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Peste Des Petits Ruminants and its Economic Importance

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Abstract: Peste des petits is a highly acute contagious and infectious viral disease of goats and sheep that is clinically similar to rinderpest and is characterized by fever, erosive stomatitis, diarrhea, conjunctivitis, gastroenteritis and pneumonia. It is caused by Peste des petits ruminants virus (PPRV). The objectives of this paper were to review peste des petits ruminants and its economic significance. The disease occurs south of the Sahara desert and north of the equator in Africa, in most of the Middle East and in parts of Asia including much of the Indian subcontinent. Peste des Petits Ruminants is common in Ethiopia and economic losses are due to loss of production, death, abortion and cost of controlling the disease. Diagnosis can be made on the basis of clinical, pathological and epizootiological findings. Laboratory confirmation is an absolute requirement, particularly in areas or countries where PPR has not previously been reported. There is no evidence that peste des petits ruminants virus infects humans. There is no specific treatment for PPR; however, drugs that control bacterial and parasitic complications may decrease mortality. Ring vaccination and/or vaccination of high-risk populations can also be helpful. It can be eradicated with a combination of quarantines, movement controls, euthanasia of infected and exposed animals and cleaning and disinfection of infected premises. Farmers/pastoralists should keep newly purchased sheep and goats separate from other animals.

Key words: Peste des petits ruminants · Goats · Sheep

INTRODUCTION

Ethiopia is known to have the largest livestock population in Africa. This livestock sector has been contributing considerable portion to the economy of the country and still promising to rally round the economic development of the country [1]. Estimate indicates that the country is a home for about 54 million cattle, 25.5 million sheep and 24.06 million goats. From the total cattle population 98.95% are local breeds and the remaining are hybrid and exotic breeds [2]. In spite of having the largest livestock population in Africa, the contribution for the economic aspect of the country is still lowest. The most important constraints are widespread endemic diseases including viral, bacterial and parasitic infestation [3-8]. The other important bottleneck for development of this sector include lack of appropriate disease control policy, lack of appropriate veterinary services and lack of attention from government [4-10]. A number of researches have confirmed the presence of PPR in Ethiopia [11-13].

Peste des petits was first described in 1942 by Gorgadennec and Lalanne, who investigated the syndrome in sheep *Oviesaries* and goat *Capra hircus* in Côte d'Ivoire, West Africa [14]. It is a highly acute contagious and infectious viral disease of goats and sheep that is clinically similar to rinderpest and is characterized by fever, erosive stomatitis, diarrhea, conjunctivitis, gastroenteritis and pneumonia [15]. The causative agent is peste des petits virus [16].

Among domesticated animals, a peste des petits ruminant is primarily a disease of goats and sheep. In addition, other species, such as camel and cattle, was also infected by the virus but without showing clear disease. The host range in wild animals is still unknown and it is possible that this disease could threaten the conservation of some wildlife species [17]. Close contact with infected animal or contaminated fomites is required for the disease to spread. Ocular, nasal and oral secretions and feces are the sources of virus [18]. Kids over 4 months and under 1 year of age are most susceptible to the disease.

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Sahelian breeds of sheep and goats are believed to be more resistant than the dwarf breeds. The disease occurs south of the Sahara desert and north of the equator in Africa, in most of the Middle East and in parts of Asia including much of the Indian subcontinent. PPR virus, like other morbilli viruses, is lymphotropic and epitheliotropic consequently; it induces the most severe lesions in organ systems rich in lymphoid and epithelial tissues [19]. The initial clinical signs were anorexia and depression, followed by fever, lacrimation, congested mucous membranes, nasal discharges, salivation and diarrhea [20]. The pathology caused by it is dominated by inflammatory and necrotic lesions in the mouth and the gastrointestinal tract [21].

PPR was suspected on clinical grounds to be present in goat herds in Afar region of Eastern Ethiopia in 1977. Moreover, serological and clinical evidences were reported by Tibbo *et al.* [12]. Economic losses are due to loss of production, death, abortion and cost of controlling the disease. Heavy losses can be seen, especially in goats; all of the affected animals in some herds may die [21].

Small ruminants infected with PPR are routinely diagnosed on the basis of clinical examination, gross pathology, histological findings and laboratory confirmation [12, 22, 23]. In the field, a presumptive diagnosis can be made on the basis of clinical, pathological and epizootiological findings. Laboratory confirmation is an absolute requirement particularly in areas or countries where PPR has not previously been reported [24]. Specimens to submit include blood in heparin anticoagulant, clotted blood or serum (if possible, paired sera), mesenteric lymph nodes, spleen, lung, tonsils and sections of the ileum and large intestine. Swabs of serous nasal and lachrymal discharges may also be useful. All samples should be shipped fresh (not frozen) on ice within 12 hours after collection [23].

There is no specific treatment for PPR. However, drugs that control bacterial and parasitic complications may decrease mortality [25]. Peste des petits ruminants can be eradicated with a combination of quarantines, movement controls, euthanasia of infected and exposed animals and cleaning and disinfection of infected premises. Ring vaccination and/or vaccination of high-risk populations can also be helpful [26]. Despite the economically important this disease, there is paucity of well documented information. Therefore, this paper was prepared with the objectives to overview an etiology, epidemiology, diagnosis and control of peste des petits ruminant along with its economic importance.

