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**ORIGINAL ARTICLE****A study on high ApoB and LDL levels in people attending a tertiary hospital in Coimbatore, India***Hariharan V<sup>1</sup>, Namrithaa S<sup>1</sup>**<sup>1</sup>Department of Biochemistry, Karpagam Faculty of Medical Sciences and Research, Othakkalmandapam, Coimbatore-641032 (Tamilnadu) India*

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**Abstract**

*Background:* It is estimated that around 17.9 million people die each year of cardiovascular disease. High LDL is an important risk factor for coronary artery disease. Apolipoprotein B (ApoB) is the primary apolipoprotein of chylomicrons, VLDL, IDL, and LDL. Increased Apo B levels and LDL levels are known to cause atherosclerotic coronary artery disease. *Aim and Objectives:* To measure LDL and Apo B levels of adult people and to calculate the prevalence of high ApoB and LDL in Coimbatore region. *Material and Methods:* This was a study done for a period of 3 months in 500 people attending a tertiary hospital in Coimbatore, Tamilnadu. Patients on statins, critically ill patients, pregnant women, and lactating women were excluded. ApoB and LDL levels were measured and the percentage of increased levels of the same was calculated. *Results:* Out of 500 people studied, 52.87% of the population had high LDL. About 45.76% of the males and 56.49% of the women had high LDL. Out of the study subjects, 4.33% of the people had high ApoB level. Pearson's correlation revealed 'r' value as 1, thereby indicating a positive correlation between ApoB and LDL. *Conclusion:* This study showed that around 50% of study subjects had high LDL levels and 4% had high ApoB levels, and there was a positive correlation between the two.

**Keywords:** Apo-B, LDL, Cardiovascular disease, Atherosclerosis, Dyslipidemia

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**Introduction**

The incidence of myocardial infarction is 3 million worldwide. According to the epidemiological reports [1-2], one in three people will be affected by cardiovascular disease during their lifetime. It was estimated that 17.9 million people die each year of cardiovascular disease. Coronary Artery Disease (CAD) is a condition which involves atherosclerotic plaque formation in the lumen of blood vessels of heart. This may lead to impairment in blood flow when the plaque ruptures or when vasospasm occurs and thus oxygen delivery to the myocardium is impaired. The risk factors of CAD can be classified into two categories namely, modifiable, and non-modifiable risk factors. Various non-modifiable risk factors are increasing age,

male gender, Asian ethnicity, and a positive family history of CAD. Modifiable risk factors are diabetes mellitus, insulin resistance, high blood pressure, sedentary lifestyle, smoking or tobacco use, dyslipidaemia, homocysteinuria and psychological stress [1, 3-4].

Lipoproteins transport lipids to many tissues. Lipids from diet and liver are transported to peripheral tissues for energy production or storage for future use. There are special functions of lipids like steroids, hormones production and bile acid synthesis.

The components of lipoproteins are cholesterol (esterified or un-esterified), Triacylglycerol (TAG), and phospholipids. These lipids cannot be

transported in plasma since they are hydrophobic. So lipoproteins have protein components called apolipoproteins. Specific apolipoproteins control and regulate plasma lipid metabolism. The major apolipoproteins are ApoA-I, ApoA-II, ApoA-IV, ApoB, ApoC-I, ApoC- II, ApoC-III and Apo-E. Their functions are to re-distribute the lipids among various cells and tissues in the body and to function as co-factors for lipid metabolism enzymes and to maintain the structure and integrity of lipoprotein particles [5-6]. They also act as structural components and ligands for cellular receptor binding. The six major lipoproteins in blood are chylomicrons, Very Low-density Lipoprotein (VLDL), Intermediate-density Lipoprotein (IDL), Low Density Lipoprotein (LDL) (figure 1); Lipoprotein A (Lp (a)), and High Density Lipoprotein (HDL) [7].

Among these lipoproteins low levels of ApoA and high levels of ApoB play a role in developing atherosclerosis in coronary vessels. Apolipoprotein B is the major protein of chylomicrons, VLDL, IDL, and LDL. It exists as two forms such as ApoB-100 and ApoB-48. ApoA-I is the main protein of HDL [6]. The diameter of Apo-B containing lipoproteins are less than 70nm. They include smaller TAG rich lipoproteins and their remnants which can cross the endothelial barrier when there is damage in endothelial cells. These interact with proteoglycans and get trapped there. These ApoB lipoproteins when trapped in arterial wall, will induce a set of process which lead to lipid deposition and formation of atheroma. [8-9].

