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Journal of Diabetic Complications and Research

RESEARCH ARTICLE

Comparative Study of Metabolic Syndrome Parameters in Myocardial Infarction Parameters versus Controls

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Citation: Sudha Y (2019) The Patient-Centered Model and Diabetes. J Diabetic Complications Res 1:103

Abstract

Metabolic syndrome is defined as a state of metabolic dysregulation characterized by insulin resistance (IR), hyperinsulinemia, and a predisposition to type 2 diabetes mellitus (T2DM), dyslipidemia, atherosclerotic vascular disease, hypertension and other disorders. Epidemiological reports from the World Health Organization and American Heart Association beginning in the late 1950s required the presence of at least two of the following for the diagnosis of myocardial infarction: characteristic symptoms, electrocardiographic changes, and a typical rise and fall in the biochemical markers.

Keywords: Child; Parents; Homicide; Murder

Aims and Objectives

1. To compare the metabolic syndrome parameters in patients with acute myocardial infarction versus healthy controls
2. To compare the serum levels of non-HDL -C between cases and controls
3. To compare serum levels of total cholesterol I HDL -C ratio between cases and controls
4. To compare serum levels triglycerides, I HDL - C ratio between cases and controls

Subjects and Methods Study Design: Case-control study (cross-sectional)

Study Duration: January 2009 -November 2010

Study Protocol: All the patients were admitted in ICCU, cardiology KGH. ECG was taken on arrival for every patient. 100 patients with Acute Myocardial Infarction admitted during the early hours of the day were taken for the study. Fasting blood samples were taken from cases of myocardial infarction within 1 hr of diagnosis of Myocardial Infarction & also for 50 age and sex-matched controls Patients were diagnosed as Myocardial Infarction based on ECG evidence & enzyme markers. Metabolic Syndrome was diagnosed based on NCEP-ATP-III guidelines. Blood pressure was recorded at the time of admission & waist circumference was measured at the time of discharge from the hospital for each patient. Blood samples were taken for lipid profile and glucose estimations in the fasting state on the day of admission. Anthropometric data and waist circumference were measured.

Results: IFG & DM were noted in 12.4% & 34% of cases and 6 & 4% of each in controls respectively. Both SBP & DBP were elevated in 42% & 41% in cases as compared to controls which showed elevation in 4% & 8% respectively. WC was increased in 40% of cases and 28% of controls. Prevalence of IFG, DM, ↑SBP, ↑DBP, ↑WC was significantly higher in cases compared to controls. Out of 40 female cases, 5(12.5%) & 17(42.5%) had impaired fasting glucose & diabetes mellitus respectively, 16 (40%) & 15 (37.5%) cases had elevation of both systolic blood pressure & diastolic blood pressure respectively & 18(45%) had increased waist circumference. Prevalence of diabetes mellitus, increased systolic blood pressure, and increased diastolic blood pressure, were significantly more in cases compared to controls whereas an increase in waist circumference was not significant. Means of total cholesterol, triglyceride, HDL-C, LDL-C, VLDL-C, were significantly elevated in cases compared to controls.

Discussion: The present study was done in 100 acute myocardial infarction patients aged > 45yrs. Most of them were between 51-60yrs. There was a male preponderance (60%) similar to A. Kumar & R. Sivakanesan, *et al.* & Hiroyasu Iso, *et al.* The metabolic syndrome parameters like fasting plasma glucose, systolic blood pressure, diastolic blood pressure, waist circumference, serum triglycerides, were significantly higher and high-density lipoprotein (HDL-C) was significantly lower in acute myocardial infarction patients compared to controls. The prevalence of impaired fasting glucose (12%) & diabetes (34%) were significantly (p0.05). The mean waist circumference in acute myocardial infarction patients (93.35 ± 13.45) was significantly (p=0.02) elevated than controls (88.24± 10.99) similar to the findings of Arun Kumar & R. Sivakanesan, *et al.* (100.77 ± 6.06 VS 93.70 ± 3.63 p<0.001).

