"STUDY OF RISK FACTORS FOR CORONARY ARTERY DISEASE IN YOUNG PATIENTS WITH ISCHEMIC HEART DISEASE"

By

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Dissertation submitted to the

SBKS MEDICAL INSTITUTE & RESEARCH CENTRE

SUMANDEEP VIDYAPEETH, PIPARIA, VADODARA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

In

MEDICINE

Under the Guidance of

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SBKS MEDICAL INSTITUTE & RESEARCH CENTRE, PIPARIA, VADODARA,

YEAR 2015-2018

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DR. JIGAR PATEL

ABSTRACT

Introduction: Coronary artery disease (CAD) remains the commonest cause of mortality worldwide. It has been documented that younger age group people are developing the diseases frequently than before in last couple of years. Various studies show that in comparison with older patients these patients have single vessel disease, has more profound hypercholesterolemia, significant positive family history and history of smoking.

Objective: To evaluate different risk factors in patients below 40 years of age suffering from coronary heart disease.

Methodology: A study of young patients with CAD was carried out in the Dept. Of Medicine who attended the Dhiraj General Hospital, Pipariya, Waghodiya, Vadodara, Gujarat. Total 63 young with the age range of 18 to 40 years patients with CAD who visited the hospital during the time of Jan'2013 to June'2015 for the treatment of it were included. A detail history of all the subjects included in the study group was obtained from the available hospital data or on follow-up.

Results: A total of 63 cases were included. Smoking was present in 33.33 % of the subjects. All the smokers in the study were males. Sedentary lifestyle as a risk factor was present in 20.64 % of the subjects. Family history of coronary artery disease was present in 47.62 % of the subjects. Generalized obesity was present in 36.51 % of our subjects. In the study, 74.8 % of the subjects had dyslipidemia as a risk factor. Increased cholesterol was noted in 14.3% subjects. Hypercholesterolemia mainly increased LDL, VLDL and decreased HDL are important risk factors. Hypertension was a risk factor in 14.29 % and Diabetes in 9.52% of the subjects.

Conclusions: Dyslipidemia was a major risk factor in young adults followed by family history, followed by obesity and smoking. Hypertension and sedentary life style were the other less important risk factors. The majority of risk factors were equally prevalent in males as well as females except smoking which was less prevalent in females. Considering the increasing incidence of the coronary heart disease in our society it is essential to assess and evaluate these risk factors at national level.

Key Words: Risk Factors, Coronary Artery Disease, Smoking, Young adults.

TABLE OF CONTENTS

Sr. No.	Content	Page No.
1.	INTRODUCTION	1-2
2.	AIMS AND OBJECTIVES	3
3.	REVIEW OF LITERATURE	4-28
4.	MATERIAL AND METHODS	29-31
5.	RESULTS	32-56
6.	DISCUSSION	57-59
7.	SUMMARY AND CONCLUSION	60
8.	BIBLIOGRAPHY	61-64
9.	ANNEXURE	65-68
10.	MASTER CHART	***

LIST OF TABLES

TABLE. NO.	TABLE	PAGE NO.
1.	Sex distribution of study group (n=63)	32
2.	Sex wise distribution of smoking as a risk factor (n=63)	32
3.	Age wise distribution of smoking as a risk factor in males (n=52)	33
4.	Distribution of smokers according to pack years (n=21)	34
5.	Sex wise distribution of sedentary life style as a risk factor (n=63)	35
6.	Age wise distribution of sedentary life style as a risk factor (n=13)	36
7.	Age wise distribution of generalized obesity as a risk factor (n=63)	37
8.	Age wise distribution of generalized obesity as a risk factor (n=23)	37
9.	Sex wise distribution of hypertension as a risk factor (n= 63)	38
10.	Age wise distribution of hypertension as a risk factor (n= 9)	39
11.	Sex wise distribution of Diabetes as a risk factor (n= 63)	40
12.	Age wise distribution of Diabetes as a risk factor (n= 6)	41
13.	Sex wise distribution of Dyslipidemia as a risk factor (n=63)	42
14.	Sex wise distribution of Total Cholesterol as a risk factor (n=9)	43
15.	Age wise distribution of Total Cholesterol as a risk factor (n=9)	44
16.	Sex wise distribution of low density lipoprotein (n=30)	45
17.	Age wise distribution of low density lipoprotein (n=30)	46
18.	Sex wise distribution of high density lipoprotein (n=31)	47

19.	Age wise distribution of High Density Lipoprotein (n=31)	**48**
20.	Sex wise distribution of Very Low Density Lipoprotein (n=50)	**49**
21.	Age wise distribution of Very Low Density Lipoprotein (n=50)	**50**
22.	Sex wise distribution of Triglycerides (n=19)	**51**
23.	Age wise distribution of Triglycerides (n=19)	**52**
24.	Sex wise distribution of family history of IHD as a risk factor (n=63)	**53**
25.	Age wise distribution of family history of IHD as a risk factor (n=30)	**54**
26.	Sex wise distribution of CAG finding (n=63)	**55**
27.	Age wise distribution of CAG finding (n=63)	**56**

LIST OF GRAPHS

GRAPH. NO.	GRAPHS	PAGE NO.
1.	Age wise distribution of smoking as a risk factor in males	33
2.	Distribution of smokers according to pack years in Male	34
3.	Sex wise distribution of sedentary life style as a risk factor	35
4.	Age wise distribution of sedentary life style as a risk factor	36
5.	Age wise distribution of generalized obesity as a risk factor	38
6.	Sex wise distribution of hypertension as a risk factor	39
7.	Age wise distribution of hypertension as a risk factor	40
8.	Sex wise distribution of Diabetes as a risk factor	41
9.	Age wise distribution of Diabetes as a risk factor	42
10.	Sex wise distribution of Total Cholesterol as a risk factor	43
11.	Age wise distribution of Total Cholesterol as a risk factor	44
12.	Sex wise distribution of Low Density Lipoprotein as a risk factor	45
13.	Age wise distribution of low density lipoprotein	46
14.	Sex wise distribution of high density lipoprotein	47
15.	Age wise distribution of High Density Lipoprotein	48
16.	Sex wise distribution of Very Low Density Lipoprotein	49
17.	Age wise distribution of Very Low Density Lipoprotein	50
18.	Sex wise distribution of Triglycerides	51
19.	Age wise distribution of Triglycerides	52
20.	Sex wise distribution of family history of IHD as a risk factor	53

21.	Age wise distribution of family history of IHD as a risk factor	**54**
22.	Sex wise distribution of CAG finding (n=63)	**55**
23.	Age wise distribution of CAG finding (n=63)	**56**

INTRODUCTION

Coronary artery disease (CAD) is the largest killer in developed countries and is rapidly becoming one in developing countries. [1 – 3]

Coronary artery disease is forecast to be the most common cause of death globally, including India, by 2020. [4]

Coronary artery disease contributed to 15.3 million deaths in 1996, of which 5.5 million was from developed countries and 9.77 million from developing countries. A rise in the prevalence of coronary artery disease in the early half of twentieth century and a subsequent decline in the second half have been well documented in the industrialized countries. However, the scenario is reversed in developing countries especially India with a steady escalation in prevalence of coronary artery disease. [5]

"Hritshoola" an equivalent of present day angina/coronary artery disease (CAD) was known to ancient physicians since 500 BC. It involves those individuals who have faulty life style including sedentary habits, increased consumption of fatty foods, smoking, hypertension, etc. Atherosclerosis starts at an early age in these individuals, these observations have been confirmed by Framingham studies. [6-8]

In a prospective autopsy study conducted on 100 cases dying of causes other than cardiovascular ailments, the earliest age of aortic and/or coronary atherosclerosis occurrence was 16 years in both sexes. [8]

It is consistently observed that Indians have premature CAD and that their risk for CAD was two to four times higher than the white European population. The prevalence of CAD has increased to 10 times in the last 40 years. [5]

CAD has a multi-factorial etiology, with many of the risk factors being influenced by lifestyle. Rapid change in dietary habits coupled with decreased physical activity in India as a consequence of urbanization may partly explain the escalation of CAD. India is at present experiencing an epidemiological transition with high rates of urbanization. One of the effects of this transition is a shift in the disease spectrum from communicable to non-communicable diseases, particularly CAD and diabetes. [5]

Previous data from different studies have indicated that smoking, hypertension & dyslipidemia if present in young individuals play an important role in the development of premature coronary artery disease. [6]

There is need to identify & correct the conventional risk factors for coronary artery disease as Smoking can increase the risk by 3-5 times & modest increase in central obesity (depicted by Waist Hip Ratio) increases the risk further. [6]

The purpose of this study was to see the prevalence of different conventional risk factors for coronary artery disease in our young population.

AIMS & OBJECTIVES

The present study was carried out with aims and objectives of:-

1. Assessment of Risk Factors of Coronary Heart Disease in Young Adults.
2. To emphasize importance of development of CAD in young subjects, with risk factors.
3. To take measures to prevent occurrence of CAD in young subjects with risk factors.
4. To compare the occurrence of CAD in specific age groups.
5. To compare the risk factors in different young age groups.

REVIEW OF LITERATURE

Coronary artery disease is defined by a joint International Society and Federation of Cardiology and World Health Organization task force as myocardial impairment due to an imbalance between coronary blood flow and myocardial requirements caused by changes in the coronary circulation. [10]

Coronary artery disease is most commonly due to atherosclerotic occlusion of the coronary arteries.

Half of all deaths in the developed world and a quarter of deaths in the developing world are due to Cardiovascular Disease which are comprised of hypertension and the diseases caused by atherosclerosis. [11]

A historical perspective:

Coronary artery disease was until recently viewed largely as a twentieth-century epidemic. In retrospect it seems clear that CAD and CAD deaths occurred before the formal medical description of myocardial infarction in the early years of the twentieth century. William Heberden in 1768 described the typical symptoms of angina pectoris and the fact that sudden death occurred as a complication. Later in the eighteenth century, Dr Samuel Black of Newry and County Down, described many cases of angina pectoris and produced a list of factors associated with increased and decreased susceptibility to angina. While angina pectoris was occasionally recorded as a cause of death, less than 2 per cent of all deaths attributed to heart disease in Britain fell into this category even by the early part of the twentieth century. [10]

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Dr Samuel Black's categorization of factors related to liability and exemption from angina pectoris [12]

Liable
The male sex
The better ranks of society
The psychologically stressed
Those with an ossific diathesis
Those with an accumulation of fat around the heart
Those with full and plethoric habits who live luxuriously
Those with insufficient exercise
The obese
Exempt
The female sex
The poor
The laborious
Those who use strong exercise
The foot-soldier
The French

In the middle of the nineteenth century, Richard Quain described a condition he called â fatty disease of the heart. A retrospective review of his case series suggests that 52 out of 83 cases probably suffered from CAD [10]. The pathologist Carl Weigert and clinician Carl Huber in Leipzig described the myocardial lesions induced by acute ischaemia and speculated that myocardial infarction and angina both reflected underlying coronary artery disease [10]. Before the turn of the century myocardial infarction and coronary thrombosis were thought of as terminal events.In 1910, for the first time two Russian doctors Obrastzow & Straschesko, diagnosed acute coronary thrombosis in living patients [10]. By 1918 Herrick was able to link clinical information with electrocardiogram patterns shown to reflect myocardial infarction in experimental canine studies, making the antemortem diagnosis of myocardial infarction easier. [10]

Atherosclerosis has retrospectively been demonstrated in Egyptian mummies, but the first contemporary descriptions occurred in the sixteenth century. [10]

Pathophysiology:

Coronary Blood Flow: [11]

The heart is an aerobic organ that is dependent for its oxygen supply entirely on coronary perfusion. Under resting condition, the myocardium extracts the maximum amount of oxygen from the blood it receives. The O2 saturation of blood returning from the coronary sinus to the right atrium has the lowest saturation of any body organ (30%).

Interruption of coronary blood flow will result in immediate ischemia. Coronary blood flow is directly dependent upon perfusion pressure and inversely proportional to the resistance of the coronary vessel.

$Q \propto$ Perfusion pressure / Vessel resistance

Coronary perfusion occurs in diastole hence diastolic pressure is more important than systolic pressure in determining coronary perfusion. Coronary vessels are divided into epicardial or conductance vessels (R1), pre capillary (R2) and microvascular vessels (R3). The epicardial vessels, the site most commonly affected by atherosclerosis, offer negligible resistance to coronary flow. Resistance to flow occurs in the pre capillary (R2), and microvascular (R3) vessels which are termed resistance vessels. The increase coronary blood flow in response to increase myocardial oxygen demand (MVO2) is achieved by the dilatation of these resistance vessels. Three factors play a key role in modifying vascular tone; the accumulation of local metabolites, endothelial factors and neural tone. The accumulation of adenosine during ischemia is an example of local metabolic factors. The most important endothelial substance mediating vasodilatation is nitric oxide (NO). Other important mediators are bradykinin, endothelium derived hyperpolarizing factor and prostacyclin. On the other hand, endothelin-1 (ET-1) is a well-known vasoconstricting substance. Angiotensin II and thromboxane A2 are other well-known endothelium derived constricting factors. Alpha receptor adrenergic stimulation results in coronary vasoconstriction whereas beta 1 receptor stimulation leads to vasodilatation. [11]

Coronary vascular resistance can be reduced to 1/5th of baseline resistance leading to a fivefold increase in the volume of perfusion in response to an increase in need. [11]

Coronary reserve is the term used to reflect the amount of increase in coronary perfusion to accommodate increased demand. Autoregulation, mediated by changes in the vascular tone of the resistance vessels, allows distal coronary perfusion to remain unaltered in the face of changes in proximal perfusion pressures. Impaired endothelial function disrupts autoregulation and may lead to ischemia. Diseases known to impair endothelial function include atherosclerosis, dyslipidemias, diabetes mellitus, hypertension, smoking (both passive and active) and hyperhomocysteinemia.[11]

Coronary arteries suffering from atherosclerosis lose the ability to release the vasodilating substances that allow the increase in coronary perfusion in the face of increased demand. Their coronary reserve is limited by the failure to dilate and reduce vascular resistance. Furchgott showed that acetylcholine, through the release of NO,results in vasodilation of the coronary vessel. However if the endothelium overlying the vascular smooth vessel was diseased (e.g. by atherosclerosis), the smooth muscle will paradoxically vasoconstrict. [10]

Blockage of the epicardial coronary vessels (coronary stenosis) of up to 60% is compensated at rest and maximal exercise by vasodilation of the resistance coronary vessels. Blockage of epicardial coronary vessels in excess of 60% will result, under conditions of increases myocardial oxygen demand, in reduced perfusion and in turn ischemia. Clinically, this translates to effort or exercise induced angina. This is the basis for performing exercise stress testing in patients suspected of having coronary artery disease. When the severity of the blockage is greater than 90%, perfusion is compromised even at rest. Clinically, this may result in resting angina, a critical stage of coronary artery disease. Ischemia is the result of the coronary vessel's inability to meet the demand of the myocardium it supplies. The imbalance between supply and

demand (↑ demand, ↓ supply) results in ischemia. Clinically this presents as chest discomfort and /or shortness of breath. [11]

The Determinants of Myocardial O2 Consumption: [11]

The major determinants of myocardial O2 consumption (MVO2) are 1) heart rate 2) ventricular wall stress (afterload) and 3) contractility (inotropy). Heart rate is the main determinant of MVO2. Ventricular wall stress, as defined by Laplace's law, is the product of the left ventricular systolic pressure and the radius of the left ventricle divided by its wall thickness. Hence, processes such as aortic stenosis and hypertension, which increase systolic pressure, mitral and aortic regurgitation, which increase LV cavity size,increase myocardial oxygen consumption. During exercise stress testing MVO2 can be described using the "double product," the product of the maximal systolic pressure and heart rate attained during maximal exercise. This formula represents the two key components of myocardial oxygen demand. [11]

Initiation of atherosclerosis: [13]

Atherosclerosis manifests itself focally not only in space, as just described, but in time as well. Atherogenesis in humans typically occurs over a period of many years, usually many decades. Growth of atherosclerotic plaques probably does not occur in a smooth linear fashion, but rather discontinuously, with periods of relative quiescence punctuated by periods of rapid evolution. After a generally prolonged "silent" period, atherosclerosis may become clinically manifest. The clinical expressions of atherosclerosis may be chronic, as in the development of stable, effort-induced angina pectoris or of predictable and reproducible intermittent claudication. Alternatively, a much more dramatic acute clinical event such as myocardial infarction, a cerebrovascular accident, or sudden cardiac death may first herald the presence of atherosclerosis. Other individuals may never experience clinical manifestations of

arterial disease despite the presence of widespread atherosclerosis demonstrated post mortem.

