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# Correlation Between Total Cholesterol, HDL, and Non-HDL Cholesterol with Apolipoprotein B in Hyperlipidemia Patients

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Hyperlipidemia is a major modifiable risk factor for atherosclerotic cardiovascular disease. Among the available laboratory markers, non-HDL cholesterol and apolipoprotein B have attracted considerable attention as more informative measures of the true atherogenic burden carried by a patient's blood. 51 hyperlipidemia patients were recruited sequentially for our cross-sectional study at the Clinical Laboratory of UNS Hospital, Sukoharjo. A power calculation for correlation studies ($\alpha = 0.05$, power = 80%) was used to estimate the sample size. A TMS 30i analyser was used to quantify total cholesterol, HDL, and apo B; non-HDL cholesterol was calculated by subtraction. For every association, Pearson product-moment correlation coefficients with 95% confidence intervals (CIs) were calculated using Fisher's z-transformation. There was a high and positive correlation between total cholesterol and apo B ($r = 0.881$, 95% CI: 0.803–0.932, $p < 0.001$). There was no significant correlation between HDL cholesterol and apo B ($r = 0.082$, 95% CI: −0.196 to 0.349, $p = 0.566$). Apo B and non-HDL cholesterol had the highest correlation by far ($r = 0.938$, 95% CI: 0.896–0.963, $p < 0.001$). In hyperlipidemic, non-HDL cholesterol followed apo B more accurately than any other parameter, supporting its usage as a useful, affordable substitute for apo B in situations when direct measurement is not possible. To strengthen these relationships, larger, prospective, multi-center studies that account for important variables are required.

**Keywords:** Apolipoprotein B, HDL Cholesterol, Hyperlipidemia, Non-HDL Cholesterol, Total Cholesterol

## INTRODUCTION

A medical disorder known as hyperlipidemia is defined by elevated blood levels of fats, such as triglycerides and cholesterol (Kala & Singhai, 2021). This tendency has been exacerbated by ageing populations, sedentary lifestyles, growing waistlines, and unhealthy diets. The World Health Organization estimated that nearly two in five adults globally had elevated total cholesterol as far back as 2008, and the situation has only worsened since (Organization, 2011). There are notable regional differences in the prevalence of hyperlipidemia. In the United States, about 94 million adults (age 20 and older) have total cholesterol levels above 200 mg/dL, with 28 million of them having levels above 240 mg/dL (Viran et al., 2020). The problem is not confined to wealthy nations: surveillance data from China, India, and South Korea all point to steep rises in hyperlipidaemia prevalence over recent decades (Joshi et al., 2014; Kim & Oh, 2013; Pan et al., 2016). In resource-limited settings, where preventive care is harder to access, these numbers carry especially heavy consequences.

Because hyperlipidemia plays a major part in atherosclerosis—the hazardous, gradual furring of artery walls that ultimately results in heart attacks and strokes—clinicians take it very seriously (Lawler et al., 2017). Lipid-carrying particles cause oxidative stress and inflammation in the artery walls, which over time develops into the plaques that cause the majority of cardiovascular deaths globally (WHO, 2021). The quality of the laboratory indicators we employ to assess atherogenic risk is crucial to identifying and stopping this process early.

Low-density lipoprotein cholesterol (LDL-C) has been the accepted metric for this purpose for many years. It is extensively accessible, thoroughly researched, and unmistakably connected to cardiovascular outcomes. However, LDL-C has a blind spot because it only counts the quantity of cholesterol contained in LDL particles. Many small, dense LDL particles can circulate at a normal LDL-C level in patients with type 2 diabetes, obesity, or hypertriglyceridemia, giving them a false sense of security (Mora et al., 2014). Researchers and medical professionals are searching for more accurate indicators as a result of this information gap.

