**Diseases of the lungs**

**PN EUM ONIA**

Pneumonia is inflammation of the pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleuritis. It is manifested clinically by an increase in the respiratory rate, changes in the depth and character of respirations, coughing, abnormal breath sounds on auscultation and, in most bacterial pneumonias, evidence of toxemia.

**ETIOLOGY**

Pneumonia may be associated with viruses, bacteria, or a combination of both, fungi, metazoan parasites and physical and chemical agents.

**Cattle**:

* Pneumonic pasteurellosis (shipping fever) - M. haemolytica, P multocida
* *Histophilus somnus*
* Enzootic pneumonia of calves
* Viral interstitial pneumonia
* Contagious bovine pleuropneumonia -*Mycoplasma mycoides*
* Atypical interstitial pneumonia
* Lungworm pneumonia – *Dictyocaulus viviparous*
* Klebsiella pneumoniae infection
* Tuberculosis
* *Fusobacterium necrophorus*

**Pigs**:

* Enzootic pneumonia (Mycoplasma & pasturella)
* Pneumonic pasturellosis
* Interstitial pneumonia – septicemic salmonellosis
* Bordetella bronchiseptica, Salmonella choleraesuis
* Anthrax by inhalation, causing pulmonary anthrax

**Horses**:

* Pleuropneumonia in mature horses due to aerobic and anaerobic (Strept, Pasturella, E.coli.,….)
* Pleuropneumonia in newborn foals caused by Streptococcus , E.coli, Actinobacilus equillini
* Pneumocystis carini
* Rhabdococcus equi
* Bronchinterstitial pneumonia in foals 1-8 month of age with uncertain etiology
* Parasitic pneumonia As a sequel of strangle ,equine viral arteritis and equine viral rhinopneumonitis

**Sheep:**

* Pneumonic pasteurellosis
* *Streptococcus zooepidemicus, Salmonella abortus-ovis*
* Severe pneumonia due to Mycoplasma sp. in lambs
* *Corynebacterium pseudotuberculosis* - sporadic cases only
* Melioidosis (*Pseudomonas pseudomallei*)
* Lungworm *(Dictyocaulus filaria*)
* Progressive interstitial pneumonia (maedi)

**Goats**:

* Chronic interstitial pneumonia with cor pulmonale as a common sequel may be associated with a number of *Mycoplasma spp*.,
* Retrovirus infection

**All species**:

* Toxoplasmosis,
* Systemic mycosis,
* Aspiration pneumonia,
* Sporadic secondary pneumonia caused by Strep., Corany., and *Dermtophllus spp*

Epidemiology:

In addition to the infectious agents which cause the pneumonia, there are risk factors which contribute to the susceptibility of the animal. Three risk factors interact in the pathogenesis of specific pneumonias

* Animal
* Environmental and management
* Pathogen.

As examples, some of the commonly recognized risk factors include:

1. The weaning of beef calves in northern climates
2. The long transportation of beef cattle to feedlots
3. The collection and mixing of animals for prolonged period
4. The transportation of Thoroughbred horses farther than 500 miles
5. Housing dairy calves in poorly ventilated overcrowded barns
6. Marked changes in weather.

Pathogenesis :

The major defense mechanisms of the respiratory tract include:

* Aerodynamic filtration by the nasal cavities
* Sneezing
* Local nasal antibody
* The laryngeal reflex
* The cough reflex
* Mucociliary transport mechanisms
* Alveolar macrophages
* Systemic and local antibody systems

The processes by which pneumonia develops varies with the causative agent and its virulence and with the portal by which its introduce into the lung.

Bacteria are introduced largely by way of the respiratory passages and cause a primary bronchitis which spread to involve surrounding pulmonary parenchyma. The reaction of the lung tissue may be in themfom1 of an acute fibrinous process as in pasteurellosis and contagious bovine pleuropneumonia, a necrotizing lesion as in infection with *F. necrophorum* or as a more chronic caseous or granulomatous lesion in mycobacterial or mycotic infections. Hematogenous infection by

bacteria results in a varying number of septic foci, which may enlarge to form lung abscesses rupture into air passages and spread as a secondary bronchopneumonia.

Viral infections are also introduced chiefly by inhalation and cause a primary bronchiolitis, but there is an absence of the acute inflammatory reaction that occurs in bacterial pneumonia. Spread to the alveoli causes enlargement and proliferation of the alveolar epithelial cells and the development of alveolar edema. Consolidation of the affected tissue results but again there is an absence of acute inflammation and tissue necrosis so that toxemia is not a characteristic development.

The pathophysiology of all pneumonias, regardless of the way in which lesions develop, is based upon interference with gaseous exchange between the alveolar air and the blood. Anoxia and hypercapnia develop, which results in polypnea, dyspnea or tachypnea. Consolidation results in louder than normal breath sounds, especially over the anteroventral aspects of the lungs, unless a pleural effusion is present to muffle the sounds. In bacterial pneumonias there is the added effect of toxins produced by the bacteria and necrotic tissue; the accumulation of inflammatory exudate in the bronchi is manifested by abnormal lung sounds such as crackles and wheezes on auscultation. Interstitial pneumonia results in consolidation of pulmonary parenchyma without involvement of the bronchi, and on auscultation loud breath sounds predominate in the early stages.

