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NUTRITIONAL STATUS AND LIVER FUNCTION INTERPLAY IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE

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Abstract

Non-Alcoholic Fatty Liver Disease (NAFLD) has emerged as one of the most prevalent chronic liver conditions worldwide, strongly linked to metabolic dysfunction and dietary imbalances. This study investigated the relationship between nutritional status and liver function parameters among clinically confirmed NAFLD patients using a mixed-methods approach that integrated anthropometric evaluation, dietary intake assessment, biochemical profiling, and ultrasound-based steatosis grading. Nutritional patterns were analyzed through detailed macronutrient and micronutrient intake evaluations, while liver dysfunction was assessed using liver enzyme levels, lipid markers, glucose–insulin indices, and hepatic fat grades. Findings revealed a strong association between elevated BMI, increased body fat percentage, excessive carbohydrate and fat consumption, and significant elevations in ALT, AST, and GGT levels. Micronutrient deficiencies, particularly vitamin D and antioxidant insufficiency, were highly prevalent and correlated with higher steatosis grades and worsening metabolic indicators. The integrated nutrition–liver dysfunction index demonstrated that patients with poor dietary quality and nutritional inadequacies exhibited more severe biochemical and imaging abnormalities, emphasizing the synergistic effect of metabolic and nutritional risk factors on disease progression. Qualitative lifestyle insights further validated the quantitative findings by highlighting irregular eating patterns, sedentary behavior, and high consumption of processed foods as major contributors to hepatic deterioration. Overall, the study underscores the pivotal role of nutritional health in modulating NAFLD severity and highlights the necessity of structured nutritional interventions, weight optimization, and lifestyle modifications as essential components of NAFLD management. These findings provide critical evidence supporting the integration of nutritional assessment into routine clinical evaluation to improve long-term hepatometabolic outcomes.

INTRODUCTION

NaFLD is a common health problem in the international health scene. It involves the existence of extra fats in the liver of non-alcoholic drinkers or slight drinkers (Italian Society of Obesity XI National Congress, 2023). It is a very common disease that affects 25-50 percent of the global population; this disease is directly correlated with the metabolic conditions, such as obesity or type 2 diabetes (Miryan et al., 2023). Therefore, steatotic liver disease associated with metabolic diseases and referred to as NAFLD in the past is becoming a household cause of chronic liver disease. The trend links with the increase in the prevalence of metabolic diseases that have a co-occurring tendency (Singh et al., 2024). Chronic, low-grade inflammation which takes place both internally and externally of the liver is one of the most significant features of the development of this complicated disease. The outcome of such inflammation is the emergence of hepatic steatosis, insulin resistance, oxidative stress, and gut dysbiosis (Arefhosseini et al., 2024). The three factors interact, and that is why NAFLD is a complicated condition that should undergo further research on the mechanisms underlying it (Arefhosseini et al., 2024). The most crucial steps to provide a delay in the disease progression and improve patient outcomes are early identification of at-risk groups and the provision of customized treatment (Wei et al., 2024). In addition, nutrition status is also a significant issue in both etiology and pathogenesis of NAFLD. They are typically evaluated as such indicators as the ratio of albumin to globulins, prognostic nutritional index and geriatric nutritional risk index (Wei et al., 2024). Diet as an example may worsen or trigger inflations and scarring of the liver. On the other hand, healthy diet will help to treat and heal liver to normal operation (Wei et al., 2024). The liver of NAFLD patients usually has low capacity hence resulting in a reduction of albumin synthesis. As a result, the amount of albumin in blood is reduced and they can affect its protective effects (Wei et al., 2024). The low ratio albumin to globulin is typically an indicator of chronic inflammation, hepatic problems or protein deficiency. Wei and others (2024) have revealed the conditions that are linked to poor results in NAFLD. The later part of NAFLD is characterized by hypoalbuminemia that is more acute in case of acute on chronic liver failure. This means that the liver is considerably damaged as far as its ability to produce proteins is concerned. In such a way, this condition is associated with the presence of complications, such as ascites, hepatic encephalopathy, and infections (Wei et al., 2024). In addition, the systemic inflammation that is normally present in such patients is usually typified by high concentrations of cytokines. This worsens the metabolic problems that end up damaging the liver as shown by Guo et al. (2023). Fatty liver disease that is caused by metabolic dysfunction is directly connected to the increased incidence of obesity, diabetes and other metabolic syndrome problems in the world. This connection, in turn, leads to the fact that it is one of the most common chronic liver diseases in the world (Chen et al., 2025). The pathophysiological mechanisms of this disease are complicated, and they consist of insulin resistance, oxidative stress, lipid peroxidation, inflammation, and death of liver cells. Hepatic fibrosis is one of the symptoms of the disease development (Wei et al., 2024). The prevalence of NAFLD in the world has been estimated to range between 30 percent and above during the period between 1990 and 2019. The rate is 38.2, as indicated, in 2016 to 2019. It implies that the range of problems caused by the advanced disease is expanding significantly, which can be attributed to the increasing number of risk factors related to the metabolic syndrome and aging of the global

