CHAPTER-I
INTRODUCTION

Goat, the poor man's cow produces high quality meat, milk and skin. It also play important role in poverty alleviation, income generation and in food production (Debnath, 1995). However different types of infectious diseases (Peste des Petits Ruminants, pneumonia, goat pox, enterotoxaemia, mastitis, tuberculosis, tetanus, papillomatosis, ringworm etc.) and non-infectious diseases (ketosis, pregnancy toxemia and vaginal prolapse etc.) are important problems to raise and rear the goat in our country.

Peste des Petits Ruminants (PPR) is an acute febrile viral disease of small ruminants, characterized by high fever (106°F), mucopurulent nasal discharge, profuse diarrhoea with foul odor and finally death due to dehydration (Sil, 2000). In South Asia, PPR was first recorded from India (Shaila *et al.,* 1989) where the disease initially occurred in Sheep and subsequently become more prevalent in goat (Kulkarni *et al.,* 1996). The disease is widely distributed in equatorial Africa, the Arabian Peninsula and part to Indian subcontinent including Bangladesh (Kulkarni *et al*., 1996; Debnath, 1995).
In Bangladesh, Dr. Taylor identified the first PPR outbreak during 1993. It is thought that the disease might have come in Bangladesh from India (Debnath, 1995). The disease is endemic in Bangladesh and causes major economic losses due to the high rates of mortality and morbidity in infected domestic animals of sheep and goats. It causes death in more than 50% of the affected animals due to high fever, pneumonia, diarrhea and dehydration. The morbidity and mortality rate are 80%-90% and 40%-80% respectively (Sil, 2000).

A wide variety of diseases are appeared with different clinical symptoms that the physical examination is not sufficient to provide a diagnosis, so blood profile can play a crucial role for accurate diagnosis. Though the physiological equilibrium is controlled by blood in the body (Ozkul *et al.,* 2002), but many physiological factors such as age and species may alter this equilibrium. Blood component may also be influenced by pathological factors (Szbo *et al.,* 2005). The importance of hemato-biochemical indices in animal is well acknowledged. The changes in hematological constituents are important

indicators of the physiological or pathological state of the animal (Ahmed *et al*., 2003). Blood examination is also performed for screening procedure to assess the general health of animal (Gutienez *et al.,* 1971; Jain, 1993; Peinado *et al.,* 1999).

The complete blood count (CBC) is an important and powerful diagnostic tool. It can be used to monitor response to therapy, to determine the severity of an illness or as a starting point for formulating a list of differential diagnosis. The evaluation of the blood parameters and its fraction provide the information required to interpret the occurrence of dehydration, infectious, immune diseases and inflammatory responses. But few limited attempts have been undertaken to diagnose the disease based on both clinical signs and corresponding hematological changes. Therefore, the study was undertaken with the following objectives:

a. To diagnose the PPR on the basis of presenting clinical signs.
b. To study the changes of hematological and biochemical parameters of the affected goats.

CHAPTER –II
REVIEW OF LITERATURE

There are so many literatures about PPR diseases, from which some important research findings are listed below along with references.

2.1 PPR

PPR is an acute viral disease of small ruminants caused by a *Morbillivirus* and characterized by fever, oculonasal discharges, stomatitis, diarrhoea and pneumonia. The disease is one of the major threats to Sheep and Goats.

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) reported that this disease followed by death in calves experimentally which was infected with peste des petits ruminants virus (PPR) infected tissue, no natural outbreak has been reported in cattle. It is generally admitted that cattle can only be infected sub-clinically. Both experimental and natural infections of PPRV have been reported in wild ruminants (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, dyspnea, coughing, enteritis, severe diarrhoea followed by death (Pawaiya *et al.,* 2004). The disease is highly contagious with morbidity and mortality rates reaching as high as 100 % and 90 % respectively (Abu-Elzein *et al.,* 1990).

2.2 Geographical distribution of PPR

PPR has comparatively a shorter history which only dates back to 1940, when it was first described by Gargadennec and Lalane (1942) in Ivory Coast of West Africa. For a longtime, 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 and the Middle East

with extension to Turkey, Pakistan, India, Bangladesh and Nepal (Shaila *et al.,* 1996; Dhar *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.*, 1994), depicts the first report of PPR from different countries. In India, PPR was first reported in 1987 from Tamil Nadu and 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). In Bangladesh, the present PPR outbreak occurred in a small flock of goats in July, 2007, the rainy season. Out of 37 goats 19 (51%) developed clinical disease, and 5 (13.5%) died (Rahman *et al.,* 2011).

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.

2.3 History of PPR outbreak in Bangladesh

In Bangladesh, Dr. Taylor identified the first PPR outbreak during 1993. 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. Investigations are proceeding to establish the extent of the spread.

2.4 Morphology

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 x10^6 DA with 16,000 ribonucleotides which is encoded with six structural proteins, 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).

