Tutorial Module 4

Classification of Pneumonia in Domestic Animals

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One of the most controversial topic in pathology and veterinary medicine is the CLASSIFICATION OF PNEUMONIAS.

Just remember that there is no best or universal classification. These are some of the many criteria used in veterinary medicine:

**EPIDEMIOLOGIC CRITERIA:** Enzootic, Contagious, Progressive

**MORPHOLOGIC CRITERIA:** Gangrenous, Lymphocytic, Proliferative, Exudative

**ETIOLOGY CRITERIA:** Bacterial, Viral, Mycotic, Parasitic, Aspiration

**MISCELLANEOUS CRITERIA:** Atypical, Cuffing, Neonatal, Calf, Shipping Fever

**CLINICAL CRITERIA:** BRD Complex, acute undifferentiated

**DISEASE CRITERIA:** Distemper pneumonia, tuberculosis, IBR,
Based on TEXTURE, DISTRIBUTION and TYPE OF EXUDATE, pneumonias in Domestic Animals can be grossly classified into FIVE morphologically distinct types:

- Suppurative Bronchopneumonia
- Fibrinous Bronchopneumonia
- Interstitial Pneumonia
- Embolic Pneumonia
- Granulomatous pneumonia

This gross classification allows you to predict the most likely port of entry, gives you a good idea for rule-outs which help you to decide which laboratory tests you should requested. Therefore, increases your chances to arrive at the most likely etiological diagnosis.
Distribution and Texture of Lesions in Pneumonias

NORMAL

Suppurative Bronchopneumonia
Craniocentral and firm

Fibrinous Bronchopneumonia
Craniocentral and hard

Interstitial Pneumonia
Diffuse and Elastic

Embolic Pneumonia
Multifocal and nodular

Granulomatous Pneumonia
Multifocal and nodular
Suppurative Bronchopneumonia

- Distribution: Cranioventral
- Texture: Firm
- Color: Red (acute) to grey (chronic)
- Cut surface: purulent exudate in bronchi
- Port of entry: Aerogenous
- Common etiologies:
  - Bacteria (low grade pathogenicity)
  - Mycoplasma
  - Aspiration of bland material
- Most common sequels: Cranioventral abscesses and bronchiectasis
Another example of a Suppurative Bronchopneumonia in a Pig

Note cranioventral consolidation (C) while the caudal lung remains unaffected (N). The texture of consolidated lung would be firmer than normal. Typically, on cut surface purulent exudate could be expressed from bronchi (arrow).
Another example of a suppurative bronchopneumonia. Note once again that consolidation is restricted to the cranioventral lung while the caudal lung (CL) remains unaffected. There is also a lobular pattern typical of suppurative bronchopneumonia (see diagram). The texture of consolidated lung would be firmer than normal and on a cut surface purulent exudate could be expressed from bronchi. The more chronic the lesion, the more mucus in the exudate expressed from bronchi.
Suppurative Bronchopneumonia

The gross appearance of the exudate in suppurative bronchopneumonia varies from purulent in acute cases to mucopurulent or mucoid in more chronic cases.

The presence of mucus in the exudate is due to severe goblet (mucus) cell hyperplasia in bronchi and goblet cell metaplasia in bronchioles.

Note the light color of the normal lung and the darker hyperemic color of the pneumonic lung.

Note also purulent exudate coming out of a major bronchus which is typical of suppurative bronchopneumonia.
The color of consolidated lung varies according to chronicity from:

- Bright red in acute suppurative bronchopneumonia (severe hyperaemia + small exudate)
- Dark red in subacute suppurative bronchopneumonia (less hyperemia and more purulent exudate)
- Pale grey resembling fish-flesh in chronic suppurative bronchopneumonia (exudate, fibrosis, and less blood because reduced volume density of pulmonary capillaries).

