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Original Article

Oxidative Stress in Fatty Liver Disease with Metabolic and Non-metabolic Etiologies

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Abstract

Background: Fatty liver disease (FLD) is classified into metabolic dysfunction-associated fatty liver disease (MAFLD) and non-MAFLD based on the presence of metabolic abnormalities. The pathophysiology of FLD is complex; however, oxidative stress (OS) seems to play a significant role in its progression.

Objectives: This study examined oxidative stress markers linked to FLD.

Methods: A total of 64 patients with FLD, including 38 MAFLD and 26 non-MAFLD patients, respectively, were evaluated and compared with 22 healthy individuals. To assess oxidative stress, in addition to the activities of catalase (CAT), glutathione peroxidase (GPx), and superoxide dismutase (SOD) enzymes, total oxidant status (TOS), malondialdehyde (MDA), and total antioxidant capacity (TAC) were measured in the serum of the participants.

Results: It was found that TOS, MDA, and TAC levels in the serum were significantly higher in the FLD group compared to the healthy individuals. Notably, these increases were more pronounced in the MAFLD group compared to the non-MAFLD group. Additionally, CAT and GPx enzyme activities were significantly elevated in patients with FLD than in the control group. The MAFLD group exhibited the highest CAT enzyme activity, while the non-MAFLD group demonstrated the highest GPx enzyme activity. Conversely, serum SOD enzyme activity was decreased across all studied groups, with no significant differences observed between them.

Conclusion: Patients with MAFLD and non-MAFLD exhibit an imbalance in their oxidative stress systems. It appears that CAT and GPx enzymes probably play crucial roles in the antioxidant defense mechanisms in these patients, respectively.

Keywords: Antioxidant enzymes, MAFLD, non-MAFLD, Oxidative stress

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Background

Metabolic dysfunction-associated fatty liver disease (MAFLD) is a significant concern for 33% of the world's population, making it incredibly common (1). This condition occurs because of the accumulation of fat in the liver, which occurs in the context of metabolic dysregulation. This condition often starts as simple steatosis and, if untreated, may advance to metabolic dysfunction-associated steatohepatitis (MASH) and end stages of liver disease (2,3). The diagnosis of MAFLD requires confirmation of hepatic steatosis through radiological imaging techniques, along with obesity, type 2 diabetes (T2D), or metabolic abnormalities (4).

While diagnostic criteria have enhanced the accuracy of patient diagnoses, certain individuals remain unrecognized by these standards and are classified as non-MAFLD patients (5). Although this group of patients exhibits varying degrees of steatosis and typically presents a milder form of the disease, they may experience greater inflammation and hepatocyte ballooning compared to patients with MAFLD (6).

The pathogenesis of MAFLD is multifactorial, involving a variety of mechanisms, among which oxidative stress (OS) could be important (7). Furthermore, researchers indicated that the accumulation of fat in hepatocytes can induce OS by disrupting the balance between the production and scavenging of reactive oxygen species (ROS) (8). This imbalance induces the accumulation of ROS, leading to structural and enzyme damage and ultimately cell death (9). Elevated generation of ROS has been observed in patients with obesity and diabetes, which are linked to MAFLD (7).

Liver cells have a strong antioxidant defense system consisting of both enzymatic and non-enzymatic

elements that help maintain ROS at physiological levels (2). Enzymatic antioxidants primarily break down ROS, whereas non-enzymatic antioxidants reduce oxidative damage by contracting free radicals and non-radical oxidizing agents (10).

This study aims to examine OS markers and the activity of antioxidant enzymes in the serum of patients with MAFLD, patients with non-MAFLD, and healthy individuals.

Materials and Methods

Study Design

A total of 64 patients with fatty liver disease (FLD) were assessed by an expert hepatologist and compared to 22 healthy controls. Based on diagnostic criteria, FLD patients were classified into two groups, those with MAFLD and those without MAFLD.