There are three hypotheses that can explain the events that initiate atherosclerosis. The first hypothesis is 'response-to-injury' hypothesis which says that when there is an injury to endothelial cells and smooth muscles, ApoB containing lipoproteins

enter through that lesion. Next is the 'oxidation' hypothesis, which says that the LDL in the lesion becomes oxidatively modified. The 'response-to-retention hypothesis' says that the accumulated oxidized LDL starts inflammatory process and becomes atherosclerosis [10-11].

Slowly the fat deposition starts increasing resulting in enlargement of the size of plaque, thereby increasing the risk of CAD. At one point where the changes in plaque composition become critical, it may lead to plaque disruption. This may lead to unstable angina or myocardial infarction or even death [12]. Sustained elevated levels of ApoB containing lipoproteins may lead to increased retaining of lipid particles in the plaque leading to its growth. It may be said that the atherosclerotic burden may be directly proportional to ApoB and LDL levels and duration of their elevation [13].

LDL levels are a measure of mass of cholesterol carried by LDL particles and contain many Apo-B proteins. It has been proved that long term lower LDL leads to lesser CAD incidence [14]. Similarly it is seen that a lifelong normal level of Apo-B leads to 90% reduced incidence of CAD [15]. Studies on prevalence of dyslipidaemia were not done at regular intervals in recent past and thus this study may aid in finding out the current prevalence of high LDL levels and Apolipoprotein B. Also this study might help find the current level of cardiovascular risk due to high LDL and ApoB levels in people attending a tertiary hospital in Coimbatore district.

#### **Material and Methods**

This was a cross-sectional study done for a period of 3 months on people attending Karpagam Hospital OPD, Coimbatore.

**Sample size calculation:** Based on a similar study [16], the expected prevalence of high LDL

and high ApoB were 22% and 18% respectively. Significance level was taken as 0.05 and desired margin of error for LDL and ApoB were taken as 4% and 3.5% respectively. Using the standard formula for sample size calculation

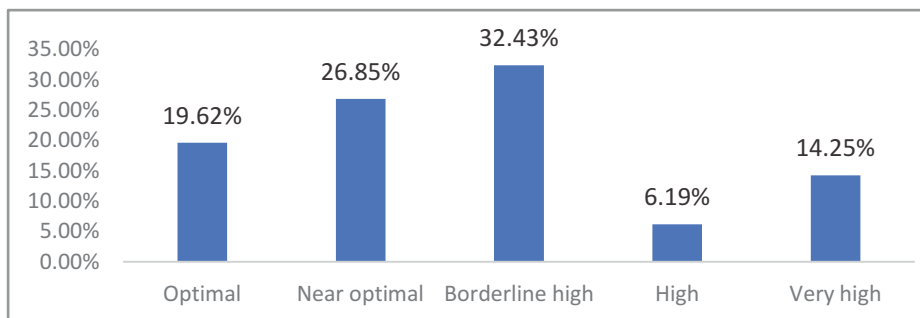
$$n = \frac{Z_{\alpha/2}^2 \times p \times (1-p)}{d^2}$$

It was calculated that 418 samples for LDL and 392 samples for ApoB were needed. So, a group of 500 individuals of Indian ethnicity, both males and females of 18-55 years of age were enrolled. Patients on statins, critically ill patients, pregnant women, and lactating women were excluded. After obtaining ethical clearance from the ethics committee, and obtaining informed consent from the participants, 2ml of blood was drawn from the subject. Blood was centrifuged within 30 minutes and serum was stored in -20° C till evaluation. ApoB and LDL were assessed by Immuno-turbometric method using ERBA EM 360 auto analyser. After obtaining the necessary parameters (i.e.) ApoB and LDL, the prevalence of the same was calculated. Normal range of ApoB was taken as 75-150 mg/dl. Reference range of LDL was taken as optimal: <100 mg/dl; near optimal: 100-129 mg/dl; Borderline high: 130-159 mg/dl; high: 160-169 mg/dl; very high: >190 mg/dl. The percentage of high LDL and ApoB was calculated using MS Excel. The correlation between ApoB

and LDL levels were calculated using Pearson's correlation coefficient r. If 'r' was 0, it indicated no correlation, and if it was 1 it indicated positive linear correlation.

