Pro Index | Journal of Science in Medicine and Life
Volume: 2 Issue: 8 Year: 2024
ISSN: 2992-9202 | http://journals.proindex.uz

Correlation between C-Reactive Protein and Lipid Profile in Patients with Heart Disease

Munther Ammar Jassim, Omar Talal Adil, Yasser Ammar Muhammed, Zaid Mahmood Shaker

Introduction

Cardiovascular disease is the leading cause of death worldwide. The number of cardiovascular deaths is higher than cancer deaths and more than any other cause of death [(1)]. The deaths reach up to 17.9million annually, which is equal to 31% of all deaths ([2]). In Iraq, the mortality rate due to cardiovascular diseases is about 33% of all deaths ([3]). Myocardial infarction is a type of coronary heart disease (CHD), that results from rupture of an arterial sclerosis plaque and a thrombus that works to block the coronary artery, which supply the heart, causing necrosis and death of part of the heart muscle ([4]). Internationally, it was reported that at least one American develops myocardial infarction approximately every 40 minutes ([1]). In the past, it was based solely on the theory of lipids role in the etiology of atherosclerosis. Currently, inflammation has been stated to play an essential role in developing atherosclerosis. Monocytes, macrophages, lymphocytes and smooth muscle cells accumulates release many cytokines or inflammatory biomarkers ([5]) The underlying cause of a heart attack due to MI is atherosclerotic, which is caused by the development of layers of fatty deposits on the inner walls of the arteries ([6]). The development of these deposits leads to narrowing and gradual blockage of blood flow in the artery. It is a long-term disease that progresses slowly ([7]).

Heart disease describes a range of conditions that affect the heart. Diseases under the umbrella term heart disease include:

1. CORONARY ARTERY DISEASE OR CAD

CAD is the most common form of heart disease. A hardening or narrowing of the arteries that lead to the heart characterizes this condition.

2. HEART ATTACK OR MYOCARDIAL INFARCTION

Unfortunately, for many people, a heart attack may be the first symptom of heart disease. A heart attack happens when the arteries leading to the heart become blocked, disrupting blood flow.

3. HEART FAILURE, OTHERWISE KNOWN AS CONGESTIVE HEART FAILURE

This condition occurs when stiffness in the heart prevents the organ from pumping blood adequately through the body.

4. HEART VALVE DISEASE

Valve disease happens when any of the four valves in the heart don’t open or close properly and interrupt blood flow. If the defect in the valve happens at birth, it’s called congenital heart disease.

5. HEART MUSCLE DISEASE OR CARDIOMYOPATHY

This condition can lead to heart failure. It occurs when the heart muscle becomes larger and stiffens, preventing it from pumping blood away from the heart. Sometimes blood can pool in the lungs.

6. ABNORMAL HEART RHYTHMS OR ARRHYTHMIA

This condition causes a fluctuation in the heartbeat that happens while at rest. If untreated, arrhythmia can be life-threatening.

Risk factors of heart disease

The most important behavioral risk factors leading to heart disease are:

- ✓ Obesity
- ✓ Diabetes Mellitus

Hypertension

- ✓ Sedentary life

Emotional stress

- ✓ Unhealthy food habit
- ✓ Smoking, and alcohol consumption
- ✓ High blood cholesterol and fat levels

It has been stated that chronic inflammation has a role in increasing the risk of cardiovascular disease, especially psoriatic arthritis and rheumatoid arthritis ([8]). Moreover, the statement of bacterial and viral infections participation in the process of atherosclerosis development, justifies mentioning that targeting infections with antibiotics can be promising as being a part of a protocol option to reduce atherosclerosis.

(9)

Diganosis of acute myocardial infarction

AMI sometimes take time unrecognized since the signs appear in a funny way. Sometimes some people become reluctant to seek medical advice since they think the pain they are feeling is due to something else. Sometimes the patient might just experience chest pains which might or might not be due to AMI and even these pains might be accompanied with heartburns or gallbladder problems. In order to identify early the main cause of the above, diagnostic process is a very important exercise to be carried out as soon as possible since by delaying medical attention; patients are at much high risk of permanent heart damage and death. As soon as possible the diagnostic process should be carried out. There are several techniques that can be used for AMI diagnosis. ([10]) .They are:

Patient history:

Present and past medical history, about medicines, physical examination, Inspection: may find cyanosis, anxiety, shortness of breath, auscultation: may find presystolic gallop and murmur sound or pericardial friction rub.

