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Epidemiology, Status and Economic Importance of Infectious Bursal Disease in Poultry Production, Ethiopia

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Abstract

Chickens rising have long been a line of tradition and the large numbers of chicken populations are kept in the backyard production system in Ethiopia. Poultry disease is among the main constraints of poultry production in the country. The infectious bursal disease is widespread viral diseases that affect chicken kept in commercial and backyard production system. Infectious bursal disease virus is a primary affect bursa fabricia of young chicken and is host-specific. This virus is prone to mutation, extremely stable and unaffected by chemicals and disinfectant. The most common route of infection is oral, conjunctiva and respiratory. The infectious bursal disease is extremely contagious and its severity is depends on age, breed of the affected birds, the degree of passive immunity and the virulence of the strain of the virus, and secondary infections associated with the immunosuppressive effects of the disease. Control of the disease has been more difficult by the popularity of variant strains of the infectious bursal disease virus. Variant viruses induce damage within the bursa fabricia in chickens, even once high and uniform protein titers are presence. The infectious bursal disease has an enormous economic impact on production varying from direct to indirect losses. Direct losses of disease are due to mortality and the financial losses due to reduced production parameters as a result of subclinical infections. The indirect economic impact of the disease is immunological disorder, growth retardation and condemnation of carcasses. With proper use of vaccine and vaccination program together with other measures like sanitation, good nutrient, high level of management in poultry farms was suggested.

Keywords: Chicken; Epidemiology; Economic; Infectious bursal disease; Ethiopia.

Introduction

Chickens are the most important species, adapted globally to different ecological conditions where human beings live and are important to subsistence, economic and social livelihoods of a large human population [1,2]. Chickens are particularly vital to women, children, and aged people, who are the foremost vulnerable member of the society in terms of under-nutrition and poverty; contribute a major role in provision animal origin protein to enhance human nutrition. The total national annual poultry meat and egg production are estimated

at 72, 300 and 78, 000 metric tons, respectively and indigenous poultry contributes almost 99% of the national egg and poultry meat production [1].

Poultry production in Ethiopia has a long traditional practice which is characterized by low input and low output [2]. Although an effort are made to enhanced chicken productivity and their contribution via introducing exotic chickens and as well by crossbreeding and distributing improved breeds to poor farmers living in the rural part of the country [3]. The introduction of diseases of assorted etiologies into many poultry farms simultaneous with the importation of exotic breeds to backyard chickens is changing into a growing concern [3]. Among those disease that might limit the potentiality of chickens are Infectious bursal disease (IBD) and Newcastle disease (NCD) have remained as two most significant infectious diseases threatening the village chicken and industrial poultry production in most part of the world [4,5]. Accompanying the intensification of poultry farming; constraints associated with the prevalence of infectious diseases are challenging factors. Among these, the infectious bursal disease is that the one that becomes to cause frequent outbreaks and a significant threat and a challenge to poultry producers [6].

The infectious bursal disease is an acute, extremely contagious, immunological disorder and economically significant poultry disease caused by Birnaviridae ribonucleic acid virus [7,8]. The disease damages the humoral immunity producing lymphoid organ bursa of fabricius and result in immunosuppression and increase the susceptibility of poultry to opportunistic secondary infections such as Marek's disease and Newcastle disease [9,10]. The infectious bursal disease is characterized by a typical clinical sign of those an acute immune depression, with depression, prostration of the affected birds, diarrhea, during the first weeks of life. The disease is spread through orally via contaminated feed and water [11].

Infectious bursal disease (IBD) is worldwide distribution that occurs in all major poultry producing areas inflicting a significant obstacle to productivity and profit within the poultry industries of each developing and developed countries [12]. The recent re-emergence of the infectious bursal disease virus (IBDV) in the form antigenic variants and hypervirulent strains has been the reason for major losses. Infectious bursal disease (IBD) has wide socio-economic importance at the international level because the disease existing in >95% of the member countries and also the incidence of acute clinical cases (vvIBDV) were stated in 80% of the country [13]. The Infectious bursal disease inflicts threat through mortality, decreased weight gain, and condemnation of carcasses as a result of marked heamorrhage in the muscle and secondary losses because of immunological disorder [13,14].

Since the contribution of poultry production to smallholder farmers and country economy is still restricted by Infectious Bursal disease (IBD) or Gumboro in addition to low inputs of feeding, poor management, and lack of applicable selection and breeding practice. This disease may cause mortalities of a lot of chicken population an estimated to range from 20% to 50% but they can rise as high as 80% during epidemics [15]. Infectious bursal disease virus (IBDV) causes prolonged immune-suppression of chicken making them vulnerable to get other infections and cause the failure of vaccinations against other diseases like Newcastle disease (NCD) [9,10]. This disease, therefore, hinders the successful control of NCD in the village chickens by vaccination method. Indirect losses due to IBD reported were impaired growth and the condemnation of carcasses [3]. Furthermore, the prices of increased use of antibiotics and chemicals to fight against secondary infections that arise as a consequence of IBD are encountered by the farmers.

