Original Research Article

Lipid profile among newly detected cases of coronary heart disease

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Abstract

Background: Burden of coronary heart disease is constantly increasing in the past few decades. There are extensive epidemiological data demonstrating that high blood cholesterol levels increase cardiovascular risk, and that this risk is dependent on levels of the different blood cholesterol fractions. Moreover, the reduction of total blood cholesterol has been clearly related to a reduction in the risk of stroke, coronary disease and overall cardiovascular death. Hence, this Study was planned to assess the pattern of fasting lipid profile among newly detected cases of Coronary Artery Disease. **Methods:** A cross-sectional study was conducted among newly detected coronary artery disease patients in a tertiary care teaching hospital from April 2015 to March 2016. A total of hundred patients were included. Following collection of detailed history, venous blood samples were collected from all participants and it was assessed for Fasting blood sugars, Fasting lipid profile, post prandial blood sugars and HbA1c. Data was analysed using SPSS software. **Results:** Findings of this study shows that higher levels of total cholesterol, LDL and triglycerides were reported among the newly diagnosed cases of coronary heart disease and at the same time HDL levels was found to be lower among them. **Conclusions:** LDL-C and triglycerides are established in clinical practice as important tools in risk prediction and therapy follow-up. The importance of checking these measurements at regular periodic intervals should be made aware to the patients in order to prevent the complication that occurs due to it.
Key Word: Coronary artery disease, Lipid profile, LDL, HDL.

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INTRODUCTION

India is undergoing a rapid health transition with rising burden of coronary heart disease (CHD).[1] Among adults over 20 year of age, the estimated prevalence of CHD is around 3-4 per cent in rural areas and 8-10 per cent in urban areas, representing a two-fold rise in rural areas and a six-fold rise in urban areas between the years 1960 and 2000.[2] Studies among Indian migrants in various parts of the world have documented an increased susceptibility to CHD in comparison to the native population studied.[3] Several factors such as genetic, metabolic, early-life, conventional and non-conventional risk factors were suspected to cause high CHD morbidity and mortality rates among Indians. However, the results from the INTERHEART study conclusively established the role of behavioural and conventional risk factors in the prediction of CHD risk among Indians.[4] Lipoprotein-a (Lp-a) is now recognised as an independent risk factor for CAD. It is a genetic risk factor. It is not affected by any level of lifestyle modifications like changes in diet and exercise. Lp-a is ten-times more atherogenic than LDL-C.[5] It promotes early atherosclerosis and thrombosis. Lp-a is a stronger risk-factor than DM for CAD in younger women. Lp-a levels above 30 mg/dl are associated with three-fold higher risk of CAD. Lp-a levels over 40 mg/dl increases the risk associated with cigarette smoking by 1.9 times, with DM by 3.4 times, with high total

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cholesterol by 4.2 times, with hypertension by 4.6 times, with high TC/HDL ratio by 6.9 times, and with high homocysteinaemia by 9.3 times[6]. In Indian patients with CAD, high triglyceride levels are found more often than high cholesterol levels. Triglycerides bring change in LDL particle size, density, distribution, and composition producing smaller, denser, and more atherogenic particles.[7] Estimation of triglyceride level gives an indirect measurement of LDL particle size. An increase of triglycerides from 90 mg/dl to 180 mg/dl is associated with doubling the incidence of CAD.[8] Increase in triglycerides by 90 mg/dl has the same effect on coronary atherosclerosis, as increase in age by 10 years.[9] Earlier, there has been an under-emphasis on the significance of triglycerides as a risk factor for CAD. Indians worldwide demonstrate a triad of high triglycerides with high LDL-C levels and low HDL levels. This triad combined with high levels of lipoprotein-a constitutes the deadly lipid quartet. Higher levels of apolipoprotein-B (Apo-B) are reported in one third of Indians males. This factor in combination with low levels of HDL and hypertriglyceridaemia results in formation of small dense LDL which increases the risk of CAD more than three times. The LDL-cholesterol types are described as phenotypes A, B, or C, which are genetically determined. Patients with LDL phenotype-B have predominantly small and dense LDL-particles which as mentioned above, constitute an important risk factor for CAD.[10] A 75% prevalence of phenotype-B is seen in Asian Indians in contrast to 25% in White population. High levels of plasminogen activator inhibitor-1 (PAI-I) in Indians are reported in association with hypertriglyceridaemia and hyperinsulinaemia. This combination promotes thrombosis by impairing fibrinolysis.[11] It is expected that individuals of Indian Asian ethnicity will account for between 40-60 per cent of global CVD burden within the next 10-15 years[8]. The astonishingly higher risk in this particular ethnic group has been attributed to underlying genetic susceptibility unmasked by environmental factors[12] or intrauterine programming which predisposes to asymmetric energy metabolism and rapid, excess accumulation of visceral body fat in adult life.[13]

