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Serum Uric Acid/Creatinine Ratio as a Potential Biomarker for Non-alcoholic Fatty Liver Disease in Type 2 Diabetes

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Abstract

Background: One of the predominant diseases among patients suffering from Type 2 diabetes mellitus (T2DM) is non-alcoholic fatty liver disease (NAFLD). Finding reliable biomarkers for predicting NAFLD severity is required for early intervention and prevention of further diseases such as fibrosis, hepatocellular cancer, and liver failure among these patients. The objective of our study was to find out that among T2DM patients can serum uric acid/creatinine ratio (SUA/CR ratio) predict NAFLD severity. **Materials and Methods:** The sample size of this cross-sectional study was 108 T2DM patients (56 males, 52 females). The participants are further divided into two groups NAFLD Grade 1 and NAFLD grade 2(Confirmed by Ultrasonography). We assessed biochemical parameters such as SUA, Creatinine, SUA/CR ratio (calculated), plasma fasting glucose (FBS), total cholesterol (TC), low density lipoprotein (LDL) cholesterol, high density cholesterol (HDL-C), glycated hemoglobin (HbA1c), Triglyceride (TG), alanine aminotransferase (ALT), and aspartate aminotransferase (AST). SUA/Cr ratio was calculated. Statistical analyses (*t*-tests, analysis of variance) were performed to identify predictors of NAFLD severity. **Results:** Grade 1NAFLD patients showed a decreased SUA/CR ratio than Grade 2 NAFLD patients with T2DM. Grade 2 NAFLD group was presented with increased SUA/CR, TGs, TC, and LDL levels and elevated ALT than Grade 1 NAFLD patients, indicating injury to the liver ($P = 0.001$). FBS, HbA1c, HDL-C cholesterol, and AST are not positively associated with SUA/CR ratio in Grade 1 and Grade 2 NAFLD groups. **Conclusion:** SUA/CR ratio is a simple and cost-effective marker linked with the severity of NAFLD, dyslipidemia, and elevated ALT among patients suffering with T2DM. SUA/CR might predict the risk of NAFLD and the severity of NAFLD among the T2DM population. In the future, large longitudinal studies should validate these findings.

Keywords: Glycated hemoglobin, Non-alcoholic fatty liver disease, Type 2 diabetes mellitus, Uric acid creatinine ratio

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Introduction

Non-alcoholic fatty liver disease (NAFLD) is defined as increased deposition of fatty acids in the hepatic cells without alcohol consumption or any other causes of liver steatosis. Approximately 25–30% of the adult population is affected by NAFLD worldwide, and it represents one of the common liver disorders across the globe.[1]

NAFLD is a multifactorial disease involving inflammation, oxidative stress, insulin resistance, Type 2 diabetes mellitus (T2DM) and dyslipidemia. An increase in NAFLD can cause hepatic manifestation and also contribute to increased manifestation of cardiovascular disease (CVD) and kidney disease.[2]

According to a study published by the Indian Council of Medical Research–India Diabetes, T2DM is a leading health concern for the Indian population. At present, 62.4 million Indian people are living with T2DM. By 2030, this number is projected to reach 100 million, positioning India at the top of the global ranking for diabetes prevalence.[3]

In humans, the end product of purine catabolism is uric acid, which is removed from the body by the kidneys. One of the functions of uric acid in plasma is that it acts as an antioxidant. When it enters the cells, uric acid increases inflammation and elevates oxidative stress.[4]

Many studies have reported the positive association of serum uric acid (SUA) and its derivatives with various inflammatory markers, which can be further linked to various disorders, such as hypertension, metabolic syndrome, diabetes mellitus Type 2, CVD, dyslipidemia, thyroiditis, NAFLD, and liver cirrhosis.[5]

Creatinine is an excretory byproduct of phosphocreatine. Creatine is an important tripeptide made up of arginine, glycine, and methionine. It is phosphorylated into creatine phosphate, which supplies energy to skeletal muscles and the brain. Creatinine is used as a marker for Glomerular filtration rate. The serum uric acid/creatinine ratio (SUA/CR ratio) might be an important factor which indicates endogenous uric acid levels after adjusting for kidney functions.[6]

