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Original Article

Biochemistry Section

Correlation of Glycaemic Control and BMI with Renal Profile in Type 2 Diabetic Patients with and without Non Alcoholic Fatty Liver Disease: A Case-control Study

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ABSTRACT

Introduction: Obesity is a risk factor for the development of diabetes, and these two are directly implicated in an individual's risk of developing Non Alcoholic Fatty Liver Disease (NAFLD), which is a major factor in Metabolic Syndrome (MS). NAFLD and Type 2 Diabetes Mellitus (T2DM) are known to frequently coexist and act synergistically to elevate the risk of hepatic as well as extrahepatic complications.

Aim: To determine the levels of renal profile, electrolytes, Glycosylated Haemoglobin (HbA1c), and Body Mass Index (BMI) in T2DM patients with and without NAFLD, as well as in control subjects, and to assess the correlation of BMI and HbA1c with renal profile and electrolytes in T2DM patients with and without NAFLD.

Materials and Methods: This case-control study was conducted in the Department of Biochemistry and the Diabetes Speciality Clinic in the Department of General Medicine at MGM Medical College and Hospital, Navi Mumbai, Maharashtra, India, from December 2021 to March 2023. The study included a total of 90 subjects divided into three groups (30 in each): Group 1-Control, Group 2-T2DM with NAFLD, and Group 3-T2DM without NAFLD. Aseptic blood collection was performed, and Renal Function Test (RFT), electrolytes, and HbA1c levels were analysed. Group comparisons were done using unpaired t-tests, and correlation analysis was conducted using Pearson's correlation with Statistical Package for Social Sciences (SPSS) software version 25.0.

Results: The authors found that 57% of the total enrolled population were female, while the remaining 43% were male, with a mean age of (49.03±5.09) years. Mean levels of HbA1c (9.55±1.78, 8.61±1.42%), BMI (28.84±4.19, 23.51±2.09) kg/m^2, Urea (31.62±6.28, 33.02±5.11) mg/dL, Creatinine (1.29±0.18, 1.36±0.10) mg/dL, and Uric acid (6.74±1.19, 6.01±0.83) mg/dL were found to be significantly higher in Group 2 and Group 3, respectively, compared to controls. A positive significant correlation of BMI with uric acid, HbA1c with urea, creatinine, and uric acid in Group 2 and 3 was observed. However, no derangement was observed concerning electrolytes in any group.

Conclusion: The correlation of urea, creatinine, and uric acid with HbA1c provides the authors with information on impaired renal function in diabetic as well as NAFLD participants. Hyperuricaemia in these individuals can aggravate the risk of T2DM and NAFLD, leading to its progression in Non Alcoholic Steatohepatitis (NASH), respectively.

Keywords: Body mass index, Creatinine, Electrolytes, Fasting glucose, Hepatic disorder, Metabolic syndrome

INTRODUCTION

There has been a rapid increase in the prevalence of Type 2 Diabetes Mellitus (T2DM) worldwide. According to the latest International Diabetes Federation (IDF) report, 537 million adults (10.5% of the global population) are living with diabetes mellitus worldwide, with 90 million residing in Southeast Asia [1]. Diabetes mellitus is a common yet potentially devastating group of metabolic illnesses characterised by hyperglycaemia brought on by defects in insulin secretion, insulin action, or both [2,3].

Obesity is a major risk factor, along with other genetic, environmental, and psychosocial factors, for the development of diabetes mellitus. Diabetes-related chronic hyperglycaemia contributes to long-term damage and dysfunction of many organs, particularly the heart, blood vessels, nerves, eyes, and kidneys [4].

Obesity often correlates with MS, a condition associated with pro-inflammatory states and considered to represent a collection of risk factors. The diagnosis of MS is made when any three of the following five risk factors are present: central obesity, high blood pressure, loss of glycaemic control, low serum High-density Lipoprotein (HDL), and high serum triglycerides [4]. Therefore, obesity and T2DM together increase an individual's risk of developing NAFLD. An international panel has now named it Metabolic Associated Fatty Liver Disease (MAFLD) [5].

Numerous studies have shown the prevalence rate of NAFLD to be around 9-32% in the general Indian population, with a higher prevalence among obese and diabetic individuals [6,7]. The prevalence rate of NAFLD in T2DM is expected to range from 12.5% to 87.5% in India [8]. NAFLD is one of the most common liver disorders and has grown to become a global public health concern. It develops when fat accounts for more than 5-10% of the liver's weight [9].