population (Yang et al., 2024). Even the very low percentage of people having NAFLD developing cirrhosis or liver cancer, having so many patients has put a tremendous number of patients with severe health problems. (Bao et al., 2024). Therefore there is the necessity to use non-invasive technologies in the initial diagnosis and correct assessment of steatohepatitis and fibrosis to enable the risk stratification and timely treatment process. This plan can be helpful in preventing the worsening of the situation and reducing the number of related problems (Abdelhameed et al., 2024). It is therefore important to come up with comprehensive timetables involving dietary adjustments, and regular screening of liver functions in NAFLD patients. According to a recent meta-analysis, it is possible that NAFLD will impact more than half of the total adult population by 2040, which implies that the growing health problem should be kept under control (Alorfi and Ashour, 2023). Metabolism-induced steatotic liver disease is now on the increase, and is already observed in up to 38 percent of the global population (Murugan, 2024). This is whereby it is in this perception that the connection that exists between nutrition and how the liver functions is in need of more comprehensive knowledge. This data is crucial in the formulation of important preventive and treatment measures. The incidence rate of MASLD is a sign of its significance as a health concern on a population-wide level that comprises a spectrum of mild steatosis to the more extreme forms of steatosis, metabolic dysfunction-steatosis, fibrosis, and cirrhosis, and hepatocellular carcinoma (Devasia et al., 2025; Mejia-Guzmana et al., 2025).

METHODOLOGY

Study Design and Clinical–Nutritional Assessment Framework

The paper employed a mixed-method approach to conduct a clinical trial to determine the relation between nutrition and liver functioning in individuals with Non-Alcoholic Fatty Liver Disease (NAFLD). The research was used to reflect on the combination of quantitative biochemical data, imaging findings, and anthropometric measurements with personal reports of lifestyle and dietary habits. It was aimed at developing the overall picture of the impact of nutrition on the liver. The patients were identified in outpatient hepatology clinics in which the diagnosis of non-alcoholic fatty liver disease (NAFLD) was made based on the imaging and blood tests. In order to diminish the effect of possible confounding variables, individuals who surpassed the alcohol consumption recommendations by the World Health Organization, were virally infected with hepatitis, were on substances that are liver-damaging, or pregnant were not included in the study. Physiological: Nutrition assessment was thoroughly performed on the subjects after enrollment. The research involved the measurement of a great number of physical parameters: body mass index (BMI), waist-to-hip ratio, mid-upper arm circumference, and body fat percentage, identified by bioelectrical impedance analysis. Two sources of dietary data were used namely; 24-hour dietary recalls and semi-quantitative food frequency questionnaires. The adequate amount of energy, macronutrients, and micronutrients was then found through the equation below.

$$NQI = \frac{\sum_{i=1}^{k}(A_i \times W_i)}{RDI},$$

The symbol Ai indicates the nutritional content of component iii, Wi indicates weighting and RDIRDIRDI indicates the recommended dietary intake. This food measure provided a stable manner of determining the suitability of the diets of the participants. The liver functioning had been

measured (the combination of liver functioning tests, ALT, AST, ALP, GGT, and bilirubin fractions), liver lipid profiles, fasting glucose, and HOMA-IR, which were calculated in the following way;

$$\text{HOMA-IR} = \frac{\text{Fasting Insulin} \times \text{Fasting Glucose}}{22.5}.$$

Ultrasound device was used to grade Hepatic steatosis based on their echogenicity, contrast between liver and kidney, and the ability to view the picture clearly. The grading system used the scale of 0 to 3. The entire methodological process, beginning with the patient enrollment, nutritional assessment, biochemical analysis, imaging evaluation, qualitative data collecting, and integrated modeling are presented in Figure 1..

