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Review on Epidemiology of Peste Des Petits Ruminants, Global Perspective

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Abstract: Peste des petits ruminants is an acute, extremely contagious and economically important transboundary viral disease of shoat that is categorized by OIE as a notifiable animal disease. It is among the main constraints of shoat production in the Globe where it is endemic. The disease is characterized in affected animals by increased respiratory rates, extension of the head and neck, dilation of nostrils, protrusion of the tongue, abdominal breathing and soft and painful cough. In post mortem finding, necrotic/hemorrhagic enteritis is usually present and linear hemorrhages or zebra stripes might be located in the colon and caecum. The disease is transmitted by either direct or indirect contact and is influenced by age, breed, flock size, exposure status of animals and climatic condition of the areas. Peste des petits ruminants has a significant economic impact on shoat production negatively affecting the livelihood of the poor farmer by causing high morbidity and mortality to their animals. Therefore, control of the disease by applying more effective measures like slaughter of infected herd, correct disinfection of contaminants and adequate disposal of carcasses, movement control, emergency vaccination and quarantine should be undertaken to mitigate the impact of the disease.

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1. Introduction

Peste des petits ruminants (PPR) is an acute, extremely contagious and economically important Transboundary viral disease of shoat that is categorized by OIE as a notifiable animal disease. It is caused by peste des petits ruminants virus (PPRV). The disease is characterized clinically by severe pyrexia, oculonasal discharge, necrotizing and erosive stomatitis, enteritis and pneumonia (Balamurugan *et al*., 2011). The virus causing the disease is a member of the genus Morbillivirus in the family Paramyxoviridae. Peste des petits ruminants virus has a non-segmented, single-strand RNA genome of 15,948 nucleotides that encodes eight proteins including six structural proteins namely the nucleoprotein (N), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin protein (H) and the large polymerase protein (L), and two non-structural proteins (Bailey *et al*., 2005). Four genetic lineages (I-IV) and several viral strains have been identified. The virus's Lineage IV has become particularly widespread in recent years and the virus is similar to rinderpest virus (CFSPCH, 2015).

Peste des petits ruminants virus is more prevalent in West and Central Africa, Arabia, the Middle East, and southern Asia. These areas encompass more of the developing countries that base their subsistence for a source of food or as export goods on small ruminants. Because of the high mortality and morbidity of PPR, this disease constitutes one of the foremost hindrances of subsistence small ruminant farming activity (Banyard *et al*., 2010).

Sheep and goats are vital livestock for supporting food security at global level because of their high reproductive capacity; faster growth rates, greater environmental adaptability and low initial investment and hence have a unique niche in smallholder agriculture (Tibbo, 2006). There is an immense opportunity for increased livestock production in Ethiopia with a growing human population, urbanization, economic development, domestic and export markets. However, the occurrence of different diseases is found to be a major constraining factor of the sector (Biruk, 2014).

The name of the disease (peste des petits ruminants; plague of small ruminants) reflects two things about the disease. Firstly, it was initially described from Francophone West Africa and secondly, it is a disease that kills a large number of sheep and goats (Tewodros and Melese, 2012). Peste des petits ruminants was first described in Ivory Coast, West Africa in 1942 and subsequently spread to other

regions such as sub-Saharan Africa, the Middle East and Asia and these areas faced severe epidemics (Kula, 2016). The disease was first reported in Ethiopia in 1991 near Addis Ababa (Abraham, 2005).

Ethiopia has the largest sheep and goats populations in Africa with an estimated population of 42,914,865 sheep and 52,463,535 goats (CSA, 2020/21). Livestock supply power for farming and transport. They also supply their owners with financial services by providing a substitute for credit and by serving as a form of insurance. The contribution of livestock to the country's agricultural GDP is estimated to reach 47% (IGAD, 2013).

