Haematological and Biochemical Study on Albino Rats Infected with 70 ± 10 Cercariae Schistosoma Mansoni

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ABSTRACT

In an attempt to study the pathogenesis of S.mansoni 70 ± 10 cercariae of S. mansoni were used in albino rats. The S.mansoni infection was evaluated by determining the following parameters; parasitological parameters which include worm burden and eggs/gm liver or intestine tissues. Biochemical parameters (total proteins, albumin, globulin and A/G ratio) and hematological indices, differential counts and MCH, MCV, MCHC were calculated accordingly. Results indicated that anemia and hypoalbumenia were recorded in all infected rats and the strain of S. mansoni was pathogenic to all infected animals. Therefore this dose of infection is highly recommended to further investigations on other aspects of the disease.

Key words: Schistosoma mansoni-pathogenesis- Biomphalaria alexandrina

Introduction

Schistosomiasis is one of the most wide spread human disease in tropical countries and is one of the major communicable diseases of public health and socio-economic importance in the developing countries [13]. Mortality due to this disease is relatively low, while morbidity is high [36]. The distribution of the disease is highly related to water development projects and according to the reports of World Health Organization (WHO), about 200 million people infected with this disease and about 120 million are symptomatic. This disease is the second most prevalent parasitic disease in the world and is second to malaria only. The S.mansoni life cycle is indirect and the three species that affect man utilize water snails to complete their life cycle which is well known.

The pathology of schistosomiasis is mainly due to chronic inflammatory lesions produced by the parasites and their eggs and sometimes the dead adult worms [4]. However, the disease progress due to large numbers of ova or lasting for several years, which lead to fibrosis of the portal tracts, urinary bladder, intestine, genitourinary tract, neural and pulmonary lesions [26].

In chronic S.mansoni infected animals, the intensity of the granulomatous lesions became maximal at 6-8 weeks post infection, and then subsequently declined reaching a minimum by 20 to 32 weeks. This phenomenon was first called endogenous desensitization and more recently called modulation [33].

Several reports on blood chemistry of this disease (total serum protein, albumin, globulin, A/G ratio) showed a marked reduction of these values. A similar trend exhibited by blood indices (PCV, HB, RBC’s) have been reported by several authors. [15,3,2]. Leukocytosis had been reported in patients suffering from schistosomiasis especially in the early stages of the disease [17], while eosinophilia had been reported in patients suffering from acute schistosomiasis [10,19].

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Despite the great deal of work which has been done on schistosomes control, chemotherapy, sanitation, health education, snail control and diagnosis still a lot of research is needed to solve many aspect of this disease. Therefore the aim of the present study is to add further information to the pathogenesis of schistosomiasis utilizing hematological and biochemical parameters.

Materials and Methods

Cercariae were shed from laboratory-bred infected snails (Biomphalaria alexandrina) 25-30 days after exposure to miracidiae. Infected snails were collected from the aquarium, washed in dechlorinated water and exposed to light for two hours [29]. The snails were then removed from the aquarium, the cercarial suspension was gently mixed and one hundred microliter of the suspension were ± 10 S. mansoni cercariae according to the method described by Olivier and Stirewalt, [27]. The maintenance of the infected snails and rats were carried out in the main lab of biology department college of Science through out the period of study.

Experimental mice divided into two groups, ten infected and ten remain as normal control. Mice were sacrificed after eight and ten weeks post infection. Blood samples were collected and sera were separated by centrifugation at 3000 r.p.m. for 15 min for biochemical analyses (total serum protein, albumin, globulin, A/G ratio) were performed by a fully automated machine in Alkarak Central Lab ministry of health.

Blood samples were collected in EDTA tubes to be used for the estimation of haematological parameters (Hb, PCV, RBC, WBC counts, Differential counts, MCH, MCV, MCHC) were performed by a fully automated machine in Alkarak Central Lab ministry of health.

The total worm burden in both liver and intestine were done according to the method of Pellegrino et al.; [29]. While egg counts (EPG) in liver and intestine were estimated according to [7, 20].

Results and Discussion

Hematological findings:

In comparison with the control infected rats showed a reduction in blood values (RBC’s, Hb, PCV, MCV, MCH and MCHC) through the two intervals times (Table 1).The PCV showed a reduction especially during the week ten post-infection. Meanwhile all other values showed a similar trend throughout the period of the study. A significant increase (8.93±0.70, 7.68±0.75) respectively (p<0.05) in total WBC’ s has been noticed during the both periods under observation eight weeks and ten weeks post study. A remarkable increase in eosinophils, this phenomenon has been observed as early as eight weeks post-infection. Also other blood cells (Neutrophils, Lymphocytes, Monocytes, Basophils) showed a clear increase in their total number (table 2).

Biochemical changes:

The biochemical values of total protein, albumin, globulin and A/G ratio of the infected rates started to drop on week eight and continue till the end of the experiment as compared to control group (table 3).

Parasitological Findings:

The Number of Ova/g Tissue in the Liver and Intestine:

Total number of eggs in liver and intestine was higher in week ten than in week eight (11728.54±1077.08, 15345.21±830.98) respectively, (10751.17±1231.32, 13840.25±638.47) respectively. However, it is much higher in intestinal tissue than liver tissue during both period of observation.

Total Worm Burden:

There was no marked differences in total worm burden of both groups (25.56±3.00, 22.86±3.12) respectively and no differences was observed in total worm burden.

Discussion

The results of the present study revealed several interesting facts concerning S. mansoni infection in rats.