Although IBD is widespread a viral disease, there is scanty of information on the epidemiology, status and economic importance of the disease in the chickens that kept under different level of the small scale and large commercial farms and local (backyard) poultry production system [3]. Unless it is to be difficult to design and implement chicken health cost-effective preventive and control programs without an understanding of the status and epidemiology disease in different level of the poultry production system. Hence, the target of this paper was to review the epidemiology, status and economic importance of infectious bursal disease in the poultry production in Ethiopia.

Infectious Bursal Disease

Etiology

Infectious bursal disease (IBD) is an acute, extremely contagious infection of young chickens (*Gallus gallus domesticus*). The etiological agent is infectious bursal disease virus (IBDV) which belongs to the family Birnaviridae, whose primary target is the lymphoid tissue of the bursa of Fabricius [13,16]. Characterization of the viral genome as bi-segmented double-strand RNA [17] allowed placing IBDV into a new family of virus the Birnaviridae contains the genera which affect chicken, fish, and insect [16]. Among the family includes three genera: (Aquabirnavirus, Entomobirnavirus, and Avibirnavirus); Avibirnavirus whose type species is infectious bursal disease virus (IBDV), which infects birds [18].

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The genome of Infectious bursal disease virus consists of 2 segments, A and B that are enclosed inside a non-enveloped icosahedral capsid [6,17]. The virus has five proteins recognized as VP1 to 5. The little section B of the genome encodes for VP1 and the large segment A encodes for VP2, 3, 4, and 5. The VP2 and VP3 are the most important proteins constituting fifty one and forty percent severally of the whole proteins. While the VP2 has the serotype-specific epitope protein contains important neutralizing antigenic sites and elicits a protective immune response and most of the amino acid (AA) changes between antigenically different IBDVs are clustered in the hypervariable region of VP2 [17,19]. Thus, this hypervariable region of VP2 is that the obvious target for the molecular techniques applied for IBDV detection and strain variation studies [6,18].

Two distinct serotypes of infectious bursal disease virus (IBDV) are legendary to indicate clinical disease in chickens younger than ten weeks [20]. Older chickens typically show no clinical signs. Antibodies are generally found in different avian species, however no signs of infection are observed. Serotype one is accountable for clinical cases of Gumboro to that commercial vaccines against Gumboro disease were mostly produced [20]. Serotype one IBD viruses are classified as attenuated (vaccine strains), classical (standard), antigenic variant, and extremely virulent (also called hypervirulent) strains based on their phenotypic traits (such as antigenicity and pathogenicity) and by the genetic traits that is, VP2 amino acid sequence differences [13,21].

Of the 4 phenotypic traits of serotype 1 that exist in the field, the hyper or very virulent IBD virus is capable of infecting chickens in the presence of maternally derived or higher levels of vaccinal antibodies causing very high mortalities and bursal damage with severe economic losses [22,23]. Serotype two antibodies are prevalent in turkeys and are seldom found in chickens and ducks. There are no reports of clinical disease caused by infection with the serotype 2 virus and are thus apathogenic for chicks [24].

Pathogenesis and clinical signs

Pathogenesis is defined as the method employed by the virus to cause injury to the host with mortality, disease or immunological disorder as a consequence. The injuries can be evaluated at the level of the host, the organ and the cell BDV usually infects young chickens between 3-6 weeks of age and causes clinical disease, but sub-clinically infecting older birds. The occurrence of Infectious bursal disease is based on strain, number of the infecting virus, the age and breed of the birds, route of inoculation and presence or absence of neutralizing antibodies [25].

Four to five hours after oral infection virus can be detected in macrophages and lymphoid cells in the cecum, duodenum, jejunum and kuppfer cell of the liver. The bursa is infected via the bloodstream and by 11 hours many cells in this organ contain antigen. A viremia follows when the virus infects other organs including the spleen, the harderian gland, and the thymus lymphocyte and their precursor appears to viral antigen may be found within the bursa up to fourteen days post-infection [26].

In some birds, the kidneys appear swollen and may have urate deposit and cell debris that most likely leads to blockage of ureters by a severely swollen bursa. The cause of muscle hemorrhage is unknown. Bursa depletion as the result of virulent IBD virus infection in early life can result in impaired immune responses to antigens and the response to IBD virus itself. Although there are reports indicating that infection as late as four weeks of age results in poor response to certain antigens. This not all the cases and the severity of the infection and whether or not maternally derived antibody (MDA) modified the disease could be important. The consequence of immunosuppression is lowered resistance to disease and suboptimal response to a vaccine is given during this time [27].

The clinical symptom is described as acute onset of depression, trembling, white and watery diarrhea, anorexia, prostration, ruffled feather, and vent feather solids with urates; in severe cases, bird became dehydrated and in terminal stages subnormal temperature and death [3,28]. Mortality commences on the third day of infection, reaches a peak by day four, then drops speedily and also the extant chickens recover a state of apparent health after five to seven days.

The severity of Infectious bursal disease is based on age and breeds sensitivity, the virulence of the strain and level passive immunity as well. If the virus persists on the farm and is transmitted to consequent flocks, the clinical sign the disease seem earlier and are gradually replaced by subclinical forms. Moreover, the first infection might also be unobvious once the infectious agent strain is of low pathogenicity or if maternal antibodies are present [29]. As cited by Tsegaye K [30] Infectious bursal disease follows one of two courses such as subclinical, and or clinical IBD, depending on the age at which chickens are infected. The subclinical variety of the disease happens in chickens less than three weeks aged. Chickens present no clinical signs of disease but experience permanent and severe in immune-suppression. The rationale young chickens exhibit no clinical signs of disease is not best-known. However, immune-suppression occurs due to damage to the bursa of Fabricius [26].

