Pure Appl. Biol., 8(2): 1214-1222, June, 2019
http://dx.doi.org/10.19045/bspab.2019.80063

Review Article

Peste des petits ruminants: A review

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Citation
Abdul Kabir, Dildar Hussain Kalhoro, Shahid Hussain Abro, Muhammad Saleem Kalhoro, Haider Ali Yousafzai, Sheeba Shams, Ihsan Ullah Khan, Ghulam Murtaza Lochi, Muhammad Qasim Mazari, Muhammad Waseem Baloch, Arab Khan Lund and Mujeeb ur Rehman Memon. Peste des petits ruminants: A review. Pure and Applied Biology. Vol. 8, Issue 2, pp1214-1222. http://dx.doi.org/10.19045/bspab.2019.80063

Received: 17/01/2019 Revised: 21/03/2019 Accepted: 10/04/2019 Online First: 18/04/2019

Abstract

Peste-des-petits-ruminants (PPR) is a notifiable, contagious and economically important transboundary viral disease of small ruminant causing high morbidity and mortality. It belongs to negative-sense, single-stranded RNA paramyxovirus of genus Morbillivirus. PPR occurs in populations of immunologically naive sheep and goats, illness and death can be high as >90%. The virus is commonly found in developing countries, especially in tropical countries where the disease is widespread. After the eradication of the rinderpest virus, which is closely relate to PPRV of small ruminants contaminated with SRMV are diagnosed having pyrexia, oculo-nasal discharges, necrotizing and erosive stomatitis, gastroenteritis, diarrhea and broncho pneumonia, whereas, gross pathology, histological findings along with laboratory confirmation of specific virus antigen, antibodies, genome in the clinical samples through a variety of serological and molecular diagnostic tests can be useful. In addition there should be social economic surveys, disease hot spot recognition and identification of role of additional species in disease transmission. SRMV control may be achieved by confinement of contaminated animals, subtraction of theoretically tainted fomites with control at import and export of sheep with goats from contaminated areas. PPR can be controlled through mass vaccination program. In the future, the preparation of a marker vaccine with a robust companion test may assist in serosurveillance for the detection of infection in vaccinated animals to control the disease.

Keywords: Control; Epidemiology; Goat; Peste des petits ruminants; Sheep; Vaccination

Introduction

Peste des petits ruminants (PPRVirus) is quick spreading viral disease that affects sheep and goat. Goats may be more severely affected than sheep by PPRV infection **[1]**. During the acute stage of disease, animals show pyrexia (40 -42^{0}C), despair, ulcers, vesicles erosion lying on tongue, smack its lips lacrimation, serous nasal discharge, abnormal breathing with coughing foul-smelling diarrhea and finally death **[2]**. In severe cases mortality and morbidity may

ranges from 90 to 100% [3]. Clinical and pathological aspect of PPRV are closely resembles with rinderpest [4]. Moreover, PPR virus is a paramyxovirus of the Morbillivirus genus [5]. Morbillivirus genus include measles virus of humans, canine distemper of dogs, rinderpest virus of cattle and buffalo and the recently identified phocine distemper virus (PDV1) of seals [6].

History

In the early 1940's, it was a fatal disease of small ruminants with high mortality, first defined

as Peste-des-petits-ruminants (PPR) in the Ivory Coast (Cote d'Ivoire) in West Africa [7]. Similar syndrome was described in Dahomy (Benin) by Cathou (1944) under the name of "Peste des especes ovine etcaprine". The disease has been reported in Senegal and Ghana [8]. Furthermore, PPR Virus was initially isolated in Senegal in 1962 [9]. During 1950 a disease that affected mostly goats, was studied in Nigeria by Johnson and Ritchie in 1968. This disease was specified with variety of names including Stomatitis pneumoenteritis complex (SPC), Pseudorinderpest and Kata. Johnson and Ritchie in 1968 further proved that PPRVirus and SPC are the same disease [6]. Rowland and co-workers proved that the tentative infection of West African dwarf in goats by PPR and Kata were clinically and pathologically same [10]. PPR was controlled to West Africa, until a disease of small ruminants in the Sudan, which was originally identified as Rinderpest (RP) in 1972 [11]. After 10 year, disease was confirmed to be PPR [12]. It is possible that cases of severe rinderpest diagnosed in small ruminants in the past may be PPR. Whereas, PPRVirus was first time reported in sheep and goat including gazelles, ibex and gemsbok at Al Ain in the Arabian Gulf [13].

Causative agent of PPR

Peste Des Petits Ruminants (PPRV) belongs to the *Morbillivirus* genus of *Paramyxoviridae* family [14]. Morbilliviruses are extremely contagious pathogens that cause destructive diseases of animal. The members of the genus *morbillivirus* are Rinderpest virus of cattle and buffaloes, Measles virus, Canine distemper virus, Phocid morbilliviruses and Porpoise distemper virus [15]. These viruses share the similar genome association though ribonucleic acid (RNA) [16]. The morbilliviruses are a closely associated group of important human and animal pathogenic viruses [5]. Morbillivirus is a non-segmented, distinct strand, pleomorphic, negative sense RNA virus measuring 200nm in diameter [17]. PPR is one of the longest sequenced Morbillivirus genomes comprising of 16 kilo base pairs [18].

