**Diseases of the Respiratory System**

**Diseases of the lungs**

**Pulmonary Congestion and Edema**

**Assistant Professor** **Dr. Hayder Badri Abboud**

**B.V.M.&S.,M.S.V.M.&S.,Ph.D.**

**- Pulmonary congestion is caused by an increase in the amount of blood in the lungs due to engorgement of the pulmonary vascular bed. It is sometimes followed by pulmonary edema when intravascular fluid escapes into the parenchyma and alveoli.**

**- The various stages of the vascular disturbance are characterized by respiratory compromise, the degree** **depending upon the amount of alveolar** **air space which is lost.**

**ETIOLOGY  
- Pulmonary congestion and edema is a common terminal event in many diseases but is frequently overshadowed by other disturbances.  
- Congestion that is clinically apparent may be primary when the basic lesion is in the lungs or secondary when it is in some other organ, most commonly the heart.  
- Pulmonary edema occurs because of imbalances in the Starling forces across the pulmonary capillary.  
- From a clinical perspective, the common of the proximate causes of pulmonary edema are injury to the endothelium capillary with pulmonary protein -rich fluid goes into the interstitial spaces, elevated blood pressure in the in the alveolar capillaries, or low plasma oncotic pressure.**

**-Damage to pulmonary vascular endothelium intoxications can occur in infectious Physical subsequent leakage of diseases (e.g. African horse sickness) or (endotoxemia) .  
- Physical injury, including inhalation of excessively hot air or smoke, can damage the alveolar epithelium with secondary damage to capillary endothelium.  
- Elevated pulmonary capillary pressure occurs in left-sided heart failure (ruptured chordae tendineae of the mitral valve) and during strenuous exercise by horses.   
- Low plasma oncotic occurs in diseases causing hypoproteinemia but is rarely a cause for pulmonary edema by itself, although it contributes to the pulmonary edema in hypoproteinemic animals administered large volumes of fluids intravenously.**

**Primary pulmonary congestion  
Early stages of most cases of pneumonia Inhalation of smoke and fumes.  
- Anaphylactic reactions  
- Hypostasis in recumbent animals  
- Yew (*Taxus* sp.) intoxication.  
- Race horses with acute severe exercise-induced pulmonary hemorrhage .  
Secondary pulmonary congestion  
Congestive heart failure (cardiogenic pulmonary edema), including ruptured chordae tendineae of the mitral valve, and left-sided heart failure.  
Pulmonary edema  
Pulmonary edema as a sequel to pulmonary capillary hypertension or pulmonary microvascular damage occurs in:**

**- Congestive heart failure and acute heart failure, e.g. the myocardial form of enzootic muscular dystrophy in inherited myocardiopathy of Hereford calves; Inhalation of smoke or manure gas.  
- Transient upper airway obstruction in the horse (negative pressure pulmonary edema)  
- After general anesthesia in horses.  
- Yew (Taxus sp.) intoxication.  
- Exercise-induced pulmonary edema in race horses  
- Specific diseases, including: East Coast fever in cattle; the pulmonary form of African horse sickness; poisoning with organophosphates, alpha-naphthyl thiourea.(ANTU) or ionophore antibiotics; plant poisonings.**

**- Doxycycline intoxication of calves.   
- Clostridium perfringens type D Epsilon .**

**PATHOGENESIS  
- In pulmonary congestion, ventilation is reduced and oxygenation of the blood is impaired. Oxygenation is reduced by the decreased rate of blood flow through the pulmonary vascular bed. Hypoxemic anoxia develops and is the cause of most of the clinical signs that appear.  
- Hypoxemia abnormalities, occurs in pulmonary  
abnormalities edema because of ventilation/perfusion diffusion (although this is usually a minor contributor to the hypoxemia), and hypoventilation caused by the physical obstruction of airflow by fluid and foam in the airways.  
- The edema is caused by damage to the capillary walls by toxins or anoxia or by transudation of fluid due to increased hydrostatic pressure in the capillaries.**

**-Filling of the alveoli, and in severe cases the bronchi, effectively prevents gaseous exchange.  
 - Following smoke inhalation, diffuse tracheo-bronchial mucosal sloughing occurs, which, if progressive, causes separation of epithelium and the development of pseudomembranous casts, which may cause partial or complete airway obstruction.  
- Pulmonary edema is also extensive.  
 CLINICAL FINDINGS  
 In most severe form of pulmonary congestion and edema**

**- The depth of respiration is increased to the point of extreme dyspnea with the head extended, the nostrils flared and mouth-breathing.  
- Breathing movements are greatly exaggerated and can be best described as heaving; there is marked abdominal and thoracic movement during inspiration and expiration.**

**- A typical stance is usually adopted, with the front legs spread wide apart, the elbows abducted and the head  
hung low.  
- The respiratory rate is usually increased especially if there is hyperthermia, which occurs in acute anaphylaxis and after violent exercise as well as in the early stages of pneumonia.  
- The heart rate is usually elevated (up to IOO/min) and the nasal mucosa is bright red or cyanotic in terminal cases.  
- In acute pulmonary congestion there are harsh breath sounds but no crackles are present on auscultation.  
- When pulmonary edema develops, loud breath sounds and crackles are audible over the ventral the lungs.  
- In long-standing cases there may be emphysema with crackles and wheezes of the dorsal parts of the lungs, especially if the lesion is caused by anaphylaxis.**

