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Rise in spread of peste des petits ruminants (PPR) lineage IV associated role of wild animals in Pakistan

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Peste des Petits Ruminants (PPR) is a highly transmissible and contagious disease in small wild and domesticated ruminants, particularly goats and sheep. Commonly called 'Goat Plague'; it's caused by Peste des Petits Ruminants Virus (PPRV). The aim of this review is to draw the attention by updating the available literature on PPRV, and stresses upon its eradication from Pakistan. Mixing of herds, movement of animals across borders, confining animals during rainy and winter seasons are risk factors for transmission of PPR. Wild small ruminants play a major role in the epidemiology of PPR. This review found out that wild animals are not responsible in the spread of PPR; there is a scope of this disease in wild ungulates. Pathological lesions and clinical signs of PPR are similar to Rinder Pest disease. Diagnosis of PPRV is possible clinical samples by several molecular and serological techniques; laboratory confirmation; post-mortem lesions; epidemiology; distinctive symptoms and general clinical observations. In Pakistan, vaccines contain Nig 75/1 strain which is being used for immunization against all PPR viruses. Current review has concluded that PPR is a significant disease of goats, sheep and small wild ruminants. However, there is a dire need of investigations to explore the role of wildlife in PPR transmission. Finally, comprehensive national programs for controlling PPR are strongly suggested.

Keywords: Peste des Petits Ruminants (PPR), PPRV, Goat plague, Rinder Pest. Wild Ruminants

INTRODUCTION

Peste des Petits Ruminants Virus (PPRV) is a highly transmissible and contagious virus responsible for a devastating disease; Peste des Petits ruminants (PPR) in small wild and domesticated ruminants, particularly goats and sheep (Li et al. 2016). This disease is also known as 'Goat Plague'. PPRV falls in the genus *Morbillivirus* (family Paramyxoviridae) (Gibbs et al.1979); and closely related to Rinderpest virus, which has been eradicated globally. Due to higher PPR-mediated mortality and morbidity cases, it is known as economically important and noticeable trans-boundary viral disease of goats and sheep

(Karim et al. 2016; Li, et al. 2018). There are four lineages of PPR viz. Lineage I, II, III and IV, and all are genetically distinct from each other (Hodgson et al. 2018). All four types are prevalent throughout African continent, but the Lineage IV prevails in Asia including Pakistan (Anees et al. 2013).

The pioneer incidence of PPR was observed in Ivory Coast in West Africa during World War II; while, in Pakistan it was recognized in early 1991 by Abubakar et al. (2015). Recently, in Punjab province of Pakistan, the outbreak of PPRV has also been reported in large ruminants like buffaloes and cattle (Abubakar et al., 2017; Li et al. 2018). It has become a hazard to livelihoods and a serious threat to food security in poor communities due to its major constraints to the production of small ruminants. Moreover, the transmission of PPRV into wild animals from the domestic stock may have significant impacts on the wildlife population (Fournié et al. 2018). Though, PPR mainly occurs in sheep and goats (Libeau et al. 2014) but certain evidences confirmed its presence in other ruminants like buffaloes, cattle and camels (Kwiatek et al. 2011) and particularly Caprine wild ungulate (Fentahun, 2012). The large ruminants are susceptible to PPR but luckily, they are unable to transmit the disease as they fail to inhibit clinical signs (Fentahun, 2012).

This disease was thought confined only in West African countries, but recently it broke out to spread throughout African continent along with Asian countries like India and China. The PPR endemic to distant areas depends on multiple factors-such as mixing of the infected animals in the herds, close contact between animals (Munir, 2014), variations in goat and sheep husbandry practices and role of wild animals towards domestic stock. The distribution and endemic of PPRV in Pakistan is hazy; but, geographically this virus is endemic throughout the country especially among small ruminant farming systems. Though, the role of wild animals in the transmission of PPR still requires a great deal of research as hardly any information is available. Thus, the role of wildlife and the possible existence of wildlife reservoirs in the transmission of PPR remain to be explicated even though many susceptible species of wildlife ruminants have been reported (Balamurugan et al. 2016).

Current review highlighted the history, epidemiology, genetics, and economic impact, control, prevention, significance, and eradication and future perspectives of PPR. Moreover, this manuscript will highlight the enhanced outbreak of PPR in Pakistan along with the role of wild animals in its transmission. We draw the attention by summarizing and updating the available literature on PPRV, shall set the direction to eradicate PPR from Pakistan.

