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Peste des petits ruminants: A comprehensive overview

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Abstract
Peste des petits ruminants (PPR) is an acute, highly contagious, OIE notifiable, fatal disease of sheep and goats caused by Morbilli virus having many common names, such as ovine rinderpest, goat plague, plague of small ruminants or Kata. The disease is endemic in many parts of the world including Africa, Asia and Middle East. The means of spread of virus is through close contact between infected and susceptible population, primary route of transmission being the respiratory route. The Peste des petits ruminants virus (PPRV) display a strong tropism for lymphoid tissues where it replicates after which the virus is disseminated to distant organs and causes profound immunosuppression. Clinical signs mainly seen are fever, mucopurulent nasal and ocular discharges, cough, dyspnoea, erosions in the oral mucosa, bronchopneumonia and diarrhea. The disease has been the cause of economic loss in developing countries where rearing of small ruminants act as source of income. Currently, the OIE recommended competitive PPRV specific anti-H monoclonal based ELISA is used and isolation of virus in cell cultures, molecular techniques PCR and RT-PCR can be used for diagnosis. Treatment is mainly based on supportive therapy, herbal medicines and sometimes antiviral therapy is used. Control measures include effective cleaning and disinfection of contaminated areas, deep burial/burning of dead animals/carcasses, close monitoring of animals and vaccination as well as mass vaccination with several PPRV vaccines alongside the Nigeria 75/1 vaccine, with live attenuated Sungri/ 96, Arasur/ 87 and Coimbatore/ 97 vaccines are being used in developing countries.
This review summarizes etiology, transmission, host susceptibility, pathogenesis, clinical signs, diagnostic methods, treatment and prevention and control measures.

Keywords: Peste des petits ruminants, small ruminants, Morbilli virus, diagnosis, vaccine

Introduction
Peste des petits ruminants is an acute, highly contagious, and frequently fatal disease caused by Morbilli virus in sheep and goats [14]. This disease is also known as ovine rinderpest, goat plague, plague of small ruminants, pneumoenteritis syndrome, pneumoenteritis complex, contagious pustular stomatitis or kata [39, 7]. PPR was first reported in 1942 in Cote d'Ivoire (West Africa) [39, 70, 14, 33,] and later from other parts of the world namely sub-Saharan Africa, the Middle East and Indian subcontinent [86, 89] and now this disease is emerging and has become endemic in many parts of the world including Asia [38, 45, 15]. Apart from small ruminants, wild animals like ibex and gazelle are also affected [88, 3]. PPR because of its high mortality and morbidity rate [57], causes devastating socio-economic impact [93] and responsible for severe economic losses as small ruminants play a major role in the food production chain of developing countries [62] as well as contributes in alleviation of poverty and food security in the livelihood of the poor and marginal farmers in the endemic countries like African and Asian continents [15, 93].

Etiology
PPRV is classified as a small ruminant morbilli virus which belongs to the largest member of the genus Morbilli of the family Paramyxoviridae (sub family Paramyxovirinae) under the order Mononegavirales [40, 15, 5]. Other morbilli virus includes Rinderpest virus, Measles virus, Canine distemper virus, Porcine distemper virus, Cetacean morbilli virus, the morbilli virus of marine mammals, and Feline morbilli virus [100]. PPR virus is closely related to the Measles virus and Canine distemper virus, [45, 38] but was later shown to be antigenically [40] and genetically distinct [31]. The PPR virus is pleomorphic in nature and has a capsid with a lipoprotein envelope which surrounds its ribo-nucleoprotein core [42, 14]. The RNA genome is single stranded, approximately 16 Kilo bases (kb) long size with negative polarity [20].The virus has 15948 nucleotides and 8 genes arranged in order of [3'-N-P/C/V-M-F-HN-L-5'] [10],

separated by inter-genic region [30] and the nucleotides follows the ''rule-of-six'' [17]. These genes are responsible to produce 6 structural proteins, N-[nucleocapsid], P-[phosphoprotein], M-[matrix protein] F-[fusion protein], HN-[haemagglutininaminidase protein], L-[large/ polymerase] and two nonstructural proteins [protein C and protein V] [10, 59, 60, 75].

