Ibn Al-Haitham Journal for Pure and Applied Sciences
Journal homepage: jih.uobaghdad.edu.iq
PISSN: 1609-4042, EISSN: 2521-3407
IHJPAS. 2025, 38(2)

Evaluation of Some Oxidants and Antioxidants Parameters in Iraqi Females with Celiac Disease

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Received: 5 May 2023 Accepted: 11 September 2023 Published: 20 April 2025
doi.org/10.30526/38.2.3459

Abstract

An inherited tendency is necessary for the manifestation of celiac disease (CD), an autoimmune, gluten-sensitive, inflammatory disorder of the small intestine. Gluten, a storage protein in cereals, causes CD. An increase in oxidant compound generation, surpassing the capacity of the endogenous antioxidant system, causes oxidative stress (OS). This study aims to evaluate some oxidants and antioxidants factors in Iraqi females with CD. This hospital-based cross-sectional study at Baghdad Educational Hospital in Baghdad, Iraq, involved 100 female participants of reproductive age. Tests have been measured, such as total oxidants (TOS), total antioxidants (TAS), malondialdehyde (MDA), catalase (CAT), nitric oxide (NO), total protein (TP), and Hb. The results of the present study showed that 72 of the female patients had Hb less than 12, whereas 28 had Hb equal to or greater than 12. The anemic patient group had elevated OS indices and a low TAS. Because of the damaged small intestine, CD patients experienced an imbalance between oxidant and antioxidant parameters, which was increased with anemia.

Keywords: Catalase, Celiac disease, Iron deficiency anemia, Malondialdehyde, Oxidative stress.

1. Introduction

Celiac disease (CD), an autoimmune, gluten-sensitive, inflammatory condition of the small intestine, requires a hereditary propensity to manifest (1). Gluten, the primary storage protein in grains, is the underlying cause of CD. Gliadins, often called prolamins and glutenins, are the two primary protein subgroups that make up gluten. Gliadin in wheat and hordein in barley are a few prolamin components connected to gluten intolerance in those with CD (1, 2).

It has been demonstrated that the gliadin sequence comprises areas that perform cytotoxic or immunomodulatory activities and are critical in CD pathogenesis. The other areas are responsible for forming oxidative stress (OS) and cytokine release that promotes inflammation (3). Anemia affects about 500 million women of reproductive age (15-49 years) worldwide and

is still a public health problem (4). Inflammation and OS are caused by an increase in oxidant compounds and a decrease in antioxidant defenses, which appear to be causing the CD (5). The body is shielded from free radical damage by antioxidant defense mechanisms that are both enzymatic and nonenzymatic. Superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione reductase (GR), glutathione S-transferase (GST), and glucose-6-phosphate dehydrogenase (G6PDH) are only a few examples of antioxidant enzymes. Glutathione (GSH), ascorbic acid (vitamin C), tocopherol (vitamin E), β-carotene, flavonoids, and binding proteins transferrin, ceruloplasmin, and albumin are examples of nonenzymatic processes (6). Total antioxidant status (TAS) measures an organism's total capacity to combat free oxygen radicals, the leading cause of oxidative damage. Vitamins are very effective nonenzymatic antioxidants. Total oxidant capacity, or TOC, measures the serum's oxidant capability (7). Changes in serum nitric oxide (NO) levels may signal CD activity and positively indicate therapy effectiveness; NO is one of all cells' most minor mammalian bioactive products (5). Increased levels of lipid peroxidation products have been linked to several chronic diseases, and MDA measurements are frequently employed as an indication of lipid peroxidation (6). Various aldehydes are produced when lipid hydroperoxides degrade in biological systems, including MDA (8). This study aims to evaluate some oxidants and antioxidants factors in Iraqi females with CD.

2. Materials and Methods

2.1. Study population

The study was conducted at Baghdad Educational Hospital in Baghdad, Iraq, from September 2021 to January 2022. This study included 141 samples, 100 Iraqi females who had just been diagnosed with CD (age 16-35 years). The Patients group was further divided into the G1 group with Hb >12% (n=28) and the G2 group with Hb <12% (n=72). Seemingly healthy females (n=41) were the control group (age 16-35 years) included in the present study. All participants in the present study were undergoing medical examinations at Baghdad Educational Hospital. The study did not consider other systemic problems such as inflammatory bowel disease, intestinal parasites, irritable bowel syndrome, cystic fibrosis, and immunological diseases. Personal interviews with pre-made questionnaires were used to collect all data.

