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Frequency and Association of Dyslipidemia with Clinical and Biochemical Parameters among Patients with Non-alcoholic Fatty Liver Disease

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ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is increasingly associated with metabolic disorders, particularly dyslipidemia, which contributes to cardiovascular risk and disease progression. Understanding this relationship is essential for early intervention. **Objectives:** To assess the frequency of dyslipidemia and its association with clinical and biochemical parameters in NAFLD patients. **Methods:** A descriptive cross-sectional study was carried out at the Department of Medicine, Jinnah Hospital, Lahore, from November 2024 to April 2025. A total of 116 ultrasonographically confirmed NAFLD patients aged 18–70 years were registered using non-probability consecutive sampling. Patients with secondary causes of liver disease, lipid-altering medications, or systemic illnesses were excluded. **Results:** Among the 116 NAFLD participants, the average age was 48.67 ± 11.57 years, and the mean body mass index (BMI) was 28.27 ± 4.60 kg/m^2. Dyslipidemia was present in 72 (62.1%) of participants. Females comprised 44 (61.1%) of the dyslipidemic group, and 44 (61.1%) were aged 46–70 years. Diabetes mellitus and ischemic heart disease were significantly associated with dyslipidemia (p=0.016 and p=0.004, respectively). Biochemical markers, including BMI (p=0.002), AST (p=0.012), and ALT (p=0.041), were significantly elevated in dyslipidemic patients. Lipid profile abnormalities, such as total cholesterol, triglycerides, and LDL-C, were significantly higher, while HDL-C was lower (all p<0.001). Dyslipidemia prevalence increased with NAFLD severity grade (p<0.001). **Conclusion:** Dyslipidemia is prevalent in NAFLD and significantly correlates with disease severity and metabolic comorbidities, highlighting the need for integrated lipid and hepatic assessment in clinical management.

INTRODUCTION

Non-Alcoholic Fatty Liver Disease (NAFLD) refers to a range of liver disorders marked by fat deposition exceeding 5% of hepatocytes, occurring without notable alcohol intake, viral hepatitis, or other secondary causes of liver disease [1]. Globally, NAFLD affects about 25 to 30% of adults, with similar rates in South Asia [2]. In Pakistan, it is estimated that 14% to 30% of the overall population is impacted by NAFLD, with higher rates (up to 61%) in urban and high-risk groups. Among individuals with metabolic disorders, prevalence is notably elevated: 48–55% in diabetes, 74% in hypertension, and 41–47% in obesity. Even non-obese individuals show a prevalence of 12–25% [3, 4]. Its burden is also rising steadily in South Asian populations, driven by dietary transitions, increasing sedentary lifestyles, as well as the growing occurrence of metabolic syndrome. The condition includes various liver abnormalities, from basic steatosis to more severe forms such as NASH, fibrosis, cirrhosis, and even liver cancer [5]. The pathogenesis of NAFLD shows a strong link with insulin resistance and widespread disturbances in metabolic

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processes. Among the metabolic abnormalities frequently observed in NAFLD, dyslipidemia plays a central role [6]. Individuals with NAFLD commonly exhibit an atherogenic lipid profile. This pattern of dyslipidemia contributes both to liver fat buildup and significantly increases the risk for cardiovascular complications [7, 8]. Although global data have established a strong link between dyslipidemia and non-alcoholic fatty liver disease, evidence from Pakistan remains limited, particularly among patients with ultrasonographically confirmed NAFLD. The association between dyslipidemia and NAFLD has been well documented across various populations. Multiple studies have demonstrated a strong relationship between NAFLD and dyslipidemia. Ullah *et al.* and Waqas *et al.* reported dyslipidemia in 26–29% of NAFLD patients with elevated TC, TG, and low HDL-C [9, 10]. Shaikh *et al.* observed 61.3% low HDL-C and worsening lipid profile with fibrosis ($p<0.01$) [11]. Cigri *et al.* and Dowla *et al.* highlighted significant dyslipidemia in pediatric NAFLD, particularly raised TG and LDL-C with reduced HDL-C [12, 13]. A study has further suggested that the severity of NAFLD, as assessed by ultrasound or liver biopsy, correlates with the degree of lipid derangement and insulin resistance [14]. Despite the increasing recognition of NAFLD and its metabolic implications, there is a relative paucity of data from Pakistan regarding the frequency and pattern of dyslipidemia among individuals with ultrasonographically confirmed NAFLD. Most regional studies remain hospital-based, with limited representation of the broader population. Moreover, while international guidelines recommend routine lipid profiling in NAFLD patients to assess cardiovascular risk, local screening practices remain inconsistent due to limited infrastructure and variable awareness [15-17].