Etiology: Pest des petits is a highly acute contagious and infectious viral disease of goats and sheep that is clinically similar to rinderpest and is characterized by fever, erosive stomatitis, diarrhea, conjunctivitis, gastroenteritis and pneumonia. The name is French for "disastrous disease of small ruminants". Goats are usually more severely affected than sheep [15].

Peste des petits ruminant's virus (PPRV) is a member of the genus Morbillivirus. It is classified in the order Mononegavirale family Paramyxoviridae, subfamily Paramyxovirinae, genus Morbillivirus on account of its genetic similarity with other members of the genus Morbillivirus that includes measles virus (MeV), rinderpest virus (RPV), canine distemper virus (CDV) and a number of other viruses that infect aquatic mammals. Structurally, the morbilliviruses are morphologically pleomorphic particles (400–500 nm) similar in appearance to other members of the family Paramyxoviridae being enveloped (cell membrane derived) with viral glycoprotein's seen as peplomers protruding from the envelope [16]. The non-segmented, negative-strand genome encodes eight proteins: the nucleocapsid protein (N), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin protein (H), the polymerase protein (L) and the two non-structural proteins and V. Haemagglutinin (H) and fusion (F) proteins are enable the virus to become attached to the target cell and to release its nucleocapsid into the cytoplasm. The neutralizing antibodies produced by the infected host are directed against these proteins. Therefore, the genes of these two proteins might be of use in producing a PPR vaccine, with the help of genetic engineering. Owing to the presence of this envelope, the virus is easily destroyed by means of lipid solvents and is very delicate, particularly outside the host [27].

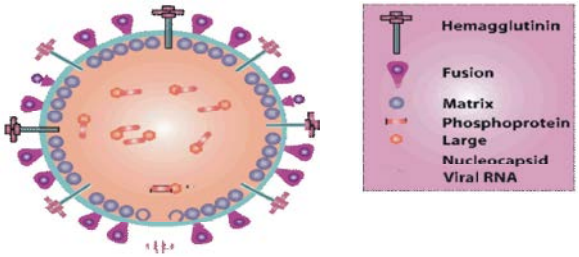


Fig. 1: A schematic illustration of the PPR virus structure Source: [27]

Epidemiology

Species Affected: Among domesticated animals, a peste des petits ruminant is primarily a disease of goats and sheep. Cattle are usually infected asymptomatically and

are not known to transmit the disease to other animals. Clinical PPR is confirmed in goats; however, its circulation in other animals has never been described. Goats were severely affected while sheep generally underwent a mild form. In addition, other species, the camel, was infected by the virus but without showing clear disease. This difference may result from a difference in cell susceptibility to the virus. Although both cattle and pigs are susceptible to infection, but do not contribute to the epidemiology as they are unable to excrete virus and they develop a humoral immune response to PPRV that protects them [17]. Peste des petits ruminants can affect some wild ungulates, but there is very limited information on species susceptibility and the occurrence of disease. Peste des petits was confirmed as the cause of two severe outbreaks, one in captive Dorcas gazelles (*Gazella dorcas*) and Thomson's gazelles (*Gazella thomsoni*) in Saudi Arabia in 2002 and the other in buffalo in India in 1995. It is also thought to have caused another outbreak that affected both gazelles and deer in Saudi Arabia in the 1980s. White-tailed deer (*Odocoileus virginianus)* can be infected experimentally. In addition, peste des petits ruminants have been reported in captive Nubian ibex, Laristan sheep and gemsbok. Whether wild ruminants are important in the epidemiology of this disease is unknown [26]. However, there is one report of naturally occurring PPR in captive wild ungulates from three families: *Gazellinae* (*dorcas gazelle*), *Caprinae* (Nubian ibex and Laristan sheep) and *Hippotraginae* (gemsbok). Experimentally, the American white-tailed deer (*Odocoileus virginianus*) is fully susceptible. The role of wildlife on the epizootiology of PPR in Africa remains to be investigated [21].

Transmission: Close contact with infected animal or contaminated fomites is required for the disease to spread. Ocular, nasal and oral secretions and feces are the sources of virus. Contact infection occurs mainly through inhalation of aerosols produced by sneezing and coughing. Peste des petits virus is shed in nasal and ocular secretions, saliva, urine and feces. It probably occurs in milk. Infection may spread to offspring by feeding them the milk of an infected dam. The exact viral survival in milk has not been demonstrated for PPR. The spread of PPRV is affected by both host density and birth rate and animals that survive infection are protected for life. Where particularly virulent isolates are involved and a naïve population is exposed to the virus, the mortality rate is often very high. Herd animals that are in constant contact with each other, like sheep and goats, are therefore very susceptible to serious outbreak. Although animals are not expected to become long-term carriers, one recent study reported that viral antigens were shed in the feces of clinically recovered goats for at least 11-12 weeks [18]. As in rinderpest (RP), there is no known carrier state. The only sources of the virus, therefore, are sheep and goats in the incubation period, or those affected by the disease. Infection is likely to be spread in the subclinical infection during the incubation period [29]. Peste des petits virus is relatively fragile in the environment and long distance aerosol transmission is unlikely; in cool temperatures and in the dark, this virus has been shown to spread for approximately 10 meters [26].

