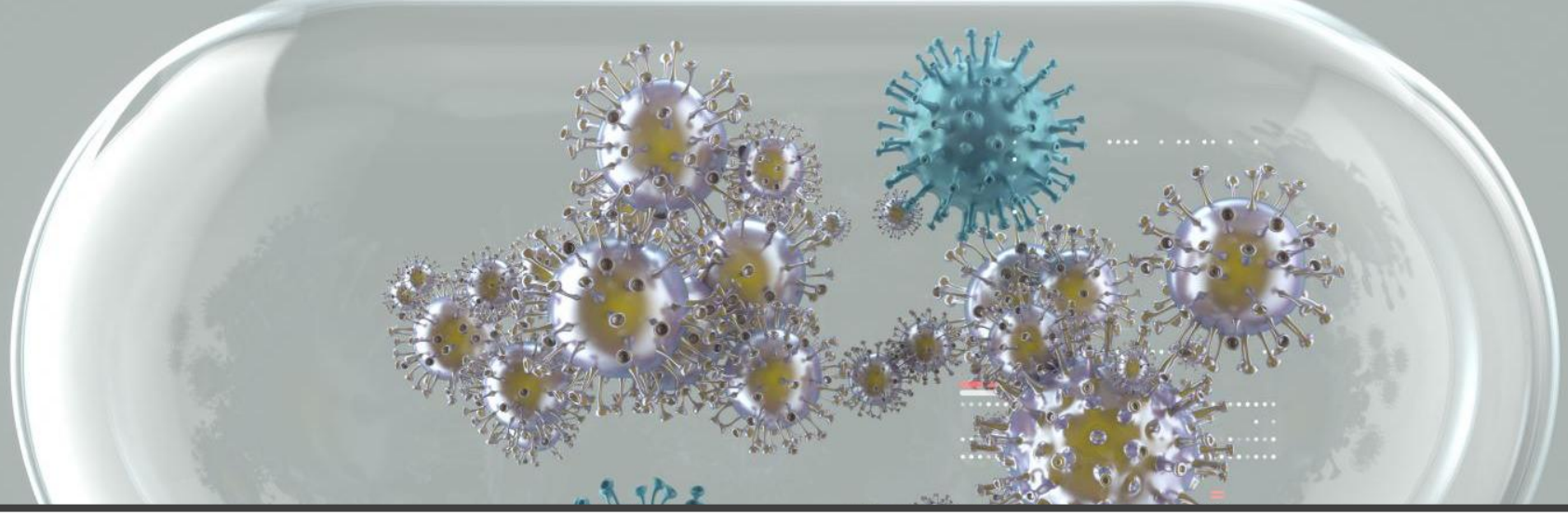


PNEUMONIA

WHAT IS PNEUMONIA?

- **Pneumonia (pneumonia, pneumonitis)** is an inflammation of the lungs.
  - **It may develop in:**
    - Alveoli (parenchyma)
    - Bronchioles and bronchi
    - Interstitial tissue
- **Pneumonia** is characterized by **exudation (fluid and cellular leakage)** or **cell proliferation (multiplication).**
- **Exudative pneumonia:** Occurs in the acute phase, where the inflammatory fluid (exudate) predominates.
- **Proliferative (interstitial) pneumonia:** In the chronic course, cellular proliferation predominates, and the inflammation is located within the interstitium.
- **Some cases also involve the pleura (lung membrane)** and are referred to as **pleuropneumonia.**

ETIOLOGY AND PATHOGENESIS

- **Pneumonia** is a **multifactorial (polyfactorial)** disease.
  - The lungs are constantly exposed to both the **external environment (airways)** and the **internal environment (blood and lymphatic vessels),** making them highly susceptible to infections.

INFECTION ROUTES

- **Aerogenous:** Agents inhaled through the respiratory tract
- **Hematogenous:** Agents disseminated via the bloodstream
- **Lymphogenous:** Agents transported through the lymphatic circulation
- **From adjacent tissues:** Spread of inflammation from neighboring structures

- **Main Causative Factors:**
- • **Physical:** Cold, humid air, dusty environment
  • **Chemical:** Irritant gases, toxins (for example, those ingested and reaching the lungs via the digestive tract)
  • **Allergic:** Pollens, fungal spores
  • **Infectious:** Viruses, bacteria, fungi, protozoa, helminths

ENVIRONMENTAL FACTORS

- **Cold and humid air:**
  - Weakens the defense mechanisms of the respiratory tract.
  - Ciliary (hair) movement stops, and mucus secretion decreases.
- **• Sudden temperature changes:**
  - Reduce resistance and increase susceptibility to infections.
  - Microorganisms that are normally harmless become active.
- **• Shelter conditions:**
  - Crowded environments → facilitate droplet infections.
  - Poor hygiene → ammonia accumulation irritates the respiratory tract.
  - Prolonged transport (by ship, train, etc.) → stress, poor nutrition, and inadequate ventilation promote infection.

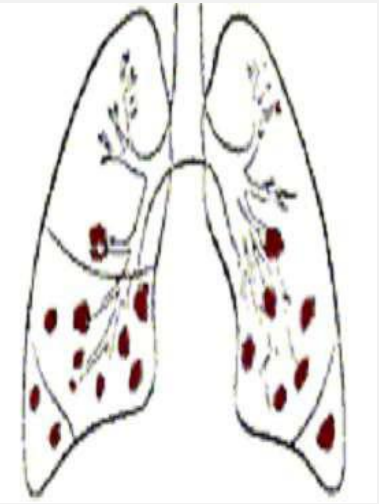
HOST-RELATED FACTORS

- **Host-Related Factors**
- Susceptibility or resistance to pneumonia depends on the **species**, **age**, and **general condition** of the host.
- • **In young animals:** The disease is common because the immune system is not fully developed.
  • **In aged animals:** Lung elasticity decreases, and defense mechanisms weaken.
- Additionally, the **type of infection** and the **characteristics of the causative agent** are decisive in pathogenesis:
  - Quantity of the agent and its toxin production
  - Route of entry (aerogenous, hematogenous, etc.)
  - Whether the infection is primary or secondary
- **Examples:**
  • **Canine distemper:** Causes primary interstitial pneumonia.
  • **Bordetella bronchiseptica infection:** Leads to secondary catarrhal bronchopneumonia.

PATTERNS OF INFLAMMATORY SPREAD IN THE LUNGS

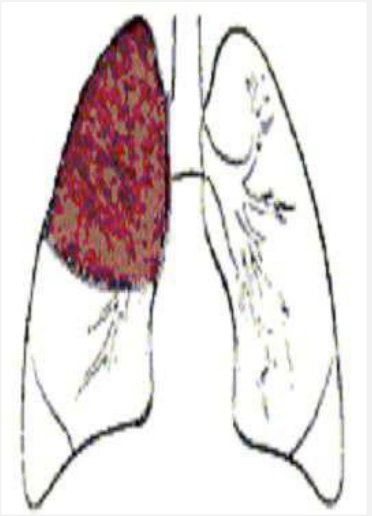
ENDOBRONCHIAL SPREAD

- Develops as a result of **bronchitis.**
- The **exudate**, through **aspiration**, reaches the bronchioles and alveoli at the termination of the bronchi.
- **Lobular pneumonia** develops:
  - The inflammation is **limited to lobules**, meaning it is discontinuous.
  - While one lobule is affected, the adjacent one may remain intact.
- If the bronchus becomes obstructed, the inflammation spreads **peribronchially** or **interalveolarly.**