2.2. Sample collection and biochemical analysis

In the present study, about 5 mL of blood sample was taken from each participant's venous blood using a sterile 5 mL syringe. The presence of CD in all the patients' samples was confirmed using a sterile 5 mL syringe. The blood sample was divided into two aliquots: 3 mL in a gel tube for serum extraction, which we used to measure the concentrations of TAS (g/dL), TOS (g/dL), nitric oxide (g/dL), MDA (mg/dL), and catalase (KU/L). A hematology analyzer computed the complete blood count (CBC) using 2 mL of an EDTA tube.

All parameters were investigated using a spectrophotometer, and BMI as weight (kg)/height (m^2) was calculated using a spectrophotometer. The data were analyzed using statistical software (SPSS 26.0; IBM Inc., Chicago, IL, USA). The mean and standard deviation of homogeneously distributed variables were calculated and compared using the one-way ANOVA test. Statistical significance was determined by P values less than $P < 0.05$.

2.2.1. Determination of serum anti-TTG

The enzyme-linked immunosorbent assay (ELISA) theory underlies the test. This method used the Diesse company kit with the CHORUS instrument. The results are expressed in IU/mL.

2.2.2. Determination of serum IgA

This method used the Ruch company kit and Cobas C 311. When the antigen in the sample reacts with anti-IgA antibodies, an antigen/antibody complex is created. This complex is turbidimetrically measured after agglutination. The results are expressed in IU/mL.

2.2.3. Determination of serum TOS

The Erel method was used to measure TOS (9). The oxidants in the sample oxidized the ferrous ion to the ferric ion to form a colored complex, the intensity of which is inversely correlated with the quantity of oxidants in the sample. Glycerol was added to speed up the oxidation reaction. The absorbance was measured at a wavelength of 560 nm. The results are expressed in μmol /L).

2.2.4. Determination of serum TAS

The TAS was measured by the Erel method (10). A standardized solution of the Fe^{+2}-*O*-dianisidine complex reacts with a standardized solution of hydrogen peroxide (H_2O_2) to produce a hydroxyl radical. These potent ROS oxidize the reduced colorless o-dianisidine molecules to yellow-brown colored dianisidyl radicals at low pH. Antioxidants in the sample prevent color production and oxidation reactions from happening. Absorbance was measured at λ=444 nm. The results of TAS were expressed as mmol /L.

2.2.5. Determination of lipid peroxidation

Malondialdehyde detection by thiobarbituric acid (TBA) reactivity is the most popular method for evaluating lipid peroxidation. A colorful substance is produced when thiobarbituric acid and MDA, as a byproduct of lipid peroxidation, are combined. The absorbance was measured at 532 nm. The results of MDA were expressed as nmol/mL.

2.2.6. Determination of serum catalase

The sample was incubated with 1.0 mL of substrate (20 mol/mol H_2O_2 in 60 mmol/L sodium-potassium phosphate buffer, pH 7.4) for 3 minutes at 37 °C to measure the catalase activity. The process was halted using titanium sulphate. Uncombined H_2O_2 and a titanium sulfate reagent are combined to create per titanic acid. The absorbance of yellow per titanic acid was estimated at 405 nm compared to a blank. The results are expressed in KU/L (11).

2.2.7. Determination of serum nitric oxide

The primary final product that was produced from nitric oxide was found to be nitrate. The most popular method relied on measuring the nitrite produced by the Griess method (12), which used sulfanilamide and naphthyl ethylene diamine dihydrochloride (NED) in an acidic environment, and the cadmium-mediated reduction of nitrate to nitrite. Sulfanilamide and NED compete for nitrite in the Griess reaction. The absorbance was measured at a wavelength of 540 nm. The results are expressed in μM/L.

2.2.8. Determination of serum total protein

A Roche company kit was used to determine serum total protein. The characteristic biuret complex, which is purple, is created when protein peptide connections react with divalent copper in an alkaline solution. The color intensity is directly proportional to the protein concentration in serum samples. The TP results were expressed as g/dL.

2.2.9. Determination of complete blood count

The blood was drawn in an EDTA tube and gently shaken for two minutes. The test starts automatically when the device's needle extracts the required blood aliquots. The Norma company's (Hungary) symbol 3 equipment was utilized.

