Changes in the Biochemical Parameters of Experimental Chicks Due to Ascaridia galli Infection

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ABSTRACT
The W.L.H. chicks carrying A. galli infection showed ascaridiasis and anemia. The biochemical responses such as glucose, protein, cholesterol, urea, acid phosphates and alkaline phosphatase level after 16 days and 32 days at low dose and high dose of infection were studied. The infection showed a significant increase in serum cholesterol and serum urea levels in all the experimental cases. The serum protein level and serum acid phosphates level decrease in all the cases of dose administered. The level of glucose and alkaline phosphatase decreased after 16 days at low dose and high dose of infection and increased at the dose level after 32 days at low dose and high dose of infection. The infected chicks showed ascaridiasis and anemia.

Key word: A. galli, WLH chicks, serum protein, glucose, cholesterol, urea, acid phosphatase, alkaline phosphatase and ascaridiasis.

INTRODUCTION
Biochemical status of a host plays an important role for various activities. Quite often prevalent disease have been found to generally decrease but sometime increases the biochemical parameters like serum protein and serum phosphatase etc. Besides parasites biochemical parameters could be altered by antibiotics, chemicals and pollutants. The parasitic infection disturbs the internal micro-environment of the host by inducing various biochemical. Pathological and immunopathological changes. Parasites also disturb the carbohydrate, lipid and protein metabolism of host [1] causing various diseases like anaemia, diarrhoea, dysentery and eosinophilia etc. Poultry industry has recently developed and most of the person depends on poultry protein. In chicken a very large nematode Ascaridia galli is frequently found. Nematode infectious effect maintain in two fold ways.
1. By directly parasitizing man and causing various disease. This in turn causes poor health. Retarded growth and in some cases death.
2. By infecting large number of domesticated animals affect the yield of milk, protein, eggs, leather and animal.

Ascaridia galli infection causes ascaridiasis in white leg hom chicks. Male A galli 5 to 8 cm long and female 6 to 12 cm long. Their life cycle is direct. The early stage of infection leads to retarded growth and weight loss. The weight gain of infected chick was much lower than that of uninfected ones with reduced egg laying capacity and more over the eggs were not fully developed. A galli leads to malnutrition and lowering of product derived from poultry. The Biochemical parameters are closely related in blood.

MATERIAL AND METHODS
Newly hatched white leg horn (WLH) chicks were obtained from M/S salim Hatchery, Meerut. They were kept in clean wood and steel cages in animal house and acclimatized to the laboratory conditions for one week before initiation of the experiment.
The parasitic form of A galli are found in the intestine of fowl. Parasites were recovered from the intestine of freshly slaughtered fowl from local slaughter house. Intestine were cut and adult parasites were collected the adult parasite were kept in lock lewis solution for egg laying. The embryonated eggs were administered to laboratory maintained chicks. The chicks were autopsied and male and female parasites were obtained from experimentally infected chicks.
Blood was collected after 16 and 32 days of post infection with A galli. Serum was obtained studied for glucose, Protein cholesterol, urea, acid phosphatase and alkaline phosphatase using standard kit method.
RESULT

The data on biochemical values of WLH chicks both uninfected and infected with *A. galli* are presented in table. The observations are the following:

**Biochemical studies of low dose of infection of 150 embryonated eggs *A. galli* in WLH chicks:**

Serum obtained from the chicks infected with low dose of 150 embryonated eggs per birds were analyzed on the 16 days and 32 days of post infection to study the alterations of various biochemical parameters. The results obtained during the experiment have been obtained in the Table.

- Serum total protein level of control chick was 5.1 mg/dl and 4.95 mg/dl respectively after 16 and 32 days of post infection at low dose of infection.
- Serum glucose level of control chick was 366.6 mg/dl and 253.16 mg/dl after 16 and 32 days of pre-infection. It decreased to 200 mg/dl at 16 days of post infection; increased to 253.7 mg/dl after 32 days of post infection at low dose of infection.
- Serum cholesterol level of control chick was 219.04 mg/dl and 220.41 mg/dl after 16 and 32 days of pre-infection respectively. Following dose administration it increased to 304.76 mg/dl and 337.14 mg/dl after 16 and 32 days of post infection at low dose of infection.
- Serum urea level of control chick was 4.4 mg/dl and 3.6 mg/dl after 16 and 32 days of pre-infection respectively. Following dose administration it increased to 5.7 mg/dl and 5.37 mg/dl after 16 and 32 days of post infection at low dose of infection.
- Serum acid phosphates level of control chick was 5.30 KA units and 5.35 KA units respectively. Following dose administration it decreased to 5.28 KA units and 4.11 KA units after 16 and 32 days of post infection respectively.
- Serum alkaline phosphatase level of control chick was 16.3 KA units and 10.28 KA units after 16 and 32 days of pre-infection respectively. Following dose administration it increased to 16.9 KA units after 32 days of post infection at low dose of infection.

