FOOD ANIMAL MEDICINE

(VCM 501)

1st semester 500 level

Viral Diseases of Food animals

PESTE DES PETITS RUMINANTS (PPR)

This is a highly contagious and infectious viral disease of domestic and wild small ruminants.

AETIOLOGY: The virus which causes PPR is called Peste des petits ruminants Virus (PPRV) which belong to the morbili virus group of the paramyxovirus family. Other members of this group which are closely related to PPR include: Rinderpest virus of cattle and buffaloes. Measles virus of humans. Distemper virus of dogs and some wild carnivores. Morbillivirus of aquatic mammals. Genetic characterization of PPR virus strains has recognised 4 groups (3 groups from Africa and 1 from Asia. The epidemiological significance of these grouping is not clear at present.

GEOGRAPHICAL DISTRIBUTION OF PPR. PPR infection has been recognised in many African countries especially those that lie between the Atlantic Ocean and the red sea. The affected areas extend North of Egypt and South to Kenya in the East and Gabon in the West. The disease is not recognised in most Northern and Southern African countries. Recently the disease has been reported in the near East and the Arabian peninsula in countries that include; Iran, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Saudi Arabia, the United Arab Emirate and Yemen. Outbreaks also common in India, Nepal, Bangladesh and Pakistan.

Probable global distribution of PPR virus infection

CLINICAL SIGNS OF PPR: Clinical signs appear 2-6 days post natural infection with the virus (incubation period). This is followed by sudden onset of fever of between 40 and 41^{0}C. There is marked depression and the animal appears sleepy. Hair stands erect giving a

bloated appearance especially in short haired breeds. After which clear watery discharges from the eyes, nose and mouth later becomes thick and yellow (mucopurulent) due to secondary Bacterial infections. This discharge causes wet chin and hairs below the eyes which becomes dry causing matting together of the eyelid, obstruction of the nose and difficulty in breathing.

FIGURE 1: PPR in a goat: purulent eye and nose discharges Discharges from the nose and eyes in advanced PPR infection; the hair below the eyes is wet and there is matting together of the eyelids as well as partial blockage of the nostrils by dried-up purulent discharges.	

One or two days post fever, the mucous membrane of the mouth and the eye becomes very reddened and congested.

FIGURE 2: PPR in a goat: inflamed (reddened) eye membranes Reddening of the mucous membranes of the eye (the conjunctiva) in the early stages of infection. Note the purulent eye	

discharges.	

The oral cavity epithelial necrosis causes small pin point greyish areas to appear on the gum, dental pad, palate, lips, inner aspect of the cheek and the upper surface of the tongue. These areas increase in number and size and join together. The lining of the mouth becomes pale and coated with dead cells. Underneath this dead surface cells are shallow erosions. Gentle rubbing across the gum and palate with a finger may yield a foul smelling material containing shreds of epithelial tissues. Similar lesions may be seen in the mucous membrane of the nose, vulva and vagina.

FIGURE 3: PPR in a goat: early mouth lesions showing areas of dead cells Early pale, grey areas of dead cells on the gums.	

FIGURE 4: PPR in a goat: later mouth lesions The membrane lining the mouth is completely obscured by a thick cheesy material; shallow erosions are found underneath the dead surface cells.	

Affected animals resist attempt to open their mouth because of the lesion and associated pains. This results in refusal of food and water.

Diarrhoea appears 2 to 3 days after fever, although in early or mild cases, it may not be obvious. Faeces initially are soft, watery, foul smelling and contain blood streaks and pieces of dead gut tissues.

FIGURE 5: PPR in a goat: signs of diarrhoea The hindquarters are soiled with liquid faeces.	

Affected sheep and goats breathe fast, thus exhibiting rocking movements with both the chest and the abdominal wall moving as the animal breaths.

Severely affected animal show difficulty and noisy breathing marked by extension of the head and neck, dilation of the nostrils, protrusion of the tongue and soft painful cough. Up to 100% of the animals in a flock may be affected in a PPR outbreak with between 20 and 90% mortality.

