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Review on Peste Des Petits Ruminants

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Abstract: Peste des petits ruminants (PPR) also known as goat plague, is a viral disease of goats and sheep characterized by fever, sores in the mouth, diarrhea, pneumonia and death. It is caused by a morbillivirus of the family of paramyxoviruses, which is related to rinderpest, measles and canine distemper viruses. The disease is considered as one of the main constraints hindering the productivity of small ruminants in enzootic regions of Africa, Asia and the Middle East. Peste des petits ruminants were first identified in Nigeria in 1942. Globally, there are four known lineages (I-IV) of the virus. PPR is a notifiable disease listed by the international animal health organization (OIE). Close contact facilitates the spread of the virus. In Ethiopia, the disease is endemic in sheep and goat rearing pastoralist areas. The per-acute and acute syndrome in sheep and goats can cause nearly 50-100% of morbidity and 10-90% fatality rate in naïve populations. Both clinical signs and mortality can vary widely depending on viral strains, breeds, co-infections and general nutrition and fitness. Prophylactic vaccination measure is the key control method to tackle the disease burden.

Key words: Peste Des Petits Ruminants Virus · Sheep · Goats · Epidemiology · Control

INTRODUCTION

Peste des petits ruminants (PPR) is caused by Peste des petits ruminants virus (PPRV). It is classified in the order Mononegavirales, family Paramyxoviridae, subfamily Paramyxovirinae and genus *Morbillivirus*. There is genetic similarity with other members of the genus *Morbillivirus* that includes Measles Virus (MeV), Rinderpest Virus (RPV), Canine Distemper Virus (CDV) and a number of other viruses that infect aquatic mammals. The non-segmented genome encodes eight proteins: the nucleocapsid protein (N), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin protein (H), the large polymerase protein (L) and the two non-structural proteins, C and V [1].

Peste des petits ruminants (PPR) is a highly contagious, fatal and economically important disease of both domestic and wildsmall ruminants. It also affects camels. Owing to high morbidity and mortality, PPR is included in the OIE (Office International des Epizooties) list of notifiable terrestrial animal diseases [2]. The disease is of emerging trans-boundary nature, which expanded from sub-Saharan Africa to Middle East, Turkey and the Indian subcontinent rapidly. Food and Agriculture Organization (FAO) has estimated that about 62.5% of the total small ruminant population is at risk of PPR around the globe [3].

Geographical Distribution: The current PPR situation is that around 70 countries have either reported infection to the OIE or are suspected of being infected. Of these, more than 60% are in Africa the other infected countries being in Asia (South-East Asia, China, South Asia and Central Asia/West Eurasia including Turkey) and the Middle East. Another 50 countries are considered to be at risk for PPR. As of May 2014, 48 countries in the world were officially recognized by the OIE as PPR free [4].

The different PPR viruses (PPRV) that have been isolated so far in all these areas were classified into four lineages (I - IV) based on partial sequence analysis of the F gene. Lineage I is represented by viruses isolated in Africa in 1970s. Lineage II which includes viruses isolated in the late 1980s in West Africa (Ivory Coast and Guinea)

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is the only African lineage that did not cross the Red Sea to the Asian countries. Lineage III is a combination of isolates from eastern Africa (Sudan and Ethiopia). Lineage IV of PPR virus isolates which includes the Asian isolates from Israel, Iran, Nepal, Bangladesh, Turkey and India, is confined to Asia [5].

Until 2000, lineage IV was confined to Asia and the Middle East. However, this lineage had recently been identified in Africa (in Sudan in the mid-2000s and in Morocco in 2008). The infection had also been identified in both Tunisia and Algeria. This situation, together with the first discovery of the disease in Uganda, Kenya and Tanzania during 2006-2007, indicated a shift in disease dynamics on the continent [6]. Recently, lineage IV has been identified in Ethiopia. The full genome sequence data of PPRV (Ethiopia/2010) clusters genetically with lineage IV isolates. This isolate was derived from the intestine of a goat suffering from severe clinical disease during the 2010 outbreak in DebreZeit, Ethiopia [7].

