

Detection of an outbreak of Inclusion Body Hepatitis in commercial broiler chickens in North Western and Western Provinces in Sri Lanka

G. I. S. PERERA^{*1}, P. S. FERNANDO¹, W. M. P. BANDARA¹, G. M. C. R. KARUNARATHNE², W. M. S. K. WIJEKOON¹ and H. M. T. DULAKSHI¹

¹Veterinary Research Institute, Peradeniya, Sri Lanka

²District Veterinary Investigation Centre, Wariyapola, Kurunegala, Sri Lanka

Corresponding author: shalivet@yahoo.com

Introduction

Inclusion body hepatitis (IBH) in poultry is caused by fowl adenoviruses or aviadenoviruses, which were formerly known as group I adenoviruses. Although widespread among avian species, adenoviruses usually cause no disease or only a mild clinical illness. However, fowl adenoviruses are responsible in causing two important conditions in poultry, namely IBH and hydropericardium syndrome, which can occur together or in isolation as two disease entities. Less commonly, these viruses are also accountable for causing necrotizing pancreatitis and/or gizzard erosions in chickens.

Out of the 12 different serotypes of fowl adenoviruses, the most common serotypes causing IBH are 4 and 8. However, involvement of serotypes 2, 5, 6, 7, 10 and 12 was also detected. The disease mainly affects meat-producing birds or broilers, but has also been reported in layer type of birds. Young broiler chicken are the most severely affected group of the disease, sometimes resulting in high mortality rates, even reaching up to 30-40%, which may depend on the pathogenicity of the virus and concurrent infections. Clinical signs of the affected birds are non-specific which include; depression, lethargy, huddling, inappetance, ruffled feathers, pallor in comb and wattle etc. The gross lesions are; enlarged pale friable liver (sometimes with necrotic foci), haemorrhages in liver, myocardium, thigh and breast muscles and anaemia. Sometimes IBH occurs in association with hydropericardium. Pathognomonic histopathological features of IBH involve detection of intra-nuclear basophilic inclusion bodies in hepatocytes in association with necrotic changes in liver. However, it is also possible to find intra-nuclear eosinophilic inclusion bodies with halo in some cases of IBH.

In Sri Lanka, although the disease was not reported previously, IBH suspected clinical cases of broilers have been encountered in the recent past. From July 2019 to March 2020, clinical outbreaks with a sudden onset of mortality ranging from 1% to 18% have been reported in 13 commercial broiler farms and one layer farm in Kurunegala, Puttalam and Gampaha districts. The age of the affected birds ranged from days 7 to 35. Major clinical signs observed were inappetance and depression, along with pallor in comb and wattle in some instances. The common post-mortem lesions encountered were; pallor of the carcass, enlarged liver with patchy yellow discolouration and crumbly texture (necrosis) sometimes associated with haemorrhage. Multifocal haemorrhages

in kidney and in some instances, in thigh and breast (pectoral) muscles were also noted. In the birds of one farm, hydropericardium was detected. Accordingly, clinical signs and gross pathological lesions observed at the field level were suggestive of IBH. Therefore, this study was conducted for diagnosis of IBH by histopathology.

Methodology

Tissue samples (mainly from liver, heart, kidney, lung, spleen and sometimes from, pancreas, proventriculus, gizzard, intestine and bursa of fabricius) from approximately 5-10 birds from each farm were fixed in 10% formalin. These were processed by paraffin-embedding technique for histopathology at the Veterinary Research Institute. After cutting the tissues within wax blocks into 3µm thick sections, they were mounted on to slides and stained with Haematoxylin and Eosin (H&E), with subsequent histological section evaluation under the microscope. Fresh tissue samples were further subjected to bacteriological testing, when concurrent disease conditions are suspected in the chickens.

Results and Discussion

Histopathological examination of liver sections revealed varying degrees of multifocal necrotizing hepatitis associated with infiltration of lymphocytes, macrophages and some heterophils in chickens submitted from all the 14 farms (n=112/123). In birds of 12 farms, intra-nuclear inclusion bodies were detected (53/123) in the liver; mostly basophilic inclusion bodies and occasionally eosinophilic intra-nuclear inclusions surrounded by a halo. Inclusion bodies were detected within and around the necrotic foci. In addition, multifocal haemorrhages of varying intensity could be observed in the liver and the kidney. Hence the condition was diagnosed as IBH, mainly based on histopathological means along with the clinical and necropsy findings.

