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Gut Microbiome in Atopic Dermatitis Patients

ERENY A. SAMAAN, M.Sc.*; EMAN M. ABD EL GAYED, Ph.D.** and AZZA G. FARAG, Ph.D.*

The Department of Dermatology, Andrology & STIs and Medical Biochemistry & Molecular Biology Department**, Faculty of Medicine, Menoufia University*

Abstract

Background: Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by recurrent eczematous skin lesions and intense itching. Worldwide, it affects 10-30% of children and 2-10% of adults, with a two- to three-fold increase in prevalence observed over the last several decades. Recent advances in sequencing technology have demonstrated that the development of AD not only associate with the skin microbiome but gut microbiota. Gut microbiota plays an important role in allergic diseases including AD. The hypothesis of the "gut-skin" axis has been proposed and the cross-talk mechanism between them has been gradually demonstrated in the researches.

Aim of Study: The aim of this work is to identify the specific patterns of gut microbiome in moderate and severe clinical severities of atopic dermatitis inpatients >3 years old.

Patients and Methods: Randomized-controlled study was carried out on 20 patients >2 years old with moderate to severe AD assessed by SCORAD index in addition to a control group of 20 healthy subjects without personal or family history of any atopic diseases to identify the specific patterns of gut microbiome in moderate and severe clinical severities of AD at Dermatology Clinic at Menoufia University Hospitals from March 2022 to May 2023.

Results: There were no statistically significant differences between severe and moderate forms of disease as regards age (p=0.077) and age of onset (p=0.482). While, there was a highly statistically significant difference between moderate and severe cases as regards duration of disease in years (p=0.005).

There was a high statistically significant difference between different study groups as regards alpha diversity of the gut microbiome (p<0.01).

There was no significant correlation between alpha diversity of gut microbiome and age of onset, duration, severity and sites of disease lesions.

Conclusion: Our study suggests that gut microbiota in patients with AD showed lower alpha diversity than healthy control subjects and supports the hypothesis that low microbial diversity is associated with an increased risk for allergic disease as AD.

Recommendations: There is a need for a profound understanding of the interactive mechanism between intestinal microbiota and host immune system. Large cohort study to detect the specific role of micro organisms in the pathogenesis and development of AD.

Key Words: *Atopic Dermatitis – Gut Microbiome – Children.*

Correspondence to: Dr. Ereny A. Samaan,
E-Mail: yoyobobo85@gmail.com

Introduction

ATOPIC dermatitis is the most common chronic inflammatory skin disease characterized by characterized by pruritus and rash, and is one of the most common skin disorders in children **[1].** AD starts in early childhood and is usually the first manifestation of the atopic march, progressing to asthma, allergic rhinitis, and allergic conjunctivitis **[2].**

The incidence and prevalence of AD have increased over the past several decades. It affects up to one fifth of the population in developed countries. Infants are particularly highly susceptible, as AD affects almost 15-20% of children in developed countries. However, AD is also becoming increasingly prevalent among adults **[3].**

Pathophysiology of AD is very complex and not yet completely elucidated. Multiple contributing factors, including epidermal barrier impairment, immune dysregulation and alteration of skin microbiota, contribute to the disease. The integration of these factors, with their different intensities and combinations, is thought to cause the varying clinical presentations of AD **[4].**

The AD lesions are changed in morphology and distribution as patients continue to age. In infancy, infantile seborrheic dermatitis is a common eruption on the face and scalp. In young children, dry skin is overt as represented by goose flesh-like skin, and eczema occurs on the antecubital and popliteal fossae. In adults, lichenified lesions are prominent, and patients may develop red face, representing

persistent dark reddish erythema on the face, and dirty neck, exhibiting poikilodermatous reticulate lesions on the neck **[5]**. Over the past few years, an increasing body of evidence has suggested that gut microbiota is involved in the regulation of a wide range of physiological processes, such as cell proliferation, tissue morphogenesis, and metabolism and immune development and function **[6]**. It is well established that the microbiota plays a central role in modulating both the innate and adaptive arms of the immune response **[7]**.