PPRV has been classified into four lineages based on molecular genotyping. Lineage I are commonly found in West Africa and have recently been reported in central Africa, lineage II isolates are found in western Africa, lineage III in eastern Africa, and some parts of the Middle East and the lineage IV are found in the Arabian Peninsula, the Middle East and South Asia [86, 96]. Recent reports showed the emergence of PPR virus lineage IV in the northeast and North Africa [18] However, a recent appearance of lineage IV, associated with a large epizootic in Morocco, undermines the probable risk of introduction of this lineage into many European countries [55]. Spread of disease is now a cause of global concern with involvement of various lineage of PPRV and especially recent introduction of Asian lineage in some African countries and presence of PPR in Europe through Western Turkey [4, 14, 55, 66].

Transmission and host susceptibility

The primary route of transmission of this virus is via respiratory route and spread of virus occurs through close contact between infected and susceptible population. Transmission could also occur through contaminated feeding troughs, water and bedding [56]. Viraemia develops 1-2 days prior to the appearance of first clinical sign while fine infective droplets are released into the air from all kind of the secretions and excretions of the infected animals [13, 84] starting from 3 to 22 days post infection [23, 63, 71]. It was reported by Abengunde and Adu (1976) that infected goats excreted the PPR virus from their nasal and ocular routes at the onset of diarrhea. Goats are considered more susceptible to PPRV compared to sheep but may infect wild small ruminants occasionally [64, 23, 94, 11]. Although natural disease in the gazelle (*Douces gazalla dorcas*), laristan sheep (*Ovis orientalis laristanica*), gemsbok (*Oryx gazella*), ibex (*Capra ibex nubiana*) and deer have been reported [73] but highly devastating condition is seen in sheep and goats [100, 87]. Besides usual hosts (small ruminants, wild ungulates), PPRV has the potential to infect large ruminants, camels and other unusual hosts [76]. Though, cattle, buffaloes, camels and pigs can become infected but there is little or no evidence of the disease associated with their infection [37]. However, the epidemiological role of wild boar and pigs in inter and intra-species transmission of PPRV has been highlighted in recent experiments [82]. Lately, outbreak of PPRV in India has been reported from an endangered species of bovine, the four-horned antelope (*Tetracerus quadricornis*), and attributed to the PPRV lineage IV [44].

Pathogenesis

After the PPRV enters the host through their oral and nasal passages, the virus is initially taken up by antigen presenting cells (APCs) present in the intraepithelial space and lamina propria of the respiratory mucosa (naso-pharyngeal/respiratory epithelium). (Kumar *et al.*, 2014) [51] from where it is transported to regional lymphoid tissues where primary virus replication takes place. The morbilli viruses display a strong tropism for lymphoid tissues where a second round of replication occurs in regional lymphoid organs, after which the virus is disseminated to distant organs and causes destruction of leucocytes which often results in profound immunosuppression [34, 69, 77].

PPR virus leads to extensive necrosis in lymphoid organs such as peyer's patches, spleen, thymus, pulmonary lymph nodes and hence results leucopenia [72, 50, 52, 49]. Clinical signs usually develop within 3-5 days following establishment of pyrexia and depends on route of infection, pre-existing conditions, nutritional status of the animal may also contribute in determining the severity of the disease as well as mortality and morbidity rates. As disease progresses, mucosal hyperaemia, mucoid nasal discharges, anorexia and diarrhea observed. Upon exposure to virulent PPR virus, susceptible animals usually develop acute pulmonary congestion and oedema and succumb to death within a week. Some of the animals in contrast, may develop a prolonged, chronic infection characterized by giant cell pneumonia, which is sometimes complicated by bronchopneumonia [67].

Clinical signs

Clinical signs of PPR disease appear as erosive stomatitis, diarrhea, pyrexia, ocular and nasal discharge [32, 78]. However, disease severity depends on various factors i.e. PPRV lineage, species, breed, and immune status of the animals [65]. It is assumed that sheep may hold some innate resistance to clinical disease [85].

Acute form: The disease is characterized by sudden onset of fever with rectal temperature of at least 40 - 41.3 °C [8, 73]. A bloated appearance is evidenced due to erection of hair. Subsequently, there is anorexia, oculonasal discharge and discharges from mouth. Initially, the discharges are watery, but later become thick, leading to matting of the eyelids, obstruction of the nose resulting in difficulty in breathing [8]. Oral necrotic and erosive lesions in lips, gums, tongue, palate and cheeks are seen. Affected animals resist attempts to open their mouths because of pain. Two to three days (2-3) after the onset of fever, diarrhea commonly appears. It may be watery, foul-smelling and may contain blood and dead tissues [36, 37, 99].