To diagnose MAFLD, it is necessary to demonstrate the presence of fatty liver using imaging techniques, and the patients need to be overweight/obese or have type 2 diabetes. If a patient has liver steatosis, does not meet the previous two criteria, and is thin, he/she needs to have at least 2 of the following 7 metabolic disorders (11):

1. Waist circumference ≤ 102 and 88 cm (for men and women, respectively)
2. Blood pressure ≤ 85/130 mmHg or use of certain medications
3. Triglyceride level ≤ 150 mg/dl or use of certain medications
4. HDL-C level > 40 mg/dl and 50 mg/dl (for men and women, respectively) or use of certain medications
5. Prediabetes (fasting blood sugar 100-125 mg/dl, 2-hour blood sugar 140-199 mg/dl, or HbA1c level 4%/7-6/5)
6. HOMA-IR ≤ 2.5
7. CRP level < 2 mg/L

A notable point regarding the diagnosis of patients based on MAFLD diagnostic criteria is that patients who were not diagnosed with non-metabolic NAFLD or lean NAFLD have fatty liver but do not have obesity, diabetes, or metabolic disorders (4,12). Accordingly, 38 and 26 patients with MAFLD and non-MAFLD were identified, respectively (Figure 1).

All participants' BMI was also calculated by dividing their weight in kilograms by the square of their height in meters.

Inclusion criteria were being at least 18 years old and having ultrasound-confirmed steatosis. Patients taking steatogenic or lipid-lowering medications, as well as those under antihypertensive treatment, were excluded from the study (13). The study design was comprehensively communicated to participants, and informed consent was obtained. The current study was approved by the Research Ethics Committee of Hamadan University of Medical Sciences (IR.UMSHA.REC.1403.602).

Blood Collection

All participants referred to the Arad Laboratory in Hamadan between 8 and 9 am and 5 mL of peripheral venous blood from subjects who underwent 12 hours of fasting was collected in a clot tube to obtain serum.

Oxidative Stress Markers Assay

Total oxidant status (TOS) was measured using the Ferrous Oxidation Xylenol Orange (FOX) assay, which quantifies the oxidation of Fe^{2+} to Fe^{3+} by oxidant compounds present in samples. In this procedure, 50 µL of serum was mixed with 950 µL of FOX reagent, and subsequent to centrifugation, the supernatant was assessed spectrophotometrically at a wavelength of 560 nm (14). Malondialdehyde (MDA) levels are typically measured using thiobarbituric acid reactive substances (TBARS) assay. In this procedure, 50 µL of serum was added to 1 mL of TBA reagent and 4 mL of distilled

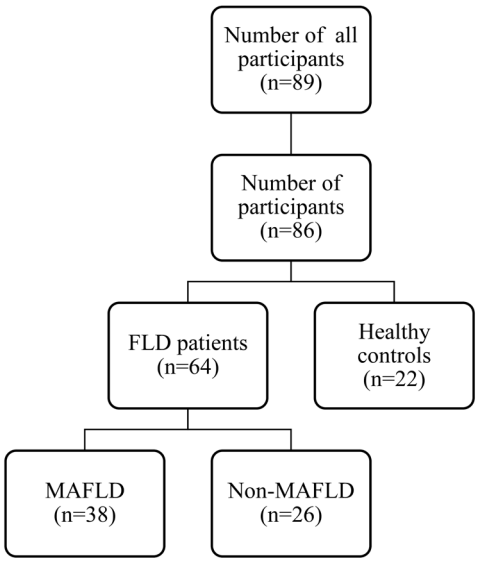

Figure 1. Flow Chart of the Study

water. The mixture was subsequently incubated in a 95 °C water bath for 60 minutes. Subsequent to cooling, 5 mL of butanol was introduced, and the liquid was completely vortexed. Finally, centrifugation was performed to separate the phases, and the absorbance of the upper layer was measured using a fluorimeter with an excitation wavelength set at 515 nm and an emission wavelength at 553 nm (15). The total antioxidant capacity (TAC) was evaluated using the Ferric Reducing Antioxidant Power (FRAP) method, which quantifies the capability of the sample to reduce Fe^{3+} to Fe^{2+} in the presence of Tripyridyl-s-triazine (TPTZ). In this assay, the formation of a bluish complex (Fe^{2+} and TPTZ) allowed for the quantification of the electron donating capacity of antioxidants. In this procedure, 100 µL of the sample was mixed with 3 mL of FRAP reagent. The resultant color intensity was subsequently quantified with a spectrophotometer at a wavelength of 593 nm (16).