Patients and Methods

We conducted a randomized-controlled study was carried out on 20 patients >3 years old with moderate to severe AD assessed by SCORAD index in addition to a control group of 20 healthy subjects without personal or family history of any atopic diseases to identify the specific patterns of gut microbiome in moderate and severe clinical severities of AD.

Ethical considerations:

All procedures were carried out in accordance with the ethical standards of the institutional and/or national research committee. The study received the approval by the Committee of Human Rights in Research in Menoufia University (No. 6/2021DERMA). Written consents from patients' guardians were taken after explaining the aim of study.

Inclusion criteria:

Include: (1) Atopic dermatitis patients >2 years old (By 36 months after birth, the baby's gut microbiota undergoes changes to become a stable system, following which 60-70% of microbiota remains unchanged throughout life) **[8]**. (2) Atopic dermatitis as defined by the Hanifin and Rajka criteria **[9]**. (3) Moderate to severe activity of atopic dermatitis as assessed by SCORAD index.

Exclusion criteria:

Include: Any participant had one or more of the following was excluded from the study: (1) Gastrointestinal disorders in the four weeks before enrolment and during the follow-up. (2) Recent use of any drug that can affect fecal microbiota diversity (steroids or calcineurin inhibitors, systemic immunosuppressant or antibiotics in the last four weeks before enrolment and during the follow-up period) **[10]**.

A total of 40 participants were divided into two equal groups, AD group (20, 12 severe and 8 moderate cases) and the control group (20). All participants included in our study were subjected to the following: (1) Full history taking (personal history as name, age, sex, residence; present history as onset, course, and duration; family historyof AD or other allergic diseases and past history of other manifestations of atopy as bronchial asthma and allergic rhinitis). (2) Clinical examination: General examination for all and dermatological examination of AD patients to determinedryness, itching, oozing, crusting thickened skin, darkening of the skin around the eyes and assess severity of AD area by scoring atopic dermatitis (SCORAD) index **[11]**. (3) Laboratory investigations: Fecal analysis for detection of microbiome patterns was done by using 16S rRNA sequencing (fecal samples were collected from all patients during clinical visits and healthy control subjects, then stored at –80°C) **[10]**. The 2 primers used were 165 Amplicon (PCR Forward Primer=5`TCGTCGGCAGCGTCAGATGTGTATAAGAGACAGCCTACGGGNGGCWGCAG and 16S Amplicon PCR Reverse Primer= 5`GTCTCGTGGGCTCGGAGATGTGTATAAGAGACAGGACTACHVGGGTATCTAATCC.

Statistical analysis:

Data entry, coding, and analysis were conducted using PSW (20), IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp. Data of this study were of both quantitative and qualitative types. Quantitative data were expressed in Mean ($\bar{x}$), and Standard Error (SEM), while qualitative data were expressed in frequency (number), and percent (%). Tests of significance used were: Student *t*-test, Chi Square test, ANOVA (Analysis of Variance) test and Post Hoc Test. The level of significance of our data was 95%, so, *p*-value ≥0.05 was considered a statistically significant difference.

Results

The mean age of the studied groups (cases & controls) was (4.71 &4.35, p=0.38), respectively, AD patients were 55% males and 45% females & controls were 35% males and 65% females (p=0.195), 65% of patients were urban residents & 35% were rural. Controls were 75% urban residents & 25% rural (p=0.773) with no statistically significant difference between studied groups (Table 1).

Table (1): Comparison of the demographic characteristics of the participants.

Studied Variable	Cases (N=20)	Controls (N=20)	*t*-test	*p*-value
Age:				
Mean ± SD	4.71±2.033	4.35±1.53	0.87	0.38
Median	4.5	4		
Range	2-9	2-7		
Gender:	No (%)	No (%)	$\chi 2$	0.195
Male	11 (55.0%)	7 (35.0%)	3.26	
Female	9 (45.0%)	13 (65.0%)		
Residence:				
Urban	13 (65.0%)	15 (75.0%)	$\chi 2$	0.773
Rural	7 (35.0%)	5 (25.0%)	0.520	

%: Percentage. SD: Standard deviation.
N: Number. $\chi 2$: Chi square.
t : Student *t*-test.

