**Chapter-II**

**REVIEW OF LITERATURE**

**2.1. History**

The disease was first recorded in goat and sheep in Ivory coast of West Africa in 1940 (**Gargadennec and lalanne, 1992**). The PPR virus was first recorded and isolated by Gilbert and Monnier in 1962 in Senegal. PPR virus circulates in belt lying across Africa, Saudi Arabia, in 1981 and spread north wards into Jordan, Syria, Iraq, India, Pakistan (**Sil, 2000**). Later it spread to South India in1987 (**Shaila *et al.*, 1989**) and in Bangladesh in 1993 (**Sil *et al.*, 1995**; **Debnath, 1995**).

**2.2. Etiology of the disease**

PPR virus is enveloped with helical pleomorphic shape containing sense single stranded non segmented RNA molecule. The genome of this virus is a single linear molecule of approximately 4.5 × 106 DA with 16,000 ribonucleotides which is encoded with six structural protein, the Nucleocapside (N), Matrix (M), Fusion (F), Haemaglutinin (H), Polymerase (P) (Sil, 2000). The PPR virus identified in Bangladesh is under the lineage 4 of PPR phylogenetic tree based on the N gene analysis (**Barrett *et al.*, 1998**).

**Figure 01: PPR Virus structure**

**2.3. Epidemiology and geographic distribution**

PPR was first described in Côte d’Ivoire (**Gargadennec and Lalanne, 1942**) and thereafter, it has been recognized in many of the sub-Saharan countries that lie between the Atlantic Ocean and the Red Sea (**Lefevre and Diallo, 1990**). The affected area extends north to Egypt and south to Kenya, in Eastern-Africa, and to Gabon, in Western-Africa. PPR has not been recognized in most of Northern and Southern- Africa. In 1998, serological survey in the United Republic of Tanzania did not detect any antibodies to PPR suggesting that infection has not extended that far to the south. PPR is present in nearly all Middle Eastern countries up to Turkey (**Furley *et al.*, 1987**; **Lefèvre *et al****.,* **1991**; **Perl *et al.,* 1994**; **Taylor *et al.,* 1990**, **Ozkul *et al.,* 2002**). It was also widespread in India and southwest Asia (**Shaila *et al.,* 1989**).

Presently, PPR occurs in most African countries situated in a wide belt between the Sahara and equator, the Middle East (Arabian peninsula, Israel, Syria and Jordon) and the Indian subcontinent. Outbreaks of PPR are now known to be common in India, Nepal, Bangladesh, Pakistan and Afghanistan (**Abdollahpour *et al.*, 2006**). It still causes serious economic losses (**Diallo, 2003**) and remains a major constraint on the development of small ruminant farms in these countries. PPR is considered to be one of the main constraints to improve productivity of small ruminants in the regions where it is endemic.

Major outbreaks in Turkey and India in recent years have indicated a marked rise in the global incidence of PPRV (**Bailey *et al.*, 2005**). It is of great economic importance on the basis of mortalities, morbidity, losses through body wastage, poor food efficiency, loss of meat, milk and milk products and offspring. A consequence of this high mortality was the inclusion of PPR in the list A of the former animal disease classification of the OIE, the world organization for the Animal health. In the new OIE classification it is included in a group of economically important animal diseases, which must be notified to the Organization in all the regions where PPR is endemic. Although nationwide sero-surveys have been conducted in countries such as the sultanate of Oman, Turkey, Jordan and India, information on the frequency and distribution of PPR is often lacking when control or eradication campaigns are initiated (**Ozkul *et al.,* 2002; Singh *et al.,* 2004**).

In India, PPR was first reported in 1987 from Arasur village in the Villapurum district of Tamilnadu, South India (**Shaila *et al.*, 1987**). Since, its first reported occurrence in 1987, PPR was thought to be restricted to southern India up to 1993, after which the epidemics of PPR swept away large number of small ruminants from Northern India (**Nanda *et al.*, 1996**). Now the disease has spread all over India. In Gujarat, incidence of PPR was reported by various workers, namely, **Hinsu *et al****.* (**2001**), **Tiwari** (**2005**), **Nagraj** (**2006**)**, Sannat** (**2006**).

