"COMPARING THE PATTERN OF ATHEROSCLEROSIS IN CAROTID WITH AORTA, RENAL AND CORONARY ARTERIES: AN AUTOPSY STUDY"

By

Dr. LOKESH HASWANI

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IN

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Under the guidance of

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DR. LOKESH HASWANI

DEDICATED

TO,
ALL THE KNOWN AND UNKNOWN FORCES THAT HAVE HELPED ME

LIST OF ABBREVIATIONS

CVD – Cardio Vascular Disease

IHD – Ischemic Heart Disease

AHA – American Heart Association

LAD – Left Anterior Descending artery

LCA – Left Circumflex Artery

RCa – Right Coronary Artery

LCa – Left Carotid Artery

AA – Ascending aorta

TA – Thoracic aorta

Ab.A – Abdominal aorta

RR – Right Renal

LR- Left Renal

DALY –Disability Adjusted Life Year

INF alpha – Interferon Alpha

PDGF – Platelet Derived Growth Factor

FGF – Fibroblast Growth Factor

TGF alpha –Transforming Growth Factor Alpha

LDL – Low Density Lipoprotein

HDL – High Density Lipoprotein

CHD – Coronary Heart Disease

GDP- Gross Domestic Product

MMP – Matrix Metallo Proteinases

NAL – Non Atherosclerotic Lesion

PAL – Progressive Atherosclerotic Lesion

NVL – No Visible Lesion

CL – Complicated Lesion

FS – Fatty streak

FP – Fibrous Plaque

FCA – Thick fibrous cap atheroma

T FCA – Thin fibrous cap atheroma

PIT – Pathological intimal thickening

IX – Intimal xanthoma

IT – Intimal thickening

RTA – Road traffic accident

ABSTRACT

Objective:

To study the gross and histomorphological atherosclerotic changes in carotid in relation to aorta, renal, distal and proximal coronary arteries in autopsy cases and to find out the relationship between atherosclerosis in proximal and distal coronary arteries with age.

Materials and Methods:

The study was carried out at The Department of Pathology, Sri Devaraj Urs Medical College, Tamaka, Kolar in co-ordination with the Department of Forensic medicine between 1st January 2011 to 31stAugust 2013. Ninety one cases that underwent autopsy during the study period mentioned were assessed for atherosclerosis in carotid's, coronaries (proximal and distal), aorta and renal arteries. Autopsy was done by conventional technique, heart and the aorta along with carotid and renal arteries were removed and fixed in 10% formalin. The heart was dissected along the direction of flow of blood and aorta along the posterior surface. Bits taken from all the arteries and histopathological study was done by routine tissue processing and H & E staining

Results:

In the present study, the atherosclerosis in carotid was 12.6%. In case of coronaries atherosclerosis in LAD is about 51%followed by RCA 35% and last LCA was 37%. Atherosclerosis was 40% in ascending aorta, 20% in thoracic aorta and 30% in the abdominal aorta. Atherosclerosis in renal arteries was only 3 %. When comparing the association of atherosclerosis in carotid, with coronary, aorta and renal arteries, the P value was <0.05 which was statistically significant.

In young age group the mean of atherosclerosis in the proximal coronary was 8.67±3.21 and mean of distal coronary was 3.67±1.53. In case of older age group mean of proximal and distal coronary atherosclerosis was 28.67±5.69 and 14±7.55 respectively. The difference between two was statistically significant ($p<0.05$).

Conclusion:

Autopsy is the gold standard test to assess the atherosclerosis. Till now none of the study both autopsy or radiological had compared the carotid atherosclerosis with coronary, aortic and renal atherosclerosis.

In the present study we conclude that if carotid atherosclerosis is present patient will be having coronary, aortic atherosclerosis but patient might have renal atherosclerosis. If carotid atherosclerosis is absent it doesn't mean that patient won't be having atherosclerosis in any other vessels.

Assessing the carotid atherosclerosis using noninvasive technique can be used as screening tool for cardiovascular, cereberovascular and kidney disease due to renal artery atherosclerosis. This is an indication that anti-atherogenic preventive measures need to be implemented as soon as carotid atherosclerosis is detected. This is to prevent premature death caused by cardiovascular, cereberovascular and kidney disease, thus burdening the national economy.

Prevalence of proximal part of coronary atherosclerosis was double that of the distal part of coronary arteries in both younger and older age group. Identification of these zones of high risk for acute coronary occlusions will lead to future advances in vulnerable plaque detection technology and potentially locally directed preventive strategies.

Key words: Atherosclerosis, autopsy, carotid artery.

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INTRODUCTION

INTRODUCTION

Atherosclerosis is a major cause of morbidity and mortality worldwide and it account for one third of all deaths from heart disease.[1] The most serious outcome of atherosclerosis are stroke, myocardial infarction and death.[1] It affects the Indian population at a younger age than in any other ethnic groups with more severe and extensive angiographic involvement.[2] Atherosclerosis is a generalized vascular disease.[3] Although there is asymmetry in involvement of various arteries in atherosclerosis but the disease usually proceeds in parallel in various organ system.[4]

Autopsy is a tool of real value rather than an exercise of tradition, when done selectively and efficiently.[5] Autopsy series are often quoted as an unbiased information source.[4] As study of atherosclerosis in the living population is difficult in many ways including its cost-effectiveness, autopsy studies have been regarded as one good way of dealing with the problem.[6] Given the limited ability of current clinical imaging methods to visualize the vessel wall, as opposed to the lumen, we are highly dependent on autopsy material for describing various different stages of atherosclerosis.[7]

It is well known fact that atherosclerosis is a systemic disease with important sequelae in many other regional circulations, including those supplying the brain, kidneys, mesentery, and limbs.[8] Once this disease is apparent in one vascular territory, there is increased risk of adverse events in other territories.[8]

Radiological studies had shown that there is close association of carotid atherosclerosis with aortic and coronary artery atherosclerosis.[9] Several studies done

angiographically had shown that renal artery atherosclerosis usually exits as one manifestation of generalized atherosclerosis.[10]

By imaging technique only few superficial vessels like carotid can be studied to assess the atherosclerosis.[11] Several radiological studies show that carotid intima media thickness can be regarded as an indicator of generalized atherosclerosis and it can predict the future cardiovascular and cerebrovascular events.[11,12] So it is very important to see whetherthe extent of disease correlates in different arterial beds is same as in carotid artery or not.[13] Till now none of the radiological or autopsy study had correlated carotid atherosclerosis with coronary aorta and renal atherosclerosis. So here the study had been conducted to see whether the carotid atherosclerosis can be correlated with the atherosclerosis of other major vessels using gold standard technique of histomorphological analysis on the autopsy specimens. So that carotid atherosclerosis can be used as a screening tool to predict the future cardiovascular, cerebrovascular and kidney disease.

The lesion in the proximal part of coronary vessels was more frequent, more severe and more prone to rupture than the distal one. [14] The proximal coronary artery is a "hot spot" for coronary artery disease (CAD).[14] Identification of these zones of high risk for acute coronary occlusions will lead to future advances in vulnerable plaque detection technology and potentially locally directed preventive strategies.[14] Proximal CAD has a hereditary basis where as distal CAD is more affected by environmental factors. [15] So here the study had been conducted to see the relationship of proximal and distal coronary atherosclerosis with age. So that it helps in early and specific detection technology of coronary atherosclerosis and start early intervention and decrease the disease burden.

AIMS AND OBJECTIVE

1. To study the gross and histomorphological atherosclerotic changes in carotid and its correlation with lesions of aorta, renal, distal and proximal coronary arteries in autopsy cases.

2. To find out the relationship between atherosclerosis in proximal and distal coronary arteries with age.

REVIEW OF LITERATURE

Marchand introduced the term -"atherosclerosis".[16] Arteriosclerosis literally means "hardening of the arteries"; It is a generic term reflecting arterial wall thickening and loss of elasticity.[11,12] Three basic patterns exist,

1) Arteriolosclerosis: Affects small arteries and arterioles.

2) Monckeberg's medial sclerosis: Characterized by calcific deposits in muscular arteries. They are usually not clinically significant.[16,17]

3) Atherosclerosis: It is derived from Greek root words for "gruel" means "hardening" and is the most frequent and clinically important pattern.[16,17]

Virchow's analysis in 1858 pointed out that historically, the term "atheroma" refers to a dermal cyst ("Grutzbalg"), a fatty mass encapsulated within a cap.[7] Autopsy based descriptions of calcified coronary arteries and atherosclerosis was initially given by Lancisi in the 1600s in his famous De SubitaneisMortibus.[18] Atherosclerosis is a disease of large and medium-sized muscular arteries and is characterized by endothelial dysfunction, vascular inflammation, and the buildup of lipids, cholesterol, calcium, and cellular debris within the intima of the vessel wall.[17] These lesions can mechanically obstruct the lumen, can rupture, cause catastrophic vessel thrombosis and plaques which can also weaken the underlying media, thereby leading to aneurysm.[16,17]

Atherosclerosis is often considered as a modern disease, yet it is evident in the remains of many ancient Egyptian mummies.[18,19] This shows that atherosclerosis is a disease of ancient times and that the epidemic of atherosclerosis which began in the 20th century is nothing more than history revisiting us.

Normal Anatomy:

Though the vascular system is complex, but the individual blood vessels are one of the simplest tissue structures in the body.[16] A blood vessel consists of two cell types: endothelial and smooth muscle cells. The earliest embryonic vascular primordia are clusters of endothelial cells that arise on the yolk sac, which recruit mesenchymal cells that become the smooth muscle cells of the media and the fibroblasts of the adventitia.[16] The simple two cell structures becomes more complex in the arteries by organization into layers called "tunica". The innermost layer is known as- tunica intima is a combination of endothelium and connective tissue on the luminal side of the internal elastic lamina.[16,17,20] The intima of the aorta is thick and contains a matrix of collagen, proteoglycans and small amounts of elastin.[16,20] Atherosclerosis mainly a disease of the tunica intima but also causesproliferation of these tunica-media's smooth muscle cells.[16] The most external layer of the vessel is the tunica adventitia, is a connective tissue sheath composed of fibroblasts, vasa vasorum and nerves. **(Fig 1)**.

Aorta and any other elastic arteries have elastic fibers interposed between smooth muscle cells.[16,20] These elastic arteries distribute blood to individual organs through large muscular arteries like coronaries and renal arteries.[16,17,20] The aorta begins at the top of the left ventricle and terminates as two common iliac arteries at the level of 4^{th} lumbar vertebra.[20] The left carotid arises directly from the arch of aorta and while the right carotid arises from brachiocephalic artery which also arise from aortic arch.[20] Both renal arteries are arise from lateral side of the aorta, immediately below the superior mesenteric artery(**Fig 2)**.[20] Atherosclerosis rarely affects the resistance vessels like the capillaries and arterioles.[16]

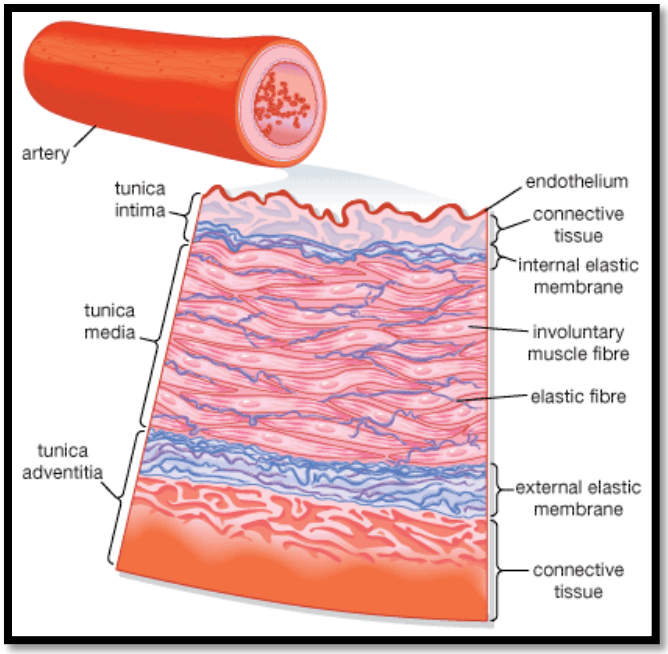


Figure 1: Normal histology of a muscular artery[16]

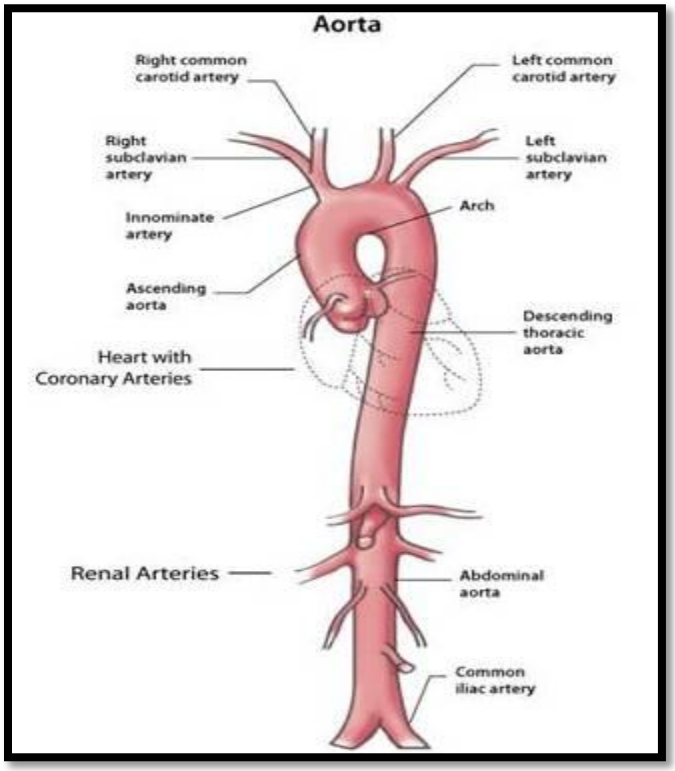


Figure 2: Aorta and its branches[20]

Vasculature of the heart:

Most of the myocardium depends on the nutrients and oxygen delivered via the coronary arteries, which arise immediately distal to the aortic valve in or just above the sinus of valsalva, initially running along the external surface of the heart (epicardial coronary arteries) and then penetrating the myocardium (intramural arteries).[16,17,20] The three major epicardial coronary arteries are (i) the left anterior descending (LAD) (ii) the left circumflex (LCA) arteries, both arising from branches of the left main coronary artery, and (iii) the right coronary artery (RCA).[17,20] The LAD supplies the anterior left ventricle, the adjacent anterior right ventricle and the anterior two thirds of the interventricular septum.[16,20] The LCA supplies the lateral wall of the left ventricle. The RCA supplies the remainder of the right ventricle and the posteroseptal region of the left ventricle including the posterior third of the interventricular septum at the base of the heart.[16,17,20]

Epidemiology:

India has one of the highest burdens of cardiovascular disease (CVD) worldwide.[21] The annual number of deaths from CVD in India is projected to rise from 2.26 million (1990) to 4.77 million (2020).[21] According to World Health Report 2002, CVD will be the largest cause of death and disability by 2020 in India.[22,23] Nearly half of these deaths are likely to occur in young and middle aged individuals (30-69 years).[22,24] Currently Indians experience CVD deaths at least a decade earlier than their counterparts in developed countries.[22,23]

The Global Burden of Disease (GBD) study estimates that 52% of CVD deaths occur below the age of 70 years in India as compared to 23% in developed countries, resulting in a profound adverse impact on its economy.[22,25] With an average GDP growth of 8.6%, India is one of the fastest growing economies of the world and was the second highest contributor to world growth in 2006.[23] World Health Organization (WHO) estimates that India lost 9 billion dollars in national income from premature deaths due to heart disease, stroke and diabetes in 2005 and is likely to lose 237 billion dollars by 2015.[23] In the year 2000, India was estimated to have lost more potentially productive years of life to CVD than any other country in the world (9.2 million years of life lost among people 35 to 64 years of age in 2000) and without effective intervention, this figure is projected to increase to nearly 18 million by 2030 (10 times the rate in the U.S.)[24] Scientific data has shown that the socio-economically disadvantaged sections of the population are now the dominant victims of CVD and its risk factors.[22,23] There is also preliminary evidence that the burden of CVD in rural areas is increasing.[23,24] **(Figure-3).** The reported prevalence of coronary heart disease (CHD) in adult surveys has risen four fold in last 40 years and in rural areas also the prevalence has doubled in the past 30 years.[23] Increase in life expectancy, urbanization, rural- urban migration adverse dietary changes in the population, poverty, maternal malnutrition, high consumption of cheap vegetable oils and fat, tobacco use and various genetic factors are the major causes for this alarming epidemic of CVD which threatens to cripple India's workforce and stunt India's growth.[23] In the face of such a huge CVD epidemic, studies regarding the environmental and genetic risk factors for CVD, prevalence and incidence of CVD and application of preventive measures are the need of the hour. Between the

year 1991 to 2001, India has produced only 294 research publications on CVD in contrast to United States of America which produced 12,502 research publications on CVD in the same time frame.[25]

Stroke is the third leading cause of death and the principalcause of long-term disability in the United States today. There are 6 lakhs new or recurrent strokes cases occurring annually.[21] The number of strokes attributable to carotid atherosclerosis approaches 20%.[8] Black populations have a 38% higher adjusted incidence of ischemic stroke than white populations and also higher stroke mortality.[8]

The incidence of end-stage renal disease in the year 2000 approximated 1 lakhs persons annually and prevalence was estimated at 3,72,000 persons.[8]

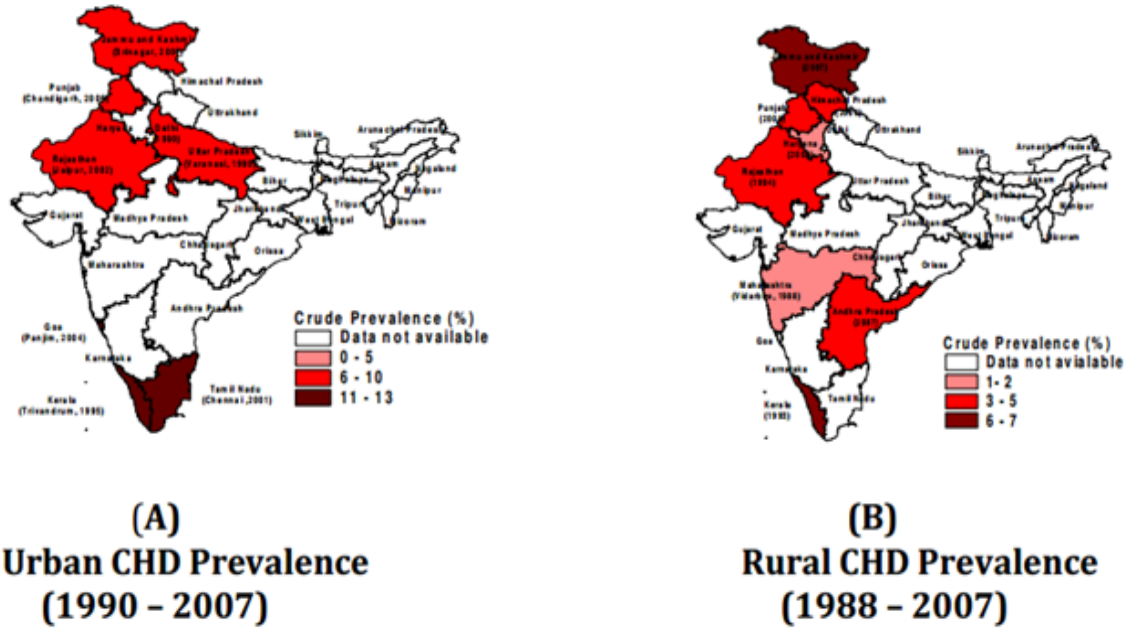


Figure 3: Crude prevalence of CHD in urban (A) and rural (B) India from 1988 – 2007.[26]

Risk factors of CVD:

The concept of coronary prone behavior has a long history but has a very short scientific past.[27] The renowned internist Sir William Osler noted, for instance, that: "It is not the delicate, neurotic person who is prone to angina, but the robust, the vigorous in mind and body, the keen and ambitious man… whose engine is at full speed ahead." [27] South Asians have twice the risk of atherosclerosis and coronary artery disease compared to Caucasians, when they are exposed to better living standards and altered lifestyle habits.[28] India is a large country, there is tremendous urban-rural differences and regional variations in the different risk factors.[29] Studies from rural areas have demonstrated a lower prevalence compared to studies from urban areas.[30] The prevalence and severity of atherosclerosis and IHD among individuals and groups are related to several risk factors, some constitutional (non-modifiable) and others acquired (modifiable).[30] Atherosclerotic process in the arteries is a result of interaction between known and unknown genetic and environmental factors.[31] Most of the data regarding the relationship between risk factors and atherosclerosis have been obtained from autopsy studies.[31] Multiple risk factors increase the probability of CVD, since cardiovascular risk factors tend to reinforce each other in their influence on morbidity and mortality.[32] The various risk factors are

Constitutional risk factors - non-modifiable:

1) Age: It has a dominant influence on atherosclerosis and IHD risk was rising with every decade.[16,17,29]

2) Gender: Male sex is a definite risk factor. Premenopausal women are protected against atherosclerosis compared to age-matched men. After menopause, however, the incidence

of atherosclerosis related diseases increases in females and at older ages it actually exceeds that of men.[16, 17, 29]

3) Genetics: Family history is the most significant independent risk factor for atherosclerosis. Familial predisposition to atherosclerosis and IHD is usually multifactorial, relating to inheritance of various genetic polymorphisms and familial clustering or other established risk factors, such as hypertension or diabetes.[16, 17, 33]

Modifiable risk factors:

1) Hyperlipidemia: In particular hypercholesterolemia is a major risk factor for atherosclerosis even in the absence of other risk factors. Higher risk is associated with increased levels of low density lipoproteins(LDL) and reduced levels of high density lipoproteins(HDL).[34,35,36] HDL mobilizes cholesterol from tissue and transports it to the liver for excretion in bile, hence the reduced risk of atherosclerosis is seen with higher levels of HDL. Patients with familial hypercholesterolemia have a mutation in the gene encoding the receptor for LDL with heterozygotes having two to three fold elevation and homozygotes having a five to six fold elevation of cholesterol levels from birth itself.[16,17,29] These individuals develop skin xanthomas, coronary, cerebral and peripheral atherosclerosis at an early age.[17]

2) Hypertension: It is a major risk factor for atherosclerosis,with both systolic and diastolic levels having major effects.[35,36,37]

3) Cigarette smoking: prolonged smoking of one pack of cigarette or more daily doubles the death rate from IHD.[35,36,38] The fatty streaks in the abdominal aorta is greater in smokers than nonsmokers.[38] Cessation of smoking reduces the risk substantially.[35]

4) Diabetes mellitus: It induces hypercholesterolemia and markedly increases the risk of atherosclerosis.[36] Other risk factors being the same, the incidence of myocardial infarction is twice as high in diabetics than in non-diabetics.