Geographical distribution

PPR has progressively expanded to cover large regions in Africa, Southern Africa, Arabian Peninsula, Middle East and Asia. PPRV was first reported in 1942 (Ivory Cost), however it occurs mainly in the African and Asian countries.

There are four lineages of PPR virus variants; lineage I and II viruses in West Africa, lineage III in East Africa, Arabian and Southern India and lineage IV in the Middle East and Asia subcontinent [19].

Extensive geographic cover of PPR has been confirmed by the setting up of surveillance and control programs, raising awareness of local populations, the provision of sensitive and specific immunosorbent and molecular diagnostic techniques and the notification of disease emergence and epizootic outbreaks since 2004 by health authorities of the countries where it is considered as compulsorily notifiable disease. Notifications can be complemented by serological detection of antibodies and virological survey in enzootic and epizootic zones to identify the viral lineage involved, monitor the movements of the virus, understand spread

factors and the impact of vaccination campaigns [20].

Host range

Peste des petitis ruminant (PPRV) is a zoonotic disease affecting small ruminants, but goats are affected more severely than sheep [21]. West African goats have been found to be more susceptible than European, and within the former group, the kids are more susceptible to the disease [22]. Cattle and pigs might be carrier to the virus without any clinical signs [23, 24]. It has been reported that the camels can be seroconvert to the PPRV [25]. Recent observations in Sudan suggested that the camels could be affected by PPR as they can show clinical expression of the disease and positive results were detected by serological tests that include RT-PCR and cell culture [26, 27]. In one study, antibodies against PPR were detected in 3 % of the 628 tested camels in Ethiopia [28].

Transmission

Natural transmission

Like other Paramyxovirus, PPRV cannot survive for long time outside the host [29]. Virus is excreted in nasal, oral and ocular discharges at the onset of pyrexia and in faeces at the onset of diarrhea. Animal from the incubation stage can infect other animals [30]. Spread of the disease can occur through interaction among susceptible animals. Infected animal and dirty equipment, oculonasal and oral discharge, loose faeces and grasping large quantity of the virus can cause transmission. The disease is transmitted between animals living in close contact by inhalation of aerosols which contain fine infectious droplets are produced by sneezing and coughing. Moreover, the disease can be spread by indirect contact through water, feed troughs and bedding contaminated with the virus [31].

Experimental transmission

Virus has been transmitted through different routes: nasal, oral, subcutaneous, intraocular, intratracheal and intravenoust [32]. The infective lymphoid tissue suspension is also used to infect goats and sheep after the propagation of the virus through three serial passages in goats. The tissue suspension failed to infect inoculated sheep [33]. While another transmission study indicated that the syndrome SPC (Stomatitis pneumo-enteritis complex) could pass from goat to goat by contact or by inoculation of susceptible goats with the infectious agent. They also reported that the disease could not be transmitted to cattle and rabbits inoculated via subcutaneous and cerebral routes [34]. Moreover, PPR is contagious due to the nature of the spread of the disease from kids to adult goats. Virus was inoculated from sheep to sheep and goats by using crude tissue material and tissue culture isolated from animals that died from Kata and SPC. Disease was transmitted through two passages in goats followed by one passage in cell culture then another one in goats [35, 36]. Pigs can be infected with PPRV by inoculation or interaction by infected goat was impotent to spread the virus either to goats or pigs [37].

Clinical signs

Small ruminants are the main host of PPRV. PPR is measured in high fever, watery oculo-nasal discharge, gradually becoming muco purulent and stick parts of the eyelids, incubation period is 3 - 4 days [38]. Affected animals are characterized by tachypnoea and rapid increase during body temperature to 39.5-41°C. Other symptoms include dilation of the nostrils, lips and tongue coughing [39]. Watery discharge is observed from nose, eyes and mouth, Fever is lay down surrounded by two days, mucous membranes and mouth, eye become marked, palate, dental pad, lips, inner aspect 10 cheeks with superior outside of tongue (Figure 1). The disease has per acute, acute and sub-acute syndromes [40]. In the majority of cases, PPR is an acute disease. The clinical signs in sheep are the same as in goats but generally less severe [41, 42]. PPR

is characterized by pyrexia, catarrhal irritation of the ocular and nasal mucous membranes, erosive stomatitis, conjunctivitis, gastroenteritis and pneumonia [12]. Clinical signs and mortality significantly depend on the virulence of virus [43].

Figure 1. Represented goats; Nasal discharge, swollen, eroded lips, later mouth lesions, Diarrhoea

Diagnosis

Disease is tentatively diagnosed by clinical observations, post-mortem lesions and laboratory confirmation by several serological and molecular techniques including PPRV detection by specific antibody in serum, detection of viral antigens [2]. Young animals are severely affected. In per acute form animals show severe depression, loss of appetite, nasal and eyes discharge, diarrhea resulting in dehydration, pregnant animals may abort [44]. Post mortem examination revealed visible black red zones congestion of lungs, large and small intestines [40]. Severe lesions include congestion and "zebra stripes" large intestine. Erosive lesions can also arise in the vulva and vaginal mucous membranes. Bronchopneumonia through consolidation and atelectasis transpires are commonly observed [45].

