Medical Science Journal for Advance Research

Vol. 5, No.2, June 2024
e-ISSN: 2774-4892

Assessment of Serum Levels of Anti-Gliadin (IgA and IgG) and Interleukin-2 in Patients with Celiac Disease

Jalal Sabr Eidan[1] and Hawraa Ameer Mubark[2]
[1,2] University of Kufa, Faculty of Medicine, Medical Microbiology, Iraq.
E-mail: jalals.taefi@student.uokufa.edu.iq, hawraa.alhaider@uokufa.edu.iq

ABSTRACT

Background: Background: *Anti-gliadin antibodies (AGA) are essential for the screening of celiac disease (CD), but high AGA levels have also been seen in a number of immune-mediated cutaneous disorders, even in the absence of clinical signs of gastrointestinal illness. The study aimed to serum levels of anti-gliadin (IgA and IgG) in patients with celiac disease and factors associated with celiac disease.* ***Materials and methods:*** *This study is case-control design was conducted in Al-Imam Ali Hospital, Imam AL-Sadiq Hospital, and Marjan Hospital. A convenience sampling of 146 samples. The study comprised from two groups include 73 patients with celiac disease (CD), 73 healthy controls. Every participant donated 5 mL of venous blood, which was placed in gel tubes (6ml). Tubed blood is centrifuged for 10 minutes at 3000 rpm. Serum for (Anti-gliadin) is stored in Eppendorf tubes at -20 ºC until thawing on ELISA day.* ***Results:*** *The results reveal that patients had significantly higher mean Gliadin IgA antibody levels (5.121 ± 3.356) than controls (2.891 ± 1.438), at statistically significant P. value <0.001. Gliadin IgG antibodies were significantly higher in patients (7.312 ± 4.575) compared to controls (3.430 ± 2.111), p-value < 0.001. Furthermore, the results found that the results found that patients had significantly higher mean levels of IL-2 (28.940±11.743) than controls (21.452±3.171).* ***Conclusions:*** *The present study found that anti-gliadin (IgA and IgG), and IL-2 levels were higher significantly in patients with celiac disease. Furthermore, the results reveal that the sensitivity of anti-gliadin (IgG) were higher compared to anti-gliadin (IgA).*

Keywords: Celiac Disease, Prevalence, Gliadin, Antibody.

Article Information
Received: April 3, 2024; Revised: May 26 , 2024; Online: June, 2024

INTRODUCTION

Celiac disease, CD, is a prevalent condition characterized by an immune-mediated enteropathy. It occurs in individuals who are genetically predisposed and is triggered by the consumption of gluten proteins found in some cereals including barley, wheat, and rye (1). In those who possess hereditary susceptibility, the enzymes used for digestion inside the intestines exhibit an inability to effectively break down the gliadin protein present in foods eaten that may cause the inflammation of the intestines (2).

https://doi.org/10.46966/msjar.v5i2.189

The worldwide prevalence of celiac disease has increased to 1–1.5% in recent decades (3). This may be due to improved diagnostic procedures and environmental variables that affect the body's gluten reaction (4). In Arabia, CD is common, although Saudi Arabia has the highest rate, 3.2% (5). Celiac illness affects 10%–11.2% of children with type 1 diabetes in Iraq, according to limited data (6). In Iraq, an independent study documented the incidence of celiac disease (CD) among people diagnosed with irritable bowel syndrome (IBS) as 12.1% (7). The identification of CD serological markers has undoubtedly altered our understanding of the illness, prompting a reevaluation of both the disease concept and the diagnostic technique. This has been particularly evident during the last two decades. Antibodies against gliadin(AGA) both IgG and IgA were the first serological markers available and this since the early 1980s(8).

The widespread usage of ELISA technologies has been solidified by the measurement of AGA (9).The human body generates anti-gliadin antibodies (specifically IgA and IgG) as a reaction to gliadin, a kind of prolamin that is present in wheat. Three distinct genes encode gliadin, which may stimulate the body to generate various antibodies. 80% of individuals with celiac disease have detectable levels of anti-gliadin IgA antibodies (10). IgA is a valuable marker for diagnosing celiac disease due to its production in the small intestine, where gluten triggers irritation and inflammation in susceptible individuals (11).

