**CHAPTER-I**

**INTRODUCTION**

Goat is one of the major constituents of livestock in Bangladesh which is known as “poor man’s cow”. The contribution of goat to boost up the rural economy of Bangladesh is very recognized than any other sector of livestock. Goats are reared by farmers mostly as a subsidiary occupation or by poor people in Bangladesh. It is more a way of life rather than a commercial enterprise and it provide a substantial part of farmer's income. Goat meat and skin ranked 38% and 28% respectively, of the total meat and skin produced from livestock in Bangladesh (FAO, 1997). There are many disease of goat prevailed in Bangladesh that cause a potential loss of farmer, of them, Peste des petits ruminants (PPR) is very common and serious exotic killer disease (Debnath, 1995).PPR virus was first isolated by Gilbert and Monnier in 1962 in Senegal (Sil, 2000).The disease is highly contagious causing varying degree of morbidity and mortality in susceptible animals (Radostits *et al*., 2000). It is characterized by high fever, ocular and nasal discharge, pneumonia, necrosis and ulceration of the mucous membrane and inflammation of gastro-intestinal tract leading to severe diarrhea (Gibbs *et al*.,1979). Morbidity and mortality rates can be as high as 100 and 90 per cent, respectively (Abu-Elzien *et al*., 1990).The causative agent of this economically important disease of small ruminants is a Morbillivirus, the Peste des Petits Ruminants Virus (PPRV), under the family Paramyxoviridae of order Mononegavirales (Murphy *et al*., 1999). In Bangladesh, the PPR virus was identified during a severe outbreak in 1993 (Sil *et al*., 1995) Which was further confirmed by World Reference Laboratory and found that the virus has a close relation with Indian isolates (West Bengal) of PPR virus at a cluster with Asian group (Barrett *et al*., 1997).PPR in goat has been recorded in 1993 from the border belt areas of south western districts of Bangladesh and then spreads like epidemics fashion throughout the country. It has been reported that the Black Bengal goats were more susceptible (67.24%) to PPR than Jamunapari breed(32.76%). Morbidity varies from 40-95% and mortality as high as 80-85% (Samad, 2001). Kids over 4 months and under 1 year of age are at higher risk and cause huge economic loss (Venkataramanan *et al*., 2005).Young kids of below one year are much more susceptible than adult one (Radostits *et al*., 1995).

Both male and female goats are equally susceptible to PPR (Samad, 2001).Vaccination reduces Peste des petits ruminants outbreak in goats (Taylor *et al*., 1990 and Majiyabe *et al*., 1994).The outbreak of this disease causes a heavy economic loss in Bangladesh. Currently Bangladesh Government has taken a national scheme of poverty alleviation through goat rearing. The total Program will be null and void if the said disease does not control in Bangladesh.

Considering all those aspects, the present studywas undertaken with following objectives:

1. To determine the prevalence of PPR in goat in study area.
2. To get a real scenario of clinical signs and symptoms in PPR infected goat.
3. To predict the association among different factors like deworming, vaccination, other disease and previous antimicrobial therapy in the magnitude of occurrence of PPR.

**CHAPTER-II**

**REVIEW OF LITERATURE**

**2.1 History**

PPR virus was first isolated by Gilbert and Monnier in 1962 in Senegal (Sil, 2000). It has been recognized in many of the sub-saharian countries that lie between the Atlantic Ocean and the Red Sea (Lefevre and Diallo, 1990).PPR is present in nearly all Middle Eastern countries up to Turkey (Lefevre *et al*., 1991 and Ozkul *et al*., 2002). It is also widespread in India and southwest Asia 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.(Shaila *et al*., 1989).PPR occurs in most African countries, the Middle East and the Indian subcontinent (Abraham *et al*., 2005; EI-Hag and Taylor, 1984 and Lefevre *et al*., 1991).Outbreaks of PPR are now known to be common in India, Nepal, Bangladesh, Pakistan and Afghanistan (Abdollahpour *et al.,*2006). Later it spread in Bangladesh at 1993 (Sil, 1995 and Debnath, 1995).

