





# **STUDY ON THE PREVALENCE OF GUMBORO DISEASE IN DINAJPUR DISTRICT**

## **A Clinical Report**

**Submitted as per approved Style and Code**

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**A Clinical Report Submitted In Part of Fulfillment of The  
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**Chittagong Veterinary and Animal Sciences University  
Khulshi, Chittagong - 4225.**

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### ABSTRACT

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The present study was conducted to investigate the status of infectious disease (IBD) in birds reared in fully intensive system for meat purpose. The study area was all five (05) upazillas (administrative unit of Bangladesh) of Dinajpur district within two months study period. 70 farms rearing 500 to 4000 birds were randomly selected from the study area. Birds were examined by clinical signs & post-mortem lesions to diagnose IBD. IBD positive cases were defined by any sort of lesions in Bursa of fabricius i.e: inflammation, haemorrhage, caseous necrosis or atrophy of the bursa. Among 70 farms 41 farms were infected with IBD. Maximum numbers of farms were infected between ages 03 to 05 weeks. Highest percentage of morbidity, mortality & prevalence were 15, 6.88 & 9.3 and lowest of those were 2.08, 0.47 & 3.09, whereas average percentage of morbidity & mortality were 5.58 & 2.07. The non-vaccinated birds were mostly affected with IBD(82.61%) than vaccinated group(46.81%).The risk factors were identified mainly wrong vaccination, overcrowding, poor ventilation heat stress and lack of bio-security. Proper vaccination was proved as the key to prevent the disease. Extensive studies are recommended for the serological and molecular study.

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**Key words:** Poultry, IBD, Clinical signs, Diagnosis, Prevalence, Morbidity, Mortality, Prevention

<b>SL. NO.</b>	<b>Chapter</b>	<b>CONTENTS</b>	<b>PAGE NO.</b>
1		ACKNOWLEDGEMENT	I
2		ABSTRACT	II
3	I	INTRODUCTION	1-3
4	II	REVIEW OF LITERATURE	4-12
5	III	MATERIALS AND METHODS	13-26
6	IV	RESULTS AND DISCUSSION	27-41
8	V	CONCLUSION	42
9	VI	REFERENCES	43-54

# CHAPTER-I

## INTRODUCTION

Livestock plays an important role in the agricultural economy of Bangladesh. The magnitude of the contribution of the livestock sub-sector to the countries gross domestic product (GDP) is 3.1 percent and to agricultural GDP it is about 11 per cent. Data generated by the Department of Livestock Service shows that the loss of livestock due to various diseases resulted in losses of approximately 140000 million taka per year (1\$=78 taka) in the mid eighties (Hassan, 1985). Losses were calculated as 25% and 60% of the total value of livestock and poultry. The loss from livestock diseases in Bangladesh could be estimated between Taka 20.70 and 49.46 billion. Since the country does not have an effective disease control program, the loss from diseases may be counted on the higher side (Nakamura, 1990). In 1997-98 the small-scale family poultry system in Bangladesh has been estimated to account for about 80% of the total poultry population (Huque, 1999). It is one of the most important income-generating activities for rural women, landless poor and marginal farmers. Bangladesh has a large potential to increase meat and egg production through improvement of indigenous practices in extensive family poultry production system.

Infectious Bursal Disease (IBD) is a highly contagious, globally occurring viral poultry disease. The disease was first reported by Cosgrove, who in 1962 observed a disease, affecting chickens on farms in the neighborhood of Gumboro, Delaware, USA (Cosgrove, 1962). Thus, Gumboro disease became synonymous for the condition. The virus causing IBD suppresses the immune system of affected birds by damaging organs of primarily the humoral cell defense, particularly Bursa Fabricii, which is why IBD has become the alternative name for the disease.