Enzymatic Anti-oxidant

Enzyme assays for catalase (ZB-CAT-96A), glutathione peroxidase (ZB-GPX-96A), and superoxide dismutase (ZB-SOD-96A) were performed utilizing ZellBio kits (ZellBio GmbH, Germany) following the manufacturer's protocol. The absorbance was measured at the wavelength recommended by the kit using a microplate reader (MicroDigital M2, Mobi, China).

Statistical Analysis

Statistical analysis was conducted using SPSS version 16.0 (Chicago, Illinois). One-way ANOVA was used for normally distributed data, while the Kruskal-Wallis test was used otherwise. *P*-values of less than 0.05 were considered statistically significant.

Results

Characteristics of Subjects

FLD patients had a higher body mass index (BMI) compared to healthy individuals. Furthermore, BMI in the MAFLD group was significantly greater than in controls and non-MAFLD patients (Table 1).

Oxidative Stress Status

In this study, an examination of OS markers revealed that serum levels of TOS, MDA, and TAC were significantly elevated in FLD patients compared to the control group ($P<0.001$) (Figure 2A).

As depicted in Figure 2B, TOS levels were significantly higher in both MAFLD and non-MAFLD patients than in controls, with $P<0.001$ and <0.05, respectively. An increase was also noted in MDA, which was significantly elevated in both the MAFLD group ($P<0.001$) and the non-MAFLD group ($P<0.05$) relative to controls. Regarding TAC, a significant increase was observed exclusively in MAFLD patients compared to healthy individuals ($P<0.001$). Although TAC also showed an increase in the non-MAFLD group, this difference did not reach statistical significance. No significant differences were found in the assessed parameters between the MAFLD and non-MAFLD groups.

Table 1. Characteristics of the Study Participants

	Control	FLD	non-MAFLD	MAFLD
Subjects (n)	22	64	26	38
Gender (male)	10	38	16	22
Age (year)	36.64 [15]	42.27 [17.5]	40.85 [17.75]	43.24 [16.25]
Grade (1/2/3)		23/31/10	14/10/2	9/21/8
BMI (kg/m^2)	23.5 ± 4	28.25 ± 3.9***	24.77 ± 1.5	30.7 ± 3.11$^{***,\#\#\#}$

The results were analyzed using one-way ANOVA and Kruskal-Wallis tests. Results are expressed as mean ± SD or median [IQR] ($^{***}P<0.001$ vs. control, $^{\#\#\#}P<0.001$ vs. non-MAFLD).

Anti-oxidant Enzymes

As presented in Figure 3A, the activity levels of CAT and GPx enzymes were notably higher in FLD patients compared to healthy participants ($P<0.001$ and $P<0.01$, respectively). Conversely, the activity levels of SOD in the fatty liver group showed a decrease; however, this difference was not statistically significant.

Further investigation revealed that CAT activity levels were significantly higher in MAFLD and non-MAFLD patients than in healthy individuals, with *P* values of <0.001 and <0.01, respectively. This increase was particularly pronounced in the MAFLD group. Regarding GPx, both patient groups showed elevated enzyme activity; however, a significant difference was only noted in non-MAFLD patients relative to controls ($P<0.01$). Moreover, the assessment of SOD activity showed decreased serum levels in both MAFLD and non-MAFLD patients, but these differences were not statistically significant. Overall, there were no significant differences found between the MAFLD and non-MAFLD groups in the analysis of the results (Figure 3B).