Fat accumulation occurs predominantly in the form of triacylglycerols as a result of an alteration in the homeostasis that regulates liver fat synthesis [10]. NAFLD has been considered a benign disease often associated with central obesity, Insulin Resistance (IR), and other MS attributes. However, recent studies have highlighted that NAFLD is a chronic condition that encompasses histologically and clinically different non alcoholic entities; fatty liver and steatohepatitis may progress to cirrhosis and, rarely, to hepatocellular cancer [9,11]. Thus, it is clear that it is a "multisystem disease," associated not only with hepatic dysfunction or hepatocellular carcinoma but also with an increased risk of developing cardiovascular disease, T2DM, and Chronic Kidney Disease (CKD) [12].

Glycosylated Haemoglobin (HbA1c) is a distinctive glycosylated protein commonly used in assessing glycaemic control [13]. Therefore, it can be assumed that patients with NAFLD are likely to have elevated HbA1c levels. There is evidence suggesting a

deranged renal profile due to a decreased estimated Glomerular Filtration Rate (eGFR) as well as electrolyte imbalances primarily in T2DM and NAFLD due to similar metabolic risk factors, indicating a potential pathophysiological link between NAFLD and CKD [14]. Thus, taking this into account, the present study was designed to assess the correlation between BMI status and HbA1c levels with renal profile and electrolytes in T2DM participants with and without NAFLD to evaluate the severity and improve the management of the participants.

MATERIALS AND METHODS

A case-control study was conducted at the Department of Biochemistry and Diabetes Specialty Clinic in the Department of General Medicine at MGM Medical College and Hospital, Navi Mumbai, Maharashtra, India, from December 2021 to March 2023. The project was approved by the Institutional Ethics Committee (ECR/457/Inst/MH/2013/RR-20). Written consent was obtained from all subjects. All participants were informed about the study procedures and enrolled with their written consent.

Inclusion criteria: Patients with Type 2 diabetes mellitus as per American Diabetes Association (ADA) guidelines for the diagnosis and classification of diabetes mellitus, with HbA1c levels >6.5% [3], without NAFLD, aged between 35 and 75 years, were included in Group 3. Diagnosed T2DM patients and NAFLD {diagnosed by Ultrasonography (USG)} with a similar age group were included in Group 2. All participants who voluntarily participated in the study were enrolled.

Exclusion criteria: Type 1 diabetes mellitus patients or any other type of diabetes were excluded from Group 3. For Group 2, type 1 diabetes mellitus patients or any other type of diabetes, and patients with a history of any other liver disease, were excluded. Detailed history of alcohol consumption (if more than 40 units/week), smokers, and pregnant women were excluded from the study groups.

Sample size calculation: The sample size was calculated using the following formula:

$$N_1 = \left\{ z_{1-\alpha/2} * \sqrt{\bar{p} * \bar{q} * \left(1 + \frac{1}{k}\right)} + z_{1-\beta} * \sqrt{p1 * q1 + \left(\frac{p2 * q2}{k}\right)} \right\}^2 / \Delta^2$$

The sample size was calculated based on the prevalence rate of NAFLD with T2DM, which is 80% as reported by Prashanth M et al., and the prevalence rate of T2DM, which is 90% as reported by the global diabetes community (UK), with a power of 80% and alpha set at 0.05 and beta at 0.3 [8,15]. The sample size obtained was 29 in each group, thus totalling 30 in each group and 90 participants overall.

Study Procedure

Diagnosed T2DM patients attending the Outpatient Department (OPD) were enrolled and further grouped based on the radiological findings of USG Abdomen. A total of 90 participants (30 in each group, i.e., Group 1-Control, Group 2-T2DM with NAFLD, Group 3-T2DM without NAFLD) were enrolled in the study.

A detailed clinical history of the participants who attended the diabetes specialty clinic was recorded, including family history of diabetes, demographics (age, sex), as well as anthropometrics (height, weight, BMI), using standard procedures and calculations. Aseptic technique was used for blood collection. A total of 5 mL of blood was drawn from each participant and transferred to plain and Ethylenediamine Tetraacetic Acid (EDTA) vacutainers for biochemical analysis, which included RFT with electrolytes and HbA1c.