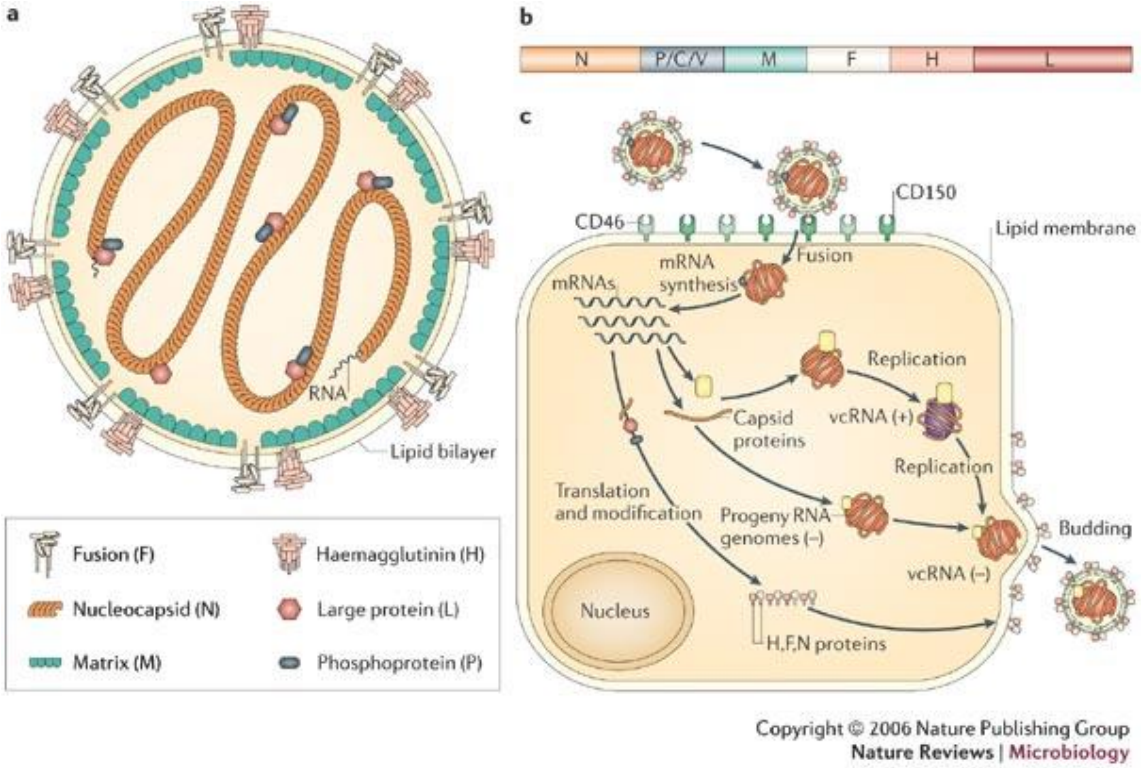

Figure 1: Morphology of *Morbillivirus*

2.5 Pathogenecity and transmission

PPR virus is not pathogenic for cattle but Rinderpest can also affect goat along with PPR (Gibbs *et al.,* 1979). Case fatality rate is higher in goat (55-85%) compared to sheep (45-75%) (Opasina *et al.,* 1985). For PPR to spread, close contact between infected and susceptible animals is needed (Ozkul *et al.,* 2002). There are several means of transmission of the virus between animals (Saliki, 1998) - inhalation of aerosols produced by sneezing and coughing of infected animals could be the main way. No carrier state is known to develop in the infected animals. Outbreaks were more frequently reported in the rainy, dry and cold seasons (Office International des Epizooties (OIE), 2002). Newly purchased animal from market and wild animal have been suspected to play a role for spreading of disease (Radostits *et al.*, 2007).

2.6 Clinical signs

The course of the disease is acute and sub acute, few of the animals die even in 36 hours of onset of the disease. The affected animal initially is severely depressed with a sudden rise in body temperature reaching almost 107.6°F in some cases, and the fevers persist for 7-8 days. From the onset of fever, most animals have serous nasal discharges which

progressively turn into mucopurulent discharge, leading to severe respiratory distress. Areas of erosions are most commonly seen on the visible nasal mucous membranes and muco-cutaneous junctions with inflammation around the mouth. The erosive and necrotic stomatitis starts as areas of hyperemia at gums, cheeks, dental pad and or anterior dorsal part of tongue with frothy salivation. The areas later develop into irregular non-haemorrhagic lesions and in some of the cases circular raised but flat non-bleeding lesions are present on the tongue. There is a great amount of necrotic debris on the older lesions. The individuals with severe oral lesions have visible swelling around mouth. Non-haemorrhagic diarrhoea is observed in all affected animals, developing 2-3 days after onset of the disease. Conjunctivitis is recorded with lachrymal discharges which become mucoid resulting in sticky eyelids. Abortion in pregnant animals is a consistent feature and mucous membranes of valva have erosive lesions very similar to that in intestinal mucosa. Subnormal temperatures precede death in animals with severe diarrhoea for few days. Temperature dropped down in the later stages of diarrhoea due to emaciation and dehydration (Sil, 2000).