MATERIALS AND METHODS

Study design and setting

This study is case-control design was conducted in Al-Imam Ali Hospital, Imam AL-Sadiq Hospital, and Marjan Hospital. The data was collected during a period from November 2023 to April 2024.

The study groups

A convenience sampling of 146 samples. The study comprised from two groups include 73 patients with celiac disease (CD), 73 healthy controls. Patients with CD defined by physician as previously or new diagnosed by gastroenterologist. Without celiac symptoms, healthy controls were identified.

Inclusion and exclusion criteria

Inclusion criteria: This study includes individuals all ages (age matched ± 5 years between study groups) and both sexes.

Exclusion criteria: Subjects with diabetes mellites, pregnancy, ischemic heart disease, other autoimmune diseases, other kidney diseases, and other gastrointestinal disorders prior to the study were excluded from consideration.

Methods of sample collection

After interviewing the members of the study groups, their age, and sex, residence, occupation, physical activity, and smoking were taken using a questionnaire designed by the researcher. After that, 5 mLof venous blood were collected from every individual in the study consecutively, the blood distributed into: gel tubes (6ml). The blood in tubes is centrifuged for 10 minutes at 3000 rpm. then the serum for (Anti-gliadin) are converted into Eppendorf tubes and preserved in a deep freezer at (-20 ºC) until thawing on the working day of ELISA.

Immunological Assays

The enzyme-linked immunosorbent assay (ELISA) technique was utilized for the evaluation of IL-2 and antigliadin antibody (IgA and IgG) serum levels, the biochemical kit used in the study for performed by a company AESKULISA.

STATISTICAL ANALYSIS

The data were gathered, arranged, analysed, and presented using Microsoft Office Excel 2010 and the statistical package for social sciences, version 27, from IBM-SPSS software. Following the determination of the normality of the variables, the numerical data's mean, standard deviation, standard error, and range were shown using the Kolmogorov-Smirnov normality test. The two independent samples t-test may be used to assess the mean difference between any two groups if the variable is normally distributed. The chi-square test (X^2) may be used to examine associations between any two category variables. Additionally, the data was analysed using the receiver operating characteristic (ROC) analysis in SPSS. ROC curves were generated by plotting sensitivity versus 1-specificity, and the area under the curve was used. The level of significance was defined as a P-value of 0.05 or less, and the very significant level was indicated by a P-value of 0.01 or less.

RESULTS

Demographic characteristics of patients with celiac disease and control subjects

Table (1) demonstrates that the majority of patients (34.2%) were lie within age group (11-20 years), while most of controls were lie within age group (21-30 years) with 38.4%. There is no a statistically significant difference (p = 0.134) was found between patient and control groups, with 72.6% and 69.9% females and 27.4% and 30.1% males. Patients (64.4%) were more likely to live in cities than controls (28.8%), with a p-value < 0.001. There was not statistically significant between patients and controls in terms of occupation (p = 0.098), in which the highest percentage of patients (41.1%) and controls (39.7%) were students. Patients were more likely to engage in moderate physical activity (53.4%) than controls (32.9%), while controls were more likely to engage in light physical activity (45.2%) than patients (42.5%), these results were statistically significant differences between patients and controls (p = 0.002). Smoking habits were not significant differences between patients (6.8%) and controls (11.0%).

Table (2) compares the levels of the patients with celiac disease and control subjects according to the Gliadin and TTG. The results reveal that patients had significantly higher mean Gliadin IgA antibody levels (5.121 ± 3.356) than controls (2.891 ± 1.438), at statistically significant P. value <0.001. Gliadin IgG antibodies were significantly higher in patients (7.312 ± 4.575) compared to controls (3.430 ± 2.111), p-value < 0.001. Furthermore, the results found that the results found that patients had significantly higher mean levels of IL-2 (28.940±11.743) than controls (21.452±3.171), at statistically significant P. value <0.001.

Receiver operator characteristic (ROC) curve analysis was used to assess the Anti-gliadin IgA level cutoff value and predict the

https://doi.org/10.46966/msjar.v5i2.189

Published by: READ INSTITUTE

Celiac disease as diagnostic tests or adjuvant diagnostic tests. The findings are displayed in table (3) and figure (1). With sensitivity, specificity, area under the curve (AUC), and P. value of 72.6%, 43.8%, 0.710 (0.627-0.792), and <0.001, the anti-gliadin IgA threshold value was >2.5.