**Results**

The overall LDL characteristics of the observed population are given in figure 1. Out of 500 people studied, 48.45% of them had optimal and near optimal level, whereas 52.87% of the population had high LDL levels. Out of the entire male subjects, 49.76% of the population had optimal and near optimal level, whereas 45.76% of the population had high LDL as given in figure 2. Out of the entire female subjects, 41.26% of the population had optimal and near optimal level, whereas 56.49% of the people had high LDL as given in figure 3. Out of the study subjects, 4.33% of the people had high Apo-B level. Out of the 4.33%, the prevalence among males and females was males-3.31% and females-1.03% as given in figure 4. On comparing the relation between LDL and Apo-B levels, the scatter plot obtained is depicted in Figure 5. On performing the student t-test to obtain the correlation between LDL and ApoB levels, the Pearson's correlation 'r' showed a value of 1, thereby indicating a positive correlation. The scatter plot also shows the same (Figure 5). Thereby we can conclude that there is a positive correlation between LDL and Apo-B.



**Figure 1: Prevalence of optimal, near optimal and high LDL among subjects**

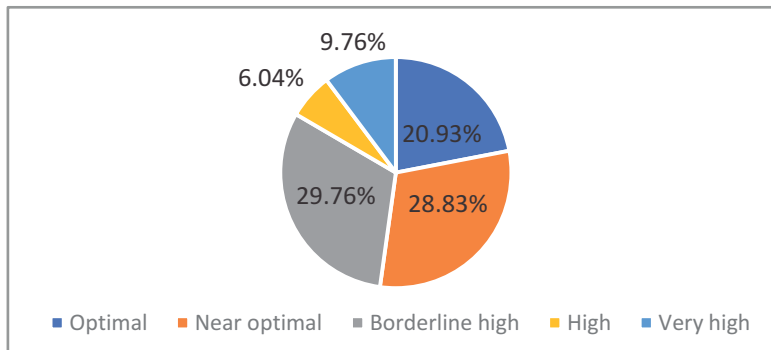


Figure 2: Prevalence of optimal and high LDL among the male subjects

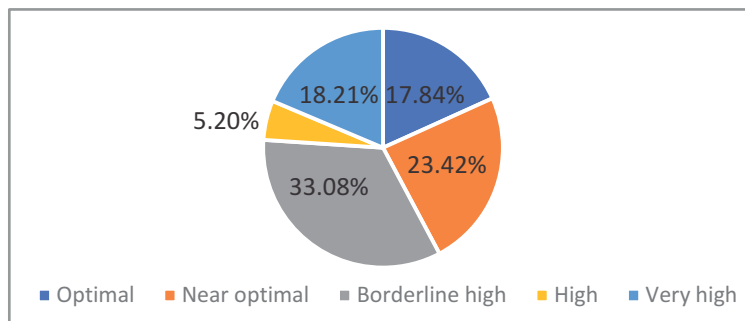


Figure 3: Prevalence of optimal and high LDL among the female subjects

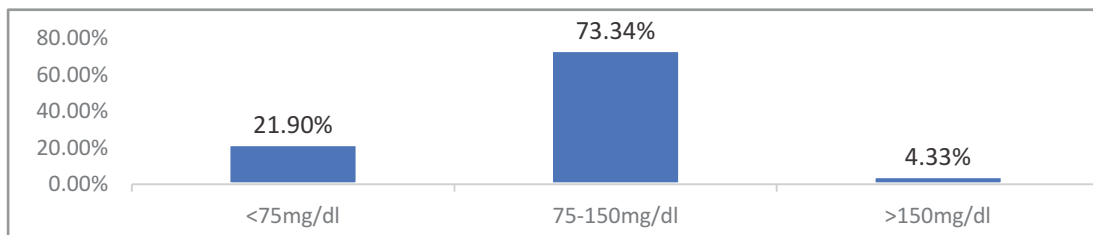


Figure 4: Prevalence of normal and high Apo-B levels among study subjects

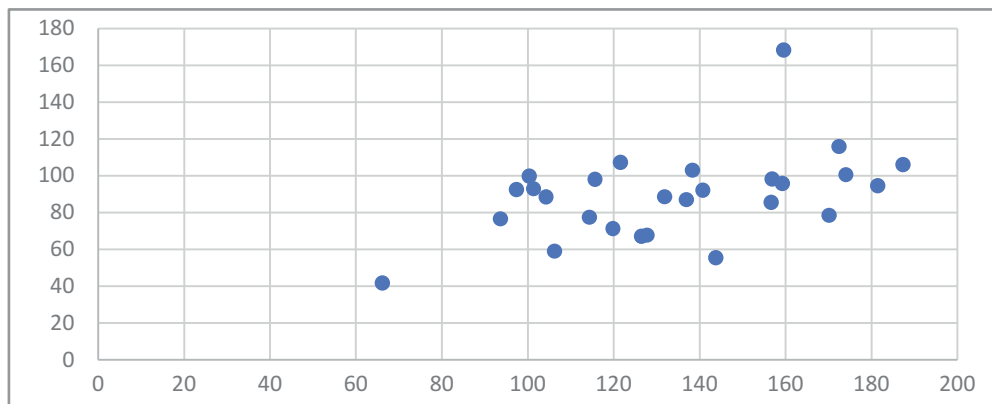


Figure 5: Scatter Plot of LDL Vs. ApoB showing a positive correlation

## Discussion

For more than 70 years, LDL levels are used to quantify the risk of developing CAD and ApoB is a recently accepted measure for the same. These measurements also help in therapeutic decision making [8, 15, 17]. In this current study, it was been found that out of the whole population, optimal and near optimal LDL was recorded only in 19.62 % and 26.85% respectively, whereas high LDL was accounted in 52.87%, which is more than half the population under study. Therefore more than half our study population was at risk of developing atherosclerosis which in turn may cause increased mortality due to cardiovascular accidents.

