ASPIRATION PNEUMONIA

(Foreign-body pneumonia, Inhalation pneumonia, Gangrenous pneumonia)

- Aspiration pneumonia is characterized by pulmonary necrosis due to inhalation of foreign material.

**Aetiology and Epidemiology**
- Faulty administration of medicines
- Drenching — animal’s tongue is drawn out, head is held high, or when the animal is coughing or bellowing.
- In sheep, poor dipping technique may cause aspiration of fluid.
- Inhalation of irritant gases or smoke is an infrequent cause.
- Aspiration of vomitus
- Disturbances to swallow, anesthetized or insensible animals, vagal paralysis, acute pharyngitis, cleft palate, abscesses or tumours in pharynx,

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Aspiration pneumonia / Acute bronchopneumonia

- Cranio-ventral consolidation.
- Monogastric animals die more acutely (shock) than polygastric animals.
- Aspiration of gastric contents or drugs typically results in a bronchopneumonia and not always is symmetrical, particularly if aspiration occurs during recumbency or surgery.
- The lesions, depending on severity and composition of aspirated material, vary from suppurative to fibrinous to gangrenous.
- Sometimes, histology is required to confirm food particles in the lung.
ASPIRATION PNEUMONIA

Aspiration pneumonia / Horse

- These lungs are from a horse that died 24 hours after a "milk-shake" was given by the owner prior to a race.
- Note: large cranioventral consolidation involving 60-70% of the pulmonary parenchyma. A large portion of the cranial lobes are covered with a thick layer of fibrin. The entire lungs were also edematous hence the rib imprints on the pleural surface.

PATHO-PHYSIOLOGY

- History
  - Horses may develop fever of 104-105°F (40-40.5°C)
  - Pyrexia is also seen in cats, dogs, and less commonly in cattle.
  - The pulse is accelerated, and respiration is rapid and labored.
  - Sweetish, fetid breath characteristic of gangrene.
  - Purulent nasal discharge that sometimes is tinged reddish brown or green.
  - May be oil droplets (aspirated contents), can be seen in the nasal discharge or expectorated material.
  - On auscultation, fluid sounds
  - In cows: toxemia is usually fatal within 1-2 days.
  - Mortality is high in all species.
  - Recovered animals often develop pulmonary abscesses.

LESIONS:

- The pneumonia — antero-ventral — unilateral or bilateral
- Marked congestion and interlobular oedema.
- Bronchi are hyperemic and full of froth.
- The pneumatic areas – cone-shaped with the base toward the pleura.
- Suppuration and necrosis — foci — soft or liquefied, reddish brown, and foul smelling.
- There usually is an acute fibrinous pleuritis, often with pleural exudate.

HAEMORRHAGIC SEPTICAEMIA
HAEMORRHAGIC SEPTICAEMIA
Acute, highly fatal septicaemic disease of cattle and buffaloes caused by certain serotypes of *Pasteurella multocida* characterized by oedema in the head-throat-brisket region.

**AETIOLOGY AND EPIDEMIOLOGY**
- The disease is caused by two serotypes B₂ and E₂, type B in buffalo, while type E in African cattle
- HS and pneumonic pasteurellosis are two different entities
- The lipopolysaccharide produced is endotoxin

• Three main antigenic component
  - B-antigen: Type specific polysaccharide, adsorbed to red cells in indirect haemagglutination procedure,
  - α-complex: Probably a polysaccharide protein complex, closely adherent to the cell wall, immunogenic, probably somewhat labile
  - γ-antigen: LPS found in organisms from all variants, derived from cell wall, each with one or more antigenic determinants responsible for different O or somatic serological varieties

HAEMORRHAGIC SEPTICAEMIA
- Organism also produces neuraminidase and B₂ strain produce hyaluronidase as well
- Disease occur in Southern and Southeast Asia and middle east as well as in Africa, Australia, Canada, Western Europe, South America
- The disease occur in monsoon season (wet season)
- In Europe, the disease is reported more in Italy
- Disease occurred in Japan in 1923 but no epidemic since 1954
- HS is known to occur occasionally in American bison
- Disease in Africa is caused by a new capsular type, viz., type E which infect cattle more frequently than buffalo
- The disease in Asia has emerged as of great economic importance
- In Sri Lanka mortality range from 20 to as high as 98 %
- In Thailand, 4000 to 10000 deaths per year are caused by this disease.

