PATHOLOGICAL INVESTIGATION OF PESTE DES PETITS RUMINANTS (PPR) OUTBREAK IN A GOAT FARM AT BAGJANA, PANCHBIBI, JOYPURHAT DISTRICT IN BANGLADESH

A THESIS

BY

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SEMESTER : JULY – DECEMBER 2013

REGISTRATION NO.: 1205101
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HAJEE MOHAMMAD DANESH SCIENCE AND TECHNOLOGY UNIVERSITY DINAJPUR-5200

DECEMBER 2013

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DEDICATED
TO MY
BELOVED PARENTS

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December, 2013 *The Author*

ABSTRACT

The present study aimed at the investigation of a PPR outbreak in a small farm of Black Bengal goats in July-December 2013. A total of 74 goats of both sexes in a goat farm at Bagjana, Panchbibi, Joypurhat district were included in this investigation. The Farm had the history of recent introduction of goats purchased from a market. Out of 74 goats, 38 were suspected with natural PPR virus infection, 10 among which died and necropsy was conducted on 6 goats. All the dead goats were young goats of less than 12 month of age. The main clinical manifestation of the suspected goats were fever ($101^0 - 107^0$F), followed by dry muzzle, anorexia followed by serous to mucopurulent nasal discharge. Severe stomatitis with necrotic lesions in the buccal cavity, diarrhoea, dyspnea with cough, frothy salivation, conjunctivitis and depression were found. Grossly, emaciated and dehydrated carcass; erosions on pillars of rumen, severe congestion throughout the intestinal tract, zebra striping in ceco-colic junction, pale and moderately enlarged liver with indented gall bladder; congested and consolidated pneumonic lungs; enlarged, congested, and edematous lymph nodes throughout the body; brush paint hemorrhages in the heart; atrophied congested spleen were recorded. Histopathologically, chronic enteritis with large mononuclear cells infiltration in the villi and lamina propria; chronic tracheitis; chronic bronchointerstitial pneumonia consistent with accumulation of fibrin; depletion of lymphocytes with infiltration of macrophages in lymph node; splenic atrophy with depletion of lymphocytes were recorded.

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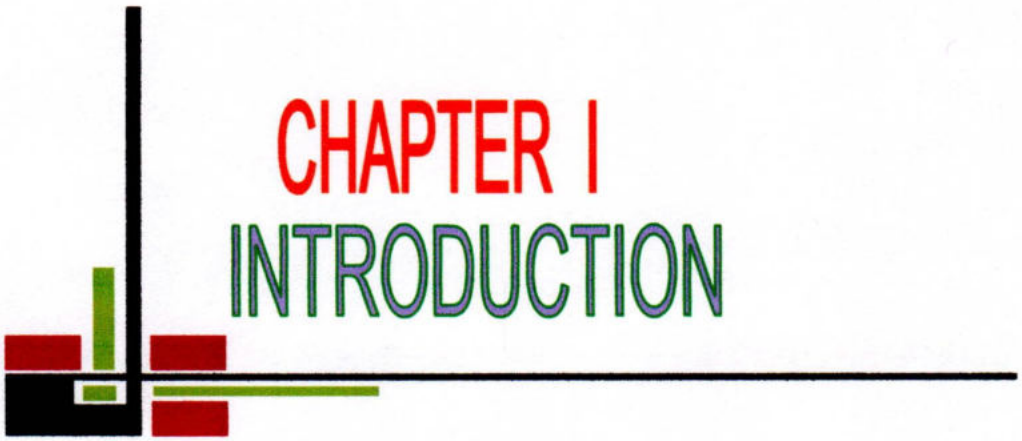
IHC	=	Immunohistrochemistry
i/m	=	Intramuscular
i/v	=	Intravenous
Ibs	=	Pound
L-Protein	=	Large protein
M	=	Molar
M-protein	=	Matrix protein
MAB	=	Monoclonal antibody
Mg	=	Milligram
Min	=	Minute
mix	=	Mixture
ml	=	Milliliter
mRNA	=	Messenger ribonucleic acid.
MS	=	Master of Science
N	=	Normal
N-protein	=	Nucleocapsid protein
OIE	=	International Office of Epizootics
P-protein	=	Phosphoprotein
PBS	=	Phosphate buffered saline
PCR	=	Polymerase chain reaction
PPR	=	Peste des petits ruminants
PPRV	=	Peste des petits ruminants virus
RNA	=	Ribonucleic acid
rpm	=	Rotation per minute
RP	=	Rinderpest
RPV	=	Rinderpest virus
RT	=	Room temperature

LIST OF ABBREVIATIONS AND SYMBOLS (Contd.)

S-ELISA		Sandwich enzyme-linked immunosorbent assay
Sec	=	Second.
TBE	=	Tri-borate EDTA
TCID (50)	=	Tissue Culture Infective Dose (50)
UV	=	Ultra Violet
wt.	=	Weight
w/v	=	Weight by volume
µg	=	Microgram
µl	=	Microlitre
^{0}C	=	Degree Celcius
^{0}F	=	Degree Fahrenheit
%	=	Percentage
<	=	Smaller than



CHAPTER I
INTRODUCTION

CHAPTER I

INTRODUCTION

The goat is the livestock that can be managed easily in Bangladesh for the purposes of meat, milk, skins etc. It is valuable particularly for low-income farmers, landless laborers and distressed women who cannot afford to rear cattle; hence it is called the "Cow of poor people'' in Bangladesh. At present, the approximate number of goats in Bangladesh is 33.5 millions (Samad, 2001). Peste Des Petits Ruminants (PPR) is an economically important transboundary viral disease of goats in Bangladesh (Kamaruddin and Islam, 2005).

PPR, caused by a *Morbillivirus* of *paramyxoviridae* family, is a fast-spreading disease of mainly domestic small ruminants (Paul *et al.,* 1979).PPR virus (PPRV) is an enveloped and pleomorphic virus particle with negative sense, single stranded, non-segmented RNA genome (Bellini *et al.*, 1994; Qzturk, 1999; Sinnathamby *et al.*, 2001). The genome encodes six structural proteins and two non structural proteins (Bellini *et al.*, 1994). PPRV may resist 60C for 60 minutes, stable between pH 4.0 and 10, may survive for long periods in chilled and frozen tissues and is susceptible to alcohol, ether, detergents and most disinfectants (Haffar *et al.*, 1999; Qzturk, 1999; Scott *et al.*, 2000; OIE, 2004). The virus can be cultivated in sheep and goat kidney cells (Losos, 1986)

PPR is primarily a disease of goats and sheep but buffaloes, camel and pigs are also susceptible to infection (Beaton, 1930; EMPRES, 1999). A case of clinical disease was reported in wildlife (Furley *et al.*, 1987). The American white-tailed deer was infected experimentally (Saliki, 1998).

CHAPTER II

REVIEW OF LITERATURE

The literature pertinent to the present study on investigation of a PPR outbreak is reviewed in this part of thesis:

2.1 PESTE DES PETITS RUMINANTS (PPR)

Peste Des Petits Ruminants (PPR), a highly contagious animal disease, is caused by the peste des petits ruminants virus (PPRV) in the genus *Morbillivirus* of the family *Paramyxoviridae* (Diallo *et al.*, 2007). It is known as goat plague which is a severe, fast-spreading disease of mainly domestic small ruminants characterized by the sudden onset of depression, fever, discharges from the eyes and nose, sores in the mouth, disturbed breathing and cough, foul-smelling diarrhoea and death (EMPRES, 1999). It is also known as pseudorinderpest, pests of sheep and goat, kata, pneumoenteritis complex, stomatitis-pneumoenteritis syndrome, goat catarrhal fever (Chakrabarti, 2002). This infection is responsible for high morbidity and mortality in sheep and goats and in some small wild ruminant species (Diallo *et al.*, 2007) and it is a disease of increasing importance in Africa and Asia wherever small ruminants form an important component of agricultural food production (EMPRES, 1999).