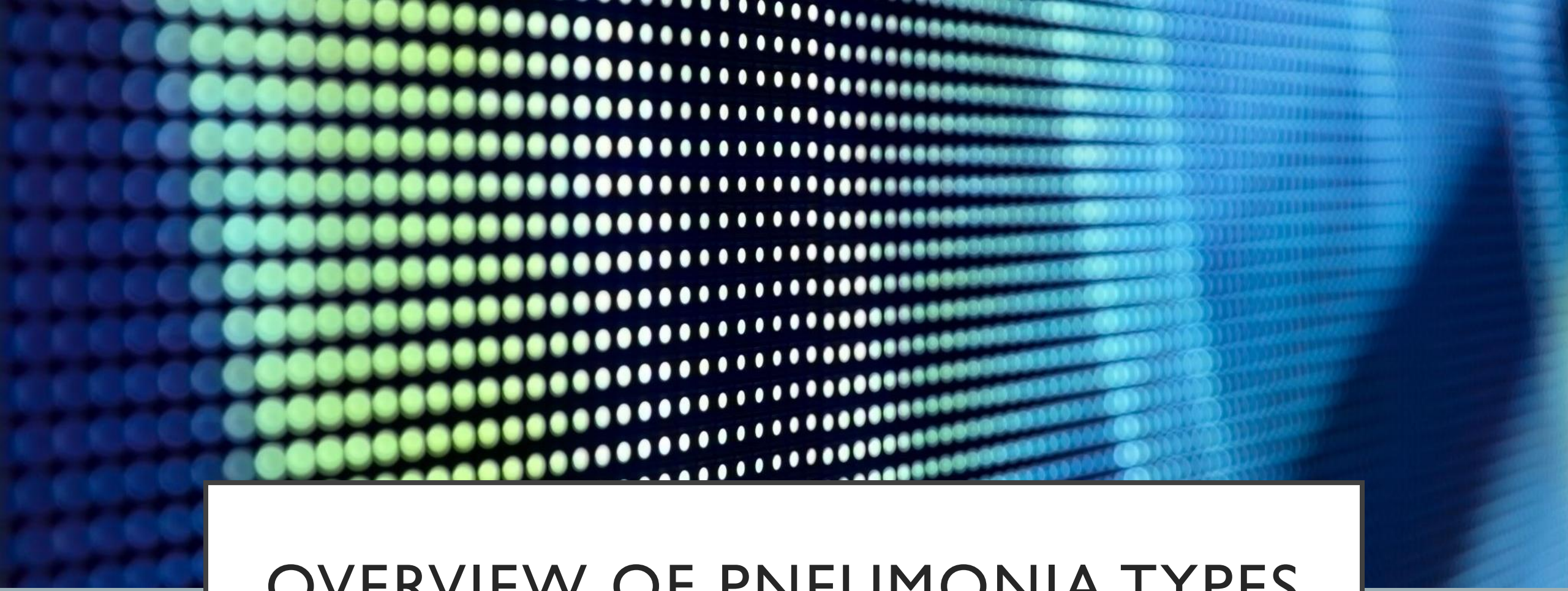
PERİBRONCHIAL SPREAD

- • The inflammation extends from the walls of the bronchi and bronchioles to the surrounding tissues, and subsequently to the alveoli connected to them.
  • During this process, **vascular thrombosis** and **obstruction of the bronchi by exudate** play a role.
  • Since the exudate cannot be expelled, the inflammation shows a **continuous (uninterrupted)** pattern of spread.



INTERALVEOLAR SPREAD

•Rarely observed.
• As a result of bronchial obstruction, the **exudate** passes from one alveolus to another through the **pores of Kohn.**
• In this way, the inflammation spreads to neighboring alveoli and bronchi.



OVERVIEW OF PNEUMONIA TYPES

Pneumonias can be classified in various ways based on their **course, distribution,** and **affected tissue regions.**

Main classification criteria:

- **According to course:**

Acute pneumonia: Sudden onset, rapidly developing, and usually **exudative** in nature.

Chronic pneumonia: Develops over a long period; characterized by **cellular proliferation** or **fibrosis.**

- **According to distribution:**

Lobular pneumonia:

Inflammation is confined to one or more **lobules** within a lobe.

Shows a **discontinuous (multifocal)** distribution.

Lobar pneumonia:

Inflammation involves the **entire lobe** or several lobes.

Forms a **large, single-focus** area of inflammation.

Historical Terminology: Distinction Between "Pneumonia" and "Pneumonitis"

• In the past, pneumonias were named according to the **histological region** where the inflammation was located:

• **Pneumonia:** Inflammation involving the **alveoli, bronchi,** and **bronchioles.**

• **Pneumonitis:** Inflammation in the **perivascular, peribronchial,** or **interlobular (between lobules)** areas — that is, within the **interstitium.**

• Later, this distinction was abandoned, and the term **"pneumonia"** came to be accepted as a **general term** referring to inflammation of the lungs.

CURRENT CLASSIFICATION OF PNEUMONIAS

- **Alveolar or broncho-alveolar pneumonias**
- **Interstitial pneumonias**
- **Special forms of pneumonia** (focal or diffuse specific types)
- **Granulomatous pneumonias** (determinant/characteristic types)
- **Pneumoconioses** (dust-induced pneumonias)
- **Allergic pneumonias**

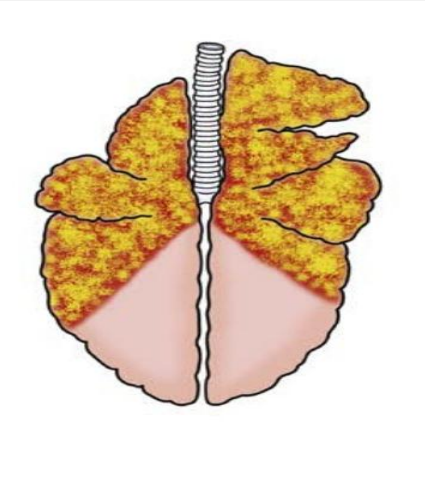
I. Alveolar / Broncho-Alveolar Pneumonias

a) Catarrhal Bronchopneumonia (Lobular Pneumonia)

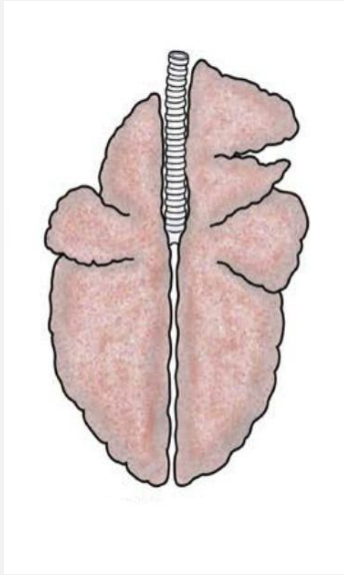
- Usually develops as a result of **secondary infections.**
- The inflammation is **limited to a few lobules.**
- **Morphological subtypes:**
  - Serous
  - Seromucous
  - Desquamative
  - Purulent (suppurative)

b) Fibrinous Bronchopneumonia (Lobar Pneumonia)

- The inflammation involves the **entire lobe.**
- **Fibrin accumulation** and **pleural involvement** are typical.
- It is **acute in course, severe,** and often of **bacterial origin.**



II. Interstitial Pneumonia



••The inflammation develops in the **connective tissue between alveoli (interstitium).**
- **Cellular proliferation** predominates, while **exudate is minimal.**
- It is usually caused by **viral** or **toxic agents.**
- May occur in **acute or chronic** forms.

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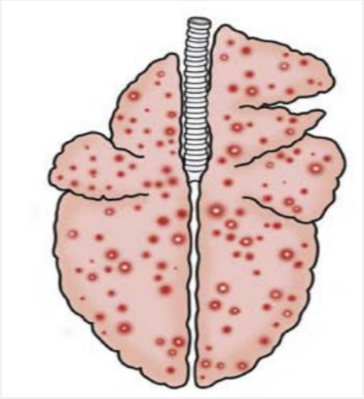
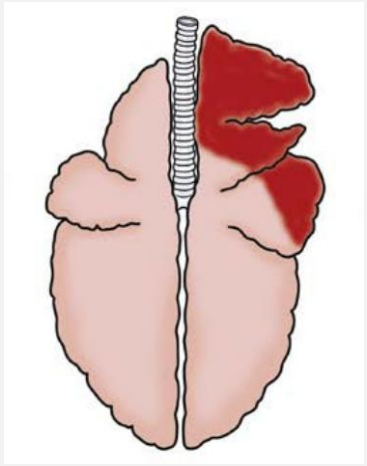
III. Special Forms of Pneumonia



Some pneumonias are given **specific names** based on their **causes** or **morphological characteristics:**

- **Embolic / Metastatic Pneumonias:**
  – Caused by microorganisms carried through the bloodstream.
  – Lead to **purulent (suppurative)** or **abscess-forming (apostematous)** lesions.

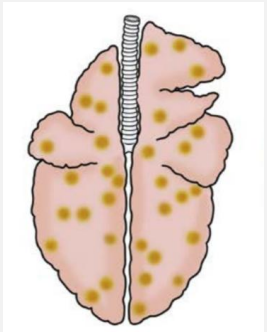


- **Aspiration Pneumonia:**
  – Occurs as a result of aspiration of foreign material (e.g., feed, gastric contents, oil).
  – May progress as **gangrenous, necrotic,** or **lipid pneumonia.**

IV. Granulomatous (Determinant) Pneumonias

Characterized by the formation of **granulomas** in the lung parenchyma.

- Usually develop in response to **chronic infections** (e.g., mycobacteria, fungi) or **foreign bodies.**
- Lesions often contain **macrophages, epithelioid cells, giant cells,** and sometimes **caseous necrosis.**
- Serve as **diagnostic (determinant)** forms due to their distinctive morphology.



Zachary, J. F., & McGavin, M. D. (Eds.). (2012). Pathologic Basis of Veterinary Disease5: Pathologic Basis of Veterinary Disease. Elsevier Health Sciences.

V. Pneumoconioses

••Chronic, **non-infectious pneumonias** that develop as a result of **inhalation of dust or particulate matter.**

- Examples include accumulations of:
  - **Coal dust (anthracosis)**
  - **Silica (silicosis)**
  - **Asbestos (asbestosis)**

VI. Allergic Pneumonias

•Develop as a result of **hypersensitivity reactions** to allergens such as **pollen, fungi,** or **organic dusts.**

- Usually **interstitial** in nature, with prominent **eosinophilic infiltration.**
- When chronic, it may lead to **alveolar fibrosis.**

BRONCHOPNEUMONIA CATARRHALIS

• Catarrhal bronchopneumonia spreads **endobronchially.**

• The inflammation is **localized** in the bronchioles and alveoli at the termination of one or several bronchi.

• The distribution is **discontinuous (lobular);** the lobules surrounding the affected ones remain normal.