Fomites such as water, feed troughs and bedding can probably transmit PPRV for a short time, but do not remain infectious for long periods. There is very little information on the survival of PPRV in the environment, but this virus is very similar to rinderpest virus, which is inactivated by ultraviolet light and desiccation within four days and normally survives for very short periods in carcasses. Temperatures above 70°C, as well as pH less than 5.6 or greater than 9.6, are also expected to inactivate PPRV. This virus may, like rinderpest virus, survive for a time in refrigerated meat and for several months in salted or frozen meat. However, PPRV is unlikely to be transmitted to sheep or goats from this source, because pigs that might be fed meat are dead-end hosts. How the virus is maintained between outbreaks is not well understood [20].

Morbidity and Mortality: The incidence of PPR in an enzootic area may be similar to that of rinderpest (RP) in that a low rate of infection exists continuously. When the susceptible population builds up, periodic epizootics (outbreaks) occur, that receive more attention than usual. Such epizootics may be characterized by almost 100 percent mortality among affected goat and sheep populations. The prognosis of acute PPR is usually poor. The severity of the disease and outcome in the individual is correlated with the extent of mouth lesions. Prognosis is good in cases where the lesions resolve within 2 to 3 days. It is poor when extensive necrosis and secondary bacterial infections result in an unpleasant, fetid odor from the animal's breath. Respiratory involvement is also a poor prognostic sign. A morbidity rate of 80-90 percent and a case fatality rate of 50-80 percent are not uncommon, particularly in goats [18]. Young animals (4 to 8 months) have more severe disease and morbidity and mortality are higher. Both field and laboratory observations indicate that PPR is less severe in sheep than in goats. Poor nutritional status, stress of movement and concurrent parasitic and bacterial infections enhance the severity of clinical signs [18].

The severity of the disease varies with the host's species, immunity and breed. Clinical signs are reported to be more common in goats than sheep in Africa, breed differences are also seen: some isolates can affect one breed of goats severely, while causing mild disease in another. The morbidity and mortality rates can reach 100%, particularly in naïve herds; however, these rates tend to be lower in endemic areas and the reported mortality rates in some individual flocks are as low as 20% [30].

High case fatality rates have been reported when PPRV infected herds of exotic ungulates. In an outbreak among buffalo in India, the case fatality rate was 96%. Fifty of 385 buffalos were affected; most (38) of these cases occurred in animals that had been recently introduced into the herd and were not yet vaccinated against rinderpest. In captive gazelles, the morbidity rate was 51% and the case fatality rate was 100%. During a countrywide outbreak among camels in Ethiopia, the morbidity rate was greater than 90% and the mortality rate ranged from 5% to 70% [20].

Risk Factors: Kids over 4 months and under 1 year of age are 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 out–break is greatly increased when a new stock is introduced or when animals are returned unsold from livestock markets [19].

Confinement and restricted movement of the animals, due to rainy seasons in tropical countries, may affect the nutritional status of the animals and hence predispose them to PPRV infection [31]. Sero-positive PPR cases was reported during the months of December, January and February followed by the months of September and October. December to February appeared to be the period of high risk for small ruminants to PPR infection. The least seroprevalence was observed from March to August. The migration of animals during the coolest months may be one of the reasons for the higher frequency of PPR outbreaks during the months of December, January and February. However, limited fodder also makes animals nutritionally deficient, resulting in an increased susceptibility to further infections. Climatic factors favourable for the survival and spread of the virus may also contribute to the seasonal distribution of PPR outbreaks. With the start of the rainy season between (June/July and August/September), the migratory activity of animals is reduced due to the increased availability of local fodder. The nutritional status of the animals is also improved, resulting in an increased resistance to infection. These factors may play a key role in limiting the transmission of disease. PPR prevail throughout the year in the country [32, 33].

Geographic Distribution: Peste des petits ruminants occurs south of the Sahara desert and north of the equator in Africa, in most of the Middle East and in parts of Asia including much of the Indian subcontinent. It is widely distributed in goat rearing areas of the countries [19].There are four virus lineages found in different geographic regions. Lineages 1 and 2 occur in parts of Africa and lineage 3 has been reported from parts of Africa, the Middle East and southern India. It is not certain whether lineage 3 has persisted in India; one study reports that there is no evidence for this virus after 1992. Lineage 4 has been found in the Middle East and the Indian sub-continent, but as of 2008, this virus has not been reported from Africa [33].

