ORIGINAL ARTICLE

Preventing Premature Coronary Artery Disease: The Synergistic Role of Biomarker Screening and Physical Activity

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Abstract

Objective: With the increasing prevalence of premature coronary artery disease (CAD), early detection and risk stratification are crucial. While physical inactivity is linked to CAD risk, its impact in the early stages remains underexplored. This study aims to identify biomarkers for early CAD diagnosis and their association with physical activity (PA), ultimately reducing morbidity and mortality rates.

Methods: This case–control study enrolled 300 subjects aged 18–45 years. They were subdivided into three categories. Additionally, the 200 subjects in groups I and II were classified into active, moderate, and sedentary categories based on World Health Organization (WHO) criteria. Serum levels of high-sensitivity C-reactive protein (hs-CRP), lipoprotein (a) [Lp(a)], apolipoprotein A1 (Apo-A1), apolipoprotein B100 (Apo-B100), and oxidized low-density lipoprotein (oxidized LDL) were analyzed, whereas non–high-density lipoprotein cholesterol (non-HDL-C) was calculated. The comparison of these biochemical parameters was done in terms of mean ± standard error of the mean (SEM) and area under the receiver operating characteristic curve (AUROC), and their significance with PA was determined using one-way analysis of variance (ANOVA) and Bonferroni test.

Results: Significant differences in hs-CRP, Apo-B100, Lp(a), non-HDL-C, and oxidized LDL were observed across groups. AUROC analysis confirmed their strong association with CAD risk. Additionally, the findings highlight that an active lifestyle is linked to a more favorable biochemical profile, which may help mitigate the risk of premature CAD.

Conclusion: The study suggests including hs-CRP, Apo-B, Lp(a), non-HDL-C, and oxidized LDL in routine screening for early CAD detection. Despite their proven effectiveness, these biomarkers are not widely used. Therefore, integrating early biomarker screening with lifestyle modifications can enhance risk assessment and improve treatment outcomes.

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Introduction

Coronary artery disease (CAD) remains a leading cause in global rates of illness and death. The early onset of CAD, referred to as premature CAD, presents significant challenges due to its aggressive nature and the need for early intervention.[1] Emerging evidence suggests that the incidence of CAD in Indians is 50–400% more than in other ethnic groups.[2–4] This vulnerability to premature CAD is primarily driven by rapid socioeconomic and cultural transitions. Over the past three decades, economic liberalization has led to significant lifestyle and dietary changes with decreased physical activity (PA) and increased mental stress.[5] Regular PA has been shown to substantially lower the risk of cardiovascular mortality rates, while decreased PA is linked with a higher risk of morbidity. Additionally, a recent science advisory from the American Heart Association highlights the detrimental relationship between sedentary behavior and cardiovascular mortality.[6]

The standard approach of assessing CAD risk primarily relies on the standard lipid profile panel, which includes total cholesterol (TC), low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides (TG). While this panel provides valuable insights, recent studies indicate that it may not be sufficient for accurate prediction, especially in young Indians at risk of premature CAD. Given the growing burden of premature CAD in this population, it is crucial to move beyond conventional lipid parameters in cardiovascular risk assessment. A more comprehensive cardiac screening panel, integrating novel biomarkers, could facilitate earlier detection of CAD and offer a deeper understanding of other contributing risk factors, such as sedentary behavior. This shift in approach would enhance prevention and intervention efforts, addressing the urgent need for more effective and personalized strategies to combat CAD. Therefore, this study seeks to identify biomarkers for the early diagnosis of CAD and explore their association with PA. The goal is to enhance screening and prevention strategies, mitigate the morbidity and mortality associated with CAD, and foster greater awareness of the role of lifestyle choices in cardiovascular health.

Methods

This case–control study took place at a tertiary healthcare facility in Northern India, enrolling 300 subjects aged 18–45 years. Premature CAD was described as the occurrence of CAD before the age of 55 in women and 45 in men.[7] Patients with diabetes mellitus, hypertension, noncardiac inflammatory conditions (such as rheumatoid arthritis or osteoarthritis), and those on statin therapy were excluded from the study.