Discussion

MAFLD is linked to OS, resulting in increased attention in past years to the effects of OS on the development and progression of FLD (17, 18). In this case-control study, the activity of antioxidant enzymes and serum concentrations of markers of OS were examined in patients with MAFLD, patients with non-MAFLD, and healthy participants. These findings indicate that patients with FLD, particularly those with MAFLD, exhibit a heightened state of OS, evidenced by increased levels of TOS and MDA concentrations. Additionally, increased antioxidant status was observed among these patients, reflected by increased TAC levels and CAT and GPx activities. However, SOD enzyme activity was found to be reduced in the FLD patients.

Previous studies on MAFLD have shown variable results. For instance, Zakaria et al (19,20) established an animal model of MAFLD in rats and reported an increase in MDA levels in the liver tissues of the MAFLD group compared to the control, which aligns with the

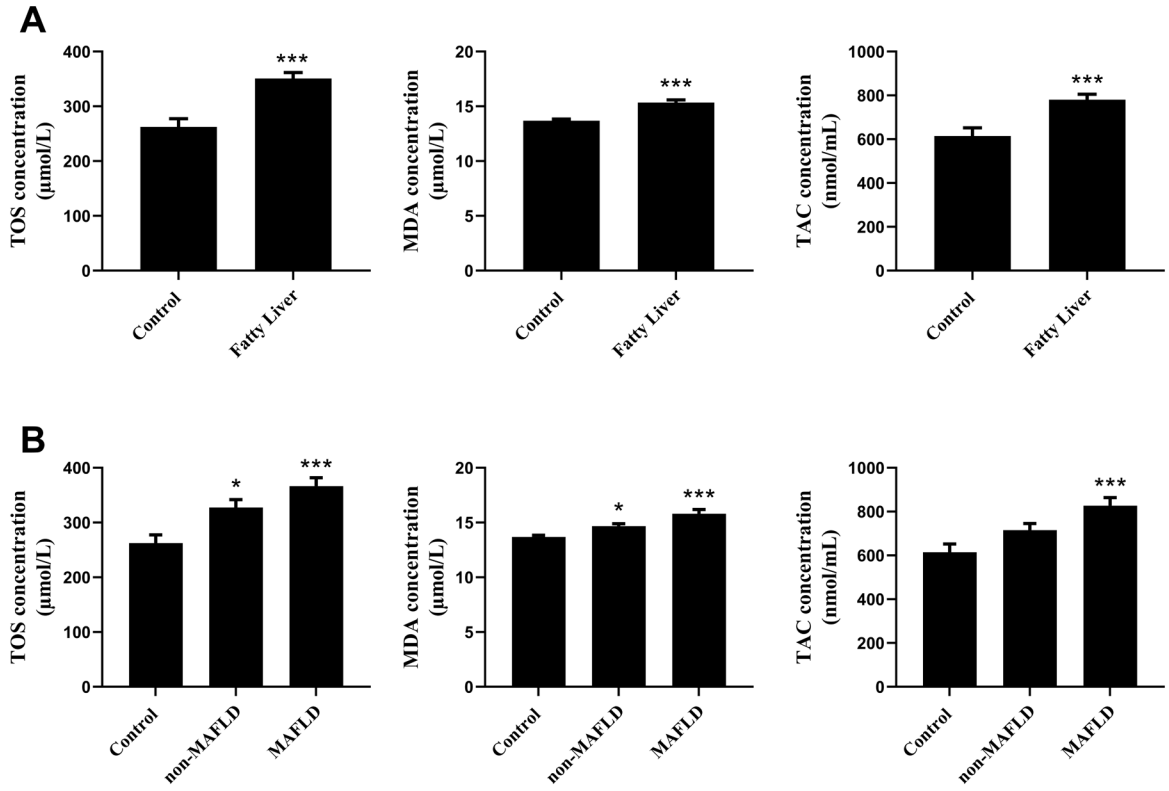

Figure 2. Oxidative Stress Markers. (A) Serum levels of oxidative stress markers in FLD patients (n=64) compared to the controls (n=22). (B) Serum Levels of oxidative stress markers in non-MAFLD patients (n=26) and MAFLD patients (n=38) compared to controls (n=22). Results are expressed as mean±SEM (*$P<0.05$ and ***$P<0.001$ vs. control)

