STUDY OF EFFECTS OF ACUTE MYOCARDIAL INFARCTION ON CHOLESTEROL AND CHOLESTEROL RATIOS

DISSERTATION SUBMITTED FOR M.D.DEGREE IN GENERAL MEDICINE BRANCH I

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CERTIFICATE

This is to certify that this dissertation entitled " ***EFFECT OF ACUTE MYOCARDIAL INFARCTION ON CHOLESTEROL AND CHOLESTEROL RATIOS"*** is a bonafide record of work done by **Dr.P.MARCHWIN KINGSTON SAMUEL** under my guidance and supervision in Tirunelveli Medical College Hospital during the period of his post graduate study for M.D(General Medicine) from 2003 – 2006.

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STUDY OF EFFECTS OF ACUTE MYOCARDIAL INFARCTION ON CHOLESTEROL AND CHOLESTEROL RATIOS

INTRODUCTION

Coronary artery disease remains the most common cause of death despite significant advancement in its prevention and treatment [4].

Myocardial infarction generally occurs with the abrupt decrease in coronary blood flow that follows a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis. The injury is facilitated by factors such as cigarette smoking, hypertension, dyslipidemia, diabetes and a number of other factors [70].

According to Davidson MA et-al, the risk of coronary artery disease in Indians is 3-4 times higher than white Americans and 20 times higher than Japanese.

Aggressive management of the risk factors is one of the crucial elements in the treatment of patients with coronary artery disease. Serum markers that are used for cholesterol risk assessment and management are total cholesterol, low density lipoprotein (LDL) cholesterol level and high density lipoprotein (HDL) cholesterol level.

Patients with acute myocardial infarctions should have plasma lipid levels determined within 24 hours of the onset of symptoms of acute infarction[6].

The studies like Mulligan IP etal (1984), Jacobson TA etal (1996), Scrass R Marshall R etal (1987) and many other studies have questioned the validity of the plasma lipid levels measures beyond 24 hours from the onset of myocardial infarction[5].

The studies have demonstrated that acute myocardial infarction results in a transient decline in the serum cholesterol levels, which becomes apparent after 24 hours of onset of myocardial infarction and may last for 2 to 3 months[11]. Therefore in situations in which plasma lipid levels are not determined within 24 hours of the onset of myocardial infarction symptoms, the cholesterol measurements are usually deferred until the effect of acute infarction is fully resolved which may result in an inappropriate delay in the management of hyper cholesterolemia based on Brugada R, Wenger, NK (Cardiology 1996 : 87: 194-199)

The ratios of total cholesterol to HDL cholesterol and of LDL cholesterol to HDL cholesterol also can be used as predictors of acute coronary events (Based on Castelli WP, AmJ Med 1984:27:4-12, Linn S.Fulwood, Carroll M et al, Am J 1991:1038 – 1043)

The purpose of the present study is to determine whether the acute myocardial infarction affects the values of the serum cholesterol ratios as it does with absolute serum cholesterol Levels.

AIM OF THE STUDY

1. To study the effect of acute myocardial infarction on absolute cholesterol and triglyceride levels.
2. To study the effect of acute myocardial infarction on the ratios of total cholesterol to HDL (High Density Lipoprotein) cholesterol and of LDL (Low Density Lipoprotein) cholesterol to HDL (High Density Lipoprotein) cholesterol.
3. To determine whether the acute myocardial infarction affects the values of the serum cholesterol ratios as it does with absolute cholesterol level.
4. To stress dyslipidemia as a major risk factor for coronary artery disease.
5. To evaluate more about primordial prevention of dyslipidemia.

REVIEW OF LITERATURE

The two coronary arteries that supply the myocardium arise from the sinuses behind two of the cusps of the aortic valve at the root of the aorta. The right coronary artery has a greater flow in 50% of individuals and the left has a greater flow in 20% and the flow is equal in 30%.

The left anterior descending Coronary artery (40to50%)	-	Anterior wall of left ventricle, anterior 2/3 of inter ventricular Septum.
Left circumflex coronary artery- (15to20%)		Lateral wall of left ventricle.
Right coronary artery (30% to 40%)	-	Infero posterior wall of left ventricle, posterior 1/3 of interventricular septum, Right atrium and right Ventricle.

MYOCARDIAL INFARCTION DUE TO CORONARY ATHEROSCLEROSIS

Myocardial infarction generally occurs with the abrupt decrease in coronary blood flow that follows a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis.

CORONARY ATHEROSCLEROSIS

The earliest lesions of atherosclerosis can be found in young children and infants in the form of fatty streak whereas the advanced lesion, the fibrous plaques generally appears during early adulthood and progress with age[45].

The abdominal aorta is involved earliest. The aorta is usually most heavily involved at or near the orifice of its branches. (Particularly at the level of coronary arteries).

In coronary arteries, raised lesions are most prominent in the main stems, the highest incidence being a short distance beyond the ostia. Atherosclerosis is usually found in the epicardial portions of the vessels, while the intra mural coronary arteries are spared, Coronary atherosclerosis is often diffuse. The degree to which the lumen is narrowed varies, but once the process has commenced, all the intima of the extra mural portions of the vessels is usually involved. Typical atheromatous fibrous plaques also develop in saphenous vein, after grafting which is used for aortocoronary by pass graft.

HYPOTHESIS OF ATHEROSCLEROSIS

❖ The response to injury hypothesis

The endothelial lining cells are exposed to repeated or continuous insults. Dysfunctional endothelial cells at the susceptible sites in the arterial tree would lead to exposure of the subendothelial tissue to increased concentrations of plasma constituents[77]. This triggers a sequence of events including monocytes and platelet adherence, migrations, platelet aggregation and formation of microthombi and release of secretary products. This causes proliferation of smooth muscle cells at these sites of injury. Monocytes become transformed to foam cells. Thus a well developed plaque is formed.

❖ Monoclonal hypothesis

This states that the intimal proliferative lesion results from the Multiplication of single, individual smooth muscle cells, as do benign tumors[78]. According to this hypothesis, the intimal smooth muscle cell that proliferate to form an atheroma are normally under feed back control by mitosis inhibitors formed by the smooth muscle cells in the continuous media and this feed back control system tends to fail with age as these controlling cells die and are not adequately replaced.

RISK FACTORS

A number of conditions and habits are present more frequently in individuals who develop atherosclerosis than in general population; these factors have been termed risk factors.

RISK FACTORS FOR ACUTE MYOCARDIAL INFARCTION [62, 71]

CATEGORY I

Non modifiable risk factors

- Age
- Male gender
- Family history of early onset coronary artery disease
- Low socioeconomic status

Modifiable risk factors

- ↑ Low density lipoprotein
- ↓ High density lipoprotein
- Atherogenic diet
- Cigarette smoking
- Hypertension
- Left ventricular hypertrophy
- Thrombogenic factors

CATEGORY II

- Diabetes mellitus
- Physical iactivity
- Elevated triglycerides
- Obesity

CATEGORY III

- Psychosocial factors
- Lipoprotein (a)
- Homocysteine
- Inflammatory markers – especially C-reactive protein
- No alcohol comsumption
- Oxidative stress
- Post menopausal status – because of reduction in plasma oestrogen levels.

DYSLIPIDEMIA

Dyslipidemia is defined as an abnormal plasma lipid status. Common lipid abnormalities include elevated levels of total cholesterol, low density lipoprotein (LDL) cholesterol, lipoprotein (a) and triglyceride; low levels of high density lipoprotein (HDL) cholesterol and a preponderance of small, dense particles. These abnormalities can be found alone or in combination[1].

Approximately 50% of U.S adults have an elevated total cholesterol level and the vast majority of patients with atherosclerotic vascular disease have some form of dyslipidemia.

Despite marked benefits of lipid therapy, 70% of individuals with dyslipidemia fail to meet the LDL cholesterol targets established by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) [73]

RISK FACTORS

Elevated levels of total cholesterol and LDL cholesterol and low levels of HDL cholesterol are major modifiable lipid risk factors for coronary artery disease[3].

It has been estimated that for each 1% decrease in LDL cholesterol and for each 1% increase in the HDL cholesterol, the risk for cardiovascular events is reduced by 2% and 3% respectively.

So routine lipoprotein analysis is recommended by the NCEP ATP III for all adults aged 20 years and older, as dyslipidemia is usually asymptomatic condition and early recognition and treatment improves prognosis[30].

LIPID THERAPY AND THE NEW CORONARY ARTERY DISEASE PARADIGM

It has been now established that the most dangerous rupture, prone atherosclerotic plaques are not necessarily those causing the most severe narrowing and that most of acute coronary syndromes are caused by lesions that were less than 70% stenotic prior to ulceration and thrombosis. Non obstructive plaques with extensive inflammation (Stimulated by oxidized lipoproteins in the vessel wall), lipid rich cores and thin fibrous caps are more prone to ulceration and rupture than longstanding obstructive lesions with extensive calcification and thick fibrous caps comprised of dense collagenous tissue[78].

Moderate reductions in low density lipoprotein (LDL) cholestrol slow the progression of coronary artery disease in most patients and lesion regression is more frequent with LDL cholesterol reduction. Nevertheless, the beneficial effects of lipid therapy are due more to plaque stabilization than to changes in stenos is severity, which are generally modest and disproportionate to the 25-80% reduction in major cardiovascular events[72].

Plaque stabilization, which can be accomplished in weeks to months with aggressive treatment of dyslipidemia may be related to resorption of macrophage and extra cellular lipid deposits, a decrease in neointimal inflammation and maintenance of fibrous cap integrity.

Effective treatment transforms the inflamed, friable plaque into a stable fibrotic plaue that is less prone to ulceration, rupture and thrombosis. In addition lipid lowering therapy, improves endothelial dysfunction caused by dyslipidemia, resulting in additional vasodilatory, anti thrombotic and anti-inflammatory effects. For patients with coronary artery disease, treatment of dyslipidemia and other concomitant risk factors is essential to improve long term prognosis[29].

Several large, randomized, places to controlled trials of statin therapy have shown reductions in cardiovascular morbidity with lipid therapy in both primary and secondary prevention[2].

Despite minor improvements in lesion severity, lipid lowering therapy results in marked (25-80%) reductions in major cardiovascular events primarily due to plaque stabilization[42].

FAMILY OF LIPIDS

AN OVERVIEW

CHOLESTEROL

It is the most abundant sterol in animal kingdom. It is not present in plant oils. It was first isolated from human gallstones. The total amount of cholesterol in human beings is about 130-160gm. The concentration in various major tissues is as follows.

	PERCENT	WT.GM
Brain and nervous system	2%	32g
Muscle	0.1%	30g
Blood	0.21%	11.3g
Liver	0.3%	5.1g
Adrenal gland	10%	1.2g
Skin	0.3%	12g
Heart, lungs, kidneys, Spleen	0.25%	5g

Cholesterol in serum can be quantitatively determined with ‘kit’ method which uses enzyme procedure.

Cholesterol as an abundant constituent of brain, nerves and spinal cord it functions as an insulating covering of impulse generations and transmitting structure.

TRIGLYCERIDES

These are formed by esterification of glycerol with fatty acids, which have a hydrocarbon group attached to carboxyl group. It is the major lipid in chylomicrons and VLDL and serves as energy substrate in liver and peripheral tissues particularly muscle. Excess energy is stores as triglyceride in adipose tissue (about 6kg).

