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Association between Antioxidant Defense Systems and Celiac Disease

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Abstract

Background: Intake of gluten activates the immune system in CD patients resulting in tissue damage to their digestive system.

Objectives: The oxidative stress status as well as antioxidant defense mechanisms in persons with CD and non-CD individuals while evaluating gluten-free dietary impact.

Methods: A total of 100 CD patients along with 100 healthy participants received inclusion for this study. The laboratory analysis determined oxidative stress through MDA, AOPP, and 8-OHdG examinations of bloodstream markers. The study measured antioxidant defenses using FRAP and TEAC techniques to examine SOD, CAT, GPx, and TAC. Tissue examination and blood tests were used to measure CD severity both before and six months into the GFD treatment.

Results: Patients with CD exhibited elevated oxidative stress levels compared to controls because their MDA reached 4.8 vs. 3.2 nmol/L whereas their AOPP reached 72.5 vs. 50.1 μmol/L and their 8-OHdG reached 5.3 vs. 3.1 ng/mL. The levels of antioxidant enzymes were below normal range in CD patients as measured by SOD at 2.1 U/mL compared to 3.5 U/mL, CAT at 28.3 U/mL versus 42.1 U/mL and GPx at 38.7 U/mL compared to 55.2 U/mL ($p < 0.001$). CD patients exhibited decreased levels of antioxidant capacity measured by FRAP values which amounted to 520 μmol/L while controls exhibited 730 μmol/L. Disease severity showed significant correlation with MDA (0.68) along with AOPP (0.55) and 8-OHdG (0.61). The patients who followed the GFD for six months showed reduced oxidative stress and higher antioxidant enzyme activities with statistical significance.

Conclusion: Rising oxidative stress during CD advancement results from reduced antioxidant mechanisms in the body. The monitoring of oxidative stress allows medical professionals to determine disease severity. A gluten-free diet acts as cellular protection which extends its therapeutic value above symptom management.

Keywords: Celiac disease, Oxidative stress, Antioxidants, Gluten-free diet, Inflammation, Malondialdehyde

1. Introduction

Persons with these health conditions develop autoimmune Celiac disease after consuming wheat barley and rye gluten products. Only the small intestine gets affected by prolonged inflammation which transforms villus tissue to scar tissue because nutrients cannot properly pass through [1]. Approximately 1% of the worldwide human population has CD but higher risk exists for people with the HLA-DQ2 or HLA-DQ8 genes. The development of CD depends on three main factors including genetic inheritance together with immune responses and external environmental factors. The consumption of gluten triggers an immune reaction which produces two types of autoantibodies: anti-tissue transglutaminase (tTG) as well as endomysial antibodies (EMA) [2]. The immune response in the intestines damages the intestinal lining which makes it difficult to absorb nutrients and maintains a healthy digestive system [3]. Researchers in the medical field have established that damaging molecules released during stress reactions lead to CD development. Greater oxidative stress progresses when reactive oxygen species accumulate above the usual protection mechanism function of the cells

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leading CD patients to develop elevated damage to their tissues and more severe inflammation. CD produces oxidative stress from immune cell activation plus problems with mitochondria and gut bacteria [4]. Overreacting immune cells produce ample ROS that causes harm to vital body organs. The functional ability of the small intestine is interrupted by inflammation, which causes cell damage through oxidation. The human body maintains multiple defensive processes which protect against oxidative stress in case such conditions develop. Two kinds of antioxidants protect human bodies from ROS injury: enzymatic agents such as superoxide dismutase [SOD], catalase [CAT], and glutathione peroxidase [GPx] and non-enzymatic agents like vitamins C and E, polyphenols, and selenium [5]. Cells receive protection from damages caused by ROS through different antioxidants which work to neutralize them. Research demonstrates that Crohn's disease patients present defective antioxidant systems that result in more extensive oxidative damage to their bodies [6].

