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Original Research Article

Study of Lipid Profile and Apolipoprotein in Patients Diagnosed with the Coronary Heart Disease

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Abstract

The cause of CHD is atherosclerosis. Atherosclerosis is a chronic inflammatory response of the arterial wall initiated by an injury to the endothelium by activation of various factors for which apolipoproteins have been implicated. Moreover, lesion progression is sustained by interaction between modified lipoproteins (e.g. oxidized low density lipoprotein), lipid laden macrophage (foam cells), T-lymphocytes and the normal cellular constituents of the arterial wall. Atherosclerosis is also characterized by thickening of the arterial wall, which protrudes into and obstructs the vascular lumen. Hence based on above findings the present study was planned for Assessment of Apolipoprotein and Lipid Profile in Patients Diagnosed with the Coronary Heart Disease.

The present study was planned in Department of Biochemistry, Shri Ramkrishna institute of medical sciences and Sanaka Hospitals, Durgapur, West Bengal, India for 1 year . The 20 cases were enrolled in Group A as cases of coronary heart disease and 20 cases were enrolled in Group B as control cases for comparative study. Estimation of serum Apo A-I and Apo B were done by Turbidimetric Immunoassay, serum cholesterol by cholesterol oxidase- peroxidase (CHOD-PAP) enzymatic colorimetric end point method, HDL cholesterol and LDL cholesterol by direct enzymatic method and serum triglycerides by glycerol phosphate oxidase peroxidase (GPO-PAP) enzymatic end point method.

The data generated from the present study concludes that the levels of apo A1 and apo B are strongly related to CHD in addition to the conventional lipid profile. Our findings support the consideration of the measurement of serum Lipo-A as a screening tool for the risk of ischemic heart disease. Therefore, this study suggests the need for routine measurement of apo A1 and apo B in the diagnosis of CHD and thus helps in early detection of myocardial damage which warrants timely intervention leading to lowered morbidity and mortality.

Keywords: Apolipoprotein, Lipid Profile, Coronary Heart Disease, etc.

Introduction

Coronary artery disease (CAD), also known as coronary heart disease (CHD) or ischemic heart disease (IHD), involves the reduction of blood flow to the heart muscle due to build-up of plaque in the arteries of the heart. It is the most common of the cardiovascular diseases. Types include stable angina, unstable angina, myocardial infarction, and sudden cardiac death. A common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw. Occasionally it may feel like heartburn. Usually symptoms occur with exercise or emotional stress, last less than a few minutes, and improve with rest. Shortness of breath may also occur and sometimes no symptoms are present. In many cases, the first sign is a heart attack.[5] Other complications include heart failure or an abnormal heartbeat.[1]

Risk factors include high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, depression, and excessive alcohol. A number of tests may help with diagnoses including: electrocardiogram, cardiac stress testing, coronary computed tomographic angiography, and coronary angiogram, among others.

Ways to reduce CAD risk include eating a healthy diet, regularly exercising, maintaining a healthy weight, and not smoking. Medications for diabetes, high cholesterol, or high blood pressure are sometimes used. There is limited evidence for screening people who are at low risk and do not have symptoms. Treatment involves the same measures as prevention. Additional medications such as antiplatelets (including aspirin), beta blockers, or nitro-glycerine may be recommended. Procedures such as percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG) may be used in severe disease. In those with stable CAD it is unclear if PCI or CABG in addition to the other treatments improves life expectancy or decreases heart attack risk. [2]

In 2015, CAD affected 110 million people and resulted in 8.9 million deaths. It makes up 15.6% of all deaths, making it the most common cause of death globally. The risk of death from CAD for a given age decreased between 1980 and 2010, especially in developed countries. The number of cases of CAD for a given age also decreased between 1990 and 2010. In the United States in 2010, about 20% of those over 65 had CAD, while it was present in 7% of those 45 to 64, and 1.3% of those 18 to 45; rates were higher among men than women of a given age. [3]

Chest pain that occurs regularly with activity, after eating, or at other predictable times is termed stable angina and is associated with narrowings of the arteries of the heart. Angina that changes in intensity, character or frequency is termed unstable. Unstable angina may precede myocardial infarction. In adults who go to the emergency department with an unclear cause of pain, about 30% have pain due to coronary artery disease. [4]

