**CHAPTER-I**

**INTRODUCTION**

Goats are reared by farmers, mostly as a subsidiary occupation in Bangladesh. It is more a way of life rather than a commercial enterprise and goat herds provide 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. Ravages of this economic animals caused by diseases, act as one of the prime production limiting factors all over the world.Infectious diseases are significant impediments to the economical rearing of small ruminants (Radostits *et al*., 2000). Peste des petits ruminants (PPR) is an acute or sub-acute viral disease of small ruminants. Other names commonly used includes, pseudo rinderpest of small ruminants; pest of small ruminants; goat plague; pest of sheep and goat; stomatitis pneumoenteritis syndrome; contagious pustular stomatitis and pneumoenteritis complex highly contagious, infectious and fatal viral disease of domestic and small ruminants The disease is characterized by high fever, severe depression, Serous nasal and ocular discharges, matting is common around the eyes, and the nose may become obstructed, necrotic stomatitis, gastroenteritis and pneumonia and death ( Food security, 2008). Some animals may recover, but a dry, [stertorous](http://en.wikipedia.org/wiki/Stertorous) coughing often persists for some days (Berrada, 2008 ).  Besides coughing, there is intensive labial dermatitis with scab formation, resembling [orf](http://en.wikipedia.org/wiki/Orf_%28disease%29) (Handbook, 2009*)*.Abortions may occur ( [Peste des Petits Ruminants](http://en.wikivet.net/Peste_des_Petits_Ruminants), 2011).

Peste des petits ruminants (PPR), is an acute contagious disease caused by a Morbillivirus in the family Paramyxoviridae ( OIE, 2013 ). It is closely related to rinderpest virus, canine distemper virus, and human measles virus, which makes the PPR an important disease of small ruminants and has created tremendous problem due to its apparent similarity to rinderpest . It was first described in 1942 in Cote d'Ivoire West Africa in 1940 and virus was isolated in 1962 from Senegal. The morbidity and mortality rates can reach 100% however, these rates tend to be lower in endemic areas and the reported mortality rates in some individual flocks are as low as 20%. 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% (Food security, 2008). When associated with other diseases such as capripox, mortality can be 100% (By Dhar 2002). The disease is widely distributed in equatorial Africa, the Arabian peninsula, part to Indian subcontinent including Bangladesh. But since summer 2008, [Morocco](http://en.wikipedia.org/wiki/Morocco) is suffering a generalized outbreak with 133 known cases in 129 provinces, mostly affecting sheep ( [FAO](http://en.wikipedia.org/wiki/FAO), 2008 ). The outbreak has precipitated the vaccination of a large number of the 17 million sheep and five million goats in the country ( AFP, 2008 ). In Bangladesh, the PPR virus was identified during a severe outbreak in 1993 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. To neutralize this virus in the host body, a thermo stable PPR vaccine has been developed experimentally by scientists of BLRI and DLS (Chowdhury *et al*.,2004).

The incubation period is typically 4–6 days, but may range between 3 and 10 days( OIE, 2013). PPR occurs mainly in 3 forms-per acute , acute and subclinical . The outbreak of this disease cause heavy economic loss in Bangladesh. These small domestic animals are their important source of family income and are known as the **‘moving banks’** of shepherds. However, this incomes often reduced by various infectious diseases**,** including peste des petits ruminants. Hence, the estimation of economic implications is important not only for a description of the actual situation, but also for how much and to what extent the losses can be avoided and risk of disease can be diminished.

The virus of PPR is antigenically , biologically related to Rinderpest. Antigenically it is also related to Canine distemper and Measles virus (Kulkarni et al 1996). In Bangladesh the disease is mainly diagnosed on the basis of history from the owner and clinical presenting signs. But few limited attempts have been undertaken to diagnosed the disease based on both clinical signs and corresponding hematological changes. Therefore, the study was undertaken with the following objectives:

1. To diagnose the PPR disease on the basis of presenting clinical signs
2. To study the changes of hematological pictures of the affected goats.

**CHAPTER-II**

**REVIEW OF LITERATURE**

**2.1 History**:

The peste des petits ruminant (PPR) was first reported by Gargadennec and Lalanne (1942) in Ivory Coast of West Africa. It was first Isolated by Gilbert and Monnier in 1962 in Senegal (Sil, 2000). For a long time, its existence was associated with West African countries. 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; Taylor *et a*l.,2001). PPR is present in nearly all Middle Eastern countries up to Turkey (Lefevre *et al*., 1991 and Ozkul *et al*., 2002).