Individuals who have celiac disease present reduced SOD together with CAT and GPx enzymes which suggests their bodies fail to defend against oxidative stress effectively. Inflammatory responses along with prolonged tissue damage happens because of the impaired antioxidant defenses. People who are vulnerable to gluten face oxidative reactions because the protein activates immune cells while releasing inflammatory proteins into their systems [7]. The strict implementation of gluten-free diets for those who have celiac disease relies on FDA monitoring systems. Commensal microorganisms within the gut maintain homeostasis between the reduction and oxidation potential of the body. The gut microbiota abnormalities in patients with CD result in higher levels of oxidative stress [8]. The antioxidant butyrate originates from specific bacterial strains yet other bacterial strains produce ROS that sustains inflammation. The severity of CD can be monitored through biomarkers including malondialdehyde (MDA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), along with advanced oxidation protein products (AOPP) according to research findings [9]. Execution of these biomarkers helps doctors assess the CD patient's condition evolution and treatment effectiveness.

The intake of dietary antioxidants provides protection from oxidative stress to people with CD. The human body works together with vitamins C and E selenium and polyphenols as antioxidants to lower ROS damage and inflammation levels [10]. People following a gluten-free diet should consume antioxidant-rich foods to gain improved health outcomes. The strict implementation of a gluten-free diet helps Crohn's disease patients decrease oxidative stress [11]. The intestinal function improves together with reduced immune system activation as major factors which contribute to this positive outcome. Patients who have CD commonly show selenium deficiency because malabsorption occurs [12]. Medical studies establish a relationship between mitochondrial dysfunction and oxidative stress in CD patients since dysfunctional mitochondria generate increased ROS production which intensifies inflammation and leads to tissue destruction [13]. Patients with Crohn's disease need additional medication treatments when they need to counteract damaging oxidative stress even though strict diet modification remains the main CD management approach. Research indicates that N-acetylcysteine (NAC) and flavonoid supplements both lower oxidative tissue damage along with maintaining gut health [14]. More scientific research is essential to comprehend the functioning of antioxidants in treating CD. Researchers should perform extensive clinical trials to study how dietary antioxidants along with pharmaceutical antioxidants benefit CD patient treatment [15].

This study aim to how CD patients differ from healthy people in terms of their oxides levels and which antioxidants protect them from damage. This research analyzes both the levels of oxidative stress and measures how a gluten-free diet affects them.

2. Materials and methods

2.1. Study design

This study type as a case-control design to test different oxidative stress factors between patients with celiac disease and healthy people. A case-control research design helps study the biochemical and molecular differences between people with medical illnesses and healthy participants.

2.2. Inclusion criteria

Cross-diagnosis of CD occurred through combined serological and histopathological testing which included anti-tTG IgA, anti-TTG IgG and EMA together with Marsh classification [16]. Age range: 18–60 years. Adherence to a gluten-free diet (GFD) for at least six months (for CD patients). Healthy control individuals without autoimmune disorders.

2.3. Exclusion criteria

Researchers excluded patients who had a secondary autoimmune disease together with new antioxidant supplement intake within the last three months and people who smoked or consumed

alcohol because both behaviors impact oxidative stress markers [17]. The researchers eliminated pregnant and lactating individuals from the study because pregnancy affects oxidative stress markers. The study did not consider participants who suffered from gastrointestinal conditions except for CD.

2.4. Samples

By using G*Power 3.1 the study authors determined the required sample quantity from past CD research outcomes [18]. Scientists found 100 CD patients and 100 healthy volunteers produced an appropriate statistical power for the research.

2.5. Blood sample collection

The study collected plasma and serum materials from 10 mL fasting blood samples that medical staff obtained. A cold centrifugation operation at 3000 rpm for ten minutes maintained at 4°C. Samples of plasma and serum were placed at –80°C for analysis before testing started.