3. Results

The current study includes 100 Iraqi females of reproductive age who are newly diagnosed with CD, with 40 healthy females serving as the control group (C). In the present study, all the individuals participating were aged from 16-35 years. Diarrhea and weight loss were the most prevalent signs at the time of presentation (64 patients); 36 of the patients were asymptomatic. Twenty-eight patients had an Hb level of 12 or more (G1, Hb=12.85±0.74 g/dL), and 72 patients had an Hb level of less than 12 (G2, Hb=9.62±1.44 g/dL). **Table 1** listed the study results, revealing no significant age difference between all the studied groups (P > 0.05).

Table 1. Mean ± SD for studied parameters among different groups (n=141).

Parameters	C (n=41)	G1 (n=28) Hb ≥ 12 g/dL	G2 (n=72) Hb < 12 g/dL	Groups	P
				C&G1	0.996
Age (year)	25.00 ± 5.80	25.10±2.76	25.04±5.18	C&G2	0.999
				G1&G2	0.998
				C&G1	0.888
Hb (g/dL)	13.03±0.64	12.85±0.74	9.62±1.44	C&G2	0.0001
				G1&G2	0.0001
				C&G1	0.0001
BMI (kg/m^2)	21.64±2.43	18.35±1.38	19.07±1.65	C&G2	0.0001
				G1&G2	0.202
				C&G1	0.0001
Anti-TTG (IU/mL)	2.73±0.70	18.33± 3.83	21.03±8.05	C&G2	0.0001
				G1&G2	0.037
				C&G1	0.0001
IgA (IU/mL)	2.05±0.49	23.23±5.16	22.45±7.80	C&G2	0.0001
				G1&G2	0.094

The G1 group had no significant difference in Hb levels (P= 0.888) compared to the C group (13.03±0.64 g/dL). In contrast, the G2 group had a highly significant decrease in Hb levels (P= 0.0001) compared to the C group and between the G1 and G2 groups (P= 0.000).

The BMI was significantly lower (P= 0.0001) in the G1 (18.35±1.38 kg/m^2) and G2 (19.07±1.65 kg/m^2) groups than in the C (21.64±2.43 kg/m^2) group, while there was no significant difference (P= 0.202) between the G1 and G2 groups.

The levels of anti-TTG in G1 (18.33±3.83 IU/mL) and G2 (21.03±8.05 IU/mL) were significantly higher (P= 0.000) as compared to the C group (2.73±0.70 IU/mL), while there was a significant decrease (P= 0.037) in G1 as compared to the G2 group. IgA in G1 (12.69±3.28 IU/mL) and G2 (16.45±11.02 IU/mL) were significantly lower (P= 0.000) as compared to the C group (2.05±0.49 IU/mL), while there was no significant difference (P= 0.094) between G1 and G2. The TOS was significantly lower in G1 (225.62±70.26 µg/dL) (P= 0.002) and G2 (279.27±67.73 µg/dL) (P= 0.000) as compared to the C group (85.41±44.94 µg/dL), while there was a significant decrease in G1 as compared to G2 (P= 0.024). The TAS was significantly lower in G1 (1.23±0.44 µg/dL) (P= 0.000) and G2 (1.40±0.35 µg/dL) (P=

0.000) as compared to the C group (2.92±0.47 µg/dL), while there was no significant difference between G1 and G2 (P = 0.168), as shown in **Table 2**.

Table 2. Mean ± SD for studied parameters among different groups (n=141).

Parameters	C (n=41)	G1(n=28) Hb ≥12 g/dL	G2(n=72) Hb <12 g/dL	Groups	P
				C&G1	0.002
TOS (µg/dL)	85.41±44.94	225.62±70.26	279.27±67.73	C&G2	0.0001
				G1&G2	0.024
				C&G1	0.0001
TAS (µg/dL)	2.92±0.47	1.23±0.44	1.40±0.35	C&G2	0.0001
				G1&G2	0.168
				C&G1	0.0001
MDA (mg/dL)	0.69±0.19	1.40±0.42	1.67±0.62	C&G2	0.0001
				G1&G2	0.050
				C&G1	0.0001
NO (µmol/L)	33.20±12.67	57.10±10.64	60.32±11.89	C&G2	0.0001
				G1&G2	0.535
				C&G1	0.578
Cat (KU/L)	62.70±2.85	61.60± 6.17	58.19±4.45	C&G2	0.0001
				G1&G2	0.002
				C&G1	0.002
TP (g/dL)	6.50±0.56	7.00±0.49	7.31±0.63	C&G2	0.000
				G1&G2	0.058

