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# CORRELATION OF APOLIPOPROTEIN B LEVELS WITH THE DEGREE OF CORONARY ARTERY STENOSIS IN CHRONIC CORONARY SYNDROME PATIENTS

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**ABSTRAK**

Apolipoprotein adalah komponen struktural dari lipoprotein plasma yang terlibat dalam metabolisme lipoprotein dan transportasi lipid, mencerminkan jumlah partikel aterogenik yang beredar. Penelitian ini bertujuan mencari korelasi kadar apolipoprotein B serum dengan derajat stenosis arteri koroner pada pasien sindrom koroner kronik. Menggunakan desain penelitian potong lintang, sebanyak 34 pasien dari total populasi 69 orang dilibatkan. Data dikumpulkan pada Juli 2024 – September 2024. Hasil menunjukkan sebagian besar responden memiliki kadar apolipoprotein B yang tinggi, dengan rerata 141,41±25,06 mg/dL dan skor SYNTAX ≥ 23 dengan rerata 24,09±2,69 mg/dL. Uji Pearson menunjukkan korelasi signifikan antara apolipoprotein B dan skor SYNTAX (p = 0,029 dan r = 0,376). Setelah dilakukan analisis multivariat didapatkan p *value* apolipoprotein B sebesar 0,017 dengan nilai koefesien regresi bernilai positif. Temuan ini menunjukan bahwa kenaikan kadar apolipoprotein B tetap berkorelasi signifikan dan berbanding lurus dengan skor SYNTAX.

**ABSTRACT**

Apolipoproteins are structural components of plasma lipoproteins that involved in lipoprotein metabolism and lipid transport, reflect the number of circulating atherogenic particles. This study aims to investigate the correlation between serum apolipoprotein B levels and the degree of coronary artery stenosis in patients with chronic coronary syndrome. A cross-sectional study design was used, involving 34 patients selected from a total population of 69 individuals. Data were collected from July 2024 to September 2024. The results showed that most respondents had elevated apolipoprotein B levels, with a mean value of 141.41 ± 25.06 mg/dL, and SYNTAX score of ≥ 23, with a mean value of 24.09 ± 2.69. Pearson's correlation test indicated a significant relationship between apolipoprotein B levels and SYNTAX scores (p = 0.029, r = 0.376). Multivariate analysis revealed a significant p-value for apolipoprotein B (p = 0.017) with a positive regression coefficient.

## INTRODUCTION

Chronic coronary syndrome (CCS) is a chronic form of coronary artery disease (CAD) that is generally stable but can progress and destabilize over time, often remaining clinically silent.[1] In 2019, the prevalence of chronic coronary syndrome in the United States was estimated at 20.1 million individuals.[2] In Indonesia, according to the Indonesian basic health research 2018, the prevalence of chronic coronary syndrome was 1,5% of the total population.[3] In 2019, the prevalence of chronic coronary syndrome in South Sumatera Province was 7,993 people, representing 8% of the population.[4]

Low-density lipoprotein (LDL) is the main atherogenic lipoprotein and the primary target in

current dyslipidemia management. However, several studies have indicated that measuring LDL is not superior to measuring apolipoproteins B in determining the risk of atherosclerosis. This is evident from the significant number of chronic coronary syndrome cases that still occur despite achieving target cholesterol levels.[5] According to the European Society of Cardiology, apolipoprotein B can be used as an option for cardiovascular disease risk identification, especially in patients with high triglyceride levels, diabetes mellitus, obesity, metabolic syndrome, or very low LDL levels.[5,6] Behbodikhah et al and Glavinovic et al stated that apolipoprotein B is a more accurate marker of atherosclerosis risk than LDL.[6,7] Apolipoprotein B represents all atherogenic lipoprotein particles. Due to the long half-life of LDL in plasma (3–4 days), more than 90% of apolipoprotein B-containing particles in plasma are LDL particles. Therefore, measurements of apolipoprotein B mostly reflect the number of LDL particles.[8] Apolipoprotein B is associated with the development of atherosclerosis, which can lead to obstruction or stenosis of coronary blood flow. The degree of coronary artery stenosis can be assessed by calculating the SYNTAX score. The SYNTAX score (Synergy between PCI with TAXUS and Cardiac Surgery) is an angiographic scoring system used to assess the complexity of coronary lesions.[9]