The distribution of PPR has steadily expanded, covering large regions in Africa, the Middle East and Asia. By 1989, PPR has spread to Egypt and by 1992 spread further south to Sudan. Between 1996 and 2001, PPR spread rapidly across Africa, the Middle East and Asia and by 2006, the most southerly affected African country was the Democratic Republic of Congo [8].

Host Range and Reservoirs: Peste des petits ruminants virus primarily infects sheep and goats, although both cattle and pigs are susceptible to infection, but do not contribute to the epidemiology as they are unable to excrete the virus. The existence of sylvatic reservoirs for PPRV has been reported with infections and deaths in captive wild ungulates from several species having been described previously [9, 10]. Clinically, PPR is seen in both sheep and goats however, goats are more susceptible than sheep. Breed of goats play an important role in susceptibility as Guinean breeds of West African dwarf goats such as Lagoon, Kirdi and Djallonké breeds are considerably more susceptible than the major Sahelian breeds [9]. Peste des petits ruminants is now recognized as an emerging disease in camelids causing respiratory syndrome in Sudan [11]. According to Abraham *et al.* [12], a 3% antibody seroprevalence was recorded in camels in Ethiopia. A serological evidence of camel exposure to PPRV was also onfirmed in Tanzania with overall seroprevalence of 2.6% [13]. Peste des petits ruminants virus antibody was also detected in dogs in India [14]. There is a report that the virus was recovered from biting midges [15].

Table 1: etection of PPRV in Wildlife Species.

Species	*Latin name*
Laristan sheep	*Ovis gmelini laristanica*
Gemsbok	*Oryx gazella*
Dorcas gazelles	*Gazella dorcas*
Thompson's gazelle	*Eudorcas thomsonii*
Nubian Ibex	*Capra nubiana*
Indian buffalo	*Bubalus bubalus*
African Grey dukier	*Sylvicapra grimma*
Arabian oryx	*Oryx leukoryx*
Bubal hartebeests	*Alcelaphus buselaphus*
Buffaloes	*Syncerus caffer*
Defassa waterbuck	*Kobus defassa*
Kobs	*Kobus kob*
Arabian mountain gazelles	*Gazella gazelle cora*
Springbuck	*Antidorcas marsupialis*
Arabian gazelles	*Gazella gazella*
Barbary sheep	*Ammotragus lervia*
Bushbucks	*Tragelaphus scriptus*
Impala	*Aepyceros melampus*
Rheem gazelles	*Gazella subguttorosa marica*
Afghan Markhor goat	*Capra falconeri*

Source: [1]

The Occurrence of PPR in the Livestock-Wildlife Interface: It is believed that PPR virus circulates in domestic ruminants and acts as a potential source of virus for wildlife. It is quite possible that in cases of pastures exchange between domestic and wild animals, the spread of PPR is facilitated between the two populations [16, 17]. In Mongolia, the massive death of wild animal populations occurred due to PPR [18]. In Africa, PPRV spillover from a domestic source was suggested in the Serengeti ecosystem in Tanzania with higher antibody prevalence in wildlife close to livestock, but without evident clinical syndromes or mortality [19].

Peste des petits ruminants was detected from different wild ruminants (Table 1).

Transmission: The transmission of PPRV mainly occurs during close contact. Inhalation is thought to be an important route of spread. This virus can be shed during the incubation period and has been found in nasal and ocular secretions, saliva, urine and diarrheic feces [20, 21]. Fomites such as water, feed troughs and bedding can probably transmit PPRV for a short time, but do not remain infectious for long periods. There is very little information on the survival of PPRV in the environment; however, this virus is very similar to rinderpest virus, which is inactivated by ultraviolet light and desiccation within 3-4 days or less and normally survives for very short periods in carcasses. Temperatures above 70°C, as well as PH less than 5.6 or greater than 9.6, are also expected to inactivate PPRV [21, 22].