2.6. Oxidative stress biomarkers

Plasma MDA levels were determined through the TBARS test method of Ohkawa *et al.*, [20]. for lipid peroxidation assessment. A spectrophotometer was programmed to read absorption at the wavelength of 532 nm. A chloramine-T assay kit enabled the evaluation of protein oxidation for advanced oxidation protein products (AOPP) at 340 nm according to Witko-Sarsat *et al.*, [21]. Tests for DNA oxidation damage detection used 8-Hydroxy-2′-Deoxyguanosine (8-OHdG) ELISA-based methods purchased from Cayman Chemical (USA).

2.7. Antioxidant enzyme activity

The test followed Marklund and Marklund, [22] to evaluate Superoxide Dismutase (SOD) Activity via the xanthine oxidase inhibition method. The researchers reported enzyme activity using values in U/mL measurement units for serum tests. CAT enzyme activity measurement involved tracking hydrogen peroxide's decomposition rate at 240 nm through the method described by Aebi, [19]. The GPx enzyme activity measurement utilized NADPH consumption assessment at 340 nm based on the approach by Paglia and Valentine [23].

Table 1. Clinical and demographic features.

Variable	Celiac disease (Mean ± SD)	Healthy controls (Mean ± SD)	p-value
Age (years)	34.2 ± 7.5	35.1 ± 8.0	0.51
Gender (Male/Female)	40/60	42/58	0.78
BMI (kg/m^2)	22.5 ± 3.1	23.0 ± 2.9	0.32
Disease Duration (years)	6.8 ± 3.4	N/A	N/A

2.8. Total antioxidant capacity (TAC)

The Total Antioxidant Measurement involved the FRAP (Ferric Reducing Antioxidant Power) test that quantified TAC by tracking Fe3+ to Fe2+ reduction at 593 nm according to Benzie and Strain's method [24].

2.9. Statistical analysis

This data was analyzed used Shapiro-Wilk to test if data samples followed the normal distribution. To analyze differences between CD victims and healthy people we ran independent t-tests or the Mann-Whitney U test. The study tested if oxidative stress levels tracked the greatest impact patient health. It was used multivariate regression analysis to measure and control data influences from normal variables including age, gender and eating habits. It was used analyses in SPSS v26.0 (IBM Corp, USA) and established the significance threshold at $p < 0.05$ for all tests.

2.10. Ethical approval

The proposal along with the sampling approach received ethical authorization from the Babylon Health Directorate Research Ethics Committee through administrative order No. 221 on 16/6/2024. The research received appropriate ethical clearance from Marjan Medical City for its execution.

3. Results

In Table 1, the age ranges and weight metrics plus patient gender status match between the CD and control groups exactly ($P > .05$). The CD course of patients lasts 6.8 years on average.

In Table 2, the oxidative stress levels of CD patients surpass those from healthy people at a very strong statistical significance ($p < 0.001$). Levels of MDA, AOPP, and 8-OHdG rise to higher amounts in patients with CD because their body tissues undergo excessive oxidation.

In Table 3, CD patients have far lower antioxidant enzyme activity than healthy individuals (test result below 0.001). Patients with CD have weak defenses