**2.2 PPR: The disease**

Peste des petits ruminants (PPR) is an acute viral disease of small ruminants caused by a Morbillivirus and characterized by fever, oculonasal discharges, stomatitis, diarrhea and pneumonia. The disease is one of the major threats to about 200 million small ruminant population of the country. The natural disease affects mainly goats and sheep, but it is usually more severe in goats where, it causes severe morbidity and mortality and is only occasionally severe in sheep (Raghavendra *et al*., 2000). Although, Mornet *et al*. (1956) reproduced the disease followed by death in calves experimentally infected with PPRV-infected tissue, no natural outbreak has been reported in cattle. It is generally admitted that cattle can only be infected subclinically. However, it is possible that cattle in poor health may develop lesions following PPRV infection, clinical signs of which would be ascribed to Rinderpest. Nonetheless, PPRV was isolated from an outbreak of Rinderpest like disease in Indian buffaloes in 1995 (Govindarajan *et al*., 1997).

PPRV was also suspected to be involved in the epizootic disease that affected single humped camels in Ethiopia in 1995–1996 (Roger *et al*., 2000 and Roger *et al*., 2001). PPRV antigen and PPRV nucleic acid were detected in some pathological samples collected during that outbreak, but no live virus was isolated. Both experimental and natural infections of PPRV have been reported in wild ruminants (Hamdy and Dardiri, 1976; Furley *et al*., 1987). Experimentally, subclinical infection in pigs has also been demonstrated (Nawathe and Taylor, 1979).

The clinical disease resembles Rinderpest in ruminants, which is acute, and after an incubation period of 3-6 days, the clinical symptoms become apparent, which include high rise of temperature, oral and ocular discharges, necrotic stomatitis, severe pneumonia, dyspnoea, coughing, enteritis, severe diarrhoea followed by death (Roeder and Obi, 1999 and Pawaiya *et al*., 2004). The disease is highly contagious with morbidity and mortality rates reaching as high as 100 per cent and 90 per cent, respectively (Abu-Elzein, 1990). The mortality is usually low in endemic areas, but when associated with other diseases such as capripox, it can approach 100 per cent (Kitching, 1988). 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 feces of the infected animals (Taylor, 1984)

**2.3 Biological property of the virus**

The etiological agent of this disease of small ruminants is a Morbiilivirus, the Peste des Petits Ruminants Virus (PPRV), under the family Paramyxoviridae of order Mononegavirales (Murphy *et al*., 1999). The virus is composed of 15, 948 nucleotides,which is encoded with six structural protein, e.g. Necleocapside (N), Matrix (M), Fusion (F), Haemagglutinin (H), Polymerase (P), The PPR virus identified in Bangladesh is under the lineage 4 of PPR phylogenetic tree based on the N gene analysis (Sil, 2000 and Barrett *et al*., 1993).

The ultrastructure of PPR virus has been examined electron microscopically by negative staining technique. The virus particle was found to be pleomorphic with a diameter of intact particles varying between 130-390 nm. The virus has an envelope of 8-15 nm thickness with spikes of 8.5-14.5 nm length. The herring bone like ribonucleoprotien strands measure approximately 14-23 nm in thickness (Durojaiye *et al*., 1985).

Genome of PPR virus is non segmented single stranded RNA of negative polarity. The genome of attenuated vaccine strain of PPRV (Nigeria 75/1) has entirely been sequenced and the physical map of the genome is the same as that of the other morbilliviruses (Rima *et al*.,1986 and Diallo *et al*., 1989).

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 in to four distinct lineages. Lineage 1 and 2 are found exclusively in West Africa, whereas lineage 3 has been found in eastern Africa and Arabia. The fourth lineage is confined exclusively in the Middle East Arabia and Indian subcontinent (Shaila *et al*., 1996). Except one isolate (TN92/1) from southern India, which belonged to lineage 3, all Indian PPRV isolates identified so far belonged to lineage 4 only (Nanda *et al*., 1996 and Dhar *et al*., 2002).

**2.4 Geographical distribution of PPR**

After development of specific diagnostic tools in late 1980s onwards, our understanding of the geographical distribution of PPR has grown very quickly (Diallo *et al*., 1995) and recent data indicates the activity of PPRV in all countries of Africa lying between Sahara and the Equator, Arabian peninsula and the Middle East with extension to Turkey, Pakistan, India, Bangladesh and Nepal (Shaila *et al*., 1996; Dhar *et al*., 2002 and Taylor *et al*., 2001). It has also been reported in Sudan (Ali and Taylor, 1984), Kenya, Uganda (Wamwayi *et al*., 1995) and also in Ethiopia (Roeder *et al*., 1994).

