



Lecture title:

Diseases of equine

Part -2

Lecturer Affiliation:

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Summary:

Ulcerative lymphangitis

(Pigeon fever, Dry land distemper)

It is an infectious chronic disease of horse characterized by formation of skin ulcers and chronic abscesses in the pectoral region and ventral abdomen, with involvement of lymph vessels on skin and limbs

The causative agent (*Corynebacterium pseudotuberculosis*) non motile , non sporing , non capsulated and gram positive rod like shape bacteria. The bacteria enter via skin wounds by arthropod vectors such as stable flies, horn flies, and house flies, or by contact with contaminated fomites or soil.

Clinical signs and lesions

Clinical signs include:

- lameness , fever , weight loss and depression
- diffuse or localized swellings
- ventral pitting edema
- ventral midline dermatitis
- draining abscesses or tracts



The lesion begins with doughy consistency, disseminated swelling in one or both hind limbs in the fetlock region.

There is small well-circumscribed gray nodules which form abscesses and when it ruptured, it forms granulating ulcers characterized by circled, irregular shape, with gray or yellowish nodules and its borders are not elevated.

Affected lymphatic vessels are thickened, hard, cords - like shape, and doughy smooth appearance. Regional lymph nodes not affected but it may be slightly enlarged

Differential diagnosis:

In the differential diagnosis of ulcerative lymphangitis, glanders, sporotrichosis, epizootic lymphangitis, and mycobacterial infections should be considered



Epizootic lymphangitis

(Pseudo glander)

It is a debilitating fungal disease seen mainly in equids. The most common form of this disease is an ulcerative, suppurative, spreading dermatitis and lymphangitis; however, other forms including pneumonia or ulcerative conjunctivitis also occur.

Epizootic lymphangitis mainly affects horses, donkeys and mules. *H. capsulatum* var. *farcinosum* has also been reported in camels, cattle and dogs, and experimental infections have been established in mice, guinea pigs and rabbits.

Causes:

Fungus : *Histoplasma farcinosum*

Clinical signs

- The most common form of epizootic lymphangitis affects the skin and lymphatics. It often occurs on the extremities, chest wall, face and neck, but can be seen wherever the organism is inoculated into a wound.
- The first symptom is a painless, freely moveable intradermal nodule, approximately 2 cm in diameter. This nodule enlarges and eventually bursts.
- the skin ulcers grow, with granulation and partial healing followed by new eruptions. The surrounding skin is edematous at first, and later becomes thickened, hard and variably painful.
- At necropsy, areas of the skin and subcutaneous tissue are thickened, and the skin may be fused to the underlying tissues.
- The regional lymph nodes may be enlarged and inflamed. Nodules in the skin have a thick, fibrous capsule and the affected lymphatic vessels are usually thickened or distended.
- Both nodules and lymphatics contain purulent exudates.
- In some cases, the lesions may extend to the underlying joints, resulting in arthritis, peri arthritis or periostitis. Multiple, small, gray-white nodules or



ulcers with raised borders and granulating bases may be apparent on the nasal mucosa, and lesions may be found on the conjunctiva and cornea.

- The lungs, spleen, liver, testes and other internal organs may also contain nodules and abscesses.

The differential diagnosis includes :

- the skin form of glanders (farcy)
- strangles
- ulcerative lymphangitis,

sporotrichosis, cryptococcosis, sarcoids and cutaneous lymphosarcoma



Contagious equine metritis (CEM)

It is a highly communicable venereal disease of horses, caused by the bacterium *Taylorella equigenitalis*.

This disease can spread widely from a single asymptomatic carrier, particularly a stallion. Infected horses do not become systemically ill or die, but reproductive success is reduced. Additional economic impacts include the cost of pre-breeding tests and treatment in endemic areas, as well as screening before importation into CEM-free countries.

Horses appear to be the only natural hosts for *T. equigenitalis*, although donkeys have been infected under experimental conditions.

Donkeys are thought to be the major hosts for *T. asinigenitalis*, but this organism has also been isolated from a small number of mares and a few stallions. Most of these horses appear to have been infected during contact with donkey jacks, especially during breeding.

Transmission *T. equigenitalis* is transmitted mainly during mating. It can also be spread by infected semen during artificial insemination (AI) or introduced to the genital tract on fomites.

Stallions are the most common source of the infection. In untreated stallions, *T. equigenitalis* can persist for months or years on the reproductive tract.

The incubation period is 2 to 14 days; most infections become apparent 10 to 14 days after breeding.

