International Journal for Multidisciplinary Research (IJFMR)
E-ISSN : 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Lipid profile in patients with and without acute coronary syndrome

Dr. Rabin Rai[1], Dr. Sangeeta Naorem[2], Dr. Victoria Kshetrimayum[3], Dr. Sachin Deba[4], Dr. Bidya Nongmaithem[5], Dr. Guiphiuliu Kamei[6]

[1,2,3,5,6]Department of Biochemistry, Regional Institute of Medical College, Imphal, Manipur, India
[4]Department of Cardiology, Regional Institute of Medical College, Imphal, Manipur, India

Address for Correspondence: Dr. Rabin Rai, Department of Biochemistry, Regional Institute of Medical College, Imphal, Manipur, India
Email: rai2017rabin@gmail.com

INTRODUCTION

Acute coronary syndrome (ACS) is a syndrome due to decreased blood flow in the coronary arteries such that part of the heart muscle is unable to function properly. ACS is one of the leading causes of increased morbidity and mortality worldwide. As per WHO 2015 report an estimated 7.4 million people died from coronary artery disease worldwide. Similarly, the incidence in India is estimated about 272 per 1,00,000 population in the year 2016. The acute coronary syndrome, the most severe form of coronary artery disease,[1] describes the continuum of myocardial ischemia, which includes unstable angina (UA), non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI).[2] Acute coronary syndrome or Ischemic heart disease (IHD) was the main cause of death globally during the last 15 years and continues to grow at exponential rates.[3] It is an immediate threat of life and the mortality rate can be high without proper therapy and patient management.[1] The common risk factors for the disease are smoking, hypertension, diabetes, hyperlipidaemia, male sex, physical inactivity, obesity, and poor nutritional practices.[4] Although substantial progress has been made in the diagnosis and treatment of acute coronary syndromes, cardiovascular disease remains the leading cause of death worldwide, with nearly half of these deaths due to ischaemic heart disease.[5]

Several studies suggested a relationship between dyslipidemia and atherogenesis which plays an important role in the pathophysiology of acute coronary syndrome. It is shown that atherosclerosis due to dyslipidemia is directly correlated with a risk of IHD. It has been directly linked to hypercholesterolemia, particularly elevated plasma levels of cholesterol in low-density lipoproteins (LDL-C).[6,7] Increased risk of AMI has been seen in patients with low plasma levels of high-density lipoprotein (HDL-C) cholesterol.[8] Hence, keeping these points in mind we have conducted the study to evaluate the association between lipid profile and acute coronary syndrome.

OBJECTIVE

To evaluate the association between lipid profile and acute coronary syndrome.

MATERIALS AND METHODS

Study design: Cross sectional study.

Study setting: Department of Biochemistry in collaboration with the Department of Cardiology, RIMS, Imphal, Manipur.

Duration: January 2021 to October 2022.

Inclusion criteria: Diagnosed cases of acute coronary syndrome above 18 years of age.

Exclusion criteria: Presence of renal diseases, presence of hepatic diseases, diabetes mellitus, recent infection, COPD, osteoarthritis, malignant diseases and peripheral artery disease.

STASTITICAL ANALYSIS

The collected data were analysed using IBM SPSS version 21.0 for windows. Statistics like mean, SD, frequency and proportion were used to summarise the findings. Continuous data like age of the participants and lipid profile were expressed in mean and standard deviation and categorical data like gender, HTN were expressed in frequency and proportion. To compare lipid profile between patients with acute coronary syndrome and the individuals without ACS, student's t-test was used. A p value <0.05 was taken as significant.

ETHICAL ISSUES

Written informed consent was obtained from each patient before recruiting for the study. Confidentiality was strictly maintained. Ethical approval was obtained from the Institutional Ethics Committee, RIMS, Imphal, before the start of the study.

RESULTS

In the present study, lipid profile was evaluated in 67 cases of acute coronary syndrome and 67 healthy individuals taken as controls in the Department of Biochemistry and the Department of Medicine, RIMS, Imphal.

Table I shows the mean±SD of age in cases was 63.69±12.11 years and in the controls, it was 45.47±11.75 years, and the difference observed was found to be statistically significant $p<0.05$. Figure-1 shows that the majority of the cases were males comprising of 49 (73.3%) and females constitute only 18 (26.7%) whereas the majority of the controls were female comprising of 36 (53.3%) and male constitute 31 (46.7%). The difference was statistically significant $p<0.05$. Figure-2 shows that 62.2% (42) of the cases had history of HTN while 37.8% (25) had no h/o HTN.