2.1.1 ETIOLOGY AND CLASSIFICATION

PPRV is classified as the fourth member of the genus *Morbillivirus* of *Paramyxoviridae* family (Paul *et al.*, 1979). At least four distinct genetic lineages (lineages I, II, III and IV) of PPRV with different geographical distributions circulate among small ruminants in regions of endemicity (Shaila *et al.,* 1996b; Dhar *et al.*, 2002) and the virus isolates from Asia over the past 2

The properties of the two hepted repeats (HRI and HR2) of the PPRV fusion protein (F) were described to obtain insights into the mechanism by which these repeats influence PPRV-mediated cell fusion (Rahaman *et al.*, 2003).

2.1.3 ANTIGENICAL RELATIONSHIP

The virus is closely related antigenically to rinderpest virus (RPV), human measles virus (MV) and canine distemper virus (Barrett *et al.*, 1993). Originally PPRV was considered as a variant of RPV adapted to small ruminants but the two viruses have separate epizootiologic cycle in natures and each exists in its own right (Dardiri *et al.*, 1976; Taylor and Abegunde, 1979).

2.1.4 CULTURAL CRITERIA

The virus can be cultivated in sheep and goat kidney cells (Losos, 1986). However, this virus grew poorly in tissue culture (Mahapatra *et al.*, 2006). PPRV form noticeable plaque (1-3 mm diameter) in Vero cells monolayer cultures grown in Petri dishes (Durojaiye and Taylor, 1984). To determine cell culture spectrum, calf kidney, lamb testicle, chick embryo fibroblast; BHK and Vero cells can be inoculated with reference as well as local isolates of PPR (Saeed *et al.*, 2004).

2.1.5 EPIDEMIOLOGY

2.1.5.1 PHYSIOCHEMICAL PROPERTIES.

PPRV may resist 60^0 C for 60 minutes and is stable between pH 4.0 and 10.0 (Haffar *et al.*, 1999; Ozturk, 1999; OIE, 2004). Virus may survive for long periods in chilled and frozen tissues (Scott *et al.*, 2000) PPRV is susceptible to alcohol, ether, detergents and also to most disinfectants (Scott *et al.*, 2000; OIE, 2004).

These physiochemical characteristics of the virus indicate that it is closely related to measles, rinderpest and canine distemper viruses (Gibbs, *et al.*, 1979)

2.1.5.2 HOSTS RANGE

2.1.5.2.1 Natural hosts

The host range has been extensively studied using reverse genetics (Bailey *et al.*, 2007) PPR is primarily a disease of goats and sheep (Ozkul *et al.*, 2002) and goats are usually more severly affected than sheep (Singh *et al.*, 2004). The clinical symptoms of the disease were firstly described in goats (Beaton, 1930). Cattle act as dead-end hosts for PPRV and show no clinical signs of infection (Ozkul *et al.*, 2002). Buffaloes, camel and pigs are also susceptible to infection but do not exhibit clinical sings (EMPRES, 1999). A case of clinical disease was reported in wildlife resulting in deaths of gazelles (Gazella dorcas), ibex (Capra ibexnubiana), gemsbok (Oryx gazelle) and Laristan sheep (Ovis orientalis laristanica) (Furley *et at.*, 1987) and the author also found the sub-clinical involvement of Nilgai (Tragelaphinae). The similar virus was found the sub-clinical involvement of Nilgai (Tragelaphineae). The similar virus was found in marine mammals (Barrett *et al.*, 1993). It can affect a broad range of species including some antelopes (Roeder and obi, 1999).

2.1.5.2.2 Experimental hosts

Pigs could be subclinically infected with PPRV by inoculation or contact with infected goats but pigs are not considered important in the epidemiology of PPRV (Nawathe *et al.*, 1979). The American white-tailed deer (Odocoileus virginianus) was infected experimentally (Hamdy and Dardiri, 1976). Experimental inoculation of a Morbillivirus strain PPR Egypt-87 was capable of reproducing plague of small ruminants in goat if inoculated i/m and it could

induce neutralizing antibody in 45 Rabbits inoculated i/m or i/v (Ismail *et al.*, 1991). In 1950s, Disease and death were recorded in calves experimentally infected with PPRV (Shaila *et al.*, 1989).

2.1.5.3. PREVALENCE, MORBIDITY, MORTALITY AND CASE FATALITY RATE

Prevalence is higher in Black Bengal Goats (67.24%) than in Jamunapari breed (32.76%) (Mondal *et al.*, 1995). Variation in prevalence is probably related to the intensity of trade of illegally imported small ruminants (Al-Naeem *et al.*, 2000). The morbidity and mortality from PPR can be as high as 100% and 90% respectively and when associated with other diseases such as capripox, mortality can be 100% (Dhar *et al.*, 2002). Both field and laboratory observations indicate that case fatality rates are much higher in goats (55-85%) than in sheep (less than 10%) (Opasina and putt, 1985; Abu Elzein *et al.*, 1990 Obi *et al.*; 1990) and young animals of both species are at higher risk than adult (Rao *et al.*, 1998; atta-ur-Rahman *et al.*; 2004). In Bangladesh ,the morbidity is 100% and mortality varied from 50 to 90% depending on the management practices and environmental condition (sil *et al*, 1995) . In India PPR causes the high rates of mortality and morbidity in infected sheep and goats (Singh *et al.*; 2004). An outbreak of PPR occurred at the Koma Village in the Ranibandh Block of the Bankura District; West bangal showed a morbidity of 18% and a case fatality of 35.3% while majority of the deaths involved 1 to 6 month old animals (Debasis and Mousumi, 2002). In Kolkata, India a total of 866 goats were studied to determine the prevalence of PPR from July 2002 to 2003 where the overall prevalence of PPR was 30.7% mortality rate was 24.0% and the disease was highly prevalent during the winter season (51.7%) in the 5-12 months age group (65%) in the Jamunapari breed (52%) (Saha *et al.*, 2005). There was a severe outbreak of pest of small ruminants virus at the regional goat breeding from in Tripura ,India where

positive, none of the goat samples were positive to PPRV antibodies and study in Tamil Nadu showed overall seropositive rate 28.81%(Krisna *et al.*, 2001; Kanani *et al.*, 2006; Brindha *et al.*, 2007). Blood samples obtained at an abattoir in Faisalabad, Pakistan between April and June, 1989 showed that 27-28% of sheep samples and 31.10% of goat samples were positive for PPR antibodies (Tahir *et al.*, 2000). A serological survey of antibodies against PPR virus in Sultanate of Oman showed that 24.5% sheep and goats was positive to PPR(Taylor *et al.*,1990).in Ethiopian camel, the surveillance results showed a global sero-prevalence of 7.8% for PPRV antibodies and 21.3% for RPV antibodies (Roger *al et.*, 2001). A study representing the first survey for serum antibodies against PPR and RP viruses, in sheep and goats in the kingdom of Saudi Arabia in which generally the prevalence of PPRV antibodies in both species was 2.3% while that of RPV was 4.3% (Al-Afaleq *et at.*, 2004).

2.1.5.5 TRANSMISSION

For PPR to spread, close contact between infected and susceptible animals is needed (Ozkul *et al.*, 2002). There are several means of transmission between animals: secretions and excretions of the sick animals (Susan and Aiello, 1998); inhalation of aerosols produced by sneezing and coughing of infected animals (Radostits *et al.*, 2000); direct contact with feces (Shanthikumar *et al.*, 1998); it is suspected that infection materials can also contaminate water and feed troughs and bedding turning them into additional sources of infection (EMPRES, 1999). No carrier state is known to exist (Taylor, 1984; OIE, 2004). In Nigeria, experimentally the virus has been transmitted nasally, orally, subcutaneously and intra-tracheally (Whitney *et al.*, 1967; Rowland *et al.*, 1971). Virus is present in semen,embryos and feeding milk from infected animals to kids or lambs can spread infection (OIE,2004).

