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Current and futuristic scope of biomarkers in acute myocardial infarction

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Abstract

Introduction: Acute myocardial infarction is a leading cause of death throughout the world. Myocardial infarction is defined as myocardial cell death due to prolonged ischemia. Objectives of the study were to look for the role of inflammatory markers (CRP, IL-6, and plasma fibrinogen) in the prediction of myocardial infarction. In recent times, IL-6 and plasma fibrinogen are not established marker in acute myocardial infarction. However, there is another biomarker i.e. CRP that have shown additional value in improving sensitivity and prognostic information. Novel biomarkers have improved assessment of outcome in acute myocardial infarction, but none have been demonstrated to alter the outcome of a particular therapy or management strategy. Thus the finding of this study may help the clinician to develop more novel therapeutic strategies for the management of myocardial infarction (MI) patients. The outcome of the study will be very beneficial as well, to the researcher working in the concerned area in order to develop more focused research approach.

KeyWords: Myocardial infarction, Coronary heart disease, Biochemical Marker, IL-6, C- reactive protein, Plasma fibrinogen, Lipid profile

Introduction

Acute myocardial infarction (AMI) results in significant mortality and morbidity. AMI is usually diagnosed on the basis of prolonged chest pain (World Health Organization (WHO) criteria), ECG changes, and increases in the levels of biochemical markers of myocardial injury. Coronary heart disease is a chronic (or long-term) condition that affects many people. Coronary heart disease is when your coronary arteries (the arteries that supply blood and oxygen to your heart muscle) become clogged with fatty material called 'plaque' or 'atheroma'. Plaque gradually builds up on the internal partition of the arteries, causing them to turn into slender. This procedure is called 'atherosclerosis'. It starts when you are youthful and can be well superior by middle age. If your arteries develop into too narrow, the blood supply to your heart muscle is compact. This may show the way to symptoms such as angina. If a blood clot forms in the narrowed artery and entirely blocks the blood supply to part of the heart, it can reason a heart attack.

Definition and Types

The word acute myocardial infarction (AMI) should be use when nearby is confirmation of myocardial necrosis in a medical setting reliable among acute myocardial ischemia. In practice, the disarray is diagnose and assess on the beginning of medical assessment, the electrocardiogram (ECG), invasive, biochemical testing, and noninvasive imaging, and pathological evaluation. Beneath these circumstances whichever one of the after that criteria meets the diagnosis for MI: Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] by way of at least one value over the 99th percentile upper reference limit (URL) and with at slightest one of the following:

- Symptoms of ischemia.
- Novel or presumed innovative important ST-segment–T wave (ST–T) change or new left bundle branch block (LBBB).
- Enlargement of pathological Q waves in the ECG.
- Admiration of an intracoronary thrombus by angiography or autopsy.

In the past, a general consensus existed for the clinical syndrome designated as myocardial infarction. Inside studies of disease incidence, the World Health Organization (WHO) defined myocardial infarction from symptoms, ECG abnormalities, and cardiac enzymes. However, the development of ever extra sensitive and myocardial tissue-specific cardiac biomarkers and extra sensitive imaging techniques now allow for recognition of very small amounts of myocardial injury or necrosis. Moreover, the administration of patients with myocardial infarction has significantly improved, resulting in less myocardial injury and necrosis, in spite of a similar clinical presentation. Moreover, it appears necessary to distinguish the various conditions which may cause myocardial infarction, such as 'spontaneous' and 'procedure-related' myocardial infarction. Accordingly, physicians, other healthcare providers, and patients require an up-to-date definition of myocardial infarction.

