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Comparison of the Efficacy of Atorvastatin and Rosuvastatin in Patients Diagnosed as Coronary Artery Disease [CAD]: An Observational Prospective Study

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A B S T R A C T

Aim and objective of the study is to compare the Efficacy of Atorvastatin and Rosuvastatin in CAD patients based on Lipid profile and to enhance the compliance of patients. It is a prospective comparative study conducted between July 2018-January 2019. A Total of 124 patient's lipid profile data case forms of age 25 years and above were collected and two follow ups were performed with a time gap of 45 days for each follow-up and efficacy of Atorvastatin-40mg and rosuvastatin-20mg was compared. Among the total no. of cases collected the lipid profile parameters were analysed for each individual patient. The efficacy of Atorvastatin-40mg was compared with Rosuvastatin 20mg and the mean differences of both the drugs were calculated. mean differences for both drugs were calculated. The mean differences of LDL cholesterol for Atorvastatin were 64.469 and Rosuvastatin was 72.683. The Mean differences of HDL Cholesterol for Atorvastatin was 22.172 and for Rosuvastatin was 32.950. Triglycerides mean difference for Atorvastatin was 124.172 and Rosuvastatin was 198.100. Total Cholesterol mean differences was 48.125 for Atorvastatin and for Rosuvastatin was 59.471. Our study concludes that the drug compliance was seen in both the patients using Atorvastatin 40mg and Rosuvastatin 20mg.But based on the mean differences and P-Valves calculated for the lipid profile parameters of each individual patient, Rosuvastatin 20mg has more efficacies with fewer side effects when compared to Atorvastatin40mg.

INTRODUCTION

Coronary artery disease (CAD) develops due to the accumulation of plaque on walls of arteries of heart leading to decreased blood and oxygen supply to heart. Complete block of arteries may cause heart attack [1]. The prevalence of CAD has increased in India as shown in many studies conducted in rural and urban India over the last 30 years. The National Statistical Survey Organisation (NSSO) survey is the largest recent study on the prevalence of CAD in India. In its 60th NSSO survey (2004–2005), a total of 390 913 subjects were evaluated. The prevalence of CAD was found to be 7% in urban and 3% in the rural population [2].

Epidemiology

According to reports from the National Commission on Macroeconomics and Health, 62 million people in India will have CAD 2015, with 23 million of these below 40 years of age. The incidence of CAD is likely to increase further because of rapid urbanisation and its accompanying lifestyle changes, including changes in diet, physical inactivity, drug and alcohol intake, as well as an increase in the prevalence of DM [3].

Causes

- Genetic predisposition
- Age
- Gender
- Atherosclerosis
- Limited blood flow to heart
- Familial hypercholesterolemia
- Tangier disease

Sign and symptoms

- Chest pain radiates to shoulders, arms, jaw.
- Chest tightness
- Nausea
- Upper body exertion
- Numbness.

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Pathophysiology of CAD

They include following -

Pathogenesis of thrombus formation

Two causes of coronary thrombosis are rupture of plaque and erosion of endothelium. Pro-thrombotic material is exposed from core of plaque. Plaque consists of phospholipids, tissue factor, platelet adhesive matrix molecules [4].

Degree of plaque disruption

Vascular injury results in platelet adhesion to the damaged wall [5]. Rupture occur when fibrous cap is thin .Active immune cells are present abundantly in this region. Inflammatory molecules, proteolytic enzymes activate cells. Platelet deposition takes place resulting in thrombosis formation [4]. Thrombus dislodge through blood flow. Fibrillar collagen promotes formation of the thrombus resulting in activation of clotting system. Transisent thrombus formation results in sign and symptoms like chest. Thrombaxane A2, serotonin present at the place of platelet aggregation results in vasoconstriction and proliferative changes. Deep Plaque disruption exposes tissue factor and other elements of the vessel leading to thrombus occlusion [5].

Degree of stenosis

Several metabolic factors contribute to lipid deposition in arteries of heart, inflammatory mediators like cytokines, tissue necrosis factor, and interleukin result in inflammation cause lipid peroxidation [4]. Platelet aggregation increases with increased stenosis. Narrowing of arteries occur resulting in decreased blood flow and oxygen supply to the heart [5].

