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Peste Des Petit Ruminants-A review

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ABSTRACT

Peste des petit ruminants [PPR] is a highly contagious viral disease which is characterized with acute or sub-acute hyperthermia, extremely contagious and mostly pernicious disease of sheep as well as goats and wild small ruminants. In this review, detailed information on etiology, transmission, clinical findings, diagnosis of method, control and elimination pathological, and epizootiological findings of Peste des petits ruminants was given.

Keywords: Peste des petits ruminants, Small ruminant

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Introduction

Peste des petits ruminants (PPR) is one of the most important diseases that severely affects small ruminants in many countries in Africa, the Middle East and parts of Asia this disease seriously hinder sheep and goat production which is highly acute contagious and infectious viral disease of goats and sheep and clinically similar to rinderpest and is characterized by fever, erosive stomatitis, diarrhea, conjunctivitis, gastroenteritis, and pneumonia (Ozkul et al., 2002). The name is French for "disastrous disease of small ruminants". Goats are usually more severely affected than sheep. The presence of PPR can have a serious impact on livestock production and trade. Economic losses are due to loss of production, death, abortion and cost of controlling the disease. The presence of the disease can limit local trade and export (Banyard et al., 2010).

1. Etiology

PPR is caused by a paramyxovirus of the genus morbillivirus. It is antigenically very similar to the rinderpest virus. For many years PPR was considered a variant of rinderpest virus, specifically adapted for goats and sheep, having lost its virulence for cattle. It is now known that the two viruses are distinct, though closely related antigenically (Ozkul et al., 2002; Lefèvre and Diallo, 1990; Diallo, 1990; Dufour, 2010). The half-life of the virus at 36-38°C was estimated at 2 hours, and at 45-50°C infectivity was destroyed in half an hour (Diallo, 1990; Banyard et al., 2010; Dufour, 2010). Peste des petits ruminants virus is very sensitive to ultraviolet radiation and desiccation as well as heat. Other studies have confirmed and clarified the thermal sensitivity of PPRV (Lefèvre and Diallo, 1990; Banyard et al., 2010). Peste des petits ruminants virus has been shown to survive in lymph nodes for 8 days at 4°C (Diallo, 1990; Banyard et al., 2010; Dufour, 2010). This virus cannot bear low pH so being destroyed after death of the animal by the low pH PPRV can stand at the pH of 5.8 and 9.5 but easily loses activity at pH lower than 4 or above 11 at room temperature (Diallo, 1990). It can survive at the pH between 7 and 8 (Dufour, 2010). PPR is divided to lineage 1 and 2 viruses in West Africa, lineage 3 in East Africa, Arabian and Southern India and lineage 4 in the Middle East and Asia subcontinent (Dhar et al., 2002).

2. History

PPR is known by fever conjunctivitis, gastroenteritis, pneumonia, stomatitis, and causes important economic losses in small ruminant's yields (Elsawalhy et al., 2010; Merck and Dohme, 2016). It was initially discovered in Côte d'Ivoire (West Africa) in 1942 (Ozkul et al., 2002; Banyard et al., 2010; Elsawalhy et al., 2010) inspectors soon after demonstrated presence of the disease in, Senegal, Ghana and Nigeria. Peste des petit ruminants is

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also known as goat plague, pest of sheep and goats, Kata, stomatitis-pneumoenteritis syndrome, contagious pustular stomatitis, and pneumoenteritis complex (Braide, 1981). In 1972 in the Sudan, a disease in goats, originally diagnosed as rinderpest, was confirmed to be PPR (Hamdy et al., 1976; Taylor, 1979a; Taylor, 1979b; Munir et al., 2012; Libeau et al., 2014). The reference to the disease as a "plague" is indicative of the highly contagious nature and economic impacts that result from this disease. The second spread in the 1990s of PPR, also other animal diseases in Africa and the Middle East, was not only belonging to biological factors, but to exacerbating standards in national veterinary services in these countries. Changes in government priorities have bring about detracted funding and a restructuring which has interrupted national veterinary services (Hamdy et al., 1976; Taylor, 1979a).

