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Chicken Infectious Anemia Virus

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Abstract

Chicken Anemia Virus (CAV), is the smallest DNA virus classified in the genus Gyrovirusof the family Circoviridae. Since its first identification in 1979, CIAV is a virus non- enveloped, stable and very resistant to the environment and disinfectants. It has a simple circular DNA chain. All CIAV strains are known until now belong to the same serotype, which means that there are no major antigenic differences, although there maybe diversity in the genome of the virus. CIAV infection has caused great economic losses for the poultry industry as the infected birds showed poor performance and high mortality rate between 10-20%, but might reach 60% in complicated cases and also due to its serious immunosuppressive potential and ability to predispose to multiple secondary bacterial infections, subsequently playing a key role in the etiology of several multifactorial diseases, immune suppression and the production of antibodies for chicken infectious anemia in field challenge. CIAV induces infection and can be closely associated many syndromes. The poultry industry needs the immunization of the breeding birds, in order to avoid the vertical and horizontal transmission of the virus and provide maternal antibody titers that give massive protection to the progeny.

This review aimed to collect available data about CIA to be available to be available to students, researchers and veterinarian in poultry practical.

Keywords: Chicken Infectious Anemia; Virus; Clinical Signs; Lesions; Diagnosis; Prevention and Control

Abbreviation: CAA: Chicken Anaemia Agent; CAV: Chicken Anaemia Virus; CIAV: Chicken Infectious Anaemia Virus; DIF: Direct Immunofluorescence; DPI : Days Post Infection; ELISA: Enzyme-Linked Immunosorbent Assay; IBD: Infectious Bursal Disease; IBDV: Infectious Bursal Disease Virus; IBH: Inclusion Body Hepatitis; IIF: Indirect Immunofluorescence; ILT: Infectious Laryngotracheitis; MD: Marek's Disease; MDV: Marek's Disease Virus; NDV: Newcastle Virus Disease ; PCR: Polymerase Chain Reaction; REV: Reticuloendotheliosis Virus; VN: Virus Neutralization.

Introduction

Chicken infectious anemia virus (CIAV) was first isolated in 1979 in Japan [1]. In fact, it was not a new disease but a newly recognized one. A retrospective serological survey evidenced that CIAV was present in the southeastern USA since at least 1959 [2], although it was first isolated in 1989 [3]. Since then, an increasing interest was paid to that virus, as it was found to have a great economic impact on poultry industry in all major chicken-producing countries of the world [4-6]. The CIAV is incriminated in a disease of young chickens, characterized by a transient severe destruction of erythrocytic and granulocytic series of the bone marrow cells, resulting in aplastic anemia and immunosuppression. The immunosuppression is caused whether directly by virus as it causes severe depletion of lymphocytes from primary and secondary lymphoid organs [1,7,8] or indirectly as it participates other immunosuppressive viruses such as IBDV [9,10], MDV or REV [9]. Experimentally, anemia was produced even when the chickens were inoculated at two or more weeks of age, or even in chickens with maternal antibody

[9,10]. The infected birds showed poor performance and high mortality rate generally between 10-20%, but might reach 60% especially in complicated cases [11]. The control of immunosuppressive diseases depends on biosecurity to avoid exposure to the causes of immunosuppressive diseases, and the increase of resistance to withstand the challenge of immunosuppressive agents through immunization and genetic selection [12].

Economic Importance

Chicken anemia virus causes an acute, immunosuppressive disease of young chickens, cutaneous, subcutaneous, and intramuscular hemorrhages; increased mortality [13,14], growth retardation [15]. Classical clinical disease is noticed in chickens of 2-4 weeks of age with characteristic anemia having lowered hematocrit (6% to 27%). Infected birds have a mortality of 5-10%, however, the mortality rate of 60% has been reported from the CIA infected flocks [16,17]. Chickens concomitantly infected with other viruses such as infectious bursal disease virus, avian adenoviruses, REV, avian reoviruses or MDV showed more severe and aggravated form of the disease [16].