The main microscopic lesion in suppurative bronchopneumonia would be neutrophils and macrophages in bronchoalveolar spaces (see next slide).
Suppurative Bronchopneumonia

Subacute, Suppurative bronchopneumonia / H&E stain/

Histological section of a lung with suppurative bronchopneumonia. Note a bronchiole plugged with purulent exudate (pe). Also note large number of neutrophils mixed with edematous fluid in alveoli, hence the name bronchopneumonia.

The pathogenesis of this lesion is related to release of chemotactic factors on mucosal surface of bronchi, bronchioles and alveoli causing migration of neutrophils into the airways.

In uncomplicated cases, the exudate moves along the mucociliary escalator. In severe cases, the excessive release of leukocytic enzymes damages the mucosa, submucosa, smooth muscle and cartilage, causing bronchiectasis (see next slide).
Pulmonary abscesses and bronchiectasis are two important sequels to suppurative bronchopneumonia.

Note large abscesses in the cranial and intermediate lobes. The caudal lobes are essentially normal. Histologically, abscesses are composed of a purulent core surrounded by connective tissue (pyogenic membrane).
Bronchiectasis is the rupture and dilation of bronchi due to enzymatic and destructive effect of neutrophils and macrophages on the bronchial wall in chronic bronchopneumonia.

Note tubular dilations in the cranioventral lung (white arrows). These tubular structures are distended bronchi filled with purulent exudate that has destroyed the bronchial walls (insert). These lesions may look like abscesses. However, microscopically instead of being lined by a capsule it is lined by remnants of bronchial walls which contain purulent exudate (red arrows).
**Fibrinous Bronchopneumonia**

- Distribution: cranioventral
- Texture: Hard
- Color: Red ➔ yellow ➔ grey
- Fibrin on pleural surface
- Cut surface: fibrin / necrosis
- Port of entry: aerogenous
- Most common etiologies:
  - Highly pathogenic bacteria (exotoxins): *Mannhemia haemolytica, Actinobacillus pleuropneumoniae*;
  - Harsh aspirated materials
- Implies severe injury to the lung
- Sequels: Sequestrum, pleural adhesions
Note cranioventral consolidation and affected lung covered with fibrin. Only a small portion of the lung appears grossly normal (asterisk).
Note cranioventral consolidation with affected lung covered with a thick layer of fibrin. Only the dorsocaudal lung appears normal (asterisk). Remember, the texture of consolidated lungs in fibrinous bronchopneumonia is typically hard.
Fibrinous Bronchopneumonia

Cut surface of lung

Fibrinous bronchopneumonia generally implies severe injury to the lung with leakage of fibrin into the airspaces.

On cut surface, fibrinous bronchopneumonia often has a mosaic appearance due to distention of the interlobular septa in areas of coagulation necrosis.

Note in this photograph a mosaic appearance of the lung. The arrows delineate distended interlobular septa that results from lymphatic thrombosis and edema.

Note also areas of coagulation necrosis (asterisks).

This particular case is Shipping Fever Pneumonia caused by the toxins of *Mannheimia haemolytica A1*.

• Highly pathogenic bacteria that produce exotoxins such as
  • *Mannheimia haemolytica*
  • *Actinbacillus pleuropneumoniae*
Fibrinous Bronchopneumonia

Histopathology H&E.

Note loss of airspaces due predominantly to exudation of fibrin (asterisks) and to a lesser extent of leukocytes. In this type of pneumonia hemorrhages are also common.

Since fibrin predominates over polymorphonuclear leukocytes, the diagnosis of fibrinous is preferred to that of suppurative bronchopneumonia.

Remember that fibrin is chemotactic for neutrophils and therefore any fibrinous pneumonia has neutrophils microscopically.
4 other examples of Fibrinous Bronchopneumonia
Pleural Adhesions are important sequel to bronchopneumonia.

Note in all cases bands of connective tissue (arrows) between the lung and the parietal pleura lining the thoracic cavity. Adhesions are commonly seen in postmortems revealing that the animal had or survived a previous episode of bronchopneumonia. Adhesions between pleura and pericardium are also common.
Lung sequestrum (plural sequestra) is also a sequel sometimes seen in fibrinous bronchopneumonia. Simply put it, a sesquestrum is a large piece of necrotic lung that becomes isolated from the surrounding pulmonary tissue.