DIFFERENTIAL DIAGNOSES. Contagious Ecthyma (ORF): No diarrhoea in ORF, whereas in PPR, there is diarrhoea and occular discharges.

Pasteurellosis caused by *Mannheimia haemolytica*: No diarrhoea, no oral lesions and the number of goats affected are very low.

Coccidiosis: No oral lesions, no coughing and mortality is very low.

***Contagious caprine pleuropneumonia* (CCPP):** No oral lesions and no diarrhoea.

DIAGNOSIS

A tentative diagnosis of PPR can be made on epidemiological and clinical features. In the event of history, oral discharges, diarrhoea, deaths with prominent breathing problems in a sheep and goat flock, no history of contact with cattle and most affected animals in the flock are adolescents; a suspicion of PPR may be made.

LABORATORY CONFIRMATION

Viral isolation from blood, lymph nodes around the lungs (mediastinal lymph nodes), spleen and alimentary tract lymph node (mesenteric lymph nodes).

Detection of viral antigen by Agar gel immunodiffusion test (AGIDT) is a simple test but this test does not discriminate between PPR and Rinderpest; therefore a further test is required.

Viral antigen can be detected by ELISA technique very rapidly. This is sensitive and differentiates between PPR and Rinderpest.

POST MORTEM LESIONS

Erosion on the gum, soft and hard palate, tongue, cheek, and into the oesophagus. Lips showed erosion with possibly scabs and nodules in later cases.

Lungs shows dark red purple areas, firm to touch mainly in the anterior and caudal lobes of the lungs (evidence of pneumonia).

FIGURE 6: PPR in a goat: the early lesions of pneumonia Note the small, red, solid areas of lung tissue caused directly by PPR virus infection.	
FIGURE 7: PPR in a sheep: advanced pneumonia Note the extensive, dark red/purple areas, firm to the touch, in the anterior and cardiac lobes of the lungs. Although such pneumonia is commonly seen in PPR, it is caused by secondary bacterial infection, most commonly *Mannheimia* haemolytica. These lesions are typical of pneumonic pasteurellosis.	

Small intestine congested with reddened lining haemorrhages and some erosions.

Large intestines (caecum, colon, and rectum) small red haemorrhages along the folds of the lining, joining together over time and becoming darker termed “Zebra Stripes”

FIGURE 8: PPR in a goat: "zebra striping" in the large intestine Note the lines of haemorrhage along the tips of the folds of the lining of the caecum and colon. Later, the individual haemorrhages join up and, after death, turn black.	

TREATMENT: Antibiotic treatment to prevent secondary bacterial infection: Penicillin - Streptomycin, Enrofloxacin

Control of PPR outbreaks relies on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk populations. Until recently, the most practical vaccination against PPR made use of tissue culture rinderpest vaccine. Recently, a homologous PPR vaccine has been developed and the vaccine is available in NVRI Vom.

The appearance of clinical PPR may be associated with any of the following:

1. History of recent movement or gathering together of sheep and/or goats of different ages with or without associated changes in housing and feeding.
2. Introduction of recently purchased animals; contact in a closed/village flock with sheep and/or goats that had been sent to market but returned unsold.
3. Change in weather such as the onset of the rainy season (hot and humid) or dry, cold periods (for example the harmattan season in West Africa); contact with trade or nomadic animals through shared grazing, water and/or housing.
4. A change in husbandry (e.g. towards increased intensification) and trading practices.

5. In endemic areas, most of the sick and severely affected animals are over four months and up to 18 to 24 months of age.

WHAT ARE THE NECESSARY PRECAUTIONARY MEASURES TO TAKE TO PREVENT OUTBREAK OF PPR BY A FARMER WHO WANTS TO ESTABLISH IN A GOAT OR SHEEP FARM?

1. Prior to acquisition of animals, a suitable house must be provided to prevent exposure of these animal to adverse weather conditions especially extreme cold / Harmattan.

2. Avoid buying of new stock of sheep/ goats from open market where there are congregation of different animals both the sick and healthy ones. Close contact in the market aid easy transfer of disease from the sick to healthy ones.

