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ORIGINAL ARTICLE

Assessment of IgA anti-endomysial antibody levels in children with autism spectrum disorder

Otizm spektrum bozukluğu olan çocuklarda IgA anti-endomisial antikor düzeylerinin değerlendirilmesi

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ABSTRACT • **Background and Aims:** The relationship between celiac disease and autism spectrum disorder is contradictory. In this study, we aimed to investigate the serum immunoglobulin A anti-endomysial antibody levels of children with autism spectrum disorder in comparison with those of healthy participants. The association between gastrointestinal symptoms and immunoglobulin A anti-endomysial antibody levels was also examined. **Materials and Methods:** We have included two main groups as study and control groups in the present study. Eighty five children with autism spectrum disorder ranging in age from 2 to 15 years were selected for the study group and 81 healthy children were selected for the control group. Peripheral venous blood samples were collected between 9 am and 10 am. ELISA was used to assay serum immunoglobulin A anti-endomysial antibody levels. Autism behavior checklist was applied to the children in the study group. **Results:** The mean immunoglobulin A anti-endomysial antibody levels for the study group and the control group were 212.8 ± 110.8 ng/ml and 142.7 ± 98.4 ng/ml, respectively. Immunoglobulin A anti-endomysial antibody levels were significantly higher in the study group compared to the control group ($p < 0.001$). The frequency of gastrointestinal complaints (e.g. diarrhea or constipation) were significantly higher in the study group ($p = 0.01$). No statistically significant relationship was found between autism behavior checklist scores, gastrointestinal symptoms and immunoglobulin A anti-endomysial antibody levels ($p > 0.05$). Age was found to be negatively correlated with immunoglobulin A anti-endomysial antibody levels in the study group. **Conclusion:** Increased immunoglobulin A anti-endomysial antibody levels and gastrointestinal complaints in children with autism spectrum disorder suggest that it may be appropriate to evaluate autistic individuals with gastrointestinal symptoms related to gluten sensitivity for celiac disease. The exact relationship between celiac disease and autism spectrum disorder needs to be supported by duodenal biopsy.

Key words: Autism spectrum disorder, gastrointestinal symptoms, celiac disease, IgA anti-endomysial antibody

ÖZET • **Giriş ve Amaç:** Çölyak hastalığı ile otizm spektrum bozukluğu arasındaki ilişki çelişkilidir. Bu çalışmada, otizm spektrum bozukluğu olan çocukların serum anti-endomisyum immünglobulin A antikor düzeylerini sağlıklı çocuklarla karşılaştırmayı amaçladık. Gastrointestinal şikâyetler ile anti-endomisyum immünglobulin A antikor düzeyleri arasındaki ilişki de incelendi. **Gereç ve Yöntem:** Çalışma ve kontrol olarak iki grup mevcut çalışmaya dâhil edildi. 2 ila 15 yaş aralığında otizm spektrum bozukluğu olan 85 çocuk çalışma grubuna ve 81 sağlıklı çocuk kontrol grubuna seçildi. Periferik venöz kan örnekleri sabah 9 ile 10 arasında toplandı. Serum anti-endomisyum immünglobulin A antikor düzeylerini ölçmek için ELISA kullanıldı. Çalışma grubundaki çocuklara otizm davranış kontrol listesi uygulandı. **Bulgular:** Çalışma grubu ve kontrol grubu için ortalama anti-endomisyum immünglobulin A antikor düzeyleri sırasıyla 212.8 ± 110.8 ng/ml ve 142.7 ± 98.4 ng/ml idi. Anti-endomisyum immünglobulin A antikor düzeyleri çalışma grubunda kontrol grubuna kıyasla anlamlı derecede yüksekti ($p < 0.001$). Gastrointestinal şikâyetlerin (örneğin ishal veya kabızlık) sıklığı çalışma grubunda anlamlı derecede yüksekti ($p = 0.01$). Otizm davranış kontrol listesi puanları, gastrointestinal şikâyetler ve anti-endomisyum immünglobulin A antikor düzeyleri arasında istatistiksel olarak anlamlı bir ilişki bulunmadı ($p > 0.05$). Çalışma grubunda yaş ile anti-endomisyum immünglobulin A antikor düzeyleri arasında negatif korelasyon tespit edildi. **Sonuç:** Otizm spektrum bozukluğu olan çocuklarda artmış anti-endomisyum immünglobulin A antikor düzeyleri ve gastrointestinal şikâyetler, gluten hassasiyetiyle ilgili gastrointestinal semptomları olan otizmli bireylerin çölyak hastalığı açısından değerlendirilmesinin uygun olabileceğini düşündürmektedir. Çölyak hastalığı ile otizm spektrum bozukluğu arasındaki kesin ilişkinin duodenal biyopsi ile desteklenmesi gerekir.