The present study was therefore designed to address this deficiency by determining the frequency of dyslipidemia and evaluating its relationship with disease severity in ultrasonographically confirmed NAFLD patients. Given the rising prevalence of NAFLD and the well-established contribution of dyslipidemia to its progression and associated morbidity, it is imperative to characterize the burden of lipid abnormalities in this patient popula Understanding the frequency and distribution of dyslipidemia among NAFLD patients may facilitate early identification of individuals at higher risk of cardiovascular complications and inform public health strategies for targeted metabolic intervention. This study aims to determine the frequency of dyslipidemia and its associated clinical patterns among patients with NAFLD diagnosed on ultrasonography.

METHODS

This descriptive cross-sectional study was conducted in the Department of Medicine at Jinnah Hospital Lahore, over a period of 6 months spanning November 2024 to April 2025. Approval for the study was granted by the Institutional Review Board of Jinnah Hospital, Lahore, with ref no: ERB132/3/17-11-2022/S1 ERB and CPSP np: CPSP/REU/MED-2021-055-18180. Eligible participants with NAFLD were recruited through a consecutive non-probability sampling method. The sample size was 116, which was calculated by assuming a proportion of dyslipidemia of 26%, with a 95% confidence level and an 8% margin of error [9]. Adults aged 18 to 70 years of either gender with ultrasonographically confirmed NAFLD and who provided informed consent were included. Exclusion criteria included chronic liver disease of other etiologies (viral hepatitis B/C, autoimmune hepatitis, hemochromatosis, Wilson's disease, drug-induced liver injury), history of alcohol intake, use of lipid-lowering drugs within the past three months, pregnancy or lactation, and chronic systemic illnesses affecting lipid metabolism, such as malignancy, chronic kidney disease, or heart failure. After obtaining informed consent from patients, those meeting the criteria were enrolled consecutively until the required sample size was achieved. At the time of enrollment, baseline demographic data, including age and gender, were recorded. Clinical information, including diabetes mellitus, hypertension, and ischemic heart disease, was obtained from patients' medical records. Ultrasonographic assessment for NAFLD grading was performed using a Samsung HS40 diagnostic ultrasound system equipped with a 3.5 MHz convex transducer, operated by experienced radiologists following standardized protocols. NAFLD was defined as increased hepatic echogenicity compared to the renal cortex, with the absence of secondary causes of liver fat accumulation. The severity of NAFLD was graded using ultrasonographic findings. Grade 0 was considered normal hepatic echogenicity. Grade 1 was characterized by a mild rise in liver echogenicity, while the diaphragm and intrahepatic vessels remained clearly visible. In Grade 2, there was a noticeable rise in liver echogenicity along with partial obscuration of the diaphragm or intrahepatic vessels. Grade 3 exhibited a significant elevation in echogenicity, often accompanied by poor or lost visualization of the diaphragm, intrahepatic vasculature, and the posterior segment of the right hepatic lobe. All participants underwent venous blood sampling following an overnight fast of 8 to 12 hours. Venous blood samples were collected after an overnight fast of 8–12 hours. Serum AST and ALT were measured using the Roche Cobas c311 automated chemistry analyzer (Roche Diagnostics, Germany) based on