3. It is recommended to get new stock from small holding goat farmers who have adequate information and history of their animals.

4. Gradual change of feed from old to new feed.

5. Acquisition of animals should be done in batches.

6. From the first day of arrival on the farm, animals should be placed on Antibiotic and multivitamins to reduce the stress of change of environment, after which PPR homologous vaccine should be administered to all apparently healthy animals.

RINDERPEST

Cattle plague, also known as Rinderpest, is a contagious disease that principally affects cattle, but occasionally can also affect sheep, goats, camels, certain wild ruminants and pigs. The disease is characterized by severe inflammation and necrosis of the mucous membrane of the digestive tract. The disease has been associated with high mortality and it is an OIE (Office International des Epizooties also known as World Organization for Animal Health) Class A disease reflecting its serious economic impact.

AETIOLOGY: The rinderpest virus (RPV) is a RNA Morbillivirus, closely related to the PPR, Measles and Canine distemper viruses. Despite its extreme virulence, the virus is particularly fragile and is quickly inactivated by heat, desiccation and sunlight.

CLINICAL SIGNS: Mortality rates during outbreaks are usually extremely high, approaching 100% in immunologically naive populations.

Initial symptoms include fever, loss of appetite, and nasal and eye discharges. Subsequently, irregular erosions appear in the mouth, the lining of the nose and the genital tract. Acute diarrhoea, preceded by constipation, is a common feature as well. Most animals die 6 to 12 days after the onset of these clinical signs.

TRANSMISSION: The disease is mainly spread by direct contact and by drinking contaminated water, although it can also be transmitted by air.

Clinical Signs: The temperature rises in the early stages. The animal is off its food, dull and the coat is starry. Sometimes shivering is noticed. The breathing is quick: a watery or mucous discharge flows from the eyes and nostrils; in the latter case there may be a slight amount of blood in the discharge. In milking cows, the secretion of milk is diminished or arrested. The membrane of the nostrils reddens, and an eruption, like grains of bran, appears in the nostrils and inside the lips and cheeks. This eruption is often followed by distinct ulceration. The animal is at first constipated, but in the later stages diarrhoea often sets in. In this case the faeces has a foul smell and is often tinged with blood. The animal rapidly loses condition and the disease usually terminates fatally in from 6 to 10 days. Cattle plague does not attack single animals in a herd, but spreads rapidly from one to another.

Rinderpest: Photos of clinical signs

	Clinical signs of Rinderpest: Purulent ocular discharge and conjunctivitis
	Clinical signs of Rinderpest: Erosions of buccal mucosa and gingival
	Clinical signs of Rinderpest: Erosions on ventral surface of tongue.

DIAGNOSIS

This is based tentatively on the clinical signs and post mortem lesions. Rising antibody titre in paired sera, serology neutralization, CFT, Agar gel diffusion tests and isolation of virus from body discharges and excretion have been used to confirm the disease.

POST MORTEM LESIONS: The principal lesions in Rinderpest are in the alimentary tract. Small necrotic areas, which later develop into sharply defined deep ulcers, occur in the mouth, pharynx and oesophagus. These ulcers later coalesce to form large erosions. Similar lesions and also numerous small haemorrhages occur in the mucosa of the abomasum. Zones of intense inflammation are found in the large intestine. Typically, these are arranged transversely giving a striped appearance "Zebra stripes". All lymph nodes are severely congested and dark red in colour.

TREATMENT AND CONTROL: No specific treatment but supportive therapy with antibiotics and fluids may reduce mortality. Annual vaccination with the Tissue culture rinderpest vaccine (TCRV). Control of outbreaks by quarantine and ring vaccination.

FOOT AND MOUTH DISEASE

This is an acute, highly contagious viral disease of cloven footed animals but most important in cattle and pigs. The disease is enzootic in NIGERIA. However it has been endemic in EUROPE in the last 1 to 3 years.

