Pulmonary Hypertension

Pulmonary hypertension is an increase in pulmonary arterial pressure above normal values due to structural or functional changes in the pulmonary vasculature. Primary pulmonary hypertension occurs in cattle with high-altitude disease. Chronic pulmonary hypertension results in right-Side congestive heart failure due to right ventricular hypertrophy

Etiology

Hypoxemia is a potent stimulus of pulmonary arterial pressure through increased pulmonary vascular resistance induced by pulmonary vasoconstriction. Pulmonary artery pressure can also increase in response to increases in cardiac output.

Alveolar hypoxia causes constriction of the precapillary pulmonary vessels, resulting in pulmonary hypertension.

Condition which may induce hypoxia include:

1- Exposure to high altitude

2- Respiratory impairment secondary to

3- thoracic wall abnormalities

4- Airway obstruction

5- Pneumonia

6- Pulmonary edema

7- Emphysema

8- Pulmonary vascular disease

9- Heaves.

At high altitudes, the low inspired oxygen tension causes hypoxic pulmonary vasoconstriction and hypertension that are common causes of corpulmonale (brisket disease) in cattle.

ATEIECTASIS

Atelectasis is collapse of the alveoli due to failure of the alveoli to inflate or because of compression of the alveoli. Atelectasis is therefore classified as obstruction (resorption), compression or contraction.

1- Obstruction

atelectasis occurs secondary to obstruction of the airways, with subsequent resorption of alveolar gases and collapse of the alveoli. This disease is usually caused by obstruction of small bronchioles by fluid and exudate. It is common in animals with pneumonia or aspiration pneumonia.

2- Compression

atelectasis occurs when intrathoracic (intrapleural) pressure exceeds alveolar pressure, thereby deflating alveoli. This occurs when there is excessive pleural fluid or the animal has a pneumothorax. In large animals it also occurs in the dependent lung or portions of lung in recumbent animals.

The clinical signs of atelectasis are not apparent until there is extensive involvement of the lungs. Animals develop respiratory distress, tachypnea, tachycardia and cyanosis.

Note / Atelectasis is reversible if the primary obstruction or compression is relieved quickly before secondary consolidation and fibrosis occur.

Acute respiratory distress syndrome (ARDS)

in animals occurs in newborns and in adult animals. The disease in some newborn farm animals is related to lack of surfactant. The causes can be infectious(e.g. influenza virus infection), chemical (smoke inhalation) or toxic such as endotoxemia.

pathogenesis

involves a common final pathway that results in damage to alveolar capillaries. The initial injury can be to either the endothelium of pulmonary capillaries or to alveolar epithelium. Damage to these structures leads to extravasation of protein-rich fluid and fibrin with subsequent deposition of hyaline membranes leading to impair respiratory gas exchanges & cause hypoxemia.

Clinical sings

The clinical signs are characteristic of acute, progressive pneumonia. Animals are anxious, tachycardic, tachypneic and have crackles and wheezes on thoracic auscultation. Severely affected animals can be cyanotic.

Treatment

includes administration of anti-inflammatory drugs (NSAIDs with or without glucocorticoids), colloids, antimicrobials and oxygen therapy.

PULMONARY HEMORRHAG

Etiology: Pulmonary hemorrhage is uncommon in farm animals but does occur occasionally in cattie, and exercise-induced pulmonary hemorrhage (EIPH) occurs in 45-75% of exercised horses. Pulmonary hemorrhage also occurs in horses with pulmonary abscesses, tumors or foreign bodies.

Tracheo-bronchoscopy, radiographic and ultrasonographic examinations are useful in identifying the site and cause of the hemorrhage.

In cattle the most common cause is erosion of pulmonary vessels adjacent to lesions of embolic pneumonia associated with venacaval thrombosis and hepatic abscessation. The onset of hemorrhage may be sudden and affected animals hemorrhage profusely and die after a short course of less than 1 hour.

Clinical signs

Marked epistaxis and hemoptysis, severe dyspnea, muscular weakness and pallor of the mucous membranes are characteristic. In other cases, episodes of epistaxis and hemoptysis may occur over a period of several days or a few weeks along with a history of dyspnea.

Diseases of pleura & diaphragm

Hydrothorax & hemothorax

The accumulation of edematous transudate or whole blood in the pleural cavities is manifested by respiratory embarrassment caused by collapse of the ventral parts of the lungs.

Etiology

Hydrothorax and hemothorax occur as part of a number of diseases.