Epidemiologic

Acute myocardial infarction is the most severe manifestation of coronary artery disease, which causes more than 2.4 million deaths in the USA, more than 4 million deaths in Europe and northern Asia, [2] and more than a third of deaths

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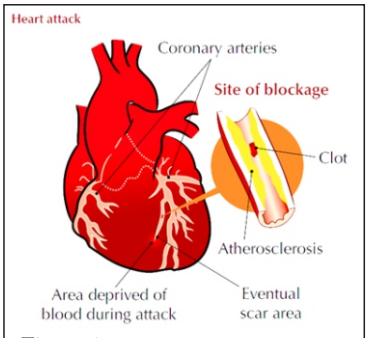

Figure 1: Diagram showing site of blockage causing Myocardial infarction (MI)

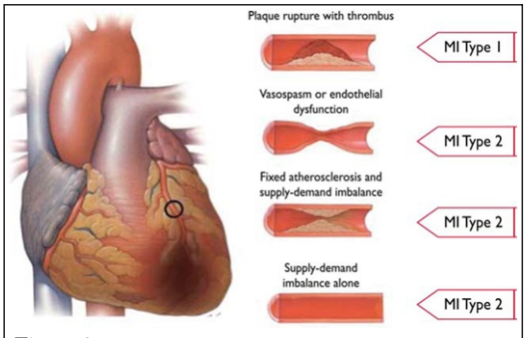

Figure 2: Discrimination among myocardial infarction (MI) types 1 and 2 according to the situation of the coronary arteries.

in developed nations annually[3]. Increased use of evidence-based therapies and lifestyle changes have spurred considerable reduction in death from coronary heart disease in recent decades. Though, myocardial infarction retains a substantial footprint on worldwide fitness, affecting more than 7 million persons universal each year. Concordantly, its financial impact is tremendous; in 2010, more than 1.1 million US hospitalizations were a result of myocardial infarction, by means of estimated direct costs of at slightest US$450 billion [4]. Biomarkers are natural molecules originate in blood and are use as markers of physiologic or pathologic processes taking place in the cadaver. The National Institutes of Health define a biomarker as "a characteristic that is objectively measured and evaluated as an indicator of a normal natural procedure and pathogenic process"[5]. In recent years, a large body of research has focused on the look for for biomarkers for early on discovery of myocardial cell injuries. The majority of these studies have evaluated patients presenting to emergency departments, underlining the need for a best biomarker for rapid gratitude of acute coronary syndrome (ACS).

The elevated prevalence of cardio metabolic risk factors between the population production the individuals prone to linked unprompted coronary artery [6]; Disorder might to be a risk factor for the progress of cardiovascular [7]. Atherosclerosis is now regarded as an inflammatory disease. Inflammation in the vessel wall seems to be significant for the development of the atherosclerotic plaque as well as for plaque destabilization [8,9]. Pro-inflammatory cytokines are thought to be involved in reperfusion injury, repair processes, and scar tissue formation after myocardial infarction [10]. Serum marker IL-6 levels are raised [11] and circulating levels of IL-6 and C-reactive protein (CRP) have been shown to correlate with infarct size[12]. inside disparity, an opposite association with earnings was noted for rates of acute myocardial infarction (1.92, 2.21, and 4.13 cases per 1000 person-years in high-, middle-, and low-income countries, respectively; P<0.001 for trend). Mitigation of the high burden of risk factors in higher-income countries was attributed to greater use of preventive measures and revascularization procedures.