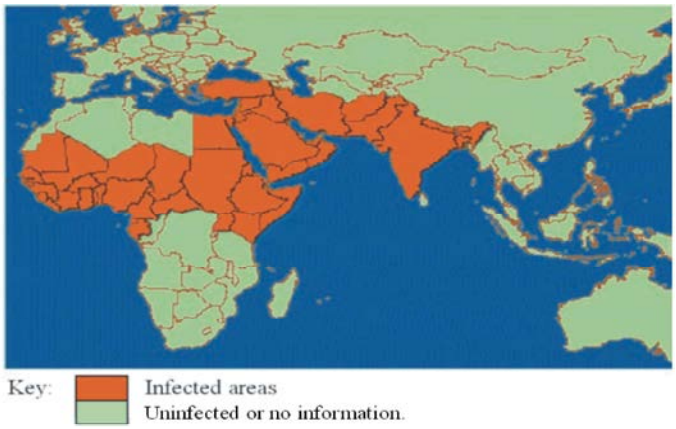


Fig. 2: Geographical distribution of PPR Key: Infected areas
Source: [34]

Pathogenesis: PPR virus, like other morbilliviruses, is lymphotropic and epitheliotropic consequently; it induces the most severe lesions in organ systems rich in lymphoid and epithelial tissues. The respiratory route is the likely portal to entry. After the entry of the virus through the respiratory tract system, it localizes first replicating in the pharyngeal and mandibular lymph nodes as well as tonsil. Viremia may develop 2-3 days after infection and 1-2 days before the first clinical sign appears. Subsequently viremia results in dissemination of the virus to spleen, bone marrow and mucosa of the gastro intestinal tract and the respiratory system [35].

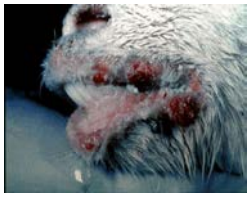
Clinical Sign: The incubation period can range from two to 10 days; in most cases, clinical signs appear in 2-6 days [20]. Most cases of PPR are acute, with a sudden fever that may last for 5-8 days before the animal either dies or begins to recover. The characteristic signs begin with a clear discharge from the nose that becomes grey and sticky. The discharge from the nose may remain mild or may progress to severe inflammation of the mucous membrane of the nose characterized by the presence of exudates that crust over, blocking the nostrils causing respiratory distress. The nasal mucous membranes may develop small areas of erosion. The conjunctiva may be congested with matted eyelids. The mucous membranes in the mouth may also be eroded. Concurrently, animals will most likely have profuse, non-hemorrhagic diarrhea resulting in severe dehydration, which may progress to emaciation, difficult breathing and die within 5-10 days. Bronchopneumonia with coughing is common late in the disease. Infection of pregnant animals with the virus has also, albeit rarely, been linked to abortion. The importance of this and the mechanisms by which it occurs are currently unknown; although co-infection with both PPRV and pestiviruses in cases of abortion has been reported [36]. The prognosis of acute PPR is usually poor. The severity of the disease and outcome in the individual is correlated with the extent of the mouth lesions. Prognosis is good in cases where the lesions resolve within 2 to 3 days. It is poor when extensive necrosis and secondary bacterial infections result in a fetid odor from the animal's mouth. Respiratory involvement is also a poor prognostic sign [29]. The severity of the disease varies with the species, as well as the animal's immunity to PPRV and its breed. Goats and sheep are not always affected to the same extent during an outbreak [20].

PPR in a goat: swollen, eroded lips The lips are swollen, oedematous and show areas of erosion.

PR in a goat: purulent eye and nose discharges. Discharges from the nose and eyes in advanced PPR.

Depression, hemorrhage, diarrhea.

Discharge from the eyes, nose, mouth and erosion in the mouth



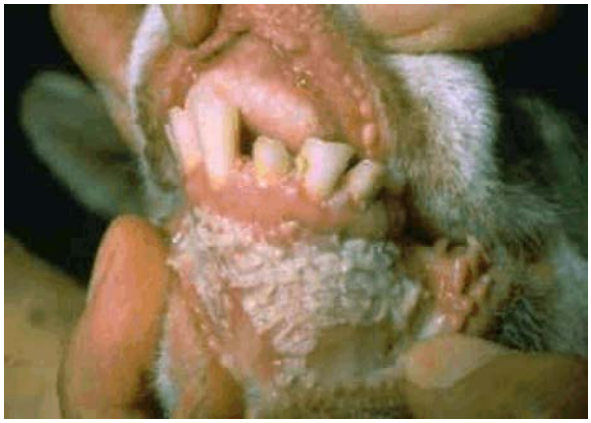
Close up view of mouth lesions

Fig. 3: Clinical signs [29].

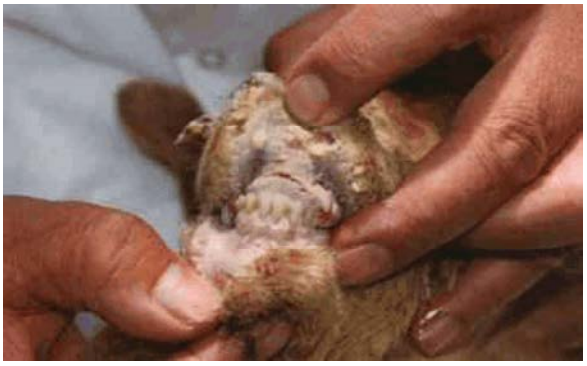
Peracute Form: Peracute cases can be seen when PPR first occurs in native populations of sheep or goats. In this form, the clinical signs are generally limited to high fever, severe depression and death. More often, paste des petites ruminants occur is as sub-acute or acute disease [29].