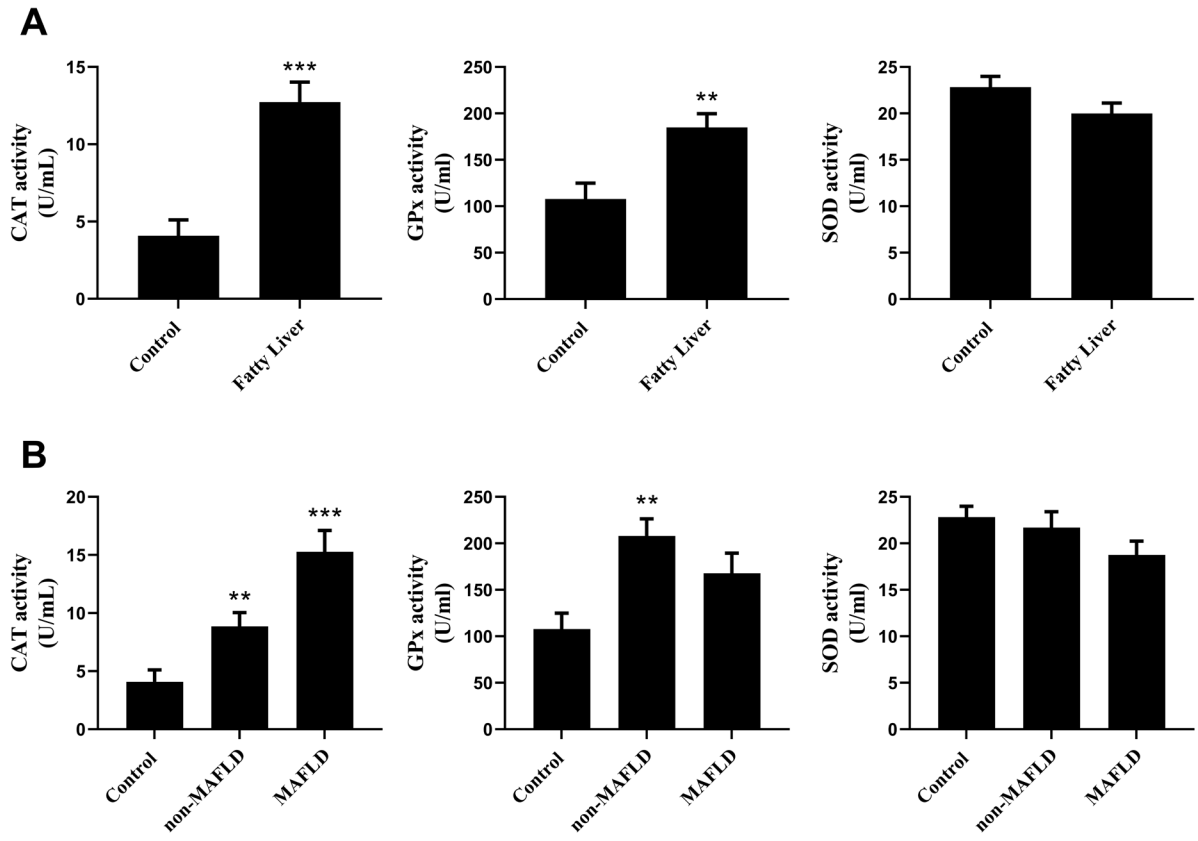

Figure 3. Antioxidant Enzymes Activities. (A) Serum levels in FLD patients (n=64) compared to controls (n=22). B. Serum levels in non-MAFLD patients (n=26) and MAFLD patients (n=38) compared to controls (n=22). Results are expressed as mean±SEM (**$P<0.01$ and ***$P<0.001$ vs. control)