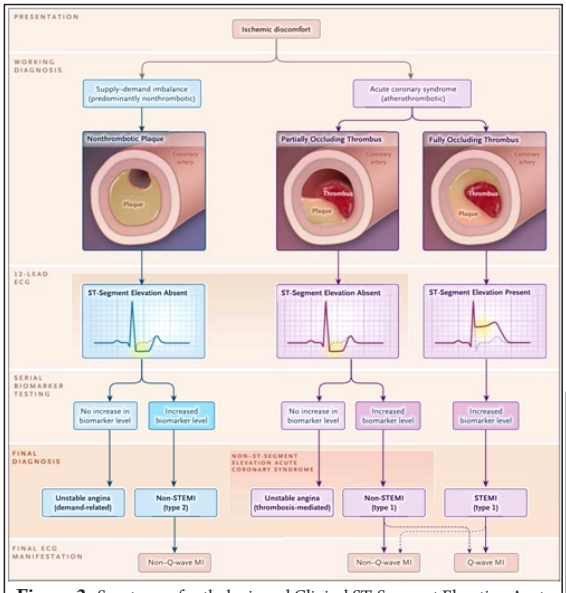

Figure 3: Spectrum of pathologic and Clinical ST-Segment Elevation Acute Myocardial Infarction (STEMI) and Non-STEMI Acute Coronary Syndromes [15] Adapted from Morrow, ECG electrocardiogram, and MI myocardial Infarction

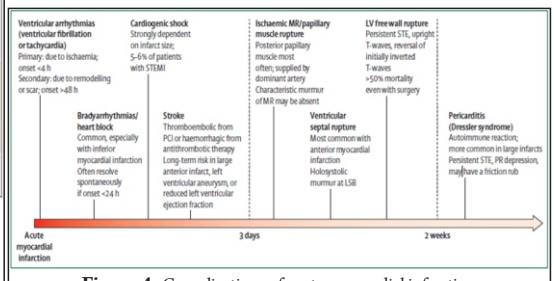

Figure 4: Complications of acute myocardial infarction

Pathophysiology

Acute myocardial infarction is divided into STEMI and NSTEMI.5 Unstable angina is also considered an acute coronary syndrome (ACS) because it is an imminent precursor to myocardial infarction. Unstable angina has a similar pathophysiology to NSTEMI, and they are together referred to as non-ST-segment elevation ACS (NSTE-ACS). They have traditionally been grouped together for management decisions. In most cases, myocardial infarction is due to disruption of a vulnerable atherosclerotic plaque or erosion of the coronary artery endothelium (type 1)[13,14]. A severe stenosis (ie, ≥70% diameter) is required to precipitate angina; however, such stenoses less commonly cause type 1 myocardial infarction, because they tend to have dense fibrotic caps that are less likely to rupture, and collateral circulation forms over time. By contrast, vulnerable plaques tend to have 30–50% stenosis, thin fibrous caps and contain more inflammatory cells such as lipid-laden macrophages [12-14]. Upon rupture, the plaque releases its thrombogenic contents, causing platelet activation, initiation of the coagulation cascade, mural thrombus formation, and embolization of atherosclerotic debris downstream. This hypercoagulable state could contribute to the rupture of additionally vulnerable fibroatheromas, and thus there can be more than one culprit lesion[14]. The end result is myocyte necrosis, detectable by elevation of cardiac biomarkers in the peripheral blood. The factors influencing the severity of ischemia include whether the vessel was partially or completely occluded, duration of occlusion, amount of myocardium supplied, the presence of collaterals, and the adequacy of reperfusion following treatment.

Complications from acute myocardial infarction

Knowledge of the cardinal features and timing of the complications of myocardial infarction is essential to recognize and properly treat these potentially fatal events. (Figure 4) Early complications result mainly from myocardial necrosis itself, while later complications reflect the