FATTY STREAK FORMATION

An integrated view of experimental results in animals and study of human atherosclerosis suggests that the "fattystreak" represents the initial lesion of atherosclerosis. The formation of these early lesions of atherosclerosis most often seems to arise from focal increases in the content of lipoproteins within regions of the intima. This accumulation of lipoprotein particles may not result simply from an increased permeability, or "leakiness," of the overlying endothelium. Rather, these lipoproteins may collect in the intima of arteries because they bind to constituents of the extracellular matrix, increasing the residence time of the lipid-rich particles within the arterial wall. Lipoproteins that accumulate in the extracellular space of the intima of arteries often associate with proteoglycan molecules of the arterial extracellular matrix, an interaction that may promote the retention of lipoprotein particles by binding them and slowing their egress from the intima. Lipoprotein particles in the extracellular space of the intima, particularly those bound to matrix macromolecules, may undergo chemical modifications. Accumulating evidence supports a pathogenic role for such modifications of lipoproteins in atherogenesis. Two types of such alterations in lipoproteins bear particular interest in the context of understanding how risk factors actually promote atherogenesis: oxidation and nonenzymatic glycation. [13]

Lipoprotein Oxidation:

Lipoproteins sequestered from plasma antioxidants in the extracellular space of the intima become susceptible to oxidative modification. Oxidatively modified low-density lipoprotein (LDL), rather than being a defined homogenous entity, actually

comprises a variable and incompletely defined mixture. Both the lipid and protein moieties of these particles can participate in oxidative modification. [13]

Modifications of the lipids may include formation of hydroperoxides, lysophospholipids, oxysterols, and aldehydic breakdown products of fatty acids. Modifications of the apoprotein moieties may include breaks in the peptide backbone as well as derivatization of certain amino acid residues. A more recently recognized modification may result from local hypochlorous acid production by inflammatory cells within the plaque, giving rise to chlorinated species such as chlorotyrosyl moieties. Considerable evidence supports the presence of such oxidation products in atherosclerotic lesions. [13]

Nonenzymatic Glycation:

In diabetic patients with sustained hyperglycemia, nonenzymatic glycation of apolipoproteins and other arterial proteins likely occurs that may alter their function and propensity to accelerate atherogenesis. [13]

LEUKOCYTE RECRUITMENT:

After the accumulation of extracellular lipid, recruitment of leukocytes occurs as a second step in the formation of the fatty streak. The white blood cell types typically found in the evolving atheroma include primarily cells of the mononuclear lineage: monocytes and lymphocytes. A number of adhesion molecules or receptors for leukocytes expressed on the surface of the arterial endothelial cell likely participate in the recruitment of leukocytes to the nascent fatty streak. Constituents of oxidatively modified LDL can augment expression of leukocyte adhesion molecules. [13]

Laminar shear forces, such as those encountered in most regions of normal arteries, can also suppress the expression of leukocyte adhesion molecules. Sites of predilection for forming atherosclerotic lesions (e.g., branch points) often have disturbed laminar flow. Ordered laminar shear of normal blood flow augments the production of nitric oxide by endothelial cells. This molecule, in addition to its vasodilator properties, can act at the low levels constitutively produced by arterial endothelium as a local anti-inflammatory autacoid, for example, limiting local adhesion molecule expression. These examples indicate how hemodynamic forces may influence the cellular events that underlie atherosclerotic lesion initiation and provide a potential explanation for the focal distribution of atherosclerotic lesions at certain sites predetermined by altered flow patterns. [13]

Once adherent to the surface of the arterial endothelial cell via interaction with adhesion receptors, the monocytes and lymphocytes penetrate the endothelial layer and take up residence in the intima. In addition to products of modified lipoproteins, cytokines (a class of protein mediators of inflammation) can regulate the expression of adhesion molecules involved in leukocyte recruitment. For example, the cytokines interleukin 1 (IL-1) or tumor necrosis factor _ (TNF-_) induce or augment the expression of leukocyte adhesion molecules on endothelial cells. Because modified lipoproteins can induce cytokine release from vascular wall cells, this pathway may provide an additional link between accumulation and modification of lipoproteins and leukocyte recruitment. The directed migration of leukocytes into the arterial wall may also result from the actions of modified lipoprotein. For example, oxidized LDL may promote the chemotaxis of leukocytes. Also, oxidatively modified lipoproteins can elicit the production by vascular wall cells of chemoattractant cytokines such as monocyte chemoattractant protein 1. [13]

FOAM CELL FORMATION:

Once resident within the intima, the mononuclear phagocytes differentiate into macrophages and transform into lipid-laden foam cells. The conversion of mononuclear phagocytes into foam cells requires the uptake of lipoprotein particles by receptor mediated endocytosis. One might suppose that the well-recognized "classical" receptor for LDL mediates this lipid uptake. Patients or animals lacking effective LDL receptors due to genetic alterations (e.g., familial hypercholesterolemia), however, have abundant arterial lesions and extraarterial xanthomata rich in macrophage-derived foam cells. Also, the exogenous cholesterol suppresses expression of the LDL receptor, such that under hypercholesterolemic conditions the level of this cell-surface receptor for LDL decreases. Candidates for alternative receptors that can mediate lipid-loading of foam cells include a growing number of macrophage "scavenger" receptors, which preferentially endocytose modified lipoproteins, and other receptors for oxidized LDL or VLDL (very low density lipoprotein), a type of lipoprotein commonly encountered in certain hypercholesterolemic states . By ingesting lipids from the extracellular space, the mononuclear phagocytes bearing such scavenger receptors may remove lipoproteins from the developing lesion. Some lipid-loaded macrophages may leave the artery wall, functioning to clear lipid from the artery. Lipid accumulation, and hence propensity to form atheroma, ensues if the amount of lipid entering the artery wall exceeds that exported by mononuclear phagocytes or other pathways. Macrophages may thus play a vital role in the dynamic economy of lipid accumulation in the arterial wall during atherogenesis. Some lipid-laden foam cells within the expanding intimal lesion perish. Some foam cells may die as a result of programmed cell death known as apoptosis. This death of mononuclear phagocytes results in formation of the

lipid-rich center, often called the necrotic core, of more complicated atherosclerotic plaques. Macrophages taking up modified lipoproteins, much like intrinsic vascular wall cells, may elaborate cytokines and growth factors that can further signal some of the cellular events in lesion complication. [13]

A number of growth factors or cytokines elaborated by mononuclear phagocytes can stimulate smooth-muscle cell proliferation and production of extracellular matrix, which accumulates in atherosclerotic plaques. Cytokines found in the plaque, including IL-1 or TNF-, can induce local production of growth factors such as forms of platelet derived growth factor (PDGF), fibroblast growth factor, and others that may contribute to plaque evolution and complication. Other cytokines, notably interferon _ (IFN-) derived from activated T cells within lesions, can inhibit smooth-muscle proliferation and synthesis of interstitial forms of collagen. These examples illustrate how atherogenesis likely depends on a complex balance between mediators that can promote lesion formation and other pathways that can mitigate the atherogenic process. [13]

FACTORS THAT MODULATE INHIBITION OF ATHEROMA

Elaboration of small molecules by activated mononuclear phagocytes and vascular wall cells in the evolving lesion may also modulate atherogenesis.

Notably, reactive oxygen species can modulate growth of smooth-muscle cells, activate inflammatory gene expression via the nuclear factor kappa B (NFB) transcriptional control system, and annihilate NO radicals, decreasing the effect of this endogenous vasodilator. However, the macrophage in the lesion may be activated to express the inducible form of the enzyme that can synthesize NO, known as inducible NO synthase. This high-capacity form of the enzyme can produce relatively

large, potentially cytotoxic amounts of NO radicals. While at the low concentrations of NO produced by the constitutive NO synthase in endothelial cells, this radical may produce beneficial effects; when overproduced by activated phagocytes, however, it may prove deleterious. Export by phagocytes may constitute one response to local lipid overload in the evolving lesion. Another mechanism, reverse cholesterol transport mediated by high-density lipoproteins (HDL), may provide an independent pathway for lipid removal from atheroma. This transfer of cholesterol from the cell to the HDL particle involves specialized cell surface molecules such as the ATP binding cassette transporter (ABCA1) and a family of scavenger receptors (the "B" family). Such "reverse cholesterol transport" explains part of HDL's antiatherogenic action. Although clear evidence supports lipoprotein disorders as predisposing factors for atheroma formation, other etiologies may contribute to or modulate atherogenesis. For example, hypertension constitutes an independent risk factor for coronary events. Male gender and the postmenopausal state also augment the risk of developing coronary artery disease. Premenopausal women have increased HDL levels compared to age-matched men. However, a favorable lipoprotein pattern only partially accounts for the protection against atherosclerosis conferred by the premenopausal state. Although laboratory studies suggest that estrogens have direct beneficial effects on the arterial wall, clinical trials have not shown that estrogen replacement therapy prevents recurrent myocardial infarction in postmenopausal women. Indeed, treatment with a combination of estrogen and progesterone appears to augment cardiovascular events in women with or without prior myocardial infarction. Diabetes mellitus aggravates atherogenesis. In addition to the wellknown microvascular complications of diabetes, macrovascular disease such as atherosclerosis causes a great deal of excess mortality in the diabetic population. Diabetes-associated dyslipidemias

strongly promote atherogenesis. In particular, the constellation of insulin resistance, high triglycerides, and low HDL, often in association with the central adiposity and hypertension frequently seen in type 2 diabetic patients, seems to accelerate atherogenesis potently. As noted above, hyperglycemia may promote the nonenzymatic glycation of LDL. LDL modified in this manner, like oxidatively modified LDL, may signal many of the initial events in atherogenesis. Triglyceriderich lipoprotein particles, often elevated in poorly controlled diabetic patients, also accentuate atherogenesis. Lipoprotein little, i.e Lp (a) provides a potential link between hemostasis and blood lipids. The Lp (a) particle consists of an apoprotein (a) molecule bound by a sulfhydryl link to the apolipoprotein B moiety of an LDL particle. Apoprotein (a) has homology with plasminogen and may inhibit fibrinolysis by competing with plasminogen. Other risk factors for atherosclerosis related to blood clotting include elevated levels of fibrinogen or of the inhibitor of fibrinolysis, plasminogen-activator inhibitor 1 (PAI-1). Another nonlipid risk factor for coronary events, elevated levels of homocysteine, may act by promoting thrombosis, although the pathophysiology of this association is uncertain at present. Although individuals with marked elevations of Lp(a) or homocysteine do appear to have heightened risk of coronary thrombosis, in the population at large these factors show a much weaker correlation with vascular events than LDL, HDL, or the global inflammatory marker C-reactive protein (CRP). The relationship between tobacco use and atherosclerosis also remains poorly understood.

The rapid reduction in risk for cardiac events after cessation of cigarette smoking implies that tobacco may promote thrombosis or some other determinant of plaque stability as well as contribute to the evolution of the atherosclerotic lesion itself. For

example, tobacco smokers have elevated fibrinogen levels, a variable associated with increased atherosclerosis and acute cardiovascular events. [13]

INFLAMMATION:

In other situations, antecedent inflammatory states may predispose toward atherosclerosis. For example, Kawasaki disease in childhood may promote development of vascular lesions in the arteries of adults. Infectious agents continue to be proposed as instigators or potentiators of atherogenesis. However, in humans, an atherogenic role for viral or microbial pathogens (e.g., Herpesviridiae, including cytomegalovirus, or Chlamydia) remains speculative. In some patients, immune or autoimmune reactions may contribute to atherogenesis. In the particular example of the accelerated form of coronary arteriopathy that plagues heart transplant recipients, immunologic factors may contribute importantly to the pathogenesis.

Known monogenic defects in lipoprotein metabolism account for only a fraction of the familial risk for coronary artery disease. Thus, other as yet undefined and perhaps multiple genetic factors may contribute to coronary risk. Mechanisms of disease susceptibility involving the arterial wall might account for some of the genetic predisposition to atherosclerosis unexplained by lipoprotein disorders. Application of molecular genetic techniques may identify new polymorphisms linked to coronary risk and may eventually shed light on new pathophysiologic mechanisms. For example, some data suggest a link between certain alleles of the genes encoding angiotensin-converting enzyme, the cytokine lymphotoxin, or PAI-1 with increased risk of myocardial infarction. Application of genomic technologies may aid identification of “modifier” genes that modulate individual responses to established risk factors. Large studies currently in progress should clarify these and other potential genetic factors that influence atherosclerosis.

ATHEROMA EVOLUTION AND COMPLICATIONS [13]

INVOLVEMENT OF ARTERIAL SMOOTH-MUSCLE CELLS

Although the fatty streak commonly precedes the development of a more advanced atherosclerotic plaque, not all fatty streaks progress to form complex atheromas. [13]

While accumulation of lipid-laden macrophages is the hallmark of the fatty streaks, accumulation of fibrous tissue typifies the more advanced atherosclerotic lesion. The smooth-muscle cell synthesizes the bulk of the extracellular matrix of the complex atherosclerotic lesion. Hence, arrival of smooth-muscle cells and their elaboration of extracellular matrix probably provides a critical transition, yielding a fibrofatty lesion in place of a simple accumulation of macrophage- derived foam cells. Recent research has provided insight into the mechanisms that may trigger migration and proliferation of smooth-muscle cells into and within the evolving intimal lesion and signal the accumulation of extracellular matrix. Cytokines and growth factors elicited by modified lipoproteins or other agents from both vascular wall cells and infiltrating leukocytes can modulate functions of the smooth-muscle cell. For example, PDGF elaborated by activated endothelial cells can stimulate the migration of smooth-muscle cells. In this manner, smooth-muscle cells resident in the tunica media may migrate into the intima. Various growth factors produced locally can stimulate the proliferation of both resident smooth-muscle cells in the intima and those that have migrated from the media. Transforming growth factor (TGF), among other mediators, potently stimulates interstitial collagen production by smooth-muscle cells. These mediators may arise not only from neighboring vascular cells or leukocytes (a "paracrine" pathway) but also, in some instances, from the same cell that responds to the factor (an "autocrine" pathway). Together, these alterations in smooth-muscle cells, signaled by these mediators acting at short distances, can hasten transformation

of the fatty streak into a more fibrous smooth-muscle cell and extracellular matrix-rich lesion. [13]

BLOOD COAGULATION:

In addition to locally produced mediators, atherogenic risk factor signals related to blood coagulation and thrombosis likely contribute to atheroma evolution and complication. Current evidence suggests that fatty streak formation begins underneath a morphologically intact endothelium. In advanced fatty streaks, however, microscopic breaches in endothelial integrity may occur. Microthrombi rich in platelets can form at such sites of limited endothelial denudation, due to exposure of the highly thrombogenic extracellular matrix of underlying basement membrane. Activated platelets release numerous factors that can promote the fibrotic response. In addition to PDGF and TGF, low-molecular-weight mediators such as serotonin can also alter smooth-muscle function. Most of these microthrombi probably resolve without clinical manifestation by a process of local fibrinolysis, resorption, and endothelial repair. [13]

MICROVESSELS:

As atherosclerotic lesions advance, abundant plexi of microvessels develop in connection with the artery's vasa vasorum. These newly developing microvascular networks may contribute to lesion complication in several ways. These blood vessels provide an abundant surface area for leukocyte trafficking and may serve as the portal of entry and exit of white blood cells from the established atheroma. [13]

The plaques' microvessels may also furnish foci for intraplaque hemorrhage. Like the neovessels in the diabetic retina, microvessels of the plaque may be friable and prone to rupture and produce focal hemorrhage. Such a vascular leak leads to thrombosis in

situ and thrombin generation from prothrombin. In addition to its role in blood coagulation, thrombin can modulate many aspects of vascular cell function including stimulation of proliferation and cytokine release from smooth-muscle cells and production of growth factors such as PDGF from endothelial cells. Atherosclerotic plaques often contain fibrin and hemosiderin, indicating episodes of intraplaque hemorrhage as an element in plaque complication. [13]

As they advance, atherosclerotic plaques also accumulate calcium. Proteins that are usually associated with bone also occur in atherosclerotic lesions. For example, osteocalcin, osteopontin, and bone morphogenetic proteins localize in atherosclerotic plaques. In fact, mineralization of the atherosclerotic plaque recapitulates many aspects of bone formation. [13]

PLAQUE EVOLUTION:

Traditionally, atherosclerosis research has focused much attention on proliferation of smooth-muscle cells, yet these cells actually replicate rather slowly in complicated atherosclerotic lesions. [13]

Estimates of the rate of smooth-muscle cell division in such lesions at a given time show a replicative rate below 1%. Such observations do not exclude bursts of proliferative activity at certain junctures in the history of an atheroma, perhaps in association with local thrombin generation due to microvascular hemorrhage or formation of a microthrombus at a site of localized endothelial denudation, as discussed earlier. On the other hand, cell death has been recognized as a component of atherogenesis since the time of Virchow in the mid-nineteenth century. Indeed, complex atheroma often have a primarily fibrous character lacking the hypercellular appearance of less advanced lesions and actually exhibiting a paucity of smooth-

muscle cells. This relative lack of smooth-muscle cells in advanced atheroma may result from the ultimate predominance of cytostatic mediators such as TGF or IFN, which can inhibit smooth-muscle cell proliferation. Also, smooth-muscle cells as well as macrophages in advanced atherosclerotic lesions can undergo programmed cell death, or apoptosis. Some of the same cytokines that activate atherogenic functions of vascular wall cells can also trigger apoptosis in these cells. Thus, during the evolution of the atherosclerotic plaque, a complex balance between entry and egress of lipoproteins and leukocytes, cell proliferation and cell death, extracellular matrix production and remodeling, as well as calcification and neovascularization contribute to lesion formation. Multiple and often competing signals trigger these various cellular events. Increasingly, we appreciate links between atherogenic risk factors and the altered behavior of intrinsic vascular wall cells and infiltrating leukocytes that underlie the complex pathogenesis of these lesions. [13]

Risk Factors for Atherosclerotic Coronary Artery Disease: [14]

1. Dyslipidemias :

Relation between triglycerides & CAD have been proven in earlier studies. Triglyceride measurement per se is not that informative as some of the triglyceride rich lipoproteins present in the plasma are not atherogenic like large VLDLs, while others like small VLDL are highly atherogenic. Increased production of small VLDL in response to hypertriglyceridemia could contribute to atherosclerosis. This is confirmed by the presence of triglyceride rich lipoproteins in the human atheroma. [14]

Moreover, increase in triglycerides levels are associated with low HDL cholesterol & with small dense LDL molecules (Phenotype B).

