Abstract

Background

Human schistosomiasis is one of the most prevalent parasitic diseases worldwide. Various host factors can affect the host–parasite interactions, including changes in metabolism, immunological responses, and the genetic background. These changes to the host may lead to the disruption of important parasite functions such as oviposition, worm development and the resulting pathology associated with the infection.

Aim

In the current study, we sort to determine the parasitological, histopathological, biochemical, and immunological status of the host impacted by *Schistosoma mansoni* infection, concurrent with host metabolic disorders through animal models of streptozotocin-induced diabetes mellitus (DM) and obesity. Aiming to identify the underlying mechanisms of schistosome host interactions leading to the induction of the pathology.

Methods

The study animals were divided into four groups. Group I is control groups, containing normal, infected, and non-infected DM1, DM2, and obese groups. The mice of the other three groups undergone induction of DM1 (Group II), DM2 (Group III) and obesity (Group IV) before being infected. All mice groups were euthanized in week 8 after cercarial challenge. Mice were subjected to body weight weekly measurement, blood glucose and insulin assessment, parasitological evaluation of tissue egg count and intestinal oogram. Histopathological and immunohistochemical studies were done using anti-glial fibrillary acidic protein (GFAP) and image analysis of Masson's trichrome stained liver sections using Image J (Fiji). Additionally immunological analysis of TNF-β, IL-5, IL-10, FOXP3 and PTX3 levels besides biochemical study of total lipid profile were evaluated.

Results

The present study revealed a significant increase in tissue egg output in the obese group compared to the infected control group. The oogram of counted eggs showed prevalence of immature eggs in DM1 group, while DM2 and obese groups showed prevalence of mature eggs. The fibrosis area percentage showed significant increase in DM2 and obese groups while it was decreased in DM1 group in comparison to infected control group. Concerning the immunological parameters, the present results

showed significant increase in the levels of TNF-β, IL-5, PTX3 in DM1, DM2 and obese groups in comparison to infected control group, whilst the levels of FOXP3 and IL-10 were increased in the infected groups in comparison to their non-infected controls. Regarding the biochemical study, infected DM1, DM2 and obese groups showed higher blood glucose and lipid profile in comparison to the infected control group. However, these parameters were improved in comparison to their non-infected controls.

Conclusions

Our study has contributed to the unravelling of the mechanisms of the interaction between schistosome infection and metabolic disorders of the host. Induction of DM2 and obesity increased the body mass, egg count, mature egg percentage, and fibrosis density, while schistosome infection induced changes in the lipid profile and blood glucose levels in infected diabetic and obese groups and impacted favorably insulin levels in obese mice. These differences may aid therapeutic and vaccine studies to better controlling these diseases and improving outcomes in endemic regions. By better understanding the complexities of host–parasite interactions, efforts to reduce the burden of these debilitating diseases can be improved.