Laboratory test:

Blood and serum enzyme test: In this WBC count is done. It shows the inflammation due to myocardial necrosis, increased ESR, increased ESR rapidly, increased creatinine level, increased LDH (lactic dehydrogenase), increased SGOT level,and lipid profile :

1- **Cholesterol**

The main sterol in human and animals , it found in all tissue and blood . (contain 27 carbon atoms)
Source of cholesterol :-

1- Exogenous (dietary cholesterol)

2- Endogenous (synthesized cholesterol)

Cholesterol synthesized mainly in the liver. Acetyl CoA is the precursor of cholesterol synthesis,synthesized cholesterol transported from the liver to extrahepatic tissue by VLDL . Main function of cholesterol;

1. Synthesis of bile acids

2. Synthesis of Vit. D .
3. Synthesis of steroid hormone .
4. Incorporated to cell membrane.

2-T.G.:

is the main lipid in the diet. which are consisting of one glycerol molecule bonded with three fatty acid molecules. The bonds between the molecules are covalent and are called Ester bonds. The average daily intake of lipids in adults is about 81 g, of which more than 90% is normally triacylglycerol (TAG, formerly called triglyceride). The remainder of the dietary lipids consists primarily of cholesterol, cholesteryl esters, phospholipids, and un esterified ("free") fatty acids.

3-Very low-density lipoprotein (VLDL): also called Pre-B lipoprotein. Synthesized in liver.

Function; deliver de-novo TG (endogenous lipids) from liver to peripheral tissues (mainly muscle and adipose tissue).

4-Lows density lipoprotein (LDL): also called B-lipoprotein. Function: deliver fat and cholesterol to the peripheral tissues. Also regulate the level of cholesterol in blood . This is called 'bad cholesterol .

5-High-density lipoprotein (HDL) also called α -lipoprotein. Synthesized in liver. Function; deliver cholesterol from tissues to the liver for elimination.

This is also called 'good cholesterol', because it carries cholesterol away from the tissues to the liver, lowering blood cholesterol levels. High HDL levels are associated with lowered risk of cardiovascular disease. HDL levels are higher with exercise, higher estrogen levels, and weight loss.

ECG (electrocardiogram) ST-T wave changes with evolution Q waves, examine Troponin, echocardiography (imaging technique),magnetic resonance (MRI).

MI is one of the progressive and severe disease conditions whichare associated with high morbidity and mortality. We can see even though the current medical and surgical treatment has reduced the mortality rate but the prognosis is still poor for MI patients.

The chief goal of AMI treatment is to restore the backflow of blood to the heart muscles which is done by quickly opening the blocked artery. When the artery is open it reduces the damage to heart muscles and thus reducing the pain. This kind of treatment offers the patient a feeling of relief because when the patient is in great pain there is a tendency of becoming discouraged and loses the meaning of living. If this opening of the artery (reperfusion) is done within first four to six hours of heart attack it is of great importance to the patient .

AMI is associated with profound systemic inflammatory response including elevated levels of circulating inflammatory mediators and activation of peripheral leukocytes and platelets. The excessive inflammatory response in AMI could be caused by a deregulated immune system.([11]) C-reactive protein (CRP) is a phylogenetically highly conserved plasma protein that participates in the systemic response to inflammation and its plasma concentration increases during inflammatory states, a characteristic that has long been employed for clinical purposes.([12]) Tissue necrosis is a potent acute phase stimulus, and following AMI, there is a major CRP response, the magnitude of which reflects the extent of myocardial necrosis · The present study was undertaken with aim to investigate lipid profile in AMI patients and correlate it with inflammatory marker i.e. CRP.

CRP and heart disease

Inflammation plays an essential role in the development, progression, and prognosis of coronary artery disease (CAD), and has been widely concerned as an independent risk factor for the development of CAD · It is involved in many steps of atherosclerosis, and the activation of inflammatory responses may be a major contributor to plaque instability ([13]) The activation of proinflammatory cells and the upregulation of adhesion molecules increase the production of cytokines and procoagulant substances. Theses molecules promote the thickening or rupture of

atherosclerotic plaques and lead to the occurrence and development of acute coronary syndrome (ACS). C-Reactive Protein (CRP) is a liver protein whose synthesis depends on several transacting cytokines of interleukin-6 (IL-6), IL-1 and tumor necrosis factor α (TNF-α).[14] Measurement of CRP is useful for the detection and evaluation of infection, tissue injury, inflammatory disorders, and associated diseases.[15] High concentrations of CRP were shown in patients with metabolic disorders such as insulin resistance, obesity, lipid metabolism and unstable angina (UA).[14] It has been suggested that high levels of CRP are associated with adverse events in various cardiovascular diseases, including heart failure, ischemic stroke, atrial fibrillation, type 2 diabetes mellitus (T2DM), hypertension and myocardial infarction (MI). CRP levels are recognized as a strong independent risk marker for the identification of individuals at risk for future cardiovascular disease, which may be useful as an independent marker of prognosis for recurrent events in patients with stable coronary disease or ACS. In this study, we investigate the relationship between CRP levels and severity of MI in patients .