Lipoproteins are high-molecular-weight complexes of lipid and protein that circulate in the blood plasma. Their physiologic functions include transport of lipids to cells for energy, growth requirements, or storage. Lipoproteins are also metabolic precursors of biologic regulators, such as prostaglandins, thromboxanes, and leukotrienes. LDLs promote atherogenesis by affecting one or several of the processes of influx and efflux of the vessel wall. Elevated LDL levels also promote thrombosis formation, and reducing LDL levels pharmacologically with statins decreases thrombosis. [14]

HDLs promote cholesterol efflux from atherosclerotic lesions. In addition, it seems that HDLs inhibit the oxidation and subsequent accumulation of LDL-cholesterol. Observational data and experiments in vitro and in transgenic mice suggest that HDLs containing apo A-I but not apo A-II are protective, whereas HDLs with both apolipoproteins are neutral. Apo A-I has been identified as a prostacyclin stabilizing factor, suggesting another possible mechanism of benefit. [14]

Evidence is growing that triglyceride-rich lipoproteins are important contributors to the development of atherothrombotic disease. Mechanisms by which hypertriglyceridemia may contribute to atherothrombotic disease risk include increased thrombogenicity, "small dense" LDLs postprandial lipidemia with increased chylomicron and very-low-density lipoprotein (VLDL) remnant particles, decreased HDL levels, and insulin resistance.An elevated lipoprotein (a) (Lp[a]) level in plasma may be a significant risk factor for atherothrombotic disease, particularly in the presence of elevated LDL levels. [14]

2. Hypertension :

JNC 7 has classified blood pressure as follows:-

Normal BP: systolic < 120 mmHg AND diastolic < 80 mmHg.

Prehypertension: systolic 120-139 OR diastolic 80-89 mmHg.

Hypertension: systolic > 140 OR diastolic > 90 mmHg.

In elevated blood pressure, endothelial dysfunction promotes atherogenesis by attenuating responses to endothelium dependent vasodilators, increasing vascular permeability to macromolecules (including lipoproteins), and increasing endothelin production and leukocyte adherence.

Hypertension also may be associated with phenotypic changes that increase the proliferative potential of vascular smooth muscle cells and their response to growth factors. [14]

3. Diabetes mellitus :

Report of the Expert Committee on the Diagnosis & Classification of Diabetes Mellitus classifies diabetes as follows:

Classification:

Normal fasting glucose < 110 mg/dL,

Impaired 110-≤ 126 mg/dL

Diabetes > 126 mg/dL

Insulin resistance in patients with type II diabetes mellitus or in patients with poorly controlled type I diabetes is accompanied by hyperinsulinemia, which may elevate

circulating insulin related growth factors such as insulin growth factor-1. In chronic hyperglycemia, glycated proteins and various local growth factors can stimulate the proliferation of the fibromuscular component of the mature atherosclerotic plaque. Levels of lipoproteins such as LDL may not be abnormal in patients with diabetes mellitus; however, lipoproteins may be glycated, resulting in abnormal function. Hypertriglyceridemia with HDL depletion is the characteristic lipid profile of insulin resistance and poorly controlled diabetes. As an important consequence of hypertriglyceridemia, abnormalities in the metabolism of triglyceride-rich lipoproteins result in modifications of LDL structure, so as to produce a smaller, denser, so-called subclass B form of LDL, which has markedly enhanced atherogenicity. Abnormalities of Lp (a) levels also are widespread in patients with poorly controlled diabetes. [14]

4. Smoking :

It is the most important modifiable risk factor for CAD. Observational data suggest that smoking exerts its atherogenic effects by inducing catecholamine release, which may elevate blood fibrinogen levels, activate monocytes, and increase platelet reactivity. These catecholamine effects may explain the increase in sudden cardiac death and acute cardiovascular events. Endothelial dysfunction, caused by catecholamines and/or nicotine, also increases in vascular tone. Smoking lowers HDL and promotes oxidation of LDL, presumably owing to the exposure of the latter to free radicals present in cigarette smoke. [14]

5. Family history of premature coronary artery disease (CAD):

First degree male relatives < 55 years or females < 65 years.

Single-gene mutations influence lipid metabolism. Complex polygenic disorders include hypertension, diabetes mellitus, and homocysteinemia and contribute to

atherogenesis. However currently identifiable genetic abnormalities only partially account for the risk predicted by a positive family history for premature coronary artery disease. [14]

6. Obesity and lack of exercise.

Obesity predisposes to hyperlipidemia, diabetes, and hypertension, but obesity itself is associated with only a small increase in the risk of coronary artery atherosclerosis, principally in youth. Physical activity favorably influences plasma lipoprotein profiles, adiposity, blood pressure, glucose tolerance, and cardiovascular and pulmonary functional capacity; individuals prone to become physically active also are prone to modify favorably their risk factors. Physical fitness, a condition that is measured more objectively than physical activity, also independently reduces the risk of coronary heart disease. [14]

7. Male sex:

Rates of death attributed to IHD in men are consistently three to four times higher than those in women. Men are more likely to smoke than women are, but differences in smoking and other established coronary risk factor levels do not appear to fully explain the observed excess of IHD seen in men. The lower risk of coronary heart disease among women has understandably led to studies of sex hormones as potential protective or risk factors for IHD. There is, however, little empirical evidence to support an important role for sex hormones. It has often been stated that women are only protected against ischaemic heart disease premenopausally, and that their risk progressively increases towards that of men after the menopause. [10]

8) Age:

Age is the strongest risk indicator for IHD incidence and mortality. Compared to men aged 40, 50 year-old-men have five times the risk, 60 year-old-men have 15 times the risk, and 70-year-old men have over 40 times the risk of dying from IHD. A similar steep gradient with age is seen for women. Risk continues to increase into older age groups and recent evidence that the elderly benefit from risk factor control, blood pressure lowering, and cholesterol lowering, and probably smoking cessation, combined with their much higher absolute level of risk than younger people. [10]

9) Others:

Recently, a number of newer cardiovascular risk factors have been identified. These factors are of great interest in native Indians where more than 60% of the CAD remains unexplained by conventional risk factors.

Comparative studies on newer risk factors illustrated that Asian Indians have higher C-reactive protein, plasminogen activator inhibitor (PAI 1) and homocysteine levels.[5]

About 20% of CAD occurs in individuals without any of the classical risk Factors ex. homocysteinemia, high sensitivity C reactive protein (hs-CRP), Fibrinogen, Lipoprotein a (Lpa), infection eg Chlamydia pneumonia. [11]

RELATED STUDIES:

S Diwedi et al (2000) [8] conducted a study titled 'Coronary Artery Disease in the Young: Heredofamilial or Faulty Lifestyle or Both' & concluded that 61.42 % of their subjects were smokers, all of these were males. They also found 51.42 % of their cases to be hypertensives, & obesity to be present in 35.7 % of the cases. Diabetes & hypercholesterolemia was present in 7.4 % & 41.66 % of the cases respectively.

Vincent Jomini et al (2002) [28] in their study 'Contribution Of Major Cardiovascular Risk Factors To Familial Premature Coronary Artery Disease– The Gamecard Project', compared the prevalence of major cardiovascular risk factors in patients with familial history of premature coronary artery disease (PCAD), to the general population , & found that compared to the general population ,patients with sporadic PCAD had a higher prevalence of Hypertension (29% vs 14%),hypercholesterolemia (54 % vs 33%) , obesity (20% vs 13%) , & smoking (76% vs 39%). These risk factors were equally or even more prevalent in patients with familial PCAD, 43%, 58 %, 21%, & 72% respectively.

Sangeeta Gulati et al (2004) [22] in their study titled 'A Comparative Study Of Risk Factors In Coronary Artery Disease' found 10.5 % of the subjects to be diabetic & 22 % of them were hypertensive . Obesity was present in 14.5 % of the study group.

In a study by **Arvind Kumar et al (2005)** [9] titled 'A Study Of Prevalence Of Risk Factors For Coronary Artery Diseases In Asymptomatic Middle Aged & Elderly Subjects' , it was found that , Hypertension , Diabetes , ↑ Low Density Lipoproteins , Hypertryglceridemia, & Central Obesity was present in 34%, 4%, 38%, 23%, & 27.50% of the cases respectively .

Noeman et al (2007) [6] in their study titled 'Coronary Artery Disease in Young: Faulty Lifestyle or Heredofamilial or Both', found that 63.4 % of the cases were smokers, whereas, Hypertension & Obesity was detected in 51.4 % & 35 % of the cases respectively. Dyslipidemia was seen in 41.66 % of the cases & 7.14 % of the cases were diabetic.

MATERIAL & METHODS

A study of young patients with CAD who attended the Dhiraj General Hospital, Pipariya, Waghodiya, Vadodara, Gujarat during the time of Jan'2013 to June'2015 which was carried out in the Dept. Of Medicine.

Total 63 young patients with CAD who visited the hospital during the time of Jan'2013 to June'2015 for the treatment of it were included as per the Inclusion criteria to study the risk factors for CAD. The study protocol was reviewed and approved by local Ethics Committee.

Inclusion Criteria:

- Male and female patients between the ages of 18 to 40 years.
- Patients with:
  - Acute coronary syndrome – STEMI, NSTEMI or unstable angina.
  - Post-myocardial infarction state – with history of coronary bypass graft or percutaneous coronary intervention with or without stenting or with history of medical management either with fibrinolytics or with heparins.
  - Chronic ischemic heart disease-evidence from coronary angiogram or from a positive stress test.

Exclusion criteria:

- The patients who fall outside the specified age group that will be <18 and >40years of age.
- Pregnant females will be excluded due to their hypercoaguable state.
- Hyperhomocysteinemia.
- Patients who are not on follow up and their past medical records not available.

Methods:

A detail history of all the subjects included in the study group was obtained from the available hospital data or on follow-up with special emphasis on Family History & History of major diseases (Hypertension, Diabetes, Ischemic heart disease) & Socioeconomic History, Sedentary Habits.

A detail history of smoking with number &/or pack's/ day & duration of smoking was obtained from all the study subjects on follow-up or from the available data.

A detailed physical examination with special emphasis on body mass index was done on follow-up visit to the hospital or available hospital data of study subjects. Routine haemogram, Fasting blood sugar (FBS), Lipid profile, resting 12 lead ECG, CAG finding were collected from the hospital data of all subjects.

The height of patient was measured in centimeter and weight in kilogram. BMI was calculated by weight in kg / height in sq. meter.

All the measured parameters were statistically analyzed using Pearson's correlation and Chi-square test.

The criteria for defining the presence of risk factors for CAD were used as mentioned in the below [(9)].

Criteria for Defining Risk Factor for CAD

Risk factors		**Criteria**
Smoking	:	correlated with smoking pack years
Generalized obesity	:	Body mass index (weight in kg/height^2 in cm) = 27 in males and 25 in females
Sedentary habits	:	Subject walking less than 14.5km/week
Socioeconomic Class	:	According to the BG Prasad's original classification
Family History	:	Of IHD
Hypertension	:	History, Blood pressure=140/90 mmHg (JNC VI criteria)
Diabetes mellitus	:	Known case, Plasma Sugar (fasting) level of =126 mg%
Dyslipidemia	:	Serum LDL=>100 mg%
	:	Serum triglyceride =>150 mg%
	:	Serum cholesterol=>200
	:	Serum HDL=<35
	:	Serum VLDL=>30

RESULTS

The present study carried out in Dhiraj hospital, Piparia, Vadodara and data was collected for study from January 2013 to June 2015. 63 individuals falling in specified criteria were included in the study.

Table 1: Sex distribution of study group (n=63)

SEX	No. of persons	%
Female	11	17.46
Male	52	82.54
TOTAL	**63**	**100.0**

As shown in table 1 there were 52 male & 11 female patients in the study.

SMOKING

Table 2: Sex wise distribution of smoking as a risk factor (n=63)

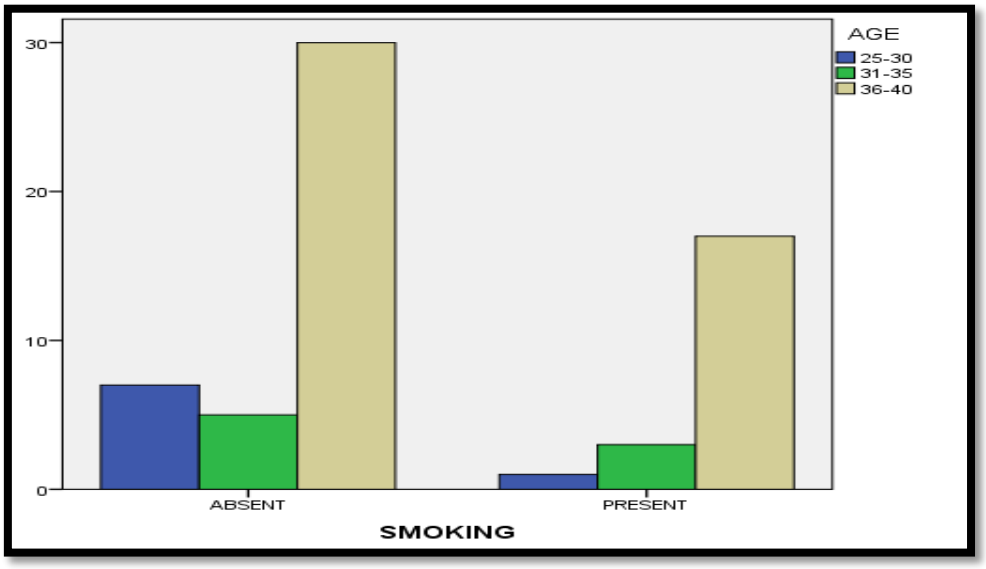
SMOKING	FEMALE	MALE	TOTAL	%
ABSENT	11	31	42	66.66
PRESENT	0	21	21	33.34
TOTAL	**11**	**52**	**63**	100

It was found that 33.34% of patients had smoking as the risk factor as shown in table 2, all of them were males.