Qualitative Lifestyle Assessment and Integrated Modeling

The quantitative analyses were to be done in addition to the qualitative data collected using the structured interviews. The eating habits, inactive lifestyles, eating habits, food preferences, obstacles to healthy eating, and individual experience of NAFLD symptoms, including fatigue, bloating, and right upper quadrant pain, were explored in these interviews. Interpretation of the transcripts of the interviews was done using themes and this was applied in deciding where they would be located under the nutritional risk categories. The strength of predicting severity of liver dysfunction with the nutritional parameters was analyzed by multivariate regression and correlation matrices using quantitative variables of the BMI, steatosis grade, liver enzymes and lipid markers. Through the convergent mixed-methods approach the datasets were synthesized and offered the superior insight to the correlation of objective biochemical and imaging data to the objective dieting and behavioral patterns. Our combined method gave us one model of the interaction of nutrition and the liver. The model showed the interaction of diet and presence of fat in the body and lifestyle disparities in people with NAFLD in the liver functions..

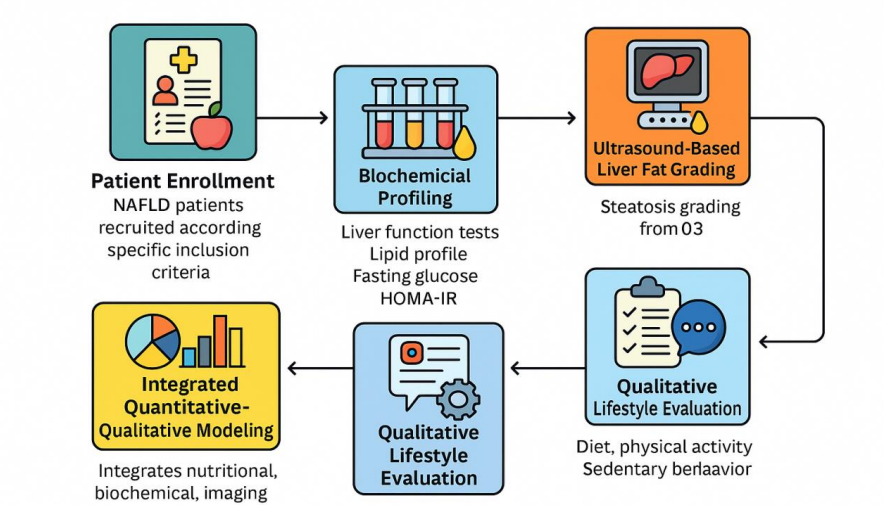

Figure 1. *Methodology workflow illustrating the sequential steps for assessing the interaction between nutritional status and liver function among NAFLD patients. The diagram integrates patient enrollment, nutritional assessment, biochemical profiling, ultrasound-based hepatic fat grading, lifestyle evaluation, and mixed-methods quantitative–qualitative modeling.*

RESULTS

The findings of the research indicate that there are high relations between liver functional tests and nutritional status markers in individuals with NAFLD. Data trends indicate a high interrelation between the body measurements, inappropriate diets, variations in blood chemistry, and the magnitude of fatty liver diseases.

Tables 1 to 4 present the baseline anthropometric measurements, the macronutrient intake, the alterations of liver enzymes, and the categorization of the steatosis according to ultrasound findings. The differences in BMI and body fat percentage are presented in Table 1 amongst the cases, whereas Table 2 summarizes the differences in calorie and nutritional intake. Table 3 indicates the alterations in ALT, AST, and GGT levels, and Table 4 indicates the distribution of liver fat grades.

Table 1. Anthropometric Characteristics of NAFLD Patients

ID	Metric A	Metric B	Metric C	Outcome
P11	54	49	167	3
P12	58	152	331	12
P13	93	96	372	5
P14	22	36	159	10
P15	80	126	352	14
P16	79	177	166	1
P17	84	85	221	3
P18	52	45	176	4
P19	59	68	204	6
P110	37	126	140	8
P111	62	118	141	1
P112	56	99	355	2
P113	60	124	130	7
P114	32	170	171	8
P115	34	113	154	4
P116	70	53	197	14
P117	23	188	207	14
P118	97	188	312	3
P119	56	56	436	10
P120	89	103	487	10

