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Diseases of equine

Part -1

Lecturer Affiliation:
Ahmed M.A. Alsaidya *, PhD*
Asst. Prof. , Department of Pathology and Poultry Diseases College of Veterinary Medicine, University of Mosul, Mosul, Iraq

Summary:

Strangles

It is an acute contagious disease mainly in young horses which is caused by *Streptococcus equi* (sub speciesequi). There is a catarrhal inflammation of the upper respiratory tract and formation of abscesses in the submaxillary and pharynagallymph nodes.

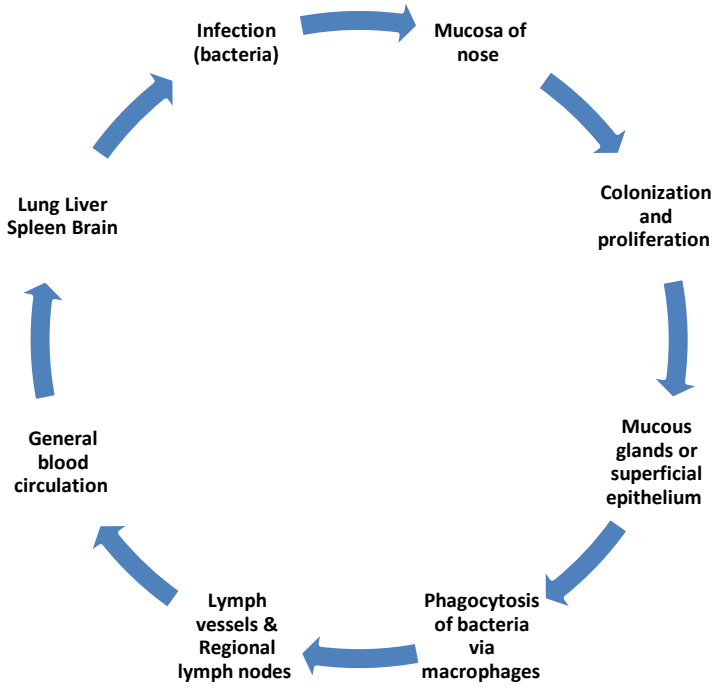
The causative agent of strangles is *Streptococcus equi* which is gram positive.

The recovered horses exist as carriers which can spread infection to other young horses. Organisms like *Str. equi* can enter the blood stream produces fever and set up metastatic lesions like abscesses. Horses over 5 years of age are resistant to *Str. equi* infection.

Contaminated utensil, bedding, food and drinking water etc., spread the infection to healthy young horses. Contact with diseased horses also spreads infection to healthy ones. Infection can enter the body by ingestion, inhalation and wound infection with discharges etc., of the animal patients.

The incubation period varies form **3** to **8** days in the horses. The important clinical signs are fever, anorexia, depression, submandibular as pharyngeal lymphadenopathy marked by abscessation, rupture of the abscesses and purulent nasal discharge.

Pathogenesis



Pathology

The main lesions are:

(1) Catarrhal inflammation of the nasal and pharyngeal mucous membrane. This results in formation of a thick yellow discharge from the nostrils.

(2) Submaxillary, pharyngeal, parotid and cervical lymph nodes are swollen and can undergo suppuration which may burst into the pharynx.

(3) Rise of temperature (pyrexia).

(4) Catarrhal bronchitis, pneumonia, pleurisy and pericarditis.

(5) Pyaemia and abscesses in the different internal organs. Septic peritonitis may arise from bursting of the abscesses in the mesenteric lymph nodes. Abscesses may occur in the lymph nodes like mediastinal, axillary, inguinal or popliteal lymph nodes etc.

Abscesses of the retropharyngeal lymph nodes drain into the guttural pouches. Empyema in such structures can be noticed. In short, caseous lymphadenopathy with rhinitis and pharyngitis, pneumonia and metastatic infection are main changes in equine strangles.

Sequelae and complications: Complications occur in about 20% of the cases. The most common fatal complication is the development of suppurative necrotic

bronchopneumonia secondary to the aspiration of pus from internal ruptured abscesses or metastatic infection of the lungs. Guttural pouch infection with empyema may also result from rupture of abscesses in the retropharyngeal lymph node. Metastatic infection, also known as "**bastard strangles**", results in the formation of abscesses in any organ or body site, but most commonly in the lungs, mesenteric lymph nodes, liver, spleen, kidneys and brain. Purpura hemorrhagica may occur as sequelae of S. equi infection as well.

Diagnosis

It is based on the symptoms, lesions and isolation of *Str. equi* from the nasal discharge and abscesses.

Differential diagnosis: Strangles should be differentiated clinically from other upper respiratory tract diseases of horses. Chronic weight loss due to metastatic infection should be differentiated from equine infectious anemia, parasitism, inadequate nutrition, and neoplasia.

GLANDERS

It is a contagious bacterial disease primarily affecting the horses and now-a-days only sporadic incidence of this disease is noticed.
Man and members of the cat family are also be affected.