Domesticated animals other than sheep and goats do not usually become ill. Cattle are usually asymptomatic; however, clinical signs have been reported in experimentally infected calves and it is possible that some cattle in poor condition might become symptomatic. If they did, the syndrome would probably resemble rinderpest. Respiratory disease was reported in camels during an outbreak that may have been complicated by *Streptococcus equi*. Experimentally infected pigs remain asymptomatic [20, 37]. Clinical signs have been described for a few exotic species. The initial signs were anorexia and depression, followed by fever, lacrimation, congested mucous membranes, nasal discharges, salivation and diarrhea. All affected animals died. A highly fatal outbreak in goat was characterized by depression, profuse salivation, conjunctival congestion and febrile [20].

Acute Form: The disease usually appears in the acute form, with an incubation period of 2 to 10 days followed by a sudden rise in body temperature to 104-106° F (40-41°C). The temperature usually remains high for about 5 to 8 days before slowly returning to normal proceeding recovery or dropping below normal before death. Affected animals appear ill and restless and have a dull coat, dry muzzle and depressed appetite. Accompanying these non specific signs is a series of changes that make up a highly characteristic syndrome. From the onset of fever, most animals have a serious nasal discharge, which progressively becomes mucopurulent. The discharge may remain slight or may progress, resulting in a profuse catarrhal exudate, which crusts over and occludes the nostrils. At this stage, animals have respiratory distress and there is much sneezing in an attempt to clear the nose. Small areas of necrosis may be seen on the visible nasal mucous membranes. The conjunctiva usually becomes congested and the medial canthus may have some crusting. As with the nose, there may be profuse catarrhal conjunctivitis resulting in matting of the eyelids [21]. Necrotic stomatitis is common. It starts as small, roughened, red, superficial necrotic foci on the gum below the incisor teeth. These areas may resolve within 48 hours or progressively increase to involve the dental pad, the hard palate, cheeks and their papillae and the dorsum of the anterior part of the tongue. Necrosis may result in shallow irregular no hemorrhagic erosions in the affected areas of the mouth and deep fissures on the tongue. Necrotic debris may collect at the oral commissures and scabs may form along the mucocutaneous junction of the lips. There may be excessive salivation but not to the extent of drooling. At the height of development of oral lesions, most animals manifest severe diarrhea, often profuse but not hemorrhagic. As it progresses, there is severe dehydration, emaciation and dyspnea followed by hypothermia and death usually occurs after a course of 5 to 10 days. Bronchopneumonia, evidenced by coughing, is a common feature in the later stages of PPR. Pregnant animals may abort. Secondary latent infections ma be activated and complicate the clinical picture [18].



PPR in a goat: early mouth lesions showing areas of dead cells Early pale, grey areas of dead cells on the gums



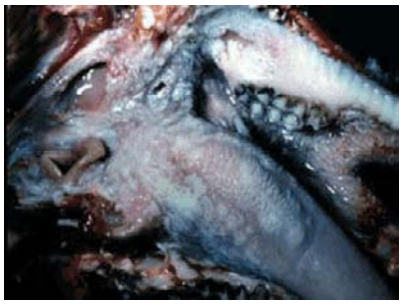
PPR in a goat: later mouth lesions
The membrane lining the mouth is completely obscured by a thick cheesy material; shallow erosions are found underneath the dead surface cells

Fig. 4. Clinical signs [29].

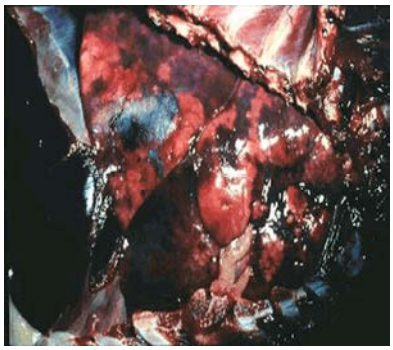
Gross Lesion: The pathology caused by PPR is dominated by inflammatory and necrotic lesions in the mouth and the gastrointestinal tract. Unlike RP, there is also a definite, albeit inconstant, respiratory system component; hence, the synonym stomatitis pneumoenteritis complex. Emaciation, conjunctivitis, erosive stomatitis involving the inside of the lower lip and adjacent gum, cheeks near the commissures and the free portion of the tongue are frequent lesions. In severe cases, lesions may also be found on the hard palate, pharynx and upper third of the esophagus [21]. The necrotic lesions do not evolve into ulcers because the basal layer of the squamous epithelium is rarely penetrated. The rumen, reticulum and omasum rarely have lesions. Sometimes, there may be erosions on the pillars of the rumen. The abomasum is a common site of regularly outlined erosions and often oozes blood. Lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages and, sometimes, erosions in the first portion of the duodenum and the terminal ileum [38]. Peyer's patches are the site of extensive necrosis, which may result in severe ulceration. The large intestine is usually more severely affected with congestion around the ileocecal valve, at the ceco-colic junction and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion ("zebra stripes") form on the crests of the mucosal folds. In the respiratory system, small erosions and petechiae may be visible on the nasal mucosa, turbinates, larynx and trachea. Bronchopneumonia may be present, usually confined to the anteroventral areas and is characterized by consolidation and atelectasis. There may be pleuritis, which may become exudative and results in hydrothorax. The spleen may be slightly enlarged and congested. Most lymph nodes throughout the body are enlarged, congested and edematous. Erosive vulvovaginitis similar to the lesions in the oral mucocutaneous junction may be present. The postmortem lesions are characterized by inflammatory and necrotic lesions in the oral cavity and throughout the gastrointestinal tract [19].

