Indian Journal of Dermatology

ORIGINAL ARTICLE
Year : 2019 | **Volume** : 64 | **Issue** : 5 | **Page** : 355--359

Atopic dermatitis in adults and irritable bowel syndrome: A cross-sectional study

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Abstract

Background: Irritable bowel syndrome (IBS) is a chronic functional gastrointestinal disorder affecting a large number of people in the world. Atopic dermatitis (AD) is a common inflammatory skin condition characterized by relapsing eczematous lesions in a typical distribution. It was first described in 1933 but exists since antiquity. **Aim and Objectives:** To determine the relationship between AD and IBS. **Materials and Methods:** A total of 109 patients with AD and 100 healthy controls were included in the study. They were defined for diagnosis of IBS according to ROME-III diagnostic criteria. Supporting findings, Bristol stool scale, frequency of defecation and history of AD and IBS were also evaluated. AD severity was assessed using Severity Scoring of Atopic Dermatitis index. **Results:** A total of 62 patients (56.9%) in the AD group and 28 patients (28%) in the control group were diagnosed with IBS ($P <$ 0.001). Supportive findings excluding abnormal stool frequency and passage of mucus were more frequent in AD patients. There was no significant relationship between disease severity according to SCORAD index and variables in AD patients. **Conclusions:** This is a rather uncultivated area in the field of AD. We observed that IBS was more common in AD group. Also, supporting findings like abnormal stool form, abdominal distension, feeling of incomplete evacuation, and straining were found more frequently in AD patients. These results may indicate the association between AD and IBS. In our opinion, patients with atopic dermatitis should be questioned in terms of IBS.

How to cite this article:
Kaya İslamog˘lu Z G, Unal M, Küçük A. Atopic dermatitis in adults and irritable bowel syndrome: A cross-sectional study.Indian J Dermatol 2019;64:355-359

How to cite this URL:
Kaya İslamog˘lu Z G, Unal M, Küçük A. Atopic dermatitis in adults and irritable bowel syndrome: A cross-sectional study. Indian J Dermatol [serial online] 2019 [cited 2021 Jul 6];64:355-359
Available from: https://www.e-ijd.org/text.asp?2019/64/5/355/265944

Full Text

Introduction

Irritable bowel syndrome (IBS) is a functional gastrointestinal disorder characterized by chronic abdominal pain or abdominal discomfort without an organic cause.[1] It affects about 15%–20% of the world population.[2] Symptoms typically occur in the 30–50 year-age range. The IBS prevelance falls with advancing age. It is more common in women.[3] Although the etiology of IBS has not been fully understood, it may result from a combination of genetic and environment factors, changes in the

gastrointestinal sensory and motor functions.[2] On the other hand, low-grade inflammation is also considered an important factor in the pathogenesis of IBS.[4] In a study, elevated serum inflammatory cytokines were detected in IBS patients compared with controls. This result shows a potential relationship between systemic inflammation and IBS.[5]

Atopic dermatitis (AD) is a common, chronic, relapsing and inflammatory skin disorder. It is usually seen under 5 years of age even though it may affect all ages.[6] Recent studies have shown that the frequency of adult AD is higher than previously thought.[7] AD results from the interaction between environmental and genetic factors. The clinical presentation of AD includes pruritus, xerosis, and eczematous lesions, also tendency to other atopic and allergic conditions including asthma, atopic rhinitis, sensitivity to irritants and allergens and Immunoglobulin E (IgE) mediated systemic manifestations.[8] Diagnostic criteria of AD are mainly based on the clinical findings.[6] Langerhans cells, other antigen presenting dendritic cells, monocytes/macrophages, lymphocytes, eosinophils, mast cells/basophils, and keratinocytes are the major cell types that take an active role in the immune regulation of AD.[9]

The skin, respiratory, and gastrointestinal tract are the three systems through which the human body interacts with the external environment. In spite of the fact that mast cells are present in many tissues, they are especially present in the skin, respiratory, and gastrointestinal tract. Although the role of mast cells in skin and respiratory diseases is well known, its role in gastrointestinal tract diseases is not well studied.[10] Many studies with IBS patients have found increased number of mast cells in the luminal mucosa.[11],[12] The density of mast cells in the intestinal system is significantly correlated with the severity of anxiety and depression. And this psychological changes can aggravate IBS symptoms in patients.[13] It has also been known for a long time that the prevalence of allergic conditions or allergic diseases in IBS patients is increased.[10],[14],[15] Mast cells and inflammation play a role both in AD and in IBS. Thus, there may be an association between AD and IBS. There is only one study done in children evaluating the relation between AD and IBS in the literature.[16] Here, we investigated the adult patients with AD for IBS frequency and the relationship between IBS and AD severity.