Coronary artery disease has a number of well determined risk factors. These include high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, depression, family history, and excessive alcohol. About half of cases are linked to genetics. Smoking and obesity are associated with about 36% and 20% of cases, respectively. Smoking just one cigarette per day about doubles the risk of CAD. Lack of exercise has been linked to 7–12% of cases. Exposure to the herbicide Agent Orange may increase risk. Rheumatologic diseases such as rheumatoid arthritis, systemic lupus erythematosus, psoriasis, and psoriatic arthritis are independent risk factors as well. [5]

Job stress appears to play a minor role accounting for about 3% of cases. In one study, women who were free of stress from work life saw an increase in the diameter of their blood vessels, leading to decreased progression of atherosclerosis. In contrast, women who had high levels of work-related stress experienced a decrease in the diameter of their blood vessels and significantly increased disease progression. Having a type A behavior pattern, a group of personality characteristics including time urgency, competitiveness, hostility, and impatience, [3,6] is linked to an increased risk of coronary disease. [6]

Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the heart's muscle cells. The heart's muscle cells may die from lack of oxygen and this is called a myocardial infarction (commonly referred to as a heart attack). It leads to damage, death, and eventual scarring of the heart muscle without regrowth of heart muscle cells. Chronic high-grade narrowing of the coronary arteries can induce transient ischemia which leads to the induction of a ventricular arrhythmia, which may terminate into a dangerous heart rhythm known as ventricular fibrillation, which often leads to death. [7]

Typically, coronary artery disease occurs when part of the smooth, elastic lining inside a coronary artery (the arteries that supply blood to the heart muscle) develops atherosclerosis. With atherosclerosis, the artery's lining becomes hardened, stiffened, and accumulates deposits of calcium, fatty lipids, and abnormal inflammatory cells – to form a plaque. Calcium phosphate (hydroxyapatite) deposits in the muscular layer of the blood vessels appear to play a significant role in stiffening the arteries and inducing the early phase of coronary arteriosclerosis. This can be seen in a so-called metastatic mechanism of calciphylaxis as it occurs in chronic kidney disease and hemodialysis (Rainer Liedtke 2008). Although these people suffer from a kidney dysfunction, almost fifty percent of them die due to coronary artery disease. Plaques can be thought of as large "pimples" that protrude into the channel of an artery, causing a partial obstruction to blood flow. People with coronary artery disease might have just one or two plaques, or might have dozens distributed throughout their coronary arteries. A more severe form is chronic total occlusion (CTO) when a coronary artery is completely obstructed for more than 3 months. [8]

Cardiac syndrome X is chest pain (angina pectoris) and chest discomfort in people who do not show signs of blockages in the larger coronary arteries of their hearts when an angiogram (coronary angiogram) is being performed. The exact cause of cardiac syndrome X is unknown. Explanations include microvascular dysfunction or epicardial atherosclerosis.[5,6] For reasons that are not well understood, women are more likely than men to have it; however, hormones and other risk factors unique to women may play a role.

For symptomatic people, stress echocardiography can be used to make a diagnosis for obstructive coronary artery disease. The use of echocardiography, stress cardiac imaging, and/or advanced non-invasive imaging is not recommended on individuals who are exhibiting no symptoms and are otherwise at low risk for developing coronary disease. [9]

In "stable" angina, chest pain with typical features occurring at predictable levels of exertion, various forms of cardiac stress tests may be used to induce both symptoms and detect changes by way of electrocardiography (using an ECG), echocardiography (using ultrasound of the heart) or scintigraphy (using uptake of radionuclide by the heart muscle). If part of the heart seems to receive an insufficient blood supply, coronary

angiography may be used to identify stenosis of the coronary arteries and suitability for angioplasty or bypass surgery. [10]

Diagnosis of acute coronary syndrome generally takes place in the emergency department, where ECGs may be performed sequentially to identify "evolving changes" (indicating ongoing damage to the heart muscle). Diagnosis is clear-cut if ECGs show elevation of the "ST segment", which in the context of severe typical chest pain is strongly indicative of an acute myocardial infarction (MI); this is termed a STEMI (ST-elevation MI) and is treated as an emergency with either urgent coronary angiography and percutaneous coronary intervention (angioplasty with or without stent insertion) or with thrombolysis ("clot buster" medication), whichever is available. In the absence of ST-segment elevation, heart damage is detected by cardiac markers (blood tests that identify heart muscle damage). If there is evidence of damage (infarction), the chest pain is attributed to a "non-ST elevation MI" (NSTEMI). If there is no evidence of damage, the term "unstable angina" is used. This process usually necessitates hospital admission and close observation on a coronary care unit for possible complications (such as cardiac arrhythmias – irregularities in the heart rate). Depending on the risk assessment, stress testing or angiography may be used to identify and treat coronary artery disease in patients who have had an NSTEMI or unstable angina.