Table-II shows, in cases the mean±SD of SBP (112.67±17.35 mmHg) and DBP (73.38±11.63 mmHg) were found to be significantly lower than the controls where mean±SD of SBP and DBP were 125.22±5.89 mmHg and 84.07±4.77 mmHg respectively. The difference was statistically significant with $p<0.05$.

Figure-3 shows in the cases the mean±SD of total cholesterol was 208.75±31.25 mg/dl and in controls 144.02±19.57 mg/dl, triglycerides in cases was 162.51±39.81 mg/dl and 98.42±16.96 mg/dl in controls and LDL (142.22±31.31 mg/dl) in the cases and 71.40±21.24 mg/dl in controls showing that the values were higher in the cases as compared to the control group and the difference was found to be statistically significant with $p<0.05$. However, the mean±SD of HDL in the cases (34.78±6.44 mg/dl) was lower as compared to the controls (49.56±5.72 mg/dl) and the difference was statistically significant with $p<0.05$. Table-III shows that 39 (57.77%) cases had TC>200mg/dl, 42 (62.2%) had TG>150mg/dl, 58 (86.66%) of the cases had LDL>100mg/dl and 43 (64.44%) had HDL<40mg/dl.

Table I: Distribution of the respondents by age stratified by cases and controls:

AGE	GROUP	NUMBER	MEAN±SD (Years)	*p*-value
	Case	67	63.69±12.11	0.00
	Control	67	45.47±11.75	

*Independent student t-test

Figure 1: Pie-chart showing gender distribution of the respondents among the cases and the controls:

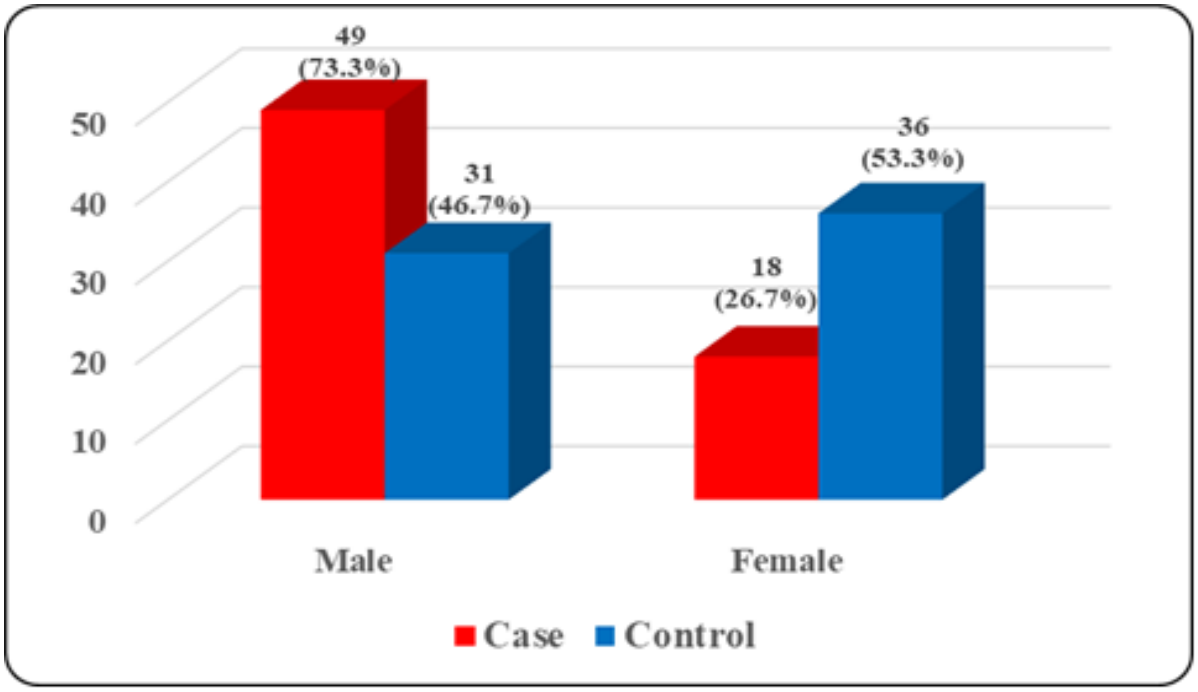

Figure 2: Distribution of the cases according to history of hypertension (HTN):

