Viral diseases of pet animals

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Simplified notes on some important viral diseases affecting pets presented

1-Infectious canine hepatitis

INFECTIOUS CANINE HEPATITIS

- caused by canine adenovirus 1
- Adenoviruses **are** cytolytic **and** cause necrosis of infected cells
- The virus has a predilection for hepatocytes, vascular endothelium, and mesothelium;

- The majority of infections are asymptomatic, and infections that result in disease may not be fatal
- **Young dogs, in the first 2 years of life**, are more likely to die of the infection than older dogs.

Pathogenesis

1. oral route by contact with urine from infected dogs.
2. Viremia lasts for 4 to 8 days
3. Virus multiplication **initially occurs in** the tonsils and produces tonsillitis, which can be severe, with **spread to** local lymph nodes and then to the systemic circulation

PM lesions

- widespread petechiae and ecchymoses
- accumulation of clear fluid in the peritoneal and other serous cavities
- the presence of fibrin strands on the surface of the liver,
- enlargement and reddening of the tonsils and lymph nodes.

- **The liver** is **moderately enlarged** and friable and may contain **small foci of hepatocellular necrosis (**centrilobular necrosis**)** → **An** enhanced lobular pattern**.**
- Characteristically, the wall of the gallbladder is thickened by edema**.**
- Foci of hemorrhage in the **lung, brain, kidneys, and the metaphysis of the long bones** may also be evident.

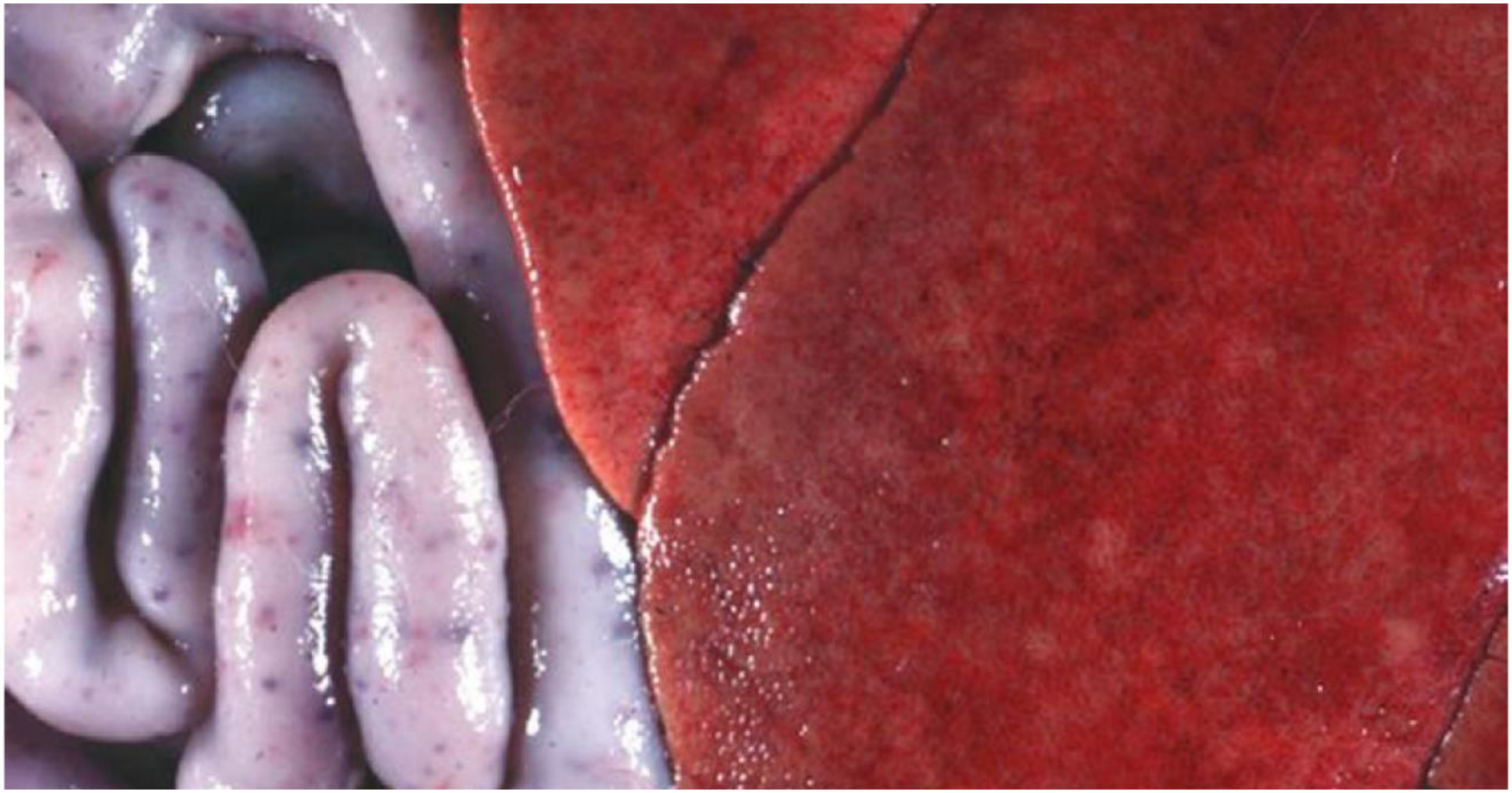


Fig. 8-74 Infectious canine hepatitis, hepatic necrosis, liver, dog.

A, The liver from a dog infected with infectious canine hepatitis (ICH) can be slightly enlarged and friable with a blotchy yellow discoloration. Sometimes, fibrin is evident on the capsular surface. Note the petechiae on the serosal surface of the intestines caused by vascular damage from canine adenovirus type I infection. **B,** Infection of hepatocytes and endothelial cells

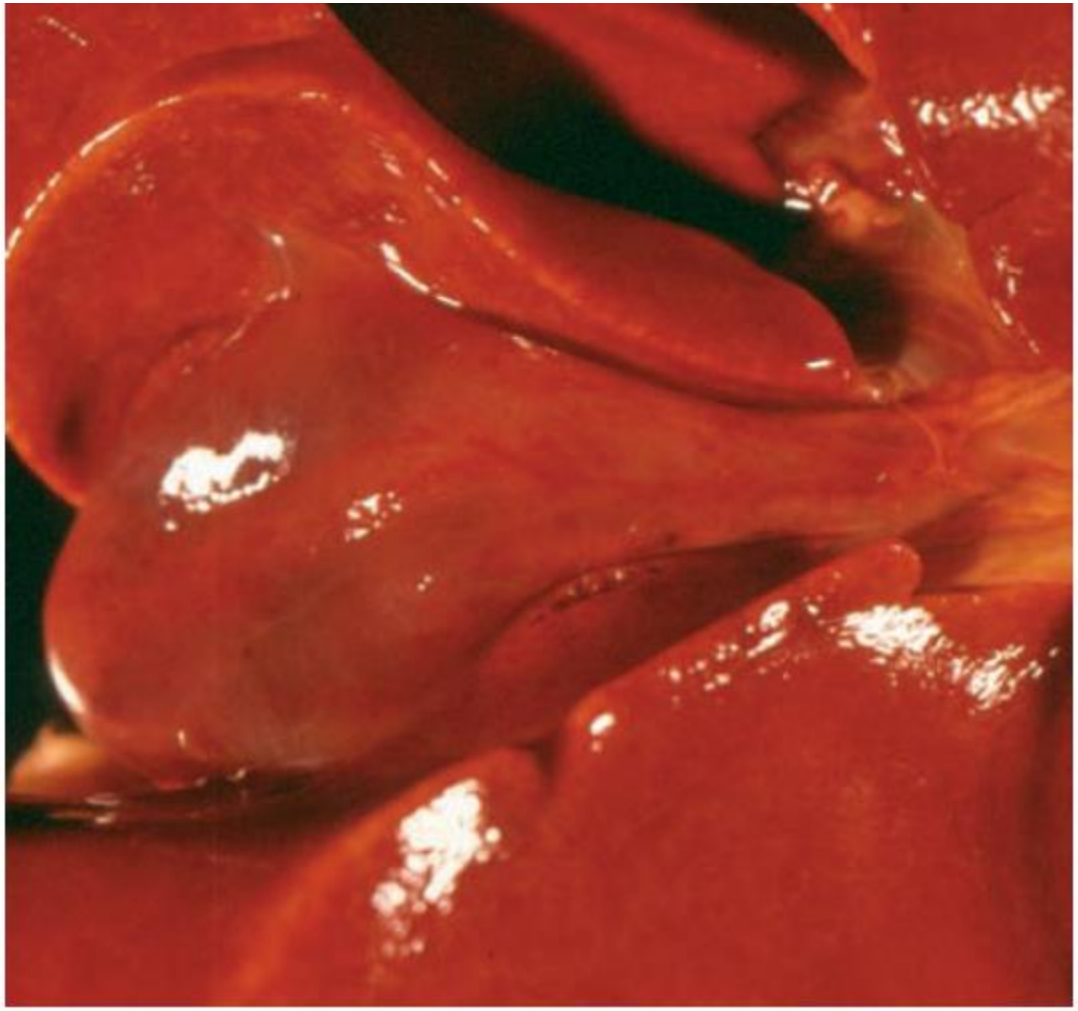
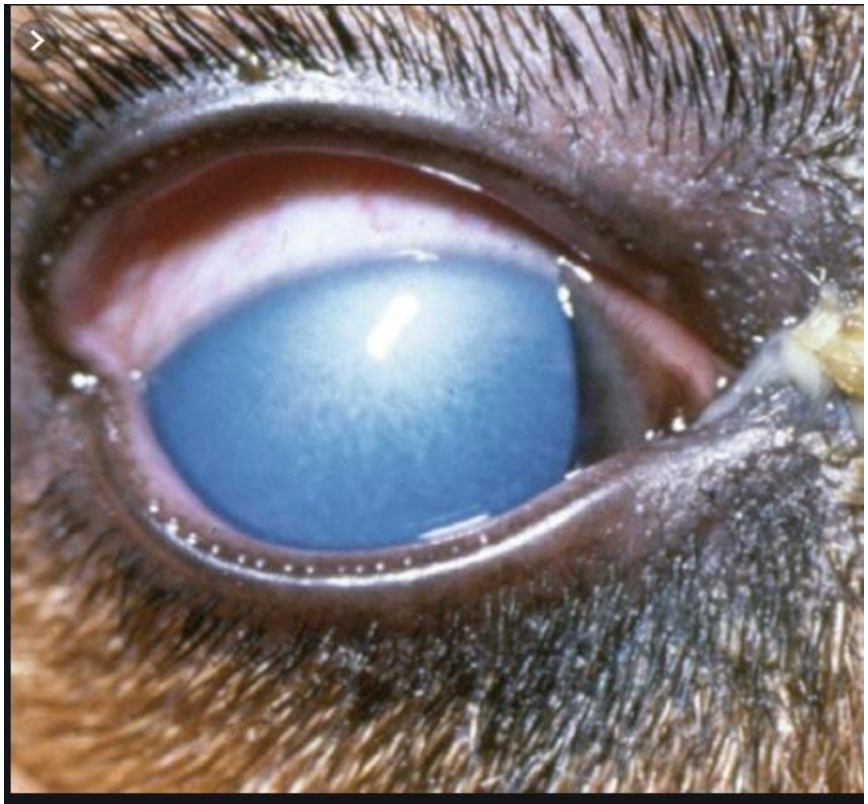


