EFFECT OF ACUTE HEAT STRESS ON CERTAIN IMMUNOLOGICAL PARAMETERS IN ALBINO RATS

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Abstract: The effect of acute heat stress on certain immunological parameters were studied in male albino rats. The test rats were exposed to an ambient temperature of 40 degrees celsius for 30 minutes and sacrificed immediately. Total WBC count, Differential count, Phagocytic index, NET reduction, organ weight body weight ratio of spleen, thymus, and popliteal lymph nodes, and soluble immune complex levels were measured in control group and the heat stressed animals. The heat stressed animals show decrease in total WBC count, and neutrophilia, eosinophilia, and lymphocytopenia. The phagocytic index showed a significant increase whereas the avidity index showed a decrease from the control value. NET reduction was also significant. The soluble immune complex level was not altered. The heat stressed animals showed a decrease in the thymus and spleen weight/body weight ratio while the lymph node/body weight ratio showed an increase compared to the control animals.

Key words: heat stress immune system

INTRODUCTION

The term stress is ambiguous and precludes accurate definition. Biological stress of various types leads to General Adaptation Syndrome (1). The effect of stress on the immunological functions in human beings, resulting in psychosomatic disorders has been an important observation (2). Heat stress is encountered by all of us in every day life. When an individual is exposed to temperatures above the zone of thermal neutrality, changes in pituitary-adrenal axis occur which may influence the immune mechanisms. Since the available literature is sparse in this field, this investigation was undertaken to study the influence of acute thermal stress on certain immune parameters.

METHODS

Healthy Wistar strain male albino rats, weighing 120-130 g, were used in this study. The animals were housed (5/cage) in standard plastic cages (30 cm × 22 cm × 14 cm) and provided with routine pellet feed and water ad libitum. Ten animals were used as the control group and another ten animals were used as the experimental group. Except the heat stress, the test and control groups were maintained under identical conditions.

Heat stress procedure: The rats were placed individually in an insulated wooden box heated by a thermostatically controlled infra red lamp. The box temperature was maintained at 40° Celsius and the test rats were individually exposed to this temperature for 30 minutes. The control rats were also placed in the same chamber to attain identical conditions for the same period of time, but without switching on the infra red lamp.

Immediately after the exposure, the rats were sacrificed by exsanguination from jugular vein under light ether anesthesia and blood thus collected was used for the different investigations. Total
WBC counts and differential counts were determined following routine methods. The phagocytic activity of the neutrophils was determined by using heat killed candida (3). Microbicidal activity of the neutrophils was determined by the NBT reduction test (4). Soluble immune complex levels were determined by the method of Seth and Srinivas (5). Animals were then sacrificed by an overdose of ether and the wet weights of spleen, thymus and popliteal lymph node determined and expressed as mg/g of body weight.

RESULTS

The effects of acute heat stress on the various parameters studied are shown in Tables I, II and III.

TABLE I: Changes in total WBC and differential count in acute heat stress. (N = Neutrophil, L = Lymphocyte, E = Eosinophil, M = Monocyte, B = Basophil) values are mean of 10 animals +/-SEM.

<table>
<thead>
<tr>
<th>Group</th>
<th>Total WBC count/ c.mm</th>
<th>Differential count in %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>18,635</td>
<td>24.5 70.0 2.4 5.1 0.3</td>
</tr>
<tr>
<td>n=10</td>
<td>+/-/ +/-/ +/- +/- +/-</td>
<td></td>
</tr>
<tr>
<td>Acute heat</td>
<td>8,840</td>
<td>28.6 59.2 9.1 2.5 0.1</td>
</tr>
<tr>
<td>stressed</td>
<td>+/- +/- +/- +/- +/-</td>
<td></td>
</tr>
<tr>
<td>n=10 (P&lt;0.001)</td>
<td>NS &lt;0.05) (P&lt;0.001) (P&lt;0.001) (P&lt;0.05) NS</td>
<td></td>
</tr>
</tbody>
</table>

TABLE II: Candida phagocytosis, NBT reduction and soluble immune complex in acute heat stressed albino rats.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Phagocytic Index</th>
<th>Avidity Index</th>
<th>NBT Reduction</th>
<th>SIC Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>56.2</td>
<td>2.937</td>
<td>11.2 17.0</td>
<td></td>
</tr>
<tr>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
<td></td>
</tr>
<tr>
<td>2.14</td>
<td>0.17</td>
<td>0.71 2.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute heat</td>
<td>70.6</td>
<td>2.259</td>
<td>6.5 11.0</td>
<td></td>
</tr>
<tr>
<td>stressed</td>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
<td></td>
</tr>
<tr>
<td>animals</td>
<td>2.21</td>
<td>0.16 0.31</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>n=10 (P&lt;0.001)</td>
<td>(P&lt;0.02) (P&lt;0.01) NS</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Phagocytic index in heat stressed animals showed a highly significant increase (P < 0.001) whereas the avidity index was significantly decreased (P < 0.02). NBT reduction was also significantly decreased (P < 0.01). The soluble immune complex levels however did not show any significant change.

Thymus and Spleen did not show any weight changes after heat stress while the weight of popliteal Lymph node was significantly increased (P < 0.01).

DISCUSSION

In the present study certain immunological parameters were studied after an acute exposure of rats to heat stress. The results suggest that such a stress influences certain immune parameters.

Our previous studies with cold stress had also shown a decrease in the total WBC count (6). Selye (1) had observed leucopenia in stressed animals. Jensen and Rasmussen (7), and Ramsey (8) and many others, have shown the involvement of adrenals in influencing the white cell contents of blood. Hence the observed decrease in white cell count of stressed animals may be attributed to the action of adrenal steroids. However, the possibility of some local chemotactic factors, causing a shift of the circulating leucocyte
population into the reservoirs, cannot be ruled out. The increase of adrenal steroids, while explaining the decrease in total WBC count, fails to offer a satisfactory explanation for the observed eosinophilia. Corticosteroids are known to produce lymphocytopenia and eosinopenia; whereas in this study while lymphocytopenia was observed, there was eosinophilia on the other hand. Similar observations have also been reported by Lee (9) in cold stressed rabbits and by Sheela and Namasivayam (10) in rats after acute cold swimming stress. In this connection it is pertinent to point out that stress induces both elevation of catecholamines and corticosteroids. Samuel (11) reported a primary and secondary leucocyte changes after the injection of epinephrine. Probably the increase in eosinophils reflects the primary response to the increase in catecholamines in the stressed animals. The absence of eosinophilia in chronically heat stressed animals (unpublished observation) also lends support to this concept of catecholamine action.

Published work on neutrophil function during stress is scanty and controversial. The increase in phagocytic index observed in this study is probably due to the action of stress produced hormones acting on the phagocytic cell receptors, which alter the metabolic activity of these cells. Similar results were reported by us when using subacute cold stress (6).

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