Irritable bowel syndrome in Upper Egypt: The role of intestinal parasites and evidence of Th2 response

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Abstract:

Background and study aims: The pathophysiology of irritable bowel syndrome (IBS) remains elusive. In countries where enteric parasitic infection is common, its role in the development of IBS is controversial. Parasites induce the Th2 immune response that elaborates cytokines such as interleukin (IL)-5, which causes eosinophilia. Eosinophilic cationic protein (ECP) is one of the mediators released during the activation of eosinophils. This study aims to determine the relationship between symptoms suggestive of IBS and parasitic infection in IBS patients and to evaluate the serum levels of IL-5, ECP and eosinophilic count as potentially useful serological tests in those patients. Patients and methods: Thirty-five IBS patients fulfilling Rome II criteria with absence of intestinal helminthic infection by direct smear method and no history of associated allergic conditions were studied. Ten healthy controls were included. Microscopic examination of stools for intestinal parasites, eosinophilic count and erythrocyte sedimentation rate were done. Colonoscopy was performed to rule out inflammatory bowel changes. Serum levels of IL-5 and ECP were measured. Results: Intestinal parasitic infection was present in 37% (13/35) of IBS patients vs. 20% (2/10) in controls. Of the 35 IBS patients, 13 (37%) had protozoal infection. Mean eosinophilic count, IL-5 level and ECP were significantly higher in IBS patients than in controls. Eosinophilic count and ECP serum level were significantly high in IBS patients with parasitic infection. Conclusion: A significant number of patients with symptoms suggestive of IBS demonstrated evidence of parasitic infection in their stool samples. The IL-5 serum level, eosinophilic count and ECP serum level might be useful tests for detecting parasitic infection aetiology in IBS patients after exclusion of conditions inducing the Th2 response. Larger case-controlled studies are required to clearly define the parasitic pathophysiology in IBS.

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