Many theories have been explained the mechanisms to prove the relationship of SUA/CR ratio with increased NAFLD risk. Increased SUA values were positively correlated with increased inflammation, which leads to elevated oxidative stress in the adipose cells, which can cause Insulin resistance. Increased uric acid levels lower nitric oxide, which elevates intracellular oxidative stress, which further damages the membranes of blood vessels. Uric acid directly increases fatty acid accumulation in the liver cells, which can cause NAFLD. As removal of uric acid from the body is influenced by renal function, we proposed the hypothesis that SUA/CR can indicate the endogenous uric acid values more accurately than urine uric acid values alone.[7-9]

T2DM patients were more prone to induce NAFLD and have increased chances of developing cirrhosis of the liver, liver failure, or hepatocellular cancer.[10-13]

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As India is the capital of T2DM and there is a positive association of T2DM with NAFLD, timely recognition of the severity of NAFLD is necessary for early intervention and prevention of further complications among these population. Liver biopsy is the gold standard test for estimating the severity of disease, but high cost and physical invasiveness limit its use in patient's care.[14] Therefore, non-invasive, cost-effective biomarkers capable of assessing NAFLD severity in T2DM patients is need of the current situation.[15]

Current studies suggest that the SUA/CR ratio might serve as a good indicator of metabolic risk better than uric acid estimation alone.[16] The potential of the SUA/CR ratio to assess NAFLD has been studied in the normal population but not in T2DM patients. Therefore, our study aim was to determine the association of the SUA/CR ratio among T2DM patients.

Given the close association between NAFLD and T2DM, the emerging utility of the SUA/CR ratio as a simple non-invasive biomarker of metabolic health, further investigation is warranted to clarify its role in this high-risk population. Therefore, this study was designed to evaluate the association between serum SUA/CR and the severity of NAFLD in the patients suffering with T2DM. Our study hypothesis was elevated SUA/CR ratio would be highly correlated with an unfavorable metabolic outcome, including NAFLD, insulin resistance and dyslipidemia.

Materials and Methods

This was a cross-sectional study in which we involved 108 T2DM participants (56 Female and 52 Male) with Ultrasonography-confirmed Grade 1 and Grade 2 NAFLD patients. Grade 1 NAFLD is mild steatosis, and Grade 2 NAFLD is moderate steatosis. We assessed biochemical parameters such as SUA levels, serum Creatinine, fasting blood glucose, glycated hemoglobin (HbA1c), triglycerides (TGs), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), alanine aminotransferase (ALT), and aspartate aminotransferase (AST). SUA/CR ratio was calculated. Statistical analyses (*t*-tests, analysis of variance [ANOVA], regression modeling) were performed to identify predictors of NAFLD severity. We have included Grade 1 and Grade 2 NAFLD patients from confirmed, proven radiology clinical data, which shows ultrasonography of the liver parenchyma relative to the renal cortex (Liver-Kidney contrast), clarity of intrahepatic vessel borders, and diaphragm visualization.

1. Grade 1 (52 patients): Slight increase in fine echoes in the hepatic parenchyma cells with normal diaphragm and intrahepatic vessels Mean Age 48.6 Years (Figure 4a).
2. Grade 2 (58 Patients): Moderate increase in fine echoes with abnormal diaphragm and intrahepatic vessels Mean Age 47 Years (Figure 4a).

We have not included Grade 3 NAFLD patients as it was correlated with advanced hepatic fat accumulation, fibrosis, and liver cirrhosis.

Inclusion Criteria

We included T2DM patients (according to ADA guidelines 2023, https://diabetes.org/newsroom/ameri can-diabetes-association-2023 standards-care-diabetes-guide-forprevention-diagnosis-treatmentpeople-living-with-diabetes) with NAFLD (according to 2023 INASL guidelines https://inasl.org.in/) aged between 18 years and 60 years of both sexes.

Exclusion Criteria

We excluded patients with a history of CVD, myocardial infarction, diabetic patients other than T2DM, cerebral hemorrhage, severe hepatorenal disorders, acute infection, patients with malignant tumors, serious and uncontrollable medical condition, pregnant women, lactating women, psychiatric patients, gout patients and patients with alcoholic fatty liver disease.

Biochemical Analysis

Blood samples from the veins were taken from each participant under sterile condition after 12-h overnight fast. Estimation of SUA and serum creatinine was done using enzymatic colorimetric methods using autoanalyzer ERBA EM 360. The SUA/CR was calculated by dividing values of SUA (mg/dL) by values of serum creatinine (mg/dL). Other biochemical parameters such as fasting blood glucose, TG, TC, LDL-C, HDL-C, HbA1c, AST, and ALT were collected from participant's clinical data.