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The typical gross lesions of the disease involve dehydration of the muscles with ecchymotic hemorrhages, enlargement and orange discoloration of kidneys. The bursa of Fabricius shows that the foremost diagnostic lesions in birds that die at the peak of the disease. It becomes enlarged and shows pale discoloration. Intra-follicular hemorrhages may be found and pinpoint hemorrhages on the skeletal muscles are usually prominent.

Figure 1: Clinical signs (severe depression and high mortality) of Gumboro disease from the farm [23].

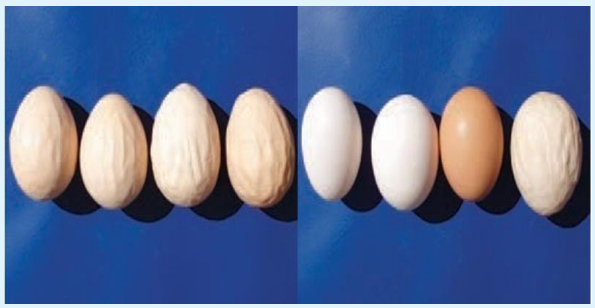

Figure 2: Deformed and pigment less eggs or eggs with soft shells and watery egg white in layer hens infected with the IB virus [21].

The infectious bursal disease produces gross lesion. The tissue distribution and severity of lesions are dependent on the subtype and pathogenicity of the virus. Infected birds are dehydrated and have darkened discoloration of pectoral muscles. Hemorrhages occur within the thigh and pectoral muscles and are stated from the mucosa at the proventriculus-ventriculus junction and on the serosal surface of the bursa. Increased mucous secretion within the intestine and renal changes are recognized in infected chicken that are because of dehydration [21]. In birds that are died or in the advanced stage of the disease, the kidneys tubules and ureters are so distended and filled with urates that they appear white in the predominant lymphoid organ affected by IBDV [31].

The cloacal bursa is that the organ for the replication of IBDV and therefore the foremost severely affected. Thus, it is important to understand the sequence of change while examining bird at postmortem. On the 3rd day following infection, bursa begins to increase in size and weight, because of the accumulation of fluid [16]. By the 4th day, bursa usually is double its normal weight and size, and then begins to decrease in size. From the 8th day onward, it is about one third its normal weights [23]. The bursa usually show necrotic foci (area of dead tissue) and cheesy mass is found within its lumen from the fallen cell of tissue. At time small-large hemorrhage on its inner surface (mucosal surface) is also seen. Sometimes widespread hemorrhage throughout the entire bursa is present in such case; a bird may pass blood in their drooping [32]. Moderate to severe splenomegaly with small gray foci uniformly distributed on the surface has been reported. Occasionally, petechial hemorrhages have been in the mucosa at the junction of Proventricules and gizzard [33].

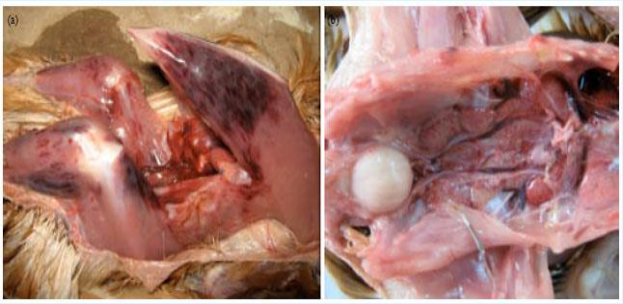

Figure 3: Gross pathology of (haemorrhagic muscles, inflamed bursa of fabricius) in the Gumboro infected bird [23].

Epidemiology of Infectious Bursal Disease

Infectious bursal disease was first reported as a particular disease of affecting the bursa of Fabricius in chickens was reported by Cosgrove in 1962. The first cases were observed in the area of Gumboro, in Delaware in the Bunting farm of USA, which is the origin of the name, although the terms 'IBD' or 'infectious bursitis' are more accurate descriptions. Between 1960 and 1964, the disease affected most regions of the USA and reached Europe in the years 1962 to 1971 [34]. From 1966 to1974, the disease was known in the Middle East, Southern and Western Africa and India. The disease is currently an international problem. Infectious bursal disease (IBD) occurs worldwide in the major poultry production area [29].

Host Range

Infectious bursal disease virus (IBDV) is host-specific. Although serologic evidence of natural infection with the virus has been reported in turkeys, ducks, guinea fowl and ostriches may be infected, the

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clinical disease occurs only in chickens [20]. It is strongly believed that the serotype IBDV one is highly hosted specific to chickens which develop IBD after infection by serotype 1 viruses. Reports have shown that serotype 2 of IBDV is more prevalent in many species of wild birds, with the natural host considered to be turkeys [35]. Infectious bursal disease virus has recently been isolated from a sparrow in China suggesting that wild birds could act as carriers [36]. The duck can also be an asymptomatic carrier of serotype 1 is no confirmation that the IBD virus could infect other animals, as well as humans.