Table 3: Age wise distribution of smoking as a risk factor in males (n=52)

		SMOKING		TOTAL	% (Present)
SEX	**AGE GROUP**	**ABSENT**	**PRESENT**		
M	18-24	0	0	0	0
	25-30	7	1	8	12.5
	31-35	5	3	8	37.5
	36-40	30	17	47	36.2
	TOTAL	**42**	**21**	**63**	

Graph 1: Age wise distribution of smoking as a risk factor in males



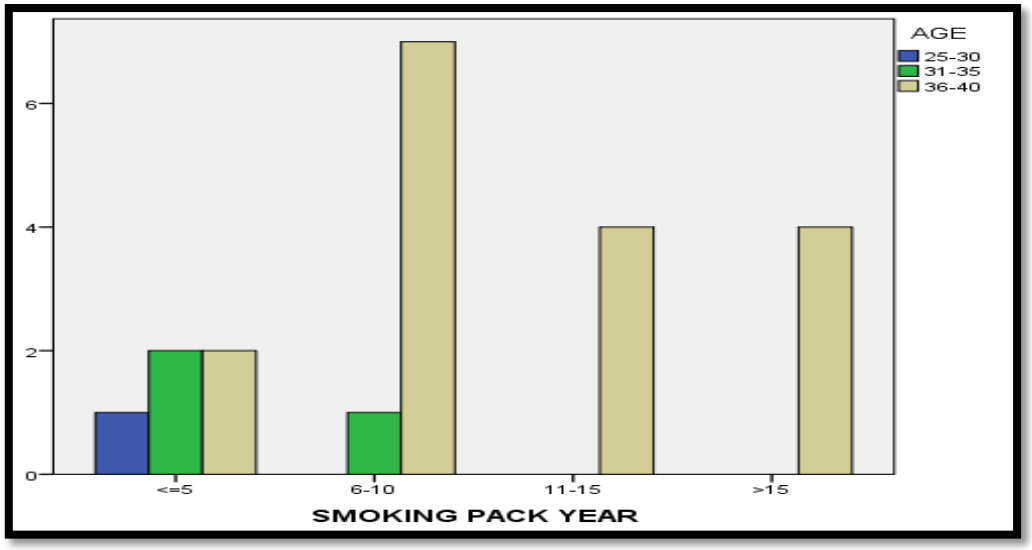
Most of these males belonged to the age group of 31-40 years as shown in table 3 & graph 1, while there were no smokers in the age group of 18– 24 yrs.

Smoking when considered sex wise, was a significant risk factor, and P <0.05. It was not statistically significant factor (P value=- 0 .40) when age wise distribution was considered.

Table 4: Distribution of smokers according to pack years (n=21)

			AGE			Total
			25-30	31-35	36-40	
SMOKING PACK YEAR	<=5	Count	1	2	2	5
		% within AGE	100.0%	66.7%	11.8%	23.8%
	6-10	Count	0	1	7	8
		% within AGE	0.0%	33.3%	41.2%	38.1%
	11-15	Count	0	0	4	4
		% within AGE	0.0%	0.0%	23.5%	19.0%
	>15	Count	0	0	4	4
		% within AGE	0.0%	0.0%	23.5%	19.0%
Total		Count	1	3	17	21
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 2: Distribution of smokers according to pack years in Male



It was found that, majority of the smokers belonged to 36 to 40years of age who had 6-10 or more pack per years of smoking as shown in table 4 & graph 2.

SEDENTARY LIFE STYLE

Table 5: Sex wise distribution of sedentary life style as a risk factor (n=63)

SEDANTARY LIFE	FEMALE	MALE	TOTAL	%
ABSENT	3	47	50	79.36
PRESENT	8	5	13	20.64
TOTAL	**11**	**52**	**63**	**100**

It was found that 20.64% of the people had sedentary life style as a risk factor, of which 8 were females & 5 were males (Table 5 & Graph 3).

Graph 3: Sex wise distribution of sedentary life style as a risk factor

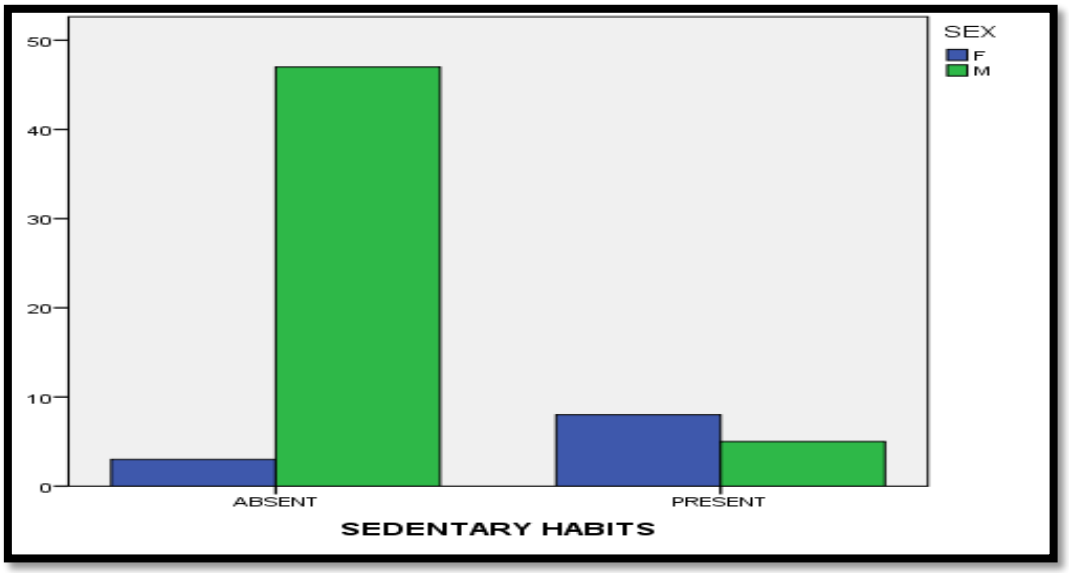
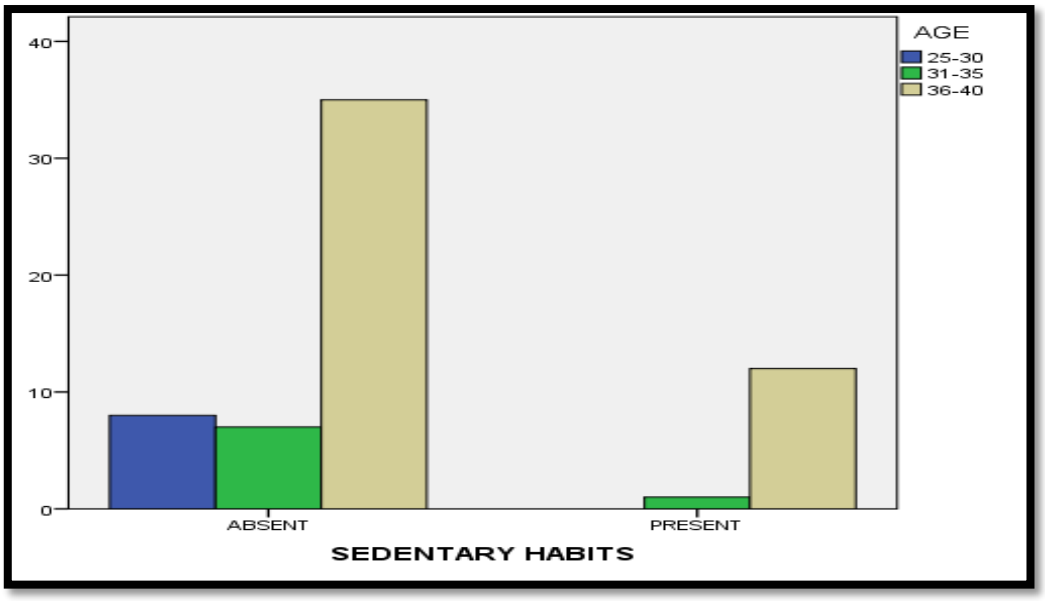


Table 6: Age wise distribution of sedentary life style as a risk factor (n=13)

			AGE			Total
			25-30	31-35	36-40	
SEDENTARY HABITS	Absent	Count	8	7	35	50
		% within AGE	100.0%	87.5%	74.5%	79.4%
	Present	Count	0	1	12	13
		% within AGE	0.0%	12.5%	25.5%	20.6%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

As observed in Table 6 & Graph 4, most of the subjects with sedentary lifestyle as a risk factor belonged to the age group of 36 – 40 years.

Graph 4: Age wise distribution of sedentary life style as a risk factor



In the present study 20.6 % individuals had sedentary life style, out of which 5 were males and 8 were females. The sedentary life style as a risk factor for CAD was statistically insignificant when considered age group wise, as the chi square value is 3.091 and P value is 0.213. However, it is a significant risk factor when considered sex wise with chi square = 22.082 and P value <0.001.

GENERALISED OBESITY

Table 7: Age wise distribution of generalized obesity as a risk factor (n=63)

G/O	FEMALE	MALE	TOTAL	%
ABSENT	8	32	40	63.49
PRESENT	3	20	23	36.51
TOTAL	**11**	**52**	**63**	**100**

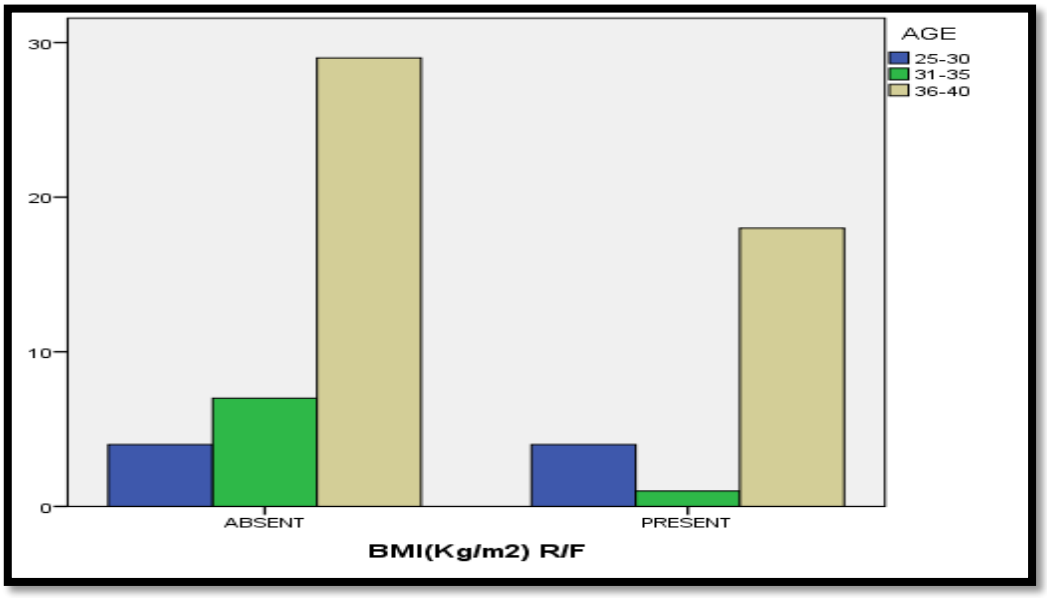
23 out of the 63 patients were having generalized obesity; of these 3 were females & 20 were males as shown in table 7.

Table 8: Age wise distribution of generalized obesity as a risk factor (n=23)

			AGE			Total
			25-30	31-35	36-40	
Generalized Obesity	Absent	Count	4	7	29	40
		% within AGE	50.0%	87.5%	61.7%	63.5%
	Present	Count	4	1	18	23
		% within AGE	50.0%	12.5%	38.3%	36.5%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Out of the total number of patients, majority of the patients having a general obesity were between the age group 36 – 40 years as shown in table 8 & graph 5.

Graph 5: Age wise distribution of generalized obesity as a risk factor



Generalized obesity did not show any statistically significant difference in age groups of the study, as the chi square = 2.682 and P value = 0.262 at 5% level i.e. P<0.05. It is also not a significant risk factor when considered sex wise as chi square = 0.490 and P= 0.484.

HYPERTENSION

Table 9: Sex wise distribution of hypertension as a risk factor (n= 63)

HTN	FEMALE	MALE	TOTAL	%
ABSENT	9	45	54	85.71
PRESENT	2	7	9	14.29
TOTAL	**11**	**52**	**63**	100

Table 9 & Graph 6 shows that there were 9 patients had hypertension out of which 7 were males and 2 were females.

Graph 6: Sex wise distribution of hypertension as a risk factor

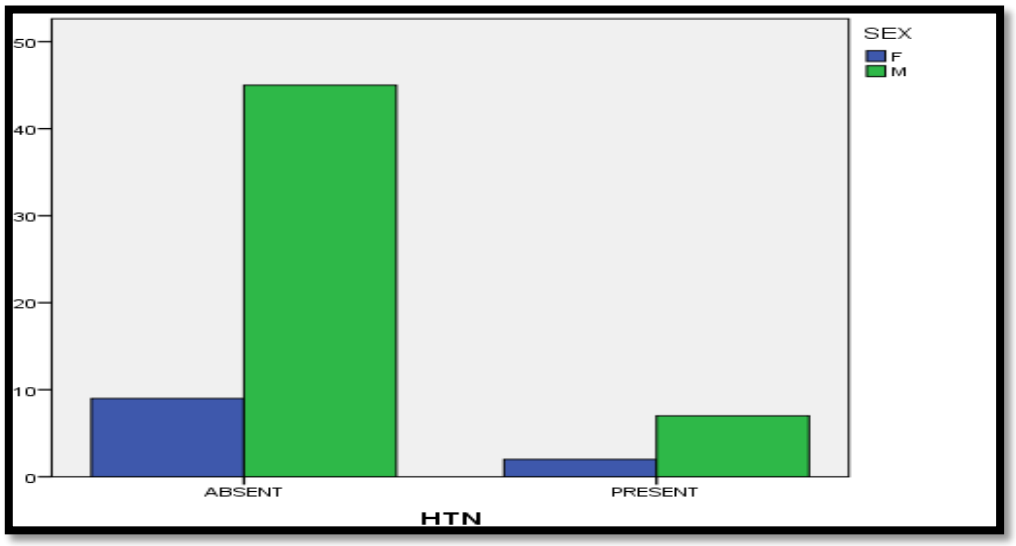
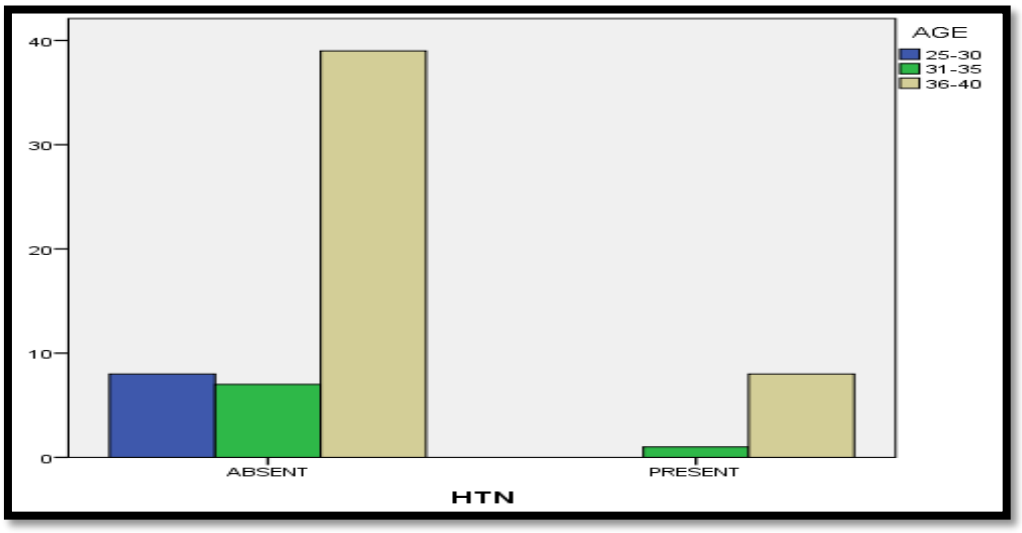


Table 10: Age wise distribution of hypertension as a risk factor (n= 9)

			AGE			Total
			25-30	31-35	36-40	
HTN	ABSENT	Count	8	7	39	54
		% within AGE	100.0%	87.5%	83.0%	85.7%
	PRESENT	Count	0	1	8	9
		% within AGE	0.0%	12.5%	17.0%	14.3%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Out of the total number of patients, majority of the hypertensive patients were between the age group 36-40yrs (Table 10 & Graph 7).

Graph 7: Age wise distribution of Hypertension as a risk factor



Hypertension was not a significant risk factor in the study group when considered in age group with chi square value = 1.641 and P= 0.440 i.e. P>0.05 at 5% level, it was also not a significant risk factor when considered sex wise with chi square value being 0.165 and P = 0.684.

DIABETES MELLITUS

Table 11: Sex wise distribution of past history of Diabetes as a risk factor (n= 63)

DM	FEMALE	MALE	TOTAL	%
ABSENT	9	48	57	90.47
PRESENT	2	4	6	9.52
TOTAL	**11**	**52**	**63**	100

In the present study there were 6 patients detected to be diabetic out of which 4 were males and 2 were females as shown in Table 11 & Graph 8.