AMI is a multifactorial disease and impaired lipid metabolism is one of the crucial factors in the development of this disease . During tissue necrosis, as seen in AMI, alteration in lipid profile is encountered.[16] Kumar et al observed significantly higher total cholesterol (TC) and triglyceride (TG) levels and lower high-density lipoprotein cholesterol (HDL) levels in AMI patients . Woo et al observed higher mean TC, LDL, and TG as well as lower mean HDL in AMI patients; high HDL was among the protective factors. It is well known fact that early treatment of hyperlipidemia following acute myocardial infarction (AMI) provides potential benefits and reduces the morbidity and mortality of coronary heart disease .[17]

Obesity is defined as having an excess of body weight. A World Health Organization (WHO) release defined obesity as a chronic disease increasing globally replacing traditional health concerns [18]. It is directly related to cardiovascular problems and children whose parents are cardiovascular patients tend to have higher weight in the childhood and develop obesity as adults . Coronary heart disease (CHD) has been estimated to become the leading cause of death in developing countries by 2020 [19]. More than 80% of global CHD burden occurs in low income countries but the knowledge regarding important risk factors is derived largely from developed countries.

Blood lipid levels are modifiable risk factors for atherosclerosis and CHD. cholesterol, cholesterol esters, triglycerides and phospholipids are transported to the other tissues in the form of lipoproteins. Major classes of lipoproteins are chylomicrons (CM), low density lipoproteins (LDL) and high density lipoproteins (HDL), named by the site of their assembly and type of lipid and apo protein they have [20].

It has been observed that many lipid/lipoprotein abnormalities are prevalent in obesity and heart problems, collectively termed as dyslipidemia, however, these dyslipidemias are often hyperlipidemia where in majority of lipids are shifted towards the upper limits of range or higher than the range.

When is a C-reactive protein test performed?

C-reactive protein (CRP) analysis is performed when inflammation in the body is suspected or when there is a need to evaluate the severity of inflammation, as follows:

- Diagnosis of inflammatory diseases such as rheumatoid arthritis, ulcerative colitis, sinusitis, hepatitis, and dermatitis.
- Monitoring how the patient responds to treatment, especially in cases of chronic diseases accompanied by infections such as diabetes, arthritis, and cardiovascular diseases.
- Evaluate the severity of inflammation in cases of acute infections such as pneumonia, urinary tract infection, dermatitis, and surgical infections.
- CRP may also be ordered as part of routine screening to monitor general health, especially for people with risk factors for chronic diseases such as obesity, high blood pressure, and smoking.

The importance of C-reactive protein analysis

CRP analysis helps doctors determine the severity of the condition and make appropriate medical decisions, as it helps in the following:

- Estimating the severity of inflammation in the body, as the CRP level rises rapidly in cases of acute and chronic inflammation.
- Diagnosis of many inflammatory diseases such as rheumatoid arthritis, ulcerative colitis, pneumonia, arthritis, and kidney infection.
- Monitor how the patient responds to treatment, as the CRP level can decrease significantly after effective treatment of inflammation.
- It is part of a routine general health assessment, especially for people with risk factors for chronic diseases such as diabetes and cardiovascular disease.

Diseases associated with high C-reactive protein

There are several diseases associated with high C-reactive protein (CRP) in the blood, according to the health line website, including:

- Rheumatoid arthritis is a type of autoimmune disease that leads to inflammation and destruction of the joints.
- Ulcerative colitis is a type of gastrointestinal disease that causes inflammation and ulceration of the intestine.
- Cancerous diseases such as lung cancer and liver cancer.
- Viral and chronic hepatitis such as hepatitis C virus or non-alcoholic steatohepatitis.
- Upper and lower respiratory tract infections such as bronchitis and pneumonia.
- Osteoarthritis and psoriatic arthritis.

Aim of the study;

To evaluate the relationship between C-reactive protein and lipid profile in patients with heart disease (MI).

Materials and Methods

2.1. Chemicals

The specific chemicals used in this work are listed in table 2-1 with their suppliers.

Table 2-1. Chemicals and reagents with their suppliers

Chemicals	Suppliers
Cholesterol kit	Human, Germany
Triglyceride kit	Human, Germany
HDL-Cholesterol kit	Human, Germany
CRP kit	

2.2 Instruments

The instruments used in this work are listed in table 2-2.

Table 2-2. Instrument with their suppliers.

Instrument	Suppliers
Water bath	Gemmy, YCW-01 Taiwan
Centrifuge	Hettich GmbH & Co. RG, Germany
Spectrophotometer	APEl Co. LTD, PD-303, Japan

2.3 Patients selection and blood sampling

The study included 25 subjects (both sex) with MI of age group 35-75 years. Out of them 25 were normal healthy individuals and they formed the normal control group. 5ml of fasting venous blood sample was taken from AMI patients and controls . Blood sample was collected in plain vial and incubated at 37˚C for 30 minutes. After incubation, clot was removed and remaining sample was taken in centrifuge test tube. Samples were centrifuged at 3000rpm for 10 to 20 minutes. Supernatant was collected in clean and dry test tube for analysis of lipid profile and inflammatory marker. Lipid parameters and inflammatory marker (CRP)were estimated .