Sub acute form: The subacute form generally occurs in sheep but may also affect goats. Necrotic ulcers are uncommon and the affected animals are usually recovered. The duration of the disease is usually more than two weeks and death is not obvious [102].

Abubakar *et al*. (2008) has reported that PPR has a possible association with mortality and occurrence of high rate of abortions in goats. There are also reports of high morbidity and mortality rates in both sheep and goats due to PPR [3, 27, 56] and in severe outbreaks, mortality can reach up to 100% [73] but these rates may vary as mortality being low as 20% [78, 2]. Das *et al*. (2007) reported the morbidity and mortality rate of 74% and 55% in PPR and these rates are higher in sucklers than in adult animals. High temperature leads to erosive and ulcerative lesions [53] resulting in concurrent bacterial, viral or parasitic infections which may further aggravate condition and mortality up to 100% [48].

Diagnosis

The methods for diagnosis, prevention and control of the agent vary widely and depend on factors such as local facilities, techniques adopted and the provision of vaccine and availability of veterinary services. OIE has recommended the

use of the competitive PPRV specific anti-H monoclonal based ELISA (cH-ELISA) [6] and virus neutralization tests [37]. However, there are several alternative methods [22, 58] including the indirect ELISA [43], immunofiltration [29], a novel sandwich ELISA [80], haemagglutination tests [28, 35] and latex agglutination tests [46]. Virus isolation in cell culture can also be attempted with several different cell lines, although recovery of virus is not always successful. Previously, a marmoset-derived cell line (B95a) was primarily used [91] although primary lamb kidney or African green monkey kidney (Vero) cell cultures have also been successful [59]. However, morbilli viruses are now recovered and grown in Vero/SLAM cells [68, 83]. Generally, cultures are examined for cytopathic effect in the days following infection of a monolayer with suspected material; the identity of the virus can be confirmed by virus neutralization or molecular techniques [89].

For molecular detection, standard RT-PCR [24, 38] has been superseded by real-time RT-PCR assays specific for PPRV [16, 54] and loop-mediated isothermal amplification techniques [97]. The generation of a standard RT-PCR product is, however, necessary in order to perform sequence analysis and subsequent phylogenetic characterization of novel virus isolates. Extensive validation of these diagnostic techniques is required before they can be accepted as approved OIE methods.

A novel pen-side lateral flow immunochromatographic assay based on the affinity and specificity of monoclonal antibody (MAb) C77 using hybridoma cells technology in a mini Perm bioreactor and purifed on a protein G HiTrap column for diagnosis of PPR was developed at The Pirbright Institute (Pirbright, UK) in 2014. Another technology using Oxford nanopore MinION sequencers has been developed which is non-PCR-based tool and used for meta-transcriptomic detection of RNA virus from the clinical samples [47]

Recently, in China, the State Key Laboratory of Agricultural Microbiology have developed a fast and ultrasensitive quantum dots lateral flow immunoassay strip to detect anti-PPRV antibodies. In this assay, the detection zone of the test strip contains immobilised N protein of PPRV and luminescent water-soluble carboxyl-functionalised quantum dots were used as signal output and were conjugated to streptococcal protein G. The performance of the test is remarkable compared to C-ELISA and the IC-LFD for PPR serum IgG antibody detection [21]

Treatment

There is no direct treatment available for PPRV infection itself, but antibiotics may be given to prevent secondary infections and subsequent other treatments can be given to alleviate the clinical signs. Generally, chloramphenicol 10 ml/kg body weight, penicillin 10,000 IU/kg, streptomycin 10 mg/kg, each given intramuscularly for 5 days), intestinal sedatives (EnteroSediv, 20 ml/kg for 4 days orally) and fluid therapy (Pedialyte, 30 ml/kg, for 4 days subcutaneously) are used to treat pneumonia, diarrhoea and restore body fluid ionic balance [101].