Up to 90% of cardiovascular disease may be preventable if established risk factors are avoided.[6,5] Prevention involves adequate physical exercise, decreasing obesity, treating high blood pressure, eating a healthy diet, decreasing cholesterol levels, and stopping smoking. Medications and exercise are roughly equally effective High levels of physical activity reduce the risk of coronary artery disease by about 25%.

Most guidelines recommend combining these preventive strategies. A 2015 Cochrane Review found some evidence that counselling and education in an effort to bring about behavioral change might help in high risk groups. However, there was insufficient evidence to show an effect on mortality or actual cardiovascular events. [11]

In diabetes mellitus, there is little evidence that very tight blood sugar control improves cardiac risk although improved sugar control appears to decrease other problems such as kidney failure and blindness. The World Health Organization (WHO) recommends "low to moderate alcohol intake" to reduce risk of coronary artery disease while high intake increases the risk. [12]

A diet high in fruits and vegetables decreases the risk of cardiovascular disease and death. Vegetarians have a lower risk of heart disease, possibly due to their greater consumption of fruits and vegetables. Evidence also suggests that the Mediterranean diet and a high fiber diet lower the risk. The consumption of trans fat (commonly found in hydrogenated products such as margarine) has been shown to cause a precursor to atherosclerosis and increase the risk of coronary artery disease. [13]

Evidence does not support a beneficial role for omega-3 fatty acid supplementation in preventing cardiovascular disease (including myocardial infarction and sudden cardiac death).[7,8] There is tentative evidence that intake of menaquinone (Vitamin K2), but not phylloquinone (Vitamin K1), may reduce the risk of CAD mortality.

Aerobic exercise, like walking, jogging, or swimming, can reduce the risk of mortality from coronary artery disease. Aerobic exercise can help decrease blood pressure and the amount of blood cholesterol (LDL) over time. It also increases HDL cholesterol which is considered "good cholesterol".

Although exercise is beneficial, it is unclear whether doctors should spend time counseling patients to exercise. The U.S. Preventive Services Task Force found "insufficient evidence" to recommend that doctors counsel patients on exercise but "it did not review the evidence for the effectiveness of physical activity to reduce chronic disease, morbidity and mortality", only the effectiveness of counseling itself.[8,7] The American Heart Association, based on a non-systematic review, recommends that doctors counsel patients on exercise. Psychological symptoms are common in people with CHD, and while many psychological treatments may be offered following cardiac events, there is no evidence that they change mortality, the risk of revascularization procedures, or the rate of non-fatal myocardial infarction.

Coronary heart disease (CHD) is the thinning or blockage of the coronary arteries, usually caused by atherosclerosis. Atherosclerosis (sometimes called “hardening” or “clogging” of the arteries) is the build-up of cholesterol and fatty deposits (called plaques) on the inner walls of the arteries. These deposits can restrict blood flow to the heart muscle by clogging the artery. This result in reduced supply of blood to the heart, thus heart becomes starved of oxygen and the vital nutrients it requires to work properly. This can lead to chest pain called angina. If blood supply to a portion of the heart muscle is cut off entirely, or if the energy demands of the heart become much greater than its blood supply, a heart attack (injury to the heart muscle) may occur. Coronary heart disease (CHD) is the leading cause of death for both men and women living in urban areas as compared to rural India.

The cause of CHD is atherosclerosis. Atherosclerosis is a chronic inflammatory response of the arterial wall initiated by an injury to the endothelium by activation of various factors for which apolipoproteins have been implicated. Moreover, lesion progression is sustained by interaction between modified lipoproteins (e.g. oxidized low density lipoprotein), lipid laden macrophage (foam cells), T-lymphocytes and the normal cellular constituents of the arterial wall. Atherosclerosis is also characterized by thickening of the arterial wall, which protrudes into and obstructs the vascular lumen. Hence based on above findings the present study was planned for Assessment of Apolipoprotein and Lipid Profile in Patients Diagnosed with the Coronary Heart Disease.

