BLOOD ANALYSIS OF BROILER CHICKS AFFECTED WITH HYDROPERICARDIUM SYNDROME (HPS) IN KARACHI

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ABSTRACT

The present study was carried out at the Poultry Physiology Unit, Department of Physiology, University of Karachi and samples collected from the poultry farms located in the suburbs of Karachi. Hydropericardium syndrome (HPS) an infection of Avian Adenovirus Type-4 was reported in Karachi (Pakistan) during 1987 but is now widespread in other poultry regions. Disease has caused heavy economic losses to the poultry industry in Pakistan. Moreover, the isolation of the causative agent has been well documented but the evaluation of blood for biochemistry and hematology in HPS affected broiler chicks (age 3 to 5 weeks) is still not known. Present study is designed in the light of analyzing the blood for formed elements and ions. Agar gel immuno diffusion test has also been performed to confirm the presence of HPS infection in the birds. Since little is known about the physiological status of the bird during the course of disease and the accumulation of jelly like fluid in the pericardial cavity strongly suggests an abnormal osmotic environment during infection. The study is highly conclusive in revealing a significant difference (P<0.05) among the control and the tested birds for the clinical chemistry and blood parameters evaluated which may be attributed for the accumulation of jelly like fluid in the pericardial cavity.

Key words: HPS, RBC, WBC, Hb, Na+, K+, Ca++, broilers

INTRODUCTION

Hydropericardium syndrome (HPS) known to be a disease of young chickens (Abe et al., 1998) was first observed in 1987 at “Angara Goth” in Karachi and has now been affecting broilers throughout Pakistan (Jaffery, 1988; Hasan, 1989; Hussain et al., 1999). This disease has caused large economic losses to the broiler industry in Pakistan (Afzal and Ahmed, 1990; Hussain et al., 1996; Iftikhar et al., 1999). The syndrome has also been reported in neighboring countries of Pakistan (Abdul Aziz and Al Attar 1991; Survashe et al 1996).

The disease was believed to be a nutritional disorder (Jaffery, 1988). Experiments and successful vaccination showed that the disease is infectious (Cheema et al., 1989; Chishti et al., 1989; Iftikhar et al., 1999) and was reported to be a viral infection in 1994 characterized as Avian Adeno virus Type 4 (Afzal et al., 1991; Akhtar, 1994; Haq et al., 1997; Mazaheri et al., 1998; Iftikhar et al., 1999; Kikuyasu et al., 1999; Kikuyasu et al., 2000; Toro et al., 2000). The mean incubation period of the disease in controlled experimental studies is 10 (range 5-18) days before the onset of HPS related sudden mortality (Akhtar, 1994; Toro et al., 2000) as high as 20 to 90 percent with completing its course in 7 to 18 days (Anjum et al., 1988). The characteristic lesions (Cheema et al., 1989; Anjum et al., 1989; Ahmed et al., 1989; Survashe et al., 1996; Morales et al., 1997; Iftikhar et al., 1999; Kikuyasu et al., 2000) of the disease included swollen pericardial sac filled with straw-colored fluid (up to 15 ml from one sac) and friable liver (Shamim and Rehmani, 1990) showing the presence of small multifocal areas of coagulative necrosis and mononuclear cell infiltration, severely affected kidneys, edematous lungs with infiltration by mononuclear cells and heterophils in the alveolar walls and enlarged spleen showing macrophagic hyperplasia but with no morbidity particularly. The heart was found misshapen and flabby (Anjum et al., 1989; Mazaheri et al., 1998). Histological examination of the hepatocytes reveals the presence of basophilic intranuclear bodies (Ahmed et al., 1989; Cheema et al., 1989; Anjum et al., 1989; Kikuyasu et al., 2000). The hemopoietic system was also found to be infected (Toro et al., 2000). The renal tubules showed degenerative changes (Anjum et al., 1989; Cheema et al., 1989; Toro et al., 2000). The syndrome has been diagnosed as an infection of Avian Adeno virus Type 4 and formalized (Khawaja et al., 1988; Chishti et al., 1989; Afzal and Ahmed, 1990) and oil emulsion vaccine (Hussain et al., 1996), is available and is being used for immunization but still outbreaks of the disease are seen.