Public Health Significance

Until recently CIAV was considered as a sole member of the Gyrovirus genus. Most commonly reported from chicken and considered of no public health significance. But, recent reports of 'Human Gyrovirus' (HGyV) from the human skin surface showing homology with CIAV [18] doubts its infectivity for human tissues. Furthermore, in 2012 two Gyrovirus (GyV3 and GyV4) were identified from the fecal samples of human consuming CIAV infected and/or vaccinated chickens [19,20]. CIAV shows many similarities with these human gyroviruses. Smuts, et al. [21] demonstrated the presence of CIAV and related gyroviruses in the South African human population with diarrhea, respiratory illness and some healthy individuals. CIAV has also been detected from the feces of mice and dogs [22]. Such instances indicate the potential ability of CIAV to threaten human health.

Epidemiology

Chickens are the only host for CIAV. The disease is spreading throughout the world mainly in countries of industrial production, the susceptibility of birds decreases with age [23]. After CIAV infection, viraemia and spread of the virus to the bone marrow, lymphoid organs, liver, heart, lung etc. Due to the hemocytoblastosis in the bone marrow, the alterations in other organs of the immune system, the birds will suffer from anaemia and immunodeficiency [24]. When a flock of breeders is negative to CIAV becomes infected during the laying period, vertical transmission occurs, which will start from 7 to 14 days after infection and may last from 3 to 9 weeks depending on the spread of the virus within the flock [25], and when the breeders seroconvert and reach sufficiently high levels of neutralizing antibodies, to avoid vertical transmission [26]. Breeders will not have clinical signs or lesions while the congenitally infected hatching bird will suffer from the disease, the first sign is an increase in mortality from 10 to 14 day of age with the peak at 21 day, and by horizontal transmission a second peak between 30 and 33 day [27].

CIA in Egypt

In Egypt, CAV is believed to be spread among chicken in Egypt since the early 1980's when several outbreaks occurred in many breeders [28]. It transmitted either vertically or horizontally [29,30]. It has been detected in most commercial poultry farms, with a high seroprevalence [4]. El-Lethi, et al. [31] reported the suspicion of CAV in dressed poultry and serological investigation have been proved the intensive exposure of commercial chicken to CAV [28,32,33]. Hussein, et al. [34] reported the molecular diagnosis of the CAV for the first time in Egypt and the isolate on of the CAV from clinical and subclinical infected broiler-breeder flock. CAV was isolated from broiler chicken in Egypt [35]. Pathological changes in bursa and thymus beside severe hypoplasia in hematopoietic cells in bone marrow of infected chickens were detected [36,37]. Mohamed, et al. [38] detected CAV DNA from 44 field samples out of 165 suspected broiler chickens of age up to 7 weeks with a percentage 26.6%. Hegazy, et al. [39] detected CAV DNA from 3 field samples out of 4 suspected cases with a percentage 75%. CAV isolate-collected from a commercial broiler flock

suffered from severe anemia and high mortality based on sequence and phylogenetic analysis of partial VP1 gene were characterize and pathogenicity and immunosuppressive potential in one day-old SPF chicks were studied by Nassif, et al. [40].

Pathogenesis

The entry route of the virus is determinant in the severity of the infection and time of appearance of the clinical signs and generation of immunity [41]. The experimental inoculation of CIAV intramuscularly in chicken birds of specific pathogens free (SPF) less than 4 weeks of age determines anaemia that begins 8 to 10 days post inoculation (PI) while birds inoculated by the oral route show a less severe reduction that starts at day 14 PI [41]. Anaemia occurs as a result of the destruction of erythroblast cells in the bone marrow. Affected

chickens show yellowish bone marrows instead of red characteristic of normal bone marrow [25]. Hematocrit has usually been restored to normal levels in surviving chicken birds around day 28 PI [42]. CIAV is also characterised to produce aplastic anaemia, intramuscular and subcutaneous haemorrhages, with causing atrophy of the thymus and the immunosuppression of young chickens [43]. It is common to observe haemorrhages in different parts of the carcass of chickens affected by CIAV, including the musculature. The cause of the haemorrhages is thrombocytopenia, which by reducing the platelet concentration decreases the coagulation capacity [44]. Weight gain is reduced following a temporal pattern similar to that observed in the hematocrit [44]. Thus, chicken birds inoculated intramuscularly reduce weight from 8 to 10 days PI while oral inoculation decreases weight gain from day 14 PI [43]. Weight loss is more drastic in chickens infected by the parenteral route. Unlike hematocrit, weight loss has not been restored until day 28 PI with differences of up to 35% lower compared to non-inoculated controls [44]. CIAV induces destruction of lymphoblasts particularly in the thymus. Histopathological lesions in the thymus are characterized by lymphoblastoid depletion, particularly in the cortex of the shy lobe, presence of intranuclear inclusion bodies and cells with apoptotic alteration [45].