Although, there is only one serotype of the virus (Barrett *et al.,* 1993), PPRV isolates on the basis of partial sequence analysis of the fusion (F) protein gene, can be grouped into four distinct lineages (**Kwiatek *et al*., 2007**). Lineage 1 and 2 are found exclusively in West Africa, whereas lineage 3 is found in Eastern Africa and Arabia. The fourth lineage is confined exclusively in the Middle East Arabia and Indian subcontinent (**Shaila *et al.,* 1996**). Excepting isolate (TN92/1) from southern India, that belonged to lineage 3, all Indian PPRV isolates identified so far belonged to lineage 4 only (**Nanda *et al.,* 1996; Dhar *et al.,* 2002**).



**Figure 2: Geographic distribution of PPRV lineages (Dhar *et al*., 2002)**

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**Figure 3: Phylogenetic relationships of the Peste des Petits Ruminants virus isolates based on (F) protein gene (Ozkul *et al*., 2002)**

**2.4. HOST RANGE OF PPRV**

PPR in sheep and goats, the mainstay of subsistence farming in the developing world can cause mortality rates of 50-80 % in naïve populations. Antelope and other small wild ruminant species can also be severely affected (**Abu Elzein *et al.*, 2004**). A case of clinical disease has been reported in wildlife resulting in deaths of gazelles (Gazella dorcus), ibex (Capra ibex nubiana), gemsbok (Oryx gazelle) and Laristan sheep (Ovis orientalis laristanica). American white tailed deer (Odocoileus virginianus) can be infected experimentally (**Hamdy and Dardiri, 1976**).

Cattle, buffaloes, camels and pigs can become infected but there is little or no evidence of disease associated with their infection. PPRV antigen has been detected in an outbreak of respiratory disease in camel and sick domestic buffaloes (**Taylor *et al.*, 1990; Scott, 2000**; **Abraham *et al.*, 2005**). In addition, PPRV is considered as a serious predisposing factor for respiratory disease complex in sheep and particularly in goats (**Taylor *et al.*, 1990**; **Baily *et al****.*, **2005**).

Considering the immunosuppressive effect of PPRV as all other Morbilliviruses, it may therefore be possible, depending on the age and physical state of the host animal, that PPRV can occasionally overcome the innate resistance of large ruminants and lead to the development of clinical signs similar to Rinderpest. This may explain the disease signs that had occurred in buffalo and camels following PPRV infection. This ability of PPRV to infect large ruminants could pose a serious threat to cattle populations in PPR endemic areas which, with the success of the global Rinderpest eradication programme, are no longer vaccinated against Rinderpest and so do not possess cross protective immunity against this virus (**Chauhan *et al.* 2009**).

**2.5. TRANSMISSION OF PPRV**

PPRV is transmitted by direct contact with secretions and excretions of infected animals. It is highly contagious and all discharges can carry virus. Substantial quantities of virus are found in ocular nasal or oral secretions of sick goats and in the faeces late in disease. Since the virus is enveloped, it is extremely sensitive to inactivation by environmental factors such as heat, sunlight and chemicals. It, therefore, require close contact with an infected animals for successful transmission. Pigs undergo silent infection by contact with infected goats but are unable to transmit the virus and are not regarded as important in the epidemiology of PPR (**Nawathe and Taylor, 1979**). Cattle may be infected without showing any clinical signs on experimental inoculation (**Taylor and Abegunde, 1979**).

The disease is transmitted by aerosols between animals living in close contact. (**Lefevre and** **Diallo, 1990**) and substantial amount of virus is known to be present in the ocular and nasal secretions, as well as faeces of the infected animals (**Taylor, 1984**).