AIMS

To Study the pattern of fasting lipid profile in newly detected cases of Coronary Artery Disease.

MATERIALS AND METHODS

A cross-sectional study was conducted among the newly detected cases of coronary artery disease presenting to Emergency department and department of general medicine in Vinayaka Missions Medical College Hospital, Salem. The study was conducted for a period of one year from April 2015 to March 2016. The patients were diagnosed to suffer from CAD by means of ECG, treadmill testing and echocardiography were included in this study and patients who are already on treatment with antilipidemic drugs, patients with chronic kidney disease, patients with liver diseases, rheumatological diseases chronic infections and hypertension patients were excluded from the study. A total of 100 patients with newly detected coronary artery disease were included in the study. After obtaining the written informed consent from the patients, detailed history was collected by the principal investigator. Height, weight, waist, hip circumference were measured. BMI was calculated by dividing weight in Kg with the square of height in meters. Waist hip ratio (WHR) was calculated. Overweight/obesity was defined as BMI $\geq$25 kg/m^2, BMI was classified into various groups for inter-group comparison of risk factors: Group I Group II 20.0-22.9, Group III 23.0-24.9, Group IV 25.0-29.9 and Group V $\geq$ 30 Kg/m2 modified from WHO classification. The waist-size was classified into five groups and the criteria used were different for men and women. In men these groups were <80 cm, 80-89 cm, 90-99 cm, 100-109 cm and 110 cm and in women these were <70 cm, 70-79 cm, 80-89 cm, 90-99 cm and 100 cm. WHR was grouped into six categories. Men were classified into <0.8, 0.80-0.84, 0.85-0.89, 0.90-0.94, 0.95- 0.99 and 1.00 and women into groups of <0.75, 0.75- 0.79, 0.80-0.84, 0.85-0.89, 0.90-0.94 and 0.95.Type 2Diabetes Mellitus was confirmed based on criteria given by American Diabetes Association-2012, hypertension (HTN) by systolic and diastolic blood pressures $\geq$140 and 90 mmHg respectively. Atherogenic dyslipidemia was defined as TG level $\geq$150 mg/dl and HDL cholesterol level (<40 mg/dl) regardless of patient's gender. Following which venous blood samples were collected from the patients. Fasting blood samples were collected after 14 h fasting. Lipids were measured by using cholesterol oxidase para aminoantipyrine, lipase/glycerol kinase (LIP/GK), enzymatic reaction respectively and low density lipoprotein (LDL) cholesterol and very low density lipoprotein cholesterol were calculated by Freidwald formula. Inter assay 3.84% and intra precision was 2% respectively for all biochemical parameters. HemoglobinA1c was measured by boronate affinity assay. Data entry was done using Microsoft excel and the statistical analysis was done using SPSS software version 17.

RESULTS

In this study 29% of the participants with coronary artery disease were between 61 to 65 years of age followed by 20% in the age group of 51 to 55 years. Least number of patients were recorded in the age group of 41 to 45 and 71 to 75 years of age i.e. 4% and 3% respectively. Regarding gender 57% of the study participants were males, 28% of the patients had family history of CAD, 24% were smokers and 22% had history of alcohol consumption, as shown in Table 1.

