasculitis leading to abortion, stillbirth, encephalomyelopathy, and respiratory disease. Fatal nonneurological EHV-1 infection in young adult horses does occur but is rare. Gross findings in the lung of a nonneurological EHV-1 infection may include severe edema (Fig. 24.6) and hydrothorax. Histological examination usually reveals pulmonary edema and perivascular hemorrhage while the alveolar spaces

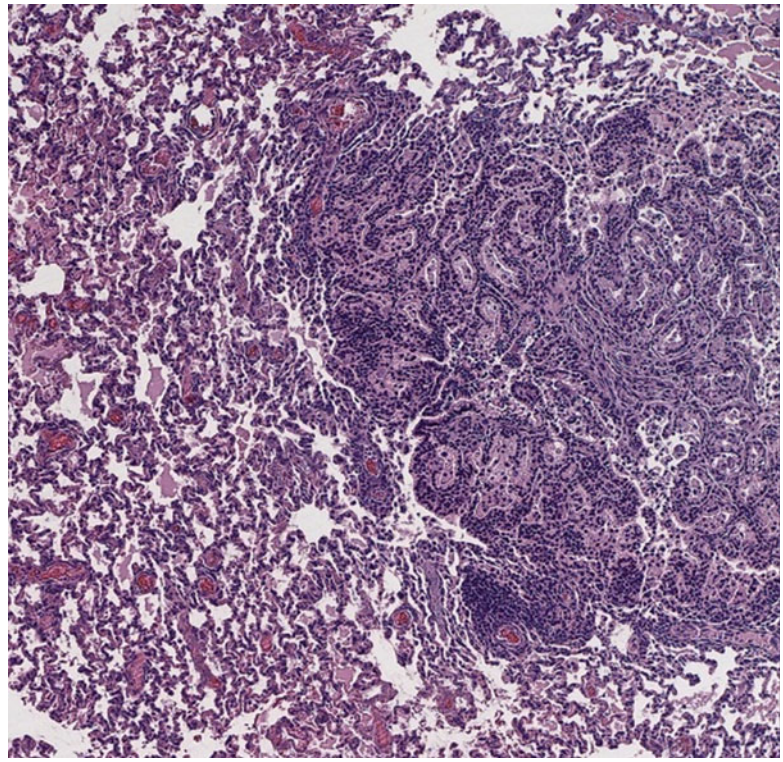


Fig. 24.2 Poxvirus. Corresponding photomicrograph reveals a papillary tumor that consists of proliferating cuboidal cells, i.e., type II pneumocytes

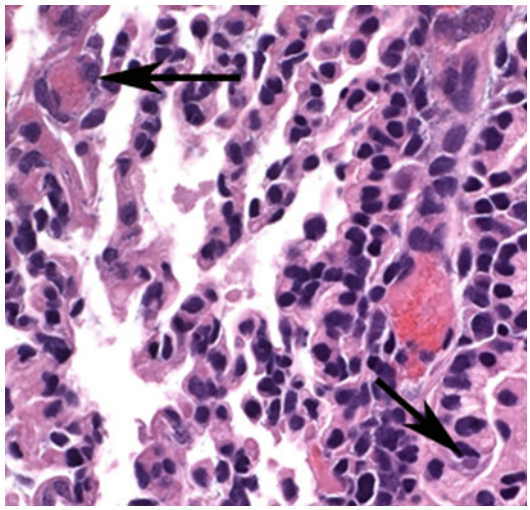


Fig. 24.3 Poxvirus. Higher magnification demonstrates intracytoplasmic inclusion bodies (*arrow*)

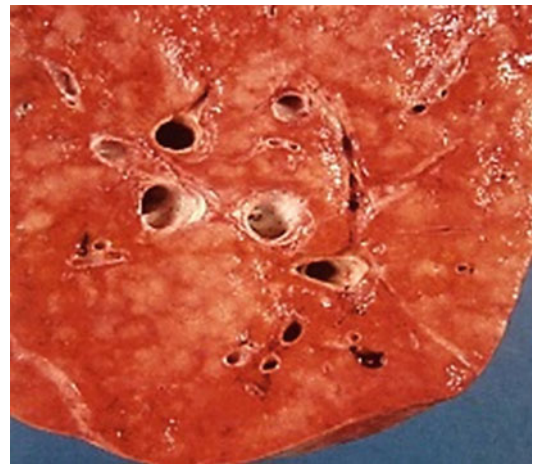


Fig. 24.4 Photomicrograph of lung from a 12 years horse infected with EHV-5 showing the most common pattern of lesion, i.e., multiple diffuse coalescing fibrotic nodules

and the conducting airways may be filled with proteinaceous fluid, fibrin, desquamated epithelial cells, and macrophages (Fig. 24.7). Prominent type II pneumocytes and perivascular infiltration by mononuclear cells can also be seen (Fig. 24.8). Intranuclear eosinophilic Cowdry type A inclusion bodies are rarely noted in endothelial cells. Immunohistochemistry is useful demonstrating the expression of EHV-1 antigen within endothelial cells (Fig. 24.9) (Del Piero et al. 2000).

24.4 Pulmonary Cytomegalovirus

Cytomegaloviruses (CMV) are host-specific viruses. They are pathogenic to various species including cats, cattle, ground squirrels, guinea pigs, horses, mice, monkeys, sheep, and swine. They affect the lung as well as multiple other organs. Rarely, the lung may be the only affected organ (Hoover and Thacker 1979). CMV infections are most often latent, but under appropriate circumstances, they may result in severe and often fatal infections. In monkeys several patterns of lesions with focal, diffuse, and a mixed pattern of disease have been reported. The lungs with the diffuse pattern of disease seen in monkeys (Baskin 1987) and sheep (Hoover and Thacker 1979) are heavy, wet, and consolidated. Microscopically, focal lesions show interstitial thickening and prominent hypertrophic pneumocytes. The alveolar spaces contain proteinaceous exudate with macrophages, neutrophils, and multinucleated giant cells with intranuclear basophilic inclusion bodies (Figs. 24.10 and 24.11) and occasionally necrotic foci. Microscopic changes in the diffuse forms are similar to those seen in the focal pattern but are generally more severe with chronic organizational changes (Baskin 1987). Electron micrographs illustrate virus present within the cell nucleus (Fig. 24.12) and viral budding from membranes (Fig. 24.13).

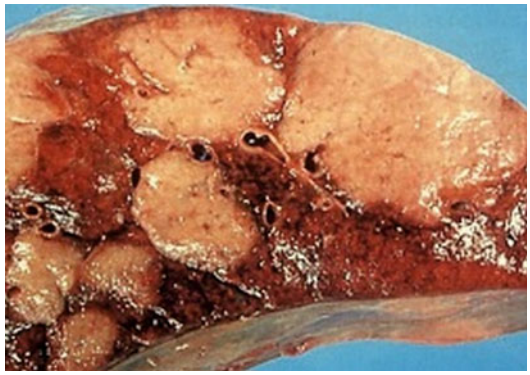


Fig. 24.5 Cross section of lung from a 4-year-old horse infected with EHV-5 revealing large discrete fibrotic nodules

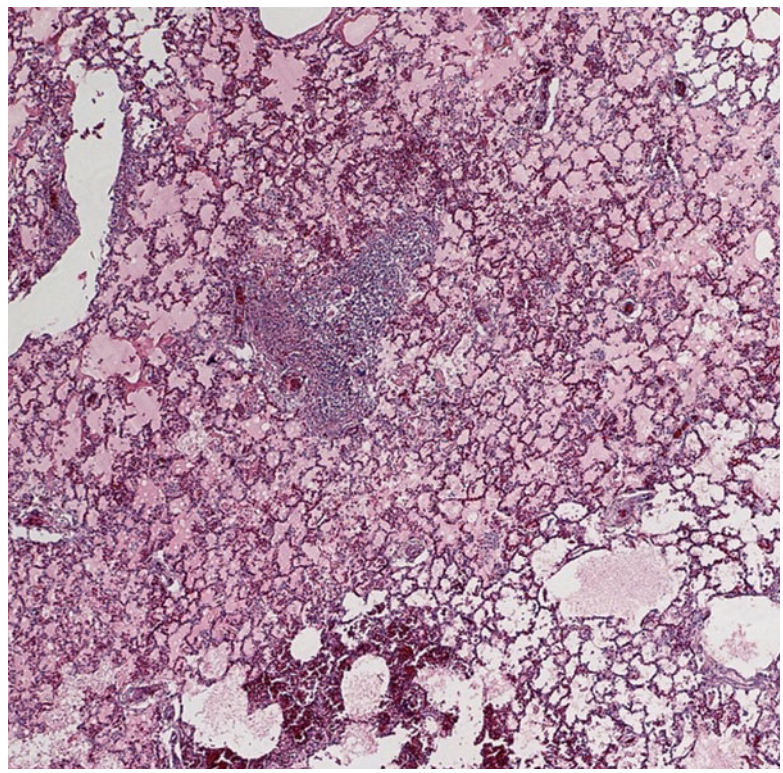


Fig. 24.6 Lung from a 1-year-old horse infected with EHV-1 showing severe pulmonary edema

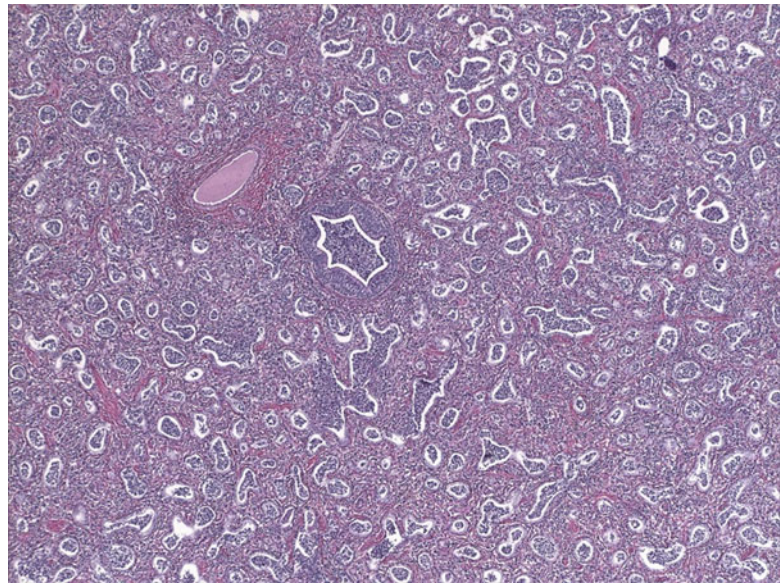


Fig. 24.7 Horse lung infected with EHV-1 is mostly edematous and consolidated. Bronchi contain an inflammatory exudate

