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REVIEW OF PESTE DES PETITS RUMINANTS (PPR) IN ETHIOPIAN CONTEXT.

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1. ABSTRACT

Peste des petits ruminants (PPR) is a highly contagious disease of sheep and goats caused by a Morbillivirus closely related to rinderpest virus and is known with high mortality rate of 90% and morbicity of 100%. It is one of the most damaging livestock diseases in Africa, the Middle East and Asia which has strong negative impact on food security and the livelihoods of poor farmers. PPR are diagnosed the animal with clinical sign like pyrexia, oculo-nasal discharges, necrotizing and erosive stomatitis, gastroenteritis, diarrhea and broncho pneumonia, in addition gross pathology and 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. Vaccination with live attenuated whole virus vaccine is the only strategy adopted for prevention and control of this disease. For implementing the proper control of PPR, there is need of strong support of diagnostic methods and proper, timely vaccination of the susceptible population. Hence, the availability of cell culture vaccine and various diagnostic techniques/kits for the diagnostic of PPR favours strong recommendation put forward for the control program of the disease. *The vaccination campaigns for diseases such as PPR is to reach post-vaccination immunity coverage of 70-80% .*

Key words: *Review, peste des petites ruminants, Etiology, Diagnosis. Control and prevention.*

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2. INTRODUCTION

Peste des petits ruminants (PPR) or goat plague is an acute or sub-acute, highly contagious viral disease of small ruminants with high morbidity as 100% and mortality reach 90% that threatens the food security and sustainable livelihood of small holders. This disease highly affects incomes from small ruminant husbandry systems and lead to loss in the profitability and productivity. The causative agent the virus is belonging to the genus morbillivirus and family Paramyxoviridae (Regmi *et al*., 2019). Clinically, the disease in small ruminants is characterized by a high fever, catarrhal ocular discharges, mucopurulent nasal discharges and erosive stomatitis in the early stages, followed by severe enteritis and bronchopneumonia. Mortality is higher in young animals compared to adults and goats tend to be more severely affected than sheep (Mdetele *et al*., 2021).

Peste des petits ruminants disease is grouped as a notifiable terrestrial animal disease by the World Organization for Animal Health (OIE). PPR was first reported in 1942 in Côte d'Ivoire, West Africa. It has infected 66 countries of the world, 38 countries in Africa, 27 countries in Asia and one country in Europe (Zhao *et al*., 2021). The disease was found to be more prevalent in Africa than in Asia and more prevalent in adult age group animals. Vaccination of young animals may limit the disease distribution and occurrence (Ahaduzzaman, 2020). zones. Among host and environment related risk factors, species, sex, agro-ecology and location of the study were significantly associated with PPR sero-positivity (Fentie *et al*., 2018).

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PPR disease was found to be more prevalent in different areas of Ethiopia. The reason for that may be due to the trans boundary movement of infected animals with inadequate quarantine, the presence of hot and humid climatic conditions that favor disease epidemiology, lack of vaccination or inappropriate vaccine administration and inadequate monitoring program facilitate disease spread, lack of awareness about *PPR* among backyard farmers and limited funding for disease eradication in developing or underdeveloped countries (Yizengaw *et al*., 2021).

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3. LITERATURE REVIEW

3.1. Aetiology of PPR disease

Peste Des Petits Ruminants is caused by a virus that was assumed for a long time to be a variant of rinderpest adapted to small ruminants. But the studies based on virus cross neutralization and electron microscopy showed that it was a morbillivirus that had the physicochemical characteristic of a distinct virus though biologically and antigenically related to RPV. It was also shown to be an immunologically distinct virus with a separate epizootiology in areas where both viruses were enzootic. The development of advanced diagnostic tools like specific nucleic acid probes for hybridization studies and nucleic acid sequencing have proved that PPR virus is quite distinct from rinderpest virus. The Peste Des Petits Ruminants virus is in the genus of Morbillivirus and under Paramyxoviridae family. The Morbillivirus genus also includes other six viruses: measles virus (MV), rinderpest virus (RPV), canine distemper virus (CDV), phocine morbillivirus (PMV), porpoise distemper virus (PDV) and dolphin morbillivirus (DMV) (Gopilo, 2005) affecting different hosts of animals and human being.