Residual thrombus formation

Residual thrombus intrudes in lumen of vessel resulting in narrowing of vessels. Activation and deposition of platelets occurs. Platelet deposition is increased two to four fold on residual thrombus formation [5].

Diagnosis

CAD is diagnosed on past history of MI , ECG changes like ST segment depression, Q-wave and T-wave changes [6].

Treatment

Treatment of CAD include both medical and surgical depending on severity, extent of the disease [7]. Treatment of CAD includes Lipids lowering drugs (statins), Antihypertensive drugs, Anti-platelet drugs [8]. Statins reduce the synthesis of cholesterol and improve the catabolism of LDL. Rhabdomyolysis are rare (less than 0.1 percent), myalgias are a common adverse effect of statin therapy [8]. Antihypertensive drugs decrease myocardial oxygen demand, due to atherosclerosis, oxygen demand is increased, and left ventricular ejection fraction is decreased thereby preventing left ventricular hypertrophy. Beta blockers are primary used as anti-hypertensives in patients with CAD. They block β1 and β2 adrenergic receptors, decreasing heart rate and cardiac contractility thereby increasing diastolic filling time. Cardioselective beta blockers (alter only β1 receptors) are preferred because they decrease adverse effects like bronchoconstriction. Betablockers with intrinsic sympathomimetic activity that increase sympathetic activity especially at rest and heart rate may not be decreased so they should be avoided. ACE inhibitors prevents formation of angiotensin I to angiotensinII , thereby reducing vasoconstriction, peripheral resistance, blood pressure. They also prevent ventricular dilation. Angiotensin receptor blockers used as alternative to ACE inhibitors. They block AngiotensinII receptors, preventing vasoconstriction. They decrease release aldosterone. Combination of ACE inhibitor and ARB causes severe renal adverse effects like Kidney failure than using ACE or ARB inhibitor alone. Calcium channel blockers used as alternative if beta blockers are not tolerated. Long acting calcium channel blockers has beneficial effect in CAD patients [8]. Antiplatelet therapy is important in management of CAD, because platelet aggregation cause MI. Most commonly used antiplatelet drugs are aspirin and clopidogrel. Aspirin is used as secondary drug in CAD management .Aspirin inhibits COX 1,2 thereby decreasing prostaglandin production and prevent platelet aggregation [8].

Surgery:

Angioplasty: Coronary arteries that are blocked can be fixed by use of angioplasty. A Catheter is placed into artery whether in wrist or groin (femoral vein) and sent to heart with angiogram. A small balloon present at end of catheter is inflated for a period of time, push plaque back against wall of coronary artery. Blood flow through arteries is enhanced. Stent or metal mesh tube small in size is placed over balloon. When balloon is exhausted and removed, stent present permanently at site of blockage, reducing the risk of narrowing of artery again [9].

METHODOLOGY

Aim of the study: Our main aim of the study is to compare the efficacy of atorvastatin and rosuvastatin in patients diagnosed as Coronary Artery disease [CAD] and counsel the patients on usage of medications in Cardiology department at Santhiram General Hospital Nandyal.

Study design: It is a prospective comparative study which includes patients diagnosed with CAD from Cardiology department of Santhiram medical college and general hospital to compare the efficacy of statins, in patients with CAD in a tertiary care teaching hospital.

Study site: Santhiram medical college and general hospital, Nandyal.

Study duration: 7 months

Study population: Patients of age 25 years and older diagnosed with CAD admitted/or present for consultation in the Cardiology department of SRGH & SRMC, NANDYAL were enrolled during the study period.

Sample size: Approx.150

Study criteria

a) Inclusion criteria: All patients diagnosed with CAD admitted/ or present for consultation in the Cardiology department of SRGH & SRMC, NANDYAL were enrolled during the study period**.**

- Subjects diagnosed as CAD
- Patients of age of 25 years and older
- Patients willing to join the study
- Gender- Both Female, Male

b) Exclusion criteria:

- Participants unwilling to join the study
- Age below 25 years

Study procedure

- **Patient consent:** study details were explained to the patients and written consent form was obtained.
- **Development of patient data collection form:** Patient data collection form was developed based on essential details to be collected from the patient. Demographic information like name, age, sex and locality were included. Parameters like description of pain, onset of pain, duration of pain, intensity of pain were included. Diagnosis and treatment protocol were included.
- **Source of data:** Patient data relevant to the study was obtained from following sources Patient data collection form, Case sheets, Prescriptions.