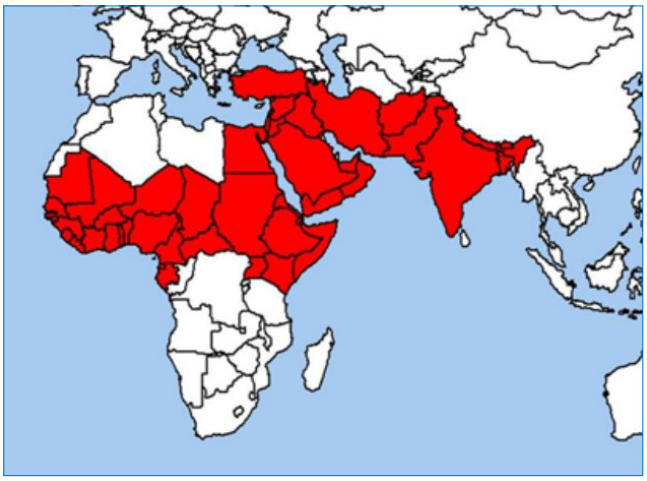


Figure 1. Global distribution of PPR prior to its southern migration into Tanzania, DRC and Angola (red parts).

3. Animals affected

PPR is a disease of sheep and goats, goats are more susceptible to PPR than sheep (Banyard et al., 2010). Cattle and pigs are known to undergo subclinical infection of PPR but they do not exhibit clinical signs. Cattle do not, however, play a role in the spread of PPR because they are apparently unable to transmit the disease to other animals (Taylor, 1984). Inother domestic animals such as camels. The disease has been reported in wild and zoo small ruminants (Ogunsanmi et al.,2003; Ezeibe et al., 2008; Kinne et al., 2010).

4. Host determinants of PPR

Host determinant factors of PPR expance have been seen in various studies, highlighting age, sex, breed and animal species (Munir et al., 2012). Young animals are more susceptible to PPRV due to less likely developed protective antibody titers (Luka et al ., 2011) which is noticed in Turkey, India, Kenya, Pakistan, and Ethiopia (Singh et al., 2004; Waret-Szkuta et al., 2008; Abubakar et al., 2009). Sex has also been shown as a risk factor of the disease (Ezeibe et al., 2008; Swai et al., 2009; Sarker, 2011; Abdalla et al., 2012). Females are more likely to demonstrate higher antibody titers than the males it has reported in Bangladesh male goats are significantly more prone to PPR than females (Sarker, 2011). In spite of this, data from Pakistan have reported no important relationship between males and females, with respect to susceptibility (Zohari and Stalli, 2008). Goat and sheep species' differences have been highlighted as major risk factor for PPRV susceptibility (Waret-Szkuta et al., 2008; Zohari and Stalli, 2008). The impression of breeds of the small ruminants on susceptibility to the PPR have also been reported (Zohari and Stalli, 2008), which are demonstrated poor differences between goat breeds however there are significant differences between sheep breeds. Breed differences to susceptibility to disease have been stated (El Hag and Taylor, 1984; Diop and Libeau, 2005; Banyard et al., 2010;).

5. Economic Impact

The presence of PPR can have a serious impact on the economics of a region which has a widespread distribution spanning Africa and Asia (Nanda et al., 1996; Shaila et al., 1996). Economic losses are due to loss of production, death, and abortion. The presence of disease can limit trade, export, import of new breeds, and the development of intensive livestock production. PPR is a major constraint on the availability of protein for human consumption as well. In addition to this economic role, sheep and goats have significant socio cultural roles. They are used as gifts or emblems for traditional rituals and religious purposes (Banyard et al., 2010).

6. Pathogenesis

The source of infection is airborne droplets. All discharge of infected sheep are contagious all over the course of the disease, however no carrier state exists (Pugh and Baird, 2012). During acute phase of the disease, agent is shed in all discharges. PPRV exhibits lympho-epitheliotropism (Gibbs, 1981). Lymphotropic nature, common to all morbilliviruses, causes a severe

leukopenia, which develops the promote of later infections by bacterial agents or parasitic opportunists due to immunosuppression (Munir et al., 2012). The virus multiplies in lymphoid organs then after spread through blood and penetrate to epithelial cells of respiratory and gastro-intestinal tract (Gopilo, 2005). PPRV causes cytopathic effect that is defered from that of other morbilliviruses (El Hag and Taylor, 1984).