It has also been reported in Sudan (Ali and Taylor, 1984), Kenya, Uganda (Wamwayi *et al*., 1995) and also in Ethiopia (Roeder *et al.,* 2002). In India, PPR was first reported in 1987 from Vallipuram district of Tamil Nadu (Shaila *et al.,*1989). The disease was restricted in southern part of the country (Shaila *et al.*1990; Krishna *et al*., 2001) until 1994, when a series of PPR outbreaks were reported from many northern states such as Himachal Pradesh, Rajasthan, and Uttar Pradesh as well as from West Bengal (Nanda *et al*.,1996). Since then, the disease has been reported regularly from different parts of the country and is considered as an endemic disease causing a great loss to small ruminants of the country. 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). In 2007 China reported PPR for the first time. In 2008 an outbreak in Morocco was the first time the disease appeared in North Africa

**2.2 History of PPR outbreak in Bangladesh** :

In Bangladesh PPR was first detected in 1993, in the west part of the country (Islam *et al.,* 2001). An epidemic ensued which covered virtually the entire country, peaking in 1995 but lasting until 1998. Since then the disease has been occurring at low incidence, and is observed sporadically. A noticeable increase in the incidence of outbreaks of PPR was detected once again in the west of Bangladesh, in February 2001, in Meherpur district of Khulna division. By early March 2001 it had spread to Rajshahi district of Rajshahi division and then has been detected in Dhaka district of Dhaka division, where it is thought to have been introduced in the second week of March.2001. PPR is spreading rapidly causing high morbidity and mortality. The outbreaks of PPR caused 74.13% morbidity and 54.83% mortality in Black Bengal goats in Bangladesh (Islam *et al*., 2001 and Das *et al.,* 2007). Investigations are proceeding to establish the extent of the spread.

**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 Mononega virales(Murphy *et al.,* 1999). PPR virus is enveloped with helical pleomorphic shape containing single stranded non segmented RNA molecule. The genome of this virus is a single linear molecule of approximately 4.5 x106DA with 16,000 (15, 948) ribonucleotides which is encoded with six structural protein, the Nucleocapside (N), Matrix (M), Fusion (F), Haemagglutinin (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 and Sil, 2000).

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 morbilli viruses (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).

The virus has got tremendous affinity for epithelial cells of gastrointestinal tract and lymphoid tissues (Fraser, 1986 ). The virus can be cultivated in sheep and goat kidney cells where in tranuclear and intracytoplasmic inclusion bodies are evident ( losos, 1986 ).

**2.4 Geographical distribution of PPR :**

Described for the first time in Côte d’Ivoire (Gargadennec and Lalanne,1942), PPR was long considered to be confined to West Africa but later it was described throughout Africa south of the Sahara and north of the equator, as well as the Middle East and Asia. Recent field and laboratory data show that PPR is spreading, with recent incursions reported into China and Bhutan, and that it is moving fast towards southern and eastern Africa where it affects a wide belt of countries south of the equator, from Gabon to Somalia. In northern Africa, the PPR epizootic that occurred in Morocco in 2008 has extended the disease’s geographical distribution to the Mediterranean. This was the first episode of PPR to be reported in a Maghreb country.



**Fig : Geographical distribution of Pests des petits Ruminants**

The global spread of PPR is probably related to the progressive control and later, eradication, of rinderpest. The cessation of rinderpest vaccination campaigns and loss of antibody cross-protection between the two diseases means that small ruminants are now fully exposed to PPR.

Its spread has certainly also been encouraged by the growing population of small ruminants, with the virus colonising new areas as a result of animal movements during seasonal transhumance, people fleeing from socio-politically and climatically insecure areas and the intensification of trade associated with human population growth. These cross-border movements have a very significant impact on the spread of many infectious agents and pose increasing problems for the surveillance and control of animal, zoonotic and human diseases (Domenech *et al*., 2006).

There is now a resurgence of PPR in some areas, with a parallel incursion of new genotypes. Four phylogenetic lineages have been ( Peste des petits ruminants evolution between 2000 and early 2011 • 2 53 © 2011, Animal Health Information Department-OIE ) identified, three of which are established in Africa (lineages I to III) and lineage IV, in Asia (Shaila *et al.,* 1996; Kwiatek *et al*., 2007). Similarly, the introduction of PPR into Morocco in 2008, which was till now free from the disease, also involved lineage IV strains (Banyard *et al*., 2010). It has now been shown that camels are susceptible to the PPR virus (Roger et al., 2001) and that the clinical expression of the disease is emerging in this species (Khalafalla *et a*l., 2010). Recent observations in Sudan suggest that camels could be victim to PPR, as well as acting as long-distance vectors (Kwiatek *et al.,* 2011). However, the scale of this phenomenon needs to be evaluated, especially from an epidemiological standpoint, by comparing long distance movements of camels with the phylogeographic distribution of PPR virus strains.

## 2.5 Epidemiology:

## 2.5.1 Hosts :

PPR is primarily a disease of sheep and goat 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 ). There have been several reports of PPR occurring in other species, particularly in captive wild ungulates from three families: Gazellinae (dorcas gazelle) ,Caprinae (nubian ibexand laristan sheep) , Hippotraginae (gemsbok) . The American white-tailed deer (*Odocoileus virginianus*) has been infected experimentally ( Saliki 2002). 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 ( EMPRES 1999). PPR is not infectious to humans.