Introduction

Metabolic Syndrome

Metabolic syndrome (MS) is defined as a state of metabolic dysregulation characterized by insulin resistance (IR), hyperinsulinemia, and a predisposition to type 2 diabetes mellitus (T2DM), dyslipidemia, atherosclerotic vascular disease, hypertension and other disorders. Affected individuals are typically obese or overweight, or show more subtle manifestations of increased adiposity such as an increase in abdominal fat or fat cell size. Based on recent NCEP ATP III diagnostic guidelines, it has been estimated that more than 50 million individuals in the USA older than 20 years. Have metabolic syndrome [1-8].

The clustering of the major components of the metabolic syndrome, such as obesity, T2DM, hypertension and dyslipidaemia has long been recognized; however, its delineation as a distinct entity took place only after its linkage to insulin resistance, hyperinsulinemia, and cardiovascular disease became more apparent [9-12].

Insulin resistance has been defined as a state in which greater than normal amounts of insulin are required to elicit a normal biologic response. In humans, it is currently diagnosed based on high levels of plasma insulin that occurs during, either fasting, or during a glucose tolerance test, or by a decreased rate of glucose infusion, or glucose uptake by muscle during a euglycemic-hyperinsulinemic clamp [13].

Shortly after the development of the Insulin immunoassay by Yalow and Berson, this suspicion was confirmed, and a whole array of disorders associated with insulin resistance and hyperinsulinemia was identified, in addition to T2DM, including cardiovascular disease and several of its risk factors. In general, most adults with insulin resistance and hyperinsulinemia were found to be obese body mass index (BMI>29 kg/m^2) or (BMI-25-29 kg/m^2) [14] (St Onge MP 2004). Significant percentages were normal weight by BMI but showed an increase in abdominal fat and/ or enlarged fat cells. The presence of central obesity has become one of the principal diagnostic criteria for metabolic syndrome.

Hyperinsulinemia & insulin resistance also were found in normal weight offspring of people with type 2 diabetes mellitus, hypertension, and hypertriglyceridemia, and in individuals at increased risk for cardiovascular disease suggesting that these are early markers of pathogenetic events for these disorders [15-18]. Presence of a high rate of ischemic heart disease in patients with type 2 diabetes mellitus at the time of diagnosis & to somewhat less extent, individuals with impaired glucose tolerance, has led to the suggestion that the treatment of the metabolic syndrome at an early stage may be needed for preventing coronary heart diseases [19-21].

Pathophysiology

Insulin Signaling Cascade

Hypothetically, the metabolic syndrome could be related to genetic abnormalities in the insulin signaling cascade. Mutations of insulin receptor substrate & insulin resistance S2 (IRS2) the initial targets of the insulin receptor tyrosine kinase have been shown to lead to insulin resistance and type 2 diabetes mellitus in transgenic mice [22]. These or other genetic defects in the insulin signaling cascade are common in humans with the Metabolic syndrome or type 2 diabetes mellitus and accounts for observed signaling defects [23,24].

Vascular Endothelial Cells

Atherogenesis is essentially an inflammatory response to a variety of risk factors and the consequences of this include coronary and cerebrovascular syndromes. An early site at which this inflammatory response appears to occur is the endothelial cell. Impaired endothelium-dependent relaxation and increase in circulating adhesion molecules, markers of cellular dysfunction and incipient atherosclerotic vascular disease, have been observed in humans with type 2 diabetes mellitus and the metabolic syndrome and in normal individuals in whom plasma free fatty acid levels are increased by a lipid infusion [25,26].

Liver

Changes like those in muscle and the endothelial cell occur in the liver in insulin-resistant states.

Pancreatic β Cell

Prolonged increases in the concentration of saturated fatty acids and glucose cause dysfunction and damage to the β cell and ultimately result in apoptosis [27,28].

Linkage of Metabolic Syndrome to Coronary Heart Disease

The notion that the metabolic syndrome, or its surrogate markers hyperinsulinemia and insulin resistance, antedate and contribute to the pathogenesis of coronary heart disease. Coronary Heart Disease can be attributed to dyslipidaemia present in people with metabolic syndrome as well as to elevations in blood pressure and the presence of procoagulant, proinflammatory state. Low levels

of adiponectin are associated with an increased risk of Coronary Heart Disease [29]. Overexpression of adiponectin or its globular subunit diminishes the severity of atherosclerosis [30]. The Metabolic syndrome per se predisposes to Coronary Heart Disease and cardiovascular disease. A two-to-four-fold increase in subsequent cardiovascular events has been described in men and women with metabolic syndrome, even in the absence of 2 diabetes mellitus or impaired glucose tolerance [31]. The presence of the metabolic syndrome had an even greater impact on the risk for developing type 2 diabetes mellitus [32,33].

