International Journal of Chemical Studies

P-ISSN: 2349–8528
E-ISSN: 2321–4902
IJCS 2017; 5(3): 458-461

Received: 06-03-2017
Accepted: 07-04-2017

Biswajit Dutta
Assistant Professor, Department of Veterinary Pathology, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati, Assam, India.

Pankaj Deka
Assistant Virologist, Department of Microbiology, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati, Assam, India.

SM Gogoi
Assistant Professor, Department of Microbiology, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati, Assam, India.

M Sarma
Jr. Scientist, LRS Mandira, Assam Agricultural University, Khanapara, Guwahati, Assam, India.

MK Bora
Ph. D. Scholar, Department of Poultry Science, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati, Assam, India.

DC Pathak
Professor, Department of Veterinary Pathology, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati, Assam, India.

Correspondence
Biswajit Dutta
Assistant Professor, Department of Veterinary Pathology, College of Veterinary Science, Assam Agricultural University, Khanapara, Guwahati, Assam, India

Pathology of inclusion body hepatitis Hydropericardium syndrome (IBH-HPS) in broiler chicken

Biswajit Dutta, Pankaj Deka, SM Gogoi, M Sarmah, MK Bora and DC Pathak

Abstract
The present work was carried out to diagnose the inclusion body hepatitis- hydropericardium syndrome (IBH-HPS) in broiler chicks using conventional technique. Materials were collected from a private broiler farm during outbreak at the age of 25 days. The clinical signs were recorded. Grossly, liver was pale, friable, enlarged with the presence of focal or diffuse areas of necrosis. The heart showed accumulation of straw coloured fluid in the pericardial sac. Microscopically, the hepatic parenchyma showed multi focal areas of coagulative necrosis with the presence of basophilic intranuclear inclusion bodies. Lymphoid organs like bursa of Fabricius, spleen showed depletion of lymphocytes from the lymphoid follicle.

Keywords: Broiler, histopathology, hydropericardium, inclusion body hepatitis

1. Introduction
Poultry industry is one of the fastest growing sectors in India. Despite being well established and well organized, this industry is still confronted with many acute and fatal diseases. In recent years inclusion body hepatitis- hydropericardium syndrome (IBH-HPS) has become one of more common impediments causing heavy economic loss, reduced weight gain and mortality. IBH- HPS that caused by aviadenovirus was first described in 1963 in the USA [10]. Since then the disease has been reported from many countries. In late eighties, a new disease in broilers with some clinical similarities to classical inclusion body hepatitis (IBH) was reported from Angara Goth near Karachi, Pakistan. The disease was mainly characterized by accumulation of a clear straw coloured fluid in the pericardial sac along with hepatitis [11] and named as Hydropericardium syndrome (HPS)/ Angara disease. In India, HPS was first noticed in the poultry belt of Jammu & Kashmir, Punjab and Delhi and named as "leechi disease" due to characteristic hydropericardium, giving the appearance of peeled Indian leechi fruit [8]. Then several outbreaks were recorded in Uttar Pradesh, Maharastra, Andhra Pradesh, Karnataka, Tamil Nadu, Kerala, Odisha and West Bengal resulting in huge economic losses to the poultry industry [4]. All the 12 serotypes of group I FAdVs have been incriminated in the field outbreaks of IBH-HPS, however FAdV serotype 4 (FAdV-4) had been mostly implicated [13]. As far as literature is concerned, research works on IBH-HPS in Assam is meagre although the disease has created severe havoc in the broiler population in last few years (Field survey data). Therefore, the present investigation was undertaken to diagnose the disease in broiler flocks during natural outbreaks.

2. Material and Methods
2.1 Study area: The materials for present study were collected from a private broiler farm located at Sonapur area (26.1172° N, 91.9802° E) of Kamrup (M) District, Assam. Age of the broiler flock was 25 days, when all of a sudden mortality started. The birds were vaccinated against RD LaSota strain on 5th day, Gumboro on 14th day and RD R_2B strain on 18th day. The clinical signs exhibited by the affected birds were recorded. The dead birds were subjected to detailed post-mortem examination and gross lesions were systematically recorded. The materials such as liver, heart, kidney, spleen, thymus, bursa of Fabricius, intestine etc. were collected from the necropsied birds at 10% formol saline solution for histopathological examination.

2.2 Histopathology: Formalin fixed tissues were processed, and sectioned at 4µ thickness and stained with Haematoxylin & Eosin (H&E) stain for histopathological examination [6]. Diagnosis was based on characteristics post-mortem findings and histopathological examination.

3. Results and Discussion

Out of the total 1010 birds 127 nos. were died due to IBH-HPS. The overall mortality was recorded as 12.57 percent which was slightly lower than earlier report [7]. But Ahamad [2] recorded a higher morbidity (32.73%) and mortality (94.44%) rate in Giriraja chicken in IBH/HPS.