2.4 Analytical methods and procedures

2.4.1 Determination of total serum cholesterol (TC)

Total serum cholesterol was determined utilizing a ready made laboratory kit for this purpose; the principle of determination was based on the enzymatic hydrolysis according to the following reaction ;

$chelesterol\ esterase$

Cholesterol ester → Cholesterol $+fatty\ acids$

$chelesterol\ oxidase$

Cholesterol → $Cholest{-}4{-}en{-}3{-}one{+}H_2O_2$

$peroxidase$

$2H_2O_2 + phenol{+}4{-}aminoantipyrine \rightarrow quinoneimine{+}4H_2O_2$ Quinoneimine, a red complex absorbing light at 500nm.

Reagent

Reagent type	Material	Concentration
Reagent (1) Buffer	Phosphte buffer Phenol Sodium cholate surfactant	100 mmol / L 5 mmol/L 23 mmol/L 1.5 mmol / L
Reagent (2) Enzymes	4-aminoantipyrine Peroxidase Cholesterol oxidase Cholesterol esterase	0.25 mmol/L ≥ 1200 Iu/L ≥ 100 I u/L ≥170 Iu/L
Standard	Cholesterol	200 mg/dL (5.17 mmol / L)

Procedure

Working solution: The contents of one vial of reagent 2 was reconstituted with the contents of one vial of reagent 1, mixed well by inventing and store in the reagent 1 bottle.

	Blank	Standard	Test
Standard	---	10 µl	--
Serum	---	---	10 µI
D.W	10 µl	--	--
Working solution	1 ml	1 ml	1 ml

Mix and measure after incubation at 37 ^{0}C for 5 min, adjust to zero by blank then read at 500 nm.

Concentration of cholesterol (mg/dl) = $\frac{Atest}{Astandard} x\ 200$

N.V = 150-250 mg /dl

2.4.2 Determination of serum triglycerides (TG)

Some triglycerides were determined utilizing a ready mode laboratory kit for this purpose.

Principle determined based on the enzymatic hydrolysis according to the following reaction ·

$$Triglycerides \xrightarrow{lipase} Glycerol + fatty\ acids$$

$$Glycerol + ATP \xrightarrow{Glycerokinase} Glycerol{-}3{-}phosphate + ADP$$

$$Glycerol\text{-}3\text{–}phosphate \xrightarrow[peroxidose]{Glycerol\text{-}3\text{-}phosphate\ oxiase} dihydroxy\ acetone\ phosphate + H_2O_2$$

$$2H_2O_2 + parachlorophenol + 4{-}amino\ antipyrine \rightarrow quinoneimine + 4H_2O_2$$

The intensity of the color formed is proportional to the triglycerides concentration in the simple .

Reagents

Reagent type	Material	Concentration
Reagent (1) Standard	Glycerol equivalent to 200(mg/dL) of TG (µw=875)	2.29 mmol/L
Reagent (2) Buffer	Tris buffer Parachlorophenol	50 mmol, pH 7.5 2 mmol/L
Reagent (2) Enzymes	4-amino antipyrine Lipase Glycerokinase Glycerol-3-phosphate oxidase Peroxidase ATP	0.1 mmol/L 150 000 u/L 500 u/L 2500 u/L 440 u/L 0.1 mmol/L

Procedure

Working solution: One bottle of reagent (3) was reconstituted with one bottle of reagent (2). Mixed well by inventing and store in the reagent 3 bottle.

	Blank	Standard	Test
Standard	---	10 µl	--
Serum	---	---	10 µI
D.W	10 µl	--	--
Working solution	1 ml	1 ml	1 ml

Mix and incubate for 5 min at t 37 ^{0}C; adjust to zero by blank and measure light absorbance at 500 nm.

Calculation Concentration of cholesterol (mg/dl) = $\frac{Atest}{Astandard} x\ 200$

N.V = 65-180 mg /dl

2.4.3 Determination of serum HDLc

The chylomicrons and lipoproteins of VLDL, and LDL contained in the serum sample were precipitates by the addition of 4% phosphotungstic acid solution, which contain 10% magnesium chloride pH 6.2. The supernatant obtained after centrifugation contains the HDL, from which the cholesterol can be determined by complementary kit used in determination of total serum cholesterol as described in .