**Biochemical studies of high dose of infection of 1500 embryonated eggs *A. galli* in W.L.H. chicks:**

Serum total protein level of control chick was 5.1 mg/dl and 4.95 mg/dl after 16 and 32 days of pre-infection. Following dose administration it decreased to 4.22 mg/dl and 4.6 mg/dl respectively after 16 and 32 days of post infection at high dose of infection.

- Serum glucose level of control chick was 366.6 mg/dl and 253.16 mg/dl after 16 and 32 days of pre-infection. It decreased to 172.22 mg/dl at 16 days of post infection; increased to 318.45 mg/dl after 32 days of post infection at high dose of infection.
- Serum cholesterol level of control chick was 219.04 mg/dl and 220.41 mg/dl after 16 and 32 days of pre-infection respectively. Following dose administration it increased to 306.66 mg/dl and 323.08 mg/dl after 16 and 32 days of post infection at high dose of infection.
- Serum urea level of control chick was 4.4 mg/dl and 3.6 mg/dl after 16 and 32 days of pre-infection respectively. Following dose administration it increased to 5.1 mg/dl and 6.15 mg/dl after 16 and 32 days of post infection at high dose of infection.
- Serum acid phosphatase level of control chick was 5.30 KA units and 5.35 KA units at pre-infection respectively. Following dose administration it decreased to 4.1 KA units and 4.6 KA units after 16 and 32 days of post infection respectively.
- Serum alkaline phosphatase level of control chick was 16.3 KA units and 10.28 KA units after 16 and 32 days of pre-infection respectively. Following dose administration it decreased to 5.5 KA units at 16 days of post infection; increased to 19.8 KA units after 32 days of post infection at high dose of infection.

**DISCUSSION**

The parasitic effects of parasite arise from their action on biological system. The potential site is blood where various biochemical parameters are affected.

During the present observation the serum protein level in white Leg horn chicks showed fall throughout the experiment. Hypoproteinemia was observed in the chicks with low as well as high doses of embryonated *A. galli* eggs. Yon Brand [1] observed that every early parasitic infestation causes any disturbances in the protein digestion in the host. But changes in the serum protein levels have the characteristic features of many parasitic infections. He also observed that the variations in the serum protein values occur depending on the stages or severity of an infection, the host species, the presence or the absences of a previous infection.

During *A. galli* infections, there is a lowered protein absorption in the gastrointestinal tracts of chicks due to its excretions or secretion. This malabsorption results fall in the serum protein level of the chicks.
A slight decline in the serum protein level during ascariasis infection in children was also reported by Venkatachalam and Patwardhan [2]. Contradictorily, an increased serum protein level was observed by Khadoon and Ansari [3] in buffalo calves with Setaria cervi infection. The fall in the serum glucose level was attributed to the disturbance in the carbohydrate metabolism in A. galli infection in chicks. Decrease in the value of serum glucose in A. galli infected chicks is due to utilization of such toxin developing by parasite. The fall in the glucose level in experiment may be attributed to decrease in adrenocorticol. Dubinsky et al [4] also reported that A. galli infection lead to a decrease in the serum glucose level. Fall in the glucose level in infected host was reported Dhanakkodi and Aruchami [5], Gordon and Webster [6] and Dubinsky [4].

Srivastava et al [7] also reported decrease in the level of serum glucose when they observed the biochemical changes in the serum of cattle immunized with tick tissue extract of Baophilus microplus. Kumar [8] and Gupta [9] was reported a decreased serum glucose level in sheep hook worm. Bunostomum trigonocephalum infected with experimental schistosomiasis. Sadun and Williams [10] also reported a decreased serum protein level in albino rats during experimental schistosomiasis.

The presence of parasitic infection in the gastrointestinal tracts of the host causes a leakage of plasma in the intestinal lumen of the injured intestine and this leakage results in the protein level of the host [11]. Thus the decreased plasma level is the result of either loss of plasma by renal excretion or when the synthesis of protein is impaired due to malnutrition and deficiency of vitamins or diseases involving the digestive organs and liver [12].

The increased cholesterol level in the serum of the infected chicks may be due to the increased lipid metabolism. This may have been brought about by the excretory and secretory substances of A. galli. The increase in the cholesterol level depends upon the severity of the parasitic infection and the inhibited activity of the enzyme involved in the anabolism of the lipid in the host tissue. Kumar [8] reported a rise in the serum cholesterol level in experimental rats with sheep hookworm. A decrease in the serum cholesterol level was also reported by Rao (1991) in white Leg Horn chicks experimentally infected with; A. galli.