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the International Federation of Clinical Chemistry (IFCC) kinetic method. Lipid profile parameters, total cholesterol, triglycerides, LDL-C, and HDL-C, were quantified by enzymatic colorimetric assays on the Roche Cobas c311 platform, employing Roche Diagnostics reagent kits, ensuring internal quality control and calibration before each batch run. Dyslipidemia, which served as the primary outcome, was identified according to predefined criteria, derived from national and international lipid guidelines. A diagnosis of dyslipidemia was established when one or more of the following thresholds were met: total cholesterol ≥200 mg/dL, triglycerides ≥150 mg/dL, LDL-C ≥130 mg/dL, HDL-C <40 mg/dL in men, or <50 mg/dL in women. Each case was monitored and reviewed at the point of laboratory reporting to ensure accurate classification. Data were statistically analyzed using SPSS, version 26.0. Categorical data were presented as frequencies and percentages, while continuous data were presented as mean and standard deviation (SD). Group comparisons between patients with and without dyslipidemia were conducted using the Chi-square test for categorical variables and independent sample t-tests for continuous variables. Associations between categorical variables were assessed using odds ratios with 95% CIs, while continuous variable comparisons were accompanied by mean differences and corresponding 95% CIs. The relationship between dyslipidemia and NAFLD severity grades was analyzed using the Chi-square test, and percentages were computed row-wise to reflect the proportion of dyslipidemia within each NAFLD grade. A p-value <0.05 was considered statistically significant.

RESULTS

In this study, 116 patients were included with a mean age of 48.67 ± 11.57 years and a mean BMI of 28.27 ± 4.60kg/m^2. There was no association between dyslipidemia and age group, with 28 (38.9%) affected patients aged 18–45 years and 44 (61.1%) aged 46–70 years. Gender distribution was also statistically non-significant, with 44 (61.1%) females and 28 (38.9%) males in the dyslipidemic group (p=0.241). In contrast, a statistically significant association was observed for diabetes mellitus, which was present in 49 (68.1%) patients with dyslipidemia versus 20 (45.5%) without (p=0.016). Ischemic heart disease also showed a strong correlation with dyslipidemia (p=0.004), affecting 32 (44.4%) versus 8 (18.2%) patients. No difference was found in hypertension prevalence (p=0.104). BMI was higher in the dyslipidemia group (29.26 ± 4.76 vs. 26.64 ± 3.84kg/m^2; p=0.002). AST (41.89 ± 11.45 vs. 36.56 ± 10.08 IU/L; p=0.012) and ALT (54.93 ± 17.09 vs. 48.59 ± 14.18 IU/L; p=0.041) were also significantly raised (Table 1).

Table 1: Association of Demographic, Clinical, and Biochemical Variables with Dyslipidemia in Patients with NAFLD (n=116)

Variables	Subgroup	Dyslipidemia Present (n=72)	Dyslipidemia Absent (n=44)	Test Statistic (χ^2 / t)	Effect Size (OR / Mean Difference, 95% CI)	p-value
Age Group (years)	18–45	28 (38.9%)	18 (40.9%)	χ^2 = 0.047	OR = 1.088 (0.506–2.339)	0.829
	46–70	44 (61.1%)	26 (59.1%)			
Gender	Female	44 (61.1%)	22 (50.0%)	χ^2 = 1.375	OR = 1.571 (0.737–3.352)	0.241
	Male	28 (38.9%)	22 (50.0%)			
Diabetes Mellitus	Yes	49 (68.1%)	20 (45.5%)	χ^2 = 5.788	OR = 2.557 (1.180–5.538)	0.016*
	No	23 (31.9%)	24 (54.5%)			
Hypertension	Yes	39 (54.2%)	17 (38.6%)	χ^2 = 2.638	OR = 1.877 (0.875–4.028)	0.104
	No	33 (45.8%)	27 (61.4%)			
Ischemic Heart Disease	Yes	32 (44.4%)	8 (18.2%)	χ^2 = 8.338	OR = 3.600 (1.469–8.820)	0.004*
	No	40 (55.6%)	36 (81.8%)			
Age (Years)	–	47.81 ± 11.73	50.09 ± 11.29	t = 1.033	MD = 2.285 (–2.099–6.669)	0.304
BMI (kg/m^2)	–	29.26 ± 4.76	26.64 ± 3.84	t = –3.095	MD = –2.63 (–4.31 to –0.95)	0.002*
AST (IU/L)	–	41.89 ± 11.45	36.56 ± 10.08	t = –2.544	MD = –5.33 (–9.49 to –1.18)	0.012*
ALT (IU/L)	–	54.93 ± 17.09	48.59 ± 14.18	t = –2.062	MD = –6.33 (–12.42 to –0.25)	0.041*

Chi-square test was used for categorical variables and an independent t-test for continuous variables. A p-value <0.05 was considered statistically significant. OR – odds ratio; CI – confidence interval; MD – mean difference; AST – aspartate aminotransferase; ALT – alanine aminotransferase; BMI – body mass index.