Characteristics of IBDV

The most interesting feature of IBDV is its ability to remain infectious for a very long period of time and its resistance to commonly used disinfectants [24]. Infectious bursal disease virus includes a short time period of 2-3 days and therefore the infection usually lasts 5-7 days. One of the earliest signs of IBDV infection is the tendency for the bird to engage in vent picking [29]. It is susceptible to mutation, highly stable and resistant to a variety of chemical and disinfectant like phenolic derivative and a quaternary ammonium compound, but iodine complex has a deleterious effect on virus; and can persist in faces, bedding, contaminated feed and water for up to four months in certain conditions. It is also resistant for treatment with chloroform and ether, remains viable from pH 2-12 and is inactivated only in 70°C for 30 minutes. The virus is unaffected by exposure for one hour at 0.5% to 30% phenol but virus infectivity was markedly reduced when exposed to 0.5% formalin for six hours. Infectious bursal disease virus (IBDV) also heat stable, viable after treatment at 56°C for 5 hours [34,37,38].

Transmission of IBD Virus

Chickens are the sole well-known avian species to develop clinical disease and distinct lesions once exposed to IBDV. The IBD transmits with a horizontal way only, with healthy subjects being infected by the oral or respiratory pathway. The most common mode of infection is through the oral route. Conjunctiva and respiratory routes may also be involved [11]. There is no evidence of egg transmission of the virus and no carrier state has been detected in chickens. The lesser mealworm (*Alphitobius diaperinus*) is recognized as a carrier and the virus has been isolated from mosquitoes (*Aedes vexans*) and evidence of infection in rats has been reported but there is no indication that either species is a reservoir for the virus. Infected subjects excrete the virus in faces as early as 48 hours after infection, and may transmit the disease by contact over a sixteen-day period. The possibility of persistent infection in recovered animals has not been studied. The IBD is very contagious and will be transmission by the movement of poultry product, equipment, feed bags, vehicles, and people. In lesser extent the disease also spread through aerosols of the dust [11,39].

There is no data that suggest IBDV is transmitted by wild birds, however direct or indirect transmission of the virus between wild birds and domestic chickens probably occurs [40]. The extreme resistance of the virus to the outside environment and its viral incubation period is about 2-3 days and can be shed as soon as 24 hours following infection and may last up to 2 weeks. In the absence of effective cleaning, disinfection, and insect control can enhance the potential for transmission when they are scavenging of dead chickens, ingestion of contaminated water, or exposure of respiratory or conjunctiva membranes to contaminated poultry dust [35,41].

Immunosuppression Effect of the Virus

Immunosuppression caused by IBDV has a significant economic impact due to the widespread nature of the disease in commercial chickens. The virus infection at early age compromises the immune responses of chickens. All generated the earliest observation regarding the immunological disorder potential of IBDV [24]. The extent of the immunosuppressive effect is related to the age at infection. The most pronounced damage results if the infection happens in the first 2-3 weeks of the hatch [24]. The birds less than 3 weeks ages do not exhibit clinical signs however are immunological disorder. After the virus was ingested by birds, the virus infected lymphoid cells and macrophage of intestine then the virus carry to bursa of Fabricius [6]. Clinical signs and lesions of IBDV seem shortly after. The infected chicken with Infectious bursal disease is more prone to secondary infections especially Newcastle disease (ND). The infected chicken had a decreased humoral response to vaccines as well. Immunosuppression resulted in lower flock performance, a lot of secondary infections, poor feed conversion, less protecting response to vaccines and a higher rate of carcass condemnation at the process level [13].

Susceptibility Factor of the Infectious Bursal Disease

The time wherever chickens are most prone to IBD is between 3 and 6 weeks once the bursa of Fabricius is at its maximum rate of development and the bursa follicles are filled up with immature lymphocytes [13]. This is because the IBD virus replicates in and cytolytically affects the actively dividing B lymphocytes in the bursa of Fabricius [13]. Infections occurring before the age of 3 weeks are typically subclinical and immunological disorder. Clinical cases may be observed up to the age of fifteen to twenty weeks [42]. Light strains of laying stock are more susceptible to disease than the heavy broiler strains.

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Morbidity and Mortality

The infectious bursal disease is extremely contagious and its severity depends on the age and breed of the affected birds, the degree of passive immunity and the virulence of the strain of the virus, and secondary infections associated with the immunosuppressive effects of the disease [13]. In infected flocks, morbidity is high, with up to 100 percent serologic conversion, once infection, while mortality is variable. Until 1987, the field strains isolated was of low virulence and caused just one to twenty of specific mortality. However, since 1987 a rise in specific mortality has been described in several part of the world. In the USA, new strains to blame for up to five percent of specific mortality were described. At a similar time, in Europe and later in Japan, high mortality rates of fifty to sixty percent in laying hens and 25% to 30% in broilers were observed. These hyper virulent field strains caused up to 100% mortality in specific-pathogen-free (SPF) chickens [13]. While in Ethiopia the mortality rate of the disease in several poultry house ranges from 45-50%. Broiler mortality was about fifty six percent whereas 25.08% for layer chickens [3].