6.1. Morbidity/Mortality

PPR is highly contagious when it first occurs in a naïve population. Periodic outbreaks may also be seen in endemic regions, particularly when animals are mixed or new animals are introduced into a herd. In endemic regions, animals between three months and two years of age are most severely affected; young animals that are still nursing and older animals tend to be spared. The severity of the disease varies with the host's species, immunity and breed. The morbidity and mortality rates can reach 100%, particularly in naïve herds; however, these rates tend to be lower in endemic areas and the reported mortality rates in some individual flocks are as low as 20%. High case fatality rates have been reported when PPR virus (PPRV) infected herds of exotic ungulates (Kahn and Kahn, 2005; Rajak et al., 2005; Diallo, 2006; Muse et al., 2012; Sahinduran et al., 2012).

7. Transmission

Transmission of Peste des petit ruminants mainly happens during close contact. Inhalation have basic role in spreading. PPRV is shed in nasal and ocular secretions, saliva, urine and feces as well as milk. Animals may also be contagious during the incubation stage. Although animals are not expected to become long-term carriers, it is demonstrated that viral antigens were shed in the feces of clinically recovered goats for at least 12 weeks. Water, feed troughs and bedding can probably transmit Peste des petit ruminants virus for a short time, but do not remain infectious for long periods. How the virus is maintained between outbreaks is not well understood (Gopilo, 2005; Zohari and Stalli, 2008; Abubakar et al.,2009; Sarker, 2011; Sarker and Islam, 2011).

8. Clinical signs

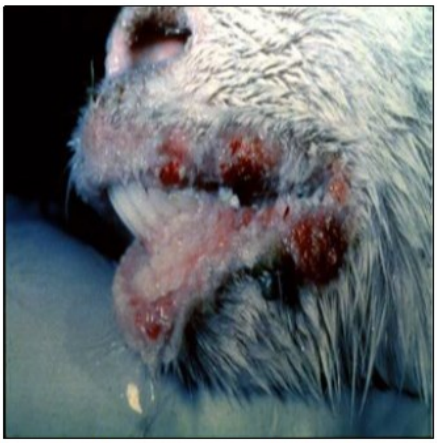
8.1. Symptoms and Clinical Signs: The incubation period can range from 2-10 days, with 2-6 days being typical. Peracute cases can be seen when PPR first occurs in native populations of sheep or goats.

8.1.1. Acute form: PPR in acute form is unusual in sheep. Most cases of PPR are acute. Symptoms generally emerage 4 to 7 days after being in contact with an infected animal (Kahn and Kahn, 2005). The disease is highly fatal in the young animals accompanied pyrexia to 40-41.2 °C (Kahn and Kahn, 2005). It is go along with serous discharge from the eyes and nostrils, dullness and sneezing. After a few days , separate lesions develop in the mouth and extend over the whole oral mucosa. Within a few days of the onset of fever, the gums become hyperemic, and small, gray, necrotic foci, covering shallow erosions, begin to appear in the mouth and appearing diaphtheric plaques. There is a deep halitosis and because of a sore mouth and swollen lips the animal is unable to eat. Mucopurulent discharge and the exudate desiccate in nasal and ocular, matting the eyelids and partially closing the external narres. Some animals abort. In the late stages of the disease and diarrhea occurs 3-4 days after the inception of fever that could be mucoid as well as blood tinged. Dyspnea and coughing occur later and due to secondary bacterial pneumonia respiratory symptoms are exacerbated. Vulva and prepuce contain erosion. Abortion has been mentioned in India and Tanzania. The cause of these lesions is unknown. Severely affected animals become dehydrated and emaciated; hypothermia can precede death. Animals that do not die often have a prolonged convalescence. Acute form of PPR is fatal and animal usually are find dead in one week after onset and show the symptoms (Muse et al., 2012).