HISTORY

Previously, PPR disease was famous with various names such as pneumoenteritis complex, stomatitis-pneumoenteritis syndrome and pseudo-rinderpest (as its clinical symptoms resembled that of rinderpest); and initially observed during World War II in Ivory Coast, West Africa. The first scientific and authentic description of disease was reported by Lalanne and Gargadennec in 1942 (Gargadennec and Lalanne, 1942). Initially PPR thought to be affecting only small ruminants as the cattle living in-contact with them apparently remained healthy. The disease then rapidly spread to other countries like Ghana, Senegal and Nigeria and other neighboring countries in early 1980s **(**Kwiatek et al. 2011; Balamurugan et al., 2014**).** Later on, the disease was transmitted to other parts of the world especially in Indian subcontinent, the Middle East and sub-Saharan Africa. In Pakistan, PPR was first reported in goats in early 1991, in Punjab province, where it was first said as 'disease like rinderpest'. Moreover, no laboratory confirmation took place and certain reports were based on post mortem findings and clinical signs (Abubakar, Irfan and Manzoor, 2015).

In all Asian regions, the first case of PPR was observed in 1987 in India, when PPR caused the first outbreak in sheep and resulted in 25% mortalities (Balamurugan et al. 2014). In China, the initial reports for the presence of PPR were first indicated in Tibet, China in 2005.(Liu et al. 2017). Then after that, few outbreaks were reported from 2008-2010. PPR cases were again reported in 2013 in Xinjiang Uygur Autonomous Region (Bao et al. 2014) and also in some other parts of China (Su et al. 2015; Liu, et al. 2017).

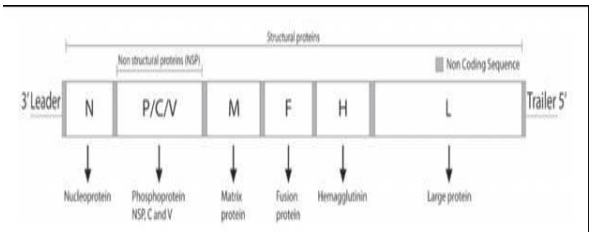
CLINICAL SYMPTOMS

Pathological lesions and clinical signs are proved to be useful for authentic diagnosis of PPR. PPR has mimicking clinical manifestation with rinderpest except for the fact that former has affinity to the lung tissues (Balamurugan et al. 2014). Clinical symptoms of the disease can even lead to death with few cases of survival has also been reported (Gargadennec and Lalanne, 1942). Clinical signs of PPR include bronchopneumonia,

diarrhea, gastro enteritis, erosive and necrotizing stomatitis, oculonasal discharges and high fever (40.6–44.2 °C) (Omani et al. 2019).

VIRAL GENOME

There are only four sereotype of PPR (Anees et al. 2013). PPR genome comprised of 15948 nucleotides (nt) and the viral body is enveloped with a negative-sense single-stranded RNA (Rajko-Nenow et al. 2017).The Indian-Sino literature reported that some novel strains emerged recently with 15 942 and 15 957 nucleotides respectively (Su et al. 2015; Bao et al. 2014). There are 6 genes in the viral genome (H- hemagglutinin protein, L- RNA-dependent RNA polymerase, F- fusion protein, M- matrix protein, P- phosphoprotein and N- nucleocapsid protein) which encodes for 6 structural proteins (Su et al. 2015; Kumar et al. 2014; Anees et al. 2013).



Genomic structure of peste des petits ruminants virus (Source: Barrett, et al. 1991)

Based on N genes, PPRV is divided into four lineages i.e., I, II, III and IV (Maganga et al. 2013). The lineage I-III is common in African populations and while in Asian goat populations, lineage IV is prevalent (Liu, et al. 2017). In Pakistan, it has been proved that lineage IV of PPRV has been circulating with genetic diversity (Abubakar, et al. 2015).

TRANSMISSION

The PPR frequently transmitted upon direct contact of goat and sheep populations with wild animals then they contract the virus (Abubakar, et al. 2015; Parida, et al. 2015). Similarly, susceptible goats and sheep may transmit the virus from infected wild animals (Fournié, et al. 2018). Additionally, introducing infected animals into herds or naïve flocks further aggravate the transmission of PPR. Close contact can easily contract the disease between animals by aerosols. Infected virus may also transmit the virus through loose feces, discharge from nose, mouth and eyes. On the contrary, indirect spread of virus can take place through bedding act, feed troughs and contaminated water (Mumba, 2015).

Shahriari, and colleagues (2019) carried out a research study to investigate the PPR virus due to increasing outbreaks in Iran.

EPIDEMIOLOGY

The epidemiology of PPR is still dealt with many gaps with respect to current situation. Various reports have specified different scenarios of animal species thought to be involved in outbreaks of PPR; like, sheep alone, goats alone or goats and sheep together (Felix, 2013). Reports also indicated that large ruminants are relatively resistant to PPR infection than small ruminants; however, other reports have revealed that PPRV is involved in rinderpest-like disease in buffaloes in India and respiratory diseases in camels of Africa (Felix, 2013). PPR is now endemic in most of South Asia, Middle East, China and particularly Africa. In 1989, PPR was first time reported in Indian subcontinent; though, was misdiagnosed as pasteurellosis and rinderpest for a long time period (Kumar, et al. 2014).