The carcass is often emaciated and/or dehydrated and may have evidence of diarrhea and serous or mucopurulent oculonasal discharges. The lips often have prominent crusty scabs and necrotic stomatitis is common. Erosions, which are shallow and sharply demarcated from normal epithelium, may be found in the mouths of some animals. In severe cases, the hard palate, pharynx and upper esophagus can also be involved. Similar lesions may be found on the vulva and vaginal mucous membranes. The rumen, reticulum and omasum are not significantly involved, although erosions are occasionally found on the pillars of the rumen. Erosions, which may ooze blood, are common in the abomasum. Hemorrhagic streaks and erosions sometimes occur in the duodenum and the terminal ileum [38]. The Peyer's patches often have extensive necrosis, which can lead to ulceration. The most severe lesions are seen in the large intestine, particularly around the ileocecal valve, at the cecocolic junction and in the rectum. "Zebra stripes" or "tiger stripes" of congestion hemorrhage or darkened tissue can sometimes be seen in the posterior part of the colon on the mucosal folds. (Zebra stripes can also be seen in animals with diarrhea and tenesmus from other causes). Respiratory lesions are also common. Congestion, small erosions and petechiae may be found in the nasal mucosa, turbinate's, larynx and trachea and blood-tinged, frothy exudates have been reported in the tracheas of some experimentally infected goats. Many animals have bronchopneumonia. The lymph nodes, particularly those associated with the respiratory and gastrointestinal tracts are generally congested, enlarged and edematous. In peracute cases, the lesions may be limited to congestion of the ileocecal valve and bronchopneumonia [20].

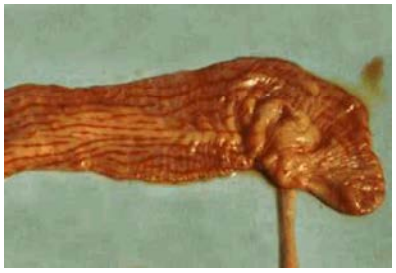
Similar lesions have been reported in buffalo and gazelles. Hemorrhagic and edematous gastroenteritis (involving the abomasum and all segments of the intestines) was reported in infected buffalo. In gazelles, small erosions were found on the tongue and the esophagus contained thick mucoid deposits along the walls. The papillae of the rumen were congested [26]. The abomasum was severely affected, with tiny hemorrhagic erosions, marked congestion and edema in the pyloric region. Congestion, hemorrhages and small erosions were found in the duodenum and congestion was seen in the jejunum. The Peyer's patches appeared shallow and were hyperemic at their edges. Congestion was seen around the ileocecal valve. The mucosal of the colon and rectum were congested, with a 'zebra stripe' pattern. Congestion was also reported in the liver, kidney, pancreas and brain. Froth was found in the trachea and bronchi and the lungs were congested. The lymph nodes and spleen were small. Unilateral corneal opacity was reported in one animal [30].



Necrotic lesions in the oral cavity



Pneumonia



Zebra stripes on intestine

Fig. 5: Post-mortem lesions [29]

Diagnosis: Successful implementation of control measures for PPR requires rapid, specific and sensitive methods for diagnosis. Small ruminants infected with PPR are routinely diagnosed on the basis of clinical examination, gross pathology, histological findings and laboratory confirmation [22, 23]. Differential diagnosis include: rinderpest, contagious caprine pleuropneumonia, bluetongue, Pasteurellosis, contagious ecthyma, foot and mouth disease, heartwater, coccidiosis, Nairobi sheep disease and mineral poisoning. The case history, geographic location and the combination of clinical signs can help differentiate some of these diseases. For PPR diagnosis swabs of the mucous membrane of eye, nose, mouth and rectal discharges should be collected. Whole blood must be collected in heparinized tubes. Samples may also be taken of the spleen, large intestine and lungs. These samples should be transported under refrigeration [29].

Field Diagnosis: In the field, a presumptive diagnosis can be made on the basis of clinical, pathological and epizootiological findings. Laboratory confirmation is an absolute requirement particularly in areas or countries where PPR has not previously been reported [24]. Peste des petits ruminants should be considered in sheep, goats or gazelles with any acutely febrile, highly contagious disease characterized by oral necrosis with ocular and nasal discharge [30].

Specimens for the Laboratory: Specimens to submit include blood in heparin anticoagulant, clotted blood or serum (if possible, paired sera), mesenteric lymph nodes, spleen, lung, tonsils and sections of the ileum and large intestine. Swabs of serous nasal and lachrymal discharges may also be useful. All samples should be shipped fresh (not frozen) on ice within 12 hours after collection [23].

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease [38]. In live animals, swabs of ocular and nasal discharges and debris from oral lesions should be collected; a spatula can be rubbed across the gum and inside the lips to collect samples from oral lesions. Whole, unclotted blood (in heparin) should be taken for virus antigen isolation and PCR. Biopsy samples of lymph nodes or spleen may also be useful. Samples for virus isolation should be collected during the acute stage of the disease, when clinical signs are present; whenever possible, these samples should be taken from animals with high fever and before the onset of diarrhea. At necropsy, samples can be collected from lymph nodes (particularly the mesenteric and mediastinal nodes), lungs, spleen, tonsils and affected sections of the intestinal tract (e.g. ileum and large intestine). These samples should be taken from euthanized or freshly dead animals. Samples for virus isolation should be transported chilled on ice. Similar samples should be collected in formalin for histopathology. Whenever possible, paired sera should be taken rather than single samples. However, in countries that are PPR-free, a single serum sample (taken at least a week after the onset of clinical signs) may be diagnostic [26].