Additional and recently implicated risk factors:

Near about 20% of all cardiovascular events occurs in the absence of hypertension, hyperlipidemia, smoking and diabetes. Other factors have been implicated in causing atherosclerosis in these patients. Few have been listed below,

1) Inflammation: It is present during all stages of atherogenesis and is intimately linked with plaque formation and rupture. Immune mechanisms interact with metabolic risk factors to initiate, propagate and activate lesions in the arterial tree by releasing cytokines, chemokines and matrix metalloproteinases (MMPs).[17,39] Circulating marker of inflammation like C-reactive protein has emerged as one of the simplest and most sensitive marker to correlate IHD risk.[17,29,39]

2) Hyperhomocystinemia: There is a strong relationship between total serum homocysteine levels and coronary artery disease, peripheral vascular disease, stroke and venous thrombosis. Elevated homocysteine levels are caused either by low intake of folate and vitamin B12 or due to inborn errors of metabolism.[17,40]

3) Metabolic syndrome: It is characterized by glucose intolerance, insulin resistance, hypertension and central obesity which are known risk factors for the development of atherosclerosis.[36]

4) Lipoprotein(a): It is an altered form of LDL. Lipoprotein(a) levels are associated with coronary and cerebrovascular disease risk, independent of total cholesterol or LDL levels.

Lipoprotein(a) levels have been consistently shown to be elevated among Asian Indians compared to other ethnic groups suggesting a genetic predisposition to coronary artery disease.[29]

5) Infections: There is tantalizing evidence that infections may drive the local inflammatory process that underlies atherosclerosis, this hypothesis has yet to be conclusively proven.[17] Herpesvirus, cytomegalovirus and Chlamydia pneumonia have been detected in atherosclerotic plaques but not in normal arteries.[17,41]

6) Elevated plasminogen activator inhibitor.

7) Lack of exercise

8) Competitive, stressful life style ("type A" personality)

9) Obesity.

Few metabolic abnormalities among the above like high triglyceride concentration, increased total cholesterol level to HDL ratio, type 2 diabetes mellitus and central obesity are known to occur more commonly in South Asians (mainly Indians) explaining the high incidence of coronary artery disease and IHD in this population.[42] Rapid economic growth, globalization, urbanization and rural-urban migration are all leading to a dramatic shift in diet and living behaviors. Consequently, adverse dietary changes, sedentary activity, increased tobacco use and other CVD risk factors are occurring at an early age in Indian population when compared to other countries.[23]

Pathogenesis:

Atherosclerosis is a multifactorial disease and its pathogenesis remains a subject of lively speculation and controversy.[43] Atherosclerosis is most common acquired abnormality of blood vessels, in which endothelial cells, leukocytes, intimal smooth muscle cells and macrophages are the major players.[16,17]

Many hypothesis have been proposed to explain the origins of atherosclerotic plaques. These hypothesis are not mutually exclusive and there is considerable amount of overlap among them.[16]

1) Insudation Hypothesis: It states that the critical event in atherosclerosis is the focal accumulation of fat in the intima which is derived from plasma lipoproteins.(Figure 4). Although this hypothesis explains the source of plaque lipid, it does not provide a complete explanation for the pathogenesis of atherosclerotic lesions.[16]

2) Encrustation Hypothesis: First suggested in the 19th century, it asserted that material from the blood is deposited on the inner surface of arteries and is the basis of the thickening of the intima.[16,17]

3) Response-to-injury hypothesis: It is the most widely accepted hypothesis.[17]It views atherosclerosis as a chronic inflammatory and healing response of the arterial wall to endothelial injury.[17] According to this hypothesis, atherosclerosis is produced by the following pathogenic events:

a) Endothelial injury: Caused byhemodynamic stressors like hypertension, hyperlipidemia, toxins from cigarette smoke, homocysteine and also infectious agents

(Figure 4). Injury leads to increased vascular permeability, leukocyte adhesion and thrombosis.[16,17]

b) Accumulation of lipoproteins in the vessel wall: Increased vascular permeability leads to lipid entry which produces oxygen free radicals, damages the tissue and oxidizes LDL into oxidized LDL. Oxidized LDL is taken up by macrophages and smooth muscle cells to form foam cells.[17]

c) Inflammation: Inflammatory cells and pathways contribute to the initiation, progression and complications of atherosclerotic lesions.[17] Macrophages in the lesion convert into foam cells, act as store house of cytokines and chemokines which attract other inflammatory cells and also produce oxygen free radicals. Inflammatory cells and macrophages produce MMPs which dissolve the connective tissue matrix leading to plaque rupture. T lymphocytes are also recruited into the plaque and produce inflammatory cytokines like INF alpha.[16,17]

d) Smooth muscle proliferation: Intimal smooth muscle proliferation and extracellular matrix (ECM) deposition convert a fatty streak, the earliest lesion into a mature atheroma and contribute to the progressive growth of atherosclerotic lesions. Growth factors like PDGF, FGF and TGF alpha are the cause for the smooth muscle proliferation and ECM synthesis.[17]

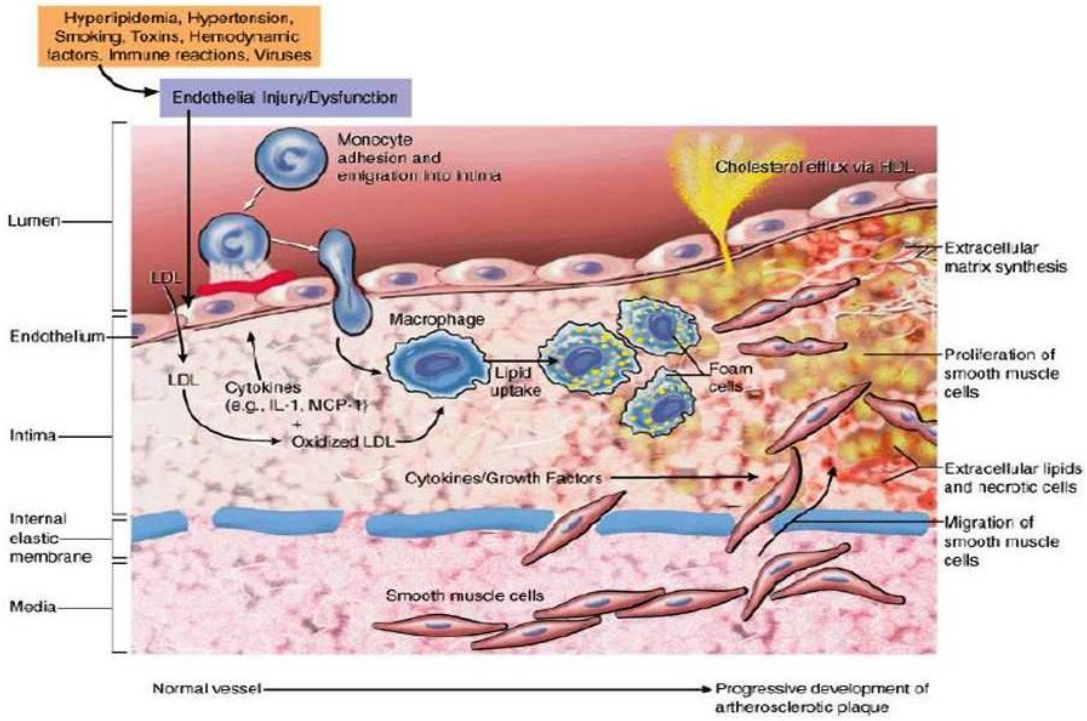


Figure 4: Diagram showing the various mechanisms involved in the formation of atherosclerotic plaques.[16]

Natural history of atherosclerosis:

The natural history of atherosclerosis, as described nearly about 50 years ago is depicted in **Figure 5.**[44] This description of origin and progression is inferred predominantly from observations of the arteries at various ages of persons autopsied.[44] It is based on the assumption that any type of lesion occurring in younger age group (for example, fatty streaks in adolescents) may be transformed into another type of lesion in an older age group occurring at the same anatomic site (for example, fibrous plaques in young adults and middle-aged persons).[44]

The fatty streak developed in childhood is depicted as a reversible process. [44] In adolescence, some of the fatty streaks accumulate more and more lipid and begin to

develop a fibromuscular cap, forming the lesion termed a fibrous plaque. In subsequent years, some of the fibrous plaques enlarge and undergo calcification, hemorrhage, ulceration or rupture, and thrombosis. [44] Thrombotic occlusion results clinical disease, depending on the artery affected. [44]

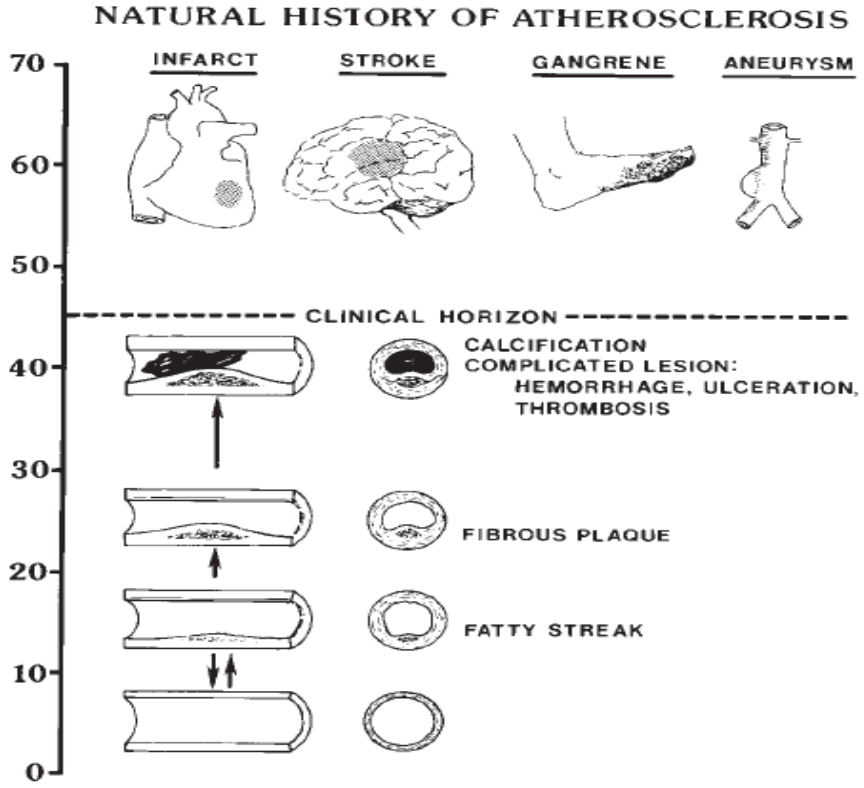


Figure 5: Progression of atherosclerosis with age. [44]

Morphology of atherosclerotic lesions:

Fatty streak is the earliest lesion of atherosclerosis.[44] It was probably present in our hominid ancestors before they emerged as a separate species because fatty streaks occur commonly present in both Old and New World nonhuman primates living in their natural habitat and in other mammalian species.[44] Fatty streak is composed of lipid-filled foamy

macrophages appearing as flat yellow spots which eventually coalesce into elongated streaks.[8] Aortas of infants less than one year old can exhibit fatty streaks, and such lesions are seen in virtually every children older than 10 years of age, regardless of geography, race, sex or environment.[16,17] Fatty streaks may evolve into plaques but not all fatty streaks are destined to become advanced lesions.[17] Microscopically they shows macrophages derived foam cells in the intima.

Atherosclerotic plaque:

Gross: Appear as white to yellow patches on the intima. The plaques have three principal components,

1) Cells, including smooth muscle cells, macrophages and T cells

2) ECM, including collagen, elastic fibers and proteoglycans.

3) Intracellular and extracellular lipid.

Microscopically, the typical plaque has a superficial fibrous cap composed of smooth muscle cells and relatively dense collagen. Beneath and to the side of the cap(the"shoulder") is a more cellular area containing macrophages, T cells and smooth muscle cells. Deep to the fibrous cap is a necrotic core, containing lipid (primarily cholesterol and cholesterol esters), debris from dead cells, foam cells, fibrin, variably organized thrombus and other plasma proteins. The periphery of the lesions shows neovascularization. Few plaques can be composed almost exclusively of smooth muscle cells and fibrous tissue and are called fibrous plaques. Atheroma's often undergo calcification.[16,17]

Consequences of atherosclerotic disease:

1) Atherosclerotic stenosis: Smaller vessels can get occluded, compromising distal tissue perfusion. At approximately 70% fixed occlusion, patients develop chest pain (angina) on exertion.[16,17]

2) Acute plaque change: Plaque rupture or erosion is typically promptly followed by partial or complete vascular thrombosis, resulting in acute tissue infarction. Ruptured plaque can embolize atherosclerotic debris and causes distal vessel obstruction.[17] At present it is impossible to reliably detect individuals who will have plaque disruption or subsequent thrombosis.[17]

3) Plaques that contain large areas of foam cells and extracellular lipid, and those in which the fibrous caps are thin or contain few smooth muscle cells or have clusters of inflammatory cells, are more likely to rupture and are therefore called "vulnerable plaques".[16,17]

4) Hemorrhage into plaque: Intra-plaque hemorrhage may expand the plaque or induce plaque rupture.[16,17]

5) Aneurysm formation: Pressure or ischemic atrophy of the underlying media, with loss of elastic tissue, causes weakness resulting in aneurismal dilation and potential rupture.[16,17]

Classification of Atherosclerosis:

The different morphological lesions was found in atherosclerosis are intimal thickening, intimal xanthoma, pathological intimal thickening, erosion, fibrous cap atheroma, thin cap atheroma and calcified nodule etc. These lesions are based on Modified AHA Classification based on Morphological Description (2000) [table1]. Atherosclerosis doesn't follow orderly, linear pattern of lesion progression. This tends to be ambiguous, because it is not clear whether there is a single sequence of events during the progression of all lesions.[chart 1][7] For example intimal thickening may progress to pathological intimal thickening or it may to fibrous cap atheroma.[7]

Chart 1: Progression of lesions according to the Modified AHA classification based on morphological description (2000) [7]

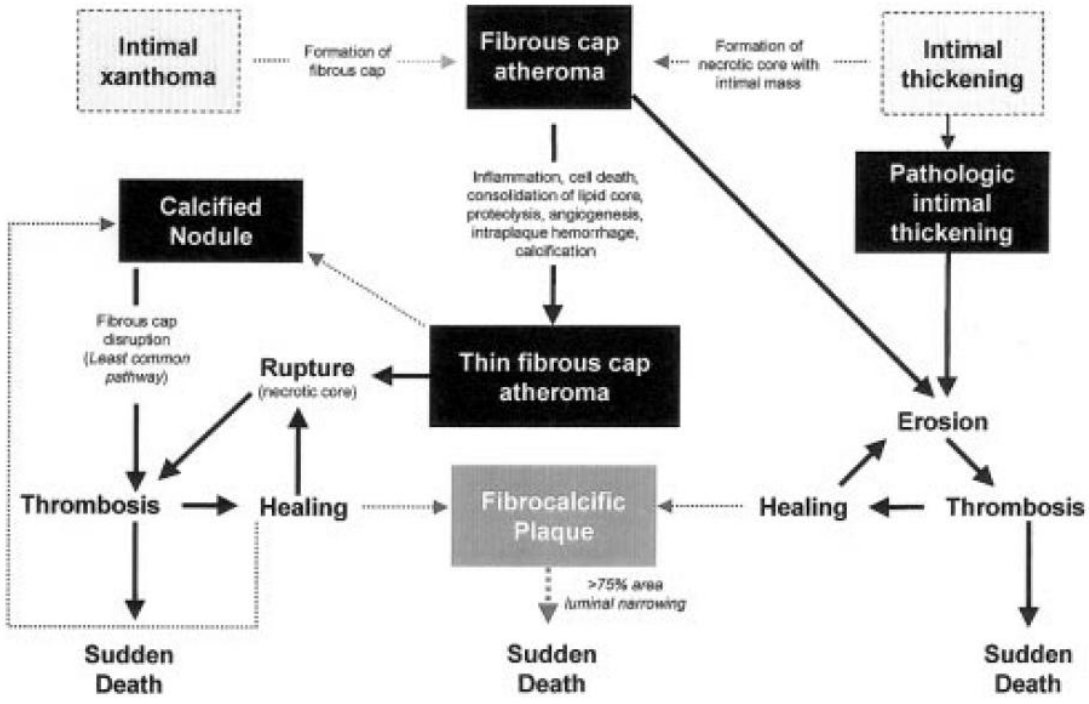


Table 1: Modified AHA Classification based on Morphological Description (2000) [7]

	DESCRIPTION	THROMBOSIS
NONATHEROSCLEROTIC LESIONS		
Intimal thickening	Normal accumulation of smooth muscle cells (SMCs) in the intima in the absence of lipid or macrophage foam cells	Absent
Intimal xanthoma or fatty streak	Luminal accumulation of foam cells without a necrotic core or fibrous cap. Based on animal and human data, such lesions usually regress	Absent
PROGRESSIVE ATHEROSCLEROTIC LESIONS		
Pathological intimal thickening	SMCs in a proteoglycan-rich matrix with areas of extracellular lipid accumulation without necrosis	Absent
Erosion	Luminal thrombosis; plaque same as above	Thrombus mostly mural and infrequently occlusive
Fibrous cap atheroma	Well formed necrotic core with an overlying fibrous cap	Absent
Erosion	Luminal thrombosis; plaque same as above; no communication of thrombus with necrotic core	Thrombus mostly mural and infrequently occlusive
Thin fibrous cap atheroma	A thin fibrous cap infiltrated by macrophages and lymphocytes with rare SMCs and an underlying necrotic core	Absent; may contain intraplaque hemorrage/fibrin
Plaque rupture	Fibroatheroma with cap disruption; luminal thrombus communicates with the underlying necrotic core	Thrombus usually occlusive
Calcified nodule	Eruptive nodular calcification with underlying fibrocalcific plaque	Thrombus usually nonocclusive
Fibrocalcific plaque	Collagen-rich plaque with significant stenosis usually contains large areas of calcification with few inflammatory cells; a necrotic core	Absent

Correlation of atherosclerosis of different vessels:

Atherosclerosis is a generalized vascular disease.[3] Although there is asymmetry in involvement of various arteries in atherosclerosis but the disease usually proceeds in parallel in various organ system.[4] Carotid, coronary aorta and renal vessels are the primary target for atherosclerosis.[8] It has been seen that atherosclerosis is a systemic disease with important sequelae in many other regional circulations, including those supplying the brain, kidneys, limbs and mesentry.[8] Once atherosclerotic disease is apparent in any one vascular territory, there is many fold increased risk for adverse events in many other territories.[8]