All AD patients had gradual onset of disease. Mean age of onset of AD was 2.333±0.142. Mean duration was 2.181±0.427 years. Chronic AD was reported in 40% of patients and relapsing in 60% of patients. 70% of patients had positive family history of AD. Regarding disease severity, 40% of cases were moderate and 60%were severe. Regarding the sites of AD lesions, the majority of the patients had anterior trunk (95%) and (90%) face lesion. Concerning precipitation factors, wool clothes (80%) and cold (70%) were the most aggravating factors of AD (Table 2).

Table (2): Clinical data of studied patients (N=20).

Variables		No. (%)
Onset	Gradual	20 (100%)
Course	Chronic	8 (40%)
	Relapsing	12 (60%)
Age of onset in years	Mean ± SD	2.333±0.142
	Range	2 - 4
Duration in years	Mean ± SD	2.181±0.427
	Range	1 - 6
Severity	Severe	12 (60%)
	Moderate	8 (40%)
Site	Face	18 (90%)
	Extensor's	9 (45%)
	Anterior trunk	19 (95%)
	Posterior trunk	14 (70%)
	Flexor's	14 (70%)
Precipitation	Cold	14 (70%)
	Wool clothes	16 (80%)
	Certain food	7 (35%)
Family history	Positive	14 (70%)
	Negative	6 (30%)

SD: Standard deviation. No: Number. %: Percent.

There were no statistically significant differences between severe and moderate forms of disease as regards age (p=0.077) and age of onset (p=0.482). While, there was a highly statistically significant difference between moderate and severe cases as regards duration of disease in years (p=0.005), patients with moderate form of the disease showed longer duration than severe form (Fig. 1).

Total number of operational taxonomic unit (OTU) in control group was 40, while in moderate cases were 48, and in severe cases were 72, with a total number of OTU 160. There was no significant statistical difference at the composition of OTU between groups (p>0.05). There was identical homogenous composition of OTU among study participants (Table 3).

There was a high statistically significant difference between different study groups as regards alpha diversity of the gut microbiome (p<0.01). The Shannon index was lower in AD patients, whilst the Simpson index of diversity was higher in patients with AD (p<0.01). These results suggest that patients with AD had a lower alpha diversity than healthy control subjects (Table 4).

Regarding alpha diversity, Chao index (species richness) was higher in control (1380.00±8.167) than severe (851.56±31.973) than moderate cases (466.00±60.06). Shannon index (species diversity) also was higher in control (6.7932±0.00951) than severe (5.7925±0.061515) than moderate cases (4.89586±0.14246). But Simpson index; its negative value correlation with microbial diversity, was higher in moderate (0.01543±0.00143) than severe (0.0067±0.000536) than control subjects (0.001545±0.000013) (Table 4).

There was no significant correlation between alpha diversity of gut microbiome and age of onset, duration, severity and sites of disease lesions (Table 5).

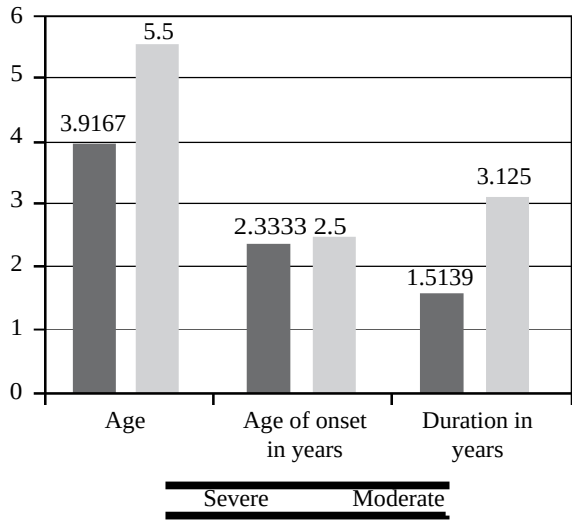

Fig. (1): Mean age, AD age of onset and duration among severe versus moderate AD cases.

