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## ISOLATION AND IDENTIFICATION OF HYDROPERICARDIUM SYNDROME VIRUS FROM OUTBREAKS IN TAMILNADU

S. Vairamuthu<sup>1</sup>, A. Raja<sup>2</sup>, G.A. Balasubramaniam<sup>3</sup>  
and B. Murali Manohar<sup>4</sup>

Department of Pathology, Madras Veterinary College, Chennai - 600 007

Hydropericardium syndrome (HPS) is a very recent and mysterious disease affecting chicken. This disease was first reported in growing chicks in Pakistan (Khawaraja *et al.*, 1988). It was mainly seen in commercial broiler chicks aged 3-5 weeks and was characterised by hydropericardium filled with straw coloured fluid in the pericardial sac. No clinical signs except sudden death upto 60-70% (Ahamed *et al.*, 1989) could be observed.

In India, the disease was first noticed in the poultry belt of Jammu in April 1994. Later it spread to the rest of the country. In Tamil Nadu, the disease was first recorded in Palladam which is considered as broiler belt of Tamil Nadu. (Karunamoorthy *et al.*,

1996). In India, the syndrome was called "leechi disease" because the heart with hydropericardium resembled shelled leechi fruit. Adenovirus was isolated from the liver of chickens affected with HPS. The serotype of the adenovirus isolated from the chickens with HPS was type 4 (Naeem *et al.*, 1995).

### Materials and Methods

Hydropericardium syndrome was recorded in a commercial broiler farm at Namakkal and five farms at Palladam in Coimbatore District. The farm particulars are furnished below (Table 1).

Complete necropsy was performed on

Table 1

Farm No.	Location	Total Stock	Total mortality	Percentage of mortality	Age at out break
1.	Namakkal	3000	2500	83%	15 <sup>th</sup> day
2.	Palladam, Coimbatore	17000	7700	45%	26 <sup>th</sup> day
3.	-do-	2800	2000	70%	20 <sup>th</sup> day
4.	-do-	7000	2800	40%	28 <sup>th</sup> day
5.	-do-	8000	3700	47%	25 <sup>th</sup> day
6.	-do-	5000	2200	44%	28 <sup>th</sup> day

1. Assistant Professor, Department of Pathology, MVC., Chennai
2. Assistant Professor, CRL., VC and RI., Namakkal
3. Associate Professor, Department of Pathology, VC and RI Namakkal
4. Professor and Head, Department of Pathology, MVC, Chennai



### Identification of Hydropericardium Syndrome virus

all the birds and tissue pieces such as liver, heart, lungs, spleen, kidneys, bursa of Fabricius were collected for histopathological studies.

Blood samples were collected from ailing birds selected at random in each flock for haematological studies.

Spleen, lungs, brain, proventriculus and intestinal contents were collected to check for the presence of Ranikhet disease virus. Filter paper samples were collected from the ailing birds to assess the Haemagglutination Inhibition (HI) antibody titre against Ranikhet disease.

Liver samples were collected aseptically for isolation of bacteria.

Liver homogenate was prepared using Phosphate Buffered Saline (PBS) and Counter Immuno Electrophoresis was performed following standard operating procedure to identify the HPS virus using HPS positive serum obtained from the Division of Avian Pathology, IVRI, Izatnagar, UP.

#### Results and Discussion

The syndrome was found to be restricted only to the broiler chicks aged from 15<sup>th</sup> day to 28<sup>th</sup> day. The total mortality

percentage varied from 40 to 83 per cent. The onset of mortality was abrupt without any clinical manifestation. The instance of mortality was found to be frequent among well nourished birds.

The haematological studies in the ailing birds indicated marked reduction of haemoglobin, packed cell volume and total RBC count (Table 2). Marked reduction of erythrocytes and/or deposition of blood pigments in the spleen, lungs and liver suggest that acute systemic reduction of erythrocytes might be due to sequestration and lysis of RBC in different organs. Haematological results of this in HPS birds corroborated the findings of Niazi *et al.*, (1989).