Table 1: Background characteristics of the participants

Variables	Percentage
Age group	
35 – 40 years	10
41 – 45 years	4
46 – 50 years	10
51 – 55 years	17
56 – 60 years	20
61 – 65 years	29
66 – 70 years	7
71 – 75 years	3
Gender	
Male	57
Female	43
Participants with positive history of	
Smoking	24
Alcohol consumption	22
Family history of CAD	28

Twenty six percentage of the participants fall in the BMI range of 27.1 – 28, followed by 15% in the range of 28.1-29, 14% in 29.1-30 and 25-26 each. Ten percent of the CAD patients were having BMI less than 25 and 8% CAD patients were above 30, shown in figure 1.

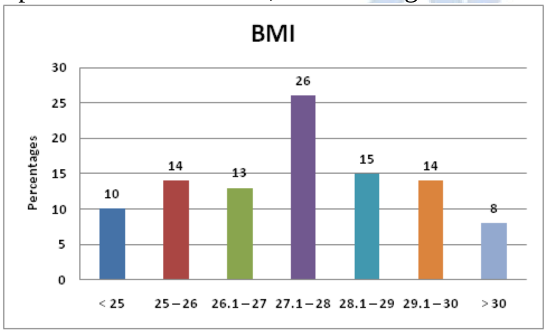

Figure 1: BMI of the participants

In this study waist hip ratio was in the range of 0.90-0.94 for 26% of the people, 0.85-0.89 for 20% of the CAD patients, 0.80-0.84 for 17% of the cases. 13% of the CAD patients had waist hip ratio between 0.76-0.79 and 24% within the range of 0.70-0.75. Total Cholesterol value was 100-150 mg/dl, 151-200 mg/dl, 201-250 mg/dl, 251-300 mg/dl and more than 300mg/dl in 17%,37%, 26%,18% and 2% respectively. Low Density Lipoprotein (LDL) was 121-140mg/dl in 28% patients. It was less than 100 mg/dl in 5 CAD cases and more than 161 mg/dl in 15% of the patients. Triglycerides were found to be less than 100mg/dl in 4% of the CAD patients and it was more than 250mg/dl in 10% of the participants. 36% of the patients had triglyceride value in the range of 151-200 mg/dl. HDL was more than 50 mg/dl in 2% CAD cases, 46-50 mg/dl in 6% of the cases. Low HDL values less than 26 mg/dl was found in 16% of the CAD patients, shown in Table 2.

Table 2: Lipid Profile of the participants

Variables	Percentage
Total cholesterol	
100 – 150 mg/dl	17
151 – 200 mg/dl	37
201 – 250 mg/dl	26
251 – 300 mg/dl	18
>300 mg/dl	2
LDL	
<100 mg/dl	5
100 – 120 mg/dl	17
121 – 140 mg/dl	35
141 – 160 mg/dl	28
161 – 180 mg/dl	13
181 – 200 mg/dl	2
Triglycerides	
<100 mg/dl	4
100 – 150 mg/dl	29
151 – 200 mg/dl	36
201 – 250 mg/dl	21
>250 mg/dl	10
HDL	
20 – 25 mg/dl	16
26 – 30 mg/dl	24
31 – 35 mg/dl	24
36 – 40 mg/dl	22
41 – 45 mg/dl	6
46 – 50 mg/dl	6
>50 mg/dl	2

The mean BMI among the study subjects were found to be 27.72±3.32, mean waist hip ratio was 0.927±0.21. The mean Fasting blood sugar and HbA1C in this study was 100.7±11.07 and 5.90±0.49. Likewise mean and standard deviation for Blood urea and Blood creatinine was 38.56±1.28 and 0.77±0.24 respectively. The mean total cholesterol was found to be 204.43±50.48, LDL 137.16±25.12. 185.72±66.99 was the mean Triglyceride value and 32.85±7.29 were the mean and SD of HDL, as shown in Table 3.