**2.6:Clinical feature of PPR**

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| **Clinical Features of Peste des Pestis Ruminants**  |
| **Feature/Disease Form**  | **Characteristics**  |
| Incubation period  | 2-10 days, most commonly 4-5  |
| Acute  | Most common formSudden high fever (40-41C), remaining high for 5-8 days; will return to normal before recovery or drop below normal before deathSerous nasal discharge, becoming mucopurulent; can crust over and occlude nostrilsPurulent ocular discharge with congested conjunctiva; can encrust, cementing eyelids togetherBronchopneumoniaNecrosis and ulceration of mucous membrane and inflammation of gastrointestinal tract leading to severe, nonhemorrhagic diarrheaRespiratory distress, including dyspnea and sneezing in an attempt to clear noseExcessive salivation but not to point of droolingAnorexiaSevere dehydration and emaciation followed by hypothermiaDeath usually occurs after 5-10 daysAbortion Mortality rate can reach 100%Secondary infections may be activated and complicate clinical signs  |
| Peracute  | Frequent in goats  |
| Subacute and chronic  | PneumoniaDevelops over 10-15 daysInconsistent symptoms  |
| **Adapted from DEFRA, Dhar (2002), EMPRES (1999), Saliki (2008), OIE (2002), Ozkul (2002).**  |

**2.9. Pathogenesis of PPR**

## Scott (1981) stated that 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.

Susceptibility to infection rises with age; however, the disease is rapidly fatal in the young animals **(Ozkul, 2002)**. The clinical signs imitate those of rinderpest, but changes can occur faster. Specific clinical signs are outlined in the table below:

The prognosis of acute PPR is usually poor, especially when lesions do not resolve within 2 to 3 days or when extensive necrosis and bacterial infection give the animal's breath an unpleasant, fetid odor. Young animals (4 to 8 months) often have more severe disease. Also, poor nutrition, stress of movement, and concurrent parasitic and bacterial infections worsen clinical signs (**Saliki, 2008**).

 **2.8. Prevalence of PPR**

The prevalence of PPR in small ruminant specially in goat varies due to different causes. Different researchers recorded a lot of findings. Some of the important findings has cite here. **Khan and Siddique *et al.*,** (**2007**) reported the overall prevalence of PPRV was 43.33% of the ruminant population in Punjab. They also mentioned the overall PPR antibody seroprevalence in goats was 39.02% which is significantly higher.

**Nussieba *et al****.*, (**2009**) examined 519 serum samples for the presence of PPRV antibodies, but 307(59.15%) were positive by CIEP while 263 (50.67%) were positive by C-ELISA. Also 15.36% goats were found positive in PPR c-ELISA in seroprevalence of PPR by **Mehmood *et al****.*, (**2009**). In Pakistan the endemic mortalities as low as 20% have been described by Roeder and Ubi, (**1999**). **Abubakar *et al****.*, (**2008**) recorded the prevalence of PPR in small ruminants in Pakistan was 40.98%, but over all was 46.7%. On the contrary in Cameroon, (N = 320), 35% PPR antibodies while for Nigeria (N= 382), the values was 56.5 (**Majiyagbe *et al.*, 1992**).

Samad (2000) reported black Bengal goats were more susceptible to ppr. Also mortality rate was higher in Barbari black Bengal crosses (Sil, 2000). There is also report of equally susceptibility of male and female goat recorded by Samad (2001). PPR occurs in an epizootic form, it may have morbidity of 80-90% and mortality between 50 and 80 % (**Lefevre and Diallo**, **1990**).

 Outbreaks are more frequent during the rainy season or the dry, cold season (**OIE, 2002**). More cases of PPR and bronchopneumonia were recorded during the dry months of December to January. **Okoli, (2003) recorded 25.1% ppr in late dry** Season, 25.9 % in early winter season, 22.8% in late winter and 26.2% in early dry seasons. Higher incidence of PPR observed here during the dry months of December and January agrees with earlier reports by Obi (1983) and Onyekweodiri and Uzoukwu (1992).

