CARTA AO EDITOR

MORPHOMETRIC STUDY OF HEPATIC GRANULOMA IN OFFSPRING OF *Schistosoma mansoni* INFECTED MICE

There is a considerable interval between the exposure to antigens and the first appearance of specific antibodies in mammals, at birth. This interval is occupied by the transmission of maternal antibodies. Specifically in mice, little antibody is transferred by the yolk sac and placenta, the passive transmission is almost exclusively postnatal by means of absorption of lacteal secretion in the intestine. In the proximal small intestine, there is a selective absorption of IgG, which is bound to an intestinal Fc fragment receptor, internalized by endocytosis and then absorbed by the vascular system. In the distal small intestine little selectivity is found. At day 16 after birth the transmission of IgG ceases, the receptors disappear from the gut, although system and the hepatic granulomatous lesions were studied in order to evaluate the effects of maternal transmission of schistosomal antigens and antibodies to offspring of *Schistosoma mansoni* infected mice in relation with the time of mother infection.

C57BL/6 female mice 8-10 weeks old were infected subcutaneously (10 ± 5 cercariae per mouse) and then challenged five times each two weeks. Infected females were separated in two groups of ten. In the first group, the females were mated ten days following the third inoculation, breeding at the 60th day after the first inoculation. The second group was mated the day after the last inoculation, breeding at the 80th day after the first inoculation. The mice born of the first group formed the experimental group A and the mice born of the second formed the experimental group B. The control group was formed by mice born of non-infected females. Offspring of infected and non-infected females were infected at 0-4 days by placing each one in a Petri dish, containing 20 ± 2 cercariae/5 ml of aquarium water at 37°C for 30 minutes. The cercariae were counted in a Petri dish before and after infection. The litters were killed at the 50th day of infection and adult worms were recovered by perfusion of the portal system. The livers were fixed in 10% formalin and paraffin sections were stained with hematoxylin-eosin. The granulomatous lesions were measured by a morphologic program video-plan (a system of image analysis managed by a computer). Only newly formed granulomatous lesions were measured, e.g. cellular lesion containing an eosinophilic miracidium. Student test was used to evaluate the differences.

Table 1 – Recovery of adult worms, measurements of granuloma and granuloma density in experimental and control groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Worm recovery</th>
<th>Area (mm²)</th>
<th>Perimeter (mm)</th>
<th>Diameter (mm)</th>
<th>Density</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>8.6 ± 6.5</td>
<td>0.11 ± 0.05</td>
<td>1.26 ± 0.37</td>
<td>0.38 ± 0.10</td>
<td>0.02 ± 0.01</td>
</tr>
<tr>
<td></td>
<td>(nm = 19)</td>
<td>(ng = 203)</td>
<td>(ng = 203)</td>
<td>(ng = 203)</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>1.7 ± 1.9</td>
<td>0.12 ± 0.06</td>
<td>1.31 ± 0.38</td>
<td>0.40 ± 0.11</td>
<td>0.01 ± 0.02</td>
</tr>
<tr>
<td></td>
<td>(nm = 9)</td>
<td>(ng = 43)</td>
<td>(ng = 43)</td>
<td>(ng = 43)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>5.7 ± 6.2</td>
<td>0.13 ± 0.06</td>
<td>1.38 ± 0.39</td>
<td>0.41 ± 0.11</td>
<td>0.02 ± 0.02</td>
</tr>
<tr>
<td></td>
<td>(nm = 7)</td>
<td>(ng = 93)</td>
<td>(ng = 93)</td>
<td>(ng = 93)</td>
<td></td>
</tr>
</tbody>
</table>

nm, number of mice; ng, number of granulomas. The results are presented as mean ± standard deviation. Granuloma density was calculated by the formula: Σ granuloma section area/ Σ hepatic section area.

We have carried out some experiments in which adult worms were recovered by perfusion of the portal system and the maternal milk still contains immunoglobulins. Others studies suggest sensitization to schistosomal antigens by means of the transfer of circulating antigens from mother to foetus through placenta or milk.

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All groups had an efficiency of infection close to 95%. The maternal transfer of antibodies (and antigens) did not contribute to change susceptibility to infection since the worm recovery was not significantly different (p > 0.05) in experimental and control groups (Table 1). Differences concerning area, perimeter and mean diameter of granulomas were not significant when groups A and B were compared, despite the difference relative to the time of mother infection. Granulomas of group A were smaller in comparison to that of control group (p < 0.05). This finding could be explained by exposure of the foetus to parasite antigens, resulting in reduced immunoresponsiveness and pathology. Although immunotolerance response on tissues in usually characterized by little or no cellular reaction around eggs in the liver, this was not observed in the present study. Further experiments are being carried out to clarify some of the discussed results.

REFERENCES