3.1 Clinical signs: The affected birds showed dullness, depression, ruffled feathers and death. The feed conversion ration and body weight gain were reduced as per the farm record. There was sudden high mortality reaching its peak within 3-4 days followed by constant death rate for 4-5 days. Similar clinical signs were also described earlier [7, 12]

3.2 Gross Pathology: The post mortem examination of birds revealed gross lesions primarily in the liver, heart and kidneys in almost all the affected birds.

3.2.1 Liver: In most of the cases, liver was pale, friable, enlarged with the presence of focal or diffuse areas of necrosis (Fig. 1 & 2). Sometimes the hepatic parenchyma showed the presence of pin point or ecchymotic haemorrhages. Occasional fatty changes were also noticed in some cases. In few isolated cases, liver was icteric with or without presence of haemorrhages. Degenerative and necrotic changes in liver are responsible for dysfunction of liver and might be responsible for decrease in osmotic pressure of blood, leading to accumulation of straw coloured fluid in the pericardial sac [18]

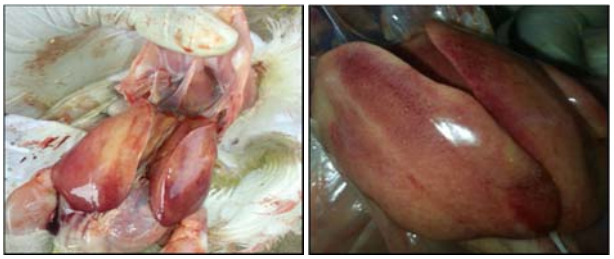

Fig 1&2: Liver showing focal or diffuse areas of necrosis (N) and haemorrhages (H)

3.2.2 Heart: The pericardial sac showed the accumulation of straw coloured fluid (Fig.3) with misshapen and flabby heart. In some cases the epicardium showed the presence of pin point haemorrhages. These findings support the observations of earlier reports [11, 12, 14].

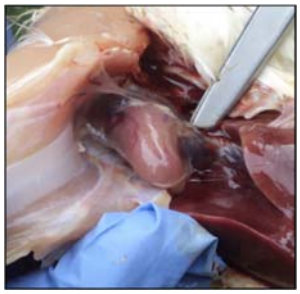

Fig 3: Heart showing accumulation of straw coloured fluid

3.2.3 Kidney: The kidneys were enlarged (Fig.4) and congested with the presence of some necrotic foci. Sometimes there was accumulation of crystals of urates in the ureter. Similar observations have been reported by several workers [2, 5]

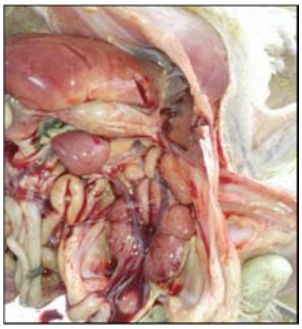

Fig.4 Kidney showing enlargement with some necrotic foci (N).

3.2.3 Lung: Lungs were congested & mildly edematous.

3.3. Histopathology:

3.3.1 Liver: The hepatic parenchyma showed congestion (Fig.5), haemorrhages (Fig.6), centrilobular or diffuse degeneration of the hepatocytes, fatty changes with dilation of sinusoidal space. Apart from these, there is presence of small multi focal areas of coagulative necrosis (Fig.7) with the presence of lymphocytes, plasma cell, macrophages and heterophils. Focal accumulation of mononuclear cells were also prominent in some cases (Fig.8). Basophilic intranuclear inclusion bodies were present in the hepatocytes and surrounded by a clear halo or sometimes filling the entire nucleus (Fig.9 & 10). Fatty infiltration with basophilic intranuclear inclusion bodies in the hepatocytes, coagulative necrosis of the hepatocytes and subacute periportal hepatitis had given typical had given typical evidence of adenoviral infection [1, 5].

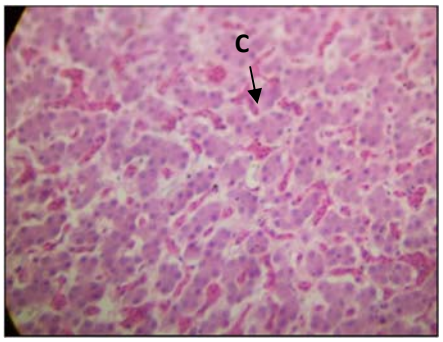

Fig 5: Liver section showing congestion (C), H&E x400;

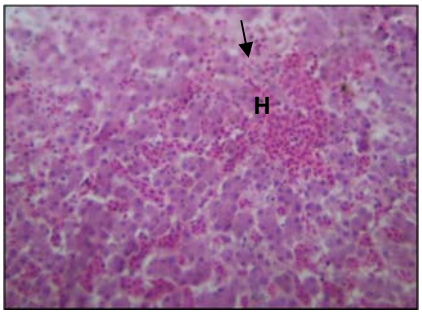

Fig 6: Liver section showing haemorrhages (H), H&E x400;