The subjects were subdivided into three categories:

- Group I: Angiographically proven cases of CAD (n = 100); subjects having ≥50% diameter stenosis in at least one epicardial coronary artery were included in the study.
- Group II: Subjects who had one or two risk factors associated with CAD were recruited in group II category (n = 100). The risk factors included positive family history and smoking.
- Group III: Noncardiac controls (n = 100); subjects without any history or risk factors associated with CAD were recruited in group III category. The selection of the noncardiac controls was based on randomization.

Additionally, the 200 subjects in groups I and II were further categorized based on their PA levels—active, moderate, and sedentary—according to the criteria established by the World Health Organization (WHO).[8]

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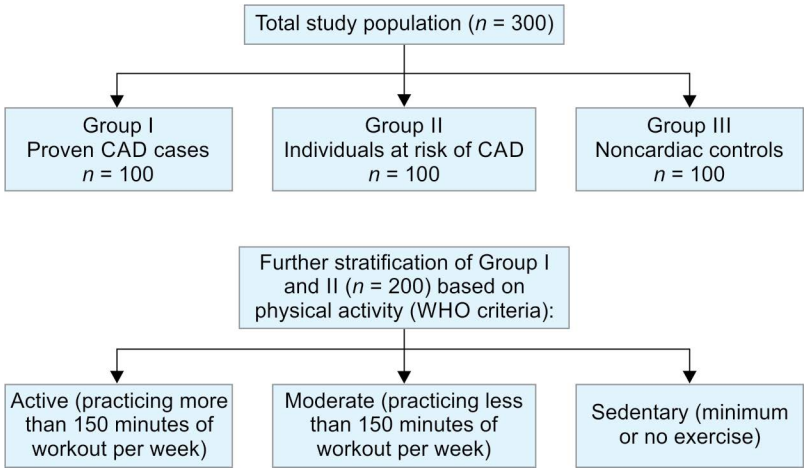

The Institutional Ethics Committee approved the study, and all participants provided informed consent. Blood samples (5 mL) were collected in plain vials, and serum was separated following standard protocols. Sample collection and preservation adhered to the STROBE-ME guidelines. The biochemical parameters analyzed in the study included high-sensitivity C-reactive protein (hs-CRP), Apo-B, lipoprotein (a) [Lp(a)], non–high-density lipoprotein cholesterol (non-HDL-C), Apo-A1, and oxidized LDL, in addition to the conventional lipid profile parameters.

After collecting blood samples, serum TG, HDL-C, and TC were assessed using the Siemens Dimensions RxL analyzer. LDL and very-low-density lipoprotein (VLDL) levels were determined using the Friedewald equation, whereas non-HDL-C was derived from TC to HDL-C. For the analysis of inflammatory and lipoprotein markers, hs-CRP was estimated using a latex-enhanced turbidimetric immunoassay, while Apo-B, Apo-A1, and Lp(a) were analyzed by the immunoturbidimetry method, and oxidized LDL was estimated using a sandwich enzyme-linked immunosorbent assay (ELISA).

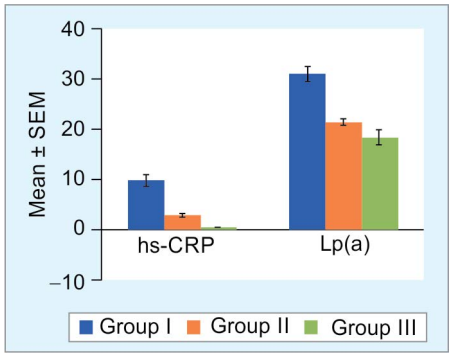

Fig. 1: Mean ± SEM level of hs-CRP and Lp(a)