The RFT with electrolytes was performed on Beckman Coulter AU480 (with reference ranges: Urea 15-40 mg/dL, Blood Urea Nitrogen (BUN) 10-18 mg/dL, Creatinine 0.6-1.2 mg/dL, Uric acid 2-6 mg/dL, Sodium 135-145 mEq/L, Potassium 3.5-5.1 mEq/L, Chloride 98-107 mEq/L), whereas HbA1c was measured using the Bio-Rad D-10 haemoglobin testing system (with a reference value of <5.7%) [16].

STATISTICAL ANALYSIS

The data was recorded and analysed using SPSS software version 25.0. Quantitative data were represented in the form of mean±Standard Deviation (SD). Odds ratio was calculated using bivariate logistic regression to determine the risk factors, and differences in the means between two groups were analysed using an unpaired t-test. Pearson's correlation analysis was also performed to determine the association between the variables. A p-value of <0.05 was considered statistically significant.

RESULTS

A total of 90 participants were enrolled in the study, which was further grouped into three groups: Group 1: Control, Group 2: T2DM with NAFLD, and Group 3: T2DM without NAFLD. Each group comprised 30 participants who were differentiated based on the radiological findings of USG abdomen. In the present study, the majority were female participants (57%) compared to male participants (43%) amongst the enrolled participants, with a mean age of 49.03±5.09 years [Table/Fig-1].

Parameters	Mean±SD		
	Group 1	Group 2	Group 3
Age (years)	44.84±6.38	47.56±11.12*	54.7±9.57$^{\#\#\#\alpha\alpha}$
Family H/o Diabetes n (%)	4 (13.33%)	28 (93.33%)***	20 (66.66%)$^{\#\#\#\alpha\alpha\alpha}$
Gender distribution			
	Group 1 (n=30)	Group 2 (n=30)*	Group 3 (n=30)$^{\#\alpha}$
Gender	Male / Female	Male / Female	Male / Female
	13 (43%) / 17 (57%)	14 (47%) / 16 (53%)	12 (40%) / 18 (60%)

[Table/Fig-1]: Demographic data of the enrolled participants in three groups respectively.
Group 1 vs 2-**p≤0.05 significant, ***p≤0.001 highly significant, *p≥0.05 non significant
Group 1 vs 3-#p≤0.05 significant, ###p≤0.001 highly significant, #p≥0.05 non significant
Group 2 vs 3-ααp≤0.05 significant, αααp≤0.001 highly significant, αp≥0.05 non significant

A total of 52 (58%) of the enrolled participants had a family history of diabetes, whereas 38 (42%) did not. When comparing the anthropometric parameters (BMI) and HbA1c of the control group with the patient groups in the study, it was found that the BMI and HbA1c levels of Group 2 and 3 (T2DM with NAFLD and T2DM without NAFLD) were significantly higher compared to those of Group 1 (control) with a p-value of <0.001 [Table/Fig-2].

Parameters	Mean±SD		
	Group 1 (n=30)	Group 2 (n=30)	Group 3 (n=30)
BMI (kg/m^2)	21.28±2.24	28.84±4.19***	23.51±2.09$^{\#\#\#\alpha\alpha\alpha}$
HbA1c (%)	4.91±0.46	9.55±1.78***	8.61±1.42$^{\#\#\#\alpha\alpha}$

[Table/Fig-2]: Comparison of BMI as well as HbA1c in the study and control groups.
Group 1 vs 2-**p≤0.05 significant, ***p≤0.001 highly significant, *p≥0.05 non significant
Group 1 vs 3-#p≤0.05 significant, ###p≤0.001 highly significant, #p≥0.05 non significant
Group 2 vs 3-ααp≤0.05 significant, αααp≤0.001 highly significant, αp≥0.05 non significant

The results indicated that urea, BUN, creatinine, and uric acid were significantly higher with a p-value <0.001 in participants with T2DM with NAFLD (Group 2) and T2DM without NAFLD (Group 3) compared to the controls (Group 1). Other biochemical parameters, such as electrolytes, were found to be normal without any significant changes in their levels [Table/Fig-3].