2.1.5.6. RISK FACTORS

The appearance of clinical PPR may by associated with introduction of recently purchased sick animals from markets (Radostits *et al.*, 2000), contact in a closed/village flock with sheep and/or goats that had been sent to market but returned unsold, trade in small ruminants at markets where animals from different sources are brought into close contact with one another and affords increased opportunities for PPR transmission, as does the development of intensive fattening units (EMPRES,1999), change in weather such as most outbreaks are associated with the winter seasons as compared to other seasons of the year (Sil *et al.*,1995; OIE,2004).In clinical records of natural infections of PPR among West African Dwarf (WAD) goats the occurrence of more cases of PPR were recorded during the dry months of December to January (Okoli,2003). Poor nutritional status, stress of movement and concurrent parasitic and bacterial infections enhance the severity of clinical signs (Taylor, 1984). The appearance of clinical PPR may also be associated with history of recent movement or gathering together of sheep and/or goats of different ages with or without associated changes in housing and feeding; contact with animals through shared grazing, water and/or housing; a change in husbandry (e.g. towards increased intensification) and trading practices; in endemic areas, animals over four months and up to 18 to 24 month of age (EMPERS, 1999).

2.1.6. PATHOGENESIS

Virus appeared to be released through the microvilli of the epithelial cells (Bundza *et al.*, 1988). The pathogenic determinants of PPR have been extensively studied using reverse genetics (Bailey *et al.*, 2007). The PPRV has a particular affinity for lymphoid tissues and epithelial of the GI and respiratory tracts, where it produces characteristic lesions (Susan and Aiello, 1998). It is suggest that death is consequence of the combined effects of the

pathology in the two systems (Smith *et al.*, 1974). After replicating in the upper respiratory tract, the virus spread to the local lymphatic tissues (House *et al.*, 1992). Replication of virus in local lymphnodes produces primary viraemia that results in the spread of virus to other lymphoid tissues and other organs including skin, kidney and GIT (Haffar *et al.*, 1999). In these various organs, the virus replicates in the endothelial cells, epithelial cells and monocytes /macrophages (Sergeny *et al.*, 1992). The ability of the PPR viruses-affected cells to fuse probably leads to the formation of multinucleated giant cells, a pathognomonic hallmark of Morbillivirus infection (Sata *et al.*, 1986; Sergeny *et al.*, 1992). The secondary viraemia is associated with the onset of prodormal phase of infection (Rossiter *et al.*, 2000). The destructive phase of the virus on lymphocytes is suggestive as a cause of lymphopenia (Scott *et al.*, 2000).

2.1.7. INCUBATION PERIOD

Zalso described that the period is of 2~10 days, most commonly 4-5 days (EMPRES, 1999).

2.1.8. CLINICAL FEATURES

2.1.8.1. Features in natural cases

Mainly the disease is characterized by three symptoms; discharges (nasal, ocular and oral), diarrhoea and death; hence it is called 3D-disease (Islam *et al.*, 2003). Where diarrhoea is not an obvious presenting sign, the insertion of a cotton wool swab into the rectum may reveal evidence of soft feces which may be stained with blood (Roeder and Obi, 1999).

The other symptoms are high fever, erosive stomatitis, pneumonia and gastroenteritis (Radositits *et al.*, 2000). All these clinical signs, apart from the respiratory symptoms, are very similar to those of Rinderpest (Diallo, 2003).

In depression, high fever (41 degrees C), anorexia, mucopulurent nasal discharge, erosive and necrotic stomatitis (dental pads; hard palate and cheeks), diarrhoea and dehydration (Abdollahpour *et al.*, 2006).

PPR occurs mainly in three forms per-acute, acute and sub-acute (Olaleye *et al.*, 1989b). Peracute and acute forms of the disease are seen in 4 phases including incubation, prodromal, pneumonic and diarrhoea / death (Radostits *et al.*, 2000). The affected animal stands apart with impaired appetite accompanied with poor rumination and constipation. Two to five days after onset of fever, mucosal erosion appears as pin heads of necrotic epithelium on the mucous membrane lining of the mouth, nasal passage and urogenital tracts (Opasina, 1980). After 2 or 3days of mucosal erosion, the fever regresses and pneumonia accompanied with diarrhoea (Scott, 1988). The peri-oral and peri-nasal areas are encrusted with muco purulent discharges (Obi *et al.*, 1990). The feces may be mucoid blood-tinged (Bundza *et al.*, 1988; Obi *et al.*, 1990). The early pyretic phase in followed by diarrhoeic phase with watery faeces (Kumar *et al.*, 2002). Death may usually occur within a week of the onset of illness (Olaleye *et al.*, 1989) .An outbreak of PPR occurred at the Bankura District of West Bengal did not exhibit signs of disease but most of the affected goats demonstrated poor growth as a result of persistent anorexia, severe diarrhoea, and pneumonia (Debasis and Mousumi,2002). An outbreak of pest of small ruminant's virus (PSRV) in approximately 100 goats was diagnosed in Rawalpindi city, Pakistan in June 1997 where clinical signs included serous ocular and nasal discharge, fever, erosive lesions in mouth, diarrhoea and pneumonia (Hussain *et al.*, 1998). Characteristic clinical signs may also be found in sheep. There is development of fever, erosive stomatitis, profuse diarrhoea, rapidly distress followed by collapse and death (Sergeny *et al.*, 1992). Dyspnea to cough may also be found (Kennerman and Senturk,

2000).Clinical signs with oculonasal discharge in goats were usually comparable but less severe in sheep (Kumar *et al.*, 2002)

2.1.8.2. Features in experimental cases

In experimental infection, the goat showed the 5 phases of PPR infection cycle which included incubation period, prodromal phase, pneumonic phase, diarrhoeic phase and prostration and /or death (Khan *et al.*, 2005). Goats and sheep with intra-nasally inoculated Malig-Yemen strain of PPR virus developed fever, nasal discharge, oral erosions, cough and diarrhoea (Bundza *et al.*, 1988).

The illness in sheep was initially manifested by low-grade pyrexia followed by; congestion of the conjunctival mucous membrane, scanty oculo-nasal discharge and change in the fecal consistency from normal pellety to semi-solid appearance; however an increase in pulse rate, respiratory rate and rumen motality was also observed (Raghavendra *et al.*, 2000). Erosion of the buccal and gingival mucosa, enlargement of the lymph nodes may be seen (Barhoom *et al.*, 2000).

2.1.8.3. Features in migratory cases

The main clinical manifestations of the PPR like outbreaks in migratory flocks of Gadd goats and sheep in Mandi district of Himachal Pradesh were increased body temperature, dry muzzle, anorexia followed by serous to mucopurulent nasal discharge, severe stomatitis with necrotic lesions in the buccal cavity, diarrhoea and respiratory distress (Wadhwa *et al.*, 2002).

2.1.9 PATHOLOGICAL FEATURES

2.1.9.1 Necropsy lesions

The necropsy findings of an outbreak of a dual infection of PPR and of virus (ORFV) in goats in Shahjahanapur, India, during October 2004 are described (Saravanan *et al.*, 2007). Postmortem examinations of an outbreak of PPR, occurred at the koma village in the Ranibandh Block of the Bankura District, west Bengal, during May-june 1999 revealed congestion and partial consolidation of the lungs, inflammation of mediastinal and mesenteric lymph nodes, as we; as congestion of the abomasum and ileo-caecal junction (Debasis and Mousumi, 2002). The goat, infected both naturally and experimentally with PPR virus revealed emaciated and dehydrated carcass (Khan *et al.*, 2005); characteristic necropsy lesions usually seen in the digestive and respiratory systems, but could be seen in other systems as well as (Saliki, 1998).