**2.5.2 Transmission :**

For PPR to spread, close contact between infected and susceptible animals is needed (Ozkul 2002). There are several means of transmission between animals (Saliki 2008):

* Inhalation of aerosols produced by sneezing and coughing of infected animals
* Outbreaks are more frequent during the rainy season or the dry, cold season ( OIE 2002).
* Direct contact with ocular, nasal, or oral secretions
* Direct contact with feces
* Fomites such as bedding, water, and feed troughs
* No carrier state is known to exist

Faeces are the main spreading agent and through it disease may occur in epidemic proportion (Shanthi *et al*, 1994). Newly purchased animal from market and wild animal have been suspected to play a role for spreading of disease (Radostits *et al*, 1994 and Fraser, 1986).

**2.5.3 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 in Bangladesh, 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).

**2.6Association of average DLC value between sexes of goats:**

The association between sex of PPR in goats was investigated (Table 2). The study revealed that the avg. DLC value of PPR in goats was higher (22.5%) in males than females (13.41%), which is in agreement with the findings of ( Rahman *et al*. 2004). The results showed that the avg. DLC value of PPR was significantly associated with the sexes of goats. Males are apparently more prone to the disease than females may be due to genetic factors.

**2.7 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).

**2.8Clinical signs:**

PPR consists of 5 phase of infection cycle as incubation period, prodermal phage, erosive phage, pneumonic phage, diarrhea and death. (BLRI, 1999).The course of the disease was acute and sub-acute, few of the animals died even in 36 hours of onset of the disease. The affected animals initially were severely depressed with a sudden rise in body temperature reaching almost 107.6°F in some cases, and the fever persisted for 7-8 days. From the onset of fever, most animals had a serous nasal discharge which progressively turned into mucopurulent discharge, leading to severe respiratory distress. Areas of erosions were most commonly seen on the visible nasal mucous membranes and muco-cutaneous junctions with inflammation around the mouth. In many of the animals, lesions similar to developed at mucocutaneous junction of mouth. The erosive and necrotic stomatitis started as areas of hyperemia at gums, cheeks, dental pad and / or anterior dorsal part of tongue with frothy salivation. PPR is a rinderpest-like contagion of goats and sheep characterized by erosive stomatitis, enteritis, pneumonia ( By Defra, 2005) . The areas later developed into irregular non-hemorrhagic lesions and in some of the cases circular raised but flat non-bleeding lesions were present on the tongue. There was a great amount of necrotic debris on the older lesions. The individuals with severe oral lesions had visible swelling around mouth. A. Non-hemorrhagic diarrhea was observed in all affected animals, developing 2-3 days after onset of the disease. Conjunctivitis was recorded with lachrymal discharge which became mucoid resulting in sticky eyelids. Abortion in pregnant animals was a consistent feature and mucous membranes vulva had erosive lesions very similar to that in intestinal mucosa. A subnormal temperature preceded death in animals with severe diarrhea for few days. Temperature dropped down in the later stages of diarrhea due to emaciation and dehydration (Chakrabarty 1997, Sil 2000).

**2.9 Pathology:**

Pathology of both natural and experimental goats revealed stomatitis, congested and/or consolidated pneumonic lungs, generalized enlargement of lymph nodes accompanied with necrosis and congestion of some lymph nodes, atrophied congested spleen and hemorrhagic gastroenteritis. Congestion of the urinary bladder, uterus and vagina in experimental goats and intestinal intussusception in dead goats of natural infection were also found. Histopathological study of both natural and experimental cases revealed congestion and edema of lungs in some cases but in other cases there were network of fibrin infiltrated with neutrophils, formation of syncytia, gaint cell and presence of pink color bacterial colony. There was infiltration of neutrophils and mononuclear cells within the alveoli, bronchioles, alveolar wall and interstitium of lungs. Lymphoid organs showed necrosis and depletion of lymphoid cell; congestion, morionuclear and neutrophilic infiltration in the lamina propria and submucosa of the abomasum, intestine, uterus and urinary bladder; loss of intestinal villi; congestion of cortical blood vessels and glomeruli of kidneys were recorded. Samples of both natural and experimental cases were confirmed as PPR by ELISA test. In this investigation, it was observed that clinical signs, gross and microscopic findings were more severe in experimental PPR infected cases than that of natural cases .( By khan *et al.* , 2005 , Bangladesh Journal of Veterinary Medicine , Vol. 3 , No. 2 ) .