Methodology:

The present study was planned in Department of Biochemistry, Shri Ramkrishna institute of medical sciences and Sanaka Hospitals, Durgapur, West Bengal, India for 1 year. The 20 cases were enrolled in Group A as cases of coronary heart disease and 20 cases were enrolled in Group B as control cases for comparative study. Estimation of serum Apo A-I and Apo B were done by Turbidimetric Immunoassay, serum cholesterol by cholesterol oxidase- peroxidase (CHOD-PAP) enzymatic colorimetric end point method, HDL cholesterol and LDL cholesterol by direct enzymatic method and serum triglycerides by glycerol phosphate oxidase peroxidase (GPO-PAP) enzymatic end point method.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: cases of coronary heart disease.

Exclusion Criteria: Patients with coronary artery disease with atrial fibrillation or pacemaker. Patients with history of stroke, intermittent claudication, peripheral vascular disease, carotid surgery, coronary artery

bypasses graft surgery or PTCA. Patient having H/O chronic alcohol consumption, hepatobiliary disorders or any other acute liver diseases and diabetes mellitus.

Results & Discussion:

CHD is one of the important causes of morbidity and mortality in most countries of the world. The debate on the value of lipids as a predictive risk factor for atherogenesis has centered for many years on TC, TGL and LDL. Recently the interest has been focused on role of apolipoprotein and inflammatory markers in atherogenesis.

Abnormalities in plasma lipoproteins and derangements in lipid metabolism are one of the best established risk-factors for atherosclerosis and coronary artery disease (CAD). Studies have shown that apolipoprotein A1 (ApoA1): Apolipoprotein B (ApoB) ratio distinguishes unequivocally between patients with and without CAD. [14] Role of other lipid parameters like lipoprotein a (Lpa) in causation of atherosclerosis and CAD is yet to be established clearly. It is a known fact that CAD in Indians occurs at levels of dyslipidemia much lower than western populations and many Indian patients with coronary artery have lipid parameters that are much lower then their western counterparts. Asian Indians tend to have higher levels of triglycerides, lower high-density lipoprotein (HDL) levels and higher Lpa. [15] The role of studying non-traditional lipid risk factors like ApoA1: ApoB ratio and Lpa in Indian CAD patients especially in patients with normal lipid parameters is not clear. [16]

Table 1: Lipid Profile & Apolipoprotein Levels

Group	**Group A**	**Group B**
Cases of	**Coronary heart disease**	**Control Cases**
No. of Cases	**20**	**20**
Total Cholesterol (mg/dl)	179.5 ± 33.4	168.2 ± 19.4
HDL Cholesterol (mg/dl)	44.5 ± 7.3	48.1 ± 5.8
LDL Cholesterol (mg/dl)	106.3 ± 23.8	97.3 ± 14.7
VLDL Cholesterol (mg/dl)	26.7 ± 11.6	18.5 ± 6.7
Triglyceride (mg/dl)	137.6 ± 34.6	113.7 ± 22.1
Apolipoprotein A-1 (mg/dl)	85.6 ± 13.6	131.4 ± 13.2
Apolipoprotein B (mg/dl)	138.4 ± 18.3	105.8 ± 15.4

There is a positive correlation between HDL-C and apo A1. Both of the variables are dependent on each other. As the concentrations of HDL-C decreases, there is a simultaneous decrease in apo A1 level thus suggesting that this correlation is statistically significant. There is a negative correlation between LDL-C and apo A1 and it is also statistically significant. Although the inverse relationship between HDL cholesterol concentrations and risk of future CHD is well recognized and HDL cholesterol has been incorporated into various algorithms for calculating CVD risk, several studies have recently demonstrated that apoA-1 might provide almost identical prognostic information as HDL, but several studies have suggested that apoA-I might even improve our ability to identify patients at risk for future CHD. [17]

A study by Gupta et al at Monilek Hospital and Research Centre, Jaipur, found out that total cholesterol levels were not significantly higher in CAD patients compared to healthy age-matched controls. [18]

Another study at Louisiana State University Medical College Center, New Orleans,

evaluating risk factors for CAD and levels of Lpa in Asian Indians of USA found average total cholesterol to be 218.9 ± 39.0 mg%. [19]

A study by Austin et al recently described atherogenic lipoprotein phenotype B characterized by moderate hypertriglyceridemia, a high proportion of small dense LDL, a high level of ApoA1 and HDL. It can inherited as a single gene trait. Atherogenic phenotype B can be differentiated from benign phenotype A by simple measurements of serum triglycerides and HDL.[20]