• Initially, **serous exudate (inflammatory edema fluid)** accumulates in the alveoli and bronchi.

• Over time, **neutrophils** and **desquamated epithelial cells** increase within the exudate, which mixes with mucus from bronchial glands to form **mucous or mucopurulent exudate.**

• It is usually **localized in the cranioventral lobes (anterior-ventral regions).**

• The cut surface appears **granular,** and its color varies from **red to yellow-gray.**

• Exudate is also found within the **bronchi and bronchioles.**

• Areas of **normal, atelectatic,** or **emphysematous** tissue may be present between pneumonia foci.

• **Pleuritis** (pleural inflammation) is **generally absent.**

Approximately **80% of pneumonias in animals** are of this type.

It occurs **mainly in young animals** (lambs, calves, kids, piglets).

ETIOLOGY

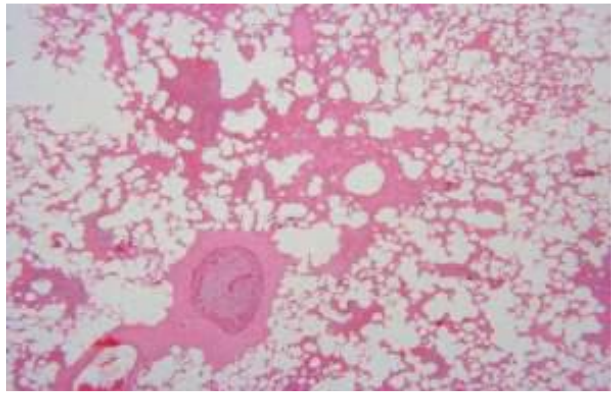
- **Bacterial Agents**
  - *Streptococcus spp.*, *Staphylococcus spp.*
  - *Escherichia coli*
  - *Pasteurella multocida*
  - *Bordetella bronchiseptica* (especially as a secondary infection following distemper in dogs)
  - *Pneumococcus sp.*, *Salmonella sp.*, *Brucella sp.* (particularly in calves)
  - *Corynebacterium pyogenes* (sheep, pigs)
  - *Corynebacterium equi* (foals; especially following adenovirus infection)
  - *Chlamydia sp.*, *Haemophilus suis*, *Salmonella choleraesuis* (in pigs)
- **Note:** Most cases develop as **secondary bacterial infections** following **viral interstitial pneumonias.**

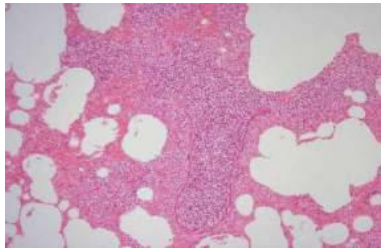
PATHOGENESIS

- Most commonly occurs due to **aerogenous infection** (via the respiratory tract).
- Pathogens reach the **bronchi and alveoli** from the **upper respiratory tract.**
- Occasionally, **hematogenous spread** may occur (e.g., through umbilical cord infection — *omphalogenous spread*).
- In such cases, **metastatic abscess-forming (purulent) pneumonia** develops.

MACROSCOPICAL FINDINGS

•The distribution is **lobular**, mostly confined to the **cranioventral lobes.**
• There is a **slight increase in volume** and a **marked increase in consistency** (the tissue feels firm).
• **Color:**
– Initially **red**,
– Turns **yellow-gray** when suppuration develops,
– Becomes **grayish-gray** in necrotic areas.
• **Cut surface:** Moist; when pressed, a **cloudy, viscous exudate** flows from the bronchial lumina.
• **Between the pneumonia foci**, areas of **atelectasis or emphysema** may be present.





HISTOPATHOLOGICAL FİNDİNGS

• The **alveolar and bronchial lumina** contain **proteinaceous inflammatory fluid (edema)**, **desquamated epithelial cells**, and **neutrophil leukocytes.**

• **Capillaries are hyperemic** (engorged with blood).

• Occasionally, **small amounts of fibrin** may also be present.

I. Bronchopneumonia catarrhalis serosa

- Represents the **initial stage** of inflammation.
- **Inflammatory edema fluid** predominates.
- **Proteinaceous pink-staining fluid** is observed in alveoli and bronchi.
- The exudate contains **few neutrophils** and **desquamated epithelial cells.**
- If mixed with mucus, it takes on a **slightly bluish tint.**

→ Also referred to as *Bronchopneumonia catarrhalis mucosa.*

II. Bronchopneumonia catarrhalis desquamativa

- The exudate is dominated by **desquamated epithelial cells.**
- Typically seen as a **secondary bacterial infection** following **viral interstitial pneumonia.**
- Epithelial desquamation may also occur due to **hypoxia.**

Histological Subtypes (3 Types):

III. Bronchopneumonia catarrhalis purulenta

- From the onset, **neutrophil leukocytes** predominate.
- Develops due to **pyogenic (pus-forming) bacteria.**
- Alveoli and bronchioles contain **mucus, edema, desquamated epithelium, and abundant neutrophils.**
- In severe cases, the exudate contains **small amounts of fibrin.**
- The **interstitial tissue** shows **edema, neutrophilic infiltration, and hyperemia.**

Healing:

- The **exudate is eliminated** through the bronchi or **resorbed by the blood vessels.**
- The **damaged epithelial layers regenerate.**

Chronic Progression:

- Develops into **chronic catarrhal pneumonia.**
- Due to **peribronchitis / peribronchiolitis**, **fibrous tissue proliferation** and **bronchial obstruction** occur.
- **Mucous exudate accumulates** in the bronchial lumen.
- On the lung section, **small nodules** (pinpoint-sized cavities) can be observed.
- **Fibrosis develops** in the interstitial tissue → **indurative pneumonia.**

Necrotic Outcome:

- If **small bronchi are obstructed**, **hypoxia and necrosis** occur → **necrotic pneumonia.**
- If necrotic areas heal, **granulation tissue forms** → **carnification** → **scarring (fibrosis).**
- Progressive fibrosis may lead to **cor pulmonale** (right heart failure).

DISEASES WITH CATARRHAL PNEUMONIA

Catarrhal→Purulent bronchopneumonia

GENERAL INFORMATION

- **Catarrhal or suppurative pneumonias are usually bacterial in origin.**
- These infections often develop under **predisposing conditions** such as viral infections, malnutrition, or poor management.
- **Opportunistic bacteria** are usually responsible.
- **Pyogenic (pus-forming) bacteria:**
  Streptococcus, Staphylococcus, Corynebacterium pyogenes, Pseudomonas aeruginosa
  → usually cause **suppurative (purulent) or abscess-forming pneumonia.**
- Depending on bacterial virulence and host resistance, **necrotic or hemorrhagic pneumonia** may also occur.

SALMONELLA SPECIES

•*S. choleraesuis* → causes **fibrinonecrotic or hemorrhagic pneumonia** in pigs.
•*S. typhisuis* → produces **chronic suppurative bronchopneumonia** with focal necrosis in pigs.
•*E. coli, Streptococcus, Actinobacillus (Shigella) equuli* → cause **septicemia, pyemia, and multifocal purulent–necrotic pneumonia** in young animals.
•These bacteria may coexist with **interstitial pneumonia agents.**

STREPTOCOCCUS PNEUMONIAE (PNEUMOCOCCAL PNEUMONIA)

- Occurs especially in **calves (3–4 weeks old)** during winter.
- **Anthropozoonosis:** can be transmitted from humans.
- **Clinical signs:** fever, dyspnea, septicemic findings.
- **Pathology:**
- **Catarrhal–purulent bronchopneumonia** in apical and cardiac lobes,
- **Rubber-like spleen** (splenic hyperplasia),
- **Serofibrinous arthritis** and **endocarditis.**
- **Differential diagnosis:** *E. coli* septicemia.

CORYNEBACTERIUM (RHODOCOCCUS) EQUI

•articularly important in **foals aged 1–6 months.**
•Causes **subacute or chronic** infections.
•**Lesions:**
•**Abscesses, catarrhal–purulent bronchopneumonia, and pyogranulomatous nodules** in the lungs,
•**Abscesses in the liver and spleen**, **ulcerative enterocolitis** may occur.
•**In other species:**
•Pneumonia in calves,
•Tuberculosis-like lymph node lesions in pigs.

BORDETELLOSIS (*BORDETELLA BRONCHISEPTICA*)

•Seen in dogs, cats, rabbits, pigs, foals, and guinea pigs.
•The bacterium attaches to **ciliated epithelium** and produces toxins.
•**In dogs:** causes **secondary catarrhal/purulent bronchopneumonia** after distemper.
•**In pigs:** causes **atrophic rhinitis**;
•in young pigs → **septicemia and bronchopneumonia**,
•in older pigs → **purulent bronchopneumonia.**
•**Pathology:** peribronchial fibrosis, endothelial damage, and congestion.

SALMONELLOSIS (*SALMONELLA* SPP.)

•Produces **fibrinous, necrotic, or hemorrhagic enteritis and pneumonia** in many species.
•Especially *S. choleraesuis* → causes **septicemic hemorrhage and pulmonary bleeding.**
•**Complication:** mycotic pneumonia may develop.