Correlation analysis of BMI with RFT and electrolytes was performed. A positive significant correlation of BMI with uric acid was observed in Group 2 and 3. The correlation analysis of BMI with urea and creatinine depicted a positive but non significant correlation. Further observations of the correlation analysis of HbA1c with RFT and electrolytes indicated a positive significant correlation of HbA1c with urea, creatinine, and uric acid in both groups 2 and 3. Electrolytes did not show any correlation with HbA1c and BMI in any of the

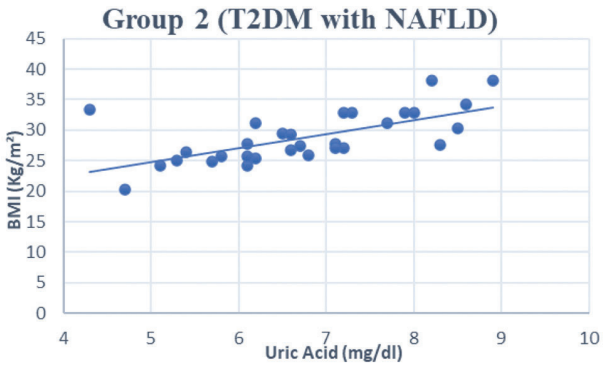
Parameters	Mean±SD		
	Group 1 (n=30)	Group 2 (n=30)	Group 3 (n=30)
Urea (mg/dL)	18.93±2.46	31.62±6.28***	33.02±5.11###α
BUN (mg/dL)	9.55±1.77	12.89±5.10***	12.90±3.70###α
Creatinine (mg/dL)	1.04±0.17	1.29±0.18***	1.36±0.10###αα
Uric acid (mg/dL)	5.26±1.52	6.74±1.19***	6.01±0.83##αα
Sodium (mEq/L)	138±2.12	137±2.28*	137.94±2.33#α
Potassium (mEq/L)	4.10±0.29	4.06±0.34*	4.00±0.35#α
Chloride (mEq/L)	98.47±2.37	98.26±1.95*	98.51±2.95#α

[Table/Fig-3]: Comparison of Biochemical parameters (RFT and electrolytes) in the study and control groups.
Group 1 vs 2-**p≤0.05 significant, ***p≤0.001 highly significant, *p≥0.05 non significant
Group 1 vs 3-#p≤0.05 significant, ###p≤0.001 highly significant, #p≥0.05 non significant
Group 2 vs 3-ααp≤0.05 significant, αααp≤0.001 highly significant, αp≥0.05 non significant

study groups [Table/Fig-4]. [Table/Fig-5,6] represent the correlation between BMI and uric acid in Group 2 and 3, respectively. Similarly, [Table/Fig-7-12] depict the correlation between HbA1c with RFT and electrolytes.

Parameters	Group 2		Group 3	
	r-value	p-value	r-value	p-value
BMI+Urea	0.189	0.314	0.113	0.552
BMI+BUN	-0.121	0.524	-0.146	0.441
BMI+Creatinine	0.224	0.234	0.129	0.495
BMI+Uric acid	0.651	0.00009	0.484	0.006
BMI+Sodium	0.193	0.306	0.074	0.694
BMI+Potassium	0.353	0.055	0.172	0.362
BMI+Chloride	0.193	0.306	-0.053	0.780
HbA1c+Urea	0.654	0.00008	0.780	<0.00001
HbA1c+BUN	0.085	0.655	0.168	0.372
HbA1c+Creatinine	0.766	<0.00001	0.709	0.00001
HbA1c+Uric acid	0.419	0.02091	0.376	0.04041
HbA1c+Sodium	-0.128	0.500	-0.028	0.883
HbA1c+Potassium	0.04	0.833	0.304	0.102
HbA1c+Chloride	0.042	0.824	0.099	0.601

[Table/Fig-4]: Represents the r and p-values of the correlational observations.

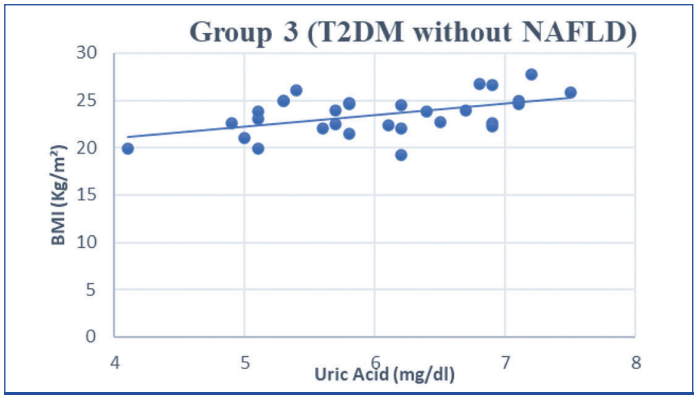


[Table/Fig-5]: Correlation of BMI with uric acid in Group 2.