There are two very noteworthy candidates. Because it includes the cholesterol carried by all atherogenic lipoprotein classes—LDL, very low-density lipoprotein (VLDL), intermediate-density lipoprotein (IDL), and remnants—non-HDL cholesterol, which is simply total cholesterol minus HDL cholesterol, provides a more comprehensive picture of atherogenic burden than LDL-C alone (Grundy et al., 2019). Apolipoprotein B (apo B) goes one step further: the plasma apo B concentration is a direct indicator of the number of atherogenic lipoprotein particles in circulation since each particle carries exactly one apo B molecule. Because of this, it may be the most accurate single indicator of atherogenic particle load that is used in daily practice (Sniderman & Marcovina, 2023). Apo B is now recommended as a secondary (or sometimes primary) treatment target by major cardiology recommendations from North America and Europe (Mach et al., 2020).

But there is a practical barrier. Immunoturbidimetry measurement of apolipoprotein B is more expensive and necessitates certain reagents that are not always available, especially in hospitals with limited resources. In contrast, non-HDL cholesterol can be computed from any conventional lipid panel and comes at no additional expense. When direct apo B testing is not feasible, non-HDL cholesterol may be a good substitute if it closely tracks apo B in patients with hyperlipidemia. This subject was the focus of the current investigation, which also looked at the relationship between apo B and total and HDL cholesterol in the same cohort. All of the associations presented here are associative because we employed a cross-sectional design; hence, judgements regarding causality cannot be made.

## METHOD

This cross-sectional observational study was conducted at the UNS Hospital's Clinical Laboratory in Sukoharjo, Central Java, Indonesia. We choose a cross-sectional technique because it is practical in a single-center scenario and well adapted to capturing concurrent connections between laboratory data in a clinical population. Because it just records a single moment in time rather than tracking patients across time, it has an inherent restriction that allows us to explain relationships but not identify which variable influences which.

Individuals with a diagnosis of hyperlipidemia who came to the lab were enrolled one at a time until the predetermined goal was accomplished. We calculated the minimum required sample size using the correlation study formula: at least 51 participants were needed with a two-tailed α of 0.05 and statistical power of 80%. Patients have to be willing to sign an informed consent form and be between the ages of 18 and 70 in order to qualify. Since the liver is the primary organ involved in lipoprotein metabolism and hepatic impairment can generate unforeseen distortions in the link between lipid markers and apo B, we did not include anyone with a history of liver disease. Before providing their agreement, each participant was thoroughly informed about the goals and methods of the study.

Each participant fasted for at least ten hours the night before a 5 mL blood sample was taken. A TMS 30i clinical chemistry analyser was used to assess total cholesterol, HDL cholesterol, and apo B using kinetic photometry and immunoturbidimetry, respectively. After that, non-HDL cholesterol was determined by subtracting HDL cholesterol from total cholesterol, which is a simple mathematical operation that doesn't require any extra chemicals. We performed two levels of internal quality control material (low and high concentration) for each analyte prior to processing any patient samples. Before moving on, the results have to be within the predetermined acceptance range (mean ± 2 SD). Additionally, precision experiments were carried out beforehand to verify that every assay fulfilled acceptable reproducibility standards.

### Ethics

The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Medicine, Sebelas Maret University (registration number: 190/UN27.06.11/KEP/EC/2024) and was conducted in line with the ethical principles set out in the Declaration of Helsinki.

### Statistical Analysis

While continuous variables are explained by mean ± standard deviation (SD), categorical variables are described by counts and percentages. Initially, we verified that the data were normally distributed using the Shapiro-Wilk test. Since all variables met the normality criterion ($p > 0.05$), Pearson product-moment correlation coefficients were used to analyse the three relationships of interest: total cholesterol with apo B, HDL cholesterol with apo B, and non-HDL cholesterol with apo B. Correlation strength was interpreted using standard thresholds: $r \geq 0.800$ was regarded as very high, 0.600–0.799 as strong, 0.400–0.599 as moderate, 0.200–0.399 as weak, and less than 0.200 as extremely weak.