Anahtar kelimeler: Otizm spektrum bozukluğu, gastrointestinal şikâyetler, çölyak hastalığı, anti-endomisyum IgA antikor

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INTRODUCTION

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by persistent deficits in social communication and social interaction, along with restricted, repetitive patterns of behavior, interests, or activities (1). ASD is a highly genetic and strongly brain-based disorder with an overall prevalence estimated to be 32.2 per 1000 (1 in 31) children aged 8 years, and an average male-to-female ratio of 3:1 (2). Given the vulnerability of the developing brain to environmental factors, a causative association between environmental factors and ASD is biologically plausible.

Celiac disease (CD) is known to be the most common gluten-triggered, immune-mediated, chronic, and multisystemic enteropathy in Western societies. It develops due to both environmental (gluten) and genetic (HLA DQ2/DQ8) factors (3). Gluten content in foods causes a T-cell-mediated inflammatory response in the proximal small intestine, resulting in mucosal damage and malabsorption (4). While patients with CD may present with gastrointestinal (GI) symptoms such as flatulence, bloating, fatigue, weight loss, and nutritional deficiencies, some patients may also have clinical symptoms unrelated to the GI system. An asymptomatic form of CD is also detected in the pediatric population (5). CD has been associated with neurological and psychiatric disorders such as ataxia, epilepsy, mood disorders, attention-deficit/hyperactivity disorder, schizophrenia, and ASD due to central nervous system involvement (6,7).

Anti-endomysial antibody (EMA) targets endomysium, a connective tissue protein found between myofibrils in smooth muscle cells of the gastrointestinal (GI) tract, and is widely used in screening for CD (8). The sensitivity and specificity of EMA in CD have been determined to be 94% and 98%, respectively. Currently, CD is diagnosed in two stages. The first stage involves serological screening for EMA, anti-tissue transglutaminase (a-tTG), and anti-gliadin antibodies (AGA). Second, duodenal biopsy is performed in patients with positive serology (9). EMA levels decrease with the implementation of a gluten-free diet and disappear with continuation. However, EMA reappears upon re-exposure to gluten (10).

The literature is contradictory about the relationship between ASD and CD. Some studies have reported no evidence of an ASD-CD link, while others have suggested a possible link or a higher prevalence of CD in children with ASD than in the general pediatric population (11). However, even in negative studies, positive CD serology was observed in autistic patients and although increased EMA and AGA levels were shown in some patients, duodenal biopsies revealed normal mucosa (12).

In light of the controversial and inconclusive literature, this study aimed to examine the serum EMA IgA levels of children with ASD in comparison with those of healthy participants. It was also investigated whether there was a relationship between GI symptoms and EMA IgA levels.

MATERIALS and METHODS

Participants

Eighty-five children with ASD ranging in age from 2 to 15 years were selected for the study group and 81 age-matched healthy children were selected for the control group. Subjects in the study group were recruited among children and adolescents who were referred to the Department of Child and Adolescent Psychiatry during a period of one year. The cases considered as ASD were enrolled consecutively in the study, and the psychiatric interviews were conducted by two expert psychiatrists within 6-month intervals.

