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Comparison of Clinico-Biochemical and Transient Elastography Findings Among Diabetic and Non-Diabetic Patients with Non-Alcoholic Fatty Liver Disease

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Abstract

Non-alcoholic fatty liver disease (NAFLD) is the commonest chronic liver disease. It is characterized by a wide range of symptoms like fatigue, pain, or dullness in the upper right quadrant of the abdomen or none at all. The risk factors linked to NAFLD are type 2 diabetes mellitus (T2DM), obesity, and genetic predisposition. In this study, the clinical, biochemical, and ultrasonography findings were compared in NAFLD patients with and without T2DM. The liver elastography (FibroScan) measured liver stiffness (LSM), and the liver fibrosis index 4 (FIB-4) were both significantly higher in diabetic patients. The controlled attenuation parameter (CAP) was not statistically significant when compared in the two groups. There was a significant negative correlation between FIB-4 and platelet counts in both groups; p = 0.006 in non-diabetics and p = 0.001 in diabetics. There was a significant positive correlation between FIB-4 and aspartate aminotransferase (AST) in the non-diabetic group only. The non-invasive diagnostic and prognostic markers of NAFLD that are LSM, and FIB-4 are more reliable than inflammatory markers. Though the dietary habits, anthropometric measurements, and physical activity were not different and did not pose risk factors in our population, we need to study the genetic and epigenetic factors in our population.

Keywords: NAFLD, liver stiffness, CAP, inflammatory markers, DM

Abbreviations: NAFLD: non-alcoholic fatty liver disease; T2DM: type 2 diabetes mellitus; LSM: liver stiffness measurement; FIB-4: fibrosis index 4; DM: diabetes mellitus; CAP: controlled attenuation parameter; AST: aspartate aminotransferase; NASH: non-alcoholic steatohepatitis; HCC: hepatocellular carcinoma; LDL: low-density

lipoprotein; FBS: fasting blood sugar; PPBS: postprandial blood sugar; HbA1c: glycated hemoglobin; ALT: alanine aminotransferase; CBC: complete blood count; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; IEC: Institutional Ethics Committee; HIV: human immunodeficiency virus; LFT: liver function test; KFT: kidney function test

Introduction

The commonest chronic liver disease worldwide is a non-alcoholic fatty liver disease (NAFLD) [1] characterized by a wide range of symptoms like fatigue, pain, or dullness in the upper right quadrant of the abdomen or none at all [2]. NAFLD can range from simple steatosis to steatohepatitis, fibrosis, and cirrhosis [3]. NAFLD and type 2 diabetes mellitus (T2DM) are two diseases that coexist frequently and there is a bidirectional relationship between the two conditions. T2DM is an important and established risk factor for the progression of NAFLD to non-alcoholic steatohepatitis (NASH), cirrhosis, or hepatocellular carcinoma (HCC) [4].

In the past century, there has been a major lifestyle change, that is, it is moving towards high-fat, high-sugar diets, and sedentary lifestyles and this has affected human metabolic status radically [4]. There is accumulating evidence that indicates high-calorie diets, especially those rich in cholesterol, fructose, saturated and trans fatty acids, increase visceral fat, central obesity, and also increased risk of NAFLD. Studies have estimated that approximately 70–75% of patients with T2DM also have fatty liver disease [5–7]. Checking liver fibrosis is essential for monitoring the prognosis in patients with NAFLD, especially among high-risk patients such as diabetes [5]. Liver biopsy is the gold standard diagnostic method for histological assessment of NAFLD [8]. However, due to its invasiveness, it cannot be used in routine clinical practice. Other methods that are available to reach the diagnosis are biochemical studies like measuring lipid profiles. Recently, transient elastography (FibroScan) has emerged as one of the best modalities to screen NAFLD in patients with diabetes. A recent feature added to the FibroScan device is the possibility of quantifying liver fat. Since this is one of the characteristics of NAFLD, assessment of steatosis is therefore important [9]. There are only a few studies from India with regard to the use of FibroScan for NAFLD screening [3, 7, 10–12]. This study is an attempt to see the prevalence of NAFLD in diabetics compared to non-diabetics using biochemical parameters and FibroScan. The primary and secondary objectives of this study were to compare the clinico- biochemical parameters between diabetic and non-diabetic NAFLD patients and analyze the association of various lifestyle parameters with the degree of NAFLD respectively.

Methodology

This study was a cross-sectional observational study conducted in the General Medicine Department of our Tertiary Care Institute after obtaining IEC approval. The participants were consecutive diabetic and non-diabetic patients visiting the outpatient clinic of the General Medicine Department, diagnosed with having fatty liver on ultrasound of the abdomen and fulfilling the selection criteria.