Risk Factors: Age is an important to risk factors, with animals aged 3 to 18 months being more severely affected than adults or unweaned young [10]. Kids over 4 months and under 1 year of age are most susceptible to the disease. Breed of sheep and goats is another risk factor. Sahelian breeds of sheep and goats are believed to be more resistant than the dwarf breeds in the humid and sub-humid zones of West Africa. In a particular flock, the risk of an outbreak is greatly increased when a new stock is introduced or when animals are returned unsold from livestock markets [23]. Recovered animals have lifetime immunity [20]. Climatic condition is also a risk factor and outbreaks are most frequent during the rainy season and/or the cold dry season [10]. Species-wise, goats were found to be more susceptible than sheep [24]. Asthe size of a flock becomes larger, the likelihood of being infected is higher [23]. Likewise female animals are more susceptible than males [25].

Pathogenesis: Peste des petits ruminants is an acute or sub-acute and highly contagious viral disease of small ruminants. Because of the respiratory signs, PPRcan be confused with contagious caprine pleura-pneumonia (CCPP) or pasteurellosis. In many cases, pasteurellosis is a secondary infection of PPR, a consequence of immunosuppression that is induced by PPRV. Peste des petits ruminants virus is transmitted mainly by aerosols between animals living in close contact [5]. Peste des petits ruminants virus penetrates the retropharyngeal mucosa, sets up a viremia and specifically damages the alimentary, respiratory and lymphoid systems. Infected cells undergo necrosis and in the respiratory system, also proliferation. Death may occur from severe diarrhea and dehydration, before respiratory lesions become severe, or is hastened by concurrent diseases such as pneumonic pasteurellosis, coccidiosis or coliform enteritis [20].

Clinical Findings: Peste des petits ruminants have an incubation period of 2-7 days [26]. It is followed by the sudden onset of pyrexia (40-42°C) that could last for 3-5 days, severe depression, anorexia and clear nasal and ocular discharges that become mucopurulent due to secondary bacterial infection. Crusts may form on the nose, resulting in the blocking of the nostrils and respiratory distress, while matting together of the eyelids may also result. One to two days following the onset of the pyrexia, the oral and ocular mucous membranes become hyperemic. This then is progressing to multifocal pin-point necrosis of the epithelium of the gingiva, dental pad, palate, lips, inner aspects of the cheeks and the upper surface of the tongue. These necrotic areas extend and may even coalesce [8].

Affected animals have obvious signs of bronchopneumonia, characterized by increased respiratory rates, extension of the head and neck, dilatation of the nostrils, protrusion of the tongue, rales, abdominal breathing and soft painful coughs. A common feature in the later stages of the disease is the formation of small nodular skin lesions onthe outside of the lips and around the muzzle. Abortions may occur in pregnant animals. Death often follows within 7-10 days from the onset of the clinical signs due to severe dehydration, emaciation, dyspnea and hyperthermia. Some infected animals may recover after a protracted convalescence[8].

Differential Diagnosis: Frequently, PPR is confused with other diseases that have grossly similar clinical signs. These diseases include rinderpest, foot and mouth disease (FMD), bluetongue, contagious ecthyma (Orf), pneumonic pasteurellosis, Nairobi sheep disease, contagious caprine pleuropneumonia (CCPP) and gastro-intestinal helminth infestations. The most frequent sources of confusion are the mouth lesions, which could be due to rinderpest, FMD, bluetongue or orf; difficult breathing, which could be due to pneumonic pasteurellosis or CCPP or diarrhea which could be due to coccidiosis or gastro-intestinal helminth infestations [21, 27].

Diagnostic Methods

Clinical Diagnosis: Clinical diagnosis of PPR in the field is based on the symptoms such as pyrexia, lachrymation, nasal discharges, oral erosion, pneumonia, diarrhea and death. Historic epidemiological information of PPR in the region or farms can help field personnel to report a suspicious case. A differential clinical diagnosis should be made with other syndromic diseases. However, it is recommended to sample sick animals for a confirmatory diagnosis [28].