Peste des petits ruminant is the disease that causes high economic losses in the small ruminants industry. The disease impend food security and livelihood of the people. Reducing the effect of PPR in endemic countries is thus common attention and could be considered a global public good (OIE and FAO, 2015). Currently, the strategy of PPR vaccination is ring vaccination to control the spread of the infection to provide a barrier between infected and clean stock. The intervention is expected to contain the outbreak of the disease and reduce losses. The major challenge in control of PPR is lack of adequate information on the dynamics of the disease and inefficiency in early detection. However, several diseases are widespread that hinder production and productivity. Similar to other global areas, there is no enough data on the epidemiology of many diseases including PPR in Ethiopia, Peste des petits ruminants is the most important disease of shoat that needs epidemiological data to mitigate its impact. Therefore, the main objective of this paper is to review the epidemiological status of peste des petits ruminants in small ruminants at global level.

2. LITERATURE REVIEW

2.1 Etiology

Peste des petits ruminants (PPR) is caused by PPR virus which is classified under the order of Mononegavirales, family Paramyxoviridae, subfamily Paramyxovirinae and the genus Morbillivirus. This virus resemblance genetically with Measles virus (MeV), Rinderpest virus (RPV), Canine Distemper Virus (CDV) and several other viruses that infect aquatic mammals. The non-segmented genome that encodes PPRV is nucleocapsid (N), phosphoprotein (P), matrix (M), fusion (F), haemagglutinin (H), large polymerase (L) and two non-structural proteins, C and V (Banyard *et al*., 2010).

2.2 Geographical distribution

At present, more than 70 countries are reporting PPR disease to OIE. Most of these countries are from Africa while others are from Asia and the Middle East. Currently, various countries are considered to be at high risk for PPR disease (OIE and FAO, 2015). 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 includes viruses isolated in the late 1980s in West Africa (Ivory Coast and Guinea), is the only African lineage that did not cross the Red Sea to the Asian countries and Lineage III is a combination of isolates from eastern Africa (Sudan and Ethiopia). Lineage IV isolates includes the Asian isolates from Israel, Iran, Nepal, Bangladesh, Turkey, and India and is confined to Asia (Abraham, 2005) and until 2000; this lineage was limited to Asian countries and the Middle East. However, this lineage had recently been recognized in African countries (Sudan, Morocco, Tunisia and Algeria) and even recently, lineage IV has been identified in Ethiopia.

In Ethiopia, the first clinically suspected case of PPR was reported from goat herds in Afar region in 1977 (Abraham *et al*., 2005) and serological evidence was reported in 1984 and later confirmed through the isolation of the virus with cDNA probe in 1991 (Pegram and Tareke, 1981; Roeder *et al*., 1994). Since then, PPR has been reported from various parts of the country with seroprevalences that varies between 12% in 2001 which was similar to that of the national serological survey conducted in 1999 and 31% from 2009–2010 in pastoral flocks (Abraham *et al*., 2005; Faris *et al*., 2012; Megersa *et al*., 2011 and Waret-Szkuta *et al*., 2008). The disease is recently considered as endemic in the country.

2.3 Host range and reservoirs

Peste des petits ruminants virus primarily infects sheep and goat, although both cattle and pigs are susceptible to infection, however, they do not contribute to the epidemiology of the disease as they are unable to excrete the virus (Abu-Elzein *et al*., 2004; Kinne *et al*., 2010). Clinically, PPR disease is seen in both sheep and goats; however, goats are more susceptible than sheep. Breed of goats that play an important role in susceptibility are goats of Guinean breeds of West African dwarf such as Lagoon, Kirdi and Djallonke that are considerably more susceptible than the major Sahelian breeds (Abu-Elzein *et al*., 2004). Peste des petits ruminants are now recognized as an emerging disease in camelids causing respiratory syndrome in Sudan (Khalafalla *et al*., 2010). Serological evidence of camel exposure to PPRV was confirmed in Tanzania with an overall sero-prevalence of 2.6% (Swai *et al*., 2011). Pest des petits virus circulates in domestic ruminants and acts as a source of virus for wildlife (Hamed *et al*., 2016).