The inclusion criteria were as follows: NAFLD patients within the age group of 20–65 years, whose alcohol intake was less than 20 g per day. They were negative for hepatitis B, C, and HIV and had normal renal function. Patients with decompensated cirrhosis of the liver, who had any infections or any connective tissue disorder or thyroid disease, or who were on any steroid medications were excluded from this study.

The sample size was calculated considering a 15% difference in low-density lipoprotein (LDL) cholesterol levels in two groups with an acceptable alpha error of 0.05 and a beta error of 0.2 (power of the study being 80%). We got a sample size of 21 in each one of the two groups (diabetic and non-diabetic). The statistical analysis was performed to compare continuous variables by t-test and Mann-Whitney U test for the dichotomous parameters. The detailed clinical record was obtained from each participant which included their dietary habits and lifestyle, biochemical, serological, and FibroScan reports.

Results

The physical and biochemical parameters were compared in the two groups (Table 1); the diabetes mellitus patients *vs.* the non-diabetics showed no significant difference in age, BMI, hematocrit values, kidney and liver function tests, and lipid profile. There was a significant difference in the glycemic control parameters that is fasting blood sugar (FBS) ($p = 0.006$), postprandial blood sugar (PPBS) ($p = 0$), glycated hemoglobin (HbA1c) ($p = 0$), and fasting insulin ($p = 0$), with levels higher in the group with diabetes mellitus. The liver elastography (FibroScan) measured the liver stiffness (LSM) and the controlled attenuation parameter (CAP). The liver fibrosis index 4 (FIB-4) was calculated using biochemical parameters like aspartate aminotransferase (AST), alanine aminotransferase (ALT), and platelet count and age of each subject [13]. The LSM and FIB-4 were significantly higher in the diabetic patients (Table 1). Among the liver function tests, serum total and indirect bilirubin levels were higher in the group without diabetes ($p = 0.021$ and 0.022 respectively). In the table (Table 2), the general dietary practices and habits compared showed no significant difference in the two groups. The correlation of glycemic control indices (Table 3) with complete blood count (CBC), liver and kidney function tests (LFT, KFT), and fasting lipid profile in the group with diabetes mellitus showed a significant positive result of platelet counts with duration of diabetes, FBS, PPBS, and HbA1c and a significant negative correlation of PPBS with total and indirect bilirubin, uric acid, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Fasting insulin had a significant negative correlation with VLDL. The risk assessment of FIB-4 done by the Kruskal-Wallis test was significant ($p = 0.004$) in the non-diabetic group of NAFLD but not in the diabetic group (Table 4). The table 5 shows the heat map of correlation showing negative in red and positive in green. There was a significant negative correlation between FIB-4 and platelet counts in both groups; $p = 0.006$ in non-diabetics and $p = 0.001$ in diabetics. There was a significant positive correlation between FIB-4 and AST in the non-diabetic group only.

	No DM		**With DM**		**P***
	Mean	**Std. Deviation**	**Mean**	**Std. Deviation**	
Age (years)	39.35	10.90	42.76	10.85	0.32
BMI (Kg/m^2)	28.72	5.95	28.78	4.23	0.87
WHR	1.04	0.06	1.03	0.08	0.61
Hematocrit (%)	39.53	5.22	41.71	5.72	0.67
RBC count ($10^6/L$)	4.59	0.67	4.53	0.57	0.21
WBC count ($10^9/L$)	7.41	3.03	7.53	1.89	0.76
Platelet count ($10^9/L$)	229.45	96.86	308.70	79.04	0.88
FBS (mg/dl)	99.37	14.72	146.92	24.08	**0.01**
PPBS (mg/dl)	130.23	18.53	287.53	60.37	**0.00**
HbA1c (%)	5.49	0.56	6.92	0.26	**0.00**
Fasting insulin (mIU/L)	13.66	9.63	21.67	32.15	**0.00**
Urea (mg/dl)	22.90	7.68	22.81	8.42	0.30
Creatinine (mg/dl)	0.96	0.23	0.96	0.21	0.97
Uric acid (mg/dl)	5.20	1.69	4.46	1.26	0.94
ALT (IU/L)	60.79	47.13	62.76	41.48	0.12
AST (IU/L)	45.51	31.43	42.19	20.98	0.89
Total bilirubin (mg/dl)	0.97	0.42	0.71	0.27	**0.02**
Indirect bilirubin (mg/dl)	0.67	0.33	0.46	0.20	**0.02**
Triglyceride (mg/dl)	197.41	52.29	183.51	48.15	0.69
LDL (mg/dl)	124.10	47.10	124.75	30.91	0.38
VLDL (mg/dl)	48.02	12.11	42.40	16.28	0.96
HDL (mg/dl)	46.86	10.47	46.60	11.13	0.22