In totally prone flocks, mortality related to infection because of classic strain might vary from 1-60% with high morbidity of up to 100%. A variant IBDV strains do not produce overt clinical signs, however cause immunological disorder and could cause mortality because of secondary opportunist infections in immune-compromised birds. In contrast, vvIBDV strains cause mortality of 50-60% in laying hens, 25-30% in broilers and 90-100% in prone leghorns. Susceptible chickens younger than three weeks of age may not exhibit clinical signs but develop subclinical infections. This results in a decreased humoral antibody response due to B lymphocyte depletion in the cloacal bursa and severe and prolonged immunosuppression. The most significant economic losses result from subclinical infections this form of IBD infection greatly enhances the chicken's susceptibility to sequels such as gangrenous dermatitis chicken anemia virus, inclusion body hepatitis, respiratory diseases and bacterial infections [13].

Diagnosis

Diagnosis of IBD involves thought of the flocks' history, and of the clinical signs and lesions. Obviously, chickens less than three week's age present no clinical signs of disease, whereas chickens larger than three weeks age presence clinical signs as described. The severity of the clinical signs can rely upon the factors described. While the clinical diagnosis of the acute forms of IBD is based on disease evolution of a mortality peak followed by recovery in five to seven days and relies on the observation of the symptoms and post-mortem examination of the pathognomonic lesions, in particular of the bursa of Fabricius [43].

Symptomatology and Gross Lesions

High mortality and severe clinical signs are the characterized of Hypervirulent IBDV. Indeed, the vvIBDVs produce disease signs almost like conventional type one infection, with a similar incubation period (4 days) however, the acute phase is exacerbated and more generalized in the affected flock. Severe outbreaks are characterized by sudden onset of depression in susceptible flocks [20]. Animals in the acute phase of the disease are prostrate and reluctant to move, with ruffled feathers and frequently watery or white diarrhea. The age susceptibility is extended, covering the entire growing period in broilers, and the peaks of mortality show a sharp death curve followed by rapid recovery clinical IBD has clearly typical signs and post-mortem lesions. A flock will show very high morbidity with severe depression in most cases lasting for 5-7 days. Mortality rises sharply for two days then declines rapidly over the next 2-3 days. Usually, between 5% and 10% of birds die, but morbidity can reach 30-40% [13].

Bursal of Fabricius is turgid, oedematous, heamorrhages and atrophic seven to ten days in chicken that died by acute stage of vvIBD through post mortem examination. This atrophy might be more rapid, even 3 to 4 days after inoculation. In addition, dehydration and nephrosis with enlarged kidneys are common, and heamorrhages in the muscle and mucosa of proventriculus are known in the most of affected chicken. Severe depletion of lymphoid cell is recognized both in bursa of Fabricius and non-bursal lymphoid tissues. Pathogenicity of IBDV has been related to virus distribution in non-bursal lymphopoietic and hematopoitic organs. Indeed, using numerous immune staining ways, a higher frequency of antigen-positive cells can be demonstrated after infection of birds with vvIBDV compared with other strains, in the thymus [11], the spleen and the bone marrow. In specific, atrophy of the thymus has been related to the acute stage of the disease and could be indicative of the virulence of the isolate, though it is not related to extensive viral replication in thymic cells. An increasing number of macrophages are found in numerous organs [44]. Thrombocytes also represent a target for IBDV, and acute disease is characterized by disseminated hemorrhages probably related to an impairment of the clotting mechanism [20].

Confirmation of a diagnosing of clinical IBD may be created at autopsy by examining the BF during the first stages of disease for characteristic gross lesions. During later stages of the disease, it is troublesome to confirm an identification of IBD by examining solely shrunken, atrophied BF, like other diseases produce similar

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changes. In birds below three weeks of age or in young chickens with maternal antibodies, IBD infection is usually subclinical. Thus, typical clinical signs are not present, and identification ought to be supported by histopathologic study of the suspect bursa of Fabricius, serological studies, or by virus isolation [43-45].

Isolation and Characterization of the Virus

Diagnosis depends on the isolation and characterization of the virus and its differentiation from endemic serotype 1 viruses; it can be made through the following methods:

Histological Diagnosis

Histological diagnosis is based on the detection of modifications occurring in the bursa. The ability to cause histological lesions in the non-bursal lymphoid organs, such as the thymus spleen or bone marrow [46] has been described as a possible characteristic of hypervirulent IBDV strains. The histological diagnostic method has the advantage of allowing for the diagnosis of both the acute and chronic or subclinical forms of the disease. Detection of viral antigens: thin sections of the bursa of Fabricius prepared to detect viral antigens specific to IBDV done by direct and indirect immunofluorescence or by immunoperoxidase staining within the bursal follicles of infected chickens between the fourth and sixth day once inoculation. No viral antigen is detectable from the tenth day. However, the virus can be isolated from bursae sampled from the second to the tenth day, with a maximum infectious titer after four days [20].