To demonstrate the accuracy of each estimate, we computed 95% confidence intervals for each correlation coefficient using Fisher's z-transformation. The sample size of 51 prevented reliable multivariable modelling, even though we acknowledge that the analysis would have been strengthened by controlling for potential confounders such age, sex, body mass index, diabetes, hypertension, and the use of lipid-lowering drugs. This is acknowledged as a study limitation. All calculations were performed using SPSS version 25.0, with statistical significance set at $p < 0.05$ (two-tailed) (IBM Corp., Armonk, NY, USA).

## RESULTS AND DISCUSSION

The study included fifty-one hyperlipidemic patients. Table 1 summarises their attributes. With nearly equal numbers of men (49%) and women (51%), the average age was 58.16 ± 11.65 years, with the youngest participant being 24 and the oldest being 70. The average

levels of total cholesterol were 192.84 ± 44.57 mg/dL, HDL cholesterol was 38.65 ± 11.29 mg/dL, non-HDL cholesterol was 153.61 ± 39.78 mg/dL, and apo B was 95.35 ± 21.33 mg/dL. When combined, these numbers point to a population with a significant atherogenic burden even when total cholesterol levels are in the borderline range.

**Table 1.**

Baseline characteristics of study participants

| Variable | Mean ± SD | Minimum | Maximum |
|---|---|---|---|
| Age (years) | 58.16 ± 11.65 | 24.00 | 70.00 |
| Sex, n (%) | | | |
| Male | 25 (49%) | — | — |
| Female | 26 (51%) | — | — |
| Total cholesterol (mg/dL) | 192.84 ± 44.57 | 118.00 | 335.00 |
| HDL cholesterol (mg/dL) | 38.65 ± 11.29 | 16.00 | 66.00 |
| Non-HDL cholesterol (mg/dL) | 153.61 ± 39.78 | 91.00 | 281.00 |
| Apolipoprotein B (mg/dL) | 95.35 ± 21.33 | 47.10 | 165.30 |

*SD, standard deviation.*

**How Closely Did Total Cholesterol Track Apolipoprotein B?**

The data points in Figure 1 clearly show an increasing diagonal distribution, which conveys a simple message: apo B rises in tandem with total cholesterol. This perception was validated by statistical analysis: r = 0.881 (95% CI: 0.803–0.932, p < 0.001), firmly placing this link in the "very strong" category (Table 2). Given the sample size, the small confidence interval indicates strong dependability, and the result is statistically significant.