In India, PPR was first reported in 1987 from Tamil Nadu (Shaila *et al*., 1989). The disease was restricted in southern part of the country (Shaila *et al*.,1990 and Krishna *et al*., 2001) until 1994, when a series of PPR outbreaks were reported from many northern states such as Himachal Pradesh, Rajasthan, Uttar Pradesh as well as from West Bengal (Nanda *et al*., 1996). A serious outbreak of PPR in goats has been recorded in 1993 from the border belt areas of southwest districts of Bangladesh and then spreads like epidemic throughout and the country (Sil *et al*., 1995).

**2.5 Prevalence of PPR**

Black Bengal goats were more susceptible to PPR(Samad, 2000). 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). The prevalence of PPR are also depend on the variation of the season. The prevalence of PPR is highest in rainy season (Bourdin, 1983).An outbreak occurred in a small flock of goats in july 2007,in the rainy season, out of 37 goats 19(51%) developed clinical disease and 5 (13.5%) died in Mymensingh, Bangladesh (Rahman *et al*., 2011).

A study is held with the clinical and epidemiological features of PPR among Sokoto Red goats in Zaria (dry Savanna). It is observed a pattern similar to that reported among West African dwarf goats in the humid zone of Nigeria. Between 25 and 88 per cent of goats were infected, but the clinical disease was rare (0.7%); 23-56 per cent of the clinically affected goats died. It is concluded, the disease was most common at the end of the dry season and the early rainy season, perhaps providing stress situations (Ezeokoli *et al.,*1986).Outbreaks of PPR occurred in Oman in 1985 and 1986. In 1985, 732 of 2129 goats were affected and 262 (12.3%) died. In 1986, 206 of 597 goats were sick and 35 (6.1%) died. The disease was diagnosed by agar gel immune diffusion assay (Ata and Al-Sumry, 1989).

First outbreak of PPR in India reported from Tamil Nadu in a flock of 800 sheep during 1987. During the outbreak, 80 animals were affected of which, 20 died. PPR was confirmed by using cDNA probes specific to nucleoprotein gene of the PPR virus. Based on cross neutralization test (Shaila *et al*., 1989).In April 1988, an Outbreak occurred of PPR affecting 90 per cent of indigenous goats in Al-Ahsa oasis in eastern Saudi Arabia. The case mortality rate was 70 per cent (Abu-Elzein *et al*., 1990). In January 1987 in Embaba Giza province of Egypt an outbreak of PPR occurredin a flock of 70 goats (32% mortality) and among Ovisornata in a zoo in the same province (Ismail and House, 1990).A survey conducted in West Bengal and it shows mortality rate due to PPRV in sheep and goat was 15.2 and 38.1 per cent respectively.

Based on clinical feature.(Bhowmick1995). An outbreak of PPR occurred that caused death of 33 of 98 Jamunapari goats in an organized farm in West Bengal (Chandra et al., 1995).In Maharashtra, 13.4 and 41.4 per cent mortality due to PPR was observed in goats and kids, respectively, in nine villages (Kulkarni *et al*., 1996).The agent responsible for an outbreak of a rinderpest-like disease afflicting sheep and goats in three states of northern India was confirmed as PPR virus using ELISA, RT/PCR, immunofluorescence with virus specific monoclonal antibodies and virus isolation (Nanda *et al*., 1996).An outbreak of PPR was recorded in 1998, in an organized sheep farm in Cuddapah district of Andhra Pradesh, India with morbidity, mortality and case fatality rates as 30.56, 13.20 and 43.20 per cent, respectively (Sreeramalu, 2000). An outbreak of PPR occurred from an organized sheep farm Chittoor district of Tamil Nadu, India with morbidity and mortality rates of 16 and 20 per cent, respectively (Rao *et al*., 2001)An outbreak of PPR occurred with 100 per cent mortality during 1995 in a group of 23 goats in Bangladesh (Islam *et al*., 2001).