findings of this study. One mechanism by which OS contributes to hepatic steatosis is the disruption of lipid metabolism, which is particularly vulnerable to damage by ROS. ROS-induced lipid peroxidation lead to the production of MDA as a downstream product of this damage (21). Increased levels of MDA can further exacerbate steatosis and contribute to the severity of the disease by interfering with the secretion of very low-density lipoprotein (22). Additionally, the products of lipid peroxidation are implicated in the progression of the disease to steatohepatitis (23). Furthermore, studies have demonstrated that elevated MDA levels can stimulate the expression of the Fas ligand gene in hepatocytes, thereby enhancing the production of inflammatory cytokines via the NF-κB pathway, which increases the risk of liver fibrosis (24). Conversely, they observed a decrease in TAC levels in the liver tissues of the MAFLD group, alongside reduced activity levels of the CAT, GPx, and SOD (19,20), which is in contrast with the findings of this study. Asghari et al (9) found elevated serum MDA levels in FLD patients compared to healthy individuals, corroborating our findings. Their study further indicated a reduction in serum TAC in patients with FLD compared to controls. They also investigated antioxidant enzymes in erythrocytes and reported that the activity levels of SOD and GPx were elevated in these patients; however, the increase in GPx activity was not statistically significant. Another study reported that plasma levels of TOS and MDA were increased in patients with FLD compared to controls (8), which aligns with the findings of this study. Additionally, this study noted an increase in the activities of SOD and GPx enzymes in patients with FLD; however, CAT enzyme activity was found to be decreased. Notably, no significant changes in plasma TAC levels were observed. Additionally, researchers categorized patients with FLD into two groups: early and advanced stages (8). It was observed that the activity level of GPx was higher in the early stage compared to the advanced stage, although this difference was not significant. Notably, patients in the early stage of FLD can be considered somewhat representative of non-MAFLD patients, which is consistent with the results obtained in this study. In general, according to past studies, an increase in the levels of oxidant factors is associated with a corresponding increase in antioxidant enzyme activity, reflecting a compensatory physiological response to mitigate these conditions (9). Although most previous studies reported decreased TAC levels in patients, this study found an increase in TAC. The possible reason for this observation could be the compensatory response of the body to OS and increased production of antioxidants. Dietary factors and consumption of antioxidant-rich foods could also be responsible for this, as patients are likely to follow a strict diet due to their disease. Regarding the reduced activity levels of SOD, we hypothesize that the mechanisms underlying OS in patients with FLD may initially disrupt the production or function of this enzyme. However, further studies are necessary to explore this relationship in greater detail. These results appear to be dependent on the type of sample analyzed. For instance, OS markers measured in liver samples from patients with FLD consistently demonstrate decreased activity or levels across all examined data. In contrast, analyses of other sample types, such as blood, plasma, and serum, reveal that antioxidant levels are often elevated in the majority of FLD cases (25). It is noteworthy that OS can serve both as a cause and a consequence of FLD (26). However, further studies are needed to establish a definitive causal relationship regarding this phenomenon.

Conclusion

Patients with MAFLD and non-MAFLD exhibit an imbalance in their oxidative systems, which is more pronounced in those with MAFLD. Furthermore, given the greater increase in GPx enzyme activity in non-MAFLD patients than in MAFLD, it can be inferred that this enzyme plays a more significant role in this group. Additionally, the ease of measuring OS markers in the serum of patients with FLD, as opposed to their liver tissue, presents an advantage that facilitates the use of these tests in clinical settings.

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Authors' Contribution

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Supervision: Sina Mohagheghi.
Validation: Sina Mohagheghi.
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Writing-review & editing: Sina Mohagheghi, Zahra Ali, Amir Mohammad Zargar.

Competing Interests

The authors declare no conflict of interests.