Carotid atherosclerosis is the one of the leading cause of ischemic stroke and most of them are due to thromboembolism.[8] The carotid atherosclerosis correlates with the prevalence of vascular end points, including stroke and coronary heart disease.[8] Many studies have shown that there is close relation between coronary, carotid and aortic atherosclerosis.[9] Atherosclerotic renal artery lesion commonly exists as one manifestation of more generalized atherosclerosis.[10] There are many major clinical syndromes associated with aortic atherosclerosis like abdominal aortic aneurysms, aortic dissection, peripheral atheroembolization, and the relatively newly recognized clinical syndromes penetrating aortic ulcer and intramural hematoma.[8] Patients with atherosclerotic carotid artery stenosis were more likely to have renal artery stenosis than patients without carotid artery stenosis.[10] Three vessels coronary artery disease were significantly associated with renal artery atherosclerosis.[45] Increased risk for severe and refractory hypertension as well as renal failure was seen with renal artery atherosclerosis.[21]

Correlation between proximal and distal coronary atherosclerosis with age:

Atherosclerosis in coronary arteries often started at proximal end and it affects more frequently and severe than distal end.[14,46] Some studies had found that atherosclerosis in distal end was more frequent in younger age group compare to proximal end of coronary artery.[46] Proximal part coronary arteries are considered as a "hot spot" for the severe atherosclerosis.[14] Identification of these zones of high risk for acute coronary occlusions will lead to future advances in vulnerable plaque detection technology and potentially locally directed preventive strategies.[14] Proximal coronary artery disease (CAD) has a hereditary basis where as distal CAD is more affected by environmental factors. [15]

Radiological Imaging of atherosclerosis:

Identifying patients at high risk for an acute cardiovascular event such as myocardial infarction or stroke and assessing the total atherosclerotic burden are clinically important.[47] One of the cost effective imaging technique is carotid artery intimal media thickness which can be taken as a indicator of generalized atherosclerosis.[11] The drawback of intimal medial thickness is that sometimes the thickening of intima-media may be due to age related changes or hypertension not only because of athtersclerosis.[13] Another drawback is that, it unable to distinguish between lesions with necrotic core in ultrasound.[13] Several other technique are available to measure carotid atherosclerosis like plaque area or volume.[13] Carotid plaque area generally considered the better predictor of an inflammatory process with atherosclerotic disease rather than intimal medial thickness.[13]

Ultrasonographic studies had shown that there is close association between asymptomatic carotid and aortic atherosclerotic plaques.[9] The likelihood of atherosclerotic renal disease in a patient with coronary or cerebrovascular disease is higher than the patient without such deadly atherosclerotic complications.[4]

Several imaging platforms are available for targeted vascular imaging to acquire information on both anatomy and pathobiology in the same imaging session using either hybrid technology (nuclear combined with CT) or MRI combined with novel probes targeting processes identified by molecular biology to be of importance.[47] CT calcium scoring has gained popularity in detecting atherosclerosis, predicting the CAD risk and has been included into various risk assessment scoring systems including the Framingham Risk Scoring System. The coronary artery calcium (CAC) score is now an independent predictor of CAD.[48]

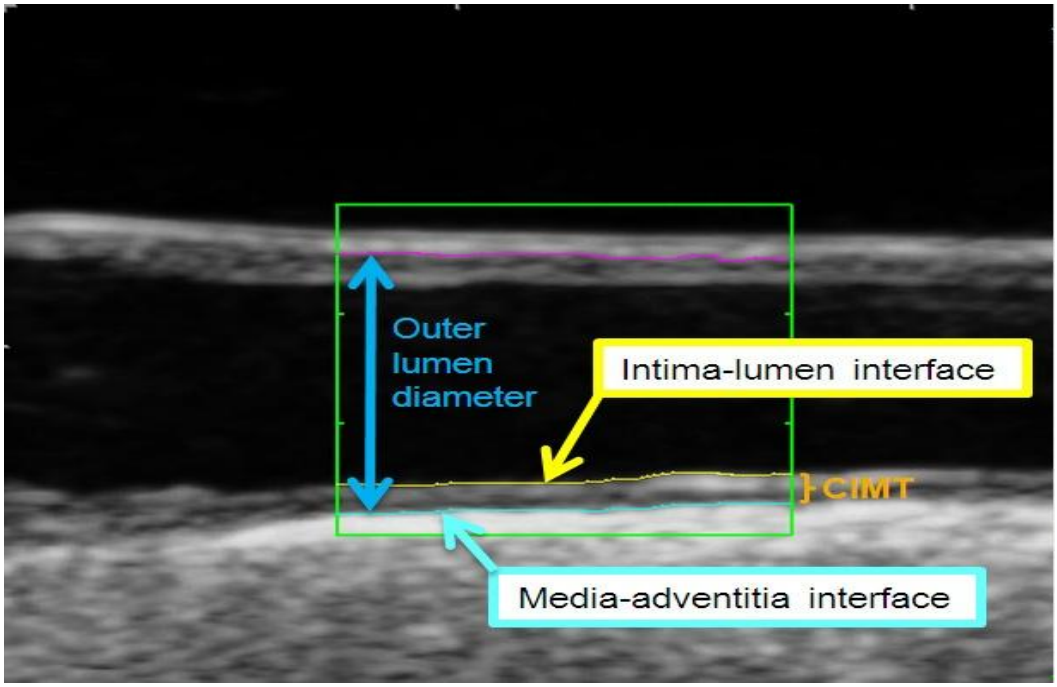


Figure 6: Carotid intimal medial thickness through ultrasound

AUTOPSY TECHNIQUES[49]

Four important techniques of autopsy are:

1. Technique of Rudolf Virchow – Organs are removed one by one. Cranial cavity, spinal cord, thoracic cavity, cervical and abdominal organs are removed in order.

2. Technique of C. Rokitansky – In-situ dissection, in part combined with the removal of organs.

3. Technique of A. Ghon – Thoracic and cervical organs, abdominal organs and urogenital system are removed as organ blocks.

4. Technique of M. Lettule – Thoracic, cervical, abdominal and pelvic are removed as one block and subsequently dissected into organ blocks.

DISSECTION METHODS OF HEART

1. Inflow – outflow technique
2. Short – axis technique
3. Four chamber technique
4. Long – axis technique
5. Base of heart technique

6. Window technique

7. Unrolling technique

8. Injection corrosion technique

9. Dissection of cardiac conduction system

10. Partition technique

❖ INFLOW – OUTFLOW TECHNIQUE

- Suitable for normal heart.
- Follows flow of blood.
- Initial cut is from the inferior venacava to the atrial appendage, sparing the superior venacava with the region of the sinus node.
- The right ventricle is cut 1 cm parallel to the interventricular septum posteriorly and this incision is continued anteriorly to join the pulmonary artery. The left atrium is cut between the right and left pulmonary veins. The left ventricle is cut inferolaterally and this incision is connected to the aorta to expose the left ventricular cavity.
- Valves are cut between commissures.

❖ SHORT – AXIS TECHNIQUE

- Suitable for evaluation of any cardiac lesion.
- Slices expose the largest surface area of myocardium.
- Correspond to the short axis plane produced by two-dimensional echocardiography.

❖ FOUR CHAMBER METHOD:

- Using a long knife, beginning at the cardiac apex, a cut is extended through the acute margin of the right ventricle, the obtuse margin of the left ventricle and the ventricular septum.
- Extend the cut through the mitral and tricuspid valves to the atria. This will divide the heart into two pieces which contain all four chambers.

❖ LONG – AXIS METHOD:

The plane is demarcated by three straight pins:

- Cardiac apex
- Right aortic sinus (adjacent to the right coronary ostium)
- Mitral valve annulus, between the right and left pulmonary veins.
- The heart can be cut along this plane from the apex to the base passing through both the mitral and aortic valves.

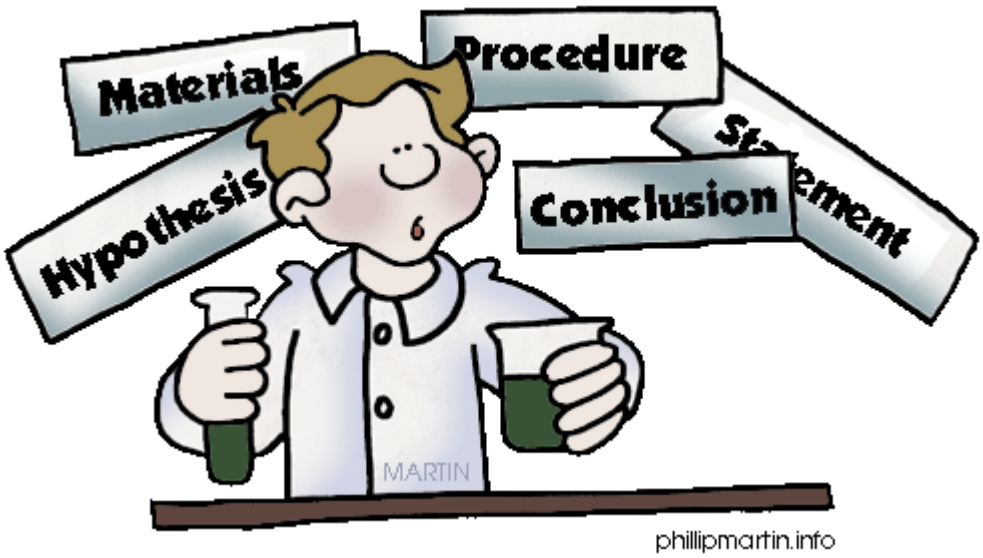
- BASE OF HEART METHOD:
  - Displays all four valves intact
  - Ideal for demonstrating anatomic relationships between valves and the adjacent coronaries and the atrioventricular conduction system.

- WINDOW METHOD
  - For preparation of dry cardiac museum specimens using paraffin and other materials.
  - Heart should be perfusion fixed.
  - Windows should be made in great vessels or chambers to view into the abnormality.

- UNROLLING TECHNIQUE:
  - Used to demonstrate opacified epicardial arteries in a single plane.

- PARTION METHOD:
  - Used to weigh each ventricle separately for detailed assessment of ventricular hypertrophy.

- INJECTION – CORROSION METHOD:
  - Plastic or latex is injected into the coronary vasculature or into cardiac chambers and great vessels.



PART II-RESEARCH METHODOLOGY

RESEARCH METHODOLOGY:

Source of Data:

Heart and aorta along with carotid and renal arteries were collected from autopsies conducted at R.L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar in coordination with the Department of Forensic Medicine.

Clinical data was obtained in each case – name, age, sex, brief relevant history and cause of death. Each autopsy subject was identified, examined and post mortem study was done after obtaining consent from next of kin in case of medical autopsy and requisition from the Police/Department of Forensic Medicine in medicolegal autopsies.

Method of Collection of Data:

a) Study Setting:

This study was carried out at The Department of Pathology, R.L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

b) Study Design:

Cross-sectional study

c) Study Duration:

The study was conducted from 1st January 2012 to 31st August 2013

d) Study Population and Sample Size:

91 cases that underwent autopsy during the study period mentioned.

e) Study procedure:

Autopsy was done by conventional technique, heart and the whole length of aorta along with carotids upto the bifurcation of common carotid artery and the renal arteries were removed and fixed in 10% formalin. The heart was dissected along the direction of flow of blood and aorta along the posterior surface. The three major coronary arteries was identified and cut transversely at 0.5 cm intervals until their entry into the musculature.

Bits from the suspicious or definite atherosclerotic lesions were taken. If no lesions were found one bit from each common carotids, left anterior descending, right coronary, left circumflex, ascending aorta, thoracic aorta and abdominal aorta were taken. From all coronaries bits from proximal and distal part was taken separately. In carotids, bits were taken just before the bifurcation of common carotid artery and from renal arteries bits were taken nearest to the renal ostia. Histopathological studywas done by routine tissue processing and H & E staining.

Special stain like Verhoeff van gieson stain was used wherever necessary.

The microscopic grading of atherosclerosis was done according to the modified American Heart Association Classification(AHA) of atherosclerosis using morphological descriptions(2000) which is better than the earlier AHA classification with regard to the description of intermediate lesions which was prone for future development of complicated atherosclerosis.[7] The atherosclerotic lesions were divided into non-atheromatous lesions (Intimal thickening and Intimal Xanthoma) and progressive atheromatous lesions [Pathological intimal thickening(PIT), Thick Fibrous cap atheroma(FCA) and Thin fibrous cap atheroma(TFCA), Calcified nodule(CN) and Fibrocalcific plaque(FCP)]. Pathological intimal thickening is the intermediate lesion that

has the potential to progress to advanced atherosclerotic lesion. Pathological intimal thickening can be associated with erosion which can lead to non-occlusive thrombus formation. Thin fibrous cap atheromas are the vulnerable plaques which have the capability to rupture and cause thrombus formation which can occlude the artery. They are usually associated with increased amount of inflammatory cells.

f) Statistical Analysis:

Statistical analysis was done using chi square test to see significance of association.

INCLUSION CRITERIA:

1. Patients of age above 3 years on whom clinical and medicolegal autopsies had been done to ascertain the cause of death.

EXCLUSION CRITERIA:

1. Autopsies on exhumed bodies.
2. Autopsies on poorly preserved bodies that was more than 3 days old where autolytic changes would have taken place.

RESULTS

AGE:

In the present study, the age ranged from 7 to 72 years. Mean age was 39.23±14.24 years. Majority of patients belonged to the 4^{th} decade of life (28.5%), followed by 3^{rd} & 5^{th} decade (19.8%).

Table 2: Age Distribution

AGE GROUP (YEARS)	NUMBER	PERCENT (%)
0-9	**1**	**1.1**
10-19	**4**	**4.4**
20-29	**18**	**19.8**
30-39	**26**	**28.5**
40-49	**18**	**19.8**
50-59	**12**	**13.2**
60-69	**9**	**9.9**
70-79	**3**	**3.3**
TOTAL	**91**	**100**

Chart 2: Age Distribution

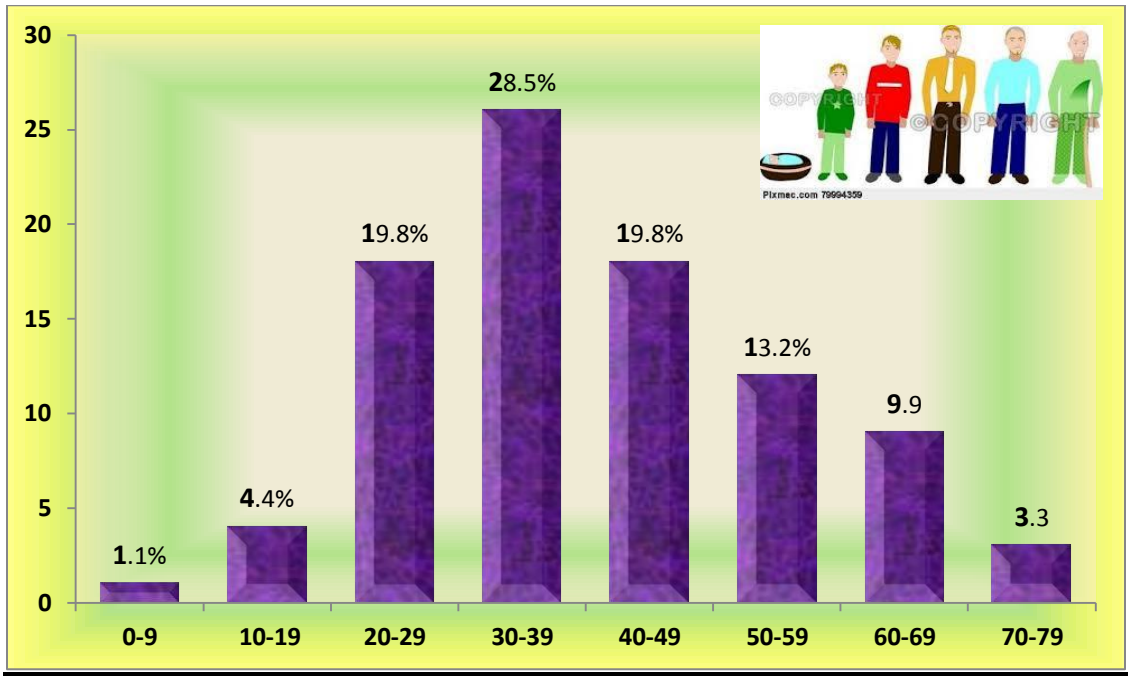
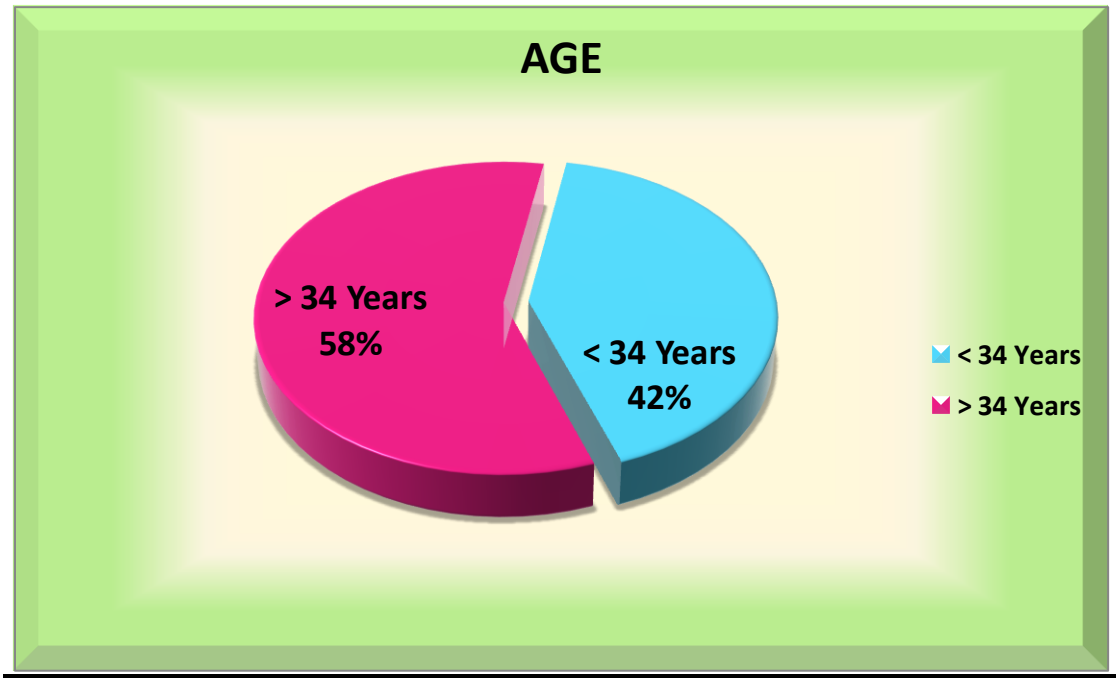


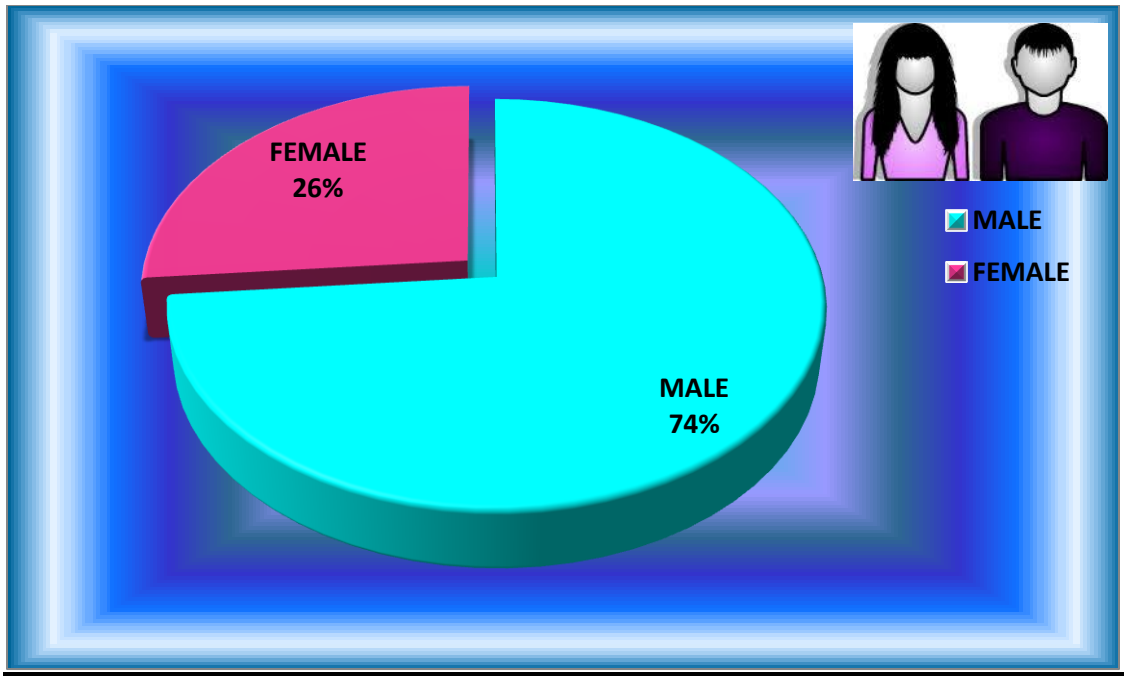
Chart 3: Age Distribution



GENDER:

In the present study, the number of males was 67(74%) and number of females was 24 (26%). The male: female ratio was 2.79.