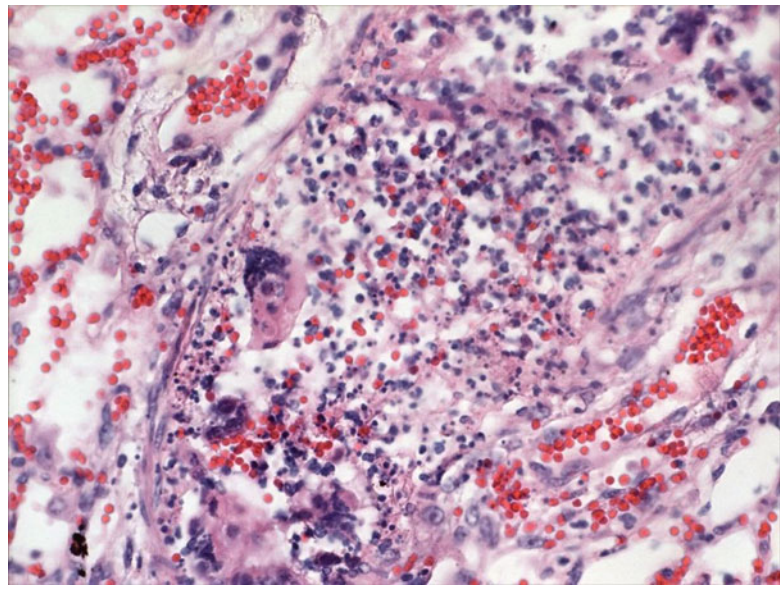


Fig. 24.8 Horse lung. Photomicrograph illustrating vascular necrosis, edema, hemorrhage, and loss of endothelial cells typical of equine herpesvirus infection

24.5 Avian Influenza Virus

Influenza is a term used to refer to an infection and/or disease syndrome caused by any type of A influenza virus. The influenza virus is capable of infecting birds as well as some mammalian species (Easterday et al. 1997). A variety of tissues besides respiratory tissue can be affected and the clinical manifestations are protean (Acland et al. 1984; Chaves et al. 2011; Woo et al. 2011). Compared to other tissues, the severity of the lesions reported in the respiratory system is relatively milder (Chaves et al. 2011). While severe inflammation occurs in non-respiratory tissues, only hemorrhage can occur in the lung. Hemorrhages in the parabronchi (Fig. 24.14), atrial spaces, and atrial walls can also be seen (Fig. 24.15).

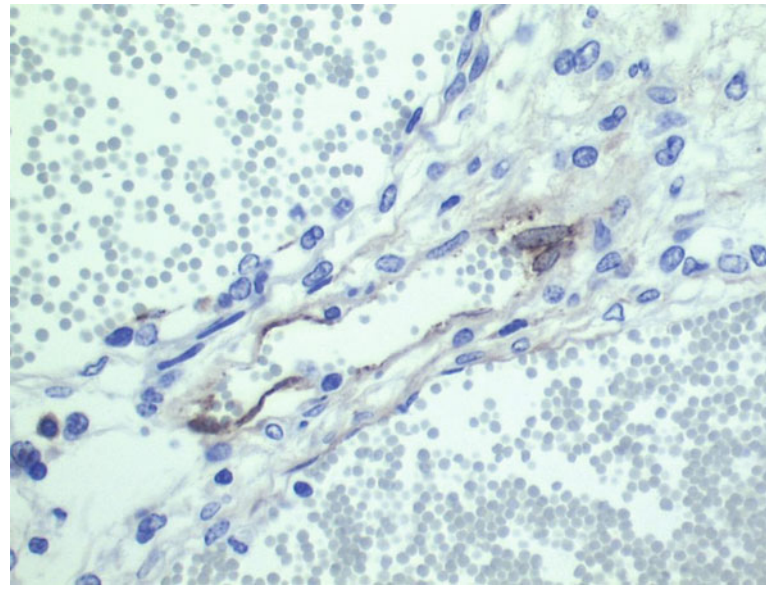


Fig. 24.9 Immunohistochemistry stain showing expression of EHV-1 antigen within endothelial cells

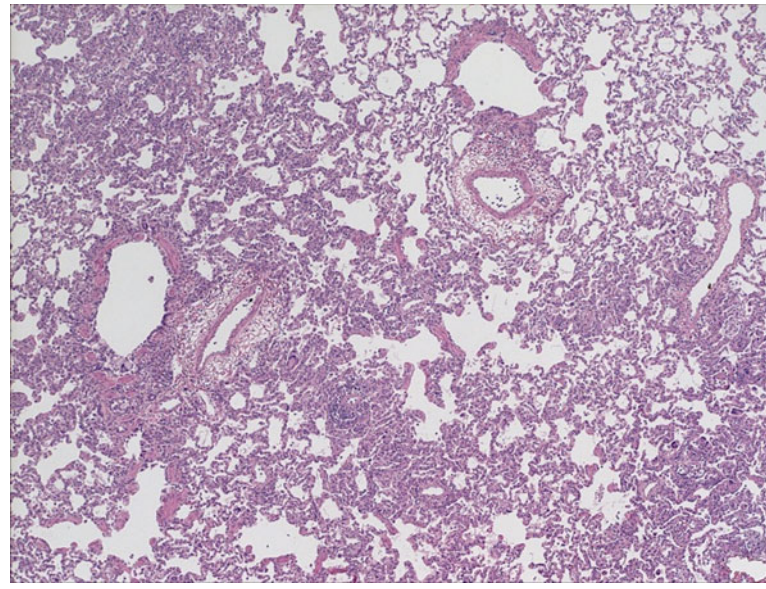


Fig. 24.10 CMV. Low-magnification photomicrograph of monkey lung revealing diffuse thickening of alveolar walls

24.6 Sendai Virus

Sendai virus (murine parainfluenza virus) is closely related antigenically to parainfluenza 1 in humans. Infection with this virus has been described in hamsters, mice, and rats. The virus is a non-cytolytic virus that selectively infects the respiratory epithelium of the nose, trachea, bronchi, bronchioles, as well as type II pneumocytes. Immunocompetent mice are susceptible. In the

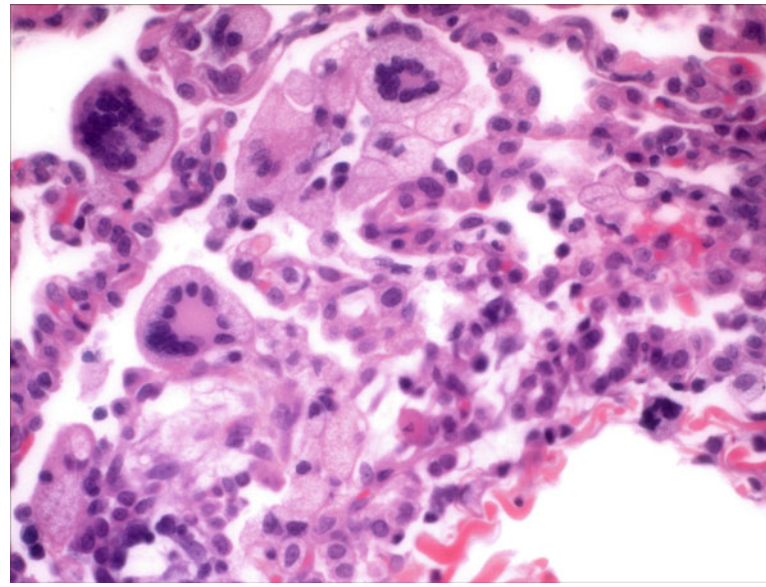


Fig. 24.11 CMV. Higher magnification demonstrating prominent pneumocytes and presence of giant cells

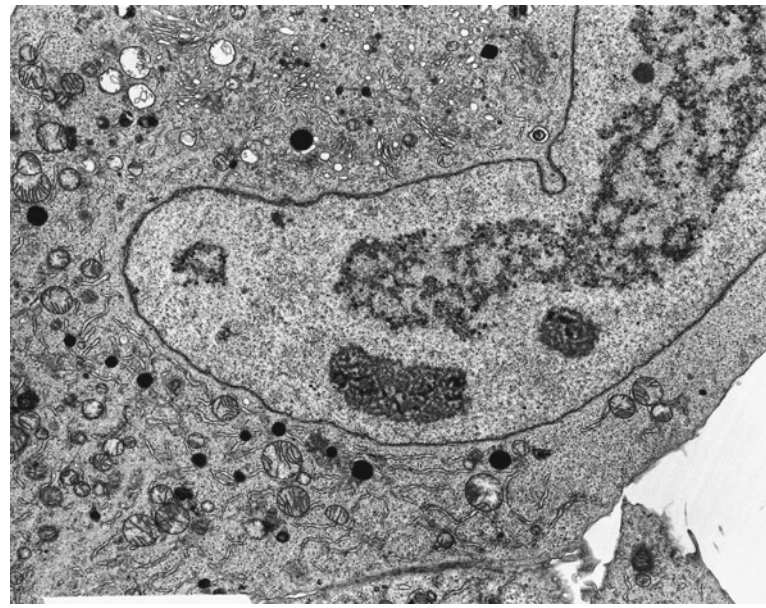


Fig. 24.12 CMV. Electron micrograph. Lung; cytomegalovirus-infected rhesus monkey. Low magnification illustrates the presence of viral particles within the nucleus

initial stage of infection, the virus is only mildly cytopathic. During the immune phase of the disease when cytotoxic T cells destroy the virus-infected cells, there is necrosis of the respiratory epithelium. This is followed by proliferating hyperplastic epithelium and increased perivascular, peribronchial cuffing, and interstitial infiltration by lymphocytes (Fig. 24.16) (Percy and Barthold 2007).