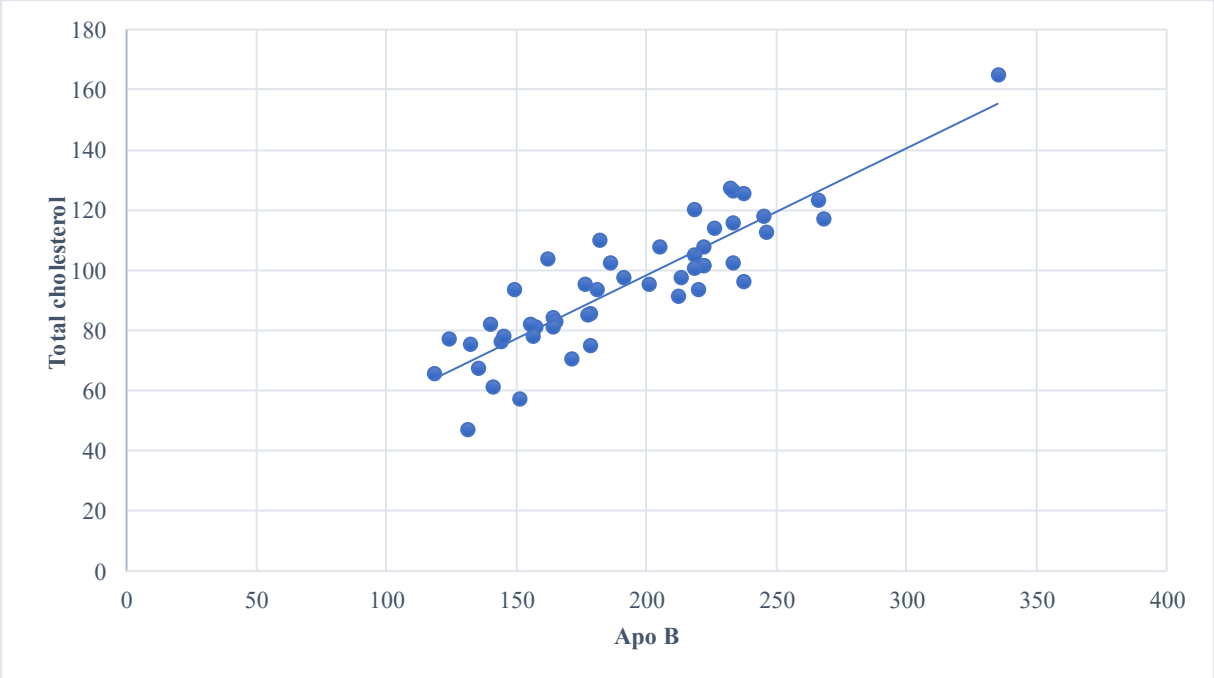


**Figure 1.** Scatterplot of the correlation between total cholesterol and apolipoprotein B in hyperlipidaemia patients

**Table 2.**

Correlation between total cholesterol and apolipoprotein B

| Variable | r | 95% CI | p-value |
|---|---|---|---|
| Total cholesterol vs. Apo B | 0.881 | 0.803 – 0.932 | < 0.001* |

Pearson product-moment correlation. *Significant at $p < 0.05$. CI, confidence interval.

**Did HDL Cholesterol Relate to Apolipoprotein B?**

The image was quite different in this instance. The correlation coefficient, r = 0.082 (95% CI: −0.196 to 0.349, p = 0.566), supported the scatterplot in Figure 2, which displays data points dispersed widely with no discernable trend (Table 3). Knowing a patient's HDL level in this sample provided virtually no information regarding their apo B level because the association is extremely weak, falls well short of statistical significance, and the confidence interval crosses zero.