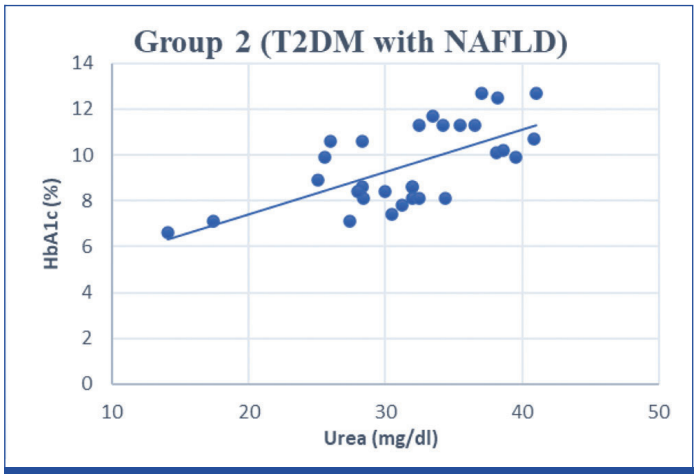
Risk factors were predicted using odds ratio. A higher odds ratio of more than 1 was observed in cases vs. control with respect to the renal profile, which included urea, BUN, creatinine, uric acid, and their risk in diabetic as well as obese participants. Electrolytes were also measured for the risk assessment, but no data was obtained due to constant values [Table/Fig-13].

DISCUSSION

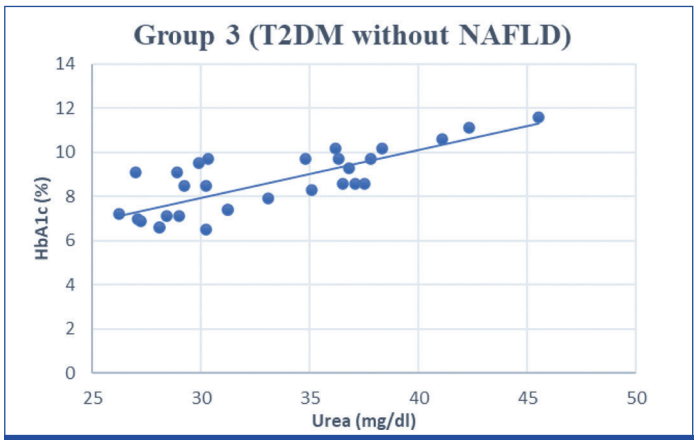
Obesity is rapidly increasing in the general population, according to World Health Organisation (WHO) estimates, and this appears to be associated with poor diet and sedentary lifestyle choices as a consequence of technological progress. The number of overweight people was expected to grow from 1.6 million in 2005 to 3.3 million in 2015. Furthermore, it is estimated to increase from 400 million to 700 million in the same time span [17]. Obesity is a complex,



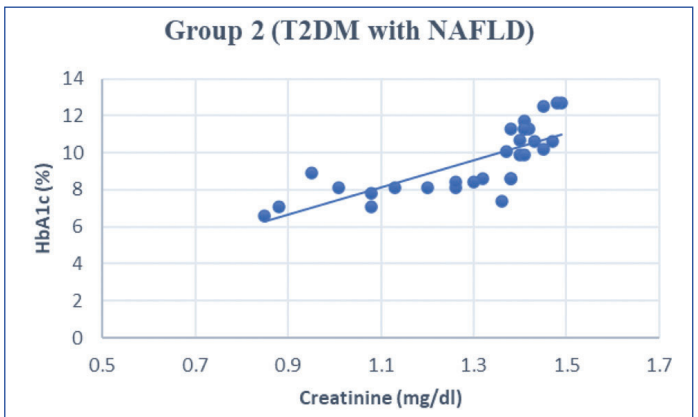
[Table/Fig-6]: Correlation of BMI with uric acid in Group 3.