Reagents:

Reagent type	Material	Concentration
Reagent (1) Precipitating agent	Phosphotungstic acid $MgCl_2$	0.55 mmol/L 25 mmol/L
Reagent (2) Standard	Cholesterol	50 mg/dl or 1.29 mmol/L
Reagent (2) Cholesterol enzymatic working solution		

Procedure

Serum 200 µl

Reagent (precipitant) 500 µl

	Blank	Standard	Test
D.W	0.05 ml	---	---
Reagent(2)	---	0.05 ml	---
Supernatant			0.05 ml
Cholesterol enzymatic working solution	1 ml	1 ml	1 ml

Mix and incubate for 5 min in 37 ^{0}C; adjust to zero by blank, and then measure light absorbance at 500 nm.

Calculation Concentration of HDLc (mg/dl) = $\frac{A\ test}{A\ standard} x conc\ of\ st.$

N.V = 35-65 mg/dl

2.4.4. Determination of serum LDL-cholesterol

LDL-cholesterol was estimated in directly by the use of friedewald formula [21]:

LDLc = TC - HDLC-VLDLc

= TC - (HDLc+VLDLc)

and VLDLc was calculated as: Concenteration of VLDLc (mg/dl) = $\frac{TG}{}$

N.V of LDL = 65-160 mg /dl

N.V of VLDL= 25-50 mg /dl

2.4.4. Determination of serum CRP

The C-Reactive Protein test is based on the principle of the latex agglutination. When latex particles complexed human anti-CRP are mixed with a patient's serum containing C reactive proteins, an visible agglutination reaction will take place within 2 minutes.

Materials required;

Materials required to perform CRP assay depend on the type of method used. These laboratory equipment and supplies are necessary to detect CRP levels using the agglutination test method.

Serum sample, CRP reagent,Positive and negative control ,Glass slide with six reaction circle,Mixing sticks ,Test tube,Saline.

Procedure

In this post, we have outlined the procedure of latex agglutination test both for qualitative assay as well as a semi-quantitative assay.

Semi-quantitative Method

- Prepare the serial dilution of samples 1:2, 1:4, 1:8, 1:16, 1:32, 1:64, and so on by using normal saline.
- Pipette each dilution of the serum sample and control (positive control and negative control) on to separate reaction circle.
- Add one drop of CRP latex reagent to each drop of diluted serum (sample) and controls.
- Using the mixing stick mix the sample and latex reagent uniformly over the entire circle.
- Immediately start a stopwatch, and rock the slide gently back and forth (120 rpm/minute) for 2 minutes.
- Observe the clump (agglutination).

Calculation:

The approximate CRP concentration in the patient sample is calculated as follows: Sensitivity (**Indicated on the label of the latex vial) (6mg /l) x CRP Titer = mg/L**

Dilution	(mg/L)
1:1	6
1:2	12
1:4	24
1:8	48
1:16	96
1:32	192

N.V = <10 mg /L normal

>10 mg / L high

Results

Table 1 and figure 1 shows the mean levels of TC, TG, HDL-C, LDL-C, VLDL-C , CRP and BMI in AMI cases and controls. The mean levels of TC, TG, LDL-C, VLDL-C and CRP were increased in AMI cases as compared to controls and were statistically highly significant ($p<0.01$) whereas mean level of HDL-C was highly significantly decreased in AMI cases as compared to controls ($p<0.01$).

Table 2 shows correlations of CRP with lipid profile,and BMI. CRP was positively correlated with total cholesterol, triglyceride, LDL and VLDL in AMI patients and was statistically highly significant where as there was significant negative correlation between CRP and HDL cholesterol.

Table 1. Mean levels of lipid profile,CRP ,and BMI in healthy subjects and AMI patients

groups	cholesterol	Triglyceride	HDL	VLDL	LDL	CRP	BMI
Healthy subjects	165±15.1[a]	91±24.6[a]	49±5.3[a]	21±6.7[a]	99±17.9[a]	2.9±1.3[a]	21±1.4[a]
patients	293±55.1[b]	230±49.9[b]	31±11.3[b]	40±9.6[b]	219±61.5[b]	13.9±4.1[b]	25±3.8[b]

Values represent mean ±SD; values with non-identical superscripts (a,b) indicated significant differences between groups (P<0. 01).