GENERAL OUTCOME

•Catarrhal or suppurative pneumonias are mostly **secondary bacterial infections.**
•They develop following **primary viral injury** or **immune suppression.**
•In chronic cases, **fibrosis, abscess formation, pleuritis, and emphysema** may occur.

PNEUMONIA FIBRINOSA (LOBAR PNEUMONİA)

GENERAL CHARACTERISTICS

- The **exudate is rich in fibrin.**
- The inflammation shows a **peribronchial spread pattern**, therefore it has a **lobar distribution** — involving a large portion or the entirety of one or more lobes.
- **Almost always accompanied by pleuritis (inflammation of the pleura).**
- The process includes **multiple stages**, but these are **not sequential** — instead, they appear **side by side within the lung**, creating a **mosaic appearance.**

→ As a result, the **lung section shows a mottled or marble-like appearance.**

ETIOLOGY

Main Bacterial Agents:

- *Mycoplasma* sp.
- *Pasteurella multocida*
- *Hemophilus* sp.

Example Diseases by Species:

- **Cattle:** "Contagious bovine pleuropneumonia"
- **Goat:** *Pleuropneumonia contagiosa capri*
- **Horse:** *Pleuropneumonia contagiosa equorum*

PATHOGENESIS (COURSE AND STAGES)

I. Inflammatory Hyperemia (Stadium incrementi)

Macroscopic Findings:

The lungs are enlarged and dark red in color.

On cut surface, a reddish fluid oozes out.

Microscopic Findings:

Capillaries are dilated and filled with blood (**hyperemia**).

In the alveolar and bronchial lumens:

- Slight serous (edematous) exudate
- A few desquamated epithelial cells
- Erythrocytes
- Scattered neutrophils

II. Red Hepatization Stage

Macroscopic Findings:

The lungs are enlarged.

The color is dark red and the consistency resembles that of the liver (**"hepatization"**).

A reddish exudate oozes from the cut surface.

Interstitial areas are widened due to inflammatory edema.

The pleura appears dull and is covered with petechial (pinpoint) hemorrhages.

Microscopic Findings:

The vessels show marked **hyperemia**.

In the alveolar and bronchial lumens:

- Desquamated epithelial cells
- Erythrocytes
- A few neutrophil leukocytes
- Fibrin strands (pink, thread-like structures)

In the interstitial tissue:

- Edema
- Fibrin deposition
- Granulocytic infiltration
- Occasional vascular thrombosis

- At this stage, the lung is firm, liver-like in consistency, and dark red in color.

III. Gray Hepatization Stage

Macroscopic Findings:

The lungs are swollen, enlarged, and have a **grayish-white color** with a **dry cut surface**.

The consistency remains firm, similar to that of the liver.

Microscopic Findings:

The alveoli are packed with **numerous neutrophil leukocytes**.

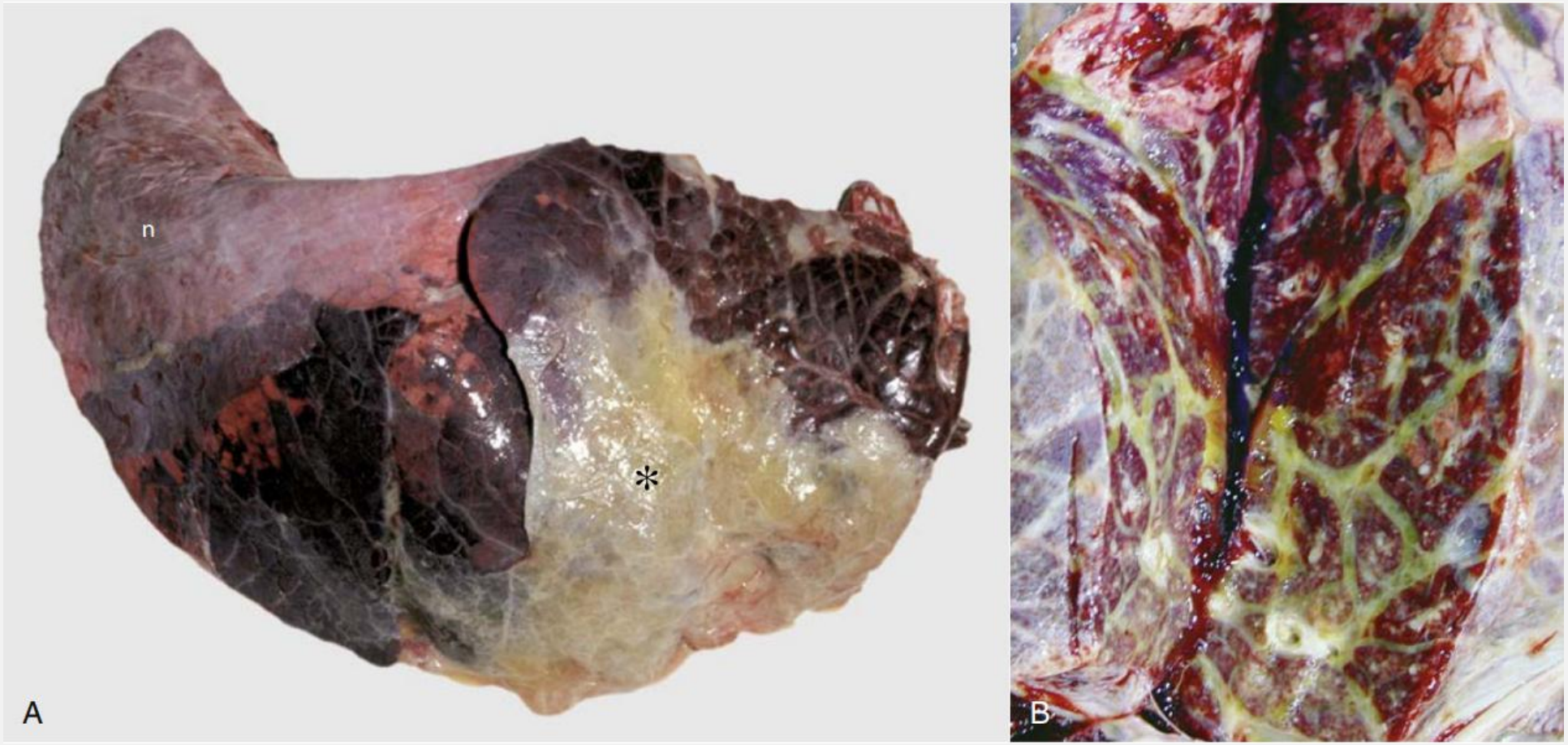
Only a few epithelial cells remain.

Due to the high cell density, **vascular ischemia** develops.

On necropsy, these areas appear **gray-yellow and hepatized**.

The interstitial tissue shows marked **edema, fibrin accumulation, neutrophilic infiltration,** and **thrombosis**.

IV. Lysis (Resolution) Stage
Macroscopic Findings:
The appearance resembles that of the **hyperemic stage**.
The color is **dark red**, and the consistency becomes **softer**.
On the cut surface, a **cloudy, grayish fluid** oozes out.
This stage is **more prominent in humans** than in animals.



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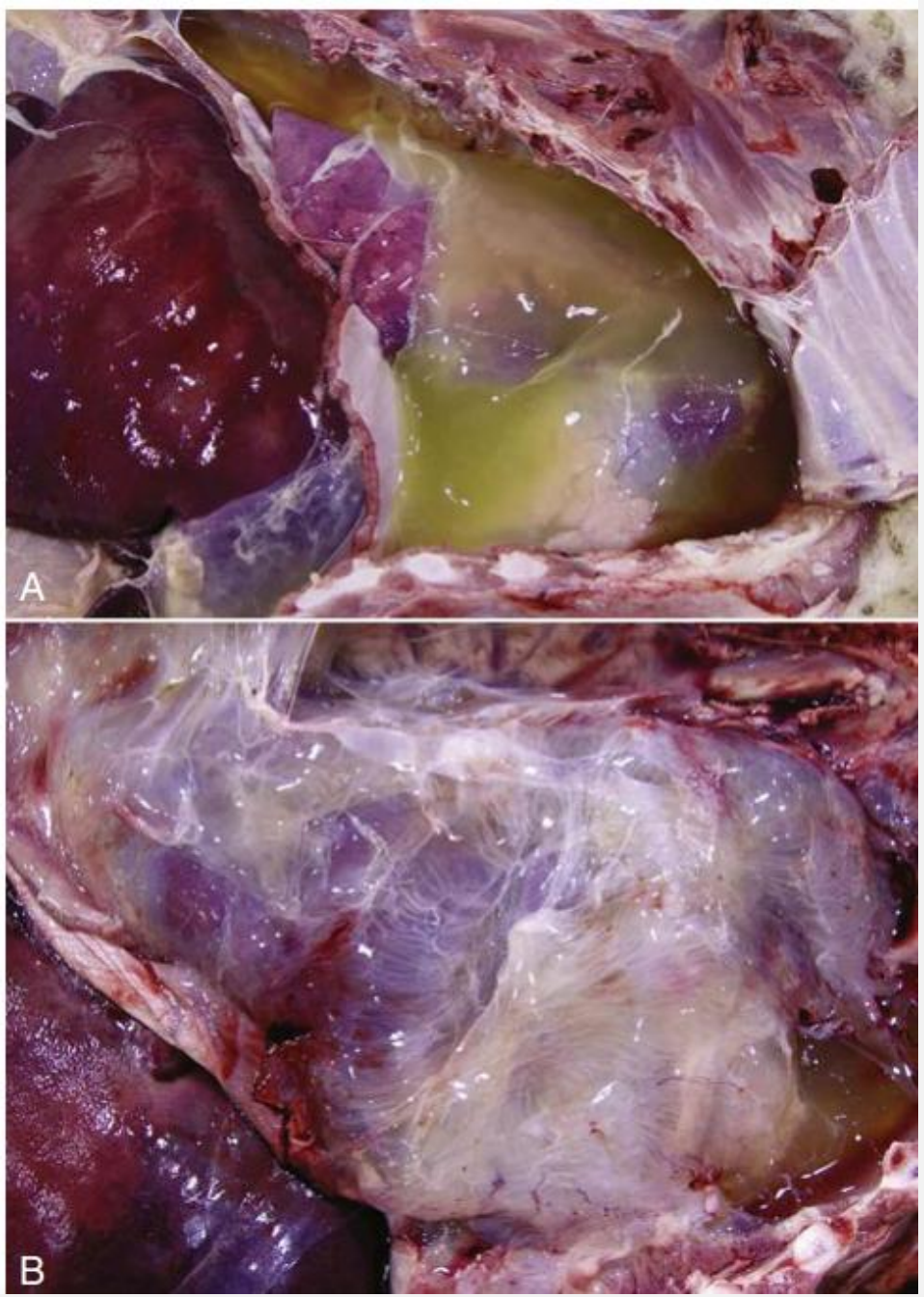