Morbilliviruses are structurally linear, non-segmented, single stranded, negative sense RNA viruses with genomes 15–16 kb in length and 200 nm in diameter. The major site of this virus propagation is lymphoid tissue and acute diseases are usually accompanied by profound lymphopenia and immunosuppression, leading to the host susceptible for secondary and opportunistic infections (Taylor, 2016).The PPRV genome is 15,948 nucleotides nearly 16 kb in length, although a variant virus with an additional 6 nucleotides has been detected in the recent Chinese epizootic. The genome contains six transcription units encoding in sequential order, the nucleocapsid (N) protein, the phospho (P) protein, the matrix (M) protein, the fusion (F) protein, the hemagglutinin (H) protein and the large (L) protein which together with the P protein forms the viral RNA-dependent RNA polymerase (Baron *et al*., 2016).

There is only one serotype of PPRV, but phylogenetic analysis based on partial N or F gene sequences groups PPRV strains into lineages I, II, III and IV. Historically, PPRV strains found in Africa belonged to lineage I and lineage II and were mainly prevalent in West Africa. Lineage III is mostly found in Arabia and recently it circulating in East Africa, but has also been isolated in

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southern India in study. Lineage IV is usually found in Asia and so termed the Asian lineage. A recent review of currently available molecular epidemiological data was carried out in Africa and showed that since 2008 lineage IV has also been continually identified in Africa (Zhao et al ,2021)

3.2.Transmission and pathogenesis

The transmission of PPR are by direct contact with (secretions and excretions) discharges from eyes, nose and mouth, and the loose faeces, contain large amount of the virus of infected animal. The fine infective droplets are release into the air, particularly when affected animals cough and sneeze and animals in close contact to inhale the droplets likely to become infected. Additional sources of infection are infectious materials may also contaminate water and feed troughs and bedding (Abubakar *et al*., 2011).

Infected animals (mostly domestic ruminants) are the only source of PPRV. At an early stage of infection virus excretion is massive in the exhaled air. Like RPV, this probably allows noncontact transmission over at least a few meters. The discharges of nasal and ocular, saliva and feces also contain large amounts of viral antigen. In goats PPRV-RNA or antigen is excreted in the feces during at least 2 months after a natural infection though it is not known if this is infectious virus. PPRV is quickly inactivated in the environment so its transmission most often occurs by direct contact between infected and healthy animals. However, indirect transmission through recently (within hours) contaminated material cannot be excluded and should be considered in epidemiological models and risk-based control measures (Baron et al., 2016).

PPR virus preferred the tissue of lymphoid and epithelial tissue for its survival. It is lymphotropic and epitheliotropic and colonizes similar way like other member of morbillIvirus. The main entry of the virus through the host is the respiratory route. It begins the attachment and infection firstly, the virus taken up by Antigen Presenting Cells (APCs) this antigen available in the intraepithelial space and lamina propria of the mucosa in the respiratory tract (naso-pharyngeal/respiratory epithelium). When the virus enters the host and settle lymphoid organ in the respiratory tract and colonizes localize tissue, it begin first replicating in the pharyngeal and mandibular in the lymph nodes as well as tonsil. The first clinical signs appear before 1-2 days infection also viremia may

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develop after 2-3 days, the dissemination of the virus to bone marrow, spleen, and mucosa of the gastrointestinal tract and the respiratory system result in viremia (Nour, 2020).

3.3.Immunosuppression of PPRV

The extensive damage of the lymphoid organs during Morbillivirus infection leads to malfunction of organ and results immunosuppression. The level of infection in peripheral blood leucocyte and lymphoid tissues is directly correlated with immunosuppression. It may also be caused by altered cytokine response, suppression of the inflammatory response, direct infection and subsequent destruction of the leucocytes (leucopenia), inhibition of immunoglobulin synthesis (due to loss of B cells) and cell cycle arrest following direct contact with viral glycoproteins (Schneider-Schaulies *et al*., 2001). Even though only a fraction of peripheral blood cells are infected, immunosuppression can last for weeks and hence increases the extent and severity of the pathological lesions. The vaccine strain may also cause a transient immunosuppression. With experiments carried out with MV and RPV, the importance of specific viral proteins (H, N and P) has been demonstrated to be responsible for inhibition of lymphoproliferation and induction of lymphodepletion (Kumar *et al*., 2014).