Statistical Analysis

Continuous variables such as Mean (M) and Standard Deviations (SD) and categorical variables such as frequencies were measured. The differences between NAFLD Grade 1 and Grade 2 were determined using one-way ANOVA for continuous variables and Chi-square test for categorical variables. The software's such as R Studio and Microsoft Excel were used for statistical analysis and plot preparations.

Results

Demographic Characteristics of Subject Population

A total of 108 T2DM patients with NAFLD are included in the study, with 56 females and 52 males, and an average age of 49.05 ± 17.5 years. The subjects are further divided into two groups NAFLD Grade 1 and NAFLD Grade 2 [Table 1].

In this study, we presented the data as M and SD. We examined our data for the normality of distribution using the Shapiro-Wilk

Table 1: The demographical and clinical data for patients in the study

S. No.	*Parameter*	*NAFLD Stage1 (n=51) (M=25, F=26)*		*NAFLD Stage 2 (n=57) (M=27, F=30)*		*P-value*
		Mean	*SD*	*Mean*	*SD*	
1.	Age (years)	48.6	7.7	47	8.4	0.3002
2.	UA (mg/dL)	4.17	1.9	5.90	1.4	0.001
3.	Cr (mg/dL)	1.36	0.9	1.06	0.2	0.018
4.	SUA/CR	3.23	0.7	5.61	0.9	0.001
5.	TG (mg/dL)	165.8	9.6	186.5	18.5	0.001
6.	TC (mg/dL)	194.3	11.0	221.7	12.5	0.001
7.	HDL-C (mg/dL)	45.09	4.1	43.73	4.1	0.090
8.	LDL-C (mg/dL)	116.8	8.5	141.0	9.8	0.001
9.	ALT (U/L)	39.5	15.6	51.8	13.8	0.001
10.	AST (U/L)	29.5	6.5	28.7	6.9	0.52
11.	FBS (mg/dL)	193.5	89.3	197.5	109.1	0.836
12.	Glycated Hb (%)	9.25	2.15	8.39	2.06	0.036

NAFLD: Non-alcoholic fatty liver disease, UA: Uric acid, Cr: Creatinine, SUA/CR: Serum uric acid/creatinine ratio, TG: Triglycerides, TC: Total cholesterol, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, FBS: Fasting blood sugar, Hb: Hemoglobin, SD: Standard deviation

test. We used the Mann-–Whitney test for the between group (Grade 1 NAFLD and Grade 2 NAFLD) comparison. Correlation analysis was carried out using Spearman's rank correlation. Correlation and differences between the Grade 1 and Grade 2 NAFLD were considered significant at $P < 0.05$.

Grade 1 NAFLD patient's data showed lower mean values of SUA, while Grade 2 NAFLD patient's data showed noticeably higher mean, indicating greater variability of uric acid levels among patients with more severe NAFLD [Figure 1a]. While there was not much difference between serum creatinine mean values among two groups [Figure 1b]. Grade 1 NAFLD patient's data showed lower mean values of serum TG and TC, while Grade 2 NAFLD patient's data showed noticeably higher mean, indicating greater variability of TG and TC levels among patients with more severe NAFLD [Figure 2a and b]. Grade 1 NAFLD patient's data showed lower mean values of serum LDL-C, while Grade 2 NAFLD patient's data showed noticeably higher mean, indicating greater variability of LDL-C levels among patients with more severe NAFLD [Figure 3a]. While there was not much difference between serum HDL-C mean values among two groups [Figure 3b]. Grade 1 patient's data showed lower median UA/CR with fewer outliers, while Grade 2 patient's data showed slightly higher median and broader range, showing more variability among

