

Effect of Inclusion Body Hepatitis disease in Iraq Broiler Chickens

Melad Ibrahim Oraibi

¹Department of Pathology and Poultry Diseases
College of Veterinary Medicine **Baghdad**
Meladibrahim1975@gmail.com

Sahar Hamdi Abdalmaged

²Department of Pathology and Poultry
College of Veterinary Medicine University of Baghdad, Iraq
sahar.h@covm.uobaghdad.edu.iq

Abstract

The viral disease known as inclusion body hepatitis (IBH) spreads rapidly among chicken flocks. This disease had been described in Iraq, since 1970s. However, in the recent few years, the severity and mortality of the disease have been dramatically increased in our region. In this article, we sought to describe and detect the causative agent serotype depending on molecular bases. For further confirmation, histological methods have also been used for better understanding. For this purpose, young broiler (18 – 22 day old) with acute IBH infection collected from four distinct geographical regions have been subject to the study. Livers were taken under sterile conditions for histopathological study. Hematoxylin and eosin (H & S) stains were used to examine liver tissue that had been sectioned at 5 microns and embedded in paraffin blocks for microscopic examination.

Our findings suggest that the serotype of the IBH in Iraq is fowl adenoviruses (FAdVs). The samples showed severe reactions where acute hepatitis is prominent with hepatic necrosis and massive basophilic and eosinophilic intranuclear inclusion bodies. Other, non-specific histopathological changes have also been reported such as infiltration of inflammatory cells, hemorrhages and edema

Keywords: Inclusion body hepatitis, Chicken, FAdVs-8b

Introduction

The condition known as inclusion body hepatitis (IBH) was first described in the United States in 1963. (Helmboldt and Frazier). As word of the pandemic spread, it was documented in countries all over the world. The various serotypes of chicken-borne adenoviruses (Franco et al., 1974; Ferran, 2000; Fitzgerald, 2008; Gomis et al., 2006; Choi et al., 2012; Dar et al., 2012), it is a sporadic illness condition. The sudden death syndrome, which mortality rate of 10% in 3–4 days; normal usually restored by day 5 after onset of clinical symptoms. During the first two to three weeks of life, chickens of all ages were discovered to be sensitive, even immunologically intact chicks. IBH was found in broiler chicks as soon as 7 days old. Birds with the condition displayed despondency, had watery droppings, and some of them limped. Additionally, some of them had ruffled feathers and were weak on their legs (Grimes, 1978; Hess et al., 2000; Villate, 2001; Zadavec et al., 2011). The current study explains the IBH disease epidemics in the district of four regions in Iraq through studies based on clinical, post-mortem, and liver histological examinations of infected broiler chickens.

Methodology

Experimental Animal Samples

A total of 110 dead broiler chicken samples were collected from four poultry houses at four governorates of southern and central parts of Iraq. The samples were as following: Al-Diwaniyah governorate 23 sample, from the of Al-Amarah governorate 21 sample, from the Dhi Qar governorate 27 samples, and from Baghdad governorate 39 sample. The dead birds were aseptically dissected and the livers were collected in sterile nylon bags and kept under cooling conditions

Histopathological Procedure and Examinations .

Dead birds that were brought in for diagnosis having undergone thorough postmortem examinations. To avoid autolysis and subsequent morphological alterations, The collected liver tissue was immediately stored in buffered formalin. (10%). After being rinsed to get rid of the formalin and dehydrated in progressively stronger ethanol solutions, the tissues were cleared and embedded in xylene and paraffin, respectively. Hematoxylin and eosin was used to stain the thin (6 m) sections in the manner reported by Campbell and Luna in 1968. (1995). Utilizing a Carl Zeiss Axioskop 20 optical microscope, each slide was meticulously examined. At the histology lab of the Department of Veterinary Medicine (University of Baghdad), Baghdad , Iraq, histopathological observations were made.