2.1.9.1.1 Digestive system

Lesions in the digestive system include inflammatory and necrotic lesion in mouth and gastrointestinal tract (DEFRA, 2005). Erosive stomatitis in inside of lower lip and adjacent gum, the cheeks near the commissures, on the ventral surface of the tongue; in severe cases, the lesions may extend to the hard palate and pharynx (Susan and Aiello, 1998). Liver was moderately enlarged and pale (Alcigir *et al.*, 1996; Yener *et al.*, 2004). Indented gall bladder may be found (Wadhwa *et al.*, 2002). Rumen, reticulum, and omasum rarely have lesions; erosions on pillars of rumen, congestion of the abomasum and ileo-caecal junction and abomasum often oozes blood (Susan and Aiello, 1998; Debasis and Mousumi, 2002). Small intestine lesions are usually moderate and include extensive necrosis of Peyer's patches resulting in severe ulceration (Saliki, 1998). Large intestine features are characterized by congestion at

cecocolic junction resulting in the characteristic "Zebra-striped" appearance (Susan and Aiello, 1998; DEFRA, 2005).

2.1.9.1.2 Respiratory system

Small erosions and petechiae were visible on nasal mucosa, turbinates, larynx and trachea while pleuritis was seen in lungs, resulting in hydrothorax (Saliki, 1998). Respiratory tract contained frothy exudates (Bundza *et al.*, 1988; Islam *et al.*, 1996; Pawaiya *et al.*, 2004) and congested and/or consolidated pneumonic lungs were a consistent feature (Islam *et al.*, 2001.)

2.1.9.1.3 Other systems

Atrophied congested spleen (Khan *et al.*, 2005) and brush paint haemorrhages in the heart may be found (islam *et al.*, 2001). Enlarged, congested and edematous lymph nodes throughout the body and erosive vulvo-vaginitis may exist (Saliki, 1998).

2.1.9.2 Microscopic lesions

Microscopically, oral cavity, intestine, trachea, lungs, spleen and lymphnodes reveal most severe changes (Pawaiya *at al.*, 2004).

2.1.9.2.1 Digestive system

The histopathologic findings of natural PPRV infection include erosive-ulcerative stomatitis, syncytial cells containing bodies in the tongue and the buccal, labial, soft palate mucosa; eosinophilic intracytoplasmic and /or intranuclear inclusions in epithelial cells lining if the abomasal mucosa ,ileum, multifocal ,coagulation necroses and conspicuous syncytial cells in the liver (Toplu, 2004, kul *et al.*,2007)

2.1.9.2.2 Respiratory system

The histopathologic findings include fibrino-necrotic tracheitis, bronchointerstitial pneumonia, conspicuous, syncytial cells in the pulmonary alveoli and cytoplasmic inclusion bodies im epithelial cells of the bronchi and bronchiole (Toplu, 2004; Kul *et al.*, 2007). Chronic infection is characterized by giant cell pneumonia, which is sometimes complicated by broncho-pneumonia (Olaleye *et al.*, 1989b; Islam *et al.*, 2001). In acute cases, lungs show fibrino-purulent pneumonia (Islam *et al.*, 2001; Kumar *et al.*, 2004), often sero-fibrinous pneumonia (kumar *et al.*, 2004). Lungs also show broncho-interstitial changes and presence of intracytoplasmic or intranuclear eosinophilic inclusions in alveolar macrophages and syncytial cells (Islam *et al.*, 2001; Kumar *et al.*, 2004; Pawaiya *et al.*, 2004).

2.1.9.2.3 Other systems

Depletion of lymphocytes are found in lymphnodes occasionally with syncytia formation (Islam *et al.*, 1996; Islam *et al.*, 2001; Kumar *et al.*, 2004). Eosinophilic intracytoplamic and/or intranuclear inclusions may be observed in epithelial cells lining the renal pelvis (Kul *et al.*, 2007).

2.1.10 IMMUNITY TO PPRV

Morbillivirus infections have been known for a long time to be associated with an acute immunosuppression in their natural hosts (Kerdiles *et al.*, 2006). It was possible to classify the animals into high and low responders according to the response with clinical presentation, pulmonary lesions and mortality pattern of goats (Olaleye *et al.*, 1989a). Yet the immunized goats develop both humoral and cell-mediated immune responses (Sinnathamby *et al.*, 2001). The level of maternal immunity is fairly high (1:50 to 1:2000 per 0.1 ml of serum) in kids; eight hour after birth, the level declines at a steady rate and at the end

of the 12th week of age tires are 1:2 which may extends up to 17 weeks of age (Ara *et al.*, 1989). Maternal antibodies in young animals are detectable up to 6 months but fell below the protection threshold level at 3.5 and 4.5 month in kids and lamb, respectively (Awa *et al.*, 2002). Animals and herds with detectable antibodies to PPR virus might be relatively immune to rinderpest, and animals with detectable antibodies against PPR failed to produce antibodies when vaccinated with rinderpest virus (Haroun *et al.*, 2002> Animals once recover from PPR remained resistant to the subsequent infection (Sil *et al.*, 1995).

2.1.11. PUBLIC HEALTH SIGNIFICANCE

There are no known health risks to humans working with PPRV as no report of human infection with the virus exists (OIE, 2004).

2.1.12. DIAGNOSTIC APPROACH

2.1.12.1. Collection of specimen

specimen can be collected on live animals: swabs of the eye (conjunctival sac), nasal secretions, and mouth and rectal lining, clotted and whole blood (with EDTA anticoagulant) or at post-mortem: fresh and preserved samples of tonsil, tongue, spleen, lungs, trachea, lymph nodes, affected areas of the alimentary tract mucosa (OIE, 2004).

2.1.12.2. Approach other than RT-PCR

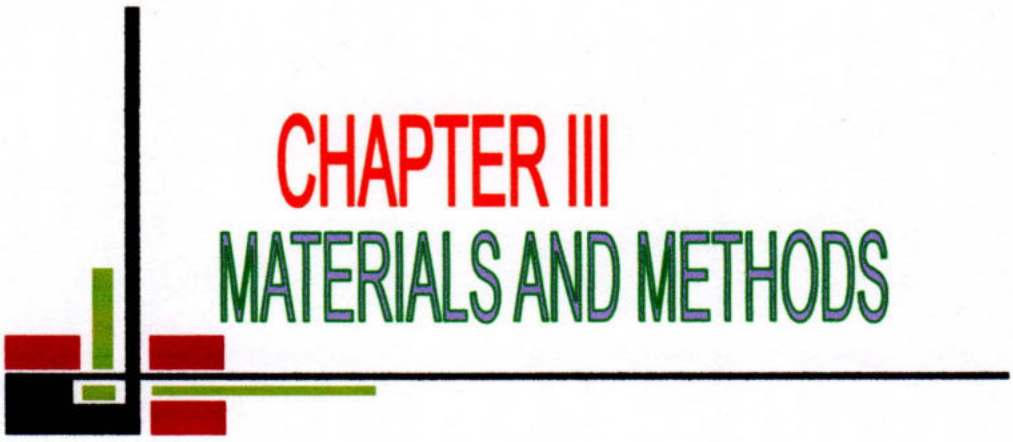
The routine diagnosis of PPR is based on clinical examination, gross pathology, histopathologic findings and laboratory confirmation which include tests for the detection of PPR antigen and PPR antibody, viral nucleic acid hybridization and recently polymerase chain reaction (Diallo *et al.*, 1995). Agar gel immunodiffusion techniques are either time-consuming, labour