Laboratory Diagnosis: A number of serological and molecular diagnostic tests are used for the detection of PPR [17].

Viral Isolation: Virus isolation in cell culture can be attempted using several different cell lines. Primary lamb kidney or African green monkey kidney (Vero) cell cultures have also been used. Cultures are examined for cytopathic effect (CPE); the identity of the virus can be confirmed by virus neutralization or other methods.Techniques for virus isolation cannot be used as routine diagnostic tests as they are time-consuming and cumbersome [39].

Antibody Detection: The neutralization test: The neutralization test is used for antibody detection. The highest neutralization titre is obtained against the homologous virus. Vero cells are commonly used for this test [40].

Competitive ELISA (cALISA): It is the most suitable choice as it is sensitive, specific, reliable and has a high diagnostic specificity (99.8%) and sensitivity (90.5%) [41].

Antigen Detection Methods: Agar-gel immunodiffusion: It is a relatively simple, fast and cheap process. It is extremely useful as an initial test, but it does not discriminate between PPR and rinderpest viruses and further tests are needed to do this. The extract of pathological samples (ocular and nasal swabs, lymph nodes, spleen or lung) is allowed to diffuse, along with known PPR positive antigen, in agar gel against a rabbit PPR hyper immune serum [42]. Immuno-capture enzyme-linked immunosorbent assay (Ic-ELISA): tests using monoclonal antibodies are often used for serological diagnosis and antigen detection for diagnostic and screening purposes [43]. The Polymerase Chain Reaction (PCR): This technique has been the most popular and highly sensitive tool so far for diagnosis of PPR [44].

Differential Diagnosis: Rinderpest: Clinical RP is rare in goats and sheep in Africa. In India, these species are quite often involved in RP outbreaks. Clinically, RP and PPR are similar, but the former should be the prime suspect if the disease involves both cattle and small ruminants. Confirmation requires virus isolation and cross-neutralization [37].

Pasteurellosis: Enzootic pneumonia or the septicemic form of pasteurellosis is characterized by obvious respiratory signs, infrequent diarrhea and a fatality rate rarely exceeding 10 percent. Contagious caprine pleuropneumonia, there is no digestive system involvement and the clinical signs and lesions are confined to the respiratory system and pericardium [37].

Bluetongue: Swelling of the lips, muzzle and oral mucosa, together with edema of the head region, should serve to differentiate bluetongue from PPR. Coronitis is common in bluetongue, is not a feature of PPR. Also, sheep are more affected than goats [38].

Heart water: There is often central nervous system involvement, including convulsions. There is no diarrhea [25].

Contagious ecthyma (contagious pustular dermatitis, orf): it is zoonotic disease caused by parapox virus. The orf virus causes proliferative, not necrotic lesions, which involve the lips rather than the whole oral cavity. The absence of nasal discharges and diarrhea also distinguish orf from PPR [37].

Foot-and-mouth disease: caused by an aphthovirus this condition is comparatively mild and the most characteristic clinical sign, lameness, is not a feature of PPR [25].

Nairobi sheep disease: Sheep are more severely affected than goats. It is limited geographically to parts of east and central Africa (Kenya, Uganda, Tanzania, Ethiopia, Somalia and Congo [formerly Zaire]). Diagnosis requires isolation and serologic identification of the virus [25].

Coccidiosis: There is no upper digestive tract and respiratory system involvement [25].

Plant or mineral poisoning: Several plants and minerals may cause severe intestinal lesions. Case history and absence of fever should distinguish poisoning from PPR [25].

Status of Ppr in Ethiopia and Economic Importance: PPR was suspected on clinical grounds to be present in goat herds in Afar region of Eastern Ethiopia in 1977. Moreover, serological and clinical evidences were reported [12]. However, the presence of the virus was only confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding land near Addis Ababa. PPR was characterized by ocular and nasal discharges, mouth lesions, pneumonia, gastro enteritis and diarrhea [38]. The disease in this outbreak caused more than 60% mortality. The disease probably was introduced into Ethiopia in 1989 in the Southern Omo river valley from where it moved eastward to Borena region and then northwards along the Rift

valley to Awash. The disease became endemic in goats [45]. Small ruminants in this country mainly thrive on free-range pasture land, shrubs and forest cover. Due to the shrinkage in pasture land and forest area, these animals move to long distance in search of fodder and water during dry season. This phenomenon is common due to different summer and winter grazing grounds depending upon the altitude. PPR is transmitted through direct contact between infected animal and susceptible population [46].