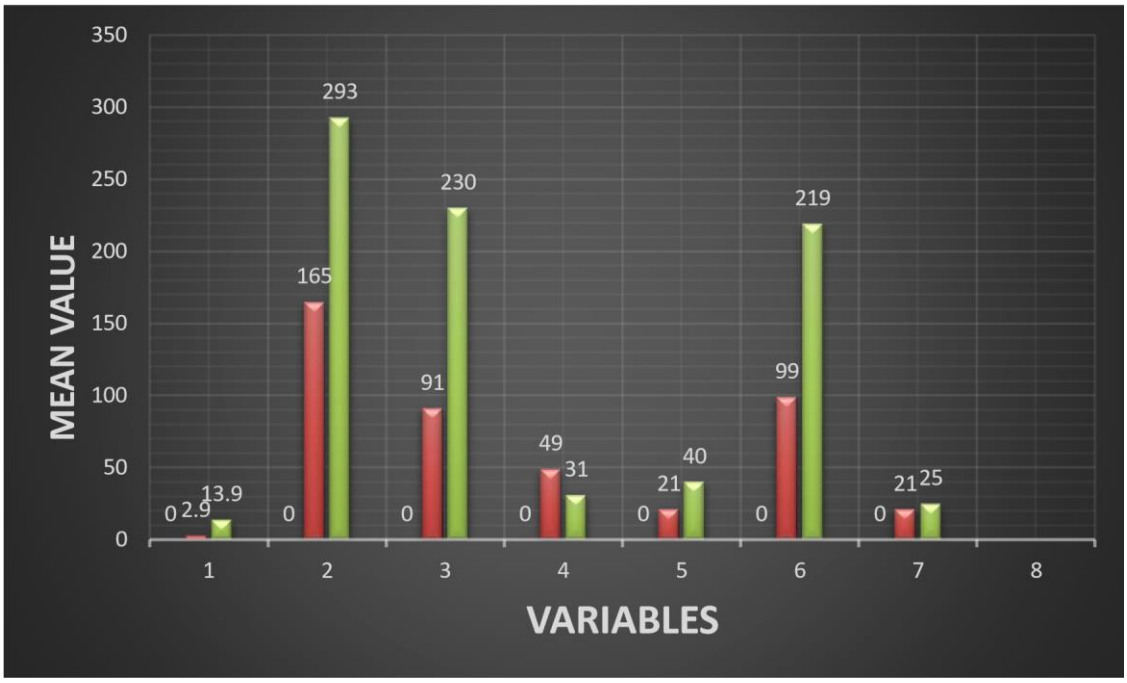

Figure 1: The status of lipid profile , CRP,and BMI in AMI cases and control.

(1=CRP , 2=Chol , 3 = TG ,4=HDL 5= 6= LDL , 7 = BMI)

Table 2; shows correlations of CRP with lipid profile,and BMI.

parameters	r value	P value
TC	0.596**	0. 01
TG	0.497*	0.05
HDL	-0.697**	0.01
VLDL	0.324*	0.05
LDL	0.584**	0. 01
BMI	0.16	0.11

Correlation** is significantat at the 0.01 level, Correlation* is significantat at the 0.05 level, TC=Total Cholesterol, TG=Triglyceride, HDL-C=High density lipoprotein cholesterol, LDL-C=Low density lipoprotein cholesterol, VLDL-C=very low density lipoprotein cholesterol, CRP= C-reactive protein,BMI=body mass index

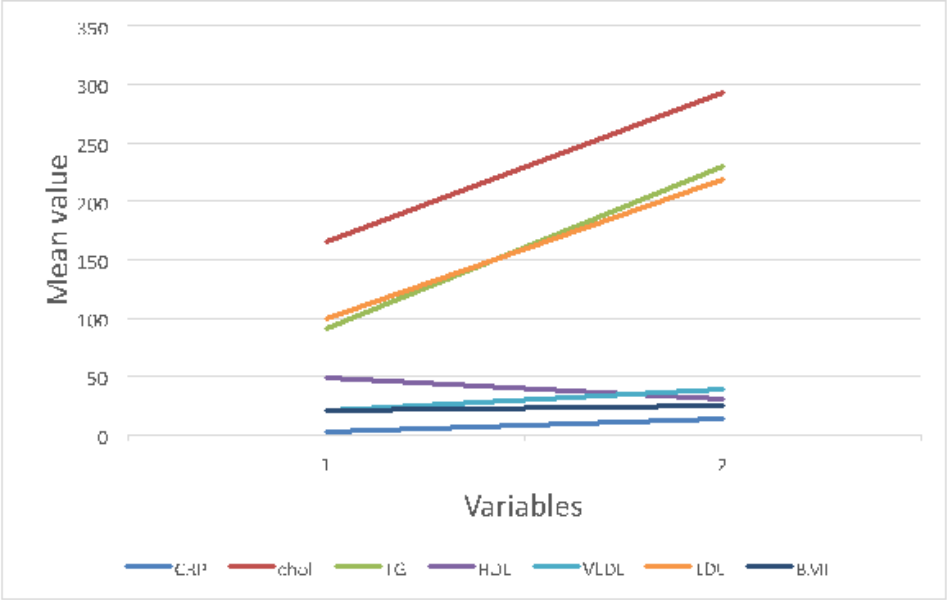

Figure 2. Correlation between status of lipid profile levels , CRP and BMI In AMI patients