**2.10Treatment and vaccination :**

There is no treatment for PPR. However, mortality rates may be decreased by the use of drugs that control the bacterial and parasitic complications. Specifically, oxytetracycline and chlortetracycline are recommended to prevent secondary pulmonary infections (OIE 2000). Today an effective etiological treatment is not available. Only supportive and symptomatic treatment can be initiated. ( By S. Geerts ,last update January 2009, Institute of Tropical Medicine, Antwerp, Belgium EAZWV Transmissible Disease Fact Sheet, Sheet No. 25). Antibiotics such as [chloramphenicol](http://en.wikipedia.org/wiki/Chloramphenicol), [penicillin](http://en.wikipedia.org/wiki/Penicillin) and [streptomycin](http://en.wikipedia.org/wiki/Streptomycin) can be used and supportive treatment may be helpful. A [vaccine](http://en.wikipedia.org/wiki/Vaccine) has been developed that may decrease death in the flock. According to the country's policy, there may be movement restrictions, slaughter of affected flocks in an attempt to eradicate the disease ( [Peste des Petits Ruminants](http://en.wikivet.net/Peste_des_Petits_Ruminants) reviewed and published by [WikiVet](http://en.wikipedia.org/wiki/WikiVet), accessed 10 October 2011).

There are no known effective drugs against virus etiology of this disease. However, hyperimmune serum and supportive treatment with fluid therapy for dehydration and antibiotics to prevent secondary bacterial infection could be used to save the life of the infected goats. Anene *et al.*, 1987 who studied the appraisement of the treatment of naturally occurring PPR in goats with oxytetracycline, chloramphenicol 25% aqueous solution and metamerazine in different groups at the recommended dose rates found recovery rate 14.29%. Islam *et al.*, 2003 stated that antibiotic combined therapy with hyperimmune serum, in which recovery rates was an average of 68.75%.Good nursing, Symptomatic treatment with broad-spectrum antibiotic/ sulphur drugs can save life of sick animal of and can improve the immunosuppressive condition of the affected goat (Sil, 2000 and Scott, 2000).

Control of PPR outbreaks can also rely on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk populations. Immunization of small ruminants with lymph node and spleen materials containing virulent virus inactivated with 1.5-5% chloroform was tried and the animals were immune to subsequent challenge 18 months latter (Braide, 2001). The tissue culture rinderpest vaccine (TCRV) at a dose of 102.5 TCID50 protected goats against PPR for 12 months and the animals were not able to transmit the infection following challenge with PPR virus (Taylor, 2000), although the antigen was detected in lachrymal swabs from vaccinated animals after challenge with virulent virus (Gibbs *et al.,* 2001). However, it was reported previously that considerable residues of virulence were detected after 32, 42, even 65 serial passages in embryonic lamb kidney cells (Taylor, 2002). This vaccine was successfully used to control PPR in some countries in West Africa and is widely used in many African countries (Lefèvre and Diallo, 2000).

**CHAPTER-III**

**MATERIALS AND METHOD**

**Study area and duration :** Thestudy was conducted at the, Upazilla, Veterinary Hospital, Bogra Sadar, Bogra from 5th May to 4th July 2013.

**Size of sample**: For this study 20 apparently PPR infected goat and 10 healthy goat were taken to compare the hematological parameters of their blood.

**Blood Collection**: Approximately 5 ml of blood was collected aseptically from the jugular vein of each 20 PPR infected goats and 10 healthy goats in vial containing Na EDTA @ 2 mg/ ml. All samples for hematological analysis were stored in 4˚C and tested within 24 hours after collection in the “Disease Investigation and Animal Nutrition Research Laboratory”, Upazilla Veterinary Hospital, Bogra Sadar, Bogra.

**Hematological examination**: The Following hematological analysis were performed: Total erythrocyte count (TEC), total leukocyte count (TLC), differential leukocyte count (DLC), Packed cell volume (PCV) and erythrocyte sedimentation rate (ESR) in “ Disease Investigation and Animal Nutrition Research Laboratory” .TEC and TLC were determined by hemocytometer. All differential counts of leukocytes were prepared as thin blood smear stained by Wright’s method. All the above parameters as TEC, TLC, PCV, ESR, and DLC were performed according to the method. Basically my study is based on DLC parameters of PPR affected Black Bengal goats and healthy Black Bengal goats.

**Vaccination history**: Each owner was asked about previous vaccination history of his/her goat suffering from PPR. Some of them are vaccinated some are not vaccinated.

**Clinical Examinations of PPR cases in goats :**

**(1)History:** Data were recorded from the owners for the Breeds/Sex/Age of the animals; probable signs of the disease are fever, diarrhea, nasal and ocular discharges, depressed appetite from the last two days of the clinical onset.

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Fig : 1.5 Diarrhea in goat

Fig : 1.4 Necrotic debris on oral lesions

Fig: 1.3 Irregular non-hemorrhagic oral lesions

Erosions at muco-cutaneous junction with inflammation around the mouth

Raised and flat circular lesions on tongue

**(2).Clinical inspection:**

Fig : 1.3 Irregular non-haemorrhagic oral lesions

The clinical examination of the affected animals revealed high fever (106-107°F), mild conjunctivitis, congestion of the third eye lids and mild ocular and nasal discharges. Erosive lesions were present on the inner side of the upper lip. All animals exhibited diarrhea. On the external examination, the carcass was dehydrated (sunken eyes) along with the soiling of hind quarters.