Table 3: Mean and SD of various parameters

Parameter	Mean ± SD
BMI	27.72±3.32
Waist hip ratio – Male	0.927±0.21
Fasting blood sugar (mgs/dl)	100.7±11.07
HbA1C	5.90±0.49
Blood urea(mg/dl)	38.56±1.28
Blood creatinine (mg/dl)	0.77±0.24
Total cholesterol (mg/dl)	204.43±50.48
LDL (mg/dl)	137.16±25.12
Triglycerides (mg/dl)	185.72±66.99
HDL (mg/dl)	32.85±7.29

DISCUSSIONS

The disorder of lipid metabolism is a key player for the occurrence of cardiovascular disease and particularly heart disease. For many years, cholesterol has been directly related to cardiovascular prognosis. This relationship is very consistent, as an increase of 2 to 3% in the incidence of coronary heart disease is expected for every 1% increase in total cholesterol.[14] For many years it was difficult to classify unequivocally Triglycerides as an independent risk factor for the occurrence of coronary heart disease, a situation presumably related to the wide fluctuations observed in their concentrations throughout the day, with the heterogeneity of triglyceride-rich lipoproteins and its inseparable association with other risk factors. However, several studies have demonstrated a clear correlation between their levels and the occurrence of coronary heart disease, indicating that the presence of high levels of Triglycerides leads to a 13% increase in the risk of cardiovascular disease in men and 37% in women.[15] With regard to HDL-cholesterol, its inverse relationship with the risk of coronary heart disease is well accepted. In fact, this risk is 2 to 3% lower for each 1mg/dl elevation of HDL-Cholesterol.[16] The protective properties of this fraction derive not only from its involvement in reverse cholesterol transport, but are also a consequence of its anti-inflammatory capacity and protection against LDL-cholesterol oxidation.[17] On the other hand, it is recognized that individuals with very low levels of HDL-cholesterol have a higher cardiovascular risk. Recent evidence further stresses the importance of determining the non-HDL-cholesterol, defined by the concentration of LDL-cholesterol + VLDL-cholesterol. This parameter can better translate the risk of cardiovascular mortality than LDL-cholesterol, as it expresses more accurately the lipoprotein atherogenicity.[18] Several large studies have demonstrated that the LDL-Cholesterol/HDL Cholesterol ratio is an excellent predictor of risk of coronary disease and an excellent way to monitor the impact of lipid-lowering therapies.[19] In the PROSPER trial, a retrospective analysis of 6,000 patients, the LDL-Cholesterol/HDLC holesterol ratio was the stronger predictor of cardiovascular events in elderly patients.[20] Among the main risk factors, dyslipidemia, especially increase in LDL levels and decrease in HDL concentrations were considered as the important factors. Table below demonstrates the Population Attributable Factors (PARs) with its 99 percent confidence interval (CI) associated with lipids by sex and geographic region.[21] In parallel to these large population studies, a series of case studies were also performed. In one study, serum lipid levels were evaluated in 500 men with a prior history of myocardial infarction. Overall 30 percent of study population had abnormal blood lipid levels.[22] Based on results of the National Health and Nutrition Examination Surveys (NHANES) from 1999 to 2004, the percentage of adults with triglyceride levels above 150 and 200 mg/dl in the United States, were 33 and 18 percent, respectively.[23] MRFIT study performed in more than 350,000 middle-aged men demonstrated that a sigmoid relationship (curvilinear) between total serum cholesterol level and prevalence of coronary artery disease especially in total cholesterol more than 240 mg/dl[24]. LRCP (Lipid Research Clinics Program) was one of the first surveys during 1970 that was conducted to determine the total cholesterol, HDL cholesterol, LDL cholesterol and triglyceride levels in American adults.[25] In another study, difference in distribution of cholesterol and its components in the blood in accordance to age were described.[26] Among patients with a prior history of myocardial infarction, an elevated total cholesterol following recovery was a major independent risk factor for re-infarction, death from heart disease and total mortality. Cardiovascular mortality is varied in different populations. The highest and lowest mortality rate was found in Finland and Japan, respectively, with a direct relationship to serum cholesterol levels.[27] The negative relationship between low HDL cholesterol and the risk of heart disease is well established in the general population.[28] Predictive role of HDL against coronary events was also well documented in patients with known heart disease. The results of Lipid and Care clinical trial showed that low levels of HDL cholesterol is a stronger predictor of heart disease incidence in presence of serum LDL cholesterol < 125 mg/dl than LDL cholesterol ≥ 125 mg/dl.[29] They also found that in serum LDL<125 mg/dl, each 10 mg /dl increase in HDL level, will cause 29 percent reduction in the incidence of cardiovascular events, while with the serum LDL cholesterol ≥ 125 mg/dl, this attenuation will be lowered to 10 percent. As mentioned previously, the cardioprotective effect of HDL was shown to be present at serum levels higher than 60 mg/dl.[30] These effects are more prominent when the serum levels of HDL cholesterol reach 75 mg/dl and higher. In the Lipid Research Clinics study, the Framingham heart Study and the HHS8 the ratio of LDL to HDL was shown to be the best predictor of cardiovascular events.[31] In HHS study, the risk of new coronary events such as myocardial infarction and sudden cardiac death in patients with LDL/HDL ≥ 5 and a concomitant serum triglycerides ≥ 200 mg /dl, was fourfold more than patients with lower LDL/HDL ratio and triglycerides levels. Overall, among men, an LDL/HDL ratio of ≥ 6.4 had 2–14 percent higher predictive value than serum total cholesterol or LDL levels. Among women the predictive value of LDL/HDL