intensive, insensitive, insensitive or expensive to perform but until recently, laboratory diagnosis relied on crude agar gel immunodiffusion tests (Scont *et al.*, 1986; Diallo *et al.*, 1995). The supernatant prepared from mesenteric lymph nodes was subjected to counter immunoelectrophrosis with Peste des petits ruminants hyperimmune serum where out of the 250 samples, 47.34% samples of Damani and 43% of Teddy giats were positive for PPR and in both the breeds, 47.05% males and 42.50% females were positive for PPR and in both the breeds, 47.05% males and 42.50% females were positive with age wise, results showed that young animals gave higher positive percentages than older oner ones (Atta-ur-Rahman *et al.*, 2004). PPRV antigen was detected in conjunctival epithelial cells obtained from goats in the early or late stage of the disease by the use of specific mAb to PPRV in an immunofluorescent antibody test (IFAT) (Sumption *et al.*, 1998). Animal inoculation for diagnosis of PPR is expensive and virus isolated in cell culture infrequently produces typical symptoms (Benazett, 1973). It could be difficult to explain the result of animal inoculation without supporting serology tests (Whitney *et al.*, 1967; Mann and isoun, 1974). After standardization, the allergic test may turn out to be a useful tool to detect immune animals in a flock (Yedloutschning and stone, 1974). The results of the immunohistochemistry (IHC) study suggested that indirect immunoperoxidase could be an alternative method in the absence of more sophisticated methods of laboratory diagnosis of PPR virus infection in goats (Kumar *et al.*, 2004). The serum beutralization test (SNT) in cell cultures is by far the most specific test for rinderpest was being used for PPR (Bourdin, 1977) but so far it has not been possible to use SNT in microtitre systems. The Complement fixation text (CFT) was used by Nduaka and Ihemelandu, (1975) to assess the immune response in goats vaccinated with chloroform-treated infected tissues. A sandwich ELOSA (S-ELISA) text using PPR specific monoclonal antibody (clone 4G6) to an epitope of nucleocapsid protein has been developed (Singh *et al.*, 2004). Competitive and immunocapture ELISAs

and differential immunohistochemical staining of tissue sections using monoclonal and polyclonal immunohistochemical staining of tissue sections using monoclonal and polyclonal antibodies are also being used (Anderson *et al.*, 1991; Libeau *et al.*, 1994; Saliki *et al.*, 1994a; Saliki *et al.*, 1994b). A MAb based S-ELISA was developed for specific detection of petits ruminants virus ,and compared with virus isolation in Vero cell cultures using 89 paired tissue and secretion samples from six experimentally infected goats, where S-ELISA was significantly more sensitive. (71.9% versus 65.2%) (Saliki *et al.*, 1994b).

An outbreak of PPR occurred at the Koma Villlage in the Ranibandh Block of the Bankura District, West Bengal, during 1999 in sheep and cattle was confirmed to be PPR on the basis of immunocapture sandwich ELISA (Debasis and Mousumi, 2002). A simple dot-enzyme-linked immunosorbent assay (dot- ELISA) using nitrocellulose membrane as solid support was developed for detection of viral antigen in caprine and ovine clinical materials (Saravanan *et al.*, 2006). Cloning and expression of nucleoprotein of nucleoprotein of PPRV in bacullo virus for use in serological diagnosis is also developed (Ismail *et al.*, 1995).

Nucleic acid hybridization, using either radiolabelled or biotinylated cDNA probes, has been used to identify these viruses which greatly speeded-up differential diagnosis (Diallo *et al.*, 1989; Pandey *et al.*, 1992)



CHAPTER III
MATERIALS AND METHODS

CHAPTER III

MATERIALS AND METHODS

The research work was performed in the Department of Pathology, Faculty of Veterinary and Animal Science, Hajee Mohammod Danesh Science and Technology University, Dinajpur, 2013. The following methods were followed in the experiment.

The major works of the present research include:

Investigation of a PPR outbreak by:

i. Epidemiological study
ii. Clinical signs and pathological study

3.1. FARM AND GOATS

A Total of 74 Black Bengal goats of a Goat farm of Bagjana, Panchbibi, Joypurhat District in Bangladesh were included in this investigation, of which 38 were suspected to be naturally infected with PPRV and 10 among which died.

3.2 QUSTIONNAIRE

For collection of date on the history and clinical signs, a questionnaire (enclosed in annexure) was prepared during the outbreak and the details were recorded from the attendant.

3.3 CLEANING AND STERILAZATION OF REQUIRED GLASSWARE AND PLASTICWARE.

New and previously used glass and plastic-ware dipped in 2% sodium hypochlorite solution and left there until cleaned. After overnight soaking in a

housed dish washing detergent solution (`Trix; Reckit Benckiser), the glassware cleaned by brushing and washed thoroughly in running tap water and rinsed four times in distilled water. The cleaned glassware were then dried on a bench at room temperature or in an oven at 50-70° C. The graduated pipettes were taken in alarge cylinder filled with water, washed thoroughly by continuous filling and removal of water the cylinder and finally washed 4 times in the same manner using distilled water. The pipettes were dried in an oven at 50°C. The graduated pipettes were cotton plugged at the neck and placed in a canister. The petridishes were wrapped with brown paper. These glassware were usually sterilized by dry heat at 160° C for one and half four in an oven. However, the bottles with plastic caps or rubber lined aluminum caps and the plasticware were sterilized by autoclaving for 20 minutes at 121° C under 15 lbs pressure per sq. inch. During autoclaving the caps were loosely fitted on the bottles, after autoclaving the glassware were immediately dried in an oven at 50-70° C and the caps of the bottles were tightened after cooling. All sterilized glassware and plastic-ware were kept in dust free place for further use.

3.4 PATHOLOGICAL STUDIES

3.4.1 Necropsy of goats and collection of samples

Necropsy of 6 dead goats was performed. At necropsy, gross tissue changes were observed and recorded carefully. The samples of trachea, lung, spleen, lymph node, intestine, kidney, stomach, heart and liver containing lesions were collected for histopathological study and fixed in 10% neutral buffered formalin until use.

3.4.2 Preparation of 10% neutral buffered formalin

37-40% formalin	100.00 ml
Distilled water	900.00 ml
Sodium phosphate monobasic	4.0 gm
Sodium phosphate dibasic (Anhydrous)	6.5 gm

3.4.3. Histopathology

Formalin fixed samples containing lesions were processed for paraffin embedding, sectioning and staining with hematoxylin and eosin according to standard method of Luna (1968) for histopathological study.

3.4.3.1.

a) The fixed tissues were trimmed properly using scalpel or handle and blade.
b) The trimmed tissues were further fixed for 24 hrs.
c) Tissues were kept in running tap water for overnight to washout formalin.
d) The tissues were dehydrated in ascending grades of alcohol using 50%, 70%, 80%, 95% and three changes of absolute for one hour in each.
e) Then sections were cleared in chloroform by tow changes, one and half hour for each.
f) The samples were then embedded with molten paraffin wax at 56^{0}C ; tow changes, one and half hour for each, and then paraffin block was prepared using a template.
g) Then the tissues were sectioned with a microtome at 5-µm thickness.

h) The section were allowed to spread on warm water bath (45^0C) and taken oil and grease free glass slides. A small amount of gelatin was added to the water bath for better adhesion of the section to the slide.

i) The section containing slides were air dried and kept in cool place until staining.

3.4.3.2 Routine hematoxylin and eosin staining procedure of the sectioned tissues

a) The sectioned tissues were deparaffinized in three changes of xyline (three minutes in each).

b) Then the sectioned tissues were rehydrated through descending grades of alcohol for two minutes; 80% alcohol for two 70% alcohol far two minutes) followed by distilled water for five minutes.

c) The tissues were stained with Harris hematoxyline for fifteen minutes.

d) Washed in running tap water for 10-15 minutes.

e) Then the tissues were differentiated in acid alcohol by 2 to 4 dips (1 part HCI and 99 parts 70% alcohol).

f) Washed in tap water for five minutes followed by 2-4 dips in ammonia water until sections were bring blue.

g) Stained with eosin for one minute.

h) Differentiated and dehydrated in alcohol (95% alcohol: three changes, 2-4 dips each; absolute alcohol: three changes 2-3 minutes for each).

i) Cleaned in xyline: three changes (five minutes each).