Ethical Approval

This study was approved by the Research Ethics Committee of Hamadan University of Medical Sciences (IR.UMSHA.REC.1403.602). All procedures were carried out following the 1964 Helsinki Declaration and the ethical guidelines established by the National Research Committee of Iran. Each participant was thoroughly informed about the purpose of the study, and written informed consent was obtained from all individuals prior to their participation.

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References

1. Pipitone RM, Ciccioli C, Infantino G, La Mantia C, Parisi S, Tulone A, et al. MAFLD: a multisystem disease. Ther Adv Endocrinol Metab. 2023;14:20420188221145549. doi: 10.1177/20420188221145549.
2. Allameh A, Niayesh-Mehr R, Aliarab A, Sebastiani G, Pantopoulos K. Oxidative stress in liver pathophysiology and disease. Antioxidants (Basel). 2023;12(9):1653. doi: 10.3390/antiox12091653.
3. Angelico F, Alcantara-Payawal D, Rani RA, Mustafa N, Thongtang N, Chaiteerakij R, et al. Review and expert opinion on MAFLD, oxidative stress and multifunctional management. Drugs Context. 2024;13:2023-9-3. doi: 10.7573/dic.2023-9-3.
4. Lin S, Huang J, Wang M, Kumar R, Liu Y, Liu S, et al. Comparison of MAFLD and NAFLD diagnostic criteria in real world. Liver Int. 2020;40(9):2082-9. doi: 10.1111/liv.14548.
5. Fan H, Liu Z, Zhang P, Wu S, Han X, Huang Y, et al. Characteristics and long-term mortality of patients with non-MAFLD hepatic steatosis. Hepatol Int. 2023;17(3):615-25. doi: 10.1007/s12072-023-10512-1.
6. Denkmayr L, Feldman A, Stechemesser L, Eder SK, Zandanell S, Schranz M, et al. Lean patients with non-alcoholic fatty liver disease have a severe histological phenotype similar to obese patients. J Clin Med. 2018;7(12):562. doi: 10.3390/jcm7120562.
7. Clare K, Dillon JF, Brennan PN. Reactive oxygen species and oxidative stress in the pathogenesis of MAFLD. J Clin Transl Hepatol. 2022;10(5):939-46. doi: 10.14218/jcth.2022.00067.
8. Świderska M, Maciejczyk M, Zalewska A, Pogorzelska J, Flisiak R, Chabowski A. Oxidative stress biomarkers in the serum and plasma of patients with non-alcoholic fatty liver disease (NAFLD). Can plasma AGE be a marker of NAFLD? Oxidative stress biomarkers in NAFLD patients. Free Radic Res. 2019;53(8):841-50. doi: 10.1080/10715762.2019.1635691.
9. Asghari S, Hamedi-Shahraki S, Amirkhizi F. Systemic redox imbalance in patients with nonalcoholic fatty liver disease. Eur J Clin Invest. 2020;50(4):e13211. doi: 10.1111/eci.13211.
10. Lü JM, Lin PH, Yao Q, Chen C. Chemical and molecular mechanisms of antioxidants: experimental approaches and model systems. J Cell Mol Med. 2010;14(4):840-60. doi: 10.1111/j.1582-4934.2009.00897.x.
11. Eslam M, Newsome PN, Sarin SK, Anstee QM, Targher G, Romero-Gomez M, et al. A new definition for metabolic dysfunction-associated fatty liver disease: an international expert consensus statement. J Hepatol. 2020;73(1):202-9. doi: 10.1016/j.jhep.2020.03.039.
12. Kang SH, Cho Y, Jeong SW, Kim SU, Lee JW. From nonalcoholic fatty liver disease to metabolic-associated fatty liver disease: big wave or ripple? Clin Mol Hepatol. 2021;27(2):257-69. doi: 10.3350/cmh.2021.0067.