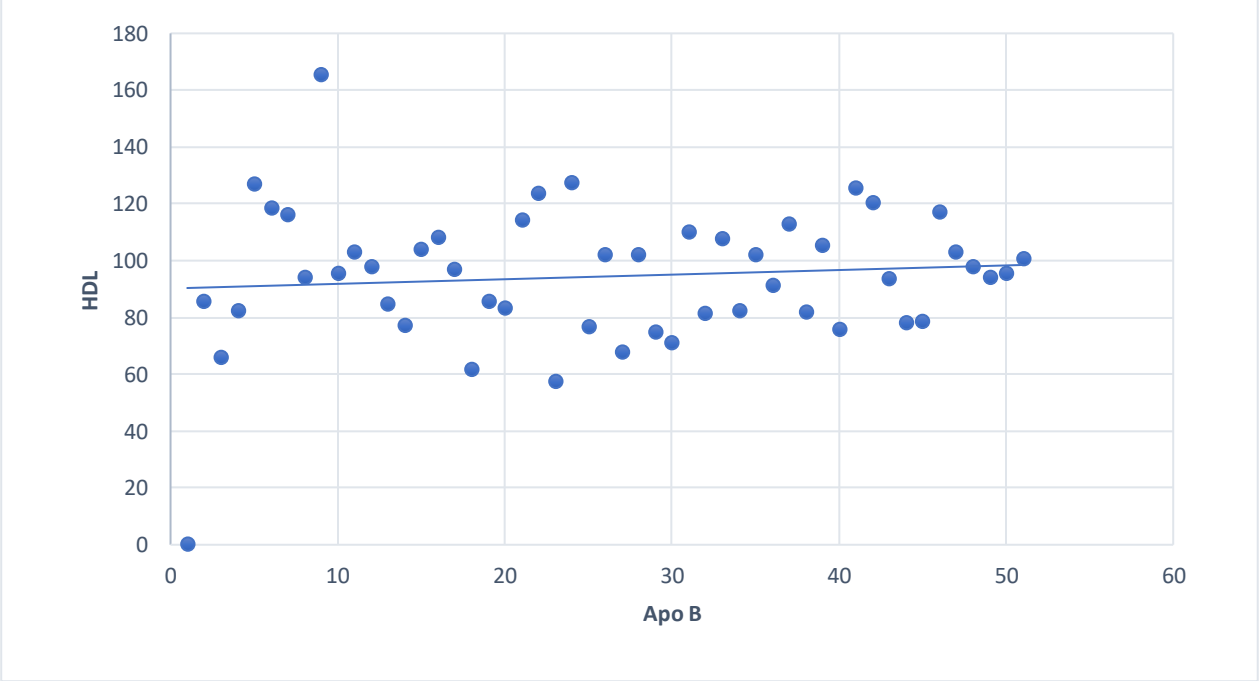


**Figure 2.** Scatterplot of the correlation between HDL cholesterol and apolipoprotein B in hyperlipidaemia patients

**Table 3.**

Correlation between HDL cholesterol and apolipoprotein B

| Variable | r | 95% CI | p-value |
|---|---|---|---|
| HDL cholesterol vs. Apo B | 0.082 | −0.196 to 0.349 | 0.566 |

Pearson product-moment correlation. CI, confidence interval.

**How Closely Did Non-HDL Cholesterol Track Apolipoprotein B?**

The most startling discovery was this. The study's greatest correlation coefficient was r = 0.938 (95% CI: 0.896–0.963, $p < 0.001$) (Table 4), and the scatterplot in Figure 3 displays data points firmly hugging the regression line. In these patients, apo B and non-HDL cholesterol moved together so regularly that non-HDL cholesterol explained about 88% of the variation in apo B levels ($r^2 = 0.88$).