During the 63rd General Session of the Office International des Epizooties (OIE, 1995), it was estimated that IBD has considerable socio-economic importance at the international level, as the disease is present in more than 95% of the Member Countries (Etteradossi, 1995). In this survey, 80% of the countries reported the occurrence of acute clinical cases. The domesticated hen (*Gallus gallus*) is the only species for which IBD virus has been reported to induce clinical disease. However, serological surveys in wild birds (Wilcox *et al.*, 1983; Gardner *et al.*, 1997;



## **CHAPTER-II**

### **REVIEW OF LITERATURE**

#### **History**

Infectious bursal disease (IBD) also known as Gumboro disease was first recognized by Cosgrove (1962) as a clinical entity, in 1957, in southern Delaware, USA. The etiological viral agent was isolated by Winterfield in 1962 (Lukert and Saif, 1997) who differentiated the disease from a previously established disease known as nephrotoxic infectious bronchitis viral infection of chickens. The term infectious bursal was proposed by Hitchner (1970).

There are two serotypes of infectious bursal disease virus (IBDV) (McFerran *et al.*, 1980). Serotype 1 is pathogenic while serotype 2 is non pathogenic for chickens. Within serotype 1 many subtypes or pathotypes have evolved (Brown and Grieve, 1992). Clinical evidence suggests, that the standard or classical serotype 1 IBDV was predominant throughout the world until early 1980s (Brown and Grieve, 1992). In 1984/85 variant strains of IBDV started to appear in Delmarava peninsula, USA with increased mortality even in vaccinated flocks, and these new American strains were antigenically different from the classical strain (Snyder *et al.*, 1988). These variant strains also differed from classical serotype 1 strains in that they produced a very rapid bursal atrophy with minimal inflammation. Vaccines prepared from classical strains did not give full protection against the variant IBDV strains (Snyder, 1990). Despite the high contagious nature, the mortality from infection with classical and variant strains of IBDV was very low. Most of the mortalities were due to immunosuppression and subsequent secondary infections (Cavanagh, 1992).

In 1987 a highly pathogenic strain (849 VB) of type 1 IBDV emerged in Holland and Belgium (van den Berg *et al.*, 1991). Mortality in exposed 3-14 weeks old layer replacement pullets attained 70% and 100% mortality in experimental infection. Gaudry (1993) reported outbreaks of vvIBDV (very virulent infectious bursal disease virus) in China and Russia in 1993, associated with 60% mortality in 10 days old Leghorn pullets. A virus responsible for outbreaks of vvIBDV in the UK designated the DV86 strain was characterized by Chettle *et al.*, (1989), who confirmed

that spontaneous enhancement of virulence had occurred without any major alteration in antigenic structure. The acute forms of the disease were then described in Japan in the early 1990s (Nunoya *et al.*, 1992; Lin *et al.*, 1993), and they have rapidly spread all over Asia and to other countries. In Bangladesh first outbreaks of IBDV occurred in the early nineties (Article 1). Since then, they have been isolated in many countries including Central Europe (Savic *et al.*, 1997), the Middle East, South America (Di Fabio *et al.*, 1999) and Asia (Cao *et al.*, 1998; Chen *et al.*, 1998; To *et al.*, 1999). On the other hand Australia, New Zealand, Canada and the US are so far unaffected (Snyder, 1990; Proffitt *et al.*, 1999; Sapats & Ignjatovic, 2000). Moreover, only a sporadic severe outbreak has been described in Finland (Nevalainen *et al.*, 1999), whereas the other northern European countries are still free (Czifra & Janson, 1999).

### **Incidence and distribution**

Infections with serotype 1 IBDV are of worldwide distribution, occurring in all major poultry-producing areas (Lukert and Saif, 1997). One exception to the ubiquitous nature of IBDV is New Zealand. It has been reported (Jones, 1986; With, 1985) that there is no evidence of IBDV infections in that country. Because of vaccination programs carried out by most producers, all chickens eventually become seropositive to IBDV.