RESULTS

Age wise distribution of subjects in the study population is given in the table 01. Gender wise distribution of subjects in study population is represented in Fig 01. Patient demographic details & health habits were given in table 02. LDL ranges of subjects in the study population, HDL ranges of subjects in the study population, Triglycerides ranges of subjects in the study population, Total cholesterol ranges of subjects in the study population were represented in Fig 02, Fig 03, Fig 04 & Fig 05 respectively..

Table 1: Age wise distribution of subjects.

Age in years	Total no. of subjects (N=124)	Percentage (%)
25-35	9	7.25
35-45	42	33.87
45-55	54	43.54
> 55 and above	19	15.32

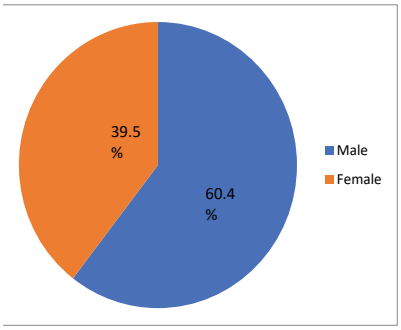

Figure 1: Gender wise distribution of subjects.

Table 2: Patient demographic data.

Parameters	Total No. of subjects (N=124)	Percentage (%)
BMI		
(<18.5)	1	0.8
Normal(18.5-24.9)	23	18.54
Pre obese(25-29.9)	57	45.96
Obese(>30)	43	34.67
Literacy status		
Illiterates	41	33.06
Less than high school	29	23.38
High School	15	12.09
More than High school	29	23
Smoking status		
Smokers	31	25
Non - Smokers	93	75
Alcohol Consumption		
Alcoholic	42	33.87
Non Alcoholic	82	66.12
Dietary Pattern		
Vegetarians	28	22.58
Non Vegetarians	96	77.41
Physical activity		
Mild	59	47.58
Moderate	47	37.90
Severe	18	14.51

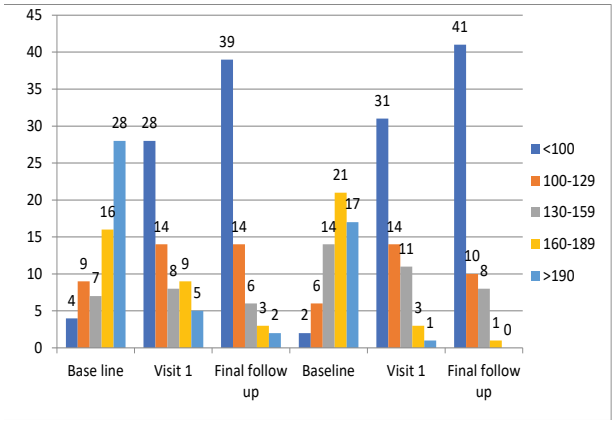

Figure 2: LDL ranges of subjects in the study population.

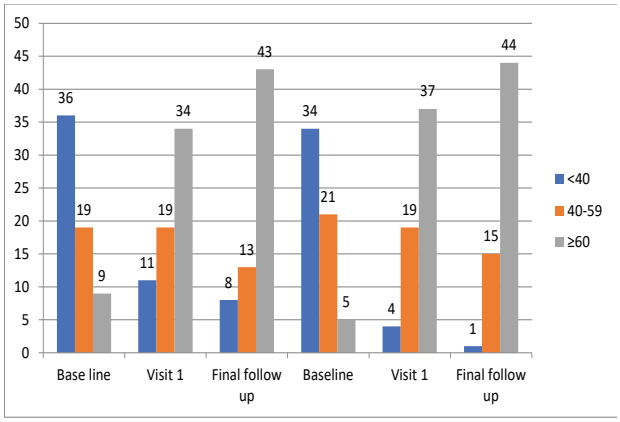

Figure 3: HDL ranges of subjects in the study population.