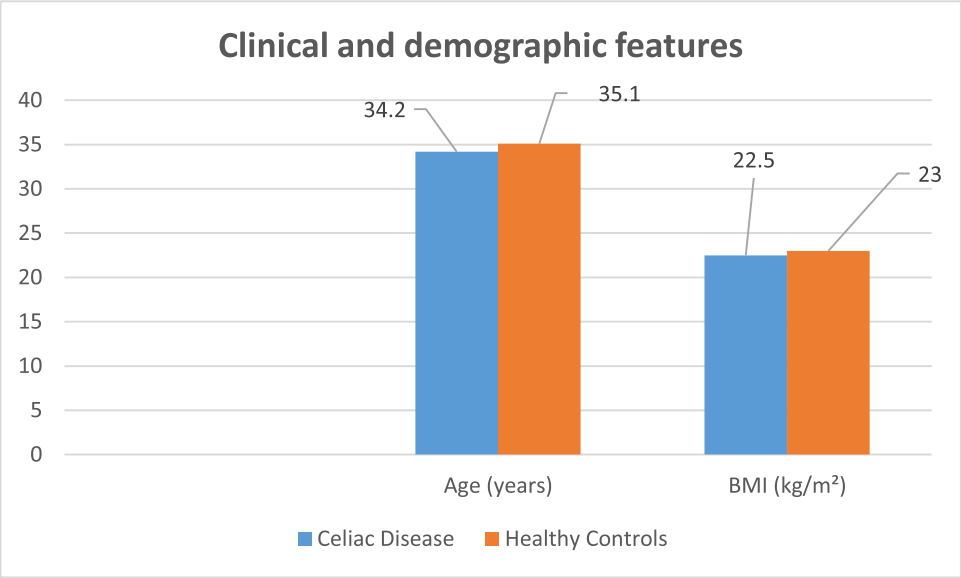

Fig. 1. Clinical and demographic features.

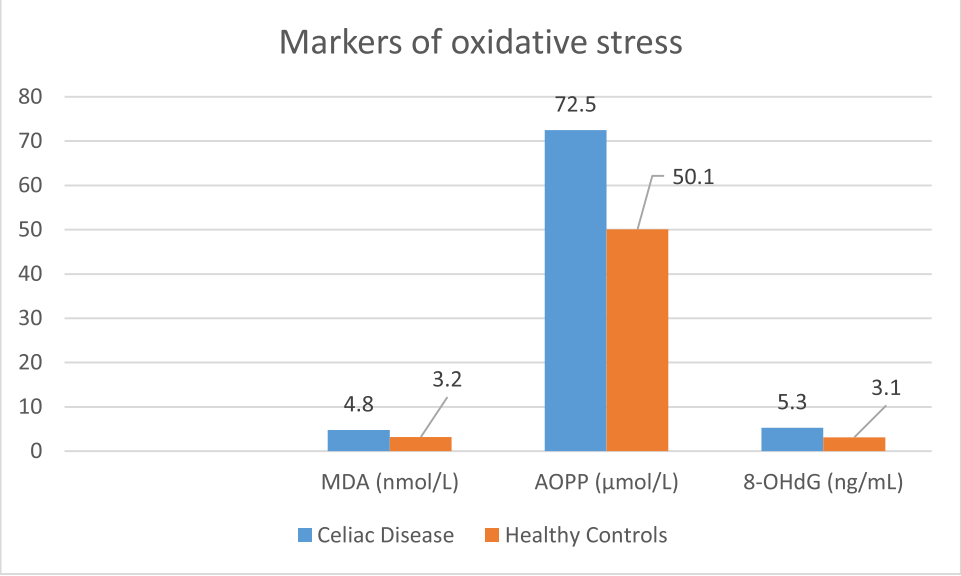

Fig. 2. Markers of oxidative stress.

Table 2. Markers of oxidative stress.

Marker	Celiac disease (Mean ± SD)	Healthy controls (Mean ± SD)	p-value
MDA (nmol/L)	4.8 ± 1.2	3.2 ± 0.9	<0.001
AOPP (µmol/L)	72.5 ± 14.8	50.1 ± 10.5	<0.001
8-OHdG (ng/mL)	5.3 ± 1.1	3.1 ± 0.7	<0.001

Table 3. Activity of antioxidant enzymes.

Enzyme	Celiac disease (Mean ± SD)	Healthy controls (Mean ± SD)	p-value
SOD (U/mL)	2.1 ± 0.4	3.5 ± 0.6	<0.001
CAT (U/mL)	28.3 ± 5.1	42.1 ± 4.7	<0.001
GPx (U/mL)	38.7 ± 6.5	55.2 ± 7.3	<0.001

against oxidative stress because their antioxidant protection is not working well.

In Table 4, Celiac disease patients show less total antioxidant capacity at 0.001 percent statistical significance. Lower FRAP and TEAC levels show that these antioxidants cannot effectively protect the body from free radical damage.