AETIOLOGY: Apthovirus of the Picona virus group. Seven serotypes of the virus has been identified and this include the following: A, O, C, SAT 1, SAT 2, SAT 3 as well as ASIA type 1 serotype. However these serotypes further comprises of over 50 sub types. Recently six new strains have been discovered in recent outbreaks in Europe.

TRANSMISSION: The major route of transmission of FMD is through aerosol. Other routes include contact with infective material or discharges, through carrier animals, by ectoparasites, experimentally by artificial insemination and use of FMD infected carcases as meat scraps in pig farming.

CLINICAL SIGNS: Initial lesions are seen on the lingual mucosa which later develops into vesicles. Dullness, anorexia and pyrexia may precede the appearance of the vesicles. Vesicles extends to the nares, buccal cavity and between the hooves which result in lameness especially in pigs. Hoof deformities may cause a persistent lameness even after remission of other signs. Drooling of saliva as a result of lesions in the oral cavity may be seen. Pregnant animals may abort and mortality is often high among calves and piglets. Mortality may be up to 100% especially in calves but rarely exceeds 1% in adults. Mammary gland involvement may lead to mastitis. In young calves, there is involvement of the heart leading to abnormal heart sounds.

DIAGNOSIS: History, clinical signs and PM lesions will give a tentative diagnosis. Virus neutralization, Agar gel precipitation and ELISA will have to be undertaken to confirm the tentative diagnosis.

POST MORTEM: Heavy presence of blisters or vesicles in the oral cavity which extends to the lips, nostrils, muzzles, teats, snout, hooves and inter digital spaces. In calves there are degenerative changes in the heart muscles.

MANAGEMENT AND CONTROL: In FMD free counties, infected animals are slaughtered and carcasses burnt or buried with Calcium oxide and whole premises decontaminated. Antibiotic therapy both systemic and topical for local lesions on the legs and oral cavity to minimize secondary complications. Quarantine, vaccination and subsequent release of in contact animal are undertaken, however such animal serve as carrier for the disease.

Bovine Viral Diarrhea and Mucosal Disease Complex

Bovine viral diarrhea (BVD) is most common in young cattle (6-24 mo old) and generally is accompanied by typical mucosal lesions; it must be distinguished from other viral diseases that produce diarrhea and mucosal lesions. These include malignant catarrhal fever and Rinderpest

ETIOLOGY

Bovine viral diarrhea virus (BVDV), the causal agent of BVD and mucosal disease complex, is classified in the genus Pestivirus in the family Flaviviridae. Although cattle are the primary host for BVDV, several reports suggest most even-toed ungulates are also susceptible. Isolates of BVDV are separated into noncytopathic and cytopathic biotypes based on cytopathic effects observed in infected cell cultures. Noncytopathic BVDV are the predominant viral biotype in nature. Cytopathic BVDV are relatively rare and arise in cattle that are persistently infected with noncytopathic BVDV

Cattle that are persistently infected with noncytopathic BVDV serve as a natural reservoir for virus. Persistent infection develops when noncytopathic BVDV is transmitted transplacentally during the first 4 mo of fetal development. The calf is born infected with virus, remains infected for life, and usually is immunotolerant to the resident noncytopathic virus. Transplacental infection that occurs later in gestation results in abortion, congenital malformations, or birth of normal calves that have antibody against BVDV. The prevalence of persistent infection varies among countries and between regions within a country. In some areas, the prevalence of persistent infection in calves may be as high as 1-2% of cattle <1 yr of age.

On a given farm, persistently infected cattle are often found in cohorts of animals that are

approximately the same age. Persistently infected cattle can shed large amounts of BVDV in their secretions and excretions and readily transmit virus to susceptible herd mates. Clinical disease and reproductive failure often are seen after healthy cattle come in contact with a persistently infected animal. Biting insects, fomites, semen, biologic products, and possibly wild ruminants also can spread BVDV. Disease induced by BVDV varies in severity, duration, and organ systems involved. Acute disease results from infection of susceptible cattle with either noncytopathic or cytopathic BVDV.