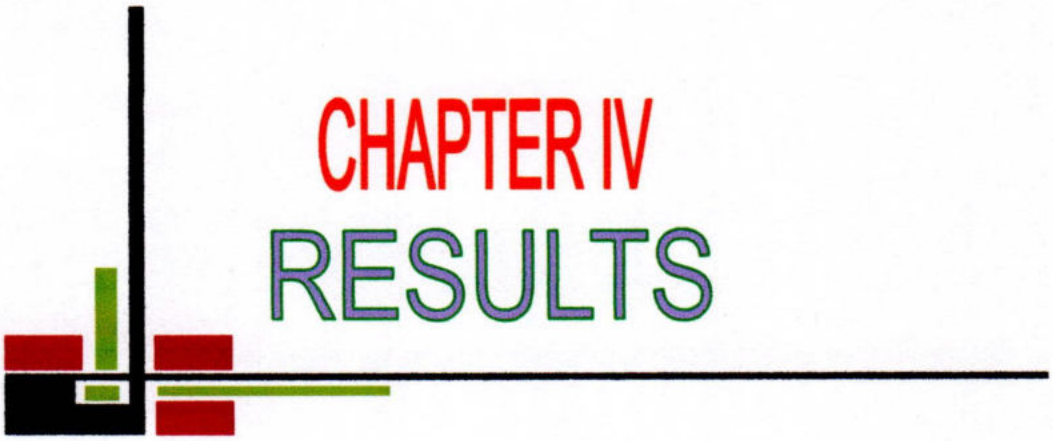
j) Finally, the sections were mounted with cover slip using DPX.

3.4.3.3. Photomicrography

Photomicrograph was taken at the Department of Pathology using photomicrographic camera (Olympus PM-C 35 Model).

3.4.3.4. Date interpretation

At the end of the study period all findings were complied, scrutinized and analyzed for comprehensive interpretation.



CHAPTER IV
RESULTS

CHAPTER 4

RESULTS

A total of 74 Black Bengal goats in the farm of Bagjana, Panchbibi ,Joypurhat District in Bangladesh were included in this investigation, of which 38 were suspected to be naturally infected with PPRV and 10 died. The results of the present study are described below.

4.1 EPIDEMIOLOGICAL FEATURES

This part of the study was completed by filling a questionnaire where details on the history and other epidemiological features of the outbreak were recorded as follows:

4.1.1 History of outbreak of PPR

The outbreak occurred in July to December, 2013 which was the Summer,Rainy & Winter season, out of 38 affected Black Bengal goats, 16 were brought from the local market. Immediately before the outbreak, goats in the neighboring village of the farm were affected by a disease with signs and symptoms similar to PPR. History also supported that these affected goats from neighboring village were usually sold in the market. So, the appearance of PPR may be associated with the introduction of recently purchased sick animals from markets.

4.1.2 Morbidity and mortality

Table 1. The morbidity and mortality of Black Bengal goats in a farm at Bagjana Panchbibi, Joypurhat District in Bangladesh.

Age groups (months)	6-8		9-11		12-16		17-24		24+		Total
Sex groups	M	F	M	F	M	F	M	F	M	F	
Population	10	6	6	12	4	6	12	12	0	6	74
Number affected	8	6	6	12	2	2	2	0	0	0	38
Number dead	4	2	2	2	0	0	0	0	0	0	10
Morbidity (%)	80	100	100	100	50	33.33	16.66	0	0	0	51.35
Mortality (%)	40	33.33	33.33	16.66	0	0	0	0	0	0	13.51

M = Male F= Female

The overall morbidity and mortality was 51.35% and 13.51% respectively. The detail may be seen in Table 1. The goat under one year affected more severely. Mortality was also only in the young goat (< 1 year).

4.2 CLINICAL SIGNS OF PPRV INFECTED GOATS

Out of 74 goats from infected farm the clinical signs and symptoms observed in 38 goats were recorded. The main clinical manifestation of the infected goats were fever, dry muzzle, anorexia followed by serous to severe dehydration . A dehydrated carcass of a goat died in the outbreak of PPR(Fig. 1.), PPR in goat: Erosive Stomatitis (Fig.2.), PPR in a goat: purulent and completely close eye (Fig.3.), PPR in a goat: Signs of diarrhoea where the hindquarters soiled with liquid feces (Fig.4.), PPR in a goat Abortion occur in a goat (Fig.5.), Aborted Fetus of PPR affected goat(Fig.6.), PPR in a goat: purulent eye and oculo-nasal discharges(Fig.7.),

were also found. The disease therefore was clinically suspected as PPR. All goats did not show all clinical signs and symptoms. The major clinical signs observed are described below:

There was an abrupt rise of body temperature. The body temperatures varied in different goats from as low as 101°F to as high as 107°F. High fever was observed at 2nd and 3rd day illness and dropped down gradually. But in some cases high temperature sustained (5th day of illness). Rise of body temperature was accompanied by watery discharges (nasal, ocular and oral), which lasted for 4 days to 5 days in goats. Then this discharge became purulent and the blocked nostril caused painful respiratory distress. Diarrhoea was common and observed immediately after rising of body temperature. The hindquarters of diarrhoeic goats were soiled with liquid faces and dehydration occurred. In severe cases, death of the animals occurred.

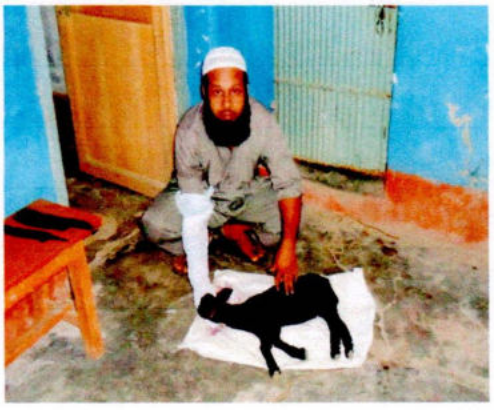


Fig.1. A dehydrated carcass of a goat died in the outbreak of PPR

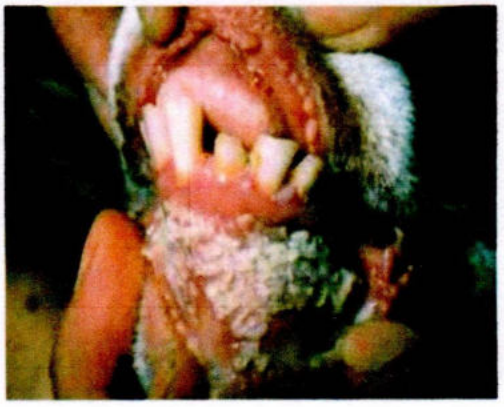


Fig.2. PPR in goat: Erosive Stomatitis

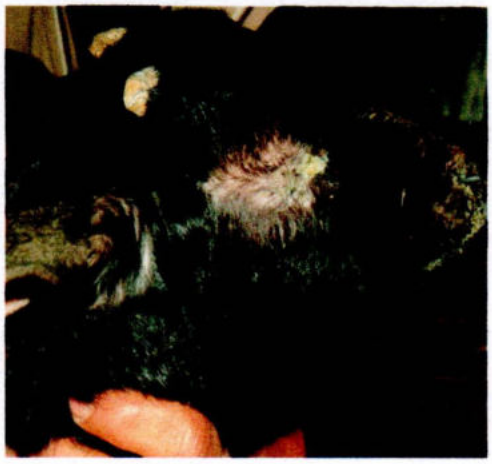


Fig.3. PPR in a goat: purulent and completely close eye

Fig.4. PPR in a goat: Signs of diarrhoea where the hindquarters soiled with liquid faeces