In Table 5, oxidative stress levels strongly match how serious the disease becomes. People who have

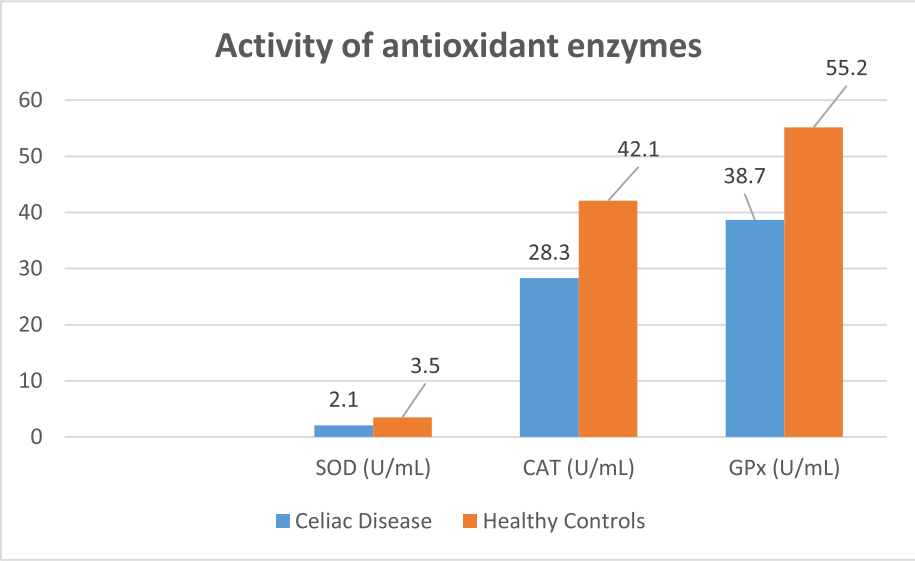

Fig. 3. Activity of antioxidant enzymes.

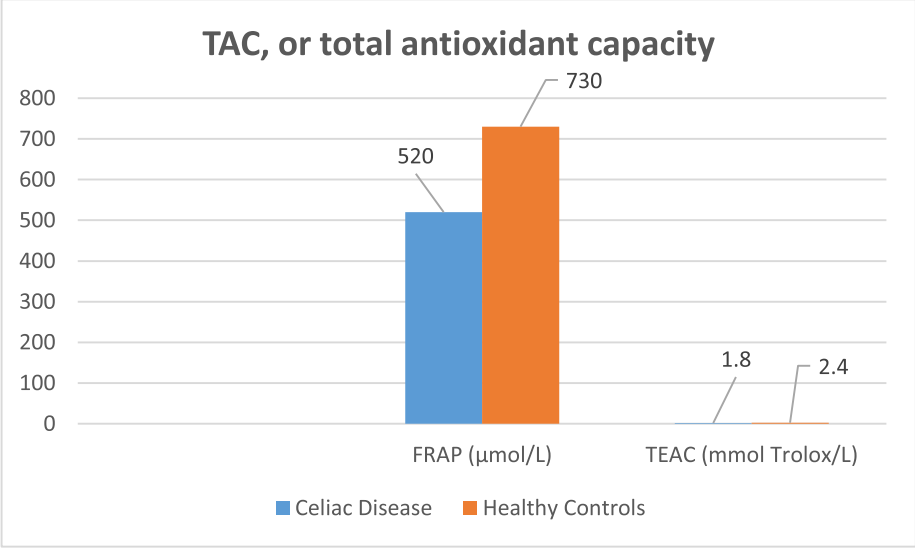

Fig. 4. TAC, or Total antioxidant capacity.

Table 4. TAC, or total antioxidant capacity.

Parameter	Celiac disease (Mean ± SD)	Healthy controls (Mean ± SD)	p-value
FRAP (μmol/L)	520 ± 75	730 ± 85	<0.001
TEAC (mmol Trolox/L)	1.8 ± 0.3	2.4 ± 0.4	<0.001

Table 5. Oxidative stress and disease severity correlation.