PHOSPHOLIPIDS

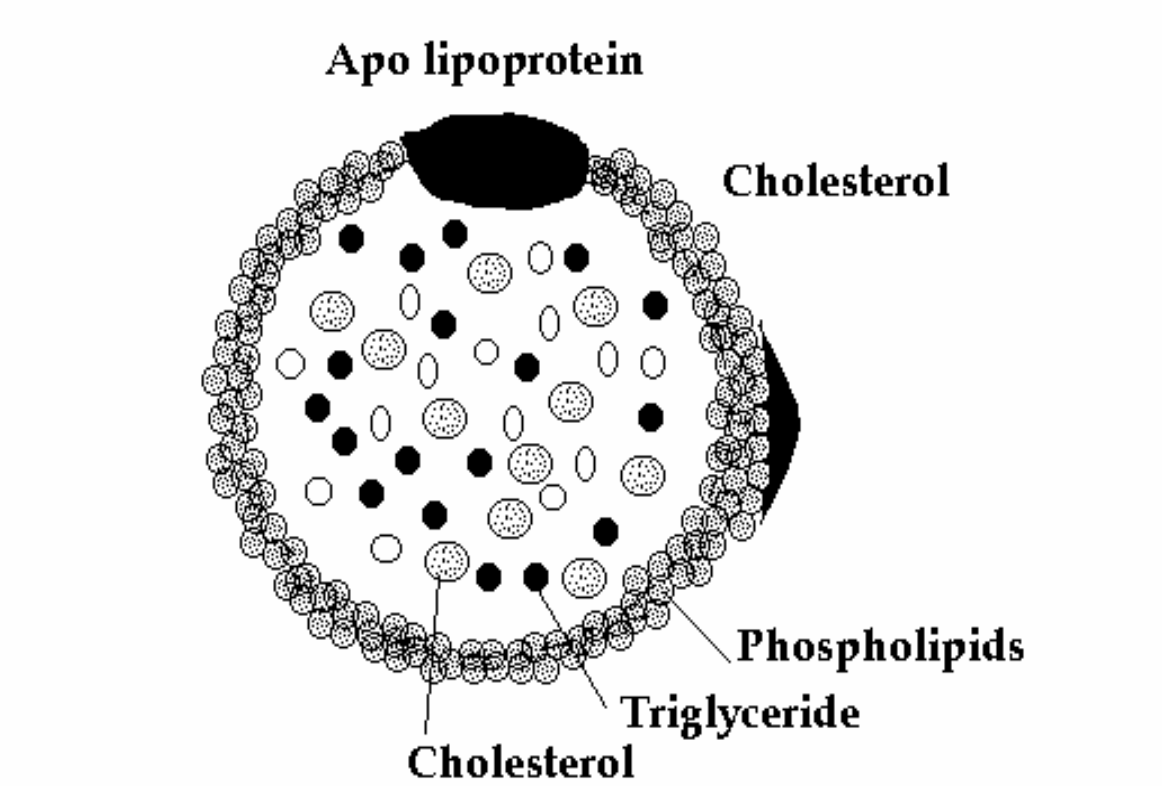
They are essential components of cell membrane. Vast majority of lipoprotein surface is formed of phospholipids that form monolayers, acting as interface with both plasma components and non-polar lipids of lipoprotein care.

APOPROTEINS

Apoproteins play important roles in lipoprotein structure, stabilization, metabolism including enzyme activation or inhibition or act as ligand for lipoprotein receptors.

MAJOR APOPROTEINS AND THEIR FUNCTIONS

	Number of amino acids	Approx. Molecular Weight(Da)	Origin	Lipoprotein distribution	Principal function
A-I	**243**	**28,000**	**Liver, Intestine**	**HDL, Chylomicrons**	**LCAT activator**
A-II	**154**	**17,000**	**Liver, Intestine**	**HDL, Chylomicrons**	**Structural protein in HDL**
A-IV	**391**	**40,000**	**Liver, Intestine**	**HDL, Chylomicrons**	**Non-specific LCAT cofactor**
B48	**2152**	**2,46,000**	**Intestine**	**Chylomicrons, Chylomicron Remnants**	**Mediates chylomicron formation and secretion**
B100	**4536**	**5,13,000**	**Liver**	**VLDL, LDL**	**Mediates hepatic VLDL formation legand for LDL receptor**
C-I	**57**	**7000**	**Liver**	**Chylomicrons VLDL, HDL**	**Inhibitor of chylomicron uptake**
C-II	**78**	**9000**	**Liver**	**Chylomicrons VLDL, HDL**	**LPL activator**
C-III	**79**	**9000**	**Liver**	**Chylomicrons VLDL, HDL**	**? Inhibitor of LPL**
D	**?**	**2000**	**Liver**	**HDL**	**? Involved in cholesterol ester transfer**
E	**299**	**3400**	**Liver**	**Chylomicrons VLDL, HDL**	**Ligand for chylomicron receptor and LDL receptor**



STRUCTURE OF LIPOPROTEIN PARTICLE

CHARACTERISTICS OF LIPOPROTEINS

Class	Diameter (A^0)	Density (g/ml)	Electro-Phoretic mobility	Chemical composition (% of dry mass)				
				Trigly-cerides	Choles-terol	Choles-terol	Phospho lipids	Proteins
Chylo microns	800-5000	0.930	α 2	86	3	2	7	2
VLDL	300-800	0.960-1.006	preβ	55	12	7	18	8
IDL	250-350	1.006-1.019	Slow preβ	23	29	9	19	19
LDL	216	1.019-1.063	β	6	42	8	22	22
HDL2	100	1.063-1.125	α_{ι}	5	17	5	33	40
HDL3	75	1.125-1.210	α_1	3	13	4	25	55
Lp (a)	300	1.055-1.085	Slow preβ	3	33	9	22	33

PLASMA LIPID ENZYMES

1. LIPOPROTEIN LIPASE

It converts lipoprotein tryglycerides in to free fatty acids and monoacids by peripheral tissues. It is synthesized in adipose tissue and in the muscle. It is transported by ill defined mechanism to surface of capillary endothelial cells where it interacts with triglyceride rich lipoproteins.

2. HEPATIC TRIGLYCERIDE LIPASE

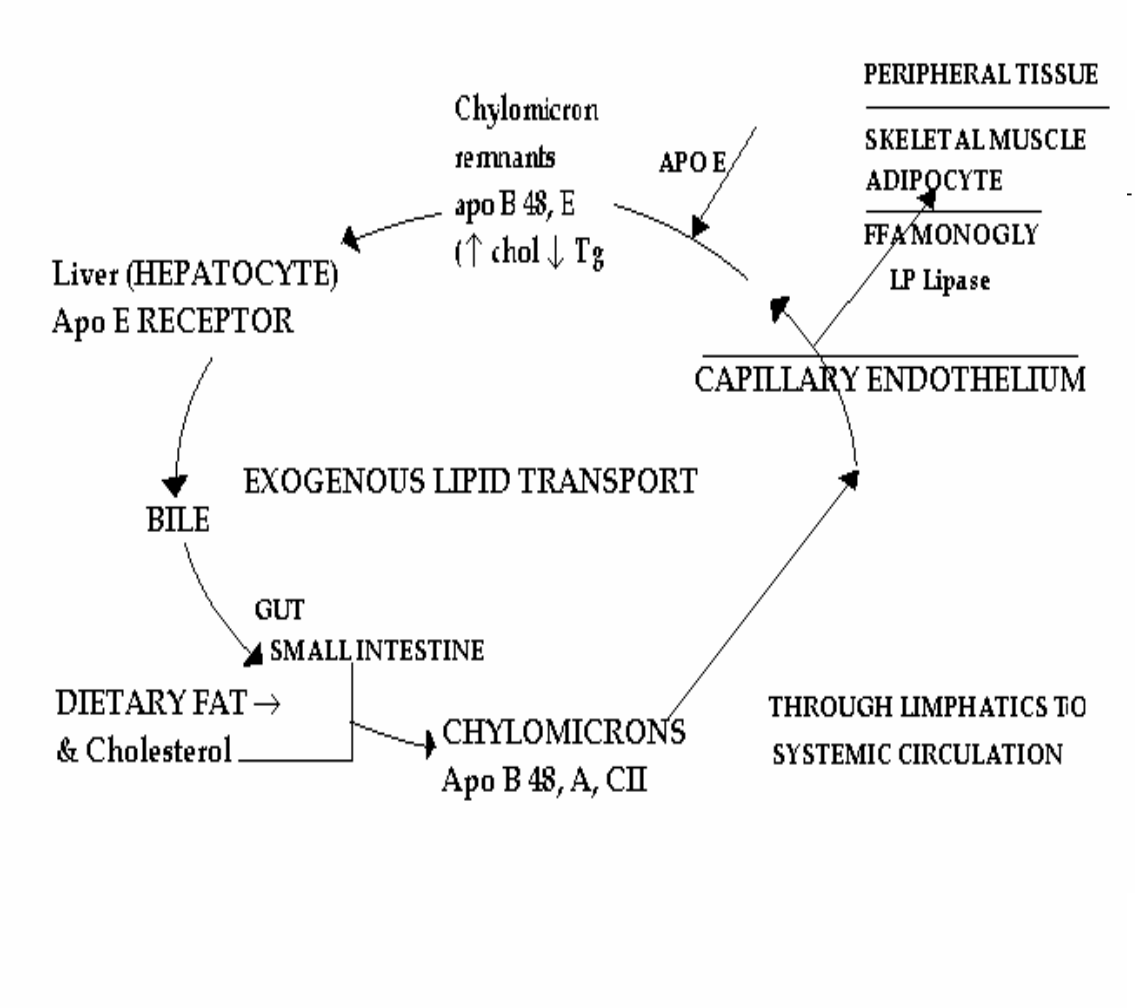
It functions as triglyceride hydrolase and phospholipase. It is an enzyme with homology to lipoprotein lipase that is synthesized in the liver. Its physiological actions are to remove triglycerides and phospholipids from chylomicrons and VLDL remnants and to possibly augment chylomicron uptake by liver.

3. CHOLESTEROL ESTER TRANSFER PROTEIN (CETP)

It is synthesized in liver and is an important plasma protein that mediates the exchange of triglycerides in VLDL, chylomicrons and remnants for cholesterol esters in HDL and LDL.

4. LECITHIN CHOLESTEROL ACYL TRANSFERASE (LCAT)

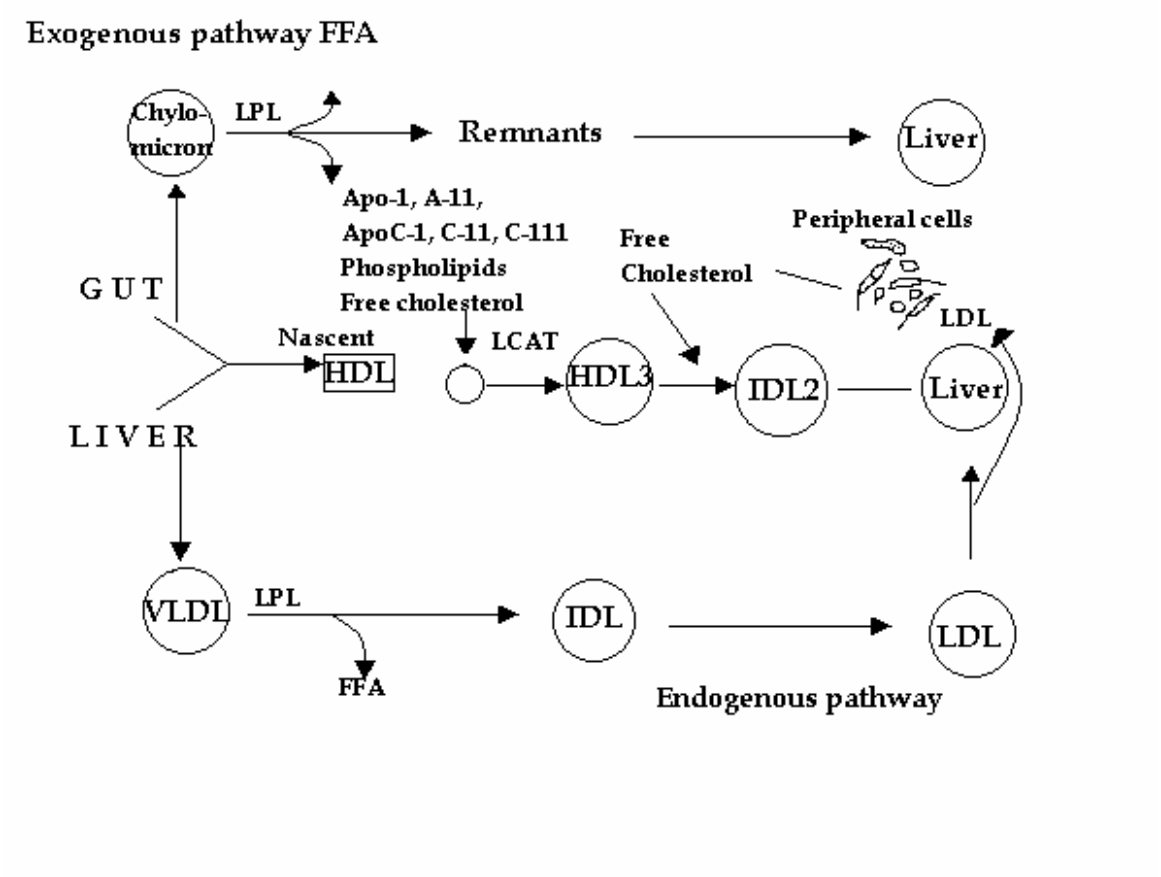
It converts HDL cholesterol into cholesterol ester. The newly formed cholesteryl ester can move from surface of high density lipoprotein to the core, allowing the particles to adsorb more free cholesterol on to their surface.