FIG. 4-3 Swollen, mottled liver with rounded lobar edges and gallbladder edema characteristic of ICH. (Photograph by Craig Greene © 2004 University of Georgia Research Foundation Inc.)

Some dogs recovering from infectious canine hepatitis develop an immunecomplex uveitis (type III hypersensitivity),

- which produces degeneration and necrosis of the corneal endothelium and resultant corneal edema clinically known as “blue eye.”



A, During the **viremic period**, CAV-1 enters the **eye** via the **uveal tract**, localizing in **vascular endothelial cells of the choroid** and causing **mild uveitis**. Virus also enters the **aqueous humor** and localizes in **the corneal endothelial cells**

B, CAV-1 **specific antibody response** increases in the blood and **reaches the eye** via the uveal tract and enters **the aqueous humor in the presence of virus.**

C, Virus is free in the aqueous and endothelial cells and viral-antibody complex formation and intranuclear inclusion body formation.

D, Complement fixation on virus-immune complexes free and in endothelial cells to chemotaxis of neutrophils. More severe uveitis and corneal endothelial injury occurs.

E, Close up showing **loss of endothelium and aqueous pump** leading to **influx of aqueous into the cornea**.

F, **Corneal endothelial cell loss** allows **aqueous to enter the cornea causing corneal edema** (**blue eye**). **After mononuclear phagocytes remove virus-immune complexes**, and **inflammation subsides**, **corneal endothelium regenerates**.

G, Uveal inflammation may lead to blockage of the filtration angle and subsequent glaucoma.

- Uveitis is inflammation of the uvea — the middle layer of the eye that consists of the iris, ciliary body and choroid

Histopathology

- scattered foci of hepatocellular necrosis (**centrilobular necrosis**). necrosis of endothelial cells that may lead to vascular stasis and local hypoxia.
- **Large deeply eosinophilic to amphophilic intranuclear inclusions** are found in **hepatocytes, vascular endothelium, and Kupffer cells**

- **Inflammation tends to be mild** and **neutrophils** are the most abundant cell type.
- **Virus-induced endothelial damage** may lead to **DIC** and **hemorrhagic diathesis**, which contribute to the **hemorrhage observed** in affected dogs.

- Viral-induced **intranuclear inclusions** are present **in glomerular capillary endothelium** in cases of infectious canine hepatitis.

2-Coronaviruses

- Feline infectious peritonitis (FIP), which is caused by a **coronavirus**
- . There are two recognized feline coronaviruses (FCoV):

❖ **feline enteric coronavirus(FECV)**

❖ **feline infectious peritonitis virus (FIPV)**

2-Feline infectious peritonitis

- FIP generally occurs sporadically in cats of all ages, but it is most common in younger cats **between the ages of 3 months and 3 years** and can be clinically significant because it can result in death.
- The disease manifests itself in **effusive (wet) or noneffusive (dry) forms.**
- in the **CNS** can include **behavioral changes, dullness, coma, paresis, ataxia, paralysis, and seizures.**

Pathogenesis

1
- ingestion of contaminated saliva or feces
- direct inoculation (e.g., cat bites, licking open wounds)
- in utero

2
- **After infection**, the virus **replicates in macrophages** that spread the virus to the **liver, visceral peritoneum and pleura, uvea, and the meninges and ependyma of the brain and spinal cord.**

3
- the **development of disease** depends on the **type and degree of immunity that develops**

Forms

Depending on the immune response, **the pathogenesis** can involve

- a **primary immune complex vasculitis (type III hypersensitivity** [**effusive form**])
- and/or delayed hypersensitivity response **(type IV hypersensitivity** [**noneffusive form**]);
- thus the lesions are **oriented around blood vessels** (**primarily capillaries and venules**) and **are granulomatous.**

- **Type III hypersensitivity** occurs when there is accumulation of immune complexes (antigen-antibody complexes) that have not been adequately cleared by innate immune cells, giving rise to an inflammatory response and attraction of leukocytes. Such reactions may progress to **immune complex diseases**.

- **Type IV hypersensitivity** is often called delayed type hypersensitivity as the reaction takes several days to develop.[1] Unlike the other types, it is not antibody-mediated but rather is a type of **cell-mediated** response. This response involves the interaction of T-cells, monocytes, and macrophages.

Forms (PM lesions)of feline infectious peritonitis

Effusive form

- **Serositis**
- accumulation of fluid in the abdominal and thoracic cavities, with varying degrees of severity of inflammation

Non-effusive form

- **There are multifocal, coalescing white to gray granulomas**
- leptomeningitis, chorioependymitis, focal encephalomyelitis, and ophthalmitis
- In the CNS, **pyogranulomatous vasculitis**
- The uvea, retina, and optic nerve sheath are also commonly involved

- **Serositis** refers to inflammation of the serous tissues of the body, the tissues lining the lungs (pleura), heart (pericardium), and the inner lining of the abdomen (peritoneum) and organs within

leptomeningitis inflammation of The **arachnoid mater and the pia mater .**

PM lesions

- The basic lesion in effusive and non-effusive FIP is a pyogranulomatous inflammation → vasculitis → vascular necrosis → infarction.
- This lesion appears to result from deposition of immune complexes, which subsequently induce an inflammatory reaction in affected vessels.
- There are multifocal, coalescing white to gray granulomas