Patients with dyslipidemia exhibited higher lipid parameters compared to those without. The mean total cholesterol level was 216.11 ± 18.74 mg/dL in the dyslipidemia group versus 205.17 ± 36.88mg/dL in the non-dyslipidemia group (p = 0.037). Triglycerides were also elevated in dyslipidemic individuals (201.99 ± 20.58mg/dL) compared to 170.05 ± 24.59mg/dL (p<0.001). LDL-C was markedly raised at 143.67 ± 15.46mg/dL versus 119.73 ± 18.46mg/dL (p<0.001). In contrast, HDL-C was significantly lower in dyslipidemic patients (35.35 ± 4.52mg/dL) than in those without dyslipidemia (41.38 ± 7.93mg/dL) (Table 2).

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Table 2: Comparison of Lipid Profile Parameters Between Patients with and without Dyslipidemia (n=116)

Lipid Parameters	Dyslipidemia Present (Mean ± SD)	Dyslipidemia Absent (Mean ± SD)	t-value	Mean Difference (95% CI)	p-value
Total Cholesterol (mg/dL)	216.11 ± 18.74	205.17 ± 36.88	−2.114	−10.94 (−21.98 to −0.69)	0.037*
Triglycerides (mg/dL)	201.99 ± 20.58	170.05 ± 24.59	−7.528	−31.95 (−40.35 to −23.54)	<0.001*
LDL-C (mg/dL)	143.67 ± 15.46	119.73 ± 18.46	−7.513	−23.94 (−30.25 to −17.63)	<0.001*
HDL-C (mg/dL)	35.35 ± 4.52	41.38 ± 7.93	5.218	6.03 (3.74 to 8.32)	<0.001*

An independent t-test was used to compare the lipid parameters between patients with and without dyslipidemia. A p-value <0.05 was considered statistically significant.

Dyslipidemia frequency increased with disease severity (p<0.001). In Grade 1, dyslipidemia was observed in 20 (41.7%) versus 28 (58.3%) without. Grade 2 included 28 (66.7%) dyslipidemic and 14 (33.3%) non-dyslipidemic patients. In Grade 3, 24 (92.3%) had dyslipidemia versus only 2 (7.7%) without, indicating a significant association between dyslipidemia and higher NAFLD grade (Table 3).

Table 3: Association Between Dyslipidemia and NAFLD Severity Grades

NAFLD Grade	Dyslipidemia Present (n=72)	Dyslipidemia Absent (n=44)	p-value
Grade 1 (Mild)	20 (41.7%)	28 (58.3%)	<0.001*
Grade 2 (Moderate)	28 (66.7%)	14 (33.3%)	
Grade 3 (Severe)	24 (92.3%)	2 (7.7%)	

The Pearson Chi-Square test was used to assess the association between dyslipidemia and NAFLD severity grades.

In this study comprising 116 participants, dyslipidemia was observed in 72 (62.1%) patients, while 44 (37.9%) patients did not have dyslipidemia (Figure 1).