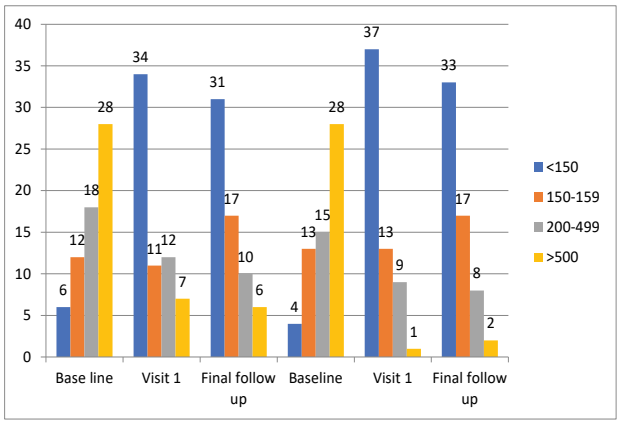

Figure 4: Triglycerides ranges of subjects in the study population.

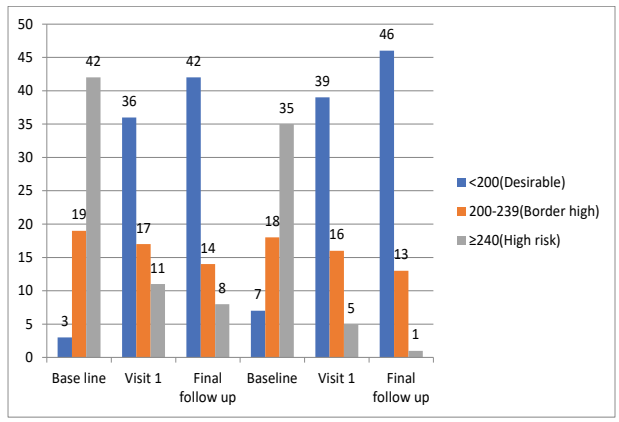

Figure 5: Total cholesterol ranges of subjects in the study population.

DISCUSSION

In this study screening for coronary artery disease was conducted for 124 subjects of age 25 years and above in Santhiram medical college and general hospital. In our study 124 subjects have been included, out of which about 60.48% were males and 39.51% were females. Table 01 conclude Subjects of age 45-55 years are more prone to development of CAD. 45.54% cases of age 45-55 years were diagnosed as CAD. New cases are diagnosed in the patients of age 25 years due to sedentary life style modifications. CAD is common cardiovascular disease with increase in age. As age increases it predispose to High rates of incidence and prevalence in both men and women. As age increases, it is associated with cellular oxidative stress, inflammation, changes in expression of gene leading to vascular stiffness, dysfunction of endothelial cells and thrombogenicity. These pathophysiological changes cause further development of CAD. Patients with older CAD have wide spread of cholesterol disposition in coronary arteries [10].

Fig 01 includes 60.48% are males and 39.51% are female. Males are prone to development of CAD due risk factors like alcohol consumption, smoking. CAD is major problem for both men and women. It include CAD is more common in men than women. Clinical condition, patient characteristics play important role in development of CAD. Women are less likely to develop CAD due to physiological reasons. Estrogen is helpful in decreasing the atherosclerotic plaque, blood pressure and increase anti-oxidative properties [11].

Table 02 conclude that 45.96% subjects are pre-obese and 34.67% subjects are obese are more prone to the development of CAD.0.8% subjects are under weight and 18.54% subjects having normal BMI are less likely prone to development of CAD. Obesity is a major risk factor for development of CAD when compared to smoking, alcoholism. It increases the risk of hypertension, diabetes mellitus, dyslipidemia, sleep apnea, cardiovascular diseases [11]. Differences in expression of gene from sex chromosomes, following differences in sex hormones affect the functions of CV system like no signalling in vascular function. All these factors cause CAD [12]. Obesity is not a significant predictor for CAD, but it is an accepted risk factor for CAD. There is an inverse relationship between Body mass index and severity of CAD. Obese people have lesser outcomes when compared to non obese people.BMI is not linked to degree of coronary atherosclerosis and death [13].