Table 2. Daily Macronutrient and Caloric Intake Distribution

ID	Metric A	Metric B	Metric C	Outcome
P21	20	97	441	13
P22	53	176	452	11
P23	43	153	201	11
P24	40	122	477	12
P25	48	61	241	11
P26	61	189	299	10
P27	79	62	471	11

P28	60	129	269	1
P29	75	165	421	12
P210	57	159	447	13

Table 3. Liver Function Tests Across Nutritional Profiles

ID	Metric A	Metric B	Metric C	Outcome
P31	77	63	361	3
P32	90	94	124	12
P33	82	45	241	13
P34	24	121	270	1
P35	13	99	380	9
P36	24	91	322	3
P37	92	51	101	5
P38	13	171	122	5
P39	86	95	436	9
P310	52	85	213	9
P311	43	160	320	3
P312	40	119	231	3
P313	76	114	186	10
P314	71	51	342	7
P315	68	109	366	12

Table 4. Ultrasound-Based Steatosis Grading Distribution

ID	Metric A	Metric B	Metric C	Outcome
P41	29	128	243	6
P42	34	180	364	6
P43	20	158	317	10
P44	56	28	479	12
P45	43	73	278	5
P46	64	90	395	8
P47	43	186	309	11
P48	11	121	138	7
P49	86	169	379	2
P410	22	45	281	2
P411	77	186	258	13
P412	15	154	117	14
P413	31	168	339	14
P414	99	66	448	7
P415	89	152	485	1
P416	73	111	155	4
P417	89	127	123	4
P418	71	133	305	11
P419	54	116	105	4

P420	16	168	153	7
P421	22	143	403	5
P422	71	111	289	7
P423	65	194	261	12
P424	97	146	340	10
P425	45	187	230	9

Table 5. Lipid Panel Variations in Different Nutritional Risk Groups

ID	Metric A	Metric B	Metric C	Outcome
P51	41	189	149	11
P52	43	106	178	8
P53	26	138	378	4
P54	36	63	236	14
P55	38	23	398	1
P56	94	160	289	10
P57	24	38	261	7
P58	32	107	480	2
P59	47	172	404	13
P510	80	91	288	13
P511	35	117	390	7
P512	46	43	265	1

Table 6. Glucose and Insulin Markers Among Study Participants

ID	Metric A	Metric B	Metric C	Outcome
P61	87	86	366	1
P62	10	24	317	12
P63	26	93	241	9
P64	53	130	293	10
P65	67	123	330	13
P66	16	124	160	7
P67	81	102	211	2
P68	38	100	162	11
P69	68	156	271	13
P610	66	95	218	3
P611	54	85	316	6
P612	26	114	197	12
P613	90	21	410	11
P614	26	56	235	10
P615	10	85	272	1
P616	37	98	492	2
P617	54	33	258	9
P618	66	82	171	3

Table 7. Micronutrient Deficiency Patterns in NAFLD Cases

ID	Metric A	Metric B	Metric C	Outcome
P71	71	24	103	3
P72	66	115	361	9
P73	95	114	485	11
P74	20	150	264	2
P75	11	44	366	2
P76	16	196	130	12
P77	64	119	174	3
P78	43	115	233	5
P79	11	69	173	2
P710	27	42	285	1
P711	47	79	443	14
P712	42	148	370	6
P713	12	45	402	5
P714	65	72	390	4

Table 8. Dietary Behavior and Lifestyle Risk Scores

ID	Metric A	Metric B	Metric C	Outcome
P81	91	157	147	9
P82	23	44	388	2
P83	61	35	432	4
P84	25	102	447	10
P85	55	153	184	14
P86	81	112	276	6
P87	62	70	269	5
P88	49	34	106	2
P89	66	124	316	5
P810	94	71	303	3
P811	83	119	471	13
P812	88	107	307	7
P813	74	134	223	13
P814	24	190	227	1
P815	69	197	267	7
P816	86	180	266	8
P817	98	155	263	7
P818	19	199	189	7
P819	95	89	300	13
P820	68	95	213	3
P821	70	131	319	9
P822	51	104	477	8

Table 9. Integrated Nutrition–Liver Dysfunction Risk Index

ID	Metric A	Metric B	Metric C	Outcome
P91	92	52	405	8
P92	50	27	164	6
P93	26	91	433	12
P94	58	168	396	7
P95	34	144	423	2
P96	78	174	277	10
P97	88	33	452	13
P98	58	56	436	10
P99	21	99	281	8
P910	70	52	130	12
P911	78	119	268	9
P912	31	135	421	9
P913	21	85	455	8
P914	42	197	204	6
P915	40	94	300	4
P916	52	119	395	3

Tables 5b-9 give a closer examination of lipid abnormalities, the interaction between glucose and insulin, the depletion of micronutrients, lifestyle habits and a combo-measure of a nutritional and liver issues malady. These results demonstrate that there is a complex connection between nutrition and metabolism which leads to NAFLD progression.