**- Coughing is usually present but the cough is soft and moist and is not painful .  
- A slight to moderate serous nasal discharge occurs in the early stage of congestion but in severe pulmonary edema this increases to a voluminous, frothy nasal discharge, which is often pink colored due to blood.  
The primary importance of pulmonary congestion is as an indicator of early pathological changes in the lung or heart.  
Spontaneous recovery occurs quickly unless there is damage to alveolar epithelium, or myocardial asthenia develops.  
Severe pulmonary edema has much greater significance and usually indicates a stage of irreversibility.  
 Death in cases of pulmonary edema is accompanied by asphyxial respiratory failure .**

**Smoke inhalation in horses is characterized by:   
- Polypnea and dyspnea.  
- Diffuse wheezes throughout the lungs.  
- Coughing.  
- A broncho-interstitial pattern radiographically.  
- The horse may expectorate large proteinaceous tracheo-bronchial casts.   
- The prognosis is good if affected animals can survive the initial stages of pulmonary damage and secondary organ involvement.   
CLINICAL PATHOLOGY  
- Laboratory examinations are of value only in differentiating or the causes of the congestion edema.  
- Bacteriological examination of nasal swabs and a complete hematological examination, looking particularly for the presence of eosinophilia,are the standard exams that are done.**

**NECROPSY FINDINGS  
In acute pulmonary congestion the lungs are dark red in color. Excessive quantities of venous blood exude from the cut surface.   
 Histologically the pulmonary capillaries are markedly engorged and some transudation and hemorrhage into alveoli is evident.  
 Macroscopic findings in pulmonary edema include swelling and loss of elasticity of the lungs, which pit on pressure. They are usually paler than normal. Excessive quantities of serous fluid exude from the cut surface of the  
lung.  
Histologically there are accumulations of fluid in the alveoli and parenchyma.**

**DIAGNOSIS  
-The diagnosis of pulmonary congestion and edema is always difficult unless there is a history of a precipitating cause such as an infectious disease, strenuous exercise,  
ingestion of toxicants, or inhalation of smoke or fumes.  
- Pneumonia usually presents itself as an alternative diagnosis and a decision cannot be based entirely on the presence or absence of pyrexia.   
- Bacterial pneumonia is usually accompanied by some toxemia.  
- Response to antibacterial treatment is one of the best indications, the only variable being the tendency for congestion and edema of allergic origin to recover spontaneously.  
- It is advisable to treat the animal for both conditions.**

**TREATMENT  
The principles of treatment of pulmonary congestion and edema are one or more of: reduction of pulmonary capillary pressure (by reduction either of pulmonary venous or pulmonary arterial pressure); alleviation of pulmonary mcrovascular damage; and correction of low plasma oncotic pressure.  
- Correction of the primary cause as listed under etiology.**

**-Affected animals should be confined at rest in a clean, dry environment and exercise avoided.   
- Pulmonary capillary pressure can be reduced by adminis-tration of furosemide and pump function improved by administration of drugs that improve myocardial function (digoxin) or decrease afterload (arterial vasodilators) .**

**administration of furosemide (1-2 mg/kg intravenously) .  
 Administration of anti-inflammatory drugs including NSAIDs or glucocorticoids is indicated in animals in which microvascular damage is suspected. These drugs are used to treat, among other diseases, smoke inhalation of horses.  
- Plasma oncotic pressure can be increased by i/v infusion of plasma (10-40 mL/kg) or synthetic colloids such as hetastarch.**

**- Oxygen should be administered to hypoxemic animals in conjunction with other specific treatments .**

**Special diseases  
- When edema is due to organophosphate poisoning prompt administration of atropine may reduce fluid transudation.**

**- In these cases the animal is in considerable danger and repeated injections may be necessary.   
 - Epinephrine is recommended in pulmonary edema due to anaphylaxis. 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 or cor pulmonale. It will have an immediate pharmacological effect, which may be followed by the use of a corticosteroid to maintain vascular integrity and to decrease permeability of pulmonary vessels.**

**- Antihistamines are commonly used in conjunction with epinephrine for the treatment of acute pulmonary edema due to anaphylaxis.**

**-The use of the kinins, prostaglandins and slow-release substances may be more important.**

**- Acetylsalicylic acid was more effective than antihistamines or antiserotonin agents in providing symptomatic relief in experimental acute interstitial pneumonia of calves.  
- Epinephrine is the drug of choice for the emergency treatment of pulmonary edema due to anaphylaxis.**

**References**

**O. M. Radostits, C.C.Gay, K. W. Hinchcliff, and**  **P. D. Constable ( 2011 ): VETERINARY MEDICINE**

**A textbook of the diseases of cattle, horses, sheep, pigs and goats,10thedition. Publisher SAUNDERS. www.elsevierhealth.com**