Table 1: Universal classification of myocardial infarction

Type 1: Spontaneous myocardial infarction
Unprompted myocardial infarction linked to atherosclerosis plaque rupture, ulceration, fissuring, erosion or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuring myocyte necrosis. The patients may have underlying severe CAD but on occasion non- observation or no CAD.
Type 2: Myocardial Infarction secondary to an ischemic imbalance
In illustration of myocardial injury by way of necrosis everywhere a condition erstwhile than CAD contributes to an between oxygen supply and /or demand, e.g. coronary artery coronary embolism, hypertension tachy-/brady-arrhythmiss, spasm, respiratory failure, anemia, coronary endothelial dysfunction, and hypertension with or without LVH.
Type 3: Myocardial infarction ensuing in death while biomarker values are unavailable
Cardiac death among symptoms indicative of myocardial ischemia and supposed new ischemia ECG changes or new LBBB, but death happening before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarker were not collected.
Type 4a: Myocardial infarction related to percutaneous coronary intervention (PCI)
Myocardial Infarction linked with PCI is arbitrarily identify by elevation of cTn value >5x99" percentile URL in patients with normal baseline value (<99"percentile URL) or a rise of cTn value 20% if the value are elevated and are stable or falling. In addition, either (i) symptoms suggestion of myocardial infarction ischemia or (ii) new ischemia ECG changes or new LBBB or (iii) angiographic loss of patency of a major coronary artery or a side branch or persistence slow- or no- flow or embolization, or (iv) Imaging demonstration of new loss of viable myocardium or new regional wall motion defect are require.
Type 4b: Myocardial Infarction related to stent thrombosis
Myocardial infarction associated with stent thrombosis is detected by coronary angiography or autopsy of myocardial ischemia and with a rise and/ or fall of cardiac biomarker values with one value above the 99" percentile URL.
Type 5: Myocardial infarction related to coronary artery bypass grafting (CABG)
Myocardial infarction associated with CABG is arbitrarily define by elevation of cardiac biomarker value >10x99" percentile URL in patients with normal baseline cTn value (<99"percentile URL). In additional either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of variable myocardial or new regional wall motion abnormality.

inflammation and healing of necrotic tissue.

Arrhythmias

In addition to arising from the electrical instability of ischemic myocardium, they can also be caused by interruption of perfusion to structures of the conduction pathway (i.e. SA node, AV node, bundle branches)

Congestive heart failure

Can be directly caused by impaired contractility resulting in both systolic and diastolic dysfunction.

More rarely results from papillary muscle infarction or rupture causing moderate to severe mitral regurgitation.

Thromboembolism

Intra-ventricular thrombus formation can arise from the stasis of blood flow in regions of impaired LV contraction, especially when the infarction involves the apex or when an aneurysm has formed.

Pericarditis

Can arise in the early post-MI period when necrosis and neutrophilic infiltrates extend from the myocardium to involve the adjacent pericardium.

A ventricular aneurysm

Develops as the ventricular wall is weakened but not perforated by the phagocytic clearance of necrotic tissue.

Cardiac tamponade

Hemorrhage into the pericardial space due to ventricular free wall rupture (structurally weakened by necrosis) leads to rapid filling of the pericardial space and severe restriction of ventricular filling; often lethal.

Cardiogenic shock

• Severely decreased cardiac output and hypotension with inadequate perfusion of peripheral tissues develops when more than 40% of the LV mass is infracted.

• The self-perpetuating mechanism whereby impaired contractility results in hypotension, decreased coronary perfusion, exacerbation of ischemic damage, the further decrease in contractile function, and so forth. Common complication following acute myocardial infarction and their approximate 50% of bradyarrhythmias are Mobitz I, 50% are Mobitz II or third degree atrioventicular block. Posterior papillary muscle rupture is the most common mechanical complication of acute myocardial infarction, most often because of infarction of the right communicating artery (which is dominant in 85% of patients). Not listed above, artial fibrillation could be seen any time after acute myocardial infarction most often in patients with left atrial enlargement. LSB=left sterna border. LV= left ventricle. MR= mitral regurgitation. PCI= percutaneous coronary intervention. STEMI= ST segment elevation myocardial infarction