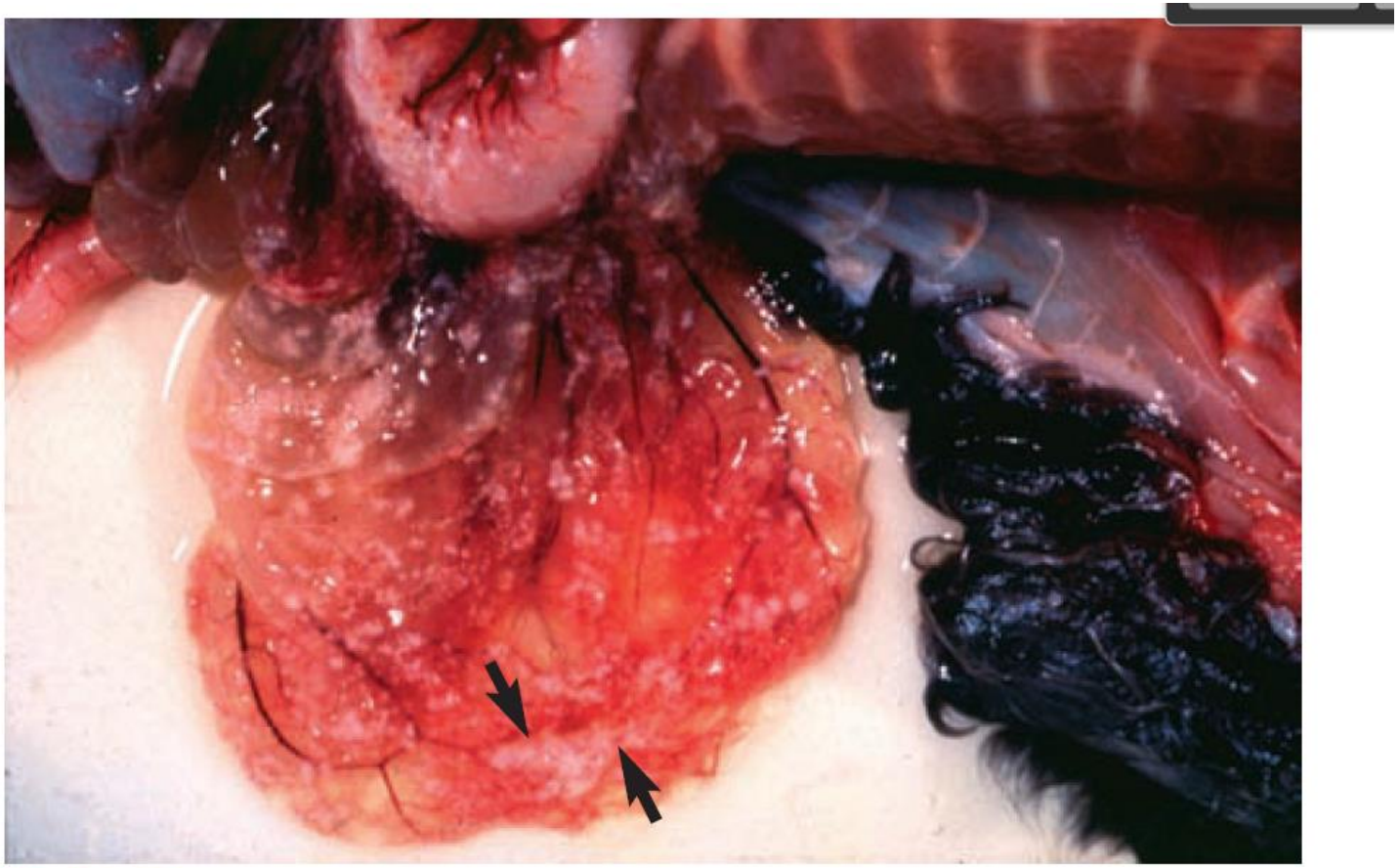
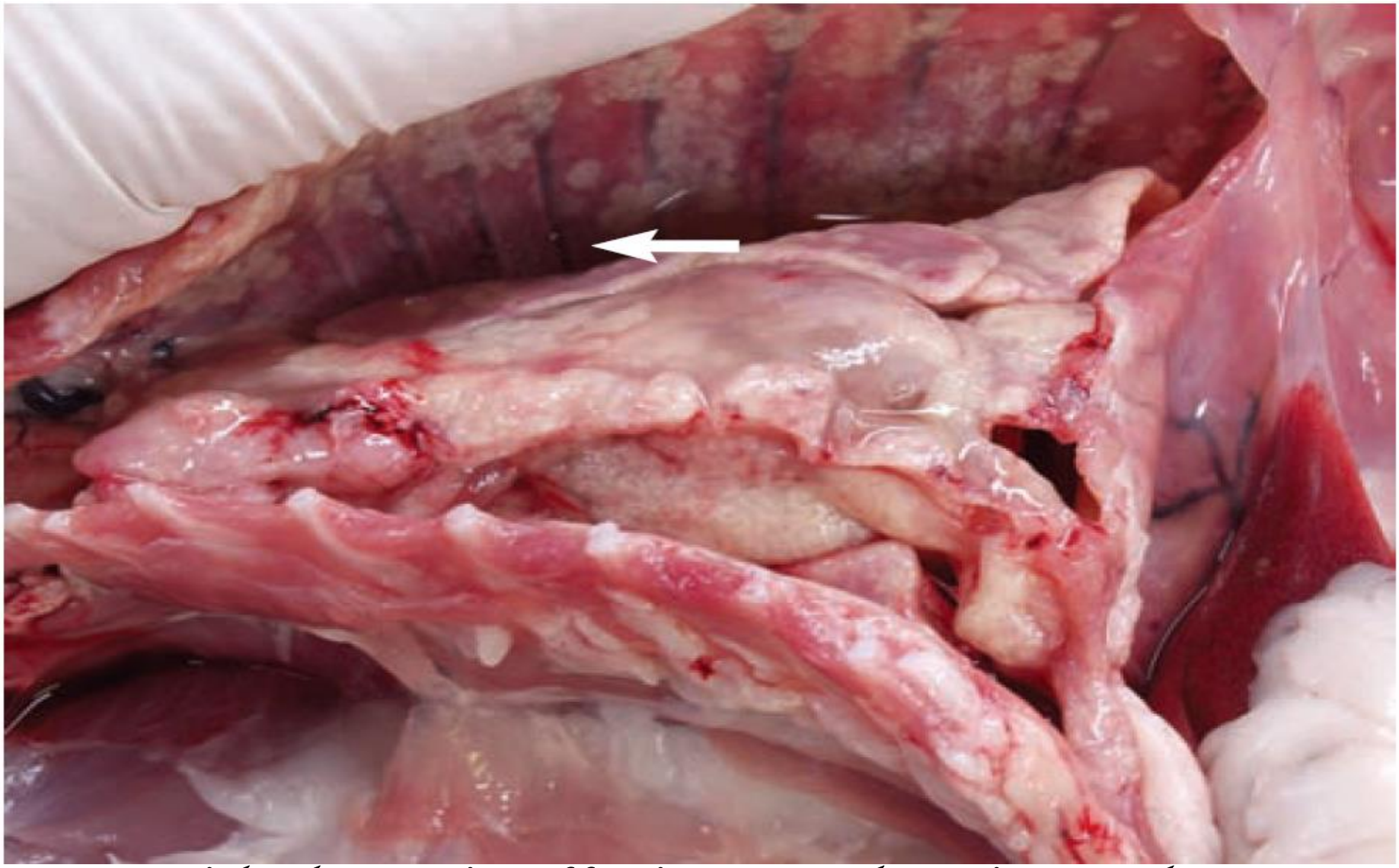
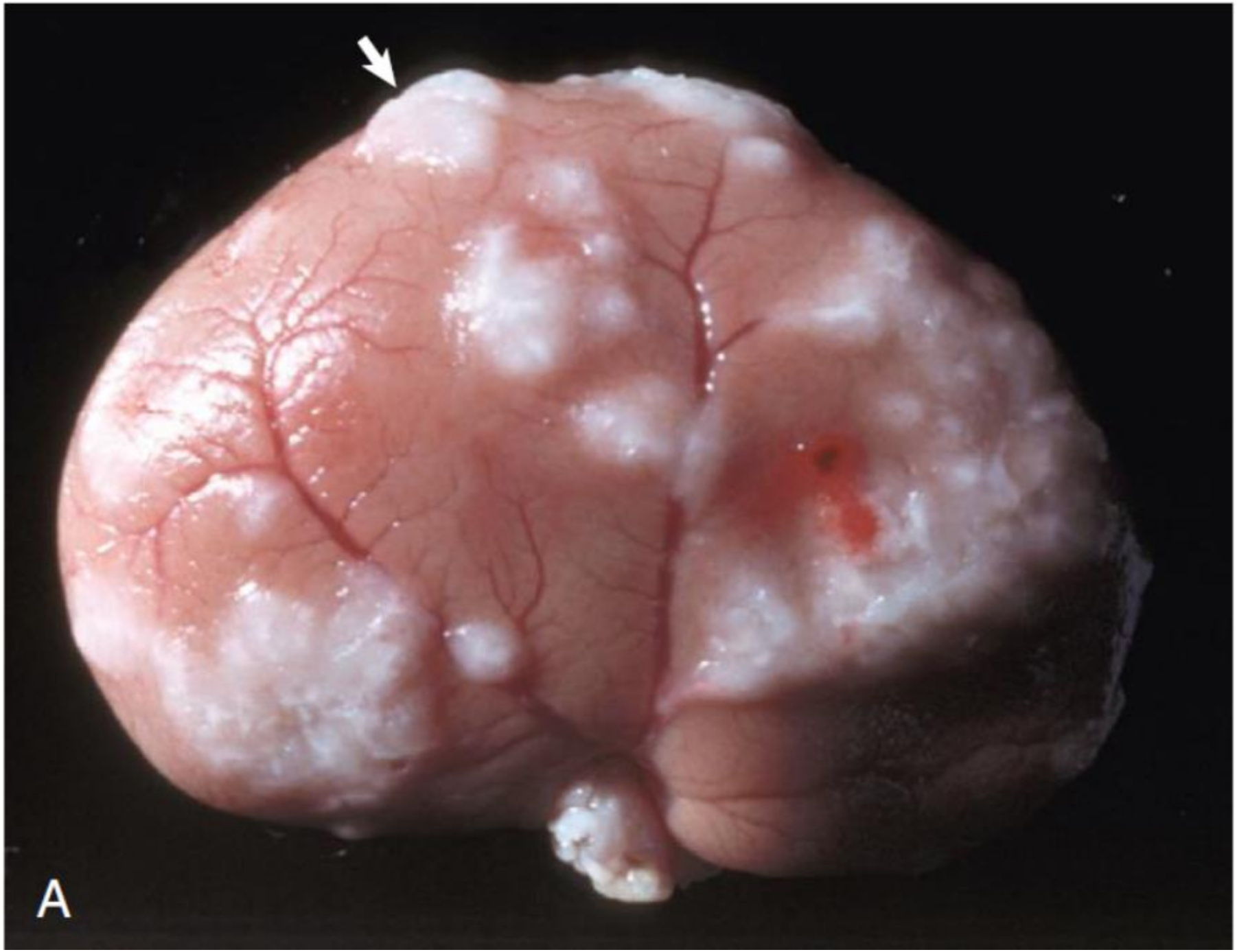