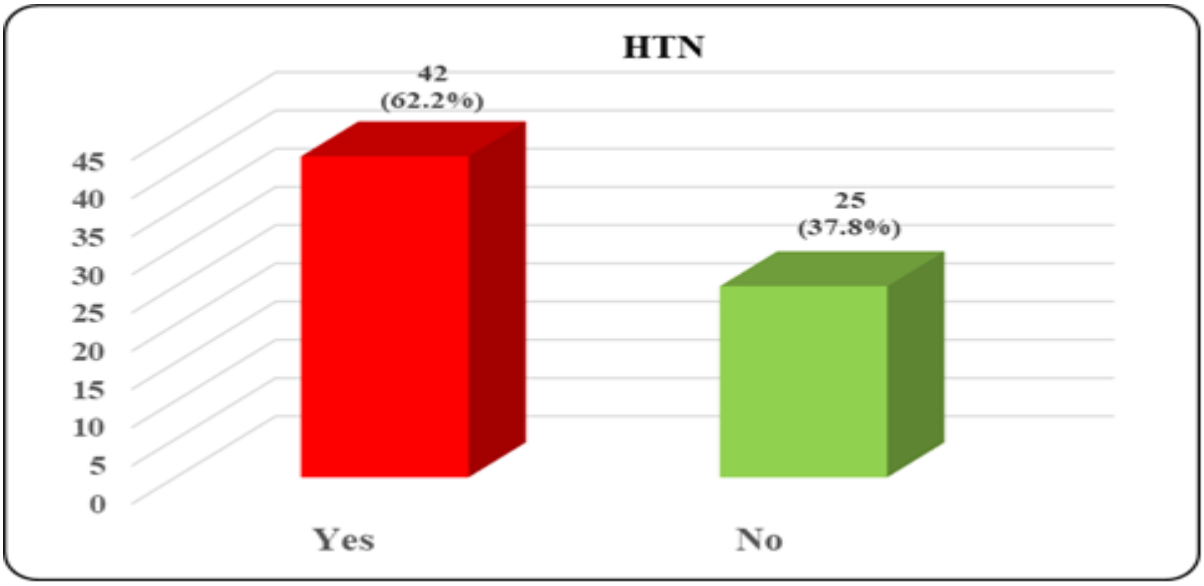

Table II: Distribution of the respondents by other variables stratified by cases and controls:

OTHER VARIABLES	Cases (n=67) Mean±SD	Controls (n=67) Mean±SD	*P* value
SBP (mmHg)	112.67±17.35	125.22±5.89	0.000
DBP (mmHg)	73.38±11.63	84.07±4.77	0.000

Figure 3: Distribution of the respondents by lipid profile in cases and controls:

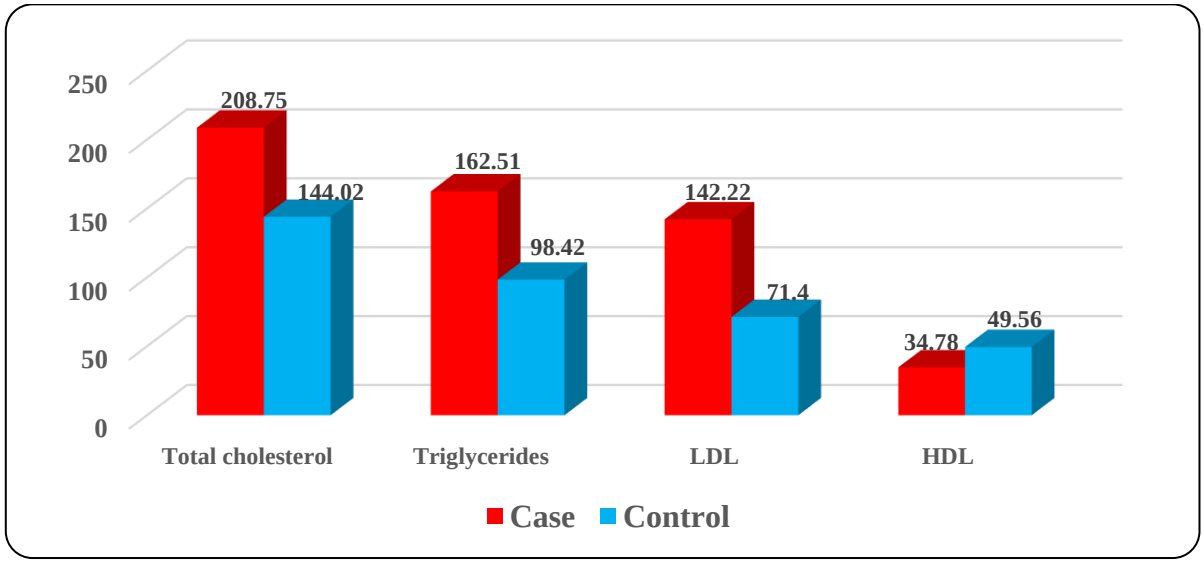

Table III: Distribution of the respondents by lipid profile in the cases:

TOTAL	TC>200mg/dl	TG>150mg/dl	LDL>100mg/dl	HDL<40mg/dl
67	39 (57.77%)	42 (62.2%)	58 (86.66%)	43 (64.44%)

DISCUSSION

In the present study, the mean±SD of age (63.9±12.11 years) was significantly higher in ACS patients compared with the control group (45.47±11.75 years) with $p<0.05$ (Table 1). The finding is supported by the study conducted by Zaky Doaa SE et al[3] where they found the mean±SD of age for the ACS cases was 56.28±10.66 years and for the controls it was 53.1±11.47 years. The elderly has the highest incidence of cardiovascular disease and frequently present with ACS. Elderly patients (>75 years of age)[9] constitute a large proportion of those patients presenting with acute coronary syndrome (ACS), and temporal trends in the incidence of myocardial infarction document a shift toward older adults.[10]

The present study (Figure-1) shows that number of males was 49 (73.3%) and of females was 18 (26.7%) in the ACS cases. Among the controls, 31 (46.7%) and 36 (53.3%) were males and females respectively. This is due to the fact that men are usually exposed to risk factors like smoking, high cholesterol and high blood pressure and they also occupy more stressful job roles. Moreover, there is higher myocardial blood flow in women compared to men.[11] The women benefit from the protective effects of endogenous estrogens, including estradiol, which may inhibit age-related vascular remodeling,[12] such as vascular smooth muscle cell proliferation and endothelial dysfunction. Various studies have reported more prevalence of ACS in males than in females which are similar to our findings. A study done by Zaky Doaa SE et al[3] also reported a higher prevalence of ACS in males (69.3%) than females (30.7%).

Dubey RK et al[13] in their study also found that men had a higher incidence of ACS than women (24.1% men & 17.0% women, p<0.001).

Figure 2 shows that 62.2% (42) of the cases had history of HTN and 37.8% (25) had no history of HTN. The findings were similar to the study conducted by Alberty R et al[14] where 83.5% (95% CI, 81.6-85.2%) of the patients with ACS had hypertension, 65.0% (62.5-67.2%) had a hyperlipidemic profile, 32.6% (30.3-34.9%) were diagnosed with diabetes, and 27.6% (25.1-29.8%) were smokers at the time of a heart-related event. Rashid MH et al[15] also found that the risk factors found in 160 cases recruited in their study were hypertension (101, 63%), lack of exercise (91, 57%), smoking (70, 44%), diabetes mellitus (61, 38%), dyslipidaemia (50, 31%). And the results were statistically significant ($p < 0.05$). The findings are also supported by the cross-sectional descriptive study conducted by Ralapanawa U et al.[16] They found that almost 51.8% NSTEMI patients, 47.8% UA patients and 29.9% STEMI patients had hypertension (P = 0.008) indicating significant association of HT with UA and NSTEMI.

As regards to blood pressure, systolic blood pressure and diastolic blood pressure were significantly lower in cases than the controls (SBP: 112.67±17.35 mmHg and 125.22±5.89 mmHg respectively; DBP: 73.38±11.63 mmHg and 84.07±4.77 mmHg respectively). This is because in most patients with right ventricular MI, the inferior wall of the left ventricle is involved (usually in the form of a STEMI) as a result of occlusion of the right coronary artery proximal to the right ventricular branch. The findings are supported by Owens P et al[17] where they found mean supine resting systolic/diastolic blood pressure was 131(24)/77(11) mmHg in coronary disease patients. Twenty patients exhibited a fall in systolic blood pressure on standing at five minutes (mean 12(14) mmHg, $p < 0.001$), while 13 patients showed a fall in diastolic blood pressure at five minutes' standing (mean 2(9) mmHg, p = NS).