Serum ferritin (ng/ml)	120.49	92.09	147.69	71.71	0.94
ESR (ml/Hr)	26.68	20.57	12.81	7.90	**0.01**
CRP (mg/dl)	2.70	2.02	4.01	2.61	0.09
CAP (dB/m)	387.55	459.98	287.90	42.43	0.29
LSM (kPa)	4.90	1.03	6.90	2.13	**0.00**
FIB-4	0.79	0.34	1.13	0.59	**0.03**

Table 1: Biochemical and physical parameters compared in the two groups. CAP*:* controlled attenuation parameter is expressed in decibels per meter; LSM: liver stiffness measurement is expressed in kilopascals; FIB-4: liver fibrosis index.

		Without DM	**With DM**	**Total**	**P for chi-square**
USG	Grade 1	12	7	19	0.121
	Grade 2	8	14	22	
Carbohydrate	Rice	14	15	29	0.92
	Wheat	6	6	12	
Diet	Veg	6	5	11	0.734
	Non-veg	14	16	30	
Oil	Mustard	11	13	24	0.331
	Sunflower	7	8	15	
	Other saturated fats	2	0	2	
Beverages	None	9	6	15	0.486
	Tea	9	11	20	
	Coffee	2	4	6	
Jaggery	No	3	4	7	0.529
	Yes	17	17	34	
Added salt	No	16	18	34	0.697
	Yes	4	3	7	
Physical activity	Sedentary	6	7	13	0.543
	Moderate	14	14	28	
Gender	Male	14	16	30	0.655
	Female	6	5	11	

Table 2: Frequency distribution table for general practices and habits.

	Duration of DM		**FBS**		**PPBS**		**HbA1c**		**Fasting insulin**	
	r	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**
Duration of DM			0.772	**0**	0.873	**0**	0.86	**0**	0.169	0.146
BMI	0.006	0.485	0.041	0.399	0.069	0.334	-0.043	0.394	-0.021	0.449
Hb	0.069	0.335	0.097	0.273	0.148	0.178	-0.061	0.353	0.097	0.272
Hematocrit	0.2	0.105	0.102	0.262	0.079	0.311	0.317	**0.022**	0.069	0.333
RBC count	-0.048	0.382	0.175	0.137	0.084	0.301	0.062	0.35	0.025	0.438
WBC count	0.024	0.441	-0.099	0.269	-0.074	0.323	-0.114	0.24	0.136	0.199
Platelet	0.418	**0.003**	0.44	**0.002**	0.424	**0.003**	0.39	**0.006**	-0.254	0.055
Urea	-0.006	0.486	-0.043	0.396	-0.018	0.455	-0.027	0.434	0.016	0.46
Creatinine	0.011	0.472	0.032	0.422	-0.043	0.394	0.054	0.369	-0.014	0.465
Uric acid	-0.246	0.06	-0.069	0.335	-0.327	**0.018**	-0.08	0.31	-0.254	0.055
ALT	0.023	0.444	0.031	0.423	0.027	0.432	-0.044	0.392	0.089	0.289
AST	-0.064	0.346	0.017	0.459	-0.062	0.35	-0.105	0.256	0.007	0.482
Tot. bilirubin	-0.357	**0.011**	-0.256	0.053	-0.366	**0.009**	-0.261	**0.05**	0.041	0.399
Indirect bilirubin	-0.361	**0.01**	-0.259	0.051	-0.335	**0.016**	-0.292	0.032	0.113	0.24

Triglyceride	-0.14	0.191	-0.054	0.37	-0.024	0.441	-0.108	0.251	-0.035	0.413
LDL	0.008	0.479	-0.031	0.423	0.064	0.345	0.049	0.382	0.086	0.297
VLDL	-0.196	0.11	-0.405	**0.004**	-0.2	0.105	-0.28	0.038	0.336	**0.016**
HDL	-0.012	0.47	-0.001	0.497	0.137	0.196	-0.043	0.395	-0.048	0.384
Ferritin	0.167	0.148	0.052	0.373	0.2	0.105	0.064	0.345	-0.143	0.186
ESR	-0.418	**0.003**	-0.327	**0.018**	-0.405	**0.004**	-0.379	**0.007**	-0.126	0.216
CRP	0.275	**0.041**	0.048	0.382	0.298	**0.029**	0.277	**0.04**	0.09	0.289
CAP	-0.156	0.164	-0.116	0.235	-0.133	0.204	-0.179	0.132	-0.052	0.374

Table 3: Correlation of glycemic parameters with history of DM and all other lab findings.