2.4 Transmission

Peste des petits ruminants virus is primarily transmitted through inhalation during close contact

with infected animals. This virus can be shed during the incubation period and has been found in nasal and ocular secretions, saliva, urine and diarrheic feces (Radostits *et al*., 2007). The virus most likely transmitted through fomites like water, feed troughs and bedding for a short time, however, infections did not persist for a long period. There is limited information on the persistence of the virus in the environment, however, the virus is inactivated by ultraviolet light and dryness within 3-4 days or less and usually persists for short periods in the carcasses. The virus may be inactivated by temperature above 70°C and PH 9.6 (CFSCH, 2015).

2.5 Risk factors

Age is an important risk factor, with animals aged 3 to 18 months being more severely affected than adults or unweaned young (Kinne *et al*., 2010). Kids over 4 months and under 1 year of age are the most susceptible to the disease. 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 new stock is introduced or when animals are returned unsold from livestock markets. Recovered animals have lifetime immunity (Radostits *et al*., 2007). Climatic condition is also an important risk factor and outbreaks are most frequent during the rainy season or the cold dry season (Kinne *et al*., 2010).

2.6 Clinical findings

Peste des petits ruminants have an incubation period of 2-10 days, followed by sudden onset of pyrexia (40- 42^0_C) that could last for 3-5 days, severe depression, anorexia and clear nasal and ocular discharges that become muco-purulent because of secondary bacterial complication. Crusts could type on the nose, leading to the interference of the nostrils and respiratory distress, whereas matting along the eyelids might additionally result. One to two days following the onset of the symptom, the oral and ocular mucus membranes became congestion. This then is aiming to multifocal pin point necrosis of the epithelial tissue of the gum, dental pad, palate, lips, inner aspects of the cheeks and also the side of the tongue. These died areas extend and should even coalesce (Sunelle, 2012).

Diarrhea normally seems to be seen 2 to 3 days once the onset of fever though, in early or gentle cases, it is going to be not obvious. The feces are at first soft then watery, malodourous and should contain blood streaks and items of dead gut tissue. Wherever, diarrhea is not presenting sign, the insertion of a cotton swab into the rectum could reveal proof of soft feces which will be stained with blood (FAO Animal Health Manual No.5). Affected animals have obvious signs of pneumonia, characterized by raised respiratory rates, extension of head and neck, dilatation of nostrils, protrusion of the tongue, rales and soft painful coughs. A standard feature within the later stages of the infection is the formation of tiny nodular skin lesions on the skin of the lips and around the muzzle. Abortions could occur in pregnant animals. Death typically follows within 7-10 days from the onset of clinical signs due to severe dehydration, emaciation and hyperthermia (Sunelle, 2012).

2.7 Differential Diagnosis

Frequently, PPR is confused with other diseases that have gross similarity in clinical signs. These diseases include rinderpest, foot and mouth disease (FMD), bluetongue, contagious ecthyma (Orf), pneumonic pasteurellosis, contagious caprine pleuropneumonia (CCPP) and gastro-intestinal helminth infection. The most frequent sources of confusion are the mouth lesion, which could be due to rinderpest, FMD, bluetongue or orf; difficulty in breathing, which could be due to pneumonic pasteurellosis or CCPP or diarrhea which could be due to coccidiosis or gastro-intestinal helminths infection (Dilli *et al*., 2011).

2.8 Diagnostic Methods

2.8.1 Clinical diagnosis

Clinical diagnosis of PPR in the field is based on symptoms such as pyrexia, lachrymation, nasal discharge, oral erosion, pneumonia, diarrhea and death. Historic epidemiologic data of PPR within the region or farms will facilitate field personnel to report a suspicious case. A differential clinical diagnosis should be created with different syndromic diseases. However, it is recommended to sample sick animals for a confirmatory diagnosis (Couacy-Hymann, 2013).