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The V and C proteins appear to be multifunctional. The V protein of morbilliviruses acts to block both the induction and action of type 1 interferons (interferon (IFN) α and β). The PPRV V protein not only binds the signaling molecules STAT1 and STAT2 but also blocks their phosphorylation through direct inhibition of the IFN receptor associated kinases Jak1 and Tyk2. Binding of STAT1 by the V protein has the additional effect of blocking the action of type 2 IFN (IFN γ) (Baron *et al*., 2016) then influence the antiviral effect of interferon.

3.4.Diagnosis of PPR disease

The measuring mechanism for diagnosis paste des petits ruminants infected small ruminants routinely diagnosed based on several combinations such as clinical examination, gross pathology, histological findings and laboratory confirmation to implement control measure. The test made is rapid, specific and sensitive methods for diagnosis (Nour, 2020). For paste des petits ruminant's diagnosis, the sample can be taken from swabs of the mucous membrane of the eye, nose and blood. Sample also can be isolate from these organs such as large intestine, lungs and spleen. After

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isolation of these samples the isolate should be transported within the cold chain and refrigeration. To detect PPR can be used for serological and molecular diagnostic tests (Abraham and Berhan, 2001).

The laboratory techniques used for the detection of the virus includes virus isolation, detection of viral antigens, nucleic acid sequencing and detection of specific antibody in serum (Mariner *et al*., 2016). PPR virus is cross-reacts with rinderpest virus. Virus isolation is a definitive test but is labor intensive, incontinent and takes a long time to complete. Currently, antigen capture ELISA and reverse transcription-PCR are the preferred laboratory tests for confirmation of the virus. For antibody detection (such as might be needed for epidemiologic surveillance, confirmation of vaccine efficacy, or confirmation of absence of the disease in a population), competitive ELISA and virus neutralization are the OIE-recommended tests (Saliki, 2015). Some of these testes are described below:

3.4.1. **Gene detection**

The PCR is the most sensitive assay for confirming PPR diagnosis. A rapid and specific TaqMan-based, real-time quantitative reverse transcription PCR (qRT-PCR) has been described for the detection of peste des petits ruminants virus (PPRV). The Primers and probe were designed based on the nucleocapsid protein gene sequence. The real-time qRT-PCR assay allows the rapid, specific and sensitive laboratory detection of PPRV in tissue samples from field cases (Bao *et al*., 2008).

3.4.2. **Virus sequencing**

Nucleotide sequencing of the PCR product offers the opportunity to differentiate specific PPR virus lineages and more effectively trace the source of outbreaks and enhance our understanding of the epidemiology of PPR virus. Such like technology is available in PPR OIE reference laboratories. The use of this assay becomes vital in understanding virus circulation, the distribution of different virus strains and the differing roles these technology might play in the epidemiology of the disease in the field (Mariner *et al*., 2016).

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3.4.3. Competitive ELISA

A mab -based competitive ELISA is available to detect PPR-specific antibodies in blood for monitoring the response of national herds (which may be multispecies) to PPR vaccination. This can be implemented as a standard assay for global use. Implementation should include a system of internal controls and performance monitoring to ensure standardization of results and international confidence in the data generated (Mariner *et al*., 2016).

3.4.4. **Serum neutralization test**

This was one of the earliest serological assays used for determination of protective immunity to PPR virus infection. It is the prescribed test for international trade in the OIE Terrestrial Manual. The principle of this test is for titrating serum antibodies by evaluation of their neutralization effect on virus infectivity on cells. To this end, serum dilutions are incubated with the viral suspension and distributed over a cell culture in tubes or plates. After one to two weeks incubation, neutralizing antibodies will inhibit visible cytopathic effects (CPE) comparatively to the virus alone. The serum titre is the last dilution of the test serum for which no CPE is observed. This reaction requires culture stocks of sensitive cells and vaccine virus (Libeau, 2015).

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PPR disease was clinically suspected for the first time in Ethiopia in 1977 in a goat herd from Afar region eastern part of the country. Clinical and serological evidence of its presence has been reported in1984 and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa. There are reports of the overall sero-prevalence of 9% in goats and 13% in sheep in different parts of Ethiopia. It was also reported that 14.6% of sheep sampled along four roads from Debre Berhan to Addis Ababa were seropositive. In 1999 national sero-surveillance of PPR conducted in Ethiopia, the overall sero-prevalence of 6.4% in both goats and sheep ranging from 0% to 52.5% was estimated. At species level sero-prevalence of sheep 46.68% was approximately the same with that of goats 50.85% which may resulted from equal exposure of sheep and goat because they are herded together and communal grazing (Gari *et al*., 2017).