During nomadism, animals come in contact with local sheep and goat population from where they pick up the infection or spread disease if nomadic flock is pre-exposed. Therefore, migratory flocks play an important role in transmission epidemiology of PPR. Movement of animals and introduction of newly purchased animals from the market also play an important role in transmission and maintenance of the virus. This could be one of the possible reasons for higher frequency of PPR outbreaks between March to June, which also correspond to lean period of kidding [17]

Although seasonal occurrence of PPR virus outbreaks is disputed, disease transmission is certainly affected by animal movement for which socioeconomic factors and variations in agro climatic conditions are responsible. Large group of animals move to large areas and even between different districts. With the start of rains, the movement of animals is restricted due to the easy availability of local fodder. Nutritional status of the animals also gets improved during the rains. This may reduce disease transmission after the start of rains and during the period of easy availability of fodder [17]. A number of researches have confirmed the presence of PPR in Ethiopia. Laboratory analysis of sample collected in goat disease outbreak reported by the regional veterinary official with the suspicion of PPR in southern Ethiopia, Arsi zone, Shirka woreda confirmed the case to be PPR [13].

There is no evidence that peste des petits ruminants virus infects humans. Nasal discharges, respiratory disease and/or gastrointestinal signs [19]. Peste des Petits Ruminants (PPR) is a disease of major economic importance and imposes a significant constraint upon sheep and goat production owing to its high mortality rate. It is an acute, highly contagious and frequently fatal disease of sheep and goats caused by PPR virus (PPRV), a member of genus morbillivirus of family Paramyxoviridae. Therefore, it poses serious threat to the development of small ruminants production in many countries where it is endemic and is a source of great financial loss to the farmers and livestock owners. Economically PPR is sometimes referred to as pseudorinderpest because its effect on sheep and goats has some parallels clinically and epidemiologically to rinderpest in cattle [47].

Treatment: There is no specific treatment for goat plague. Affected goat with stomatitis, enteritis and pneumonia were treated with penicillin and streptomycin reinforced with broad-spectrum chloramphenicol [48]. However, mortality rates can be reduced by the use of drugs that control the bacterial and parasitic complications. Specifically oxytetracyclineand chlortetracycline is recommended to prevent secondary pulmonary infections [49]. Valuable sick animals in the early stages of the disease should be isolated and given hyperimmuneserum, which may be obtained from cattle hyperimmunized against rinderpest. Lesions around the eyes, nostrilsand mouth should be cleaned and good nursing provided the disease may precipitate other infections and the animals harbor without clinical signs, such as blood and internal parasites. Thus, it may be necessary to administer antiprozoal and anthelmintics as well [50].

Prevention and Control: The disease can be prevented by not introducing new stock from unknown sources, especially animals bought at livestock markets. In addition animals returned unsold from markets should be segregated unless the entire herd or flock has been vaccinated. Vaccination is the most effective way to gain control epidemic PPR. It is important to vaccinate animals in areas where the disease does not often happen if there is a risk of sick animals from elsewhere being brought through the area [51]. The vaccine is currently used in many African countries for vaccination against PPR. Recently, a homologous PPR vaccine has been developed and the vaccine seed is available through the PANVAC at Debre Zeit, Ethiopia. The vaccine can protect small ruminants against PPR for at least for 3 years. Ring vaccination and/or vaccination of high-risk populations can be helpful [52].

Animals that recover develop good immunity, which persists for at least four years and possibly lifelong. To prevent infections in susceptible wildlife and captive wild animals such as gazelles, they should be prevented from having contact with sheep and goats. Vaccination might also be possible in these species [30]. Eradication is recommended when PPR appears in new areas.

Methods that have been successfully applied for RP eradication in many areas would be appropriate for PPR. These should include quarantine, slaughter and proper disposal of carcasses and contact fomites, decontamination and restrictions on importation of sheep and goats from affected areas [25].

Carcasses of dead animals and contaminated items should be buried or burned. The rapid in activation of PPRV in the environment aids eradication; this virus is thought to remain viable for less than four days outside the animal. Peste des petits virus can be inactivated by many disinfectants including alkalis (sodium carbonate, sodium hydroxide), halogens (sodium hypochlorite), phenolic compounds, citric acid, alcohols and iodophores. Care should be taken to prevent the virus from spreading to susceptible or potentially susceptible wild populations such as deer, gazelles, wild sheep or feral goats [26].

CONCLUSIONS AND RECOMMENDATIONS

Peste des petits is highly contagious virul disease of small ruminant. It is primarily a problem of sheep and goats in Africa (from Tropic of Cancer to Equator), the Middle East and the Indian subcontinent. Economically it is the most important small ruminants disease. Goats are severely affected while sheep undergo mild form. In a particular flock, the risk of an out–break is greatly increased when a new stock is introduced or when animals are returned unsold from livestock markets. There is no specific treatment for the disease Therefore, it is very important to support and implement control programmes so as to prevent further spread of the disease. Advances in knowledge and development/ design of control tools for PPR disease including diagnostics and vaccines provide an excellent prospect for improved control programs.

Based on the fact and information mentioned in the review the following recommendations are forwarded.

- Farmers/pastoralists should keep newly purchased sheep and goats separate from other animals.
- The veterinary services in the country must review their preparedness plans, strengthen border control and improve surveillance.
- Government should be involved in the control of the disease in integrated manner among the ministries of health, agricultures and other social associations to save economic loss due to the death of animals and should also provide regular public awareness education on the prevention and control of PPR.
- The Government State and federal veterinarians should be informed immediately of any suspected cases of peste des petits ruminants.

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