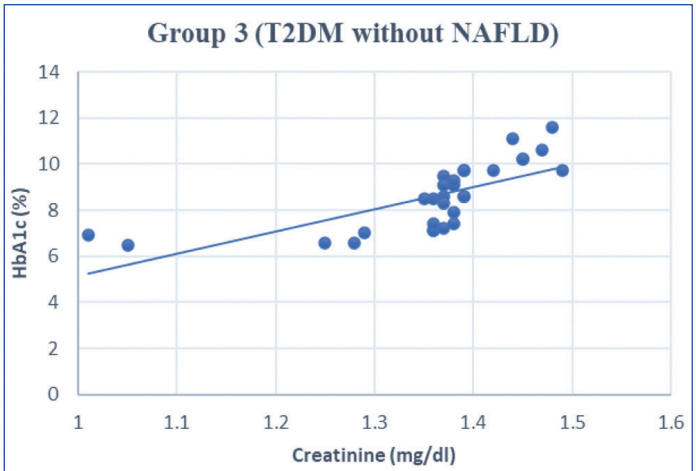
[Table/Fig-7]: Correlation of HbA1c with urea in Group 2.



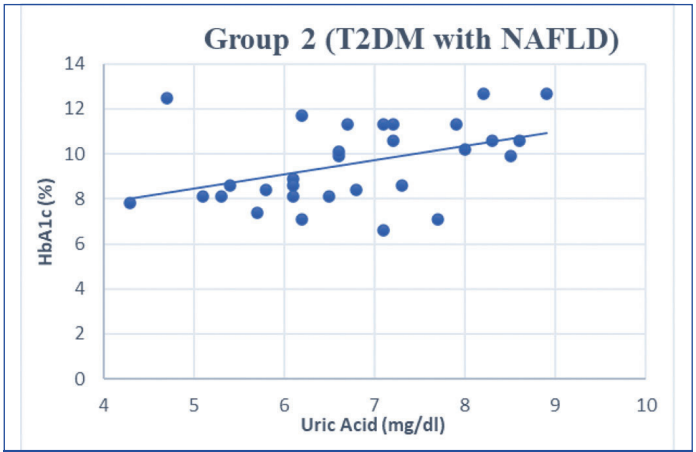
[Table/Fig-8]: Correlation of HbA1c with urea in Group 3.



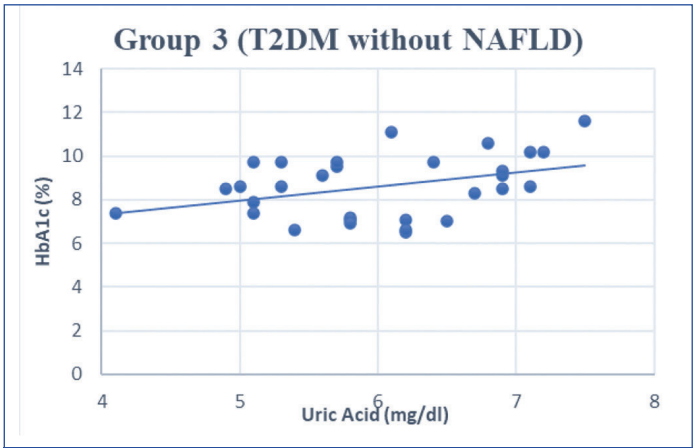
[Table/Fig-9]: Correlation of HbA1c with creatinine in Group 2.



[Table/Fig-10]: Correlation of HbA1c with creatinine in Group 3.



[Table/Fig-11]: Correlation of HbA1c with uric acid in Group 2.



[Table/Fig-12]: Correlation of HbA1c with uric acid in Group 3.

	Group 1+Group 2		Group 1+Group 3	
	HbA1c with RFT			
Parameters	Odds ratio	95% CI	Odds ratio	95% CI
Urea (mg/dL)	1.07	0.97-1.17	1.11	0.98-1.25
BUN (mg/dL)	1.11	0.98-1.25	1.11	0.98-1.25
Creatinine (mg/dL)	17.87	4.73-67.43	26	6.53-103.49
Uric acid (mg/dL)	9.03	2.80-29.13	3.59	1.21-10.63
	BMI with RFT			
	Odds ratio	95% CI	Odds ratio	95% CI
Urea (mg/dL)	1.07	0.97-1.19	4.25	0.33-54.16
BUN (mg/dL)	0.55	0.04-6.47	4.25	0.33-54.16
Creatinine (mg/dL)	10.83	3.23-36.27	1.61	0.32-7.91
Uric acid (mg/dL)	13.80	3.93-48.41	10.73	1.20-95.95

[Table/Fig-13]: Represents the risk factors in the enrolled population.

chronic, non communicable disease affecting more than one-third of the global population [18]. Obese patients exhibit typical metabolic alterations such as IR and type 2 diabetes mellitus. IR is characterised by impaired insulin-induced glucose uptake and metabolism in adipocytes and skeletal muscle, as well as impaired regulation of hepatic glucose synthesis, and it is a major aetiological factor for diabetes mellitus [4].