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4. Control and Prevention of PPR disease

Since the transmission of PPRV from virus excreting infected to naive animals is mainly by close contact, the most important sanitary preventive measure consists of restricting the importation or movement of susceptible animals from infected to disease free areas. Outbreaks can be controlled by stamping out followed by disinfection of premises and compensation of affected farmers. However, such drastic measures are difficult to implement because most of the PPR endemic regions are in developing countries. So, vaccination is the main means available for the effective prevention and control of PPR in those countries (Baron *et al.*, 2016).

For implementing the proper control of PPR, there is need of strong support of diagnostic methods and proper, timely vaccination of the susceptible population. Hence, the availability of attenuated cell culture vaccine and various diagnostic techniques/kits for the diagnostic of PPR favours strong recommendation put forward for the control program (Balamurugan *et al*., 2014).

On the other methods for control and prevention of this virus recommended such as sanitary measure, testing and killing positive animal thus composition, destruction of the carcass in standard as well infected material, isolation and quarantine of infected and slaughter. In addition, infected fomites should be buried and banned, such as banes tools and other infected material that facilitates the risk of contamination and transmission of the virus by using disinfected with a common disinfectant such as sodium hydroxide 2%, alcohol, ether and phenol, detergents that recommended by OIE (Nour, 2020).

Most of the affected countries cannot afford drastic sanitary control measures, like the stamping out policy. Therefore, for this country the only effective means to control PPR is vaccination. This approach takes advantage of the fact that hosts that recover from morbillivirus infection develop lifelong immunity. In the case of rinderpest and PPR and contrary to measles and canine distemper, The National Veterinary Institute (NVI) is the producing laboratory of the PPR vaccines in the country and also exports the vaccine for neighboring countries. Improving the production and stability of the PPR vaccines produced at NVI will contribute to the control of PPR (Silva *et al*., 2014).

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4.1.accination and sero-conversion

Vaccination is recommended to support control and eradication efforts thus limit the economic losses due to disease. The countries like India has practiced focused vaccination in outbreak of different places of some states before the global framework was developed to mitigate PPR burden in sheep and goats. Now the disease has been brought under control and the occurrence and severity of the disease have progressively declined in areas under progressive vaccination. This scenario warrants the studies to be undertaken to know the effect of vaccination on the occurrence and disease severity pattern in different regions of vaccinated vs. non-vaccinated areas including status of vaccination and effectiveness of control programme (Balamurugan et al., 2018).

Vaccination against PPR is an effective means of controlling and eradicating the disease.. However, after the eradication of rinderpest, it was necessary to restrict the use of this vaccine. The first attenuated vaccine developed against PPR involved using the lineage I African isolate, Nigeria 75/1. Subsequently, three more attenuated vaccines were developed in India by using the lineage IV Indian isolates (Sen *et al*., 2010).

5. Conclusion

In Ethiopia currently, the strategy of PPR vaccination is ring vaccination to control the spread of PPR infection that help to provide a vaccinated barrier between infected animals and clean stock. The intervention is expected to control the outbreak of the disease and reduce losses. In a cross-sectional study conducted in selected districts of Ethiopia reported the seroprevalence result indicated that herd immunity level against PPR was very low in the study area prior to vaccination (1.70%), but an overall rise to 61.1% sero-conversion had been observed after the vaccination campaign. Using vaccine made from the same Nigeria 75/1 strain from National Veterinary Institute in Debre-Zeit (Faris *et al*., 2012).Vaccination of all stock, however, led to herd immunity levels rising every year particularly for low vaccination efficiencies. After four years vaccination of all stock at 55% efficiency had produced a herd immunity of 86% whereas the comparable levels for vaccination of calves twice and once only were 62% and 54% respectively (Rossiter and James 1989).

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The targeted Vaccination is recognized that an animal effectively vaccinated will be immune for life from infection by all known PPR viruses helping for eradication. Young animals under the age of 6 month may not respond correctly to vaccination owing to the presence of maternally derived Abs and thus will require revaccination once assuredly susceptible to vaccine take. The vaccination seldom reaches all animals and not all animals given vaccination will respond due to problems in application of vaccines in the field, including maintaining the viability of the vaccine (hence the need for post vaccination Seromonitoring). Thus, a targeted vaccination strategy, linked with other disease control approaches, makes absolute sense.

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