Peste Des Petits Ruminants: A Major Threat to Small Ruminant Health and Production

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Abstract
Peste des petits ruminants (PPR) is a highly contagious and often fatal disease affecting sheep, goats, and wild small ruminants. It presents as an acute or sub-acute febrile condition, featuring symptoms such as fever, erosive stomatitis, conjunctivitis, gastroenteritis, and pneumonia. The disease poses a significant economic threat to small ruminant production. Peste des petits ruminants is prevalent in Sub-Saharan Africa, extending to the Arabian Peninsula, the Middle East, and Asia. The PPR virus spreads through close contact between infected and susceptible animals, commonly occurring in shared grazing and watering areas. Infected animals release the virus through exhaled air, secretions, and excretions. Field diagnosis is based on clinical, pathological, and epizootiological observations. Laboratory confirmation involves virus isolation, detection of viral antigens, nucleic acid isolation and sequencing, as well as identifying specific antibodies in the serum. To combat the disease, the Food and Agricultural Organization and the Office International des Epizooties have collaborated on a global eradication strategy. The goal is to control and eliminate PPR by the year 2030.

Keywords: Peste des petits ruminants, Sheep, Epidemiology, Control

Introduction

Peste des petits ruminants [PPR] is a highly contagious disease that severely affects small ruminants in nearly 70 countries across Africa, the Middle East, and parts of Asia. This disease causes annual losses of USD 1.5 to 2 billion in regions that are home to over 80% of the world's sheep and goats, impacting the livelihoods of more than 330 million of the world's poorest people who depend on these animals. The threat of PPR extends to food security and the economic potential of animal husbandry sectors.Peste des petit ruminants [PPR] is an acute or sub-acute febrile, highly contagious, and often fatal disease of sheep, goats, and wild small ruminants [1]. It is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, pneumonia, and leads to significant economic losses in small ruminant production [2,3]. The disease was first identified in Cote d' Ivoire, West Africa [4], and is also known by various names such as goat plague, pest of sheep and goats, Kata, stomatitis-pneumoenteritis syndrome, contagious pustular stomatitis, and pneumoenteritis complex [5]. The reference to the disease as a "plague" highlights its highly contagious nature and the resulting economic impacts. Only in the late 1970s was PPR confirmed to be a distinct virus from rinderpest virus through serology, biochemical, and cross-protection experiments [6-8]. Initially thought to be limited to West Africa, PPR has now been confirmed in several African, Middle Eastern, Central and South Asian countries, as well as China [9,10].

Etiology

Peste des Petits Puminants [PPR] is caused by the Paramyxovirus of the Morbillivirus genus. It was first described in 1942 in Côte d'Ivoire, West Africa, and is closely related to rinderpest virus, canine distemper, and human measles virus. The Peste des petits ruminants virus has an envelope derived from the host-cell plasma membrane, containing two transmembrane glycoproteins surrounding a nucleocapsid. The presence of the envelope makes virions sensitive to heat, lipid solvents or detergents, non-ionic detergents, formaldehyde, and oxidizing agents. Peste des petits ruminants virus is also very sensitive to ultraviolet radiation and desiccation. Like all enveloped viruses, PPRV is highly sensitive to heat. The half-life of the virus at 37° C was estimated at 2 hours, and at 50° C infectivity was destroyed in 30 minutes [11]. Other studies have confirmed and clarified the thermal sensitivity of PPRV [12,13]. The PPR virus is also sensitive to low pH, being destroyed after the animal's death due to the low pH that accompanies rigor mortis. Peste des petits ruminants virus is stable at a pH between 5.8 and 9.5 but rapidly loses activity at a pH below 4 or above 11 at room temperature [14].

The optimum pH for the survival of PPRV is between 7 and 8 [15]. Four lineages of PPR viruses have been identified: 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 [16].

Epidemiology

Peste des Petits Ruminants (PPR) is endemic in Sub-Saharan Africa and extends to the Arabian Peninsula. Historically, the disease was primarily associated with West Africa, but it extends in a belt across Africa immediately south of the Sahara, reaching into the Arabian Peninsula [17-19]. Adverse climatic factors, whether seasonal or not, affect the availability of pasture and water, leading to increased movements of small stock in search of better nutrition and shelter, thereby aiding the spread of the PPR virus to susceptible groups [20]. The seasonal epidemiologic patterns of the PPR disease differ in different ecological systems, geographical areas, and are dependent on the culture and livelihood patterns of small stock owners [21]. In Pakistan, seasonal outbreaks of PPR were alluded to [22,23], suggesting that seasonal grazing patterns among nomadic livestock keepers during winter encourage disease transmission [24]. Similar observations were made by [25], who associated PPR outbreaks in Bangladesh with winter grazing. PPR outbreaks among sheep and goats in India are described to occur at any time of the year, but are most frequent during the wet [April to September/October] or cold dry [January and February] seasons [26]. Peste des Petits Ruminants virus is transmitted by close contact between infected and non-infected susceptible animals, likely to occur in common grazing areas. Infected animals shed PPRV in exhaled air, secretions, and excretions approximately 10 days after the onset of fever. Sneezed or coughed out droplets by infected animals contain large amounts of the virus, which can spread the infection. Transmission between animals in the vicinity can occur through inhalation over a distance of about 10 meters. Infected fomites can act as a source of infection, although it is unlikely considering the rapid inactivation of the PPRV in external dry conditions. Peste des Petits Ruminants can be transmitted to the offspring by feeding them milk from an infected dam. The virus is thought to be present in milk from 1-2 days before the signs appear and last until 45 days after complete recovery [27].