Schematic representation of Exogenous Lipid

Transport & Metabolism

LP Lipase : Lipoprotein Lipase

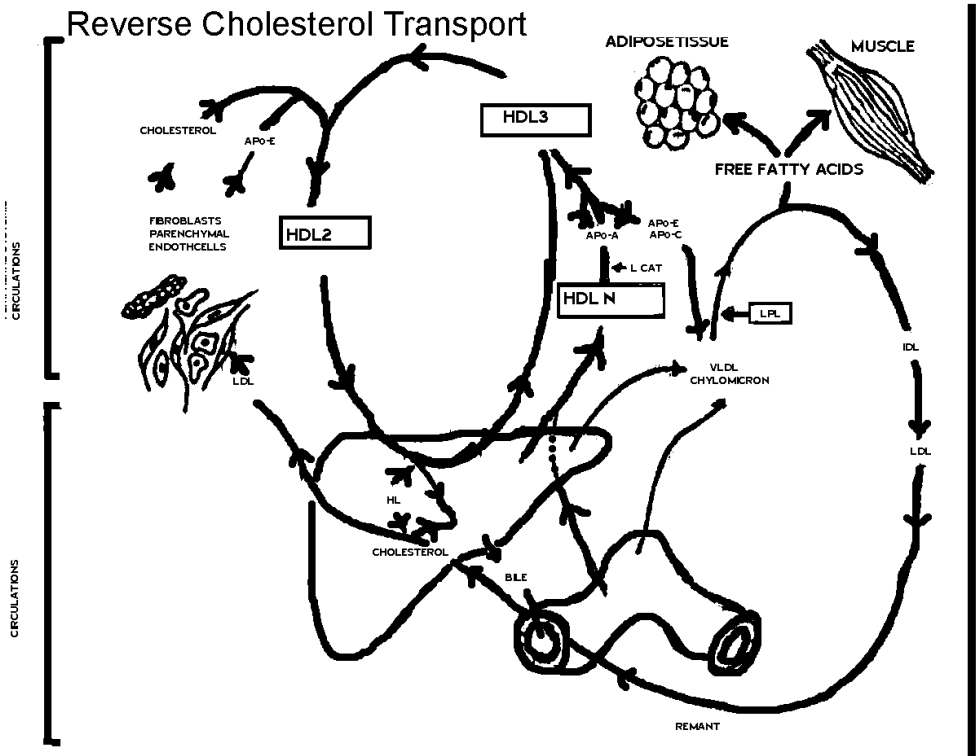


Lipid Transport – Endogenous Pathway (Diagrammatic Pathway)

LPL – Lipoprotein Lipase, HL – Hepatic Lipase

CETP – Cholesterol Ester Transport Protein

FFA – Free Fatty Acids



Diagramatic representation of HDL and reverse Cholesterol transport
HDLn : Nasent HDL
LCAT : Lecithin Cholesterol Transferase
LPL : Lipoprotein Lipase, HL : Hepatic Liapse
FFA : Free Fatty Acid

ROLE OF RECEPTORS IN LIPID DISORDERS

THE LDL RECEPTOR

Of all lipoprotein receptors, the most important is the low density lipoprotein (LDL) receptor. It is a highly regulated receptor and takes major part in cholesterol metabolism.

It is present in almost on all cell membrane though in variable number. The amino terminal of clathrin present on the cytosolic side of the receptor is exposed outside which works as the binding site for APO-B-100, but not for other apportions including APO-B-48. After binding with the receptor, the LDL molecule is taken in as a whole by the process of endocytosis. After leaving the LDL particle in the cytosol, the receptor returns back to the original site, so in this process the receptor is not destroyed but recycled.

When the intracellular cholesterol level rises, LDL receptor activity is down regulated and when the intra cellular cholesterol falls, LDL receptor activity is upgraded.

High Density Lipoprotein (HDL) transfers cholesterol to the VLDL and chylomicron but ultimately to the LDL which is taken in via the LDL receptor.

ABNORMAL LDL AND ROLE OF RECEPTORS

Normal LDL does not cause foam cell formation when incubated with cultured macrophages or smooth muscle, but when the LDL undergoes lipid peroxidation (Oxidized LDL) it becomes a ligand for an alternative scavenger pathway for uptake into the cells.

These scavenger receptors are present on the endothelial cells and macrophages. This pathway is less regulated than the LDL receptor pathway.

Though LDL is the most harmful lipoprotein, elevated VLDL and Triglycerides possibly enhances atherosclerosis. It has been seen that cholesteryl esters enriched VLDL isolated form cholesterol fed animals can be taken by receptors on macrophages and smooth muscle cells and cause foam cell formation.

This cholesteryl ester rich VLDL is enriched in APO-E and is probably the representative of VLDL remnant. This APO-E receptor has been termed as a LDL receptor related protein (LRP). APO-E rich lipoproteins bind to LRP and thereby help in the uptake of his remnants by the liver.

Familial hyper cholesterolemia and familial defective APO-E-100 are the disorders related to receptor abnormality.

LIPID DISORDERS AND DYSLIPIDAEMIAS

Hyperlipidaemia or hyperlipoproteinaemias are disturbances of lipid transport that result from the accelerated synthesis or retarded degradation of lipoproteins that transport cholesterol and triglycerides through plasma. Hyperlipidaemia consists of an excessive accumulation of one or more of the major lipids transported in plasma and is a manifestation of one or more abnormalities of metabolism or transport.

It is not easy to define the exact cut off point for high cholesterol or triglycerides. However as a working rule hyperlipoproteinaemia is considered to be present whenever the plasma cholesterol level exceeds 5.2 mmol/L. (200mg/dl) or the triglyceride level exceeds 2.2 mmol/l (200mg/dl).

The value of cholesterol represents the total cholesterol, which includes both cholesterol esters and unesterified cholesterol.

An isolated elevation in plasma trigylcerides indicates that the concentration of chylomicrons or VLDL is increased. On the other hand, an isolated elevation of plasma cholesterol nearly always indicates that the concentration of LDL is increased.

The various combinations of elevated lipoproteins have been divided into six lipoprotein types or patterns by Freidrickson on the basis of specific electrophoretic patterns of the various plasma lipoproteins.

PATTERNS OF LIPOPROTEINS IN PLASMA

IN HYPERLIPOPROTEINEMIAS

LIPOPROTEIN PATTERN	MAJOR ELEVATIONS IN PLASMA	
	Lipoprotein	**Lipid**
Type – I	Chylomicrons	Triglycerides
Type – II a	LDL	Cholesterol
Type – II b	LDL and VLDL	Cholesterol and Triglyceride TG/Chol<5:1
Type – III	Chylomicrons remnants & IDL	TG and Cholesterol TG/Chol > 5:1
Type – IV	VLDL	Triglycerides (TG)
Type – V	VLDL and Chylomicrons	Triglycerides (TG) and Cholesterol TG/Chol > 5:1

PATHOPHYSIOLOGICAL CLASSIFICATION OF THE HYPERLIPIDAEMIA

Mechanism	Disorders		Lipoprotein Abnormalities	Common Xanthomas	Early Athero sclerosis
	Primary	Secondary			
Increased triglyceride production Increased Endogenous VLDL synthesis	Familial Hypertri-glyceridemia	Hyper insulinemic states Obestiy Estrogen therapy Glucocorticoid therapy Type 2 DM, treated Growth Hormone excess Alcoholism Pregnancy	Increased VLDL Increased VLDL And chylomicrons	None; eruptive	None ?
Decreased triglyceride removal Abnormal LPL function	LPL deficiency LPLactivator (apo C II) deficiency LPL Inhibitation	Low insulin (untreated diabetes mellitus) Hypothyroidism Uremia Dysglobulinaemia (systemic erythematosus, myeloma, lymphoma, macroglobulinaemia)	Increased VLDL Chylomicrons	Eruptive	None
Decreased remnant removal: Core lipid accumulation	Dysbetalipo-proteinaemia (broad – beta disease)	Hypothyroidism	Increased remnants, VLDL, chylomicrons, Abnormal apo E	Planar (palmar) Tuberous Tubero eruptive disease	Coronary peripheral vascular

PATHOPHYSIOLOGICAL CLASSIFICATION OF THE HYPERLIPIDAEMIA

Table Continued…

Mechanism	Disorders		Lipoprotein Abnormalities	Common Xanthomas	Early Athero sclerosis
	Primary	Secondary			
Surface lipid Accumulation Decreased LDL removal	LCAT deficiency Familial cholesterolaemia Familial defective Apo-B-100	Liver disease Hypothyroidism Anorexia Nervosa	Disc-shaped HDL LP-X Increased LDL	Tendon Xanthomas	Coronary
Mechanisms unknown combined hyperlipid-aemias (multiple lipoprotein phenotypes)		Hypothyroidism Nephrotic Syndrome Glucocorticoid therapy	Increased LDL and / or VLDL Increased Apo B	Nil	Coronary

GENETIC HYPERLIPOPROTEINEMIAS[36]

Disorders	Primary defect	Plasma Lipoprotein pattern	Genetic Mechanism	Clinical findings	Estimated Population Frequency
Familial Hypercholesterolaemia	LDL receptor	2a, 2b	Autosomal dominant	Palmar, Tuberous xanthomas, Premature atherosclerosis	1-2/1000
Polygenic Hypercholesterolaemia	Unknown	2a, 2b	Polygenic	Palmar, Tendinous, xanthomas, premature atherosclerosis	-
Familial Hypercholesterolaemia	Unknown	4,5	Autosomal dominant	Nil	2/1000
Familial combined Hyperlipidaemia	Unknown	2a,2b,4,5	Autosomal dominant	Eruptive xanthomas Premature atherosclerosis	3-5/1000
Familial dysbeta Lipoproteinaemia	Apo E	3	Autosomal recessive	Premature atherosclerosis Peripheral vascular disease	1/10,000
LPL deficiency	Lipoprotein lipase	1,4	Autosomal recessive	Chylomicronaemia Syndrome, eruptive xanthomas	- Rare

GENETIC HYPERLIPOPROTEINEMIAS[36]

Table Continued…

Disorders	Primary defect	Plasma Lipoprotein pattern	Genetic Mechanism	Clinical findings	Estimated Population Frequency
Hepatic Lipase Deficiency	Hepatic lipase	-	Unknown	-	Rare Rare
Apo C-II deficiency LCAT deficiency Lipid transfer protein deficiency	apo C II Lecithin cholesterol acyl transferase Lipid transfer protein	1,4 - -	Autosomal recessive Autosomal recessive Unknown	Chylomicronaemia syndrome Corneal opacities Nephrotic syndrome, Renal failure -	Rare