Chart 4: Gender Distribution

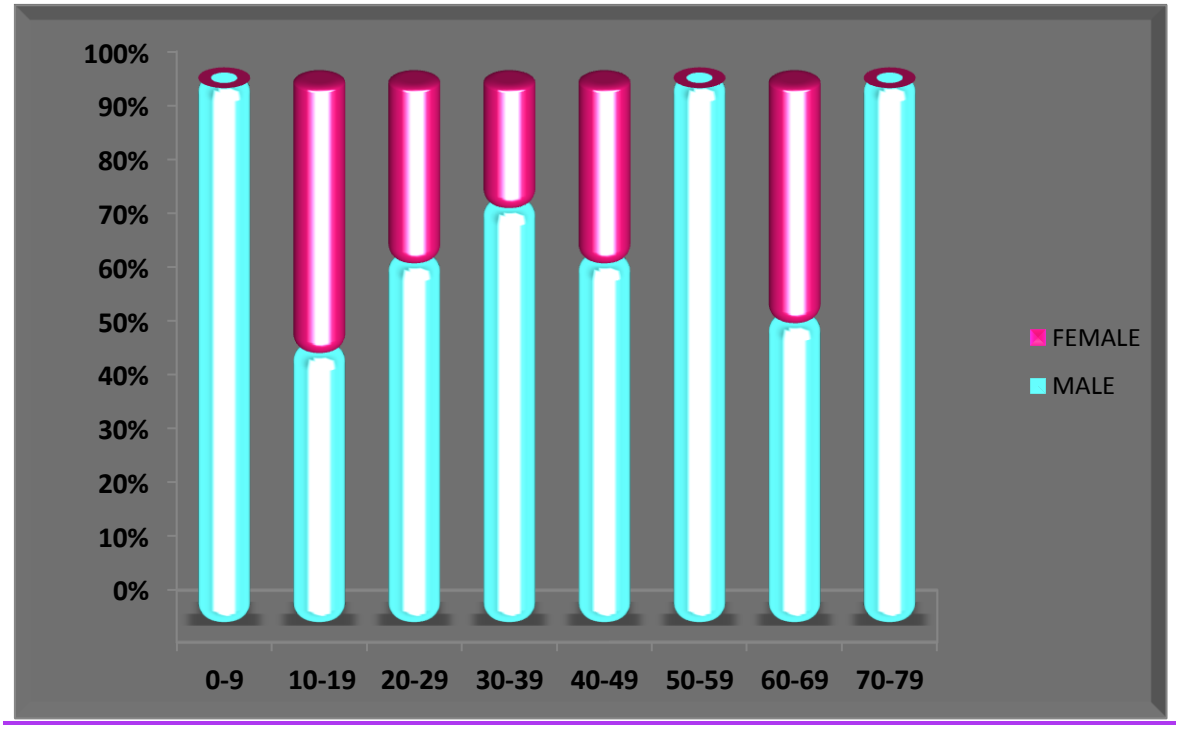


DEMOGRAPHIC ANALYSIS OF AGE AND GENDER DISTRIBUTION

Table 3: Age and Gender Distribution

AGE GROUP (YEARS)	FEMALE	MALE	NUMBER
0-9	1	0	1
10-19	2	2	4
20-29	12	6	18
30-39	20	6	26
40-49	12	6	18
50-59	12	0	12
60-69	5	4	9
70-79	3	0	3
TOTAL	24	67	91

Chart 5: Age and Gender Distribution



In present study the mean age for females was 37.33±13.9 years and mean age for males was 40±14.39 years.

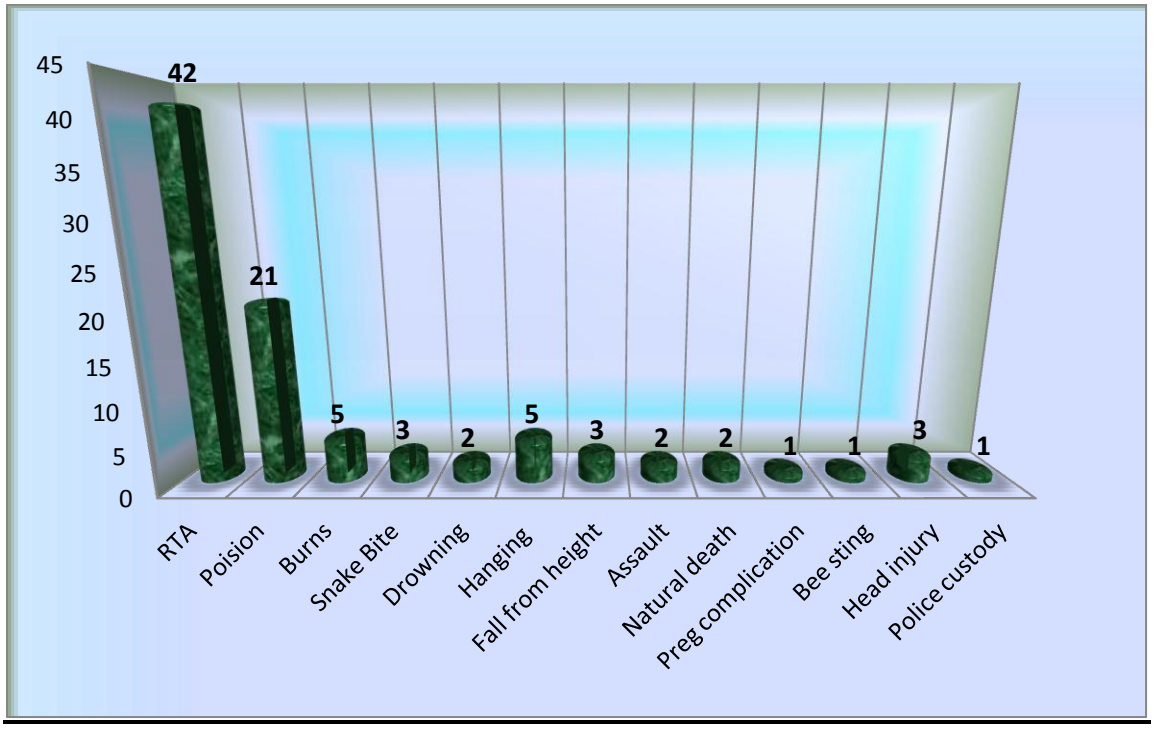
CLINICAL/FORENSIC CAUSE OF DEATH:

In the current study, the most common cause of death was road traffic accidents (RTA) which occurred in 42(46.1%) cases, followed by poisoning 21(23.1%) cases, then hanging and burns 5(5.5%) cases in each category.

Table 4: Cause of Death Distribution

CAUSE OF DEATH	NUMBER	PERCENT (%)
RTA	42	46.1
Poison	21	23.1
Burns	5	5.5
Snake Bite	3	3.3
Drowning	2	2.2
Hanging	5	5.5
Fall from height	3	3.3
Assault	2	2.2
Natural death	2	2.2
Pregnancy complication	1	1.1
Bee sting	1	1.1
Head injury	3	3.3
Police custody	1	1.1
TOTAL	91	100

Chart 6: Cause of Death



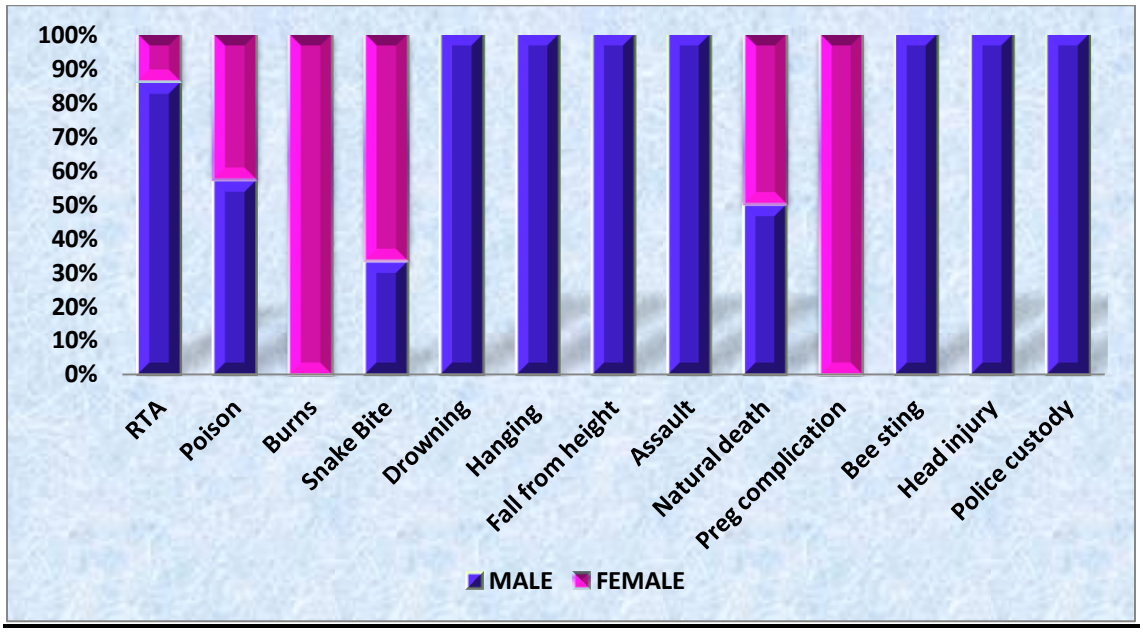
CLINICAL/FORENSIC CAUSE OF DEATH AND GENDER DISTRIBUTION:

In this study, maximum victims of RTAs and all victims of drowning, hanging and assault were males and all victims of burns were females.

Table 5: Cause of Death and Gender Distribution

CAUSE OF DEATH	MALE	FEMALE	NUMBER
RTA	**36**	**6**	**42**
Poison	**12**	**9**	**21**
Burns	**0**	**5**	**5**
Snake Bite	**1**	**2**	**3**
Drowning	**2**	**0**	**2**
Hanging	**5**	**0**	**5**
Fall from height	**3**	**0**	**3**
Assault	**2**	**0**	**2**
Natural death	**1**	**1**	**2**
Pregnancy complication	**0**	**1**	**1**
Bee sting	**1**	**0**	**1**
Head injury	**3**	**0**	**3**
Police custody	**1**	**0**	**1**
TOTAL	**67**	**24**	**91**

Chart 7: Cause of Death and Gender Distribution



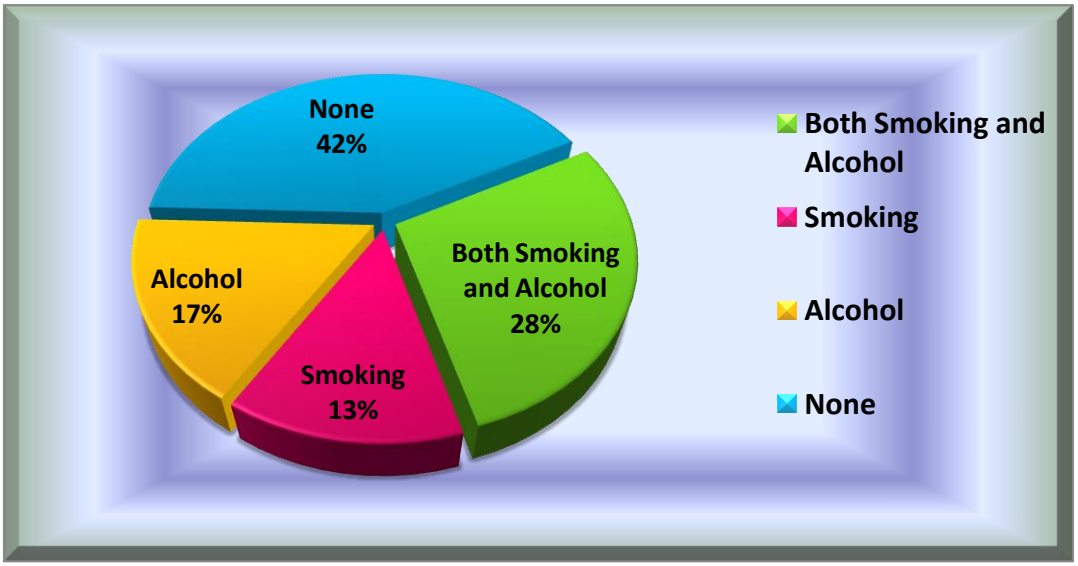
HISTORY OF SMOKING AND ALCOHOL:

In present study history of alcohol, and smoking were taken in 60 cases, out of which 25 (42%) cases had no history of smoking and alcohol consumption.

Table 6: History of Alcohol and Smoking

HISTORY	NUMBER	PERCENT (%)
Both Smoking and Alcohol	**17**	**28**
Smoking	**8**	**13**
Alcohol	**10**	**17**
None	**25**	**42**

Chart 8: History of Alcohol and smoking



GENDER DISTRIBUTION WITH HISTORY OF SMOKING AND ALCOHOL:

In present study none of the female case had history of smoking and alcohol consumption

HISTORY OF SMOKING/ALCHOL WITH ATHEROSCLEROSIS:

Table 7: History of Alcohol and Smoking with Atherosclerosis:

	Non Atherosclerotic lesions		Progressive Atherosclerotic lesions	
	Number	**Percent**	**Number**	**Percent**
Both Smoking & Alcohol	1	9%	16	91%
Alcohol	4	40%	6	60%
Smoking	2	29%	5	71%
None	11	44%	14	56%

In the Patients with history of both smoking and alcohol consumption, the progressive-atherosclerotic lesion (PAL) was found in 16(91%) cases and non-atherosclerotic lesion (NAL) was found in only 1(9%) case. Those who don't have history of both smoking and alcohol consumption, the PAL was found in 14(56%) and NAL was found in 11(44%) cases. In the patient with history of only smoking, the number of PAL and NAL was found in 5(71%) and 2(29%) cases respectively. Patient with history of only alcohol consumption, the number of PAL was 6(60%) cases and NAL was 4(40%) cases.

BMI and Atherosclerosis:

In the present study, total 91 cases were collected and Body Mass index (BMI) was calculated in each case using the formula [BMI = Weight (kg) / [Height (m) x Height (m)]. The degree of atherosclerosis was studied in each case. The Normal BMI cut off for all the groups was 18.5 to 24.9 kg/M^2, below this range were considered as underweight and above this range were the overweight cases.[28] According to data taken from the National Family Health Survey - III, 2005-2006. [NFHS 3, Adult BMI in India] state of Karnataka, which shows that 37% of women and 30% of males were overweight. The mean of BMI for all the cases in present study were 23.17 ± 3.73 kg/M^2. [28]

In the females only 1(4%) case was overweight and 4(17%) cases were underweight whereas in males overweight and underweight cases were 22(33%) and 5(7%) respectively. The only one overweight female had progressive atherosclerotic lesion (PAL) while the females with normal range BMI, Number of NAL and PAL cases were 6 (33%) and 12 (67%) respectively. In the underweight females number of NAL and PAL cases were 3(60%) and 2 (40%) respectively. In underweight males number of

NAL and PAL cases were 3(60%) and 2(40%) respectively while males with normal BMI, number of NAL and PAL cases were 10(26%) and 29(74%) respectively. In overweight males number of NAL and PAL cases were 7(30%) and 16(70%) respectively.

Chart 9: BMI and atherosclerosis in females:

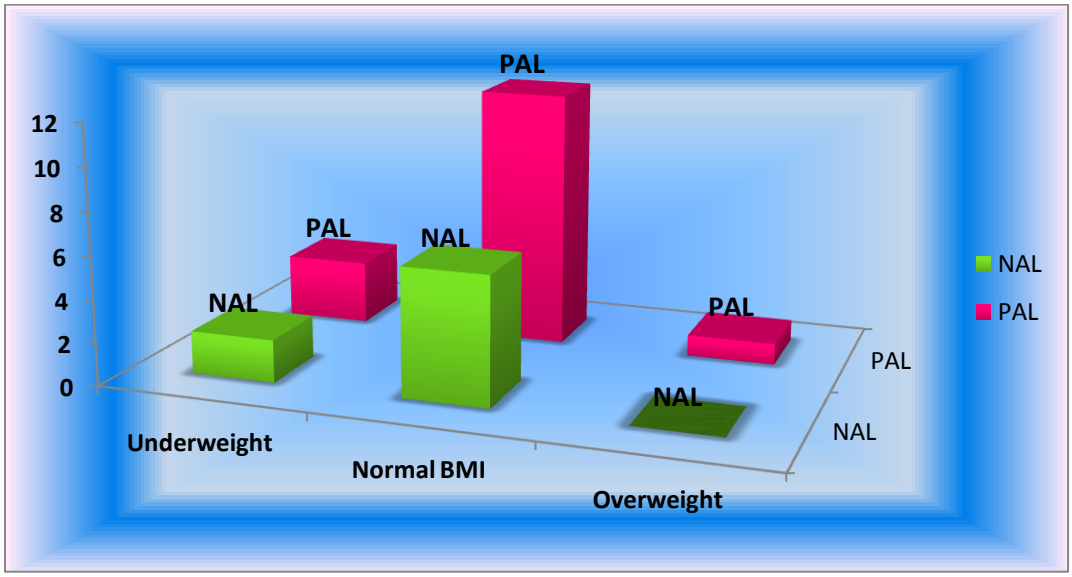
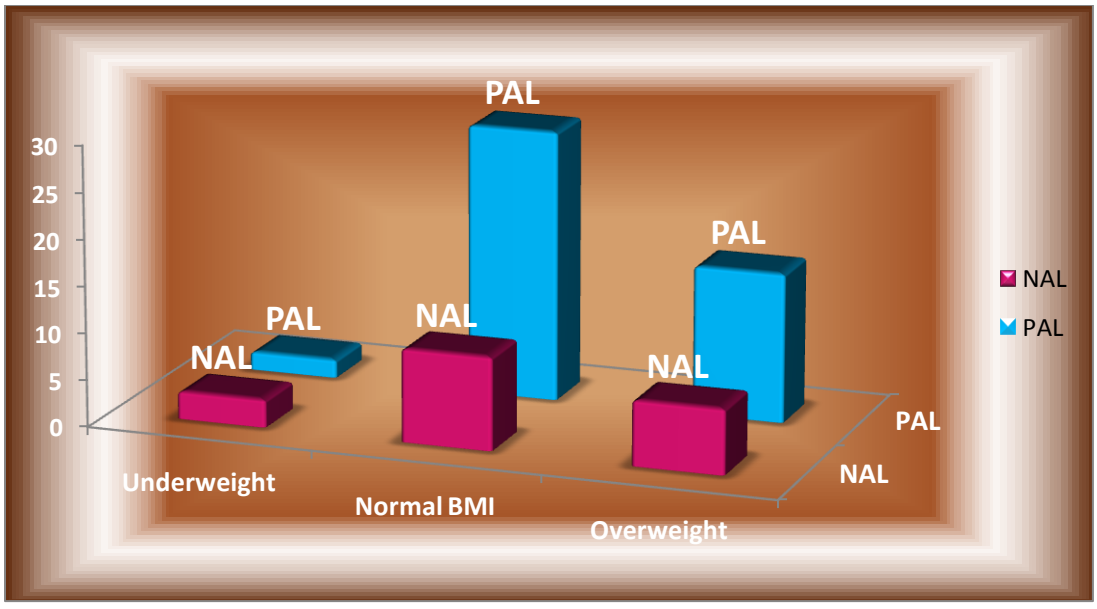


Chart 10: BMI and atherosclerosis in males:



GROSS FINDINGS

Gross Examination of Carotid Arteries:

Table 8: Gross examination findings of carotid arteries based onage and sex distribution:

	Right Carotid Artery				LeftCarotid Artery			
	NVL	**FS**	**FP**	**CL**	**NVL**	**FS**	**FP**	**CL**
Overall	42	37	11	1	46	36	8	1
Age <34 Y	28	10	0	0	24	14	0	0
Age >34 Y	14	27	11	1	22	22	8	1
Female	4	13	7	0	5	14	5	0
Male	38	24	4	1	41	22	3	1

NVL- No visible lesion; FS- Fatty streak; FP- Fatty plaque; CL- Complicated lesion

On gross examination, the atherosclerotic lesions was divided into no visible lesion, fatty streak, fibrous plaque and complicated atherosclerotic lesions like calcification, ulceration and thrombosis.[44] Combined fibrous plaque and complicated atherosclerotic lesions are considered as raised lesions.[44]

Gross examination of right coronary artery had shown that, in older individuals 12(23%) cases and in females 7(29%) cases had raised lesions.