The rate of cardiovascular events was higher in patients who had type 2 diabetes mellitus and metabolic syndrome than in individuals with only the metabolic syndrome [34].

Children and Adolescents

Recent studies indicate that the prevalence of metabolic syndrome is increasing in children and adolescents in parallel with the increase in obesity and type 2 diabetes mellitus in this population. It has been associated with insulin resistance, central obesity, dyslipidemia, elevations in blood pressure, and increases in intramyocellular and intrahepatic lipid [35,36]. The prevalence of non-alcoholic fatty liver disease and polycystic ovarian syndrome is increased. The prevalence of cardiovascular diseases in these individuals later in life may also be increased [37].

Metabolic Syndrome: Definitions

Several definitions have been proposed for metabolic syndrome by, World Health Organisation (WHO) (1999) (Table 1), National Cholesterol Educational Programme Adult Treatment Panel (NCEP ATP III) (2001) (Table 2), American Association of Clinical Endocrinologist criteria, and International Diabetes Federation (IDF) [38-40].

The one followed in this study is as per National Cholesterol Educational Programme Adult Treatment Panel (NCEP ATP III) (2001) [41,42].

The main purpose of identifying metabolic syndrome was to identify clustering of features that were associated with increased cardiovascular disease risk [43].

Metabolic Syndrome: Definitions WHO (1999)

	Glucose intolerance, IGT or diabetes and/ or insulin resistance + two or more of the following:
Fasting plasma glucose	> 110 mm / dl
Blood Pressure	≥140 / 90 mmHg
Triglycerides	Raised plasma triglycerides: > 1. 7 mmol/l (150 mg / dl)
HDL Cholesterol	Men: <0.9 mmol/l (35 mg/ dl) Women: < 1.0 mmol/1 (39 mg/ dl)
Obesity	Men: waist-hip ratio >0.90 Women: waist-hip ratio > 0.85 and / or BMI >30 kg/m2.
Microalbuminuria	Urinary albumin excretion rate 20 mg / min or albumin: creatinine ratio ≥30 mg/g

Table 1: Metabolic Syndrome: Definitions WHO (1999)

NCEP ATP III (2001)

	Three or more of the following five risk factors:
Fasting plasma glucose	≥5.6 mmol/1 (100 mg/ dl)
Blood Pressure	≥130 / 85 mmHg
Triglycerides	≥1.7 mmol/l (150 mg/dl)
HDL Cholesterol	Men: <1.03 mmol/1 (40 mg/ dl) Women: <1.29 mmol/1 (50 mg/dl)
Obesity	Men: waist circumference > 102 cm Women: waist circumference >88 cm

Table 2: National Cholesterol Educational Programme Adult Treatment Panel (NCEP ATP III) (2001)

Myocardial Infarction

Myocardial infarction in India has assumed epidemic proportions and is increasing at an alarming rate. The 2001 global burden of diseases study confirmed the growing importance of non-communicable diseases in most low and middle-income countries. The cardiovascular disease burden afflicts both men and women. The 1999 World Health report showed that cardiovascular deaths

account for 34% of all deaths in women and 28% in men. Between 1990 and 2020, the increase in ischemic heart disease mortality (120% in women and a 137% in men) in the developing countries is expected to be much greater than the developed countries (29% and 48%) respectively. India is currently witnessing a demographic and epidemiological transition. Epidemiological studies show that cardiovascular disease affects 3-5% of the rural and 7-10% of the urban adult population. This is likely to rise in future [44-47].

Myocardial infarction in India occurs 5-10 years younger compared to western population. As this affects young individuals in the prime time of life, the disability to the individual and the economic burden on the society increases. The incidence of myocardial infarction and symptomatic cardiovascular disease in the young adults is 3-6% in most of the studies. The fact that clinically manifest cardiovascular disease is relatively uncommon in young individuals prevents them to seek medical attention at the earliest [48].