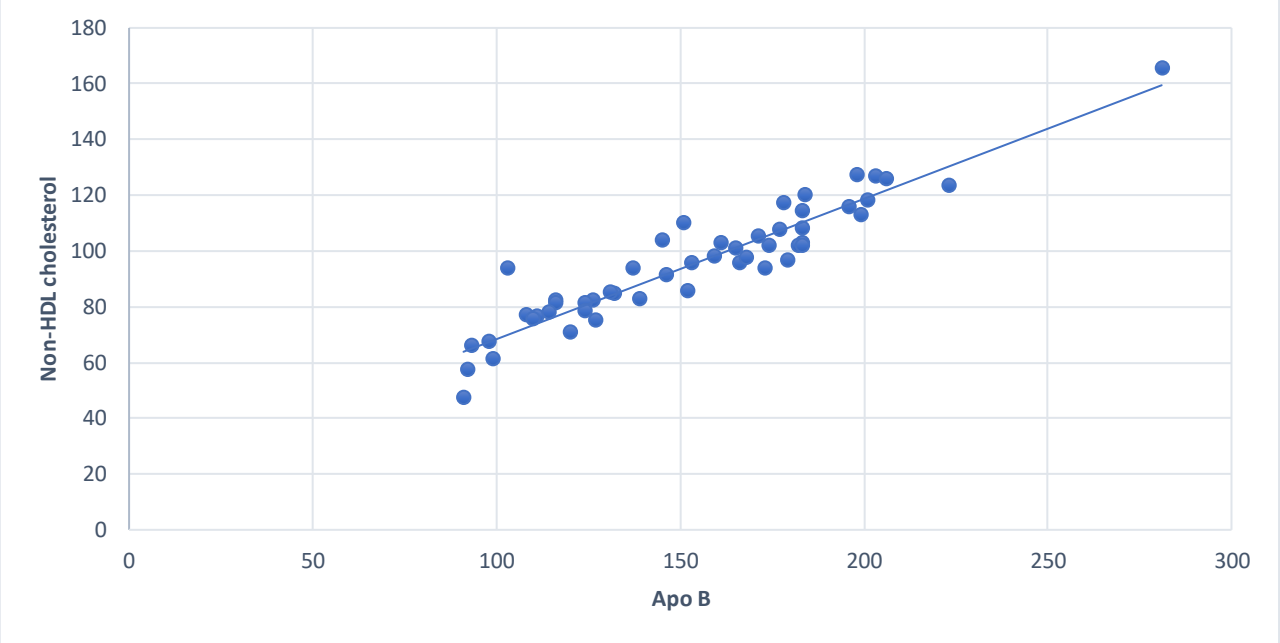


**Figure 3.** Scatterplot of the correlation between non-HDL cholesterol and apolipoprotein B in hyperlipidaemia patients

**Table 4.**

Correlation between non-HDL cholesterol and apolipoprotein B

| Variable | r | 95% CI | p-value |
|---|---|---|---|
| Non-HDL cholesterol vs. Apo B | 0.938 | 0.896 – 0.963 | < 0.001* |

Pearson product-moment correlation. *Significant at $p < 0.05$. CI, confidence interval.

**Total Cholesterol and Apo B: A Relationship Grounded in Biology**

The very significant connection we found between total cholesterol and apo B (r = 0.881) makes perfect sense given how these two variables are associated at the molecular level. Apo B-containing lipoproteins, mainly in LDL particles, which each contain a single apo B molecule, carry the majority of blood cholesterol (Sniderman & Marcovina, 2023). As a result, it is entirely predicted that apo B will grow in parallel with an increase in total cholesterol, which is mostly brought on by LDL accumulation.

This observation sits comfortably alongside recent literature. In the Copenhagen General Population Study, Johannesen et al., (2021) followed 13,015 statin-treated patients over eight years and confirmed the tight colinearity between LDL-C, total cholesterol, and apo B at the population level— while also pointing out that individuals with metabolic syndrome or hypertriglyceridemia have a breakdown in this tidy association. Similarly, Sniderman et al., (2011) showed in a meta-analysis of 233,455 adults that the three primary cholesterol markers cluster closely together in unselected groups, with significant divergence only occurring in those with aberrant lipoprotein composition. This message was reaffirmed by the 2024 National Lipid Association (NLA) consensus and Contois et al., (2023): on average, the markers agree well, but clinically significant discordance—where apo B is increased despite normal LDL-C or total cholesterol—occurs, especially in insulin-resistant patients (Contois et al., 2023; Soffer et al., 2024).

One reason our correlation coefficient (r = 0.881) may sit at the higher end of published estimates is that our cohort was entirely composed of hyperlipidaemic patients. In such a group, the predominant lipoproteins are cholesterol-rich LDL particles, which reduces the variability introduced by cholesterol-depleted apo B particles and thereby tightens the statistical relationship. This is worth bearing in mind when comparing our findings to population-based studies that include a broader spectrum of metabolic profiles. And, as with all cross-sectional observations, this remains an association; the direction of influence between total cholesterol and apo B cannot be inferred from these data.

**HDL Cholesterol and Apo B: Two Markers, Two Very Different Stories**

The near-zero correlation between HDL cholesterol and apo B (r = 0.082, p = 0.566) may initially seem surprising, but it reflects one of the most fundamental

distinctions in lipoprotein biology. HDL particles do not carry apo B at all—they are built around apolipoprotein A-I instead, and they serve an almost opposite physiological function, retrieving excess cholesterol from peripheral tissues and ferrying it back to the liver (Mach et al., 2020). It would therefore be remarkable if HDL cholesterol and apo B were closely correlated.