FIG. 10-10 Omentum of a cat with effusive FIP. Note gelatinous appearance and small, white perivascular pyogranulomata *(arrows)* typical of effusive FIP on gross postmortem examination.

gelatinous appearance and small,white perivascular pyogranulomata *(arrows)* typical of effusive FIP



cat with thoracic effusive FIP, showing a clear, amber effusion *(arrow)* , fibrin on the pleura, and pyogranulomata within the lung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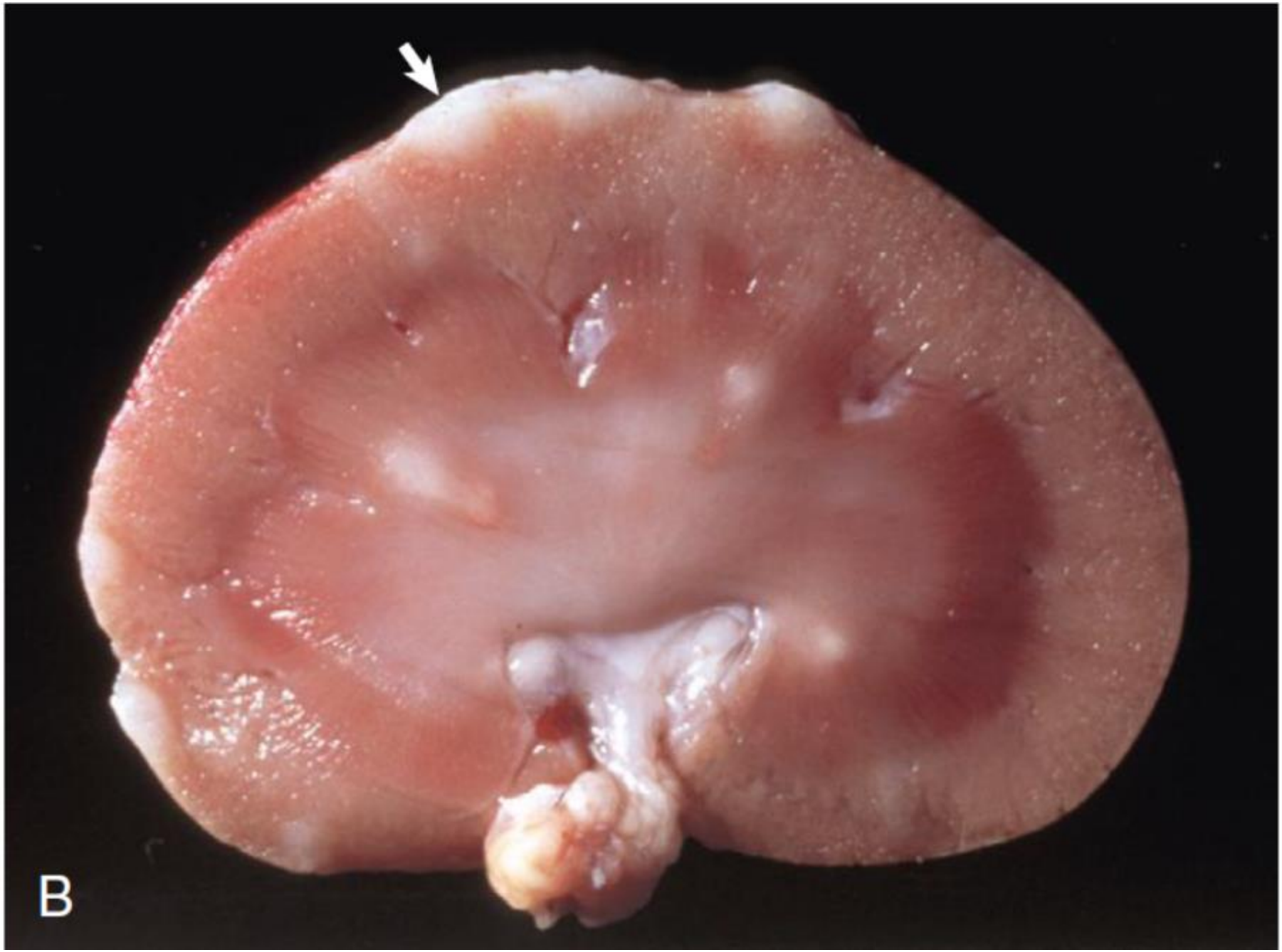
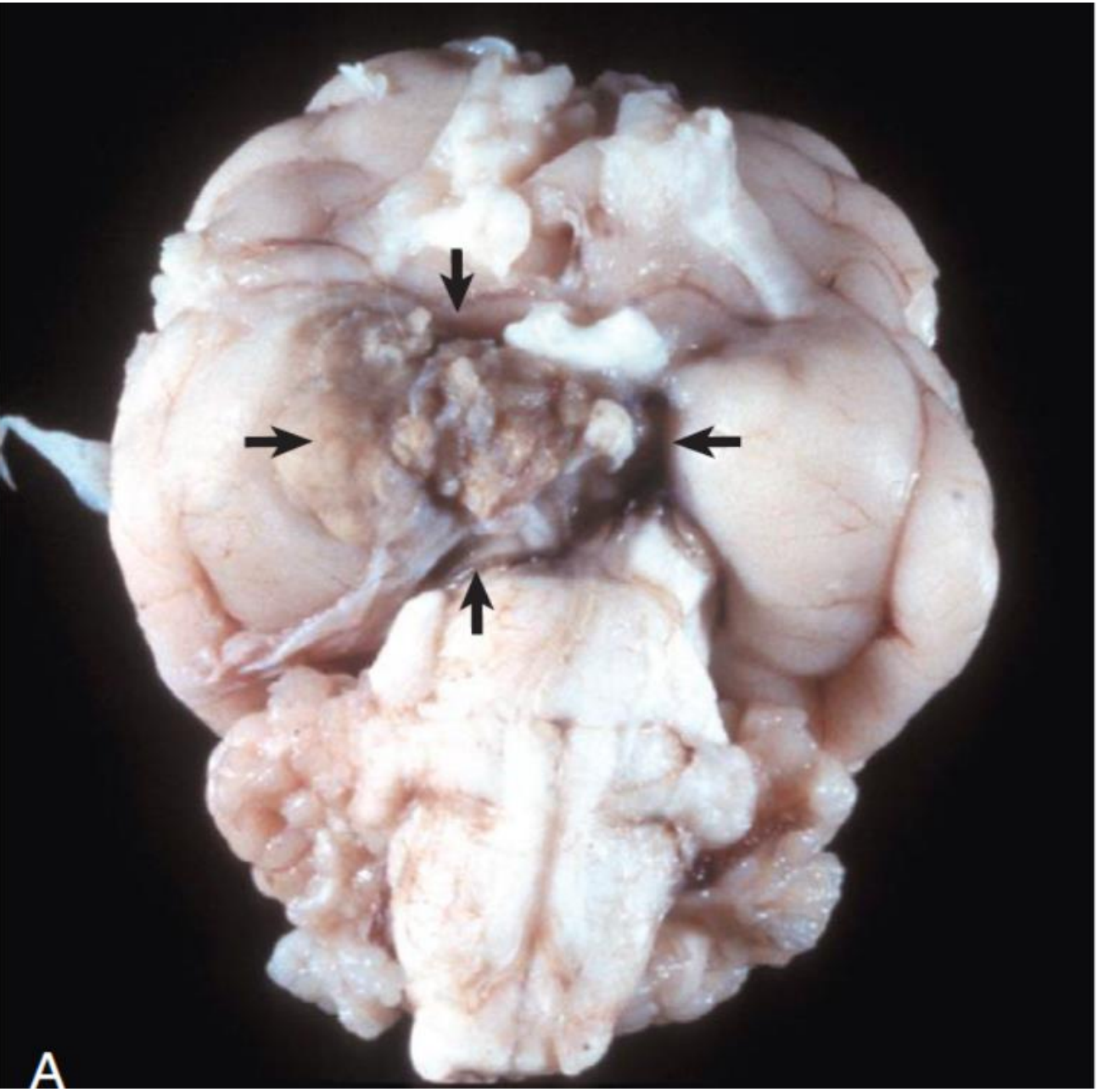