## METHOD

This study was a cross-sectional, analytical observational study.The inclusion criteria were patients with chronic coronary syndrome who underwent coronary angiography in the Cardiac Catheterization Unit of the Internal Medicine Department at Dr. Mohammad Hoesin Hospital Palembang from July to September 2024, aged over 18 years, with plaques detected that partially obstructed the coronary vessel lumen, and who were willing to participate and signed informed consent. The exclusion criteria were patients with severe chronic liver or kidney disease, malignancy, blood disorders, or severe infections; patients with microvascular or vasospastic angina, new-onset heart failure or left ventricular dysfunction, a history of revascularization, or those who had been taking lipid-lowering drugs for less than 2–6 weeks. The SYNTAX score was calculated by a consultant cardiovascular internist. Data were processed using SPSS 22. A significance level of $p < 0.05$ was considered statistically significant. Ethical approval for this study was obtained from the Health Research Ethics Committee of Dr. Mohammad Hoesin Hospital Palembang, with approval No.DP.04.03/D.XVIII.6.8/ETIK/110/2024.

## RESULT AND DISCUSSION

### General Characteristics of Subjects

In this study, the mean age of the research subjects was 61.29 ± 9.63 years. Most of the subjects were male, 29 subject (85.3%) and 5 subjects (14.7%) were female. In terms of education level, the majority had completed junior high school (13 subjects, 38.2%), followed by high school (12 subjects, 35.3%). Subjects with a Bachelor's degree accounted 7 subject (20.6%), and only 2 subjects (5.9%) had completed elementary education. Regarding occupation, 8 subjects (23.5%) were civil servants, military, or police officers, 20 subjects (58.8%) were private sector workers, and 6 subjects (17.6%) were unemployed.

**Risk Factor Characteristics of Study Subjects**

7 subjects (20.6%) were smokers, while the majority, 27 subjects (79.4%), were non-smokers. Regarding type 2 diabetes mellitus, 5 subjects (14.7%) were diagnosed with this condition, 29 subjects (85.3%) did not have type 2 diabetes mellitus. Hypertension was present in 11 subjects (32.4%), 23 subjects (67.6%) had no history of hypertension. Dyslipidemia was found in 26 subjects (76.5%), 8 subjects (23.5%) did not have dyslipidemia. The mean body mass index (BMI) of the subjects was 25.64 ± 3.85 kg/m². A total of 11 subjects (32.4%) had normal weight, 5 subjects (14.7%) were overweight, and 18 subjects (52.9%) were categorized as obese.

**Laboratory Findings of the Subjects**

Mean total cholesterol (175.88 ± 36.01 mg/dL), low-density lipoprotein (LDL) (123.59 ± 35.54 mg/dL), and apolipoprotein B (141.41 ± 25.06 mg/dL). The median high-density lipoprotein (HDL) level was 42 mg/dL (range: 29–80 mg/dL), the triglyceride level was 89 mg/dL (range: 40–289 mg/dL), and the median fasting blood glucose was 88.5 mg/dL (range: 54–303 mg/dL).

**SYNTAX Score Results**

The SYNTAX score was calculated based on the points assigned to each significant lesion identified using the coronary artery segment scheme. The mean SYNTAX score was 24.09 ± 2.69, indicating that patients with chronic coronary syndrome generally had a severe degree of arterial stenosis. There were 7 subjects (20.6%) with a SYNTAX score < 23 and 27 subjects (79.4%) with a score ≥ 23.

**Correlation of SYNTAX Score with Various Other Variables**

A significant correlation was found between age and SYNTAX score (p = 0.039, r = 0.591). The mean age of the subjects was 61.29 ± 9.63 years, indicating a wide age range within the sample. This finding underscores the importance of the age factor in the development of coronary artery stenosis. Among the lipid parameters studied, a significant correlation was found between the SYNTAX score and triglycerides (p = 0.025, r = 0.384). The median triglyceride value was 89 mg/dL, with a wide range from 40 to 289 mg/dL. This correlation emphasises the potential role of triglycerides in the pathogenesis of atherosclerosis. Interestingly, other lipid parameters such as total cholesterol with a mean of 175.88±36.01 mg/dL, LDL with a mean of 123.59±35.54 mg/dL, and HDL with a median of 42 (29-80) mg/dL showed no significant correlation with SYNTAX score. For smoking risk factors, 14.29% had SYNTAX scores < 23 and 85.71% had scores ≥ 23, diabetes mellitus 0% had scores < 23 and 100% had scores ≥ 23, hypertension 18.8% had scores < 23 and 81.82% had scores ≥ 23, and dyslipidaemia 15.38% had scores < 23 and 84.62% had SYNTAX scores ≥ 23.