CLINCAL SIGNS

Acute BVD, also termed transient BVD, often is an inapparent to mild disease of high morbidity and low mortality. Biphasic fever (~104°F [40°C]), depression, decreased milk production, transient inappetence, rapid respiration, excessive nasal secretion, excessive lacrimation, and diarrhea are typical signs of acute BVD. Clinical signs of disease usually are seen 6-12 days after infection and last 1-3 days. Transient leukopenia may be seen with onset of signs of disease. Recovery is rapid and coincides with production of viral neutralizing antibody. Gross lesions seldom are seen in cases of mild disease. Lymphoid tissue is a primary target for replication of BVDV, which may lead to immunosuppression and enhanced severity of intercurrent infections.

Some isolates of BVDV induce clinically severe disease that manifests as high fever (~107°F [41-42°C]), oral ulcerations, eruptive lesions of the coronary band and interdigital cleft, diarrhea, dehydration, leukopenia, and thrombocytopenia. In thrombocytopenic cattle, petechial hemorrhages may be seen in the conjunctiva, sclera, nictitating membrane of the eyes; and on mucosal surfaces of the mouth and vulva. Prolonged bleeding from injection sites also occurs. Swollen lymph nodes, erosions and ulcerations of the GI tract, petechial and ecchymotic hemorrhages on the serosal surfaces of the viscera, and extensive lymphoid depletion are associated with severe forms of acute BVD. The duration of overt disease may be 3-7 days. High morbidity with moderate mortality is common. Severity of acute BVD is related to the virulence of the viral strain infecting the animal and does not depend on viral biotype or genotype.

In pregnant cattle, BVDV may cross the placental barrier and infect the fetus. The consequences

of fetal infection usually are seen several weeks to months after infection of the dam and depend on the stage of fetal development and on the strain of BVDV. Infection of the dam near the time of fertilization may result in reduced conception rates. Infection during the first 4 mo of fetal development may lead to embryonic resorption, abortion, growth retardation, or persistent infection. Congenital malformations of the eye and CNS result from fetal infections that occur between months 4-6 of development. Fetal mummification, premature birth, stillbirth, and birth of weak calves also are seen after fetal infection.

DIAGNOSIS

BVD is diagnosed tentatively from disease history, clinical signs, and gross and microscopic lesions. Diagnostic laboratory support is required when clinical signs and gross lesions are minimal.

Laboratory tests for BVDV include virus isolation and assays that detect antibody in serum or detect viral RNA or viral antigen in clinical specimens and tissues. Because antibody against BVDV is prevalent in most cattle populations, a single serologic test is seldom sufficient for diagnosis. A >4-fold increase in antibody titer in paired serum samples obtained 2 more weeks apart is necessary to verify recent infection. Isolation of BVDV from blood, nasal swab specimens, or tissues confirms active infection. Identification of persistent infection requires detection of virus in clinical specimens obtained at least 3 wk apart. At necropsy, tissues of choice for viral isolation include spleen, lymph node, and ulcerated segments of the GI tract.

DDX

Laboratory support also is required in some outbreaks of mucosal disease or clinically severe acute BVD because either disease may appear similar to rinderpest, FMD and malignant catarrhal fever. These include malignant catarrhal fever which is sporadic disease in more matured cattle and rinderpest can be seen in outbreaks form but is exotic in most countries

TREATMENT

Treatment of BVD is limited primarily to supportive therapy. Control is based on sound

management practices that include use of biosecurity measures, elimination of persistently infected cattle, and vaccination. Replacement cattle should be tested for persistent infection before entry into the herd. Quarantine or physical separation of replacement cattle from the resident herd for 2-4 wk should be considered, and vaccination of replacement cattle for BVD should be done before commingling with the resident herd. Embryo donors and recipients also should be tested for persistent infection. If vaccination of embryo donors or recipients is warranted, it should be done at least 1 estrous cycle before embryo transfer is performed. Because BVDV is shed into semen, breeding bulls should be tested for persistent infection before use. Artificial insemination should be done only with semen obtained from bulls free of persistent infection.