### **Epidemiology**

Infectious bursal disease is usually a disease of three to six week old chickens. An early subclinical infection before three weeks of age (Lukert and Saif, 1997), even in newly hatched chicks (Fadley and Nazerian, 1983), may occur. The disease has also been reported to occur up to 20 weeks of age in chickens (Okoye and Uzoukwu, 1981). All breeds are affected but severe reactions with highest mortality rate were observed in White Leghorn (Lukert and Saif, 1997). Chowdhury *et al.*, (1996) observed higher mortality rate (70-80%) in the Fayoumi breed as compared to White Leghorn (40%) in a limited number of field outbreaks. Thirteen to 85% mortality due to IBDV was found in different breeds of chickens in field outbreaks (Article 1). Mortality due to IBD on various farms ranged from 1 to 40% in broilers and from 2 to 40% in layers (Kurade *et al.*, 2000) and from 1.5 to 30% in native and broiler flocks respectively (Saif *et al.*, 2000). However, Meroz (1966) found that there was no difference in mortality between heavy or light breeds. Natural infections of turkeys and ducks have been reported (McFerran *et*

*al.*, 1980). The disease spreads rapidly by direct contact because of the highly contagious nature (Benton *et al.*, 1967a). There is no report of egg transmission of IBDV. Infected birds have excreted the virus in their droppings for at least 14 days (Baxendale, 2002). Fishmeal in the feed contaminated with IBDV may act as a transmitter of the disease (Yongshan *et al.*, 1994), while lesser mealworm as well as mosquito may act as a reservoir of IBDV (Snedeker *et al.*, 1967; Howie and Thorson, 1981; McAllister *et al.*, 1995).

### **Structure of the virus**

IBDV is a naked icosahedral, double-stranded RNA virus with a diameter of 55-60 nm (Hirai and Shimakura, 1974; Nick *et al.*, 1976; Dobos *et al.*, 1979; Jackwood *et al.*, 1982) belonging to the family Birnaviridae (Kibenge *et al.*, 1988). The prototype of the family is infectious pancreatic necrosis virus (IPNV) of fish. Other members of the family can affect insects and molluscs. The molecular weight of the virus ranged from 2.2 to 2.5 X 10<sup>6</sup> daltons (Nick *et al.*, 1976; Müller *et al.*, 1979) with the buoyant density of 1.34 g/ml (Hirai and Shimakura, 1974; Nick *et al.*, 1976; Dobos *et al.*, 1979; Jackwood *et al.*, 1982). The virion has a single capsid shell composed of 32 capsomers and a diameter of 60 to 70 nm. The larger segment A (approximately 3400 base pairs) is monocistronic and encodes a polyprotein that is auto-processed after several steps into mature VP2, VP3 and VP4 (Müller & Becht, 1982; Azad *et al.*, 1985; 1987; Hudson *et al.*, 1986; Kibenge *et al.*, 1997). Segment A can also encode VP5, a short 17kDa protein (Mundt *et al.*, 1995). The smaller segment B (approximately 2800 bp) encodes VP1, the viral RNA polymerase of 90 kDa (Müller & Nitschke, 1987; Spies *et al.*, 1987).

### **Pathogenesis**

Susceptibility of different breeds of chicken has been described with higher mortality rates in light than in heavier breeds (Bumstead *et al.*, 1993; Nielsen *et al.*, 1998). Inoculation of IBDV in other avian species fails to induce disease (McFerran, 1993). Bursectomy can prevent illness in chicks infected with virulent virus (Hiraga *et al.*, 1994). The severity of the disease is directly related to the number of susceptible cells present in the bursa of Fabricius. Therefore, the highest age of susceptibility is between 3 and 6 week, when the bursa of Fabricius is at its maximum development. This age susceptibility is broader in the case of vvIBDV strains (van den Berg *et al.*, 1991; Nunoya *et al.*, 1992)

After oral infection or inhalation, the virus replicates primarily in the lymphocytes and macrophages of the gut-associated tissue. Then virus travels to the bursa via the blood stream, where replication occur. By 13h post-inoculation (p.i.), most follicles are positive for virus and by 16h p.i a second and pronounced viraemia occurs with secondary replication in other organs leading to disease and death (Müller *et al.*, 1979).

