PESTE DES PETITS RUMINANTS (PPR)

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Introduction:

Peste des petits ruminants also known as goat plague or kata is a highly contagious and infectious disease of wild small ruminants characterised by fever, oculonasal ischarges, stomatitis, diarrhoea and pneumonia. The true extend of the disease has only become apparent in recent years and the extend of occurrence is still being investigated. Many veterinarians, animal health workers and livestock owners in areas where PPR is absent or recently introduced are not familiar with its clinical and pathological features. As the Global Rinderpest Eradication Programme (GREP) proceeds towards the anticipated eradication of rinderpest by the year 2010, it becomes increasingly important that PPR and Rinderpest be differentiated because, at this stage of the programme any outbreak of Rinderpest anywhere represents an international emergency.

It was first described in the Ivory Coast of West Africa in1942. The viral agent was isolated in 1956 and was thought to be a Rinderpest virus that had adapted to sheep and goats. For many years it was believed to be restricted to the African continent until a disease of goats in Sudan, which was originally diagnosed as Rinderpest in 1972, was confirmed to be PPR. The true extent of the disease has only become apparent in recent years and is still being clarified. The realization that many of the cases diagnosed as Rinderpest among small ruminants in India may, instead have involved the PPR virus, together with the emergence of the disease in other parts of western and south Asia point to its ever increasing importance.

PPR may have passed unrecognized for years because it is frequently confused with other diseases which cause respiratory problems and mortality of small ruminants. The clinical signs of PPR closely resemble those of Rinderpest as well as many other diseases making a differential diagnosis difficult. Hence accurate diagnosis of PPR is of paramount importance to ascertain the magnitude and the extend of this infection among small ruminant population of India.

The disease:

The virus which causes Peste des petits ruminants virus (PPRV) belongs to the Morbilli virus group of the family Paramyxoviridae. It is closely related to the Rinderpest virus of cattle and buffalo, the measles virus of humans, the distemper virus of dogs and wild carnivores and the Morbilli virus of aquatic mammals. The African and Asian strains of PPRV have some differences, implying that both strains may have evolved separately, presumably from the goat-adapted Rinderpest vaccine introduced six decades ago.

The disease occurs in goats and less often in sheep. Cattle, buffaloes and pigs can become infected but there is little or no evidence of the disease associated with their infection. PPR infection has been recognized in many of the African, Arabian and the middle-east countries. Outbreaks of PPR are now known to be common in India, Nepal, Pakistan, Bangladesh and Afghanistan.

Transmission and spread

Close contact with infected animal or contaminated faeces is required for the disease to spread. Large amounts of the virus are present in all body secretions and excretions especially diarrhoeic faeces. Fine infective droplets are released to the air, particularly when the animal coughs or sneezes. Other animals which inhale the droplets are likely to become infected. Although close contact is the most important way of transmitting the disease, it is suspected that infectious materials can also contaminate water and feed troughs and bedding turning them into additional sources of infection. Trade in small ruminants, at markets where animals from different sources are brought into close contact with one another, affords increased opportunities for PPR transmission.

Recognizing the disease in a herd or flock

When PPR occurs in an area for the first time, it is possible that acute high fever with extreme depression and death occurs before other typical signs are seen. Sometimes a fast spreading syndrome in sheep goats characterized by a sudden onset of depression, discharges from eyes, nose and mouth, abnormal breathing with coughing, diarrhoea and deaths. The outbreak will not involve cattle, whether Rinderpest vaccinated or not (even if they are not in contact with the affected sheep or goat).

The appearance of typical PPR may be associated with:

- History of recent movement or gathering together of goat/sheep of different ages without associated changes in housing and feeding.
- Introduction of recently purchased animals
- Contact with goats or sheep which have returned unsold from the market
- Change in weather such as onset of rainy season or dry cold periods.

Clinical signs:

Clinical signs appear an average of 2 – 6 days after natural infection with the virus; this is followed by a sudden onset of fever with rectal temperature of at least 40 – 41ÚC. The affected animals are markedly depressed. Soon after this stage a clear watery discharge is noticed from eyes, nose and mouth which later become thick and yellow as a result of secondary bacterial infections. The discharges wets the chin and hair below the eyes, dries up, causing matting together of the eyelids, obstruction of the nose and difficulties in breathing.

A day or two later, the mucous membrane of mouth and eye become very reddened and epithelial necrosis causes small pin point grayish areas to appear in the gum, dental pad , palate, lips, inner aspects of cheek and upper surface of tongue. The lining of mouth becomes pale and in later stages becomes obscured by a thick cheesy material. Shallow erosions are seen beneath the dead surface epithelial cells. As the disease progresses, a characteristic foul smell (halitosis) exude from the mouth and the animal is unable to eat because of a sour mouth and swollen lips.