The post-mortem examination in the affected birds revealed uniform manifestation of gross lesions. The carcass appeared pale, soft, moist and well nourished. The predominant lesion was seen with heart showing typical hydropericardium filled with 5 to 10 ml of straw coloured fluid. The pH of the fluid was 7.2 and the fluid clotted on exposure to air. The heart after removal of pericardial sac appeared reddish, glistening with white patches and occasionally with haemorrhagic spots. The lungs were congested and oedematous. The livers were markedly enlarged, yellow, mottled and haemorrhagic. The spleen appeared

Table - 2

	Control birds	Affected birds
Haemoglobin g/dl	10.50	6.70
Packed Cell Volume per cent	31.20	24.20
RBC millions/c m.m.	2.76	1.29



congested and mottled. Kidneys appeared pale, tumified with prominent tubules. The mesentery vessels were engorged and intestine showed mucoid enteritis. The abdominal fat was cloudy grey and in some cases appeared yellowish. There was no ascities in these cases.

Histopathological studies revealed hepatocellular degeneration, bile duct hyperplasia and intranuclear inclusions in a few hepatocytes. Focal congestion with degeneration of myocardial fibres was noticed. Heterophilic infiltration in secondary bronchi with fibrinous exudate was seen in lung. Kidneys revealed congestion and degenerative changes in tubular epithelial cells. Lymphoid depletion and glandular transformation were noticed in the bursal follicles and fusion of villi with increased goblet cell activity was also noticed in intestine.

In the present study, the results of histopathological studies were in accordance with Ahmed *et al.*, (1989) and Sreenivasa Gowda, (1944) Hydropericardium, a pathognomonic lesion of HPS, has never been noticed in any report of IBH field cases and experimental cases (Gallina *et al.*, 1973). Compared with IBH, the high mortality rate is characteristic of HPS. Hydropericardium is seen predominantly in dead birds. Cardiac lesions might therefore be associated with death (Toshiaki *et al.*, 1998). Ahmed *et al.*, (1989) reported that the chicken inoculated with liver homogenate from HPS chickens died and showed hydropericardium. Hydropericardium might have been caused indirectly by HPS adenovirus infection.

In ascites syndrome in broiler chickens, hydropericardium as well as

ascites are observed (Julian, 1993). In this condition heart failure induces liver damage and ascites. Dilation of right ventricles was seen commonly in ascites syndrome, which was not noticed in the present study. Therefore pathogenesis of hydropericardium in HPS may be different from that of ascites.

No bacteria were isolated. The HI antibody titre against RD was less than 16. In HA test, there was absence of RD virus. Bursa of Fabricius was atrophied. HA and HI titre of RD revealed that there was no involvement of the disease.

In CIE, the suspected materials produced classical precipitation lines with the known HPS positive serum raised against adenovirus type IV. In the present study, the results of CIE indicate that the HPS recorded was due to adenovirus serotype 4, the same serotype as that of the Pakistan strain of HPS (Qureshi, 1997).

Sudden mortality, post-mortem findings, histopathology, immunodiagnostic techniques, suggested that the chickens with HPS were infected by group I adenovirus serotype 4. No agent such as Ranikhet disease virus and Infectious bursal disease virus could be demonstrated. Hence it is possible that the present group I adenovirus strain (serotype 4) may be very virulent and its pathogenicity needs to be studied experimentally.

### Summary

Outbreaks of HPS among broiler flocks at Namakkal and Palladam areas were recorded. Affected birds were in the age group of 15<sup>th</sup> day to 28<sup>th</sup> day. The consistent postmortem lesion was hydropericardium and intranuclear inclusions in the hepatocytes.

8. Discussion

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### Identification of Hydropericardium Syndrome virus

No NDV on IBDV involvement could be observed. Immuno diagnostic techniques revealed the involvement of group I adenovirus (serotype 4) in the present study.

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#### NEWS

##### MADRAS VETERINARY COLLEGE CENTENARY CELEBRATION

Madras Veterinary College, an institution of national and International reputation, is to celebrate its centenary during 2003. To commemorate this unique event Professional Societies and Associations are invited to hold their annual conventions, symposium and seminar for 2003 at Madras Veterinary College, Chennai - 600 007.

Please contact for further details:

Dr. K.S. Palaniswami  
 Chairman, Scientific Services Committee Centenary Celebration  
 and Director of Research 1/C  
 Tamil Nadu Veterinary and Animal Sciences University,  
 Madhavaram Milk Colony,  
 Chennai - 600 051. e-mail : drtanuvas@rediffmail.com