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(OUTCOMES)

- **Healing (Resolution):**

More commonly observed in humans.
Fibrin dissolves, and thrombi are resolved.
Exudate is **resorbed** through lymphatic or blood vessels, or expelled via the bronchi.
The epithelial lining is **renewed (regeneration)**.

- **In Animals (Complicated Course):**

Thrombi usually do not dissolve, and the exudate cannot be expelled → **necrosis develops**.
Necrotic areas appear **grayish-brown, soft**, leading to **necrotic-fibrinous pneumonia**.

Possible Developments After Necrosis

- **Small Necrotic Areas:**

Undergo **organization**, forming **granulation tissue**.
The tissue becomes **meaty in consistency → carnification**.
Over time, **fibrous tissue increases**, leading to **sclerosis** and **induration**.

- **Extensive Necrotic Areas:**

Sequestration: The necrotic area separates from viable tissue.
Gangrenous Pneumonia: Putrefactive, foul-smelling necrosis develops.
Abscesses (Pneumonia apostematosa): Pus accumulates within the necrotic focus; sometimes it ruptures into bronchi, leading to **cavity (cavern) formation**.

COMPLICATIONS

- **Pleural Empyema (Purulent Pleuritis):**

Occurs when a necrotic area ruptures into the pleural cavity.

- **Fibrinous Pleuritis:**

The **most common complication** of fibrinous pneumonia.

The causative agent spreads to the pleura via **peribronchial lymphatic vessels**.

DISEASES ASSOCIATED WITH FIBRINOUS PNEUMONIA

MYCOPLASMA SPP. INFECTİONS

- ◆ **General Characteristics:**
- *Mycoplasma* species have a **wide host spectrum** — they cause **pneumonia** and other **systemic diseases** in goats, cattle, sheep, pigs, horses, dogs, cats, rats, and humans.
- ◆ **Transmission:**
- Typically **aerogenous (airborne)**; spreads easily under **stable or crowded conditions**.
- Animals that recover from the disease may remain **carriers** and continue to spread the infection.

CONTAGIOUS BOVINE PLEUROPNEUMONIA (CBPP / "CATTLE LUNG PAIN")

- **Causative Agent:** *Mycoplasma mycoides* (various subspecies)
- **Distribution:** Common in **Asia**, **Africa**, and **Eastern Europe**.

Species such as **zebu, bison, yak,** and **camel** are susceptible.
Experimentally, the disease can also infect **sheep** and **goats**.

PATHOGENESIS

- The pathogen reaches the lungs **via the aerogenous route**.
- It first spreads **endobronchially**, then **peribronchially**, eventually extending to the **pleura**.
- **Acute vasculitis** develops, leading to **thrombosis**, **necrosis**, and **fibrinous exudation**.
- This vasculitis results from an **antigen–antibody (hypersensitivity) reaction** — typically **Arthus-type** or **mixed-type**.
- The disease may become **chronic** in cases of **reinfection** or **immunosuppression**.

CLINICAL FINDINGS

- **Incubation period:** Up to **1 month**.
- **Mortality rate: 10–70%**.
- Signs include **fever**, **depression**, **initially dry then painful cough**, and **respiratory distress**.
- Animals that recover develop **long-term immunity** lasting **over 2 years**.