Post Mortem Diagnosis: Erosive lesions may be extensive, extending from the oral cavity to the reticulo-rumen junction. There is an apical pneumonia, with enlarged, edematous and congested lymph nodes. Pleuritis and hydrothorax may be present. The spleen is congested and enlarged and necrotic lesions may be

present. Necrotic or hemorrhagic enteritis is usually present and linear hemorrhages or zebra stripes may be located in the colon and caecum [8, 21]. Small erosions and petechiae lesions are observed on the nasal mucosa, turbinates, larynx and trachea [21].

Laboratory Diagnosis

Virus Isolation: This technique needs cell culture facilities which are not common in many laboratories in the developing countries. Where this is possible, primary cell culture from lamb or kid kidney and lung, were used for the virus isolation along with different cell lines such as: Vero cells, MDBK (Madin-Darby Bovine Kidney), marmoset-derived cell line (B95a). Recently, it has been developed a new and very sensitive cell line using the monkey cell expressing sheep-goat SLAM (Signaling Lymphocyte Activation Molecule) receptor. Usually, cultures are examined for the cytopathic effect in the days following infection of a monolayer with suspect material. The identity of the virus can be confirmed by virus neutralization or molecular techniques. Alternatively, specific antigens and antibodies can be detected [21, 28].

Antigen Detection: Counter-Immuno-Electrophoresis (CIEP) and Agar Gel Immuno-Diffusion (AGID) tests using hyper-immune serum:

- ✓ Agar Gel Immuno-Diffusion test is simple and can be performed in a basic laboratory but remains relatively insensitive. Moreover, it cannot distinguish PPRV from RPV.
- ✓ Counter-Immuno-Electrophoresisis sensitive and specific and able to differentiate PPRV from RPV sample [28].

Immunohistochemestry (IHC) on Tissue Samples: It allows the localization of specific PPRV antigens in a pathological tissue sample [28].

Antibodies Detection: Viral neutralization test (VNT): is applied to a serum sample; this technique needs also cell culture facilities [28].

Molecular Techniques

Antigen Detection: Immuno-capture Enzyme Linked Immuno-Sorbent Assay (IC-ELISA): It is sensitive and specific method to detect the presence of PPRV antigens. It is easy to run and is well established in many laboratories in developing countries [28].

Antibodies Detection: Detection of antibodies against PPRV is carried out byusing ELISA techniques. Currently the use of competitive PPRV-specific anti-H (H-cELISA) or anti-N (N-cELISA) monoclonal based ELISA is routinely effective in laboratories where the disease exists. Both competitive ELISA tests can be used equally for the detection of PPRV antibodies [28].

Genome Detection: Real-time Polymerase Chain Reaction (RT-PCR) is an accurate, rapid and reliable method that can be used for the detection and also for the quantization of specific DNA molecules [29].

The conventional RT-PCR has been developed for the specific amplification of the NP gene or for the amplification of the fusion (F) gene and is established in various laboratories. The real-time PCR assay specific for PPRV and the loop-mediated isothermal amplification technique (LAMP-RT-PCR) are also available for the genome detection of PPRV [28].

Treatment, Control and Prevention: There is no treatment for PPR but it helps to give broad-spectrum antibiotics to stop secondary bacterial complications [30].

The controls of PPR require an effective vaccine and for this purpose several vaccines including both homologous and recombinant vaccines have been developed [31, 32]. Nowadays, efficient live attenuated PPR vaccines are available that can induce lifelong protective immunity in vaccinated animals [4, 33]. The challenges in control activities arise include it is not possible to distinguish vaccinated animals from those that have recovered from natural infection. A differentiation of infected from vaccinated animals (DIVA) vaccine/test would improve epidemiological data by allowing tracking of infection in areas where there has been partial vaccination. Animals that have been infected are detected by the presence of antibodies to the N protein, while vaccination coverage can be assessed by the presence of antibodies to the H protein in the absence of antibodies to the N protein [33]. Thermo-stabilizing PPR vaccines are compatible with the shipment of the vaccine to remote areas without the need for a cold chain. Currently, Pan African Vaccine Center (PANVAC) which is found in Ethiopia is producing and distributing effective PPR vaccine for Ethiopia and some African countries.