8.1.2. Sub-acute form: Subacute disease can also be seen in some animals; this form usually lasts 10-15 days. This form is prevalent in sheep as well as goats. This form is characterized with long incubation period of about 1 week. In this form of disease due to lack characteristic clinical signs mortality is seldom. The symptoms are variable, but often include respiratory signs. Asymptomatic infections also occur. As dose contagious ecthyma lesions such as oral crusts due to mucosal discharges may appear and therefore deferential diagnosis could be hard. Body temperature is about 39-40 °C and meliorate of about 2 weeks and remain immune protected (Diallo, 2006). PPR should be considered in sheep or goats with pyrexia, highly contagious oral erosions as well as gastrointestinal symptoms (Diallo, 2006; Muse et al., 2012). The photo shows dried exudate on the muzzle and around the eye resulting from rhinitis and conjunctivitis (Figure 2-7).

Depression, hemorrhage, diarrhea.

Discharge from the eyes, nose and mouth



Close up view of mouth

Figure 2. Clinical sign

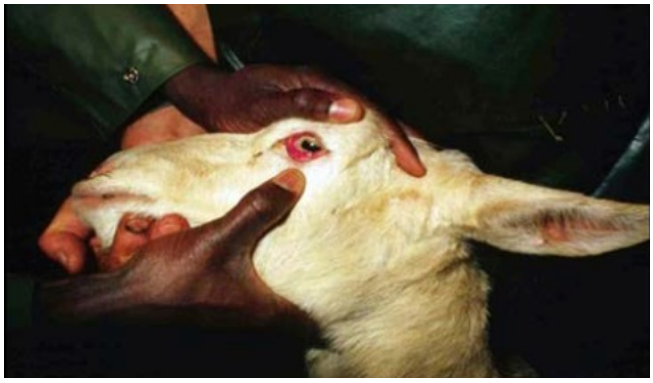


Figure 3. Hyperaemia and congestion of the conjunctiva

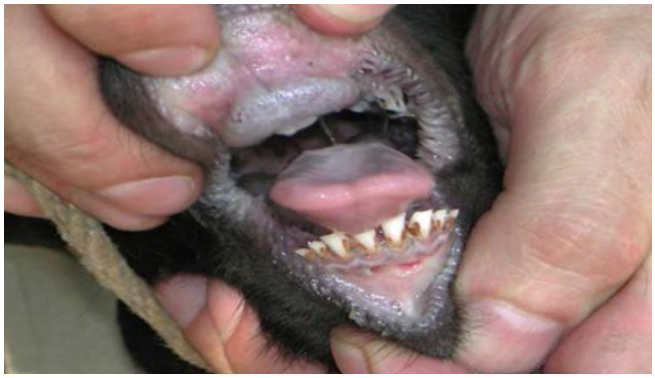


Figure 6. Erosions and ulcerations on the mucous membranes of the lips

Figure 4. Mucopurulent discharges from the eyes and nasal cavities

Figure 5. Severe purulent nasal exudate

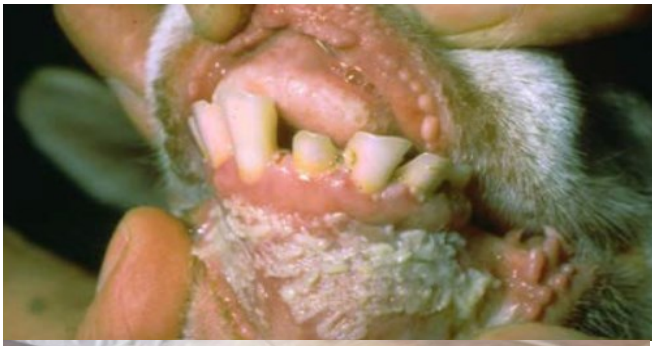


Figure 7. Necrotic tissue accumulating on the buccal mucosa

8.2. Experimental infection: A severe experimental form of disease has been reproduced in sheep and goats (Bundza et al., 1988). In spite of natural PPR, experimental infection of susceptible cases with PPRV considered with high morbidity rate and low mortality rate. (Osman et al., 2009; Maina et al., 2015). Sheep experimentally infected with PPRV shown significant lesions at the lip commissure (Figure 8), intense diarrhea (Figure 9) enteric congestion (Figure 10) and gooseberry-like mesenteric lymph nodes (Figure 11) (Maina et al., 2015).