### **Clinical aspects of IBD**

The incubation period of IBD ranges from 2 to 4 days. Infection of susceptible broilers or layer pullet flocks is characterized by acute onset of depression. Birds are disinclined to move and peck at their vents (Cosgrove, 1962) and pericloacal feathers are stained with urates (Landgraf *et al.*, 1967). Helmboldt and Garner (1964) detected histologic evidence of infection in cloacal bursa within 24 hours. Müller *et al.*, (1979), using immunofluorescence techniques, observed infected gut-associated macrophages and lymphoid cells within 4-5 hr after oral exposure to IBDV.

The European strains responsible for vvIBD produce clinical signs similar to conventional type 1 infection. The initial outbreaks were characterized by high morbidity (80%) and correspondingly significantly mortality, attaining 25% in broilers and 60% in pullets over a 7-day period (Chettle *et al.*, 1989; van den Berg *et al.*, 1991; Nunoya *et al.*, 1992).

### **Gross Pathology**

Chickens which die acutely of primary IBD infection show dehydration of the subcutaneous facial and pectoral musculature (Cosgrove, 1962). Gross lesions of IBD have been well described (Cheville, 1967; Helmboldt & Garner; 1964; Landgraf *et al.*, 1967; Ley *et al.*, 1983; Skeeles *et al.*, 1979a). The bursa of Fabricius is the principal diagnostic organ in which gross changes occur following exposure to IBDV. Autopsy of birds dying in the acute phase 3-4 days after infection, reveals dehydration and swelling of the bursa to about twice its normal size due to hyperaemia and oedema. In severe cases, there is marked inflammation of the mucosa and a serous transudate giving the serosal surface a yellow appearance. Petechial hemorrhage on the mucosal surface is common. Similar findings (Article 2) confirmed the challenge infection of

IBD. By the 5th day after infection, the bursa has returned to normal size and by the 8th day it has atrophied to about one third of its original weight.

Swelling and white appearances of the kidneys and associated dilatation of the tubules with urates and cell debris are features encountered in some outbreaks but do not seem to be a consistent findings and increased mucus in the intestine (Baxendale, 2002; Cosgrove, 1962). The splenic enlargement was documented by Morales and Bocclair (1993) who showed highly significant differences in bursa:spleen weight ratio of 2.4 for controls compared with 0.9 in chicks seven days after challenge. Very often small grey foci are uniformly dispersed on the surface of the spleen (Rianldi *et al.*, 1965). Occasionally, hemorrhages are observed in the mucosa at the juncture of the proventriculus and gizzard (Lukert and Saif, 1997).

### **Histopathology**

IBD affects primarily the lymphoid structures- cloacal bursa, spleen, thymus, harderian gland and cecal tonsil, gut-associated lymphoid tissue (GALT), head associated lymphoid tissues (HALT) (Lukert and Saif, 1997). All lymphoid follicles were affected by 3 or 4 days postinfection. Lymphocytes were soon replaced by heterophils, pyknotic debris and hyperplastic reticuloendothelial cells (Article 2). Hemorrhages often appeared but were not a consistent lesions (Helmbodt and Garner, 1964; Cheville, 1967; Mandelli *et al.*, 1967; Peters, 1967). Following lytic changes, follicles are replaced by cysts lined by columnar epithelium surrounded by a fibroplastic interfollicular stroma (Okoye and Uzoukwu, 1990). Cystic cavity develops after subsiding the inflammatory reaction and there was a fibroplasia in interfollicular connective tissue (Cheville, 1967) (Article 2) Lukert and Saif (1997). One of the recent isolates (variant A) of IBDV was reported to cause extensive lesions in bursa but the inflammatory response was lacking (Sharma *et al.*, 1989).

In spleen following initial perivascular reticuloendothelial hyperplasia, lymphoid necrosis was observed in the germinal centres by the 3rd day after infection (Helmbodt and Garner 1964), Repopulation commences by the fifth day and is complete in eight days (Okoye and Uzoukwu, 1990). Type 1 IBDV infection in 1-day-old broilers devoid of maternal IBD antibody was investigated by Dohms *et al.*, (1981) who showed that plasma cells, which normally populated the Harderian gland by 3 weeks of age, were significantly reduced in numbers compared with non-infected controls.

