Rinderpest (Cattle plague)

It's a viral disease affected all ruminants,characterized by High fever, oculonasal discharge, salivation, ulcerative stomatitis,diarrhea, dehydration and death .

Etiology

Rinderpest is associated with a morbillivirus (family Paramyxoviridae) and there are many strains with considerable variation in virulence between them but all are immuno logically identical. the immunity which develops after infection or vaccination with one strain protects against all other strains or isolates.

Epidemiology

1-rinderpest was a most old disease spread from Asia to Europe, the Middle East and Africa, usually as a sequel to wars.

2-the disease is endemic in Iraq and some other countries, Cattle and buffalo of all ages are susceptible to rinderpest

3-All ruminants and pigs are susceptible to infection with rinderpest virus. Natural infection occurs commonly only in domestic cattle, buffalo ,but in some out breaks, sheep and goats become infected and show clinical signs.

4-hight morbidity and mortality rate with high losses

5-the disease transmitted between animals by direct contacts through contaminated feed or by inhalation of infected aerosol and The virus is excreted by infected animals in urine, feces, nasal and ocular discharges, and saliva.

6-the virus is present in most animal tissues but its very sensitive and inactivated in cadavers within 24 h as a result of pH changes and putrefaction, and it is readily destroyed by heat, dryness and most disinfectants.

7- The virus of rinderpest is not pathogenic to humans.

Pathogenesis…

1- The virus is inhaled in infected droplets; it penetrates through the epithelium of the upper respiratory track and multiplies in tonsils and regional lymph nodes.

2- From these sites, the virus enters the blood and disseminated through out the body, The virus has high affinity for lymphoid tissues and alimentary mucosa and replicates in monocytes, lymphocytes and epithelial cells

3- The focal, necrotic stomatitis and enteritis which are characteristic of the disease are the direct result of viral infection and replication in epithelial cells in the limentary tract.

4- Death is usually from severe dehydration, but in less acute cases, death may occure due to secondary infection because the animal is immunosuppressed as a result of the destruction of lymphoid organs by the virus.

Clinical findings…

Clinical sings and symptoms mostly depend on the virulence of the virus , susceptibility of the host and the presence or absence of concurrent diseases. and In general, the disease may be peracute, acute,subacute or inapparent

The peracute form

its not common except after experimental administration of the virus. It is characterized by high fever, congested mucous membranes,respiratory distress and death 1-3 d later.

Acute form…

It manifested by …

phase of prodromal fever which characterized by An incubation period

of 6-9 days with high fever (40.5-41) without mucosal lesions but with Anorexia,decrease milk production , lacrimation and a harsh, staring coat .

mucosal phase…which characterized by

1-inflammation of buccal, nasal and conjunctival mucosae(conjunctivitis) and, in some cases, hyperemia of vaginal mucosa and swelling of vulva. The lacrimation becomes more profuse and then purulent and is accompanied by blepharospasm. Bubbly فقاعي (frothy)salivation of clear blood stained saliva is followed by purulent saliva and halitosis رائحة الفم الكريهه.A serous nasal discharge which becomes purulent.

2- Discrete, grayish, raised necrotic lesions (1-5 mm in diameter) develop,appearing first on the inside of the lower lip and adjacent gum, on the cheek mucosa at the commissures, and on the lower surface of the tongue. Later they become general in the mouth, including the dorsum of the tongue, and may become so extensive that they coalesce. Similar lesions are common on nasal, vulval and vaginal mucosae. The necrotic material sloughs, leaving raw, red areas with sharpe dges and these may coalesce to form shallow ulcers. Vesicles are not present.

3- Severe diarrhea, and sometimes dysentery with tenesmus, appear as lesions develop in abomasum and intestines

4- Skin lesions affecting the perineum,scrotum, flanks, inner aspects of thighs and the neck are less common. The skin becomes moist and reddened and later covered with scabs.

5- Other signs include dyspnea, cough, severe dehydration and sometimes abdominal pain. decrease in body temperature to subnormal levels occur on days 6-12, after which death usually occurs within 24 h

6- A few animals may survive and go into a convalescent phase during which the mucosal lesions heal rapidly,the diarrhea stops and recovery of body condition takes several weeks. Pregnant cattle may abort at this stage, discharging infective virus in the fetus and vaginal secretions for up to 24 h.

Subacute form…it characterized by

1- mild fever accompanying by anorexia

2- The inflammation of the mucosa is catarrhal only and there is no dysentery.