Table (3): Number of Common / Unique OTU among different study groups.

OTU	Studied groups					
	Controls n=20	Cases (n=20)			χ^2	*p*-value
		Total	Moderate n=8	Severe n=12		
	No. (%)	No. (%)	No. (%)	No. (%)		
ASV1	2 (5%)	5 (5%)	1 (2.1%)	5 (6.9%)		
ASV2	2 (5%)	5 (5%)	1 (2.1%)	5 (6.9%)		
ASV3	2 (5%)	5 (5%)	1 (2.1%)	5 (6.9%)		
ASV4	2 (5%)	5 (5%)	2 (4.2%)	4 (5.6%)		
ASV5	2 (5%)	5 (5%)	4 (8.3%)	2 (2.8%)		
ASV6	2 (5%)	5 (5%)	3 (6.2%)	3 (4.2%)		
ASV7	2 (5%)	5 (5%)	3 (6.2%)	3 (4.2%)		
ASV8	2 (5%)	5 (5%)	1 (2.1%)	5 (6.9%)		
ASV9	2 (5%)	5 (5%)	3 (6.2%)	3 (4.2%)		
ASV10	2 (5%)	5 (5%)	3 (6.2%)	3 (4.2%)		
ASV11	2 (5%)	5 (5%)	4 (8.3%)	2 (2.8%)		
ASV12	2 (5%)	5 (5%)	2 (4.2%)	4 (5.6%)		
ASV13	2 (5%)	5 (5%)	1 (2.1%)	5 (6.9%)	0.00	1.00
ASV14	2 (5%)	5 (5%)	3 (6.2%)	3 (4.2%)		
ASV15	2 (5%)	5 (5%)	2 (4.2%)	4 (5.6%)		
ASV16	2 (5%)	5 (5%)	2 (4.2%)	4 (5.6%)		
ASV17	2 (5%)	5 (5%)	3 (6.2%)	3 (4.2%)		
ASV18	2 (5%)	5 (5%)	2 (4.2%)	4 (5.6%)		
ASV19	2 (5%)	5 (5%)	2 (4.2%)	4 (5.6%)		
ASV20	2 (5%)	5 (5%)	5 (10.4%)	1 (1.4%)		
Total	40	120	48	72		

OTU: Operational Taxonomic Unit. ASV: Amplicon Sequence Variant. %: Percentage. $\chi 2$: Chi square.

Table (4): Alpha diversity of the gut microbiome among different study groups.

	Controls N=40 Mean±SE	Cases		ANOVA	*p*-value	LSD
		Moderate N=48 Mean±SE	Severe N=72 Mean±SE			
Abundance	0.00076±0.00011	0.00872±0.002369	0.00841±0.001435	6.145	0.003*	p_1<0.001 p_2<0.001 p_3=0.86
Abundance 100	0.07607±0.01077	0.87212±0.236848	0.84099±0.14347	6.145	0.003*	p_1<0.001 p_2<0.001 p_3=0.740
Chao	1380.00±8.167	851.56±31.973	466.00±60.06	106.45	0.000*	p_1<0.001 p_2<0.001 p_3=0.001
Simpson	0.001545±0.000013	0.0067±0.000536	0.01543±0.00143	57.092	0.000*	p_1<0.001 p_2<0.001 p_3=0.001
Shannon	6.7932±0.00951	5.7925±0.061515	4.89586±0.14246	94.52	0.000*	p_1<0.001 p_2<0.001 p_3=0.67

*ANOVA: Analysis of Variance.
OTU: Operational Taxonomic Unit.
* : Significant.

p1: Control group vs Moderate cases.
p2: Control group vs Severe cases.
p3: Moderate cases vs Severe cases.

Table (5): Correlation between alpha diversity of gut microbiome and age of onset, duration, severity and sites of disease lesions.