Marker	Correlation coefficient (r)	p-value
MDA vs Disease Severity	0.68	<0.001
AOPP vs Disease Severity	0.55	<0.001
8-OHdG vs Disease Severity	0.61	<0.001

bad CD symptoms show higher levels of oxidative stress according to statistical results ($p < 0.001$).

In Table 6, the tests showed that following a GFD lowered oxidative stress levels in all patients at ($p < 0.001$). The gluten-free diet restores cellular balance and decreases oxidative damage in people with CD.

CD patients showed increased oxidative stress markers together with decreased antioxidant enzyme activity when compared to healthy individual results. The extent of disease severity showed a positive relationship with oxidative stress levels. A gluten-free dietary regimen showed its therapeutic benefits by enhancing the parameters of oxidative stress.

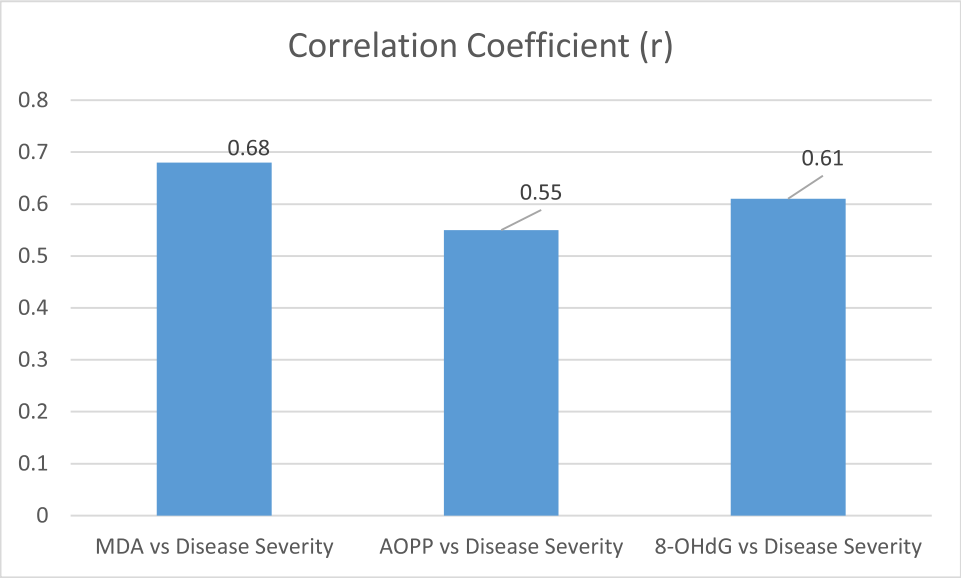

Fig. 5. Oxidative stress and disease severity correlation.