Materials and Methods

This study has been approved by the Institutional Review Board (No: 2017/927) and informed consent was obtained from all participants. A total of 109 patients clinically diagnosed with AD were included in this study. In the clinical diagnosis, we used Hanifin and Rajka criteria. Healthy volunteers who did not have AD or any other dermatologic disease were used as the control group. Patients or controls with any gastrointestinal disease (except IBS) were excluded from the study. The demographic features were recorded for all subjects. Severity of AD was calculated using Severity Scoring of Atopic Dermatitis Index (SCORAD Index).[17] Also, duration and family history of AD were noted. Diagnosis of IBS was made according to the ROME-III diagnostic criteria.[18] Supportive findings including abnormal stool frequency, abnormal stool form, passage of mucus, abdominal distension, feeling of incomplete evacuation, and straining were asked to the patients and controls. Also, they identified fecal shape and consistency according to the Bristol stool form scale. Participants with alarming findings were not included in the study.[18] Frequency of defecation and "is there an association between AD and IBS episodes?'' were elicited.

The data obtained in the study were analyzed using SPSS (Statistical Package for Social Sciences) for Windows 22.0 program. Number, percentage, mean, and standard deviation were used as descriptive statistical methods in the evaluation of the data. The t-test was used to compare quantitative continuous data between two independent groups. Pearson correlation analysis was applied among the continuous variables of the study. The findings were evaluated at the 95% confidence interval and at the 5% significance level.

Results

A total of 109 patients (73 females - 67.0% and 36 males - 33.0%) with AD and 100 healthy controls (42 females - 42.0% and 58 males - 58.0%) were included in the study. The mean age of the patient group (35.24) was higher than that of the control group (27.22). The mean "duration of AD" of the cases was determined as 7.78±11.13 months (min = 0.5, max = 60). And sixty two patients (56.9%) in AD group and 28 patients (28%) in control group were diagnosed with IBS according to Roma III diagnostic criteria and this difference was statistically significant (P <0.001). Abnormal stool form,

abdominal distension, feeling of incomplete evacuation, and straining which are supportive findings were more frequent in the AD group but abnormal stool frequency and passage of mucus were similar between two groups [Table 1]. Distribution of stool patterns according to Bristol stool form scale of participants is shown in [Table 2]. In AD group, 11 (10.1%) were 1, 10 (9.2%) were 2, 23 (21.1%) were 3, 41 (37.6%) were 4, 2 (1,8%) were 5,20 (18.3%) were 6, 2 (1.8%) were 7; in healthy controls, 4 (4%) were 1, 5 (5%) were 2, 25 (25%) were 3, 50 (50%) were 4, 4 (4%) were 5, 7 (7%) were 6 and 5 (5%) were 7. The family history of AD patients and history of IBS in both groups can be seen in [Table 1]. There was no significant relationship between the groups and family history. The association between AD and IBS episodes was not significant. There were 98 of patients (89.9%) said ''no''and 11 of patients (10.1%) said ''yes''.{Table 1}{Table 2}

When analyzing the correlation between SCORAD index, age, Bristol stool form, defecation frequency, duration of AD; there was a correlation between duration of the disease and age ($r = 0.272$) ($P = 0.004$). Relations between other variables were not statistically significant ($P > 0.05$) [Table 3].{Table 3}

There was no significant relationship between disease severity according to SCORAD index and variables (frequency of IBS, abnormal stool frequency, abnormal stool form, passage of mucus, abdominal distension, feeling of incomplete evacuation and straining) in AD patient group ($P >0.05$).

Discussion

To our knowledge, there is no study examining the frequency of irritable bowel syndrome in adult AD and relationship with disease severity. Our data demonstrate that IBS was more common in AD group than in healthy controls. Supportive findings excluding abnormal stool frequency and passage of mucus were more common in AD patients. But there was no association between frequecy of IBS and supporting finding with SCORAD index. According to Bristol stool form scale, AD patients tend to have stiffer stool.