3- small pustules develop on the neck, over the withers, inside the thighs and on the scrotum. Most affected animals recover and convalescence is short

4-secondary infection are mostly manifested by anaplasmosis

Inapparent form…

It mostly occur in pigs and some times buffalo and manifested by mild sings

Note… Signs and lesions similar to those which occur in cattle develop in sheep and goats

Clinical pathology…

1- A marked leucopenia occurs at the peak of the infection and after vaccination in cattle

2- A rapid chromatographic strip test (Penside test) that can detect rinderpest antigen in lachrymal fluid is useful tool for field diagnosis

3- agar gel diffusion test,CFT,ELISA ,PCR and isolation of the virus

PM…

1- The important necropsy findings are in the alimentary and upper respiratory tracts and in the external genitalia

2- The carcass is dehydrated, emaciated and soiled with fetid feces. Small,discrete, necrotic areas develop on the oral mucosa and separation of the necrotic material leaves sharply walled,deep erosions with a red floor which may coalesce to form large erosions or ulcers.
3- lesions may extend to the pharynx, upper esophagus and abomasum, particularly the pyloric region.
4- Zones of hemorrhage and erythema running transversely across the colonic mucosa produce a characteristic striped appearance, the so-called 'zebra stripes خطوط الحمار الوحشي
5- The nasal turbinates and septa are coated with a tenacious mucopurulent exudate beneath which is an eroded and ulcerated surface. Lesions may extend to the upper trachea and the lungs are usually not affected. Congestion,swelling and erosion of the vulval and vaginal mucosae may occur.

Clinical pathology…
1-leuckoenia
2-serological tests

D.D….
The disease must be differentiated from
FMD.MCF.BVD,mucosal disease
Treatment ..
No specific treatment
Control…steps for control must include
1-slaughter and rigid quarantine measures In endemic areas
2-surveillance of new outbreaks
3-Hygenic disposal of dead animals
4-prevent movement of animals to infected areas
5-Vaccination …using

- Attenuated tissue culture rinderpest vaccine (TCRV)produced in calf kidney cells ,the vaccine were prepared by passage of the virus in goats, rabbits and chicken eggs to produce caprinized, laprinized and avianized vaccines, respectively,its easy to produce and can be lyophilized and produce long life immunity.
- The measles vaccine

Peste des petits ruminants (PPR, Goat Plague or Kata)

It's a rinderpest of small ruminant caused by PPR virus, a morbillivirus (family paramyxoviridae) closely related to the rinderpest virus as well as the viruses of canine distemper in dogs, phocine distemper in seals, and measlesin humans.

Epidemiology…

1-The disease is endemic in iraq and other countries and occurs mostly in goats and sheep in all Ages , Kids over 4 months and under 1 year of age are most susceptible to the disease .the disease is more sever in goats than sheep .

2-morbidity rate is 50-90% and mortalities is 55-85%.

3- close contact with an infected animal or contaminated fomites is required for the disease to spread. Large amounts of the virus are present in all body excretions and secretions, especially in diarrheic feces. Infection may also transmitted by inhalation and through the conjunctiva and oral mucosa.

Clinical findings… The disease can be acute or subacute

Acute form…

1- The acute form is seen mainly in goats and is similar to rinderpest in cattle except that severe respiratory distress is a common feature of PPR

2- A high fever (above 40°C) is accompanied by dullness, sneezing and serous discharge from the eyes and nostrils. followed by discrete necrotic lesions develop in the mouth and extend over the entire oral mucosa, forming diphtheric plaques with bad odor of the mouth and the animals unable to eat.

3- Diarrhea develops 3-4 d after the onset of fever, It is profuse and the mix feces with mucos and blood .

4- Dyspnea and coughing occur later and the respiratory signs are more sever when there is secondary bacterial pneumonia occur.

5-. Erosions have been described in the vulva and prepuce and abortions have been reported during outbreaks.

6- Death usually occurs within 1 week of the onset of illness.

Subacute form….

Subacute forms are more common in sheep but they also occur in goats. The signs and lesions are less marked and a few animals may die within 2 weeks, but most recover. Contagious ecthyma (orf) may develop in surviving animals.

PM…

The carcass is severely dehydrated, the hindquarters are soiled with fluid feces, crusts of exudate are present around eyes, nose and lips. Discrete or extensive areas of erosion, necrosis, and ulceration are present in the oral mucosa, pharynx,and upper esophagus and may extend to the abomasum and distal small intestine.Hemorrhagic ulceration is marked in the ileocecal region, colon and rectum where they produce typical 'zebra stripes

DD…

- Pneumonic pasteurellosis
- Contagious caprine pleuropneumonia in
- goats
- Contagious bovine pleuropneumonias
- Helminthosis
- Coccidiosis
- Contagious ecthyma