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# A STUDY OF CORRELATION BETWEEN NON-ALCOHOLIC FATTY LIVER DISEASE AND SERUM LIPOPROTEIN A LEVEL

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## ABSTRACT

Non-alcoholic fatty liver disease (NAFLD) is a prevalent metabolic disorder with significant implications for cardiovascular health.

Lipoprotein(a) [Lp(a)] is a genetically determined lipoprotein associated with atherosclerosis and cardiovascular risk. However, its relationship with NAFLD remains unclear. Some studies suggest an inverse association, where lower Lp(a) levels correlate with increased hepatic fat accumulation and advanced fibrosis. Other studies report no significant association or even elevated Lp(a) levels in NAFLD patients. Given these inconsistencies, the role of Lp(a) in NAFLD pathogenesis, progression, and cardiovascular risk requires further investigation. This review highlights current evidence, explores potential mechanisms linking Lp(a) to liver metabolism, and discusses its potential as a biomarker for NAFLD severity and related complications. Future studies with larger, well-defined cohorts and mechanistic insight are needed to establish the clinical relevance of Lp(a) in NAFLD.

**Keywords:** Lipoprotein (a), Non alcoholic fatty Liver disease (NAFLD).

## INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most prevalent chronic liver condition globally, affecting approximately 25% of the adult population. It encompasses a spectrum of liver abnormalities, from simple steatosis to non-alcoholic steatohepatitis (NASH), which can progress to fibrosis, cirrhosis, and hepatocellular carcinoma. NAFLD is closely associated with metabolic syndrome components, including obesity, insulin resistance, dyslipidemia, and hypertension, and is recognized as an independent risk factor for cardiovascular diseases (CVD)[1].Lipoprotein(a) [Lp(a)] is a genetically determined plasma lipoprotein consisting of a low-density lipoprotein (LDL) particle attached to apolipoprotein(a). Elevated Lp(a) levels have been identified as an independent risk factor for atherosclerotic CVD due to their pro-atherogenic and pro-thrombotic properties. However, the relationship between Lp(a) levels and NAFLD remains ambiguous, with studies yielding conflicting results[2].Some research indicates an inverse association between Lp(a) levels and NAFLD. For instance, a study involving 600 adults with biopsy-confirmed NAFLD found that lower Lp(a) concentrations were associated with more advanced liver fibrosis or cirrhosis. Conversely, other studies have reported no significant correlation or even elevated Lp(a) levels in NAFLD patients. A systematic review encompassing ten observational studies with 40,045 participants concluded that the association between Lp(a) levels and

hepatic steatosis is
inconclusive[3].Understanding the interplay between Lp(a) and NAFLD is crucial, given the significant cardiovascular risk associated with both elevated Lp(a) levels and NAFLD. Clarifying this relationship could enhance risk stratification and inform therapeutic strategies for individuals with NAFLD. This review aims to synthesize
current evidence regarding the association between serum Lp(a) levels and NAFLD, explore potential underlying mechanisms, and identify areas requiring further research to elucidate the clinical implications of this association[4].

## METHODOLOGY

**STUDY DESIGN:** An Analytical cross sectional study
**SAMPLE SIZE:** A total of 15 patients who were having NAFLD( sonological) were studied.
**PERIOD OF STUDY:** The study was conducted over a period of 3 months, between NOVEMBER 2024 to JANUARY 2025.
Assessment of fatty liver disease.
Fatty liver disease was diagnosed based on abdominal ultrasonography .

**To send Serum lipoprotein a .**
- 8hrs of an fasting period, blood samples were collected from all the subjects. Normal serum Lp a level 12-30 mg/dl
reference ranges are: based on the risk of coronary artery disease and stroke.
Lp(a) in mg/dL:
- Less than 10 mg/dL → Low risk 10–30 mg/dL → Mild risk
30–50 mg/dL → Moderate risk Above 50 mg/dL → High risk

## INCLUSION CRITERIA

-Age >18 yrs
-Who underwent abdominal ultrasonography and found to have Fatty liver.

## EXCLUSION CRITERIA

-Patients who are T2DM, Hypothyroid.
-Any liver diseases like Hepatitis, Liver cirrhosis, HCC at baseline were excluded
-Alcoholic: individuals with alcohol consumption exceeding 30gm in men, 20gm for women.
-Patients who are on lipid lowering drugs.

## DATA ANALYSIS:

Sample size: 15

**SERUM LIPOPROTEIN (a) LEVEL IN THE STUDY GROUP**

| Lipoprotein (a) Level (mg/dL) | Number of Patients | Percentage |
|---|---|---|
| <12 mg/dL | 8 | 53.3% |
| 12–30 mg/dL | 6 | 40% |
| 30–50 mg/dL | 1 | 6.6% |
| >50 mg/dL | 0 | 0% |

**Total patients = 15**

**Chi-square test p-value = 0.0076 (significant deviation from uniform distribution)**

**RESULTS:**

**1.** Majority Have Low Lp(a) Levels (<30 mg/dL)
G3.3% (14 out of 15 patients) have Lp(a) below 30 mg/dL.
This suggests that high Lp(a) is uncommon in NAFLD patient group.
**2.** G3.3% of NAFLD patients have Lp(a) <30 mg/dL (relatively low).
No patients had Lp(a) >50 mg/dL, which is generally considered high.
The p-value (0.0076) suggests a non-random distribution, meaning low Lp(a) levels are more common than expected.

**This pattern supports the idea of an inverse relationship:**
**- As NAFLD is present, Lp(a) levels appear lower than expected.**

**DISCUSSION:**

The relationship between serum lipoprotein(a)
[Lp(a)] levels and non-alcoholic fatty liver disease (NAFLD) has been widely debated, with studies yielding inconsistent findings. Some research suggests an inverse association, where lower
Lp(a) levels are linked to increased hepatic fat accumulation and advanced fibrosis, while others report no significant correlation or even elevated Lp(a) levels in NAFLD patients. This
variability indicates a complex interplay between lipid metabolism, liver function, and
cardiovascular risk.

**Potential Mechanisms Linking Lp(a) to NAFLD**

Several mechanisms may explain the association between Lp(a) and NAFLD:

**1. Lipid Metabolism and Hepatic Fat Accumulation:**
Lp(a) is primarily synthesized in the liver and shares metabolic pathways with other lipoproteins, such as low-density lipoprotein (LDL) and very-low-density lipoprotein (VLDL), both of which are involved in NAFLD pathogenesis. Lower Lp(a) levels in NAFLD patients may reflect altered hepatic lipid metabolism and impaired lipoprotein synthesis.

**2. Inflammation and Fibrosis Progression:**
Lp(a) contains oxidized phospholipids that promote inflammation and fibrosis. Elevated Lp(a) levels have been linked to cardiovascular diseases due to their pro-inflammatory properties, yet some studies suggest that in NAFLD, lower Lp(a) levels might contribute to greater liver inflammation and fibrosis progression. The exact mechanism behind this paradox remains unclear.

**3. Genetic and Ethnic Variability:**
Lp(a) levels are largely genetically determined and vary significantly among individuals and ethnic groups. Studies suggest that genetic predisposition may influence both Lp(a) levels and NAFLD susceptibility, contributing to the inconsistent findings in different populations.

## CONCLUSION:

There is a inverse correlation between the NAFLD and serum Lipoprotein (a) Level. Understanding this association could improve disease risk assessment, guide personalized treatment approaches, and enhance cardiovascular risk management in NAFLD patients.

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