Subjects with any genetic syndrome (e.g., Down syndrome, fragile X, Rett syndrome) and any med-

ical disorder (e.g., epilepsy, clinically active infection, and morbid obesity) were excluded from the study group. One hundred and seven children with ASD presented to the outpatient clinic, but three children diagnosed with genetic syndromes and 19 children with clinically active infection or chronic medical/neurological conditions were excluded from this study. Healthy children, living in similar addresses with patients, similar in age, and having no history of psychiatric illnesses were selected as the healthy control group. The exclusion criteria for the control group were neurodevelopmental disorders (e.g., ASD, intellectual disability, communication disorders), the presence of any neurological disorder, and clinically active infection. The study was carried out with the permission of the Dicle University Medical Faculty Ethics Committee for Non-interventional Studies (Date: 27.11.2015, Decision No: 68). We obtained an informed consent form from all patients for the procedure. All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Measures

Autism Behavior Checklist (ABC)

ABC was developed by Krug et al. (1980) (13). It has been used to evaluate the severity of autism symptoms. ABC consists of five subscales which have a 57-item scale including sensory, relationship building, the use of the body and objects, language skills, social and self-care skills. The lowest score of the scale is 0 and the highest score is 159. The scale has been adapted to Turkish by Irmak et al. (2007) (14).

Biochemical analysis

Blood samples were obtained in the morning between 09:00 and 10:00 h. The samples were collected in gel tubes. After withdrawal, blood samples were allowed to rest for 15 min for clotting. Then, blood samples were centrifuged at 5000 rpm for 6 min. The sera were transferred to 1.5 ml polypropylene tubes and stored at –80 °C until the analysis. Serum levels of EMA IgA were determined with ELISA method (Shanghai LZ Biotech Co., Ltd, China, catalogue number isYHB0246Hu), according to the manufacturer's instructions.

Procedure

Firstly the diagnosis process of ASD was conducted in referred subjects. Two expert child and adolescent psychiatrists examined the subjects consecutively and diagnosed them as ASD according to Diagnostic and Statistical Manual of Mental Disorders, 5th ed (APA, 2013). Patients with given the same diagnoses by two experts were included and cases without diagnostic consensus were excluded. The severity of autistic symptoms was assessed with the ABC scale. Children with ASD having a loss of language and social skills between 18-30 months were also diagnosed with regressive type of autism. Blood draw processes were performed by experienced pediatric nurses and phlebotomists. Blood samples of children were collected regardless of the time they wake up between 9 and 10 am once a day. ELISA was used to assay serum levels of EMA IgA. Family history of having any psychiatric disorder and CD, and complaints of GI system were also evaluated in both study and control groups.

Statistical Analysis

The student's t-test was used to compare normally distributed variables in independent groups, and the Mann-Whitney U test was used to compare nonparametric or ordinal variables. The effects of age and gender were adjusted using a two-way ANOVA and ANCOVA tests. The significance of the difference between the groups in terms of gender, history of psychiatric disorders in family members and relatives and consanguineous mar-

riage were evaluated using the chi-square test or Fisher's exact test where appropriate. Pearson's test was used to evaluate correlation coefficients and statistical significance of normally distributed variables, and Spearman's test was used to evaluate non-normally distributed variables. The values were given as mean ± standard deviation (SD). The p-value below 0.05 was considered statistically significant.

RESULTS

The study group consisted of 85 children (73 males, 12 females) with a mean age of 44.2 ± 22.6 months and the control group consisted of 81 healthy children (58 males, 23 females) with a mean age of 46.5 ± 14.1 months. There was no significant difference between the groups in terms of mean age of the participants, parental education level, number of siblings, rate of consanguineous marriages, mean age of the fathers, duration of breastfeeding and family history of CD ($p > 0.05$). There was a significant difference between the groups in terms of mean maternal age ($p < 0.05$). The presence of any psychiatric disorder in the family history and the frequency of GI complaints (e.g. diarrhea or constipation) were significantly higher in the study group compared to the control group ($p = 0.01$).