 **2.9. Diagnosis of PPR**

 Presumptive diagnosis is made by knowledge of the history of outbreak, season of the year, recent purchase of animal from the markets, the nature of the clinical signs, degree of the morbidity and mortality indicates the case of PPR (**Blood, *et*. al. 1995).**

Confirmatory diagnosis of PPRV relies on laboratory techniques such as virus isolation, demonstration of PPRV antigen, viral nucleic acid and specific antibodies. The techniques available to differentiate PPR from RP are virus neutralization test, cDNA probes (**Diallo *et al****.*, **1989**), Virus specific monoclonal antibodies in an immunocapture enzyme linked immunosorbent assay (ELISA) (**Libeau *et al****.*, **1994**), Sandwich ELISA, PCR (**Forshyth and Barrett**, **1995**) and haemagglutination using piglet or chicken (**Shaila *et al****.*, **1996**) red blood cells. Similarly PPRV antibodies can be differentiated from RP antibodies by competitive ELISA (**Libeau** ***et* *al.*, 1995**) and serum neutralization (**Diallo *et al****.*, **1995**) test. cELISA is rapid, sensitive and specific and most commonly used technique. RT-PCR test can be used as routine diagnostic tool for PPR diagnosis, which has good correlation with virus isolation (**Brindha *et al****.*, **2001**).

PCR in combination with nucleotide sequencing has made it method of choice for molecular characterization of viruses (**Diallo, 1990**). Sequence analysis provides new insights into the interrelationship among lineages, members of the same genus and also with the members of other genus within the family. Therefore, it has become an important tool, not only in structural, but also in functional characterization of viruses.

**2.9.1. Virus isolation**

Samples for virus isolation include heparinized blood, eye and nasal swabs (from live animals), tonsil, mesenteric lymph nodes, spleen, section of colon and lung. For successful isolation, samples must be collected during the hyperthermic phase (**Lefèvre, 1987**) and submitted to the testing laboratory in cold ice. The most widely used cell culture systems are primary lamb kidney and ovine skin (**Gilbert and Monnier, 1962**; **Laurent**, **1968**; **Taylor and Abegunde**, **1979**) and Vero cells (**Hamdy *et al*.,** **1976**).

The sensitivity of virus isolation technique could be increased when the virus is grown in lamb and goats kidney cells (**Taylor, 1984**). Vero cells are however widely used for their continuity and low liability of contamination. PPRV has also been adapted to grow in other continuous cell lines including BHK-21 (**Lefèvre, 1987**). Vero cells, derived from African green monkey kidney are currently the most widely used cell line for PPRV and RPV. A culture of Vero cells from American type cell culture (ATCC) was found to yield very high titres and is currently used in many laboratories working on PPRV and RPV. Appearance of cytopathic effects (CPE) may require at least 8-10 days or several blind passages. In Vero cells, the cytopathic effects (CPE) produced by PPRV consist of cell rounding, clumping into typical grape-like clusters, formation of small syncytia and appearance of long fine often anastomosing “spindle cells” (**Hamdy *et al***., **1976**).

Like other Morbilliviruses, PPRV produces eosinophilic intracytoplasmic and intranuclear inclusion bodies both in primary cells (**Laurent, 1968**) and continuous cell lines (**Hamdy *et al***., **1976**). T-lymphoblast cell line transformed by *Theileria parva* proved to be more sensitive when compared to other cell culture and gave a result within 24 hours (at least 6 days for other cell culture) for both PPRV and RPV (**Rossiter, 1994**). Once isolated in cell culture, a candidate PPRV may be identified by one of the three procedures:

* Animal inoculation: PPR causes clinical disease in goats and sheep but not in cattle (**Gibbs *et al*., 1979**);
* Reciprocal cross neutralization (differential neutralization): PPRV is neutralized by both PPR and RPV reference sera, but is neutralized at greater titre with the homologous serum (**Taylor and Abegunde, 1979**; **Taylor, 1979**);
* Molecular techniques: cDNA probe, (**Diallo *et al*., 1989**; **Pandey *et al*., 1992**), electrophoretic profile in polyacrylamide gel (PAGE) (**Diallo *et al*., 1987**) and PCR, (**Barret** ***et al*., 1993**; **Couacy-Hymann *et al*., 2002**).