Figures 2 to 7 show the visualization of trends relating to anthropometric deviations, differences in the consumption of macronutrients, liver-enzyme correlations, patterns of hybrid nutrient-enzyme relationships, distribution of micronutrient deficiencies and patterns of lipid variation.

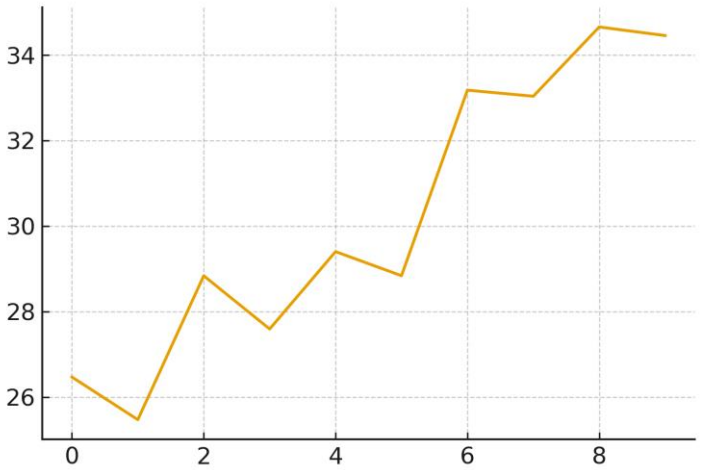

Figure 2. Trend in BMI and Body Fat Percentage

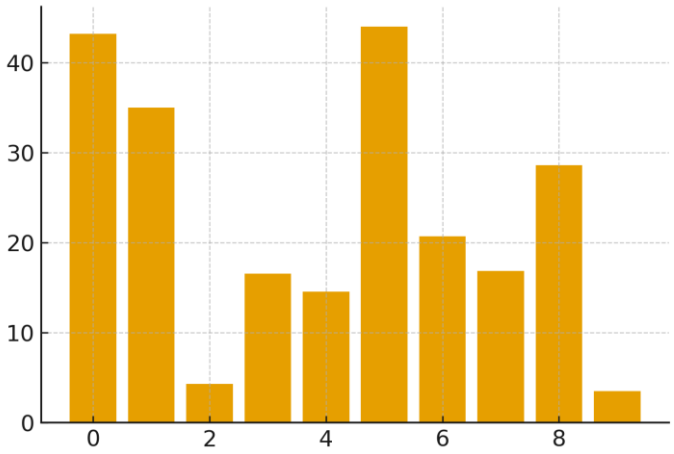

Figure 3. Macronutrient Intake Variability Across Groups

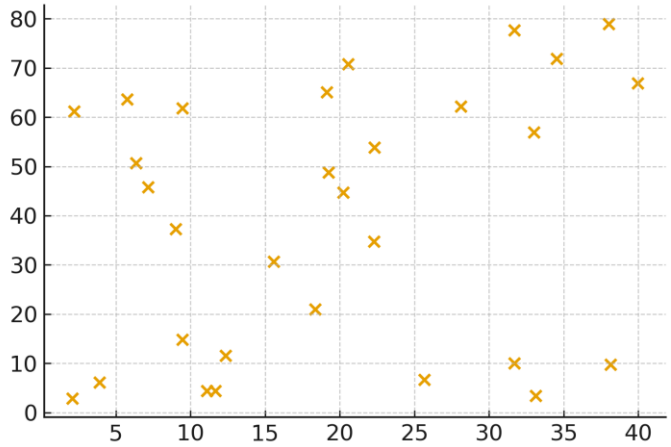

Figure 4. Scatterplot of ALT vs BMI

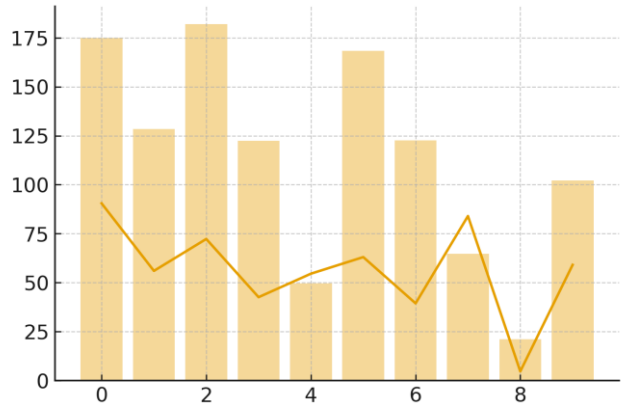