13. Zargar AM, Tayebinia H, Hasanzarrini M, Bahiraei M, Mohagheghi S. Differential levels of thyroid hormones, cortisol, and apolipoprotein M in fatty liver disease. Horm Mol Biol Clin Investig. 2025. doi: 10.1515/hmbci-2024-0074.
14. Erel O. A new automated colorimetric method for measuring total oxidant status. Clin Biochem. 2005;38(12):1103-11. doi: 10.1016/j.clinbiochem.2005.08.008.
15. Yagi K. Assay for blood plasma or serum. Methods Enzymol. 1984;105:328-31. doi: 10.1016/s0076-6879(84)05042-4.
16. Celik SE, Ozyürek M, Güçlü K, Apak R. Solvent effects on the antioxidant capacity of lipophilic and hydrophilic antioxidants measured by CUPRAC, ABTS/persulphate and FRAP methods. Talanta. 2010;81(4-5):1300-9. doi: 10.1016/j.talanta.2010.02.025.
17. Pai SA, Munshi RP, Panchal FH, Gaur IS, Mestry SN, Gursahani MS, et al. Plumbagin reduces obesity and nonalcoholic fatty liver disease induced by fructose in rats through regulation of lipid metabolism, inflammation and oxidative stress. Biomed Pharmacother. 2019;111:686-94. doi: 10.1016/j.biopha.2018.12.139.
18. Cichoż-Lach H, Michalak A. Oxidative stress as a crucial factor in liver diseases. World J Gastroenterol. 2014;20(25):8082-91. doi: 10.3748/wjg.v20.i25.8082.
19. Zakaria Z, Othman ZA, Bagi Suleiman J, Che Jalil NA, Wan Ghazali WS, Nna VU, et al. Hepatoprotective effect of bee bread in metabolic dysfunction-associated fatty liver disease (MAFLD) rats: impact on oxidative stress and inflammation. Antioxidants (Basel). 2021;10(12):2031. doi: 10.3390/antiox10122031.
20. Zakaria Z, Othman ZA, Bagi Suleiman J, Che Jalil NA, Wan Ghazali WS, Mohamed M. Protective and therapeutic effects of orlistat on metabolic syndrome and oxidative stress in high-fat diet-induced metabolic dysfunction-associated fatty liver disease (MAFLD) in rats: role on Nrf2 activation. Vet Sci. 2021;8(11):274. doi: 10.3390/vetsci8110274.
21. Martín-Fernández M, Arroyo V, Carnicero C, Sigüenza R, Busta R, Mora N, et al. Role of oxidative stress and lipid peroxidation in the pathophysiology of NAFLD. Antioxidants (Basel). 2022;11(11):2217. doi: 10.3390/antiox11112217.
22. Esterbauer H, Schaur RJ, Zollner H. Chemistry and biochemistry of 4-hydroxynonenal, malonaldehyde and related aldehydes. Free Radic Biol Med. 1991;11(1):81-128. doi: 10.1016/0891-5849(91)90192-6.
23. Bellanti F, Villani R, Facciorusso A, Vendemiale G, Serviddio G. Lipid oxidation products in the pathogenesis of non-alcoholic steatohepatitis. Free Radic Biol Med. 2017;111:173-85. doi: 10.1016/j.freeradbiomed.2017.01.023.
24. Tsikas D. Assessment of lipid peroxidation by measuring malondialdehyde (MDA) and relatives in biological samples: analytical and biological challenges. Anal Biochem. 2017;524:13-30. doi: 10.1016/j.ab.2016.10.021.
25. Ore A, Akinloye OA. Oxidative stress and antioxidant biomarkers in clinical and experimental models of non-alcoholic fatty liver disease. Medicina (Kaunas). 2019;55(2):26. doi: 10.3390/medicina55020026.
26. Smirne C, Croce E, Di Benedetto D, Cantaluppi V, Comi C, Sainaghi PP, et al. Oxidative stress in non-alcoholic fatty liver disease. Livers. 2022;2(1):30-76. doi: 10.3390/livers2010003.