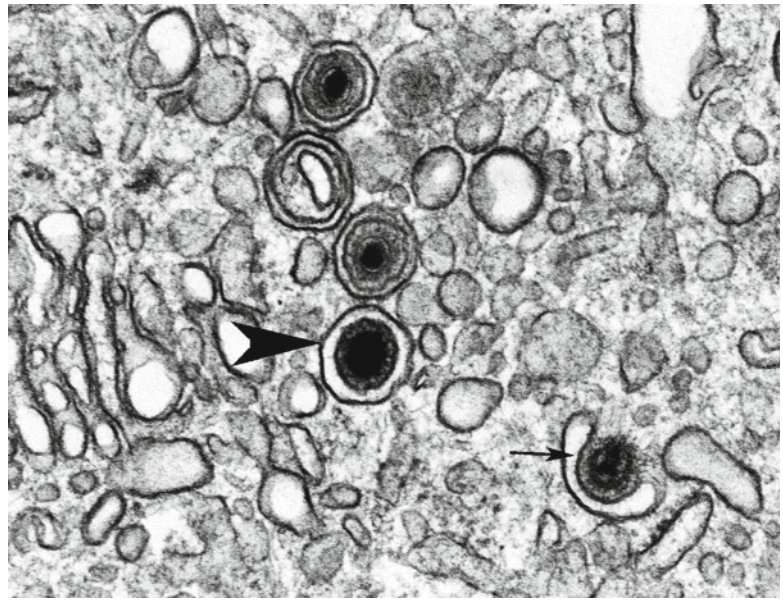


Fig. 24.13 CMV. Electron micrograph. Lung; cytomegalovirus-infected rhesus monkey. High magnification. Note viral budding (*arrow*) and mature virions (*arrowhead*) in cytoplasmic vacuoles

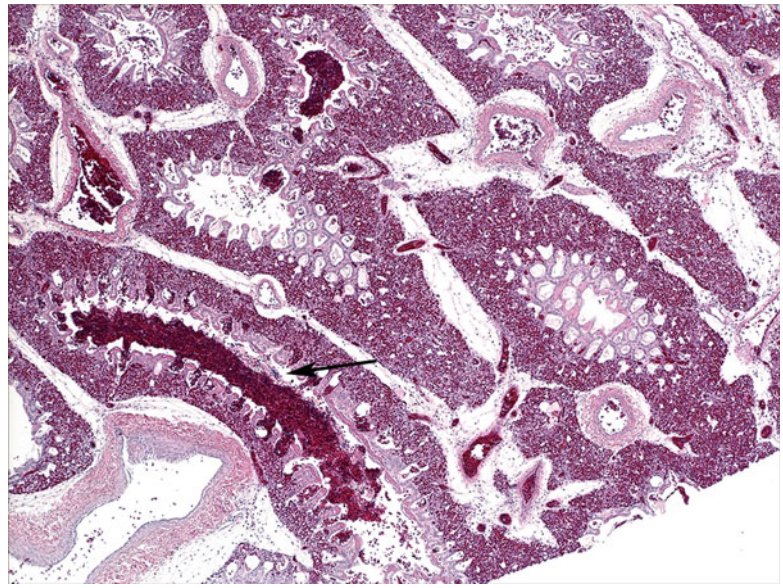


Fig. 24.14 Low magnification of chicken lung infected with avian influenza. Note marked hemorrhage in the parabronchi (*arrow*)

24.7 Measles Virus

Measles virus infects several species of monkeys including rhesus, baboons, and marmosets. In some species, such as marmosets and colobus monkeys, measles infection is more severe resulting in primary giant cell pneumonia followed by secondary bacterial bronchopneumonia (Jones et al. 1997). Histological findings include necrotizing bronchiolitis, diffuse

alveolar injury, and prominent perivascular and peribronchial lymphoid tissue (Fig. 24.17). The presence of multinucleated giant cells containing intranuclear and intracytoplasmic eosinophilic inclusion bodies is a characteristic finding (Fig. 24.18)

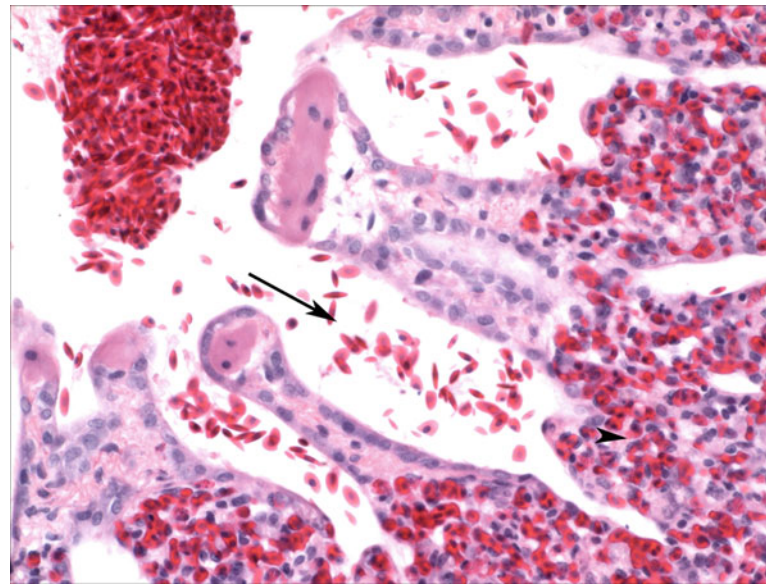


Fig. 24.15 Avian influenza. Higher magnification demonstrating hemorrhage in the parabronchus, atrial space (*arrow*), and the atrial wall (*arrowhead*)

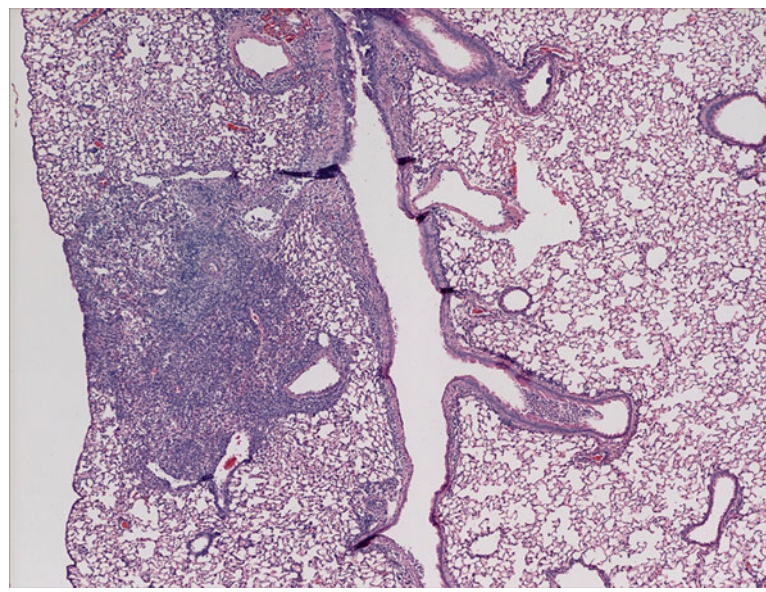


Fig. 24.16 (**a**) Sendai parainfluenza. Low magnification of mouse lung infected with Sendai virus. Hypercellular focus. (**b**) Higher magnification demonstrates alveolar walls lined by cuboidal cells and alveolar spaces containing necrotic material

Fig. 24.16 (continued)

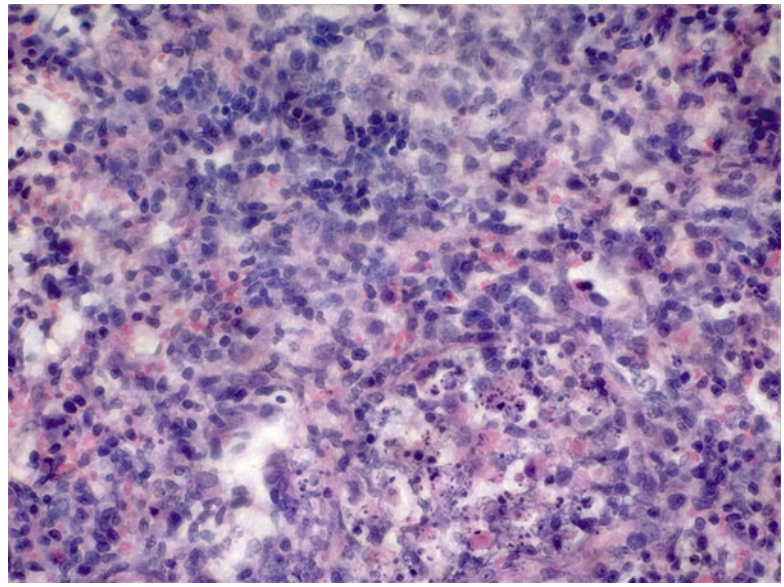
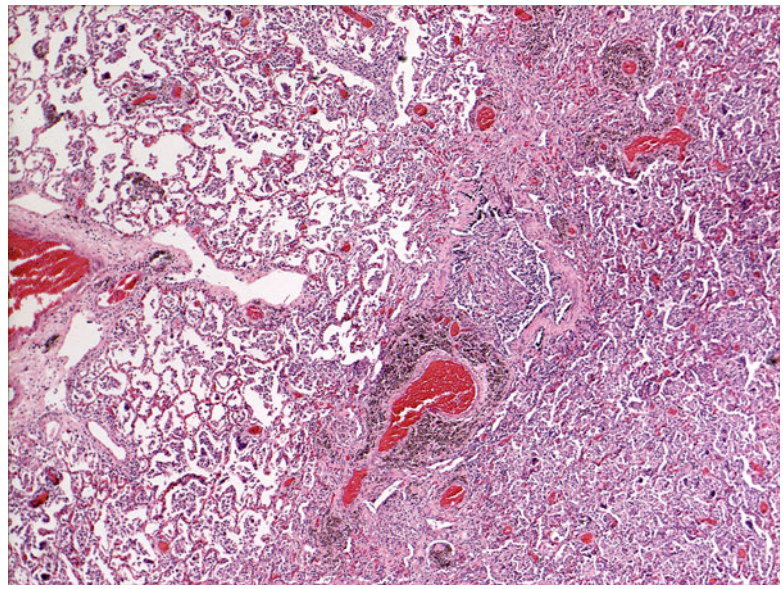