Histologic lesions of the kidney are nonspecific (Peters, 1967) and probably occur because of severe dehydration of affected chickens. Helmboldt and Garner, (1964) found kidney lesions in less than 5% of birds examined. The liver may have slight perivascular infiltration of monocytes (Peters, 1967).

### **Immunosuppression and interaction with other pathogens**

The first published description of the immunosuppressive effect of IBDV in the chicken demonstrated a diminished antibody response to Newcastle disease vaccination (Faragher *et al.*, 1974). Pattison and Allan (1974) demonstrated the persistence of Newcastle disease virus in the respiratory tract of chickens which had earlier been exposed to IBD. There was moderate suppression when chicks were infected at 7 days and negligible effects when infection was at 14 or 21 days (Faragher *et al.*, 1974). Hirai *et al.*, (1974) demonstrated decreased humoral antibody response to other vaccine as well. Panigraphy *et al.*, (1977) reported that IBDV infections at a young age caused a prolonged skin graft rejection. However, other workers (Giambrone *et al.*, 1977 and Hudson *et al.*, 1975) found no effect from early IBDV infections on skin graft rejection or tuberculin-delayed hypersensitivity reactions. Sivanandan and Maheswaran (1981) observed suppression of cell-mediated immune (CMI) responsiveness, using the lymphoblast transformation assay. In a sequential study of peripheral blood lymphocytes from chickens inoculated with IBDV, a transient depression of mitogenic stimulation was reported (Confer *et al.*, 1981). Sharma and Lee (1983) reported an inconsistent effect of IBDV infection on natural killer cell toxicity and a transient early depression of the blastogenic response of spleen cells to phytohemagglutinin. Depression in plasma cell activity in the Harderian gland is caused by IBDV (Pejkovski *et al.*, 1979 and Dohms *et al.*, 1981).

Chickens infected with IBDV, day old at age, were completely deficient in serum IgG and produced only a monomeric IgM (Ivanyi, 1975; Ivanyi and Morris, 1976). The number of B cells in peripheral blood was decreased following infection with IBDV but T cells were not appreciably affected (Hirai *et al.*, 1979; Sivanandan and Maheswaran 1980). The virus appears to replicate primarily in B lymphocytes of chickens (Hirai and Calnek 1979; Ivanyi 1975; Yamaguchi *et al.*, 1981). Apparently IBDV has a predilection for actively proliferating cells (Müller, 1986), and it was suggested that the virus affected "immature" or precursor B lymphocytes to a greater extent than mature B lymphocytes (Sivanandan and Maheswaran 1980).

Chicks infected early with IBDV were more susceptible to inclusion body hepatitis (Fadley *et al.*, 1976), coccidiosis (Anderson *et al.*, 1977), Marek's disease (Cho, 1970; Sharma, 1984), hemorrhagic-aplastic anemia and gangrenous dermatitis (Rosenberger *et al.*, 1978), infectious laryngo tracheitis (Rosenberger *et al.*, 1978), infectious bronchitis (Pejkovski, *et al.*, 1979), chicken anemia agent (Yuasa *et al.*, 1980), and salmonella and colibacillosis (Wyeth, 1975).

### **Diagnosis**

Classical IBD is characterized by acute onset, relatively high morbidity and low flock mortality in 3-6 weeks old broilers or replacement pullets. Diagnostic lesions include muscle haemorrhages and bursal enlargement (Hanson, 1967).

### **Isolation**

Hitchner (1970) demonstrated that chorio allantoic membrane (CAM) of 9 -11 days old embryos was the most sensitive route for isolation of the IBDV which could subsequently be adapted to the allantoic sac and yolk sac route of inoculation. Hitchner (1970) observed that most mortality occurred between the 3rd and 5th days post inoculation. Affected embryos had edematous distention of the abdomen, petechiae and congestion of the skin and occasionally ecchymotic hemorrhages in the toe joints and cerebrum. Bursal samples from the infected ducklings were able to infect chick embryos that died in 96-120 hours after inoculation and the embryos showed the pathological lesions of infectious bursal disease (Bian *et al.*, 1999). Similar findings were observed by the author (Article 1). Variant strains of IBD differ from standard viruses in that they induce splenomegaly and liver necrosis of embryos and produce little mortality (Rosenberger *et al.*, 1985). McFerran *et al.*, (1980) reported that three of seven chicken isolates of IBDV failed to grow in chicken embryo fibroblast (CEF) cells but propagated in embryonating eggs.