2.7 Association among PPR, age and breed 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.*, 2007). The young goat may die due to anoxia. Black Bengal breed is more susceptible to PPR than Jamunapari (Mondal *et al.*, 1995). Also noticed a higher prevalence of PPR in Black Bengal goats (57.34%) than in Jamunapari in the West Bengal, India.

2.8 Haemato-biochemical parameters

There were statistically significant differences between all parameters. White blood cells (WBCs), Mean corpuscular volume（MCV), Mean corpuscular hemoglobin (MCH) and Mean corpuscular hemoglobin concentration (MCHC) parameters were significantly high, but hemoglobin (HB), Red blood cells (RBCs) and PCV parameters were low in infected group compared to those of control group. (Aytekin *et al.*, 2011).

Total serum protein values decreased but globulin concentration increased indicating immune response towards infection. (Kataria *et al.*, 2007).

2.9 Treatment and vaccination

Goat vaccinated with escaped mutant virus were 100% sera converted following 15 days post vaccination and developed protective immunity against the field strains of PPR for more than 2 years (BLRI, 1999). 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, 2000). Thermostable PPR vaccine has been developed experimentally by scientists of BLRI and DLS (Chowdhury *et al*., 2004)

Hyper immune serum can be used successfully along with long acting antibiotic (Antibiotic combined Hyper immune serum Therapy) to limit the spread of virus and recover those animals which are under incubation and in early stage of infection. Good nursing, symptomatic treatment with broad spectrum antibiotic / Sulphur drugs can save life of sick animals and also can improve the immunosuppressive condition of the affected goat (Sil, 2000).

CHAPTER –III
METHODOLOGY

3.1 Place and period of study

A cross sectional study was conducted on PPR affected goats at S.A Quaderi Teaching Veterinary Hospital (SAQTVH) of CVASU, Chittagong for a period of three months from September to November, 2013.

3.2 Collection of information of PPR affected goats

A total of 20 cases of goats were brought to SAQTVH for treatment during my internship period. After admission at the hospital, demographic information, vaccination along with deworming status and clinical signs were recorded. PPR was diagnosed presumptively on the basis of clinical signs (Kataria *et al.*, 2007).

3.3 Presumptive diagnosis: All PPR affected goats show more or less similar signs. Diagnosis was based on the following clinical signs (Chakrabarti, 2006).

Table 1: Methods Used for detail clinical examination of PPR affected goats.

Close inspection	***Indirect***	***Indirect***
Oculo-nasal discharge	Body temperature	**(**Auscultation)
Hind quarter soiled with feces	High fever (104°F-107° F)	Respiratory rate
Shooting diarrhea with foul odor		
Erosion in mouth in some cases		
Lowering head and arched back		

3.4 Laboratory testing

3.4a Collection of sample

Approximately 5ml of blood was collected aseptically from the jugular vein of each 20 PPR infected goats. 3ml blood was kept in a vial containing Ethyline diamine tetra acetic acid (EDTA @1mg / ml) and 2ml was kept in another vial for smooth coagulation. Blood samples were transported to the Physiology laboratory of Department of Physiology, Biochemistry and Pharmacology, CVASU within half an hour and kept in the chiller part of refrigerator for TLC, DLC, Erythocyte sedimentation rate (ESR), PCV and for serum preparation.

3.4b Serum Sample Preservation

2ml of coagulated blood was kept overnight in refrigerator for collecting the serum. The serum samples obtained after centrifugation and stored at 4ºC in refrigerator for further biochemical test.

3.4c Hematological Examination

Different hematological parameters were studied according to the methods described by Alcon, 2000. The detail of the procedure is described as follows:

3.4c.1 TLC

A dry and clean counting chamber was placed under the microscope. There after the chamber was examined under low power objective without the cover slip in order to understand the ruling. Blood mixed properly with EDTA was sucked into the WBC pipette upto the mark 0.5 keeping the pipette nearly horizontal. The diluting fluid was sucked into the pipette until the mixture of blood and the fluid reaches 11 marks above the bulb. The pipette was rotated and inverted several times to ensure thorough mixture blood with diluting fluid. A few drop of mixture was discarded from the pipette and wiped out its tip. A small drop of diluting fluid mixture was allowed to form at the tip of the pipette. The tip was placed on the surface of the chamber touching the edge of the cover slip at an angle of 45^0 to the horizontal. The dilute blood was allowed to flow evenly and slowly under the cover slip by capillary action. There should be no overflow into the central or side trenches as the excess fluid will lift up the cover slip and gives false high count. A few minutes were spent before the count to allow the corpuscles to settle.

