**** Foot and mouth disease:**

Foot and mouth disease (FMD) is an acute widespread infectious epitheliotropic viral disease of all cloven-hoofed animals and characterized by the formation of vesicles and erosions in the mouth and on the feet in adult animals and high morality in young age .

syn :Aphthous fever.

Causative agent:- Aphtho virus, presently 7 immunological and serological main types and

60 sub-types have been identified. In this state '0', 'A', 'C' and Asia-I are main types and A-22 a sub-type. Smallest of all the viruses and has affinity towards epithelial tissue of mouth and foot (Picorma viridae).

Animals Susceptible Cattle, buffaloes, followed by sheep, goat & pigs in that order Wild animals like Neelgai, yak, Antelopes and deer

. Exotic and Cross-breed animals are more susceptible.

Route of infection and pathogenesis

The infected animals excrete the virus in the saliva, nasal discharges, urine, feces, milk and semen. Recovered cattle can carry the virus up to 2 years.

The virus can be carried not only by the infected animals but also mechanically by humans and others animals, shoes, migratory birds, and animals byproducts.

The infection is spread directly through air born route and ingestion. Following infection, the virus replicates in the pharynx or respiratory tract, then reaches the lymphatics in the mucous membranes of the oropharyngeal region to the blood with subsequent to viremia and less localization particularly in the epithelium of the mouth and feet, and to a extent, the teats.

The signs usually related to lesions. After incubation period of 2-6 days, fever anorexia, salivation and painful eating with smacking of the lips and the tongue is noticed. Vesicles appear on the tongue, dental pad, and buccal mucosa. Moreover, the vesicles can be seen on lightly haired skin such as udder, vulva and conjunctiva.

Lameness occurs due to development of vesicles in the feet, particularly the cleft, heel and coronet

In young animals such as calves and lambs mortality rate is very high due to myocarditis and gastroenteritis Mortality rate in adults is about 2%, but in calves it is up to 20%

Gross lesions

The distribution of vesicles and erosion in buccal cavity is characteristic. The vesicles are seen on oral mucosa, dental bad, over the lips , palate and dorsum of the tongue

Also, vesicles and erosions near the coronary band and adjacent to interdigital clefts are common. Moreover, vesicles and erosion are seen in lightly haired skin as teats and vulva. Rumen, reticulum, omasum show vesicles also.

Abomasum may shows focal hemorrhages and diffuse edema of the mucosa. The small and large intestine show focal hemorrhages, congestion, or diffuse edema of the mucosa.

In young calves and lambs, lesions in the myocardium are most common in the fatal disease. These consist of small, grayish foci of irregular size in the wall and septum of left ventricle. The necrosis may give the myocardium a somewhat stripped appearance (tiger heart).

Microscopic appearance

The lesions of the epithelium of tongue, dental pad, buccal mucosa are started in stratum spinosum. The intercellular bridges of the epithelial cells retract and then disappear, and the cells become loosened from one another. The cells undergo hydropic degeneration and liquefactive necrosis. These changes are associated with infiltration of edematous fluid leading to the formation of small vesicle (aphthae). The vesicles coalesce to form larger ones (bullae) which compressed the surrounding epithelium with intact basal cell layer

The rupture of the vesicles lead to formation of erosions. The dermis is heavily congested and infiltrated by inflammatory cells. Vesicles in the feet and erosions are formed in the same manner as that in the oral mucosa.

Heart shows hyaline degeneration and coagulative necrosis of the cardiac muscle fibers besides intense infiltration of inflammatory cells, mainly lymphocytes, between the cardiac muscle fibers.

Histogenesis of vesicle: The most favourable cells for the reproduction of the virus appear to be those in the middle layer of stratum spinosum. The cell become swollen, rounded and the cytoplasm becomes acidophilic. The **nuclei pyknotic.**

Inflammatory exudate derived from the hyperemia vessels of the papillae of the corium collect between loosened cells these cell may undergo liguefactive necrosis. The cells over these foci undergo hydropic degeneration forming thus microvesicles. Necrotic cell attract

neutrophiles to the areas and these cause liquefaction. Coalescence of the neighboring foci result in vesicles

Diagnosis.

1-clinical symptoms and lesions.

2-Compliment Fixation Test (C.F.T.)

3-The virus can isolated in tissue culture and suckling mice

4-The disease should be differentiated from vesicular stomatitis,vescular exanthema, rinder pest and mucosal disease.

*****MALIGNANT CATARRHAL FEVER**:

It is an acute infectious disease of cattle and buffaloes, which characterized by high fever, emaciation, catarrhal and mucopurulent inflammation of eye and nostrils, erosion in oral mucosa, enlargement of lymph nodes, corneal opacity and nervous manifestation. It is generalised and usually fatal disease affecting many species of Artiodactyla. The disease has been most often described as affecting species of the subfamily Bovinae and family Cervidae,

Causes:

Malignant catarrhal fever is caused by viruses in the genus *Macavirus* of the family Herpesviridae (subfamily Gammaherpesvirinae).

Route of infection and susceptible hosts

The disease is not transmitted by direct contact with infected animals. Infection may occur through insect bits. Congenital infection may occur. Cattle and buffaloes are susceptible to natural infection.

Signs and gross lesions

Fever and catarrhal conjunctivitis and rhinitis with stream mucopurulent discharge from nostril and conjunctiva. Later on the discharge dries and adheres.

Rapid emaciation, corneal opacity beside dries and eroded skin of the muzzle is seen

Congestion of the oral mucosa may be with sharply irregular erosion is seen with the start of fever Diarrhea may be noticed beside nervous manifestation in the final stage. The esophagus, omasum, rumen, abomasum and intestine are congested edematous and eroded

Brain shows cooked appearance with broth odor. Generalize enlargement of the lymph nodes are constant signs. The cut section is granular and pink. Moreover, in mild form thickening and peeling of neck, axillae and perineum skin are seen.

Enlargement of liver and kidneys with grayish white foci are seen.

Microscopic appearance

The blood vessels in almost all organs show a pathognomonic lesion in the form lymphocytic arteritis, which characterize by necrosis of media, endothelial swelling of the intema and infiltration with lymphocytes

The lymph nodes show hyperplasia of the reticuloendothelial cells, edema and dilated lymphatics. Destruction and loss of the mature small lymphocytes and much of their debris is ingested by macrophage.

The liver and kidneys show foci of lymphocytic aggregations.

Alimentary and upper respiratory epithelium show erosions and necrosis. Infiltration of inflammatory leukocytic cells in the submucosal connective tissue is noticed.

Brain shows meningioencephalitis characterized by edema of the meninges and leukocytic infiltration of all types of inflammatory cells in the meningeal spaces. Moreover, perivascular edema and lymphocytic cuffing infiltration in Virchow. Robin space are seen. Necrotic vasculitis and neuronal degeneration are recorded.

Corneal epithelium undergoes degeneration and vesiculation beside edema of the lamina propria.

Iridocyclitis beside fibrinous exudate in the anterior and posterior chambers are observed

Differential diagnosis:

The clinical signs of the ‘head and eye’ form of MCF resemble those of other diseases that cause oral lesions . Thus BVD/mucosal disease, rinderpest, foot and mouth disease,

bluetongue and vesicular stomatitis may be considered as potential differential diagnoses where MCF is suspected. A clear diagnosis of MCF may be supported by additional evidence such as detection of MCF virus DNA, virus-specific antibody response and/or histopathology consistent with MCF.