B

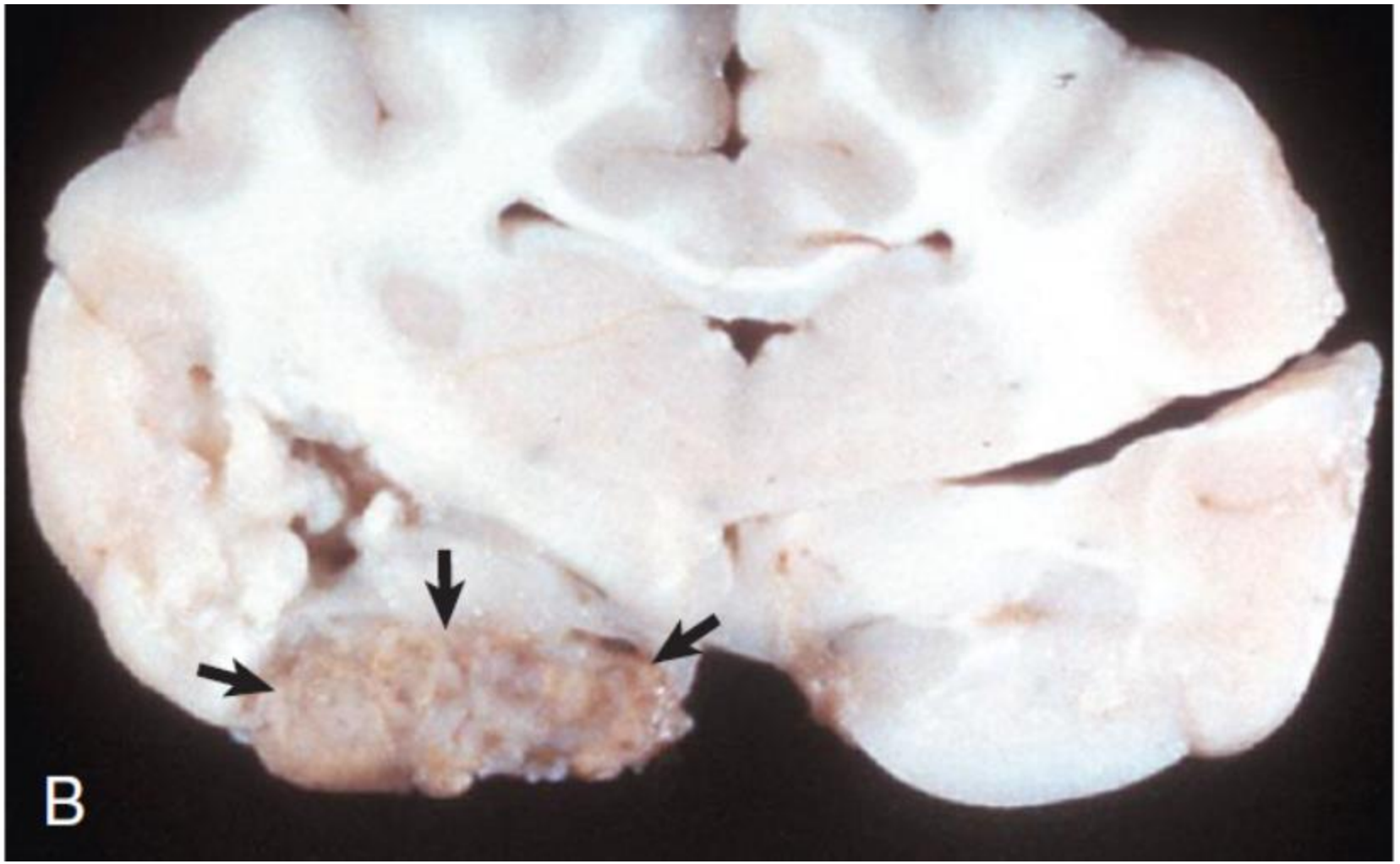
Fig. 11-75 Granulomatous nephritis, feline infectious peritonitis kidney, cat.

A, Lesions are typical of the noneffusive (dry) form of feline infectious peritonitis. There are multifocal, coalescing white to gray granulomas *(arrow)* which can be confused with the nodular form of lymphosarcoma, thus warranting histologic examination. **B,** Dorsal section. Multifocal, coalescing white to gray granulomas extend into the cortical parenchyma *(arrow)*. The pathogenesis of this lesion is determined by the effectiveness and/or ineffectiveness of both humoral and cellular immune responses. Depending on the immune response, the pathogenesis can involve a primary immune complex vasculitis (type III hypersensitivity [effusive form]) and/or delayed hypersensitivity response (type IV hypersensitivity [noneffusive form]); thus the lesions are oriented around blood vessels (primarily capillaries and venules) and are granulomatous. (Courtesy Dr. M.D. McGavin, College of Veterinary Medicine, University of Tennessee.)



Periventricular white matter (arrows) beneath the fourth ventricle (between the medullary velum and medulla

The type III hypersensitivity and pyogranulomatous inflammation cause vascular and perivascular injury, vasogenic edema, and parenchymal disruption



Pyogranulomatous inflammation

Fig. 14-105 Pyogranulomatous vasculitis, feline infectious peritonitis, cat.

A, Ventral brain, cerebral vasculature of the circle of Willis. A white-yellow pyogranulomatous inflammation distorts and obscures the blood vessels. Lesions are attributed to deposition of immune complexes (type III hypersensitivity), and in some cases possibly with a cell-mediated component, in the vessel walls that results in inflammation *(arrows)*. The character of the inflammatory response can vary from an exudate with accumulation of serous fluid and fibrin mixed with neutrophils and histiocytes to a reaction that is more pyogranulomatous, and in which commonly there are lymphocytes and plasma cells. The severity and magnitude of the lesion depicted here is much more dramatic than usual. **B,** A cross-sectional view of **A.** The pyogranuloma *(arrows)* is principally in the subarachnoid space and has compressed the adjacent cerebral cortex. (**A** and **B,** Courtesy Dr. J. Sundberg, College of Veterinary Medicine, University of Illinois.)

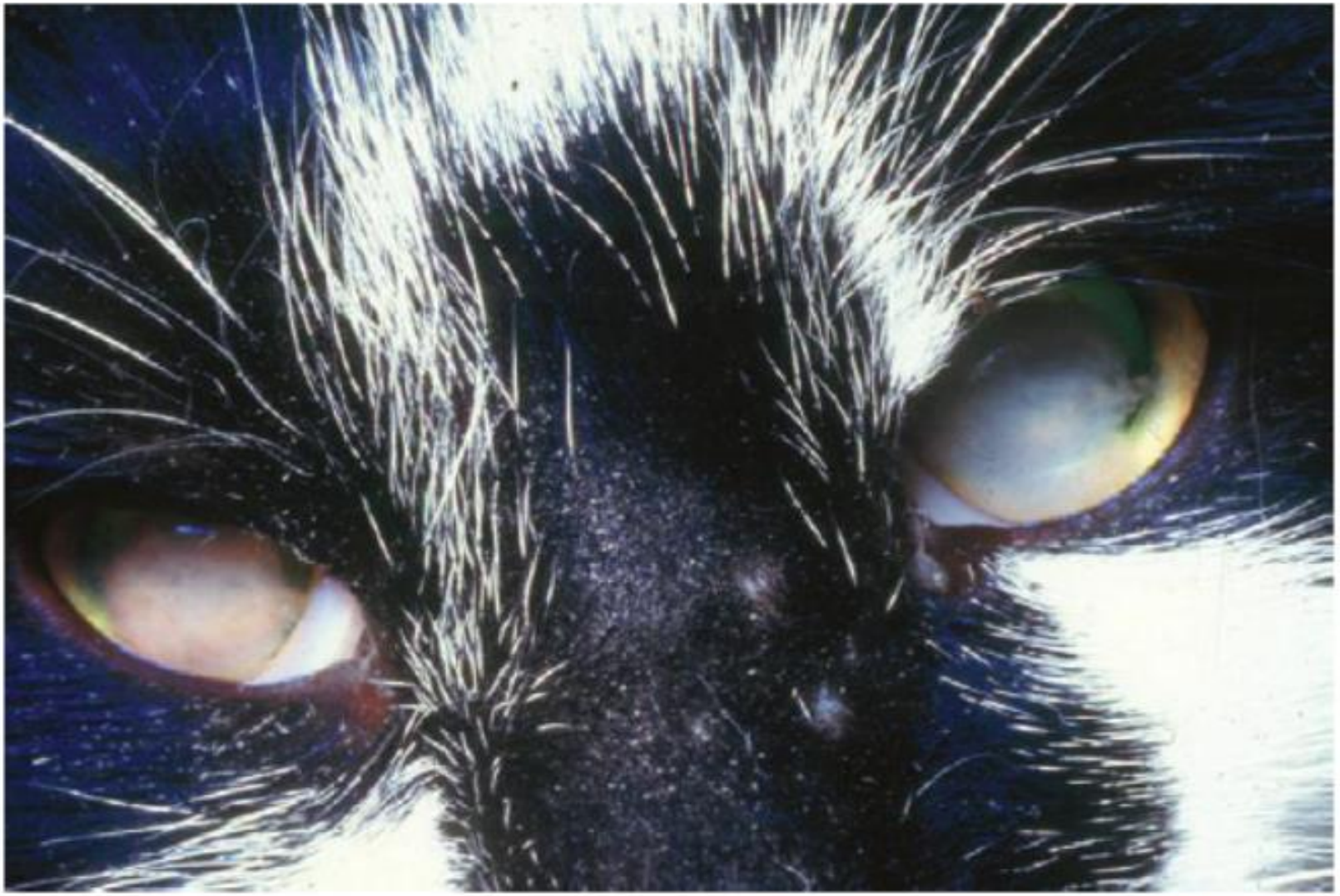


Fig. 20-69 Hypopyon (bilateral), feline infectious peritonitis, cat.
A mixture of fibrin and neutrophils (white-gray opacity) is present within the anterior chamber of the eyes. (Courtesy Dr. B. Wilcock, Ontario Veterinary College.)