≥ 5.6 was 25–45 percent greater than serum total cholesterol or LDL level.[32] The relationship between hypertriglyceridemia and CVD was determined in the population based Stockholm prospective study.[33] In this study, 3,486 subjects were followed for 14.5 years. An independent relation between hypertriglyceridemia and CVD was observed in this study, which was stronger than the relationship between hypercholesterolemia and CVD. Meta-analysis of several large population–based prospective studies showed similar results.[34] As mentioned previously in the HHS study, not only there is an interaction between triglycerides and total cholesterol/HDL ratio, but also an inverse association between triglycerides and HDL levels exists.[27]Additionally, hypertriglyceridemia is associated with increased mortality in patients with known CHD and also reduces the event–free survival after coronary artery bypass graft surgery (CABG).[35] Nevertheless, because hypertriglyceridemia is an independent risk factor for CVD, measurement of triglycerides as a part of routine cholesterol screening is recommended by NECP ATPIII guidelines.[35] Fasting triglyceride measurement is important for evaluating the risk of heart disease especially in cases who are suffering from diabetes, glucose intolerance, insulin resistance syndrome, obesity and low HDL. Although, triglyceride measurement is commonly done after 8–12 hours fasting, an association between nonfasting triglyceride levels and CVD is also present.[36] As mentioned by the previous studies the results of our study had also proven that the lipid parameters can act as an independent risk factor in the occurrence of coronary artery diseases. Total cholesterol levels, LDL cholesterol and triglycerides had shown a strong positive correlation in the occurrence of CVD and the HDL levels had shown a negative correlation in the occurrence of CVD among our study subjects in spite of their blood sugar levels and blood pressure being maintained at normal levels.

CONCLUSIONS

Based on this analysis of the lipid metabolism, its importance in the prediction of cardiovascular risk is reiterated. Classic measurements, such as LDL-C, HDL-C and triglycerides are established in clinical practice as important tools in risk prediction and therapy follow-up. Besides them, it is important to find other markers, due to the considerable number of individuals that suffer cardiovascular events with few or none of the risk factors Lipid metabolism has several elements with a potential role in the risk prediction and monitoring that are targets for the development of new therapeutic strategies for CVD, as many of these makers take part in the physiopathology of atherosclerosis. The next guidelines of ATP IV are being determined, and possibly Lp(a) will be included as a reference measure for the treatment and monitoring of CVD. There are great expectations for apo B, non-HDL cholesterol, and total cholesterol/ HDL-C ratio as aids in the evaluation of cardiovascular risk. It may be concluded that the fast evolution of knowledge on the subject may justify periodic reassessment of lipid metabolism.