Graph 8: Sex wise distribution of Diabetes as a risk factor

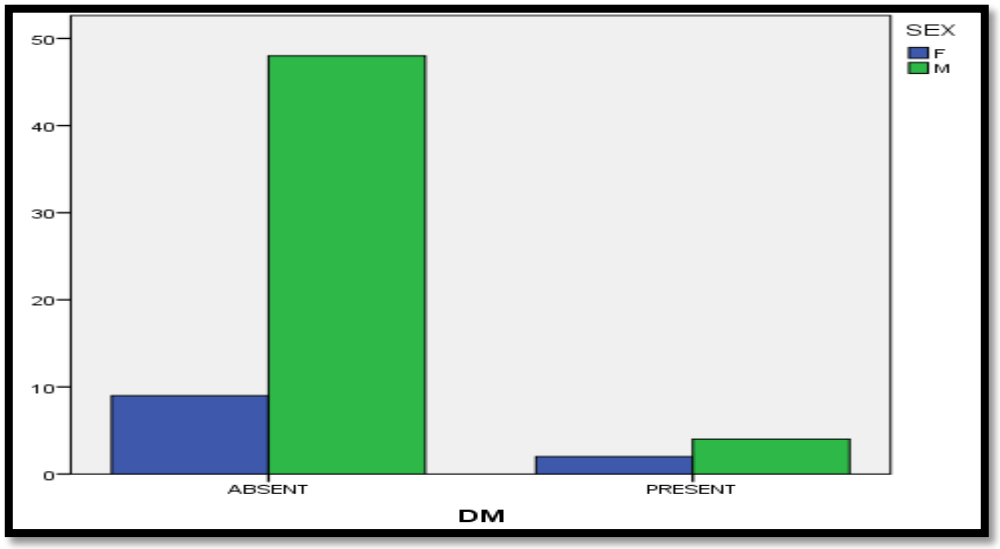
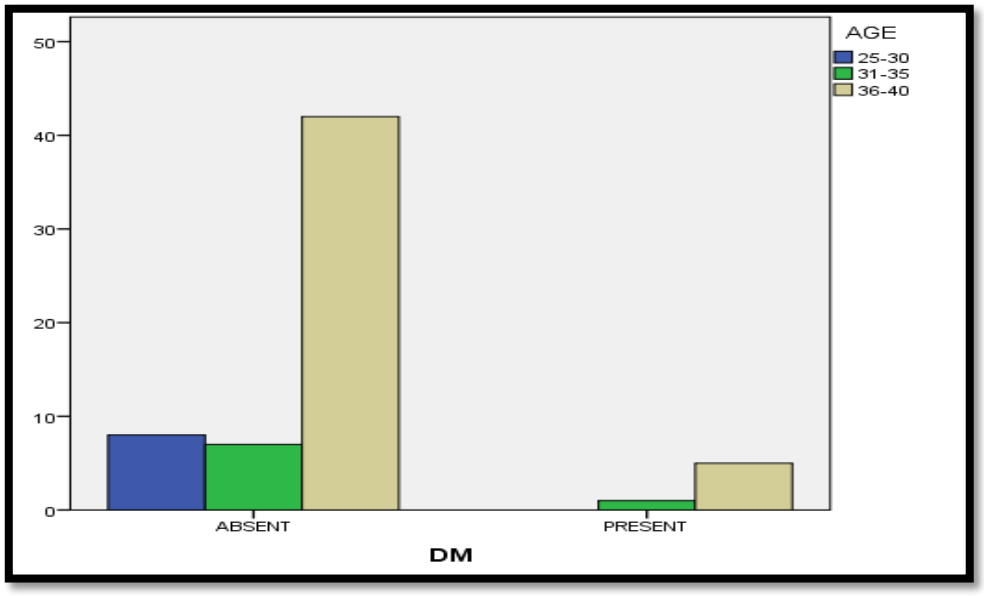


Table 12: Age wise distribution of Diabetes as a risk factor (n= 6)

			AGE			Total
			25-30	31-35	36-40	
DM	Absent	Count	8	7	42	57
		% within AGE	100.0%	87.5%	89.4%	90.5%
	Present	Count	0	1	5	6
		% within AGE	0.0%	12.5%	10.6%	9.5%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0 %	100.0%

Out of the total number of patients, majority of the diabetic patients were between the age group 36-40yrs (Table 12 & Graph 9).

Graph 9: Age wise distribution of Diabetes as a risk factor



Diabetes was not a significant risk factor in the study group when considered in age group with chi square value = 0.992 and P= 0.609 i.e. P>0.05 at 5% level, it was also not a significant risk factor when considered sex wise with chi square value being 1.159 and P = 0.282.

DYSLIPIDEMIA

Table 13: Sex wise distribution of Dyslipidemia as a risk factor (n=63)

DYSLIPIDIEMIA	FEMALE	MALE	TOTAL	%
ABSENT	2	2	4	25.20
PRESENT	9	50	59	74.80
TOTAL	**11**	**52**	**63**	100

59 out of 63 of the studied patients had dyslipidemia, of which 50 were males and 9 were females (Table 13).

TOTAL CHOLESTEROL

Table 14: Sex wise distribution of Total Cholesterol as a risk factor (n=9)

			SEX		Total
			F	M	
TC	<200	Count	9	45	54
		% within SEX	81.8%	86.5%	85.7%
	>200	Count	2	7	9
		% within SEX	18.2%	13.5%	14.3%
Total		Count	11	52	63
		% within SEX	100.0%	100.0%	100.0%

Graph 10: Sex wise distribution of Total Cholesterol as a risk factor

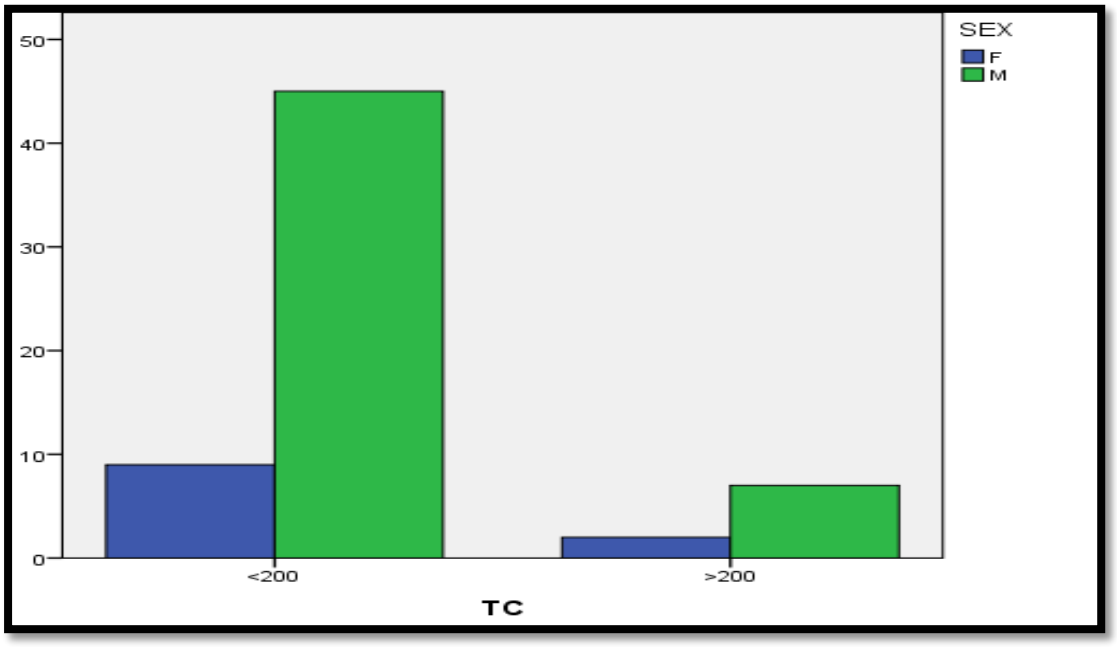
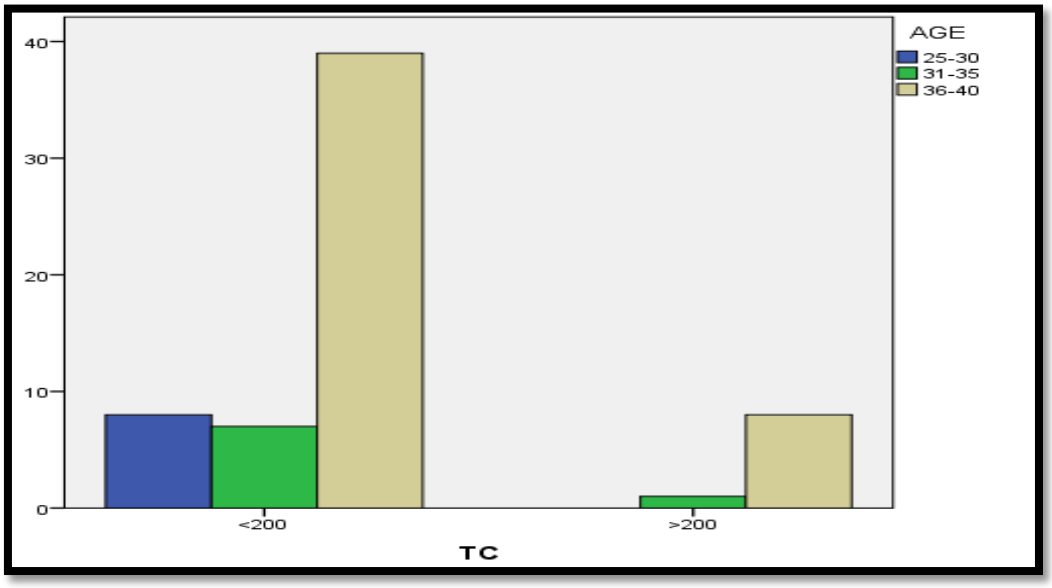


Table 14 & Graph 10 shows that Total cholesterol as a risk factor was present in 7 out of 52 males and 2 out of 11 females.

Table 15: Age wise distribution of Total Cholesterol as a risk factor (n=9)

			AGE			Total
			25-30	31-35	36-40	
TC	<200	Count	8	7	39	54
		% within AGE	100.0%	87.5%	83.0%	85.7%
	>200	Count	0	1	8	9
		% within AGE	0.0%	12.5%	17.0%	14.3%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 11: Age wise distribution of Total Cholesterol as a risk factor



Total cholesterol was a major risk factor in age group of 36-40years as shown in Table 15 and Graph 11.

Total cholesterol was not a significant risk factor in the study group when considered in age group with chi square value = 1.641 and P= 0.440 i.e. P>0.05 at 5% level, it was also not a significant risk factor when considered sex wise with chi square value being 0.165 and P = 0.684.

LOW DENSITY LIPOPROTEIN

Table 16: Sex wise distribution of low density lipoprotein (n=30)

			SEX		Total
			F	M	
LDL	<100	Count	5	28	33
		% within SEX	45.5%	53.8%	52.4%
	>=100	Count	6	24	30
		% within SEX	54.5%	46.2%	47.6%
Total		Count	11	52	63
		% within SEX	100.0%	100.0%	100.0%

Graph 12: Sex wise distribution of Low Density Lipoprotein as a risk factor

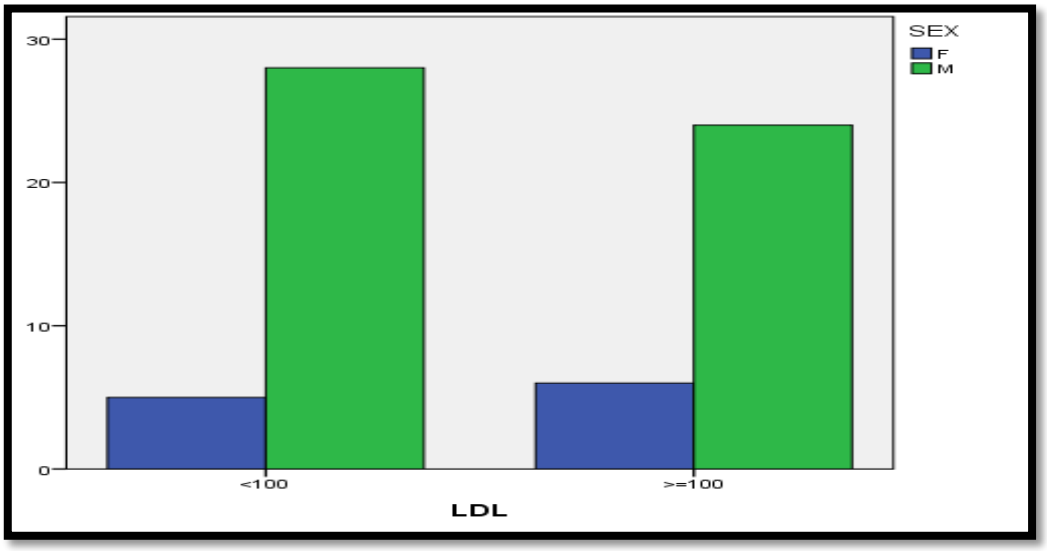
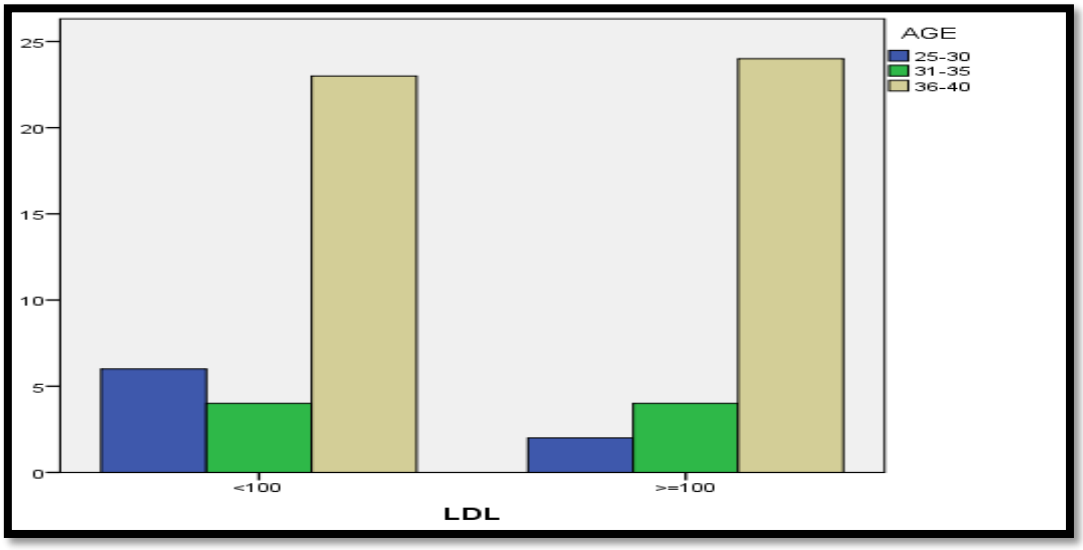


Table 16 & Graph 12 shows that, Low Density Lipoprotein was present as a risk factor in 6 females and 24 males.

Table 17: Age wise distribution of low density lipoprotein (n=30)

			AGE			Total
			25-30	31-35	36-40	
LDL	<100	Count	6	4	23	33
		% within AGE	75.0%	50.0%	48.9%	52.4%
	>=100	Count	2	4	24	30
		% within AGE	25.0%	50.0%	51.1%	47.6%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 13: Age wise distribution of low density lipoprotein



Low density lipoprotein was a major risk factor in age group of 36-40years (Table 17 & Graph 13).

Low density lipoprotein was not a significant risk factor in the study group when considered in age group with chi square value = 1.883 and P= 0.390 i.e. P>0.05 at 5% level, it was also not a significant risk factor when considered sex wise with chi square value being 0.256 and P = 0.613.