Large-scale evidence supports this interpretation. Information from the New Risk Factors The preventive effect of HDL against cardiovascular disease is regularly demonstrated by collaboration and other prospective studies to be essentially independent of apo B (Welsh et al., 2019). However, there is a nuance that should be noted: low HDL frequently co-occurs with increased apo B in patients with atherogenic dyslipidemia, which is characterised by low HDL, elevated triglycerides, and an abundance of tiny dense LDL particles. In that situation, sufficiently large and metabolically diverse samples may show an inverse connection (Soffer et al., 2024). The small sample size (reflected in the wide confidence interval: −0.196 to 0.349) and lack of thorough metabolic phenotyping of our subjects are probably the reasons we did not see this in our study. Future research would be better suited to examine this subtlety with bigger sample sizes and triglyceride data. Non-HDL Cholesterol and Apo B: The Closest Partnership.

The strongest correlation in this study—and its most clinically useful finding—was the near-unity agreement between non-HDL cholesterol and apo B (r = 0.938). This is not coincidental. Non-HDL cholesterol is the sum of cholesterol carried by every atherogenic lipoprotein class: LDL, VLDL, IDL, and remnants—all of which, without exception, carry apo B. In a sense, non-HDL cholesterol and apo B are two different ways of measuring the same underlying phenomenon: the combined atherogenic lipoprotein burden. Non-HDL cholesterol measures the cholesterol mass within those particles; apo B counts the particles themselves.

Our outcome is in good agreement with a seminal prospective analysis. In the Copenhagen General Population Study, Johannesen et al., (2021) discovered that whereas elevated LDL-C in discordant cases did not predict all-cause mortality and myocardial infarction in statin-treated patients, elevated apo B and elevated non-HDL cholesterol did. Even though the study methods differ significantly, our results have some external validity because a cross-sectional analysis of 51 patients from a single Indonesian hospital finds a correlation (r = 0.938) congruent with data from a large Northern European prospective cohort.

Nevertheless, the recent literature offers crucial warnings against overinterpreting this correlation's closeness as indicating that the two measures may be used interchangeably in every clinical setting. While non-HDL cholesterol and apo B track each other well in most people, a 2025 systematic review of 15 studies with 593,354 participants found that when apo B is disproportionately high compared to non-HDL cholesterol, cardiovascular risk aligns with apo B rather than non-HDL (Johannesen et al., 2025; Sniderman et al., 2024). People with hypertriglyceridemia, insulin resistance, or the tiny dense LDL phenotype are more likely to experience this discrepancy—exactly the patients for whom apo B's special ability to quantify particles independent of cholesterol quantity is most beneficial.

This point was further clarified by the 2024 NLA Expert Consensus, which identified particular clinical subgroups—patients with diabetes, metabolic syndrome, chronic kidney disease, or very low LDL-C on intensive lipid-lowering therapy—in which non-HDL cholesterol may either overestimate or underestimate the actual atherogenic particle burden, making direct apo B measurement the more reliable option (Soffer et al., 2024). We cannot determine how many of our 51 participants fell into these higher-risk metabolic categories because our study did not collect data on diabetes status, triglyceride concentrations, or medication use. As a result, our findings cannot be assumed to apply consistently to all hyperlipidemic patients.

From a guideline standpoint, The status of apo B has been gradually improved by the most current international recommendations. Apo B is advised as a secondary therapeutic target in the 2019 ESC/EAS guidelines, with treatment objectives of < 65 mg/dL for very-high-risk patients and < 80 mg/dL for high-risk patients (Mach et al., 2020). Going one step further, the 2021 Canadian Cardiovascular Society guidelines allow apo B to take the position of LDL-C as the main objective. Apo B testing is specifically advised in individuals with diabetes and hypertriglyceridemia according to the 2023 ESC preventive cardiology guidelines (Visseren et al., 2023). In light of this, Langlois & Sniderman, (2020) suggestion that non-HDL cholesterol be used as a practical, affordable stand-in for apo B in healthcare settings where immunoturbidimetric apo B assays are not frequently available is practically supported by the strong non-HDL–apo B correlation we found in a real-world Indonesian hyperlipidemic cohort.