2.8.2 Post mortem diagnosis

Widespread erosive lesions occurred that extends from the oral cavity to the reticulo-rumen junction. Besides, apical pneumonia, enlarged, edematous and congested lymph nodes, pleuritis and hydrothorax might be also present. The spleen is congested and enlarged and necrotic lesions might be present. Necrotic or hemorrhagic enteritis is usually present and linear hemorrhages or zebra stripes may be located in the colon and caecum (Sunelle, 2012).

2.8.3 Laboratory diagnosis

2.8.3.1 Conventional techniques

a) Virus isolation

This technique needs cell culture facilities which are not common in many laboratories in the developing countries. In areas 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) and marmoset-derived cell line (B95a). A new and very sensitive cell line has been developed recently by using 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 suspected material. The identity of the virus can be confirmed by virus neutralization or molecular techniques. Alternatively, specific antigens and antibodies can be detected (Couacy-Hymann, 2013).

b) Antigen detection

- Agar Gel Immuno-Diffusion test- this is simple and can be performed in a basic laboratory but remains relatively insensitive and it cannot distinguish Peste Des Petits Rumunants Virus (PPRV) from Rinder Pest Virus (RPV).
- Counter-Immuno-Electrophoresis- this is sensitive and specific and able to differentiate PPRV from RPV sample.
- Immuno Histochemistry (IHC) on tissue samples: this one allows the localization of specific PPRV antigens in a pathological tissue sample (Couacy-Hymann, 2013).

c) Antibody detection

Viral neutralization test (VNT): is applied to a serum sample; this technique needs also cell culture facilities (Couacy-Hymann, 2013).

2.8.3.2 Molecular techniques

a) Antigen detection

Immuno-capture Enzyme-Linked Immuno-Sorbent Assay (IC-ELISA): this test 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 (Couacy-Hymann, 2013).

b) Antibody detection

Detection of antibodies against PPRV is carried out by using 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 (Couacy-Hymann, 2013).

c) 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 (Vinayagamurthy *et al*., 2012). The conventional RT-PCR has been developed for specific amplification of the NP gene or amplification of the fusion (F) gene and is established in various laboratories. The RT-PCR assay specific for PPRV and the loop-mediated isothermal amplification technique (LAMP-RT-PCR) is also available for the genome detection of PPRV (Couacy-Hymann, 2013).

2.9 Prevention and Control

There is no specific treatment against PPR disease but it is important to give broad-spectrum antibiotics to stop secondary bacterial complications/infections (Bharath *et al*., 2016). Control of PPR in non-infected countries might be achieved by using classical measures such as restriction of importation of shoat from affected areas, quarantine, slaughter and proper disposal of carcasses and contact fomites and decontamination of affected premises in case of introduction. Control of PPR outbreaks can also be relied on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk population. Immunization of shoat with lymph node and spleen materials containing virulent virus inactivated with 1.5-5% chloroform was tried and the animals were immune to subsequent challenge 18 months latter (Braide, 1981).

Until recently, the most practical vaccination against PPR was based on the use of tissue culture adapted rinderpest vaccine. Vaccination of animals with RP attenuated virus has been practiced for a long time. The tissue culture rinderpest vaccine (TCRV) at a dose of 102.5 TCID50 protected goats against PPR for 12 months and the animals did not transmit the infection following challenge with PPR virus (Taylor, 1979a), although the antigen was detected in lachrymal swabs from vaccinated animals after exposure to virulent virus (Gibbs *et al*., 1979). However, it was reported previously that considerable residues of virulence were detected after 32, 42 and even 65 serial passages in embryonic lamb kidney cells (Taylor, 1979a). This vaccine was successfully used to control PPR in some countries in West Africa (Bourdin, 1973) and is widely used in many African countries (Lefèvre and Diallo, 1990). It has been withheld from being used because of its interference with the Pan-African Rinder pest Campaign (PARC), since it is impossible to determine if seropositive small ruminants have been vaccinated or naturally infected with RPV. Sera from animals vaccinated with RP vaccine contain substantial level of RP antibodies with little or no cross neutralising antibodies to PPR but after challenge with PPR, neutralizing antibodies to PPR increase sharply. Rinderpest thermostable vaccine was developed for protection of goats against PPR (Stem, 1993).