Fig. 24.17 Measles. Low-magnification photomicrograph of a rhesus monkey lung infected with measles virus. Note prominent perivascular lymphoid cuffing, some collapsed alveoli, and some alveoli filled with desquamated pneumocytes



24.8 Distemper Virus

Distemper is an RNA virus that belongs to the *Morbillivirus* genus group within the family of *Paramyxoviridae*. Distemper affects multiple species including dogs, wolves, coyotes, ferrets, fox, minks, raccoons, and various aquatic mammals such as dolphins and seals (Kennedy 1998). The virus targets and damages epithelial, mesenchymal, neuroendocrine, and hematopoietic cells in various organs. Secondary bacterial and mycoplasma infections are common in terrestrial mammals.

Pneumonia is the predominant pathologic process in infected dolphins. In lungs of aquatic

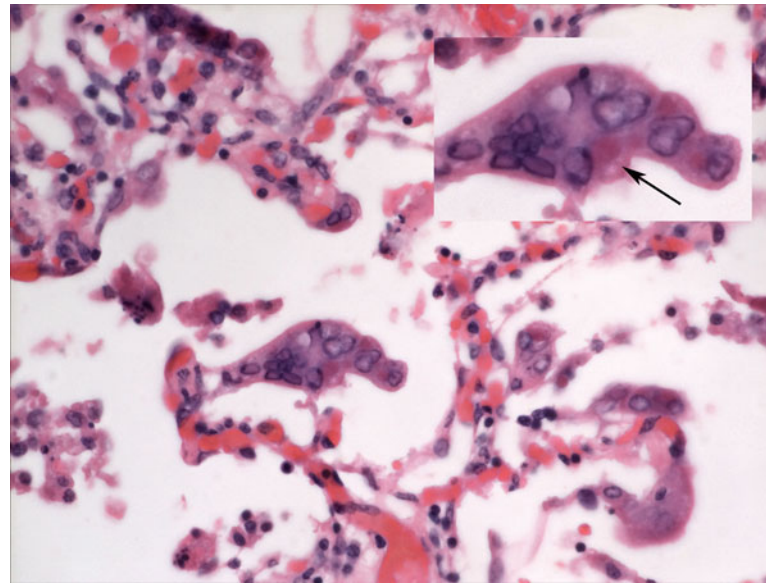


Fig. 24.18 Measles. Closer view reveals the presence of multinucleated giants cells and desquamated pneumocytes. *Inset*. Higher magnification of multinucleated giant cell with eosinophilic cytoplasmic inclusion bodies (*arrow*)

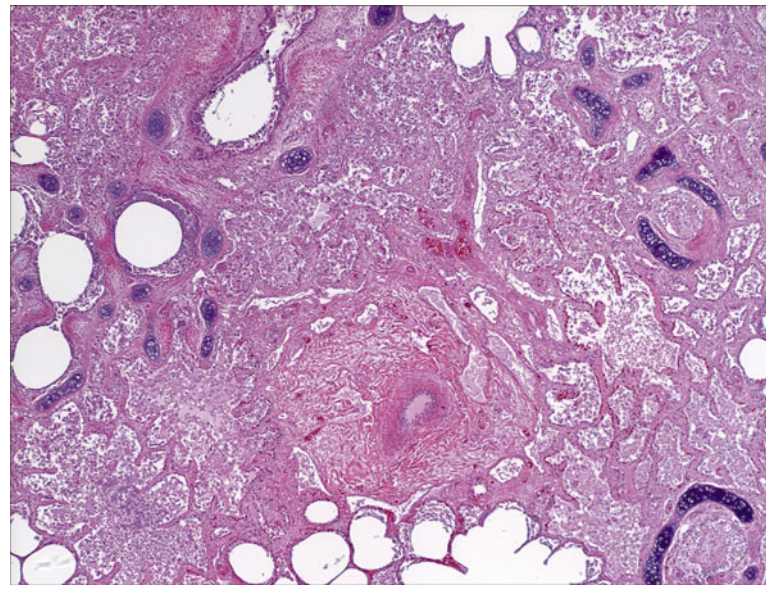


Fig. 24.19 Distemper. Low-magnification photomicrograph of dolphin lung illustrating cartilaginous tissue adjacent to bronchioles

mammals, cartilage plates are present adjacent to bronchioles (Fig. 24.19). The pneumonia is a bronchointerstitial pneumonia with presence of serofibrinous exudate and a mixed population of inflammatory cells in the conducting airways and the alveolar spaces. There is also diffuse alveolar injury with formation of hyaline membranes, hemorrhages, and proliferation of type II pneumocytes (Fig. 24.20). Occasionally syncytial cells are noted in the alveoli and conducting airway cells. Eosinophilic cytoplasmic and nuclear inclusions are observed in type II pneumocytes, in bronchiolar and bronchial cells. Immunohistochemical studies have demonstrated distemper antigens in cells of conducting airways, pneumocytes and mononuclear cells.

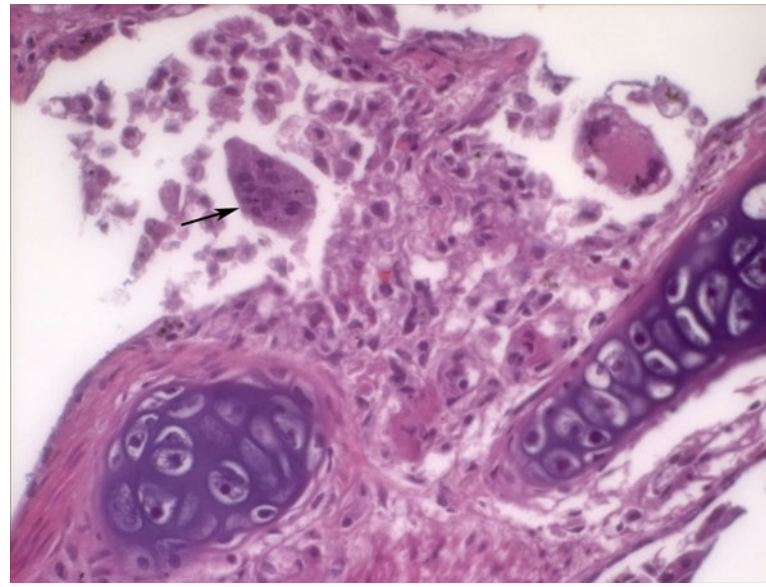


Fig. 24.20 Distemper. Dolphin lung. Closer view revealing the presence of a mixed population of mononuclear cells within the bronchiolar spaces and including multinucleated giant cells (*arrow*)

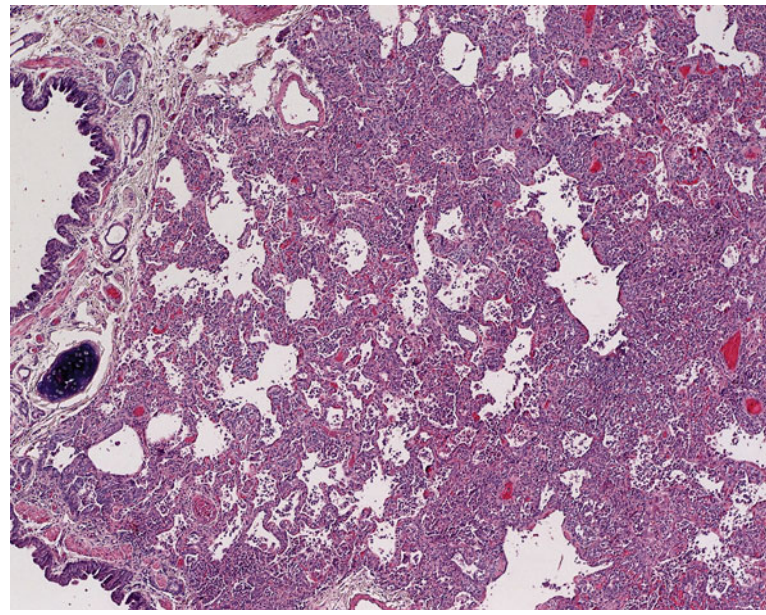


Fig. 24.21 Distemper. Low-magnification photomicrograph of interstitial pneumonia of raccoon's lung infected with distemper

Interstitial fibrosis is apt to occur in chronic stages of the disease.

A common but serious disease in dogs, distemper is caused by a morbilliform distemper virus. The distemper canine virus enters the lung through the upper airways. Affected lungs are heavy and edematous. Microscopically, necrotizing bronchiolitis, desquamation of respiratory airway cells, and hyperplastic type II pneumocytes can be seen. Histologically, canine distemper is further characterized by interstitial infiltration by mononuclear cells (Fig. 24.21). Large multinucleated giant cells are often noted (Fig. 24.22). Eosinophilic cytoplasmic and nuclear inclusion bodies are seen in type II pneumocytes and alveolar macrophages.