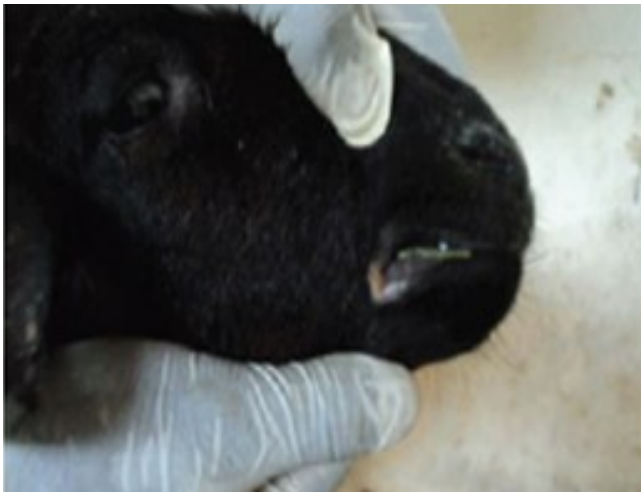


Figure 8. An experimentally infected sheep having ulcerated oral lesions at the commissure of the mouth on day 12 post infection.

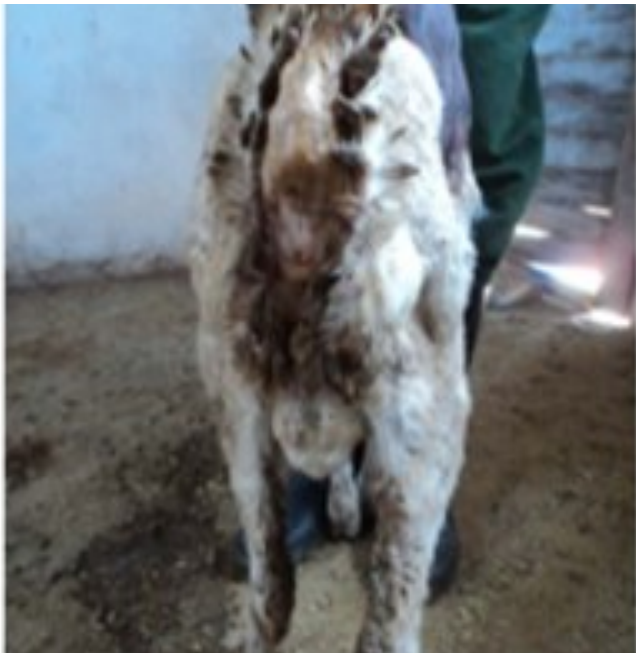


Figure 9. An experimentally infected sheep having severe watery diarrhea with soiling of the hind limbs on day 13 post infection.

Figure 10. Intestinal mucosa from an experimentally infected sheep appering severely congested.

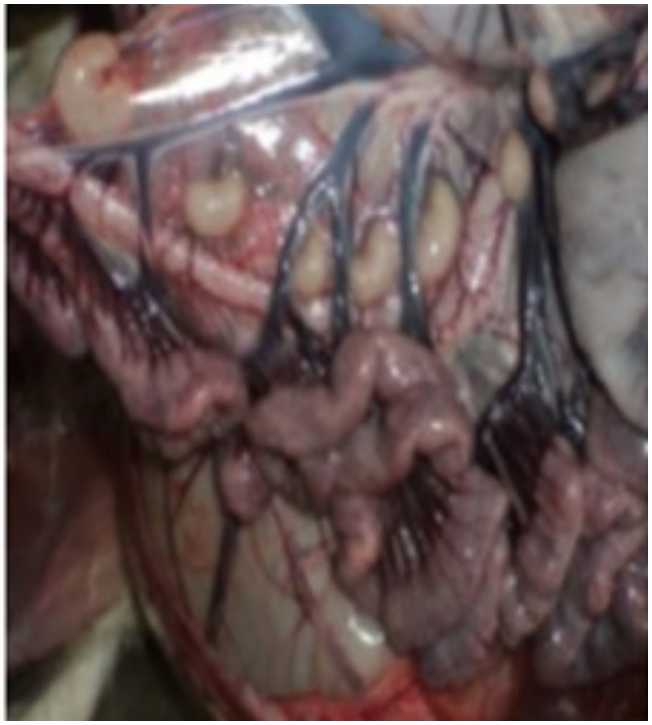


Figure 11. Swollen and enlarged mesenteric lymph nodes (arrow) from an experimentally infected sheep having a goose berry-like appearance (the sheep died on day 17 post infection).