**Figure 1.** Severe fibrinous pneumonia in a cow showing focal areas of acute necrosis (small circle). If the animal had survived, these areas of necrosis could become isolated from the remaining lung forming a sequestrum.

**Figure 2.** These lungs show a large wall-off pulmonary sequestrum. This was a case of Contagious Bovine Pleuropneumonia (*Mycoplasma pleuropneumoniae*) that is enzootic in some countries in Asia and Africa, but non-existent in the Americas. If you were to cut this sequestra it would be composed of pure necrotic tissue.
INTERSTITIAL PNEUMONIA

- **Distribution**: Diffuse
- **Texture**: Elastic
- **Cut surface**: Meaty
- **Gross Features**:
  - Rib imprints
  - Lungs failed to collapse when thorax is opened
- **Port of entry**: Aerogenous or Hematogenous
- **Most common etiologies**: Virus, toxins, type III hypersensitivity, toxicants

- **Pathogenesis**: Injury centered in the alveolar wall (endothelium or pneumocytes)
- **Histology**: Thickening of the alveolar walls
- **Important Feature**: Often difficult to diagnose grossly
Acute, severe, diffuse interstitial pneumonia / Horse.

Note discreet rib imprints on pleural surface which resulted from lack of deflation at the time when the negative pressure was removed by opening the thorax.
Acute, severe, diffuse interstitial pneumonia / Lamb.

This is another example of an interstitial pneumonia. Note again rib imprints on the pleural surfaces (arrows) of this lamb that died acutely with adenovirus pneumonia.

Remember that the pleural imprints result from lack of deflation at the time when the negative pressure is removed by opening the thorax.

Changes are often subtle, difficult to diagnose grossly and generally require histopathological confirmation.

On cut surface, the lung tissue in interstitial pneumonia has an elastic texture and a meaty appearance.

The primary lesion is centered in the alveolar wall. Thickening of alveolar walls results from interstitial infiltration of mononuclear cells or from proliferation of type II pneumocytes. In chronic interstitial pneumonia, there is alveolar fibrosis.
Steer.

This lung belonged to a steer that died a few days after severe respiratory distress. The lungs failed to collapse when the thorax was opened and there were rib imprints on pleural surface. The texture of this lung was notably elastic.

Note some edematous distention of the interlobular septa (arrows) and "meaty" appearance of the parenchyma.

Interstitial pneumonia is perhaps the most difficult type of pneumonia to diagnose grossly. In most cases it is imperative to do histopathology. The basic lesion in interstitial pneumonia is the thickening of alveolar septa which is what gives it the elastic texture and prevents alveoli from collapsing.

Microscopic view of the basic interstitial pneumonia is illustrated in the next slide.
Interstitial Pneumonia

Steer. Histopathology. HE stain

Note thick alveolar walls (arrows) due to increased cellularity of the alveolar walls and interstitium (asterisks).

Thickening of alveolar walls can result from influx of inflammatory cells or fluid into the interstitium or from proliferation of hyperplastic type II pneumonocytes.

In chronic cases, interstitial pneumonia may progress to interstitial fibrosis and the lesion is often called "fibrosing alveolitis."

Interstitial pneumonia results from alveolar damage caused by viral infections, deposition of antigen-antibody complexes, inhalation of toxic gases (nitrogen dioxide, sulfur dioxide, ozone, oxygen toxicity), or ingestion of toxicants that damage bronchioles and alveoli such as the herbicide paraquat or some mycotoxins.
Embolic Pneumonia

- **Distribution**: Multifocal
- **Texture**: Nodular
- **Port of entry**: Hematogenous
- **Color**: red when acute, pale when chronic
- **Most common etiologies**:
  - Rupture of hepatic abscesses into the vena cava in cattle
  - Vegetative endocarditis (right side of the heart)
  - Jugular thrombosis
  - Embolic foreign body (hair, septic emboli, etc).