Gross examination of right coronary artery had shown that, in older individuals 9(17%) cases and in females 5(21%) cases had raised lesions.

Gross Examination of Coronary Arteries:

Table 9: Gross examination findings of coronary arteries based on age and sex distribution:

Gross examination findings of coronaries arteries												
	LAD				RCA				LCA			
	NVL	FS	FP	CL	NVL	FS	FP	CL	NVL	FS	FP	CL
Overall	4	40	43	4	11	48	30	2	10	49	29	3
Age<34	4	23	10	1	8	25	5	0	10	20	8	0
Age>34	0	17	33	3	3	23	25	2	0	29	21	3
Female	2	13	9	0	4	13	7	0	5	14	5	0
Male	2	27	34	4	7	35	23	2	5	35	24	3

NVL- No visible lesion; FS- Fatty streak; FP- Fatty plaque; CL- Complicated lesion

Gross examination of left anterior descending artery had shown that, in older individuals 36(68%) cases and in males 38(57%) cases had raised lesions

Gross examination of right coronary artery had shown that, in older individual's 27(51%) cases and in males 25(37%) cases had raised lesions.

Gross examination of right coronary artery had shown that, in older individuals 24 (47%) cases and in males 27(36%) cases had raised lesions.

Gross examination Aorta:

Table 10: Gross examination findings of aorta based on age and sex distribution:

	Ascending Aorta				Thoracic Aorta				Abdominal Aorta			
	NVL	**FS**	**FP**	**CL**	**NVL**	**FS**	**FP**	**CL**	**NVL**	**FS**	**FP**	**CL**
Overall	22	32	18	19	20	50	16	5	20	46	17	8
Age<34	17	15	6	0	14	21	3	0	17	17	4	0
Age>34	5	17	12	19	6	29	13	5	3	29	13	8
Female	9	6	3	6	8	13	1	2	6	13	5	0
Male	13	26	15	13	12	37	15	3	18	33	12	8

NVL- No visible lesion; FS- Fatty streak; FP- Fatty plaque; CL- Complicated lesion

Gross examination of the ascending aorta had shown that, in older individuals 31(59%) cases and in males 28(42%) cases had raised lesions.

Gross examination of the thoracic aorta had shown that, in older individual's 18(34%) cases and in males 18(27%) cases had raised lesions.

Gross examination of the abdominal aorta had shown that, in older individuals 21(40%) cases and in males 20(29%) cases had raised lesions.

Gross examination of Renal arteries:

Table 11: Gross examination findings of renal arteries based on age and sex distribution:

	Right Renal Artery				Left Renal Artery			
	NVL	**FS**	**FP**	**CL**	**NVL**	**FS**	**FP**	**CL**
Overall	73	15	2	1	67	22	1	1
Age <34 Y	33	5	0	0	32	6	0	0
Age >34 Y	40	10	2	1	35	16	1	1
Female	21	3	0	0	20	4	0	0
Male	52	12	2	1	45	18	1	1

NVL- No visible lesion; FS- Fatty streak; FP- Fatty plaque; CL- Complicated lesion

Gross examination of the right renal artery had shown that, in older individuals 3(6%) cases and in males 3(4%) cases had raised lesions

Gross examination of the left renal artery had shown that, in older individuals 2(4%) and in males 2(3%) cases had raised lesions.

GROSS COMPARISION OF RAISED LESIONS IN ALL ARTERIES:

Raised lesion consisting of both fibrous plaque and complicated lesions.[44] It determines the risk of clinically manifest coronary artery disease both for individuals and for population.[44]

Table 12: Overall distribution of Raised lesions in all arteries:

Overall distribution of Raised lesions in all arteries (percent)									
R Ca	L Ca	LAD	RCA	LCA	A.A	T.A	Ab A	R R	L R
10	12	52	35	35	41	23	28	3	2

Maximum raised lesion was found in LAD followed by abdominal aorta

Chart 11: Overall distribution of Raised lesions in all arteries:

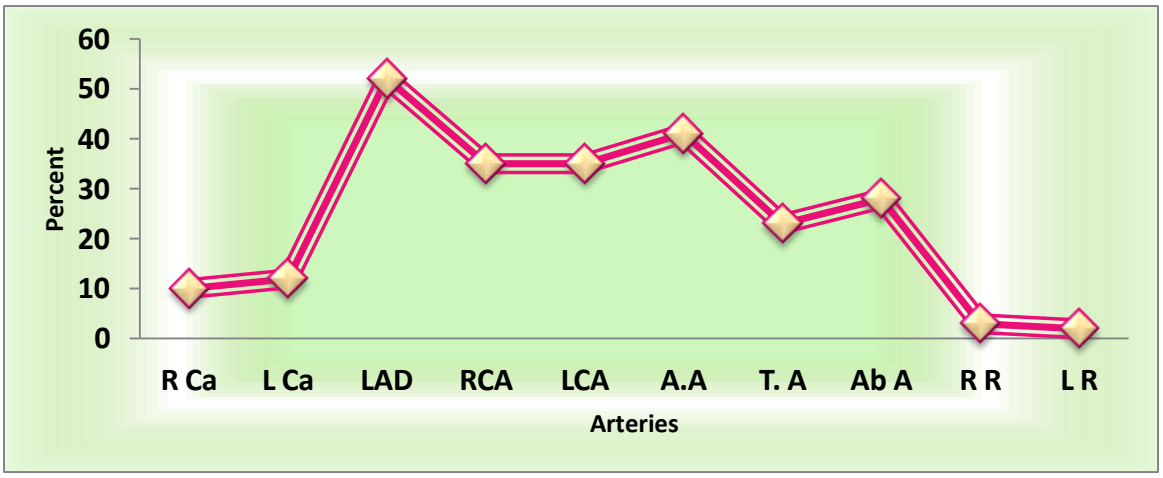


Table 13: Age distribution of Raised lesions in all arteries:

	Age distribution of Raised lesions in all arteries (percent)									
	R Ca	L Ca	LAD	RCA	LCA	A.A	T.A	Ab A	R R	L R
< 34 Y	0	0	29	13	21	16	8	11	0	0
> 34 Y	23	17	68	51	45	59	34	40	6	4

LAD had highest number of raised lesion both in younger and older age group.

Chart 12: Age distribution of Raised lesions in all arteries:

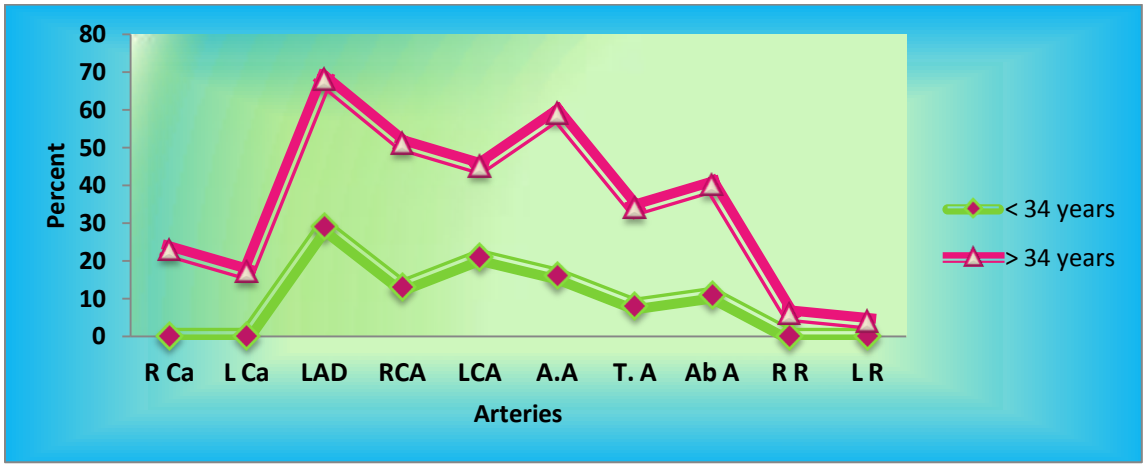
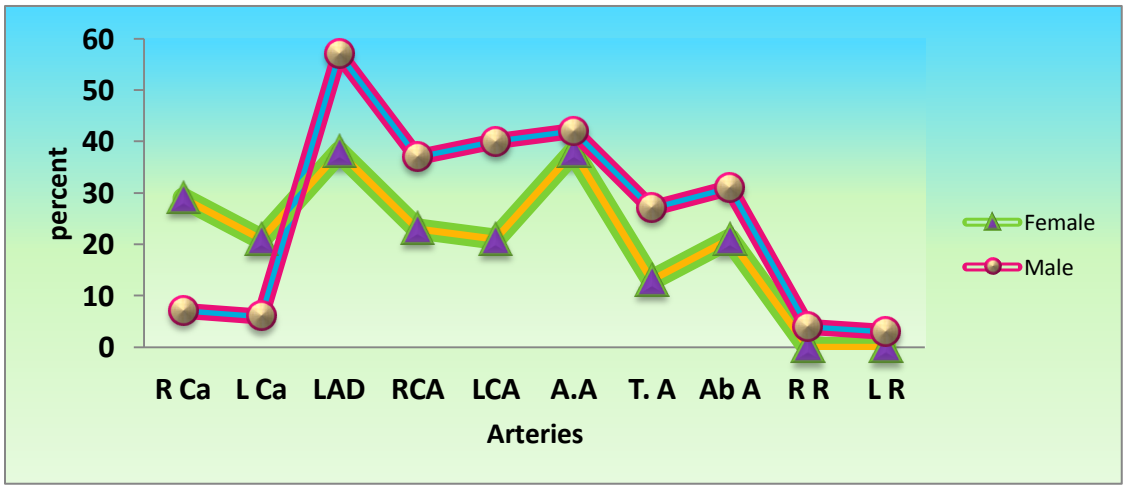


Table 14: Sex distribution of Raised lesions in all arteries:

	Sex distribution of Raised lesions in all arteries (percent)									
	R Ca	L Ca	LAD	RCA	LCA	A.A	T.A	Ab A	R R	L R
Female	29	21	38	29	21	38	13	21	0	0
Male	7	6	57	37	40	42	27	31	4	3

In female maximum raised lesion was found in LAD and abdominal aorta while in males maximum raised lesion was found in LAD.

Chart 13: Sex distribution of Raised lesions in all arteries:



MICROSCOPIC FINDINGS

Microscopic examination of Carotid Arteries:

Table 15: Microscopic examination findings of Carotid arteries with age and sex distribution:

	Right Carotid Artery						Left Carotid Artery					
	IT	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**	**IT**	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**
Overall	44	35	10	0	1	1	49	32	9	1	0	0
Age<34	28	10	0	0	0	0	26	12	0	0	0	0
Age>34	16	25	10	0	1	1	23	20	9	1	0	0
Female	16	7	1	0	0	0	17	6	1	0	0	0
Male	28	28	9	0	1	1	32	26	8	1	0	0

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

IT and IX was considered as non-atherosclerotic lesion (NAL) whereas PIT, FCA, TFCA and FCP are considered as progressive atherosclerotic lesion (PAL).

There was no PAL in young age group in both carotids. PAL was significantly more in older individuals and in males in both carotid arteries. Males had 11(16%) cases of PAL and female had only 1(4%) case of PAL i.e male had 4 times more atherosclerotic lesion then female.

Microscopic examination of Left Anterior Descending Artery:

Table 16: Microscopic examination findings of left anterior descending artery with age and sex distribution:

	Proximal LAD						Distal LAD					
	IT	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**	**IT**	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**
Overall	4	41	38	6	0	2	15	51	20	1	3	1
Age<34	4	23	10	0	0	1	13	21	3	0	0	1
Age>34	0	17	28	6	0	1	2	30	17	1	3	0
Female	2	14	6	2	0	0	7	13	4	0	0	0
Male	2	27	32	4	0	2	8	38	16	1	3	1

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

PAL was significantly more in older individuals and in males in both proximal and distal LAD. In proximal and distal LAD number of PAL cases were 44(51%) and 25(27.5%) respectively. Number of PAL cases in older age group in proximal and distal LAD were 35(66%) and 21(40%) respectively. One young male had FCP in the distal LAD. Three older male had TFCA in the distal LAD.

Microscopic examination ofRight Coronary Artery:

Table 17: Microscopic examination findings of right coronary artery with age and sex distribution:

	Proximal RCA						Distal RCA					
	IT	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**	**IT**	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**
Overall	12	47	28	4	0	0	23	60	7	0	1	0
Age<34	9	24	5	0	0	0	16	20	2	0	0	0
Age>34	3	23	23	4	0	0	7	40	5	0	1	0
Female	4	13	7	0	0	0	6	18	0	0	0	0
Male	8	34	21	4	0	0	17	42	7	0	1	0

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

PAL was significantly more in older individuals and in males in both proximal and distal RCA. In proximal and distal RCA number of PAL cases was 32(35%) and 8(9%) respectively. Number of PAL cases in older age group in proximal and distal RCA was 27(51%) and 6(11%) respectively. One of the older male had TFCA in the distal RCA.

Microscopic examination of Left Circumflex Coronary Artery:

Table 18: Microscopic examination findings of left circumflex coronary artery with age and sex distribution:

	Proximal LCA						Distal LCA					
	IT	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**	**IT**	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**
Overall	12	45	28	5	0	1	28	43	18	0	0	2
Age<34	12	16	8	2	0	0	18	15	5	0	0	0
Age>34	0	29	20	3	0	1	10	28	13	0	0	2
Female	5	14	5	0	0	0	7	12	5	0	0	0
Male	7	41	23	5	0	1	21	31	13	0	0	2

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

PAL was significantly more in older individuals and in males both in proximal and distal LCA. In proximal and distal LCA number of PAL cases were 34(37%) and 20(22%) respectively. Number of PAL cases in older age group in proximal and distal LCA was 24(45%) and 15(28%) respectively. Two young male had FCA in proximal LCA. Two older male had FCP in the distal LCA which is more compared to the proximal coronaries.

Microscopic examination of Ascending Aorta:

Table 19: Microscopic examination findings of ascending aorta with age and sex distribution:

	IT	IX	PIT	FCA	TFCA	FCP
Overall	28	27	14	4	0	18
Age<34	21	11	5	1	0	0
Age>34	7	16	9	3	0	18
Female	12	3	2	1	0	6
Male	16	24	12	3	0	12

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

PAL was significantly more in older individuals. Prevalence of PAL in males and females were 27(40%) and 9(37.5%) cases which was near about in equal proportion. One of the important finding noted here was 18 (19.8%) of older individuals had the FCP.

Microscopic examination of Thoracic Aorta:

Table 20: Microscopic examination findings of thoracic aorta with age and sex distribution:

	IT	IX	PIT	FCA	TFCA	FCP
Overall	20	53	11	3	0	4
Age<34	16	21	1	0	0	0
Age>34	4	31	10	3	0	4
Female	8	13	0	1	0	2
Male	12	40	11	2	0	2

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

PAL was found in 1(2.65%) case of the younger age group and 17 (32%) cases in the older age group. It shows that older age group had 12 times more prevalence of atherosclerosis compare to younger individuals. Male had 15 (22%) cases and whereas female had 3 (12.5%) cases of PAL i.e males had 5 times more prevalence of atherosclerosis then female in thoracic aorta.

Microscopic examination of Abdominal Aorta:

Table 21: Microscopic examination findings of abdominal aorta with age and sex distribution:

	IT	IX	PIT	FCA	TFCA	FCP
Overall	22	42	15	6	2	4
Age<34	19	13	6	0	0	0
Age>34	3	29	9	6	2	4
Female	7	10	4	1	2	0
Male	15	32	9	5	2	4

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

In abdominal aorta PAL was significantly more in older individuals and in males. In younger age group PAL was found in 6 (15.8%) cases and in older individual it was found in 21 (39.6%) cases. All the lesions which were present in the younger age group were PIT. Males had 20 (29.9%) cases and female had 7 (29.2%) cases of PAL.

Microscopic examination of Renal Arteries:

Table 22: Microscopic examination findings of renal arteries with age and sex distribution:

	Right Renal Artery						Left Renal Artery					
	IT	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**	**IT**	**IX**	**PIT**	**FCA**	**TFCA**	**FCP**
Overall	73	15	2	1	0	0	70	19	1	1	0	0
Age<34	32	6	0	0	0	0	31	7	0	0	0	0
Age>34	41	9	2	1	0	0	39	12	1	1	0	0
Female	22	2	0	0	0	0	20	4	0	0	0	0
Male	51	13	2	1	0	0	50	15	1	1	0	0

IT-Intimal Thickening; **PIT-Pathological Intimal Thickening;**
IX-Intimal Xanthoma; **FCA - Fibrous Cap Atheroma;**
FCP-Fibrocalcific plaque; **TFCA-Thin Fibrous Cap Atheroma;**

No PAL was found in younger age group in both right and left renal artery. In case of males the number of PAL cases in right and left renal artery was 3(3.3%) and 2(2.2%) respectively. None of the females had PAL in any of the renal arteries. Only 1 FCA was found in older age group male.

Microscopic examination of Proximal and Distal Coronary Atherosclerosis in relation to Age groups:

Table 23: Microscopic examination of proximal and distal coronary atherosclerosis in relation to age groups:

Age distribution of proximal and distal coronary atherosclerosis (No of PAL cases)				
	Age less than 34 years		Age more than 34 years	
Artery	**Proximal**	**Distal**	**Proximal**	**Distal**
LAD	11(29%)	4(11%)	35(66%)	21(40%)
RCA	5(13%)	2(5%)	27(51%)	6(11%)
LCA	10(26%)	5(11%)	24(45%)	15((28%)

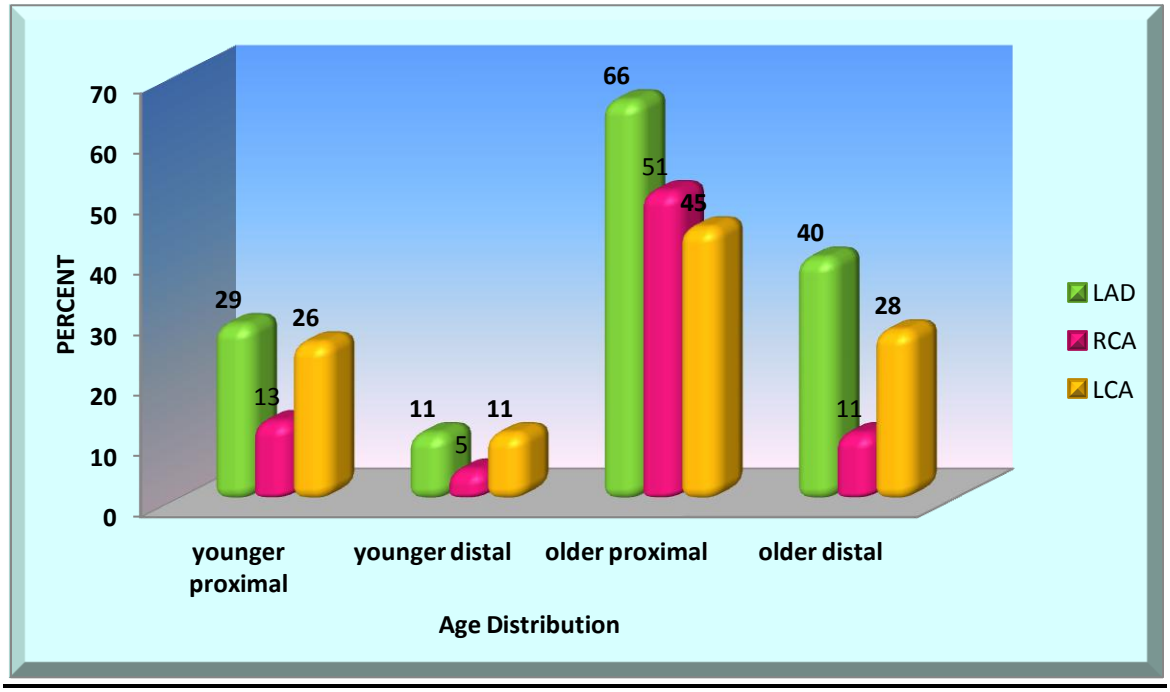
LAD – Left anterior descending artery; **RCA – Right coronary artery**
LCA – Left circumflex coronary artery: **No – number**

In present study in younger age group, number of PAL cases in proximal part of LAD, RCA and LCA were 11(29%), 5(13%) and 10(26%) respectively.

In older age group, number of PAL cases in proximal part of LAD, RCA and LCA were 35(66%), 27(51%) and 24(54%) respectively.

From above date it is seen that proximal part had double the prevalence of atherosclerosis than distal part of coronaries.