		Risk group with FIB-4	**N**	**Mean rank**	**p[a]**
No DM	FIB-4	Low	15	8	0.004
		Moderate	4	17.5	
		High	1	20	
		Total	20		
DM	FIB-4	Low	20	10.5	0.099
		Moderate	1	21	
		Total	21		

Table 4: Risk assessment using categories of FIB-4 in non-diabetic and diabetic patients. a: Kruskal-Wallis Rank test.

	No DM		**DM**	
	FIB-4		**FIB-4**	
	r	**p**	**r**	**p**
FBS	0.425	0.062	-0.173	0.453
PPBS	-0.274	0.242	-0.172	0.456
HbA1c	0.207	0.381	-0.039	0.865
Fasting insulin	0.418	0.066	0.191	0.406
Platelet	-.596**	0.006	-.660**	0.001
ALT	0.393	0.086	-0.062	0.791
AST	.580**	0.007	0.123	0.596
HDL	0.228	0.335	-0.086	0.711
ESR	-0.107	0.655	-0.152	0.511
CRP	-0.339	0.144	-0.120	0.603
CAP	-0.272	0.246	0.162	0.483
LSM	0.227	0.335	-0.118	0.61

Table 5: Correlation of glycemic and NAFLD parameters with FIB-4 in both groups.

Discussion

The commonest liver disease in adults is NAFLD which is associated with risk factors like obesity, dyslipidemia, and diabetes mellitus. Our findings indicated that there was no difference in markers of obesity (BMI, WHR) or the fasting

lipid profile compared in the two groups of NAFLD; the non-diabetics *vs.* the diabetics. However, there was a decrease in the total and indirect bilirubin in the diabetes mellitus patients. Bilirubin, a toxic metabolite of heme catabolism, is handled by the liver by converting it to a nontoxic and soluble compound. Bilirubin is known for its antioxidant and anti-inflammatory effects [14, 15] and has a cardio-protective role [16, 17]. Due to these reasons, it is seen that the serum bilirubin is inversely proportional to the development of NAFLD [18, 19], which was similar to our findings. Among the markers of inflammation, there was no difference in WBC count, CRP, and ferritin in the two groups compared but the latter two were higher than the normal limits. ESR was lower in the diabetics with NAFLD though it is expected to rise with liver stiffness, probably because of variability in immune response and/or genetic profile or due to anti-inflammatory actions of medications used in the management of DM [20, 21]. CAP, a measure of hepatic steatosis, and LSM, a measure of liver fibrosis were reciprocal in non-diabetics and DM patients [4, 10]. By combining transient elastography with CAP, clinicians can obtain information on both liver stiffness (indicative of fibrosis) and the degree of hepatic steatosis in a non-invasive manner.

The diet in the form of complex carbohydrates, vegetarians or beverages, and physical activity did not show any significant difference in the FibroScan findings in the two study groups in our study. A beneficial effect on NAFLD can be achieved with the combination of physical activity, weight loss, intake of useful phytochemicals, and improving the gut microbiome [22]. The relationship between FIB-4 and diabetes mellitus is more nuanced. In our study, FIB-4 was a significant risk factor among non-diabetic subjects but not so in DM. It is suggested that once NAFLD is detected, FIB-4 can be used as a prognostic marker to assess the disease progression and treatment outcomes [13, 23]. As FIB-4 calculation uses platelet count in the denominator, they have an inverse relation as seen in our study. It is related to the fact that liver fibrosis can lead to portal hypertension [24]. One consequence of portal hypertension is splenic sequestration of platelets. The spleen acts as a reservoir for platelets, and when portal hypertension occurs, it can trap and sequester platelets, reducing the overall platelet count in the peripheral blood [25, 26]. Though both the groups showed an elevation of AST levels, it was statistically significant only in the non-diabetics. It could be explained as an increase in insulin sensitivity due to the treatment of DM or by the fact that a significant population of hepatocytes is replaced by fibrocytes to give slightly elevated or normal AST levels [9, 27].

Limitations of the study include our sample size calculation based on LDL cholesterol, which was not different in the two groups assessed. The drug history of the DM and non-diabetics was not considered. The disease progression and severity of NAFLD can be variable in individuals due to genetic predisposition and epigenetic changes. It will facilitate answering such queries in larger studies considering the confounding factors listed above.

Conclusion

The non-invasive diagnostic and prognostic markers of NAFLD that are LSM and FIB-4 are more reliable than inflammatory markers. Though the dietary habits, anthropometric measurements, and physical activity were not different and did not pose risk factors in our population, we need to study the genetic and epigenetic factors in our population.

Conflicts of Interest

The authors declare that there are no conflicts of interest in any form; personal, professional, or financial to the conduct and publishing of this study.

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