Hypopyon is a condition involving inflammatory cells in the anterior chamber of the eye.

- Iridocyclitis: inflammation of iris and ciliary body

3-canine distemper

- It is caused by a **Morbillivirus (**family **Paramyxoviridae**) and has a worldwide distribution.
- Morbilliviruses other than CDV include measles virus, rinderpest virus, peste des petits ruminants virus, phocine distemper virus of seals, equine Morbillivirus, and dolphin and porpoise Morbilliviruses.

Viral tropism

- The virus is pantropic and has a particular affinity for lymphoid and epithelial tissues (lung, gastrointestinal tract, urinary tract, skin) and the CNS (including the optic nerve) and eye.
- In the CNS, there is demyelination without any substantial amount of inflammation.

- **Canine distemper** virus preferentially infects lymphoid, epithelial, and nervous cells.

- The distemper virus spreads from the **tonsil** and **tracheobronchial lymph nodes** to the spleen, bone marrow, and distant lymph nodes, where it causes lymphoid necrosis.

- **The cortices of lymph nodes** of dogs infected with canine distemper are **depleted of lymphocytes** 6 to 9 days after exposure. This **loss of lymphocytes** is also reflected hematologically by a **profound lymphopenia**.

- Paramyxovirus ~ Canine distemper ~ **Demyelination/encephalitis/myelitis**

Pathogenesis

- CDV is spread between dogs by aerosol transmission. The virus is trapped in the mucosa of the nasal turbinates (centrifugal turbulence), infects local macrophages, and is spread by macrophages (leukocytic trafficking) to regional lymph nodes (retropharyngeal).

- CDV replicates in these regional lymph nodes and replication is followed by a primary viremia that infects systemic lymph nodes, spleen, and the thymus approximately 48 hours after exposure

A, Sequential pathogenesis of CDVinfection.

1, CDV enters the respiratory tract via aerosols and colonizes the local lymphoid tissues such as the tonsils

2, **Primary viral replication** occurs in the **tonsils, retropharyngeal nodes**, **bronchial lymph nodes**, and **gastrointestinal (GI) lymphoid tissue.**

3, From these sites of primary replication, **macrophages containing CDV** enter **lymphatics** traveling back to the **heart**, where they enter the blood as **a mononuclear cell-associated viremia**

4, Virus enters the central nervous system (CNS) via the cerebral circulation. There it is deposited in the perivascular spaces of fine blood vessels.

5, Alternatively, virus enters the vessels of the choroid plexus and eventually the cerebrospinal fluid (CSF) and ventricular system.

6, As an uncommon phenomenon in dogs, CDV can travel from the nasal passage, through the cribiform plate and anterograde via the olfactory nerve to the olfactory bulb and CNS. There it localizes, predominantly in the pyriform lobes of the cerebral cortex.

- With infection of the **lymphoid system**, **immunosuppression** can occur, **resulting in secondary bacterial infection**s, such as conjunctivitis, rhinitis, and bronchopneumonia, which are commonly seen in CDV infections

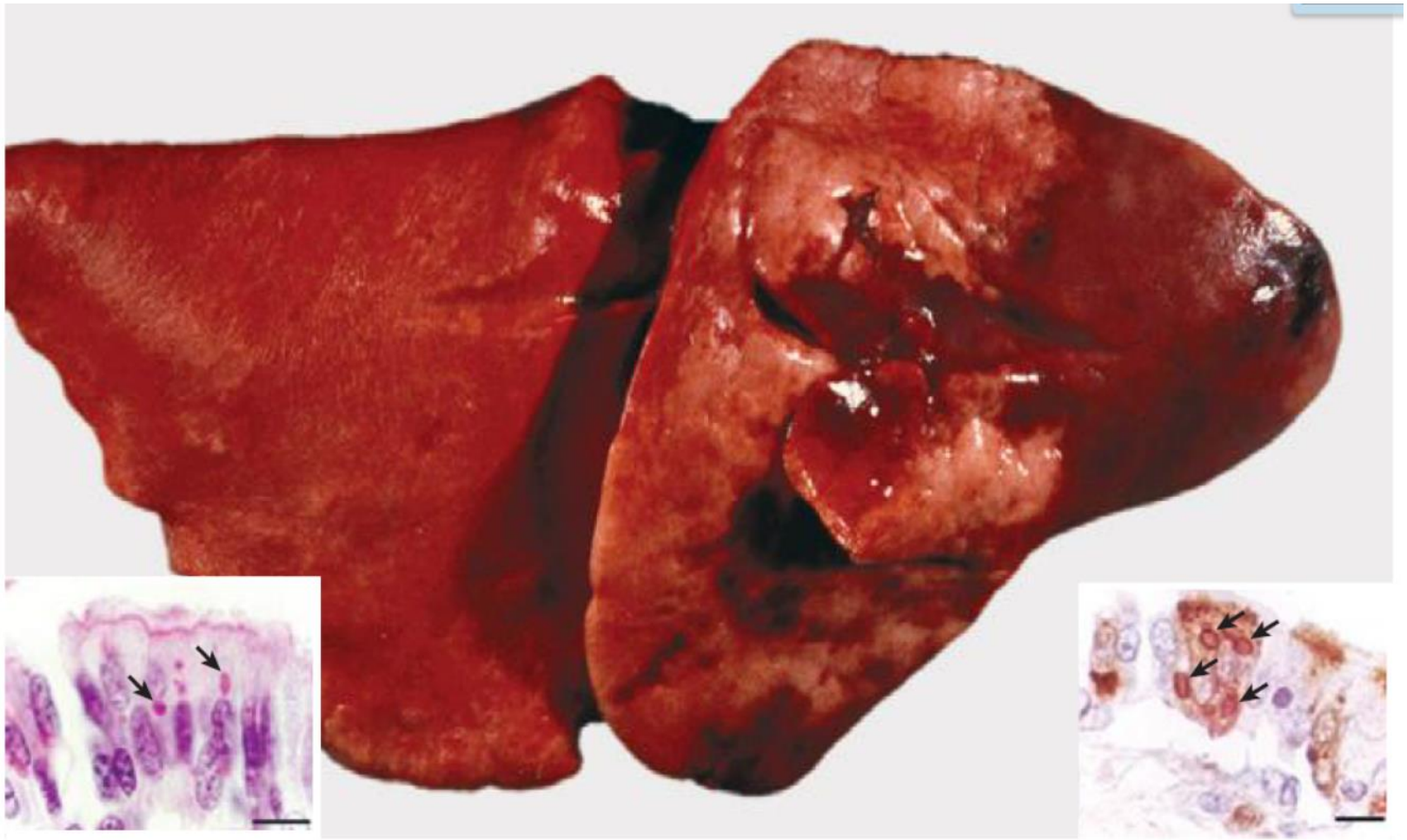
Gross lesions

- in the acute stages include **serous to catarrhal to mucopurulent nasopharyngitis** and **conjunctivitis.**

- **The lungs** are **edematous** and have a **diffuse interstitial pneumonia** (Fig. 9-87)

- Fig. 9-87 Interstitial pneumonia, canine distemper, lungs, dog.

The lungs are heavy, edematous, and rubbery, with costal (rib) imprints on the pleural surface.