In general, the control of the disease is more effective by applying measures such as the slaughter of the infected herd, correct disinfection and adequate disposal of carcasses, movements control, emergency vaccination

and quarantine [28]. Other preventive actions include public awareness creation, quick report, surveillance and treatment of products and by-products [34].

PPR in Ethiopia: Owing to their high fertility, short generation interval and adaptation even in harsh environments, sheep and goats are considered as an important asset of poor farmers and they are exploited for diverse purposes in the Ethiopia [35]. The first suspected clinical case of PPR was identified in 1977 in Ethiopia. Currently, Lineage III and IV are found in Ethiopia. Lineage III has been found to be circulating in East Africa countries such as Kenya, Sudan, Uganda and Tanzania [1]. In 1999, a serological survey on PPR was conducted in Ethiopia with the aim of informing a subsequent vaccination campaign which would be the first large scale vaccination campaign against PPR in the country. The result showed district level seroprevalence estimates ranged from 0% for Guba in the Benishangul region or Abala in the Afar region to 52.5% for Dolo Odo in the Somali region [36]. Districts with the higher prevalence levels seem to be mainly those in areas of low altitudes where pastoral management systems prevail over sedentary ones. Similarly, all villages in Afar and 97% of the studied villages in Gambella regions were affected by the PPR virus [25]. Reasons for this may be related to different production systems with exchanges and movements in areas of lowlands being more frequent and involving larger numbers of animals. This is particularly important during the dry season and in low altitude areas. In addition, animals are exchanged between households and flocks as a result of social practices and changes in economic conditions that exhibit seasonal patterns. The seasonality of animal movements could partly explain the occurrence of the disease in Ethiopia mainly between the months of March and June [36].

Molecular epidemiological analysis of outbreaks made in different parts of the Ethiopia showed that samples were positive for the virus [37]. A cross-sectional study conducted in Kafa and Bench Maji zones in SNNPR and showed an overall prevalence of 2.1% (n= 968, 95% CI= 1.2 - 3.0%) at individual animal level and 18.8% (95% CI= 10.9 - 26.6%) at flock level [23]. The overall seroprevalence of PPR in sheep and goats in Eastern Amhara region in unvaccinated flock was 26.9% (n=133) and 28.6% (n=196), respectively [38]. A cross-sectional seroprevalence study conducted in southern region of Tigray revealed 47.5% prevalence in goats [39]. A study showed PPR antibody seroprevalence in unvaccinated herd/flock was 3% (n=628) in camels, 9% (n=910) in cattle, 9% (n=442) in goats and 13% (n=835) in sheep in Ethiopia [12]. Similarly, a report showed an overall seroprevalence of 29.2% (n= 390) in Gurage and Siltie zones in sheep and goats [24]. A serological survey conducted in lowland pastoralist areas in Afar and Gambella regions, a prevalence of 31% (n= 1163) was found [25].

Table 2: Prevalence of PPR in the Seven Surveyed Regions of Ethiopia

Region	Number of samples collected	Prevalence with 95% CI
Afar	1653 (12.1%)	15.3% (13.6-17.0)
Amhara	5992 (43.9%)	4.6% (4.0-5.1)
BenishangulGumuz	729 (5.3%)	8.0% (6.0-9.9)
Oromia	2290 (16.8%)	1.7% (1.2-2.2)
SNNPR	1622 (11.9%)	1.8% (1.1-2.4)
Somali	465 (3.4%)	21.3% (17.6-25.0)
Tigray	900 (6.6%)	15.3% (13.6-15.9)
Total	13,651 (100%)	6.4% (6.0-6.8)

Source: [36]

Table 3: Summary of PPR studies in Ethiopia.