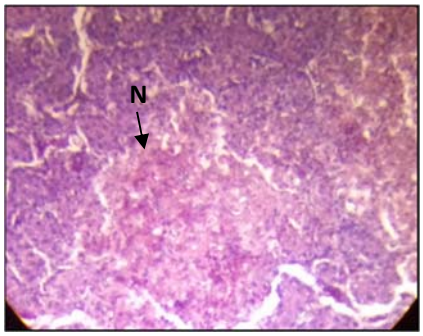

Fig 7: Liver section showing focal areas of coagulative necrosis (N), H&E x100

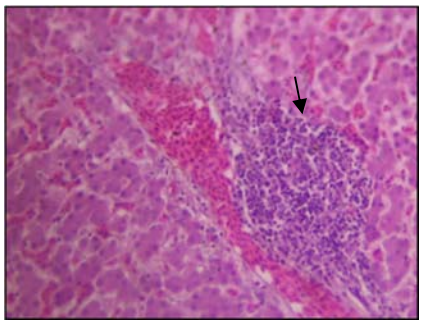

Fig 8: Liver section showing focal accumulation of mononuclear cells, (arrow) H&E x400

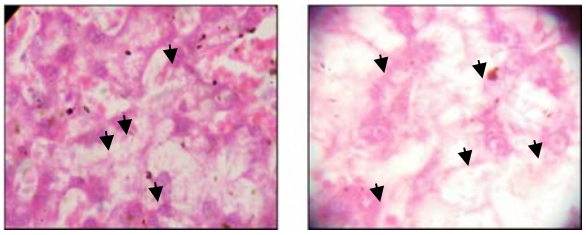

Fig 9 & 10: Liver section showing basophilic intranuclear inclusion bodies (arrow).

3.3.2 Heart: In the epicardium, the blood vessels were congested and haemorrhagic. Degeneration of the cardiac muscle fibre with focal accumulation of mononuclear cells (Fig.11) was prominent. Similar cardiac lesions were described earlier [2]. The heart being the very active organ cannot sustain the reduced osmotic pressure so long and hence the vascular endothelium is damaged.

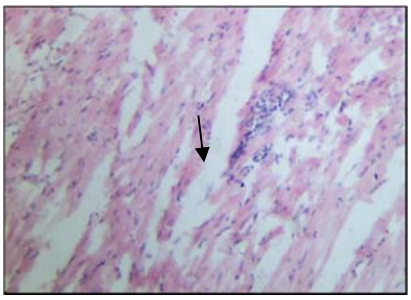

Fig 11: Heart section showing focal accumulation of mononuclear cells, H&E x100

3.3.3 Kidney: The renal parenchyma showed tubular degeneration characterized by swelling of the tubular epithelium and pyknosis of the nucleus. Interstitial haemorrhages were also observed. The degenerative changes in the renal tubular epithelium reduces the renal perfusion leading to deposition of crystals of urate in the kidney [3].

3.3.4 Lung: Lungs showed congestion, haemorrhages, edema and infiltration of the mononuclear cells (Fig.12). The damage to the pulmonary vessels and edema might be result due to persistent intravascular pulmonary pressure [2, 17].

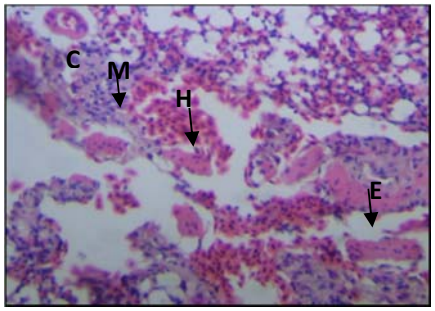

Fig 12: Lung section showing congestion (C), haemorrhages (H), edema (E) and infiltration of the mononuclear cells (M), H&E x400;

3.3.5 Bursa of Fabricius: Mild to moderate depletion of lymphocytes (Fig.13) from the lymphoidal follicles was observed. Depletion of the lymphocytes from lymphoidal organs were described by earlier worker [5], which indicate immunosuppression [16].