As regards to the lipid profile in the study (Figure-3), it was found that the mean±SD of total cholesterol was 208.75±31.25 mg/dl in the cases and 144.02±19.57 mg/dl in the controls, triglycerides in cases was 162.51±39.81 mg/dl and 98.42±16.96 mg/dl in controls and LDL was 142.22±31.31 mg/dl in the cases and 71.40±21.24 md/dl in controls showing that the values were higher in the cases as compared to the control group and the difference were found to be statistically significant with $p<0.05$. However, the mean±SD of HDL in the cases 34.78±6.44 mg/dl was lower as compared to the controls (49.56±5.72 mg/dl) and the difference was statistically significant with $p<0.05$. In this study, it was observed that there were high TC, TG and LDL and low HDL. The underlying pathophysiology in ACS is decreased blood flow to part of heart musculature which is usually secondary to plaque rupture and formation of thrombus. Sometimes ACS can be secondary to vasospasm with or without underlying atherosclerosis. The result is decreased blood flow to a part of heart musculature resulting first in ischemia and then infarction of that part of the heart.[18] Rupture of an atherosclerotic plaque that results in partial or complete occlusion of an epicardial coronary artery is the most common mechanism responsible for ACS. Plaque disruption exposes subendothelial collagen, which results in activation of platelets and the coagulation cascade, leading to thrombus formation.[19] Reduction in blood flow due to coronary occlusion and/or distal embolization of thrombus into coronary microcirculation results in symptoms of ischemic heart disease. Thrombus may be completely or partially occlusive. Dyslipidaemia is an independent major risk factor for CAD. A combination of low HDL-C and high TG referred to as atherogenic dyslipidemia, have been implicated as important predictors of CAD.[20] The findings of this study are also supported by the study conducted by Zaky Doaa SE et al.[3] In this study, 39 (57.77%) patients were observed to have TC>200mg/dl, 42 (62.2%) had TG>150mg/dl, 58 (86.66%) had LDL>100mg/dl and 43 (64.44%) had HDL<40mg/dl (Table-VI). A study conducted by Penalva RA et al[21] where a total of 107 patients were included, they found 64 (59.8%)

patients had TC<200mg/dl, 33(30.8%) had HDL<40 mg/dl, and 38(35.5%) had LDL<100mg/dl. The imbalance between TC and HDL levels plays a more important role in the pathophysiology of atherogenesis. It is important to consider that the atheroprotective function of HDL is not restricted to reverse cholesterol transport, but can also transport antioxidant enzymes, break down oxidized lipid fractions, and neutralize their proinflammatory effects.[22]

LIMITATIONS OF THE STUDY

The study had some limitations that the sample size was relatively small and this was a single centre study done in Imphal. Hence, the results of this research need to be proved through multi-centric surveys with larger sample sizes.

CONCLUSION

The present study was carried out to evaluate the levels of lipid profile in acute coronary syndrome (ACS) subjects and normal healthy controls without ACS. The study shows the level of total cholesterol, triglycerides and LDL were higher in cases of ACS as compared to the healthy controls. However, the level of HDL was lower in cases of ACS. The findings were found to be significant. Many studies show dyslipidemia in ACS. Thus, it can be concluded that dyslipidemia is a risk factor for development of ACS.

FINANCIAL SUPPORT AND SPONSORSHIP

Nil.

CONFLICTS OF INTEREST

There are no conflicts of interest.