Pathogenesis

- Spread from an acutely infected or carrier mare.
- Spread from a carrier stallion.
- Infection can be spread venereally at breeding, or indirectly by personnel and contaminated fomites.
- Bacterial invasion of the uterus → acute inflammatory response with an influx of neutrophils to the endometrium usually about 2 days post-mating.
- Smears of discharge may show the organism within the neutrophils.
- Acute self-limiting infection which may → carrier state.



- Potentially, foals born to mares which harbor CEM in the uterus during pregnancy may be born infected.

Clinical Signs

Infected stallions display no clinical signs. Mares can develop metritis and become temporarily infertile, although they have no systemic signs. Some of these infections are subclinical; the only sign may be a return to estrus after a shortened estrus cycle. In other cases, a mucopurulent, grayish-white vaginal discharge develops a week or two after breeding

in severe cases, the discharge is copious. Mixed bacterial infections may result in a gray to yellow exudate. Variable degrees of endometritis, cervicitis and vaginitis can sometimes be found if the reproductive tract is examined with a speculum. Infertility usually lasts a few weeks, with the discharge often disappearing in a few days to two weeks. Long-term effects on reproduction have not been reported; however, some mares can carry *T. equigenitalis* for a time. Carriers are usually asymptomatic, although a few mares may have an intermittent vaginal discharge. Most infected mares do not conceive. Those that do, usually give birth to a normal full-term foal, which may carry the organism asymptotically. Some infected mares have an intermittent vaginal discharge during the pregnancy, while others do not.

Abortions also occur, but appear to be rare. *T. asinigenitalis* has not been reported to cause disease in donkeys or horses under natural conditions; however, some experimentally infected mares developed cervicitis and metritis, with vaginal and cervical discharges. These mares had a shortened estrus cycle and failed to conceive. The clinical signs were milder than in mares infected with *T. equigenitalis*.

Post Mortem Lesions

The most severe lesions are usually found in the uterus. The endometrial folds may be swollen and edematous, and a mucopurulent exudate may be apparent.

Edema, hyperemia and a mucopurulent exudate may be seen on the cervix. Salpingitis and vaginitis also occur. The lesions are most apparent approximately 14 days after infection, then gradually decrease in severity over the next few weeks. They are not pathognomonic for contagious equine metritis.



Equine Viral Arteritis (EVA) *Equine Typhoid,* *Epizootic Cellulitis–Pinkeye*

Equine viral arteritis is an infectious disease of horses characterized by fever, depression, edema, conjunctivitis, Palpebral edema , nasal discharges, and abortions. Mortality is rare except in old, young, and debilitated horses

Etiology

Equine viral arteritis is caused by equine arteritis virus, a RNA virus in the genus *Arterivirus* (family *Arteriviridae*).

Transmission

Equine arteritis virus can be transmitted by the respiratory and the venereal routes. Acutely affected horses excrete the virus in respiratory secretions; aerosol transmission is common when horses are gathered at racetracks, sales, shows and other events. This virus has also been found in urine and feces during the acute stage. It occurs in the reproductive tract of acutely infected mares, and both acutely and chronically infected stallions. In mares, EAV can be found in vaginal and uterine secretions, as well as in the ovary and oviduct, for a short period after infection. Mares infected late in pregnancy may give birth to infected foals. Stallions shed EAV in semen, and can carry the virus for years. Transmission from stallions can occur by natural service or artificial insemination. Some carriers may eventually clear the infection.

Pathogenesis

Virus reaches in body through inhalation and viraemia occurs causing severe damage to small arteries in intestines, lymph nodes and adrenals that leads to diarrhoea and abdominal pain. It causes severe myometritis, conjunctivitis and pulmonary oedema.



Clinical signs

The incubation period varies from 2 days to 2 weeks. Infections transmitted venereally tend to become apparent in approximately one week. Most EAV infections, especially those that occur in mares bred to long-term carriers, are asymptomatic. Fulminant infections with severe interstitial pneumonia and/ or enteritis can be seen in foals up to a few months of age. Systemic illness also occurs in some adults. In adult horses, the clinical signs may include fever, depression, anorexia, limb edema (particularly in the hindlimbs), and dependent edema of the prepuce, scrotum, mammary gland and/or ventral body wall. Conjunctivitis, photophobia, periorbital or supraorbital edema and rhinitis can also be seen. Some horses develop urticaria; the hives may be localized to the head or neck, but are sometimes generalized.