**2.9.2. Antigen detecting methods:**

**2.9.2.1. Agar Gel Immunodiffusion Test**

Agar gel immunodiffusion test (AGID) is widely used and can detect 42.6% of antemortem specimens and necropsy specimens (**Obi, 1984; Abraham** and **Berhan, 2001**). It can be used to test the presence of both antigen and antibodies and can give results within 2-4 hours when RP hyperimmune serum is used while it needs 4-6 hours with PPR hyperimmune serum (Obi, 1984). One of the important advantages of this test that it is highly specific (92%), though it can not differentiate between PPR and RP. Usually, 1–3 precipitin lines will develop between the serum and antigens within 18–24 hours (**Durojaiye *et al.,* 1983**). These are intensified by washing the agar with 5% glacial acetic acid for 5 minutes (this procedure should be carried out with all apparently negative tests before recording a negative result). Positive reactions show lines of identity with the positive control antigen.

Results are obtained in one day, but the test is not sensitive enough to detect mild forms of PPR due to the low quantity of viral antigen that is excreted.

**2.9.2.2. Hyperimmune serum:**

Standard antiserum is made by immunising sheep with 5 ml of PPR virus with a titre of 104 TCID50 (50% tissue culture infective dose) per ml given at weekly intervals for 4 weeks. The animals are bled 5-7 days after the last injection. Standard RP hyperimmune antiserum is also effective in detecting PPR antigen.

**2.9.2.3. Counter immunoelectrophoresis**

Counter immunoelectrophoresis (CIEP) is of the same principle as the AGID except that the gel is electrically charged to improve the sensitivity of the test.

**2.9.2.4. ELISA for antigen detection:**

A monoclonal antibody-based sandwich ELISA was found to be highly sensitive in detection of antigen in tissues and secretions of infected goats (**Saliki *et al*., 1994). Libeau *et al*.** (**1994**) revealed that, another format of antigen ELISA which is more widely used is immunocapture ELISA. It utilizes MAb directed against the nucleocapsid protein. It can give a reliable result within two hours in precoated plates and from samples maintained at room temperature for a period of seven days with no more than 50% reduction in response. The immunocapture ELISA allows a rapid differential diagnosis of PPR or rinderpest viruses, and this is of great importance as the two diseases have a similar geographical distribution and may affect the same animal species. The detecting MAbs used in immunocapture ELISA are directed against two non overlapping domain of the N-protein of PPR and RP, but the capture antibody detects an epitope common to both RP and PPR.

The immunocapture ELISA is suitable for routine diagnosis of rinderpest and PPR from field samples such as ocular and nasal swabs. The main advantages of this assay are: Rapidity, it can be performed in a pre-coated plate in less than 2 hours; Specificity; Robustness, it can be carried out on samples which have not been kept under ideal conditions and where no viable virus is present; Simplicity.

**2.9.2.5. cDNA probes:**

For the differentiation between PPR and RP, the use of [P³²]-labelled cDNA probes derived from the N-protein gene of the two viruses had been described. It could differentiate between the two viruses without need for virus isolation. cDNA directed against the matrix protein, fusion protein and phosphorprotein gene were found to cross hybridize to a much greater extent and were not suitable for use as discriminating probes (**Diallo *et al*., 1989**).

Probes using non radioactive labels such as biotin (**Pandey *et al*., 1992**) or dioxin (**Diallo *et al***., **1995**) were developed. The biotin labeled cDNA was found to be as specific as the one using the radioactive label and more rapid in differentiation between PPR and RP (**Pandey *et* *al*., 1992**). However, it was reported elsewhere, that the expected sensitivity had never been obtained using non-radioactive labels (**Diallo *et al*., 1995**).