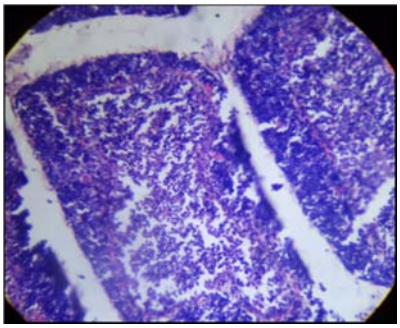

Fig 13: Bursa of Fabricius showing depletion of lymphocytes, H&E x100

3.3.6 Spleen: Vascular congestion and focal areas of haemorrhages (Fig.14) with depletion of the lymphocytes [5]. In IBH/HPS, avian adenovirus caused immnosuppression as the virus was found to have predilection for lymphoid organs was capable of producing disease in healthy chicken [15]

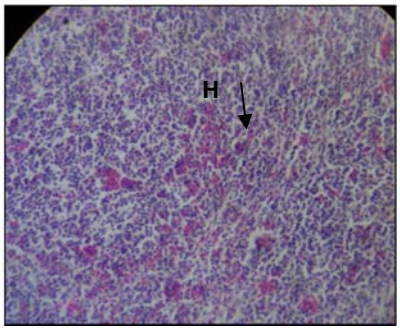

Fig 14: Spleen section showing haemorrhages (H), H&E x100.

4. Conclusion: In conclusion, the present study recorded an outbreak of IBH/HPS, in the agroclimatic condition of Assam

based on the pathomorphological study. The immunosuppression and mortality causes severe economic losses to the poultry industry. It is therefore advisable an emerging disease.

5. Acknowledgement: Authors are thankful to the Head, Department of Pathology, College of Veterinary Science, AAU, Khanapara, Assam, India for proving necessary facilities to carry out this work.

6. References

1. Abdul Aziz TA, Hasan SY. Hydropericardium syndrome in broiler chickens: its contagious nature and pathology. Res Vet Sci. 1995; 59:219-221.
2. Ahamad DB, Selvaraj J, Sasikala M, Prasath NB. Pathological study of an outbreak of hydropericardium syndrome in Giriraja chicken. Indian J. Vet. Pathol. 2017; 41(1):53-56.
3. Asrani RK, Gupta BK, Sharma SK, Singh SP, Katoch RC. Hydropericardium- hepatopathy syndrome in Asian poultry, Vet Rec. 1997; 141:271-273.
4. Asthana M, Chandra R, Kumar R. Hydropericardium syndrome: Current status and future developments. Arch. Virol. 2013; 158(5):921-931.
5. Cheema AH, Ahmad J, Afzal M. An adenovirus infection of poultry in Pakistan. Rev Sci Tech offInt Des Epizoot. 1989; 8:789-795.
6. Culling CFA. Handbook of histological & histochemical technique. 3rd edn. Butterworth and Co. 1974.
7. Das T, Panda SK, Panda HK, Acharya AP, Das N. Pathology of inclusion body hepatitis and hydropericardium syndrome in broiler chicken in natural outbreaks in Odisha. Indian J. Vet. Pathol. 2015; 39(1):46-49.
8. Gowda RNS, Satyanarayana ML. Hydropericardium syndrome in poultry. Indian J. Vet. Pathol. 1994; 18:159-161.
9. Goyal D, Singh A, Sood N, Gupta K, Sood NK. Pathological changes in naturally occurring inclusion body hepatitis and hydropericardium syndrome in poultry. Indian J. of Vet. Pathol. 2009; 33:105-106.
10. Helmboldt CF, Frazier MN. Avian Hepatic inclusion bodies of unknown significance. Avian Disease. 1963; **7**:446-450.
11. Jaffery MS. A treatise on Angara disease (hydropericardium pulmonary oedema – hepatonephritis syndrome). Pak. Vet. Med. Assoc.1988*;* 1-33.
12. Kumar R, Chandra R, Kumar V, Bhatt P, Shukla SK, Dhama K, *et al.* Hydropericardium Syndrome (HPS) Virus: Immunofluorescence Studies on Aspects of Pathogenesis in Chickens. Advances in Animal and Veterinary Sc. 2013; 1(3):25-29.
13. Mazaheri A, Prusas C, Vob M, Hess M. Some strains of serotype 4 fowl adenoviruses cause inclusion body hepatitis and hydropericardium syndrome in chickens. Avian Pathology. 1998; 27:269-276.
14. Meenakshi, Bal MS, Kumar H, Sandhu KS. Pathology of hydropericardium syndrome in an outbreak in broilers. Indian J. Vet. Pathol. 2005; 29(1):46-47.
15. Naeem K, Niazi T, Malik Sa, Cheema AH. Immunosuppressive potential and pathogenicity of an Avian Adeno virus isolate involved in Hydrop aricardium syndrome in broilers. Avian Dis. 1995; 39:723-728.
16. Roy P, Murulimonohar B, Koteeswaran A, Omprakash AV. Experimental studies on hydropericardium syndrome in two different synthetic lines of broiler chickens. Veterinarski Arhiv. 2004; 74:157-164.
17. Sharma S, Asrani RK, Singh G, Gulathi BR, Patil PK, Gupta VK *et al.* Outbreak of hydropericardium syndrome associated with ascites and liver rupture in caged broilers. Vet Res Int. 2014; 2:33-45.