**2.6 Hosts**

Natural disease affects mainly goats and sheep, but it is usually more severe in goats where it causes heavy losses and is only occasionally severe in sheep Black Bengal goats are more susceptible (67.24 %) to PPR than Jamunapari breed (32.76 %) (Shaila *et al*., 1989).Cattle, buffaloes, camels, and pigs are also susceptible to infection but do not exhibit clinical signs and are unable to transmit the disease to other animals (Ozkul, 2002 and Lefevre and Diallo, 1990) Susceptibility to infection rises with age. However, the disease is rapidly fatal in the young animals (60.87 %) especially at 7-12 months of age (Blood *et al*., 1995). PPR is a major constrain in the development of goat industry due to high morbidity (50-90%) and case fatality (50- 85%) rates. Kids over 4 months and under 1 year of age are at highest risk and cause huge economic loss per year (Venkataramanan *et al*., 2005).

**2.7 Association between PPR and breed of goat**

There are a number of goat breeds such as Black bangle, jamunapari etc. It was said that Black Bengal goats were more susceptible to PPR than Jamunapari (Samad, 2000). Highest mortality rate (65%) in Barbari-Black Bengal cross bred goats in Regional Goat Breeding Farm in Tripura (Sil, 2000).

**2.8 Association between PPR and age of goat**

Young kids of below one year are much more susceptible than adult one. The maximum proportionate of PPR was encountered 37.5% at the category of 7 to 12 month subacute manner (Radostits *et al*, 1995). The young goat may die due to anoxia. Kids over 4 months and under 1 year of age are at higher risk and cause huge economic loss (Venkataramanam *et al*, 2005).

**2.9 Association between PPR and sex of goat**

There is no detail finding about the PPR infection of goat in different sex.Both male and female goats are equally susceptible to PPR (Samad, 2001).

**2.10 Association between PPR and vaccination in goat**

Vaccination contributes to lowering Peste des petitsruminans outbreak in goats (Taylor *et al*., 1990 and Majiyabe *et al*., 1994).

**2.11 Association among PPR, previous disease exposure anddeworming history in goat**

Poor nutritional status, stress of movement, and concurrent parasitic and bacterial infections enhance the severity of clinical signs of PPR disease (Rautmare, 2010). Parasite reduce immunity by causing malnutrition in animal because some gastrointestinal parasites serious blood suckers (Githigia *et al*., 2004). Parasite cause malnutrition so any viral disease may cause severity (Al-Quaisy, *et al*., 1985).From these observations it can be said that history of previous disease exposure and the history of no deworming enhance the PPR disease susceptibility

**2.12 Pathogenicity**

Case fatality rate is higher in goats (55-85%) (Opasina *et al*., 1985). The high morbidity (100%) and mortality (50-90%) rates in goats caused by PPR have been described inBangladesh, followed by evaluation of ELISA as field diagnostic method and inactivated vaccine to control this disease (Sil *et al*., 2000-2001).when a susceptible population builds up, periodic epizootics (outbreak) occurs, some of which might lead to almost 100% mortality among affected goat and sheep at risk(Taylor, 1984., Lefevre and Diallo, 1990).

**2.13 Transmission**

The discharges from eyes, nose, and mouth as well as the loose feces contain large amount of the virus. Fine infective droplets are released into the air from this secretions and excretions, particularly when affected animals cough and sneeze. Other animal inhale the droplet and are likely to become infected. Although close contact is the most important way of transmitting the disease it is suspected that infectious materials can also contaminate water and feed trough and bedding, turning them into additional source of infection. (Roeder, P.L. and Obi, T.U. 1999).Newly purchased animal from market and wild animal have suspected to play a role spreading of disease (Fraser, 1986).

**2.14 Pathology**

PPR virus causes epithelial necrosis of the mucosa of the alimentary and respiratory tracts marked by the presence of eosinophilic, intracytoplasmic and intranuclear inclusion bodies. In the spleen, tonsil and lymph nodes, the virus causes necrosis of lymphocytes evidenced by pyknotic nuclei and karyorrhexis (Rowland *et al*, 1969). PPR virus, like other morbilliviruses, is lymphotropic and epithelio tropic (Scott, 1981).The alveoli were altered with the proliferation of type ΙΙ pneumocytes, multinucleated giants cell with eosinophilic intracytoplasmic inclusion body. In PPR infection lungs were become highly consolidated in apical lobe, echynyotic and brush paint haemorrage were found in epicardium (Islam, *et al*., 2001). Zebra stripes are formed by haemorrage in the fold of rectum. Lymphocytes percentage declines (up to 21 %) with onset of duration of diseases increases at the same time neutrophilia (up to 72 %) occur (Sil, 2000).