Taxonomy and Characteristics of CIAV

CIAV is the smallest DNA virus classified in the genus Gyrovirusof the family Circoviridae, of 25 nm, the etiology behind CIAV, has gained great importance as an emerging poultry pathogen worldwide [46]. CIAV is 23.5-25 nm in size. It is believed that CIAV genome replicates through rolling circle model [47] and is very hardy, difficult to inactivate thermally or with common disinfectants, which limits the utility of normal sanitization practices. CIAV is generally considered to be omnipresent in both egg and meat-type chickens worldwide. Affected young birds appear anorexic, depressed, and with aplastic anaemia, which is characterized by PCV values ranging from 6 to 27%, with consequences including generalized lymphoid depletion and immunosuppression. In the breeding periods, CIAV is responsible for increased mortality, reduced performance and decreased resistance to viral and bacterial diseases [48].

Transmission

The horizontal transmission is produced by direct contact with diseased and indirect birds with infected material. The route of entry of the virus is by ingestion or inhalation [27]. The virus, when transmitted through the transovarial route, can cause severe offspring disease, characterized by anaemia, subcutaneous hemorrhage, and a decrease in resistance to secondary bacterial diseases such as gangrenous dermatitis [21]. Affected birds, if co-infected with the IBV, can develop deep immunosuppression with increased susceptibility to a wide range of viral and bacterial pathogens [23]. Clinical form of the disease it occurs when susceptible chicks are infected very early (breeders with active infection) [49]. It is of acute presentation, occurs between 7 and 14 day of age. Birds show depression and there is peak mortality between 17 to 24 day of age. There may be a second peak of mortality at 30 to 34 day of age, probably due to horizontal transmission [24].

Subclinical Form of the Disease

Maternal antibodies remain for 2 to 3 weeks, after which the birds become susceptible to infection [50]. Subclinical infection leads to a decrease in productive performance with consequent economic losses [51]. Subclinical infection of CIAV results in immunosuppression. Indirect evidence includes inadequate responses to vaccination such as NVD, ILT and MD [52] high initial mortality and MD in pullets, as well as an increase in the pathogenicity of several agents such as MDV, Reovirus, IBDV, NDV, IBH, Staphylococcus aureus and Cryptosporidium spp [53]. The virus inoculated in birds older than 6 weeks of age can be found in several organs. It is very difficult to isolate the virus after 2 weeks of infection. After the natural atrophy of the thymus it is more difficult to find the virus or lesions. Bursectomized birds may suffer from the clinical form for longer [53].

Clinical Signs and Lesions

In birds infected at very early ages, the manifestations they present are not specific to the disease such as: depression, growth retardation, ruffled feathers, increase in dead and culls from 10 to 14 days after infection [54]. Mortality is usually between 5 and 10%, although depending on the pathogenicity of the strain, simultaneous infections can cause much higher levels; the morbidity within the flock is almost 100% with the consequent production decreases [27]. Macroscopic lesions are more frequently observed at necropsy, with a pale or yellowish bone marrow due to anaemia, severe atrophy of the thymus, hemorrhages in the proventriculus mucosa as well as muscular and subcutaneous [42]. In the latter case they may appear in the wing, and may be complicated by secondary bacterial infections and gangrenous or blue wing dermatitis. The hematocrit will be below 20% in birds suffering from CIA anaemia from 8 to 10 PI [27].