Variables	Abundance	Abundance 100	Chao	Simpson	Shannon
Age of onset years:					
r	–0.218	–0.218	0.131	–0.266	0.221
p-value	0.356	0.356	0.583	0.257	0.349
Duration years:					
r	0.141	0.141	–0.132	0.08	–0.102
p-value	0.552	0.552	0.578	0.738	0.669
Severity:					
r	–0.111	–0.111	–0.117	0.083	–0.096
p-value	0.64	0.64	0.622	0.729	0.689
Face Site:					
r	0.386	0.386	–0.374	0.326	–0.363
p-value	0.093	0.093	0.104	0.161	0.115
Extensors Site:					
r	0.048	0.048	0.164	–0.048	0.095
p-value	0.841	0.841	0.489	0.84	0.691
Anterior trunk Site:					
r	0.235	0.235	–0.257	0.224	–0.25
p-value	0.319	0.319	0.273	0.342	0.288
Posterior trunk Site:					
r	–0.008	–0.008	–0.09	0.16	–0.131
p-value	0.972	0.972	0.704	0.502	0.582
Flexors Site:					
r	0.1	0.1	–0.035	–0.071	0.029
p-value	0.673	0.673	0.883	0.765	0.904

r: Pearson correlation.

Discussion

Atopic dermatitis is a chronic inflammatory skin disease characterized by recurrent eczematous skin lesions and intense itching. Worldwide, it affects 10–30% of children and 2-10% of adults, with a two- to three-fold increase in prevalence observed over the last several decades *[12]*.

The gut is the most important source of postnatal microbial stimulation of the immune system. Atopic children may have a different gut microbiome compared with their nonatopic peers. Although it has been claimed a gap of information in school-age children as microbiome studies are concentrated in early infants and adults *[13]*, differences have been found between cases of eczema and healthy controls as well as between countries with a high and low incidence of atopic diseases *[14]*.

In this study, the mean age in AD patients was 4.71 years. 55% were males and 45% were females; 65% were urban residents and 35% were rural residents. Similarly, in Egypt, the mean age of the studied children was 3.90±1.81 years. 56% of the studied children came from urban areas *[15]* and male predominance *[16]*. However, a female predominance of AD (58% of the cases) was quite apparent in a study by Abdel-Maguid et al. *[15]*. It is quite probable that the presence of the disease in female children leads to more anxiety and distress due to the parents' worry over problems in marriage and post marriage relationships *[15]*.

In agreement with our findings, Abdel-Maguid et al. *[15]* reported a similar result including a positive high family history of AD (86%), the age of onset ranged between 1 and 8 years, with mean age within 1.65±1.21 years, while the mean duration of disease was 2.58±1.29 years, and the majority (67%) of the children had severe dermatitis. Regarding the sites of AD lesions, that most of the children had eczema in the face (89.4%), followed by the flexural sites (78.8%) and to less extent in the extensor surfaces (44.7%) in Egypt *[16]*.

In consistence with our findings as regard precipitation factors, cold and dry weather conditions are known to increase the risk of flares in AD by decreasing skin barrier function and increasing susceptibility towards mechanical stress. Skin becomes more reactive towards irritants and allergens as pro-inflammatory cytokines, and cortisol are released by keratinocytes, and the number of dermal mast cells increases *[17]*. Additionally, seasonal changes, urbanization, and woolen cloths were principal causative and exacerbating factors for AD *[18]*.

Similarly, Ye et al. *[19]* revealed that the OTU results showed that patients with AD had greater component similarity than healthy controls and in terms of the alpha diversity index, no significant differences were observed between the two groups (AD cases and healthy controls). These results suggest that patients with AD had a lower alpha diversity than healthy control subjects.

Our results were consistent with previous findings of low intestinal microbial diversity in AD *[10,20,21,22]*, which supports the theory of 'microbial deprivation syndromes of affluence *[23]*. According to this theory, reduced intensity and diversity of microbial stimulation led to an abnormal immune maturation in early childhood. In fact, limited microbial pressure results in insufficient Th1 cell induction and the failure to suppress Th2 responses. The switching of the immune stimulation towards a pronounced Th2-phenotype is suggested to be a major mechanism to explain allergy development and maintenance *[23,24]*.