Risk factors:

The spread of PPR outbreaks has long been associated with social, cultural, and economic activities such as conflicts, disasters, livestock trade, cultural festivals, and changes in husbandry practices, nomadism, and seasonal climatic and environmental variations [28,29]. A study conducted in Turkana, Kenya, revealed that the crowding of sheep and goats at watering points during the dry season was a significant risk factor for PPR outbreaks in 2009. Similarly, in 2010, the sharing of grazing and water by sick adult goats and sheep with lambs and kids was found to be a significant source of PPR outbreaks [30].

Host range:

Peste des petits ruminants primarily affects sheep and goats. Other domestic animals such as camels, cattle, and pigs can undergo subclinical infection of PPR [31]. The disease has been reported in wild small ruminants in zoos and in the wild [32-34]. Young animals are less likely to have developed protective antibody titers and are therefore more susceptible to PPRV [35]. This high susceptibility in the young has been reported in Ethiopia, Kenya, Pakistan, India, and Turkey [36-38]. In Oman, the disease is reported to maintain itself in susceptible yearling populations, with an increase in incidence reflecting the recruitment of susceptible young goats/sheep [39]. Results have shown insignificant differences between goat breeds but significant differences between sheep breeds. Breed differences in susceptibility to PPR have been reported in other studies [42,43].

Economic significance:

Peste de Petit Ruminants virus has a widespread distribution spanning Africa and Asia [44,45]. These areas encompass much of the developing world, which heavily relies on subsistence farming to supply food or goods for trade, and small ruminants provide an excellent supply of both. Unfortunately, in many areas of Asia and Africa, small ruminant production, and therefore the livelihoods of poor farmers, are threatened by PPR, among other trans-boundary animal diseases [TADs]. With its associated high morbidity and mortality, PPRV constitutes one of the major obstacles to subsistence farming [46].

Pathogenesis:

The route of infection is respiratory and is spread by airborne droplets. All secretions and excretions of infected sheep are contagious throughout the course of the disease, but no carrier state exists [47]. During the acute phase of the disease, the virus is shed in all secretions and excretions. Peste des petits ruminants virus exhibits lymphoepitheliotropism [48]. The virus causes cytopathic effects that are distinguished from those of other Morbilliviruses by its characteristic appearance of multinucleated cells capable of forming round mini syncytia and intracytoplasmic inclusion bodies and eosinophilic intranuclear inclusion bodies [49]. The fusion between an infected cell and neighboring cells (syncytia), aided by the viral fusion protein (F) expressed on the surface of infected cells, is one way the virus spreads. This process of spreading the infection from cell to cell through fusion allows the virus to continue the infectious process free of neutralizing antibodies, as nucleocapsids migrate from cell to cell without passing through the external environment.

Clinical Signs

The acute form of PPR is rare in sheep. Signs generally appear 3 to 6 days after being in contact with an infected animal, [50]. It is accompanied by dullness sneezing serous discharge from the eyes and nostrils. A day or two day later, discrete lesions develop in the mouth and extend over the entire oral mucosa, forming diaphtheric plaques. There is a profound halitosis and the animal is unable to eat because of a sore mouth and swollen lips. Nasal and ocular discharge becomes mucopurulent and the exudate dries up, matting the eyelids and partially occluding the external narres. Diarrhea develops 3-4 days after the onset of fever. It is a profuse and faeces may be mucoid and blood tinged. Dyspnea and coughing occur later and the respiratory signs are aggravated when there is secondary bacterial pneumonia. Erosion has been described in the vulva and prepuce. Abortion has been reported during outbreak in India and Tanzania [51]. Death usually occurs within one week of the onset of the illness.The sub-acute form is more common in sheep but they also occur in goats. This form has a longer incubation period of about 6 days. Sub-acutely affected animals are not severely affected and lack characteristic clinical signs and therefore mortality is usually very low. Lesions such as oral crusts due to mucosal discharges may appear making the disease to be confused with contagious ecthyma. Infected animals develop low grade pyrexia [39-40o C] and recover in 10-14 days and remain immune protected [52]. Hemorrhages in the digestive system and the liver reduce number of erythrocytes and hematocrit values significantly in animals naturally infected with PPRV. The virus has affinity for lymphoid organs contributing to marked immune-suppression as indicated by leucopenia, monocytes depletion and lymphopenia [53-54].