Study areas	Sample Size	Prevalence	Author
Bench Maji and Kafa zones	968	2.1%	23
Gurage and Silte zones	390	29.2%	24
Afar and Gambella regions	1163	31%	25
East Amhara	196	28.1%	38
Southern Tigray	240	47.5%	39
Awi, East Gojam, West Gojam, North Gonder, South Gonder	672	18.3%	40
East Sewa and Arsi zones	700	48.43%	41

Ethiopia has launched progressive PPR control strategy. The strategy will adopt geographic approaches. The initial area of operation will include at least the epidemiologically interconnected pastoral areas of the country, where a progressive control (ultimately leading to eradication) program will be implemented in strategically defined epidemiologically important (sub)-ecosystems. These areas include Somali and Afar regional states, pastoral districts of Oromia and South Omo zone of SNNPR. The strategy of the highland lowland interface will be similar to that of the pastoral areas. It includes districts immediately adjacent to the pastoral areas of the country and epidemiologically closely linked to the pastoral areas through seasonal grazing and marketing [42].

The approach towards controlling PPR can be divided into three inter-dependent phases based on the epidemiology of the disease and prioritizing available resources. The first phase will establish a better understanding of the disease situation and implement disease control strategies that will progressively control the PPR until the stage that there is evidence that the (sub)-ecosystem is clinically PPR disease free.

The second phase will start when the veterinary services stops vaccination in the (sub)-ecosystem and intensifies clinical disease search/surveillance to verify absence of clinical disease for a period of about two years and simultaneously prevent reintroduction of PPR. The third phase will serologically verify the absence of circulating antibodies and by this the absence of possible mild disease circulating in all susceptible species [42]. In general, mass annual vaccination programs have been practiced since 2005 with annual vaccination coverage reaching nearly 6 million heads (20%) of small ruminant are vaccinated [43].

Economic Significance: Peste des petits ruminants is generally considered a major constraint for small ruminant production; however, the economic impact of the disease has not been fully evaluated [44, 45]. The morbidity and mortality rates of PPR can be up to 100% in severe outbreaks but in milder outbreaks, mortality rate may be reduced to 50% while morbidity rate still remains high in both cases [46]. This is of particular concern for the economics of small rural farms, where sheep and goats are reared as the sole source of income. Moreover, PPR is most prevalent in countries that rely heavily on subsistence farming of small ruminants for trade and food supply [2]. Collectively, a review showed that PPR causes a loss of US$1.5 million annually in Nigeria, US$39 million in India and at least US$1.5 million in Iran [2]. It was estimated that about US$15 million loss in Kenya [47].

CONCLUSION

Peste des Petits Ruminants (PPR) is a viral disease affecting predominantly small ruminants. The clinical signs in diseased animals include pyrexia, naso-ocular discharge, respiratory tract infection leading to pneumonia, ulcerations and inflammation of the gastrointestinal tract leading to severe diarrhea. Compulsory notification to the World Animal Health Organization (OIE) of the presence of PPR in a country leads to restrictions on the movements of livestock and animal products. Given the high morbidity and mortality of PPR infection in immune-naïve small ruminants, the economic and food security impact of outbreaks is large for small-holder farmers. A better understanding of the epidemiology of PPR in the region and its contributing factors will be necessary for eradication. Due to its trans-boundary nature, regional coordination of control strategies will be the key to the success of the on-going PPR eradication campaign.

Recommendations:

- Seasonal vaccination practice needs to be applied in countries to contain an outbreak of the disease.
- Thermostable vaccines need to be timely dispatched to remote pastoralist areas.
- More research needs to be conducted in livestock-wildlife interfaces.
- Endemic countries should attempt their maximum effort to comply with PPR eradication time frame to achieve the goals.
- Researches in large animals should be conducted to understand their role in the epidemiology of the disease.
- Socioeconomic assessment of the disease has to be conducted to design and implement the control strategy well.

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