Table 2 also revealed that MDA was significantly lower in G1 (1.40±0.42 mg/dL) (P= 0.0001) and G2 (1.67±0.62 mg/dL) (P= 0.0001) as compared to the C group (0.69±0.19 mg/dl), while there was no significant difference between G1 and G2 (P= 0.050). Nitric oxide was significantly lower in G1 (57.10±10.64 µmol/L) (P= 0.0001) and G2 (60.32±11.89 µmol/L) (P= 0.0001) as compared to the C group (33.20±12.67 µmol/L), while there was no significant difference between G1 and G2 (P= 0.535). The catalase activity was significantly lower (P= 0.0001) in G2 (58.19±4.45 KU/L) in comparison to C group (62.70±2.85 KU/L); also, there was a significant difference (P= 0.002) between G1 (61.60±6.17 KU/L) and G2 (58.19±4.45 KU/L), while there was no significant difference between G1 and C group (P= 0.578). The TP was significantly increased in G1 (7.00±0.49 g/dL) (P= 0.002) and G2 (7.31±0.63 g/dL) (P= 0.000) as compared to the C group (6.50±0.56 g/dL), while there was no significant difference between G1 and G2 (P= 0.058).

4. Discussion

Oxidative stress results from an imbalance between the capacity to produce oxidant compounds and neutralize them in living things (13). The gastrointestinal tract (GIT), vital for nutrient absorption and immunological function, can also significantly contribute to ROS production (14). Continuous exposure to a wide range of potentially hazardous substances serves the intestinal epithelia as a selective barrier between the tissues and the luminal environment of the GIT. Numerous stresses lead to the production of free radicals, OS, and inflammatory reactions in the GIT, which includes the epithelium and immune/inflammatory cells (15). Highly reactive chemicals such as reactive oxygen species (ROS), reactive nitrogen species (RNS), and reactive sulfur species (RSS) can cause OS when they build up in tissues

and organs faster than the body's natural antioxidant defense system can deal with them. As a result, cellular damage and dysfunction occur, leading to various disorders (16). The results showed that most newly diagnosed patients had higher levels of OS parameters than healthy subjects. In G2, where IDA was present, the TAS was higher than in G1, where it was not. This increased OS led to decreased antioxidant parameters, which aligned with the findings of the previous studies (17,18). Untreated CD patients have decreased antioxidant potential, which may indicate a greater need for antioxidants to counteract the high levels of ROS produced in the body and prevent the harmful effects of ROS. Due to the overproduction of ROS brought on by gluten consumption, CD patients experience OS on both the small intestine wall and the organism as a whole. Oxidative stress can result in free radical damage to crucial cellular components, negatively affecting how those cells function. Inflammation of the gastrointestinal system, abdominal pain, low stature or stunted growth, diarrhea, anemia, weight loss, steatorrhea, and other symptoms are among the classic signs of CD or celiac enteropathy. Minor intestinal inflammation harms the villi, leading to villous atrophy, where the villi shorten and eventually flatten out. This results in poor nutrient absorption, which causes weight loss (19). The current results showed that the byproduct of lipid peroxidation, MDA, was also elevated in the two groups of patients (G2 had a higher value than G1) compared to healthy subjects, which was identical to previous studies' findings (20, 21). Induced NO synthase is one of the pro-inflammatory cytokines and enzymes activated by the oxidative imbalance of gliadin peptides in enterocytes. This increased synthesis of NO metabolites favors OS (22). Increased mucosal permeability may make it easier for intestinal toxins to reach the liver, and excessive NO generation may also cause the liver to become inflamed due to OS. Both patient groups (G2 had a higher value than G1) showed elevated nitric oxide compared to healthy subjects. Our result was identical to the previous studies (23-26). The catalase enzyme's main component of the antioxidant system is lowering OS, which is frequently associated with various clinical illnesses (27-29). CAT activity was lower in anemic celiac patients compared to the control group, while it remained relatively stable in non-anemic CD patients compared to healthy subjects (30). The current results confirm past studies' findings that OS may significantly contribute to the pathophysiology of this enteropathy and that it is associated with CD (31).

5. Conclusion

In conclusion, the results demonstrated that CD patients' inflammatory minor intestinal condition led to OS. The CD patients were imbalanced in oxidant and antioxidant parameters, which increased with anemia. These results provide evidence of the role of OS in the pathophysiology and monitoring of CD.

Acknowledgment

The authors thank the Department of Chemistry at the College of Science/ University of Baghdad for their assistance.

Conflict of Interest

There was no conflict of interest.

Funding

There was no financial support for this work.

Ethical Clearance

The local Scientific Committee at the College of Science / University of Baghdad approved this study (Ref.: CSEC/1123/0126 on 28/11/2023).

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