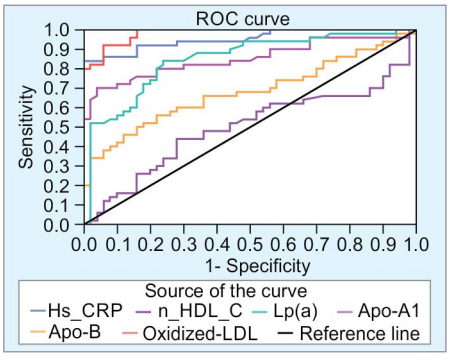

Fig. 3: ROC curve for all the parameters

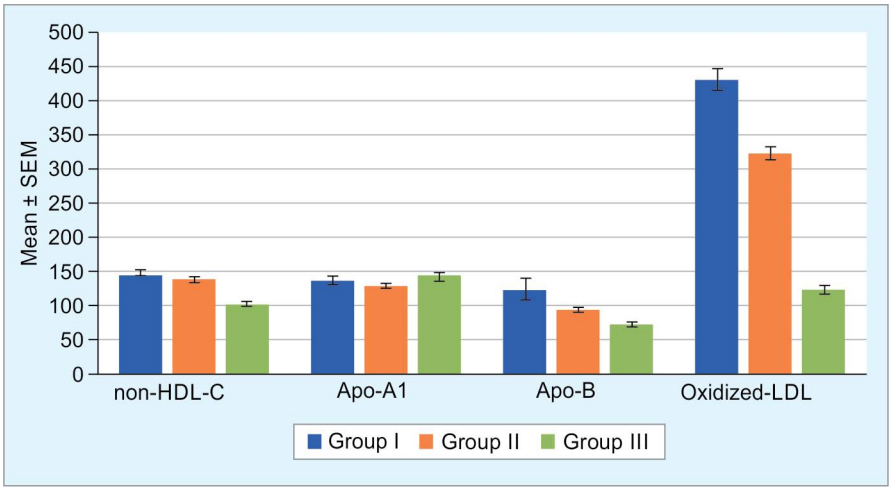

Fig. 2: Mean ± SEM level of non-HDL-C, Apo-A1, Apo-B, and oxidized LDL

Statistical Analysis

Biochemical parameters were compared in terms of mean ± standard error of the mean (SEM) level and area under the receiver operating characteristic curve (AUROC) analysis. The association between biochemical parameters and PA was evaluated using one-way analysis of variance (ANOVA) followed by a *post hoc* analysis (Bonferroni) to identify the mean differences between the groups (active, moderate, and sedentary) for each biochemical parameter. All analyses were done by SPSS version 24. A *p*-value below 0.05 was interpreted as statistically significant.

Results

Serum hs-CRP, Lp(a), apolipoprotein A1 (Apo-A1), Apo-B, and oxidized LDL were measured, while non-HDL-C was calculated for all the subjects. The findings revealed that hs-CRP was markedly elevated in group I but decreased substantially in group III. Similarly, Lp(a), non-HDL-C, Apo-B, and oxidized LDL exhibited the highest values in group I and progressively decreased across groups II and III, whereas the results were nonsignificant for Apo-A1. Figures 1 and 2 show the mean ± SEM levels of hs-CRP and Lp(a), and non-HDL-C, Apo-A1, Apo-B, and oxidized LDL, respectively.

The predictive values of hs-CRP, non-HDL-C, Lp(a), Apo-A1, Apo-B, and oxidized LDL were compared using ROC curve analysis. The AUROC for oxidized LDL was found to be 0.982 (95% confidence interval: 0.963, 1.000), followed by hs-CRP, 0.952 (95% confidence interval: 0.912, 0.992), followed by non-HDL-C, 0.852 (95% confidence interval: 0.773, 0.931), followed by Lp(a), 0.847 (95% confidence interval: 0.769, 0.924), followed by Apo-B, 0.680 (95% confidence interval: 0.574, 0.787), whereas Apo-A1 had the least AUROC of 0.498 (95% confidence interval: 0.381, 0.614) (Fig. 3).