SECONDARY HYPERLIPOPROTEINEMIAS

Disease	Plasma Lipoprotein Pattern							Proposed Mechanism for hyper lipoproteinemias	Associated Abnormality of carbohydrate metabolism
	CHY	IDL	VLDL	LDL	CHY+ VLDL	LDL+ VLDL	Type		
Endocrine Diabetes Mellitus, Severe, untreated	+				+		**1,5**	**Increased secretion of VLDL**	**Insulin deficiency**
Moderate		+	+		+	+	**2b,3,4,5**	**Decreased LPL&hence decreased catabolism of VLDL abd chy&VLDL**	**Insulin resistance**
Corticosteroid therapy High dose Low dose: Cushing's	+		+	+	+	+	**1&5 2a,2b,4**	**Increased VLDL secretion in & accelerated conversion to LDL**	**-**

SECONDARY HYPERLIPOPROTEINEMIAS

Table Continued…

Disease	Plasma Lipoprotein Pattern							Proposed Mechanism for hyper lipoproteinemias	Associated Abnormality of carbohydrate metabolism
	CHY	IDL	VLDL	LDL	CHY+ VLDL	LDL+ VLDL	Type		
Hypothyroidism		+	+	+	+	+	**2a,2b,3, 4&5**	**Decreased LPL activity Decreased receptor mediated LDL degradation**	**Insulin deficiency or Insulin resistance**
Hypopituitarism (atellotic dwarfism)			+		+		**4,5**	**Decreased LPL activity Decreased VLDL, &CHY degradation**	
Acute Hepatitis (non fulminant)			+				**4**	**Decreased LCAT**	-
Hepatoma				+			**2a**	**Lack of feedback inhibition of hepatic chol synthesis by dietary chol**	-

SECONDARY HYPERLIPOPROTEINEMIAS

Table Continued…

Disease	Plasma Lipoprotein Pattern							Proposed Mechanism for hyper lipoproteine mias	Associated Abnormality of carbohydrate metabolism
	CHY	IDL	VLDL	LDL	CHY+ VLDL	LDL+ VLDL	Type		
Antihypertensive therapy (Thiazides, β-blockers))		+	+				3,4	?Unopposed adrenergic activity	-
Iso retinal therapy			+	+	+		4,5	Unknown	-
Stress: emotional, Ml, burns, septicemia			+	+	+		4,5	Decreased LPL activity: Decreased catabolism, Increased secretion VLDL	Insulin Resistance
Acromegaly			+				4	Increased secretion of VLDL	Insulin resistance
Anorexia Nervosa			+				2a	Decreased biliary secretion in of chol and bile acids	-

SECONDARY HYPERLIPOPROTEINEMIAS

Table Continued…

Disease	Plasma Lipoprotein Pattern							Proposed Mechanism for hyper lipoproteinemias	Associated Abnormality of carbohydrate metabolism
	CHY	IDL	VLDL	LDL	CHY+ VLDL	LDL+ VLDL	Type		
Estrogen or oral contraceptive therapy			+		+		**4,5**	**Increased VLDL secretion in females genetically predisposed to hypertriglyceridemia**	**Insulin resistence**
Lipodystrophy (congenital or acquired)			+		+		**4,5**	**Increased VLDL secretion**	**Insulin resistance**
Von Gierke's disease (glycogen storage disease, Type I)			+		+		**4,5**	**Decreased LPL leads to decreased catabolism of VLDL&CHY Increased VLDL secretion**	**Hypoglycemia with decreased Insulin secretion**

SECONDARY HYPERLIPOPROTEINEMIAS

Table Continued…

Disease	Plasma Lipoprotein Pattern							Proposed Mechanism for hyper lipoproteinemias	Associated Abnormality of carbohydrate metabolism
	CHY	IDL	VLDL	LDL	CHY+ VLDL	LDL+ VLDL	Type		
Non-Endocrine Renal disease Nephrotic syndrome			+	+	+	+	**2a,2b, 4,5**	**Increased secretion of VLDL increased secretion of VLDL Increased secretion form liver, decreased catabolism of VLDL**	**-**
Uremia		+	+		+		**3,4,5**	**Decreased LPL activity Decreased catabolism of VLDL**	**Insulin Resistance**
Alcoholism			+		+		**4,5**	**Increased VLDL secretion individuals genetically predisposed to hyper triglyceredimea**	**-**

SECONDARY HYPERLIPOPROTEINEMIAS

Table Continued…

Disease	Plasma Lipoprotein Pattern							Proposed Mechanism for hyper lipoproteinemias	Associated Abnormality of carbohydrate metabolism
	CHY	IDL	VLDL	LDL	CHY+ VLDL	LDL+ VLDL	Type		
Dysglobulinemia	+	+		+	+		1,2a, 3,5	IgG, IgM0 antibodies to heparin chylomicron remenants in VLDL resulting in low LPL activity and metabolism	-
Werner syndrome	+				+		1,5	Unknown	Insulin Resistance
Acute intermittent porphyria				+		+	2a,2b	Unknown	-

CHY – Chylomicron

IDL – Intermediate Density Lipoprotein

VDL – Very low Density Lipoprtoein

LDL – Low Density Lipoprotein

CHOL – Cholesterol

LPL – Lipoprotein Lipase

LCAT – Lecithin Cholesterol Acyl Transferase

ASSAY PROCEDURES FOR SERUM LIPOPROTEINS

Ultra centrifugation of lipoproteins remains the standard procedures for separation of lipoprotein fractions.

LIPOPROTEIN ELECTROPHORESIS is a simple procedure. The electrophorosis can be carried out on agarose sel. However there is a problem of automation. In case of dyslipoproteinemia difficulties are faced in obtaining true hipoprotein separation and quantification.

SPECIFIC PRECIPITAION OF LIPOPROTEINS is based on the original work of Burstein. Their simplicity and possibility of automation has made them widely used procedures.

High density lipoprotein (HDL) cholesterol can be determined by ultracentrifugation, electrophoresis, and precipitation based methods. Several approaches for direct measurement of HDL-Cholesterol in the serum have been proposed including the use of magnetically responsive particles such as polyanion metal combinations.

Lipid and lipoprotein measurements are usually performed in the fating state. But recent evidences show that post-prandial lipaemia which includes Triglyceride rich, APO-B containing lipoproteins are atherogenic.

LIPID LEVELS IN INDIANS

Category	**LDL – C (mg/dL)**	**Cholesterol (mg/dL)**	**Tryglycerides (mg/dL)**
Desirable	< 100	< 150	< 150
Borderline	100 – 130	150 - 200	150 - 170
High	> 130	> 200	> 170

(EnasEA – Why is there an epidemic of malignant CAD in young Indians ?

Asian J Clin Cardiol 1998 ; 1 : 43)

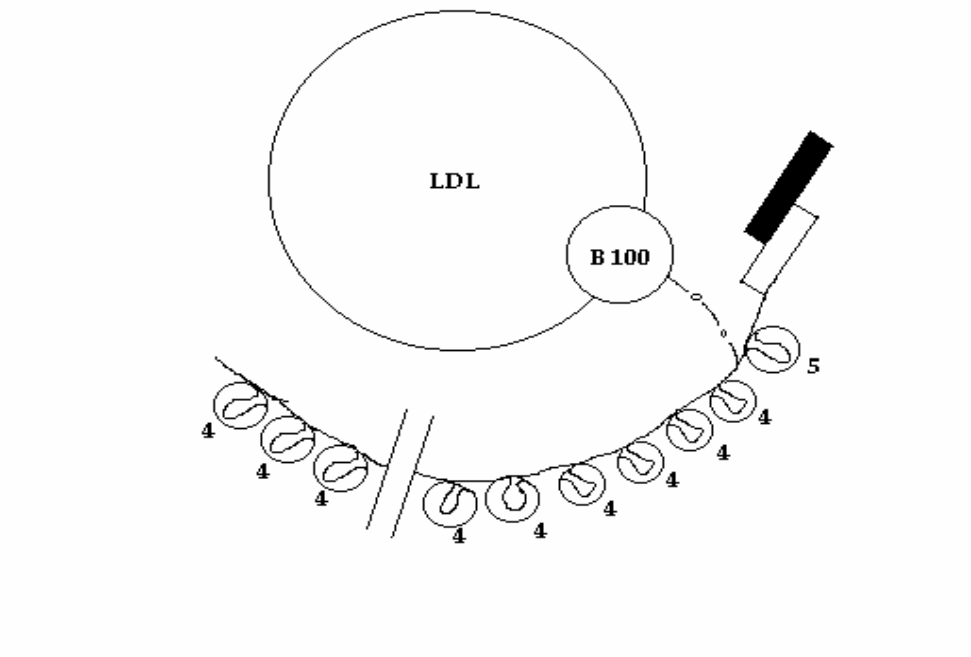
LIPOPROTEIN (a) – Lp(a) is a predictor of macro vascular disease.

It was found that the level of Lp(a) was genetically determined by an autosomal dominant made of inheritance.

It is likely that Lp(a) is secreted directly by the liver and then associates with LDL. LP(a) has structural homology to LDL in its protein and lipid composition. It has the capacity to bind to the fibrin and to membrance proteins of endothelial cells, monocytes and LP(a) on the surface of fibrin and cell membranes favours finbrin and cholesterol deposition at sites of endothelial injury. On the top of this pathogenesis of insufficient activation of transforming growth factor-Beta due to low plasmin activity, result in migration and proliferation of smooth muscle cells into the vascular intima[53].

LP(a) is widely recognized as a biological marker for familial coronary artery diseas and high levels of LP(a) a can be considered to have the same importance as history of premature coronary artery disease in patients.

Three well known studies like PROLAM study, Framingham Heart study and Quebec Cardiovascular study demonstrated that LP(a) has independent risk factor for coronary artery disease but appears to have increased risk when associated with other lipid risk factors.



Lipoprotein (a) consists of LDL jointed by a single disulphide Bridge to apoprotein (a), which consist of the protease domain, kringle 5 and a variable number of kringle 4 repeats of plasminogen

Myocardial infarction and cardiac death has been well correlated with the serum level of Lp(a). Many studies also showed a linear relationship with the severty and extent of coronary artery involvement with the serum Lp(a) level.

Evidence has emerged that male and female migrants from the Indian subcontinent have a higher mortality from Coronary Artery Disease than the native white population because of increased levels of Lp(a).

MANAGEMENT OF DYSLIPIDEMIA

THERAPEUTIC LIFESTYLE CHANGES (TLC) DIET[47,57]

Initiation of the TLC diet as recommended by NCEP-ATP III has been estimated to lower LDL cholesterol levels by 10-20%

Therapeutic lifestyle changes (TLC) Diet

FOOD COMPOSITION	RECOMMENDATION
Total fat saturated fat polyunsaturated fat Monounsaturated fat	25-35% of total calories <7% of total calories up to 10% of total calories up to 20% of total calories
Carbohydrates	50-60% of total calories
Fiber	20-30 gm/day
Protein	15% of total calories
Cholesterol	<200 mg/day
Total calories	Sufficient to achieve or maintain desirable body weight.

PHYSICAL ACTIVITY

Regular exercise that increases heart rate 60-80% of maximal peak heart rate for 30 minutes on all or most days of the week can raise HDL cholestezol levels up by 30% and can prevent or improve hypertension, insulin resistance and type 2 diabetes, obesity, anxiety and depression[70].

Regular exercise can also help smokers quit, reduce the risk of myocardial infarction and stroke by 50% or more, reduce the risk of death following infarction 25% and improve functional capacity in patients with claudicating from peripheral arterial disease.

Recent studies indicate that physical activity does not need to be performed in a traditional structured exercise program to provide health benefits and that a lifestyle – based exercise program incorporating physical activity into daily living is effective at improving risk factors, weight and long term cardiovascular prognosis. (JAMA 1999: 281; 327-34) This can be accomplished by encouraging patients to use the stairs, walking whenever possible, gardening playing actively with children etc.

Examples of moderate physical activity from the Surgeon General's Report on Physical Activity and health (JAMA 1996: 276: 522)

Include:

- Washing and waxing a car or washing windows or floors for 45 minutes.
- Gardening, dancing fast (social) or rating for 30 minutes
- Walking 1 ¾ miles in 35 minutes (20min/mile)
- Pushing a stroller 1 ½ miles or bicycling 5 miles in 30 minutes
- Stair walking, shoveling snow or jumping rope for 15 minutes.