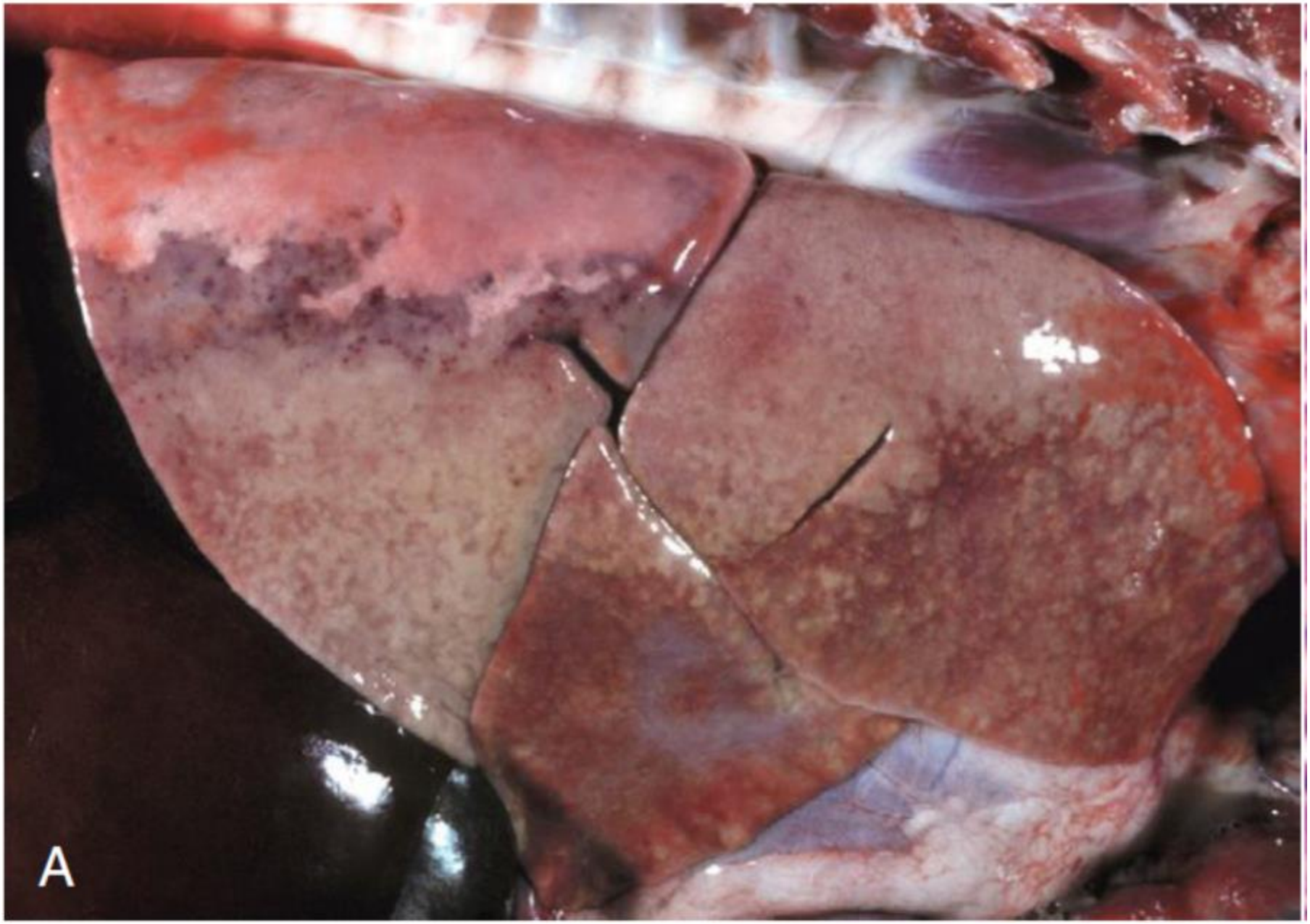


Gross lesion

- **Secondary infections** with **Bordetella bronchiseptica** and **mycoplasmas** are common and induce life-threatening **suppurative bronchopneumonia.**

- **Suppurative (purulent) inflammation, secondary bacterial bronchopneumonia, infectious canine distemper, puppy.**

A, The **cranioventral areas** of the lung are **firm** and **beige to brown**. This lesion is caused by **neutrophils transmigrating into alveoli** in an **acute inflammatory response** secondary to bacterial infection of the lung.


A

- **Nasal and/or digital hyperkeratoses** have a variety of underlying causes, including infectious disease (e.g., canine distemper

- **Retinitis** as the sole ocular lesion is rare. When it does occur, however, it is almost always in the course of neurotropic virus infections, such as canine distemper,