**Table 1. Characteristics of the Research Subjects According to the Degree of Coronary Artery Stenosis**

| Variable | SYNTAX Score | |
|---|---|---|
| | <23 (n=7) | ≥ 23 (n=27) |
| Smoking | 1 (14,29%) | 6 (85,71%) |
| DM Type 2 | 0 (0%) | 5 (100%) |
| Hypertension | 2 (18,8%) | 9 (81,82%) |
| Dyslipidemia | 4 (15,38%) | 22 (84,62%) |

**Table 2**. **Characteristics of the Research Subjects**

| Characteristics | n (34) (%) | p |
|---|---|---|
| **Age (years)** | ≥ 18 (100) | 0,039[1] |
| **Gender, n (%)** | | 0,544[3] |
| Male | 29 (85,3) | |
| Female | 5 (14,7) | |
| **Smoking, n (%)** | | 0,713[3] |
| Yes | 7 (20,6) | |
| No | 27 (79,4) | |
| **DM Type 2, n (%)** | | 0,530[3] |
| Yes | 5 (14,7) | |
| No | 29 (85,3) | |
| **Hypertension, n (%)** | | 0,897[3] |
| Yes | 11 (32,4) | |
| No | 23 (67,6) | |
| **Dyslipidemia** | | 0,019[3] |
| Yes | 26 (76,5) | |
| No | 8 (23,5) | |
| **Body Mass Index** | | 0,791[1] |
| Normoweight | 11 (32,4) | |
| Overweight | 5 (14,7) | |
| Obese | 18 (52,9) | |
| **Laboratory (mean/median)** | | |
| Total Cholesterol | 175,88±36,01 | 0,191[1] |
| Low Density Lipoprotein | 123,59±35,54 | 0,648[1] |
| High Density Lipoprotein | 42 (29-80) | 0,167[2] |
| Triglycerides (mg/dL) | 89 (40-289) | 0,025[2] |
| Blood Glucose (mg/dL) | 88,5 (54-303) | 0,035[2] |
| Apolipoprotein B (mg/dL) | 141,41±25,06 | |
| **SYNTAX score** | 24,09±2,69 | |

*[1]Pearson test, [2]Spearman's test, [3]Independent t-test*

## Correlation Results Between Apolipoprotein B and SYNTAX Score

The results of this study showed a significant positive correlation between apolipoprotein B levels and the SYNTAX score. The correlation coefficient was r = 0.376, with a p-value of 0.029, indicating statistical significance ($p < 0.05$). This finding suggests that higher apolipoprotein B levels are associated with a greater degree of coronary artery stenosis. The figure below presents a scatter plot illustrating the correlation between apolipoprotein B levels and the SYNTAX score

**Table 3. Correlation Test Results Apolipoprotein B and SYNTAX Score**

| Variable | SYNTAX Score | |
|---|---|---|
| | Direction | p value |
| Apolipoprotein B | Positive | 0,029* |

*Pearson test

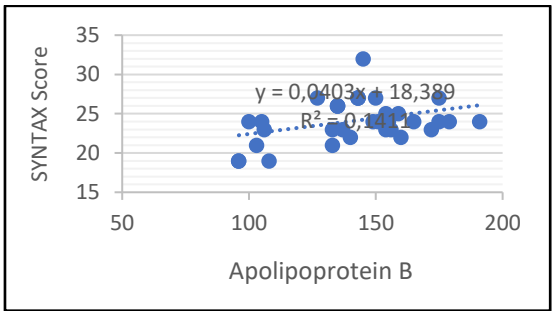


**Figure 1. Correlation Scatter Diagram of Apolipoprotein B Levels and SYNTAX Score**

## DISCUSSION

### General Profile of the Research Subjects

Of these 34 patients, 29 subjects (85.3%) were male and 5 subjects (14.7%) were female. These results are consistent with previous research by Muszynski et al., which showed that males are more likely to develop chronic coronary syndrome.[10] Another study conducted in Kuwait also reported that chronic coronary syndrome is more commonly found in male patients.[11] Men are considered to be at higher risk of developing chronic coronary syndrome, partly due to hormonal differences between men and women that can affect endothelial function and vasodilation. Estrogen has a protective effect on blood vessels, which can reduce the risk of atherosclerosis. processes and inhibits vascular smooth muscle. In addition, men tend to have more conventional cardiovascular risk factors such as smoking, hypertension, and dyslipidemia.[12]