The important factor is to accumulate at least 30 minutes of moderate Physical activity all or most days of the week, which can be split in three 10 minutes blocks.

Health benefits may plateau at 3500 Kcal per week, the equivalent of moderately intense jogging or bicycling for one hour per day.

Patients with cardiovascular or respiratory disease or sedentary patients with multiple coronary artery disease risk factors interested in participating in a vigorous exercise program should be considered for stress testing.

WEIGHT CONTROL

Over weight and obesity increase the risk of all cause mortality and they increased morbidity from hypertension, dyslipidemia, type 2 diabetes, coronary artery disease, stroke, Gall bladder disease, osteo arthrosis, sleep apnea, respirators problems and cancers of endometrium, breast and colon[56].

Weight control improves blood pressure, triglycerides, LDL and HDL cholesterol, blood glucose and hemoglobin AIc levels in type 2 diabetics.

CLASSIFICATION OF OBESITY

CATEGORY **OBESITY CLASS**	**BMI (BODY MASS INDEX)** **= wt in kg** **(ht in m)^2**	**Waist circumference**	**Risk for type 2 diabetes, hypertension, CAD**
I	30 – 34.9	N ↑	High Very high
II	35.0 – 39.9	N or ↑	Very high
III	≥ 40.0	N or ↑	Extremely high

WAIST - HIP RATIO

Waist circumference is the minimum circumference measured between costal margin and iliac crest and hip circumference is measured over the buttocks. Waist – Hip circumference ratio more than 0.83 in females and 0.93 in males were taken as abnormal.

The treatment of overweight and obesity may require the combination of

- Dietary restriction – low carbohydrate and other 'fad' diets may facilitate early weight loss. The best approach to diet is to eat smaller portions of a well rounded (TLC) diet.
- Increased physical activity.
- Behavior therapy.
- Pharmacotherapy can be a useful adjunct to dietary restriction, increased physical activity and behaviour modifications but is unlikely to be effective as monotherapy. The anti-obesity drugs approved by the FDA are sibutramine[56] and orlistat. (Lipase inhibitor)
- Weight loss surgery - Gastric restriction or bypass should be reserved for motivated patients with extreme obesity (BMI $\geq$ 40 kg/m^2) despite non-surgical intervention.

AN OVERVIEW OF DRUG THERAPY

FOR DYSLIPIDEMIA

For patients with dyslipidemia who require drug therapy the drug should be added to not substitute for diet therapy and lifestyle modification[42].

EFFECTIVE CHOLESTEROL – LOWERING DRUGS[59,63]

DRUG(USUAL STARTING DOSE / MAX)	EFFECT ON LIPIDS	COMMENTS
HMG – COA Reductase Inhibitors (Statins)		
Rosuvastatin Atorvastatin[74] (10/80mg/d) Fluvastatin (20/80mg/d) Lovastatin(20/80 mg/d) Pravastatin[68] (20/80mg/d) Simvastatin[64](20/80mg/d)	LDL – decrease by 18-55% HDL – increase by 5-20% TG- Decreases by 7-30%	Overwhelmingly the drug class of choice for elevated LDL levels. <u>Adverse effects:</u> 1. Myopathy 2. Elevation of liver transaminases. Monitoring to be done every six months. 3. Fibrates and nicotinic acid should be used with caution in combination.

EFFECTIVE CHOLESTEROL – LOWERING DRUGS[59,63]

Table continued…

DRUG(USUAL STARTING DOSE / MAX)	EFFECT ON LIPIDS	COMMENTS
Nicotinic acid (Niacin)[42]		
Immediate release form (1.5-3/4.5g/day) Sustained – release form (1.2 /2g/day) Extended – release form (1-2/2g/day)	LDL – decrease by 5-25% HDL – increase by 15-35% TG- Decreased by 20-50%	Useful in nearly all dyslipidemias. <u>ADVERSE EFFECTS</u> 1. Flushing 2. Hyperglycemia 3. Hyperuricemia 4. Gastritis 5. Hepatotoxicity
Bile acid sequestrants		
Colesevelam 2.6 - 3.8/4.4g/d) Cholestyramine (4-16 / 24g/d) Colestipol (5-20/30g/d)	LDL –decreases by 15-30% HDL- increases by 3-5% TG-usually not affected may actually increase	Useful in moderate hypercholesterolemia, younger patients with elevated LDL cholesterol and women with elevated LDL cholesterol, who are considering pregnancy? Useful as adjunctive therapy with statins. <u>ADVERSE EFFECTS</u> 1. Gastritis 2. Decreased absorption of several drugs.

EFFECTIVE CHOLESTEROL – LOWERING DRUGS[59,63]

Table continued…

DRUG(USUAL STARTING DOSE / MAX)	EFFECT ON LIPIDS	COMMENTS
Fibric acid Derivatives		
Gemfibrozil (600mg bid) Fenofibrate (160 mg/d) Clofibrate (1000 mg bid)	LDL – decreases by 5-20% HDL – increases by 10-35% TG- Decreased by 20-50%	Major uses: 1. Hypertriglyceridemia 2. Atherogenic dyslipidemia (Especially type 'z' diabeties) ADVERSE EFFECTS 1. Dyspepsia 2. Gastritis 3. Cholesterol Gall stones 4. Myopathy
Cholesterol absorption inhibitor		
Ezetimibe (10mg/day)	LDL –decreases by 18% HDL- increases by 1% TG-decreases by 8%	Used as an adjunct to statins when further LDL lowering is required. Not recommended in moderate or severe hepatic in sufficiency. ADVERSE EFFECTS: Gastritis.

DRUG THERAPY BASED ON LIPID PROFILE

PROFILE	TREATMENT[48]	COMMENTS
(A) High LDL, Normal TG (Type II a)		
LDL ≥ 190 TG < 150	Mono therapy statin combination: Statin + ezetimibe, Statin + niacin, Statin + biteacid Sequestrants	Bile acid sequestrant is preferred as monotherapy if liver disease is present.
LDL ; 160 – 190 TG : 150 - 400	Monotherapy : Statin Alternative: Niacin, ezetimibe, Bile acid sequestrants	Statin, niacin, Bile acid sequestrants have process long term. Safety profiles
(B) Mixed Hyperlipidemia (↑LDL, ↓ TG) Type II		
LDL : 160 – 190 TG : 150 - 400	Monotherapy : Statin Alternative : Niacin, Fibric acid derivative, ezetimibe. <u>COMBINATION:</u> 1. Statin + niacin 2. Statin + fibric acid derivative 3. Statin + ezetimibe 4. Fibric acid derivated +bile acid sequestrant.	Niacin reduced myocardial infarction by 27% in the coronary drug project. Gemfibrozil reduced coronary artery disease by 34% in the Hesink Heart study.

DRUG THERAPY BASED ON LIPID PROFILE

Table continued…

PROFILE	TREATMENT[48]	COMMENTS
(C) Hyper Triglyceridemia (Type IV, Type V)		
LDL < 130 TG > 400	Monotherapy: Niacin, Fibric acid derivations, fish oils COMBINATION: 1. Fibric acid derivatives + fish oils[55]. 2. Niacin + fish oils. 3. Fibric acid derivatives + Niacin	1. Fish oils may be used as adjunctive therapy. 2. Treat to decrease the risk of pancreatitis.
(D) Isolated low HDL (HDL < 40 MG/DL)[49]		
LDL < 130 TG < 150	Monotherapy: 1. Niacin 2. Fibric acid derivatives 3. Statin COMBINATION: 1. Niacin + statin Niacin + fibric acid derivatives.	1. Low HDL greatly increase coronary artery disease risk even in the absence of elevated LDL. 2. Gemfibrozil reduced myocardial infarction (or) coronary death by 22% (VA-HIT study)

MATERIALS AND METHODS

The study was conducted at the intensive coronary care unit and medical wards of Tirunelveli Medical College Hospital, Tirunelveli during the period of August 2004 to August 2005.

- Hundred patients who were admitted with a confirmed diagnosis of acute myocardial infarction were enrolled in the study.
- The diagnosis of acute myocardial infarction was made if patients had ischemic type chest pain for ≥ 30 minutes with evidence of ST – segment elevation of ≥ 1 mm in two anatomically contiguous leads on the ECG or the appearance of a new left bundle branch block.
- Patients who had symptoms suggestive of acute myocardial infarction but did not meet the ECG diagnostic criteria, needed to have serum creatinine – kinase MB levels that were more than twice the upper limit of normal.
- Exclusion criteria were the following

  1. Symptoms suggestive of acute myocardial infarction ≥ 12 hours.
  2. Hospital stays of < 4 days.
  3. Already receiving lipid – lowering medications.

- All the patients were followed from the day of admission to the day of discharge.

LIPID MEASUREMENTS

Besides clinically examination and routine investigation, the serum lipid profile was measured within the first 24 hours of the onset of symptoms of myocardial infarction and again at day 4 post myocardial infarction.

The serum total cholesterol, triglyceride levels were measured by colorimetric test and HDL cholesterol is measured by precipitation assay.

The LDL cholesterol value was calculated by using the Friedewald formula.

LDL cholesterol = total cholesterol –HDL cholesterol – (triglyceride/5)

The cholesterol ratios then were calculated by using the total cholesterol / HDL cholesterol and LDL cholesterol / HDL cholesterol ratios. All the blood samples were 12 hours fasting samples.

STATISTICAL ANALYSIS

Continuous variables were expressed as the mean ± standard deviation (SD) and the categoric variables were expressed as a percentage. The student's't' test was used to compare lipid values and ratios between day 1 post M I and day 4 post M I. A two tailed 'P' value of < 0.05 was considered to be significant.

OBSERVATION AND ANALYSIS

1. Eighty one percent (81%) of the patients studied were men.
2. Hypertension was present in Thirty –three (33%) percent of the patients studied.
3. Twenty nine (29%) percent of the patients studied were diabetic.
4. Thirty seven (37%) percent of the patients studied were smokers.
5. Sixteen (16%) percent of the patients had family history of coronary artery disease.
6. Out of the hundred patients studied, non ST elevation myocardial infarction was diagnosed in twenty (20%) patients.
7. Eighty (80%) of the patients studied were diagnosed to have ST elevation myocardial infarction.
8. Sixteen (16%) of the patients studied were obese.