Results

In the infected poultry houses, there was a significant rate of illness and mortality. The broiler chickens were mostly discovered dead, but on rare occasions, they were discovered in a gravely depressed state just before passing away. Within a few hours of the first symptom detection, death happened. A thorough clinical examination indicated that the patient's food and water consumption had both decreased. The clinical evaluation was expanded to include euthanized broilers and some necropsy examinations of dead animals. The liver was where the most consistent findings in this illness were seen. The livers in all epidemics seemed extremely similar upon visual inspection. The principal organ afflicted in these birds is the liver, which is enlarged, pale yellow in color, and covered in petechial hemorrhages (Fig 1). The liver's parenchyma is with a fragile consistency. (Fig 2).

The post-mortem lesions seen in this examination were comparable to those that were described by (Howell et al.,1970; Grimes et al.,1978; Fitzgerald, 2000; Hess, 2011).



Figure 1 : Liver of chicken (age 22 day old) infected with IBH. The liver shows enlargement with pale and yellow texture. The liver parenchyma shows many foci of petechial hemorrhages



Fig 2 : Liver of chicken (age 22 day old) infected with IBH. The liver shows deep yellowish color , round edges fatty degeneration and fragile consistency

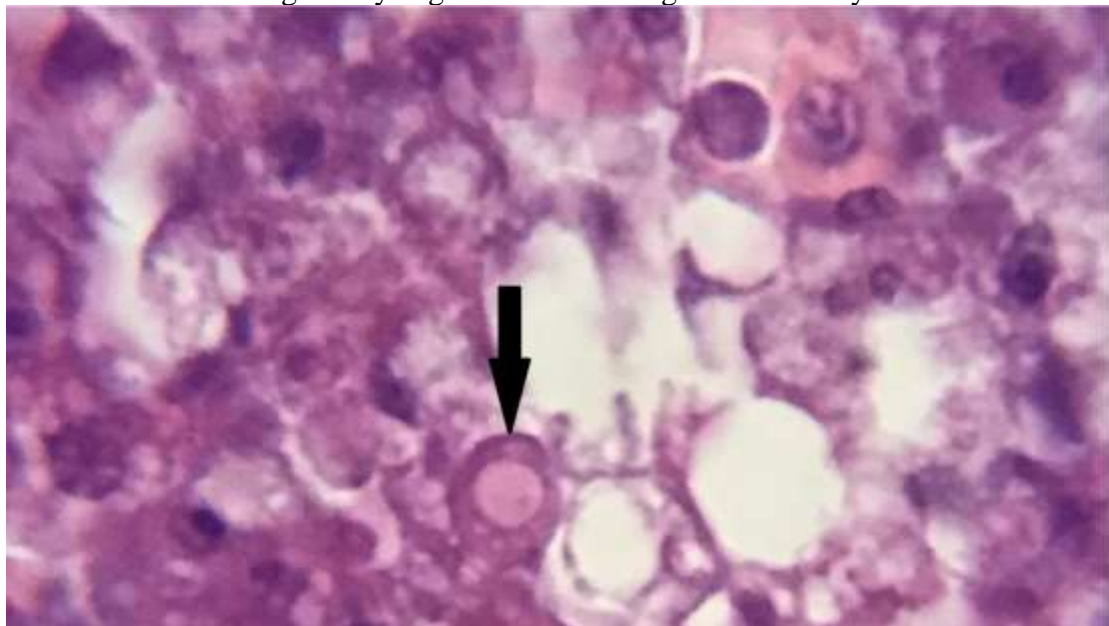


Fig 3 : Liver of chicken (age 22 day old) infected with IBH. The section shows eosinophilic inclusion bodies (arrow), which has a round or irregular shape and are surrounded by a halo. H & E, 40X.