WBC Counting: The leucocytes were counted in four large squares (each having 1 sq. mm is divided into 16 small squares) at the four corners of the counting chamber. This means that the white blood corpuscles in 64 small squares were counted. The white blood cells were appeared as round faintly dark and refractive dots under low power where as high power the stained nucleus were visible with clear granular cytoplasm around it. In counting the cells which touched the left hand line or upper line of the square were taken to be within that square and those which touched the lower and right lines were omitted as outside the square. This technique was followed to avoid the duplicate counting and counting was started from upper left chamber.

Calculation factor

a. Area of chamber: 4 sq. mm
b. Depth of chamber: 1/10 mm
c. Dilution obtained: 1:20 =1/20

Total volume: 4*1/10*1/20 cu. mm

=1/50 cu. mm

Suppose 1/50 cu. mm contains x number of cells

So, 1 cu. mm contains x*50 number of cells.

The total numbers of cells were multiplied by the calculation factor. i.e. 50 and expressed the result in thousand/cu.mm by dividing the total number of cell by 1000.

3.4c.2 DLC

Several clean grease free slide with smooth unbroken end were selected, then a drop of blood was placed at right end of the slide. The slide was taken on a piece of paper on the table holding firmly by the force or thumb finger on left hand. The even edge of the second slide was placed near the drop of blood towards the middle of slide. The second slide was drawn towards the drop of the blood at an angle of 45^0 and the blood was spread along the edge. Keeping the second slide in the same angle a quick even push was given with a uniform force toward the other end of slide. When the blood film dried, it was placed in the staining rack. 8-10 drops of Wright's stain was poured over the slide to cover the blood smear and allowed to stain for 2 minutes. Distilled water above double quantity of stain was added and mixed by blowing pipe and allowed to stand for 5 minutes. Then the film was washed with distilled water without much distribution the slide till the slide film become pinkish. The slide was placed against the support and allowed to dry in air.

DLC Counting: The slide was placed on a fixed place and a drop of immersion oil on the slide. Then the cells were identified by using high power objective 100x following parallel stripe method. The counting was continued till 100 cells were counted. Different types of cells were counted by tally method.

3.4c.3 ESR by Wintrobe's method

With the help of special loading pipette the Wintrobe's tube was filled with blood starting at its bottom and withdrawing the pipette as the tube was filled from below up-wards. The tube was filled up to the mark 10 air bubble removed from the top of the column of blood so that it stood exactly at 10. The tube was placed in the rack in vertical portion. The reading taken after one hour by noting down the level of lower end of the clear plasma.

3.4c.4 Determination of PCV

With the help of special loading pipette the Wintrobe's tube was filled with blood starting at the bottom and withdrawing the pipette as the tube was filled from below upwards. The tube was filled up to the mark 10; air bubble was removed from the top of the column of blood. So that it would be stood exactly at 10. The Wintrobes tube was centrifuged at a rate of 3000 rpm for about 30 minutes. After 30 minutes the tube were taken out of centrifuged machine and PCV was read directly from the calibrated on the right side of the tube. The result was expressed in percentage.

3.4d Biochemical examination:

TP, Sodium (Na^+), Potassium (K^+) and Chloride (Cl^-) were determined by automated biochemical analyzer (Humalizer, 3000) established in Biochemistry laboratory of CVASU.

Photometric Colorimetric Test for Total Proteins (Biuret Method*)*

Contents

Reagents:	4×100 ml or 1×1000 ml Color reagent	
	Sodium hydroxide	200mmol/l
	Potassium sodium tartrate	200mmol/l
	Copper sulfate	12 mmol/l
	Potassium iodide	30 mmol/l
	Irritant R 36/38	

Standard	1×3 ml Standard	
	Protein	8g/dl
	Or	80g/l
	Sodium azaid	0.095%

Assay

Wavelength: Hg 546 nm, 520-580 nm

Optical path: 1 cm

Temperature: 20 - 25^0C

Measurement: Against reagent blank

Only one reagent blank per series was required.

Pipetting Scheme: 1000 µl of reagent was taken in previously marked three eppendorf tubes for reagent blank, sample and standard separately by micropipette. 20µl of sample and standard were added in the previously marked eppendorf tube, respectively. Reagent blank eppendorf tube only contain reagent. Sample and standard for total protein were mixed separately in each eppendorf tube and incubated for 10 minutes at 25^0C. Absorbance of the sample and standard were measured against the reagent blank within 30 min (ΔA) λ_{max} in 546 nm by spectrophotometer of the Humalyzer 3000®.