Irritable bowel syndrome is a functional gastrointestinal disorder that occurs without any organic cause. It is characterized by chronic or recurrent abdominal pain associated with either relief or exacerbation by defecation, or a change in bowel habit. In routine clinical practice, a diagnosis of IBS is made on the basis of typical symptoms.[1] A selected panel of tests can be used to exclude known organic diseases such as inflammatory bowel disease or celiac disease that present with similar symptoms.[19] In our study, we used ROME-III criteria and supporting findings for diagnosis.[18]

The pathogenesis of the disease has not been clearly understood, and several factors, such as psychosocial stress, alteration of serotonin signaling, mast cells, brain-gut axis, low-grade inflammation, microbiota, barrier dysfunction on mucosal surfaces, and immune reaction to food are thought to be related to this condition.[20] There are some findings that atopic conditions or allergic diseases are more common in functional bowel diseases.[10],[11],[12],[13],[14],[15],[16] The role of allergies and mast cells in IBS is being increasingly comprised.[10],[15] Shalom et al. and Pearson et al. described two cases of positive clinical responses to omalizumab in patients with IBS with diarrhea and concomitant chronic spontaneous urticaria and asthma.[15],[21] Also, Shalom et al. showed that IBS increases in chronic urticaria patients (1.7% in urticaria and 0.8% in the control group). One thousand and one hundred of these chronic urticaria patients were AD and 6.9% of AD patients had IBS.[10] Mast cell stabilizers such as ketotifen reduce visceral hypersensitivity in IBS patients and we can understand the important role of mast cells from here.[15],[22]

In a study by Ekiz et al., they investigated the relationship between chronic pruritus of undetermined origin and IBS. They found the frequency of IBS was higher in patients with chronic pruritus than in healthy controls. The interactions of stress, biological, and environmental factors are important in the association they inferred.[23]

Inflammation is also considered an important factor in the pathogenesis of both AD and IBS. Elevated serum inflammatory cytokines, such as IL-6, IL-8, and tumor necrosis factor alpha (TNF-α), and decreased serum anti-inflammatory cytokines, such as IL-10, were detected in IBS patients compared with controls.[5],[24],[25] This result can represent the relationship between IBS and AD.

Importance of the role of food in IBS generally has been given falling preference. But, IgE-mediated food allergy has been suggested in a subset of patients with diarrhea predominant IBS and a history of atopy. In a study, they applied skin patch test of food panels to IBS patients and showed at least 1 doubtful or positive patch test results. They said food-related type-4 hypersensitivity reactions may

conduce the pathogenesis of IBS.[26] IgE-mediated food allergies have a clear association with AD. Adult AD patients have a significantly higher risk of food sensitization than adults without AD.[27] This may be another entity describing the association of AD and IBS and can also support our study results. Again, an 8-year population-based cohort study with childhood AD, they said AD children had a greater risk of developing IBS.[16]

The limitations of this study include the small number of subjects and the patient selection does not reflect general population. Besides this, our study was not a prospective study. In addition to IBS frequency, supplementary findings such as supporting findings, the Bristol stool scale, weekly stool frequency, family history were questioned and compared with controls. Also, the relationship between AD severity and IBS was investigated.

In summary, the relationship between adult AD and IBS were not worked before. Also the other studies with IBS did not show how various characteristics of two diseases (severity of AD, history of IBS and AD, number of defecation, findings supporting IBS diagnosis, etc.) affect each other. We found a strong association between AD and IBS in adults. Further prospective researches with larger numbers of the patients are needed to confirm the relation.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1 Hungin AP, Whorweel PJ, Tack J, Mearin F. The prevelance, patterns and impact of irritable bowel syndrome: An international survey of 40.000 subjects. Aliment Pharmacol Ther 2003;17:643-50.
2 Longstreth GF, Wilson A, Knight K, Wong J, Chiou CF, Barghout V, *et al*. Irritable bowel syndrome, health care use, and costs: A US managed care perspective. Am J Gastroenterol 2003;98:600-7.
3 Houghton LA, Heitkemper M, Crowell MD, Emmanuel A, Halpert A, McRoberts JA, *et al*. Age, gender, and women's health and the patient. Gastroenterology 2016;150:1332-43.
4 Barbara G, Cremon C, Annese V, Basilisco G, Bazzoli F, Bellini M,*et al*. Randomised controlled trial of mesalazine in IBS. Gut 2016;65:82-90.
5 Choghakhori R, Abbasnezhad A, Hasanvand A, Amani R. Inflammatory cytokines and oxidative stress biomarkers in irritable bowel syndrome: Association with digestive symptoms and quality of life. Cytokine 2017;93:34-43.
6 Spergel JM, Paller AS. Atopic dermatitis and the atopic march. J Allergy Clin Immunol 2003;112:S118-27.
7 Silverberg JI. Public health burden and epidemiology of atopic dermatitis. Dermatol Clin 2017;35:283-9.
8 Diepgen TL. Atopic dermatitis: The role of environmental and social factors, the European experience. J Am Acad Dermatol 2001;45:S44-8.
9 Taylor RS, Baadsgaard O, Hammerberg C, Cooper KD. Hyperstimulatory CD1a+CD1b+CD36+Langerhans cells are responsible for increased autologous T lymphocyte reactivity to lesional epidermal cells of patients with atopic dermatitis. J Immunol 1991;147:3794-802.
10 Shalom G, Magen E, Babaev M, Horev A, Freud T, Ben Yakov G, *et al*. Chronic urticaria and irritable bowel syndrome: A cross-sectional study of 11 271 patients. Br J Dermatol 2018;178:e204-6. doi: 10.1111/bjd.15997.
11 Cremon C, Gargano L, Morselli-Labate AM, Santini D, Cogliandro RF, De Giorgio R, *et al*. Mucosal immune activation in irritable bowel syndrome: Gender-dependence and association with digestive symptoms. Am J Gastroenterol 2009;104:392-400.
12 Walker MM, Talley NJ, Prabhakar M, Pennaneac'h CJ, Aro P, Ronkainen J, *et al*. Duodenal mastocytosis, eosinophilia and intraepithelial lymphocytosis as possible disease markers in the irritable bowel syndrome and functional dyspepsia. Aliment Pharmacol Ther 2009;29:765-73.
13 Piche T, Saint-Paul MC, Dainese R, Marine-Barjoan E, Iannelli A, Montoya ML, *et al*. Mast cells and cellularity of the colonic mucosa correlated with fatigue and depression in irritable bowel syndrome. Gut 2008;57:468-73.
14 Cole JA, Rothman KJ, Cabral HJ, Zhang Y, Farraye FA. Incidence of IBS in a cohort of people with asthma. Dig Dis Sci 2007;52:329-35.
15 Pearson JS, Niven RM, Meng J, Atarodi S, Whorwell PJ. Immunoglobulin E in irritable bowel