Virological Diagnosis

Infectious bursal disease virus is also detected within the bursa of Fabricius of chicks in acute phase of infection, ideally inside the primary 3 days following the looks of clinical signs. Isolation: A filtered homogenate of the bursa of Fabricius is inoculated in nine to eleven-day-old embryonated eggs originating from hens free of anti-BDV antibodies. The most sensitive route of inoculation is the chorioallantoic membrane (CAM); the yolk sac route is also practicable, and the intra-allantoic route is the least sensitive [20]. The specificity of the lesions observed must be demonstrated by neutralizing the effect of the virus with a monospecific anti-IBDV serum. Isolation in embryonated eggs does not require adaptation of the virus by serial passages and is suitable for vvIBDVs. In the absence of lesions, the embryos from the first passage should be homogenized in sterile conditions and clarified, and two additional serial passages should be done [21].

Serological Diagnosis

An AGID, VN or enzyme-linked-immunosorbent serologic assay may be conducted on serum samples. The infection typically spreads speedily in a flock of birds. Attributable to this, solely small percentage of the flock must be tested to find the presence of antibodies. If positive reactions are found in susceptible birds then the total flock should be thought to be infected [20]. Isolation of IBDV is not conducted as a routine procedure. Specific antibody-negative chickens may be used for this purpose, as may cell cultures or embryonated eggs from specific antibody-negative sources. On the other hand, some difficulty may be practiced in using latter two systems as virus does not readily adapt to them. If effective, the identity of the virus is often confirmed by the virus neutralization (VN) test. The agar gel Immuno-diffusion (AGID) test can be used to detect viral antigen in the bursa of Fabricius. A segment of the bursa is removed, make uniform and used as antigen in test against known positive antiserum. This is particularly useful in the early stages of the infection, before the development of an antibody response. An immune-fluorescence test using IBDV-specific chicken antiserum can also be used to detect the antigen in bursal tissue. Antigen-capture enzyme-linked immunosorbent assays (ELISAs) supported plates coated with IBDV- specific antibodies have also been described for the demonstration of infectious agent RNA within the bursa of Fabricius [20].

Current serological tests cannot distinguish between the antibodies induced by pathogenic IBDV and those induced by attenuated vaccine viruses, so serological diagnosis is of little interest in endemic zones wherein areas contaminated by IBDV, most broiler flocks have anti-IBDV antibodies when leaving the farm. Nonetheless, the quantification of IBDV-induced antibodies is important for the medical prophylaxis of the disease in young animals, in order to measure the titer of passive antibodies and determine the appropriate date for vaccination [47] or in laying hen so verify the success of vaccination. Serology is likewise essential to confirm the disease-free status of flocks. Each serological analysis must include a sufficient number (at least twenty) of individual serum samples representative of the flock under study.

Molecular Identification

Molecular diagnostic assays are the most frequently used to determine IBDV in diagnostic samples. They use reverse-transcriptase PCR to seek out the viral genome in bursa tissue. Sequence analysis of the VP2 coding region has been used to further characterize the viruses [42]. Most efforts at molecular identification have concentrated on the characterization of the larger fragment of IBDV (segment A) and particularly of the vVP2 cryptography region.

Several protocols have been published on characterization using restriction endonucleases of RT-

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PCR products. These methods are mentioned to as RTPCR/RE or RT-PCR-RFLP (restriction fragment length polymorphism) [48]. The usefulness of the information they provide depends on the identification of enzymes that cut in restriction sites that are phenotypically relevant. Some sites involved in antigenicity have already been identified, however, restriction sites reliably related to virulence still need to be defined and validated. Nucleotide sequencing of RT-PCR products, although more expensive than restriction analysis, provides an approach to assessing more precisely the genetic relatedness among IBDV strains. Markers have been demonstrated experimentally, using a reverse genetics approach, for cell culture-adapted strains, which exhibit amino acid pairs 279 N–284T. In virulent viruses, four characteristic amino acids are existed (222 A, 256 I, 294 I and 299 S) [49]. However, it is not yet known whether these amino acids play a task in virulence or if they're simply a sign of the being origin of most vvIBDV isolates. Several recent studies indicate that although VP2 is an important virulence determinant, it may not be the only one. It has been reported that segment A and B of IBDV essentially co-evolve (i.e. most vital IBDV clusters, like vvIBDV-related strains, is also known by analysis of both genome segments), however, some potentially re-assorting viruses are known [50].

Control and Preventions

There is no specific therapy for the disease. Facilitate access to water to prevent dehydration. As with every disease optimize climate and reduce stress to a minimum. The use of antibiotics can sometimes be advisable to limit the impact of secondary infections [51]. Control of IBD has been more difficult by the popularity of variant strains of the IBD virus. Variant viruses induce damage within the BF in chickens, even once high and uniform protein titers are presence. Variant strains do not cause obvious clinical disease, but immune-suppression. In Chicken affected by classical IBDV the bursa of Fabricius undergo rapid atrophy (lymphocyte depletion) without inflammatory changes observed early in the infection. These variants are not from a different serotype, but are antigenically different enough to cause immunosuppression problems [24]. An additional important feature of IBD is its immunosuppressive action that may interfere with the efficiency of vaccination programs.

Usually IBD could be a significant problem in integration and loss ensue persistent efforts at reducing field virus's exposure through a biosecurity program, maintenance of adequate and uniform maternal titers and an effective broiler vaccination program with a suitable vaccine and at a proper age. In this case, consideration should be given to vaccinating breeders with inactivated vaccines containing standard and variant strains of the IBD virus occurs [24]. Immunization of breeders is a vital a part of the IBD management program. Antibodies produced by the hen are gone through the egg to the broiler chick. These maternal antibodies, if existing in adequate levels, defend the chicks against subclinical IBD.