Chart 14: Age distribution of proximal and distal coronary atherosclerosis:



Morphological photographs of atherosclerotic lesions:

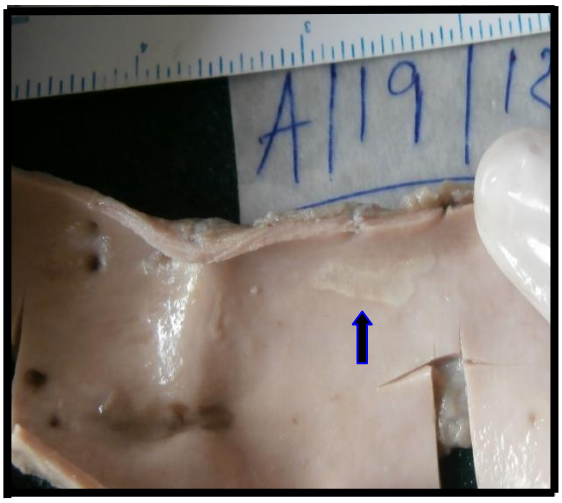


Figure 7: Gross photograph of aorta showing fatty streaks

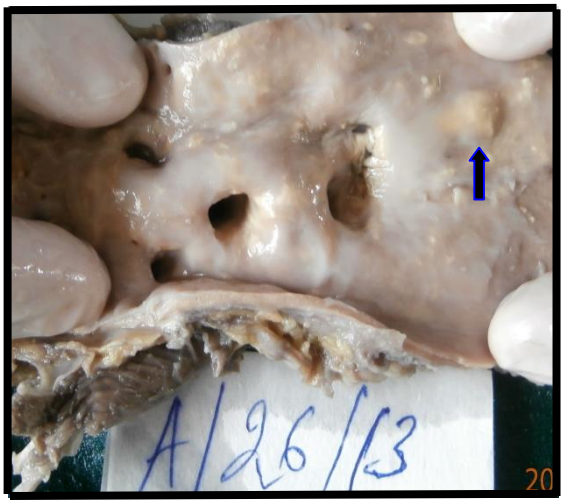


Figure 8: Gross photograph of aorta showing fibrous plaques

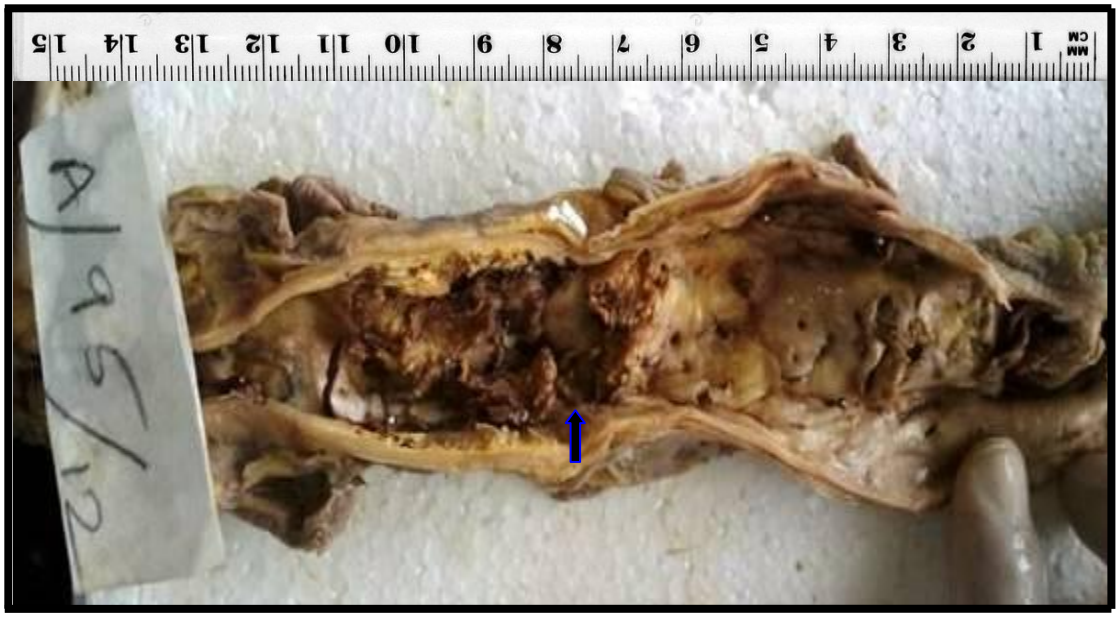


Figure 9: Gross photograph of aorta showing complicated atherosclerotic lesions

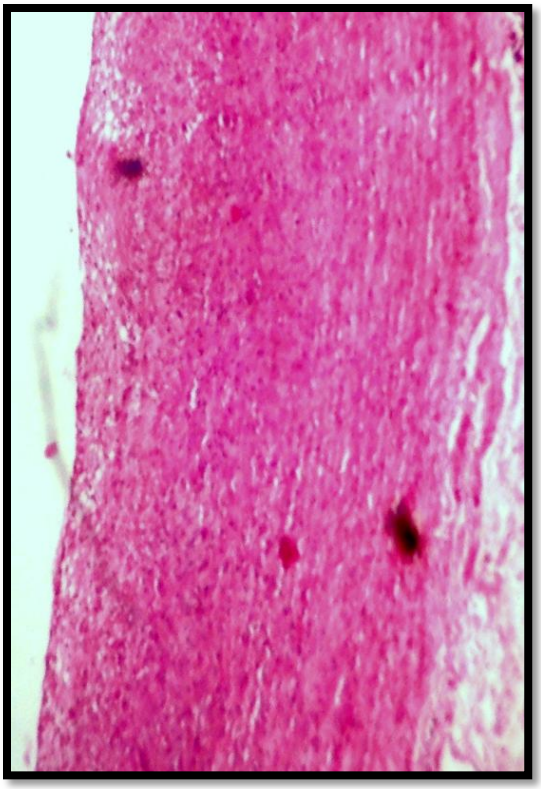


Figure 10: Microscopic photograph of carotid artery showing intimal thickening

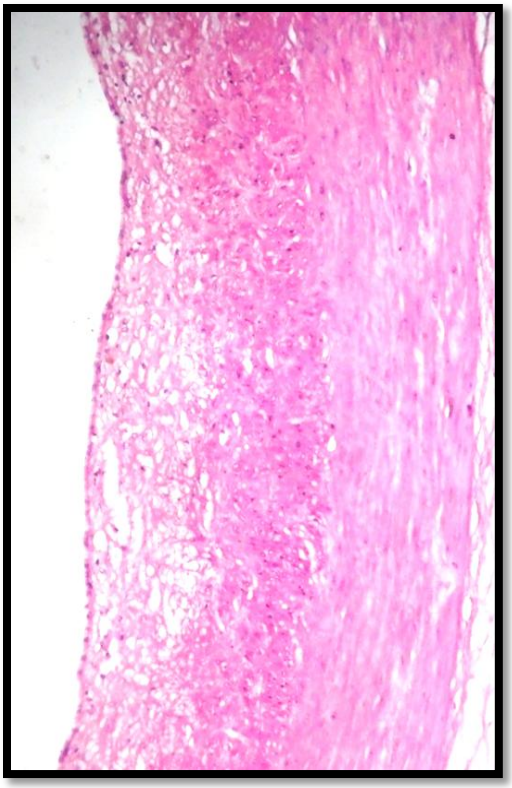


Figure 11: Microscopic photograph of renal artery showing intimal xanthoma

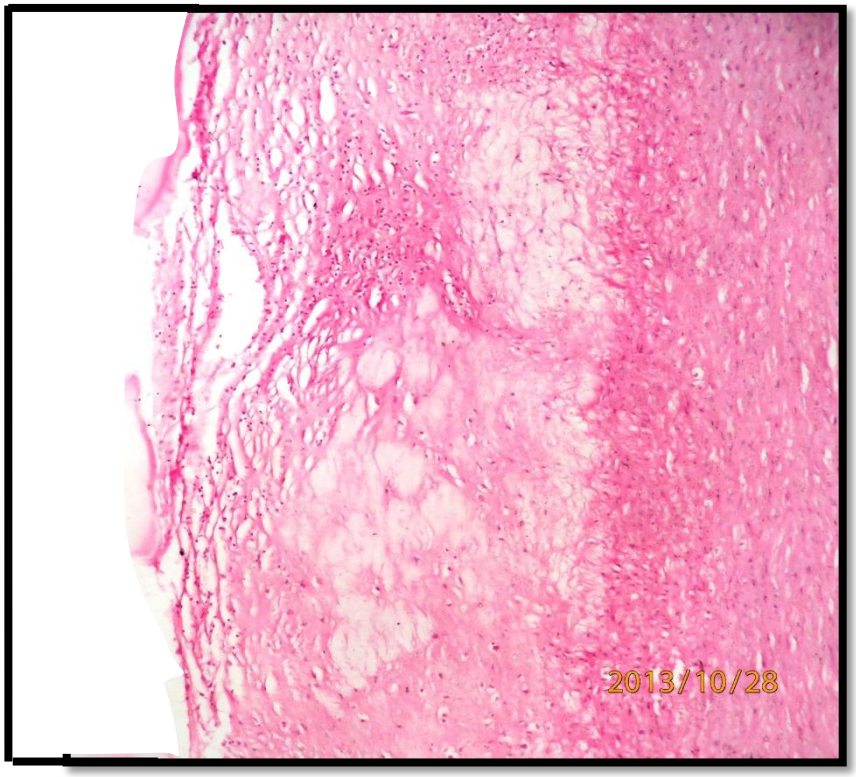


Figure12: Microscopic photograph of aorta showing pathological intimal thickening

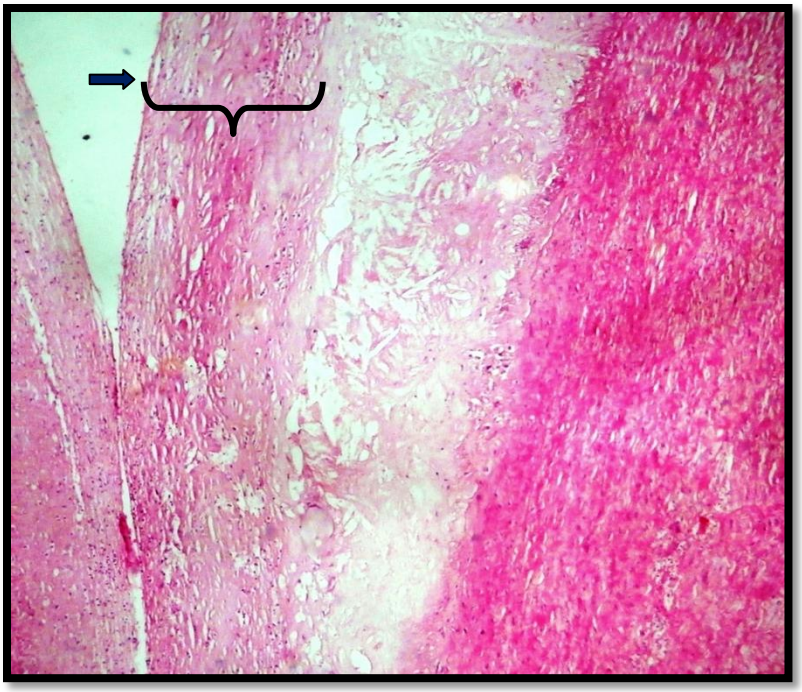


Figure 13: Microscopic photograph of aorta showing thick fibrous cap atheroma

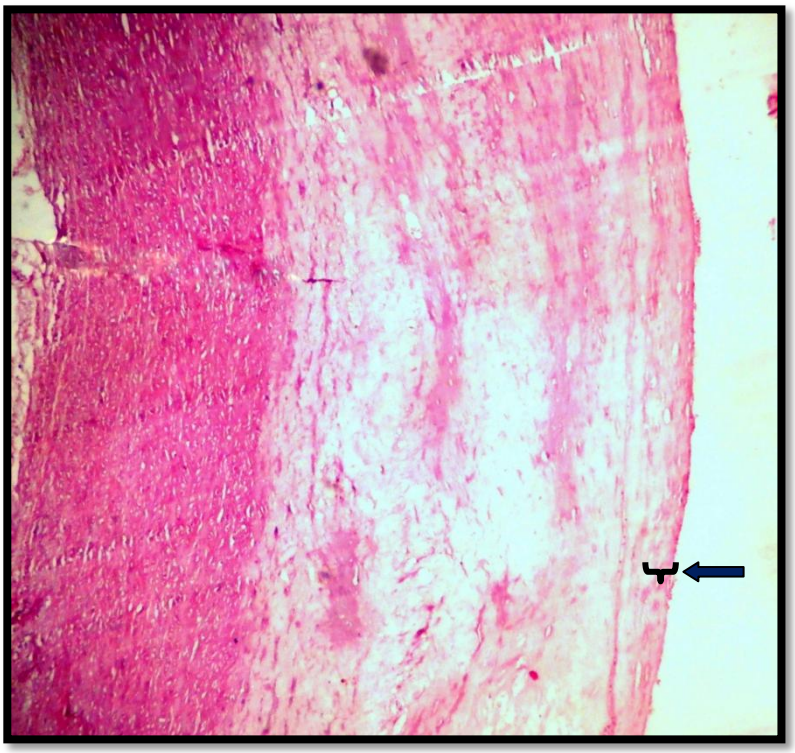


Figure 14: Microscopic photograph of coronary arteryshowing thin Fibrous cap atheroma

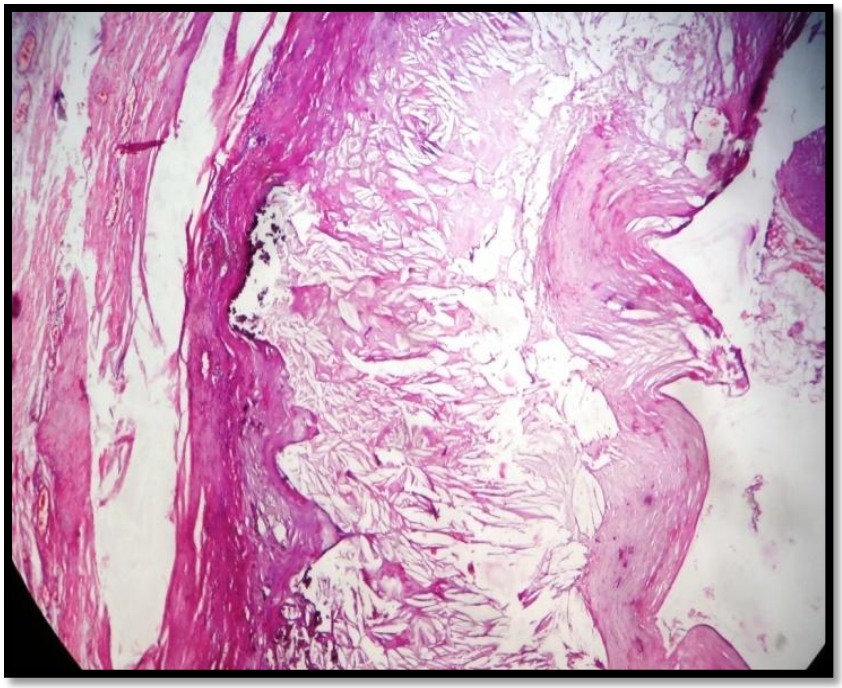


Figure 15: Microscopic photograph of aorta showing Fibrocalcific plaque

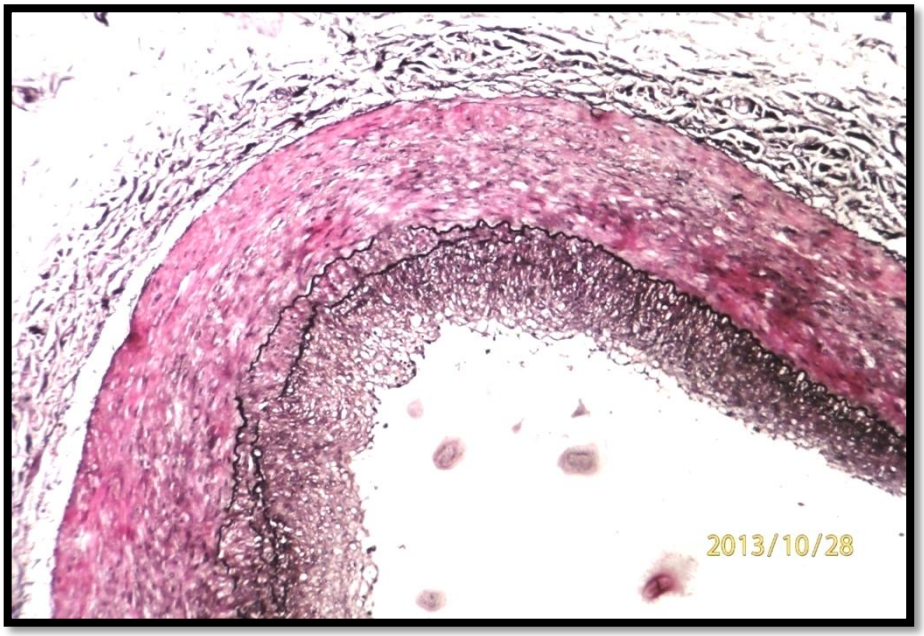


Figure 16: Microscopic photograph of coronary artery withVerhoeff van gieson stain

DISCUSSION

DISCUSSION

Atherosclerosis is a major cause of morbidity and mortality world-wide with outcomes being myocardial infarction, stroke and death.[50] Atherosclerosis is the generalized vascular disease.[11,12,51] It affects all the vascular beds including coronary, carotid, aorta and other peripheral vascular arteries and is present years before a cardiovascular event.[50] Although disease runs in parallel in various vessels.[4] Many epidemiological and autopsy studied had demonstrated the association between carotid and aortic atherosclerosis.[22] There is strong correlation between carotid and coronary atherosclerosis.[52] Renal artery atherosclerosis is usually present as one of the manifestation of generalized atherosclerosis.[10] Atherosclerosis begins early in childhood and transformed into another type of lesion occurring at the same anatomic site in an older age group.[8]

Age Based Distribution of Atherosclerosis:

In the present study, the age ranged from 7 to 72 years and mean of all age was 39.23±14.24 years. Majority of patients belonged to the 4^{th} decade of life (28.5%), followed by 3^{rd} & 5^{th} decade (19.8%). Since atherosclerosis is known to increase with ageing, the need of the hour is to assess the incidence of atherosclerosis in the young population who are being exposed to atherogenic influences like sedentary lifestyle and poor dietary habits.[5] In the present study 38 cases had age < 34 years [defined as young individuals in the Pathobiological Determinants of Atherosclerosis in Youth Study(PDAY)].[5]

In young age group, both carotid and renal arteries show 100% of NAL. Prati et al had also reported no carotid lesion in age less than 30 years.[54] In proximal coronary arteries, the mean of NAL was 29.33±3.21 and mean of PAL was 8.66±3.21 and in case of distal coronaries mean of NAL was 34.33±1.53 and mean of PAL was 3.66±1.53. So it had been seen that in younger age group proximal lesion had more of than the distal lesion which is same as elderly age group. According to Hochmann et al distal atherosclerosis in coronaries were more in younger patients.[48]

Table24: Comparison of other studies of coronary atherosclerosis in young

ATHEROSCLEROSIS IN YOUNG		
Study	**Coronary Artery Disease**	**Population**
Present	**9%**	**Indian**
ThejMJ	**16%**	**Indian**
Fausto N	**10%**	**Western**
Noeman A	**5%**	**Western**
Noeman A	**12-16%**	**South Asian**

In aorta of younger age group, the mean of NAL was 33.67±2.88 and PAL was 4.33±2.88. The PAL was least in thoracic aorta whereas both ascending and abdominal aorta had equal number of PAL. Thej MJ et al has reported 79% atherosclerotic lesion in ascending aorta, 84% in thoracic aorta and 90% in abdominal aorta in all age group and 88% of PAL in younger age group in aorta.[53] So here we saw that maximum PAL was found in proximal coronaries followed by aorta, distal coronaries, carotid and renal arteries in descending order.

The hypothesis proposed and pioneered by Dr David Barker stated that the deaths from ischemic heart were commoner in men who had been small at birth and at 1 year of age. This has been attributed to permanent metabolic and endocrine changes caused by impaired nutrition in fetal life. These findings of low birth weight in Indians can be a cause for the increased atherosclerosis at a young age. As atherosclerosis is multifactorial, such a deduction needs to be proved by future, larger studies.[54]

In first two decades of life, atherosclerotic lesions are expected to be foam cell lesions and fatty streaks involving lesion growth by LDL-C accumulation which makes up the types I-III lesions. Types I and II lesions, sometimes combined under the term early lesions (NAL in AHA 2000 classification), generally are the only ones that occur in infants and children, although they also occur in adults.[55] Type III lesions may evolve soon after puberty and, in their composition, form the bridge between early and advanced lesions. By the third decade, lesions may have progressed to atheroma, which makes up type IV lesion (also called PAL in AHA 2000 classification).[55]

In case of older age group (age > 34 years) in carotids mean NAL was 42±1.41 and mean PAL was 11±1.41. In case of proximal coronaries, mean of NAL was 29.33±12.5 and mean of PAL was 28±6.88 and in case of distal coronaries mean of NAL was 39.33±7.55 and mean of PAL was 14.±7.55. In case of aorta the mean of NAL was 30±6.24 and PAL was 22±7. In case of renal arteries mean NAL was 50.5±0.71 and mean PAL was 2.5±0.71.