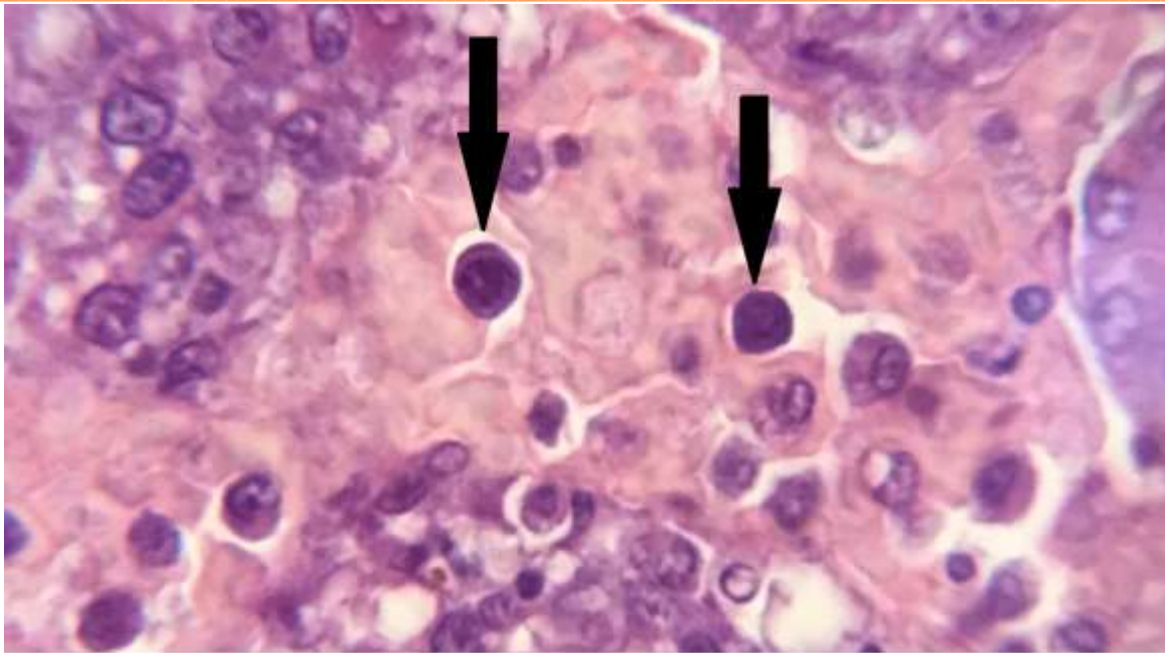


Fig 4 : Liver of chicken (age 25 day old) infected with IBH. The section shows multiple basophilic intranuclear inclusion bodies on the background of severe degenerative necrotic parenchymal lesions (Arrow). H&E, 40X