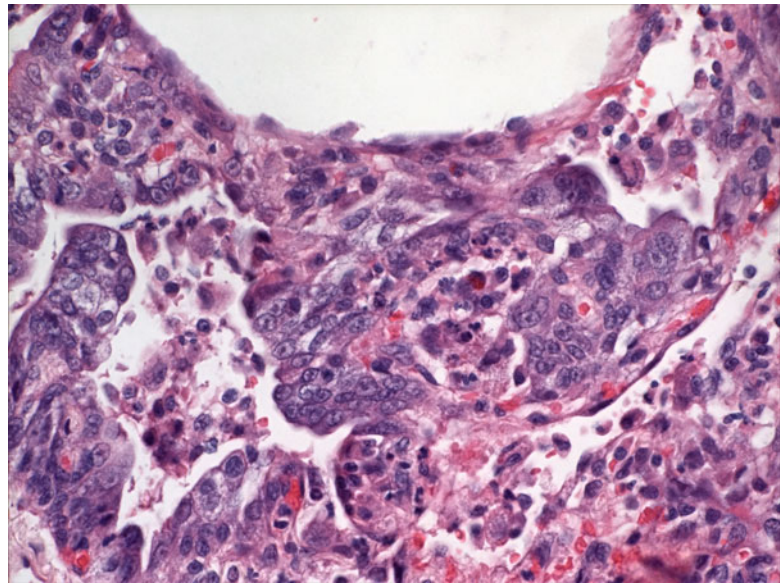


Fig. 24.22 Distemper. Raccoon lung. Higher magnification demonstrating the presence of multinucleated giant cells

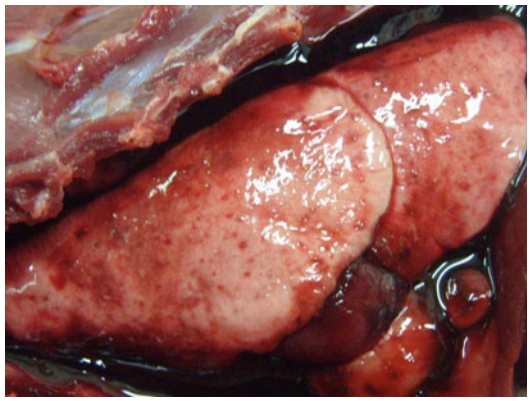


Fig. 24.23 Nipah virus. Photograph of lung from infected pig illustrating very prominent lesions on the ventral portion of the lung lobes

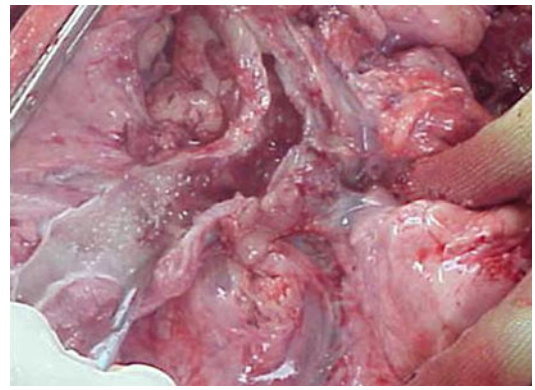


Fig. 24.24 Nipah virus. Photograph of open trachea and lung of an infected pig revealing the presence of prominent inflammatory deposits on the tracheal surface

24.9 Nipah Virus

The Nipah virus, a virus of the *Paramyxoviridae* family, is carried primarily by fruit bats and affects both humans and swine. In pigs, gross examination of infected lungs shows prominent consolidation of the ventral portion of the lung lobes (Fig. 24.23) and also lining of the trachea. The tracheal surface is lined by pseudomembranous membranes (Fig. 24.24). There is also evidence of bronchial and bronchiolar infiltration by lymphocytes, some neutrophils, macrophages, and multinucleated syncytial cells (Fig. 24.25). Intracytoplasmic eosinophilic inclusion bodies are seen in syncytial cells (Hooper et al. 2002). The formation of syncytia within endothelial and epithelial cells can be seen both in cases of Nipah and Hendra virus infections and is related to the fusogenic properties of viral glycoproteins G and F (Weingartl et al. 2009). Immunohistochemistry is helpful to demonstrate the presence of the virus in respiratory epithelium (Fig. 24.26), vascular endothelium, and lymphocytes (Fig. 24.27) (Middleton et al. 2002).

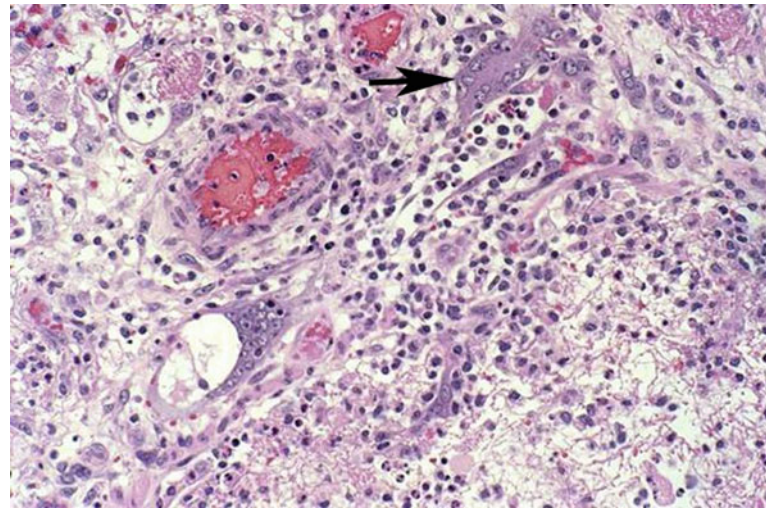


Fig. 24.25 Nipah virus. Photomicrograph of pig lung showing heavy infiltration by a mixed population of inflammatory cells and some syncytial cells (*arrow*)

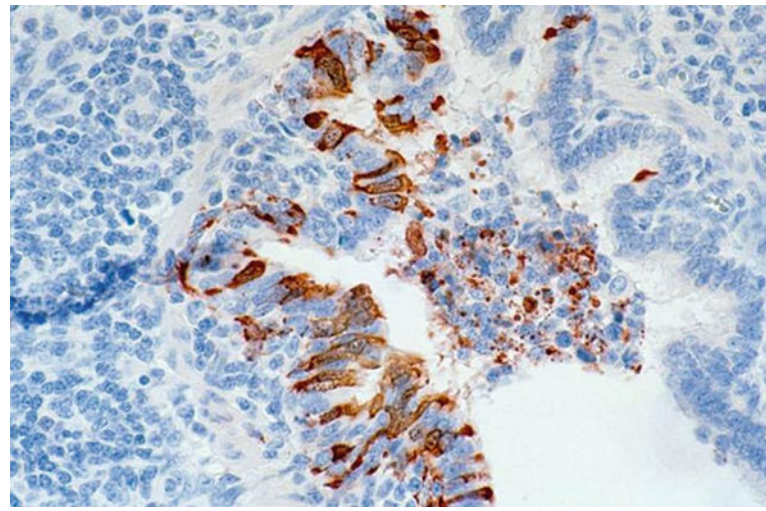


Fig. 24.26 Nipah virus. Immunohistochemical stain of swine lung with rabbit polyclonal anti-Nipah antibodies demonstrating the presence of Nipah virus in respiratory epithelium

24.10 Hendra Virus

The Hendra virus causes a severe respiratory disease in both horses and humans (Murray et al. 1995; Paterson et al. 1998). The virus is carried by species of fruit bats (Young et al. 1996). In horses, gross findings of the lungs include prominent pulmonary edema and dilation of subpleural lymphatic network (Hooper et al. 1997). Histological findings are varied, including mild to moderate interstitial edema, mild infiltration by mononuclear cells (Fig. 24.28), presence of alveolar macrophages, hemorrhagic foci, and capillary thrombosis as well as the presence of syncytial cells and eosinophilic inclusion bodies. Immunohistochemistry studies have demonstrated the presence of Hendra virus antigen in endothelial cells (Fig. 24.29).

24.11 Feline Infectious Peritonitis

Feline infectious peritonitis (FIP) is caused by a coronavirus that typically leads to vasculitis. In turn, the vasculitis results in systemic

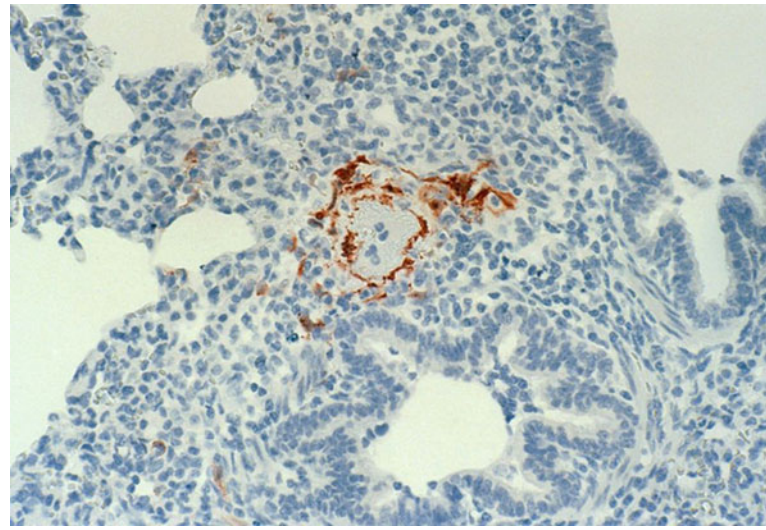


Fig. 24.27 Nipah virus. Immunohistochemical stain of swine lung with rabbit polyclonal anti-Nipah antibodies highlighting the presence of Nipah virus in vascular endothelium and some lymphocytes