REFERENCES

1. Becker D, Merkely B. Acute coronary syndrome–2012. Orvosi hetilap. 2012 Dec 1;153(51):2009-15
2. Zaky Doaa SE, Mabrouk FM, Zaki ER, Hendy OM. The value of YKL-40 in ischemic heart disease patients. The Egyptian Journal of Internal Medicine. 2016 Jun;28(2):71-7.
3. Azeredo-Da-Silva AL, Perini S, Soares PH, Polaczyk CA. Systematic review of economic evaluations of units dedicated to acute coronary syndromes. Value Health. 2016 Mar 1;19(2):286-95.
4. Bracey A, Meyers HP. Posterior Myocardial Ischemia. InStatPearls [Internet]. StatPearls Publishing. 2021 Aug 6.
5. Feigin V. GBD 2017 Causes of Death Collaborators. Global, regional, and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980–2017: a systematic analysis for the Global Burden of Disease Study 2017. The Lancet. 2018;392(10159):1736-88.
6. Syed Zahed R, Shafiq A, Abdus S. Effect of statins on triglycerides in the management of hypercholesterolemia in patients with coronary heart disease.
7. Moon JC, Bogle RG, Minas R. Statins audit: wrong question, wrong conclusions. The Lancet. 2007 Feb 24;369(9562):640.
8. Khan HA, Alhomida AS, Sobki SH. Lipid profile of patients with acute myocardial infarction and its correlation with systemic inflammation. Biomarker insights. 2013 Jan;8:BMI-S11015.
9. Roger VL, Weston SA, Redfield MM, Hellermann-Homan JP, Killian J, Yawn BP, et al. Trends in myocardial infarction incidence and survival. Olmsted County, Minnesota–1979 to 1994. Ann Int Med. 2002;136(5):341-8.

10. Duan JG, Chen XY, Wang L, Lau A, Wong A, Thomas GN, et al. Sex differences in epidemiology and risk factors of acute coronary syndrome in Chinese patients with type 2 diabetes: a long-term prospective cohort study. PLoS One. 2015 Apr 1;10(4):e0122031.
11. Murthy VL, Naya M, Taqueti VR, Foster CR, Gaber M, Hainer J, Dorbala S, Blankstein R, Rimoldi O, Camici PG, Di Carli MF. Effects of sex on coronary microvascular dysfunction and cardiac outcomes. Circulation. 2014 Jun 17;129(24):2518-27.
12. Oommen AM, Abraham VJ, George K, Jose VJ. Prevalence of coronary heart disease in rural and urban Vellore: a repeat cross-sectional survey. Indian Heart J. 2016 Jul 1;68(4):473-9.
13. Dubey RK, Imthurn B, Barton M, Jackson EK. Vascular consequences of menopause and hormone therapy: importance of timing of treatment and type of estrogen. Cardiovascular research. 2005 May 1;66(2):295-306.
14. Alberty R, Studenčan M, Kovář F. Prevalence of conventional cardiovascular risk factors in patients with acute coronary syndromes in Slovakia. Cent Eur J Public Health. 2017 Mar 1;25(1):77-84.
15. Rashid MH, Yaseen G, Ghaffar U, Khan AA, Kabir A, Aisha A, et al. Prevalence of Acute Coronary Syndrome and Various Risk Factors in Acute Stroke Patients. Cureus. 2020 Aug 4;12(8).
16. Ralapanawa U, Kumarasiri PV, Jayawickreme KP, Kumarihamy P, Wijeratne Y, Ekanayake M, et al. Epidemiology and risk factors of patients with types of acute coronary syndrome presenting to a tertiary care hospital in Sri Lanka. BMC cardiovascular disorders. 2019 Dec;19(1):1-9.
17. Owens P, O'Brien E. Hypotension in patients with coronary disease: can profound hypotensive events cause myocardial ischaemic events?. Heart. 1999 Oct 1;82(4):477-81.
18. Singh A, Museedi AS, Grossman SA. Acute coronary syndrome. StatPearls Publishing, Treasure Island (FL). 2017 Oct 31.
19. Naghavi M, Libby P, Falk E, Casscells SW, Litovsky S, Rumberger J, et al. From vulnerable plaque to vulnerable patient: a call for new definitions and risk assessment strategies: Part I. Circulation. 2003 Oct 7;108(14):1664-72.
20. Khot UN, Khot MB, Bajzer CT, Sapp SK, Ohman EM, Brener SJ, et al. Prevalence of conventional risk factors in patients with coronary heart disease. Jama. 2003 Aug 20;290(7):898-904.
21. Penalva RA, Huoya MD, Correia LC, Feitosa GS, Ladeia AM. Lipid profile and intensity of atherosclerosis disease in acute coronary syndrome. Arquivos brasileiros de cardiologia. 2008;90:24-30.
22. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002 Mar 5;105(9):1135-43.