syndrome: Another target for treatment? A case report and literature review. Therap Adv Gastroenterol 2015;8:270-7.
16 Tsai JD, Wang IC, Shen TC, Lin CL, Wei CC. An 8-year population-based cohort study of irritable bowel syndrome in childhood with history of atopic dermatitis. J Investig Med 2018;66:755-61.
17 Oranje AP, Stalder JF, Taïeb A, Tasset C, de Longueville M. Scoring of atopic dermatitis by SCORAD using a training atlas by investigators from different disciplines. ETAC study group. Early treatment of the atopic child. Pediatr Allergy Immunol 1997;8:28-34.
18 Can G, Yılmaz B. Approaches in the diagnosis and treatment of irritable bowel syndrome. Güncel Gastroenteroloji Dergisi 2015;19:171-81.
19 Pimentel M, Talley NJ, Quigley EM, Hani A, Sharara A, Mahachai V. Report from the multinational irritable bowel syndrome initiative 2012. Gastroenterology 2013;144:e1-5.
20 Saha L. Irritable bowel syndrome: Pathogenesis, diagnosis, treatment, and evidence-based medicine. World J Gastroenterol 2014;20:6759-73.
21 Magen E, Chikovani T. Case report of irritable bowel syndrome responding to omalizumab. Georgian Med News 2015;243:42-5.
22 Jones MP, Walker MM, Ford AC, Talley NJ. The overlap of atopy and functional gastrointestinal disorders among 23,471 patients in primary care. Aliment Pharmacol Ther 2014;40:382-91.
23 Ekiz Ö, Balta I, Özug˘uz P, Şen BB, Rifaiog˘lu EN, Ekiz F, *et al*. Irritable bowel syndrome in patients with chronic pruritus of undetermined origin. J Eur Acad Dermatol Venereol 2014;28:1034-9.
24 Seyedmirzaee S, Hayatbakhsh MM, Ahmadi B, Baniasadi N, Bagheri Rafsanjani AM, Nikpoor AR, *et al*. Serum immune biomarkers in irritable bowel syndrome. Clin Res Hepatol Gastroenterol 2016;40:631-7.
25 Bashashati M, Rezaei N, Shafieyoun A, McKernan DP, Chang L, Öhman L,*et al*. Cytokine imbalance in irritable bowel syndrome: A systematic review and meta-analysis. Neurogastroenterol Motil 2014;26:1036-48.
26 Stierstorfer MB, Sha CT, Sasson M. Food patch testing for irritable bowel syndrome. J Am Acad Dermatol 2013;68:377-84.
27 Manam S, Tsakok T, Till S, Flohr C. The association between atopic dermatitis and food allergy in adults. Send to Curr Opin Allergy Clin Immunol 2014;14:423-9.