Table 2: Causes of risk factors for Myocardial Infarction

Modifiable risk factors	Targets
Smoking	Smoking damages artery walls and puts extra strain on the heart – the only safe number of cigarettes to smoke is none at all.
Cholesterol	artery wall. Total cholesterol should be less than 4 mmols/L with LDL ('bad cholesterol') being less than 2 mmols/L. You can take steps to eat a diet that helps protect your cardiovascular system by:
	• Reducing saturated/trans fat intake
	• Eating 2-3 oily fish portions per week
	• Increasing fibre in diet• Aiming for 5 portions of fruit and veg per day.
Obesity (BMI of 30 and above or central obesity)	Being the correct weight for your height is important and is calculated by your BMI (Body Mass Index) which should ideally be 20 -25. People who are 'apple' shaped (centrally obese) are at higher risk. Waist measurement should be less than 102cm for men (90cm for Asian men) and less than 88cm for women (80cm for Asian women). You can take steps to reduce your weight by:
	• Reducing calorie intake (reducing amounts of high fat/sugar foods and also portion sizes)
	• Increasing activity levels.
Lack of physical activity/ exercise	The recommendation is 30 minutes of moderate activity 5 days a week e.g. a brisk walk, climbing stairs, cleaning windows, cycling. If you have not exercised for some time or have had a recent heart event, you will be advised to build up your exercise levels gradually. Cardiovascular rehabilitation can help to support you in becoming more active.
High blood pressure/ hypertension	People with cardiovascular disease should aim for a blood pressure reading of less than 130/80mmHg. Steps to reduce your blood pressure include: • Reducing salt intake • Sticking to guidance on alcohol -not regularly drinking more than 14 units per week and spreading this evenly, with at least two alcohol free days per week
	• Aiming to maintain a healthy weight aking regular exercise.
Diabetes	Diabetics are at a higher risk of cardiovascular disease. This risk increases if your diabetes is not well controlled. Diabetics should aim to have a blood sugar between 4 and 7mmols/l before mealtimes. You should liaise with your GP/diabetic nurse to discuss long-term diabetes management.
Stress	Stress can affect some of the above risk factors and so being aware of your own stress levels and how to manage stress is important. Relaxation, seeking support and exercise can all help.

Rationale for Novel Biomarkers of Myocardial Infarction Prediction

This review focuses specifically on established and emerging inflammatory biomarkers involved in the prediction of a Myocardial Infarction. In particular, beyond CRP, IL-6, and Plasma Fibrinogen warrant special emphasis as inflammatory biomarkers at least as research tools, if not currently appropriate for routine clinical use. We will not discuss at this point markers involved in prediction and risk stratification that are assessable in serum by ELISA or markers involved in risk stratification at the time of an acute coronary syndrome for the secondary avoidance of cardiovascular disease. Markers of oxidative stress, LDL oxidation, and heart failure are treated elsewhere.

The only blood biomarkers presently recommended for use in Myocardial Infarction risk prediction by the Adult treatment panel are LDL cholesterol (LDL-C), HDL cholesterol (HDL-C), and triglycerides[16]. However, plasma total cholesterol concentrations alone poorly discriminate risk for coronary heart disease, as more than half of all vascular events occur in individuals with below-average total cholesterol concentrations [17, 18].

Emerging Inflammatory Biomarkers in Myocardial Infarction Assessment

C-reactive protein

CRP is a broadly recognized acute phase protein, formed by the liver in response to pro-inflammatory cytokines, and especially IL-6. Although it is a non-specific inflammatory marker, it appears to be a stronger predictor of cardiovascular risk compared to most of the other known circulating inflammatory molecules. The C-reactive protein (CRP) is synthesized by the liver after a stimulus such as tissue injury, inflammation and/or infection. Its production also occurs in atherosclerotic lesions by smooth muscle cells and macrophages, kidneys, neurons, pulmonary alveoli and adipose tissue [19, 20]. CRP biomarker reflecting relevant events would also be of great use in the ischemic cascade [21]. As the methods traditionally employed to measure CRP do not have good sensitivity, it is recommended to measure us CRP to evaluate

atherothrombotic disease, which usually presents lower CRP levels than the other inflammatory processes. C-reactive protein (CRP) is the most widely studied pro-inflammatory molecule. In healthy individuals, CRP levels are only minimal. Under acute conditions, levels of CRP in the circulation increase during the first 6 to 8 hours and reach a peak value after approximately 48 hours. CRP is an effective clinical marker because of its analytical stability, reproducible results, and commercial availability of high sensitivity assays with good precision. Recent studies suggest that CRP may have direct pro-inflammatory effects and contributes to the initiation and progression of atherosclerotic lesions. Functionally, CRP activates circulating monocytes and induces their recruitment to the arterial wall. It also mediates LDL uptake by macrophages and induces expression of cell adhesion molecules and tissue factor. Measuring CRP levels will also be helpful in determining efficacy of treatment[22]. Serum CRP measurements are simple, rapid, cost-effective [23]. Latest epidemiological studies have supported the significance of CRP level in predicting future cardiovascular risk.