Receiver operator characteristic (ROC) curve analysis was performed in order to assess the Anti-gliadin IgG level cutoff value and to forecast the Celiac disease as diagnostic tests or adjuvant diagnostic tests. The findings are displayed in table (4) and figure (2). With sensitivity, specificity, area under the curve (AUC), and P. value of 82.2%, 60.3%, 0.791(0.720-0.862), and <0.001, the anti-gliadin IgG cutoff value was >3.1.

Table (4) shows celiac disease patients' parameter correlation coefficients. Gliadin IgA and IgG exhibit a significant positive moderate correlation ($r = 0.451$, $p < 0.001$). Gliadin IgA also has positive correlations with IL-2 ($r = 0.280$, $p = 0.017$) respectively. Gliadin IgG has moderate positive correlations with IL-2 ($r = 0.473$, $p < 0.001$).

Table (1): The distribution of the patients with celiac disease and control subjects according to demographic characteristics

Demographic characteristics		GROUPS				X^2	P. value
		Patients		Control			
		No.	%	No.	%		
Age groups	≤10 years	5	6.8	4	5.5	5.934	0.313
	11-20 years	25	34.2	21	28.8		
	21-30 years	17	23.3	28	38.4		
	31-40 years	18	24.7	10	13.7		
	41-50 years	5	6.8	5	6.8		
	>50 years	3	4.1	5	6.8		
	Mean± SD (Range)	**25.1±12.3 (5-65)**		**25.5±11.9 (8-53)**			
Sex	Male	20	27.4	22	30.1	0.134	0.715
	Female	53	72.6	51	69.9		
Residence	Urban	47	64.4	21	28.8	18.608	**<0.001**
	Rural	26	35.6	52	71.2		
Occupation	Employed	13	17.8	5	6.8	6.308	0.098
	Self-employed	4	5.5	10	13.7		
	Student	30	41.1	29	39.7		
	Housewife	26	35.6	29	39.7		
Physical activity	Light	31	42.5	33	45.2	12.529	**0.002**
	Moderate	39	53.4	24	32.9		
	Vigorous	3	4.1	16	21.9		
Smoking	Yes	5	6.8	8	11.0	0.760	0.383
	No	68	93.2	65	89.0		

Levels assessment of the Anti-gliadin (IgA and IgG) and Interleukin-2

Table (2): Comparison between levels of the patients with celiac disease and control subjects according to the anti-gliadin (IgA and IgG) and IL-2.

	GROUPS	Mean ± SD	SE	t.test	P. value
Anti-Gliadin IgA	Patients	5.121±3.356	0.393	5.216	**<0.001**
	Control	2.891±1.438	0.168		
Anti-Gliadin IgG	Patients	7.312±4.575	0.535	6.582	**<0.001**
	Control	3.430±2.111	0.247		
IL_2	Patients	28.940±11.743	1.374	5.259	**<0.001**
	Control	21.452±3.171	0.371		

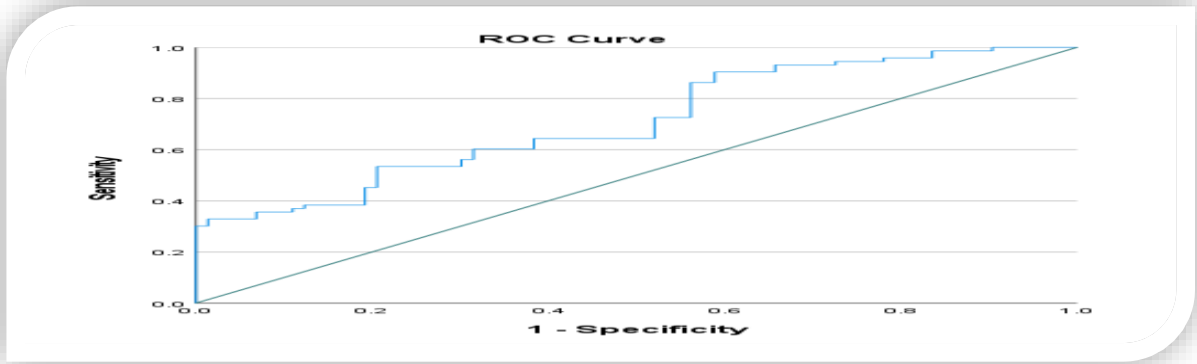

Figure (1) : Roc curve of Anti-gliadin IgA level in patients with Celiac disease.