In this study, The mean age of the subjects was 61.29 ± 9.63 years. This suggests that chronic coronary syndrome tends to occur in middle-aged individuals, although with considerable variation. The average age found in this study is similar to that reported by Alexander et al., which noted that patients most commonly affected by chronic coronary syndrome were aged ≥ 65 years.[13] Salimi et al in Iran on the potential of the HEART score to detect the severity of coronary artery disease based on the SYNTAX score in patients with chronic coronary syndrome, the mean age was 58.42 ± 12.42 years.[14] As age increases, a natural process of biological aging occurs in the blood vessels, affecting their structure and function. Blood vessels undergo a decline in elasticity (due to reduced elastin, increased deposition of extracellular matrix, increased collagen, and fibrosis), thickening of the intima-media layer, and reduced endothelial function. Older individuals tend to have more risk factors for atherosclerosis, including increased levels of glycated proteins and higher activity of matrix metalloproteinase enzymes, which contribute to vascular thickening and stiffness. Atherosclerosis occurs when pro-inflammatory cytokines increase in endothelial cells and vascular smooth muscle cells. An average age of over 60 years indicates that the incidence of atherosclerosis and chronic coronary syndrome increases with age.[15]

**Characteristics of Risk Factors among the Research Subjects**

In this study, 20.6% of the subjects were smokers, while 79.4% were non-smokers. Statistical analysis showed no significant correlation between smoking history and the studied outcomes. This finding is consistent with previous studies indicating variations in the clinical characteristics of patients with cardiovascular disease. Similarly, reports Gariepy et al. reported no significant difference in the intima-media thickness of the carotid and femoral arteries between smokers and non-smokers ($p \geq 0.05$).[16] Kiriyama et al. reported no significant difference in mean intima-media thickness (or plaques ≥ 1.1 mm) between smokers and non-smokers. However, smokers were found to have a higher prevalence of high-risk (vulnerable) plaques. Prolonged smoking duration with a high number of cigarettes per day (≥ 10 cigarettes/day for 5–10 years), and genetic or metabolic variations may be required to contribute to the development of atherosclerosis.[17]

In this study, the incidence of hypertension was found in 32.4% of subjects, 67.6% had no history of hypertension. Statistical analysis showed no significant correlation. This finding is consistent with the study by Mathiesen et al., which found that hypertension did not independently predict plaque progression after adjusting for other factors such as LDL levels and age.[18] Morelli et al. found cholesterol levels (total cholesterol, LDL-C, and the LDL/HDL ratio) were significantly associated with carotid intima-media thickness, whereas systolic blood pressure did not show a significant independent correlation with intima-media thickness. These findings validate that, in certain populations, cholesterol levels may play a more dominant role than blood pressure in structural changes to blood vessels.[19]

In this study, only 14.7% of the subjects were diagnosed with type 2 diabetes mellitus, 85.3% did not have type 2 diabetes mellitus, and statistically, no significant correlation was found. Boden et al. reported no significant difference in the progression of stenosis between patients with well-controlled type 2 diabetes mellitus and non-diabetic patients.[20] These findings contrast with other studies indicating that blood glucose levels can affect vascular structure. Chronic hyperglycemia can cause endothelial damage and accelerate atherosclerosis, potentially increasing the degree of coronary artery stenosis. In this study, the duration of diabetes diagnosis was unknown, and patients' blood glucose levels were generally well controlled. This suggests that optimal glucose control can help reduce the progression of atherosclerosis.