Interleukin-6 (IL-6)

The IL-6 is an important immune cell activator and can participate in the destabilization of the atherosclerotic plaque. The IL-6 also reflects the cardiovascular risk factors in a model that is similar to that of CRP and its levels increase with age. In addition to the association with CHD, IL-6 levels were also strongly associated with a number of established risk factors and other inflammatory markers. For example, there were moderate associations of IL-6 with smoking, diabetes, and dyslipidemia. Accordingly, the strength of the association between IL-6 and CHD varied between models including different sets of covariates and was attenuated when covariates strongly correlated with IL-6 were included. Circulating IL-6 is a physically powerful autonomous marker of increased mortality in unstable CAD and identifies patients who advantage most from a strategy of early invasive management[24]. Studies suggest that the use interleukin-6 (IL-6), for risk stratification identifies individuals who might benefit from targeted interventional or intensive medical therapy [25]. At present, the large circadian difference of IL-6 levels and the lack of positive studies limit the function of IL-6 as a biomarker of ACS [26].

Fibrinogen

Several large-scale epidemiologic studies disclose that baseline fibrinogen concentrations predict future danger of myocardial infarction in addition as stroke [27-29]. compared head-to-head with CRP, clotting factor looks a fewer effective analyst of vessel events [30]. Illustrating the import of detection methodology, while clotting factor is measured among an even and high-quality immunochemical assay, there's a major association between higher concentrations of clotting factor and CRP, alone at the side of together with incident upset in apparently match feminine over a 10-year follow-up amount [31].

Fibrinogen as an inflammatory marker

Fibrinogen is a constituent of coagulation with a determinant of blood viscosity. Elevated fibrinogen levels and boosts platelet reactivity[32]. There is some inconsistency as to whether fibrinogen is considered an inflammatory marker. It is clear that it is responsive to inflammatory stimuli; during the acute-phase, its levels can increase 100–200% over baseline. There is a strong interaction between the inflammatory and the haemostatic systems. Studied healthy individuals have demonstrated a direct and independent association between the plasma fibrinogen levels and the risk of coronary events, of total and cardiovascular mortality. Among the elderly, it also seems to be a risk factor for general and cardiovascular mortality, ischemic CVA and deep venous thrombosis. This platelet hyper-reactivity in diabetes may result from increased fibrinogen levels because fibrinogen acts as across bridge between platelets. Poor diabetic control is also associated with higher levels of fibrinogen.

General risk factors for acute coronary syndromes

A series of modifiable risk factors (e.g. hyperlipidaemia, hypertension, diabetes and metabolic syndrome) and non-modifiable risk factors (e.g. gender and age) relate to the development of atherosclerosis and the risk of presenting with ACS.

These risk factors cannot be changed:

1. Age – risk increases as you get older,
2. Gender – < 60 age of men are at greater risk than women
3. Your Family History – your risk may increase if close blood relatives experienced early heart disease.

But there are other risk factors that you can change:

- Raised or altered levels of blood cholesterol
- Raised triglycerides with low HDL-cholesterol
- High blood pressure
- Diabetes
- Smoking
- Overweight and obesity
- Being inactive
- Excessive alcohol
- Excessive stress

Having extra than one risk factor means the generally risk of coronary heart disease is greatly upper.