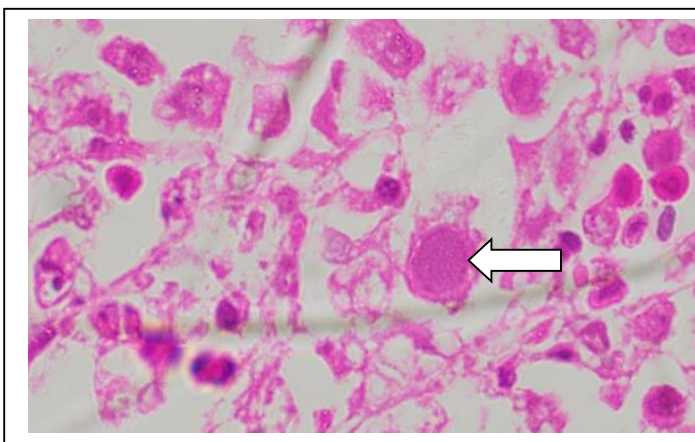


Figure 1. Large basophilic intra-nuclear inclusion body in a hepatocyte (arrow) and associated inflammatory reaction. H&E, x1000

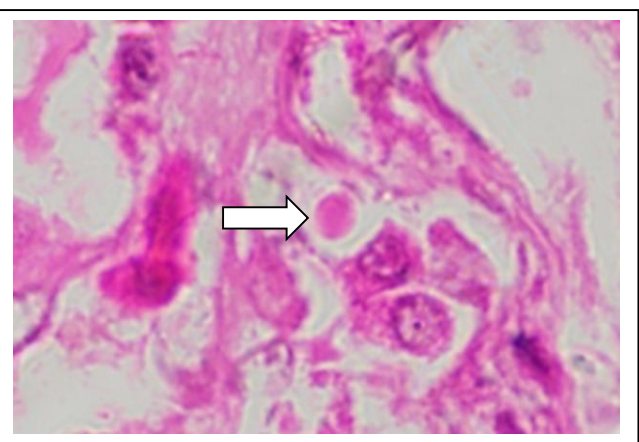


Figure 2. Intra-nuclear eosinophilic inclusion body with a halo in a hepatocyte (arrow). H&E, x1000

Histopathological findings detected were consistent with previous studies conducted on detection of IBH through histopathology (Mariappan *et al.*, 2018; Nakamura *et al.*, 2011). According to the previous workers, liver has been identified as the primary organ affected during IBH infection and detection of intra-nuclear basophilic inclusion bodies in hepatocytes was considered as pathognomonic, which we have observed in the current study (Mariappan *et al.*, 2018). However, we have not witnessed prominent histopathological lesions in pancreas or gizzard, although these have been noticed in few of the former studies (Mariappan *et al.*, 2018; Ono *et al.*, 2003). Yet, it was described that the occurrence of lesions in gizzard and pancreas is rare compared to the liver lesions. Moreover, the number of tissue samples that we have gained for histopathological testing from gizzard and pancreas were less in compared to the liver, from which, the samples were tested in each and every bird included in the study.

Apart from IBH, the chicken of some of the affected farms showed concurrent infections of colibacillosis, necrotic enteritis and respiratory diseases. Since there is no specific treatment for IBH, the affected birds in majority of the farms were treated for these other conditions. In some of the farms where only IBH signs prevailed, antibiotic treatment was practiced to prevent occurrence of secondary bacterial infections. In most of the farms vitamin supplements were provided. With the treatment, birds that were not severely affected with a concurrent infection recovered, approximately within 2 weeks of time.

Poultry getting infected with other disease conditions could be anticipated with IBH infection, since fowl adenoviruses can affect the immune competency of infected chickens. On the other hand, immunosuppressive diseases such as infectious bursal disease and chicken anaemia can also predispose birds to secondary IBH infection (Nakamura *et al.*, 2011). However, recent studies have identified fowl adenoviruses as primary pathogens in chicken causing significant disease without prior immunosuppression or predisposing factors (Mariappan *et al.*, 2018). Therefore currently avian adenoviruses and IBH are gaining more global attention with targeting of prevention of occurrence.

Conclusion

To our knowledge, this is the first reported outbreak of IBH in Sri Lanka. Therefore, further testing on the disease, coupling histopathology with additional diagnostic tools is recommended, and identification of the serotypes involved in the Sri Lankan context is warranted. This will facilitate recognition of the true magnitude of the condition, according to which the future preventive measures of the disease should be opted to reduce the economic losses to the poultry industry in Sri Lanka.

References

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