1-Hydrothorax

- As part of a general edema due to congestive heart failure or hypoproteinemia
- As part of African horse sickness or bovine viral leucosis
- Secondary to thoracic neoplasia

2-Hemothorax

- Traumatic injury to thoracic wall, a particular case of which is rib fractures in newborn foals
- During Lung biopsy
- Incase of haemangiosacroma of pleura
- Excessive exercise in the horse

Pathogenesis

Accumulation of fluid in the pleural cavities causes compression atelectasis of the ventral portions of the lungs and the degree of atelectasis governs the severity of the resulting dyspnea. Compression of

the atria by fluid may cause an increase in venous pressure in the great veins, decreased cardiac return and reduced cardiac output.

Extensive hemorrhage into the pleural space can cause hemorrhagic shock.

CLINICAL FINDINGS

In both diseases there is an absence of systemic signs, although acute hemorrhagic anemia may be present when extensive bleeding occurs in the pleural cavity. There is dyspnea, which usually develops gradually, and an absence of breath sounds, accompanied by dullness on percussion over the lower parts of the chest.

The accumulation of the fluid or blood is evident by radiographic or ultrasonagraphic.

CLINICAL PATHOLOGY

Thoracocentesis may yield a flow of clear serous fluid in hydrothorax, or blood in recent cases of hemothorax. The fluid is bacteriologically negative and total nucleated cell counts are low (5×10^9 /liter)

TREATMENT

1- Treatment of the primary condition
2- If the dyspnea is severe, aspiration of fluid from the pleural sac causes a temporary improvement but the fluid usually re-accumulates rapidly.
3- blood transfusion in severe hemothorax.

PNEUMOTHORAX

Pneumothorax refers to the presence of air (or other gas) in the pleural cavity. Entry of air into the pleural cavity in sufficient quantity causes collapse of the lung and impaired respiratory gas change with consequent respiratory distress.

Etiology

Pneumothorax is defined as either spontaneous, traumatic, open, closed, or tension.

- Spontaneous cases occur without any identifiable inciting event.
- Open pneumothorax describes the situation in which gas enters the pleural space other than from a ruptured or lacerated lung, such as through an open wound in the chest wall. Closed pneumothorax refers to gas accumulation in the pleural space in the absence of an open chest wound. Tension pneumothorax occurs when a wound acts as a one-way valve, with air entering the pleural space during inspiration but being prevented from exiting during expiration by a valve-like action of the wound margins. The result is a rapid worsening of the pneumothorax. The pneumothorax can be unilateral or bilateral.

PATHOGENESIS

Entry of air into the pleural cavity results in collapse of the lung. There can be partial or complete collapse of the lung.

- Collapse of the lung results in alveolar hypoventilation, hypoxemia, hypercapnia, cyanosis, dyspnea, anxiety, and hyperresonance on percussion of the affected thorax.
- Tension pneumothorax can also lead to a direct decrease in venous return to the heart by compression and collapse of the vena cava.
- The degree of lung collapse varies with the amount of air that enters the cavity.
- small amounts are absorbed very quickly . but large amounts may cause fatal anoxia.

CLINICAL FINDINGS

There is an acute onset of inspiratory dyspnea, which may terminate fatally within a few minutes if the pneumothorax is bilateral and severe. If the collapse occurs in only one pleural sac, the rib cage on the affected side collapses and shows decreased movement. There is a compensatory increase in movement and bulging of the chest wall on the unaffected side. On auscultation of the thorax, the breath sounds are markedly decreased in intensity and commonly absent. The mediastinum may bulge toward the unaffected side and may cause moderate displacement of the heart and the apex beat, with accentuation of the heart sounds and the apex beat. The heart sounds on the affected side have a metallic note and the apex beat may be absent. On percussion of the thorax on the affected side, a hyperresonance is detectable over the dorsal aspects of the thorax.

PLEURITIS (PLE U R I SY)

Pleuritis refers to inflammation of the parietal and visceral pleura. Inflammation of the pleura almost always results in accumulation of fluid in the pleural space. Pleuritis is characterized by varying degrees of toxemia, painful shallow breathing, pleural friction sounds and dull areas on acoustic percussion of the thorax because of pleural effusion. Treatment is often difficult because of the diffuse nature of the inflammation.

ETIOLOGY

Pleuritis is almost always associated with diseases of the lungs. Pneumonia can progress to pleuritis, and pleuritis can cause consolidation and infection of the lungs.

- **Primary pleuritis** is usually due to perforation of the pleural space and subsequent infection. Most commonly this occurs as a result of trauma,l but it can occur in cattle with traumatic reticuloperitonitis and in any species after perforation of the thoracic esophagus.