**2.15 Clinical signs**

The clinical disease resembles Rinderpest in ruminants, which is acute, and after an incubation period of 3-6 days, the clinical symptoms become apparent, which include high rise of temperature, oral and ocular discharges, necrotic stomatitis, severe pneumonia, dyspnoea, coughing, enteritis, severe diarrhoea followed by death (Roeder and Obi, 1999 and Pawaiya et al., 2004). Just after showing the clinical sign the dehydration is not remarkably. After few days of diarrhea the animal become severely dehydrated which lead to animal die (Rodestits *et al*., 2000). Susceptibility to infection rises with age; however, the disease is rapidly fatal in the young animals (Ozkul, 2002).Incubation period of PPR disease is 2-10 days, commonly 4-5 days.(Barrett, 1994). After incubation period patients develop sudden high fever (40'C-41'C), remaining high for 5-8 days, will return to normal before recovery or drop below normal before death.(Sil, 2000).After infection, the clinical signs are as Serous nasal discharge, becoming muco purulent can crust over and occlude nostrils. Purulent ocular discharge with congested conjunctiva and bronchopneumonia generally found. (Lefevre and Diallo, 1990 and Rowland *et al*., 1969).

**2.16 Control of outbreak**

Control of PPR outbreaks relies on movement control (Quarantine) combined with the use of focused (ring) vaccination and prophylactic immunization in high risk populations. Until recently the most practical vaccination against PPR made use of tissue culture rinderpest vaccine. Recently a homologous PPR vaccine has been developed. This vaccine of choice is becoming increasingly available. The vaccine can protect small ruminant against PPR for atleast three years (Roeder, P.L. and Obi, T.U. 1999).The quarantine, slaughter, proper disposal of carcasses and avoiding contact fomites, decontamination mination of facilities and equipment, restrictions on importation of sheep and goats from infected areas (Nanda et al., 1996)

**2.17 Treatment and vaccination**

Recently a homologous PPR vaccine has been developed.This vaccine of choice is becoming increasingly available. The vaccine can protect small ruminant against PPR for atleast three years (Roeder, P.L. and Obi, T.U. 1999).PPR being an exotic disease in Bangladesh, there is a very little knowledge about its epidemic nature, diagnostic techniques and control strategy. A control programme using locally produced tissue culture live attenuated rinderpest vaccine, as well as imported rinderpest vaccine were adapted against PPR in this country, but failed (Sil *et al*., 2000-2001). The efficacy of the thermo stable PPR vaccine incubated at room temperature for 14 days. Hyper immune serum can be used successfully along with long acting antibiotic to limit the the spread of virus and recover those animals which are under incubation and in early stage of infection. Good nursing, symptomatictreatment with broad spectrum antibiotic, sulphur drugscan safe life of sick animal and also can improve the immunosuppressive condition of the affected goat (Sil, 2000).

**CHAPTER-III**

**MATERIALS AND METHODS**

**3.1 Study area and population**

The study was conducted from 16 July 2012 to 6 September 2012, at the Upazilla Veterinary Hospital, Sadar, Dinajpur. The study was conducted on natural PPR infected goats of various age, sex and breed that were brought to the Hospital over the study period. A total of 130 cases were recorded in internship period in Upazilla Veterinary Hospital.

**3.2 Data collection**

All the required data were collected by direct interviewing with the animal’s owner, clinical history and by examining clinical signs of enrolled goat. Apre-tested questionnaire was filled during the examination, containing various types of information regarding demographic (age, sex, breed) characteristics, previous disease exposure history and any preventive measures taken by them.