Fig.5. PPR in a goat Abortion occur in a goat

Fig.6. Aborted Fetus of PPR affected goat

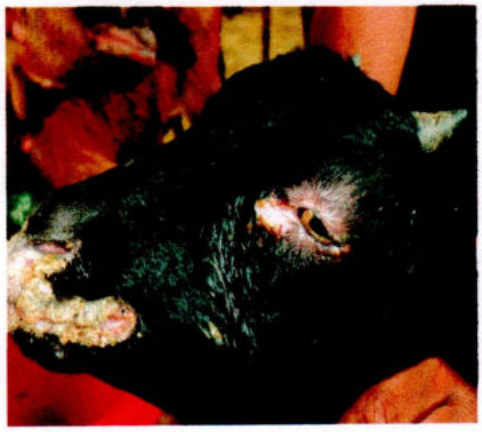


Fig.7. PPR in a goat: purulent eye and oculo-nasal discharges

PATHOLOGICAL FINDINGS OF PPRV INFECTED GOATS

4.1.1 Post mortem findings of PPRV infected goats

During the outbreak, among 38 affected goats 10 were dead. Postmortem examinations were done on 6 goats. Erosions on pillars of rumen severe congestion throughout the intestinal tract (Fig.8.), pale and moderately enlarged liver with indented gall bladder (Fig.9.), zebra striping in ceco-colic junction were main findings. Congested and consolidated pneumonic lungs (Fig.10.), Congestion &Erosion in the Trachea (Fig. 11.). Atrophied Spleen (Fig.12.), Profuse Haemorrhage in the eleo-cecal junction (Fig. 13.), Zebra stripe lesion with haemorrhage in the eleo-cecal(Fig. 14.)

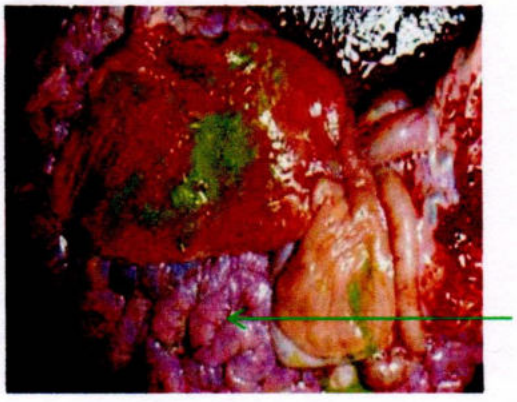


Fig.8. Exterior view of intestine gastroenteritis with severe congestion throughout the intestinal tract

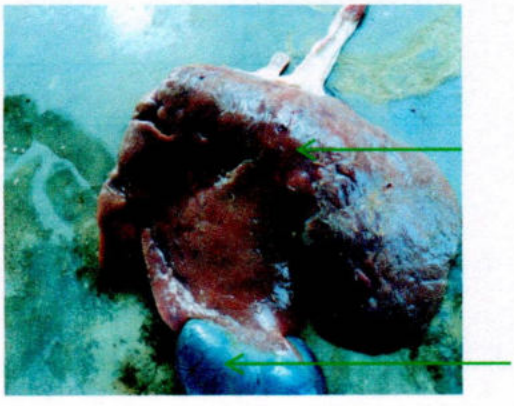


Fig.9. Pale and moderately enlarged liver with indented gall bladder

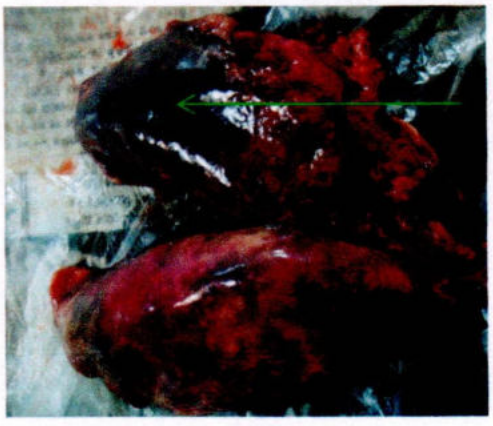


Fig.10. Congested and consolidated pneumonic lung

Fig.11. Congestion and Erosion in the Trachea

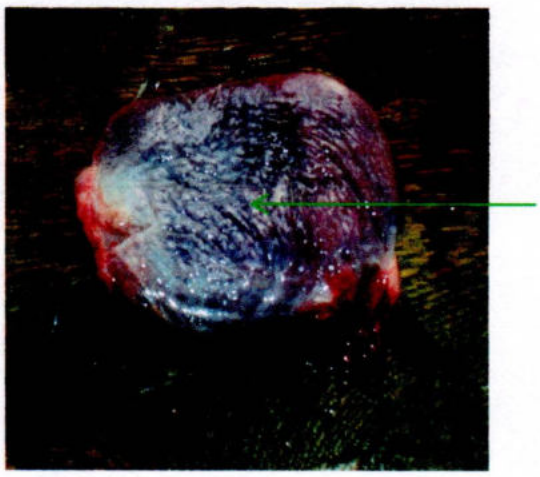


Fig.12. Atrophied Spleen

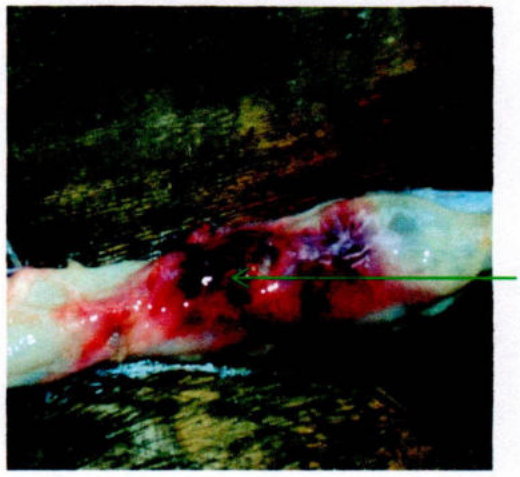


Fig.13. Profuse Haemorrhage in the eleo-cecajunction

Fig.14. Zebra stripe lesion with haemorrhage in the eleo-cecal junction

4.3.2 Histopathological findings of PPRV infected goats:

Intestine: Congestion and infiltration of inflammatory cells (mostly large mononuclear cells in the lamina propria were the major lesions. The villi lost their lining epithelial cells and were severely infiltrated with large mononuclear cells in the core.

Trachea: There were sloughing of tracheal mucosa and infiltration of inflammatory cells in the lamina propria. The infiltration was along the epithelial lining.

Lung: The whole lungs were affected and the lesions were diverse. The lungs parenchyma was partly consolidated; lung alveoli and interstitium were severely infiltrated with large mononuclear cells (Fig.15). In some places, alveoli lost pneumocyte I and lined by pneumocyte II. The large mononuclear cells infiltrated the interstitium and were also found in the alveoli (Fig. 16). In some sections fibrins were accumulated and severely infiltration with large mononuclear cells.