Post mortem lesions

- Acute cases
 - Edema, congestion, hemorrhages
 - Fluid accumulation in body cavities
- Foals
 - Pulmonary edema, interstitial pneumonia, splenic infarcts, enteritis
- Aborting mares
 - Endometrial hemorrhages
 - Fetuses may be partially autolyzed.

In acute cases, the lesions are characterized by edema, congestion and hemorrhages of the subcutaneous tissues, visceral organs and lymph nodes. These changes are often found in the subcutaneous tissues of the limbs and abdomen, the thoracic and abdominal lymph nodes, and the small and large intestines (especially the colon and cecum), but may occur throughout the body.

Accumulations of clear, yellowish fluid may be found in the peritoneal cavity, pleura and pericardium. Foals may also have pulmonary edema, interstitial pneumonia, emphysema, splenic infarcts and enteritis. In mares that abort, the endometrium may be swollen and congested, and can contain hemorrhages.



Aborted fetuses are often partially autolyzed, but may be well preserved. In some fetuses, the only gross lesions may be excess fluid in the body cavities and signs of interlobular interstitial pneumonia.

Differential Diagnosis

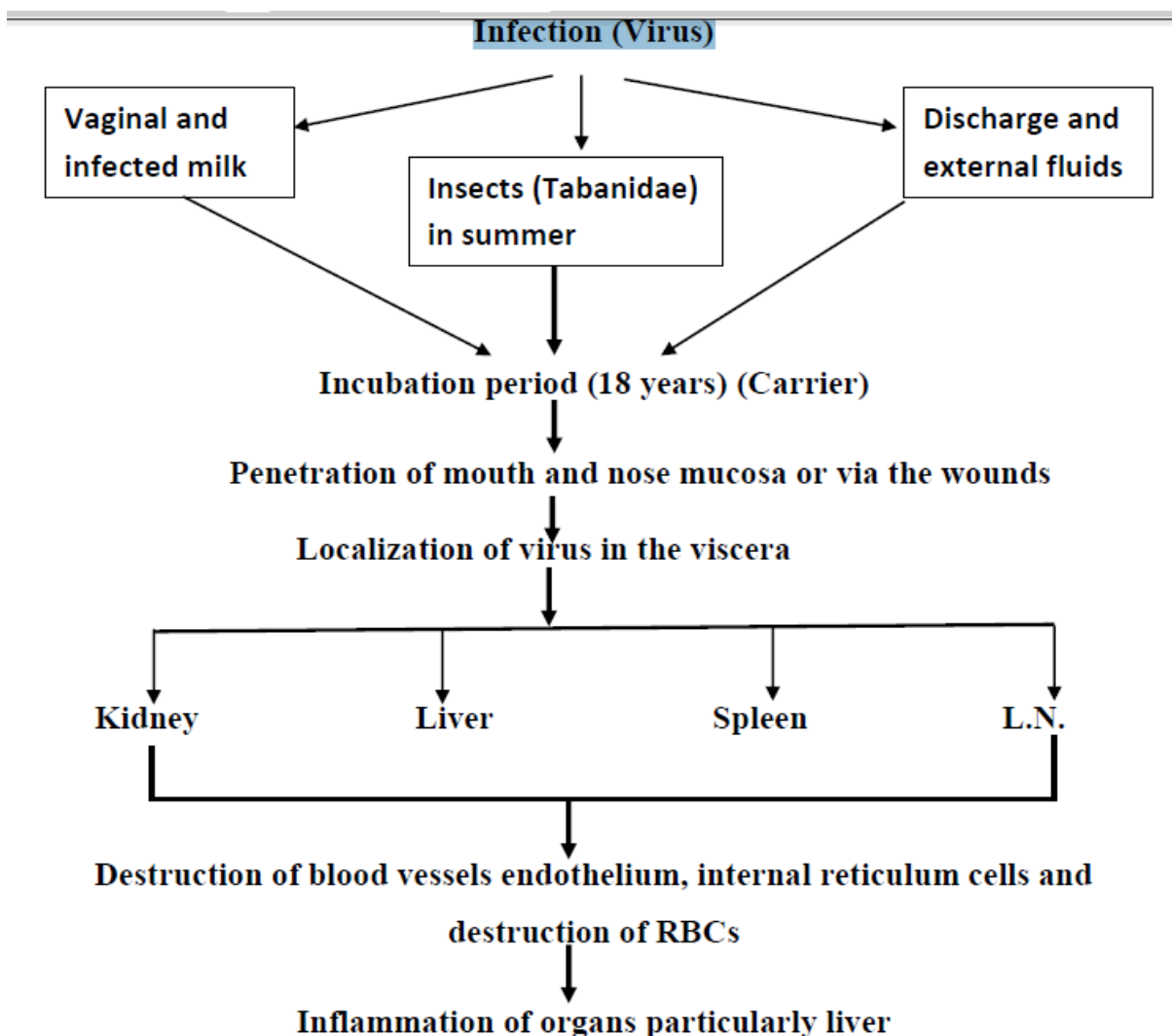
- Equine influenza
- African horse sickness
- Equine rhinitis
- Equine adenoviruses
- Equine herpesviruses
- Purpura hemorrhagica