Many IBDV isolates have been adapted to primary cell cultures of chicken embryo origin, including chicken embryo kidney (CEK) cells and chicken embryo fibroblasts (Lukert and Davis, 1974; McNulty *et al.*, 1979). Because these cells produce low yields of virus (Lukert *et al.*, 1975; Müller, and Becht 1982), there is a need for cell cultures that will produce higher yields of infectious virus required for experimental purposes. Cells susceptible to the virus other

than cells of chicken origin include turkey and duck embryo cells (McNulty *et al.*, 1979), mammalian cell lines derived from rabbit kidneys (RK-13) (Rianldi *et al.*, 1972), Vero cells (Jackwood *et al.*, 1987; Leonard, 1974; Lukert *et al.*, 1975), derived from African green monkey kidneys; BGM-70 cells (Jackwood *et al.*, 1987), from givet monkey kidneys; and MA-104 cells (Jackwood *et al.*, 1987), from fetal rhesus monkey kidneys.

### **Serology**

The agar gel diffusion precipitin test (AGDP) was the original qualitative method to detect antibody. Bursal homogenate is used as the antigen to demonstrate antibody 7 days after infection (Rosenberger, 1989; Article 1). The serum virus neutralization procedure is extremely sensitive (Weisman and Hitchner, 1978) and is sufficiently specific to differentiate between serotypes of IBD virus (Chin *et al.*, 1984).

The ELISA procedure (Engvall and Perlman, 1971) was adapted for IBDV serology and represents a rapid, quantifiable, sensitive and reproducible procedure, which can be automated (Marquardt *et al.*, 1980). The practice of sequential sampling of flocks to monitor antibody level as influenced by vaccination, field exposure and time-related decay in titre was facilitated by the introduction of commercial ELISA test kits (Briggs *et al.*, 1986, Article 2). Automated assays and computerized processing, storage and retrieval of data are the basis of flock profiling (Snyder *et al.*, 1986). The relative advantages and applications for three methods of assaying IBDV antibody titre have been summarized by Box (1988). Quantitative agar gel diffusion proved to be relatively insensitive, especially when monitoring sera from chicks to determine patterns of maternal antibody decay and age of susceptibility (van den Berg *et al.*, 1991).

### **Prevention of IBD**

Maintaining commercial flocks free of IBDV requires the application of sound biosecurity coupled with effective vaccination of parents and progeny (Lukert and Saif, 1997). Since decontamination alone is ineffective (Parkhurst, 1964), prevention of conventional type 1 strain IBDV is dependent on appropriate vaccination of parent breeders and broiler stock. The first vaccines to prevent IBD in broilers and replacement pullets were prepared by adaptation of field isolates in embryonated eggs (Edgar and Cho, 1965; 1973).



Attenuation of IBDV by passaging chick embryo kidney adapted cultures on a non-avian (VERO) tissue culture system (eight passages) resulted in a non-pathogenic virus. This candidate vaccine was shown to be ineffective when administered orally but capable of stimulating high levels of antibody when injected subcutaneously (Lukert *et al.*, 1975). Three commercial IBD vaccines were evaluated for pathogenicity and protective in specific pathogen free (SPF) chicks by Naqi *et al.*, (1980). Wood *et al.*, (1988) reported on the development of a candidate IBD vaccine strain designated 002-73 isolated in Australia. *In vivo* neutralization and passive protection suggested that the isolate would be effective against field strains prevalent in Europe.