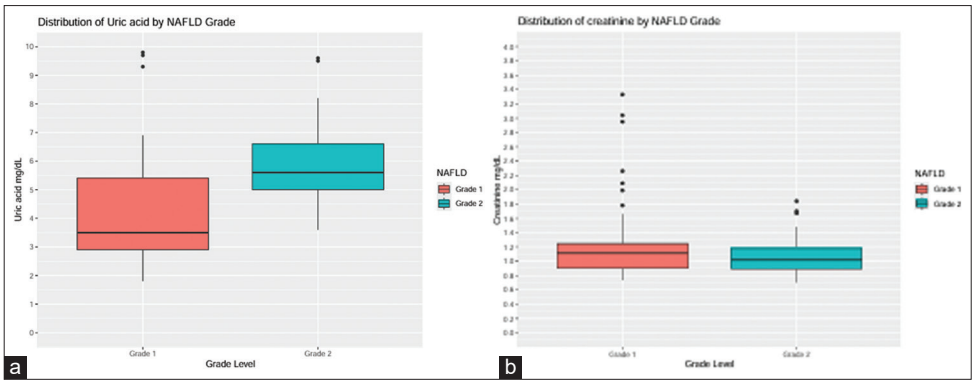

Figure 1: (a) Distribution of serum uric acid among Grade 1 and Grade 2 Type 2 diabetes mellitus (T2DM) patients. (b) Distribution of serum creatinine among Grade 1 and Grade 2 T2DM patients

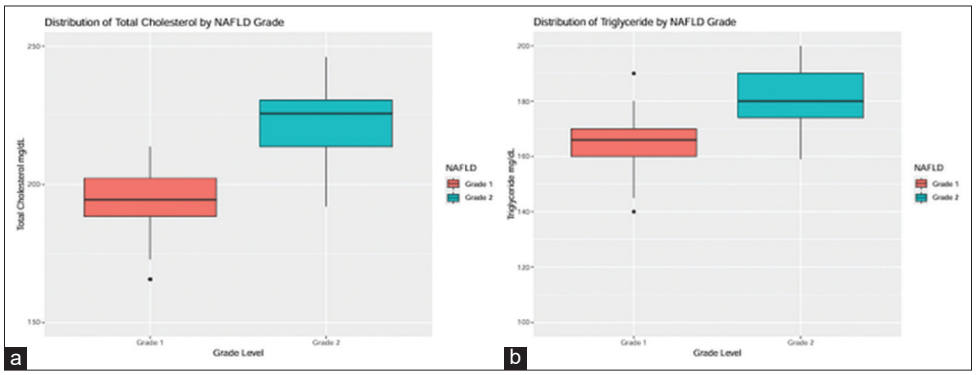

Figure 2: Distribution of serum total cholesterol among Grade 1 and Grade 2 Type 2 diabetes mellitus (T2DM) patients. (b) Distribution of serum triglyceride among Grade 1 and Grade 2 T2DM patients

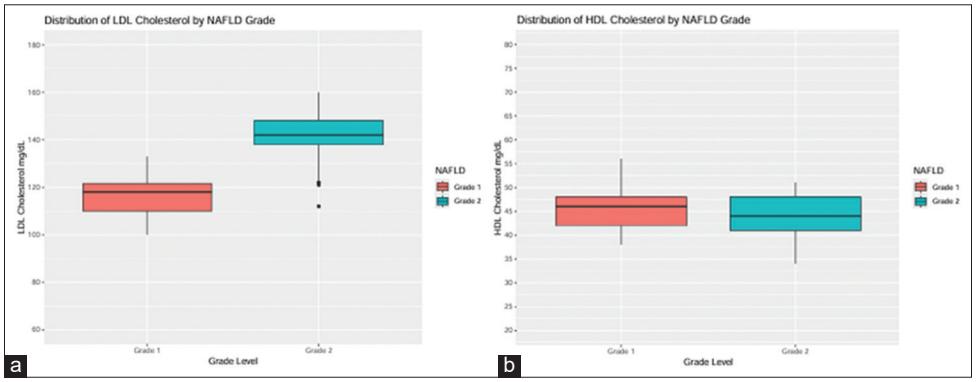

Figure 3: (a) Distribution of low-density lipoprotein cholesterol among Grade 1 and Grade 2 Type 2 diabetes mellitus (T2DM) patients. (b) Distribution of high-density lipoprotein cholesterol among Grade 1 and Grade 2 T2DM patients