Secondary pleuritic

In Cattle :

- Secondary to Mannheimia haemolytica pneumonia in cattle.
- Tuberculosis.
- Sporadic bovine encephalomyelitis.
- Contagious bovine pleuropneumonia.

In horse :

- Rarely case in horse include lymphosacroma , equine infectious anemia

In sheep & goat :

- Pleuropneumonia associated with Mycoplasma spp., including Mycoplasma mycoides subsp. Mycoides and Haemophilus spp.
- Streptococcus dysgalactiae in ewes.

PATHOGENESIS

Contact and movement between the parietal and visceral pleura causes pain due to stimulation of pain end organs in the pleura. Respiratory movements are restricted and the respiration is rapid and shallow. There is production of serofibrinous inflammatory exudate, which collects in the pleural cavities and causes collapse of the ventral parts of the lungs, thus reducing vital capacity and interfering with gaseous exchange. If the accumulation is sufficiently severe there may be pressure on the atria and a diminished return of blood to the heart. Clinical signs may be restricted to one side of the chest in all species with an imperforate mediastinum. Fluid is resorbed

in animals that survive the acute disease and adhesions develop, restricting

movement of the lungs and chest wall but interference with respiratory exchange is usually minor and disappears gradually as the adhesions stretch with continuous movement In all bacterial pleuritis, toxemia is common and usually severe. The toxemia may be severe when large amounts of pus accumulate.

CLIN ICAL FINDINGS

The clinical findings of pleuritis vary from mild to severe. depending on the species and the nature and severity of the inflammation. In peracute to acute stages of pleuropneumonia there are fever, toxemia, tachycardia, anorexia, depression, nasal discharge, coughing, exercise intolerance, breathing distress, and flared nostrils. The nasal discharge depends on the presence or absence of pneumonia. It may be absent or copious and its nature may vary from mucohemorrhagic to mucopurulent. The odor of the breath may be putrid, which is usually associated with an anaerobic lesion.

- Pleural pain (pleurodynia) is common and manifested as pawing, stiff forelimb gait, abducted elbows and reluctance to move or lie down. In the early stages of pleuritis, breathing is rapid and shallow, markedly abdominal and movement of the thoracic wall is restricted.
- The breathing movements may appear guarded, along with a catch at end-inspiration. The animal stands with its elbows abducted and is disinclined to move.
- The application of hand pressure on the thoracic wall and deep digital palpation of intercostal spaces usually causes pain manifested by a grunting sound , a spasm of the intercostal muscles also this may be audible over the area friction sounds during the initial stage of the disease they are not audible when fluid accumulates in the pleural space
- Subcutaneous edema of the ventral body wall extending from the pectorals to the prepubic area is common in horses with severe pleuritis Presumably this edema is due to blockage of lymphatics normally drained through the cranial lymph nodes.
- Pleural effusion due to exudate In cattle, an inflammatory pleural effusion is often limited to one side because the pleural sacs do not communicate.

- Bilateral pleural effusion may indicate either a bilateral pulmonary disease process or non-inflammatory abnormality such as right- sided congestive heart failure or hypoproteinemia, Also Dullness on the percussion the area over the fluid -filled area of the thorax is characteristic of pleuritis

- In the presence pleural effusion both normal & abnormal lung sound are diminished in intensity depending on the amount of effusion.

- Dyspnea may still be evident, particularly during inspiration
- Animals with pleuritis characteristically recover slowly over a period of several days or even weeks.
- The toxemia usually resolves first but abnormalities in the thorax remain for some time because of the presence of adhesions and variable amounts of pleural effusion
- Rupture of the adhesions during severe exertion may cause fatal hemothorax.

- Chronic pleurisy, as occurs in tuberculosis in cattle and in pigs, is usually subclinical, with no acute inflammation or fluid exudation occurring

Diagnosis

1- Clinical signs & examination of the animal (percussion & palpation)
2- Medical energy such as radiographic, ultrasonographic & pleuroscopy

Note / ultrasonographic more realable for detection of pleural fluid in horse and cattle than radiographic

3- Clinical pathology which include thoracentesis (pleurocentesis) to obtain a sample of the fluid for laboratory examination is necessary for a definitive diagnosis. The fluid is examined for its odor, color and viscosity, protein concentration and presence of blood or tumor cells, and is cultured for bacteria. It is important to determine whether the fluid is an exudate or a transudate.