Table (3): Sensitivity and specificity of Anti-gliadin IgA level in patients with Celiac disease

ROC Analysis (Cutoff Point =2.5)			Group	
			Patients with CD	Healthy Control
Anti-gliadin IgA level	**Predictive-Patients >2.5**	No.	53	41
		%	72.6%	56.2%
	Predictive-Control ≤2.5	No.	20	32
		%	27.4%	43.8%
	Sensitivity %	72.6%		
	Specificity %	43.8%		
	AUC (95% CI)	0.710 (0.627- 0.792) **"Fair"**		
	P. value	<0.001*		

https://doi.org/10.46966/msjar.v5i2.189

Published by: READ INSTITUTE

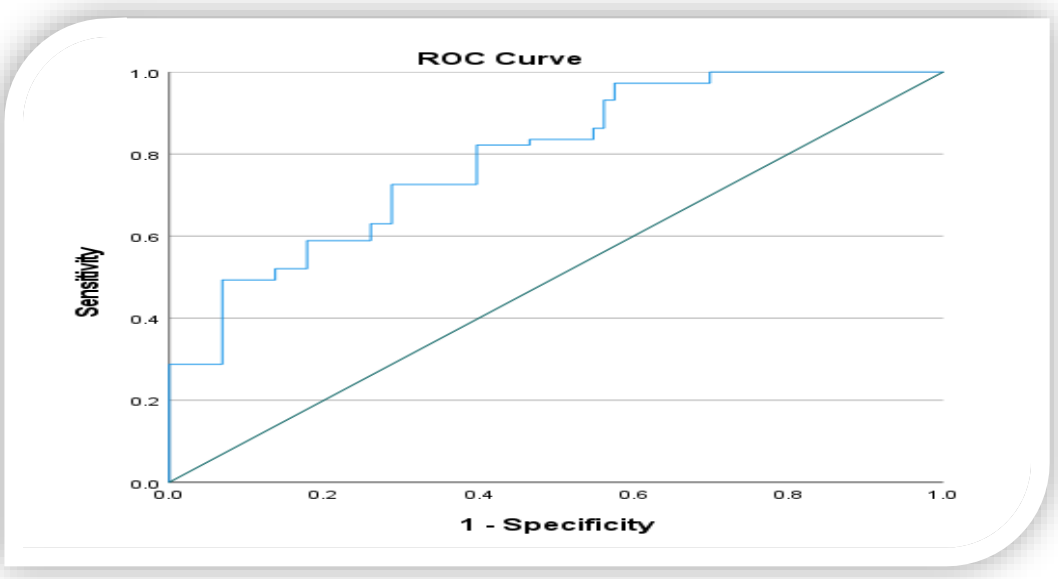

Figure (2) : Roc curve of Anti-gliadin IgG level in patients with Celiac disease.

Table (4): Sensitivity and specificity of Anti-gliadin IgG level in patients with Celiac disease

ROC Analysis (Cutoff Point =3.1)			**Group**	
			Patients with CD	**Healthy Control**
Anti-gliadin IgG level	**Predictive-Patients >3.1**	No.	60	29
		%	82.2%	39.7%
	Predictive-Control ≤3.1	No.	13	44
		%	17.8%	60.3%
	Sensitivity %	82.2%		
	Specificity %	60.3%		
	AUC (95% CI)	0.791 (0.720- 0.862) **"Fair"**		
	P. value	<0.001*		

Table (4): Correlation between the studied parameters in patients with celiac disease

		Gliadin IgA	Gliadin IgG	IL_2
Gliadin IgA	r	1	**0.451****	**0.280***
	P. value		**<0.001**	**0.017**
Gliadin IgG	r		1	**0.473****
	P. value			**<0.001**

****. Correlation is significant at the 0.01 level (2-tailed).**

***. Correlation is significant at the 0.05 level (2-tailed).**

DISCUSSION

In this study, there is no a statistically significant difference (p >0.05) was found between patient and control groups in terms of age and gender. These results are consistent with the previous study findings conducted by Alamoudi *et al.*, (2020) reported there was matching between case and control regarding age and gender (P. value >0.05). This can explain that gender/age matching is essential for study design to control bias effects. Researchers can reduce the impact of gender/age-related variables by ensuring that both case and control groups have equal numbers of men and women. Gender/age matching reduces bias and improves study validity by allowing more accurate case-control comparisons.