The obligate causative organism is *Burkholderia (Pseudomonas) mallei* which produces an acute or more usually a chronic disease in horses, mules and asses. This disease ends fatally and fibro-caseous nodules are formed in the upper respiratory tract, lungs and skin etc.

Pigs and cattle are immune to this disease. Ingestion of food or water contaminated with discharges of excretion of the infected animals produces it in healthy animals. Skin infection through contamination of open wounds in the body may occur in some cases.

Infection by inhalation is unusual.

Incubation period ranges from 2 weeks to 2 or 3 months or longer.

The organisms are rod shaped, Gram negative, non-sporulating and non-motile .

Pathogenesis

The incubation period is a few days to several weeks. It varies with the form of the disease: septicemia or localized disease usually becomes apparent after 1 to 5 days, while the pulmonary form typically develops after 10 to 14 days.

After infection occurs primary multiplication and lesions at point of entry, then the organism pass to regional lymph nodes in which they propagate and via lymphatic it reach

to blood causing septicemia (acute form) or bacteremia (chronic form). Then localization of organism occurs in lung, other viscera as liver, spleen, nasal mucosae and skin with formation of typical nodules (pyogranulomatous).

Glanders can be divided into the following types:

(i) Nasal glanders
(ii) Pulmonary glanders
(ill) Cutaneous glanders

It may develop in either acute, sub acute or chronic form in animals. The characteristic lesions in the glandered animals are pneumonia, nodules or ulcers in the respiratory tract (e.g., larynx and trachea etc.), skin and subcutaneous tissues

along lymphatics. Vary-sized nodules in the subcutaneous tissues of hind limbs, neck, face and liver etc. are called farcy buds.

(i) Nasal Glanders

Sub mucosal nodules are found on the nasal septum and these nodules break down to give rise to shallow, crater like ulcers or erosions which liberate a thick, sticky or oily yellowish brown discharge. Later, these ulcers turn into stellate shaped scars. Swelling is noticed in regional lymph nodes.

The ulcers on the nasal septum may have characteristic punched out appearance. There is an irregular contour of these lesions with raised or eroded borders. Ulcers can penetrate into cartilage to cause perforations. The discharge from the ulcers may be often blood tinged in nature. Usually isolated ulcers are found in the larynx and trachea.

Ulcers can also be found on the turbinate bones and in the guttural pouches and Eustachean tubes.

(ii) Pulmonary Glanders

Greyish, firm round encapsulated nodules like tubercles of Mycobacterium tuberculosis infection are found in the lungs.

Diffuse pneumonic changes may be present in such lungs. The nodules can be found sub-pleurally like small shots in the lung parenchyma.

The nodules are red at the earliest stages but later show yellowish centers projecting above the surrounding red zone of hyperaemia. Later, yellowish grey nodules are formed in the lungs from such lesions.

Capsules are found in the old lesions. Pleurisy is found with a covering of fibrinous material.

Bronchial and thoracic lymph nodes may show degenerative changes or abscesses.

Microscopically, the lesions in the lungs show alveoli filled with leucocytes and the alveolar walls can be seen disappearing or disintegrating at places. The degenerative necrotic changes occur in the affected tissues with appearance of nuclear chromatin as fragmented or scattered particles (phenomenon of karyorrhexis). At peripheral areas of such changes, red cells and fibrinous material are found and the surrounding lung tissue is hyperaemic.

Endothelioid cells are seen around these central degenerated areas in the older nodular lesions. Necrotic center with surrounding endothelioid cells can be found marginal to fibrous capsule.

Giant cells are found in such areas and calcification may be present occasionally. Highly virulent strains of P.mallei produce acute lesions marked by necrosis and disintegration of the cellular elements where as endothelioid cells, fibrous tissue and giant cells are noticed in the less acute lesions i.e., subactue or chronic ones.

Conversion of lesions of chronic glanders into acute ones is seen in cases of acute glanders arising from chronic glanders. Catarrhal or croupous pneumonia and haemorrhagic infards are found in the pulmonary glanders.

in addition to nodular changes. Such nodular lesions are also
found in the organs like liver and spleen. These nodules may be firm and grey with central softening in these organs.
Submaxillary lymph nodes are most commonly infected and show enlargement, oedema and one or two yellowish-grey centres. Later, these become hard, fibrous and fixed to the jaw of horses.

(iii) Cutaneous glanders

It is also called farcy. Nodules appear along the lymphatic channels, particularly often on limbs and break through the skin discharging a thick, sticky and yellowish grey pus. Such ulcers heal very slowly. Farcy is characterized by chronic lymphangitis and lymphadenitis affecting one or more limbs(especially the hind limbs).

Small round nodules (farcy buds) appear in chains in subcutaneous tissues along the course of lymph channels. These buds turn into abscesses which later give rise to ulcers. Such lesions can be found on
face, neck and body of the infected horses.

Diagnosis

It is based on the following :
(i) Symptoms, lesions and detection and isolation of P. mallei from glanders lesions, nasal discharge, ulceration, chronic cough, submaxillary lymphadenitis. Cutaneous lesions help diagnosis of glanders in the affected animals.
(ii) Cultures from the lesions of glanders.