HIGH DENSITY LIPOPROTEIN

Table 18: Sex wise distribution of high density lipoprotein (n=31)

			SEX		Total
			F	M	
HDL	>35	Count	4	28	32
		% within SEX	36.4%	53.8%	50.8%
	<=35	Count	7	24	31
		% within SEX	63.6%	46.2%	49.2%
Total		Count	11	52	63
		% within SEX	100.0%	100.0%	100.0%

Graph 14: Sex wise distribution of high density lipoprotein

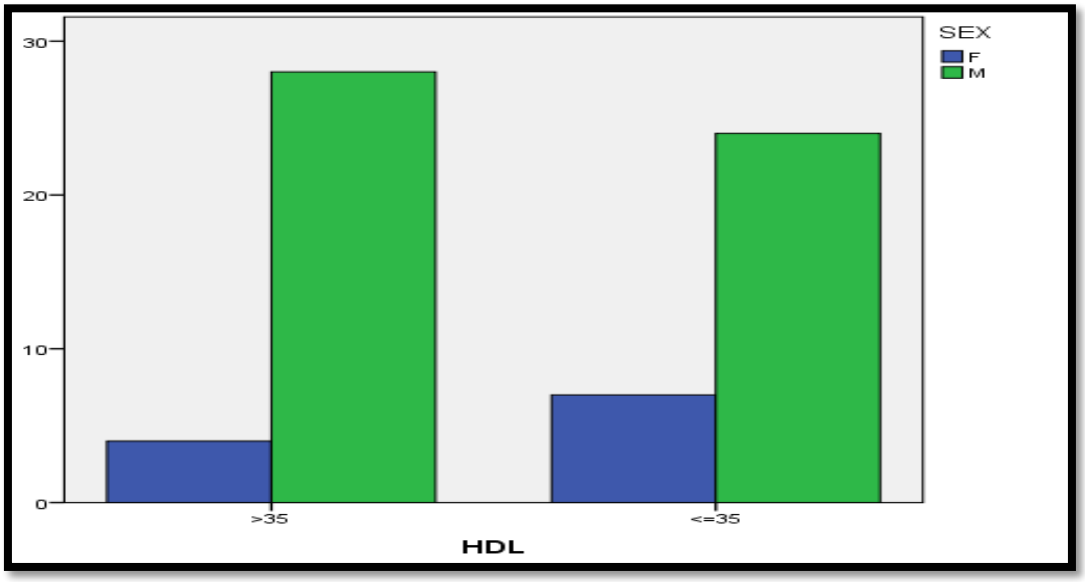
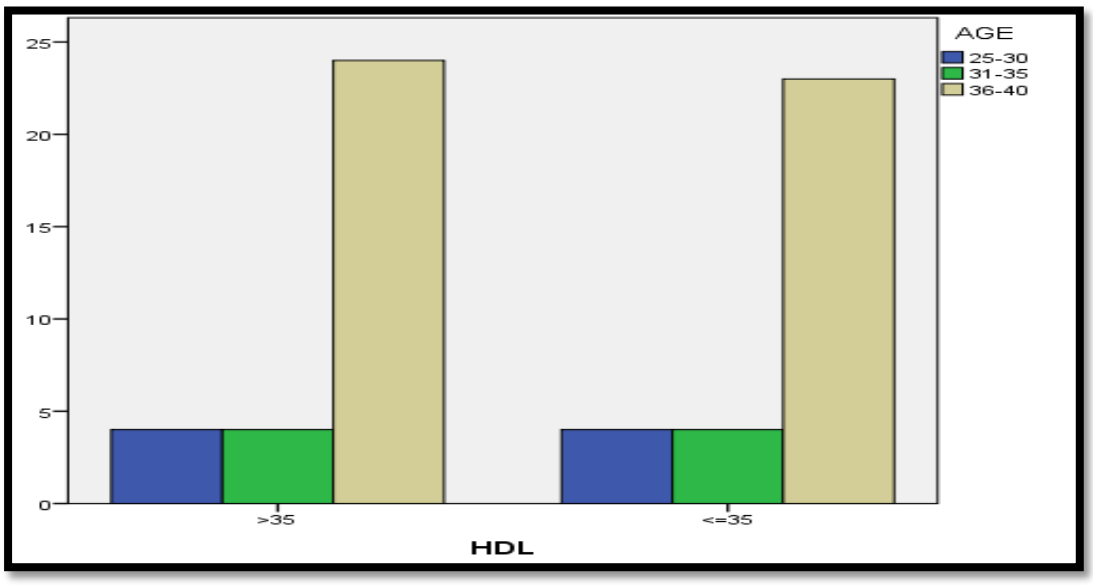


Table 18 & graph 14 shows that HDL as a risk factor was present in 24 males and 7 females.

Table 19: Age wise distribution of High Density Lipoprotein (n=31)

			AGE			Total
			25-30	31-35	36-40	
HDL	>35	Count	4	4	24	32
		% within AGE	50.0%	50.0%	51.1%	50.8%
	<=35	Count	4	4	23	31
		% within AGE	50.0%	50.0%	48.9%	49.2%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 15: Age wise distribution of High Density Lipoprotein



High density lipoprotein was a major risk facror in age group of 36-40years (Table 19 & Graph 15).

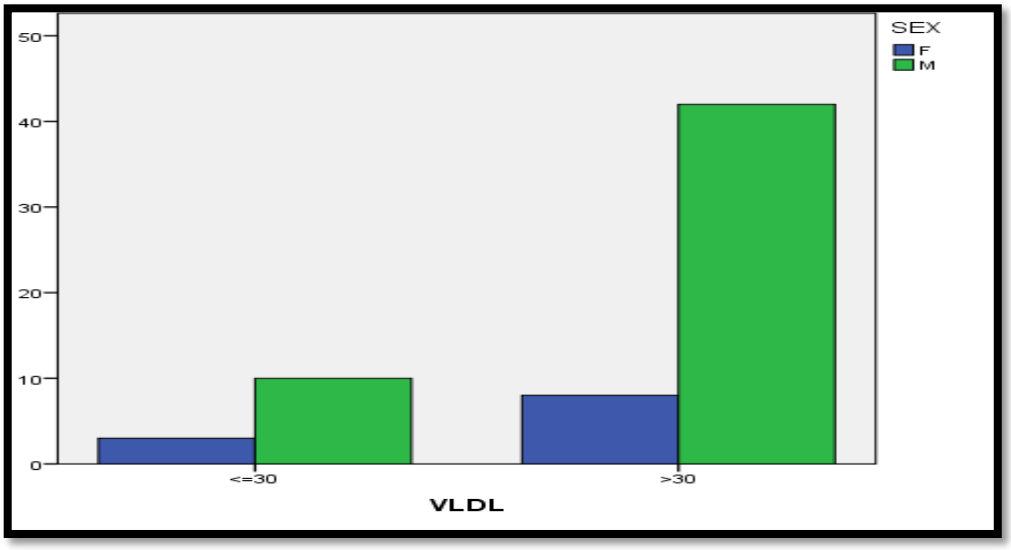
High density lipoprotein was not a significant risk factor in the study group when considered in age group with chi square value = 0.005 and P= 0.997 i.e. P>0.05 at 5% level, it was also not a significant risk factor when considered sex wise with chi square value being 1.110 and P = 0.292.

VERY LOW DENSITY LIPOPROTEIN

Table 20: Sex wise distribution of Very Low Density Lipoprotein (n=50)

			SEX		Total
			F	M	
VLDL	<=30	Count	3	10	13
		% within SEX	27.3%	19.2%	20.6%
	>30	Count	8	42	50
		% within SEX	72.7%	80.8%	79.4%
Total		Count	11	52	63
		% within SEX	100.0%	100.0%	100.0%

Graph 16: Sex wise distribution of Very Low Density Lipoprotein

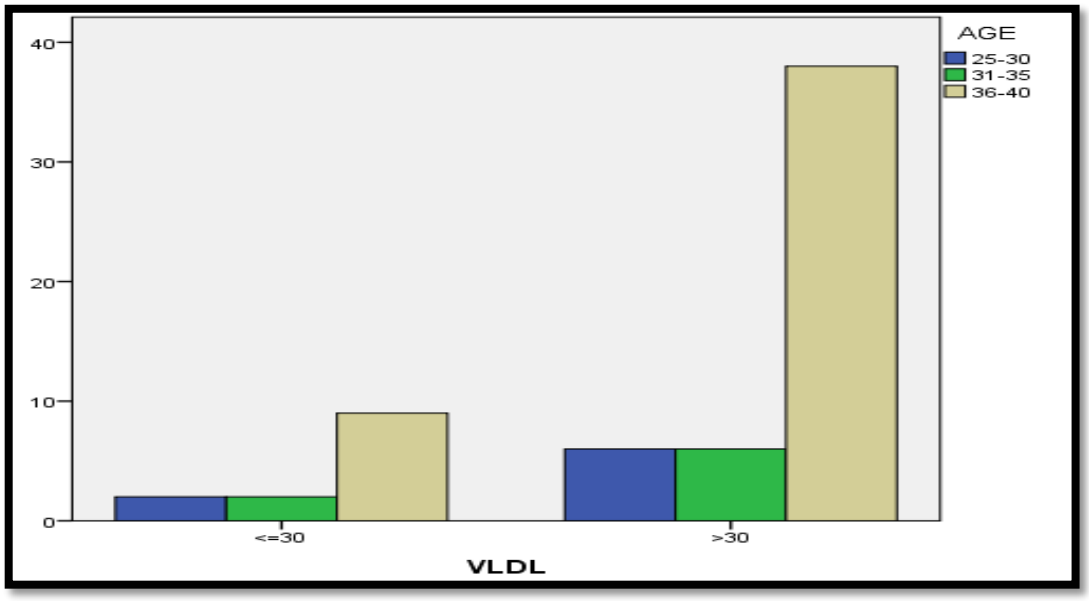


VLDL as a risk factor was present in 42 out of 52 males and 8 out of 11 females (Table 20 & Graph 16).

Table 21: Age wise distribution of Very Low Density Lipoprotein (n=50)

			AGE			Total
			25-30	31-35	36-40	
VLDL	<=30	Count	2	2	9	13
		% within AGE	25.0%	25.0%	19.1%	20.6%
	>30	Count	6	6	38	50
		% within AGE	75.0%	75.0%	80.9%	79.4%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 17: Age wise distribution of Very Low Density Lipoprotein



Very low density lipoprotein was a major risk factor in age group of 36-40years as shown in Table 21 and Graph 17.

Very low density lipoprotein was not a significant risk factor in the study group when considered in age group with chi square value = 0.250 and P= 0.883 i.e. P>0.05 at 5% level, it was also not a significant risk factor when considered sex wise with chi square value being 0.359 and P = 0.549.

TRIGLYCERIDES

Table 22: Sex wise distribution of Triglycerides (n=19)

			SEX		Total
			F	M	
TGS	<150	Count	10	34	44
		% within SEX	90.9%	65.4%	69.8%
	>=150	Count	1	18	19
		% within SEX	9.1%	34.6%	30.2%
Total		Count	11	52	63
		% within SEX	100.0%	100.0%	100.0%

Graph 18: Sex wise distribution of Triglycerides

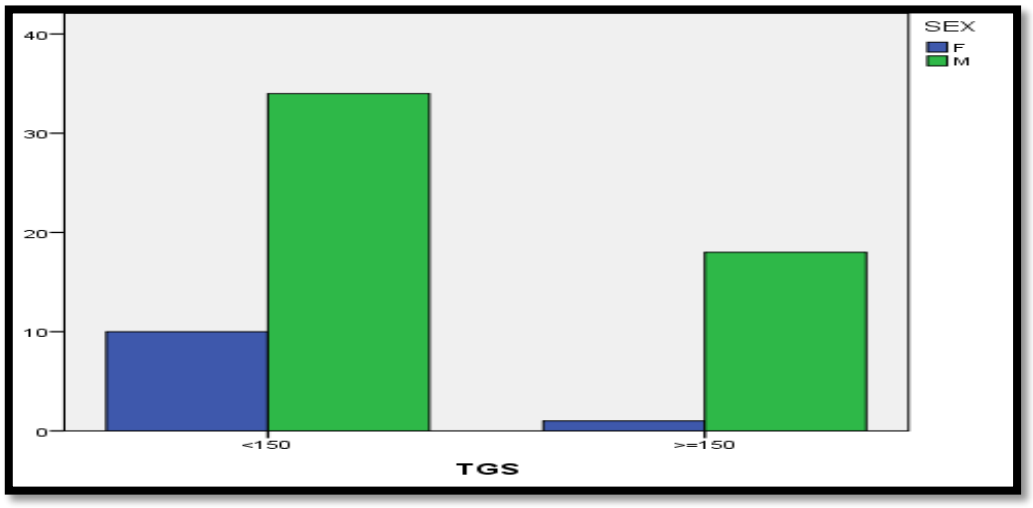
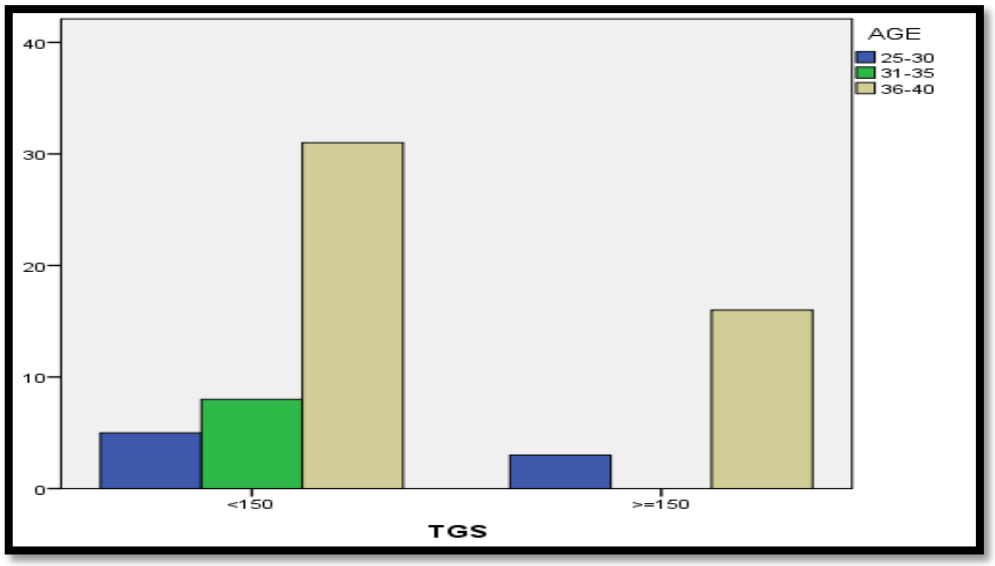


Table 22 & Graph 18 shoes that Triglycerides as a risk factor was present in 18 males and 1 female.

Table 23: Age wise distribution of Triglycerides (n=19)

			AGE			Total
			25-30	31-35	36-40	
TGS	<150	Count	5	8	31	44
		% within AGE	62.5%	100.0%	66.0%	69.8%
	>=150	Count	3	0	16	19
		% within AGE	37.5%	0.0%	34.0%	30.2%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 19: Age wise distribution of Triglycerides



Triglycerides was a major risk factor in age group of 36-40years (Table 23 & Graph 19).

Triglycerides was not a significant risk factor in the study group when considered in age group with chi square value = 3.996 and P= 0.136 i.e. P>0.05 at 5% level, it was also not a significant risk factor when considered sex wise with chi square value being 2.808 and P = 0.094.

FAMILY HISTORY OF ISCHEMIC HEART DISEASE

Table 24: Sex wise distribution of family history of IHD as a risk factor (n=63)

F/H	FEMALE	MALE	TOTAL	%
ABSENT	5	28	33	52.38
PRESENT	6	24	30	47.62
TOTAL	**11**	**52**	**63**	100

Family history of IHD as a risk factor was present in 30 patients of which 6 were females and 24 were males as shown in Table 24 & Graph 20.