Clincal pathology and Histopathology

The hematocrit, which under normal conditions is around 27%, will be below 20% in birds suffering from

anaemia from 8 to 10 days after infection [27]. Sakr, et al. [36] observed marked depletion of the lymphocytes in the thymus and bursa of Fabricius beside severe hypoplasia in hematopoietic cells in bone marrow. In addition, some bursal changes with various degrees of atrophy in the lymphoid follicles with scattered necrotic foci were probably attributed to secondary infections [37]. Infection with CIAV in chicken produces decreased hematocrit, leucopenia, erythropenia, and thrombocytopenia [55]. Necrotic foci were observed in the liver, kidneys, lungs, proventriculus and caecal tonsils. Lymphoid follicles had been depleted of cells and appeared swollen. The liver was swollen and sinusoids were dilated. Eosinophilic intranuclear inclusion can be observed in bone marrow and thymus of infected birds [56]. Occasionally, intranuclear inclusions can be detected in the spleen, proventriculus, lung, kidney, bursa of Fabricius and skin [16]. Chicks that manage to survive, repopulation of the thymus with lymphocytes become distinct by 20-24 days and appear normal after 32-36 days [57]. Bursa had mild to severe lymphoid follicle atrophy with occasional small necrotic foci in folded epithelium along with hydrophic epithelial degeneration, perifollicular edema and proliferation of reticular cells [58]. Spleen had depletion of T cells with hyperplasia of reticular cells in the lymphoid follicle as well as in Schweiger-Seidl sheath [59].

Diagnosis

For the diagnosis CIAV we must take into account some factors such as: that the signs and lesions are suggestive to CIAV, the age of presentation, the productive parameters, among others. Being a virus of global distribution is relatively easy to find [27]. As direct methods of diagnosis, viral isolation and detection of genetic material can be performed by PCR or DIF [60]. Serological techniques such as VN, ELISA and IIF can be used for diagnosis, where the most sensitive and effective is VN but too complex as a routine technique, the ELISA test is reliable and practical for bird monitoring [27].

Prevention and Control

The control of immunosuppressive diseases depends on biosecurity to avoid exposure to the causes of immunosuppressive diseases, and the increase of resistance to withstand the challenge of immunosuppressive agents through immunization and genetic selection [12,61]. Today, with the expansion of the chicken flock batches, the litter (bedding) is reused due to economic and environmental limitations, cleaning and disinfection must become seasonal events instead of occurring after each batch [59]. Strategies for the control of immunosuppression in commercial chickens (broilers and layers) are largely based on vaccination programs for broilers breeder's progeny, and management to minimize stress during breeding [12].

CIA Vaccines types, Aim and Applications

Attention should be paid to forestall immunosuppression by environmental factors, nutritional deficiencies and by other viral and bacterial infections. Proper immunization and genetic selection should be practiced to increase the resistance so that challenges of immunosuppressive agents can be bear up by the flock [12]. There are many types of vaccines agonist CIA including inactivated vaccines, subunit vaccine combined with some good adjuvants and DNA vaccine. The immunization of breeder flocks against CIAV has been reported to ensure more protective levels of passive immunity or the progeny chicks during the first few weeks of life [62]. Maternal anti-CIAV antibodies in chicks help to minimize vertical transmission, therefore regular vaccination and sero-monitoring (six weeks post vaccination) of breeder flocks during the rearing period is necessary to avoid vertical transmission [61]. The live attenuated virus vaccines are available to prevent CIA and it is recommended to vaccinate the breeder flock between 9 and 15 weeks of age [57]. Since, concurrent infections with immunosuppressive agents such as REV, MDV, or IBDV causes a delay in the development of age resistance and increased pathogenicity of CIAV, immunosuppressive disease control has been suggested to be integrated into the CIAV control program. Therefore, the introduction of the CIAV vaccination program requires consideration to the nature and immunopathogenesis of CIAV infection in relation to other agents [61].

Conclusion

CIA can be considered as a threat for poultry production due to it causes losses due mortality, low growth rate and immunosuppression. Prevention depends mainly on good hygiene and vaccination especially in breeder flocks to avoid vertical transmission. The control of immunosuppressive diseases depends on biosecurity to avoid exposure to the cause's immunosuppressive agents through immunization and genetic selection improvement.

Declarations

All data collected in this review are included in this published article.

Competing Interests

The author declares that they have no competing interests.

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