- Pleural fluid from horses affected with anaerobic bacterial pleuropneumonia may be foul-smelling. Examination of the pleural fluid usually reveals an increase in leukocytes up to and protein concentrations of up to 50 giL
- The fluid should be cultured for both anaerobic & aerobic bacteria & mycoplasma spp.

4- hematological examination in peracute bacterial pleuropneumonia in horses and cattle, leukopenia and neutropenia with toxic neutrophils are common.

- In acute pleuritis with severe toxemia, hemoconcentration, neutropenia with a left shift and toxic neutrophils are common.
- In subacute and chronic stages normal to high leukocyte counts are often present. Hyperfibrinogenemia, decreased albumin-globulin ratio and anemia are common in chronic pleuropneumonia.

DIFFERENTIAL DIAGNOSIS

• The presence of inflammatory fluid in the pleural cavity

• Pleural friction sounds, common in the early stages of pleuritis and loud and abrasive; they sound very close to the surface, do not fluctuate with coughing common in the early stages and may continue to be detectable throughout the effusion stage

• The presence of dull areas and a horizontal fluid line on acoustic percussion of the lower aspects of the thorax, characteristic of pleuritis and the presence of pleural fluid

• Thoracic pain, fever and toxemia are common.

The diseases include

1- **Pneumonia** occurs commonly in conjunction with pleuritis and differentiation is difficult and often unnecessary. The increased intensity of breath sounds associated with consolidation and the presence of crackles and wheezes are characteristic of pneumonia.
2- **Pulmonary emphysema** is characterized by loud crackles, expiratory dyspnea, hyperresonance of the thorax and lack of toxemia unless associated with bacterial pneumonia.
3- **Hydrothorax and hemothorax** are not usually accompanied by fever or toxemia and pain and pleuritic friction sounds are not present. Aspiration of fluid by needle

puncture can be attempted if doubt exists. A pleural effusion consisting of a transudate may occur in corpulmonale due to chronic interstitial pneumonia in cattle.

4- **Pulmonary congestion and edema** are manifested by increased vesicular murmur and ventral consolidation without hydrothorax or pleural inflammation.

TREATMENT

The principles of treatment of pleuritis are

a- pain control
b- elimination of infection
c- prevention of complications.

1-Antimicrobial therapy The primary aim of treatment is to control the infection in the pleural cavities using the systemic administration of antimicrobials' which should be selected on the basis of culture and sensitivity of pathogens from the pleural fluid. Before the antimicrobial sensitivity results are available it is recommended that broad spectrum antimicrobials be used. Long term therapy daily for several weeks may be necessary.

2-Drainage and lavage of pleural cavity Drainage of pleural fluid removes exudate from the pleural cavity and allows the lungs to re-expand. Criteria for drainage include:

- An initial poor response to treatment
- Large amount of fluid causing respiratory distress
- Putrid pleural fluid
- Bacteria in cells of the pleural fluid.
- Pleural lavage may assist in removal of fibrin, inflammatory debris, and necrotic tissue; it can prevent loculation, dilute thick pleural fluid and facilitate drainage. One chest tube is placed dorsally and one ventrally; 5-10 L of sterile, warm isotonic saline is infused into each hemithorax by gravity flow. After the infusion the chest tube is re connected to the unidirectional value & lavage fluid is allowed to drain.

3-Thoracotomy has been used successfully for the treatment of pericarditis and pleuritis and lung abscesses in cattle Claims are made for the use of dexamethasone at 0.1 mg/kg BW to reduce the degree of pleural effusion. In acute cases of pleurisy in the horse analgesics such as phenylbutazone are valuable to relieve pain and anxiety, allowing the horse to eat and drink more normally.

4- Fibrinolytic therapy Pleural adhesions are unavoidable and may become thick and extensive with the formation of location which traps to form of pleural fluid,

However Fibrinolytic agents such as streptokinase promote removal, thickening pleural fluid, lyse adhesion & facility drainage of fluid.

EQUINE PLEUROPNEUMONIA

(PLEURITIS, PLEURISY)

Pleuropneumonia of horses is almost always associated with bacterial infection of the lungs, pleura, and pleural fluid.

Etiology Most infections are polymicrobial combinations of *S. equi var. zooepidemicus, Actinobacillus sp., Pasteurella sp., Enterobacteriaceae* and anaerobic bacteria, including *Bacillus fragilis* . Disease due to infection by a single bacterial species occurs. Other causes are *Mycoplasma felis*, penetrating chest wounds and esophageal perforation.

EPIDEMIOLOGY

Pleuropneumonia occurs worldwide in horses of all ages and both sexes, although most cases occur in horses more than 1 and less than 5 years of age, The case fatality rate varies between 5 - 65 %, with the higher rate reported.