Firstly: All the pathophysiological changes that accompanied the course of the infection indicated that S. mansoni is a pathogenic parasite. Where a severe anemia and hypoalbuminemia were practiced by all the infected rats. These findings are in agreement with [5,31,3].

Secondly: It seems that the reduced levels of protein and albumin in association with schistosomal infection could be due to impaired synthesis of these substances in liver and/or increased leakage of albumin through intestinal mucosa induced by the activity of these flukes [11].

Thirdly: Anemia of S. mansoni infections at present study is either according to their diminished production or acceleration loss of erythrocytes [35]. hypochromia and microcythemia were moderate as indicated by MCV, MCH and MCHC. However, hypochromia due to schistosomiasis was not uncommon and it was reported in different animals and human beings evidenced by reduced MCH and
MCHC [32,6,23]. Many investigators explained the above findings of anemia due to blood loss, increased hemolysis and iron deficiency [16,14,28]. The direct loss of erythrocytes may arise from the extrusion of schistosomal ova or because of the consumption of blood by schistosomes [24,22].

Fourthly: Infection with S. mansoni was found to cause an elevation in WBC’s counts and differential WBC’s which has been indicated by the increase of Neutrophils, Lymphocytes, Eosinophils, Monocytes and Basophils in response to S. mansoni infections. However, it’s not uncommon to find an increase in the total counts of eosinophils in helminthic infection [9,25,34,30,21,18,1].

Finally: Our results indicated that there was a good correlation between worm burden and the pathogenesis induced by these parasites.

Acknowledgment

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In fact I owe much to the stuff member of Al-Karak Central Lab Ministry of Health for the facilities so freely placed at my disposal and stuff of the Animal House of Theodor Bilharz Research Institute (T.B.R.I.), Giza, Egypt for supplying infected snails.

Table 1: Haematology values pre and post infection with S. mansoni.

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>Red Blood Cells (RBCs) value×106/µl</th>
<th>Hemoglobin Hb g/dl</th>
<th>PCV %</th>
<th>MCV fl</th>
<th>MCH Pg</th>
<th>MCHC g/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 Weeks</td>
<td>Control (n=6) 7.62 0.23</td>
<td>13.65 0.20</td>
<td>41.48 0.60</td>
<td>54.03 2.38</td>
<td>18.22 0.77</td>
<td>33.5 0.18</td>
</tr>
<tr>
<td></td>
<td>Pos-infection (n=6) 6.85 0.26</td>
<td>11.95 0.38</td>
<td>36.00 1.13</td>
<td>51.57 2.59</td>
<td>16.93 0.89</td>
<td>32.22 0.27</td>
</tr>
<tr>
<td>10 Weeks</td>
<td>Control (n=6) 7.73 0.14</td>
<td>13.27 0.22</td>
<td>39.93 0.71</td>
<td>51.67 0.95</td>
<td>17.20 0.38</td>
<td>33.25 0.28</td>
</tr>
<tr>
<td></td>
<td>Pos-infection (n=6) 6.54 0.19</td>
<td>10.30 0.88</td>
<td>31.93 2.51</td>
<td>48.67 3.15</td>
<td>15.65 1.08</td>
<td>31.60 0.56</td>
</tr>
</tbody>
</table>

Table 2: Differential WBC’s counts pre and post infection with S. mansoni.

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>Neutrophils value×10³/µl</th>
<th>Lymphocytes value×10³/µl</th>
<th>Eosinophils value×10³/µl</th>
<th>Monocytes value×10³/µl</th>
<th>Basophils value×10³/µl</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 Weeks</td>
<td>Control (n=6) 5.02 0.27</td>
<td>1.648 0.144</td>
<td>3.196 0.180</td>
<td>0.017 0.011</td>
<td>0.159 0.018</td>
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<tr>
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<td>Pos-infection (n=6) 8.93 0.70</td>
<td>2.947 0.491</td>
<td>5.090 0.188</td>
<td>0.699 0.159</td>
<td>0.179 0.033</td>
</tr>
<tr>
<td>10 Weeks</td>
<td>Control (n=6) 4.98 0.26</td>
<td>1.63 0.128</td>
<td>3.20 0.161</td>
<td>0.033 0.019</td>
<td>0.116 0.018</td>
</tr>
<tr>
<td></td>
<td>Pos-infection (n=6) 7.68 0.75</td>
<td>2.32 0.279</td>
<td>4.53 0.542</td>
<td>0.666 0.092</td>
<td>0.153 0.024</td>
</tr>
</tbody>
</table>

Table 3: Biochemical changes due to S. mansoni-infection.

<table>
<thead>
<tr>
<th>Animal groups</th>
<th>Total protein (g/dl)</th>
<th>Albumin (g/dl)</th>
<th>Globulin (g/dl)</th>
<th>A/G ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 Weeks</td>
<td>Control (n=8) 7.14 0.30</td>
<td>3.13 0.32</td>
<td>4.00 0.24</td>
<td>0.82 0.11</td>
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<td>Pos-infection (n=9) 6.00 0.51</td>
<td>2.46 0.24</td>
<td>3.54 0.38</td>
<td>0.74 0.09</td>
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<tr>
<td>10 Weeks</td>
<td>Control (n=7) 7.19 0.44</td>
<td>3.39 0.28</td>
<td>3.80 0.55</td>
<td>1.11 0.28</td>
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<td>Pos-infection (n=7) 5.46 0.44</td>
<td>2.30 0.27</td>
<td>3.16 0.35</td>
<td>0.77 0.12</td>
</tr>
</tbody>
</table>

References