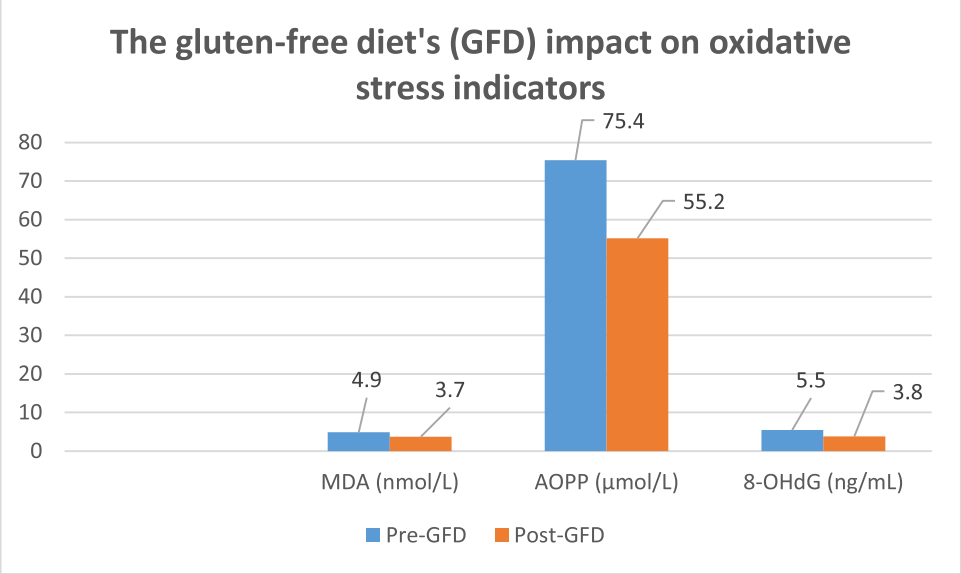

Fig. 6. The gluten-free diet's (GFD) impact on oxidative stress indicators.

Table 6. The gluten-free diet's (GFD) impact on oxidative stress indicators.

Marker	Pre-GFD (Mean ± SD)	Post-GFD (Mean ± SD)	p-value
MDA (nmol/L)	4.9 ± 1.3	3.7 ± 1.0	<0.001
AOPP (µmol/L)	75.4 ± 15.1	55.2 ± 12.0	<0.001
8-OHdG (ng/mL)	5.5 ± 1.2	3.8 ± 0.9	<0.001

4. Discussion

Celiac disease (CD) impacts oxidative stress and the antioxidant system during intestinal injury. The body develops autoimmunity against inflammation following the absorption of gluten in intestinal tract tissue. Activated macrophages along with T-cells generate reactive oxygen species (ROS) together with tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) to enhance oxidative stress [21]. Cell membranes undergo harm from ROS while lipid peroxidation produces malondialdehyde (MDA) during this process [20].

The immune response in CD leads to increased MDA levels. Enduring oxidative stress affects proteins which eventually results in the production of advanced oxidation protein products (AOPP). The degree of protein damage in CD patients

increases because of persistent inflammatory conditions. Increased oxidative stress attacks DNA which leads to the production increase of 8-hydroxy-2′-deoxyguanosine (8-OHdG) molecules [8]. CD patients experience intestinal cell damage together with persistent tissue harm because DNA oxidation occurs in their bodies. CD places significant strain on antioxidant defenses due to excessive ROS production.

Benzie and Strain demonstrated that CD patients show lower activity of protective enzymes superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) [24]. The damage to intestinal villi leads to selenium deficiency combined with lowered vitamin C and E and zinc levels while simultaneously impeding GPx basic functioning. The three enzymes SOD, CAT, and GPx come from intestinal cells damaged by CD. Reduced functional enzyme levels create an inability for the human body to defend against free radicals. Diarrhea causes patients with CD to consume limited nutrients thus leading to reduced levels of vitamin E, selenium, and polyphenols in their bodies [25]. Total Antioxidant Capacity (TAC) shows a decrease in level using Ferric Reducing Antioxidant Power (FRAP) and Trolox Equivalent Antioxidant Capacity (TEAC) testing methods. CD causes such extensive ROS generation that it drains antioxidant reserves according to research [26].

An increased level of oxidative stress results in worse gut tissue damage that produces higher ROS amounts and extends intestinal damage. Organ damage increases proportionally with the elevated amounts of MDA, AOPP, and 8-OHdG in the body. Villus atrophy becomes more severe because ROS damages intestinal cells which reduces the capacity to absorb nutrients [8]. The removal of gluten helps decrease both inflammatory reactions and ROS production levels. Starving patients from gluten through a gluten-free diet blocks immune system activation which creates less oxidative stress [21]. The absorption of selenium along with zinc and vitamin E restores normal functioning of antioxidant enzymes.