MATERIALS AND METHODS

Blood samples were pooled in heparinized and non-heparinized tubes from the infected broiler farms located in the suburbs of Karachi for clinical chemistry and whole blood evaluations using standard kit methods. The
hematological parameters evaluated were total red blood cell count, total white blood cells count, hemoglobin concentration, the differential white blood cell count and blood indices. For the clinical chemistry evaluation the parameters were pH, blood glucose, plasma protein, serum triglycerides, serum uric acid, serum sodium, serum potassium and serum calcium. In addition a control broiler flock was also reared at the Poultry Physiology Unit, Department of Physiology for a comparison with the field samples. However, control flock was regularly monitored for the presence of HPS antigen via agar gel immuno diffusion test (Shamim, 1990) and any other viral infection. The results so obtained were subjected to statistical analysis on MS Excel 2000. The level of significance was 0.05

RESULTS AND DISCUSSION

Hydropericardium syndrome a known viral infection of Avian Adeno virus type IV causes a mortality rate to as high as 20 to 90% with in 24 hrs. The disease causes an accumulation of straw colored fluid (up to 15 ml) in the pericardial cavity of chicken, which causes a significant rise in mortality rate.

This study was designed in the light that no data has yet been reported on the physiological and biochemical changes that are occurring during the course of infection, which may be one of the causes for the accumulation of excess fluid in the pericardial cavity.

The blood evaluation included the whole blood analysis and biochemical estimations as pH and glucose, triglycerides, plasma proteins, uric acid and ions as Na\(^+\), K\(^+\) and Ca\(^{++}\) concentration.

The results obtained after the blood analysis are summarized in Table 1. The complete blood profile revealed a significant rise in the number of red blood cells from (RBC) 1.19 x 10\(^9\) ± 0.05 (25) /cu mm in control to 2.21 x 10\(^9\) ± 0.02 (25) /cu mm in infected birds along with a significant decrease in mean corpuscular volume from (MCV) 146 ± 0.5 (25) in control to 135 ± 0.4 (25) in infected birds. In addition, the hemoglobin concentration was significantly increased from 7.24 ± 0.1 (25) gm/dl in control to 10.7 ± 0.08 gm/dl (25) in tested birds with a significant rise in mean corpuscular hemoglobin (MCH) percentage and mean corpuscular hemoglobin (MCHC) concentration from 38.1 ± 0.7 (25) % in control to 47.5 ± 0.4 (25) % in tested birds and from 28.5 ± 0.8 gm/dl (25) in control to 34.5 ± 0.3 gm/dl (25) in infected birds. This rise may be attributed to enhanced erythropoietic activity of the system (Sturkie, 1965). In addition an elevated number of white blood cells from 3.1 x 10\(^9\) ± 0.5 /cu mm (25) in control to 5.0 ± 0.3 x 10\(^9\) /cu mm (25) in infected birds gave a significant difference. This significant rise indicated the presence of an infection in the body (Sturkie, 1965). The differential count for the white blood cells revealed a significant difference for the heterophiles and lymphocytes. The significant rise in the heterophiles percentage from 20.9 ± 2.0 (25) in control to 45.0 ± 3.0 (25) in infected birds was an outcome of secondary infection and disease stress, whereas a significant drop in lymphocytes percentage was noted from 73.3 ± 3.0 (25) in control to 44.0 ± 2.0 (25) in diseased birds. A non-significant difference was noted for the monocyte, eosinophile and basophile percentage among the control and infected birds (Table 1, Fig. 1).

Table 1. Complete blood picture of broiler chicken (age 3 weeks)