Regarding residence, the results found Patients (64.4%) were more likely to live in cities compared to 71.2% of controls live in rural regions, with a p-value < 0.001. Perhaps due to several variables for example urban residents may have higher celiac disease diagnosis rates due to better access to healthcare facilities and specialized services. Urban regions have more dietary options, which may increase the likelihood of celiac disease patients seeking medical assistance for gluten-related symptoms. Urban lives may also predispose people to disease-causing diets and environments.

In this study, controls were more likely to engage in vigorous physical activity (21.9%) than patients (4.1%). These results agreed with the study findings done by Martínez-Rodríguez *et al.*, (2021) which found that after 12 weeks of intervention, celiac women that followed exercise showed higher scores on health scale than celiac women without intervention. Active exercise may lower celiac disease risk for several reasons. First, exercise may lessen celiac disease autoimmune reactions by modulating the immune system. Regular exercise increases intestinal motility and blood flow, which may lessen celiac disease-related intestinal inflammation. Exercise often improves lifestyle habits including balanced meals rich in fiber and minerals, which may lower celiac disease risk. Physical exercise decreases systemic inflammation, a key celiac disease component.

The findings showed no significant smoking status differences between patients and controls (P>0.05). These findings

https://doi.org/10.46966/msjar.v5i2.189

Published by: READ INSTITUTE

supported Ludvigsson et al., (2005) findings that smoking did not cause CD. In addition, Wijarnpreecha *et al.*, (2018) observed that former smokers had the same celiac disease risk as never-smokers. These findings contradicted (Ludvigsson et al., 2014). Contrary to previous findings, smoking does not cause celiac disease. Some causes may cause this. Sample size, demographics, and research design may impact outcomes. Advances in celiac disease pathogenesis may reveal newly unknown details, rendering old linkages outdated.

The results reveal that patients had significantly higher mean Gliadin IgA antibody levels than controls, at statistically significant P. value <0.001. These results are consistent with the previous study findings conducted by Pallav *et al.*, (2016) and Al-Hussaini *et al.*, (2023). We can discuss that Gliadin IgA antibody levels in celiac disease are mostly caused by the immune system responding to gluten, a protein in wheat, barley, and rye. Gluten causes an inappropriate immune response in celiac disease, causing inflammation and small intestinal damage. After eating gluten, the immune system creates antibodies like Gliadin IgA. Gluten contains gliadin, which IgA antibodies target, causing celiac disease symptoms and tissue damage (Gujral et al., 2012) (20). Thus, increased Gliadin IgA antibodies indicate persistent gluten-induced immune reactivity and diagnose celiac disease.

In this study, the results report that gliadin IgG antibodies were significantly higher in patients (7.312 ± 4.575) compared to controls (3.430 ± 2.111), p-value < 0.001. These results agreed with the study findings done by Elia et al., (2018), which found that control samples were lower significantly for IgA- anti reticulin, IgA anti gliadin, IgG- anti reticulin and IgG-anti gliadin compared to patients with CD. In addition, these results agreed with the recent studies (22) (23). Thus, increased Gliadin IgG antibodies help diagnose and treat celiac disease.

The results of this study found that sensitivity, and specificity anti-gliadin IgA level 72.6%, and 43.8%, respectively. This supported by (24) which found that the sensitivity of gliadin IgA was significantly greater in celiac patients. Furthermore, the results of this study found that sensitivity, and specificity anti-gliadin IgG level 82.2%, and 60.3%, respectively. This matches (Elia et al., 2017) research, which found 80.0% gliadin IgG sensitivity and specificity.

Concerning IL-2, the results found that patients had significantly higher mean levels (28.940±11.743) than controls (21.452±3.171), at statistically significant P. value <0.001. these results are consistent with the study findings done by (26) reported that a significant plasma IL-2 increase occurred only in subjects with CD. Also, a study by (27) revealed that IL-2 in CD patients compared to controls ($p = 0.001$). in addition, these results supported by the recent studies (28) (29) which found that elevating of IL-2 level in patients with celiac disease. Due to its dysregulated immune response, celiac disease patients have elevated IL-2 level. Gluten causes an immunological reaction in the small intestine, causing inflammation and tissue damage in celiac disease. IL-2, generated by activated T cells,

regulates immunological responses and T cell proliferation (30) (31).