Competitive Enzyme-Linked Immunosorbent Assay [C-ELISA]

The C-ELISA is considered suitable for large scale testing due to its simplicity and availability of the recombinant antigen [55]. C-ELISA sensitivity is 99.4 % and specificity 94.5%. A competitive ELISA based on PPRV monoclonal antibodies specific for haemagglutinin [H] protein [56] or nucleoprotein [N] [55] was developed for detection of antibodies to PPRV in serum samples of sheep and goats.

Experimental infection

In contrast to natural PPR infections, experimental infection of susceptible animals with PPRV results in a clinical disease with high morbidity rate and low mortality rate [57,58]. A severe experimental form of disease has been reproduced in sheep and goats [59].

Necropsy finding

The carcass of a PPR affected animal is usually emaciated, the hindquarters soiled with soft/watery faeces and the eyeballs sunken. The eyes and nose contain dried-up discharges. Lips may be swollen and possibly scabs or nodules in late cases. The nasal cavity is congested [reddened] lining with clear or creamy yellow exudates and erosions. The pathology caused by PPR is dominated by necrotizing and ulcerative lesions in the mouth and the gastro-intestinal tract. Erosion in the oral cavity is a constant feature affecting the gums, soft and hard palates, tongue and cheeks and into the oesophagus. The abomasum is congested with multiple haemorrhages. The rumen reticulum and omasum rarely exhibit lesions. Occasionally, there may be erosions on the pillars of the rumen. The omasum is a common site of regularly outlined erosions often with oozing blood. Lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages and, occasionally, erosions in the first portions of the duodenum and the terminal ileum. The large intestine is usually more severely affected, with congestion around the ileo-cecal valve, at the ceco-colic junction and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion “zebra stripes” form on the crests of the mucosal folds. In the respiratory system, small erosion and petechiae may be visible on the nasal mucosa, turbinates, larynx and trachea. Bronchopneumonia may be present, usually confined to the antero-ventral areas, and is characterized by consolidation and atelectasis. The lung is dark red or purple with areas firm to the touch, mainly in the anterior and cardiac lobes show evidence of pneumonia. Lymph nodes associated with the lungs and the intestines are soft and swollen [60].

Histopathology of the disease

Peste des petits ruminants virus causes epithelial necrosis of the mucosa of the alimentary and respiratory tracts marked by the presence of eosinophilic intracytoplasmic and intranuclear inclusion bodies [61]. Infiltration of the lymphocytes, plasma cells and histiocytes into the alveolar septae leads to its hypertrophy and desquamation with alveolar casts [9]. Intestinal lesions are characterized by blunted villi, degeneration of surface and crypt epithelial cells; expansion of lamina propria by a primarily mononuclear infiltration with scattered syncytial cells [49].

Virus isolation

Detection of the virus is done by isolation of the PPR virus in cultured cells. This method of diagnosis can be very valuable as it provides live virus for biological characterization studies and the isolated viruses are stored for later studies [50]. Samples for virus isolation include heparinized blood, eye and nasal swabs [from live animals], tonsil, mesenteric lymph nodes, spleen, section of colon and lung from necropsied cases. For successful isolation, samples must be collected during the hyperthermic phase [62] and submitted to the testing laboratory in cold ice. The most widely used cell culture systems are primary lamb kidney and ovine skin [63,64] and Vero cells [65].

Molecular Techniques

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 [66,67]. This technique is 1000 times more sensitive than classical virus titration on Vero cells [66] with the advantage that results are obtained in 5 hours, including the RNA extraction, instead of 10–12 days for virus isolation. Tears- cotton buds or swabs of absorbent cotton wool are inserted into the conjunctival sac and swirled around to collect tears. The bud of swab is broken off and placed in Phosphate buffered saline. Gum debris- this material is scraped with a spatula from the gums and placed in Phosphate buffered saline. The tissues to sample include: Lymph nodes found around the lungs [mediastinal] and alimentary tract [mesenteric], portions of the spleen and the lungs. Two sets of each tissues are required; one set of chilled but not frozen and the other is put in 10% formalin solution to preserve the samples. It was found that even after three years of storage in formalin, this sample can still be used to recover RNA for PPR confirmation [68]. Unclotted blood is needed for virus isolation and should be collected in bottles containing anticoagulant [Heparin or Ethylenediaminetetr acetic acid [EDTA], serum is needed for antibody detection.

Prevention and Control

There is no specific treatment against PPR. 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 [69]. 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 [70]. Although PPRV is classified into four lineages based on F and N genes, they all belong to a single serotype. Therefore vaccination using vaccine prepared from any of the lineage will provide protection against all the lineages. A live attenuated culture vaccine based on Nigeria75/1 strain is widely used for vaccination and immunization in almost all the PPRV endemic areas of the world. This vaccine is safe for pregnant dams and induces immunity in at least 98% of the vaccinated animals in the field [71]. 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 degrees Celsius 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 [69].

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