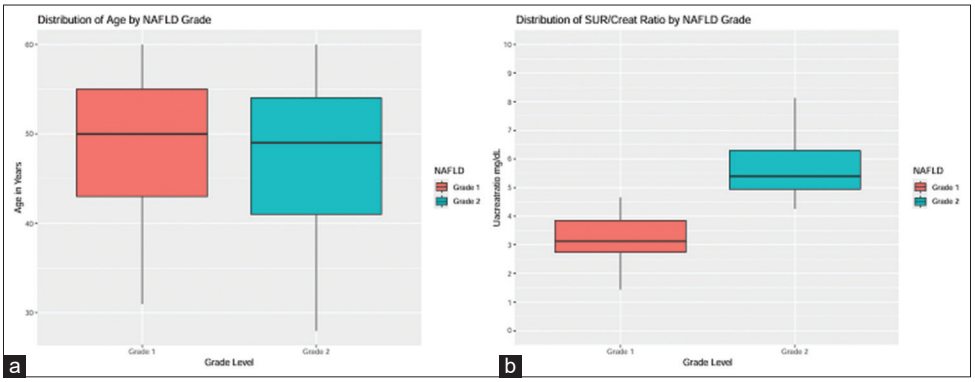

Figure 4: (a) Distribution of age among Grade1 and Grade 2 T2DM patients. (b) Distribution of SUA/creat ratio among Grade1 and Grade 2 T2DM patients

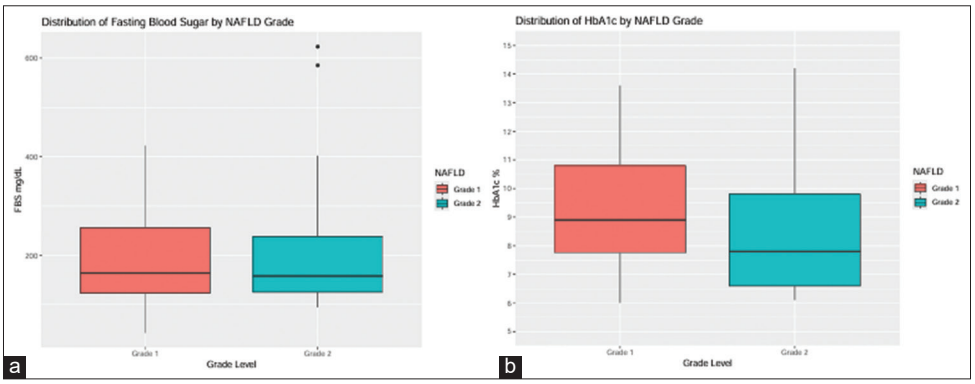

Figure 5: (a) Distribution of fasting blood sugar among Grade 1 and Grade 2 Type 2 diabetes mellitus (T2DM) patients. (b) Distribution of HbA1c among Grade 1 and Grade 2 T2DM patients

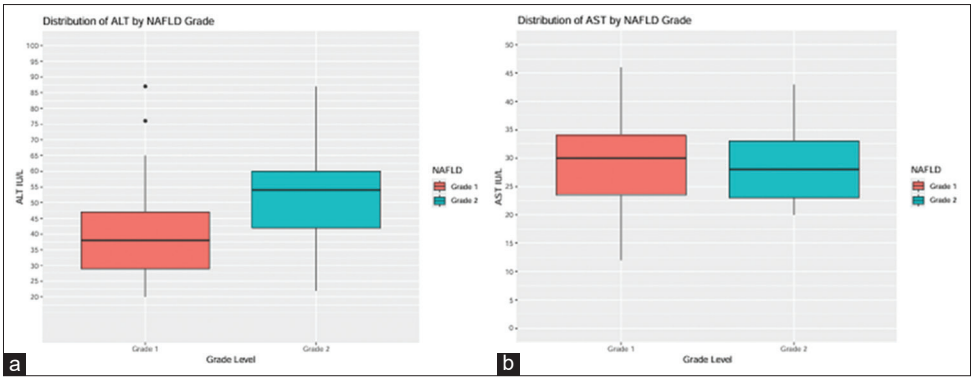

Figure 6: Distribution of alanine aminotransferase among Grade 1 and Grade 2 Type 2 diabetes mellitus (T2DM) patients. (b) Distribution of aspartate aminotransferase among Grade 1 and Grade 2 T2DM patients

patients with advanced NAFLD [Figure 4b]. Grade 1 NAFLD patient's data showed higher mean HbA1c values, with fewer high outliers, while Grade 2 NAFLD patient's data showed lower mean HbA1c values [Figure 5b], while there was not much difference between fasting blood glucose mean values between Grade 1 and Grade 2 NAFLD patients [Figure 5a]. Grade 1 NAFLD patient's data showed lower mean ALT values, while Grade 2 NAFLD patient's data showed higher mean ALT values, while there was not much difference between serum AST mean values between Grade 1 and Grade 2 NAFLD patients.