9. Post mortem lesion necropsy finding

Post mortem lesions are similar to rinderpest, with inflammatory and necrotic lesions in the oral cavity and throughout the gastrointestinal (GI) tract. The carcass of a PPR affected animal is generally emaciated, the

hindquarters soiled with soft/dilute faeces. Erosion in the oral cavity is a constant feature affecting the gums, soft and hard palates, tongue and cheeks and into the oesophagus. The eyes and nose contain desiccated discharges and the eyeballs sunken. Lips may be blown and possibly nodules in late cases. The nasal cavity is congested with yellow exudates and erosions (Roeder et al., 1994). The abomasum is congested with multiple haemorrhages. The rumen, reticulum and omasum seldom content lesions. Between times, erosions may be exhibit on the pillars of the rumen. Lesions in the small intestine are commonly mild, just surrounded with small lines of hemorrhages and, sometimes, erosions present in the first part of the duodenum and in the end of ileum. The large intestine is more susceptible, with congestion around the ileo-cecal valve, at the ceco-colic junction and in the rectum. The most severe lesions are seen in the large intestine, with congestion and "zebra stripes" of congestion on the mucosal folds of the posterior colon. Erosive lesions may also occur in the vulva and vaginal mucous membranes. About respiratory system, bronchopneumonia may be present, usually confined to the antero-ventral areas, and is characterized by consolidation and atelectasis occurs frequently. Nasal mucosa consist small erosion and petechiae, as well as larynx and trachea. The lung is dark red or purple with areas stiff to the touch. Lungs and the intestines lymph nodes are soft and swollen Figure (10-16) (Roeder et al., 1994).

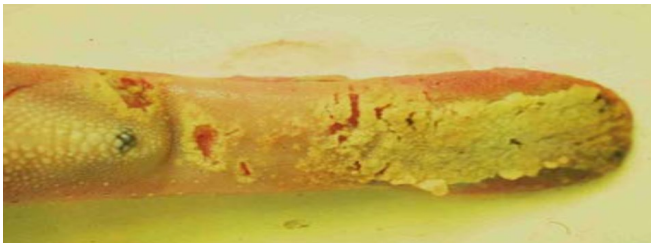


Figure 12. Post-mortem lesions (Accumulation of necrotic material and ulcerations on the dorsal surface of the tongue).

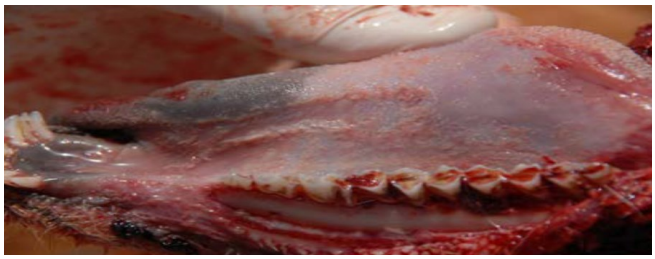
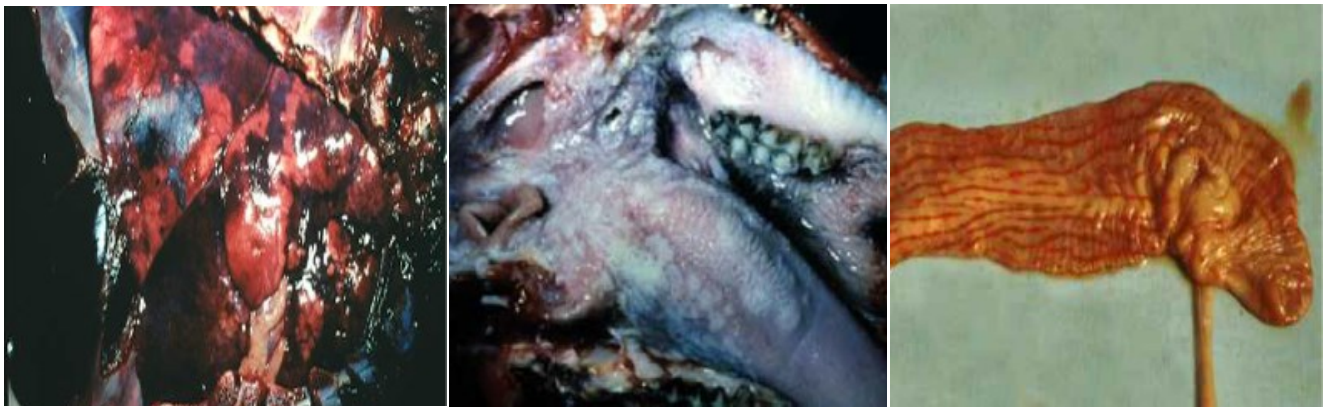