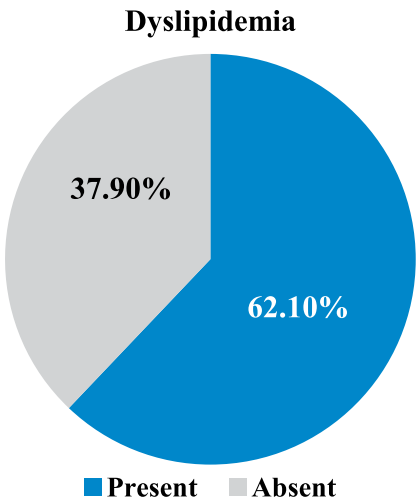


Figure 1: Frequency of Dyslipidemia among NAFLD Patients

DISCUSSION

This study evaluated the association between dyslipidemia and clinical, biochemical, and radiological features of non-alcoholic fatty liver disease (NAFLD) among 116 patients. Age and gender did not show significant associations with dyslipidemia in our study (p=0.829 and p=0.241, respectively). Comparable findings were noted by Ferreira *et al.* and Krishan, where demographic variables like age and sex did not significantly influence the presence of dyslipidemia among NAFLD patients [18, 19]. However, Dowla *et al.* documented age and race-dependent variations, particularly earlier NAFLD onset and higher dyslipidemia rates among Hispanic children, suggesting that population-specific genetic and environmental factors may modulate lipid patterns [13]. In the present study, diabetes mellitus (DM) was significantly more prevalent among dyslipidemic NAFLD patients (68.1% vs. 45.5%, p=0.016), with an odds ratio of 2.557. This finding resonates with studies by Ferreira *et al.* and Méndez-Sánchez *et al.* which identified type 2 diabetes mellitus (T2DM) as an independent predictor of advanced fibrosis and dyslipidemia in NAFLD patients [18, 20]. The interplay of insulin resistance with hepatic lipid metabolism is a well-established driver of steatosis and subsequent liver injury. Ischemic heart disease (IHD) also demonstrated a robust association with dyslipidemia (p=0.004), with an OR of 3.6, underscoring the shared pathophysiological axis between hepatic fat accumulation and atherogenesis. These observations are supported by Dowla *et al.* and Risal *et al.* who emphasized cardiovascular comorbidity as an essential component of the NAFLD metabolic phenotype. Although hypertension showed a higher prevalence among dyslipidemic individuals (54.2% vs. 38.6%), statistical significance was not attained (p=0.104) [13, 21]. In contrast, Méndez-Sánchez *et al.* demonstrated a significant link between hypertension and advanced fibrosis, with hypertension emerging as an independent risk factor (OR 2.59, p=0.014) [20]. The mean BMI in the dyslipidemic group was higher (29.26 ± 4.76 vs. 26.64 ± 3.84kg/m^2, p=0.002), reaffirming obesity as a central contributor to both hepatic steatosis and lipid dysregulation. This finding is corroborated by Cigri *et al.* who reported significantly elevated BMI in pediatric NAFLD patients (23.5 vs. 22.1 kg/m^2, p<0.001), and Risal et al. where NAFLD patients had significantly higher BMI than controls (26.41 ± 4.03 vs. 23.48 ± 2.85kg/m^2, p<0.001) [12, 21]. Similarly, Ferreira *et al.* showed significantly increased BMI and waist circumference in NAFLD individuals [18]. Transaminases were significantly elevated in dyslipidemic NAFLD patients in our study. AST levels were higher (41.89 ± 11.45 vs. 36.56 ± 10.08 IU/L, p=0.012), and ALT values also showed significance (54.93 ± 17.09 vs. 48.59 ± 14.18 IU/L, p=0.041).