**Presumptive diagnosis:** All PPR affected goats show more or less similar signs. Diagnosis was based on the following clinical signs:

|  |  |  |
| --- | --- | --- |
| Methods Used for detail clinical examination of PPR affected goats | | |
| Close inspection | *Direct* (Palpation) | *Indirect*(Auscultation) |
| Oculo-nasal discharge  Hind quarter soiled with faeces  Shooting diarrhea with foul odor  Erosion in mouth in some cases  Lowering head and arched back | Body temperature  (High fever (104°F-107° F)  Skin fold test for the estimation of the degree of dehydration | Respiratory rate |

**Treatment:**

The infected animals is treated with sulphonamide and also other symptomatic treatment is given like fluid therapy for diarrhea, dehydration, Respiratory stimulants were also given. Lesions around the eyes, nostrils and mouth should be cleaned and good nursing provided.

**CHAPTER-IV**

**RESULTS AND DISCUSSION**

An overview statistics on DLC on the number of PPR-affected goats and healthy goats were investigated in the study is summarized in Table 1. The study was ended when the numbers of PPR-cases in Black Bengal goats reached the number 20 and in healthy goats were 10 in no. In table no, 2 and 3 agewise average Differential Leukocyte Count was counted. In table no. 4 and 5 the average male and female goats DLC values were shown in a significant manner respectively.

In my study the Table shows that the neutrophil range in affected goats were 4-31% and in healthy goats were 26-39%. The average value was 17.3 in PPR affected goats and 31.4 in healthy goats (Table 1). We found that neutrophil numbers were lower in affected goat than healthy goats. It sugges ted due to viral infection in host body destruction of neutrophil is occur in PPR affected goats .so the neutrophil % is lower in PPR affected goats then healthy goats(Figure.1). The author reported similar values for west African Dwarf goat as was obtained in this study on goat (Aikhuomobhogbe *et al*, 2006 ) and also observe the same value in osmanabadi goats under stress condition (Ambor *et al* , 2009)

In case of lymphocyte the ranges in affected goats were 44% - 89% and in healthy goats were 52% - 65%. The average value was 68.15 in PPR affected goats and 58.5 in healthy goats(Table 1). We found that lymphocyte numbers were higher in affected goats than healthy goats. It suggested due to viral infection in host body try to eliminate the organism and give rise to such high lymphocyte.So the lymphocyte % is higher in PPR affected goats then healthy goats (Figure.1). The author reported similar values for west African Dwarf goat as was obtained in this study on goat (Aikhuomobhogbe *et al*, 2006 ) and also observe the same value in osmanabadi goats under stress condition (Ambore *et al*, 2009) .

In case of eosinophil the range in affected goats were 1% - 22% and in healthy goats were 3% - 8%. The average value was 6.3 in PPR affected goats and 5.4 in healthy goats. We found that eosinophil numbers were higher in affected goats than healthy goats. It suggested due to viral infection in host body try to eliminate the organism and give rise to such high eosinophil. So the eosinophil % is higher in PPR affected goats then healthy goats. The author reported similar values for west African Dwarf goat as was obtained in this study on goat (Aikhuomobhogbe *et al*, 2006 ) and also observe the same value in osmanabadi goats under stress condition (Ambore *et al*, 2009).

In case of basophil the range in affected goats were 1% - 4% and in healthy goats were 0% - 2%. The average value was 2.15 in PPR affected goats and 1 in healthy goats. We found that basophil numbers were higher in affected goats than healthy goats. It suggested due to viral infection in host body try to eliminate the organism and give rise to such high basophil. So the basophil % is higher in PPR affected goats then healthy goats. The author reported similar values for west African Dwarf goat as was obtained in this study on goat (Aikhuomobhogbe *et al*, 2006 ) and also observe the same value in osmanabadi goats under stress condition (Ambore *et al*, 2009).

In case of monocyte the range in affected goats were 3% - 19% and in healthy goats were 2% - 5%.. The average value was 7.55 in PPR affected goats and 3.7 in healthy goats. We found that Monocyte numbers were higher in affected goats than healthy goats. It suggested due to viral infection in host body try to eliminate the organism and give rise to such high Monocyte.So the monocyte % is higher in PPR affected goats then healthy goats. The author reported similar values for west African Dwarf goat as was obtained in this study on goat (Aikhuomobhogbe *et al* , 2006 ) and also observe the same value in osmanabadi goats under stress condition (Ambore *et al* , 2009).