Discussion

The results of our study revealed that an increased levels of SUA/CR ratio were significantly associated with an increased severity of NAFLD in individuals with T2DM. With increased severity of NAFLD, there are increased levels of TG, TC, LDL-C, and ALT, which suggests the presence of hepatic injury. Thus SUA/CR ratio not only indicates NAFLD severity but also indicates increased risk of cardiometabolic complications. Many studies have shown that even mild or moderate grades of NAFLD are associated with

increased risk of atherosclerosis and myocardial infarction. In this study, we also demonstrated that the increased SUA/CR ratio is not associated with fasting blood glucose, HbA1c, HDL-C, and aspartate aminotransferase. The correlation of NAFLD grades with SUA/CR ratio, TG, TC, LDL-C, HDL-C, fasting glucose, HbA1c, ALT, and AST was illustrated in Figures 1-6 and Table 1.

In adipose tissue, uric acid increases an inflammatory endocrine imbalance, which leads to increased oxidative stress. Uric acid-induced increased intracellular and mitochondrial oxidative stress causes disturbance in the TCA cycle, leading to decreased fatty acid oxidation, which ultimately increases fat synthesis. The combined effect of these factors results in visceral obesity, insulin resistance, T2DM, and NAFLD. SUA levels are increased due to reduced glomerular filtration. Therefore, the SUA/CR ratio can be used in prediction of NAFLD with decreased confounding effect caused by kidney dysfunction.

The strength of our study was it focuses on affordable and easily available biomarker, SUA for metabolic functions, serum creatinine for renal aspects, and together the SUA/CR ratio for both metabolic and renal aspects increases its importance. The limitations of this study are as follows: First, in this cross-sectional study, we used lower sample size, and this is single center study. Second, in this study, we used abdominal ultrasonography to confirm NAFLD grade status, which is a widely used and non-invasive tool; it has lower sensitivity compared to advanced imaging techniques. Third, we did not compare the SUA/CR ratio among type 2 diabetic patients without NAFLD. To prove SUA/CR ratio will be the great potential biomarker for early detection of the severity of NAFLD the larger and multicenter studies are required.

Conclusion

The conclusion of our study was that the results of our study demonstrated that SUA to CR was positively associated with increased severity of NAFLD in T2DM population, along with dyslipidemia and liver enzyme elevation. The mechanism pathways linking SUA/CR to NAFLD severity might be increased oxidative stress, inflammation, and adipose tissue dysfunction. As SUA/CR can be done easily at low cost and has clinical relevance with NAFLD, it might be act as a potential biomarker for the prediction of risk stratification of NAFLD and early intervention among T2DM patients. In future large studies should be warranted to demonstrate whether lowering SUA/CR might improve metabolic outcomes and decrease the NAFLD progression rate in T2DM patients.

Authors' contributions

VKH: Writing-original draft, writing-review and editing, conceptualization, funding acquisition, investigation, methodology, project administration. HOS: Formal analysis, methodology, project administration, validation, writing-review, and editing. All authors have reviewed and approved the final manuscript and agree to be accountable for all aspects of the work.

Declaration of Helsinki

In this study, we followed all the ethical principles outlined in the Declaration of Helsinki (revised 2013), which elaborates the guidelines for medical research involving human participants, including studies on identifiable human material and data. The approval was taken from the Institutional Ethics Committee of the Oxford Medical College, Hospital and Research Centre before the initiation of the study.

Declaration of patient consent

We have obtained the written informed consent from all participants after giving a full explanation of our study objective, study procedures, potential benefits, and possible risks. Participants were given time to consider participation and guaranteed them that they can refuse to participate from the study any time without affecting their medical care. The right of the participant to refuse to participate or to discontinue participation without providing reasons and without prejudicing further treatment was respected. Before the study commencement, approval was obtained from the Institutional Review Board of The Oxford Medical College, Hospital and Research Centre under approval code 117/2024-25.

Data availability statement

We will share the supportive data of our study upon reasonable request. Due to ethical consideration and the privacy of participants, the raw data is not shared publicly but may be considered for sharing to legitimate academic or research purposes.

Acknowledgment

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