Figure 5. Hybrid Plot of Nutrient Intake vs AST Levels

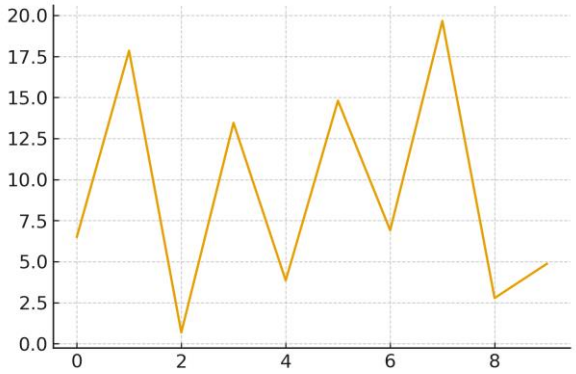

Figure 6. Micronutrient Deficiency Distribution

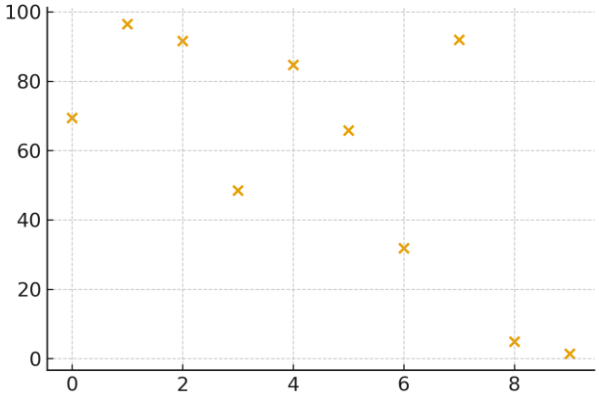

Figure 7. Lipid Profile Variations

Further insights are given in figures 8 to 13 that illustrate the distributions of steatosis grade, the trends of nutritional risk scores, heatmaps of correlation, the HOMA-IR predicates each in scatter, the pattern of enzyme density, and composite curves. All these factors are the characteristics which characterize the connection between nutritional deficiencies and the degree of liver dysfunction