Mallein Test

It is done by subcutaneous, intradermo-palpebral, ophthalmic and cutaneous methods. Intradermo-palpebral mallein test is the most reliable one. 0.1 ml of concentrated mallein is injected into the dermis of the lower eyelid about a one-

fourth of inch below the lashes. A voluminous oedema develops in the positive cases. The oedema is very hot and painful one.

Direct smears are made from a farcy bud and stained by Gram's method after fixation over heat. Films are examined for the presence of slender Gram negative rods. For cultural examination, pus is inoculated on to blood agar plates and the plates are examined after 3 - 4 days for the
growth of organisms. Animal inoculation is done to diagnose glanders.

Pus or contaminated material or culture is inoculated into two male guinea pigs subcutanceously and examination of guinea pigs is done for the development of orchitis daily after 3 days. Complement fixation test confirms its diagnosis.

Differential Diagnosis

- Strangles (*Streptococcus equi*);
- Epizootic lymphangitis (*Histoplasma farciminosum*);
- Sporotrichosis (*Sporothrix schenckii*)
- Ulcerative lymphangitis (*Corynebacterium pseudotuberulosis*)

African Horse Sickness

African horse-sickness is an infectious but not contagious febrile seasonal disease of horses which is caused by a filterable virus (an Orbivirus of the family Reoviridae).

It is characterized by fever, oedema of the lungs and subcutaneous tissues in the affected animals. Culicoides are responsible for its transmission to healthy horses from diseased ones. Standing or stagnant water and warm humid conditions give a suitable environment for the multiplication of these vectors.

Antigenically, different types of viruses (1-9-main immunological types) have been identified and the virus is present in the blood, other affected tissues and even in the fetus of the affected animals. Horses are highly susceptible to infection and donkeys can also be affected.

Pathogenesis

Horses is bitten by an infected insects >initial viral replication in regional lymph nodes >primary viremia > infection of target organs (endothelial cells and mononuclear cells of the lung, spleen, and lymphoid tissue) secondary viremia > virally induced endothelial cell damage and activation of infected macrophages with subsequent cytokine production (IL-1, TNFa) > increased vascular permeability > edema.

Signs

Incubation period varies from 2 to 21 days. An average one is 6 days. Fever is seen in the sick horses. .

Three types of African horse sickness are as follows :

1. Dikkop horse sickness (marked by hydropericardium).
2. Dunkop horse sickness (marked by respiratory distress).
3. Mixed or Acute type.

Dikkop (thick head) horse sickness

(The subacute edematous or cardiac form)

Subcutaneous oedema of head and neck, shoulders and chest, fever (reaching its height after about 2 weeks of its infection) are noticed. Such symptoms arise from abnormality of heart and circulation. The affected animals show drowsiness, arched back, restlessness, hanging of the head,
loss of natural wrinklings in the mandibular space and jugular furrows.

Dunkop (thin head) Horse Sickness (The peracute or pulmonary form)

This is an acute form of the disease affecting primarily the lungs of the animals. These animals show pulmonary edema and death at a later stage. Dyspnoea, paroxysmal coughing and escape of frothy fluid from the nose and struggle, severe breathing problems are noticed in the sick animals.

The acute or mixed form

Clinical signs of both the pulmonary and cardiac forms are seen in the mixed form. In most cases, the cardiac form is subclinical and is followed by severe respiratory distress. Occasionally, mild respiratory signs may be followed by edema and death from cardiac failure. Although the mixed form is common, it may not be recognized except at necropsy.

Pathology

In Dikkop horse sickness (thick head form), the main pathological lesions are the following:

1. Oedema of the subcutaneous and serous tissues.
2. Oedema in the head, neck and region of temporal fossa is seen and it can extend to shoulders, sternum and abdomen.Ascites is a marked lesion in this viral infection.

3. Excess of pericardial fluid (hydropericardium and subendocardial and subepicardial haemorrhages), myocardial degeneration and anasarca.

4. In Dunkop (thin head horse sickness), there is no subcutaneous oedema but the pulmonary oedema is massive and the interlobular tissue is infiltrated with fluid of gelatinous consistency. The trachea and bronchi are filled with forthy material. Hydrothorax is also seen in such cases.

Diagnosis

Its diagnosis is based on the symptoms, leucopenia and lesions. The immunological type of the virus infecting the horse is also identified. Other tests are detection of the virus by culture and reverse transcriptase - polymerase chain reaction (RTPCR) in blood or tissues.

Post Mortem Diagnosis: Gross findings in acute cases include severe hydrothorax and pulmonary edema and moderate ascites. The liver is acutely congested and

there is edema of the bowel wall . It is possible to find 3-5 litter of fluid in the chest cavity .

In cases of cardiac horse sickness there is marked hydro pericardium, endocardial hemorrhage, myocardial degeneration and anasarca, especially of the supra orbital fossa

At necropsy, the lungs are distended and heavy and frothy the conjunctiva fluid may fill the trachea, bronchi and bronchioles. This frothy exudate may ooze from the nostrils.