Literacy can be defined as process of understanding and communication that is necessary to make decisions related to health. It plays an important in supervision of CAD patients. Knowledge about the disease condition, complexity should be known by the patient or patient's representatives which is necessary to prevent the further complications, co-morbid condition. Low literacy lead to decreased compliance with recommended treatment. Hence literacy rate is important in maintaining health of the patients [14]. Smoking is considered as risk factor for the development of coronary artery disease. It is a major cause of morbidity and mortality. Platelet activation is enhanced by smoking and it leads to formation of thrombosis. It leads to narrowing of arteries, further causing CAD [15]. Alcohol acts on the liver causing increase of hepatic production leading to apolipoprotein formation. They increase triglyceride levels, LDL, decrease circulation of HDL. Alcohol consumption reduces fibrinogen levels, clotting factors, platelet aggregation [16]. Excessive consumption of alcohol is associated with CAD. It causes toxic cardiomyopathy. Enhanced coronary vasavasorum causes CAD [17]. This table conclude that 22.58% are vegetarians, 77.41 % are non vegetarians. Diet plays a important role in development of Coronary artery disease. Fatty acids and Cholesterol present in diet regulates the serum cholesterol levels. Cholesterol rich LDL fraction regulates the development of atherosclerotic plaque. Genes, hormones, diet control the serum cholesterol level. Saturated fatty acids increase the LDL cholesterol levels. Complex interaction between diet and life style has an important role in formation of atherosclerosis. Healthy diet and life style modifications reduce the risk of coronary artery disease. Intake of vitamins with antioxidant properties reduce of coronary artery disease [18]. Table 02 concludes that 47.58 % subjects are having mild physical activity, 37.90% subjects are having moderate physical activity, 14.51% subjects are having severe physical activity. Physical inactivity is one of the major risk factor for coronary artery disease. Various physiological mechanisms have harmful effects on blood pressure, lipid profile. All these changes have effect on atherosclerotic plaque. Physical inactivity also causes non fatal myocardial infarction, sudden coronary death, mortality. Physical inactivity is modifiable risk factor [18].

Fig 02 concludes that Rosuvastatin 10mg showed greater reduction in LDL cholesterol than atorvastatin. Both rosuvastatin and atorvastatin reduced LDL cholesterol in first 2 weeks of treatment regimen. Rosuvastatin 10mg reduced LDL cholesterol considerably when compared to starting doses of Atorvastatin, Simvastatin, Pravastatin. LDL cholesterol reduction was 41 and 42 with rosuvastatin 5mg in two groups was concluded. LDL cholesterol reduction was 41 and 42 with rosuvastatin 5 mg and 10mg in two groups were concluded [19].

Fig 03 includes HDL levels are important in determine the risk for CAD.HDL has a major role in decreasing the risk of CAD. HDL is inversely associated with CAD. Higher the HDL level lower the risk of CAD.HDL is potential antiatherogenic factor. It is powerful inverse risk predictor. Genetic disorders characterized by low levels of HDL dispose to the development of CAD. Post menopausal estrogen therapy increase HDL levels there by reducing the risk of CAD. Factors like cigarette smoking, androgens, physical inactivity, thiazide diuretics decrease HDL levels. Increase in weight about 5 pounds (2.27 kg) is associated with 5% decrease in HDL level with increase in LDL and triglyceride levels. Persons who smoke have HDL levels 3 to 5 mg/dl less [19].

Fig 04 concludes Triglycerides are considered as independent risk factor for CAD. Increased triglyceride levels remnants lead to development of atherosclerotic plaque. This results in development of coronary artery disease. Triglyceride lowering medications such as omega-3-fatty acids has beneficial effect in reducing the CAD risk [20].

Fig 05 concludes that increased total cholesterol levels are associated with development of CAD. Hyperlipidemia can be defined as high levels of cholesterol in blood above normal range. Hyperlipidemia increase atherosclerotic plaque. High cholesterol levels are associated with CAD in young adults. Low levels of cholesterol reduce the risk of CAD. Hyper-cholestermia is associated with metabolic and hormonal disturbances [21].

CONCLUSION

Coronary artery disease (CAD) develops due to the accumulation of plaque on walls of arteries of heart leading to decreased blood and oxygen supply to heart. Complete block of arteries may cause heart attack.

Our study concludes that the drug compliance was seen in both the patients using Atorvastatin 40mg and Rosuvastatin 20mg. But based on the mean differences and P-Valves calculated for the lipid profile parameters of each individual patient, Rosuvastatin 20mg has more efficacies with fewer side effects when compared to Atorvastatin 40 mg.

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CONFLICT OF INTEREST:

No authors declared conflict of interest.