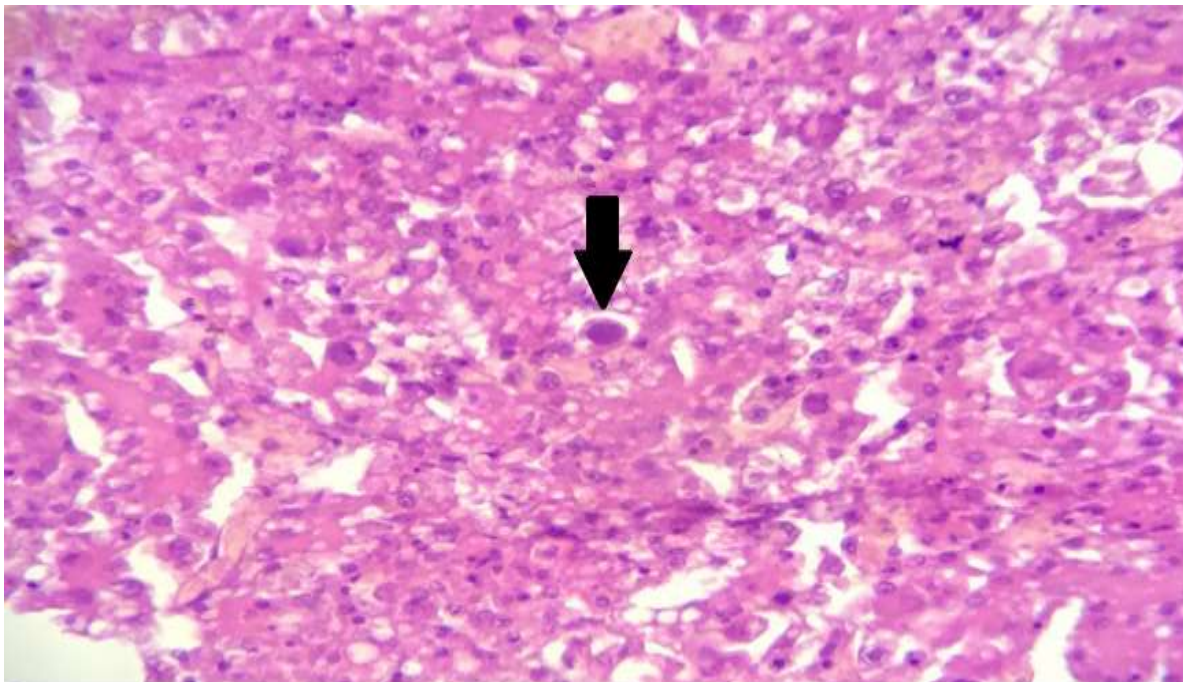


Fig 5 : Liver of chicken (age 22 day old) infected with IBH. The section demonstrates fatty degeneration, hepatocyte necrosis, and the presence of intensely basophilic intranuclear inclusion bodies. A light halo surrounds these inclusion bodies. (arrow). Inflammatory cell infiltration are consist together with lymphocytes, macrophages, and heterophils. H&E, 40X.

Discussion

In the majority of the liver cells are with eosinophilic inclusion bodies, which has a round or irregular shape and are surrounded by a halo and agree with Wolf, and other in 2018 liver parenchyma shows extensive necrosis and dystrophic changes at the microscopic level. Hepatocytes have been shown to have inclusion bodies that are either basophilic or eosinophilic in their cytoplasm. In contrast to the eosinophilic inclusion bodies, which are typically spherical or irregular in shape and surrounded by a faint

halo, basophilic inclusion bodies are typically dense and fill the entire inner space of the nucleus (Wolf et al., 2018). Pyknosis, karyorrhexis, and karyolysis were also seen in varying degrees (Fig 3). Numerous hepatocytes showed large intranuclear inclusion bodies with halo surrounding. A distinct area encircled them, and their shapes were erratic. There were a lot of inclusion bodies in various parts of the liver (Fig 4). In other hand Wolf, and other Both the typical gross lesions and the patient's medical history play a role in making the diagnosis. Histological examination is a primary method in IBH diagnostics because it aids in the detection of intranuclear inclusion bodies. Differentiating IBH from IBD and avian infectious anemia is of utmost importance (CIA).

As a preventative and control measure against infectious broiler hemorrhagic disease (IBH), it is recommended that infected broiler parent flocks' eggs not be used for hatching purposes. The virus could be carried and spread by wild birds, so they should be kept out (Wolf et al., 2018). Inflammatory cell infiltration are consist together with lymphocytes, macrophages, and heterophils. (Fig 5). However Hepatocytes show basophilic intranuclear inclusion bodies, vacuolar degeneration, and multifocal necrosis. are hallmarks of the severe hepatic damage caused by the hepatotropic fowl adenovirus (Yuan et al., 2021). For additional research into the infection-induced apoptosis of chicken liver cells (Liu et al., 2021).

Conclusions

In conclusions we found that the disease is endemic in Iraq. The disease show severe illness with high morbidity and mortality. As usual, the target for the virus is the liver, however, other organs might be effected as well. With current virus serotype, the disease is striking at early age with severe illness.