Nowadays, efficient live attenuated PPR vaccines are available that can induce lifelong protective immunity in vaccinated animals (OIE and FAO, 2015; Rebecca et al., 2015). The challenges in control activities arise from that 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 (Rebecca *et al*., 2015). Thermo-stabilizing PPR vaccine is well-suited for transport without icebox cold chain. Currently, the Pan African Vaccine Center (PANVAC) which is found in Debre Zeit, Ethiopia is producing and distributing effective PPR vaccines for Ethiopia and some African countries.

Generally, control of the disease is more effective by applying measures such as slaughter of infected herd, correct disinfection of contaminants and adequate disposal of carcasses, movement control, emergency vaccination and quarantine (Couacy-Hymann, 2013). Other preventive actions include public awareness creation, immediate report, surveillance and proper treatment of products and by-products (AUSVETPLAN, 1996).

2.10 Economic impacts of PPR

Peste des petits ruminants epidemics can cause mortality rates of 50–80% in naive sheep and goats populations (Kitching, 1988). Because of confusion with other diseases, the economic impacts of PPR are probably underestimated, but it is believed that the disease is one of the major constraints of shoat production in tropic (Taylor, 1984). Based on assumption that goats experience an outbreak every 5 years, Opasina and Putt (1985) estimated an annual sum ranging from 2.47£ per goat at highest loss and 0.36 £ per goat at lowest loss. The loss due to PPR in Nigeria was estimated to be 1.5 million dollars annually (Hamdy *et al*., 1976). An economic analysis for assessing benefits of vaccination of shoat against PPR in Niger revealed that such a program was highly beneficial with an anticipated net present value (NPV) return in five years of 24 million USD following an investment of two million USD.

Peste des petits is economically important disease in Ethiopia that excludes the country from profitable international markets thereby greatly reducing the country's foreign exchange earnings (ESGPIP, 2008). According to FAO (2013), PPR can result in huge losses due to mortality in susceptible flocks from 10 to 100 percent and morbidity from 50 to 100 percent with significant economic, food security and livelihood impacts. Central statistical Agency 2015/16 survey revealed that, the economic loss from small ruminant mortality due to the disease was estimated to be 4,989,677 heads of sheep and 5,582,924 heads of goat. In spite of the fact that, PPR and other disease outbreaks are under reported, due to poor reporting system in the country, this figure can say a lot about the impact of diseases on the livelihood of the people in Ethiopia and peste des petits ruminants could be one of the causes for such huge losses (CSA, 2015/16). Sheep and goats contribute a quarter of the domestic meat consumption, half of the domestic wool requirements, 40 percent of fresh skins and 92 percent of the value of semi-processed skin and hide export trade in Ethiopia (Anteneh and Moges, 2017).

In Ethiopia, many households depend on sheep and goat production to feed and educate their families, pay their immediate expenses and women often have access and control over small ruminants making it an important resource of income for them despite the fact that when they lose their shoat, they fall out of livestock production leading to and turn to selling of firewood, grass, charcoal and others to sustain their livelihood (Beyene *et al*., 2018).

2.11 Conclusion

Peste des petits ruminant is a serious viral disease that has high economic impact in areas where there is shoat production and the disease is endemic. The disease is widely distributed in almost all parts of developing countries. Peste des petits ruminant is transmitted by either direct or indirect contacts. Peste des petits ruminants virus primarily infects sheep and goats and it is influenced by age, breeds, flock size, exposure status of animals and climatic condition of the areas. As it is one of the most important diseases of shoat, effective prevention and control measures that involve the use adequate disposal of carcasses, movement control, emergency vaccination and quarantine should be applied to mitigate its impact.

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