Altered serum lipid and lipoprotein levels are considered as independent modifiable risk factors for hypertension which can be corrected by diet, drugs and exercise. The untreated hypertensives are more prone for dyslipidemia than normotensives and studies reveal that there is an increase in blood pressure with rise in lipid level. Studies by Osuji et al., reveal that hypertensives do not have specific pattern of dyslipidemia and total cholesterol, triglycerides and all fractions of lipoproteins tend to be more frequently abnormal in the hypertensives than the general population [9]. Recent studies suggest that plasma concentration of Apo A1 and Apo B100 and their ratio are more sensitive and specific biochemical markers for the risk of coronary heart disease than the conventional lipid and lipoprotein measurement. [21-22]

Hypercholesterolaemia and hypertriglyceridaemia are considered the independent risk factors but most of the earlier studies in this area have considered only the fasting lipids and lipoproteins. Recently it has been proposed that postprandial lipoproteins may be better indicators of deranged lipoprotein metabolism and hence of atherosclerosis and CHD. Postprandial hypertriglyceridaemia (PHTG) and delayed triglyceride (TG) rich lipoprotein clearance have been found to impair endothelial function significantly either directly or by increasing superoxide anions. As these lipoproteins are rich in cholesterol as well as triglyceride content, their uptake by macrophages can result in formation of cholesterol laden foam cells. It has also been reported that magnitude and duration of postprandial lipidaemia is positively related to the pathogenesis and progression of CHD. [23]

Dyslipidemia is a primary, widely established as an independent major risk factor for coronary artery disease (CAD) and may even be a prerequisite for CAD, occurring before other major risk factors come into play. [24] Studies have reported higher prevalence of lipid abnormalities among Asians compared with non-Asians. [25] Low HDL cholesterol and high TG concentrations have been implicated as possible independent predictors of CVD and the combination of these two conditions are called as atherogenic dyslipidemia. [26] Asian Indian have a higher prevalence of low HDL cholesterol and lower prevalence of high cholesterol than non-Asian Indians, [27] which suggests impaired reverse cholesterol transport. These findings suggest the importance of high TG and low HDL cholesterol in Asian Indians compared with high cholesterol, which is more prevalent in western countries, which may have therapeutic implications. [28]

Conclusion:

The data generated from the present study concludes that the levels of apo A1 and apo B are strongly related to CHD in addition to the conventional lipid profile. Our findings support the consideration of the measurement of serum Lipo-A as a screening tool for the risk of ischemic heart disease. Therefore, this study suggests the need for routine measurement of apo A1 and apo B in the diagnosis of CHD and thus helps in early detection of myocardial damage which warrants timely intervention leading to lowered morbidity and mortality.

References:

1. Coronary Artery Disease (CAD). 12 March 2013. Archived from the original on 2 March 2015. Retrieved 23 February 2015.
2. Rezende PC, Scudeler TL, da Costa LM, Hueb W. Conservative strategy for treatment of stable coronary artery disease. World Journal of Clinical Cases. February 2015; 3(2): 163–70.
3. Centers for Disease Control Prevention (CDC). Prevalence of coronary heart disease- United States, 2006-2010. MMWR. Morbidity and Mortality Weekly Report. October 2011; 60(40): 1377–81.
4. Kontos MC, Diercks DB, Kirk JD. Emergency department and office-based evaluation of patients with chest pain. Mayo Clinic Proceedings. March 2010; 85(3): 284–99.
5. Esdaile JM, Abrahamowicz M, Grodzicky T, Li Y, Panaritis C, du Berger R, et al. Traditional Framingham risk factors fail to fully account for accelerated atherosclerosis in systemic lupus erythematosus. Arthritis and Rheumatism. October 2001; 44(10): 2331–7.
6. McCann S.J.H. The precocity-longevity hypothesis: earlier peaks in career achievement predict shorter lives. Pers Soc Psychol Bull. November 2001; 27(11): 1429–39.
7. Rhodewalt, Smith. Current issues in Type A behaviour, coronary proneness, and coronary heart disease. In Snyder, C.R.; Forsyth, D.R. (eds.). Handbook of social and clinical psychology: the health perspective. New York: Pergamon. 1991;197–220.
8. Ambrose JA, Singh M. Pathophysiology of coronary artery disease leading to acute coronary syndromes. F1000prime Reports. 2015;7:08.
9. Aziz S, Ramsdale DR. Chronic total occlusions- a stiff challenge requiring a major breakthrough: is there light at the end of the tunnel? Heart. June 2005; 91 Suppl 3 (suppl_3): iii; 428.
10. 10. American Society of Echocardiography. Five Things Physicians and Patients Should Question. Choosing Wisely: An Initiative of the ABIM Foundation. Archived from the original on 26 February 2013. Retrieved 27 February 2013.
11. Coronary Angiography. National Heart, Blood, and Lung Institute. Retrieved 10 December 2017.
12. Ebrahim S, Taylor F, Ward K, Beswick A, Burke M, Davey Smith G. Multiple risk factor interventions for primary prevention of coronary heart disease. The Cochrane Database of Systematic Reviews. January 2011; (1): CD001561.
13. Population nutrient intake goals for preventing diet-related chronic diseases. WHO. Archived from the original on 29 December 2015. Retrieved 26 October 2015.
14. Wang X, Ouyang Y, Liu J, Zhu M, Zhao G, Bao W, Hu FB. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. BMJ. July 2014; 349: 4490.
15. Stamler J, Wentworth D, Neaton JD. Is the relationship between serum cholesterol and risk of premature death from coronary heart disease continuously graded? Findings in 356, 222 primary screenees of the Multiple Risk Factor Intervention Trial (MRFIT). JAMA. 1986; 256(20): 2823-8.
16. Gupta R, Gupta VP, Sarna M, Bhatnagar AS, Thanvi J, Sharma V, et al. Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch. Indian Heart J. 2002; 54(1):59-66.