<table>
<thead>
<tr>
<th>PARAMETERS</th>
<th>CONTROL</th>
<th>TESTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC (10(^{12}))</td>
<td>1.19 ± 0.05 (25)</td>
<td>2.21 ± 0.02 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>WBC (10(^9))</td>
<td>30.4 ± 0.5 (25)</td>
<td>50 ± 0.3 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>Hb g/dl</td>
<td>7.24 ± 0.1 (25)</td>
<td>10.7 ± 0.08 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>MCV cu</td>
<td>146 ± 0.5 (25)</td>
<td>135 ± 0.4 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>MCH %</td>
<td>38.1 ± 0.7 (25)</td>
<td>47.5 ± 0.4 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>MCHC g/dl</td>
<td>28.5 ± 0.8 (25)</td>
<td>34.5 ± 0.3 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>Heterophiles %</td>
<td>20.9 ± 2.0 (25)</td>
<td>45 ± 3.0 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>Lymphocytes %</td>
<td>73.3 ± 3.0 (25)</td>
<td>44 ± 2.0 (25) P &lt; 0.05</td>
</tr>
<tr>
<td>Monocytes %</td>
<td>3.1 ± 1.0 (25)</td>
<td>0.5 ± 2.0 (25) P &gt; 0.05</td>
</tr>
<tr>
<td>Eosinophils %</td>
<td>1.9 ± 2.0 (25)</td>
<td>0.5 ± 1.0 (25) P &gt; 0.05</td>
</tr>
<tr>
<td>Basophils %</td>
<td>3.1 ± 2.0 (25)</td>
<td>0 ± 1.0 (25) P &gt; 0.05</td>
</tr>
</tbody>
</table>

All Values are shown as Mean ± Standard Error
Results of blood analysis for the clinical chemistry values are summarized in Table 2. Evaluation of blood pH in controlled and infected birds illustrated a non-significant difference. On the other hand the level of glucose was significantly decreased from $291 \pm 4$ (25) gm/dl in control to $165 \pm 18$ (25) gm/dl in infected birds. This sharp decrease indicated an impaired carbohydrate metabolism, which could be attributed to low feed intake and stress condition.

A significant rise of serum triglycerides from $64.48 \pm 13.2$ (25) mg/dl to $98.25 \pm 9.27$ (25) mg/dl shows a high rate of lipolysis owing to an increase body demand for energy because of impaired carbohydrate metabolism (Bell and Freeman, 1971; Oser, 1976). Thus elevating the serum triglycerides. A significant decrease of plasma protein from $4 \pm 0.33$ (25) gm/dl to $2.24 \pm 0.23$ (25) gm/dl shows hepatic dysfunction since blood proteins are synthesized in the liver (Bell and Freeman, 1971). Therefore a decrease plasma protein was altered due to hepatic dysfunction, as this is one of the target sites of the virus.

A marked increase in serum uric acid and Ca$^{2+}$ from $9 \pm 0.5$ gm/dl to $18.24 \pm 2.7$ gm/dl and $57.75 \pm 7.95$ m mol/l to $89.56 \pm 10.2$ m mol/l respectively, which is a significant rise indicates decrease urinary excretion as a result of kidney dysfunction (Sturkie, 1965; Oser, 1976).

Table 2. Clinical chemistry values in blood of broiler chickens (age 3 weeks)

<table>
<thead>
<tr>
<th>PARAMETERS</th>
<th>CONTROL</th>
<th>TESTED</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>7.42 ± 0.19</td>
<td>7.59 ± 0.19</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Glucose mg/dl</td>
<td>291 ± 4</td>
<td>165 ± 18</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Plasma Protein g/dl</td>
<td>4 ± 0.33</td>
<td>2.24 ± 0.23</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Uric Acid mg/dl</td>
<td>9 ± 0.5</td>
<td>18.24 ± 2.7</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Triglycerides mg/dl</td>
<td>64.48 ± 13.2</td>
<td>98.25 ± 9.27</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Sodium m mol/l</td>
<td>168 ± 27</td>
<td>146 ± 21</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Potassium m mol/l</td>
<td>9.94 ± 1.86</td>
<td>13.13 ± 1.28</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Calcium m mol/l</td>
<td>57.75 ± 7.95</td>
<td>89.56 ± 10.2</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

All Values are shown as Mean ± Standard Error

The decrease in level of sodium ion concentration from $168 \pm 27$ (25) m mol/l to $146 \pm 21$ (25) m mol/l was found non-significant. Similarly the potassium concentration of $9.94 \pm 1.86$ (25) m mol/l increased to $13.13 \pm 1.28$ (25) m mol/l was also found non-significant. The study is conclusive in the aspect of evaluating the physiological and biochemical
changes during Hydropericardium syndrome. But is unable to answer the query for the accumulation of straw colored fluid in the pericardial cavity. Since no significant difference is seen in the concentration of biologically important ions sodium and potassium. The study recommends that an extensive study be carried out to evaluate the cause for the fluid accumulation (Table 2, Fig. 2).

![Fig. 2. Blood biochemistry of broiler chicks (average of 4 weeks).](image)

**REFERENCES**


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