In this study, 76.5% of the subjects were found to have dyslipidemia. Abnormal lipid levels can accelerate plaque formation, promote the atherosclerotic process, and contribute to coronary artery stenosis. Statistical analysis showed a significant correlation between dyslipidemia and the SYNTAX score. This finding is consistent with the study by Akhtaruzzaman et al., which examined the relationship between dyslipidemia and chronic coronary syndrome based on angiographic findings, and found that patients with dyslipidemia were more likely to experience stenosis, particularly in the inferior segments.[22]

The mean BMI of the study subjects was 25.64 ± 3.85 kg/m². A total of 14.7% of subjects were classified as overweight and 52.9% as obese. This finding is consistent with the findings of Lind et al. Previous studies have demonstrated that overweight and obesity are significant factors contributing to dyslipidemia.[23] This association is primarily linked to insulin resistance, which is a key mechanism underlying dyslipidemia in overweight and obese individuals. Obesity impacts the modification of lipoproteins and factors related to systemic and vascular inflammation, as well as endothelial dysfunction. Abnormal concentrations of lipids and apolipoproteins can lead to changes in the production, conversion, or catabolism of lipoprotein particles. These changes may contribute to increased lipolysis in obesity, leading to the release of free fatty acids into the

circulation and a pro-atherogenic phenotype. Obesity is often associated with other cardiovascular risk factors such as hypertension, dyslipidemia, and insulin resistance may contribute to the progression of atherosclerosis.[24,25]

**Laboratory Profile of the Study Participants**

The results showed a mean total cholesterol level of 175.88 ± 36.01 mg/dL, which falls within the normal range. Statistically, no significant correlation was found between total cholesterol and the SYNTAX score (p = 0.191). This finding is consistent with a study by Ravnskov et al., which also reported no significant correlation between total cholesterol and cardiovascular disease.[26] However, recent research by Ference et al. demonstrated that even at cholesterol levels considered normal, the lifetime exposure to elevated LDL can increase the risk of cardiovascular disease.[27]

The mean LDL level in this study was 123.59 ± 35.54 mg/dL, which is within the normal range but slightly above the recommended threshold for primary prevention. Furthermore, the p-value of 0.648 indicates no significant correlation. This result is consistent with a study by Inoue et al., which found no statistically significant association between LDL and coronary atherosclerosis based on CT angiography.[28] In addition, recent research by Packard et al. emphasized that it is not only LDL concentration that matters, but also the size and number of lipoprotein particles, as well as the presence of endothelial dysfunction.[29]

Apolipoprotein B is believed to provide a better assessment of the severity of coronary artery stenosis. All lipoproteins containing apoB100 with a diameter of less than 70–80 nm including remnant VLDL, IDL, and triglyceride-rich LDL can freely circulate across the endothelial barrier and may become trapped within the arterial wall. Once retained, these particles undergo oxidation, which induces local inflammatory and immune responses, such as increased inflammatory cytokines and the expression of leukocyte chemoattractants on endothelial cells. Circulating monocytes then infiltrate the intimal layer of the vessel wall, where macrophages phagocytose the retained lipid particles, leading to the formation of foam cells that eventually develop into atherosclerotic plaques. Based on the findings of this study, LDL alone is not the sole major atherogenic lipoprotein, additional risk factors are required to drive plaque formation.[30]

In this study, the median HDL level was 42 mg/dL (range: 29–80 mg/dL). Statistical analysis revealed no significant association (p = 0.167). This finding is consistent with the study by Inoue et al., which also found no significant relationship between HDL levels and atherosclerosis.[28] HDL is known to have anti-inflammatory and antithrombotic properties. HDL prevents oxidation and facilitates reverse cholesterol transport by carrying excess cholesterol from macrophages in the arterial wall (atherosclerotic plaques) back to the liver for excretion. Epidemiologically, higher HDL levels are generally associated with a lower risk of cardiovascular disease. However, HDL is not a single homogeneous molecule but a heterogeneous class of particles varying in size, protein composition, and lipid content. Not all HDL particles are equally effective at promoting cholesterol efflux. Under chronic inflammatory conditions such as diabetes mellitus and metabolic syndrome HDL can lose its protective function and even acquire pro-inflammatory properties. Genetic evidence from the study by Voight et al. showed that individuals with genetic mutations resulting in higher HDL-C levels did not have a reduced risk of cardiovascular disease.[31] Furthermore, pharmacological interventions aiming to raise HDL levels, such as CETP inhibitors (torcetrapib, dalcetrapib, evacetrapib), have failed to demonstrate clear benefits in reducing atherosclerotic events.[32]