The serum urea level was found to increase throughout the experimental period. The larvae and adult forms of A. galli present in the lumen of the intestine of the WLH chicks excrete and secrete certain toxic substances, causing nephrotoxicity, caused by the ingestion of these toxic substances may be the main cause of the elevated serum urea level [12]. The elevation in the serum urea level may be also cause of the increased nitrogen metabolism due to the presence of A. galli in the gastrointestinal tract of chicks. Toxic metal are also accumulate by the kidney so produce toxin and increase urea level in the blood. Kumar [8] and Gupta [9] also observed an increased serum urea level in Bunostomum trigonocephalum infected albino rats.

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The alterations caused by the parasites, A. galli in the various biochemical parameters of the experimental host. WLH chicks may be due to the various toxic substances secreting by the parasite via its excretions and secretion into lumen of the gastrointestinal tract of the host. These toxic substances cause leakage of the plasma and affect the carbohydrate, protein and lipid metabolism of the host toxic substances also affect the all activity of the host. Liver and kidney are the targeting organs which accumulate toxic substance. This toxic substance affects the host homestasis.

The present observation revealed a slight decrease in the serum acid phosphatase level in the chicks with an experimental A. galli infection. Pandey and Rai [14] reported no marked difference in the acid phosphatase activity during experimental taeniasis in pups. Also reported no increase or any significant changes in the serum acid phosphatase activity in chicks with an A. galli infection. The decrease in the serum acid phosphatase level may be due to disturbed metabolism in chicks intestinal tissue during A. galli infection. Rao [13] also demonstrated an increase in the serum acid phosphatase level in WLH chicks experimentally infected with A. galli.

Rao [13] also observed an increased in alkaline phosphatase activity in chicks with experimental ascariasis. Panday and Rai [14] observed alkaline phosphatase activity in the mucosa of the intestine of pups with experimental taeniasis as compared to the control. Kumar [8] also reported an elevated serum alkaline phosphatase level in the albino rats with an experimental B.
Table: Biochemical responses in blood of W.L.H. treated with *Ascaridia galli* infection.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control 16 days</th>
<th>Control 32 days</th>
<th>Low dose infection 16 days</th>
<th>Low dose infection 32 days</th>
<th>High dose infection 16 days</th>
<th>High dose infection 32 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Protein (mg/dl)</td>
<td>5.1 ±0.1 ±0.0577</td>
<td>4.95 ±1.3 ±0.07505</td>
<td>3.35 ±0.01 ±0.0057</td>
<td>3.6 ±0.5 ±0.2880</td>
<td>4.22 ±0.02 ±0.0115</td>
<td>4.6 ±0.8 ±0.4618</td>
</tr>
<tr>
<td>Serum Glucose (mg/dl)</td>
<td>366.6 ±0.4 ±0.2309</td>
<td>253.16±5.9 ±3.4237</td>
<td>200 ±14 ±0.0831</td>
<td>253.7 ±4.4 ±2.5404</td>
<td>172.22±3.02 ±1.7436</td>
<td>318.45±2.83 ±1.6339</td>
</tr>
<tr>
<td>Serum Cholesterol (mg/dl)</td>
<td>219.04 ±0.06 ±0.0346</td>
<td>220.41±0.54 ±0.3117</td>
<td>304.76±0.83 ±0.4792</td>
<td>337.14±0.89 ±0.5130</td>
<td>306.66±0.83 ±0.4792</td>
<td>323.08±0.27 ±0.1558</td>
</tr>
<tr>
<td>Serum Urea (mg/dl)</td>
<td>4.4 ±1.2 ±0.6928</td>
<td>3.6 ±0.5 ±0.2867</td>
<td>5.7 ±0.5 ±0.2886</td>
<td>5.37 ±0.61 ±0.3521</td>
<td>5.1 ±0.2 ±0.1155</td>
<td>6.15±0.18 ±0.1039</td>
</tr>
<tr>
<td>Serum Acph (K.A. units)</td>
<td>5.30 ±0.03 ±0.0173</td>
<td>5.35 ±0.38 ±0.2194</td>
<td>5.28 ±0.06 ±0.0346</td>
<td>4.1 ±1.09 ±0.6293</td>
<td>4.10±0.9 ±0.5196</td>
<td>4.60±0.33 ±0.1905</td>
</tr>
<tr>
<td>Serum Alph (K.A. units)</td>
<td>16.3 ±0.3 ±0.1732</td>
<td>10.28±0.5 ±0.2886</td>
<td>13.6 ±0.6 ±0.3460</td>
<td>16.9±1.8 ±1.0392</td>
<td>15.5±0.7 ±0.4042</td>
<td>19.8±0.9 ±0.5196</td>
</tr>
</tbody>
</table>

Results are mean ± S.E.(n=3)

REFERENCES