Figure 13. Post-mortem lesions (Necrosis and erosions on the side and at the base of the tongue in a sand gazelle)

Figure 14. Post-mortem lesions (Erosions and ulcerations on the tongue and soft palate).



Necrotic lesions in the oral cavity Pneumonia Zebra stripes on intestine

Figure 15. Post-mortem lesions

10 . Samples

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. Call before sampling as a USDA trained Foreign Animal Disease Diagnostician (FADD) will need to collect and ship the samples.

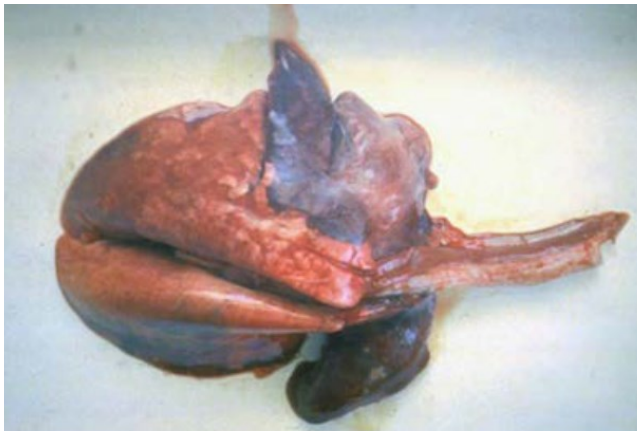


Figure 16. Post-mortem lesions (Advanced stage pneumonia in apical lung lobes)

11. Diagnosis and Treatment

11.1. Differential diagnosis: The differential diagnoses include rinderpest (although many reports of 'rinderpest' among small ruminants may have been PPR), bluetongue, contagious ecthyma, foot and mouth disease, heartwater, coccidiosis and mineral poisoning. The respiratory signs can resemble contagious caprine pleuropneumonia (CCPP) or pasteurellosis; pasteurellosis can also be a secondary complication of Peste des petits ruminants.

11.2. Diagnosis of the disease: Diagnosis of PPR can be made on the basis of clinical, pathological, and epizootiological findings. However laboratory verification is an absolute necessity. Diagnosis of PPR may be carried out through virus isolation, detection of specific antibody in the serum, detection of viral antigens, nucleic acid isolation and sequencing (Gopilo, 2005).

11.3. Virus isolation: Process of finding virus is implemented by isolation of the Peste des petit ruminants virus in cultured cells. This way of diagnosis very important as it enables live virus for biological qualification studies and the isolated viruses are kept for later studies (Kahn and Kahn, 2005). Samples for virus isolation consist of heparinized blood, eye and nasal swabs, tonsil, mesenteric lymph nodes, spleen, section of colon and lung samples must be collected during the pyrexia (Libeau et al., 1995) and rendered to the testing laboratory in ice. The lamb kidney and ovine skin and Vero cells are vastly used in cell culture systems. (Hamdy et al.,1976; Taylor and Abegunde, 1979; Adombi et al., 2011)

11.4. Laboratory diagnosis: Peste des petit ruminants virus antigens can be detected by immunocapture ELISA (ICE), counter immunoelectrophoresis (CIEP) or agar gel immunodiffusion (AGID). CEIP and ICE can distinguish PPRV from rinderpest virus, but the AGID test cannot differentiate these two viruses. Immunofluorescence and immunochemistry can be used on conjunctival smears and tissue samples collected at necropsy. Viral nucleic acids can be detected with reverse transcription PCR (RT-PCR). Serological examinations consists virus neutralization and competitive ELISA assays. Both tests can distinguish PPR from rinderpest. In live animals, swabs of ocular and nasal discharges, and debris from oral lesions should be collected. Whole, unclotted blood (in heparin or EDTA) should be taken for virus isolation and PCR (Libeau et al., 1995; Luka et al., 2011; Maina et al., 2015).