Pipette into cuvettes	Reagent blank	Sample/Standard
Sample/Standard	------	20µl
Reagent	1000µl	1000µl

Mixed and incubated for 10 min. at 20---25^0C. Absorbance of the sample and standard were measured against the reagent blank within 30 min(ΔA)

Calculation of the Protein Concentration

With standard-

$C=80\times \Delta A_{\text{sample}} / \Delta A_{\text{standard [g/l]}}$

3.4 Statistical analysis

The data obtained were first stored in Microsoft Excel -2007 (Microsoft Corporation, USA). Data were then analyzed by using STATA 11.0 (STATA Corporation, Texas, USA) and significant results were expressed as t-test ($p \leq 0.05$).

IMAGES

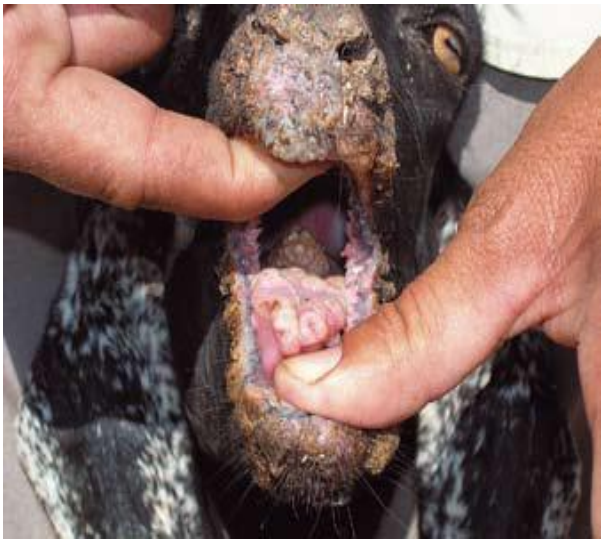

Figure: 2 Raised and flat circular lesions on tongue.

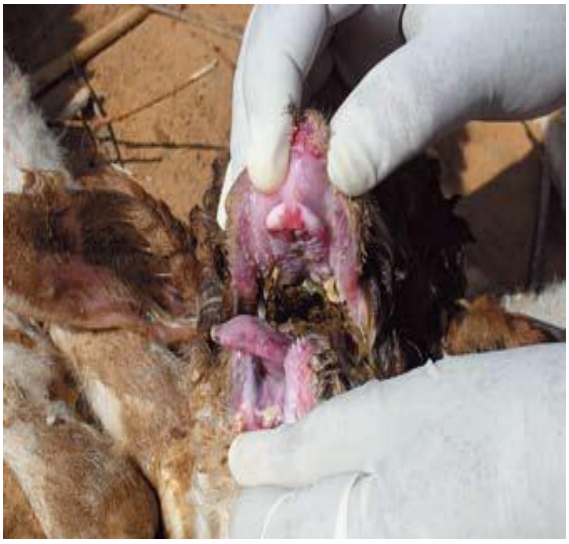

Figure: 3 Irregular non-hemorrhagic oral lesions.

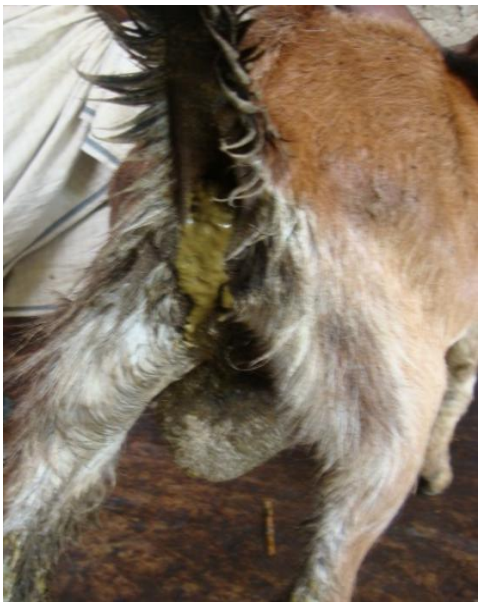

Figure: 4 Diarrhoea in goat.

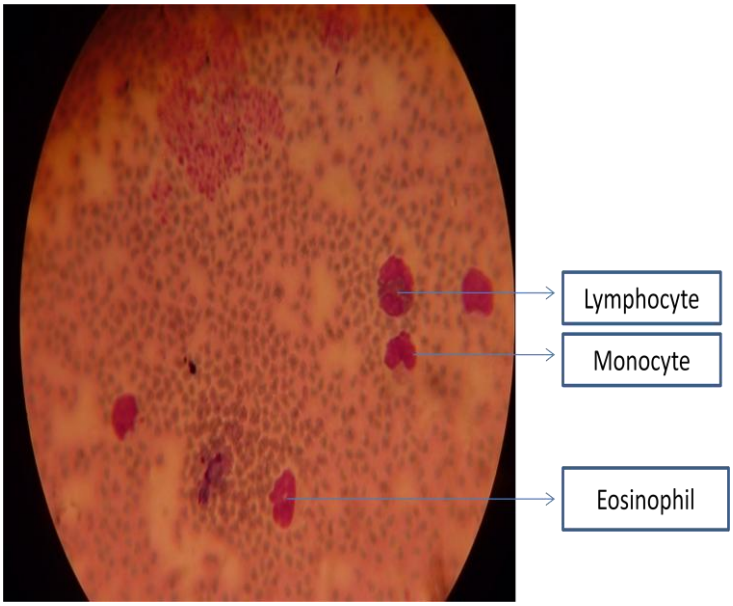

Figure: 5 Differential Leucocytes Count (DLC).