**3.3 Clinical diagnostic procedure**

A tentative diagnosis was performed on the basis of clinical history and clinical examination of animal. PPR consists of 5 phase of infection cycle as incubation period, prodermal phage, erosive phage, pneumonic phage, diarrhea and death. (BLRI, 1999). Depending upon the early stage and later stage of clinical signs and symptoms the animals were divided into two groups namely, prodermal-erosive phage and pneumonic-diarrhea phase as early and later stage of infection respectively. Several clinical signs were observed related to PPR in goat including high fever (106-107°F), mild conjunctivitis, congestion of the third eye lids, oculonasal discharge (Figure 2)erosive lesions on the inner side of the upper lip (Figure 1), diarrhea and dehydration (Figure 3) (Lefevre and Diallo, 1990).

|  |
| --- |
| Figure 1. Erosion in lips and buccal mucosa |
| Figure 2. Severe diarrhea |
| Figure 3. Purulent nasal discharge |

**3.3.1 Anamnesis**

History of the cases were taken from the owner and carefully recorded in case sheet individually.

**3.3.2 Clinical examination**

The following clinical examinations were done carefully and the findings were recorded.

**Distance inspection**

The posture and gait of each animal was inspected by careful observation from distance.

**Close inspection**

Close inspections were performed properly in order to observe the presenting signs such as a sharp rise of temperature of 104°F – 106°F, oculonasal discharge, diarrhea, respiratory distress and any other significant clinical signs.

**Temperature**

Per rectal temperature was recorded with the clinical thermometer.

**Indirect auscultation**

Normal and abnormal sound of respiratory and cardiovascular system were observed initially by direct hearing and then with the help of stethoscope.

**Dehydration test**

On the basis of skinfold test three types of dehydration were examined namely, Severe, Moderate and Slight.

**3.4 Data analysis**

All the collected data were entered into an excel spread sheet (Microsoft office excel 2010) and analyzed by using SPSS statistical software (**Ver.13 for windows, SPSS)**. Chi-square (χ2) test was used to examine the variation and any significant difference of prevalence in different groups of animal.

**CHAPTER-IV**

**RESULTS AND DISCUSSION**

The total goat population in the Dinajpur district is 57374 (BBS, 2008), of the130 goat examined, 82 were found to be clinically PPR affected. The overall prevalence of PPR in goats was found to be 63%. Similar prevalence (51%) of PPR in another region in the country was reported by Rahman *et al*., (2011). Conversely, the finding of this study was lower than the prevalence of 90% reported in Al-Ahsa oasis in eastern Saudi Arabia in 1988. The case mortality rate was 70 per cent (Abu-Elzein *et al*., 1990). It is difficult to draw any conclusions becauseof the differences in sampling procedures in the different studies that affect their representativeness (Ozkul *et al*., 2002).

**Table 4.1 Prevalence of PPR in goat in different breed**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Breed | N | No. positive | % Prevalence (95% CI) | χ2-value | P-value |
| Black Bengal | 89 | 65 | 73 (63-82) | 12.947 | 0.0015 |
| Jamunapari | 28 | 13 | 46 (28-66) |
| Cross breed | 13 | 4 | 31 (9-61) |
| Total | 130 | 82 | 63 (54-71) |  |  |

N= Number of animal examined; CI= Confidence Interval

The prevalence was higher (73%) in Black Bengal goat amongthe three type of breed included in this study. Asignificant (p<0.01) variation of the prevalence of PPR in breed susceptibility was observed. Shaila, *et al*., (1989) reported 67.24% and 32.76% prevalence in Black Bengal and Jamunapari goat respectively which is similar to the present study (Table 4.1). The prevalence of PPR according to sex is mentioned in Table 4.2. The female goat showing higher rate of prevalence (65%) compared to male (60%). There was no significant difference (P>0.05) in the prevalence between male and female goat. Similar result was described by Samad (2001) who stated that both male and female goats are equally susceptible to PPR.

**Table 4.2 Prevalence of PPR in goat in different sex**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Sex | N | No. positive | % Prevalence (95% CI) | χ2-value | P-value |
| Male | 45 | 27 | 60 (44-74) | 0.114 | 0.735 |
| Female | 84 | 55 | 65 (54-76) |
| Total | 130 | 82 | 63 (54-71) |  |  |

N= Number of animal examined; CI= Confidence Interval

The prevalence among different age group of animal are demonstrated in Table 4.3. Among the 56 adult (more than 12 months), 66 young (4 to 12 months) and 8 kid (Less than 4 months) the prevalence was 62%, 67% and 38% respectively.