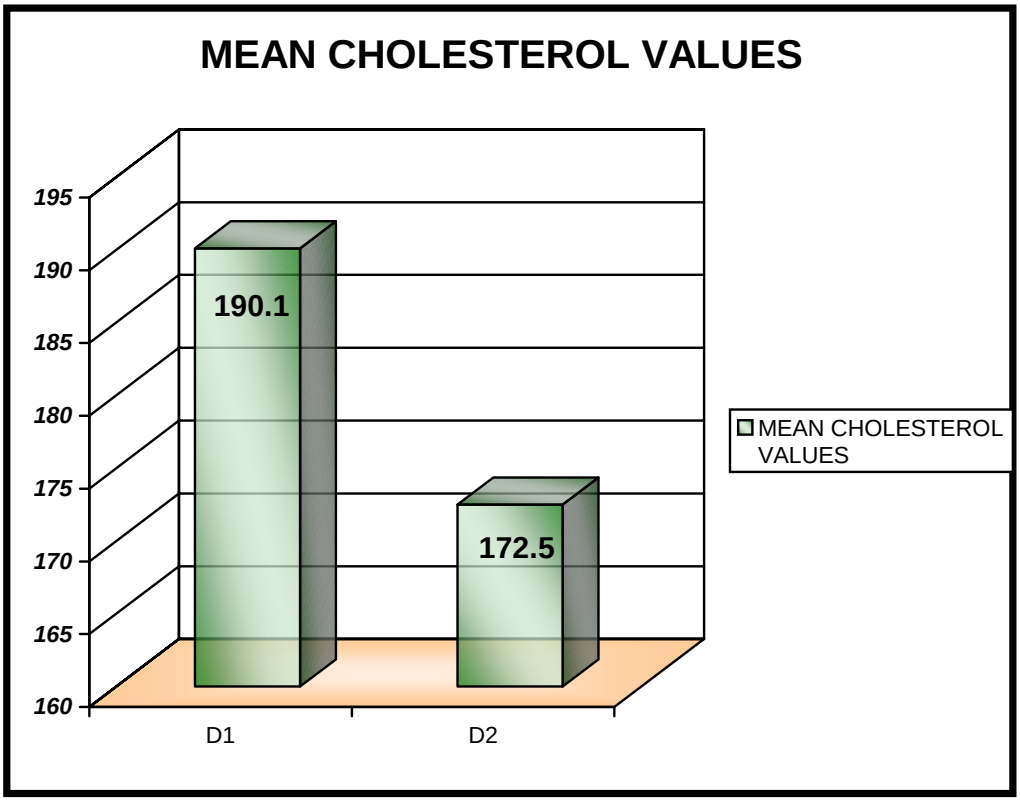
CLINICAL CHARACTERISTICS OF THE STUDY POPULATION

Total number of cases studied = 100

Sl.No	CHARACTERISTICS	PATIENTS No (Percentage)
1.	Hypertension	33%
2.	Diabetic mellitus	29%
3.	Smokers	37%
4.	Positive family history	16%
5.	Non ST elevation Myocardial infarction	20%
6.	ST elevation myocardial infarction	80%
7.	Obesity	16%

OBSERVATION IN LIPID PROFILE

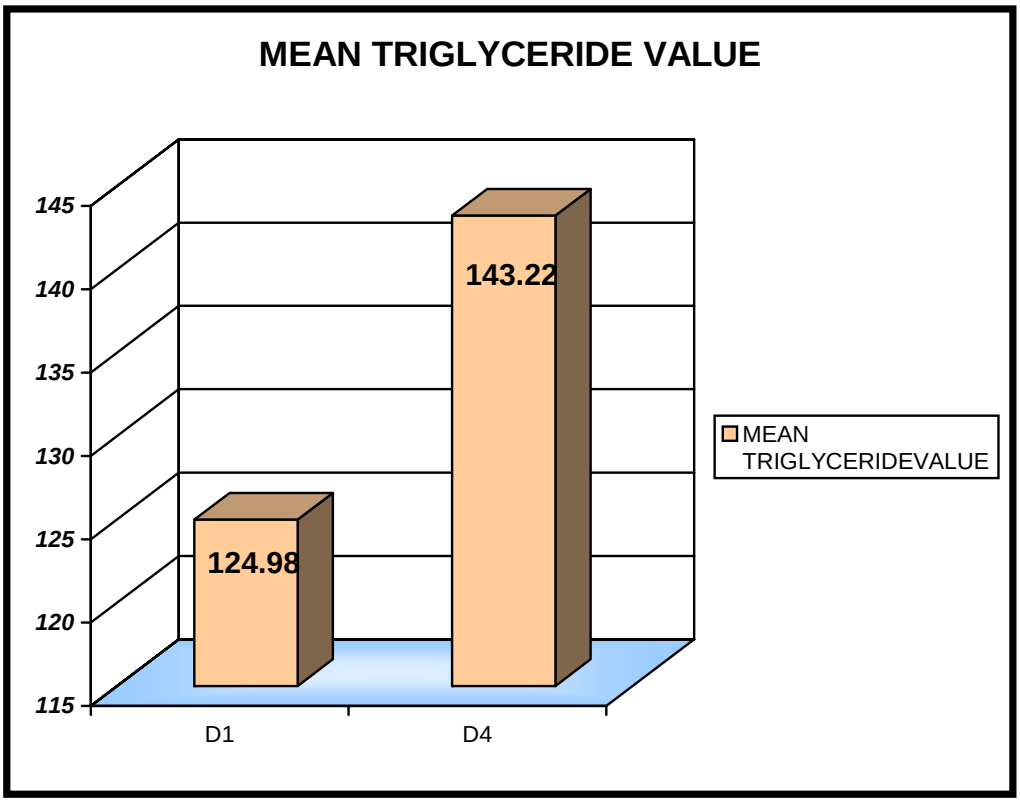
1. All serum lipid levels changed significantly between day 1 post myocardial infarction (within 24 hours) and day 4 post myocardial infarction.
2. On day 1 post myocardial infarction the mean total cholesterol value is 190.10.
3. On day 4 post myocardial infarction, the mean total cholesterol value is 172.50.



Mean Cholesterol Value – D1
Post Myocardial infarction = 190.10
Mean Cholesterol Value – D4
Post Myocardial infarction = 172.50

OBSERVATION IN LIPID PROFILE

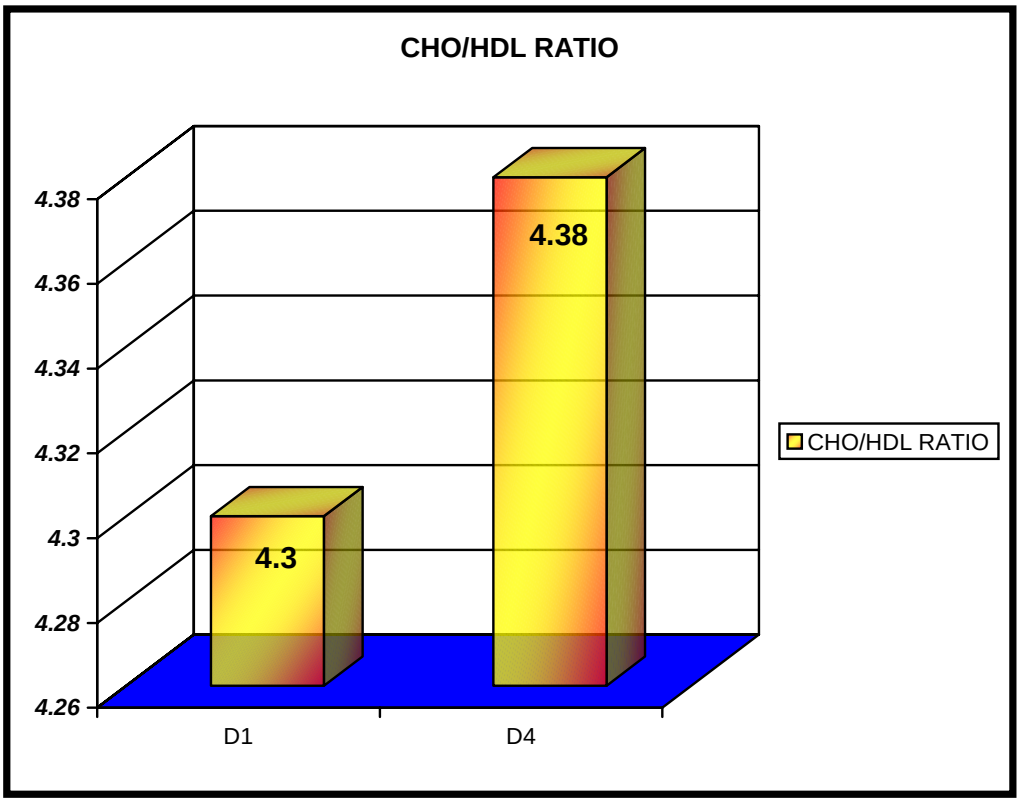
4. On day 1 of post – myocardial infarction, the mean triglyceride value was 124.98.
5. On day 4 of post – myocardial infarction, the mean triglyceride value was 143.22



Day – 1 Post Myocardial Infarction
Mean TriglycerideValue = 124.98
Day – 4 Post Myocardial Infarction
Mean TriglycerideValue = 143.22

OBSERVATION IN LIPID PROFILE

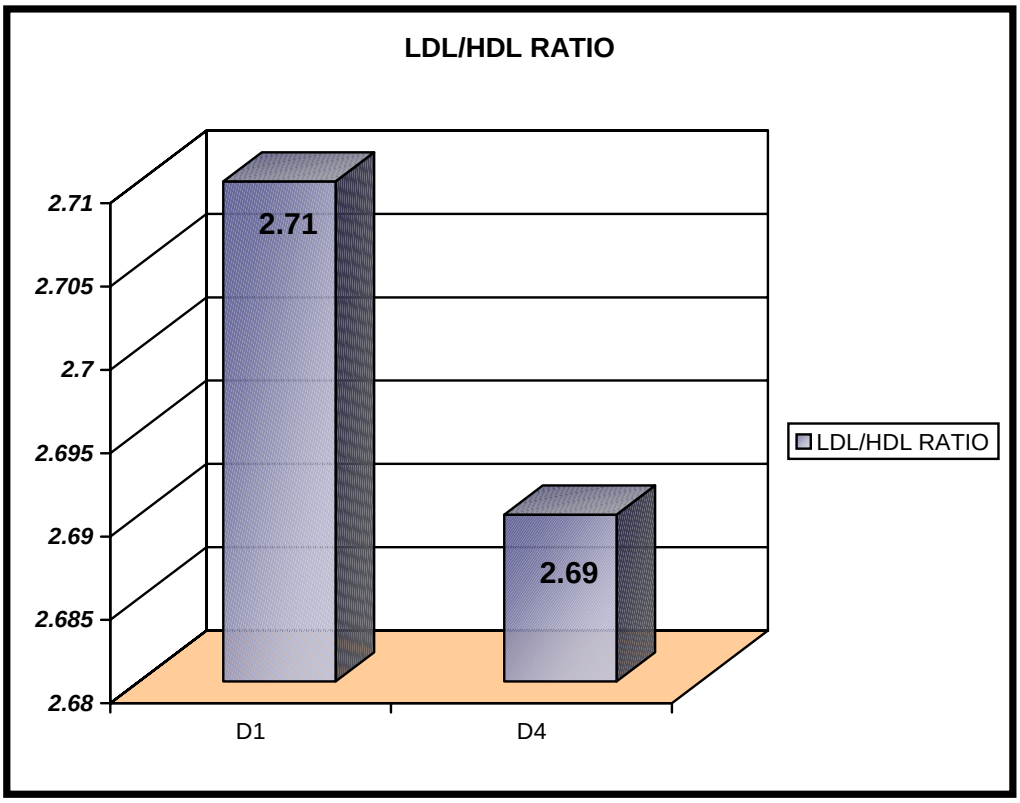
6. Regarding the Cholesterol ratios, the ratio at total cholesterol to High Density Lipo protein cholesterol (Cholesterol / HDL ratio) on day – 1 post myocardial infarction and day 4 post myocardial infarction were 4.300 and 4.380 respectively.



Day – 1 Cholesterol / HDL Ratio = 4.300
Day – 4 Cholesterol / HDL Ratio = 4.380

OBSERVATION IN LIPID PROFILE

7. The ratio at low density lipoprotein cholesterol to high density lipoprotein cholesterol (LDL/HDL ratios) on day – 1 post myocardial infarction and day- 4 post myocardial infarction were 2.710 and 2.690 respectively.



Day - 1 LDL / HDL Ratio	=	2.710
Day - 4 LDL / HDL Ratio	=	2.690

COMPARISON OF THE SERUM LIPID VALUES AND RATIOS BETWEEN WITHIN 24 HOURS OF MYOCARDIAL INFARCTION AND DAY-4 POST MYOCARDIAL INFARCTION

Serum lipids	Within 24hrs of Myocardial infarction	Day-4 post Myocardial infarction	'Z' and 'P' Values
Total cholesterol (mg/dl)	190.10±25.75	172.50±27.05	6.33
Triglycerides (mg/dl)	124.98±38.35	143.22±35.64	4.02
Total cholesterol / HDL cholesterol ratio	4.30±0.36	4.38±0.36	0.33
LDL cholesterol / HDL cholesterol ratio	2.71±0.40	2.69±0.38	0.06

The values are expressed as mean ± standard deviation.

If 'z' is > 1.96 and if 'P' is < 0.05 then the change is significant.

DISCUSSION

Many studies in the past few decades have shown that acute myocardial infarction results in a significant decrease in the serum levels of total cholesterol, LDL cholesterol and HDL cholesterol [5,7].

The acceptable time for the measurement of plasma lipids after an acute myocardial infarction is within 24 hours after the onset of symptoms and the plasma lipid levels measured beyond 24 hours are mostly considered to be invalid [6].