CHAPTER –IV

RESULTS

In this study, a total of 20 clinically identified PPR cases were thoroughly examined, haematological and biochemical parameters were analyzed. We found increased lymphocytes and decreased PCV %, TP and sodium level in the serum which has been shown in Table-2.

Table 2: Different hematological and biochemical parameters of PPR affected goats (N = 20).

Parameters	Mean ± SD	Reference value
ESR mm (1^{st} hour)	0 ± 0	0.0
PCV %	19.95 ± 7.14	22-38
Lymphocyte %	69.6 ± 7.92	50-55
Monocyte %	7.5 ± 3.23	0-4
Neutophil %	16.55 ± 7.9	30-48
Eosinophil %	4.8 ± 3.98	1-8
Basophil %	1.55 ± 0.94	0-1
TLC ($\times 10^3/mm^3$)	8.61 ± 1.13	4-13
Sodium (mmol/L)	104.21 ± 35.68	133.5-154
Potassium (mmol/L)	5.49 ± 1.42	4.6-9.8
Chloride (mmol/L)	110.5 ± 12.62	105-120
TP (gm/dl)	5.38 ± 1.05	6.4-7.4

*(Goatlink.com, 2009)

The table shows that hemato-biochemical parameters of PPR affected goats are changed from normal value. The mean ± SD PCV (19.95 ± 7.14 %), Lymphocyte (69.6 ± 7.92 %), Neutrophil (16.55 ± 7.9 %), Monocyte (7.5 ± 3.23 %), and TLC (8.61 ± 1.13$\times 10^3/mm^3$) respectively in the PPR-affected goats. We found that, lymphocyte and monocyte counts were higher in PPR affected goats than normal value. PCV decreased in PPR affected goats than reference value.

The study revealed that Sodium (mmol/L), Potassium (mmol/L), Chloride (mmol/L) and TP (gm/dl) were found 104.21 ± 35.68, 5.49 ± 1.42, 110.5 ± 12.62 and 5.38 ±1.05 respectively. In our study total protein level was decreased in PPR affected goats Sodium level was also decreased.

Table 3: Different hematological and biochemical parameters of PPR affected Male and Female goats.

Parameters	Male (n = 12) Mean ± SD	Female (n = 8) Mean ± SD	*p* value
ESR mm (1^{st} hour)	0 ± 0	0 ± 0	-
PCV %	18.16 ± 4.21	22.62 ± 9.84	0.18
Lymphocyte %	67.75 ± 7.22	72.37 ± 8.6	0.21
Monocyte %	8.5 ± 3.45	6 ± 2.32	0.09
Neutrophil %	18.03 ± 6.21	14.25 ± 9.9	0.3
Eosinophil %	4.17 ± 3	5.75 ± 4.06	0.33
Basophil %	1.5 ± 1	1.62 ± 0.91	0.79
TLC ($\times10^3/mm^3$)	8.51 ± 0.94	8.76 ± 1.42	0.64
Sodium (mmol/L)	115.57 ± 33.86	87.15 ± 33.15	0.08
Potassium (mmol/L)	5.77 ± 1.37	5.07 ± 1.48	0.29
Chloride (mmol/L)	112.11 ± 14.12	108.07 ± 10.48	0.5
TP (gm/dl)	5.21 ± 1.05	5.63 ± 1.07	0.4

The table shows that, PCV % (18.16 ± 4.21, 22.62 ± 9.84), Lymphocyte % (67.75 ± 7.22, 72.37 ± 8.6), Monocyte % (8.5 ± 3.45, 6 ± 2.32), Neutrophil % (18.03 ± 6.21, 14.25 ± 9.9), Eosinophil % (4.17 ± 3, 5.75 ± 4.06), TLC$\times10^3/mm^3$ (8.51 ± 0.94, 8.76 ± 1.42) in male and female PPR affected goats respectively. Where PCV %, Lymphocyte % and TLC ($\times10^3$ / mm^3) are higher in female than male PPR affected goats.

The study also revealed that, Sodium (115.57 ± 33.86, 87.15 ± 33.15 mmol/L), Potassium (5.77 ± 1.37, 5.07 ± 1.48 mmol/L), Chloride (112.11 ± 14.12, 108.07 ± 10.48 mmol/L) and Total Protein (5.21 ± 1.05, 5.63 ± 1.07 gm/dl) were found in PPR affected male and female goats respectively. Sodium and chloride were more in male than female but TP near about similar in PPR affected male and female goat.