The Gut microbiota imbalance of CD patients results in an elevation of ROS production. When implemented the GFD reinstates microbial equilibrium while reducing systemic oxidation [12]. The research establishes both an exclusive connection between oxidative stress and CD as well as demonstrates how a GFD can curb oxidative damage. The results showed that patients diagnosed with CD have elevated oxidative stress markers together with reduced antioxidant enzyme functions and decreased TAC levels when compared to individuals without CD. The disease course of CD worsens due to oxidative stress while the severity of the condition shows direct correlation with oxidative stress levels. The adherence to GFD as a treatment provides CD patients with reduced oxidative stress levels. The levels of MDA, AOPP and 8-OHdG are elevated in CD patients when compared to the normal population. Mitochondrial dysfunction together with chronic inflammation causes immune responses which lead to increased production of ROS [19].

The oxidative stress process promotes lipid peroxidation into MDA while converting proteins to AOPP and it leads to DNA oxidation which results in 8-OHdG formation [21]. Scientific research findings demonstrate CD patients show higher concentrations of oxidative stress markers when compared to those without CD thus validating oxidative stress as a CD disease driver [16]. Influxes of gluten activate immune cell activation which produces inflammatory cytokines and boosts ROS generation [21]. Persistent oxidative stress damages the intestinal barrier to an extent that it causes increased villous atrophy while diminishing absorption of nutrients. CD patients show impaired endogenous antioxidant protection because their SOD, CAT and GPx activity levels are lower. These antioxidant enzymes serve as crucial protectors against ROS in addition to their function of neutralizing ROS in cells. Along with its enzymatic function GPx transforms hydrogen peroxide into less reactive chemical compounds [12]. The disintegration of antioxidant enzyme function due to oxidative stress leads to SOD and CAT and GPx activity loss [6]. The activity of GPx depends on selenium nutrition while SOD requires zinc for its operation and CAT needs iron for functioning [7]. CD-related injury causes detrimental effects on enzyme synthesis by intestinal cells since these cells naturally produce them. Research indicates CD patients demonstrate reduced blood selenium together with zinc and vitamin C and vitamin E content-all important factors for defending cells against free radicals [8].

The findings of this study establish how CD malabsorption deteriorates antioxidant protection systems in patients who become more susceptible to oxidative damage. Patients with severe intestinal damage show stronger correlations between oxidative stress markers (MDA, AOPP, and 8-OHdG) and disease severity [19]. Oxidative stress deepens when inflammation occurs which results in more serious intestinal damage. Done by ROS the disturbance of tight junction proteins results in intestinal permeability also known as “leaky gut” [14]. The continued stress caused by oxidative situations leads to abnormal immune responses which intensify the progression of the condition [13].

Medical professionals recognize oxidative stress as a measurement tool for CD severity that enables treatment effectiveness assessments. Medical tests measuring MDA, AOPP and 8-OHdG show that following a gluten-free diet restores homeostasis of

oxidative stress. Lab results show six months of total adherence to a GFD tends to decrease measurement of oxidative stress [14]. Eliminating gluten inhibits immune activation and suppresses T-cell proliferation and cuts down inflammatory cytokine creation that slows ROS formation [19]. The recovery of intestines from GFD adherence enables better absorption of all three antioxidants selenium, zinc and vitamin E supporting enzyme antioxidant functions [13]. Healthy gut microbiota restoration plays an additional role in lowering oxidative stress levels [14].

5. Conclusion

This study revealed the importance of oxidative stress in celiac disease health issues and shows its value as a disease measure in patients. It was proveed that CD patients have higher oxidative stress levels shown by MDA, AOPP, and 8-OHdG markers while their antioxidant enzyme activities (SOD, CAT, GPx) decrease with lower total antioxidant capacity (TAC) compared to non-CD individuals. Intestinal harm and inflammatory digestive conditions grow as a result of these changes. Oxidative stress levels reinforce their role in starting CD symptoms and worsening their impact on patient health. Oxidative stress levels decrease when people with CD stick to a gluten-free diet which showed why this treatment method goes beyond managing symptoms. The gluten-free diet helped control inflammation as well as restores normal gut function which enhances nutrient uptake while strengthening antioxidants and balancing gut microbiome to reduce oxidative damage in the system.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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