One additional contextual observation deserves mention. Although the mean total cholesterol of 192.84 mg/dL is only in the borderline normal range, the mean apo B of 95.35 mg/dL in our sample places the majority of individuals at or above the threshold for treatment consideration in high-risk patients (80 mg/dL). In contrast to an increased number of cholesterol-depleted particles, this gap suggests that many participants may have been carrying relatively cholesterol-dense LDL particles. This pattern may be consistent with the metabolic profile of middle-aged and older Asian adults, in whom cardiovascular risk may manifest at lower absolute cholesterol levels than in Western populations (Viran et al., 2020). This contextual variation highlights the need of apo B and its surrogate non-HDL cholesterol as supplementary instruments to LDL-C in clinical practice, especially in Asian healthcare environments.

Limitations

Several aspects of this study limit the confidence with which its findings can be applied. Most importantly,

the cross-sectional design means that the associations we describe reflect simultaneous measurements at one point in time—they tell us nothing about which marker drives changes in another or about future cardiovascular outcomes. We were unable to account for significant confounders like body mass index, triglyceride levels, diabetes, hypertension, or the use of statins and other lipid-lowering medications, all of which are known to independently affect both lipid levels and apo B concentrations, because the sample of 51 patients, while adequate for the primary correlation analyses by power calculation, left little room for subgroup analyses or multivariable modelling. Our findings might not apply to other groups with different metabolic or demographic characteristics due to the single-center approach. We were also unable to completely describe the participants' lipoprotein phenotypes due to the lack of LDL-C values. To fill these gaps, future studies should use prospective cohort designs with bigger, ethnically diverse, multi-center populations, thorough metabolic data, and multivariable adjustment.

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**CONFLICT OF INTEREST**

The authors have no competing interests to declare.

**CONCLUSION**

When we set out to understand how closely routine cholesterol measurements track apolipoprotein B in hyperlipidaemic patients, the results were clear and clinically meaningful. Non-HDL cholesterol emerged as the strongest predictor of apo B levels in our cohort ($r = 0.938$, 95% CI: 0.896–0.963, $p < 0.001$), followed closely by total cholesterol ($r = 0.881$, 95% CI: 0.803–0.932, $p < 0.001$). HDL cholesterol, as expected from its distinct biological role, showed no meaningful relationship with apo B ($r = 0.082$, 95% CI: −0.196 to 0.349, $p = 0.566$).

The majority of the atherogenic information given by apo B in typical clinical populations is captured by non-HDL cholesterol, according to a growing body of evidence from large prospective studies and guideline consensus statements. Calculating non-HDL cholesterol from a regular lipid panel seems to be a reasonable and affordable alternative for identifying patients at elevated atherogenic risk in hospitals and clinics in Indonesia and similar settings where direct apo B testing may not be routinely accessible.

However, these conclusions should be accompanied by three crucial cautions. First, as this was a cross-sectional study, the correlations we found cannot be understood as causal linkages because they represent co-occurrence at a particular time point. Second, our estimates carry statistical uncertainty and may not apply to all hyperlipidemic patients, especially those with diabetes, hypertriglyceridemia, or insulin resistance—subgroups in which direct apo B measurement continues to provide distinctive diagnostic value that non-HDL cholesterol alone cannot replicate. This is due to the small, single-center sample and the lack of data on metabolic confounders. Third, none of these correlations have been connected to specific clinical outcomes in our group; only a prospective investigation can determine whether these correlations are predictive of future cardiovascular events. We anticipate that future studies will expand on these first results using the larger, multi-center, longitudinal designs required to convert them into conclusive clinical recommendations.

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