CONCLUSIONS

The current study found that celiac disease patients had significantly higher IgA and IgG levels of anti-gliadin than health controls. Compared to IgA, anti-gliadin (IgG) had higher sensitivity. Furthermore, celiac diseases were less common in rural areas and people experienced physical activity.

Ethical approval

The study followed a protocol approved by a local ethics committee at University of Kufa/ Medicine College/department of medical microbiology and Babylon Health Directorate under the reference number ( ), dated July 14 (09/2023).

Acknowledgment

The authors would like to express their gratitude to the individuals who generously agreed to participate in this survey. Additionally, we extend our sincere appreciation to employees working in Al-Imam Ali Hospital, Imam AL-Sadiq Hospital, and Marjan Hospital.

REFERENCES

1. Kumar S, Singh A, Singh AP, Ram S, Gupta OP, Pandey V, et al. Gluten-Related Disorders: Current Understanding, Myths, and Facts. In: Wheat Science. CRC Press; 2024. p. 321–38.
2. Orjiekwe OA. Nutritional Management of Celiac Disease. J Clin Exp Immunol. 2023;8(2):561–72.
3. Singh P, Arora A, Strand TA, Leffler DA, Catassi C, Green PH, et al. Global prevalence of celiac disease: systematic review and meta-analysis. Clin Gastroenterol Hepatol. 2018;16(6):823–36.
4. King JA, Jeong J, Underwood FE, Quan J, Panaccione N, Windsor JW, et al. Incidence of celiac disease is increasing over time: a systematic review and meta-analysis. Off J Am Coll Gastroenterol ACG. 2020;115(4):507–25.
5. El-Metwally A, Toivola P, AlAhmary K, Bahkali S, AlKhathaami A, AlSaqabi MK, et al. The epidemiology of celiac disease in the general population and high-risk groups in Arab countries: a systematic review. Biomed Res Int. 2020;2020.
6. Mansour AA, Najeeb AA. Coeliac disease in Iraqi type 1 diabetic patients. Arab J Gastroenterol. 2011;12(2):103–5.
7. Abdullah SH, Al-Badran AI. Some Immunological characters in Women with Celiac Disease from Thi-Qar province-South of Iraq. 2022;
8. Volta U, Bai JC, De Giorgio R. The role of serology in the diagnosis of coeliac disease. Gastroenterol Hepatol From Bed to Bench. 2023;16(2):118.
9. Di Simone N, De Spirito M, Di Nicuolo F, Tersigni C, Castellani R, Silano M, et al. Potential new mechanisms of placental damage in celiac disease: anti-transglutaminase antibodies impair human endometrial angiogenesis. Biol Reprod. 2013;89(4):81–8.
10. Dhamad GD, Mahmood WT, Kareem NGA, Jafar AK. Association between Diabetes Mellitus Type-1 and Celiac Disease in Growth Retardation Iraqi Patients. Indian J Forensic Med Toxicol. 2022;16(1):1375–9.
11. Lindblom-Dreis S. Stability Analysis of Immunogenic Gliadin Accumulation in