The post myocardial infarction decline in serum cholesterol occurs because of the acute – phase response[19], and is of greatest extent by days 4 to day 5 post – myocardial infarction[9].

Acute myocardial infarction like any other tissue injury, initiates various local and systemic reactions[20].

The local response includes vasodilation, leucocyte infiltration and chemotaxis, monocyte and macrophase activation and cytokine release[22].

The cytokines act on the systemic targets, including the liver to generate changes in the concentration of various heterogenous plasma proteins, collectively known as acute – phase reactants including lipoproteins[23,24].

By day 4 to 5 post – myocardial infarction, there is a significant decrease in the serum concentrations of apoprotein A-1 and apoprotein-B reflecting the maximum decrease in the serum cholesterol level by the time[25].

While the serum cholesterol level decreases after an acute myocardial infarction, the serum triglyceride level increases.

This paradoxical rise in serum triglycerides is due to and increase in serum – C reactive protein level which may increase to levels that are several hundred – fold higher than baseline 4 days after and myocardial infarction[24].

The C- reactive protein binds selectively with very LDL and interferes with its catabolism thereby increasing the serum triglyceride concentration[26].

The magnitude of the decrease in serum cholesterol level after an myocardial infarction is positively correlated with the infarct size and is not dependent on the patients age or sex, the development of arrhythmias, the medications being received or the development of heart failure[28].

The decrease in serum cholesterol levels after acute myocardial infarction is transient and these levels gradually return to the baseline pre-myocardial infarction values in 2 to 3 months[6].

Therefore most experts recommend measuring the serum cholesterol levels within the first 24 hours after the onset of an acute infarction[18] (or) otherwise deferring measurement until 2 to 3 months after the myocardial infarction[11].

However deferring the measurement of serum cholesterol levels in patients whose cholesterol levels were not determined with in the first 24 hours after the onset of acute infarction can lead to a delay in initiating the appropriate cholesterol lowering therapy for the secondary prevention of future coronary events[71].

The National cholesterol Education program guidelines recommend using the absolute values of total cholesterol, LDL cholesterol and HDL cholesterol as determined of the cholesterol risk and the therapeutic goals have been set forth using these absolute serum cholesterol levels.

These guidelines emphasized the issue stating that LDL cholesterol and HDL cholesterol are independent risk factors requiring individual attention.

Several large – scale epidemiologic studies have shown that the total cholesterol / HDL cholesterol ratio are also strong predictors of coronary artery disease events because these ratios sum up the importance of both the total cholesterol or the LDL cholesterol and HDL cholesterol collectively.

The study has shows that in certain situations in which the plasma cholesterol levels are not measured within the first 24 hours after the onset of acute myocardial infarction, cholesterol ratios determined from the serum cholesterol measurements taken after 24 hours of the onset of acute infarction could be used reliably for cholesterol risk assessment[9].

Because at day 4 post-myocardial infarction when the absolute values of serum total cholesterol. Significantly decreased from the baseline value at day one post – infarction, the ratios at total cholesterol to HDL cholesterol and LDL cholesterol to HDL cholesterol remained unchanged.

The ratios of total cholesterol to HDL cholesterol and LDL cholesterol to HDL cholesterol that have been reported to correlate with the development of acute coronary events are > 4.3 and > 2.7 respectively.

In the study the mean (± standard deviation) total cholesterol to HDL cholesterol ratio was 4.30 ± 0.36 at day one. Post myocardial infarction and did not change significantly at day – 4 posts – infarction.

These findings suggest that the cholesterol ratios could be used to determine cholesterol risk in patients who experienced acute myocardial infarctions and may have an advantage in situations in which the absolute total and fractionated cholesterol levels are longer applicable because of the effect of the acute myocardial infarction. (Beyond 24 hours after the onset of acute myocardial infarction)[10].

The results observed in the studies of acute myocardial infarction on cholesterol and cholesterol ratios like Jackson R et al[10], chamsi-Pasha et al[11], Brugada R et al[6], Heldenberg D et al[7] and PyFe T et al[9] are consistent with the present study.

CONCLUSION

- Following acute myocardial infarction the total serum cholesterol level falls significantly and the triglyceride level rises significantly.
- Therefore measurement of absolute levels of serum cholesterol and triglycerides following acute myocardial infarction are not valid in risk assessment 24 hours after infarction.
- But acute myocardial infarction does not affect the cholesterol ratios. (Cholesterol / HDL and LDL / HDL cholesterol ratios) even 24 hours after infarction.
- Therefore following acute myocardial infarction, the cholesterol ratios are valid and very useful in risk assessment.
- Treatment for hypercholesterolemia may be done on the basis of these cholesterol ratios in situations in which absolute levels of serum cholesterol and triglycerides are not valid. (After 24 hours of onset of acute myocardial infarction).

SUMMARY

A study of 100 patients admitted with acute myocardial infarctions has revealed that, acute myocardial infarction significantly reduces the total serum cholesterol levels and increases the serum triglyceride levels. But the acute myocardial infarction has no significant effect on the cholesterol ratios (LDL cholesterol / HDL cholesterol ratios and cholesterol / HDL cholesterol ratio). So after 24 hours of acute myocardial infarction assessment of cholesterol ratios will be more appropriate than assessing total cholesterol levels.

KEY NOTES FOR MASTER CHART

Sl.No	-	Serial Number
IP No	-	In Patient number
SE Status	-	Socio Economic Status
dur’n	-	duration
hrs	-	hours
DM	-	Diabetes mellitus
HT	-	Hypertension
STEMI	-	ST Elevation myocardial infarction
NSTEMI	-	Non ST Elevation Myocardial Infarction
↑	-	Increased
MI	-	Myocardial Infarction
CHOL	-	Cholesterol
TGL	-	Triglyceride
HDL	-	High Density Lipoprotein
LDL	-	Low Density Lipoprotein

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PROFORMA

NAME :

AGE :

SEX :

OCCUPATION :

SOCIO ECONOMIC STATUS :

EDUCATIONAL STATUS :

DATE OF ADMISSION :

PRESENTING SYMPTOMS : Y(Yes) N(No)

1. Chest pain

Time of onset

2. Angina equivalents

(Specify)

PAST HISTORY

H/O CAHD

HT

DM

STROKE

HYPERLIPIDEMIA

PERSONAL HISTORY:

DIET

SMOKING

ALCOHOL

ORAL CONTRACEPTIVES

OBSTETRIC HISTORY

FAMILY HISTORY

GENERAL EXAMINATION

GENERAL APPEARANCE Comfortable

Anxious

Yes No

CYANOSIS

CLUBBING

PEDAL OEDEMA

ANAEMIA

COLD CLAMMY SKIN

VITAL SIGNS

Pulse	-	Rate
		Rhythm
		Volume
		Vessel wall thickness
		Peripheral pulses
BP	-	Upper limb
		Lower limb

CVS Examinations

1. JVP
2. Apical impulse
3. Parasternal Pulsations
4. Heart sounds – S_1 S_2 – Normal intensity

   (or)

   Muffled

   (or)

   Loud intensity
5. Additional heart sounds
6. Murmur
7. Pericardial Rub.

RS Examination : Crackles Wheeze

INVESTIGATIONS

ECG in 15 leads

Cardiac Enzyme CPK-MB 0 hours 6-12hours > 12 hours

SGOT

Hb% Blood Glucose

Urea

Serum Creatinine

Electrolytes

Sl.No	LIPID PROFILE	Within 24 hours of M 1	Day 4 post M 1
1.	Total Cholesterol		
2.	LDL cholesterol		
3.	HDL cholesterol		
4.	Triglycerides		
5.	Cholesterol / HDL ratio		
6.	LDL / HDL ratio		

Chest X- Ray PA view

ECHO CARDIOGRAPHY

MASTER CHART I

															DAY - 1 POST MI				DAY – 4 POST M I			
Sl. No	IP	Age	Sex	SE Status	Angina	DM	HT	Smoking	Obesity	FamiIly History	Stemi (or) Nstemi	Enzymes	Echo	Killip class	Total Chol	TGL	Cho/ HDL	LDL/ HDL	Total Chol	TGL	Cho/ HDL	LDL/ HDL
1	238614	65	M	L	3	-	-	+	-	-	S	↑	+	II	190	160	4 .1	2 .4	172	189	4 .3	2 .4
2	238619	68	M	L	½	+	-	-	+	-	S	↑	+	I	223	165	4 .1	2 .5	204	200	4 .3	2 .5
3	238677	45	M	M	½	-	-	-	-	-	N	↑	+	I	213	198	4 .0	2 .3	197	227	4 .1	2 .2
4	238742	56	F	L	½	-	+	-	+	+	S	↑	+	I	160	80	4 .4	3 .0	143	118	4 .7	3 .0
5	238509	48	M	L	1	+	+	+	-	+	S	↑	+	III	140	60	5 .4	3 .9	125	100	6 .2	4 .2
6	238622	52	M	L	1	-	-	+	-	-	N	↑	+	I	228	180	3 .8	2 .2	210	208	3 .8	2 .2
7	238755	65	M	M	½	-	+	+	+	-	S	↑	+	II	163	80	4 .6	3 .0	146	112	4 .8	3 .1
8	238756	70	M	L	½	+	-	-	+	+	S	↑	+	I	240	190	3 .9	2 .2	220	220	4 .0	2 .2
9	238905	70	M	M	1	-	-	+	-	-	N	↑	+	I	138	58	5 .3	3 .8	121	87	5 .5	3 .9
10	238915	58	M	L	¾	-	-	-	-	-	S	↑	+	II	187	121	4 .3	2 .7	171	150	4 .3	2 .6
11	239080	50	M	L	1	-	-	+	-	-	S	↑	+	I	163	82	4 .4	3 .0	146	113	4 .6	3 .0
12	239100	27	M	L	1	+	-	-	-	+	S	↑	+	I	138	60	5 .0	3 .5	120	81	5 .2	3 .6
13	239109	67	M	L	¾	-	+	-	-	-	S	↑	+	I	192	120	4 .1	2 .5	173	150	4 .2	2 .5
14	239183	53	F	L	½	-	+	-	-	-	N	↑	+	I	215	162	4 .0	2 .4	198	183	4 .2	2 .4
15	239198	69	F	M	1	+	-	-	-	-	S	↑	+	I	241	200	3 .8	2 .2	227	218	4 .0	2 .2
16	239223	70	F	L	2	-	+	-	-	-	S	↑	+	II	191	121	4 .1	2 .6	173	152	4 .1	2 .5
17	239284	65	M	L	2	-	-	+	-	-	S	↑	+	I	140	62	4 .8	3 .2	115	88	4 .8	3 .1
18	239295	46	M	L	1	+	+	+	+	+	S	↑	+	II	239	205	3 .9	2 .1	227	217	4 .0	2 .2
19	239340	68	F	L	2	-	+	-	-	-	S	↑	+	I	163	82	4 .4	2 .9	142	115	4 .5	2 .9
20	239391	60	M	L	1 ½	-	-	+	-	-	S	↑	+	II	213	160	4 .0	2 .4	198	183	4 .1	2 .4
21	239400	70	M	L	2	-	+	-	+	-	N	↑	+	I	138	58	5.0	3.5	113	98	4.9	3.2
22	239438	43	M	M	1	-	-	-	-	-	N	↑	+	I	164	80	4.4	3.0	142	110	4.4	2.9
23	239551	54	M	L	2 ½	+	-	-	+	-	S	↑	+	I	238	198	3.8	2.1	228	277	3.9	2.1
24	239626	55	M	M	1	-	+	-	-	-	S	↑	+	I	213	160	4.0	2.4	198	183	4.2	2.5
25	239640	67	F	L	2	-	+	-	-	-	S	↑	+	II	189	120	4.3	2.7	171	150	4.3	2.6