RISK FACTORS

PPR is endemic in all neighboring countries of Pakistan like Afghanistan, Iran, India, China. Particularly with Afghanistan, Pakistan shares a large border that is why there is a lot of cross border movement. Most importantly, Afghani nomads have been crossing the border for centuries; this shows that the persistence and transmission of PPR among both of these countries is evident (Zahur, et al. 2008). Thus, movement of animals across borders is thought to be a risk factor for the transmission of PPR disease. Another risk factor for PPR is its seasonal occurrence. PPR incidence lessened during rainy season due to availability of ample amount of food and which ultimately enhances immunity against the disease. Other risk factors for spread include inadequate veterinary services, visit of animals to the animal market and large flock size (Abubakar et al. 2015).

Various environment stresses directly impacts the morbidity of disease like confining animals during rainy and winter seasons (Li et al. 2016). Though, environmental effects on the incidence of PPR lie on the basis of the socio-economic status of the farm owner and nature of animal husbandry (Munir, 2015). Moreover, seasonal variations in environmental conditions impact the animal movement, mixing patterns between and within husbandry systems, grazing of animals and the demographic process as a

whole (Abubakar et al. 2015), thus cumulatively aggravate the transmission of PPR. Similarly, trade patterns of animals change as per the religious, seasonal or other festivals; being celebrated by the countries for other livestock species (Baudon et al. 2017). Transmission rates of PPR vary with different seasons and can be identified for finding out the most suitable time for vaccination (Fournié et al. 2018).

ROLE OF DOMESTIC AND WILD LIFE IN SPREADING PPRV

The main natural hosts of PPRV are only wild, small and domestic ruminants. Still, there are many other species which are being infected by the virus like camels, Asiatic lion, buffalo, and cattle (Balamurugan et al. 2012). There is limited information regarding the severity, occurrence and susceptibility of the disease in species of wild ungulates. Though, it has been indicated by the recent reports that in the epidemiology of PPR, wild small ruminants play a major role (Munir et al. 2013). The severity of PPR may differ in small ruminants depending on the season, sex, breed and age (Rahman et al. 2016). It has been observed that in general under same environmental conditions no any severe clinical signs appear in sheep like that in the goats. This depicts that sheep have stronger antibodies against PPRV than goats (Fakri, et al. 2017). It also has been noted that even without affecting sheep herds, PPR infection can circulate between the goats only; however, mixed raising of goats and sheep lead to high risk of sero-positivity in sheep (Al-Majali, et al. 2008). Zahur and his fellows in (2008) threw light on the factors that play a significant role in the transmission of PPRV to the new areas. They described that communal grazing and the introduction of new animals into the stock have thought to be responsible for outbreak of PPR. Moreover, it was noticed that livestock markets that are held around the Muslim festival also play a key role in the subsequent spread of PPR in an area (Zahur et al. 2008).

Another study carried out in Punjab province of Pakistan found out that sex, species and geographical location are risk factors for sero-positivity to PPRV. Feeding pattern and age were formally considered as significant risk factors to PPR;

The occurrence of PPR in China is attributed to the wild small ruminants present in the vicinity of Xinjiang borders reported by Liu, and colleagues (2017). These wild small ruminants are responsible for cross-border dissemination of PPRV as there is an abundance of small wild ruminant population near the boundaries of Xinjiang province. They are playing a key role in wide spread of infection in both the domestic and wild small ruminants in China (Li, et al. 2018). Additionally, sheep transportation increases due to certain events in the Xinjiang region, hence the virus moves from one area to another. Uighurs/Uyghurs in Xinjiang region are majority Muslims so they have greater need of products of sheep and goats (Liu, et al. 2017; Wu et al. 2015). Last but not the least, the New Silk Road strategy led to increase in sheep production which further drove the development of sheep raised in Xinjiang and thus all this facilitated the cross border transfer of sheep and goats. Thus, Liu, and colleagues (2017) deduced a comprehensive analysis that wild small ruminants from neighboring countries introduced PPR in China which re-emerged in 2013. The outbreak of PPR of 2013 in China was probably disseminated from Pakistan and Tajikistan (Liu et al. 2017; Wu et al. 2015; Bao et al. 2014).