Equine infectious anemia (EIA) *Swamp Fever*

Equine infectious anemia (EIA) is a noncontagious, infectious disease of horses and other Equidae. It is caused by an RNA virus classified in the *Lentivirus* genus, family Retroviridae. The most frequently encountered form of the disease is the unapparent, chronically infected carrier.

Transmission and pathogenesis





Lesions:

Gross lesions frequently seen in acute cases of EIA include enlargement of the spleen, liver, and abdominal lymph nodes; dependent edema; and mucosal hemorrhages. Chronic cases of infection are characterized by emaciation, pale mucous membranes, petechial hemorrhages, enlargement of the spleen and abdominal lymph nodes, and dependent edema.

Histopathologically, there is a nonsuppurative hepatitis and, in some cases, a glomerulonephritis, periventricular leukoencephalitis, meningitis, or encephalitis. Proliferation of reticuloendothelial cells is evident in many organs, especially in the liver, where there is also accumulation of hemosiderin in Kupffer cells. Perivascular accumulation of lymphocytes can be found in various organs.



Equine TRYPANOSOMIASIS

It is an infectious disease of animals caused by protozoan parasite, The trypanosomes are spindle-shaped protozoal parasites, most of which propel themselves with a flagellum and undulating membrane. Infection of horses with *Trypanosoma equiperdum*, *Trypanosoma evansi*, and *Trypanosoma brucei brucei* has traditionally been associated with the diseases **dourine**, **surra**, and **African animal trypanosomiasis (AAT)**.

Dourine

Dourine is a chronic trypanosomal disease of horses that is transmitted predominantly by coitus and is characterized by genital edema, neurologic dysfunction, and death.

Etiology

Trypanosoma equiperdum

Pathogenesis

Equids are considered the only natural host for *T. equiperdum*. Clinical signs are less obvious in donkeys than in horses, and these animals may be a reservoir for infection.

The organism is present in the urethra of infected stallions and in vaginal discharges of infected mares. Transmission in horses primarily occurs by coitus, although mechanical transmission by arthropod vectors is also possible. *T. equiperdum* can pass through intact mucous membranes. Transmission is considered most likely during the early stages of disease. The incubation period between exposure and initial clinical signs is highly variable, it may be as short as 1 to 2 weeks or as long as several years.

Foals born to mares infected with *T. equiperdum* may be infected in utero or may become infected during parturition. Transmission to foals by ingestion of infected colostrum or milk is considered rare. Foals that ingest colostrum from infected mares will become seropositive due to passive transfer of antibodies; these foals are usually seronegative by 4 to 7 months of age.



Signs and Lesions

The first signs of dourine in mares are vaginal discharge, with edema of the vulva, perineum, mammary gland, and ventral abdomen. Some mares exhibit signs of vulvitis and vaginitis with polyuria or other signs of perineal discomfort.

Abortion may occur if mares are infected with virulent strains. In stallions, initial clinical signs include edema of the external genitalia and perineum.

Cutaneous plaques, when they occur, are considered pathognomonic for dourine (“silver dollar plaques”); however, these plaques do not occur with all strains of the parasite.

Conjunctivitis and keratitis may occur in some infected horses. Chronically infected horses develop signs of neurologic dysfunction with progressive weakness and ataxia, leading ultimately to recumbency and death. These horses usually exhibit wasting despite a good appetite and frequently have anemia. Clinical signs may wax and wane for many months or years before death, depending on the strain of infecting parasite and the host immune response.

Cachexia and genital edema are often seen at necropsy. In stallions, the scrotum, sheath and testicular tunica may be thickened and infiltrated. The testes may be embedded in sclerotic tissue and may not be recognizable. In mares, a gelatinous infiltrate may thicken the vulva, vaginal mucosa, uterus, bladder and mammary glands.

Depigmentation of the genital area, perineum, and udder may occur. Lymph nodes, particularly in the abdominal cavity, are hypertrophied, softened and, in some cases, haemorrhagic.