Buffaloes are more susceptible than cattle
- Disease in goats is low
- The carrier animals harbor the infection for long time
- Infection occur by direct contact
- The organism die soon in dry soil
- moist conditions and the presence of blood & tissue fluids may prolong the survival for few days
- In virgin areas, the disease cause very high mortality in all age groups
- It is generally accepted that this disease causes more losses in young animals under two years of age than adults
- Incidence of disease is found to be 4-5 times greater in large herds (over 50 animals)
- Some animals are naturally immuned to the infection and it was assumed that presence of strains of *P. multocida* antigenically related to type B₂ or other bacteria with common antigenic components may account for this natural immunity
HAEMORRHAGIC SEPTICAEMIA

**PATHOPHYSIOLOGY**

- Infection occurs by inhalation or ingestion
- Initial site of replication is proposed to be tonsillar region
- Endotoxins (LPS – a cell wall complex) are the main cause of pathological changes and death
- Endotoxins not only are antigenic but also stimulate the production of IL-1 which is responsible for malaise (unease), temperature changes and weakness
- These endotoxins also trigger arachidonic acid metabolite production such as prostaglandins and leukotrienes that cause vasodilation, hypotension and other circulatory problems
- Endotoxins also activate the complement pathway resulting in platelet aggregation and release of pro-coagulants resulting in thrombus formation
- Being extracellular organism, the immunity produced is humoral with polymorphonuclear response and little monocytic response

**CLINICAL COURSE**

- The incubation period of the disease is short (30 hours to 2-3 days)
- The course of disease is also short especially in buffaloes than cattle
- Death occurs from 6 to 24 hours after the first recognized signs.
- The typical clinical syndrome of HS is characterized by three phases
  - The initial phase is temperature elevation with inappetence and sometimes salivation
  - Second phase is respiratory distress with profuse salivation and nasal discharge
  - In the third phase recumbency sets in leading to terminal septicemia
- Under field conditions, often the initial phase is not observed
- In many cases submandibular edema becomes evident and may spread to the brisket region and occasionally down the forelimbs
HAEMORRHAGIC SEPTICAEMIA

AT POSTMORTEM
• On opening the first lesion is the subcutaneous infiltration of a yellow *sero-gelatinous* (or rarely *sero-sanguineous*) fluid in the submandibular, throat, pharyngeal and brisket region.
• There may be additional fluid in the *pericardial sac* and the pleural cavity.
• Petechial and *ecchymotic haemorrhages* are seen to a varying degree on the *epicardium* of the heart, particularly involving the auricular region and base of the ventricles.
• In some cases, there is marked *serofibrinous pericarditis, pleuritis* and *peritonitis* with adhesions.
• The principal change in the *lungs* is *congestion* but there may also be some consolidation with thickening of the interlobular septa.

MORBIDITY AND MORTALITY
• *Husbandry, weather* and immunity affect morbidity.
• In endemic areas, from 10 to 50 % of the cattle or buffalo populations acquire solid immunity through exposure or subclinical infection.
• Close herding and wetness predispose to an increased morbidity.
• Most animals that develop clinical signs die – case fatality rate is high.

DIAGNOSIS
• History, clinical signs and gross pathological lesions
• *Isolation* of the organism from *blood, bone marrow*
• Serological typing
• *Animal inoculation tests* (mouse)

DIFFERENTIAL DIAGNOSIS
• Must be differentiated from causes of sudden death including
  – Snake bite,
  – Lightening,
  – Anthrax,
  – Rinderpest
  – Blackleg
CONTAGIOUS BOVINE PLEUROPNEUMONIA

Contagious bovine pleuropneumonia (CBPP) is a highly infectious acute, subacute, or chronic disease, primarily of cattle, affecting the lungs and occasionally the joints, caused by Mycoplasma mycoides mycoides.

Etiology
• Contagious bovine pleuropneumonia is caused by M. mycoides mycoides small-colony type (SC type).
• M. mycoides mycoides large-colony type is pathogenic for sheep and goats but not for cattle.
• Does not survive in meat or meat products and does not survive outside the animal in nature for more than a few days.
• Many of the routinely used disinfectants effectively inactivate the organism.