The 200 subjects from groups I and II were further categorized based on their PA levels, with results showing that more than half of the participants exhibited sedentary behavior, 38% were moderately active, and only 9% were physically active (Fig. 4).

The one-way ANOVA analysis of biochemical parameters in relation to PA demonstrated a significant association with hs-CRP, Lp(a), non-HDL-C, Apo-B, and oxidized LDL levels, while results were nonsignificant for Apo-A1 (Table 1).

Additionally, the Bonferroni test was conducted, revealing a significant mean

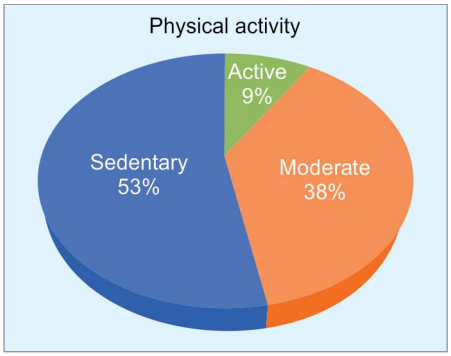

Fig. 4: Distribution of 200 subjects based on PA

difference among the active, moderate, and sedentary groups only with non-HDL-C and oxidized LDL-C. For hs-CRP, Lp(a), and Apo-B, the mean difference was significant between the active and both the moderate and sedentary groups, whereas no notable difference was detected between the moderate and sedentary groups. Regarding Apo-A1, no statistically significant differences were detected across the groups (Table 2).

Figure 5 highlights the relationship between physical activity levels and biochemical parameters.

Discussion

The rising prevalence of premature CAD among young Indians has become a growing concern as affluence and urbanization have led to significant lifestyle and dietary changes, along with a decline in PA, which has exacerbated this public health challenge, underscoring the urgent need for preventive measures and awareness.[9,10] Numerous biomarkers have been identified to assess cardiovascular risk, yet conventional risk prediction algorithms still fall short of providing reliable and accurate

Table 1: ANOVA analysis of biochemical parameters in relation to PA

		Sum of squares	*Df*	*Mean square*	*F*	*Significance*
hs-CRP	Between groups	1661.18	2	830.591	15.05	<0.001***
	Within groups	16390.541	297	55.187		
	Total	18051.722	299			
Non-HDL-C	Between groups	88044.28	2	44022.14	42.07	<0.001***
	Within groups	310750.29	297	1046.29		
	Total	398794.58	299			
Lp(a)	Between groups	2035.49	2	1017.74	5.5	<0.01**
	Within groups	54955.07	297	185.03		
	Total	56990.56	299			
Apo-A1	Between groups	2036.05	2	1018.02	0.491	0.612
	Within groups	615662.57	297	2072.93		
	Total	617698.62	299			
Apo-B	Between groups	76187.59	2	38093.79	4.161	<0.05*
	Within groups	2718791.42	297	9154.18		
	Total	2794979.01	299			
Oxidized LDL	Between groups	2529770.63	2	1264885.31	63.346	<0.001***
	Within groups	5930501.17	297	19968.017		
	Total	8460271.80	299			

Levels of statistical significance are denoted as follows: *$p < 0.05$ (significant), **$p < 0.01$ (highly significant), ***$p < 0.001$ (very highly significant)