But, severe leucopoenia could have been due to the inhibition of peripheral blood lymphocytes proliferation by PPR virus (Heaney *et al.,* 2002). A marked lymphocytopoenia, monocytopoenia, neutrophilia and eosinopoenia in present investigation could have been due to the combined effect of virus infection and stress as evidenced by elevated cortisol levels (Kataria and Kataria, 2004).

**Table 1**:Result of Differential leukocyte count (DLC) in PPR affected and normal Black Bengal goats:

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Affected goat** | | | | | | | | Healthy goat | | | | | | | |
| **SL No** | **DLC feature** | | | | | | | **SL No** | DLC feature | | | | | | |
|  | **Age** | **M** F | **N** | **E** | **B** | **L** | **M** |  | **Age** | **M**  **F** | **N** | E | **B** | **L** | **M** |
| **1** | 6 month | Male | 11 | 15 | 2 | 65 | 7 | **1** | 3 yr | Male | 39 | 5 | 0 | 53 | 3 |
| **2** | 9 month | Male | 5 | 13 | 1 | 78 | 3 | **2** | 1.5 yr | Male | 26 | 6 | 1 | 65 | 2 |
| **3** | 5 yr | Male | 32 | 3 | 2 | 56 | 7 | **3** | 5.2 yr | Male | 39 | 4 | 0 | 54 | 3 |
| **4** | 2.4 yr | Male | 18 | 22 | 4 | 44 | 12 | **4** | 4 yr | Male | 29 | 5 | 2 | 60 | 4 |
| **5** | 4 month | Male | 9 | 6 | 1 | 76 | 8 | **5** | 1 yr | Male | 29 | 7 | 1 | 61 | 2 |
| **6** | 1.3 yr | Male | 4 | 10 | 2 | 82 | 5 | **6** | 1.7 yr | Male | 27 | 8 | 1 | 59 | 5 |
| **7** | 1.5 yr | Male | 4 | 6 | 2 | 89 | 15 | **7** | 2 yr | Female | 38 | 4 | 2 | 52 | 4 |
| **8** | 4.2 yr | Male | 21 | 8 | 3 | 65 | 3 | **8** | 9  month | Female | 26 | 6 | 0 | 63 | 5 |
| **9** | 1.7 yr | Male | 13 | 3 | 1 | 81 | 7 | **9** | 5 yr | Female | 32 | 3 | 1 | 58 | 6 |
| **10** | 2 yr | Male | 17 | 3 | 1 | 65 | 14 | **10** | 4.5yr | Female | 29 | 6 | 2 | 60 | 3 |
| **11** | 2.5 yr | Male | 15 | 2 | 2 | 76 | 5 |  |  |  |  |  |  |  |  |
| **12** | 3 yr | Male | 12 | 3 | 1 | 76 | 8 |  |  |  |  |  |  |  |  |
| **13** | 2.4 yr | Female | 16 | 4 | 3 | 72 | 5 |  |  |  |  |  |  |  |  |
| **14** | 4.5 yr | Female | 24 | 1 | 4 | 57 | 19 |  |  |  |  |  |  |  |  |
| **15** | 3.5 yr | Female | 20 | 3 | 1 | 68 | 8 |  |  |  |  |  |  |  |  |
| **16** | 3. 2 yr | Female | 22 | 4 | 2 | 68 | 4 |  |  |  |  |  |  |  |  |
| **17** | 4yr | Female | 26 | 7 | 3 | 60 | 4 |  |  |  |  |  |  |  |  |
| **18** | 5.2 yr | Female | 31 | 4 | 1 | 58 | 6 |  |  |  |  |  |  |  |  |
| **19** | 2.3 yr | Female | 24 | 3 | 4 | 65 | 4 |  |  |  |  |  |  |  |  |
| **20** | 3.5 yr | Female | 22 | 6 | 3 | 62 | 7 |  |  |  |  |  |  |  |  |
| Avg. | | | 17.3 | 6.3 | 2.15 | 68.15 | 7.55 | Avg. | | | 31.4 | 5.4 | 1 | 58.5 | 3.7 |

###### N = Neutrophil , E = Eosinophil , B = Basophil

**L = Lymphocyte , M = Monocyte**

**Fig : 1 Difference in avg. neutrophil and lymphocyte percentage between affected and healthy goats.**

From the below table ( 2 , 3 ) we can see that, The average neutrophil % in 0 – 1 , 1 – 2, 2 - 3, 3 - 4, 4 - 5, >5years old PPR affected Black Bengal goats were 8.33%, 9.5%, 17%, 21.25%, 26.33% ,31%and in healthy goats were 26.5%, 32%, 39%, 29%, 30.5%, 39%(Table no. 2 & 3). So,in every years the average. neutrophil % in PPR affected Black Bengal goats were less than the healthy goats. It suggests that due to disease condition where might severe destruction of neutrophil which yields such lower number (Table no. 2 &3).