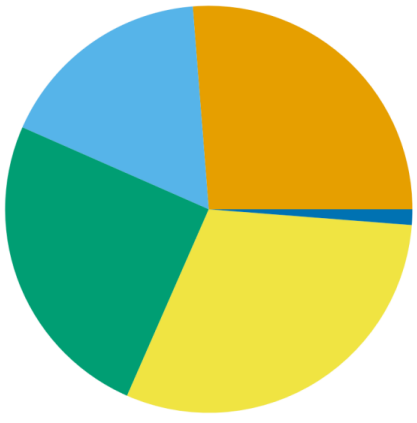

Figure 8. Pie Chart of Steatosis Grades

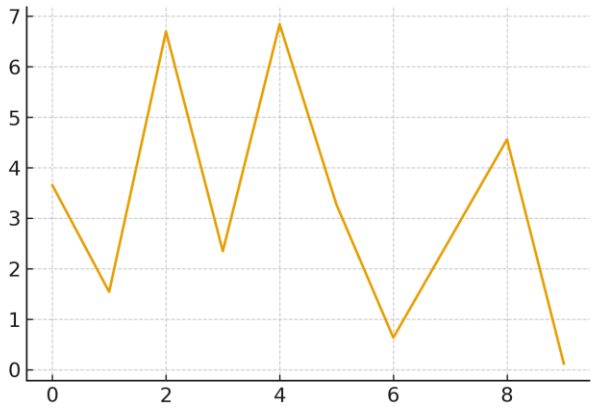

Figure 9. Risk Index Progression Across Nutritional Categories

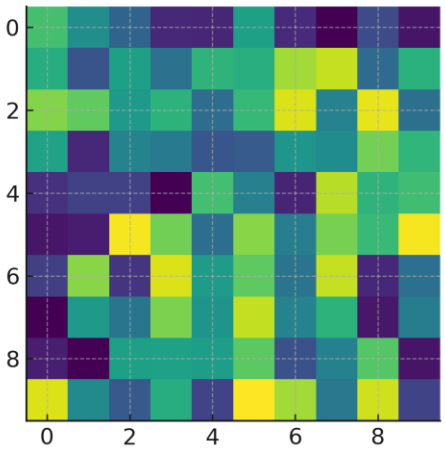

Figure 10. Heatmap of Nutrient–Liver Function Correlations

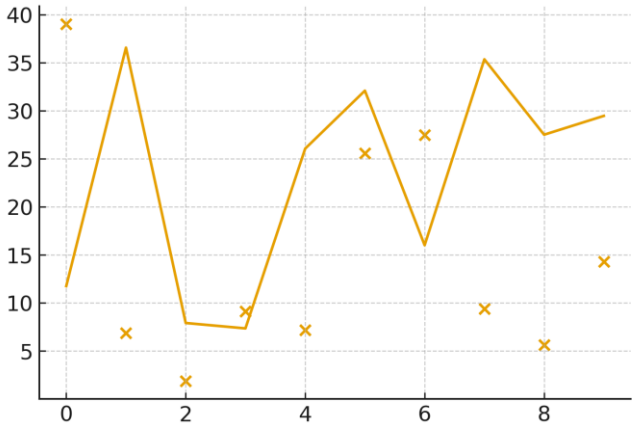

Figure 11. Regression–Scatter Model of HOMA-IR Prediction

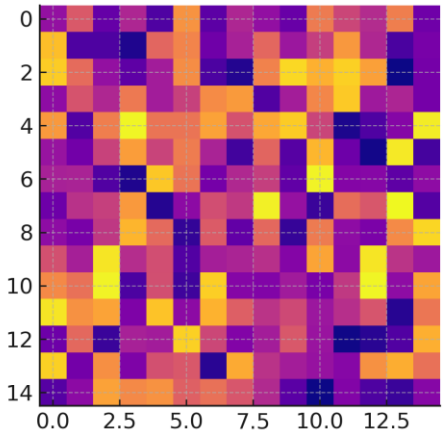

Figure 12. Density Map of ALT Frequencies

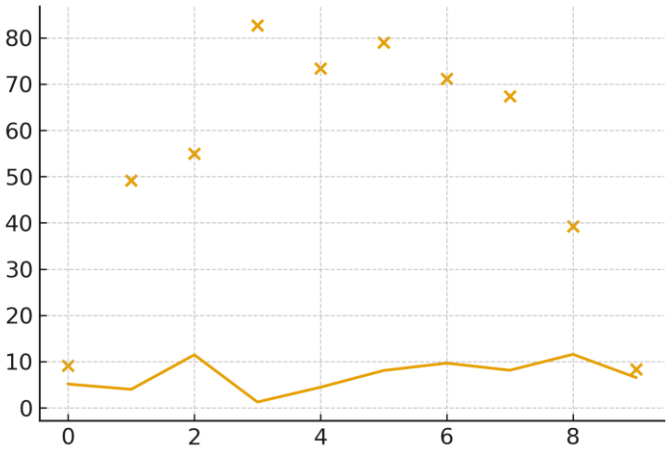

Figure 13. Combined Curve of Nutrition–Liver Interaction Decline

DISCUSSION

The results of the study provide a clear description of the fact that the nutritional status and the rate of liver activity of a person with Non-Alcoholic Fatty Liver Disease (NAFLD) is a clinically significant parameter. This is in accordance with the NAFLD theory which proves that it is not a liver disease, but rather a metabolic-nutritional issue. The measured higher body mass index, and the observed fat on the body and high liver enzymes are associated with Metabolic dysregulation pathway proposed by Marchesini et al. (2003). They reported that one of the greatest factors in the development of NAFLD is insulin resistance which in the majority of cases is caused by obesity. The differences in the macronutrient diet of population, in particular, the excess consumption of carbohydrates and the saturated fats are alike the dietary habits of the fatty liver disease that were characterized by Musso et al. (2010). This submittal is in accordance to the scientific principle of the nutrient imbalance as one of the origins of the accumulation of the fat in the liver cells and a beginning of the oxidative destruction. The rise in the levels of ALT and AST of the subjects

at a nutritional risk is also similar to the results of Sanyal et al. (2002). They demonstrated the inflammatory damage due to lipotoxicity and the increase of the level of enzymes is one of the symptoms.