Table 2: Bonferroni test of biochemical test in relation to PA

Dependent variable	*(I) Is*	*(J) Is*	*Mean difference (I–J)*	*Std. error*	*p-value*	*95% confidence interval*	
						Lower bound	*Upper bound*
hs-CRP	1	2	−4.494	1.096	<0.001	−7.132	−1.856
		3	−5.892	1.100	<0.001	−8.539	−3.244
	2	3	−1.398	0.997	0.486 (NS)	−3.799	1.003
Non-HDL-C	1	2	−27.846	4.770	<0.001	−39.331	−16.360
		3	−43.838	4.788	<0.001	−55.366	−32.310
	2	3	−15.992	4.342	<0.001	−26.447	−5.538
Lp(a)	1	2	−5.752	2.006	<0.05	−10.581	−0.922
		3	−6.108	2.014	<0.05	−10.956	−1.260
	2	3	−0.357	1.826	1	−4.753	4.040
Apo-A1	1	2	−0.564	6.715	1	−16.730	15.602
		3	−5.719	6.740	1	−21.945	10.507
	2	3	−5.155	6.112	1	−19.870	9.560
Apo-B	1	2	−33.984	14.110	0.05	−67.956	−0.013
		3	−38.236	14.163	<0.05	−72.335	−4.138
	2	3	−4.252	12.843	1	−35.174	26.670
Oxidized LDL	1	2	−115.978	20.840	<0.001	−166.152	−65.805
		3	−234.002	20.917	<0.001	−284.362	−183.641
	2	3	−118.023	18.969	<0.001	−163.693	−72.354

Note: 1 = active, 2 = moderate, and 3 = sedentary; The mean difference was considered significant at the 0.05 level

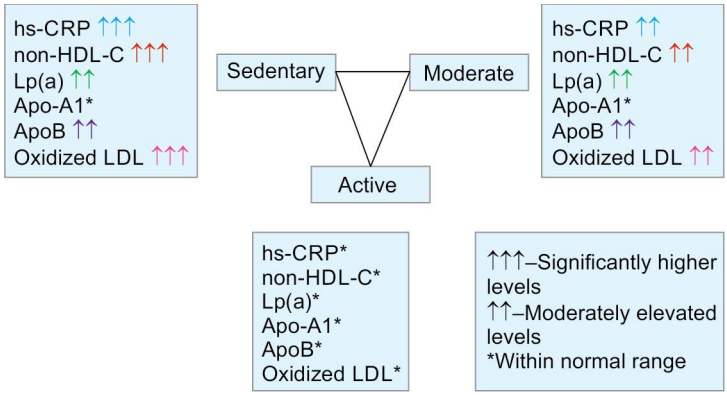

Fig. 5: The relationship between PA levels and biochemical parameters

markers for CAD.[11] Therefore, this study was designed to evaluate biomarkers for early risk prediction of premature CAD and their potential correlation with PA.

The mean ± SEM level of hs-CRP, a key inflammatory marker, was significantly elevated in group I, moderately elevated in group II, and within the normal range in group III (Fig. 1). The identified cutoff value for hs-CRP was 0.795 mg/L. The results are consistent with findings from studies by Gupta et al.[12] and Lee et al.,[13] where a significant difference in hs-CRP levels was observed between cardiac and noncardiac individuals. Similarly, Mokhtar et al.,[14] in their study of 45 subjects, reported findings comparable to those of the present study. Extensive research, including observational studies and clinical trials, has also highlighted the role of elevated hs-CRP levels in the development of atherosclerotic vascular disease.[15–18]

Similarly, the mean ± SEM level of non-HDL-C was significantly higher in group I, moderately elevated in group II, and within the normal range in group III (Fig. 2). These findings align with those reported by Li et al.,[19] Kathariya et al.,[20] and Cui et al.[21] The cutoff value for non-HDL-C in this study was found to be 113.65 mg/dL. A large-scale study spanning 44 cohorts across multiple countries, involving 5,24,444 individuals, utilized multivariate analysis to establish a significant relation between levels of non-HDL-C and long-term risk of CVDs.[22] Furthermore, the Lipid Association of India recognizes non-HDL-C as a coprimary target for assessing CAD risk.[23]