Obesity and diabetes mellitus are regarded as major risk factors for NAFLD, and they have a productive link with both the existence and progression of the disease. Obese and diabetic individuals have a higher incidence of hepatic steatosis, cirrhosis, and a greater probability of developing Hepatocellular Carcinoma (HCC) [19]. Lipotoxicity and glucotoxicity are key factors in the development of basic steatosis in the liver and its progression to NASH. A high-fat and carbohydrate diet, which obese persons are more likely to follow, promotes fat deposition in the liver, contributing to the advancement of NAFLD [10].

Over the years, the relationship between uric acid and BMI has been widely studied. As we all know, uric acid is the end result of purine degradation. At increased concentrations, uric acid can act as a pro-oxidant and thus can be used as a marker of oxidative stress [20]. Uric acid is mostly eliminated by the kidneys (>70%), with a lesser percentage through intestinal and biliary secretion [21]. In obese individuals, abnormalities in Serum Uric Acid (SUA) metabolism and decreased excretion by the kidneys, as well as increased exogenous protein consumption and endogenous uric acid synthesis, are additional variables that contribute to hyperuricaemia [22-24]. Elevated uric acid levels have been linked to an increased risk of MS, atherosclerosis, and chronic renal disease [25].

According to a study conducted by Duan Y et al., in 2015 on 3529 participants, it was demonstrated that there is a positive significant correlation between obesity and serum uric acid, which aligns with the present study findings of Group 2 and 3 when correlated between BMI and uric acid. Several other studies with similar findings have shown a strong correlation between uric acid and BMI of MS participants wherein obesity is a major factor [26]. The link between Hyperuricaemia (HUA) and obesity can be explained by a number of mechanisms. Obesity or excess body fat may be amalgamated with increased uric acid production due to increased intracellular adenosine (uric acid precursor) which is a derivative of higher Adenosine Monophosphate (AMP) concentrations due to increased synthesis of Fatty-acyl-Coenzyme (CoA) in peripheral tissues [27], and inadequate evacuation as a result of IR and/or hyperinsulinemia, leading to an impaired uric acid metabolism and even HUA. Meanwhile, HUA can induce obesity by accelerating liver and peripheral fat synthesis [28]. A comparison of findings of previous study with the present study has been tabulated in [Table/Fig-14] [28-33].

Similarly, when serum uric acid was correlated with HbA1c, which was used as a measure of blood glucose metabolism, a positive significant correlation was observed in Group 2 and 3. The key factor on which this correlation of HbA1c with uric acid mostly relies on is insulin levels [25,34]. This condition could be explained by the action of insulin on uric acid and glucose metabolism. Hyperinsulinemia may stimulate the hexose phosphate shunt, promoting purine biosynthesis and transformation, thereby increasing the rate of uric acid production [35]. Additionally, insulin may stimulate uric acid reabsorption from the kidneys by activating the urate anion transporter on the border membrane of the proximal tubular brush, leading to an increase in serum uric acid concentration. Insulin can also enhance renal tubular sodium reabsorption [36], which in turn can reduce renal excretion of uric acid. Hyperinsulinaemia could contribute to hyperuricaemia by increasing the rate of xanthine oxidase synthesis, an enzyme involved in UA production [37]. Studies conducted by Hussain A et al., in 2018 and Donkeng M et al., in 2021 have reported similar correlational observations between HbA1c and UA [33,38]. A comparison of the findings of previous studies with the present study has been tabulated in [Table/Fig-15] [33,38,39].