The mean EMA IgA levels for the study group and the control group were 212.8 ± 110.8 ng/ml and 142.7 ± 98.4 ng/ml, respectively. EMA IgA levels were significantly higher in the study group compared to the control group ($p < 0.001$).

Two-way analyses of variance (ANOVA and ANCOVA) were conducted in order to assess the contribution of age, gender and GI symptoms on EMA IgA levels of the groups. There was a negative correlation between age and serum EMA IgA levels in the study group ($r = -0.290$, $p = 0.007$). A one-month increase in age was associated with a 1.2 ng/ml decrease in serum EMA-IgA level. Gender and GI symptoms had no significant effect on EMA-IgA levels ($p > 0.05$).

The mean total ABC score was 78.7 ± 20.0 in the study group. The ABC subscale scores were found to be 9.5 ± 3.9 for sensory, 20.1 ± 5.6 for relating, 17.9 ± 5.8 for body and object use, 18.5 ± 5.5 for language, and 12.6 ± 3.6 for social and self-help. The regressive type of autism was observed in 23.5% of the subjects with ASD ($n = 20$). The total ABC scores and language, social and self-help subscale scores were significantly higher in subjects with regressive autism than those without regression ($p < 0.05$). The contribution of ABC total and subscale scores, and the regressive type of autism, to EMA IgA levels was also assessed in the study group. ABC scores and the regressive type of autism had no significant effect on EMA IgA levels ($p > 0.05$ and $p = 0.39$, respectively).

Table 1 shows the socio-demographic attributes and EMA IgA levels in the study and control groups.

DISCUSSION

The present study investigated the EMA IgA levels between children with ASD and their healthy peers. The results revealed a possible association between ASD and CD, as children with ASD were found to have significantly higher EMA IgA levels and more GI symptoms compared to healthy controls. Additionally, there was no significant difference in EMA IgA levels between children with GI symptoms and those without. The severity of ASD symptoms (ABC scores and regressive type) also had no effect on serum EMA IgA levels. Interestingly, age was found to be negatively associated with EMA IgA levels in children with ASD.

EMA IgA levels of children with ASD were found to be significantly higher than in healthy controls in the present study. Several recent studies supported the evidence of an increased immune response

Table 1 Socio-demographic variables and EMA IgA levels in the study and control groups

	Autism (n = 85)	Control (n = 81)	t value	p value
Age (months)	44.2 ± 22.6	46.5 ± 14.1	-1.195	0.23
Gender (male/female)	73/12	58/23	Chi-square	**0.03**
Mother's age (years)	30.5 ± 5.7	32.8 ± 5.3	-2.715	**0.01**
Mother's education period (years)	5.7 ± 4.9	7.2 ± 5.2	-1.955	0.05
Father's age (years)	35.4 ± 6.1	36.7 ± 5.3	-1.552	0.12
Father's education period (years)	9.0 ± 4.4	10.1 ± 4.9	-1.451	0.15
Number of siblings	2.7 ± 1.2	3.0 ± 1.5	-1.287	0.20
Breast milk (months)	17.6 ± 9.7	17.6 ± 7.3	-0.042	0.97
Consanguineous marriage (yes/no)	24/61	22/59	Chi-square	1.00
History of psychiatric disorders* (yes/no)	19/66	6/75	Chi-square	**0.01**
History of celiac disease* (yes/no)	3/82	0/81	Chi-square	0.25
Gastrointestinal complaints (yes/no)	14/71	3/78	Chi-square	**0.01**
Anti-endomysial antibody immunoglobulin A (ng/ml)	212.8 ± 110.8	142.7 ± 98.4	4.314	**0.001**

*History of celiac disease and psychiatric disorders in the immediate family and first-degree relatives.