On the contrary, wild animals are not responsible in the spread of PPR, reported by different studies because mainly this is a disease of goats and sheep (Fentahun and Woldie, 2012). Though it has been reported many times that PPR has occurred in wild ungulates as well; still, no evidence of involvement of wildlife in transmission of PPR. Also, camels, buffaloes and cattle are susceptible to PPR but they are unable to transmit the disease to other animals as they do not inhibit clinical signs (Fentahun and Woldie, 2012). Similarly, PPR was diagnosed in eastern Mongolia, in many wildlife populations like goitred gazelle (Gazella subguttorosa), ibex (Capra sibirica) and saiga antelope (Saiga tatarica mongolica) (Baron, et al. 2017). But, it cannot be stated that the wildlife population plays a role in the transmission of disease. This area needs additional focus to figure out the role of wildlife associated with the spread of PPR (Baron et al. 2017).

A recent study argues the same view point regarding the susceptibility of wildlife species to PPR is obvious but their role in the spread of PPR is still uncertain. Only survey based on serological evidence gave the information regarding the incidence of disease in free-ranging wildlife. There is very scarce data on genetic nature of circulating PPR strains. Current outbreaks of wild ungulates in many countries suggested that there is a scope of this disease in wild ungulates (Munir 2014; Baazizi et al. 2017). Additionally, through the

Phylogenetic analysis of fusion genes and nucleoproteins, it was found that the isolated PPRVs belonged to lineage IV-causing outbreaks in wild ungulates (Munir, 2014). However, there is a dire need of pouring additional investment in this area of finding the role of wild animals in the epidemiology of PPR; as the recent disease surveillance efforts are insufficient. Thus, it is affirmed that PPR is a potential source of virus for domestic species as it circulates in wild animals as well. There is also a need to address the susceptible species of antelopes and their role in PPR transmission; irrespective of the fact that wild small ruminants are possible reservoirs of PPRV (Munir, 2014).

DIAGNOSIS

This disease can be diagnosed by several molecular and serological techniques; laboratory confirmation; post-mortem lesions; epidemiology; distinctive symptoms and general clinical observations. Recently, more rapid and specific diagnosis of PPR is possible through the advent of molecular biological techniques and cell culture (Balamurugan, 2014). Generally, PPR is diagnosed through advanced methods like ELISA using PPRV-specific Monoclonal Antibodies (MAbs), Virus Neutralization Test (VNT), immuno histochemical detection, hemagglutination utilizing chicken or piglet's red blood cells, nucleic acid hybridization, Counter-Immuno-Electrophoresis (CIE), Agar Gel Immuno-Diffusion test (AGID)/AGPT and simply by virus isolation (Balamurugan, Hemadri, Gajendragad, Singh and Rahman, 2014).

ERADICATION OF PPR

Significant efforts are now being made to eradicate and control PPR; following the successful eradication of Rinderpest (Baron, 2017). Owing to the unique disease mechanisms, epidemiological patterns and identical features of the virus; it has been considered as an appropriate target for control and eradication of PPR. Now, the virus can be detected in all possible clinical samples by using sensitive molecular assays and for the purpose of immunization, by efficient and potential vaccines (Balamurugan et al. 2014). Additionally, for improving the field applicability and thermostability of the PPR vaccines, significant efforts have been made in recent years. For appropriate control and eradication of the disease across the globe, the vaccines should have been on easy access and cost effective. Several wild life species need to be monitored for prevalence of PPRV (Munir, 2013). In Pakistan PPR is thought an endemic disease of goats and sheep (Aziz-ul-Rahman, et al. 2016). Despite of several clinical, preventive measures and strict vaccination programs, the outbreaks of PPR are still frequent in many parts of the country (Abubakar and Manzoor 2015; Abubakar et al. 2015). PPRV has only one serotype; despite of several genotypes. In Pakistan, vaccines contain Nig 75/1 strain is used for immunizing against all PPR viruses (Zahur, et al. 2014). Thus, control and eradication of PPR in Pakistan is somehow simplified recently.

CONCLUSION

Current review has concluded that PPR is a significant disease of goats, sheep and small wild ruminants. It is endemic in many countries and causes significant losses to the animal population. In-depth analysis and review of literature has been confirmed that the wildlife population also plays a key role in the spread of PPR disease. Therefore, the spread of PPR in wild small ruminants should closely be monitored and should not be ignored. However, there is a dire need of investigations to explore the role of wildlife in PPR transmission. Moreover, the pathogenesis of PPRV and the mechanism of infection need further investigation.

For the global eradication of disease, similarly like RP, a great deal of extensive research is required for PPR. Finally, comprehensive national programs for controlling PPR are strongly suggested.

CONFLICT OF INTEREST

The authors declared that present study was performed in absence of any conflict of interest.

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AUTHOR CONTRIBUTIONS

MMJ and KR designed the idea and also wrote the manuscript. MKM, HN, UW, WA, FH, MR helped in writing of manuscript and data analysis. ZN, RH, MKS, BA, IAK and MR designed experiments and reviewed the manuscript. All authors read and approved the final version.

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