Graph 20: Sex wise distribution of family history of IHD as a risk factor

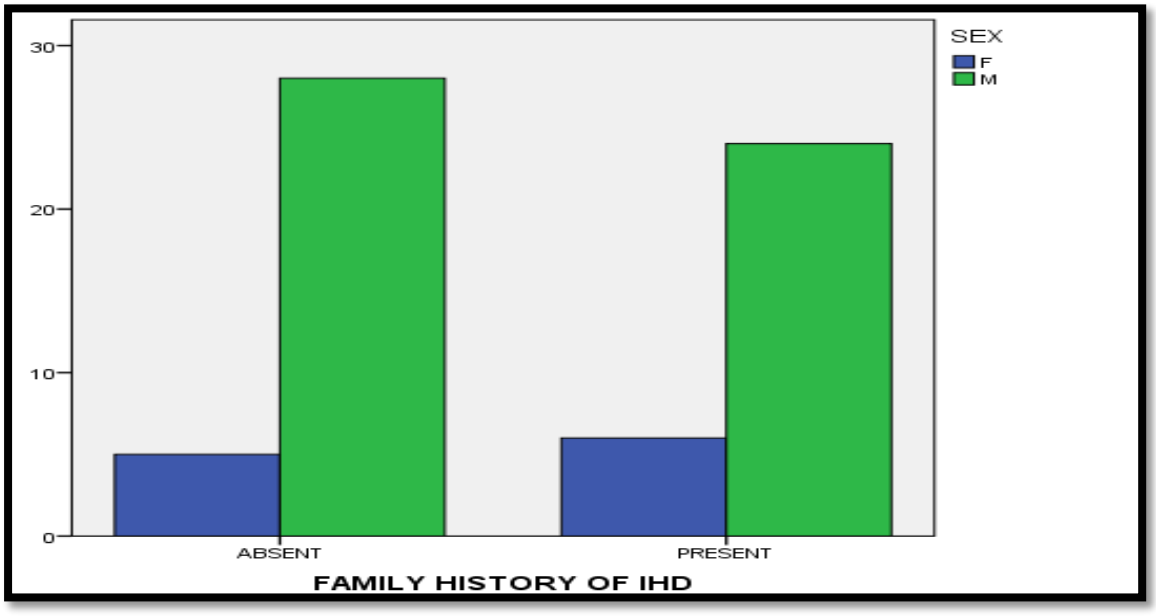


Table 25: Age wise distribution of family history of IHD as a risk factor (n=30)

			AGE			Total
			25-30	31-35	36-40	
Family History Of IHD	Absent	Count	1	4	28	33
		% within AGE	12.5%	50.0%	59.6%	52.4%
	Present	Count	7	4	19	30
		% within AGE	87.5%	50.0%	40.4%	47.6%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 21: Age wise distribution of family history of IHD as a risk factor

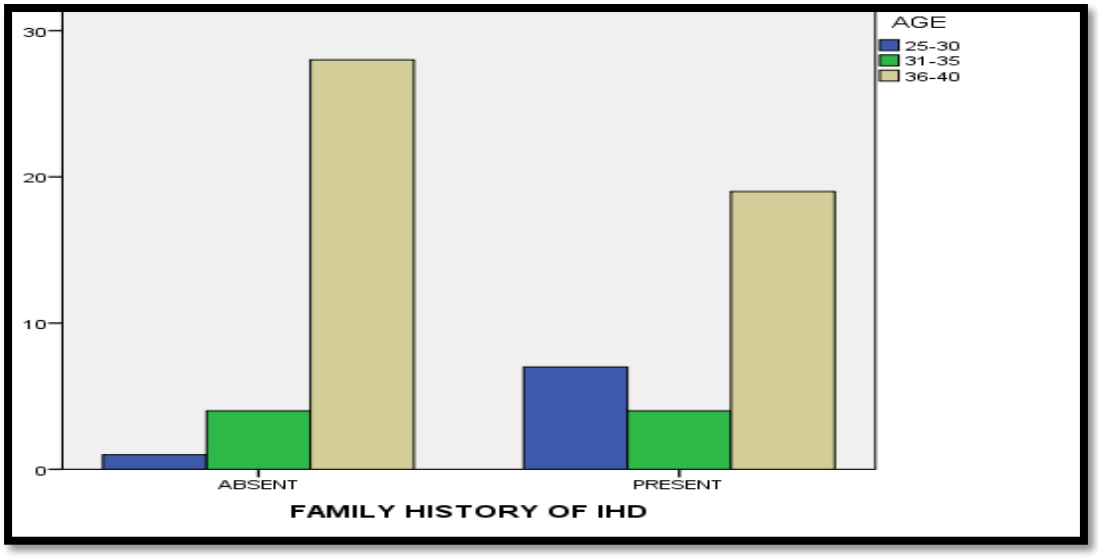


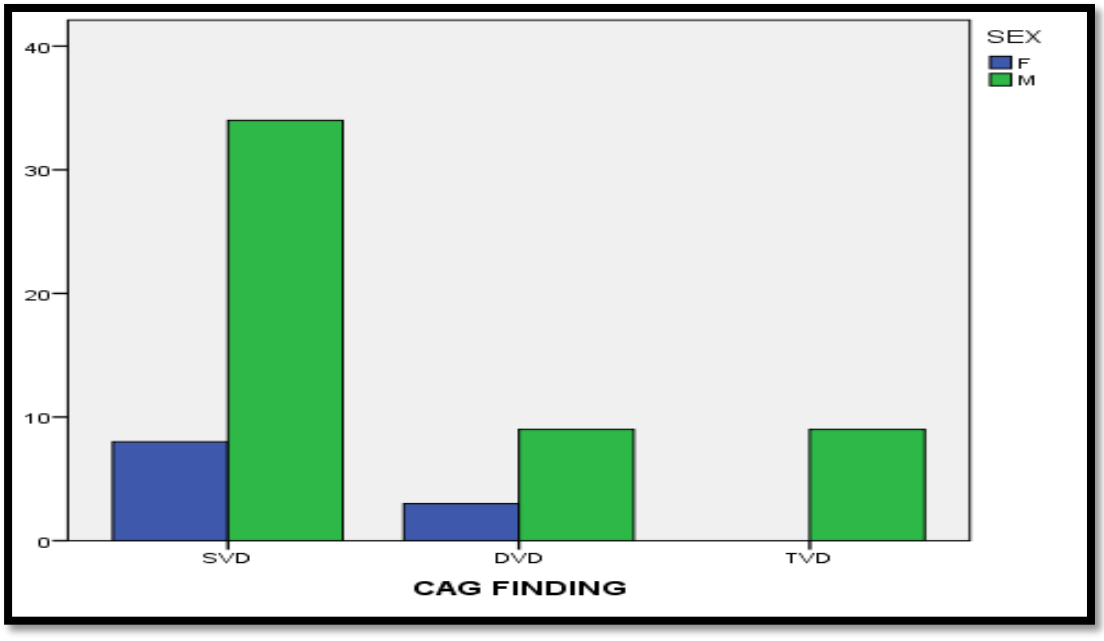
Table 25 & Graph 21 shows that family history was present in 24 of the 52 males , most of which belonged to the 36 – 40 years age group , no patients in the age group of 18 – 24 had family history as the risk factor.

CAG FINDING

Table 26: Sex wise distribution of CAG finding (n=63)

			SEX		Total
			F	M	
CAG FINDING	SVD	Count	8	34	42
		% within SEX	72.7%	65.4%	66.7%
	DVD	Count	3	9	12
		% within SEX	27.3%	17.3%	19.0%
	TVD	Count	0	9	9
		% within SEX	0.0%	17.3%	14.3%
Total		Count	11	52	63
		% within SEX	100.0%	100.0%	100.0%

Graph 22: Sex wise distribution of CAG finding (n=63)

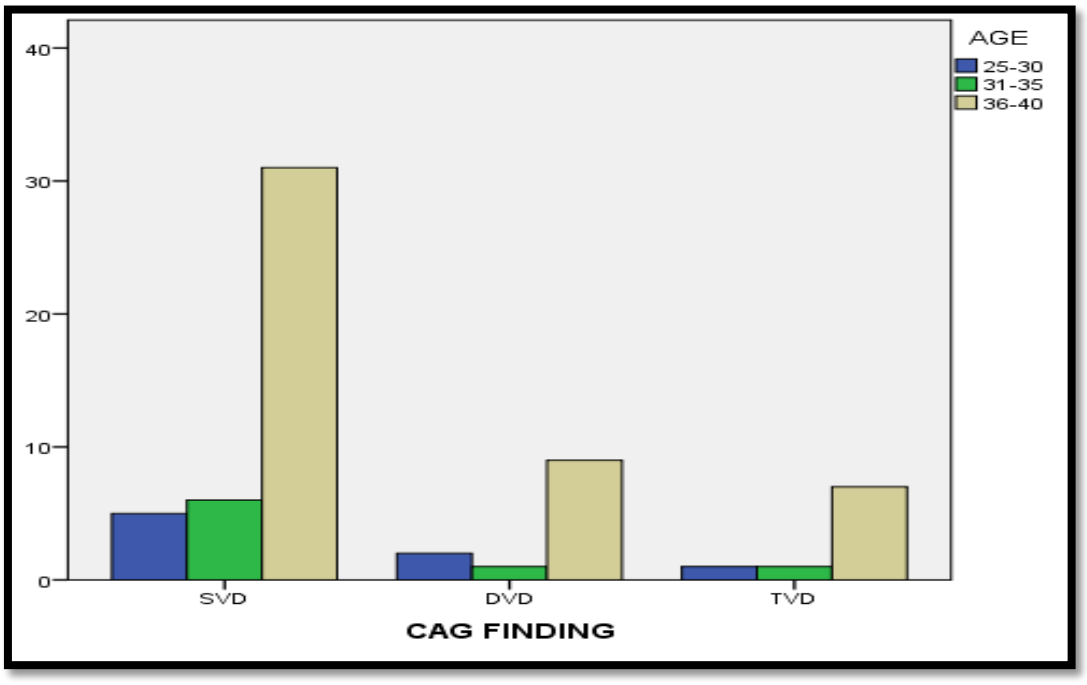


As shown in Table 26 & Graph 22, Single vascular disease was found in 8 females and 34 males, Double vascular disease was found in 3 females and 9 males and 9 males were found who had Triple vascular disease.

Table 27: Age wise distribution of CAG finding (n=63)

			AGE			Total
			25-30	31-35	36-40	
CAG FINDING	SVD	Count	5	6	31	42
		% within AGE	62.5%	75.0%	66.0%	66.7%
	DVD	Count	2	1	9	12
		% within AGE	25.0%	12.5%	19.1%	19.0%
	TVD	Count	1	1	7	9
		% within AGE	12.5%	12.5%	14.9%	14.3%
Total		Count	8	8	47	63
		% within AGE	100.0%	100.0%	100.0%	100.0%

Graph 23: Age wise distribution of CAG finding (n=63)



As shown in Table 27 & Graph 23, CAG finding was more common in 36-40 years of age group.

DISCUSSION

The prevalence of coronary heart disease has been tremendously increasing in last decade. In this study there were 82% males compared to 17% females.

Smoking: (Table 2, 3 & 4)

Smoking is important and modifiable risk factor known for CAD**.**

In present study smoking was present in 33.34 % of the study group. All the smokers were males & most of them belonged to the age group of 31 – 40.

A little higher prevalence of 40 & 42.4% of smoking had been reported by H S Ressam et al (2003) [25] & Gupta R et al (1994) [23] and 63 % smoking had been reported by various other workers. (Naeman et al 2007[6], S Diwedi et al 2000 [8], & Arvind Kumar et al 2005 [9]).

Mahilmaran (2005) [21] & R Panchal et al (2005) [17] had reported a low prevalence (17 % & 8.9 %) of smoking in study group of their studies.

Sedentary life style: (Table 5 & 6)

Sedentary life style was found in 20.64 % of study group in the present study.

T N Sugath et al (2008) [15] reported almost similar findings of 23 % cases with sedentary life style in their respective studies.

A higher prevalence of 63.5 % had been reported by R Panchal et al (2005) [17] in their study. Gupta et al (1994) [23] had also reported almost similar findings of 69.3 % prevalence in their study.

Arvind Kumar et al (2005) [9] found 80 % of the study group having sedentary life style as risk factor for CAD.

Generalized Obesity: (Table 7 & 8)

Higher BMI was documented in 36.51% of patients. Twenty three were obese. There was no statistical difference comparing males with females.

Our findings match with those of other investigator as shown in below:

STUDY	PERCENT
Present study	36.51 %
K Rmaiya et al (1991) [20]	13.3
Sangeeta et al (2004) [22]	15.4
Gupta R et al (1994) [23]	20.8

However generalized obesity was present in only 4.40 % of the study group, in a study done by Arvind Kumar et al (2005) [9].

Sangeeta et al (2004) [22] reported higher prevalence of 48.5 % of generalized obesity in their study.

Hypertension: (Table 9 & 10)

In present study 14.29 % of the subjects had hypertension & this finding is not consistent with most of other studies.

Sangeeta et al (2000) [22] had reported low prevalence of hypertension (5.8 %) in their study.

However a higher prevalence (51.42 % & 51.4 %) had been reported by S Diwedi et al (2000) [8] & Noeman A et al (2007) [6] in their studies respectively.

S Diwedi et al (2000) [8] also found 41 % of the study group to be hypertensive.

Diabetes mellitus (Table 11 & 12)

Diabetes mellitus was noted in 9.52%. The disease is more prevalent in males compared to females and in older patients as compared to younger patients. This fact has been documented in a number of previous studies. Sangeeta et al (2004) [22] & K L Ramaiya et al (1991) [20] reported 9.8 % of cases to be diabetic in their studies. Our results are similar to these with 9 % of study population showing history of Diabetes.

S Diwedi et al (2000) [8] & Naeman et al (2007) [6] reported 7.14 % of their cases to be diabetic in the study group, similar results are reported by Vivek Singh et al (2006)[23].

However a low prevalence of only 4 % had been reported by Gupta R et al (1994) [23].

Dyslipidiemia: (Table 13 to 23)

Increased cholesterol was noted in 9 (14.3%) patients. Hypercholesterolemia mainly increased LDL, VLDL and decreased HDL are important risk factors.

In the present study, dyslipidiemia was found to be present in 74.80 % of the studied cases.

However, Noeman et al (2007) [6] & S Diwedi et al (2000) [8] reported a prevalence of dyslipidemia in 41.66 % of their subjects .Vincent Jomini (2002) [28], also reported a prevalence of dyslipidemia (33 %) in their study group.

Family History of IHD: (Table 24 & 25)

A positive family history of IHD was found in 47.62 % of cases in our study.

The other workers have reported a lower prevalence (18.8 % & 6.2 %) of family history in their studies (Noeman et al (2007) [6], R Panchal et al (2005) [17]. While S Diwedi et al (2000) [8] found 42.8 % of their study group with positive family history.

SUMMARY & CONCLUSION

The study was conducted on 63 people who were randomly selected, these people were between 18 – 40 years of age. The mean age of the subjects was 38.91 years.

Smoking was present in 33.33 % of the subjects. All the smokers in the study were males. Sedentary lifestyle as a risk factor was present in 20.64 % of the subjects. Family history of coronary artery disease was present in 47.62 % of the subjects. Generalized obesity was present in 36.51 % of our subjects. In the study, 74.8 % of the subjects had dyslipidemia as a risk factor. Increased cholesterol was noted in 9 (14.3%) patients. Hypercholesterolemia mainly increased LDL, VLDL and decreased HDL are important risk factors. Hypertension was a risk factor in 14.29 % of the subjects and Diabetes was a risk factor in 9.52% of subjects. Coronary artery disease is a major burden with increasing number of cases in developing countries.

The problem is getting more severe as much younger population of productive age group is getting affected. The disease is also associated with morbidity and mortality.

Many conventional and non-conventional risk factors have been recognized and studied over the years. Early recognition of these risk factors in the young population is an important step towards prevention of CAD in young individuals.

The present study was carried out with the aim to identify risk factors in young individuals & to emphasize need for their controls if present. Study of non-conventional risk factors in population needs to be studied in future.

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ANNEXURE-A

LIST OF ABBREVATIONS USED

CAD:	Coronary Artery disease
LV:	Left ventricle
PDGF:	Platelet derived growth factor
JNC:	Joint national committee
STEMI:	ST elevation myocardial infraction
NSTEMI:	NON ST elevation myocardial infraction
ECG:	Electrocardiogram
DM:	Diabetes mellitus
HTN:	Hypertension
IHD:	Ischemic heart disease
SBP:	Systolic blood pressure
DBP:	Diastolic blood pressure
BMI:	Body mass index
TLC:	Total Leucocyte count
TC:	Total cholesterol
LDL:	Low density lipoprotein
HDL:	High density lipoprotein
VLDL:	Very low density lipoprotein
TGS:	Triglycerides
CAG:	Coronary angiography
SVD:	Single vessel disease
DVD:	Double vessel disease
TVD:	Triple vessel disease

ANNEXURE-B

CLINICAL PROFORMA

The data will be used for this study: history will be taken from the patients and their previous medical records.