Table 4: Different hematological and biochemical parameters of PPR affected goat according to breed category.

Parameters	Non Descriptive (n = 9) Mean ± SD	Jamnapari (n = 3) Mean ± SD	Black Bengal (n = 8) Mean ± SD
ESR mm (1^{st} hour)	0 ± 0	0 ± 0	0 ± 0
PCV %	17.55 ± 3.94	17.67 ± 5.85	23.5 ± 3.91
Lymphocyte %	69.88 ± 8.53	68 ± 8	69.87 ± 8.23
Monocyte %	7.66 ± 2.91	9 ± 1	6.75 ± 4.09
Neutophil %	15.44 ± 7.85	20 ± 8	16.5 ± 8.6
Eosinophil %	5.88 ± 5.08	2.33 ± 0.57	4.5 ± 3.02
Basophil %	1.11 ± 0.78	0.67 ± 0.57	2.37 ± 0.51
TLC ($\times 10^3/mm^3$)	8.48 ± 1.37	8.37 ± 0.51	8.85 ± 1.06
Sodium (mmol/L)	93.49 ± 16.41	153.86 ± 10.34	97.64 ± 15.33
Potassium (mmol/L)	4.88 ± 1.22	6.63 ± 0.49	5.76 ± 1.60
Chloride (mmol/L)	114.39 ± 14.79	109.7 ± 10.19	106.42 ± 10.68
TP (gm/dl)	5.65 ± 1.01	4.6 ± 1.25	5.37 ± 1.02

The table shows that, ESR mm1^{st} hour (0 ± 0, 0 ± 0 and 0 ± 0), PCV % (17.55 ± 3.94, 17.67 ± 5.85 and 23.5 ± 3.91), Lymphocyte% (69.88 ± 8.53, 68 ± 8 and 69.87 ± 8.23), Monocyte% (7.66 ± 2.91, 9 ± 1 and 6.75 ± 4.09), Neutrophil% (15.44 ± 7.85, 20 ± 8 and 16.5 ± 8.6), Eosinophil% (5.88 ± 5.08, 2.33 ± 0.57 and 4.5 ± 3.02), TLC$\times 10^3/mm^3$ (8.48 ± 1.37, 8.37 ± 0.51 and 8.85 ± 1.06) in PPR affected non descriptive, Jamnapari and Black Bengal goat respectively.

The biochemical parameters were Sodium (93.49 ± 16.41, 153.86 ± 10.34 and 97.64 ± 15.33 mmol/L), Potassium (4.88 ± 1.22, 6.63 ± 0.49 and 5.76 ± 1.60 mmol/L), chloride (114.39 ± 14.79, 109.7 ± 10.19 and 106.42 ± 10.68 mmol/L), Total Protein (5.65 ± 1.01, 4.6 ± 1.25 and 5.37 ± 1.02 gm/dl) in PPR affective Non Descriptive, Jamnapari and Black Bengal goats.

Table 5: Different hematological and biochemical parameters of PPR affected goat according to age ground.

Parameters	>1 Year (n = 8) Mean ± SD	4-12 Months (n = 12) Mean ± SD
ESR mm (1^{st} hour)	0 ± 0	0 ± 0
PCV %	16.83 ± 4.13	24.62 ± 3.65
Lymphocyte %	68.33 ± 8.59	71.5 ± 6.9
Monocyte %	8.33 ± 3.67	6.25 ± 2.05
Neutophil %	17.92 ± 7.77	14.50 ± 4.70
Eosinophil %	4.03 ± 3.44	5.87 ± 4.7
Basophil %	1.33 ± 0.88	1.87 ± 0.9
TLC ($\times 10^3/mm^3$)	8.53 ± 1.31	8.74 ± 0.85
Sodium (mmol/L)	81.24 ± 6.45	119.52 ± 13.38
Potassium (mmol/L)	5.52 ± 1.43	5.64 ± 1.50
Chloride (mmol/L)	108.99 ± 8.58	112.76 ± 7.52
TP (gm/dl)	5.13 ± 0.84	5.76 ± 1.27

The table shows that, PCV % (16.83 ± 4.13, 24.62 ± 3.65), Lymphocyte% (68.33 ± 8.59, 71.5 ± 6.9), Monocyte% (8.33 ± 3.67, 6.25 ± 2.05), Neutrophil% (17.92 ± 7.77, 14.50 ± 4.70), Eosinophil% (4.03 ± 3.44, 5.87 ± 4.7), TLC$\times 10^3/mm^3$ (8.53 ± 1.31, 8.74 ± 0.85) in PPR affected older (> 1 year) goats and young (4-12 months) goats respectively. Here PCV and lymphocyte are higher in young goat than older.