PPR is rapidly fatal in the young animals (60.87 %) especially at 7-12 months of age (Blood et al., 1995). This study shows that the young goats which are 4 to 12 months of age are more susceptible to PPR and the prevalence percentage is 67, it is somewhat lower than the previous study. Some study reported that, young kids of below one year are much more susceptible than adult one. The maximum proportionate of PPR was encountered 37.5% at the category of 7 to 12 month subacute manner (Radostits *et al*., 1995). Kids over 4 months and under 1 year of age are at higher risk and cause huge economic loss (Venkataramanan *et al*., 2005). The observation of Venkataramanan *et al*., (2005) also agrees with this study.The Table 4.4 specifies the number of dewormed animal in relation to prevalence of PPR. Among 46 numbers of goats which have deworming history, 31 were PPR affected and on the other hand, among 84 numbers of goats which does not have deworming history, 51 were PPR affected.

**Table 4.3 Prevalence of PPR in goat among different age groups**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Age group | N | No. positive | % Prevalence (95% CI) | χ2-value | P-value |
| Adult (more than 12 month) | 56 | 35 | 62 (49-75) | 2.62 | 0.27 |
| Young (4 to 12 month) | 66 | 44 | 67 (54-78) |
| Kid (less than 4 month) | 8 | 3 | 38 (13-69) |
| Total | 130 | 82 | 63 (54-71) |  |  |

N= Number of animal examined; CI= Confidence Interval

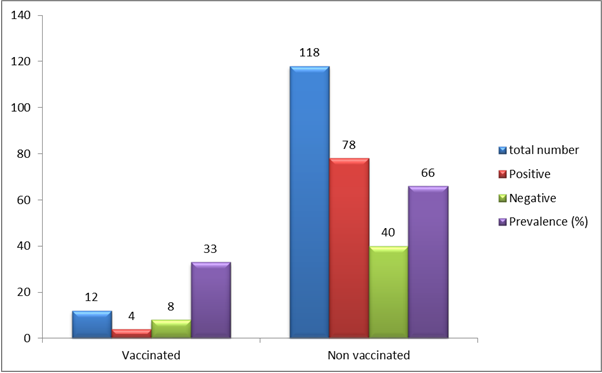
**Table 4.4 Association between Prevalence of PPR and deworming in goat**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Deworming history | N | No. positive | % Prevalence (95% CI) | χ2-value | P-value |
| Yes | 46 | 31 | 67 (53-79 ) | 0.3184 | 0.5726 |
| No | 84 | 51 | 61 (49-71) |
| Total | 130 | 82 | 63 (54-71) |  |  |

N= Number of animal examined; CI= Confidence Interval

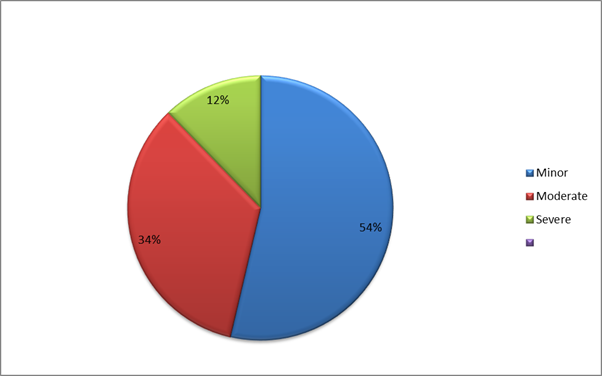
There is no significant association (P>0.05) between deworming and Prevalence of PPR. But the occurrence of infectious disease like PPR depends upon several management factors including Poor nutritional status, stress of movement, and concurrent parasitic and bacterial infections (Rautmare, 2010). Parasite reduces immunity by causing malnutrition in animal because some gastrointestinal parasites serious blood suckers (Githigia *et al*., 2001). Parasite cause malnutrition so any viral disease may cause severity (Al-Quaisy *et al*., 1985).

Impact of vaccination in the prevalence of PPR are illustrated in figure 4.1. The rate of vaccination against PPR among rural community is very poor. Among 130 cases, only 12 cases having the history of vaccination. The prevalence of PPR in the vaccinated group (33%) is much lower than that of non vaccinated group (66%). Similar result was described by Taylor *et al*., (1990) and Majiyabe *et al*., (1994). It is well documented that Vaccination contributes to lowering Peste des petitsruminants outbreak in goats (Taylor *et al*., 1990 and Majiyabe *et al*., 1994).