Likewise, the mean ± SEM level of oxidized LDL was found to be elevated in group I as compared to groups II and III, and its cutoff value was found to be 224 ng/mL in this study (Fig. 2). Oxidized LDL plays a crucial role in the atherosclerotic process, with its proinflammatory and proatherogenic properties contributing substantially to the development of cardiovascular disease.[24] Atherogenicity of oxidized LDL is due to alteration in its biological properties due to oxidative modification of LDL, resulting in increased chemotaxis, more retention in subendothelial cells, macrophages, cytokine production from smooth muscle cells, and alteration of growth factors and endothelial cells.[25] Numerous studies have identified oxidized LDL as a valuable lipid marker and risk factor for atherosclerotic cardiovascular disease.[26–28] The study carried out by Augsburg revealed notably elevated levels of oxidized LDL in CAD patients compared to the controls. Zhao et al.[29] observed elevated oxidized LDL levels in individuals with CAD compared to controls, which aligns with our study's results. Similarly, our findings are consistent with studies by Koenig et al.[30] and Bansal et al.[31] who also compared the oxidized LDL in terms of mean ± SD levels and found increased oxidized LDL levels in CAD patients as compared to the noncardiac controls.

Regarding Lp(a) and Apo-B, the mean ± SEM level was significantly higher in group I, moderately elevated in group II, and within the normal range in group III, as depicted in Figures 1 and 2, respectively. These findings are consistent with the studies conducted by Leistner et al.,[32] Tsimikas,[33] Nathir et al.,[34] and Walldius and Jungner.[35]

Recent meta-analyses link sedentary PA to higher CAD mortality.[36] To assess its impact, this study evaluated PA using a standard questionnaire.[37] Results showed that 53% subjects had sedentary PA, while 38% had moderate PA, suggesting its role in premature CAD.[38] The *post hoc* analysis revealed that individuals with an active PA had significantly lower hs-CRP levels than those with moderate or sedentary activity, suggesting that increased PA is associated with reduced inflammation, which is in accordance with the study conducted by Koeder et al.[39] Similarly, active individuals exhibited significantly lower non-HDL-C levels than those in the moderate and sedentary groups, further highlighting the cardiovascular benefits of increased PA. Likewise, oxidized LDL was significantly lower in active individuals compared to both moderate and sedentary groups. Additionally, moderate activity was associated with significantly lower oxidized LDL levels than a sedentary activity, emphasizing the role of PA in reducing oxidative stress and cardiovascular damage.

In the context of Lp(a), individuals with an active PA exhibited significantly lower Lp(a) levels compared to those with moderate or sedentary activity. This observation indicates that increased PA is linked to lower Lp(a) levels. Interestingly, no significant difference was observed between individuals with moderate PA and those leading a sedentary PA, indicating that merely moderate activity does not confer the same benefit in terms of Lp(a) reduction. Therefore, maintaining active PA is essential for achieving lower Lp(a) levels, rather than simply engaging in moderate PA. Likewise, Apo-B levels were notably reduced in physically active individuals relative to those in moderate and sedentary groups, while no notable difference was observed between the moderate and sedentary groups, indicating a protective effect of PA against elevated Apo-B levels. Therefore, addressing lifestyle factors is crucial for CAD prevention,[40] emphasizing the need to replace sedentary PA with an active PA.

Limitation of the Study

The study was limited to a relatively small sample size from a single center. Therefore, extensive studies with larger and more diverse populations are necessary to enhance understanding and validate these findings.

Conclusion

A comprehensive cardiac screening panel, including hs-CRP, Apo-B, Lp(a), non-HDL-C, and oxidized LDL, could enable earlier detection of CAD and significantly benefit public health. Moving beyond the traditional lipid profile, which still leaves residual risk despite achieving desired LDL levels, is crucial to address the growing global burden of premature CAD. Timely detection and accurate diagnosis through the inclusion of additional biomarkers would allow for disease prevention at an early stage, even before clinical symptoms appear. Incorporating this expanded panel into leading institutes and laboratories could notably reduce the CAD burden.

The study also highlights the positive relationship between an active lifestyle and improved biochemical markers, offering enhanced cardiovascular protection and lowering the risk of premature CAD. These

findings emphasize the need to combine early biomarker screening with lifestyle modifications to improve risk assessment and clinical outcomes.