Likewise, in terms of the alpha diversity index, the Shannon index was lower in patients with AD, whilst the Simpson index of diversity was higher in patients with AD, and thus being significantly different among the groups. These results suggest that patients with AD had a lower alpha diversity than healthy control group *[19]*.

The conflicting results may be explained by methodological differences, difficulties with isolation and identification of gut bacterial species, and the complexity of the interactions between the gut microbiota and external factors. Methods for detection of bacteria have evolved through the years and are much more sensitive compared with last years [25].

Our findings revealed that there was no significant correlation between alpha diversity of gut microbiome and age of onset, duration, severity and sites of disease lesions.

In the same context, there was no significant correlations were observed between eczema severity and the alpha diversity of the gut microbiota or the relative abundance according to Cheung et al. [26]. In addition, Chan et al. *[27]* reported that there was no significant correlation between the relative abundance of gut microbiota and severity of AD. On the other hand, another study has reported a lower alpha diversity in the gut microbiota of moderate and severe cases, suggesting an association between the severity of eczema and alpha diversity of the gut microbiota, at least at certain time points. Also, a negative correlation between Shannon diversity and eczema severity at 12 months of age was reported *[28]*. A similar negative correlation has been reported at 6 months of age *[20]*. The difference between our findings and the other studies may be attributed to the age variation among the participants.

Conclusion: Our study suggests that gut microbiota in patients with AD showed lower alpha diversity than healthy control subjects and supports the hypothesis that low microbial diversity is associated with an increased risk for allergic disease as AD.

Recommendations: There is a need for a profound understanding of the interactive mechanism between intestinal microbiota and host immune system. Large cohort study to detect the specific role of microorganisms in the pathogenesis and development of AD.

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الميكروبيوم المعوى فى مرضى التهاب الجلد التأتبى

يعد مرض التهاب الجلد التأتبى مرض جلدى مزمن مصاحب بحكة جلدية وهو يصيب حوالى ١٠-٢٠٪ من الأطفال عالمياً. من بين المصابين ٦٠٪ تظهر عليهم الأعراض قبل بلوغ عام من عمرهم، و٨٥٪ تزداد حدة الأعراض فى أول خمس سنوات من العمر. يعتمد تشخيص التهاب الجلد التأتبى على مجموعة النتائج السريرية التالية: الحكة، أكزيما الوجه والباسطات عند الرضع والأطفال، الأكزيما فى الانثناءات عند البالغين والتهاب الجلد المزمن.

الهدف من الدراسة: هو تحديد الأنماط المحددة للميكروبيوم المعوى فى الحالات السريرية المتوسطة والشديدة لالتهاب الجلد التأتبى.

تم إجراء هذه الدراسة على ٢٠ مريضاً تزيد أعمارهم عن عامين مصابين بالتهاب الجلد التأتبى المتوسط إلى الشديد والذى تم تقييمه بواسطة مؤشر SCORAD بالإضافة إلى مجموعة مراقبة مكونة من ٢٠ شخصًا أصحاء ليس لديهم تاريخ شخصي أو عائلى لأى أمراض تأتبية بعيادة الأمراض الجلدية بمستشفيات جامعة المنوفية من مارس ٢٠٢٢ إلى مايو ٢٠٢٣.

وقد خلصت الدراسة إلى أن الميكروبيوم المعوى فى المرضى الذين يعانون من التهاب الجلد التأتبى أظهرت تنوعاً ألفا أقل من الأشخاص الأصحاء وتدعم الفرضية القائلة بأن التنوع الميكروبى المنخفض يرتبط بزيادة خطر الإصابة بأمراض الحساسية مثل التهاب الجلد التأتبى. ومع ذلك، دور بعض مسببات الأمراض فى تطور التهاب الجلد التأتبى يحتاج إلى إثبات فى دراسة أترابية كبيرة.