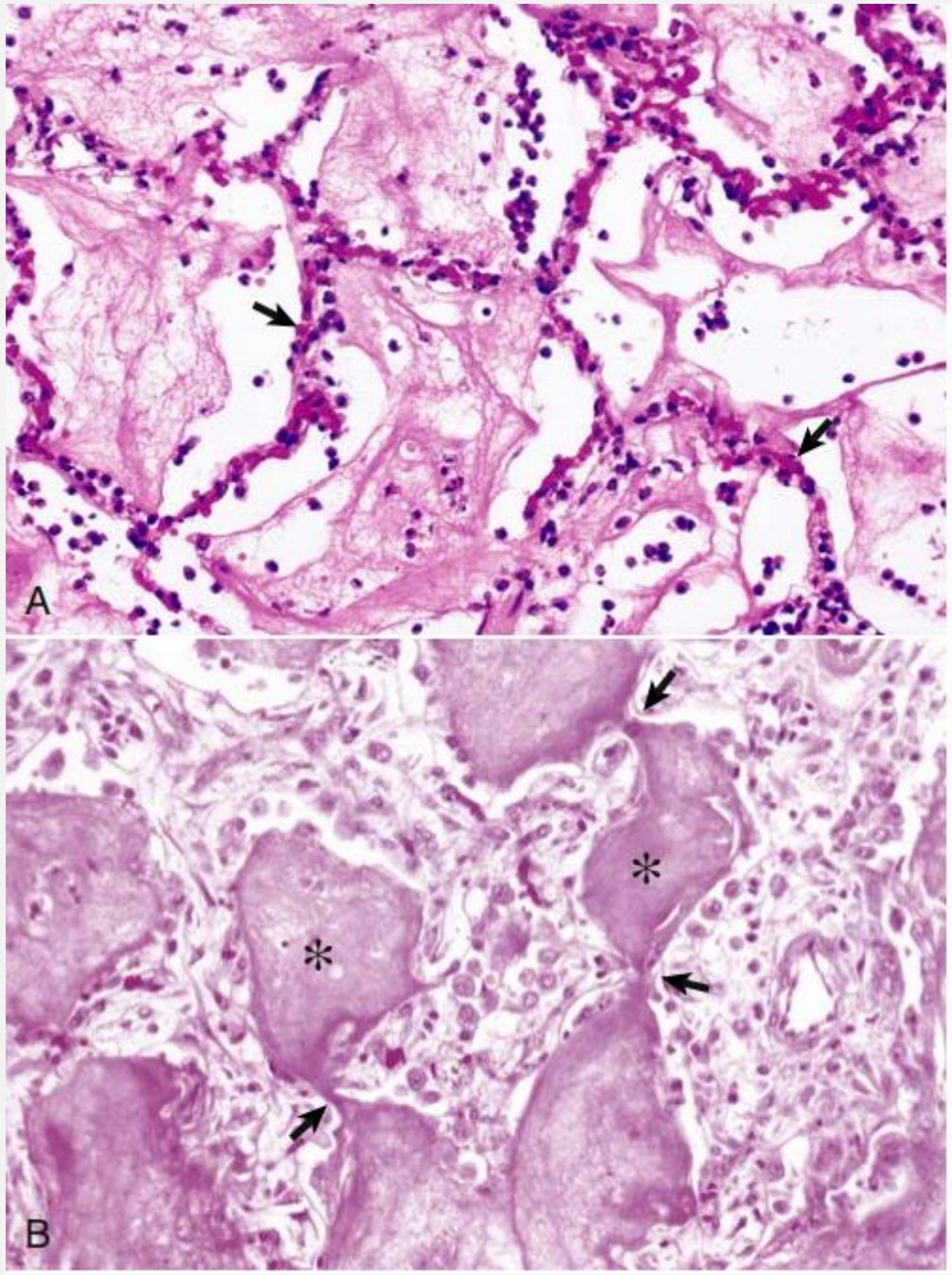
MACROSCOPICAL FINDINGS

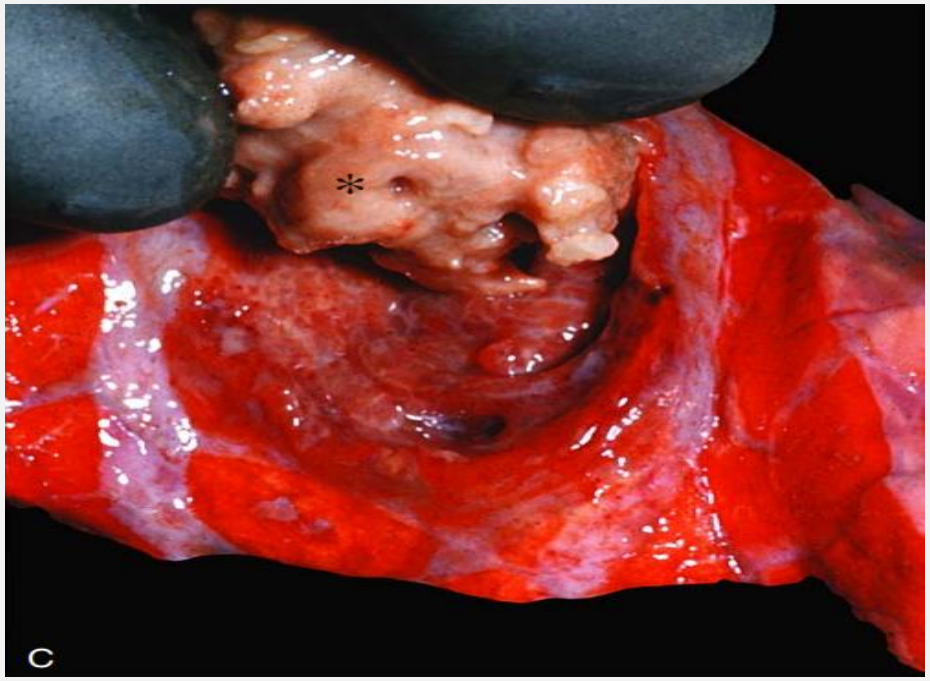
•Initially, numerous **small foci** form, which gradually expand to involve an entire **lobule or lobe**.
•The condition is **always complicated by fibrinous pleuritis**.
•**Pleura:** Thickened and covered by a **yellow-gray fibrinous layer**, with **adhesions** forming between pleural surfaces.
•**Thrombosis** leads to extensive **yellow-gray necrotic areas**.
•During healing, **carnification** and especially **large sequestra formation** are characteristic.

Zachary, J. F., & McGavin, M. D. (Eds.). (2012). Pathologic Basis of Veterinary Disease5: Pathologic Basis of Veterinary Disease. Elsevier Health Sciences.

MICROSCOPIC FINDINGS

- Exhibits a **typical fibrinous (croupous) pneumonia** pattern.
- **Severe vascular thrombosis** leading to **extensive necrosis**.
- During the healing stage, **peribronchial** and **perivascular organization** becomes a **distinct diagnostic feature**.





Zachary, J. F., & McGavin, M. D. (Eds.). (2012). Pathologic Basis of Veterinary Disease5: Pathologic Basis of Veterinary Disease. Elsevier Health Sciences.

***Mycoplasma mycoides* ssp. *Mycoides*, Sığır.**

A, Bakteri başlangıçta kan-hava bariyerindeki kapillerlerde *(oklar)* şiddetli bir vaskülitise neden olur ve bunun sonucunda plazma proteinleri alveollere sızar. Bakteriyel toksinler ve diğer faktörler, pulmoner parankimde koagülasyon nekrozuna yol açar.

B, Zamanla fibrin birikimi *(yıldızlar)* ve koagülasyon nekrozu ağırlaşarak bakterilerin fibrinli pıhtı içinde izole olmasına neden olur. Bakteriler, Kohn porları *(oklar)* aracılığıyla komşu alveollere yayılabilir. Alveoler septalar ödemlidir ve inflamatuvar hücreler ile reaktif pnömositlerle doludur.

C, Majör bir pulmoner damarın trombozu veya bakterilerin salgıladığı nekrotizan toksinlerin etkisiyle oluşan pulmoner sekestralar *(yıldız),* zamanla fibröz bağ dokusu ile çevrelenir

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

EXTRAPULMONARY FINDINGS

- **In young animals:** Polyarthritis
- **In pregnant animals:** Abortion
- **Liver:** Mononuclear infiltration in portal areas and anoxic necrosis
- **Spleen:** Enlarged germinal centers and increased plasma cell density

DIFFERENTIAL DIAGNOSIS

- May be confused with **Pasteurellosis**, but:
- **Thrombosis and necrosis** are much **more extensive** in the early stages.
- **Sequestrum formation** is **more pronounced**.

ENZOOTIC PNEUMONIA (IN CALVES AND YOUNG CATTLE)

•In **calves**, enzootic pneumonia cases manifest as **bronchitis**, **bronchiolitis**, or **broncho-interstitial pneumonia**, arising from the **synergistic effect** of multiple agents.
•Some cases are **subclinical**, and such **subclinical bronchiolitis and pneumonias** are commonly associated with:
Mycoplasma dispar, M. bovis,* and *M. ureaplasma species.
•Additionally, ***M. bovigenitalium*** may sometimes be **isolated** from such cases.

Predisposing viral infections: PI-3, BRSV, IBR, Adenovirus.

Pathogenesis:

Viral damage to airways → Mycoplasma colonization → bronchitis, bronchiolitis, and interstitial inflammation.

- There are **no characteristic signs of fibrinous pneumonia**. Instead, **catarrhal, purulent,** and partially **interstitial pneumonia** lesions are observed. Occasionally, it may also be identified as **atypical interstitial pneumonia**.

Findings:

Lesions are localized in **broad cranioventral areas**, which appear **dark red and atelectatic**.

Catarrhal bronchitis and **bronchiolitis**, along with **interstitial pneumonia**, are evident.

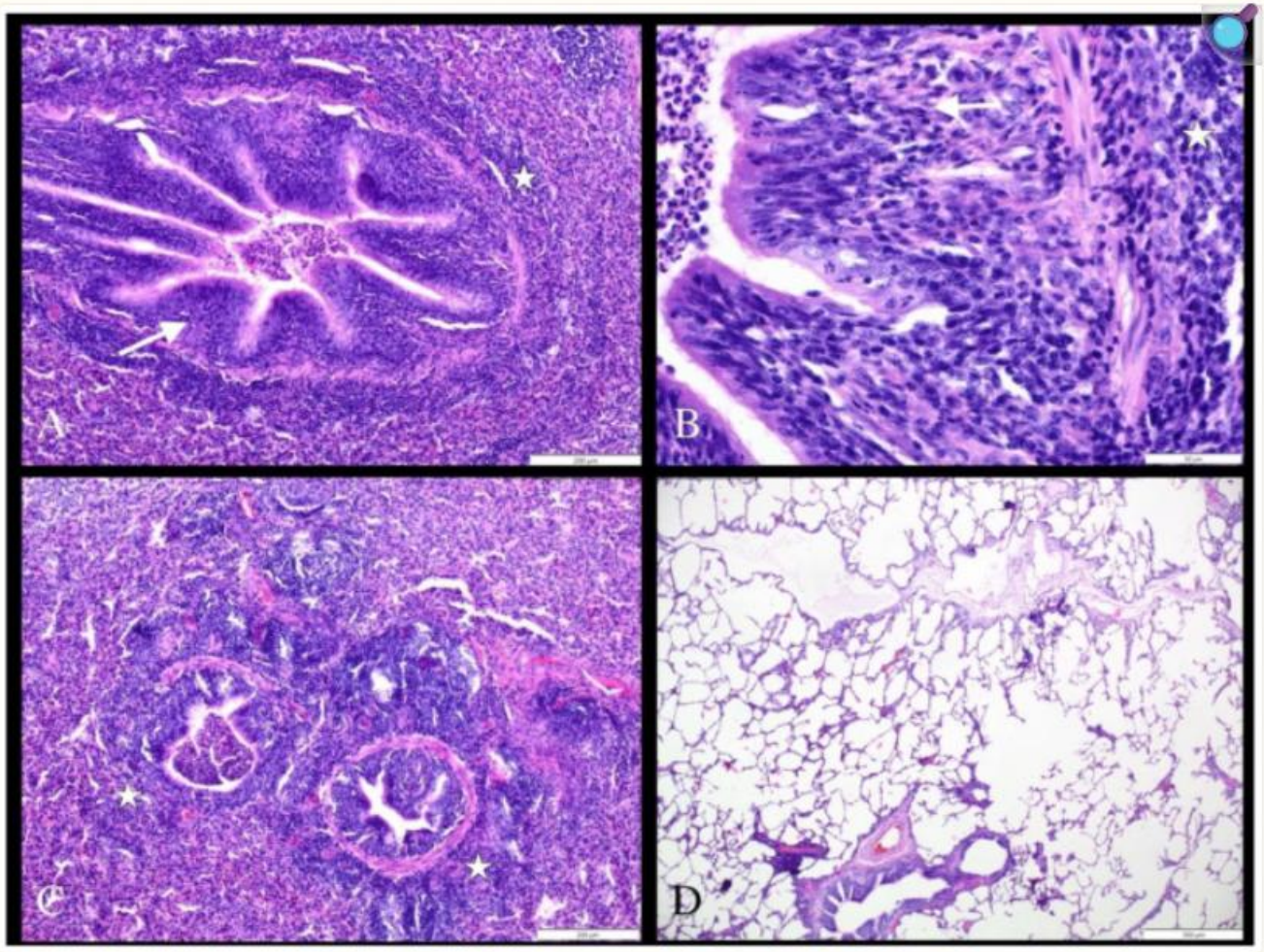
In **catarrhal bronchitis, bronchiolitis, and pneumonia**, the **bronchial and alveolar lumens** contain **exudate rich in neutrophilic leukocytes**.