Stage of pneumonia :

1. Active hyperemia ,serous exudatation , capillary are extended with blood ,alveoli filled with serous fluid ,neutrophils and macrophage mixed with fibrin.
2. Consolidation (Red hepatization).
3. Gray hepatization erythrocyte disappear from the lungs.
4. Stage of resolution (hyperemia subside ,regeneration of cells and destroy of micro-organism ).

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1. Rapid, shallow breathing is the cardinal sign of early pneumonia
2. Coughing

Bacterial bronchopneumonia moist painful cough

viral interstitial pneumonia frequent, dry ,hacking and paroxysmal cough

3- Nasal discharge may or may not be present

4- Oder of breath Oder of decay when there is pus or putrid when gangrene is present.

5- Open mouth breathing ( in severe cases).

6- On auscultation there is increase of vesicular murmurs ,moist rales, pleuritic friction rub when pleurisy, muffled pulmonary sound in second stage of pleurisy ,cripitation sound in case of compensatory emphysema.

7- Large area of consolidation detected by percussion.

8- Cyanosis in late stage.

9- Heart sound can be heard in consolidation area.

10- Variation in the movement between two sides of the chest .

11- Fever , anorexia , depression ,increase puls rate and tendency to down and finally dehydration.

**Clinical pathology**:

* **Respiratory secretions**:

The laboratory examination of the exudates and secretions of the respiratory tract is the most common diagnostic procedure performed when presented with cases of pneumonia. Nasal swabs, tracheobronchial aspirates and bronchoalveolar lavage samples can be submitted for isolation of viruses, bacteria and fungi, cytological examination and determination of antimicrobial sensitivity.

* **Thoracocentesis:**

When pleural effusion is suspected, thoracocentesis can be used to obtain pleural fluid for analysis.

* **Heamatology :**

1. The hematocrit will be elevated in severely toxemic animals that are not drinking water.
2. Marked changes in the leukon in Severe bacterial bronchopneumonia and pleuritis.
3. Markedly elevated of plasma fibrinogen concentration in horses with bronchopneumonia.

* **Serology:** to diagnosis of the specific disease.
* **Fecal samples:**

When lungwonn pneumonia is suspected, fecal samples can be submitted for detection of the larvae.

**Necropsy findings:**

Gross lesions are usually observed in the anterior and dependent parts of the lobes; even in fatal cases where much of the lung is destroyed, the dorsal parts of the lobes may be unaffected. The gross lesions vary a great deal depending upon the type of pneumonia present. Bronchopneumonia is characterized by the presence of serofibrinous or purulent exudate in the bronchioles, and lobular congestion or hepatization.

In the more severe, fibrinous forms of pneumonia there is gelatinous exudation in the interlobular septae and an acute pleurisy, with shreds of fibrin present between the lobes.

In interstitial pneumonia the bronchioles are clean and the affected lung is sunken, dark red in color and has a granular appearance under the pleura and on the cut surface. There is often an apparent finn thickening of the interlobular septae.

In chronic bronchopneumonia of cattle there is consolidation, fibrosis, fibrinous pleuritis, interstitial and bullous emphysema, bronchi filled with exudate, bronchiectasis and pulmonary abscessation.

**Differential diagnosis:**

1. Pulmonary emphysema
2. Hydrothorax, heamothorax and pneumothorax
3. Pleurisy
4. Lung abscess
5. Pulmonary congestion and oedema
6. Congested heart failure
7. Hyperthermia and acidosis
8. Laryngitis, tracheitis and bronchitis

**Treatment:**

* Antimicrobial therapy
* Bronchodilators such as clenbuterol and steroidal anti inflammatory drugs
* Supporative therapy and housing

**Aspiration pneumonia**

Aspiration or inhalation pneumonia is a common and serious disease of farm animals. Most cases occur after careless drenching or passage of a stomach tube during treatment for other illness, for example administration of mineral oil to horses with colic. Other causes include the feeding of calves and pigs on fluid feeds in inadequate troughing.

**Pathogenesis:**

If large quantities of fluid are aspirated after passage of the stomach tube into the trachea,death may be almost occurs but with smaller quantities the outcome may depended on the composition of aspirated materials. Absorption from the lung is very rapid and soluble substance such as chlorlhydrate and magnesium sulfate exert their systemic pharmacological effects vary rapidly. With insoluble substances and vomitus the most common occurrence is the development of pneumonia with toxemia which is usually fatal in 48-72. Hours.

**Clinical signs:**

Signs of pneumonia including Polypnea, cough and the presence of rales, consolidation and associated pleruitic friction rub are present but the later may be localized . the severity of aspiration pneumonia depended largely on bacteria which are introduced although in animals the infection is usually mixed causing in many cases gangrenous pneumonia which may be manifested by putrid oder on the breath or extensive pulmonary suppuration .Occasionally animals survive the acute stage but persist in stage of chronic ill-health due to the presence of pulmonary abscess.