References

1. Campbell, T.W. (1995). Avian hematology and cytology. Iowa State University Press, Ames, 2 Eds. p:104.
2. Choi, K.S., Kye, S.J., Kim, J.Y., Jeon, W.J., Lee, E.K., Park, K.Y., and Sung, H.W. (2012). Epidemiological investigation of outbreaks of fowl adenovirus infection in commercial chickens in Korea. *Poult., Sci.* 91: 2502-2506.
3. Dar, A., Gomis, S., Shirley, I., Mutwiri, G., Brownlie, R., Poter, A., Gerdt, V. and Tikoo, S.K. (2012). Pathotypic and molecular characterization of a fowl adenovirus associated with inclusion body hepatitis in Saskatchewan chickens. *Avian Dis.*, 56: 73-81
4. Di Franco, E., Lussier, G., Berthiaume, L., Cloutier, S. and Marois, P. (1974). Hépatite à corps d'inclusion chez le poulet de grill isolément d'un agent viral. *Can. Vet. J.*, 15(5):144-147.
5. Fitzgerald, S.V., and Mc Connell, A. (2008). Group I adenovirus infections. In: Saif, Y.M. Diseases of Poultry. 12th Eds. Iowa, Wiley-Blackwell, pp: 252-266.
6. Gomis, S., A.R. Goodhope, A.D. Ojki and P. Wilson. 2006. Inclusion body hepatitis as a primary disease in broilers in Saskatchewan, Canada. *Avian Dis.* 50, 550-555
7. Grimes, T.M., Fletcher, O.J. and Munnell, J.F. (1978). Comparative study of experimental inclusion body hepatitis of chickens caused by two serotypes of avian adenovirus. *Vet. Pathol.*, 15: 249-263.
8. Helmboldt, C.F. and Frazier, M.N. (1963). Avian hepatic inclusion bodies of unknown significance. *Avian Dis.*, 7: 446-450.
9. Hess, M. (2000). Detection and differentiation of avian adenoviruses: a review. *Avian Pathol.*, 29: 195-206
10. Hess, M. (2001). Fowl adenovirus infections in chicken: current status and control approaches. In: Proceeding of the XVII World Veterinary Poultry Congress, Cancun, Mexico, pp: 41-48
11. Howell, J., Mac Donald, D.W. and Christian, R.G. (1970). Inclusion body hepatitis in chickens. *Can. Vet. J.*, 11(5): 99-101
12. Liu, J., Shi, X., Lv, L., Wang, K., Yang, Z., Li, Y., & Chen, H. (2021). Characterization of Co-infection With Fowl Adenovirus Serotype 4 and 8a. *Frontiers in Microbiology*, 12.

13. Luna, L.G. (1968). Manual of Histologic Staining Methods of the AFIP 3rd Eds., Mc Graw-Hill, NY, p: 131.
14. Mc Ferran, J.B. and Adair, B.M. (2003). Group I adenovirus infections. In: Saif Y.M. Diseases of Poultry. 12 th Eds. Iowa, Wiley-Blackwell, pp: 214-227.
15. Schonewille, E., Singh, A., Gobet, T.W., Gemer, W., Saalmuller, A. and Hess, M. (2008). Fowl adenovirus (FAdV) serotype 4 causes depletion of B and T cells in lymphoid organs in specific pathogen-free chickens following experimental infection. *Vet. Immunopathol.*, 121:130-139.
16. Villate, D. Maladie des volailles, . Agricole, Wolf, J. C., & Wheeler, J. R. (2018). A critical review of histopathological findings associated with endocrine and non-endocrine hepatic toxicity in fish models. *Aquatic Toxicology*, 197: 60-78.
17. Yuan, F., Hou, L., Wei, L., Quan, R., Wang, J., Liu, H., & Liu, J. (2021). Fowl adenovirus serotype 4 induces hepatic steatosis via activation of liver X receptor- α . *Journal of Virology*, 95(6): e01938-20.
18. Zadavec, M., Slavec, B. Krapez, U., Kajan, G.L., Racnik, J., Juntos, P.,
19. Jursic, R., Cizerl, M. Benko and Rojs, O.Z. (2011). Inclusion body hepatitis associated with fowl adenovirus type 8b in broiler flock in Slovenia. *Slov. Vet. Res.*, 48 (3/4): 107-113.