In case of older age group proximal coronaries had more PAL than distal one, similar results was found by Hochmann et al.[48] Garg at el reported the significant atheroma in third decade (14.3%) onwards.[56]

In present study, carotids atherosclerosis in older age group was found in 22.6% and Kuroda et al reported 19.7% in same age group.[10] In case of renal atherosclerosis it was 5.6% in our study and Kuroda et al reported 10.4% in same artery and age group.[10]

One of the significant finding in our study in ascending aorta was that, 18(34%) cases had FCP in older age group.

Sex based distribution of Atherosclerosis:

Men have greater atheroma burden, more eccentric atheromas and more diffuse epicardial endothelial dysfunction than women.[57] This difference in atherosclerosis between females and males can be explained by the protective effect of oestrogen in females and the reduced amount of atherogenic risk factors in females compared to males.[58]

In present study the percent of atherosclerosis (PAL) in different arteries in male and female is as follow.

Table 25: Sex distribution of atherosclerosis in all arteries

Arteries	Female	Male
Carotids	4%	15%
Coronaries	28%	46%
Aorta	26%	31%
Renal	0%	4%
Average of all arteries	**14.5%**	**24%**

As has been seen in other studies conducted both on Western and Indian populations, the incidence of atherosclerosis was more in males compared to females.[57,58,59] Similar results were found in our study.

In present study carotid atherosclerosis was found in only 4 % of females compared to male it was 15%. In the study conducted by Prati et al carotid atherosclerosis in men and women was 25% and 27% respectively.[54] Erete et al had reported 40% in males and 33% in the females.[60] The wide difference between the present study and the studies done by Prati et al and Erete et al might be due to the fact that they had used the earlier AHA classification and considered even grade I and II lesions as atherosclerosis. In the present study since we have used the modified AHA classification of atherosclerosis, the earliest lesions like intimal thickening and intimal xanthoma were not considered as atherosclerotic lesions, partly explaining the difference in the incidence of atherosclerosis.

In case of coronary atherosclerosis males and females in different studies is as follow

Table 26: Sex distribution of coronary atherosclerosis in different study

Sex distribution of coronary atherosclerosis in different study		
Study	**Female**	**Male**
Present	**28%**	**46%**
Thej MJ	**48%**	**62%**
Singh H	**27%**	**68%**
Yazdi	**61.5%**	**73.1%**

In various studies done on different age groups, the frequency of atherosclerosis were reported to be between 16% to 75%.[57,58,59,61] The reason for this diversity can be the variability of race, culture (varied dietary habits, different economic status) and various other environmental factors.[59]

CAUSE OF DEATTH AND ATHEROSCLEROSIS:

In the current study, the most common cause of death was RTA (46.1%), followed by poisoning (23.1%), then Hanging and Burns (5.5%) each. Similar results were found by both Thej MJ et al and Singh H et al.[53,61] These two studies were done on the Indian population. No significant relation was found between cause of death and atherosclerosis may be because of less number of cases in each sub group.

Table 27: Cause of death distribution in different studies

Cause of death distribution in different studies			
	RTA	**Poisoning**	**Burns**
Present	**46.1%**	**23.1%**	**5.5%**
Thej MJ	**46%**	**37.1%**	**5.3%**
Singh H	**61.8%**	**18.8%**	**4.2%**

The cause of death seems to play a minor role in explaining the difference in degree of atherosclerosis except for in individuals who had committed suicide, since suicide is an indicator of depression and stress in the individual.[62] In humans, psychological stress, hopelessness and peoression can be risk factors for disastrous cardiovascular events such as stroke, coronary ischemia and myocardial infarction, which occur secondary to increased atherosclerosis.[62]

HISTORY OF SMOKING AND ALCOHOL WITH ATHEROSCLEROSIS:

In the present study history of both alcohol and smoking were present in 17(28%) cases, only history of smoking was present in 8(13 %) cases and only history of alcohol consumption was present in 10(17%) cases. Singh H et al had reported history of smoking in 32(16%) and alcohol consumption in 20(10%) cases.[61]

Table 28: Prevalence of smokers and alcoholics in different studies

Prevalence of smokers and alcoholics in different studies		
	Smokers	**Alcoholics**
Present	**13%**	**17%**
Siraj Ahmed	**48%**	**46%**
Singh H	**16%**	**10%**

In smokers the prevalence of PAL was found in 17(68%) cases and in alcoholics it was 18(69.2%) cases. In case of non-smoker and non-alcoholics prevalence of PAL was 13(40.5%) and 12(38.7%) cases respectively. According to Siraj ahmed et al prevalence of atherosclerosis was 19(79.2%) and 18(78.3%) cases for smokers and alcoholics respectively and in non-smoker and non-alcoholics it was 14(53.8%) and 15(55.6%) cases respectively.[43] It is well known fact that both smoking and alcohol are atherogenic and together both having synergetic effect; smoking causes endothelial injury and alcohol induce hypertension and also free radical mediated injury to vessel wall.[16,17]

Table 29: Distribution of atherosclerosis in smokers and alcoholics different studies:

Distribution of atherosclerosis in smokers and alcoholics different studies:		
	Present	**Siraj Ahmed**
Smokers	68%	73.2%
Non Smokers	40.6%	53.8%
Alcoholics	69.2%	78.3%
Non-alcoholics	38.7%	55.6%

BODY MASS INDEX AND ATHEROSCLEROSIS:

In present study, the prevalence of PAL in underweight, normal weight and overweight patients was 5(5.5%), 41(45%) and 17(18.7%) cases respectively. From the above study no correlation was found between atherosclerosis and BMI. Nazarat et al had also not found any association between atherosclerosis and BMI in the autopsy study done on Iranian population.[63] In the cross sectional study done by J Auer et al on 1100 patient also failed to detect any positive association between BMI and coronary artery disease detect by angiography.[64]

Many investigators emphasize the role of obesity in acute clinical events rather than atherosclerosis progression.[64] A problem that can obscure potential associations of coronary atherosclerosis with obesity may be that patients with more severe CAD could present lower BMI as a result of their long-standing disease.[64] The mechanism by which normal or low BMI patients, as compared to obese patients, may have relative excess of CAD is not clear but could be related to the presence of severe, non-cardiovascular, underlying diseases in very lean patients, as seen in many previous clinical trials.[64]

ATHEROSCLEROSIS IN DIFFERENT ARTERIES:

Atherosclerosis in carotids:

In present study, the mean of carotid atherosclerosis was 12.6%. Prati et al had reported the same as 26% for all age group.[54] Erete et al had found 20.8% of the carotid atherosclerosis in the study done in Nigeria.[60] Nezarat et al had reported 50% of carotid atherosclerosis in the study done in Iran.[63] The involvement of right carotid has slightly higher than the left in present study. Similar results was found by the Nazarat et al.[63] The reason behind variation in atherosclerotic lesion in right and left carotid is because of difference in flow dynamics and geometry of both arteries i.e left carotid directly arises from arch of aorta and right carotid arises from brachiocephalic artery which originate from arch of aorta which is more complex circulation in right carotid compare to left result in variation in atherosclerotic lesion.[65]

Atherosclerosis in coronaries:

In present study, the most common coronary artery involved by atherosclerosis was LAD in about 51% of cases followed by LCA and RCA 37% and 35% of cases. The degree of atherosclerosis in LAD was significantly more when compared to the RCA and LCA. This pattern correlates with results from other Western and Indian data.[3,61] Thej MJ et al reported the incidence of coronary atherosclerosis as 51%, 50% and 51% in LAD, RCA and LCA respectively.[53] Similarly Yazdi et al had found atherosclerotic prevalence as 60%, 50% and 42.5% in LAD, RCA and LCA respectively.[58] Golshahi et al had reported coronary atherosclerosis as 28.4% in population of Iran.[59] Many studies of the effects of flow-related variables have focused on the left coronary artery, which is geometrically

more complex than the RCA, because the left coronary artery branches into the circumflex and left anterior descending arteries.[35] This may be reason behind LAD and LCA had higher prevalence of atherosclerosis compared to RCA.

Atherosclerosis in Aorta:

In present study, atherosclerosis in aorta was 40% in ascending aorta, 20% in thoracic aorta and 30% in the abdominal aorta. Thej MJ et al had found 69%, 81% and 84% in ascending, thoracic and abdominal aorta respectively.[53] Siraj Ahmed et al had reported 80% of aortic atherosclerosis in Indian population.[43] Atherosclerosis is affected by multiple risk factors; the variation in physiological and anatomic characteristics of ascending, thoracic and abdominal aorta may be reason behind the different trend of atherosclerotic lesions in these areas.[35]

Atherosclerosis in Renal arteries:

In present study, the atherosclerosis in renal arteries was seen in only 3 % of cases. Kuroda et al had reported prevalence of renal atherosclerosis as 10.4%.[10] The higher prevalence is because of cases which were taken in the study group had age above 40 years in the Kuroda et al study.[10] The prevalence of renal artery stenosis reported by Aggarwal et al study was 13.8%.[66]

RELATION OF CAROTID ATHEROSCLEROSIS WITH OTHER ARTERIES:

Atherosclerosis has the distinctive topographical distribution in the intimal surface of each artery.[35] Hypothesis which can explain the difference in atherosclerotic lesion are (i) hemodynamic stresses related to arterial geometry; (ii) anatomic, cellular or biochemical variation in arterial wall, particularly in the endothelium cells, smooth muscle cells mutations leading to monoclonal proliferation and (iii) infectious agent.[35] In recent decades many studies had been done on carotid intima-media thickness by B mode of ultrasound to assess atherosclerosis non-invasively in carotid artery.[8,12,67,68] Many radiological studies done using ultrasound had told that carotid atherosclerosis is the indicator of generalized atherosclerosis.[8,12,51,67] and it can predict the future adverse cardiovascular and cerebrovascular events.[8,12] Both arterial beds share the risk factors that contribute to the progression of atherosclerosis in their vascular bed.[52]

Association between carotid and coronary atherosclerosis:

In the present, study mean of carotid atherosclerosis was 11.5±0.71 and mean of coronary atherosclerosis was 37.33±7.57. Nazarat et al had reported 50% and 85% of carotid and coronary atherosclerosis respectively.[63] Most Indian studies has shown steep rise in coronary atherosclerosis after second decade of life.[61] Advanced lesion in the coronary artery start appearing as early as in third decade.[69] In present study the none of the patient less than 34 year had carotid atherosclerosis. Prati et al. also reported none of the carotid atherosclerosis in less than 30 years.[54] Both the above studies had shown that carotid

atherosclerosis may start appearing after fourth decade compare to coronary which show steep rise after second decade only.

It was observed in our study that the coronary arteries were invariably involved showing atherosclerosis when carotid artery was affected and the percentage distribution of both arteries being involved when carotid artery showed atherosclerotic changes was 100%. When statistical analysis was done between carotid with LAD, RCA and LCA "P" value using chi square test and Fisher's Exact Test was 0.002, 0.003 and 0.050 respectively, all of three are statistically significant. By above data it had been shown that carotid atherosclerosis has close association between LAD followed by RCA and last LCA. Bari et al had reported 89.5% of carotid atherosclerosis had coronary atherosclerosis done on radiology study.[70] Tanimoto et al reported 29.8% of carotid involved by atherosclerosis had multivessels coronary artery disease on echographic study done on Japanese population.[71] Ogata et al had reported left main coronary artery atherosclerosis with common carotid atherosclerosis by USG.[52]

From above discussion it is clear that carotid atherosclerosis is closely related to coronary atherosclerosis. But when there is atherosclerosis in the coronary, it is not necessary that carotid will also be having atherosclerosis.

Association between carotid and aortic atherosclerosis:

The mean of aortic atherosclerosis was 27±9 which was higher compared to the carotid atherosclerosis observed in this study. Earliest age of aortic atherosclerosis was observed in 20 year where as earliest age of carotid atherosclerosis was observed at 40 years. Siraj Ahmed et al had observed earliest aortic atherosclerosis in the second decade.[43]

When carotid had atherosclerotic lesion 100% of patient had aortic involvement and most frequently associated with the ascending and abdominal aorta. Kallikazaros et al had found 74.3% of incidence of carotid plaque in the subgroup with aortic plaque.[9] and also found that absence of carotid plaque may not reflect the absence of aortic plaque.[9]

When statistical analysis was done between carotid and ascending, thoracic and abdominal aorta Fisher's Exact Test value for all the three were 0.001 which was statistically significant.

From above discussion it is clear that carotid atherosclerosis is closely related to aortic atherosclerosis but absence of carotid atherosclerosis may not reflect that aorta will not be involved.

Association between carotid and renal artery atherosclerosis:

The mean of renal artery atherosclerosis was 2.75±0.71 which was much lower compared to the carotid atherosclerosis observed in our study. Only 25% of cases of carotid atherosclerosis had renal atherosclerosis. The difference may be due to the prevalence of renal artery atherosclerosis was much lower than the carotid atherosclerosis. Kuroda et al had reported 22.8% patient with carotid atherosclerosis had renal atherosclerosis.[10] Aggarwal et al reported advanced lesion like fibrous plaque was rarely observed before fifth decade in renal arteries.[66]

When atherosclerosis presents in renal artery all (100%) the cases also had carotid atherosclerosis. It had been seen that 45%-100% of patient with renal atherosclerosis had comorbid carotid artery atherosclerosis.[10] Studies had shown that severe carotid

atherosclerosis associated with renal atherosclerosis and also the chronic kidney disease.[72]

From above discussion it is clear that carotid atherosclerosis is related to renal artery atherosclerosis but absence of renal atherosclerosis may not reflect that carotid will not be involved.

Association between proximal and distal coronary atherosclerosis with age:

In present study the atherosclerosis was most common in the proximal coronary when compared to the distal. In case of the LAD proximal part had 46(50.5%) atherosclerotic lesion compared to the distal part had 25(27.5%) atherosclerotic lesions. In case of RCA prevalence proximal and distal atherosclerosis was 32(35.2%) and 8(8.8%) respectively. In case of LCA prevalence proximal and distal atherosclerosis was 34(37.4%) and 20(22%) cases respectively.

In young age group the mean of atherosclerosis in the proximal coronary was 8.67±3.21 and mean of distal coronary was 3.67±1.53. In case of older age group mean of proximal and distal coronary atherosclerosis was 28.67±5.69 and 14±7.55 respectively. The difference between two was statistically significant. ($p < 0.05$)

From above data it was clear that prevalence of proximal coronary atherosclerosis was double the prevalence of distal part in both the young and older age group. Hochman et al had reported the prevalence of proximal coronary atherosclerosis was 83% and distal

was 32% which was consistent with our study.[46] Hochmann et al also found that atherosclerosis at the distal stenosis were more in younger age group compare to proximal stenosis.[46] The lesion in the proximal part of coronary was more frequent, more severe and more prone to rupture than the distal one.[14] Reason behind the proximal predominance of atherosclerotic involvement is because of exposed to high shear stress in the proximal part compared to distal part of coronaries.[14,15] The another reason was given that the origin of proximal part of coronary artery is distinct from the peripheral part i.e proximal part is developed from truncus arteriosus whereas distal portion is a the forerunner of the subepicardial branches of the coronary arteries and develops as a subepicardial vascular network.[15] Proximal part coronary arteries are the hot spot for the severe atherosclerosis.[14] Identification of these zones of high risk for acute coronary occlusions will lead to future advances in vulnerable plaque detection technology and potentially locally directed preventive strategies.[14] Proximal coronary artery disease(CAD) has a hereditary basis whereas distal CAD is more affected by environmental factors.[15]


Summary &
Conclusion

SUMMARY

1) This is a cross sectional study to compare the pattern of atherosclerosis in carotid with aorta coronary and renal arteries, undertaken in the Department of Pathology, Sri Devaraj Urs Medical College.

2) A total of 91 cases were studied.

3) Majority of patients belonged to the 4^{th} decade of life, followed by 3^{rd}& 5^{th} decades.

4) Number of males were more than females. The male: female ratio was 2.79.

5) Majority of them are died of RTA

6) Male had higher atherosclerosis than the female

7) Older age group had more atherosclerosis then the younger age group

8) Maximum number of atherosclerosis was found in coronary followed by aorta then carotid and last renal artery.

9) In coronary maximum atherosclerosis was found in LAD followed by LCA and last RCA.

10) Prevalence of proximal coronary atherosclerosis was double that of distal coronary both young and adult age group.

11) Ascending aorta had maximum atherosclerosis followed by abdominal aorta and last thoracic aorta.

12) Ascending aorta had maximum number of FCP, compare to any other artery.

13) Right carotid had more atherosclerosis then the left.

14) Right renal had slightly higher prevalence of atherosclerosis then left.

15) Carotid atherosclerosis was strongly associated with the coronary and aortic atherosclerosis then the renal atherosclerosis.

CONCLUSION

This study was done to compare the pattern of atherosclerosis in carotid with aorta coronary and renal arteries.

In last decade many radiological studies had shown that carotid atherosclerosis is the indicator of generalized atherosclerosis. Carotid is the superficial vessel and it is easy to detect the atherosclerosis in carotid using noninvasive technique compare to other vessels. Many studies have shown that carotid atherosclerosis can predict future stroke and cardiovascular events. Carotids atherosclerosis is also associated with the chronic kidney disease. There are various methods to assess the carotid atherosclerosis using ultrasound. Accuracy of all of these methods is still a debate.

Autopsy is the gold standard test to assess the atherosclerosis. Till now none of the study both autopsy or radiological had compared the carotid atherosclerosis with coronary, aortic and renal atherosclerosis.

In present study we conclude that if carotid atherosclerosis is present patient will be having coronary, aortic atherosclerosis but patient might have renal atherosclerosis. If carotid atherosclerosis is absent it doesn't mean that patient won't be having atherosclerosis in any other vessels.

Assessing the carotid atherosclerosis using noninvasive technique can be used as screening tool for cardiovascular, cereberovascular and kidney disease due to renal artery atherosclerosis. This is an indication that anti-atherogenic preventive measures need to be implemented as soon as carotid atherosclerosis detected. This is to prevent premature death caused by cardiovascular, cereberovascular and kidney disease, thus burdening the national economy.

Many earlier disease studies had found that proximal coronary atherosclerosis had hereditary basis and distal coronary had environmental basis. In present study we found that prevalence of proximal coronary atherosclerosis was double that of the distal part of coronaries. Same pattern of coronary atherosclerosis was found in both younger and older age group. From above it has shown that atherosclerosis may have the hereditary factor but other factors also affect it. So need further molecular studies to prove this. Proximal part coronary arteries are the hot spot for the severe atherosclerosis. Identification of these zones of high risk for acute coronary occlusions will lead to future advances in vulnerable plaque detection technology and potentially locally directed preventive strategies.

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ANNEXURE- 1

DEPARTMENT OF PATHOLOGY

CONSENT FORM

Date:

Autopsy No:

Name of Deceased:

I hereby consent to allow the Department of Pathology, SDUMC to take the following organs for Histomorphological diagnosis in relation to the Cause of Death.