REFERENCES

1. Jayita dasgupta et al. Study of lipid profile in patients of coronary artery disease among rural population. IOSR journal of pharmacy and biological sciences. 2015; Vol 10 (1), pp 51-54.
2. Abhishek et al. Trend in prevalence of coronary artery disease and risk factors over two decades in rural Punjab. Heart asia. 2017; 9(2): e010938. Published online 2017 sep14. Doi: 10.1136/heartasia-2017-010938.
3. T sekhri et al. Prevalence of risk factors for coronary artery disease in an urban Indian population. BMJ open 2014; 4:e005346. Doi:10.1136/bmjopen-2014-005346
4. Göran k, Hansson et al. Mechanisms of disease inflammation, atherosclerosis, and coronary artery disease. N Engl J Med. 2005; 352; 16.
5. Valentin et al. Pathogenesis of coronary artery disease. 1992 vol 326(5), pp.310-317.
6. Viswanathan mohan et al. Prevalence of coronary artery disease and its relationship to lipids in a selected population in south India. Journal of the American college of cardiology. 2001; vol 38 (3), 200.
7. Eswar kandaswamy et al. Recent advances in treatment of coronary artery disease: role of science and technology id. Int. J. Mol. Sci. 2018, 19, 424.

8. Matthew pflieger et al. Medical management of stable coronary artery disease. Afp Am Fam Physician. 2011; Vol 83(7):819-826.
9. Ruskin Street et al. Coronary Artery Disease A Guide for Patients and Families. 2011.
10. Daniel E. Forman et al. CAD and the Elderly: Diagnostic and Therapeutic Considerations. The Cardiology Advisor > Decision Support in Medicine > Cardiology > CAD and the Elderly: Diagnostic and Therapeutic Considerations Copyright © 2017, 2013 Decision Support in Medicine, LLC.
11. Amal Jamee et al. Gender Difference and Characteristics Attributed to Coronary Artery Disease Glob J Health Sci. 2013 Sep; 5(5): 51–56. doi: 10.5539/gjhs.v5n5p51 PMCID: PMC4776843.
12. Hassan Alkhawam et al. Coronary artery disease in patients with body mass index ≥30 kg/m²: a retrospective chart analysis. J Community Hosp Intern Med Perspect. 2016; 6(3): 10.3402/jchimp.v6.31483. PMCID: PMC4942517 PMID: 27406452
13. Anne Gregory et al. The Relationship between Body Mass Index and the Severity of Coronary Artery Disease in Patients Referred for Coronary Angiography. Cardiology Research and Practice. Vol 2017, Article ID 5481671, 10 pages https://doi.org/10.1155/2017/5481671
14. Gabriela Lima de Melo Ghisi et al. Health literacy and coronary artery disease: A systematic review. 2004; V 2(1); 8. http://dx.doi.org/10.1016/j.pec.2017.09.002 PMC2669461.
15. Doron aronson et al. Coronary artery disease and diabetes mellitus. Cardiol clin cardiol clin. 2014; 32(3): 439–455. Doi: 10.1016/j.ccl.2014.04.001
16. Marc J Mathews et al. The mechanism by which moderate alcohol consumption influences coronary heart disease. Nutr J. 2015; 14: 33. PMC4389579 doi: 10.1186/s12937-015-0011-6 PMCID: PMC4389579.
17. Diane L. Lucas et al. Alcohol and the Cardiovascular System: Research Challenges and Opportunities. Journal of the American College of Cardiology. 2005; Vol 45 (12), pp.1916-1924. https://doi.org/10.1016/j.jacc.2005.02.075
18. Jo-Ann Eastwood et al. Anginal Symptoms, Coronary Artery Disease, and Adverse Outcomes in Black and White Women: The NHLBI-Sponsored Women's Ischemia Syndrome Evaluation (WISE) Study. 2013; 22(9):724732. doi:10.1089/jwh.2012 4031PMCID: PMC3768228 PMID: 23992103
19. Peter W.F. Wilson et al. High-Density Lipoprotein, Low-Density Lipoprotein and Coronary Artery Disease. Am J Cardiol. 1996.
20. Awadhesh Kumar Singh et al. Triglyceride and cardiovascular risk: A critical appraisal. Indian J Endocrinol Metab. 2016; 20(4): 418–428.doi: 10.4103/2230-8210.183460. PMCID: PMC4911828. PMID: 27366705
21. Luther T. Clark, Brooklyn et al. Cholesterol and Heart Disease: Current Concepts in Pathogenesis and Treatment. Journal of the National Medical Association, 1986; Vol 78, (8).