Foot and mouth disease (FMD)

(Aphthus fever)

It is an acute highly contagious viral disease of cloven hoofed animals characterized by vesicular lesions, erosions, and ulcers in the mouth and on the muzzle, teats, inter digital area and coronary band.

Etiology:

The FMD virus is a Picornaviridae of genus *Aphthovirus*. At least seven immunologically distinct types of FMD virus have been identified: A, O, C, SAT1, SAT2, SAT3 and Asia 1. Within the seven types at least 60 subtypes have been recognized. Vaccine against one subtype may not protect against another.

The virus is rapidly inactivated by high or low pH, sunlight and very high temperature but very resistant to normal environmental conditions and drying. Sodium hydroxide, sodium carbonate and acetic acid are effective disinfectants but many common disinfectants are ineffective.

Epidemiology:

- FMD affect all cloven footed animals.
- The disease generally occurs in the form of outbreaks that rapidly spread from herd to herd before it controlled.

- Of the seven types, O, A, and C are prevalent in all continents when the disease occurs, SAT 1 is found in Africa and Asia, SAT 2 and SAT 3 are limited to Africa whereas Asia 1 occur in Asia.
- The morbidity rate in outbreaks of FMD in susceptible animals can rapidly approached 100 % and case fatality rate is low (about 2 % in adults and 20 % in young)
- The disease is transmitted by ingestion and inhalation or by direct or indirect contact with infected animals.
- The disease is most important in cattle and pigs but goats and sheep are also affected.

Pathogenesis:

The primary site of replication of inhaled virus in the pharynx and lymphoid tissue of upper respiratory tract. FMD virus then enter the blood stream and distributed around the body following secondary replication in other glandular tissues appears in the body fluids such as milk, urine, respiratory secretions and semen before the appearance of frank clinical signs of FMD. However, it is during the early vesicular stage of the disease that the majority of virus is secreted into environment.

Clinical findings:

- The incubation period is between 2 – 14 days depending on the rout of infection, the dose, the strain of virus and the susceptibility of the host.

- ♦ Following an initial pyrexia (40 – 41 C°) lasting one or two days, a variable numbers of vesicles (1 – 2 cm in diameter) develop on the tongue, hard palate, dental pad, lips and muzzle, these rapture within 24 hours leaving raw painful surface which heal in about one week. The vesicles are thin walled ruptured easily and contain a thin straw colored fluid.

- ♦ Concurrently with oral lesions vesicles appears on the feet particularly in the cleft and on the coronate. Rapture of vesicles cause acute discomfort and the animal is grossly lame, often recumbent with marked painful swelling of coronate. Secondary bacterial infection of feet lesions may interfere with the healing and lead to severe involvement of the deep structure of the foot.

- ♦ Acutely infected animals salivate profusely (the saliva hanging in long, ropy strings with characteristic smacking of the lips) and develop the nasal discharge which cover the muzzle and the animal stamp their feet to relief the pressure on first one foot and then another.

- ♦ Vesicles may occur on the teats and when the teat orifice is involved, severe mastitis is often follow.

- ♦ The vesicles on the tongue frequently coalesce and large proportion of the dorsal epithelium of the tongue may be displaced.

- ♦ Pregnant animals may abort.

- ♦ Rapid loss of condition and decrease milk production.

- ♦ Eating resumed in 2 – 3 days as lesions heal but the period of convalescence may be as long as 6 months.

- Young animals may die before the development of vesicles because the predilection of the virus to invade and destroy the cells of developing heart muscles.

Sequels of FMD in cattle:

A sequels of FMD in cattle due probably to endocrine damage is a chronic syndrome of dyspnea, anaemia, over growth of hair and lack of heat tolerance (panting).

Necropsy findings:

- The lesions of FMD consist of vesicles and erosions in the mouth and on the feet and udder. In some cases, vesicles may extend to the pharynx, esophagus, forestomach, and intestine as well as trachea and bronchi.
- In malignant form and in neonatal animals, epicardial haemorrhage with or without pale area are also present. Grossly the ventricle walls appear streaked with patches of yellow tissue interspersed with apparently normal myocardium giving the typical tiger heart appearance. If animal survives there is replacement fibrosis and the heart is enlarged and flabby.

Clinical pathology:

Identification of the virus by:

- Tissue culture.
- CFT.
- ELISA.
- Experimental transmission in test animals.

Differential diagnosis:

- ♦ Bovine popular stomatitis.
- ♦ Blue tongue.
- ♦ Rinderpest.
- ♦ Malignant catarrhal fever (MCF).
- ♦ Infectious bovine rhinotrichitis (IBR).
- ♦ Bovine herpes mammalitis.
- ♦ Vesicular stomatitis.
- ♦ Bovine viral diarrhea (BVD).

Treatment:

Treatment with mild disinfectants and protective dressing to inflamed area to prevent secondary infection is recommended in endemic countries, Where a slaughter policy is not in force.

Prevention and control:

- In endemic areas vaccination and quarantine are the basis for prevention and control. In free areas, the method of choice is rapid identification of an outbreak, quarantine and slaughter of all affected animals.
- Vaccine must be type specific. Most European and South American countries use trivalent inactivated vaccine against types A, O and C from cell culture virus. In Iraq vaccine should contain A, O and Asia 1 types of vaccine.
- Vaccine induced and naturally occurring immunity is short lived and vaccination must be repeated two to three times a year. Protection is partial so the infection usually results in sub clinical or mild disease.
- Calves nursing immune dams are likewise partially protected for up to 5 months. In an outbreak the most effective vaccine is outogenous