Live vaccines are administered to achieve active immunity but interference of maternally derived antibody (MDA) is the crucial problem in determining a successful live IBDV vaccination schedule. Vaccinating chickens in the presence of high levels of maternally derived antibodies results in vaccine virus neutralization and no immunity [52]. Currently as reported by Hagazi F [53] in Ethiopia, determining the proper time for administration of live intermediate IBD vaccine important than giving IBD vaccine to chickens whose parents that have taken IBD vaccine without determining maternally derived antibodies (MDA) concentration and age for vaccination. And therefore, in order to have chickens protected from IBDV field challenge, it is crucial to determine the optimal timing for IBD vaccine delivery. The optimum timing is commonly foreseen supported serological data following the detection of IBDV MDA by ELISA system throughout the first-week post-hatch. The "Deventer formula" was developed to estimate the optimum vaccination time supported the half-life time of the MDA, the age of the chicken at sampling, genetic background, breakthrough concentration of the immunizing agent, and also the requested proportion of the flock having antibody levels below the breakthrough concentration of the immunizing agent at the time of administration [54].

The dramatic impact of a very virulent IBD virus can be reduced by proper clean-up and disinfection between flocks, and that traffic (people, equipment and vehicles) onto the farm be controlled. The development and implementation of a comprehensive biosecurity program is that the most significant factor in limiting losses because of the IBD virus is extremely resistant and might survive for over a hundred days in contaminated area. Phenolic and formaldehyde compounds are shown to be effective for disinfection of contaminated premises [45]. Since the virus is extremely stable for months. It is largely excreted through feces hence contaminated litter, feed and water have to be burnt or buried deep under the lime cover. Besides these other measures are; lower stocking densities, increasing intervals between flocks and complete removal of organic waste between batches. In areas where management practices to reduce virus concentration are used, the disease tends to occur at a later age, and immunosuppressive form of infection is reduced [55]. Vaccination of inactivated vaccines to breeder hens induces long-lasting and high levels of antibodies in the hatched chicks. However in some

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areas wherever terribly virulent IBD virus has caused important losses, the producers do not adopt inactivated vaccination. However intensive live virus vaccination program is employed in hatched chicks from the susceptible breeder hens. Such chicks escape the strong risk of an immunosuppressive form of the disease [56].

Current Status of Infectious Bursal Disease in Ethiopia

Devastating outbreaks of the disease have been reported in many parts of the world and recently in Ethiopia [3]. The importance of the disease is represented by the high mortality, reduced productivity amongst infected chicks and accrued prone to other infections (accordingly, chickens also develop a poor immune reaction to vaccination against alternative pathogens [3,11].

The IBDV infection has as spread to all commercial farms and multiplication centers occur at an average outbreak rate of 3-4 farms per year. The disease was encountered usually in backyard poultry production systems similarly. Gumboro disease investigation was conducted by the NAHDIC in different Regions and with the result of overall prevalence rates to be about 77.48 % from the 706 samples collected and analyzed [57].

There were also studies carried out by different authors where the disease is prevalent in different parts of our country. Seroprevalence of 45.05% (173/384) of Infectious bursal disease (IBD) in chicken reared under backyard poultry production systems in Mekele town was reported [58]. Out of 552 serum samples tested 458 (83%) in backyard chickens at selected districts of Eastern Ethiopia by Tadesse B & Jenbere S [59] of 82.2% (227/276) reported on backyard chickens in both peasant associations and kebelles of Debre Zeit revealed the presence of IBDV specific antibody in the absence of vaccination, which indicate the presence of field exposure of household chickens to the virus by Tesfaheywet Z & Getnet F [14] and 27.8% of with a case fatality rates of 98.56% and 77.73% the incidence of IBD in chickens owned by 775 households in Amhara region of Bahir Dar and Farta district respectively by Hailu M, et al. [60]. Agar gel Immuno-diffusion test revealed the presence of antibodies against IBD in the serum of most recovered birds from IBD. Thus, it's of very importance design cost-efficient management ways against IBD so as to enhance the productivity and welfare of village chickens and additionally to conserve the indigenous chicken genetic resource [60].

The study conducted in eight districts of Ethiopia showed that among the total of 2,597 chicken serum samples, 83.1% (2158/2597) positive for IBD examined using ELISA. Among the predisposing factors location, age, breed are influenced the occurrence IBD. The highest seroprevalence was recorded in Mekele (90.3%) while the lowest was recorded at Gondar (69.8%). Moreover, higher seroprevalence was reported in crossbreed of chicken (91.4%) while the lowest was recorded in indigenous breed of chicken (81.4%). The production system can as well influence the occurrence of IBD [61].