https://doi.org/10.46966/msjar.v5i2.189

Published by: READ INSTITUTE

Hard Red Spring Wheat (Triticum Aestivum L.). 2018;
12. Alamoudi NM, Alsadat FA, El-Housseiny AA, Felemban OM, Al Tuwirqi AA, Mosli RH, et al. Dental maturity in children with celiac disease: a case–control study. BMC Oral Health. 2020;20:1–10.
13. Martínez-Rodríguez A, Loaiza-Martínez DA, Sánchez-Sánchez J, Rubio-Arias JA, Alacid F, Prats-Moya S, et al. Effects of 12 weeks of strength training and gluten-free diet on quality of life, body composition and strength in women with celiac disease: a randomized controlled trial. Appl Sci. 2021;11(22):10960.
14. Ludvigsson JF, Montgomery SM, Ekbom A. Smoking and celiac disease: a population-based cohort study. Clin Gastroenterol Hepatol. 2005;3(9):869–74.
15. Wijarnpreecha K, Lou S, Panjawatanan P, Cheungpasitporn W, Pungpapong S, Lukens FJ, et al. Cigarette smoking and risk of celiac disease: a systematic review and meta-analysis. United Eur Gastroenterol J. 2018;6(9):1285–93.
16. Ludvigsson JF, Nordenvall C, Järvholm B. Smoking, use of moist snuff and risk of celiac disease: a prospective study. BMC Gastroenterol. 2014;14:1–7.
17. Pallav K, Xu H, Leffler DA, Kabbani T, Kelly CP. Immunoglobulin A deficiency in celiac disease in the United States. J Gastroenterol Hepatol. 2016;31(1):133–7.
18. Al-Hussaini A, Al-Jurayyan A, Alharbi S, Salman Bashir M, Troncone R. Performance of deamidated gliadin peptide antibodies as first screening for celiac disease in the general pediatric population. Front Pediatr. 2023;11:1279825.
19. Gujral N, Freeman HJ, Thomson ABR. Celiac disease: prevalence, diagnosis, pathogenesis and treatment. World J Gastroenterol WJG. 2012;18(42):6036.
20. Yu XB, Uhde M, Green PH, Alaedini A. Autoantibodies in the extraintestinal manifestations of celiac disease. Nutrients. 2018;10(8):1123.
21. Elia Z, Mustafa N, Othman G. Evaluation of Diagnostic Efficiency of Anti-reticulin and Anti-gliadin antibody test in Celiac Disease. Polytech J. 2018;8(1):294–306.
22. Uhde M, Caio G, De Giorgio R, Green PH, Volta U, Alaedini A. Subclass profile of IgG antibody response to gluten differentiates nonceliac gluten sensitivity from celiac disease. Gastroenterology. 2020;159(5):1965–7.
23. Catassi GN, Pulvirenti A, Monachesi C, Catassi C, Lionetti E. Diagnostic accuracy of IgA anti-transglutaminase and IgG anti-deamidated gliadin for diagnosis of celiac disease in children under two years of age: a systematic review and meta-analysis. Nutrients. 2021;14(1):7.
24. Rashtak S, Ettore MW, Homburger HA, Murray JA. Comparative usefulness of deamidated gliadin antibodies in the diagnosis of celiac disease. Clin Gastroenterol Hepatol. 2008;6(4):426–32.
25. Elia ZN, Hussain SG, Mustafa NW. Assessment of Anti–Gliadin (IgA & IgG), Thyroid Stimulating Hormon and Growth Hormon Level in Celiac Disease Patients in Erbil City–IRAQ. J Garmian Univ. 2017;4(ICBS Conference):581–92.
26. Cartee AK, King KS, Wang S, Dzuris JL, Anderson RP, Van Dyke CT, et al. Plasma IL-2 and symptoms response after

acute gluten exposure in subjects with celiac disease or nonceliac gluten sensitivity. Off J Am Coll Gastroenterol ACG. 2022;117(2):319–26.

27. Ganjali F, Asri N, Rostami-Nejad M, Hashemi M, Ainy E, Masotti A, et al. Expression analysis of IL-2, TBX21 and SOCS1 in peripheral blood cells of celiac disease patients reveals the diagnostic potential of IL-2. Mol Biol Rep. 2023;50(6):4841–9.

28. Vorobjova T, Tagoma A, Oras A, Alnek K, Kisand K, Talja I, et al. Celiac disease in children, particularly with accompanying type 1 diabetes, is characterized by substantial changes in the blood cytokine balance, which may reflect inflammatory processes in the small intestinal mucosa. J Immunol Res. 2019;2019.

29. Tye-Din JA, Daveson AJM, Goldstein KE, Hand HL, Neff KM, Goel G, et al. Patient factors influencing acute gluten reactions and cytokine release in treated coeliac disease. BMC Med. 2020;18:1–10.

30. Tye-Din JA, Daveson AJM, Ee HC, Goel G, MacDougall J, Acaster S, et al. Elevated serum interleukin-2 after gluten correlates with symptoms and is a potential diagnostic biomarker for coeliac disease. Aliment Pharmacol Ther. 2019;50(8):901–10.

31. Dunne MR, Byrne G, Chirdo FG, Feighery C. Coeliac disease pathogenesis: the uncertainties of a well-known immune mediated disorder. Front Immunol. 2020;11:513850.

https://doi.org/10.46966/msjar.v5i2.189

Published by: READ INSTITUTE