The study also revealed that Sodium (81.24 ± 6.45, 119.52 ± 13.38 mmol/L), Potassium (5.52 ± 1.43, 5.64 ± 1.50 mmol/L), Chloride (108.99 ± 8.58, 112.76 ± 7.52 mmol/L) and Total Protein (5.13 ± 0.84, 5.76 ± 1.27 gm/dl) were found in PPR affected older (> 1 year) goats and young (4-12 months) goats respectively. In biochemical TP, Sodium and Chloride were higher in young than older goat.

CHAPTER –V
DISCUSSION

TLC was increased in PPR affected goats and corresponding PCV (%) was also decreased that is agreed with (Aytekin *et al.*, 2011). It could be due to dehydration and anemia. Anemia was developed due to poor nutrition and irregular anthelmentic program. In differential leukocyte count percentage is varies from normal values. In which lymphocyte and monocyte counts were increased in PPR affected goats. It suggested that due to viral infection in host body try to eliminate the organism and give rise to such high lymphocyte monocyte but decreased neutrophil count.

In our study total protein level was decreased in PPR affected goat due to anorexia and off feeding which is similar to the findings of Kataria *et al.* (2007). Sodium level was also decreased due to dehydration and electrolytes loss.

5.1 Sex basis

The lower lymphocytes in males compared to females may be attributed to physiological stress response arising from their social behavior which consist of aggressiveness and hierarchical fights. Similar observation was found by Zapata *et al*. (2003). DLC value of PPR-goat was significantly associated with the sex (Rahman *et al*., 2011). Males are apparently more prone to the disease than females may be due to genetic factors.

TP was near about similar in PPR affected male and female goat. It is agreed with the findings of Aikhuomobhogbe and Orheruata, (2006). In case of sodium and chloride level female was lower than male.

5.2 Breed basis

PCV %, Lymphocyte % and TLC ($\times 10^3/mm^3$) in PPR affected Black Bangel goats were higher than PPR affected Jamnapari goats. It is reflected that, Black Bengel is more susceptible to PPR than Jamnapari. That is agreed with Mondal *et al.* (1995).

Sodium level was so much lower in Black Bengal and non descriptive than Jamnapari but TP (gm/dl) was lower in Jamnapari. It could be due to poor nutritional status of the animal.

5.3 Age basis

PCV % and Lymphocyte % was higher in PPR affected young goats (4 -12 months) than older (> 1 year). This finding is agreed to the findings of Kataria *et al. (*2007). PCV % is increase in young goat due to hemoconcentation. It is reflected that young are more susceptible to PPR than older that is similar with Radostits *et al*. (2007)

In biochemical analysis TP, Sodium and Chloride were higher in young than older goat. The same observation was found by Kataria *et al.* (2007). Sodium and Chloride level were higher in case of hemoconcentation.

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CHAPTER –VI
CONCLUSION

Presenting **c**linical signs are primary tools to presumptive diagnose the PPR in field condition but haematological and biochemical parameters can be useful to confirm the diagnosis. In PPR affected goats most of the value of haematological parameters was higher than normal value except neutrophil. In biochemical parameters, decreased total protein, sodium, potassium and chloride concentration was found in serum level. So, it can be used as diagnostic tools to diagnose PPR disease in goat.

Black Bengal goats are more susceptible to PPR than Jamunapari. Appropriate knowledge on the epidemiology of the virus and the disease has to be generated in order to develop a control strategy for Bangladesh.

CHAPTER –VII

LIMITATION

There were some limitations of this study. These are as follows:

1. The study period was very short and so the result may vary due to time constraint.
2. Sample sizes were very small.
3. Clinical signs basically were used to diagnose the case definition.
4. The hematological tests have been done manually. If auto analyzer is used more accurate results might be obtained.

CHAPTER –VIII
RECOMMENDATIONS

Finally hematological and biochemical values are an efficient tool for evaluate the patient is diseased or not by alteration the values from normal value as well as physiological status, metabolic disorders and management problems of the animal. The study suggests further intensive study on hematobiochemical parameters and globulin level assessment.

CHAPTER –IX
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APPENDIX

Recordkeeping sheet:

1) Date:----------

2) Case no:----------

3) Owners Name:----------

4) Address:----------

5) Occupation:----------

6)Breed:----------7)Age:----------8) Sex:----------

9) Color:----------10) Vaccination: yes/no------11) Deworming: yes/no---

12) Clinical findings:----------

Temperature:

Respiration:

Heart rate:

Observation:

13) Laboratory test (a.Hematological test):----------

TLC ($\times 10^{3/}mm^3$):

PCV (%):

ESR (mm in 1^{st} hour):

Lymphocyte (%):

Monocyte (%):

Neutrophil (%):

Eosinophil (%):

Basophil (%):

b. Biochemical test:

Sodium (mmol/L):

Potassium (mmol/L):

Chloride (mmol/L):

TP (gm/dl):

14) Diagnosis:

15) Treatment**:**