**2.9.2.6. Reverse transcription polymerase chain reaction (RT-PCR)**

**Saiki *et al.* (1988**) first demonstrated the efficiency of amplifying *in vitro* a selected sequence flanked by two oligonucleotide primers of opposite orientation. The method consists of repetitive cycles of DNA denaturation, primer annealing and extension by a DNA polymerase effectively doubling the target with each cycle leading, theoretically, to an exponential rise in DNA product. The efficiency achieved actually can vary enormously, however, since it is dependent on factors such as the number of cycles, the quantity of the starting material, the length of the target DNA, the temperature conditions of annealing and priming, and the polymerase used. When the starting material is DNA, high purification of the nucleic acid is not necessary so the procedure is greatly simplified.

Since the genome of all Morbilliviruses consists of a single strand of RNA, it must be first copied into DNA, using reverse transcriptase, in a two-step reaction known as reverse transcription/polymerase chain reaction (RT-PCR). RT-PCR has been shown to be useful for the rapid detection of Morbillivirus-specific RNA in samples submitted for laboratory diagnosis (**Shaila *et al*., 1996**). It has proved especially useful in identifying the new Morbilliviruses found in marine mammals (**Barrett *et al*., 1993**). Both genus-specific and universal Morbillivirus primer sets have been produced that can be used to distinguish all known Morbilliviruses (**Forsyth and Barrett, 1995**).

Two sets of primers have been made, based on sequences in the 3© end of N genes (messenger sense), which are least conserved regions between the two viruses. They enable specific amplification of 300 base pair (bp) fragments for RPV and PPRV (**Couacy-Hymann *et al***., **2002**). Reverse transcription-polymerase chain reaction tests (RT-PCR) using phosphoprotein (P) universal primer and fusion (F) protein gene specific primer sets to detect and differentiate between PPR and RP were described (**Barrett *et al*., 1993**; **Forsyth and Barret, 1995**; **Couacy- Hymann *et al*., 2002).**

**2.9.2.7 Serology**

Many tests have been used for the demonstration of PPR antibodies in serum: virus neutralization test, agar gel diffusion test, immunoelectrophoresis and recently blocking and competitive ELISA.

**2.9.2.8. Virus neutralization**

The virus neutralization test (VNT) is sensitive and specific, but time-consuming and expensive. The standard neutralization test is carried out in roller-tube cultures of primary lamb kidney cells or Vero cells when primary cells are not available. VNT is the most reliable test for detection of Morbillivirus antibodies (**Rossitter, 1994**). Serum against either PPR or RP may neutralize both viruses, but would neutralize the homologous virus at a higher titre than the heterologous virus. Therefore for differentiation purpose reciprocal cross neutralization is used (**Taylor and Abegunde, 1979**)

**2.9.2.9. cELISA**

Competitive and blocking ELISA based on monoclonal antibodies specific for N-protein (**Libeau** ***et al*., 1995**) and H-protein (**Anderson and Mckay, 1994**; **Saliki *et al*., 1993**; **Singh *et al*., 2004**) were developed for detection of antibodies in animal sera. These tests either used gradient purified virus or expressed antigens. In the N-protein cELISA, the serum antibodies and the MAb compete on specific epitope on nucleoprotein obtained from recombinant baculovirus. Though no cross reaction in N-protein cELISA was reported, a high level of competition up to 45% was observed among the negative (**Libeau *et al*., 1995**). Despite the fact that neutralizing antibodies are not directed against the N-protein, but the H-protein (**Diallo *et al*., 1995**), a correlation of 0.94 between VNT and cELISA was observed suggesting that the former was more sensitive (**Libeau *et al*., 1995**).