Triglycerides showed a statistically significant correlation (p = 0.025) with a median value of 89 mg/dL (range: 40–289 mg/dL). This finding is consistent with the study by Zheng C et al which demonstrated a significant association between triglyceride levels and arterial stenosis.[24] A study by Darmawan and Irfannuddin in Palembang also found a relationship between obesity and levels of total cholesterol, VLDL, and triglycerides, with age and sex acting as modifying factors. Notably, abdominal obesity had the strongest influence on VLDL and triglyceride levels compared to other lipid parameters, highlighting their role as important cardiovascular risk factors.[33] These results align with growing evidence supporting the role of triglycerides in atherosclerosis. Furthermore, research by Ala-Korpela suggested that elevated triglyceride levels can increase circulating free fatty acids and promote greater flux of free fatty acids from adipose tissue to non-adipose tissues. Additionally, the retention of triglyceride-rich, ApoB-containing remnant particles within the coronary arterial wall is believed to contribute directly to the atherosclerotic process.[34]

The lack of a significant correlation for total cholesterol, LDL, and HDL (p > 0.05) contrasts with findings from several recent large-scale studies. For instance, the FOURIER trial demonstrated substantial cardiovascular benefits from intensive LDL lowering.[35] This discrepancy may be explained by factors such as differences in population characteristics, study design, or unmeasured confounding variables in the present study. Furthermore, recent genomic research additional complexity in lipid metabolism, identifying novel genetic variants that influence lipid profiles and cardiovascular risk. These insights highlight the importance of considering genetic predispositions and other modifying factors when interpreting lipid profiles and their relationship with cardiovascular outcomes.[36]

The median fasting blood glucose level among subjects was 88.5 mg/dL, with a range from 54 to 303 mg/dL. The statistical analysis revealed a correlation coefficient of r = 0.362 with a p-value of 0.035, indicating a significant correlation between fasting blood glucose levels and the SYNTAX score. Previous studies have shown that elevated blood glucose can adversely affect vascular structure; chronic hyperglycemia may lead to endothelial dysfunction and accelerate atherosclerosis, thereby potentially increasing the degree of coronary artery stenosis.[21] The findings of this study support this direct correlation, underscoring that blood glucose levels are an important indicator in assessing cardiovascular risk.

**Correlation of Apolipoprotein B with SYNTAX Score**

Studies have shown a positive correlation between serum apolipoprotein B levels and the SYNTAX score. A correlation coefficient of $r = 0.376$ with a $p$-value of 0.029 ($p < 0.05$) indicates a significant association between serum apolipoprotein B levels and the severity of coronary artery stenosis as measured by the SYNTAX score. This result is consistent with studies by Lin Taiwu *et al.*, Hua *et al.*, This finding suggests that higher levels of apolipoprotein B are associated with more severe coronary artery stenosis. Apolipoprotein B is believed to be a better marker for assessing the severity of coronary artery stenosis.[30,37] All lipoproteins containing apoB100 with a diameter smaller than 70–80 nm including remnant VLDL and triglyceride-rich LDL can freely circulate across the endothelial barrier and become trapped within the arterial wall. These retained particles undergo oxidation, which induces a local inflammatory and immune response, such as increased inflammatory cytokines and the expression of leukocyte chemoattractants on endothelial cells. Circulating monocytes then migrate into the intimal layer of the blood vessel, where macrophages phagocytose the retained lipid particles, forming foam cells. The correlation between apolipoprotein B levels and the degree of coronary artery stenosis provides insight into the

severity of stenosis in patients with chronic coronary syndrome. High apolipoprotein B levels are associated with an increased number of atherogenic lipoproteins that can penetrate the vascular endothelium.**7** An elevated SYNTAX score reflects ongoing atherosclerotic processes in the coronary arteries and corresponds to the degree of stenosis present. In this study, a correlation coefficient of $r = 0.376$ with a $p$-value of 0.029 ($p < 0.05$) demonstrates that serum apolipoprotein B levels are useful for identifying individuals at high risk for atherosclerosis. Early intervention in patients with elevated apolipoprotein B levels may help prevent the progression of atherosclerosis and related cardiovascular complications.

## CONCLUSION

The study results indicated a significant positive correlation between serum apolipoprotein B concentrations and the severity of coronary artery stenosis as assessed by the SYNTAX score.

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