The average lymphocyte % in 0 – 1 , 1 – 2 , 2 - 3, 3 - 4, 4 - 5, >5 years old PPR affected Black Bengal goats were 73%, 79.25%, 66.6%, 61.5%, 60%, 58% and in healthy goats were 62%, 58.67%, 53%, 60%, 60.5%, 59%(Table no. 2 & 3). So, the avg. Lymphocyte % in PPR affected Black Bengal goats were more than the healthy goats in the years of 0 – 1 , 1 – 2 , 2 - 3, 3 - 4 and less in4 - 5 , >5 years old goats(Figure no. 2 &3).

The average eosinophil % in 0 – 1 , 1 – 2 , 2 - 3, 3 - 4, 4 - 5, >5years old PPR affected Black Bengal goats were 11.33% ,5.5%, 6.8%, 5, 4%, 1 %and in healthy goats were 6.5%, 6, 5%, 5%, 4.5% ,4% (Table no. 2 & 3). So, the avg. eosinophil % in PPR affected Black Bengal goats and healthy goats were more or less same.

The average basophile % in 0 – 1 , 1 – 2 , 2 - 3, 3 - 4, 4 - 5, >5 years old PPR affected Black Bengal goats were 1.33%, 1.5%, 2.8%, 2.25%, 3%, 1% and in healthy goats were 0.5%, 1.33%, 0%, 2%, 1.5%, 6%(Table no. 2 & 3). So, the avg. Basophill % in PPR affected Black Bengal goats were more than the healthy goats in the years of 0 – 1 , 1 – 2 , 2 - 3, 3 - 4, 4 - 5 and less in>5 years old goats.

###### The average monocyte % in 0 – 1 , 1 – 2 , 2 - 3, 3 - 4, 4 - 5, >5 years old PPR affected Black Bengal goats were 6%, 10.25%, 6.8%, 5.75%,9.67%, 6% and in healthy goats were 3.5%, 3.67%, 3%, 4%, 4.5%, 3%(Table no. 2 & 3). So, in every year the avg. Monocyte % in PPR affected Black Bengal goats were more than the healthy goats.

###### Table 2: Average% of DLC Result of PPR affected Black Bengal goat on the basis of age

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Age**  **(Year)** | **No. of animal** | **Neutrophil**  **Avg.** | **Eosinophil**  **Avg.** | **Basophil**  **Avg.** | **Lymphocyte**  **Avg.** | **Monocyte**  **Avg.** |
| 0 - 1 | 3 | 8.33 | 11.33 | 1.33 | 73 | 6 |
| 1 - 2 | 4 | 9.5 | 5.5 | 1.5 | 79.25 | 10.25 |
| 2 - 3 | 5 | 17 | 6.8 | 2.8 | 66.6 | 6.8 |
| 3 - 4 | 4 | 21.25 | 5 | 2.25 | 61.5 | 5.75 |
| 4 - 5 | 3 | 26.33 | 4 | 3 | 60 | 9.67 |
| >5 | 1 | 31 | 1 | 1 | 58 | 6 |

**Fig : 2 Difference in average. neutrophil and lymphocyte percentage in PPR affected goats on the basis of age group.**

In this graph Row- 1 indicates the average neutrophil and lymphocyte % within 0-1 years of age, Row- 2 indicates 1-2 years of age, Row- 3 indicates 2-3 years of age, Row - 4 indicates 3-4 years of age, Row -5 indicates 4-5 years of age, Row- 6 indicates >5years of age ,in PPR affected goats.

###### Table 3 : Average% of DLC Result of Healthy Black Bengal goats on the basis of age

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Age**  **(Year)** | **No. of animal** | **Neutrophil**  **Avg.** | **Eosinophil**  **Avg.** | **Basophil**  **Avg.** | **Lymphocyte**  **Avg.** | **Monocyte**  **Avg.** |
| 0 - 1 | 2 | 26.5 | 6.5 | 0.5 | 62 | 3.5 |
| 1 - 2 | 3 | 32 | 6 | 1.33 | 58.67 | 3.67 |
| 2 - 3 | 1 | 39 | 5 | 0 | 53 | 3 |
| 3 - 4 | 1 | 29 | 5 | 2 | 60 | 4 |
| 4 - 5 | 2 | 30.5 | 4.5 | 1.5 | 60.5 | 4.5 |
| >5 | 1 | 39 | 4 | 6 | 59 | 3 |

**Fig : 3 Difference in avg. neutrophil and lymphocyte percentage in healthy goats on the basis of age group.**

In this graph Row- 1 indicates the average neutrophil and lymphocyte % in 0-1 years of age, Row- 2 indicates 1-2 years of age, Row- 3 indicates 2-3 years of age, Row - 4 indicates 3-4 years of age, Row -5 indicates 4-5 years of age, Row- 6 indicates >5years of age in healthy goats .