- Recent prolonged transport, racing, viral respiratory disease & anesthesia increase the incidence of the disease in horse .
- Aspiration of wound material secondary to esophageal obstruction or dysphagia also cause the disease.

PATHOGENESIS

Bacterial pleuropneumonia develops following bacterial colonization of the lungs with subsequent extension of infection to the visceral pleura and pleural space Organisms initially colonizing the pulmonary parenchyma and pleural space are those normally present in the upper airway, oral cavity, and pharynx, with subsequent infection by Enterobacteriaceae and obligate anaerobic bacteria Bacterial colonization and infection of the lower airway is attributable to either massive challenge or a reduction in the efficacy of normal pulmonary defense mechanisms or a combination of these factors.

- Confinement the head elevated for 12-24 hours, such as occurs during transport of horses or decreases mucociliary transport and increases the entering of thorax at difficult location.
- Bacterial multiplication in pulmonary parenchyma is associated with the influx of inflammatory cells, principally neutrophils, tissue destruction and accumulation of cell debris in alveoli and airways. Infection spreads both through tissue and via airways.
- Infection spreads both through tissue and via airways.
- Extension of inflammation, and later infection, to the visceral pleura and subsequently pleural space causes accumulation of excess fluid within the pleural space.
- Pleural fluid accumulates because of a combination of excessive production of fluid by damaged pleural capillaries (exudation) and impaired reabsorption of pleural fluid by thoracic duct .

CLINICAL FINDING

The acute disease is characterized by the sudden onset of a combination of fever, depression, inappetence, cough, exercise intolerance, respiratory distress, and nasal discharge.

The respiratory rate is usually elevated as is the heart rate.

Nasal discharge ranges from serosanguineous to mucopurulent, is usually present in both nares and is exacerbated when the horse lowers its head.

TREATMENT

1- The principle treatment by using broad spectrum antimicrobial therapy such as Procaine penicillin G 22,000 – 44,000 IU B.W, I.M. / 6 hrs
2- Thoracic drainage.
3- Pleural lavage.

RHINITIS

Rhinitis (inflammation of the nasal mucosa) is characterized clinically by sneezing, wheezing, and stertor during inspiration and a nasal discharge that may be serous, mucoid, or purulent in consistency depending on the cause.

ETIOLOGY

Rhinitis usually occurs in conjunction with inflammation of other parts of the respiratory tract. It is present as a minor lesion in most bacterial and viral pneumonia such as:

In Cattle

1- Catarrhal rhinitis in infectious bovine rhinotracheitis, adenoviruses.

2-Ulcerative/erosive rhinitis in bovine malignant catarrh, mucosal disease, rinderpest.

3-Rhinosporidiosis caused by fungal infection.

4-Bovine nasal eosinophilic granuloma due to Nocardia Sp.

In horse

Glanders, strangles, epizootic lymphangitis equine infectious rhinopneumonitis.

In sheep & goat

Blue tongue, orf, sheep pox, oestrus ovis infestation, allergic rhinitis, purulent discharge & otitis associated with pseudomonas aerogenous in sheep shower with contaminated wash.

PATHOGENESIS

- Rhinitis is of minor importance as a disease process except in severe cases when it causes obstruction of the passage of air through the nasal cavities.
- Its major importance is as an indication of the presence of some specific diseases.
- The type of lesion produced is important.
- The erosive and ulcerative lesions of rinderpest, bovine malignant catarrh and mucosal disease, also lesions of glanders, melioidosis, and epizootic lymphangitis.

CLINICAL FINDINGS

- The primary clinical finding in rhinitis is a nasal discharge, which is usually serous initially but soon becomes mucoid and, in bacterial infections, purulent. Erythema, erosion, or ulceration may be visible on inspection.
- The inflammation may be unilateral or bilateral. Sneezing is characteristic in the early acute stages and this is followed in the later stages by snorting and the expulsion of large amounts of mucopurulent discharge.
- A chronic unilateral purulent nasal discharge lasting several weeks or months in horses suggests nasal granulomas associated with mycotic infections

Differential Diagnosis

Allergic rhinitis in cattle must be differentiated from fungal infection but Rhinitis in the horse must be differentiated from inflammation of the facial sinuses

TREATMENT

Specific treatment aimed at control of individual causative agents. Thick tenacious exudate that is causing nasal obstruction may be removed gently and wash nasal cavities with saline.

Treatment with broad spectrum antimicrobial therapy.