Host Range
• Cattle (Bos taurus), zebu (Bos indicus) and water buffalo (Bubalus bubalis) are susceptible
• Wild bovids and camels are resistant

Geographic Distribution
• CBPP is endemic in most of Africa.
• It is a problem in parts of Asia --- India, Pakistan and China.
• Periodically, CBPP occurs in Europe, and outbreaks within the last decade have occurred in Spain, Portugal, and Italy.
• Eradicated from the United States in the 19th century.

Transmission
• Spread by inhalation of droplets from an infected animal.
• Relatively close contact is required
• It is widely believed that recovered animals harbor infectious organisms within a pulmonary sequestrum, may become active shedders when stressed.
• Transmission up to several kilometers has been suspected under favorable climatic conditions.
CONTAGIOUS BOVINE PLEUROPNEUMONIA

Incubation Period
- Generally quite long.
- It has been shown that healthy animals placed in a CBPP-infected herd may begin showing signs of disease 20 to 123 days later.
- During an outbreak of natural disease, only 33% of animals present symptoms, 46% are infected but have no symptoms (subclinical forms) and 21% seems to be resistant.

Clinical Signs
- Depression, in-appetence and fever
- Coughing may be the next sign
- Evidence of thoracic pain and an increased respiratory rate.
- As pneumonia progresses, animals are inclined to stand with elbows abducted.
- Auscultation of the lungs reveals variety of sounds, depending
  - Crepitating rales, and pleuritic friction rubs are all possible.
  - Percussion over affected areas reveals dullness.
- When pulmonary involvement is extensive and severe --- labored respiration, sometimes open-mouth breathing.

Gross Lesions
- The gross pathologic features of CBPP are quite characteristic.
  - extensive and marked inflammation of the lung and associated pleura.
- In severe cases, abundant fluid in the thoracic cavity (upto 30 liters).
  - The affected pulmonary parenchyma is odorless and often has all stages of lesions with both acute and chronic inflammatory changes adjacent to one another.
  - The predominant gross change is consolidation, or thickening, of individual lobules, widened interlobular septa, resulting marbled appearance.
  - Interlobular septa become distended first by edema, then by fibrin, and finally by fibrosis.
  - The pleura may be very thickened, fibrosed, often resulting in adhesions between parietal and visceral pleurae.
CONTAGIOUS BOVINE PLEUROPNEUMONIA

Gross Lesions
• within an affected lung will be found a sequestrum – area with coagulative necrosis and sealed off.
• Such sequestra may even be found in recovered animals.
• The organism can survive within these sequestra for months or possibly longer.

Morbidity and Mortality
• It is not thought to be a highly contagious disease.
• With increased confinement of animals, morbidity rises.
• The mortality – 10 to 70%

Diagnosis
• Clinical diagnosis of CBPP is difficult.
• At postmortem the gross lesions of CBPP are somewhat distinct.
• Often there is an extensive deposition of fibrin and a large quantity of straw-colored fluid in the thoracic cavity with a prominent marbling of pulmonary parenchyma.
• Generally, all stages of pathologic changes, from acute through to chronic, are present in one animal.
• In some chronic cases the nodules of inflammation may not be readily apparent from the pleural surface but can be palpated within the parenchyma.
• Unlike many other pneumonias, CBPP is often unilateral.

Specimens for Laboratory
• From a live animal, nasal swabs, transtracheal washes, or pleural fluid obtained by thoracic puncture all provide good samples for isolation attempts.
• From a dead animal that has had severe clinical disease, the best specimens to submit are affected lung, swabs of major bronchi, tracheo-bronchial or mediastinal lymph nodes, and joint fluid from those animals with arthritis.

Laboratory Diagnosis
• A definitive diagnosis is made by isolating and identifying the organism.
• Serology is helpful in the diagnosis of CBPP.

Differential Diagnosis
• bovine pasteurellosis: bovine pasteurellosis spread much more rapidly and consequently the epidemiologic picture may be distinct.

WARNING
Besides lung cancer and cardiovascular diseases.... there is also unequivocal scientific association between Cigarette smoking and pulmonary emphysema.