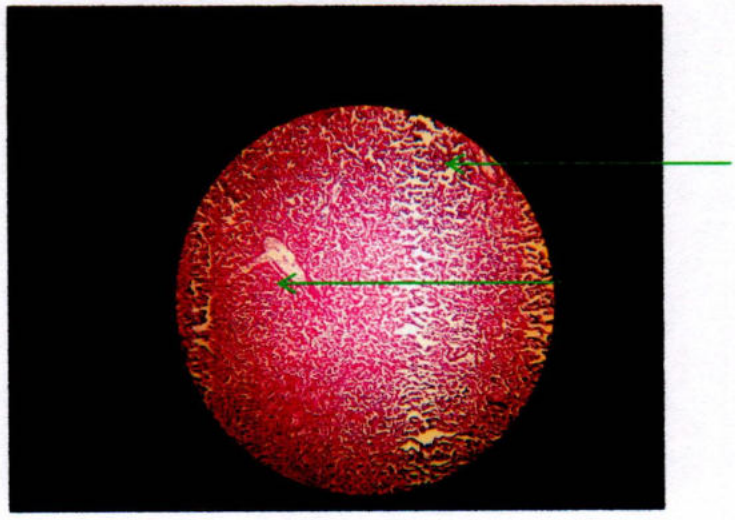


Fig.15. Section of lung of PPRV infected goat: Showing infiltration of inflammatory cells (mostly large mononuclear cells) in the alveoli and interstitium, loss of pneumocyte I and proliferation of pneumocyte I (H & E, ×82.5)

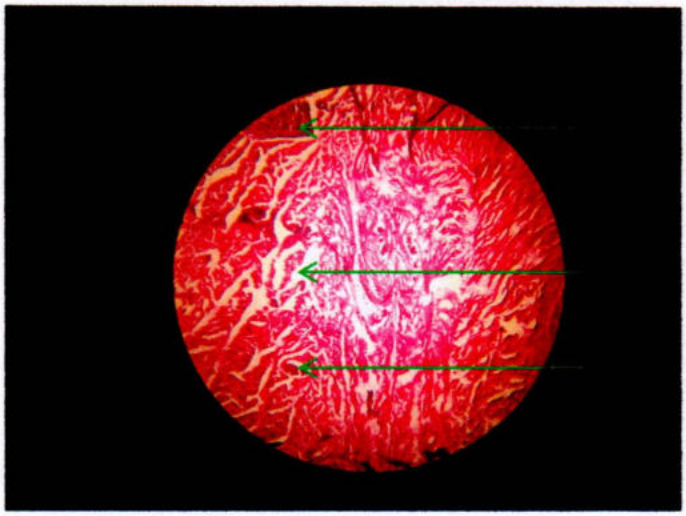
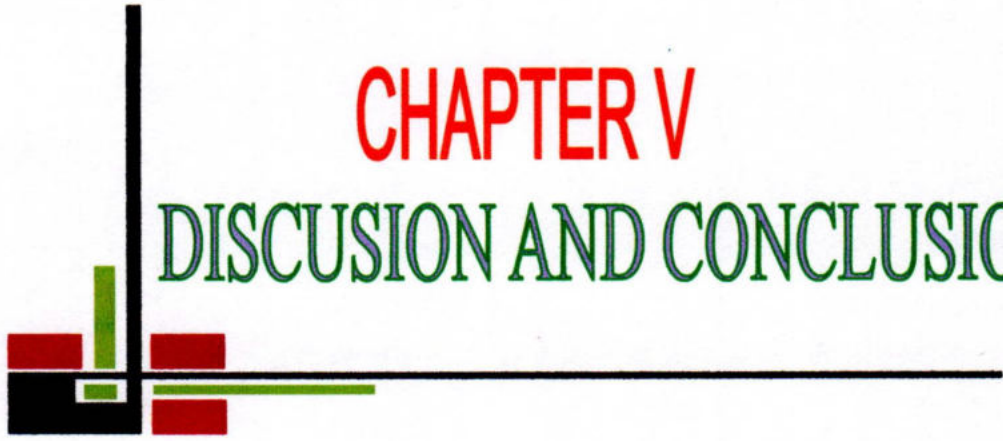


Fig 16. Section of lung of PPRV infected goat: Showing infiltration of inflammatory cells (mostly large mononuclear cells) in the alveoli and interstitium, loss of pneumocyte I and proliferation of pneumocyte II, closure view of (H & E,× 330)



CHAPTER V

DISCUSION AND CONCLUSION

CHAPTER V

DISCUSION AND CONCLUSION

The study was carried out in an experimental goat Farm of Bagjana, Panchbibi, Joypurhat in Bangladesh to investigate the suspected PPR outbreaks. The investigation included epidemiological and clinico-pathological observation.

The outbreak occurred in July-December/ 2013, which was the summer,rainy & winter season and the appearance of clinical PPR was associated with introduction of recently purchased animals from markets. Such events were quite common in the goat farms in PPR endemic area (Whitney *et al.*, 1967; Sil *et al.*, 1995; Radostitis *et al.*, 2000).

The overall morbidity and mortality were 51.35% and 13.51% respectively which was similar to the result of EI-Hassan *et al.*, (1994) when the young goat between 5-8 months old had the highest morbidity rate, Sex difference did not influence the morbidity rate. The same result also obtained by Opasina and Putt (1985).

Infected goats showed depression, inappetance, variation in body temperature (101^{0}F-107^{0}F), purulent eye and oculo-nasal discharges, erosive stomatitis, respiratory disterbence, diarrhoea with soiled hind quarter, dehydration and death. There were some variations in clinical signs in different goats The clinical signs and symptoms observed in the present outbreak were similar to those reported in case of PPR of goats from different geographical areas (Mondal *et al.*, 1995; Shanker *et al.*, 1998; Radistitis *et al.*, 2000; Kumar *et al.*, 2001; Islam *et al.*, 2001; Debasis and Mousumi, 2002; Dhand *et al.*, 2002; Wadhawa *et al.*, 2002., Diallo, 2003; Islam *et al.*, 2003; Pawauya *et al.*, 2004; Islam, 2006).

Emaciated and dehydrated carcass was found at time of postmortem examination which was also observed by khan *et al.*, (2005). The important necropsy findings were erosions on pillars of rumen, severe congestion throughout the intestinal tract, zebra striping in ceco-colic junction, pale and moderately enlarged liver with indented gall bladder, congested and consolidated pneumonic lungs, enlarged edematous lymph nodes with congestion throughout the body, brush paint hemorrhages in the heart and atrophied congested spleen. Congestion in almost all organs might be due to damage of endothelial lining cells and which might be due to involvement of endothelial cells in the pathogenesis of PPRV infection. The similar necropsy findings were described for PPRV by several other authors (Alcigir *et al.*, 1996; Islam *et al.*, 1996; Saliki *et al.*, 1998; Susan and Aiello, 1998; Islam *et al.*, 2001; Wadhwa *et al.*, 2002; Yeaner *et al.*, 2004; Khan *et al.*, 2005).

The important histopathological findings were congestion and infiltration of inflammatory cells (mostly large mononuclear cells) in the villi and lamina propria of intestine; sloughing of mucosa and infiltration of inflammatory cells in the trachea; infiltration of inflammatory cells (mostly large mononuclear cell) in the alveoli and interstitium, loss of pneumocyte I and proliferation of pneumocyte II, fibrinous pneumonia and bronchointerstitial pneumonia that is characterized by accumulation of fibrin, infiltration of large mononuclear cells in the alveoli, interstitium and parenchyma of lung; depletion of lymphocytes with infiltration of macrophages in lymphnode, spleenic atrophy with depletion of lymphocytes, indicated by the presence of numerous trabeculi in spleen. The similar histopathological findings were described for PPRV by several authors (Islam *et al.*, 1996; Islam *et al.*, 2001; Kumar *et al.*, 2004; Pawaiya 2004; Kul *et al.*, 2007). The proliferation of macrophages in different organs indicates primarily non-purulent inflammation in the affected goat. Depletion of lymphocytes from lymphnode

and spleen may be due to particular affinity of the virus to lymphoid tissue and the involvement of lymphocytes in the pathogenesis of PPRV infection (Susan and Aiello, 1998).

Further study should be carried out for isolation, characterization with full molecular studies, pathogenesis and development of effective vaccine from local isolates of PPRV in near future to protect our goat population in Bangladesh.

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NECROPSY FINDINGS (If any):

- Lungs
- Liver
- Mesenteric Lymphnode
- Bronchial Lymphnode
- Heart
- Intestine

Date & Signature