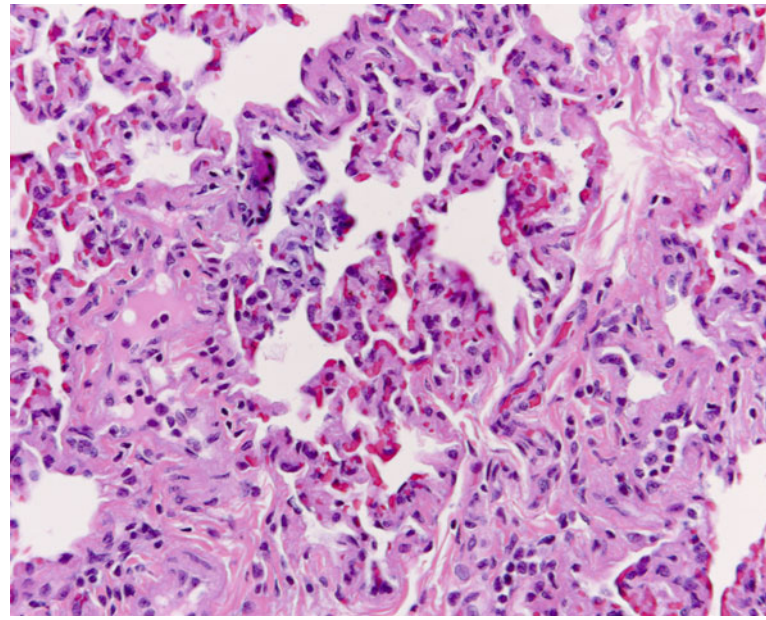


Fig. 24.28 Hendra virus. High magnification of horse lung infected with Hendra virus demonstrating mild to moderate interstitial edema

inflammatory disease. The virus may also cause chronic immunologic mediated disease. It has two forms, effusive and noneffusive forms. In cats, about 25 % of the infected population harbor a pleural effusion. The effusions generally contain a large amount of yellow, viscous fluid and fibrin strands. There are gray-white granular membranous deposits of exudate on the pleural surface and severe pulmonary edema. Microscopic findings include generalized vasculitis, hemorrhagic perivasculitis, and edema (Fig. 24.30) plus multiple pyogranulomas made up of neutrophils, lymphocytes, and macrophages (Fig. 24.31) (Dungworth 2007).

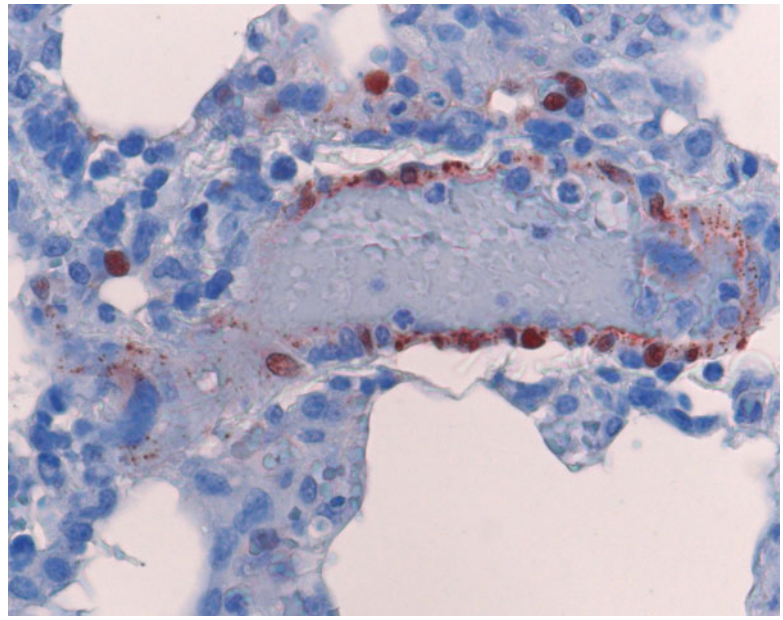


Fig. 24.29 Hendra virus. Moderate magnification of horse lung stained with anti-Hendra antibodies revealing the presence of Hendra virus antigen in the cytoplasm of endothelial cells (*arrow*)

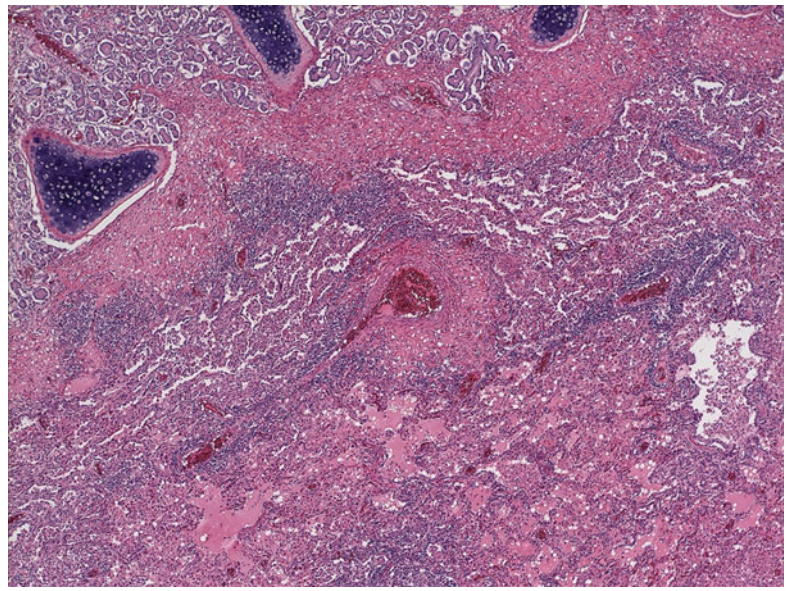


Fig. 24.30 Coronavirus. Low-magnification photomicrograph of cat lung with feline infectious peritonitis illustrating pulmonary edema, congestion, hemorrhage, and multiple pyogranulomas

24.12 Ovine Pulmonary Adenocarcinoma

A unique lung disorder affecting the lungs of sheep is ovine pulmonary adenocarcinoma (OPA). The disorder has a wide geographic distribution and is also known as jaagsiekte, a word derived from the *Afrikaans* word meaning "driving sickness." The causal agent is the RNA jaagsiekte sheep retrovirus (JSRV), currently regarded as a betaretrovirus (Griffiths 2010). The age of affected sheep varies widely from 2 months to 11 years. Initially OPA was regarded as a proliferative disorder with metastatic potential (Nobel

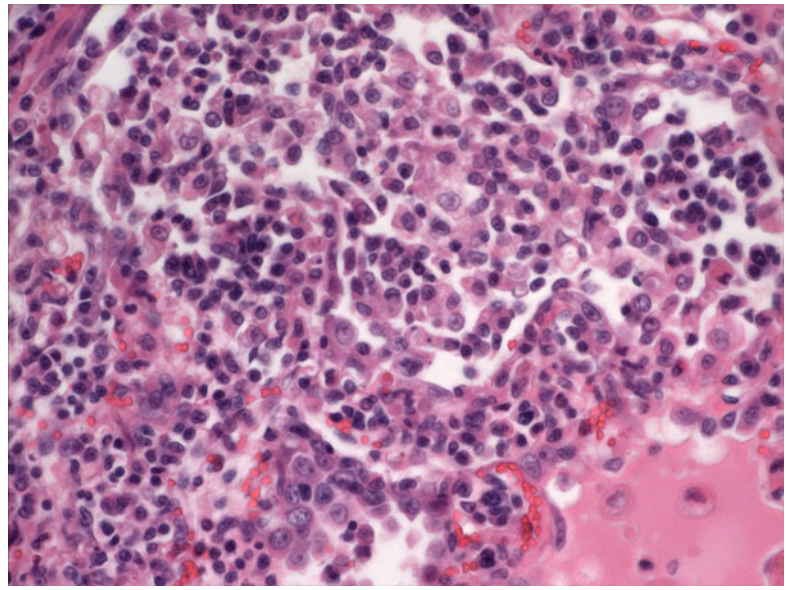


Fig. 24.31 Coronavirus. Higher magnification of a pyogranuloma showing a mixed population of neutrophils, lymphocytes, and macrophages

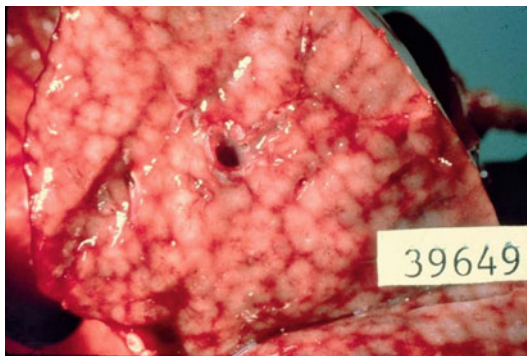


Fig. 24.32 Ovine pulmonary adenocarcinoma. Cross section of sheep pulmonary carcinoma illustrating firm, consolidated pulmonary nodules

et al. 1969). A viral etiology was first reported in 1974 (Perk et al. 1974). Clinically, the disease progresses slowly. Initial manifestations include coughing and exercise intolerance, followed by wheezing, nasal discharge, and crackles. Grossly, white firm consolidated pulmonary nodules are present (Fig. 24.32). Histological examination generally shows papillary and acinar structures lined by columnar to cuboidal cells replacing the alveolar spaces of the lung (Fig. 24.33). Immunohistochemical studies show the presence of surfactant proteins in the majority of affected cells that generally correspond to type II alveolar cells (Platt 2002). Some cells are known to express CC10, a protein of Clara cell origin. Ultrastructural studies have demonstrated the presence of lamellated membrane structures within type II pneumocytes (Fig. 24.34).

24.13 Ovine Progressive Pneumonia

Ovine progressive pneumonia is caused by the maedi-visna virus, a member of the lentivirus genus within the Retroviridae family. Sheep and occasionally goats can be affected with a chronic progressive pneumonia. Grossly, the lungs do not collapse and show firm large white consolidated foci (Fig. 24.35). Histologically, the pneumonia is characterized by the presence of perivascular, peribronchial, and peribronchiolar areas of lymphofollicular proliferation (Fig. 24.36). Interstitial infiltration by lymphocytes, interstitial fibrosis, and smooth muscle hyperplasia can also be seen (Fig. 24.37). There is in addition hyperplasia of pulmonary lymph nodes with formation of germinal centers within the lymphoid follicles (Lairmore et al. 1986). Electron microscopic examination shows the presence of viruses with features of lentiviruses.