Selection of vaccines from the 'mild', 'intermediate', and low attenuation or 'hot' classification depends on managemental and stock-related factors, level and uniformity of maternal antibody transfer, virulence of field virus strains, and risk of challenge. High parental immunity was recognized as beneficial in protecting young chicks from field virus challenge during the critical first 2 weeks when the bursa is most vulnerable to damage induced by IBDV (Hitchner, 1976). In contrast, high maternal antibody interferes with stimulation of IBD antibody induced by live attenuated vaccines (Wyeth and Cullen, 1978a). Propagation of virus on bursal tissue to produce inactivated oil emulsion vaccines (Wyeth and Cullen, 1979) produces a more immunogenic agent than virus prepared in specific pathogen free embryos (Wyeth and Chettle, 1982).

Selection of vaccination programmes to protect broilers against vvIBDV strains that emerged in Europe in 1987 has required extensive evaluation of the dynamics of vaccine antigenicity, pathogenicity of virus, and maternal antibody. Challenge studies in SPF chicks conducted in Belgium evaluated intermediate and mild vaccines against the vvIBDV isolate designated 849VB (van den Berg *et al.*, 1991). Chicks were vaccinated at 10 days of age and challenged 3-6 days later. Intermediate cloned D78 was mildly pathogenic to bursae, but was immunogenic and provided 80% protection against mortality when challenged 4 days after vaccination. Mild IBD strain vaccines were ineffective in protecting against 849VB virus.

## **CHAPTER-III**

### **MATERIALS AND METHOD**

The survey was carried out in some selected areas of Dinajpur district of Bangladesh using a questionnaires, developed mainly for collection of information on Infectious Bursal Diseases (IBD), especially on prevalence and economic losses from in at farm level, The survey was designed to collect data from farmers of different farm categories such as large and small farms from each of 2 farm categories, 70 farm owners were planned to be supplied with questionnaire. The data collected through questionnaire was analyzed finally.

#### **Duration of the study:**

The study was conducted at July 16, 2012 to September 15,2012 (Total 60 days).

#### **Selection of the study areas:**

The experience was conducted in 14 villages of five upazilla in Dinajpur district of Bangladesh. Among this villages 05 farms from village i.e. total 70 farms were again randomly selected. In all selected farms, the number of birds was ranged from 500-4000.

#### **Geographical description:**

Dinajpur is bounded by Panchagarh districts in the north, Gaibandha and Joypurhat districts in the south, Nilphamari and Rangpur districts in the east, and the state of West Bengal, India in the west. It lies between latitude  $25.63^{\circ}$  N and longitude  $88.65^{\circ}$  E. The total area of the district is  $3,437.98 \text{ km}^2$  Dinajpur experiences a hot, wet and humid tropical climate. The district has a distinct monsoonal season and monthly means varying between  $18^{\circ}\text{C}$  ( $64^{\circ}\text{F}$ ) in January and  $29^{\circ}\text{C}$  ( $84^{\circ}\text{F}$ ) in August. Annual average highest temperature  $33.5^{\circ}\text{C}$  and lowest  $10.5^{\circ}\text{C}$ ; annual rainfall 2,536mm. Main rivers are Dhepa, Punarbhaba, and Atrai.

## CHAPTER-IV

### RESULT AND DISCUSION

#### **Farms covered under respective areas (Dinajpur district)**

During study of IBD 14 villages were taken as the target area. The percentage of IBD affected farms are shown below.

**Table:1 Farms covered under respective areas (Dinajpur district)**

SL NO.	Name of the village	Total no. of farms visited	No.of farms affected	% of Gumboro affected farms
01	Basherhat	05	03	60%
02	Banahar	05	04	80%
03	Parikata	05	02	40%
04	Ushahar	05	03	60%
05	Nabogram	05	02	40%
06	Pachbar	05	04	80%
07	Cowga	05	03	60%
08	Fawkol	05	03	60%
09	Chapuli	05	02	40%
10	Ayra	05	03	60%
11	Gobindopur	05	03	60%
12	Ramkrishnopur	05	04	80%
13	Shibpur	05	02	40%
14	Ulapur	05	03	60%