Hypertension

There is a well-built connection between hypertension as well as coronary heart disease with there is a mainly strong influence of elevated blood pressure on stroke[33]. The Blood Pressure Lowering Treatment Trialists Collaboration has examined the influence of blood pressure lowering on mortality and the development of major cardiovascular events[34]. Excess alcohol intake seems to be an important contributor to high blood pressure. Thus, blood pressure lowering is critically important not only in secondary prevention but in the primary prevention of major cardiovascular events including the development of ACS.

Diabetes and metabolic syndrome

Clinical and animal studies demonstrate the importance of diabetes as a risk factor for the development of atherosclerosis as well as for ACS, and the increasing prevalence of obesity in specific populations is implicated in the increased frequency of metabolic syndrome and type 2 diabetes. Having diabetes puts people with the condition at a much higher risk of CHD.

C-reactive protein (CRP) and interleukin-6 are markers of inflammation and their levels can be used as predictors for future cardiovascular events; the concentrations of these markers rise with the extent of

obesity. In addition, adiponectin, a biomarker of insulin sensitivity which also has a role in preventing atherosclerosis development, is decreased in obesity [35]. The findings provide a link between obesity, metabolic syndrome and future risk of acute coronary events. Past study was shown; oxidative stress has been implicated in the progression of major health problems by inactivating the metabolic enzymes and damaging important cellular components, oxidizing the nucleic acids, leading to cardiovascular diseases[36]. As elevated HbA1c and dyslipidemia are independent risk factors of CVD, diabetic patients with elevated HbA1c and dyslipidemia can be considered as a very high risk group for CVD[37]. Old study was also been finding able to prove that there is increased Lipid Profile and dyslipidemia in Non-vegetarian person which show Heart Failure, myocardial infarction, Atherosclerosis[38].

Lipid abnormalities

Wide studies have established that elevated low density lipoprotein (LDL) cholesterol and very-low-density lipoprotein (VLDL) cholesterol are associated with atherogenesis and that lowering total cholesterol and LDL cholesterol is associated with reduced atherogenesis [39]. Elevated levels of HDL-cholesterol are protective whereas reduced levels of HDL confer increased risk. Multiple large-scale cholesterol-lowering trials have demonstrated a reduced number of cardiovascular events among treated individuals without manifest coronary artery disease. More detailed consideration of hyperlipidaemia and coronary risk is described elsewhere.

Conclusion:

We conclude that patients with Myocardial infarction with previous biochemical markers now play a crucial role in the detection of disease, risk stratification and the monitoring of therapy. In past prospective analysis of CRP proved to be the strongest and most significant predictor of the risk of future myocardial infarction. These observations suggest the advantage of screening for circulating IL-6 concentrations and the use of anti-inflammatory treatment (such as aspirin and statins) for those thought to be at high risk of future MI, though although further studies are necessary to confirm this premise. Fibrinogen represents an inflammatory marker that appears to be implicated in the pathophysiology and prognosis of myocardial infarction. However, studied to investigate the value of adding information on CRP or fibrinogen levels to conventional models for the prediction of cardiovascular risk among people without known cardiovascular disease.

The clinical outlook, it is significant to recognize that the immediate measurement of lipids, particularly LDL cholesterol, and IL-6 improves the prediction of risk of future MI-coronary death compared with that associated with lipids or IL-6 alone. With the continuing explore in this field, future holds hopes of finding an ideally specific marker of myocardial infarction, but until then biochemical markers should be used in conjunction with clinical evaluation and electrocardiography in making the diagnosis of myocardial infarction, and the patients should not be treated merely on the basis of elevated serum levels of cardiac biochemical markers.

Abbreviations

MI: Myocardial Infarction CRP: C- reactive protein; AMI: Acute myocardial infarction; IL-6: Interleukin -6; ECG: Electrocardiogram; cTn: Cardiac troponin; CAD: Coronary artery disease; ACS: Acute coronary syndrome; LDL-C: Low Density Lipoprotein- cholesterol, HDL: High Density Lipoprotein- cholesterol, VLDL: Very-low-density lipoprotein

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