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References

1. Al-Khlaiwi T, Habib SS, Bayoumy N, et al. Identifying risk factors and mortality rate of premature coronary artery disease in young Saudi population. Sci Rep 2024;14(1):12727.
2. Sharma M, Ganguly NK. Premature coronary artery disease in Indians and its associated risk factors. Vasc Health Risk Manag 2005;1(3):217–225.
3. Nag T, Ghosh A. Cardiovascular disease risk factors in Asian Indian population: a systematic review. J Cardiovasc Dis Res 2013;4(4):222–228.
4. Ardeshna DR, Bob-Manuel T, Nanda A, et al. Asian-Indians: a review of coronary artery disease in this understudied cohort in the United States. Ann Transl Med 2018;6(1):12.
5. Holmes SD, Krantz DS, Rogers H, et al. Mental stress and coronary artery disease: a multidisciplinary guide. Prog Cardiovasc Dis 2006;49(2):106–122.
6. Eaton CB. Sedentary behaviour and risk of cardiovascular disease. J Am Coll Cardiol 2025;85(5):487–488.
7. Khoja A, Andraweera PH, Lassi ZS, et al. Risk factors for premature coronary artery disease (PCAD) in adults: a systematic review protocol. F1000Res 2021;10:1228.
8. Bull FC, Al-Ansari SS, Biddle S, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. Br J Sports Med 2020;54(24):1451–1462.
9. Iyengar SS, Gupta R, Ravi S, et al. Premature coronary artery disease in India: coronary artery disease in the young (CADY) registry. Indian Heart J 2017;69(2):211–216.
10. Aggarwal A, Srivastava S, Velmurugan M. Newer perspectives of coronary artery disease in young. World J Cardiol 2016;8(12):728–734.
11. Dhingra R, Vasan RS. Biomarkers in cardiovascular disease: statistical assessment and section on key novel heart failure biomarkers. Trends Cardiovasc Med 2016;27(2):123–133.
12. Gupta S, Gupta VK, Gupta R, et al. Relationship of high-sensitive C-reactive protein with cardiovascular risk factors, clinical presentation and angiographic profile in patients with acute coronary syndrome: an Indian perspective. Indian Heart J 2013;65(3):359–365.
13. Lee H, Park HE, Yoon JW, et al. Clinical significance of body fat distribution in coronary artery calcification progression in Korean population. Diabetes Metab J 2021;45(2):219–230.
14. Mokhtar ER, Saleh BI, Aboualia AM, et al. Relationship between monocyte subsets, IL-6 and hs-CRP with the severity of coronary artery disease in stable angina pectoris patients. Am J Biochem 2017;7:114–126.
15. Sharma SB, Garg S, Veerwal A, et al. hs-CRP and oxidative stress in young CAD patients: a pilot study. Indian J Clin Biochem 2008;23(4):334–336.
16. Kamath DY, Xavier D, Sigamani A, Pais P. High sensitivity C-reactive protein (hsCRP) & cardiovascular disease: an Indian perspective. Indian J Med Res 2015;142(3):261–268.
17. Benzaquen LR, Yu H, Rifai N. High sensitivity C-reactive protein: an emerging role in cardiovascular risk assessment. Crit Rev Clin Lab Sci 2002;39(4–5):459–497.
18. Song Y, Wang M, Li Y, et al. The evaluation value of atherogenic index of plasma and high-sensitivity C-reactive protein for the degree of coronary artery lesion in premature coronary artery disease. BMC Cardiovasc Disord 2024;24(1):410.
19. Li C, Ford ES, Tsai J, et al. Serum non-high-density lipoprotein cholesterol concentration and risk of death from cardiovascular diseases among U.S. adults with diagnosed diabetes: the Third National Health and Nutrition Examination Survey linked mortality study. Cardiovasc Diabetol 2011;10:46.
20. Kathariya G, Aggarwal J, Garg P, et al. Is evaluation of non-HDL-C better than calculated LDL-C in CAD patients? MMIMSR experiences. Indian Heart J 2020;72:189–191.