#### **The morbidity and mortality of poultry with IBD:**

Morbidity and mortality of poultry were varied in different age groups. The highest percentage of morbidity was found in the age group of 2-3 weeks old which was 15% and highest percentage of mortality was found in age group of 4-5 weeks old which was 6.88%.The lowest

## **CHAPTER-V**

### **CONCLUSION AND RECOMMENDATION**

The prevalence of Gumboro disease of poultry depends on the management systems of the farms, quality of vaccines, bio-security etc. most of the farmers vaccinated their flocks without maintaining cold chain and indiscriminately. The principles of food-safety are yet difficult to implement by the specific grower because of various socio-economic barriers. Quality feed stuffs, vaccine, medicine chicks and proper disease diagnostic facilities are still far from reaching to the majority of specific farmers. If anyhow, the detection of antibody level of chicks after vaccination is possible; whether the antibody develop or farmers can get benefit in preventing the disease.

Finally, it is suggested that specific and sensitive diagnostic tools should be developed and vaccine should be produced from existing strains of infectious bursal disease virus so that effective vaccines can be produced and level of maternal antibody should be known for successful immunization. As faulty management is the principle cause of the disease, the farmers should be give proper understanding about improved poultry management by suitable extension program. The company should test vaccine efficiency before releasing to the market for sale. Before vaccination the following points should be considered to prevent vaccination failure:

1. Use of quality vaccine.
2. In vivo vaccination should be done at hatchery level.
3. Vaccination schedule should be maintained properly.
4. Have to check antibody titre.
5. Application of vaccine quickly after mixing in appropriate route.
6. Dose should be maintained properly.
7. Diseased or risk birds should not be vaccinated.
8. After all, before vaccination the farmers should take consultancy form veterinary doctor.

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# QUESTIONNAIRE

## Diseases diagnosed in broiler chickens by postmortem examination

DATE: \_\_\_\_\_

SAMPLE NO.: \_\_\_\_\_

1. Name of the farm.....
2. Name of the owner & address:.....
3. Type of farm:.....
4. Total no. of birds:.....
5. Total death to date:.....
6. Age of the birds:.....
7. Type of feed: a) mash b) pellet .....
8. Strain:.....
9. Total number of shed:.....
10. Infected shed no.....
11. Batch no:.....
12. When the bird was died:.....
13. Clinical signs described by the owners:.....
  
14. Postmortem findings:
  - Head.....
  - Trachea.....
  - Lung .....
  - Liver .....
  - Intestine .....
  - Bursa .....
  - Thai muscle.....
  - Spleen.....
  - Proventriculus.....
  - Gizzard.....
  - Air sac.....

# STUDY ON THE PREVALENCE OF GUMBORO DISEASE IN DINAJPUR DISTRICT

## ABSTRACT

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The present study was conducted to investigate the status of infectious disease (IBD) in birds reared in fully intensive system for meat purpose. The study area was all five(05) upazillas ( upazillas is the administrative unit of Bangladesh) of Dinajpur district & study period 16<sup>th</sup> July 2012 to September 15, 2012 ( Total 60days). 70 farms rearing 500 to 4000 birds were randomly selected from the study area. Birds were examined by clinical signs & post-mortem lesions to diagnose IBD. IBD positive cases were defined by any sort of lesions in Bursa of fabricius i.e: inflammation, haemorrhage, caseous necrosis or atrophy of the bursa. The post-mortem lesions of 100 birds of which 43 farms with only IBD & 27 farms with IBD mixed infection i.e: total 70 farms were infected with IBD. Maximum numbers of farms were infected between ages 03 to 05 weeks. Highest percentage of morbidity, mortality & prevalence were 75.71, 22 & 46.76 and lowest of those were 19.38, 3.8 & 32.7 where as average percentage of morbidity, mortality & prevalence were 41.89, 9.50 & 41.89. The risk factors were identified mainly wrong vaccination, over crowding, poor ventilation heat stress and lack of bio-security. Proper vaccination was proved as the key to prevent the disease.

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**Key words:** Poultry, IBD, Clinical signs, Diagnosis, Prevalence, Morbidity, Mortality, Prevention.