MASTER CHART II

															DAY - 1 POST MI				DAY – 4 POST M I			
Sl. No	IP	Age	Sex	SE Status	Angina	DM	HT	Smoking	Obesity	Family History	Stemi (or) Nstemi	Enzymes	Echo	Killip class	Total Chol	TGL	Cho/ HDL	LDL/ HDL	Total Chol	TGL	Cho/ HDL	LDL/ HDL
26	239776	65	M	M	1	-	-	-	-	-	S	↑	+	I	138	60	5 .3	3 .8	121	91	5 .4	3 .7
27	239935	75	M	L	1 ½	+	-	-	-	-	S	↑	+	III	240	200	3 .8	2 .1	222	230	3 .8	2 .1
28	240000	65	M	L	2	-	-	-	-	+	S	↑	+	I	162	80	4 .5	3 .1	144	108	4 .7	3 .1
29	240005	65	F	L	3 ½	-	-	-	+	-	S	↑	+	I	192	121	4 .2	2 .6	171	150	4 .2	2 .5
30	240025	70	M	L	1	+	+	+	-	-	S	↑	+	I	215	160	3 .9	2 .3	197	190	4 .0	2 .3
31	240073	49	M	L	1	-	-	+	-	-	S	↑	+	II	242	202	3 .8	2 .1	225	235	3 .8	2 .1
32	240090	76	F	M	2	+	-	-	-	-	S	↑	+	I	214	161	4 .0	2 .3	197	192	4 .0	2 .3
33	240255	40	M	L	1	-	+	+	-	-	N	↑	+	I	138	56	5 .1	3 .7	120	87	5 .5	3 .9
34	240316	67	M	L	½	+	-	-	-	-	S	↑	+	II	165	79	4 .5	3 .0	147	108	4 .7	3 .0
35	240447	75	M	L	½	-	+	-	-	-	N	↑	+	I	189	121	4 .1	2 .5	171	150	4 .2	2 .5
36	240484	56	M	M	1	+	-	+	-	-	N	↑	+	I	136	56	5 .3	3 .9	118	87	5 .8	4 .0
37	240720	45	M	L	2	-	-	-	-	-	S	↑	+	II	216	162	4 .0	2 .3	197	191	4 .1	2 .3
38	240842	86	M	L	3	-	-	-	-	-	S	↑	+	IV	163	80	4 .5	3 .1	145	110	4 .6	3 .0
39	240869	30	F	M	½	+	-	+	-	+	S	↑	+	I	187	118	4 .2	2 .7	169	151	4 .2	2 .6
40	241416	42	M	L	½	-	+	-	-	-	S	↑	+	I	238	200	3 .9	2 .2	227	217	4 .0	2 .2
41	241433	41	M	L	1	-	-	+	-	-	S	↑	+	II	163	81	4.5	3.0	142	115	4.5	2.9
42	241442	55	M	L	1	-	-	-	+	+	S	↑	+	II	135	60	4.9	3.3	113	90	4.9	3.2
43	241525	35	M	L	1 ½	-	+	+	-	+	S	↑	+	I	241	205	3.8	2.2	226	217	4.0	2.2
44	241775	62	M	L	1	+	-	-	-	-	N	↑	+	I	214	158	3.9	2.3	198	183	4.2	2.4
45	241794	60	M	M	1	-	+	-	-	-	S	↑	+	II	187	121	4.3	2.7	171	150	4.3	2.6
46	241899	32	M	L	2	-	-	+	-	-	S	↑	+	I	213	160	4.0	2.4	198	183	4.2	2.4
47	242439	80	F	L	1	-	-	-	-	-	N	↑	+	I	240	200	3.8	2.2	227	218	3.9	2.2
48	242440	65	M	L	1	+	-	-	-	-	S	↑	+	II	162	81	4.5	3.0	141	114	4.5	2.9
49	242579	56	F	L	2	-	+	-	-	-	S	↑	+	I	135	58	5.1	3.7	112	101	5.1	3.3
50	242736	54	M	L	2	-	-	-	-	-	S	↑	+	I	241	201	3.8	2.2	228	219	4.0	2.2

MASTER CHART III

															DAY - 1 POST MI				DAY – 4 POST M I			
Sl. No	IP	Age	Sex	SE Status	Angina	DM	HT	Smoking	Obesity	Famly History	Stemi (or) NsteMi	Enzymes	Echo	Killip class	Total Chol	TGL	Cho /HDL	LDL/ HDL	Total Chol	TGL	Chol /HDL	LDL/ HDL
51	242434	70	M	M	3	+	+	+	-	-	N	↑	+	I	138	51	4 .9	3 .4	112	90	4 .7	3 .0
52	243449	86	M	L	½	-	+	+	-	-	S	↑	+	II	162	80	4 .5	3 .0	142	116	4 .5	2 .8
53	243468	29	M	L	½	-	-	+	-	-	S	↑	+	I	188	119	4 .2	2 .6	171	150	4 .3	2 .6
54	244526	49	M	M	½	+	+	-	+	-	S	↑	+	II	242	200	3 .9	2 .2	228	218	4 .0	2 .2
55	243548	41	M	M	1	-	-	-	-	-	S	↑	+	I	215	159	4 .0	2 .4	198	184	4 .2	2 .4
56	243931	50	M	L	3	-	-	-	-	-	S	↑	+	II	161	93	4 .2	2 .7	151	126	4 .4	2 .7
57	244503	54	M	L	2	+	-	+	-	+	N	↑	+	I	152	65	4 .7	3 .3	132	103	4 .7	3 .0
58	244516	49	M	M	1	-	-	+	-	-	S	↑	+	I	206	171	3 .7	2 .1	189	172	4 .2	2 .5
59	245345	58	F	L	1	+	-	-	-	-	S	↑	+	II	189	121	4 .2	2 .6	170	148	4 .3	2 .6
60	245392	46	M	L	2	-	-	-	-	-	S	↑	+	I	224	171	4 .0	2 .4	206	194	4 .2	2 .4
61	245529	50	F	L	3	+	-	-	-	-	S	↑	+	II	183	114	4 .7	3 .0	166	145	4 .8	3 .0
62	245583	60	M	M	2	-	-	+	-	-	S	↑	+	I	173	104	5 .7	4 .0	155	134	6 .0	4 .1
63	245612	65	M	L	1	-	-	-	-	+	S	↑	+	I	203	134	3 .4	1 .9	185	164	3 .4	1 .9
64	245719	62	M	L	2	-	-	+	-	+	S	↑	+	II	183	114	4 .6	2 .9	165	144	4 .8	2 .9
65	245772	45	M	M	2	-	-	+	-	-	S	↑	+	I	187	118	4 .3	2 .7	169	118	4 .4	2 .7
66	246263	50	M	L	3	-	-	-	+	+	N	↑	+	I	218	149	2 .9	1 .5	201	179	2 .9	1 .4
67	246280	48	M	L	1	-	-	+	-	-	S	↑	+	I	176	108	7 .0	5 .0	158	137	6 .0	4 .7
68	246466	40	F	L	2	+	+	-	-	-	S	↑	+	I	200	131	3 .5	2 .0	182	161	3 .5	2 .0
69	246934	60	M	M	1	-	-	+	-	-	N	↑	+	I	195	125	3 .8	2 .3	177	156	3 .9	2 .3
70	347056	76	M	M	2	-	+	-	+	+	S	↑	+	III	191	122	4 .0	2 .4	173	152	4 .1	2 .4
71	246760	60	M	L	2	-	-	+	-	-	N	↑	+	I	171	93	4 .4	2 .9	152	127	4 .4	2 .7
72	247343	65	M	L	1	-	-	-	-	+	S	↑	+	II	175	99	4 .3	2 .8	157	132	4 .4	2 .8
73	247373	64	M	M	2	-	+	-	-	-	N	↑	+	I	201	139	4 .1	2 .5	185	166	4 .3	2 .5
74	248212	57	M	L	2	+	-	-	-	-	S	↑	+	I	186	117	4 .4	2 .8	168	147	4 .4	2 .8
75	248545	40	F	L	3 ½	-	-	-	-	-	S	↑	+	II	206	146	4 .1	2 .5	189	171	4 .2	2 .5

MASTER CHART IV

															DAY - 1 POST MI				DAY – 4 POST M I			
Sl. No	IP	Age	Sex	SE Status	Angina	DM	HT	Smoking	Obesity	Family History	Stemi (or) Nstemi	Enzymes	Echo	Killip class	Total Chol	TG	Chol /HDL	LDL/ HDL	Total Chol	TGL	Chol /HDL	LDL/ HDL
76	248555	47	M	L	1	-	+	-	-	-	S	↑	+	I	178	103	4 .4	2 .8	159	135	4 .4	2 .7
77	248998	60	M	M	1	+	+	+	+	-	S	↑	+	I	170	82	4 .3	2 .9	152	127	4 .3	2 .7
78	249035	70	M	L	1 ½	-	-	+	-	-	S	↑	+	II	208	145	4 .1	2 .5	189	172	4 .2	2 .5
79	249413	69	M	M	1	-	+	-	-	-	S	↑	+	I	202	140	4 .1	2 .4	184	166	4 .1	2 .4
80	249595	52	M	M	2	+	-	-	-	-	S	↑	+	I	187	118	4 .3	2 .7	169	148	4 .4	2 .7
81	249685	70	M	L	1	-	-	-	-	-	S	↑	+	II	152	65	4 .6	3 .0	132	105	4 .6	2 .9
82	249712	61	M	L	2	-	+	-	-	-	S	↑	+	II	224	173	4 .0	2 .4	208	193	4 .1	2 .4
83	249800	65	F	L	1	+	+	-	-	-	S	↑	+	I	162	78	4 .6	3 .0	142	115	4 .7	3 .0
84	249886	42	F	M	1 ½	+	-	-	-	-	S	↑	+	I	215	160	4 .0	2 .4	198	183	4 .2	2 .4
85	249974	65	M	L	1	-	-	+	-	-	S	↑	+	I	189	120	4 .1	2 .5	172	151	4 .1	2 .5
86	250542	47	M	L	1 ½	-	-	+	-	-	S	↑	+	II	205	146	4 .1	2 .5	189	171	4 .2	2 .5
87	250750	50	M	M	2	-	-	-	-	-	S	↑	+	III	227	180	4 .0	2 .3	212	202	4 .1	2 .3
88	251437	55	M	L	1	+	-	-	-	-	N	↑	+	I	162	79	4 .5	3 .0	142	115	4 .5	2 .9
89	251500	74	M	L	2	-	-	+	-	-	N	↑	+	I	170	92	4 .4	2 .9	151	127	4 .4	2 .8
90	251952	46	F	M	1	-	+	-	-	-	S	↑	+	I	206	146	4 .0	2 .5	190	172	4 .2	2 .5
91	252057	53	F	M	2	-	-	-	-	-	S	↑	+	II	214	159	4 .0	2 .4	206	146	4 .0	2 .4
92	252090	45	M	L	1	+	-	-	-	-	S	↑	+	II	162	78	4 .6	3 .0	142	114	4 .7	3 .0
93	252204	76	M	L	2	-	-	+	-	-	S	↑	+	III	214	160	4 .0	2 .4	199	183	4 .2	2 .4
94	252979	52	M	L	1	-	-	+	-	-	S	↑	+	I	201	139	4 .1	2 .5	183	167	4 .1	2 .5
95	253105	57	M	M	2	-	-	-	-	-	S	↑	+	I	186	117	4 .3	2 .8	168	147	4 .4	2 .7
96	253110	45	M	L	1	-	-	+	-	-	S	↑	+	I	198	135	4 .2	2 .6	181	163	4 .3	2 .6
97	253952	75	M	L	2	-	-	-	+	-	S	↑	+	I	175	98	4 .3	2 .7	155	132	4 .4	2 .7
98	255062	44	M	M	1	-	+	-	-	-	S	↑	+	I	187	118	4 .3	2 .7	169	148	4 .4	2 .7
99	255170	45	M	M	2	-	+	-	+	-	N	↑	+	I	164	79	4 .4	2 .9	139	113	4 .7	3 .0
100	255176	65	M	M	1 ¾	+	+	+	+	+	S	↑	+	III	242	202	3 .8	2 .1	227	219	3 .9	2 .2