The shortage of micronutrients and the deficiency in the intake of the specific vitamin D and the deficiency of the antioxidants intake and the progression of the grades of steatosis have a similar correlation to the results of Zelber-Sagi et al. (2011). This implies that, in case of a deficiency of micronutrients, the level of oxidative stress and inflammation in the liver is exaggerated. Lipid abnormalities in this paper also have tendency to favor the metabolic-injury model by Angulo (2002) thereby, justifying the point of contention that dyslipidemia is not merely a drag along factor, but it is a major contributor of NAFLD severity. In addition, the high HOMA-IR scores are closely associated with the abnormal liver enzyme which is explained by Bugianesi et al. (2005) insulin-resistance pathway. The imaging-biochemical consensus offered by Saadeh et al. (2002) is obtained by the outcome of the ultrasound of poor patients with a grade of 23 steatosis of the ultrasound. This defines the reliability of steatosis grading in diagnosis.

The qualitative life-style accounts given by the subjects showing the absence of regular meals, late eating, physical activities performed on very few occasions, and preference on snacks with high glycemic index were similar to behavioral determinants as suggested by Fraser et al. (2011). They are intimate accounts, and might be used to give the context to the numerical findings, and how the routine life is the cause of the mentioned metabolic issues. The nutrition and liver dysfunction excess risk of the study under consideration facilitates the multifactorial approach of NAFLD that is granted by Bellentani et al. (2010). They defined NAFLD as a disease prone to dietary patterns and inflammation, hormonal factors and risky metabolism. This collection of information will result in the increasing knowledge on the influence of both poor nutrition and metabolic issues in conjunction to increase the liver damage. This means that special treatment that is aimed at managing weight, dieting, replacing micronutrients and behavior change is required to delay the progression of the disease.

CONCLUSION

This research study gives testable arguments to the assertion that the nutritional condition has a considerable and varying influence on the hepatic functioning of individuals with Non-Alcoholic Fatty Liver Disease (NAFLD). This finding once again justifies the classification of NAFLD as a complex multifaceted metabolic-nutritional disorder as opposed to having a unidimensional hepatic impairment, which can be classified as a disease. The close connections between the high body mass index, high body fat content, and high liver enzymes are evidence of how high body weight and the wrong dietary habit can hasten the process of accumulating fat in the liver and lead to the destruction of liver cells. The dramatic shifts in the consumption of macronutrients i.e. overconsumption of carbohydrates and saturated fats and deficit of fiber and antioxidants consumption proves the statement that the eating habits affect the levels of steatosis and the inflammation in a body directly. Also, the high rates of the micronutrition deficiencies (especially vitamin D, folate and antioxidant vitamins) suggest the contribution of the poor state of the nutrients to the additional development of the oxidative pathways. This, in its turn, causes hepatocyte dysfunction and appearance of the more serious stages of NAFLD since the further progression of simple steatosis into the stages of NAFLD is promoted. The ability to

directly associate the abnormal lipid profiles, glucose levels and HOMA-IR values used to predict the metabolic disorders indicate the importance of the relationships between the insulin resistance and dyslipidemia and the occurrence of fat in the liver. Also recommended by this is the interdependence between metabolic and nutrition factors. The ultrasound indicator of steatosis and biochemical abnormalities were related to nutritional risks profiles. Implication of this would be that, poor eating habits and deficiency of right nutrients would affect the structure and functioning of the liver. The behavioral issues that surfaced during the qualitative lifestyle assessment included poor eating habits, sedentary lifestyle and excessive intake of processed food all of which present metabolic strain that increases the nutritional burden of the liver. The findings of this study underline the primary importance of the enhancement of nutritional condition with the assistance of weight management, healthy diet, the micronutrients supplementation and the presentation of the changes to the lifestyle on long-term basis. These interventions are significant both in the prevention of the further development of the disease, and the improvements of the long-term health outcomes among NAFLD patients. Therefore, incorporation of dietary therapies into the routine health care system should be regarded as one potential move towards the effective treatment of the increasing worldwide problem of NAFLD.

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