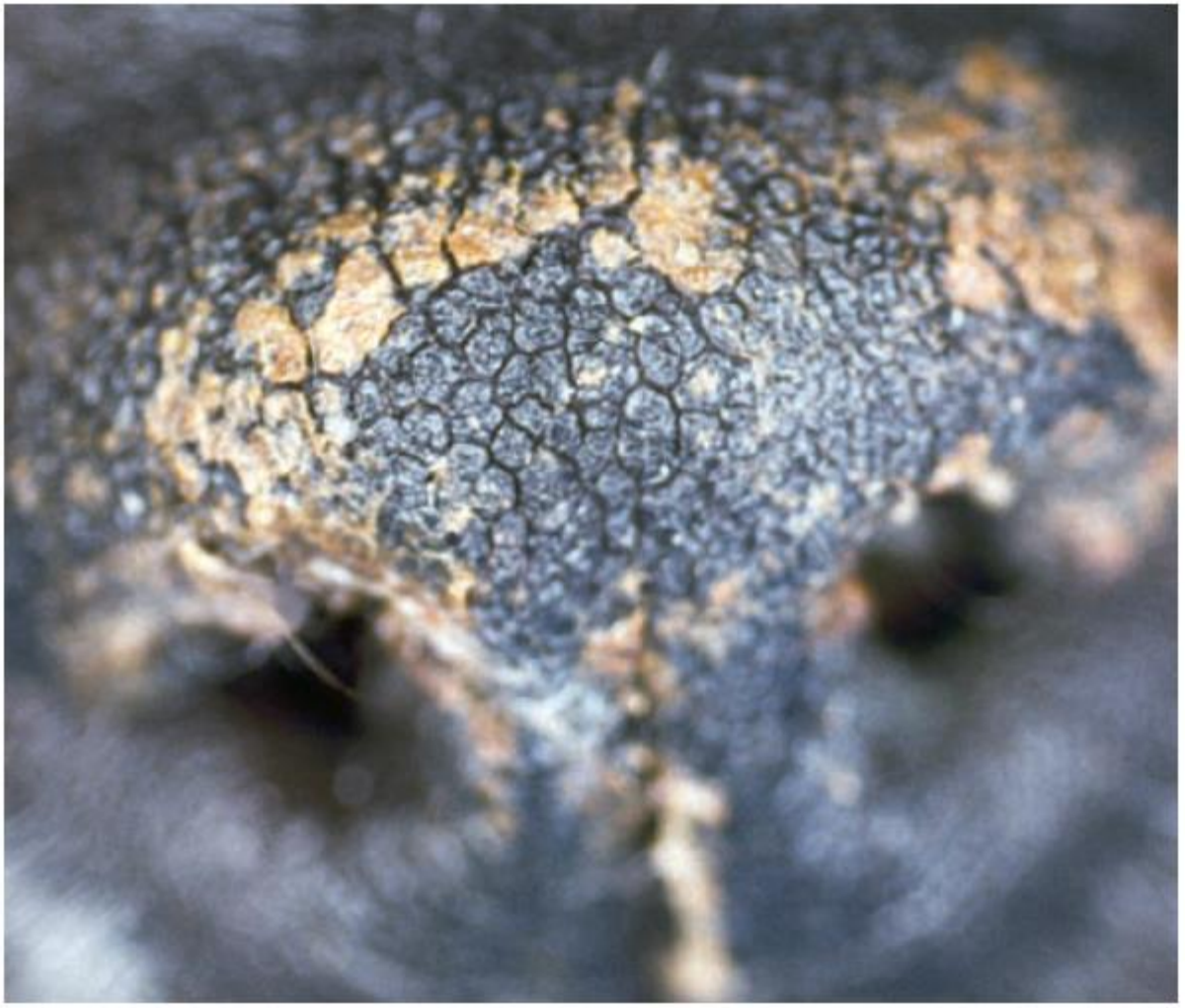


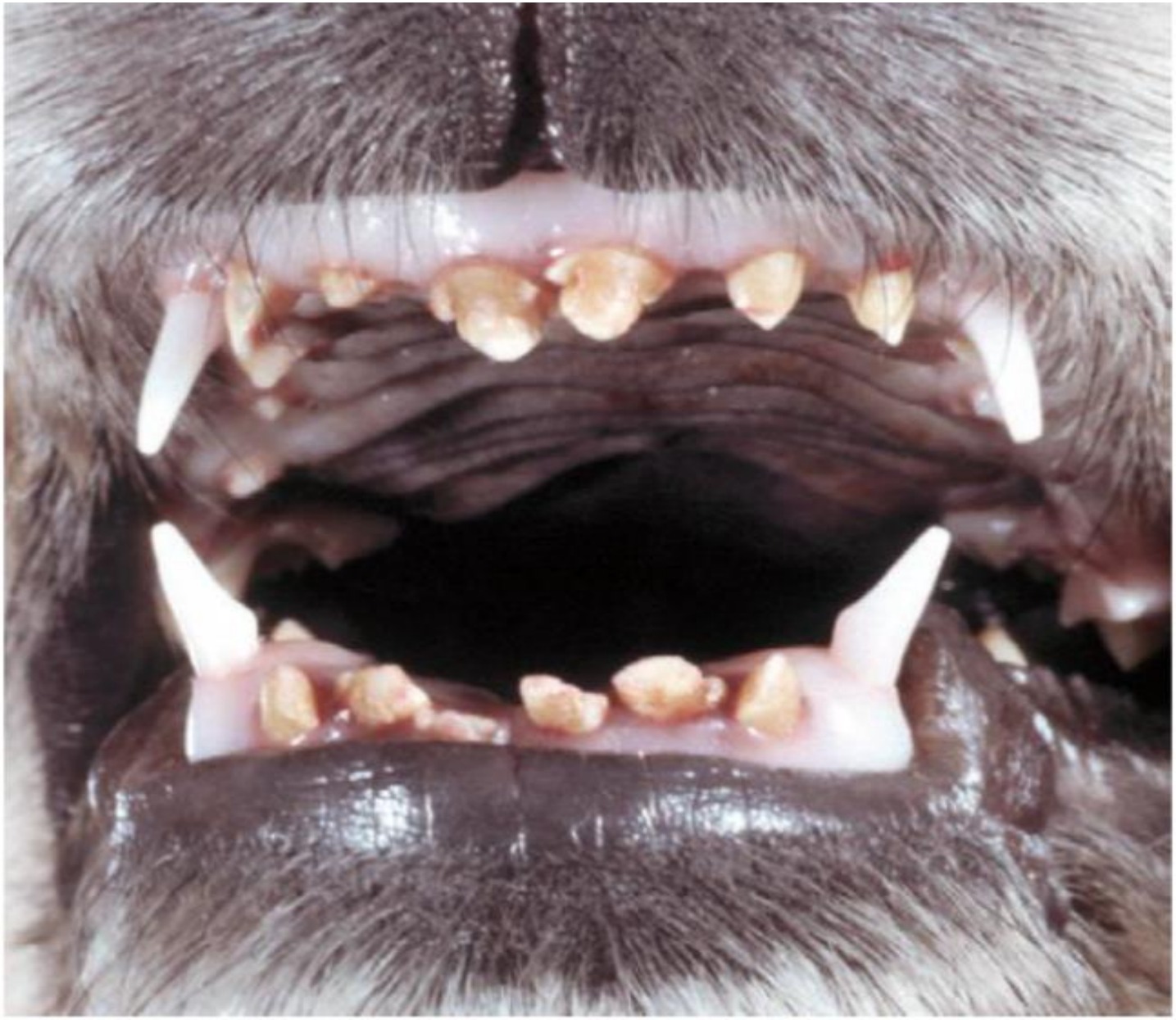
FIG. 3-4 Nasal hyperkeratosis in a dog with systemic distemper. (Photograph by

FIG. 3-5 Digital hyperkeratosis ("hard pads") in a dog dying of distemper encepha-

❑ Enamel hypoplasia, permanent incisor teeth, dog.

There is a **lack of enamel formation** with resultant **discrete deep pits and exposure of the dentin** (**light yellow to beige areas of the teeth**), the result of infection with canine distemper virus and **necrosis of the ameloblasts during enamel formation.**

- Permanent adult teeth (shown in illustration) are infected with virus before their eruption and while they are still within their sockets (dental alveoli).



ig. 7-17 Enamel hypoplasia, permanent incisor teeth, dog.

- The **thymus may be small relative** to the age of the animal because of viral-induced **lympholysis.**

Histopathology

- necrotizing bronchiolitis, necrosis and exfoliation of pneumonocytes, mild alveolar edema
- Bronchial epithelium contains intracytoplasmic eosinophilic inclusion bodies (arrows). H&E stain.

- Immunoperoxidase stain revealing **canine Morbillivirus antigen (arrows)** in the **cytoplasm** and apical borders of **bronchial epithelial cells.**

- **eosinophilic inclusions** are present in the epithelial cells of many tissues, **in the nuclei or cytoplasm, or in both**

.

They appear early in the **bronchiolar epithelium** but are most prominent in the epithelium of the **lung, stomach, renal pelvis, and urinary bladder**, making these tissues good choices for diagnostic examination.

- **Suppurative (purulent) inflammation, secondary bacterial bronchopneumonia, infectious canine distemper, puppy.**

Microscopically, **alveoli** contain **numerous neutrophils (suppurative exudate)** and **sloughed pneumocytes**. H&E stain.

(Courtesy Dr. M.D. McGavin, College of Veterinary Medicine, University of Tennessee.)

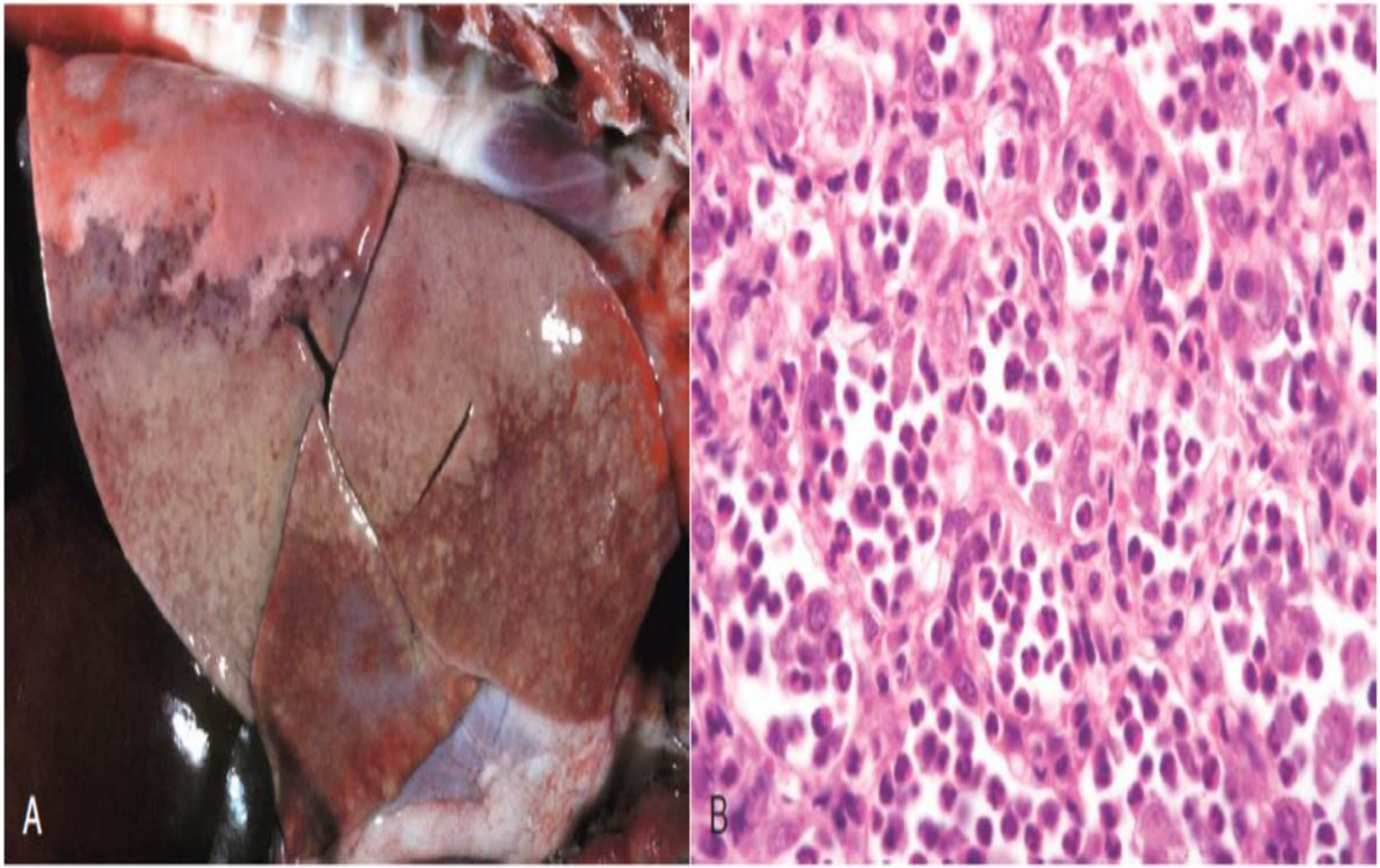


Fig. 3-19 **Suppurative (purulent) inflammation, secondary bacterial bronchopneumonia, infectious canine distemper, puppy.**

A, The cranioventral areas of the lung are firm and beige to brown. This lesion is caused by neutrophils transmigrating into alveoli in an acute inflammatory response secondary to bacterial infection of the lung. B, Microscopically, alveoli contain numerous neutrophils (suppurative exudate) and sloughed pneumocytes. H&E stain. (Courtesy Dr. M.D. McGavin, College of Veterinary Medicine, University of Tennessee.)

- The earliest evidence of **myelin injury** is a **ballooning change resulting from a split in the myelin sheath**, or more **degenerative changes including axonal swelling**.

- This lesion is also variably associated with **astroglial and microglial proliferation.**

- Microscopically, in addition to demyelination there is status spongiosus, astrocytic hypertrophy and hyperplasia with focal and variable syncytial cell formation, reduced numbers of oligodendroglia, and variable neuronal degeneration

- **A late stage of demyelination**, which is a reflection of an affected animal's improved immune status, is more pronounced and is characterized by **nonsuppurative inflammation (perivascular cuffing, leptomeningitis, and choroiditis)** and also can be accompanied by **tissue degeneration and accumulation of gitter cells**.

Sequelae to distemper

Sequelae to distemper include:

- ✓ the **nervous** and **pneumonic complications** and **various systemic infections**, such as **toxoplasmosis** and **sarcocystosis**, because of **depressed immunity.**

- ✓ **Persistent viral infection** occurs in some dogs that survive the disease, and they may **become carriers and the source of infection for other susceptible animals**