REFERENCES

1. Reddy KS, Shah B, Varghese C, Ramadoss A. Responding to the threat of chronic diseases in India. Lancet 2005; 366: 1744-9.
2. Gupta R, Joshi P, Mohan V, Reddy KS, Yusuf S. Epidemiology and causation of coronary heart disease and stroke in India. Heart 2008; 94: 16-26.
3. McKeigue PM, Miller GJ, Marmot MG. Coronary heart disease in South Asians: a review. Clin Epidemiol 1989; 42: 579-609.
4. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, *et al*; INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004; 364: 937-52.
5. Lawn RM. Lipoprotein (a) in heart disease. Sci Am 1992; 266: 54-60.
6. Bhatnagar D, Anand I, Durington PN *et al*. Coronary risk factors in people from Indian subcontinent in West London and their siblings in India. Lancet 1995; 345: 405-9.
7. Hopkins PN, Wu L, Hunt S *et al*. Lipoprotein (a) interactions with lipid and nonlipid risk factors in early familial coronary artery disease. Arterioscler Thromb Vasc Biol 1997; 17: 2783-92.
8. Lamarche B, Tchernof A, Moorjani S *et al*. Small dense low-density lipoprotein particles as a predictor of the risk of ischaemic heart disease in men. Prospective results from Quebac Cardiovascular Study. Circulation 1997; 95: 69-75.
9. Jeppesen J, Hein H, Suadicani P, Gyntelberg F. Relation of high TG-low HDL cholesterol and LDL cholesterol to the incidence of ischemic heart disease: An 8-year followup in the Copenhagen Male study. Arterioscler Thromb Vasc Biol 1997; 17: 1114-20.
10. Drexel H, Amann F, Beran J *et al*. Plasma triglycerides and three lipoprotein cholesterol fractions are independent predictors of extent of coronary atherosclerosis. Circulation 1994; 90: 2230-5.
11. Hamstein A, Walldius G, Szamosi A *et al*. Plasminogen activator inhibitor in plasma: risk factor for recurrent myocardial infarction. Lancet 1987; 3-9.
12. Chambers JC, Elliott P, Zabaneh D, Zhang W, Li Y, Froguel P, *et al*. Common genetic variation near MC4R is associated with waist circumference and insulin resistance. Nat Genet 2008; 40: 716-8.
13. Lev-Ran A. Human obesity: an evolutionary approach to understanding our bulging waistline. Diabetes Metab Res Rev 2001; 17: 347-62.
14. Carlson, L.A.; Bottiger, L.E.; Ahfeldt, P.E. (1979). Risk factors for myocardial infarction in the Stockholm

Prospective Study. A 14-year follow-up focusing on the role of plasma triglycerides and cholesterol. Acta Med Scand, 206:351-360.