**Treatment:**

If the lesion are well advanced treatment is not often effective but treatment with broad spectrum antibiotic or sulfonamide may prevent development of the disease if administrated soon after aspirated occurs.

**Pulmonary abscess:**

The development of single or multiple abscesses in the lung causes a syndrome of chronic toxemia, cough, and emaciation. Suppurative bronchopneumonia may follow.

**Etiology:**

Pulmonary abscesses may be part of a primary disease or arise secondarily to diseases in other parts of the body.

**primary disease**

* *Corynebacterium (Rhobdococcus)* *equi* and *Streptococcus zooepidemicus* pulmonary abscesses of foals
* Sequestration of an infected focus, e.g. strangles in horses, caseous lymphadenitis in sheep
* Tuberculosis
* Actinomycosis Aerogenous infections with ' systemic' mycoses, e.g. coccidioidomycosis, aspergillosis, histoplasmosis, and cryptococcosis.

**Secondary disease**

* Sequestration of an infected focus of pneumonia, e.g. pleuropneumonia in bovine & horses often after prolonged antimicrobial therapy.
* Pulmonary abscesses secondary to ovine estrosis.
* Emboli from endocarditis, caudal or cranial vena caval thrombosis, metritis, mastitis, omphalophlebitis.
* Aspiration pneumonia from milk fever in cows, drenching accident in sheep - residual abscess .
* Penetration by foreign body in traumatic reticuloperitonitis.

**Pathogenesis:**

Pulmonary abscesses may be present in many cases of pneumonia and are not recognizable clinically. In the absence of pneumonia, pulmonary abscess is usually a chronic disease, clinical signs being produced by toxemia rather than by interference with respiration. When the spread is hematogenous and large numbers of small abscesses develop simultaneously, tachypnea occurs. In these animals the respiratory embarrassment cannot be explained by the reduction in vital capacity of the lung. However, in more chronic cases the abscesses may reach a tremendous size and cause respiratory difficulty by obliteration of large areas of lung tissue. In rare cases, erosion of a pulmonary vessel may occur, resulting in pulmonary hemorrhage and hemoptysis.

**Clinical finding :**

1. In typical cases there is dullness, anorexia, emaciation and a fall in milk yield in cattle.
2. The temperature is usually moderately elevated and fluctuating.
3. Coughing is marked. The cough is short and harsh and usually not accompanied by signs of pain.
4. Intermittent episodes of bilateral epistaxis and hemoptysis may occur, which may terminate in fatal pulmonary hemorrhage following erosion of an adjacent large pulmonary vessel.
5. Respiratory signs are variable depending on the size of the lesions.
6. When the abscesses are large (2-4 cm in diameter) careful auscultation and percussion will reveal the presence of a circumscribed area of dullness over which no breath sounds are audible.
7. Crackles are often audible at the periphery of the lesion.
8. Multiple small abscesses may not be detectable on physical examination but the dyspnea is usually more pronounced.
9. There may be a purulent nasal discharge and fetid breath but these are unusual unless bronchopneumonia has developed from extension of the abscess.

**Clinical pathology:**

1. Examination of nasal or tracheal mucus may determine the causative bacteria but the infection is usually mixed and interpretation of the bacteriological findings is difficult.
2. Radiologic examination can be used to detect the presence of abscesses and give some information on its size and location.
3. Hematological examination may give an indication of the severity of the inflammatory process In lung abscesses in foals and adult horses, hyperfibrinogenemia and neutrophilic leukocytosis are common.

**Necropsy findings**:

An accumulation of necrotic material in a thick-walled fibrous capsule is usually present in the ventral border of a lung, surrounded by a zone of bronchopneumonia or pressure atelectasis.

**Differential diagnosis:**

* Splenic and hepatic abscesses
* Tuberculosis
* Focal parasitic lesion such as hydrated cyst
* Pulmonary neoplasm
* Pneumonia and pleurisy

**Treatment:**

**Antimicrobials** :The daily administration of large doses of antimicrobials for several days may be attempted but is usually not effective and slaughter for salvage or euthanasia is necessary.

The recommended treatment of Rhabdococcus equi in foals is a combination of erythromycin 25 mg/ Kg three time daily with rifampicin 5mg/Kg twice daily for 4-9 weeks.

**Pulmonary and pleural neoplasm**

* Primary neoplasms of the lungs, including carcinomas and adenocarcinomas, are rare in animals and metastatic tumors also are relatively uncommon in large animals.
* Pulmonary adenocarcinoma is the most commonly reported primary lung tumor in cattle.
* Squamous cell type tumor through to be a benign papilloma has been in goats.
* Malignant melanomas in adult gray horses
* Lymphomatosis in young cattle
* Pleural mesothelioma
* Thymoma or lymphosarcoma.