From the below table (table(4) and table(5) we can see that, In total hematological studies the average value of male and female almost same. It shows that sex does not play a significant role in hematological parameter. However in my study the lymphocytes number were slightly higher in female than the male. The reason is not clear to the author.

In our study we found that in virus affected average, neutrophil% in male goats was 22.5, female goats was 13.41(Table4) and the healthy male goats was 31.5, female goat was 31( Table 5). The neutrophil numbers were reduced in PPR affected male and female Black Bengal goats then healthy individuals. The result is similar with West African Dwarf goats where neutrophil % of PPR affected male is also higher than PPR affected female (Aikhuomobhogbe *et al* , 2 May 2006, 2 May 2006 ).

In case of eosinophil, the average eosinophil % in PPR affected male goats was 7.83, female goats was 4 (Table 4) and the healthy male goats was 5.83, female goat was 4.75 ( Table 5) . The average , eosinophil % in PPR affected males were more than healthy male goats and PPR affected females were less then healthy female goats.

In case of basophil, The average basophil % in PPR affected male goats was 1.83, female goats was 2.62( Table 4) and the healthy male goats was 0.83, female goat was 1.25( Table 5) .So, The avg. basophil% in PPR affected male and female Black Bengal goats were more than the healthy Black Bengal goats .

In case of lymphocyte, The average, lymphocyte % in PPR affected male goats was 63.75 , female goats was 71.08 ( Table 4) and the healthy male goats was 58.67, female goat was 59.25( Table 5). So, The average, lymphocyte% in PPR affected male and female Black Bengal goats were more than the healthy Black Bengal goats. So, the lymphocytes numbers were higher in infected goat than healthy goats. The result is similar with the West African Dwarf goats where PPR affected male goat had lower % of lymphocyte than PPR affected female goats (Aikhuomobhogbe *et al*., 2 May 2006, 2 May 2006)..

In case of. monocyte, The avg. monocyte % in PPR affected male goats was 7.83, female goats was 7.12 (Table 4) and the healthy male goats was 3.16 , female goats was 4.5( Table 5). So, the avg. monocyte% in PPR affected male and female Black Bengal goats were more than the healthy Black Bengal goats.

**Table 4 : Average.% of D ifferential Leulocyte Count Result of Peste des Petits ruminants affected Black Bengal goats on the basis of sex**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | No. of animal | Neutrophil  Avg. | Eosinophil  Avg. | Basophil  Avg. | Lymphocyte  Avg. | Monocyte  Avg. |
| Male | 12 | 22.5 | 7.83 | 1.83 | 63.75 | 7.83 |
| Female | 8 | 13.41 | 4 | 2.62 | 71.08 | 7.12 |

**Table 5 : Average% of Differential leukocyte count Result in Healthy Black Bengal goats on the basis of sex**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | **No. of animal** | **Neutrophil**  **Avg.** | **Eosinophil**  **Avg.** | **Basophil**  **Avg.** | **Lymphocyte**  **Avg.** | **Monocyte**  **Avg.** |
| Male | 6 | 31.5 | 5.83 | 0.83 | 58.67 | 3.16 |
| Female | 4 | 31 | 4.75 | 1.25 | 59.25 | 4.5 |

**CHAPTER-V**

**CONCLUSION**

PPR is highly contagious disease with higher mortality and morbidity in goat. In differential leukocyte counts percentage of various leukocytes counts were decreased in affected goats in comparison to healthy goats but only increased lymphocyte counts. So, it can be concluded that total lymphocyte counts were increased in PPR affected goats and others hematological pictures were also increased. But due to small sample sizes the result may vary.

**ABBREVIATIONS**

**DLC =** Differential Leukocyte count.

**ESR =** Erythrocyte sedimentation rate.

**PCV =** Packed cell Volume.

**PPR =** Pesti des Petits ruminant**.**

**TEC =** Total Erythrocyte count.

**TLC =** Total Leukocyte count.

**BLRI =** Bangladesh Livestock Research Institute**.**

**CVASU =** Chittagong Veterinary and Animal Sciences University

**EMPRES** = Emergency Prevention System for Transboundary

Animal and Plant Pests and Diseases.

**FAO** = Food and Agricultural Organization.

**OIE** = Office International des Epizooties/World Organization

for Animal Health.

**CHAPTER-VI**

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**ANNEX-I**

**Questionnaire for Data Collection**

1. Owners Name:…………………….Address…………….…………Date…………..….
2. Species:………………………………………………………………………….
3. Farm Size : :………………………………………………………….………
4. Age: :…………………………………………………………………………
5. Rearing system:…………. …………………………………………………..
6. Number of PPR affected goats:… ……………………………………………
7. Vaccination status…………………………………………………………….
8. Clinical signs…………………..……………………………………………
9. Tentative Diagnosis……………………………………………………………
10. Treatment…………………..…………………………………………………..

Name of the interviewer…………….

Date…………..……………………....

Signature………………………………