Our study is in accordance with ICMR-INDIAB study [18] which showed increased prevalence of high LDL and high ApoB in Tamilnadu population. On comparing the LDL levels of males and females in the sample population, it was found that prevalence of high LDL was higher among the female population. The levels of high LDL were 45.56% and 56.49% in men and women, respectively. Therefore more stringent levels of controlling the LDL levels have to be adopted in the females for better health outcome. High ApoB prevalence is also measured in the population and it is found that the prevalence of high apolipoprotein is 4.33% and among the population 3.31% of males and 1.03% of females have high apolipoprotein.

Generally, LDL-C, and ApoB concentrations are very highly correlated. They provide similar information regarding risk of CAD. Positive correlation was found between LDL and ApoB in our sample population with the 'r' value = 1 (Pearson's correlation coefficient).

The high prevalence of higher LDL and ApoB may be due to genetic, or food and other environmental

factors. A radical change in diet may lower LDL and Apo-B thereby reducing heart risk. This anti-atherosclerotic property of foods like fruits, non-starchy vegetables, legumes, nuts, fish, vegetable oils, yoghurt, and whole grains are well documented. Lowering intake of red meat, processed meats, refined carbohydrates and sugar may also help achieve the same [8, 18].

Some of the LDL lowering agents are statins, cholesterol absorption inhibitors, bile acid sequestrants, pro-protein convertase, subtilisin/kexin type 9 inhibitors, Lomitapide, fibrates, omega 3 fatty acids and nicotinic acid. They aim at lowering the LDL and thereby are protective in nature [19-22]. The prevalence of high LDL and high ApoB apolipoproteins play a pivotal role in identifying the population at risk, so that the required measures can be adopted to decrease the incidence of atherosclerotic coronary vascular disease and thereby improve the health of the population.

## Conclusion

Studies on prevalence of dyslipidemia are very rare in our country. This study assessed the lipid levels of people attending a tertiary hospital in state of Tamilnadu. And it was seen that many had high levels of LDL and Apo B which can be a cause for increased incidence of cardiovascular diseases in our region. The study has some limitations such as people of all socio-economic statuses were taken and dietary habits were not studied. Even then this study shows a picture of cardiovascular risk and emphasize on measures that should be taken to reduce them.