- NAME : AGE :
- SEX : REG.NO :
- ADDRESS :
- DATE OF PRESENTATION / ADMISSION: DATE OF DISCHARGE:
- CHIEF COMPLAINTS :
- PAST HISTORY :
- PERSONAL HISTORY :
- FAMILY HISTORY :
- TREATMENT HISTORY:
- SOCIO-ECONOMIC HISTORY:
- WORK RELATED STRESS
- GENERAL EXAMINATION :
- B.P:
- HEIGHT : WEIGHT : BMI :

Investigations*: AS PER THE AVAILABLE RECORD WILL BE ANALYSED.*

1. CBC:

2. RBS:

3. FBS:

4. PP2BS: (IF INDICATED):

5. GLYCOSYLATED HB

6. RFT:

7. S.ELECTROLYTES:

8. LIPID PROFILE (TC, LDL, HDL, VLDL, TGS):

9. ECG:

10. STRESS TEST:

11. 2D-ECHO:

12. CARDIAC BIOMARKERS (IF AVAILABLE)

13. CAG FINDINGS:

KEYS TO MASTER CHART

DM	**:**	**Diabetes mellitus**
HTN	**:**	Hypertension
IHD	**:**	Ischemic heart disease
R/F	**:**	Risk factor
SBP	**:**	Systolic blood pressure
DBP	**:**	Diastolic blood pressure
Ht (cm)	**:**	Height in centimeter
Wt (Kg)	**:**	Weight in kilogram
BMI	**:**	Body mass index
Hb	**:**	Haemoglobin
CBC	**:**	Complete blood count
TLC	**:**	Total Leucocyte count
RBS	**:**	Random blood sugar
FBS	**:**	Fasting blood sugar
PP2BS	**:**	Post prandial blood sugar
TC	**:**	Total cholesterol
LDL	**:**	Low density lipoprotein
HDL	**:**	High density lipoprotein
VLDL	**:**	Very low density lipoprotein
TGS	**:**	Triglycerides
CAG	**:**	Coronary angiography
SVD	**:**	Single vessel disease
DVD	**:**	Double vessel disease
TVD	**:**	Triple vessel disease

MASTER CHART

SR NO.	REG. NO(I.P.D/O.P.D,CATH)	AGE	SEX	DATE OF ADMISSION	DATE OF DIACHARGE	CHIEF COMPLAINT		PAST HISTORY				SMOKING PACK YEAR	FAMILY HISTORY OF IHD	SOCIO ECONOMIC STATUS	SEDENTARY HABITS	GENERAL EXAMINATION		GENERALISED OBESITY				CBC		BLOOD SUGAR					S.CREAT(mg.dl)	S.K+ (meq/ltr)	DYSLIPIDEMIA (R/F=+)					CAG FINDING
						CHEST PAIN	DYSPNOEA	DM	HTN	IHD	R/F		R/F		R/F	PULSE	SBP/DBP	Ht(cm)	Wt(Kg)	BMI(Kg/m2)	R/F	Hb(%)	TLC(Cells/Cumm)	RBS(mg/dl)	FBS(mg/dl)	PP2BS(mg/dl)	R/F	HbA1C(%)			TC	LDL	HDL	VLDL	TGS	
1	I1503020090	35	M	02/03/2015	06/03/2015	+	-	-	-	-	-	2.5	-	3	-	78	106/74	180	76	23	-	13.2	7500	-	124	-	-	-	1.2	4.3	136	128 +	38	36	96	SVD
2	I1403190092	38	M	19/03/2014	22/03/2014	+	-	-	-	-	-	10	-	3	-	86	126/78	180	76	23	-	11.9	10800	96	-	-	-	-	1.3	4.2	170	136 +	38	36	124	SVD
3	C-2559	26	M	11/04/2014	13/04/2014	+	-	-	-	-	-	-	+	3	-	70	110/74	164	64	24	-	12.8	11600	121	-	-	-	-	0.9	4.4	146	48	34 +	36	110	SVD
4	I1504080114	40	M	08/04/2015	08/04/2015	+	+	-	-	-	-	12	-	3	-	84	128/74	164	64	24	-	13.2	11200	124	-	-	-	-	0.8	4.6	186	136 +	30 +	80 +	179 +	SVD
5	I1503300094	37	M	30/03/2015	30/03/2015	+	-	-	-	-	-	-	-	1	-	84	126/76	168	88	31	+	13.6	6800	124	-	-	-	-	1.3	3.8	226 +	126 +	32 +	80 +	160 +	SVD
6	I1503200009	38	M	20/03/2015	24/03/2015	+	-	-	-	-	-	-	+	3	-	76	120/76	167	80	29	+	11.7	6900	124	-	-	-	-	1.2	3.6	154	98	36	54 +	96	SVD
7	1503180029	39	M	18/03/2015	18/03/2015	+	-	-	-	-	-	12	-	1	-	78	180/74	172	73	25	+	13.2	8400	122	-	-	-	-	1.2	3.8	176	146 +	26 +	88 +	136 +	TVD
8	I1503160141	39	M	16/03/2015	16/03/2015	+	-	-	-	-	-	20	-	3	-	76	114/76	167	80	29	+	12.2	8300	136	-	-	-	-	0.9	3.6	184	140 +	34 +	76 +	115	TVD
9	I1503120203	40	F	12/03/2015	12/03/2015	-	+	-	-	-	-	-	+	5	-	78	116/74	180	76	23	-	12.8	12900	116	-	-	-	-	0.8	3.9	136	76	36	26	116	DVD
10	C-3046	39	M	14/07/2014	21/07/2014	+	-	-	-	-	-	5	-	3	+	120	126/84	168	88	31	+	12.8	14800	-	134	-	-	-	1.2	4	265 +	103 +	26 +	108 +	540 +	SVD
11	C-3125	38	M	26/07/2014	30/07/2014	+	-	-	-	+	+	5	-	3	+	100	100/70	164	56	21	-	13.8	8900	119	-	-	-	-	1.5	4.2	180	133 +	37	118 +	59	SVD
12	C-3321	40	M	09/09/2014	15/09/2014	+	-	+	-	-	+	-	-	3	+	104	110/76	167	80	29	+	13.3	9800	-	234	280	+	10.8	1.1	3.9	111	57	32 +	22	110	SVD
13	C-3328	40	M	14/09/2014	14/09/2014	-	+	-	-	+	+	-	+	3	-	96	92/70	172	73	25	+	13.3	11000	116	-	-	-	-	1.2	4	171	110 +	28 +	110 +	180 +	TVD
14	C-3499	40	M	15/10/2014	17/10/2014	+	-	-	-	-	-	15	-	5	-	104	106/84	168	88	31 +	+	12	6100	124	-	-	-	-	0.9	4.8	110	59	30 +	21	105	DVD
15	C-3480	40	F	11/10/2014	20/10/2014	+	-	-	-	-	-	-	+	5	-	104	94/60	164	64	24	-	9.6	9800	118	-	-	-	-	1.2	3.4	194	84	28 +	26	106	DVD
16	C-3702	40	M	02/12/2014	06/12/2014	+	-	-	-	-	-	-	+	3	-	56	130/90	180	76	23	-	13	9200	122	-	-	-	-	1.2	4.5	166	86	26 +	24	120	SVD
17	C-3655	38	M	03/12/2014	05/12/2014	+	-	-	-	-	-	-	+	5	-	76	110/80	166	57	21	-	10.1	7200	110	-	-	-	-	1.2	4.1	128	46	42	39 +	197 +	DVD
18	I1501130089	37	F	13/01/2015	15/01/2015	+	-	-	-	-	-	-	+	3	+	90	122/84	168	68	24	-	12.6	9300	126	-	-	-	-	1.1	3.8	154	96	36	57 +	116	SVD
19	I1502110108	37	F	11/02/2015	15/02/2015	+	+	-	-	-	-	-	+	3	-	94	146/86	168	88	31	+	12.8	12200	98	-	-	-	-	1.3	5	176	140 +	34 +	126 +	68	SVD
20	I1502270151	40	F	27/02/2015	02/03/2015	+	-	+	-	-	+	-	-	3	+	96	130/90	180	76	23	-	12.6	9800	-	296	380	+	13.1	1.1	4	230 +	176 +	30 +	118 +	128	SVD
21	I1503170052	40	F	17/03/2015	19/03/2015	+	-	-	-	-	-	-	-	5	+	80	118/74	155	54	22	-	12.6	12600	116	-	-	-	-	0.8	3.9	186	126 +	26 +	166 +	124	SVD
22	I1503240008	37	M	24/03/2015	26/03/2015	+	-	-	-	-	-	15	-	4	-	80	130/80	180	76	23	-	11.6	6800	116	-	-	-	-	0.9	4.2	168	116 +	34 +	60 +	204 +	DVD
23	C-2679	34	M	05/05/2014	05/05/2014	+	-	-	-	-	-	10	-	5	-	86	124/76	168	68	24	-	12.3	7900	114	-	-	-	-	1.2	3.6	156	124 +	38	58 +	146	SVD
24	I1501050102	38	M	05/01/2015	10/01/2015	+	-	-	+	-	+	-	-	1	-	84	160/104	164	56	21	-	12.6	10600	116	-	-	-	-	1.2	4.8	165	126 +	38	36 +	128	SVD
25	I1501050142	40	F	05/01/2015	06/01/2015	+	-	+	+	-	+	-	-	3	+	84	174/90	164	56	21	-	12.2	9500	-	120	240	+	7.7	0.9	4	220 +	136 +	36	76 +	128	DVD
26	C-2029	32	M	26/12/2013	30/12/2013	+	-	-	-	-	-	-	+	5	-	74	116/70	180	76	23	-	12.9	9800	120	-	-	-	-	0.9	4	176	116 +	30 +	96 +	120	SVD
27	C-1973	39	F	19/12/2013	23/12/2013	+	-	-	-	-	-	-	-	3	+	80	118/76	167	80	29	+	12.2	9600	116	-	-	-	-	0.7	3.9	146	116 +	30 +	76 +	116	SVD
28	I1403310103	40	F	31/03/2014	02/04/2014	+	-	-	-	-	-	-	+	3	+	86	128/74	164	56	21	-	13.2	7200	118	-	-	-	-	0.8	4.3	138	110 +	32 +	58 +	188 +	SVD
29	C-2663	40	M	01/05/2014	01/05/2014	+	-	-	-	-	-	-	+	3	+	72	126/70	167	80	29	+	11	6700	112	-	-	-	-	0.8	4.6	154	96 +	38	48 +	188 +	TVD
30	C-2682/426	38	M	03/05/2014	06/05/2014	+	-	-	+	-	+	-	+	3	-	68	126/80	164	64	24	-	11.5	5800	110	-	-	-	-	1	4.3	146	94	30 +	44 +	148	DVD
31	C-2690	33	M	08/05/2014	08/05/2014	+	-	-	-	-	-	-	+	3	-	74	116/80	168	68	24	-	13.4	7500	116	-	-	-	-	1.1	3.9	150	86	38	36 +	76	TVD
32	C-2704	36	M	11/05/2014	15/05/2014	+	-	-	-	-	-	20	-	5	-	80	124/70	164	56	21	-	11.8	8400	122	-	-	-	-	1.1	3.9	144	56	38	86 +	196 +	SVD
33	C-2741	40	M	19/05/2014	20/05/2014	+	-	-	-	-	-	-	+	3	-	84	90/60	180	76	26	-	12.3	8600	114	-	-	-	-	1.2	4.2	164	84	40	94 +	190 +	SVD
34	I1503300010	37	M	30/03/2015	04/04/2015	+	-	-	-	-	-	6	+	3	-	56	120/74	167	80	29	+	11.6	10200	108	-	-	-	-	1	3.8	156	98	36	46 +	186 +	SVD
35	C-2435	36	M	19/03/2014	22/03/2014	+	-	-	-	-	-	-	+	1	-	84	128/74	164	64	24	-	13.4	11400	116	-	-	-	-	0.9	4.2	116	48	28 +	88 +	116	SVD
36	C-2811	39	M	31/05/2014	31/05/2014	+	-	-	+	-	+	6	-	3	-	86	148/94	180	76	23	-	12.9	8200	121	-	-	-	-	0.8	4	204 +	126 +	38	46 +	96	TVD
37	C-2433	40	M	19/03/2014	22/03/2014	+	-	-	+	-	+	10	-	5	-	80	154/90	180	76	23	-	12.9	5600	121	-	-	-	-	0.9	4.3	143	134 +	38	30	110	SVD
38	C-2259	40	M	15/02/2014	15/02/2014	+	-	-	-	-	-	-	-	5	-	84	126/74	168	88	31	+	11.4	8600	120	-	-	-	-	0.8	4	206 +	126 +	38	84 +	184 +	DVD
39	I150411091	40	M	11/04/2015	13/04/2015	+	-	-	-	-	-	20	-	3	-	84	120/74	168	88	31	+	12.8	7800	124	-	-	-	-	0.7	3.9	146	98	36	80 +	206 +	SVD
40	I1502280100	27	M	28/02/2015	03/03/2015	+	-	-	-	-	-	-	+	1	-	96	122/84	164	74	28	+	11.4	9400	106	-	-	-	-	1	4.3	160	76	30 +	106 +	186 +	SVD
41	C-2165	40	M	27/01/2014	27/01/2014	+	-	-	-	-	-	-	+	3	-	86	126/80	168	68	24	-	13.6	11300	112	-	-	-	-	0.9	4	130	48	34 +	28	108	SVD
42	C-2141	40	M	21/01/2014	26/01/2014	+	-	+	+	-	+	-	-	3	-	84	124/76	178	80	26	+	14	8600	260	-	-	+	10.6	0.9	4.8	156	86	34 +	36 +	94	SVD
43	C-2136	40	M	20/01/2014	25/01/2014	+	-	-	-	-	-	-	+	3	-	86	124/90	178	80	26	+	11.6	8200	120	-	-	-	-	1	4.6	213 +	126 +	32 +	50 +	130	SVD
44	C-2129	30	M	16/01/2014	16/01/2014	+	-	-	-	-	-	-	+	3	-	86	118/74	180	76	23	-	12.8	7800	120	-	-	-	-	0.9	3.9	116	46	38	46 +	136	DVD
45	C-2048	40	M	27/12/2013	29/12/2013	+	-	-	-	-	-	6	-	1	-	86	106/70	164	56	21	-	11.8	6700	96	-	-	-	-	0.9	3.8	130	42	38	58 +	136	SVD
46	I1503200026	30	M	20/03/2015	25/03/2015	+	-	-	-	-	-	-	+	4	-	80	116/80	178	80	26	+	13.2	7400	122	-	-	-	-	0.9	3.7	164	108 +	28 +	46 +	166 +	SVD
47	C-2044	40	M	27/12/2013	29/12/2013	+	-	-	-	-	-	10	-	5	-	80	128/84	164	56	21	-	11.4	6700	123	-	-	-	-	1.1	5	176	128 +	38	60 +	180 +	DVD
48	C-1958	39	F	17/12/2013	19/12/2013	+	-	-	-	-	-	-	+	3	+	80	134/90	180	76	23	-	11.1	6900	111	-	-	-	-	1.2	3.9	132	86	38	26	130	SVD
49	C-1897	25	M	09/12/2013	09/12/2013	+	-	-	-	-	-	-	+	3	-	100	106/74	164	56	21	-	12.6	8200	90	-	-	-	-	0.8	3.6	130	48	36 +	28	108	TVD
50	I1401080073	36	F	08/01/2014	10/01/2014	+	-	-	+	-	+	-	-	3	+	84	154/96	167	80	29	+	10.9	5800	94	-	-	-	-	1	4.2	190	96	32 +	46 +	116	SVD
51	I1401280126	35	M	28/01/2014	30/01/2014	+	-	-	-	-	-	-	+	3	-	84	130/90	164	64	24	-	11.3	9800	96	-	-	-	-	0.9	4.2	126	48	34 +	28	110	SVD
52	C-2637	39	M	28/04/2014	28/04/2014	+	-	-	-	-	-	8	-	5	-	88	116/78	180	76	23	-	12.9	6600	116	-	-	-	-	1.2	3.9	146	136 +	36	114 +	126	TVD
53	C-2560	34	M	11/04/2014	14/04/2014	+	-	-	-	-	-	-	+	3	-	80	114/76	164	56	21	-	13.2	7000	98	-	-	-	-	1	4.2	141	36	38	28	106	SVD
54	I1504210134	38	M	21/04/2015	22/04/2015	+	-	+	-	-	+	-	-	5	-	80	126/74	164	56	21	-	12.6	9500	-	284	360	+	13.4	1.2	4.3	204 +	118 +	36	58 +	176 +	SVD
55	I1311220072	28	M	22/11/2013	26/11/2013	+	-	-	-	-	-	-	+	3	-	84	114/70	172	73	25	+	13.4	11600	98	-	-	-	-	1	3.9	138	44	36	30	98	SVD
56	I1404190115	33	M	19/04/2014	24/04/2014	+	-	-	+	-	+	3	-	5	-	80	104/76	166	57	21	-	13	10400	111	-	-	-	-	1.2	3.6	174	126 +	32 +	40 +	116	DVD
57	I1404160059	40	M	16/04/2014	19/04/2014	+	+	-	-	-	-	-	+	3	-	84	110/70	164	64	24	-	11	6800	98	-	-	-	-	1.1	3.9	144	44	36	38 +	94	SVD
58	I1404290147	35	M	29/04/2014	03/05/2014	+	-	+	-	-	+	-	-	5	+	76	106/76	167	80	29	+	11.4	7600	-	260	286	+	11.1	1.2	4.6	208 +	48	34 +	176 +	126	SVD
59	I1404030138	30	M	03/04/2014	05/04/2015	+	-	-	-	-	-	-	+	3	-	78	114/76	164	64	24	-	13.6	9800	116	-	-	-	-	0.9	4	142	50	36	38 +	90	SVD
60	I1403200082	38	M	20/03/2014	21/03/2014	+	-	-	+	-	+	-	-	5	-	84	100/60	180	76	23	-	10.9	11100	116	-	-	-	-	1	4	126	84	32 +	30	132	TVD
61	I1403240166	37	M	24/03/2014	25/03/2014	+	-	-	-	-	-	-	+	3	-	78	108/74	164	64	24	-	11.2	7300	124	-	-	-	-	1	4.4	170	46	38	36 +	94	SVD
62	C-2542	30	M	26/09/2014	29/09/2014	+	-	-	-	-	-	3	-	5	-	90	130/70	168	88	31	+	11.9	7800	116	-	-	-	-	1	3.8	176	116 +	34 +	60 +	188 +	DVD
63	I1403258553	38	M	14/12/2013	17/12/2013	+	-	-	-	-	-	17	-	3	-	76	124/76	180	76	23	-	11.4	6900	116	-	-	-	-	0.8	4.3	140	104 +	36	68 +	210 +	SVD