11.5. Molecular Techniques

11.5.1. Nucleic acid recognition methods : Reverse transcription polymerase chain reaction (RT-PCR) techniques based on the amplification of parts of the N and F protein genes has been developed for the specific diagnosis of PPR (Forsyth and Barrett, 1995; Couacy-Hymann et al., 2002). This technique is 1000 times more sensitive than classical virus titration on Vero cells with the advantage that results are obtained in 5 hours, including the RNA extraction, instead of 10–12 days for virus isolation (Couacy-Hymann et al., 2002).

12. Treatment

There is no specific treatment for PPR. However, drugs that control bacterial and parasitic complications, as well as supportive care, may decrease mortality.

13. Disease in human

PPRV does not infect humans.

14. Preventation, Control and Vaccination

As mentioned before there is no specific treatment against PPR. State or federal authorities should be notified immediately. Barns, tools and other items that have been in contact with the sick animals must be cleansed and disinfected with common disinfectants (phenol, sodium hydroxide 2%, virkon) as well as alcohol, ether, and detergents. Control of the disease in previously non-infected countries can be effected through strict quarantine, movement controls, restriction of importation of sheep and goats from affected areas, rapid identification, humane slaughter,

disposal of affected animals and burning or burying carcasses and effective cleaning and disinfection of contaminated areas and clothing with lipid solvent solutions of high or low pH. Effective disinfectant agents include alcohol, ether, phenol, sodium hydroxide and common detergents. In areas where PPR is endemic, the commonly employed control mechanism is vaccination (Kihu et al., 2015). The virus can survive for long periods of time in chilled or frozen tissues. New animals should be quarantined for three weeks before allowing them to mix with the flock. In a case of PPR outbreak, animals with signs of PPR should be isolated immediately and sheep and goats around the outbreak area should be vaccinated as soon as possible (Blood et al., 1983; Diallo et al., 1995; Spickler and Roth, 2006; Kihu et al., 2015). Vaccination is the most effective way to gain control epidemic PPR. In a situation where goats are reared together with sheep, the mixed herd model established that sheep were the main drivers of PPR transmission. Peste des petits ruminants disease in sheep herds was seen to persist longer than was the case in the goats and thus may serve as the reservoir for virus in between outbreaks. A simulation of the model showed that vaccination coverage of 50% of combined sheep and goats herds was enough to curtail the spread of the PPR disease within 254 days in Turkana, Kenya (Blood et al., 1983; Peacock, 1996). This vaccine is safe for pregnant dams and induces immunity in at least 98% of the vaccinated animals in the field (Thrusfield, 2013). This vaccine protects immunized small ruminants for a period of up to 3 years. The major drawback in using this vaccine is thermo stability especially since PPRV is a disease of tropical countries. Recently, a freeze dried form of this vaccine has been prepared in an excipient containing trehalose to make it thermo stable. This fortified vaccine is resistant to temperature as high as 45 °C for 14 days with negligible loss in efficacy. The use of this vaccine to protect small ruminants will lead to effective control of PPR in developing countries (Kihu et al., 2015).

15. Disinfection

PPRV is thought to remain live for about four days outside. This virus can be loosen by many disinfectants including alkalis phenolic compounds, (sodium carbonate, sodium hydroxide), citric acid, alcohols, iodophores and halogens (sodium hypochlorite). Over all animals suspected with Peste des petit ruminants should be isolated, and the farm should be quarantined until certain diagnosis is determined. PPR can be eliminated with a combination of quarantines, movement controls, eradication and euthanasia of infected animals, and cleaning and disinfection of infected presupposition, as dose rinderpest also in PPR methods applied for eradication would be appropriate

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