Discussion

In this study, lipid parameters in AMI subjects were studied and correlated with inflammatory marker. In our study, we found significant increased levels of TC, TG, LDL, VLDL and significant decreased level of HDL in AMI cases as compared to controls ($p<0.01$). Kumar et al in their study observed significantly higher total cholesterol (TC) and triglyceride (TG) levels and lower high-density lipoprotein cholesterol (HDL) levels in AMI patients, which are in agreement with our study.[22] In agreement with the present study, Woo et al also observed higher mean TC, LDL and TG as well as lower mean HDL in AMI patients.[23]Similar results have been also observed by Shirafkan et al who also found higher levels of TC, LDL, VLDL and TG as well as lower levels of HDL in AMI patients.[24]

LDL carries the most of cholesterol in the plasma and increasing of LDL depends on increasing of total cholesterol level.[25] Nigam et al reported increasing trend of triglyceride after myocardial infarction with a significant increase on day three and predischarge as compared to day one[26] There is a different mechanism about elevation of triglycerides after AMI. It has been reported that elevated triglyceride levels may depend on genetic basis and nutritional habits.[27] The levels of triglyceride may also change because of inherited abnormality of very low density lipoprotein. It may happen because of elevated flux of fatty acids and impaired elimination of VLDL from the plasma.[26] Epidemiologically high serum levels of HDL are associated with reduced risk for the development of atherosclerotic disease. HDL particles are believed to be antiatherogenic, secondary to their capacity to drive reverse cholesterol transport and antagonize pathways of inflammation, thrombosis, and oxidation[28] Al Aqeel et al., observed that HDL appears to be the main lipid risk factor in patients of AMI, suggesting that primary prevention strategies should focus on treatment modalities that increase HDL.[29]

AMI is a multifactorial disease, in which inflammatory processes play a central role.28 In this regard, CRP is considered to be the most important marker and it has been extensively studied in recent years.[30] In our study, we found significant increased mean level of CRP in AMI cases as compared to controls. This is in agreement with the study done by Sesani et al who also reported increased mean level of CRP in AMI patients as compared to controls. Kausadikar et al showe elevated C-reactive protein in patients who present with an anterior wall myocardial infarction reflecting the greater amount of myocardial damage .[31]

In the present study, we found strong significant positive correlation of serum CRP levels with total cholesterol, triglyceride, LDL and VLDL in AMI patients. In the other hand, we found significant negative correlation of serum CRP levels with HDL-cholesterol. Khan et al in their study found significant inverse correlation between CRP and HDL in AMI patients.7 The findings of our study support the fact that there occurs co-existence of inflammation and impaired lipid metabolism.Also we found weak significant positive correlation of serum CRP levels with BMI.

Conclusion

In present study, we found alterations in the lipid profile and inflammatory marker in AMI cases, which play significant role in incidence of AMI. Hence, all the people should undergo regular checkup including lipid profile evaluation and inflammatory marker such as CRP to decrease the incidence, morbidity and mortality from the disease.

References

1. American Heart Association. Heart Disease and Stroke Statistics—2019 at a Glance. (2019).
2. Wierer, M., Prestel, M., Schiller, H. B., Yan, G., Schaab, C., Azghandi, S., ... and Aherrahrou, Z.. Compartment-resolved proteomic analysis of mouse aorta during atherosclerotic plaque formation reveals osteoclast-specific protein expression. Molecular and Cellular Proteomics, (2018);17 (2), 321-334.
3. Turk-Adawi, K., Sarrafzadegan, N., Fadhil, I., Taubert, K., Sadeghi, M., Wenger, N. K., ... and Grace, S. L.. Cardiovascular disease in the Eastern Mediterranean region: epidemiology and risk factor burden. *Nature Reviews Cardiology*, (2018);*15* (2), 106.
4. Rathore, V., Singh, N., Rastogi, P., and Mahat, R. K.. Correlation of Inflammatory Marker with Glycogen Phosphorylase BB (GPBB) in Patients of Acute Myocardial Infarction. (2017).
5. Beverly, J. K., and Budoff, M. J.. Atherosclerosis: Pathophysiology of insulin resistance, hyperglycemia, hyperlipidemia, and inflammation. (2019).
6. Amirfakhryan, H.. Vaccination against atherosclerosis: An overview. Hellenic Journal of Cardiology. (2019)
7. Lacy, M., Atzler, D., Liu, R., de Winther, M., Weber, C., and Lutgens, E.. Interactions between dyslipidemia and the immune system and their relevance as putative therapeutic targets in atherosclerosis. Pharmacology and therapeutics, (2019), 193, 50-62.
8. Groh, L., Keating, S. T., Joosten, L. A., Netea, M. G., and Riksen, N. P.). Monocyte and macrophage immunometabolism in atherosclerosis. In Seminars in immunopathology, (2018, February); (Vol. 40, No. 2, pp. 203-214). Springer Berlin Heidelberg.
9. Moriya, J.. Critical roles of inflammation in atherosclerosis. Journal of cardiology, (2019); 73 (1), 22-27.
10. Horne R, James D, Petrie K, Weinman J, Vincent R.. Patients' interpretation of symptoms as a cause of delay in reaching hospital during acute myocardial infarction, 2000; 388–393.
11. Fang L, Moore XL, Dart AM, Wang LM. Systemic inflammatory response following acute myocardial infarction. J Geriatr Cardiol. 2015;12(3):305-12.
12. Black S, Kushner I, Samols D. C-reactive Protein. J Biol Chem. 2004;279(47):48487-90.
13. Bouzidi N, Betbout F, Maatouk F. Relationship of activin A levels with clinical presentation, extent, and severity of coronary artery disease. Anatol J Cardiol 2017; 18: 402–409.
14. Kaur R, Matharoo K, Sharma R, et al. C-reactive protein + 1059 GNC polymorphism in type 2 diabetes and coronary artery disease patients. Meta Gene 2013; 1: 82–92.
15. Stefanutti C, Mazza F, Steiner M, et al. Relationship between sustained reductions in plasma lipid and lipoprotein concentrations with apheresis and plasma levels and mRNA expression of