15. Castelli, W.P. (1986). The triglyceride issue: a view from the Framingham. Am Heart J, 112:432-437.
16. Gordon, D.J.; Probstfield, J.L.; Garrison, R.J.; *et al.* (1989). High-density lipoprotein cholesterol and cardiovascular disease: four prospective american studies. Circulation, 79:8-15.
17. Ansell, B.J.; Navab, M.; Watson, K.E.; *et al.* (2004). Anti-inflamatory properties of HDL. Rev Endocr Metab Disord, 5:351-358.
18. Cui, Y.; Blumenthal, R.S.; Flaws, J.A.; *et al.* (2001). Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. Arch Intern Med, 161:1413- 1419.
19. Manninen, V.; Tenkanen, L.; Koskinen, P.; *et al.* (1992). Joint effects of serum triglyceride and LDL cholesterol and HDL cholesterol concentrations on coronary heart disease risk in the Helsinki Heart Study. Implications for treatment. Circulation, 85:37–45.
20. Packard, C.J.; Ford, I.; Robertson, M.; *et al.* (2005). The PROSPER Study Group: Plasma lipoproteins and apolipoproteins as predictors of cardiovascular risk and treatment benefit in the PROspective Study of Pravastatin in the Elderly at Risk (PROSPER). Circulation, 112:3058–3065.
21. Labarthe DR (2011). Coronary heart disease, In: Epidemiology and prevention of cardiovascular disease: a global challenge, Labarthe DR, pp. 59-87, Jones and Bartlett, ISBN-13: 978–0– 7637–4689–6, Canada.
22. Goldstein JL, Hazzard WR, Schrott HG, Bierman EL, Motulsky AG (1973). Hyperlipidemia in coronary heart disease I. Lipid levels in 500 survivors of myocardial infarction. J Clin Invest; 52:1533–43.
23. Ford ES, Li C, Zhao G, Pearson WS, Mokdad AH (2009). Hypertriglyceridemia and its pharmacologic treatment among US adults. Arch Intern Med; 169: 572–8.
24. Stamler, J, Wentworth, D, Neaton, JD (1986). Is relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? Findings in 356,222 primary screenees of the Multiple Risk Factor Intervention Trial (MRFIT). JAMA; 256:2823–8.
25. Heiss G, Tamir I, Davis CEo (1980). Lipoprotein – Cholesterol distributions in selected North American populations: the Lipid Research Clinics Program Prevalence Study. Circulation; 61: 302–15.
26. Glueck CJ, Stein EA (1979). Treatment and management of hyperlipoproteinemia in childhood. In: Levy R, Rifkind B, Dennis B, Ernst N. Nutrition, Lipids, and Coronary Heart Disease, New York: Raven Press; pp. 285–307.
27. Bansal S, Buring JE, Rifai N, *et al* (2007). Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA; 298:309–16.
28. Abbott RD, Wilson PWF, Kannel WB, Castelli WP (1988). High density lipoprotein cholesterol, total cholesterol screening, and myocardial infarction: the Framingham Study. Arteriosclerosis; 8: 207–11.
29. Sacks FM, Tonkin AM, Craven T, Pfeffer MA, Shepherd J, Keech A, *et al* (2002). Coronary Heart disease in patients with low LDL–cholesterol: benefit of pravastatin in diabetics and enhanced role for HDL– cholesterol and triglycerides as risk factors. Circulation; 105: 1424–8.
30. Castelli WP (1983). Cardiovascular disease and multifactorial risk: Challenge of the 1980s. Am Heart J; 106: 1191–200.
31. Manninen V, Tenkanen L, Koskinen P, Huttunen JK, Manttar M, Heinonen OP, *et al* (1992). Joint effects of serum triglyceride and LDL–cholesterol and HDL–cholesterol concentrations on coronary heart disease risk in the Helsinki Heart Study: implications for treatment. Circulation: 85: 37–45.
32. Kinosian B, Glick H, Garland G (1994). Cholesterol and coronary heart disease: predicting risk s by levels and ratios. Ann Intern Med; 121: 641–7.
33. Carlson LA, Böttiger LE, Ahfeldt PE (1979). Risk factors for myocardial infarction in the Stockholm prospective study. A 14–year follow-up focussing on the role of plasma triglycerides and cholesterol. Acta Med Scand;206(5):351–60.
34. Hokanson IE, Austin MA (1996). Plasma triglyceride level is a risk factor in cardiovascular disease independent of high density lipoprotein cholesterol level: a meta – analysis of population - based prospective studies. J Cardiovasc Risk; 3: 213–9.
35. Haim M, Benderly M, Brunner D, *et al* (1999). Elevated serum triglyceride levels and long term mortality in patients with coronary heart disease: the Bezafibrate Infarction Prevention (BIP) Registry. Circulation; 100: 475–82.
36. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A (2007). Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA; 298:299–308.

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