Jenberie S, Lynch SE et al. [62] stated that phylogenetically, Ethiopia infectious bursal disease virus characterizes two genetic lineages: very virulent infectious bursal disease virus or variants of the classical attenuated vaccine strain (D78). The nucleotide identity between Ethiopian vvIBDVs ranged between zero and 2.6%. Ethiopian vvIBDVs are clustered phylogenetically with the African IBDV genetic lineage, difference from the Asian/European lineage. This report demonstrates the circulation of vvIBDV in business and breeding poultry farms in Ethiopia. Besides, among all IBDV strains represented within the study for phylogenetic comparison of VP2 nucleotide sequences, Ethiopian strains type cluster inside the vvIBDV lineage. There was also shown that Ethiopian IBDV strains have mutations in the VP1 region. This report could help to select the most appropriate vaccination program for the genomic sequences of field strains through diagnostic testing [63].

Study area	Prevalence	Authors
Gondar and west Gojjam	73.50%	Kassa and Molla [64]
Southwest showa of Ethiopia	76.64%	Hailu et al. [60]
Mekelle town	45.05%	Zegeye et al. [58]
Debre-Zeit	82.20%	Tesfaheywet and Getnet [14]
Andassa poultry farm	98.90%	Solomon and Abebe [44]
Eastern Ethiopia	83%)	Tadesse and Jenbere [59]
Debre Brehan	94.70%	Animal Health Yearbook [57]

Table 1: Reported prevalence of IBD in Ethiopia.

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Economic importance of Infectious Bursal Disease

Even though Infectious Bursal Disease (IBD) also called Gumboro; was first recognized more than 50 years ago, in 1962 in Gumboro, Delaware, USA. The IBD virus belongs to Avibirnaviruses caused to the immune system (bursa of Fabricius) causes severe immunological disorder by destroying B-lymphocyte precursors found inside the bursa of Fabricius that impairs the chickens' ability to develop antibodies therefore, lowered resistance to different infectious agents and poor response to usually used vaccines. Immunosuppressed flocks have poor performance that results in reduced economic returns [65]. It has been a greater concern for the poultry industry for long time, however significantly for the past decade. Indeed, its "re-emergence" invariant or highly virulent forms have been the cause of significant economic losses. Until 1987, the strains of the virus were of low virulence, inflicting less than two percent specific mortality, and satisfactorily controlled by vaccination. But in 1986 and 1987, vaccination failures were described in different parts of the world [13].

It was assessed that IBD has significant socio-economic importance at the international level, because the disease is present in more than ninety fife percent of the member countries [66]. In this survey, 80% of the countries reported the occurrence of acute clinical cases. The disease still causes significant economic losses and well known to worldwide poultry farmers resulting in a major setback to productivity and profitability in the poultry business all countries [12].

The infectious bursal disease has an enormous economic impact on production varying from direct losses to indirect losses. Direct losses IBD is because of mortality to secondary infections and also the monetary losses because of reduced production parameters as a result of subclinical infections. The indirect economic impact of the disease is also considerable, has most of the economic devastation associated with IBD due to its immunosuppressive effect that leads to secondary infections, growth retardation and condemnation of carcasses.

Moreover, the raised use of antibiotics against concurrent infections constitutes an increasing public health concern. Mortality is variable and tends to have an effect on layers more than broilers however can be up to 100 percent with very virulent strains of the disease. Even if birds survive, the resulting immunosuppression and effect on egg production in layer birds are significant [6].

In Ethiopia, an outbreak investigation was carried out in 2002 on suspected IBD case which was reported from a commercial poultry farm in Debre-Zeit town [3]. At the time of investigation started there was the mortality of 22,437 broiler chicken and 2508 layer chicken, and 40, 000 Hubbard broiler chickens and 10,000 Lohman Brown layer chicks were at risk in 4 weeks' time of an outbreak in the farm. While, economic losses associated with outbreaks and or occurrences of IBD in the studied farms, back yard village chickens and small scale poultry owned farmers of different part of the country may appeared unimaginable to the farm owners and farmers as the owner did not relent efforts to restock his farm. Infectious bursal disease (IBD) has been reported earlier to be a vital reason for economic losses within the poultry trade. Typically, the poultry producer concern is that this financial mortality cost of lost flock and never beyond if were the birds to survive. These combined losses are usually high, unthinkable and alarming if properly quantified as indicated clearly above. Infectious bursal disease is continues to be a serious disease problem of commercial and rural chickens and constitutes a serious threat to poultry production.

Conclusion

Conclusively infectious bursal disease is a serious viral disease that has a great economic impact throughout poultry production areas. This is recognized as an essential disease of young chickens worldwide. In Ethiopia infectious bursal disease is the main constraint to both commercial and backyard poultry production system. This disease is widely distributed in almost all part of the country imposes great losses on the economic development of the country. The most common mode of infection is through the oral route, but conjunctiva and respiratory routes may also be involved. Immunosuppression caused by infectious bursal disease virus has a significant economic impact due to the widespread nature of the disease in commercial chickens. The infectious bursal disease is host-specific and extremely contagious. The disease severity is depends on age, breed of the affected birds, the degree of passive immunity and the virulence of the strain of the virus, and secondary infections associated with the immunosuppressive effects of the disease. An effective Infectious bursal disease prevention and control method should involve an efficient breeder vaccination program, an efficient bio-security program and broiler vaccination program immunization of breeders is a vital a part of the infectious bursal disease management.

Competing Interests

The author declared not any competing of interests

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