PTX3 and plasma levels of hsCRP in Patients with HyperLp(a)lipoproteinemia. Mediators Inflamm , 2016: 4739512.

16. Brindha G, Padmavathi C, Muthukumar D. Lipid profile in myocardial infarction-controversies revisited. Int J Sci Res. 2016;5(4);53-4.
17. Balci B. The modification of serum lipids after acute coronary syndrome and importance in clinical practice. Curr Cardiol Rev. 2011;7:272-6
18. Javed A, Jumean M, Murad M, Okorodudu D, Kumar S, Somers V, et al. Diagnostic performance of body mass index to identify obesity as defined by body adiposity in children and adolescents: a systematic review and meta-analysis. Pediatric Obesity. 2014;10(3):234–244.
19. Organization WH. The double burden: emerging epidemics and persistent problems. Switzerland: The world health report; 1999. p. 221–7.
20. Chen J, Corbin I, Zhang Z, Zheng G. High-density lipoprotein-like peptide-phospholipid scaffold ('hpps') nanoparticles. Canada: US Google Patents; 2009.
21. Friedewald,W.T.,Levy,R.I.and Fredrickson,D.S.ClinChem 1972;81:499.
22. Kumar A, Nagtilak S, Sivakanesan R, Gunasekera S. Cardiovascular risk factors in elderly normolipidemic acute myocardial infarct patients-a case controlled study from India. Southeast Asian J Trop Med Public Health. 2009;40:581-92.
23. Woo J, Ho SC, Wong SL, Woo KS, Tse CY, Chan KK et al. Lipids, lipoproteins and other coronary risk factors in Chinese male survivors of myocardial infarction. Int J Cardiol. 1993;39:195-202
24. Shirafkan A, Marjani A, Zaker F. Serum lipid profiles in acute myocardial infarction patients in Gorgan. Biomed Res. 2012;23(1):119-24
25. More HV, Pujari KN, Jadkar SP, Patil CG. Biochemical Parameters in Acute Myocardial Infarction with or Without Co-Morbidities. J Med Sci Clin Res. 2017;5(2):17299-304
26. Nigam PK, Narain VS, Hasan M. Serum lipid profile in patients with acute myocardial infarction. Indian J Clin Biochem. 2004;19(1):67-70.
27. Watts GF, Jackson P, Mandalia S, Brunt JN, Lewis ES, Coltart DJ. Nutrient intake and progression of coronary artery disease. Am J Cardiol. 1994;73(5):328-32.
28. Davidson MH, Toth PP. High-density lipoprotein metabolism: potential therapeutic targets. Am J Cardiol. 2007;100(11A):32-40
29. Al Aqeel A, Mojiminiyi OA, Al Dashti R, Al Ozairi ES. Differences in physician compliance with guideline on lipid profile determination within 24 h after acute myocardial infarction. Med Princ Pract. 2005;14:41-5
30. Sesani S, Vijayabhaskar M, Madhulatha MI, Lokary V. Assessment of C-reactive protein in cases of acute myocardial infarction and its correlation with risk factors. Int J Sci Study. 2016;4(7):140-143.
31. Kausadikar SR, Mehra HA, Pathak KP. Study of C-reactive protein in patients with acute myocardial infarction attending tertiary care teaching hospital in Saurashtra region;2019.
32. C-reactive protein analysis...to detect infections in the body: Noura Allam, article published in Al-Ain Newspaper, Wednesday 5/8/2024.