Lungs -Consolidation of cranioventral lobes.

MANIMARAN, K., HEMALATHA, S., RAMESH, A., JAISREE, S., KUMANAN, K., RAVIKUMAR, G., ... & SUBRAMANIAN, S. A REPORT ON MASSIVE OUTBREAKS OF FMD WITH CONCURRENT MANNHAEMIA HEMOLYTICA INFECTION AMONG CATTLE IN TAMIL NADU. *LEGUME RESEARCH*.

As evidence of interstitial pneumonia:

- **Thickening of alveolar walls**, **hyperplasia of type II pneumocytes**, and **peribronchial lymphocytic cuffs (cuffing)** are observed.
- **With bronchial obstruction, atelectasis** develops.
- Additionally, in the **submucosal glands** of the bronchi and in the **goblet cells** of the lamina epithelialis, **secretory activity increases**; in chronic cases, **mucosal epithelial hyperplasia** occurs.
- These findings help distinguish **mycoplasmal pneumonia** from **true fibrinous pneumonia**.



Histopathological findings in lung tissue samples. (**A**–**C**) Gross cranioventral pulmonary consolidation associated with **enzootic pneumonia**. There are distinct peribronchiolar inflammatory cell cuffs composed of macrophages, lymphocytes, plasma cells, and eosinophils (asterisks). The bronchial mucosa is thickened and infiltrated with variable numbers of inflammatory cells (arrows) (**A**,**B**). (**D**) Healthy lung tissue. No microscopic changes are observed. HE. Scale: 20 µm (**A**); 50 µm (**B**); 200 µm (**C**); 500 µm (**D**).

Przyborowska-Zhalniarovich P, Maes D, Otrocka-Domagała I, Paździor-Czapula K, Wiszniewska-Łaszczych A, Sołtysiuk M. Association between Enzootic Pneumonia-like Lung Lesions and Carcass Quality and Meat pH Value in Slaughter Pigs. Animals (Basel). 2023 Jul 5;13(13):2210. doi: 10.3390/ani13132210. PMID: 37444007; PMCID: PMC10340002.

OTHER MYCOPLASMA INFECTİONS

Mycoplasma spp. Infections in Various Species

- **Cattle:** *M. bovis* → chronic pneumonia, otitis media, arthritis, mastitis
- **Goats:** *M. capricolum* → contagious caprine pleuropneumonia
- **Pigs:** *M. hyopneumoniae* → enzootic pneumonia of swine
- **Sheep:** *M. ovipneumoniae* → chronic progressive pneumonia
- **Poultry:** *M. gallisepticum* → chronic respiratory disease (CRD) in chickens
- **Turkeys:** *M. meleagridis* → air sacculitis and reduced hatchability

CONTAGIOUS CAPRINE PLEUROPNEUMONİA

Causative Agent: *Mycoplasma mycoides* subsp. *capri* var. *mycoides* (PPLO)

- **Cattle are not susceptible** to this disease.
- It is characterized by **fibrinous pneumonia**.
- **Necrosis and sequestration** occur in advanced cases.
- **Fibrinous pleuritis** often develops, which may **progress to adhesive pleuritis**.

Additional Lesions:

- In **goat kids**: fibrinous pericarditis, meningitis, interstitial pneumonia (similar to lesions in cattle).
- In **adult goats**: arthritis, peritonitis, abortion, and mastitis (as seen in cattle).

PleuroPneumonia-Like Organisms

PASTEURELLA INFECTIONS

- *Pasteurella spp.* are **Gram-negative, rod-shaped, bipolar-staining bacteria**. They have a **broad host range**, causing both **primary infections** (alone) and **secondary infections** (in conjunction with other pathogens) in humans and animals.
- Some species are **normal inhabitants** of the **nasopharynx and oral mucosa**:
- **P. pneumotropica:** found in the respiratory tracts of mice, rabbits, and other laboratory animals; also in the nasal–pharyngeal mucosa of humans and dogs.
- **P. ureae:** can exist **saprophytically in human nasal mucosa**, but may also participate in **secondary infections**.

Main Species

- **Pasteurella multocida** (*syn. P. septica*)
- **Mannheimia haemolytica** (*formerly Pasteurella haemolytica*)
- **Pasteurella pneumotropica** and **Pasteurella ureae** — *opportunistic species*

Pasteurella multocida (*P. septica*)

- **Significance:** Important in **cattle, sheep, buffalo, deer, pigs, rabbits, and poultry**.
- **Robert classification:** Types **I–VI**
- **Carter classification** *(based on capsular antigen):* Types **A, B, C, D, E**
- **Characteristics by type:**
- **Type B (I):** Cattle, deer, buffalo → *hemorrhagic septicemia*
- **Type A (II):** *Fowl cholera*, pneumonia in cattle and pigs
- **Type C (III):** Less significant
- **Type D (IV):** Swine pneumonia, sporadic distribution
- **Type E (V):** Also reported in swine pneumonia

Mannheimia haemolytica

Formerly known as *Pasteurella haemolytica*.
It is hemolytic in nature, with multiple serotypes (mainly A1, A2, A6, etc.).
Causes **fibrinous or fibrinonecrotic pneumonia** and **septicemia** in sheep, goats, and cattle.
In lambs, infections are often associated with *Bibersteinia trehalosi* (formerly *P. haemolytica biotype T*).

PRIMARY PASTEURELLOSIS (HEMORRHAGIC SEPTICEMIA / CLASSICAL PASTEURELLOSIS)

Causative Agent

- **P. multocida Type B (I)** and, to a lesser extent, **Type D (IV)**.
- Common in **ruminants** such as **cattle, buffalo, and deer** — widespread in **Asia and Africa**.

Transmission and Pathogenesis

- The agent is shed in **saliva, feces, urine, and milk**; transmission occurs **via the alimentary route**.
- It spreads from the **pharyngeal lymphoid tissue** into the bloodstream, leading to **bacteremia**.

Endotoxin Effects:

- Increases **vascular permeability**, causing **edema and inflammation**.
- Induces **intravascular coagulation**, resulting in **thrombosis**.
- Consequently, **edema**, **hemorrhage**, and **fibrinous pneumonia** develop.

Predisposing Factors:

- **Stress**, **elevated corticosteroid levels**, **climatic changes**, and **poor husbandry conditions**.

CLINICAL FINDINGS

- Although **subclinical cases** may occur, **mortality can reach 100%.**
- **Peracute form:** *Hemorrhagic septicemia* — death occurs within **a few hours to one day**.
- **Acute form:** Characterized by **subcutaneous and submucosal hemorrhages and edema**, especially **under the jaw, on the neck, chest,** and **eyelids**, causing visible swelling.
- Clinical signs include **fever**, **respiratory distress**, **fibrinous pneumonia** in the *pectoral form*, and **hemorrhagic gastroenteritis** in the *intestinal form*.

PATHOLOGICAL FINDINGS

- **Peracute form:** No distinct macroscopic lesions.
- **Acute form:**
  - **Petechial hemorrhages** on **serous membranes**, **lungs**, **muscles**, and **internal organs**.
  - **Yellowish, coagulated edema** in the **pharynx, larynx, eyelids,** and **submandibular/neck regions**.
  - **Bloody fluid** in serous cavities and **enlarged lymph nodes**.
  - **Acute catarrhal or hemorrhagic gastroenteritis.**
- **Fibrinous pneumonia:** shows **less peribronchial organization** and **minimal sequestration**, which serves as a **key distinguishing feature** from *Mycoplasmosis*.

DIFFERENTIAL DIAGNOSIS

- **Hemorrhagic septicemia**
- **Anthrax**
- **Mycoplasma pneumonia**

SECONDARY PASTEURELLOSIS (SHIPPING FEVER / CALF PASTEURELLOSIS)

(shipping = ship by ship; fever = fever)
Transport, transport, animal market fever, Railway Fever

Causative Agents:

- **Pasteurella multocida** *Type A (formerly Type II)*
- **Mannheimia haemolytica** *Serotype A l* (formerly *Pasteurella haemolytica*)

Associated Primary Agents

Parainfluenza-3, IBR (Infectious Bovine Rhinotracheitis), RSV (Respiratory Syncytial Virus), Chlamydia sp., Mycoplasma sp.

→ Develops **secondarily** to these **primary infections.**

•*Shipping fever* is a **multifactorial respiratory disease** complex of cattle.
•It often follows **stress**, **transport**, or **viral infections** (e.g. *IBR, PI3, RSV, BVD*).
•*Mannheimia haemolytica A1* is the **primary bacterial pathogen**,
while *P. multocida A* acts **secondarily** or **co-infects**.

Lesions

Typically **catarrhal**, partially **fibrinous**, **abscess-forming**, or **interstitial pneumonia**

patterns.