Organs – 1) Heart

2) Aorta along with both carotids and renal arteries

Name:

Signature:

Relation:

ANNEXURE- 2
PROFORMA

Name :
Age:
Sex:
IP no:
Cause of death:

Date of death:
Autopsy No:
Height:
Weight:
BMI:

Gross Examination:

	LAD		RCA		LCA	
	Proximal	**distal**	**proximal**	**distal**	**proximal**	**distal**
1] Presence of thrombus						
2] Gross atheroma seen						
3] Number of bits taken						

	AA	TA	Ab A
1] Gross Atheroma			
2] Calcification			
3] Number of bits taken			

Carotid	Right	Left
1] Gross Atheroma		
2] Calcification		
3] Number of bits taken		

Renal	Right	Left
1] Gross Atheroma		
3] Calcification		
4] Number of bits taken		

Microscopy:
Coronary arteries:

Carotid arteries:

Aorta:

Renal arteries:

Any other Observation:

Signature of Guide

Signature of candidate

ANNXURE - 3

KEY TO MASTER CHART

M - Male

F - Female

Y – Years

C O D – Cause of death

S – Smoking

A – Alcohol

NK – Not known

P.LAD – Proximal Left anterior descending

P.RCA - Proximal Right coronary artery

P.LCA –Proximal Left circumflex coronary artery

D.LAD – Distal Left anterior descending

D.RCA - Distal Right coronary artery

D.LCA – Distal Left circumflex coronary artery

AA – Ascending aorta

TA - Thoracic aorta

Ab.A- Abdominal aorta

RCa – Right carotid

LCa- Left Carotid

RR – Right renal

LR – Left renal

NVL- No visible lesion

FS - Fatty streak

FP - Fibrous plaque

CL - Complicated lesion

IT – Intimal thickening

IX – Intimal xanthoma

PIT – Pathological intimal thickening

FCA – Thick fibrous cap atheroma

T FCA – Thin fibrous cap atheroma

RTA – Road traffic accident

GENERAL							GROSS FINDINGS										MICROSCOPIC FINDINGS											
S.No	Autopsy No	AGE	SEX	C O D	S or A	BMI	LAD	RCA	LCA	AA	TA	AbA	RCa	LCa	LR	RR	P.LAD	P.RCA	P.LCA	D.LAD	D.RCA	D.LCA	AA	TA	AbA	RCa	LCa	LR
1	04\12	26	F	Burning	N K	21.2	FS	FS	FS	FS	FS	FS	FS	FS	NVL	NVL	IX	IX	IX	IX	IT	IX	IT	IT	IT	IT	IT	IT
2	05\12	56	M	Poisioning	N K	23.8	FP	FS	FP	CL	FP	FP	FP	FP	NVL	NVL	PIT	IX	PIT	PIT	IX	IX	FCA	PIT	PIT	PIT	PIT	IT
3	06\12	38	M	RTA	N K	28.3	FP	FP	FS	FS	FS	FS	FS	FS	FS	NVL	PIT	PIT	IX	IT	IX	IT	IX	IX	IX	IX	IT	IT
4	08\12	28	M	Head injury	N K	23.1	FS	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	IX	IX	IX	IT	IX	IX	IX	IX	IX	IT	IT	IX
5	10\12	35	M	Poisioning	N K	22.8	FS	FS	FS	FS	FS	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IT	IX	IX	IX	IX	IT	IT	IT
6	11\12	20	M	Head injury	N K	22.5	FP	FS	FP	FS	NVL	NVL	FS	FS	NVL	NVL	PIT	IX	PIT	IX	IT	IT	IX	IT	IT	IX	IX	IT
7	12\12	37	F	RTA	N K	22.7	FS	NVL	FS	FS	FS	FS	NVL	NVL	NVL	NVL	IX	IT	IX	IX	IT	IX	IT	IX	IX	IT	IT	IT
8	13\12	50	M	Poisioning	N K	25.4	FS	FS	FS	NVL	FS	FS	NVL	NVL	NVL	NVL	IX	IX	IX	IX	IX	IT	IT	IX	IX	IX	IT	IT
9	14\12	42	F	Snake Bite	N K	23.8	FS	FS	FS	FS	NVL	NVL	NVL	NVL	NVL	NVL	IX	IX	IX	IT	IX	IX	IT	IX	IT	IT	IT	IT
10	15\12	60	M	RTA	N K	27.6	FP	FS	FS	FP	FP	FP	FP	FP	FS	FS	PIT	IX	IX	IX	IT	IT	PIT	IX	PIT	PIT	IT	IT
11	19\12	50	M	RTA	A	20.5	FS	FS	FS	FP	FS	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IX	IX	IX	IX	IX	IX	IT	IT
12	20\12	45	M	RTA	S+A	21.7	FP	FP	FP	CL	FS	FS	FS	FS	FS	FS	PIT	PIT	PIT	PIT	PIT	PIT	FCP	IX	IX	IT	PIT	IX
13	23\12	24	M	Poisioning	NO	23.9	FS	FS	FS	FS	FS	FS	NVL	FS	NVL	NVL	IX	IX	IX	IX	IT	IX	IX	IX	IX	IT	IX	IT
14	25\12	45	M	RTA	S+A	20.2	FP	FP	FP	FP	FP	FP	FS	FS	NVL	NVL	PIT	PIT	PIT	IX	IT	IT	PIT	PIT	PIT	IX	IX	IT
15	27\12	30	M	RTA	S+A	25	FS	FS	FS	FS	NVL	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IX	IX	IX	IX	IX	IX	IT	IT
16	33\12	18	F	Burning	NO	21.6	FS	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IX	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT
17	42\12	28	F	Snake Bite	NO	18.4	FP	FP	FP	NVL	FS	FS	NVL	NVL	NVL	NVL	PIT	PIT	PIT	IX	IX	PIT	IT	IX	IX	IX	IT	IT
18	43\12	32	M	RTA	NO	23.8	FS	FS	FS	FP	NVL	NVL	FS	FS	NVL	NVL	IX	IT	IX	IX	IX	IT	PIT	IT	IT	IX	IX	IT
19	44\12	26	M	RTA	S	29.3	FS	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	IX	IX	IT	IX	IT	IT	IT	IT	IT	IT	IT	IT
20	45\12	33	F	RTA	N0	20.4	FP	FS	FS	FP	FS	FP	NVL	NVL	NVL	NVL	PIT	IX	IX	IX	IT	IT	PIT	IX	PIT	IT	IT	IT
21	46\12	46	M	RTA	S+A	32.8	FP	FP	FP	FS	FS	FS	NVL	NVL	NVL	NVL	FCA	FCA	PIT	IX	IX	IX	IX	IX	IX	IX	IT	IT
22	54\12	15	M	Fall from height	NO	16.6	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IT	IT	IT	IT	IT	IX	IT	IT	IT	IT	IT	IT
23	95\12	40	M	Drowning	N K	23.8	CL	CL	CL	CL	CL	CL	CL	CL	CL	CL	FCA	FCA	FCA	PIT	PIT	PIT	FCP	FCP	FCP	FCP	FCA	FCA
24	96\12	23	M	RTA	S	18.5	NVL	NVL	FS	NVL	FS	FS	FS	NVL	NVL	NVL	IT	IT	IT	IX	IT	IX	IT	IX	IX	IX	IT	IT
25	97\12	19	F	Poisioning	NO	17.2	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT
26	98\12	40	F	RTA	N K	18	FS	FP	FS	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IX	PIT	IX	IX	IX	IX	IT	IT	IT	IT	IT	IT
27	110\12	55	M	Head injury	S+A	25.7	FP	FP	FP	FS	FP	FP	FS	FS	FS	FS	FCA	PIT	FCA	IX	IX	IX	IX	PIT	PIT	IT	IT	IT
28	111\12	70	M	Fall from height	S+A	21.3	FP	FS	FS	CL	FS	FP	FP	FP	FS	FS	PIT	IX	IX	PIT	IX	IT	FCP	IX	PIT	PIT	PIT	IT
29	112\12	35	M	RTA	N K	21.7	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	NVL	IX	IX	IX	IX	IX	IX	IX	IX	IT	IT	IT	IT
30	113\12	50	M	Poisioning	S	28.4	FP	FS	FP	NVL	FS	FS	FS	FS	NVL	NVL	PIT	IX	PIT	FCA	PIT	PIT	IT	IX	IX	IX	IX	IT
31	114\12	66	F	Burning	NO	22.2	FP	FP	FP	FP	FP	FP	FP	FP	FS	FS	IX	PIT	PIT	PIT	IX	PIT	FCA	FCA	FCA	PIT	PIT	IX

GENERAL							GROSS FINDINGS										MICROSCOPIC FINDINGS										
S.No	Autopsy No	AGE	SEX	C O D	S or A	BMI	LAD	RCA	LCA	AA	TA	AbA	RCa	LCa	LR	RR	P.LAD	P.RCA	P.LCA	D.LAD	D.RCA	D.LCA	AA	TA	AbA	RCa	LCa	LR
32	115\12	70	M	Poisioning	S+A	26.6	CL	CL	CL	FP	CL	CL	FP	FP	FS	FS	FCA	FCA	FCA	PIT	PIT	FCP	PIT	FCA	FCA	PIT	PIT	IX
33	123\12	33	M	Police custody	N K	24.1	FS	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	IX	IX	IX	IX	IX	IX	IX	IX	IX	IT	IT	IT
34	124\12	20	M	Poisioning	NO	21.5	FP	FP	FP	NVL	FP	FS	NVL	NVL	NVL	NVL	PIT	PIT	PIT	IX	IX	IX	IT	PIT	IX	IT	IT	IT
35	125\12	35	M	RTA	A	29	FP	FP	FP	CL	FP	FP	FP	FS	NVL	NVL	PIT	PIT	PIT	PIT	IX	PIT	FCP	PIT	PIT	PIT	IX	IT
36	127\12	48	M	Poisioning	S	23.9	FS	FS	FP	CL	FS	FP	FS	FS	FS	FS	IX	IX	PIT	IX	IX	IX	FCP	IX	FCA	IX	IX	IX
37	128\12	32	F	Poisioning	A	18	FP	FS	FP	FS	FS	FS	FS	FS	FS	NVL	PIT	IX	PIT	PIT	IX	PIT	IX	IX	IX	IX	IT	IX
38	129\12	53	M	RTA	S+A	29.1	FP	FS	FS	FP	FS	FS	FS	FS	FS	FP	PIT	IX	IX	IX	IX	IX	PIT	IX	IX	IX	IX	IX
39	147\12	32	M	RTA	A	21.5	FP	FP	FP	FP	FS	FP	FS	FS	NVL	NVL	PIT	PIT	PIT	IX	IX	PIT	PIT	IX	PIT	IX	IX	IT
40	1\13	40	F	Poisioning	NO	22.8	FP	FS	FS	FP	FS	FP	NVL	NVL	NVL	NVL	PIT	IX	IX	IX	IX	IX	PIT	IX	PIT	IT	IT	IT
41	2\13	45	M	Poisioning	N K	21.7	FP	FP	FP	CL	FS	FS	FS	FS	FS	FS	PIT	PIT	PIT	PIT	PIT	PIT	FCP	IX	IX	IT	PIT	IX
42	3\13	7	M	RTA	NO	16.5	FS	NVL	FS	NVL	NVL	NVL	NVL	NVL	FS	FS	IX	IT	IX	IT	IT	IT	IT	IT	IT	IT	IT	IX
43	4\13	20	F	Poisioning	N K	18.8	FS	FS	FS	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IX	IX	IX	IT	IX	IX	IT	IT	IT	IT	IT	IT
44	5\13	32	M	Poisioning	S+A	29.4	FP	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	PIT	IX	IX	PIT	IX	IX	IX	IX	IX	IT	IT	IT
45	6\13	55	M	RTA	S+A	23.1	FP	FP	FP	FP	FP	CL	FP	FS	NVL	NVL	PIT	PIT	PIT	IX	IX	IT	PIT	PIT	FCP	PIT	IX	IT
46	7\13	23	M	RTA	NO	20.7	FS	FS	FP	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IX	IX	PIT	IX	IX	IX	IT	IT	IT	IT	IT	IT
47	17\13	40	M	RTA	N K	26.3	FP	FS	FS	FS	FS	FS	FS	NVL	NVL	NVL	PIT	IX	IX	IX	IT	PIT	IX	IX	IX	IX	IT	IT
48	18\13	32	M	Bee sting	S+A	32.7	FS	NVL	FS	FS	FS	NVL	NVL	FS	NVL	NVL	IX	IT	IX	IX	IT	IT	IX	IX	IT	IT	IX	IT
49	19\13	33	F	RTA	NO	17.6	FS	FS	FS	NVL	FS	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IX	IT	IT	IX	IX	IX	IT	IT
50	20\13	48	F	RTA	NO	23.6	FS	FS	FS	CL	FS	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IX	IX	FCP	IX	IX	IX	IT	IT
51	21\13	60	F	Burning	N K	23.1	FP	FP	FP	CL	CL	FP	FS	FS	NVL	NVL	FCA	PIT	PIT	PIT	IX	PIT	FCP	FCP	T FCA	IX	IX	IT
52	22\13	22	M	RTA	NO	21.5	FS	FS	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IX	IX	IT	IX	IX	IT	IT	IT	IT	IT	IT	IT
53	23\13	43	M	RTA	S+A	21.2	FP	FP	FP	FS	FS	FS	FS	NVL	FS	NVL	PIT	PIT	PIT	IX	IX	PIT	IX	IX	IX	IX	IT	IX
54	24\13	26	M	RTA	S+A	23.3	FS	FS	FS	NVL	NVL	NVL	NVL	FS	FS	NVL	IX	IX	PIT	IT	IT	IT	IT	IT	IT	IT	IX	IX
55	25\13	32	M	RTA	S+A	24.1	CL	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	FCP	IX	IX	FCP	IX	IX	IX	IX	IX	IT	IT	IT
56	26\13	72	M	Natural death	N K	18	FP	FP	FP	CL	CL	CL	FS	FS	NVL	NVL	PIT	PIT	PIT	T FCA	IX	PIT	FCP	FCP	FCP	IX	IX	IT
57	27\13	20	F	preg CLlication	NO	18.7	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT	IT
58	28\13	30	M	RTA	S+A	20.1	FP	FP	FP	FP	FP	FP	NVL	FS	FS	FS	PIT	PIT	FCA	IX	PIT	PIT	PIT	IX	PIT	IT	IX	IX
59	29\13	26	F	Poisioning	N K	18.7	FS	FS	NVL	NVL	FS	FS	NVL	NVL	NVL	FS	IX	IX	IT	IT	IX	IT	IT	IX	PIT	IT	IT	IT
60	30\13	35	M	Poisioning	N K	20.5	FS	NVL	FS	NVL	FS	FS	FS	FS	NVL	NVL	IX	IT	IX	PIT	IX	IX	IT	IX	IX	IX	IX	IT
61	31\13	55	M	Hanging	N K	17.4	FP	FP	FP	CL	FP	FP	FS	FS	NVL	NVL	PIT	PIT	PIT	T FCA	IX	IX	FCP	PIT	PIT	IX	IX	IT
62	32\13	60	F	Poisioning	NO	19.2	FP	FS	FS	CL	FS	FS	NVL	FS	FS	NVL	PIT	IX	IX	IX	IX	IX	FCP	IX	IX	IT	IX	IX

GENERAL							GROSS FINDINGS										MICROSCOPIC FINDINGS											
S.No	Autopsy No	AGE	SEX	C O D	S or A	BMI	LAD	RCA	LCA	AA	TA	AbA	RCa	LCa	LR	RR	P.LAD	P.RCA	P.LCA	D.LAD	D.RCA	D.LCA	AA	TA	AbA	RCa	LCa	LR
63	33\13	50	M	RTA	N K	22.2	FP	FP	FP	FP	FS	FS	FS	FS	NVL	NVL	PIT	PIT	PIT	PIT	IX	IX	PIT	IX	IX	IX	IX	IT
64	34\13	40	M	RTA	NO	19.5	FP	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	PIT	IX	IX	IX	IX	IX	IX	IX	IX	IT	IT	IT
65	35\13	28	M	Hanging	NO	24.6	FS	FS	NVL	FS	FS	NVL	FS	FS	NVL	NVL	IX	IX	IT	IT	IX	IX	IT	IX	IT	IX	IX	IT
66	36\13	65	M	RTA	S+A	24.6	FP	FP	FS	CL	FP	CL	FP	FP	FS	FS	PIT	PIT	IX	PIT	IX	IX	FCP	PIT	FCA	PIT	PIT	IX
67	37\13	62	M	Assault	S+A	31.3	CL	FP	CL	CL	FP	CL	FP	FP	FP	FP	FCP	FCA	FCP	PIT	T FCA	FCP	FCP	FCA	FCA	T FCA	PIT	PIT
68	38\13	34	M	RTA	N K	21.3	FS	FS	FS	FP	FS	FS	NVL	NVL	NVL	NVL	IX	IX	IX	IX	IT	IT	FCA	IX	IX	IT	IT	IT
69	39\13	44	F	RTA	NO	20.7	FS	FP	FS	FS	NVL	FS	NVL	NVL	NVL	NVL	IX	PIT	IX	IX	IX	IX	IX	IT	IX	IT	IX	IT
70	40\13	50	M	RTA	A	27	FS	FS	FS	FS	NVL	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IX	IT	IX	IT	IX	IX	IT	IT
71	65\13	40	F	Poisioning	N K	23.7	FS	FP	FS	FS	NVL	FS	NVL	NVL	NVL	NVL	IX	PIT	IX	IX	IX	IX	IX	IT	IX	IT	IX	IT
72	66\13	60	M	RTA	S+A	23.4	FP	FP	FS	CL	FP	CL	FP	FP	FS	FS	PIT	PIT	IX	PIT	IX	IX	FCP	PIT	FCA	PIT	PIT	IX
73	67\13	47	M	RTA	N K	25.4	FP	FS	FS	FS	FS	FS	FS	NVL	NVL	NVL	PIT	IX	IX	IX	IT	PIT	IX	IX	IX	IX	IT	IT
74	68\13	53	M	Snake Bite	NO	20.7	FP	FP	FP	FP	FS	FS	FS	FS	NVL	NVL	PIT	PIT	PIT	PIT	IX	IX	PIT	IX	IX	IX	IX	IT
75	69\13	38	F	Burning	NO	18.7	FP	FS	FS	CL	FS	FS	NVL	FS	FS	NVL	PIT	IX	IX	IX	IX	IX	FCP	IX	IX	IT	IX	IX
76	70\13	43	M	RTA	S	22.9	FP	FP	FP	FS	FS	FS	FS	NVL	FS	NVL	PIT	PIT	PIT	IX	IX	PIT	IX	IX	IX	IX	IT	IX
77	71\13	17	M	RTA	N K	24.5	FS	FS	NVL	NVL	NVL	NVL	NVL	NVL	NVL	NVL	IX	IX	IT	IX	IX	IT	IT	IT	IT	IT	IT	IT
78	72\13	65	M	Drowning	N K	25.4	FP	FP	FP	CL	FP	FP	FS	FS	NVL	NVL	PIT	PIT	PIT	T FCA	IX	IX	FCP	PIT	PIT	IX	IX	IT
79	73\13	39	F	Poisioning	NO	21.8	FS	FS	FS	CL	FS	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IX	IX	FCP	IX	IX	IX	IT	IT
80	74\13	51	M	Fall from height	A	28.7	FS	FS	FS	FS	NVL	FS	FS	NVL	NVL	NVL	IX	IX	IX	IX	IX	IT	IX	IT	IX	IX	IT	IT
81	75\13	29	M	RTA	NO	20.8	FP	FP	FP	FP	FP	FP	NVL	FS	FS	FS	PIT	PIT	FCA	IX	PIT	PIT	PIT	IX	PIT	IT	IX	IX
82	76\13	38	M	Hanging	N K	28.1	FP	FP	FP	FP	FP	CL	FP	FS	NVL	NVL	PIT	PIT	PIT	IX	IX	IT	PIT	PIT	FCP	PIT	IX	IT
83	77\13	42	M	RTA	NO	25	FP	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	PIT	IX	IX	IX	IX	IX	IX	IX	IX	IT	IT	IT
84	78\13	30	M	RTA	NO	24.2	FS	FS	NVL	FS	FS	NVL	FS	FS	NVL	NVL	IX	IX	IT	IT	IX	IX	IT	IX	IT	IX	IX	IT
85	79\13	32	M	Hanging	N K	24.8	FP	FS	FS	FS	FS	FS	NVL	NVL	NVL	NVL	PIT	IX	IX	PIT	IX	IX	IX	IX	IX	IT	IT	IT
86	80\13	61	F	Natural death	NO	28.1	FP	FP	FP	CL	CL	FP	FS	FS	NVL	NVL	FCA	PIT	PIT	PIT	IX	PIT	FCP	FCP	T FCA	IX	IX	IT
87	81\13	33	M	Assault	A	32.7	FS	NVL	FS	FS	FS	NVL	NVL	FS	NVL	NVL	IX	IT	IX	IX	IT	IT	IX	IX	IT	IT	IX	IT
88	82\13	26	F	Poisioning	N K	23.3	FS	FS	NVL	NVL	FS	FS	NVL	NVL	NVL	FS	IX	IX	IT	IT	IX	IT	IT	IX	PIT	IT	IT	IT
89	83\13`	37	M	RTA	A	25.3	FS	NVL	FS	NVL	FS	FS	FS	FS	NVL	NVL	IX	IT	IX	PIT	IX	IX	IT	IX	IX	IX	IX	IT
90	84\13	25	M	Poisioning	S	20.9	FS	FS	FS	NVL	NVL	NVL	NVL	FS	FS	NVL	IX	IX	PIT	IT	IT	IT	IT	IT	IT	IT	IX	IX
91	85\13	36	M	Hanging	S	24.4	FS	FS	FS	FP	FS	FS	NVL	NVL	NVL	NVL	IX	IX	IX	IX	IT	IT	FCA	IX	IX	IT	IT	IT

RR
IT
IT
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IT
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