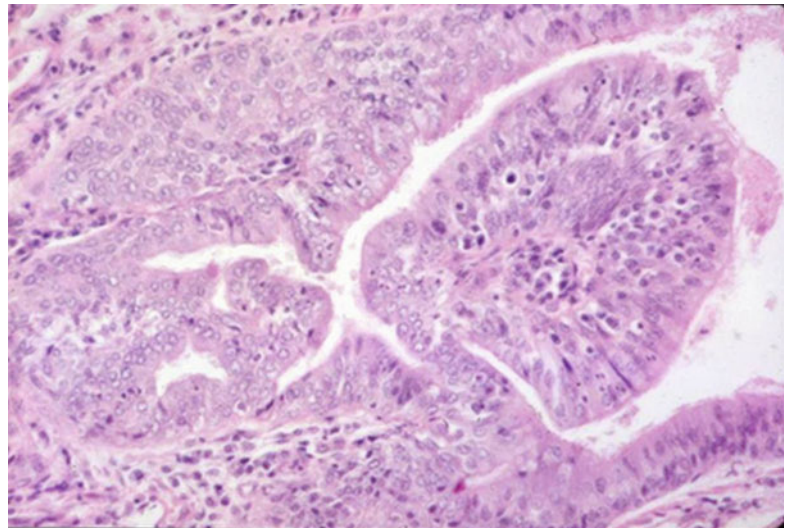


Fig. 24.33 Ovine pulmonary adenocarcinoma. High magnification revealing papillary projections lined by columnar neoplastic epithelial cells

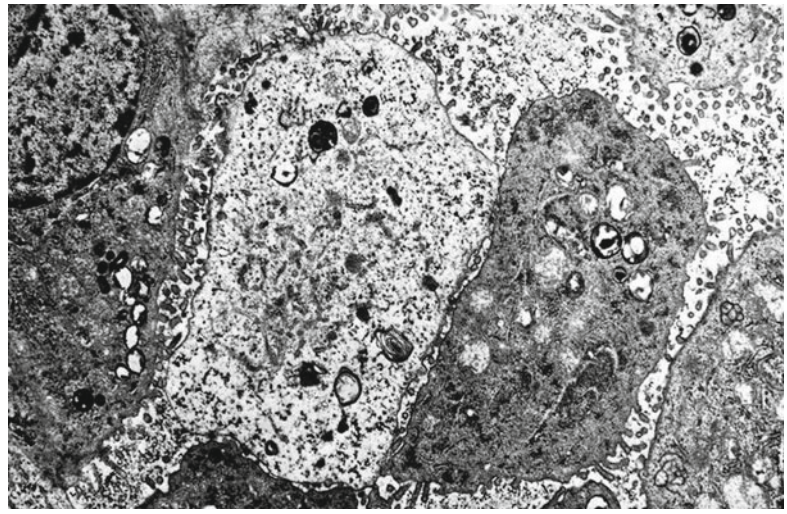


Fig. 24.34 Ovine pulmonary adenocarcinoma. Electron micrograph showing neoplastic cells with microvilli and cytoplasm with membrane-bound vesicles that contain surfactant

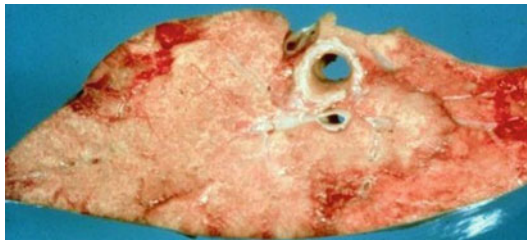


Fig. 24.35 Ovine progressive pneumonia. Sheep lung with a large area of consolidation that occupies most of the lung substance

24.14 Simian Immunodeficiency Virus

Simian immunodeficiency virus (SIV) is a lentivirus in the family of Retroviridae. Infections secondary to SIV occur spontaneously and asymptomatically in several species of monkeys and macaques, the latter sometimes developing an immunodeficiency syndrome (Baskin et al. 1991).

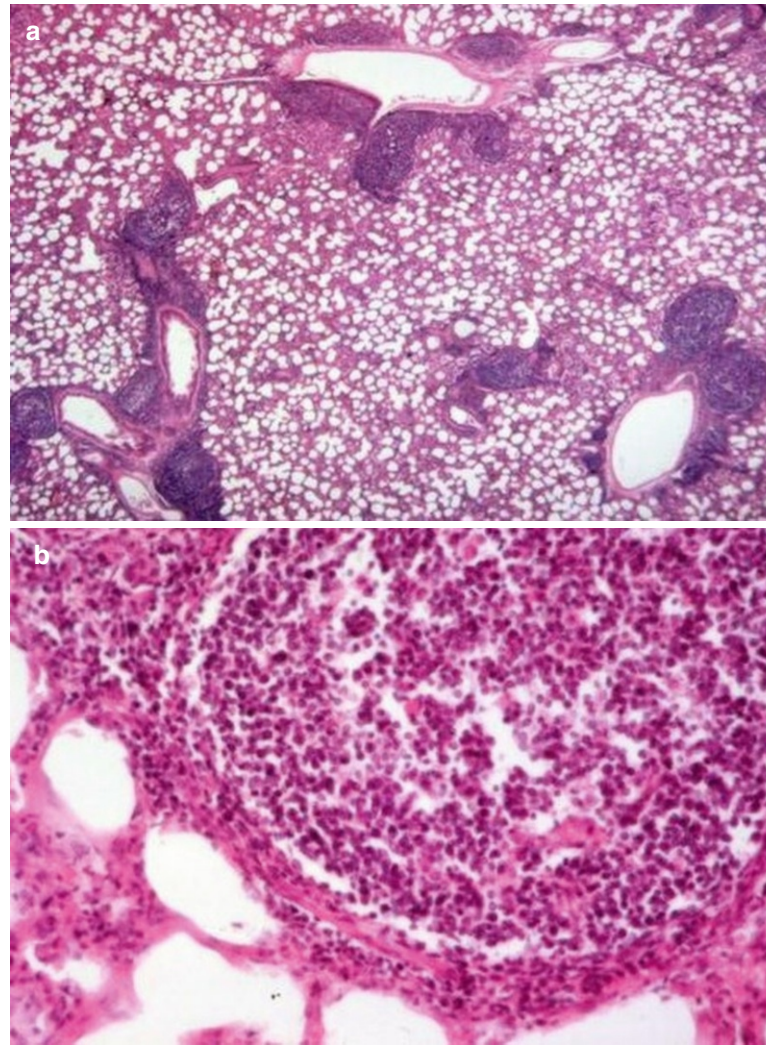


Fig. 24.36 (**a**) Ovine progressive pneumonia. Low-magnification photomicrograph demonstrates the presence of perivascular and peribronchial lymphoid infiltrates (*arrow*). (**b**) Corresponding high magnification

Gross findings in the lungs include diffuse pleural thickening, with some irregular firm foci of consolidation. The lesions seen in early stage of the infection tend to be localized. Microscopically, perivascular infiltration by lymphocytes and histiocytes around small vessels (Fig. 24.38) as well as infiltration of the alveolar septa by mononuclear inflammatory cells (Fig. 24.39) can be seen. Syncytial multinucleated giant cells occur during the stage of rapid multiplication (Fig. 24.40). In advanced infections the lesions are diffuse and/or patchy (Fig. 24.41) and are associated with alveolar spaces filled with exudate that include fibrin, histiocytes, lymphocytes, plasma cells, neutrophils, and syncytial cells (Fig. 24.42). In advanced stages there is mild fibrosis of the alveolar septae and fibrosis of the visceral pleura (Baskin et al. 1991). Mature virions are present in cytoplasmic vacuoles in syncytial cells and macrophages (Fig. 24.43).

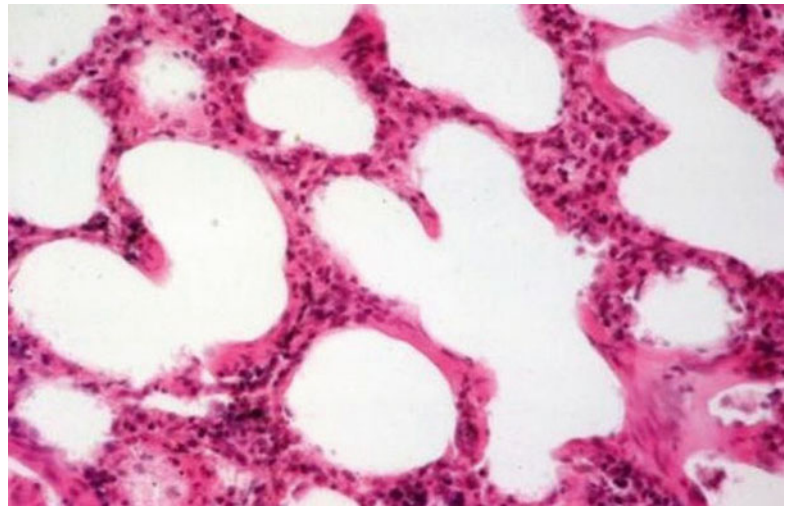


Fig. 24.37 Ovine progressive pneumonia. Note moderate magnification showing thickened alveolar walls