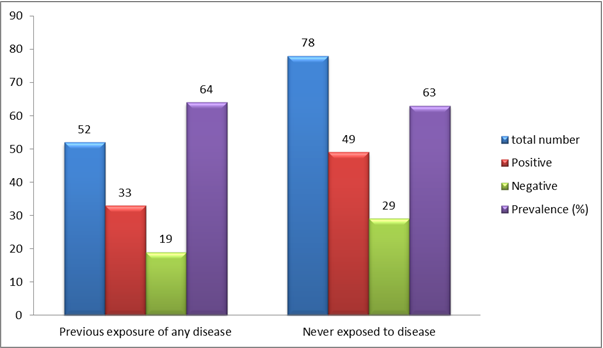
**Fig 4.1 History of vaccination and prevalence of PPR in goat**

The degree of dehydration among the PPR affected goats is described in figure 4.2. Three major types of dehydration were observed. Major percent (54%) of animal was grouped as minor dehydrated, and only 24% and 12% of animal was recorded as moderate and severely dehydrated respectively. In PPR affected goat the animal become severely dehydrated after few days of diarrhea which lead to animal die (Radostits *et al*., 2000). In this we found a maximum normal dehydrated animal because the maximum animals were hospitalized just after showing the clinical signs.

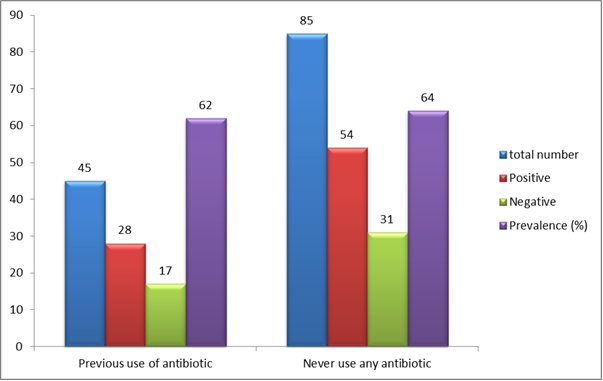


**Fig 4.2 Degree of dehydration among PPR affected goat**

The association between the prevalence of PPR and the previous disease exposure of goats are represented in figure 4.3. A total 52 cases found that having the history of previous disease expossure and 78 cases reported never expossed to disease. The prevalence of PPR in this two group of animal was 64% and 63% in disease exposed and never exposed group respetively. There was no significant (P>0.05) difference between this two group.



**Fig 4.3 Previous histories of disease exposure and Prevalence of PPR in goat**



**Fig 4.4 Previous use of antibiotic and prevalence of PPR in goat**

Poor nutritional status, stress of movement, and concurrent parasitic and bacterial infections enhance the severity of clinical signs of PPR disease (Rautmare, 2010). Parasite reduces immunity by causing malnutrition in animal because some gastrointestinal parasites serious blood suckers (Githigia *et al*., 2001). Parasite cause malnutrition so any viral disease may cause severity (Al-Quaisy *et al*., 1998).

Previous disease exposure either of bacterial, parasitic, metabolic or nutritional disease, it enhance the PPR susceptibility, and the above given result also agree with this observation, Previous disease exposure increase the PPR, may be as because previously occurred disease cause immunosuppression and enhance the viral disease susceptibility.

Among the 45 goats, 28 were affected by PPR in which antibiotics were used previously and the prevalence percentage was 62 (figure 4.4). Among the 85 goats, 54 were PPR positive in which antibiotics were never used before and the prevalence percentage was 64. Therefore it was found that the prevalence percentage of PPR was more in those goats which were not treated with any antibiotics before than those goats which were treated with antibiotics previously.

**CHAPTER-V**

**CONCLUSION**

PPR is highly contagious disease with higher mortality and morbidity in goat. In this study it is revealed that Black Bengal goat were more susceptible (73%) compared to others. According to age groups,4 to 12 months age of goats were highly infected as 67% in comparison with others. Here female goats were more susceptible to PPR infection than male.The goats which had previous disease exposure history were more susceptible to PPR than those goats which had no history of disease exposure. It was also observed that vaccination reduces the PPR disease susceptibility. Although against virus, there is no specific treatment, combined therapy containing antibiotic, antihistamines as well as fluid therapy noticed line of treatment in PPR infection of goat.

**CHAPTER-VI**

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