

Histopathological, Morphometric and Stereological Analysis of the Effects of Alcoholism on the Intestine of Schistosomiasis-Infected Mice

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Although the impact of ethanol and *Schistosoma mansoni* infection on small intestine damage has been extensively studied individually, the intestinal histopathological effects of their comorbidity remain uncertain. To investigate this gap, male Swiss Webster mice infected with 80 cercariae received 200 μ L of 18% ethanol orally for 28 consecutive days. The animals were divided into the following groups: C (Control), E (Uninfected and ethanol intake), I (infected without ethanol intake), and IE (infected and ethanol intake). After nine weeks of infection, the mice were euthanized, and the small intestine (jejunum) was removed and fixed in 10% formalin. A fragment of the intestine underwent routine histological processing and Hematoxylin-Eosin staining for histopathological, morphometric and stereological analysis. The group C presented the four intestinal layers preserved and with characteristic morphological features. In group E, altered Paneth cells, protein denaturation in intestinal glands extending to the mucosal layer, submucosa, and muscularis due to ethanol-influenced pH changes, and reduced vascularization of enterocytes were observed. In group I, schistosomiasis-induced inflammation in the mucosa and submucosa, with Paneth cells, goblet cells, and leukocyte infiltration in all layers except the serosa, led to villus destruction. In group IE, there was an increase in the muscular layer thickness compared to both group E (129%) and group I (61%). Additionally, an increase in the number of Paneth cells was observed compared to both group E (200%) and group I (125%), and a reduction in goblet cells counts (-67%) compared to both groups. Leukocyte infiltration increased by 133% in quantity compared to the ethanol group (E). However, the inflammatory response was comparable to that of the infected group (I). The association of schistosomiasis infection with ethanol intake exacerbated the inflammatory process, leading to greater tissue damage in the intestine.

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