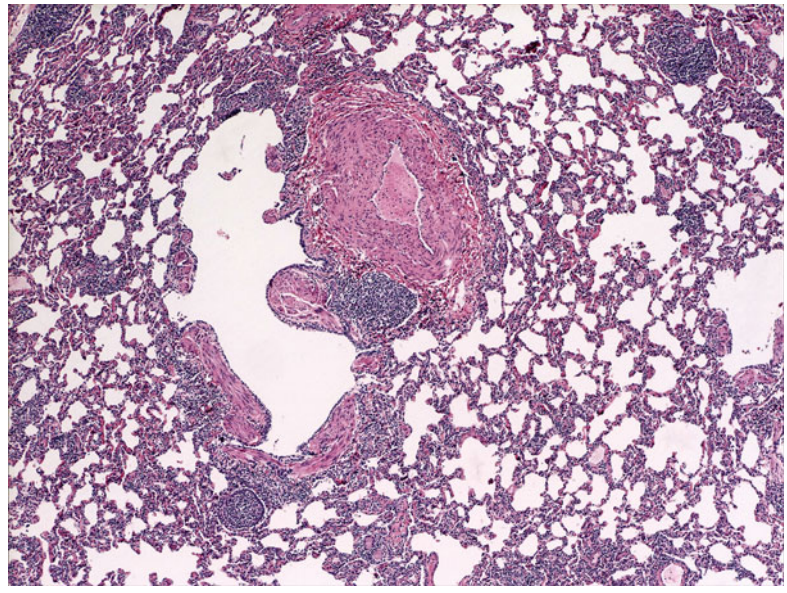


Fig. 24.38 Simian immunodeficiency virus. Low-magnification photomicrograph of early stage of lung infection revealing normal size bronchial-associated lymphoid tissue (BALT) and multiple foci of small perivascular infiltration

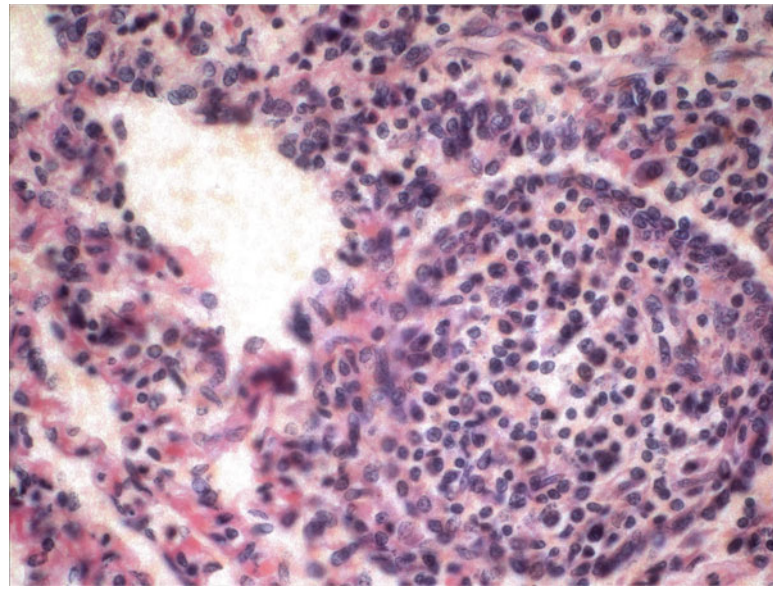


Fig. 24.39 Simian immunodeficiency virus. Higher magnification showing infiltration of the alveolar septae by mononuclear cells, presence of a few neutrophils, and BALT lymphoid tissue

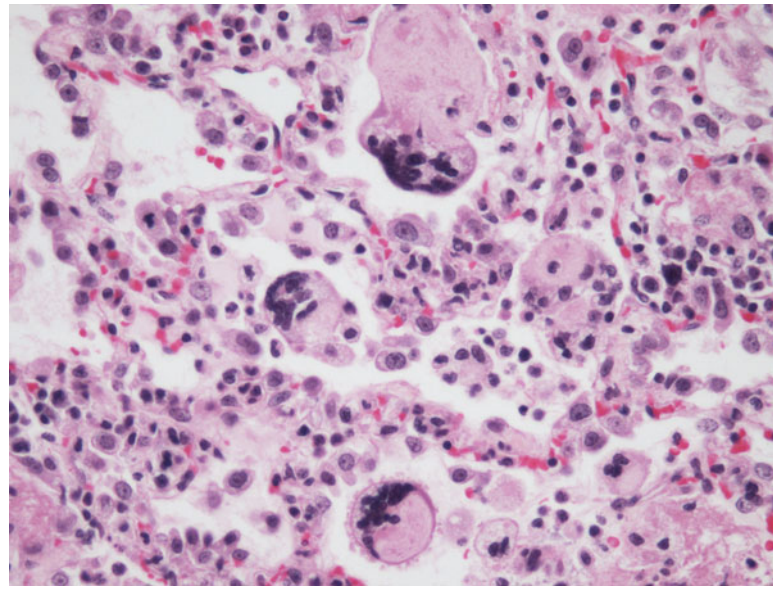


Fig. 24.40 Simian immunodeficiency virus. Syncytial multinucleated giant cells occur during the stage of rapid multiplication

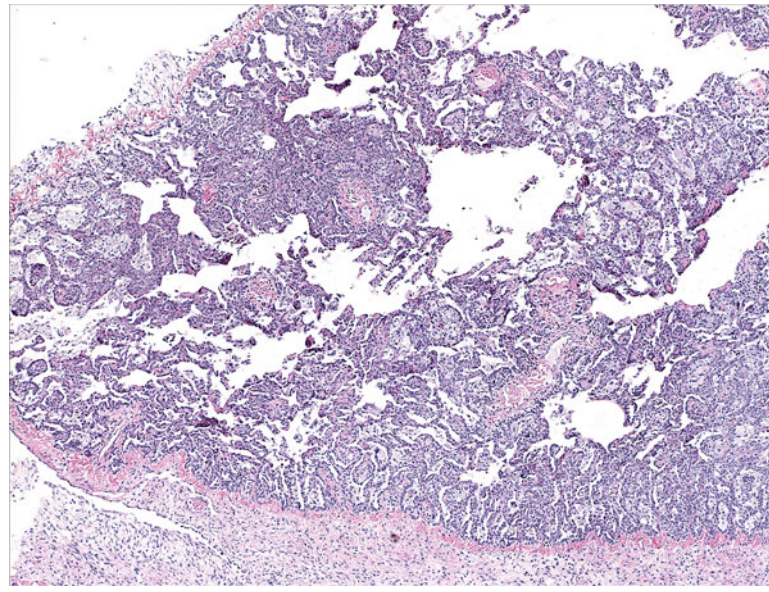


Fig. 24.41 Simian immunodeficiency virus. Low-magnification photomicrograph of a severe chronic lung infection demonstrating diffuse lesions with alveolar space filled by inflammatory exudate and heavy infiltration of the lung parenchyma by mixed inflammatory cells (*arrow*)

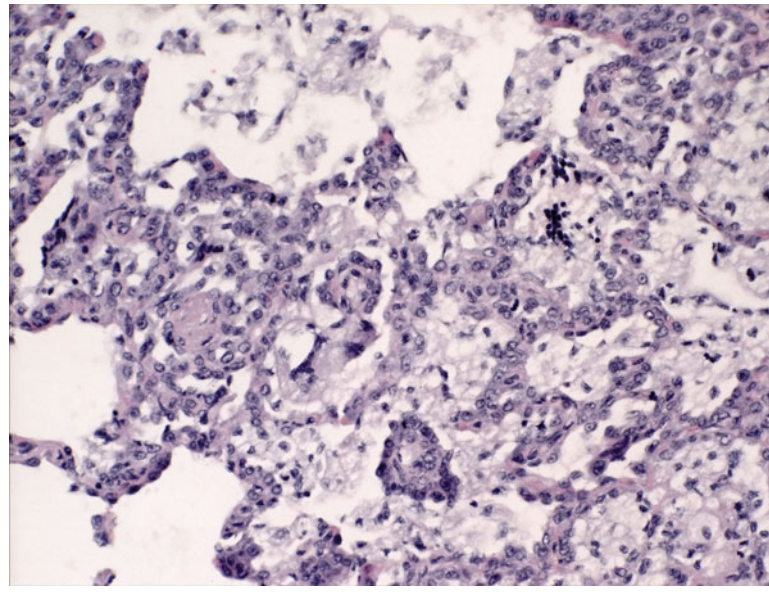


Fig. 24.42 Simian immunodeficiency virus. Closeup view highlighting the presence of alveolar exudate, multinucleated giant cell, and infiltration of the alveolar walls by lymphocytes

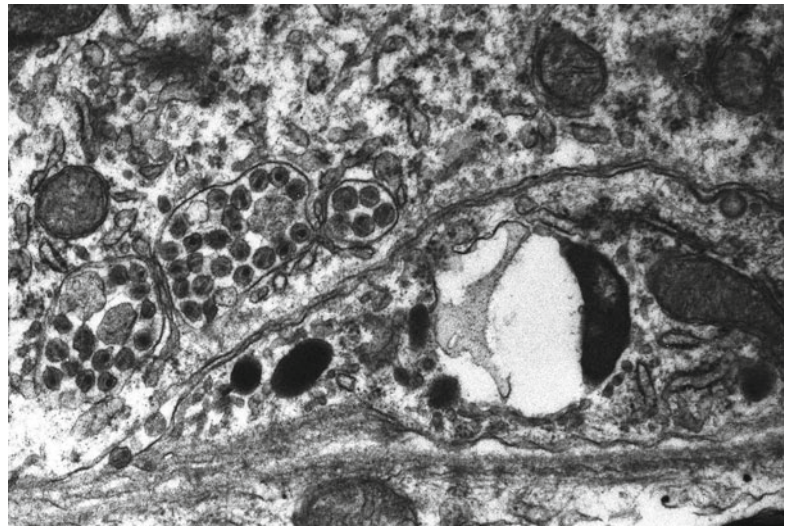


Fig. 24.43 Simian immunodeficiency virus. Electron micrograph. Lung of infected rhesus monkey showing viral particles within a cytoplasmic vacuole in an alveolar macrophage (Courtesy Dr. Susan Westmoreland, Harvard Medical School/New England Primate Research Center)

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