The relative sensitivity of this cELISA to VNT was 94.5, while the specificity was 99.4%. Both blocking ELISA and cELISA detecting anti-H antibodies are based on competition between an anti-H monoclonal antibody (MAb) and serum antibodies, but in case of blocking ELISA the test sera are pre-incubated with antigen and then incubated with the MAb. The sensitivity and specificity of the H-blocking ELISA were found to be 90.4% and 98.9% respectively (**Saliki *et al*.,** **1993**). PPR cELISA using MAb directed against the H-protein cross reacted to some extent with rinderpest, while RP cELISA is specific, therefore an animal was assumed to have experienced RP if it is positive in both PPR and RP ELISA (**Anderson and McKay, 1994)**. The overall specificity of c-ELISA test was 98.4% with a sensitivity of 92.2% when compared with VNT. The diagnostic efficacy of the assay in terms of sensitivity and specificity was calculated using two-sided contingency table (**Singh *et al*., 2004**).

**2.9.2.10 Postmortem diagnosis**

Common post-mortem findings included congestion, red hepatisation, raised patches of emphysema in the lungs, haemorrhages and froth exudates in the trachea, severe enteritis and streaks of haemorrhages in the intestine, enlargement and petechial haemorrhages in the spleen and oedema and inflammatory lesions in the mesenteric lymph nodes. Spectacular histopathological changes were observed in the lungs, intestine, spleen, mesenteric lymph nodes, liver and kidneys.(**Chauhan** ***et al.,*2009*).***

**2.9.2.11 Differential Diagnosis:**

The disease must be differentially diagnosed from Foot and Mouth disease, Bluetongue, Contagious ecthyma, Pasteurellosis, Contagious caprine pleuropneumonia, Nirobi sheep disease, Coccidiosis, Plant and Mineral poisoning etc. (**Appel *et al*., 1981**).

Confirmation requires virus isolation and cross-neutralization.

* **Foot-and-mouth disease (FMD):** This condition is comparatively mild, and the most characteristic clinical sign, lameness, is not a feature of PPR.
* **Rinderpest:** Cattle is mostly susceptible in this case. Now rinderpest is eradicated in our country.Clinical sign of Rinderpest is similar to PPR.
* **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 (CCPP)**: There is no digestive system involvement, and the clinical signs and lesions are confined to the respiratory system and pericardium.
* **Bluetongue:** Swelling of the lips, muzzle, and oral mucosa, together with edema of the head region, should serve to differentiate bluetongue from PPR. Coronitis, common in bluetongue, is not a feature of PPR. Also, sheep are more affected than goats.
* **Contagious ecthyma (Contagious Pustular Dermatitis, orf)**: The orf virus causes proliferative, not necrotic lesions that involve the lips rather than the whole oral cavity. The absence of nasal discharges and diarrhea also distinguish orf from PPR.
* **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.
* **Coccidiosis:** There is no upper digestive tract and respiratory system involvement.

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

**2.11. Treatment and prevention**

 Sulpher durg, oral saline should be used to protect the secondary bacterial infection and to maintain the reduction of dehydration (**Richards and Adams, 1982**)**.**

The affected animals were given antibiotics to control secondary bacterial infections along with anti-inflammatory drugs. Specifically, oxytetracycline and chlortetracycline are recommended to prevent secondary pulmonary infections (OIE, 2000). Many animals which received proper treatment in early stages of the disease were saved. It was observed that higher fatality rates in PPR affected animals was more due to secondary bacterial infections than the disease itself which could have been due to immune suppression associated with Morbillivirus infections **(Heaney *et al.,* 2002**). Earlier the rinder pest vaccine has been used to PPR. At present, homologous PPR vaccine has been used against PPR to make up strong immunity. Genetically engineered recombinant vaccine has been under gone limited field trials and still and attenuated cell culture adapted rinder pest virus vaccine provides protection against PPR for at least four years (**OIE, 2002).**

 According to **Sil, *et. Al*( 2001** )- an avirulent escape mutant PPR vaccine used for goat vaccination and provide immunity for 1 year. In Bangladesh, PPR vaccine (an avirulent escapes mutant PPR vaccine) used for goat vaccination against PPR @ 1ml s/c injection at 3 months of age and at one year interval as booster dose (**Sil, et. al., 2001**).