At the height of development of oral lesions, most animals manifest severe diarrhea, often profuse but not hemorrhagic. It may not be obvious in early or mild cases. The faeces are initially soft, then watery, foul smelling and may contain blood streaks and pieces of dead gut tissue. As it progresses, there is severe dehydration, emaciation, and dypsnoea followed by hypothermia. Affected animals breathe fast and severely affected cases show difficult and noisy breathing marked by extension of head and neck, dilatation of nostrils, protrusion of tongue and soft painful coughs.

Death usually occurs after a course of 5 to 10 days. Bronchopneumonia, evidenced by coughing, is a common feature in the later stages of PPR. Pregnant animals may abort. Secondary latent infections may be activated and complicate the clinical picture.

Erosions may also be seen in the mucous membranes of nose, vulva and vagina .In the later stages of the disease small nodular lesions may be seen in the skin on the outside of lips around the muzzle.

Up to 100% of the animals in a flock may be affected in a PPR outbreak with between

20 – 90% dying. PPR infection can be suspected if a combination of symptoms occurs:

- Sudden onset of febrile disease affecting sheep and or goats
- Oculonasal discharges with sores in mouth, with or without scabs or nodules around mouth
- Pneumonia, Diarrhoea
- Significant death rate

Post mortem findings

Inflammatory and necrotic lesions in the mouth and the gastrointestinal tract dominate the pathology caused by PPR. Unlike RP, there is also a definite, albeit inconstant, respiratory system component; hence, the synonym stomatitis-pneumoenteritis complex. The carcass is severely dehydrated and the hindquarters are soiled with fluid faeces. The eyes and nose have dried up discharges

- Extensive areas of erosion, necrosis and ulceration in oral mucosa, pharynx, upper oesophagus which may extend to abomasum and distal small intestine.
- Typical "zebra stripes" (hemorrhagic ulcerations) in ileocaecal region, colon, rectum and on crests of mucosal folds.
- Mucopurulent exudate in areas between

nasal opening and larynx with terstitial pneumonia in lungs.

Differential diagnosis

PPR is frequently confused with other diseases that present fever and grossly similar signs especially when it is newly introduced. Other diseases to be considered are;

Mouth lesions - Blue tongue or Contagious ecthyma (Orf)

Difficult breathing - Pneumonic pasteurellosis

Contagious caprine pleuropneumonia (CCPP)

Diarrhoea - Coccidiosis or gastrointestinal helminth infections

Diagnosis

Tentative diagnosis of PPR is mainly based on clinical signs and post mortem lesions. Because of the necessity to detect PPR amidst a number of other acute diseases with grossly similar presenting signs and to differentiate it from Rinderpest, some laboratory tests need to be carried out. These tests may detect the virus itself, evidence of the presence of the virus (viral antigen or genetic material) or antibodies against the virus found in blood serum.

- Detection of viral antigens by agar gel immunodiffusion test (AGID), Immunocapture ELISA (ICE)
- Detection of virus genetic material by the reverse transcriptase polymerase chain reaction (RT PCR) which can be combined with nucleotide sequencing for epidemiological studies.
- Detection of the virus by isolation of the PPR virus in cultured cells.
- Detection of antibodies by Competitive ELISA. Surveys for antibodies are very useful to determine the presence or absence of infection and its extent in a population.

Samples required for laboratory testing

The collection of specimens at the correct time is important to achieve diagnosis by virus isolation and they should be obtained in the acute phase of the disease when clinical signs are still apparent.

- Conjunctival swabs in PBS
- Gum debris
- Lymph nodes
- Sections of the ileum and large intestine
- Portions of spleen, lungs
- Unclotted blood
- Serum (if possible, paired sera)

Treatment

There is no treatment for PPR. However, mortality rates may be decreased by the use of drugs that control the bacterial and parasitic complications. Specifically, oxytetracycline and chlortetracycline are recommended to prevent secondary pulmonary infections

Control

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. The use of Rinderpest vaccine to protect small ruminants against PPR is now contraindicated because its use produces antibodies to rinderpest which compromise serosurveillance for Rinderpest, and thereby the Global Rinderpest Eradication Programme. A homologous PPR vaccine is now available and gives strong immunity for at least 3 years. There are also genetically engineered recombinant vaccines undergoing limited field trials

Methods for Rinderpest eradication may be appropriate for PPR which include;

- Quarantine
- Slaughter
- Proper disposal of carcasses and contact fomites
- Decontamination of facilities and equipment
- Restrictions on importation of sheep and goats from infected areas. ❑

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5. Organic farming needs research and development in order to apply the most modern knowledge and improve its performance.

6.Training and extension should be provided to all categories of stakeholders.

7.Government has to make legislation in order to ensure the regulatory framework, where all stakeholders can play a fair level ground.

Conclusion

Organic farming is an emerging area for crop and livestock production, processing, marketing, trade and consumption and therefore, for research all over the world. In developed countries it has made significant inroads but the developing countries especially India is in the stage of consumption only, as far as organic livestock production is concerned. ❑