## References

1. Malakar AK, Choudhury D, Halder B, Paul P, Uddin A, Chakraborty S. A review on coronary artery disease, its risk factors, and therapeutics. *J Cell Physiol* 2019;234(10):16812-16823.
2. Roth GA, Forouzanfar MH, Moran AE, Barber R, Nguyen G, Feigin VL, *et al.* Demographic and epidemiologic drivers of global cardiovascular mortality. *N Engl J Med* 2015 Apr 2;372(14):1333-1341.
3. Bauersachs R, Zeymer U, Brière JB, Marre C, Bowrin K, Huelsebeck M. Burden of coronary artery disease and peripheral artery disease: A literature review. *Cardiovasc Ther* 2019;2019:8295054.
4. Dong H, Chen, W, Wang, X, *et al.* Apolipoprotein A1, B levels, and their ratio and the risk of a first stroke: a meta-analysis and case-control study. *Metab Brain Dis* 2015; 30(6): 1319-1330.
5. Mehta A, Shapiro MD. Apolipoproteins in vascular biology and atherosclerotic disease. *Nat Rev Cardiol* 2022;19(3):168-179.
6. Seishima M. Physiological function of apolipoproteins and atherosclerosis. *Rinsho Byori* 2016;64(2):186-192.
7. François Mach, Colin Baigent, Alberico L Catapano, Konstantinos C Koskinas, Manuela Casula, Lina Badimon, *et al.* ESC Scientific Document Group, 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk: The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS), *Eur Heart J* 2020; 41(1):111-188.
8. Tabas I, Williams KJ, Borén J. Subendothelial lipoprotein retention as the initiating process in atherosclerosis: update and therapeutic implications. *Circulation* 2007;116(16):1832-1844.
9. Borén J, Williams KJ. The central role of arterial retention of cholesterol-rich apolipoprotein-B-containing lipoproteins in the pathogenesis of atherosclerosis: a triumph of simplicity. *Curr Opin Lipidol* 2016;27(5):473-483.
10. Xu X, Song Z, Mao B, Xu G. Apolipoprotein A1-Related proteins and reverse cholesterol transport in antiatherosclerosis therapy: Recent progress and future perspectives. *Cardiovasc Ther* 2022;2022:4610834.
11. Sniderman AD, Marcovina SM. Apolipoprotein A1 and B. *Clin Lab Med* 2006;26(4):733-750.
12. Emerging Risk Factors Collaboration; Di Angelantonio E, Gao P, Pennells L, Kaptoge S, Caslake M, *et al.* Lipid-related markers and cardiovascular disease prediction. *JAMA* 2012;307(23):2499-506.
13. Ference BA, Graham I, Tokgozoglul L, Catapano AL. Impact of Lipids on Cardiovascular Health: JACC Health Promotion Series. *J Am Coll Cardiol* 2018; 72(10):1141-1156.
14. Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL *et al.* ESC Scientific Document Group. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. *Eur Heart J* 2016;37(29):2315-2381.
15. Ohira T, Iso H. Cardiovascular disease epidemiology in Asia: an overview. *Circ J* 2013;77(7):1646-52.
16. Kumar R, Singh, P. Prevalence of High LDL and Apo B levels in different ethnic groups: A multicenter study. *Int J Clin Lipidol* 2020; 50(2): 203-210.
17. Kavousi M, Elias-Smale S, Rutten JH, Leening MJ, Vliegthart R, Verwoert GC, *et al.* Evaluation of newer risk markers for coronary heart disease risk classification: A cohort study. *Ann Intern Med* 2012; 156(6):438-444.
18. Joshi SR, Anjana RM, Deepa M, Pradeepa R, Bhansali A, Dhandania VK, *et al.* Prevalence of dyslipidemia in urban and rural India: The ICMR-INDIAB Study. *PLoS One* 2014; 9(5): e96808.
19. Brugts JJ, Yetgin T, Hoeks SE, Gotto AM, Shepherd J, Westendorp RG, *et al.* The benefits of statins in people without established cardiovascular disease but with cardiovascular risk factors: Meta analysis of randomised controlled trials. *BMJ* 2009;338:b2376.
20. Cholesterol Treatment Trialists' (CTT) Collaborators; Mihaylova B, Emberson J, Blackwell L, Keech A, Simes J, *et al.* The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomised trials. *Lancet* 2012;380(9841):581-590.

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21. Khan MD, Ahmad MK, Alam R, Khan S, Jaiswal G, Jahan A, Khan MM. Association of central obesity with risk factors for cardiovascular disease in North Indian population: A case-control study. *J Krishna Inst Med Sci Univ* 2023; 12(2):81-93.
22. Kottagi SS, Rathi DB, Dongre NN. Evaluation of LDL-Cholesterol / HDL-Cholesterol ratio as predictor of dyslipidemia in subclinical hypothyroidism. *J Krishna Inst Med Sci Univ* 2014; 3(1): 34-40.
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