17. Enas EA, Jacob S. Coronary artery disease in Indians in the USA. In: Coronary Artery Disease in Indians: A Global Perspective. Sethik, (Ed.), Cardiological Society of India; Mumbai. 1998:32-43.
18. Walldius G, Jungner I: The apoB/apoA-I ratio: a strong, new risk factor for cardiovascular disease and a target for lipidlowering therapy — a review of the evidence. J Intern Med. 2006; 259:493–519.
19. Gupta R, Kaul V. Prakash H, Saran M, Gupta VP. Lipid abnormalities in coronary heart disease: a populationbased case-control studies. Indian Heart J. 2001; 52(3): 332-6
20. Enas A, Yusuf S, Mehta JL. Prevalence of coronary artery disease in Asian Indians. Am J Cardiol. 1992;70(9): 945-9.
21. McKeigue PM, Miller GJ, Marmot MG. Coronary heart disease in south Asian's: a review. J Clin Epidemiol. 1989;42(7):597-609.
22. Naito HK. Apolipoproteins: Biochemistry, metabolism and clinical significance developed by lipid and lipoprotien division. Clin Chem. 1987;33:1051-52.
23. Stein EA. Lipids. In: Text Book of Clinical. Chemistry. Ed Teitz NB. London, WB. Saunders Co. 1986; 829-900.
24. Bae JH, Schwemmer M, Lee IK, et al. Postprandial hypertriglyceridemia induced endothelial dysfunction in healthy subjects is independent of lipid oxidation. Int J Cardiol. 2003; 87: 259-67.
25. Genest JG Jr. Dyslipidemia and coronary artery disease. Can J Cardiol. 2000;16 Suppl A:3A-4.
26. Karthikeyan G, Teo KK, Islam S, McQueen MJ, Pais P, Wang X, et al. Lipid profile, plasma apolipoproteins, and risk of a first myocardial infarction among Asians: An analysis from the interheart Study. J Am Coll Cardiol. 2009;53:244-53.
27. Fruchart JC, Sacks F, Hermans MP, Assmann G, Brown WV, Ceska R, et al. The residual risk reduction initiative: A call to action to reduce residual vascular risk in patients with dyslipidemia. Am J Cardiol. 2008;102:1K-34.
28. McKeigue PM, Miller GJ, Marmot MG. Coronary heart disease in south Asians overseas: A review. J Clin Epidemiol. 1989; 42:597-609.
29. Batool D. R., Jamal D. K., Sheroze D. M. W., Bhatti D. I. A., Jaffar D. N., Haider, D. G., & Faridi D. M. A. Clinical features of colorectal carcinoma at the Jinnah Postgraduate Medical Centre, Karachi, Pakistan: a cross-sectional study. Journal of Medical Research and Health Sciences. 2022; 5(9): 2203–2209.