The Laboratory confirmation of the disease is usually done by detecting virus or viral antigen, detection of genetic material from the virus and detecting antibodies against virus [46]. Current laboratory tests includes gene detection, Agar Gel Immuno-diffusion (AGID) Immunocapture enzyme-linked immunosorbent assay (ICE ELISA for antigen detection lateral flow device (LFD;field test) conventional RT-PCR, Real-time RT-PCR, LAMP PCR (field test) [47]. Serological tests are virus neutralization and (c-ELISA) for antibody detection [17].

Risk factors

Age

Young animals of two years of age are affected. Older animals are much probable toward sero-positive of PPR than young one [48].

Specie

The goats are more susceptible than sheep as confirmed by various studies carried out in many parts of Pakistan [49]. PPR is also reported in the Sindh Ibex [50].

Sex

Ewes are more affected and positive for PPR than ram. This might be due to poor management system practiced in Pakistan [51]. Male animals are slaughtered in early stage of life while the female goat and sheep are kept live for breeding and milk [52].

Season

The climatic conditions highly affect the spread of PPR as in humid season PPR infection decreases due to restricted animal movement and availability of fodder with high nutritional status that causes enhance immune power against PPR disease [50]. Occurrence of PPR outbreaks may increase

in months of December to February due to dry environment and dusty season along with reduced fodder presence in grazing area which supports the disease propagation [53].

Vaccines

Live attenuated homologous non thermo-stable PPR vaccines have been accessible in the market for certain time. They contain a variety of PPRV isolates, for example Nigeria 75/1 and Shun-gri/96, which are attenuated over serial passages in Vero cells [54]. The vaccines have been demonstrated to be highly effective and are suggested to provide lifelong immunity in both sheep and goats. These popular vaccines are used in many PPRV endemic countries [55]. Though until now no vaccine is available that can serologically distinguish vaccinated animals from diseased ones, a so called Differentiating Infected from Vaccinated Animals (DIVA) vaccine [56]. The available PPR vaccines do not support the DIVA principle. Possible DIVA vaccines based on recombinant viruses are good vaccine candidates. One vaccine based on recombinant adenoviruses has shown to be promising but has not undergone extensive or long term testing [57]. Thermo stable vaccines and other strategies for improving the stability of PPR vaccines are under study [58]. Some research has been done on synthetic short interfering RNAs (siRNAs) which might kill the virus while preserving the serological status of treated animals [59].

Treatment

There is no treatment for PPR but mortality rates may be decreased by use of broad spectrum antibiotics and antiparasiticides. Oxytetracycline and Chlortetracycline are suggested to inhibit secondary pulmonary infections [62]. Supportive care including fluid therapy can also decrease deaths due to dehydration and subsequent electrolyte imbalance [63]. Clinical cases of acute PPR can be adequately and successfully treated even in advanced cases particularly if treatment is started early [64].

Clinical cases of PPR disease in goats are preferably treated symptomatically by the use of broad spectrum antibiotics, intestinal sedatives and fluid therapy to treat pneumonia, diarrhoea and restore body fluid ionic balance [63]. Goats are treated with norfloxacin together with oral and i/v administration of electrolytes and a liver detoxifying agent [65]. It was also observed that management of hyperimmune serum to animals incubating the disease or in the early stages of the disease before the start of diarrhea resulted in safety and recovery [66].

Control and prevention

Different control and preventive strategies can be used. At very first stage, separate the diseased animals from healthy animals to minimize the casual of transmission of PPR virus from diseased animals to healthy animals. Secondly slaughtering of apparent diseases animals and seropositive animals [67]. Furthermore, vaccination of animals is a good option to minimize the risk of incidence in healthy animal population [68]. Worldwide different immunization strategies against PPR has been used i.e. earlier immunization of small ruminants was done with lymph node and spleen materials containing contagious virus inactivated with 1.5-5 % chloroform, attenuated tissue culture rinderpest vaccine (TCRV) but now PPR homologous vaccine is available which is prepared by a new freeze-drying process and addition of stabilizing agents [69].

Conclusion

Peste des petits ruminants is a severe, highly infectious and fatal viral disease of small ruminants all around the world including Pakistan and generally circulated in sheep and goat rearing areas**.** PPR transmission occurs through contact with infected animals. Goats are more susceptible than sheep. Clinical signs of PPR should be confirmed by laboratory testing. Vaccination of animals

against PPR has a positive impact to control virus outbreak in the country. Thus, Mass vaccination program should be carried out in affected areas to reduce the PPR infection among sheep and goats.

Authors' contributions

Conceived and designed the experiments: A Kabir, DH Kalhoro & SH Abro, Performed the experiments: A Kabir & DH Kalhoro, Analyzed the data: MS Kalhoro, HA Yousafzai, MR Memon & S Shams, Contributed materials/ analysis/ tools: GM Lochi, MQ Mazari, AK Lund & MW Baloch, Wrote the paper: A Kabir & DH Kalhoro.

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