Often accompanied by **fibrinous pleuritis.**

Lesions are **mainly localized** in the **cranioventral lobes.**

Sequestrum formation is **minimal.**

MICROSCOPICAL FINDINGS

- **Vascular thrombosis and necrosis** due to **endotoxin effects.**
- **Alveolar lumina** contain **elongated leukocytes** with a characteristic **“oat cell” appearance.**
- ***M. haemolytica*** → causes **fibrinous pneumonia.**
- ***P. multocida*** → causes **fibrinopurulent pneumonia.**
- Depending on the **primary agent**, **syncytial giant cells** and **inclusion bodies** may also be present.

Complications

Meningitis (in calves aged 2–4 months)

Polyarthritis

Abortion (*P. haemolytica*)

Mastitis:

- *P. haemolytica* → **Peracute**, **hemorrhagic**, and **necrotic mastitis**
- *P. multocida* → **Chronic mastitis** characterized by **fibrosis** and **atrophy**

PASTEURELLOSIS IN SHEEP AND GOATS

Causative Agents

- **Mannheimia haemolytica** → *Primary pathogen*
- **Pasteurella multocida** → *Secondary pathogen*

Pathogenesis

- Transmission occurs **aerogenically** (via inhalation).
- The organism may normally reside in the **nasal cavity, pharynx, and tonsils**.
- **Predisposing factors:** poor hygiene, malnutrition, vitamin A deficiency, climatic changes, transportation, shearing, and stress.
- **Viral interstitial pneumonias**, **Chlamydia infections**, and **lung parasites** also facilitate the development of the disease.
- In **primary hemorrhagic septicemia**, the **endotoxin** is the major pathogenic factor.

Clinical Findings

Hemorrhagic Septicemia Form:

High fever, **tachycardia**, **respiratory distress**, **frothy discharge** from the mouth, and marked **hyperemia of mucous membranes.**

Death usually occurs **within a short period.**

Pneumonic Form:

Acute fibrinous pneumonia, accompanied by **nasal discharge**, **coughing**, **respiratory difficulty**, and **fever.**

Pathological Findings

Lymph nodes: Edematous and hemorrhagic.
Pleura, pericardium, peritoneum, mesentery: Show **petechial and ecchymotic hemorrhages.**
Subcutaneous tissue: Contains **yellow, edematous areas.**
Lungs: Edematous, hyperemic, and marked with **ecchymoses.**
Trachea: Contains **slightly bloody, frothy fluid.**
Liver: Displays **focal necroses** resulting from **portal vein thrombosis.**

Microscopic Findings:

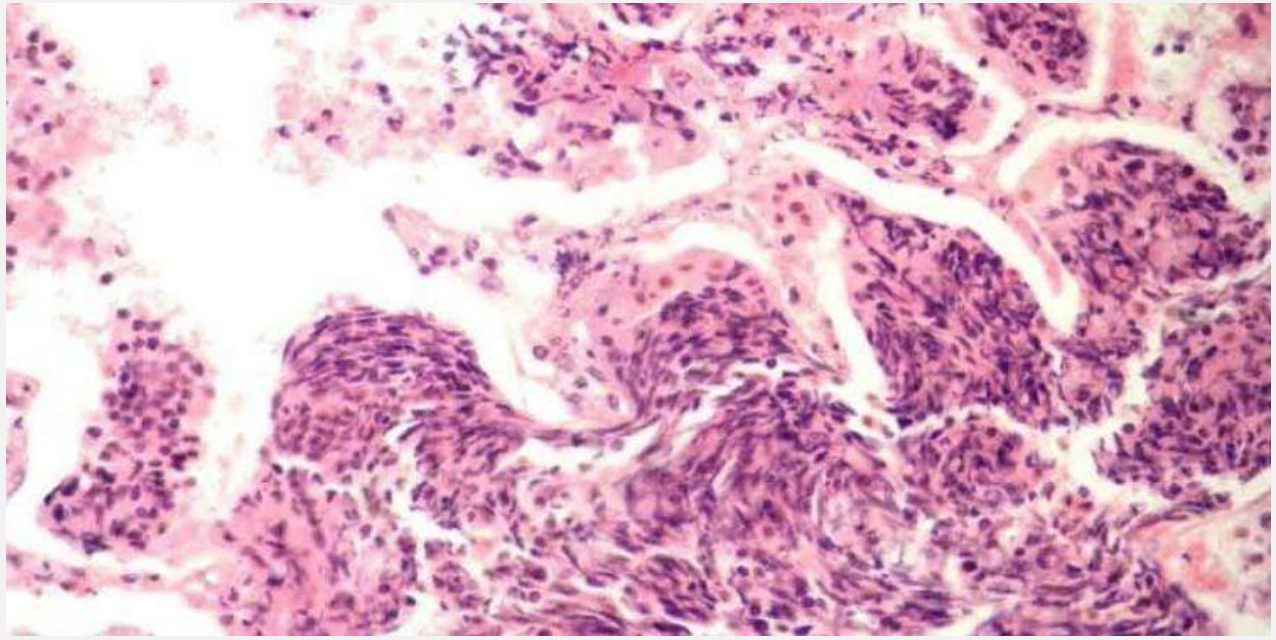
Around necrotic areas, there is a **lack of neutrophils (nonreactive inflammation)**.

Presence of **microbial emboli** and characteristic **"oat cells."**

Joints, meninges, and pericardium: Show **inflammatory reactions.**

Lungs: Exhibit **fibrinous to fibrinonecrotic pneumonia** and **serofibrinous pleuritis.**

In **subacute cases**, **adhesive pleuritis** and **abscess formation** may occur.



https://vetrehberi.com/yulaf-bicimli-hucreler-nedir/

HAEMOPHILUS SPECIES AND THE DISEASES THEY CAUSE

- *Haemophilus spp.* are **Gram-negative, pleomorphic bacteria** that may appear **coccoid, rod-shaped, or filamentous**.
- There are significant differences among species in terms of **virulence, pathogenicity,** and **geographical distribution**.
- The **course and outcome** of infection vary depending on the **host species**, **age**, **physiological condition**, and **immune status**.
- Therefore, **Haemophilus infections** can present with **different clinical manifestations** in **animals and humans**.

Tür	Konak	Oluşturduğu Hastalıklar
H. pleuropneumoniae	Domuz	Contagious pleuropneumonia, meningitis, arthritis
H. parasuis	Domuz	Polyserositis (pleuritis, peritonitis, perikarditis), arthritis, keratitis, Glässer hastalığı, pnömoni
H. somnus	Sığır (özellikle buzağı)	Infectious thromboembolic meningoencephalitis, pnömoni, arthritis
H. agni	Koyun (özellikle kuzular)	Septisemi, meningitis, arthritis, pnömoni
H. haemoglobinophilus	Köpek	Genital kanalda kommensal olarak bulunur
H. paragallinarum	Kanatlılar	Infectious coryza (üst solunum yolu enfeksiyonu)
H. influenzae	İnsan	Üst solunum yolu enfeksiyonları ve pnömoni
H. equigenitalis	At	Contagious equine metritis (CEM)

CATTLE (ESPECIALLY CALVES) – *HAEMOPHILUS SOMNUS*

Causative Agent:

H. somnus typically causes **embolic meningoencephalitis**.

Lesions:

Brain and spinal cord:

Numerous **focal hemorrhages**

Infarct (necrotic) areas ranging from **1–15 mm** in diameter

May also cause **serofibrinous laryngitis, tracheitis, pleuritis, pericarditis, peritonitis,** and **polyarthritis**.

Lymph nodes are usually **enlarged**.

In the **urogenital system**, can lead to **necrotic metritis**; in **pregnant cows**, **abortion** often occurs around the **7th month** (sporadic in nature).

Clinical Findings

Fever and **depression**

Nervous system signs: ataxia, opisthotonus, blindness, and paralysis

Histopathological Findings

Characterized by **vascular thrombosis**, **septic emboli**, and **vasculitis**.

Macroscopic findings correspond to **microscopic vascular damage and necrosis**.

SHEEP (ESPECIALLY LAMBS) – *HAEMOPHILUS AGNI*

Causative Agent:

H. agni primarily causes **septicemia** in **lambs**.

Clinical Findings:

Fever

Depression

Necropsy Findings:

Hemorrhages in skeletal muscles and various organs

Focal necroses in the liver

Splenomegaly (enlarged spleen)

Fibrinopurulent arthritis

Meningitis (especially at the base of the brain)

Choroiditis may also be observed.

Histopathological Findings

Vasculitis resulting from **bacterial emboli** is the most prominent feature.

Other lesions develop **secondarily** to these **vascular disturbances**.

Overall, the lesions are **similar to those observed in cattle.**

HORSE – *HAEMOPHİLUS EQUİGENİTALİS*

Disease: *Contagious Equine Metritis (CEM)*
Causative Agent:
H. equigenitalis, which localizes in the **genital organs**.
Lesions:
Mucopurulent exudation
Proliferation and vacuolation of epithelial cells
Mononuclear cell infiltration in affected areas
Outcome:
Causes **inflammatory reactions** and **reproductive disorders** in the genital system.
Leads to **infertility** and **transmission through infected breeding** in both **male and female horses.**