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These findings mirror the results of Singh *et al.* and Cigri *et al.* where ALT and AST levels were higher in patients with elevated triglycerides and NAFLD [22, 12]. Dowla *et al.* further highlighted strong correlations between ALT and other metabolic markers, including GGT, TG, and non-HDL-C [13]. Notably, Cigri *et al.* identified ALT as a highly sensitive diagnostic biomarker for NAFLD (AUC = 0.986), with sex-specific cut-off values suggesting the clinical utility of ALT in early screening [12]. Dyslipidemic NAFLD patients exhibited significantly higher total cholesterol (216.11 ± 18.74 vs. 205.17 ± 36.88mg/dL, p=0.037), triglycerides (201.99 ± 20.58 vs. 170.05 ± 24.59mg/dL, p<0.001), and LDL-C (143.67 ± 15.46 vs. 119.73 ± 18.46mg/dL, p<0.001), with HDL-C levels being significantly lower (35.35 ± 4.52 vs. 41.38 ± 7.93mg/dL, p<0.001). These findings are in agreement with Singh *et al.* who reported significantly elevated triglycerides (194.3 ± 104.7 vs. 131.9 ± 53.1 mg/dL) and lower HDL-C in NAFLD patients [22]. Similarly, Méndez-Sánchez *et al.* found triglycerides and LDL-C as independent predictors of fibrosis (OR for TG = 4.96, LDL-C = 3.04, both p<0.05) [20]. Risal *et al.* and Dowla *et al.* also reported lipid derangements, particularly higher TG, LDL-C, and non-HDL-C levels in NAFLD subjects compared to controls or normolipidemic subgroups [13, 21]. The current study's mean LDL-C of 143.67 mg/dL in the dyslipidemic NAFLD group falls well within the elevated range identified in these prior analyses, further validating the link between hepatic fat accumulation and atherogenic lipid patterns. The frequency of dyslipidemia was notably high (62.1%) among NAFLD individuals, consistent with the dyslipidemic burden documented in global and regional literature. The frequency of dyslipidemia in NAFLD in the present study aligns with figures reported by Singh *et al.* (NAFLD n=2436), who observed a dyslipidemia frequency exceeding 60%, and Krishan, where 65% of T2DM patients had NAFLD and a significantly high burden of lipid abnormalities [19, 22]. Similarly, Risal *et al.* identified dyslipidemia in a majority of NAFLD cases compared to controls, particularly low HDL-C and high LDL-C [21]. The link between dyslipidemia and NAFLD likely reflects insulin resistance–driven hepatic lipid accumulation. Increased free fatty acid flux to the liver enhances triglyceride synthesis, while impaired VLDL secretion and reduced HDL formation create an atherogenic lipid profile that perpetuates hepatic steatosis. A significant correlation was observed between the presence of dyslipidemia and NAFLD severity grades (p<0.001), with 33.3% of dyslipidemic patients categorized in Grade 3 compared to only 4.5% among the non-dyslipidemic group. This trend of increasing dyslipidemia with advancing steatosis is supported by Krishan, who observed worsening lipid parameters across NAFLD grades, and by Risal *et al.* whose grade III NAFLD subgroup showed the highest non-HDL-C and atherogenic ratios [19, 21]. The pattern underscores the progressive metabolic derangement associated with steatohepatitis. Furthermore, Méndez-Sánchez *et al.* demonstrated that dyslipidemia not only coexists with but also contributes to fibrosis progression and cirrhosis risk in biopsy-confirmed NASH, with LDL-C and TG emerging as key markers [20]. Given the statistically significant correlations between dyslipidemia and key metabolic and hepatic parameters, early lipid profiling in NAFLD patients is essential. Integration of lipid management into NAFLD care protocols may reduce not only hepatic complications but also the cardiovascular burden, as emphasized by multiple global studies. The study's strength lies in its comprehensive analysis of dyslipidemia in relation to NAFLD severity, incorporating both clinical and biochemical parameters using robust statistical methods. The use of ultrasound grading and detailed lipid profiling enhances diagnostic reliability.

However, limitations include the cross-sectional design, absence of lifestyle variable adjustment, and single-center data collection, potentially affecting generalizability. Absence of liver biopsy restricts histological correlation. Future studies should employ longitudinal designs with larger, multicenter cohorts and incorporate non-invasive fibrosis markers or liver histopathology to validate findings. Investigating therapeutic responses to lipid-lowering agents in NAFLD subgroups may also offer valuable clinical insights.

CONCLUSIONS

This study demonstrates a high frequency of dyslipidemia (62.1%) among patients with non-alcoholic fatty liver disease, with significant associations observed with diabetes mellitus, ischemic heart disease, elevated body mass index, and worsening hepatic enzyme levels. Dyslipidemia was significantly linked with higher NAFLD grades, suggesting its potential role in disease progression. These findings underscore the importance of routine lipid profiling and metabolic screening in NAFLD patients to guide timely intervention and risk stratification for cardiometabolic complications.

Authors' Contribution

Conceptualization: AU, UA
Methodology: AA, SS, AB, AU, UA, MIJ
Formal analysis: AA, MIJ
Writing and Drafting: AA, UA, MIJ
Review and Editing: AA, SS, AB, AU, UA, MIJ

All authors approved the final manuscript and take responsibility for the integrity of the work

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Conflicts of Interest

All the authors declare no conflict of interest.

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