Name of the author	Place and year of study	Sample size	Study population	Correlation findings
Li F et al., [28]	China-2021	153	Obese participants with Metabolic Syndrome (MS)	Pearsons correlational analysis showed serum uric acid positively correlated with BMI.
Yu H et al., [29]	China-2021	553	NAFLD with T2DM	Elevated SUA was associated with a significant increased prevalence of NAFLD and BMI.
Kohichiroh Y et al., [30]	Japan-2011	174	Obese NAFLD	Presence of chronic kidney disease was associated with a higher BMI and presence of NASH.
Arersa KK et al., [31]	South-western Ethopia-2019	287	T2DM patients	Hyperuricaemia was relatively common among type 2 DM patients. The prevalence of hyperuricaemia was common among patients with obesity.
Singh SK et al., [32]	India-2023	402	T2DM patients	A significant positive association between SUA and generalised obesity among newly diagnosed diabetic subjects was observed.
Hussain A et al., [33]	Oranjestad-2018	162	T2DM patients	A positive significant correlation of Hyperuricaemia with increased BMI was observed.
Present study	India-2023	90	T2DM patients with and without NAFLD	A positive significant correlation between serum uric acid and BMI was observed.

[Table/Fig-14]: Comparison of correlation studies between past and present findings [28-33].

Name of the author	Place and year of study	Study size	Sample population	Correlation findings
Hussain A et al., [33]	Oranjestad-2018	162	T2DM patients.	A positive significant correlation between HbA1c and serum uric acid levels due to hyperinsulinemia was observed.
Donkeng M et al., [38]	Dschang-2021	80	T2DM patients with central obesity.	Hyperuricaemia significantly associated to uncontrolled diabetes i.e., HbA1c levels.
Čaušević A et al., [39]	Sarajevo, BH-2010	43	T2DM patients.	This study showed a significantly elevated urine/serum ratio of uric acid in patients with T2DM as compared to healthy control subjects.
Present study	India-2023	90	T2DM patients with and without NAFLD.	A positive significant correlation between serum uric acid and HbA1c levels was observed.

[Table/Fig-15]: Comparison of correlation studies between past and present findings [33,38,39].

When HbA1c was correlated with other parameters of RFT such as urea and creatinine, a positive significant correlation was demonstrated. Given that diabetic nephropathy is a common condition, correlating the provided parameters yielded significant results in Group 2 and 3, which consisted of subjects with T2DM with NAFLD and T2DM without NAFLD, respectively. Type 2 hyperglycaemia typically manifests after the age of 40 years, when the kidneys are already experiencing the long-term effects of ageing and other chronic renal damage promoters such as arterial hypertension, dyslipidaemia, and obesity. This is likely the reason for elevated serum creatinine and urea levels in Type 2 diabetes. According to a study conducted in the Indian diabetic community by Unnikrishnan RI et al., poor glycaemic control is a significant factor responsible for the micro- and macrovascular alterations that occur in diabetes, predisposing diabetic patients to complications [40].

Studies have suggested that long-term diabetes causes higher serum creatinine and urea concentrations, which are risk factors for the progression of kidney damage. Prolonged hyperglycaemia and its associated risks, where excess glucose binds with collagen and tissue proteins, resulting in non enzymatic glycosylation similar to the formation of HbA1c, can lead to microvascular and macrovascular damage. Over time, elevated blood sugar levels damage millions of nephrons, the microscopic filtering units in each kidney, leading to abnormalities in metabolite filtration [41]. T2DM plays an important role as a risk factor for the impairment of RFTs in NAFLD subjects as well. These two being a prominent part of MS makes it even more evident.

Limitation(s)

The present study had a few limitations. Since it was limited to one tertiary care facility, it is possible that the findings may not apply to a broader population. Secondly, the study did not include detailed anthropometric data and the most sensitive biomarkers such as micro-albumin, cystatin C, and GFR for renal impairment, which could have provided further insight into the causal relationship between obesity, diabetes, and NAFLD.

CONCLUSION(S)

The NAFLD is closely associated with IR, obesity, and T2DM, resulting in detrimental hepatic as well as extra-hepatic consequences. Elevated levels of urea, creatinine, and uric acid, and their positive correlation with HbA1c, establish a connection between NAFLD and renal impairment in T2DM. Hyperuricaemia in these individuals can exacerbate the risk of T2DM and NAFLD progression to NASH, respectively. However, no statistically significant difference could be observed with respect to electrolytes in any of the study groups.

Regular assessment of renal parameters would provide early detection and might help in planning both therapeutic and preventive implications. Weight loss can be recommended through proper exercise and a healthy diet plan, which can help limit the damage.

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