Note multifocal hemorrhagic lesions randomly distributed in all pulmonary lobes (arrows). Also note that the center of the hemorrhagic foci is often white, suggesting neutrophilic inflammation.
Note numerous foci of inflammation scattered throughout the lungs (arrows)
See Figure 9-41 Pathologic Basis of Veterinary Diseases
Embolic Pneumonia

Foal.
Sometimes the embolic lesions are very small and difficult to see as in the lungs of this foal. Look closely in the inset and you will notice small dark foci with white center (arrows).
Embolic Pneumonia

Note foci of neutrophilic inflammation in the lung.
Endocarditis (arrow) is a common cause of embolic pneumonia.

Note numerous foci of inflammation in the lungs. The focal lesion have a white color because they are chronic and the initial hemorrhage has been resolved. These focal lesions are evolving towards small abscesses.
Sequel to Embolic Pneumonia: Pulmonary abscesses

Note numerous small abscesses resulting from septic embolisms due to vegetative endocarditis affecting the tricuspid valve (right side) of the heart. Septic emboli are easily trapped in pulmonary vasculature causing embolic pneumonia and its sequel pulmonary abscesses. Bronchopneumonia may also have abscesses as sequels, except that in this latter type of pneumonia the distribution is cranioventral and not random as in embolic pneumonia.
Granulomatous Pneumonia

- Distribution: Multifocal
- Texture: Nodular (no pus)
- Cut surface: granulomas
- Port of entry:
  - Aerogenous
  - Hematogenous
- Etiology:
  - *Myocbacterium spp*
  - Systemic mycoses
  - Parasitic ova
  - Trapped food particles (starch)
  - Dead parasites
Granulomatous Pneumonia in Bovine Tuberculosis

- Note numerous focal to coalescing granulomas scattered throughout the lung parenchyma.

- Granulomatous pneumonia is typically caused by organisms or particles that cannot be readily destroyed by macrophages or in other words resistant to phagocytosis such as Mycobacteria, fungus, parastic ova, dead larvae, etc.

- The texture of this lung would be nodular.
Granulomatous Pneumonia

Chronic, severe, multifocal granulomatous pneumonia / Canine Blastomycosis.

Note entire lung parenchyma filled with medium sized, often confluent granulomas. The distribution of granulomatous pneumonia is random and multifocal affecting all pulmonary lobes. The port of entry in granulomatous pneumonia could be either aerogenous or hematogenous. Texture is typically nodular.
Granulomatous Pneumonia

Numerous focal to coalescing granulomas in lung parenchyma.

Tuberculosis can affect any tissue including bones, bone marrow, gonads, kidneys, meninges, etc.

*Mycobacterium tuberculosis* generally affects humans, but cross infections with *M. bovis* and *M. avium* can also occur. Pigs can be affected by all three species. Remember that typical granulomatous lesions seen in cows and sheep (caseous necrosis and calcification) with tuberculosis are not seen in horses, cats and dogs. In these other animals tubercles have the appearance of tumoral growths (sarcomatous).

Sometimes tumor and granulomas are not easily distinguishable.

Acid fast stain reveals the TB bacillus.
Granulomatous Pneumonia

Note typical granuloma macrophages in the center and an external band of fibrous connective tissue (double arrows) infiltrated by lymphocytes and plasma cells (not seen at this magnification).

In most granulomatous pneumonias, the etiologic agent can be detected with special stains (to be shown later). Among the most common causes of granulomatous pneumonia in animals are:

- Tuberculosis
- Systemic mycosis (*Cryptococcus neoformans, Blastomyces dermatitides, Coccidiodes immits*)
- Aberrant parasitic larvae, foreign body (food particles), algae.
- Feline Infectious Peritonitis in cats, etc.
SUMMARY

The 5 gross morphologic types of Pneumonia in Domestic Animals

Fibrinous Bronchopneumonia

Embolic Pneumonia

Interstitial Pneumonia

Granulomatous Pneumonia

Suppurative Bronchopneumonia
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Also, if you find any errors or typos please let me know too.

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The End