and sensitivity to gluten in ASD patients, even in the absence of full-blown CD. De Magistris et al. found that AGA-IgG and anti-deamidated gliadin peptide (DPG-IgG) were more prevalent in patients with ASD compared to the healthy population (15). Damage to the intestinal epithelial barrier and increased intestinal permeability due to the immune system encountering partially digested gluten particles lead to an increased antibody response in ASD (16). In this context, higher EMA levels detected in the present study may be associated with this increased antibody response. Additionally, a correlation has been reported between EMA IgA levels and intestinal villus atrophy (17). Lau et al. found higher IgG antibody levels against gliadin in children with ASD compared to healthy controls but found no significant relationship between increased AGA IgG levels and HLA-DQ2/DQ8 concluding that the increased immune response to gliadin in children with ASD was not associated with CD (16). However, Józefczuk et al. showed that, contrary to the so-called "leaky gut" hypothesis, there is no deterministic correlation between the presence of gluten-induced autoantibodies and increased intestinal permeability in ASD patients (18).

There are also studies suggesting no relationship between CD and ASD. Batista et al. found no greater prevalence of CD or gluten sensitivity in children with ASD, and similarly, the prevalence of ASD was not greater than in a group of biopsy-proven CD patients in this respect (19). A recent cohort study by Zambrano et al. showed that there was no statistically significant difference between the CD seroprevalence in their ASD patients and healthy group and regular CD screening was not recommended in patients with ASD (20). In another recent study, Prosperi et al. also demonstrated no higher prevalence of CD in ASD children than in the control population, but they suggested the utility of routine CD screening, given its frequent atypical clinical presentation in this population (21). We partially agree with this suggestion. CD should be kept in mind especially in children with ASD who have persistent GI symptoms.

We found that GI symptoms such as diarrhea and constipation were more common in children with ASD compared to healthy controls in our study. Many studies reported that children with ASD experience more constipation, loose stools, recurrent abdominal pain, excessive gas and bloating compared to healthy children (22). One of the possible reasons for this situation could be higher EMA IgA levels causing GI symptoms in children with ASD compared to the control group. Another important reason could be the change in the structure of the intestinal flora due to the restricted dietary habits of children with ASD, especially the reduced intake of fibrous foods such as vegetables (23). However we found no relationship between EMA levels and GI symptoms in the present study and since we didn't question children's dietary habits, it is difficult to interpret the last mechanism.

Although CD can occur at any age, it is reported that EMA IgA appears after the age of two and antibody sensitivity is age-dependent (24). CD is more common in women with an average female to male ratio of 2:1 or 3:1 (25). Although our study determined that gender does not have a confounding effect on EMA IgA levels, interestingly, age was found to be negatively correlated with EMA IgA levels in children with ASD. An independent relationship has been reported between advanced maternal and paternal age and ASD risk (26), but in our study, the mean age of the mothers in the ASD group was found to be lower than that of the control group. It is known that the tendency to develop psychiatric disorders increases in the families of autistic children (27). Similarly, in our study, it was found that the incidence of psychiatric diseases increased in the families and close relatives of the ASD group. A complex interaction between CD and psychiatric disorders was also proposed in the literature (28,29). Higher antibody levels of children with ASD found in the present study may be associated with these psychiatric disorders in this respect.

Our study has several limitations that should be addressed. The EMA IgA test is considered a specific test for the diagnosis of CD due to its very high specificity (98-100%), but the fact that duodenal biopsy was not performed as the gold standard is an important limitation of our study. Another important limitation of our study is that the dietary habits of the participants, which may affect antibody levels, were not examined.

Despite these limitations, our study stands out due to its larger sample size compared to most previous studies. The results showed a possible link between CD and ASD due to increased EMA IgA response and higher GI symptoms found in our study. To clearly demonstrate the exact association between CD and ASD, higher EMA IgA levels should be supported by duodenal biopsy and other related antibodies like AGA, A-tTG and DPG. Furthermore, our study suggests that it may be appropriate to evaluate children with ASD who have persistent GI symptoms for CD, but this should not be routine for every child with ASD.

Ethics: *The study protocol was approved by the Dicle University Medical Faculty Ethics Committee for Non-interventional Studies (Date: 27.11.2015, Decision No: 68).*

Conflicts of Interest: *None of the authors have any potential conflicts of interest associated with this research.*

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