Antiviral treatment strategies

Research showed that antiviral therapy can effective method for control of PPR [3]. However, it is important to check the efficacy, drug administration route and compound clearance from the body of the animals because those animals are the source of food and relevant bi-products for humans, e.g., milk, cheese and meat etc [41]. De-Almeida *et al*. (2007) studied about silencing of the gene expression by using RNA interference that is an antiviral strategy to control PPR. PPR virus replication can be inhibited by use of the RNA interference. It has been reported that synthesized compounds like 4, 4′ (arylmethylene) bis (3 – methyl – 1 – phenylpyrazol – 5 - ols) have outstanding antiviral activity against PPRV and have more effectiveness than the standard ribavirin drug used [3, 92].

Use of hyperimmune serum and fluid therapy in the early stages of the disease is recommended. Hyperimmune serum should be used in case of valuable sick animals. Lesions around the nostrils, eyes and mouth should be cleaned along with providing good nursing [74]. Use of symptomatic and supportive treatment. Wosu (1989) illustrated via an experiment that PPR cases treated symptomatically. Intestinal sedatives, broad spectrum antibiotics and fluid therapy is commonly used for the treatment of diarrhea, pneumonia and the restoration of the body fluid ionic balance along with good feeding and hygienic conditions. The survival rate of goats by this treatment was raised to 13.3%. Supportive treatment includes broad spectrum antibiotics to prevent secondary bacterial infections.

Herbal treatment strategies

Ethnoveterinary medicine and herbal strategies when combined with vaccination showed good results [79]. Wosu *et al.* (1989) reported that lemon fruit and *Citrus aurantium* are effective for the treatment of the orf-like labial scabs and increase the chances of recovery from PPR. Various transgenic plants expressing vaccine antigens has been shown to produce specific immune responses for oral immunization [61].

Prevention and control

The World Organization for Animal Health (OIE) and the Food and Agriculture of the United Nations (FAO) released a strategy in 2015 aiming for global eradication of PPR by 2030. [10, 49]. Control strategies require a strong support of diagnostic methods as well as proper and scheduled vaccination of the susceptible population which may vary from country to country based on the prevalence of disease but choices are limited in developing or under-developed countries. Control measures also include strict quarantine of any new animal (s) brought into the flock for at least 2-3 weeks to ascertain their health status and control of animal movements should also be monitored. Migratory flocks should be avoided as they may possess threat to local sheep and goat. Effective cleaning and disinfection of contaminated areas, deep burial/burning of dead animals/carcasses, close monitoring of animals for any developing clinical signs of disease, isolation of sick animals from the flock and examination of sick animals by veterinarians are important parts of containment of the disease [12, 95]. Current control of the disease include focused vaccinations in high-risk populations of sheep and goats, followed by mass vaccination campaigns by administration of a live-attenuated vaccine or a thermostable live-attenuated conventional or recombinant vaccine to avoid cold chain-associated problems in tropical and subtropical countries [84, 95].

The tissue culture rinderpest vaccine (TCRV) was used formerly to protect sheep and goats from PPRV which provided cross-neutralising antibody protection for at least 12 months but during the rinderpest eradication the need to stop

vaccinating animals with the TCRV led to the requirement of a homologous PPRV vaccine, Nigeria 75/1, generated by serial passages of a virulent PPRV strain in cell culture and was reported to protect goats and sheep from wild-type PPRV isolates for at least 3years post-vaccination. [19, 81] The existence of only one serotype of PPRV made this vaccine able to afford protection against all four PPRV lineages [95]. Vaccination against PPR has been practiced in some states of India since 2002 [89] and has resulted in decreased numbers of outbreaks as well as changes in the severity of disease patterns which might be due to the effectiveness of live attenuated vaccines, scheduled vaccination of sheep and goats, and circulation of a single Asian lineage IV PPRV, since the disease was first reported in India [12] Currently, several PPRV vaccines alongside the Nigeria 75/1 vaccine, with live attenuated Sungri/ 96, Arasur/ 87 and Coimbatore/ 97 vaccines are being licensed to be used in India [81]. In countries like China, mass vaccination with vaccine based on lineage II PPRV Nigeria 75/1 has being implemented routinely as a major control strategy [61]. However test and slaughter of infected population has been considered as the best control measure to maintain the free status in certain countries free from PPR [90].

Conclusions

Peste des petis ruminants disease is emerging at an accelerating rate throughout the worldwide and draws a major concern as it continues to disrupt economic as well as social condition. Speading and prevention of PPR in endemic countries must be contained by effective control programme by vaccination. Affected animals must be given essential treatment and care. Rapid and accurate diagnosis is of paramount importance with the various diagnostic tools to prevent further spread of the disease.

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