21. Cui Y, Blumenthal RS, Flaws JA, et al. Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. Arch Intern Med 2001;161(13):1413–1419.
22. Brunner FJ, Waldeyer C, Ojeda F, et al. Application of non-HDL cholesterol for population-based cardiovascular risk stratification: results from the multinational cardiovascular risk consortium. Lancet 2019;394:2173–2183.
23. Aggarwal J, Kathariya G, Verma PK. LDL-C, NON-HDL-C and APO-B for cardiovascular risk assessment: looking for the ideal marker. Indian Heart J 2021;73(5):544–548.
24. Maiolino G, Rossitto G, Caielli P, et al. The role of oxidized low-density lipoproteins in atherosclerosis: the myths and the facts. Mediators Inflamm 2013;2013:714653.
25. Witztum JL, Steinberg D. Role of oxidized low-density lipoprotein in atherogenesis. J Clin Invest 1991;88(6):1785–1792.
26. Kelishadi R, Nadery GA, Asgary S. Oxidized LDL metabolites with high family risk for premature cardiovascular disease. Indian J Pediatr 2002;69:755–759.
27. Hong CG, Florida E, Li H, et al. Oxidized low-density lipoprotein associates with cardiovascular disease by a vicious cycle of atherosclerosis and inflammation: a systematic review and meta-analysis. Front Cardiovasc Med 2023;9:1023651.
28. Mosca L, Rubenfire M, Tarshis T, et al. Clinical predictors of oxidized low-density lipoprotein in patients with coronary artery disease. Am J Cardiol 1997;80(7):825–830.
29. Zhao X, Zhang HW, Xu RX, et al. Oxidized-LDL is a useful marker for predicting the very early coronary artery disease and cardiovascular outcomes. Per Med 2018;15(6):521–529.
30. Koenig W, Karakas M, Zierer A, et al. Oxidized LDL and the risk of coronary heart disease: results from the MONICA/KORA Augsburg study. Clin Chem 2011;57(8):1196–1200.
31. Bansal SK, Agarwal S, Daga MK. Conventional and advanced lipid parameters in premature coronary artery disease patients in India. J Clin Diagn Res 2015;9(11):BC07–BC11.
32. Leistner DM, Laguna-Fernandez A, Haghikia A, et al. Impact of elevated lipoprotein(a) on coronary artery disease phenotype and severity. Eur J Prev Cardiol 2024;31(7):856–865.
33. Tsimikas S. A test in context: lipoprotein(a): diagnosis, prognosis, controversies, and emerging therapies. J Am Coll Cardiol 2017;69(6):692–711.
34. Nathir I, Aziz FA, Hashim R. Association between apolipoprotein B and coronary artery disease among hypertensive patients: a systematic review of the prospective and retrospective studies. Cureus 2023;15(12):e49854.
35. Walldius G, Jungner I. The apo B/apo-AI ratio: a strong, new risk factor for CVD and target for lipid-lowering therapy—a review of evidence. J Intern Med 2006;259(5):493–519.
36. Young DR, Hivert MF, Alhassan S, et al. Sedentary behaviour and cardiovascular morbidity and mortality: a science advisory from the American Heart Association. Circulation 2016;134(13):e262–e279.
37. Nigg CR, Fuchs R, Gerber M, et al. Assessing physical activity through questionnaires—a consensus of best practices and future directions. Psychol Sport Exerc 2020;50:101715.
38. Teo KK, Rafiq T. Cardiovascular risk factors and prevention: a perspective from developing countries. Can J Cardiol 2021;37(5):733–743.
39. Koeder C, Anand C, Husain S, et al. Exploratory analysis of the effect of a controlled lifestyle intervention on inflammatory markers—the Healthy Lifestyle Community Programme (cohort 2). BMC Nutr 2023;9(1):25.
40. Kanemitsu K, Hassan BD, Mdivnishvili M, et al. The impact of lifestyle intervention programs on long-term cardiac event-free survival in patients with established coronary artery disease. Cureus 2024;16(12):e76585.