Much of the knowledge regarding the cardiovascular disease and its risk factors in India are lacking and it is being extrapolated from the western studies. The subject of cardiovascular disease in Indians has become a challenge to many research centers worldwide. The conventional and newer risk factors namely hypertension, diabetes mellitus, hypertriglyceridemia, low levels of HDL-C, central obesity, Lp(a), high LDL-C, rapid modernization associated with sedentary and stressful lifestyle in summation are suggested as additional risk factors for coronary artery disease [49,50].

Review of Literature

Metabolic syndrome has several names including Syndrome X, Insulin Resistance syndrome, the deadly quartet, Cardiometabolic syndrome and the hypertensive syndrome, but the WHO has given it an official name i.e., Metabolic syndrome (Metabolic syndrome) [51]. It is a clustering of cardiovascular disease risk factors: abdominal obesity, atherogenic dyslipidaemia, elevated blood pressure, and insulin resistance. It has become a large public health issue. Its prevalence is increasing [52-57].

Asian Indians have long been a "high-risk population" for both metabolic syndrome and cardio vascular disease. Despite the various criteria devised for metabolic syndrome diagnosis, most studies agree that the prevalence of Metabolic syndrome in Asian Indians is underrepresented when NCEP ATP –III or WHO criteria are employed, considering the high propensity of this ethnic group to develop Metabolic syndrome and its various co-morbidities [58]. It is well known that Asian Indians have a smaller built and excess body fat with predominant abdominal adiposity as compared with Caucasians. To this effect, the WHO has recognized the need for a population-specific modification of anthropometric measures [59]. The recommended lower BMI cut-off for defining 'overweight' in Asian Indians is 23 kg/m^2 (WHO 2004), modified waist circumference measures is 94 cm and 80 cm, and a waist-hip ratio (WHR) of 0.89 and 0.81 for men and women, respectively.

Prevalence

The prevalence of metabolic syndrome based on National Health and Nutrition Examination Survey (NHANES - 2001-02) has been estimated to be 36.8% according to NCEP-ATP III definition and 39.9% according to the International Diabetes Federation (IDF) definition [60,61]. It is estimated that 12million adults greater than or equal to 40yrs of age have diagnosed or undiagnosed diabetes, of whom most have metabolic syndrome (69.9% for whites, 64.8 Waist % for Blacks and 62.4% for Mexican Americans). Some estimates suggest that as many as 90% of persons with established type 2 diabetes mellitus have metabolic syndrome. An estimated 41 million people aged 40-74 years have pre-diabetes (raised blood glucose levels insufficiently high to be called diabetes mellitus) [62].

The Indian subcontinent is undergoing an epidemiological transition as non-communicable diseases like coronary heart disease and type 2 diabetes mellitus are fast rapidly replacing infections as the leading cause of morbidity and mortality. India is already the 'Diabetic capital of the world [63].

S. Kanjilal, *et al.* in 'The Indian Atherosclerosis Research Study (IARS)', found that NCEP ATP-III criteria identified a significantly higher proportion of people with metabolic syndrome compared to WHO [64]. They found that the revised criteria for metabolic syndrome with lowered cut-offs for waist circumference and body mass index are critical for the accurate assessment of metabolic syndrome among Asian Indians [28]. Metabolic syndrome was diagnosed a decade earlier in unaffected subjects compared with those with cardio vascular disease /diabetes using the modified metabolic syndrome criteria. Waist circumference correlated significantly with BMI and waist-hip ratio (WHR). Among Metabolic syndrome components, high density lipoprotein cholesterol and BMI contributed significantly in males and females respectively [40].

Significant differences exist in the prevalence of various components of the metabolic syndrome even within an urban environment and this appears to be influenced by socioeconomic status. V. Mohan, *et al.* in their study in south Indian urban population, found that geographical area (higher social class) had a strong association with components of metabolic syndrome even after inclusion of other risk factors like age, body mass index into the model [58].

M. Deepa, *et al.* in 'The Chennai Urban Rural Epidemiological study', reported an increased risk of probable cardio vascular disease in Metabolic syndrome subjects diagnosed by WHO criteria (odds ratio (OR) 3.86, 95% Confidence Interval (CI), 2.37--6.29, $p < 0.001$), compared to NCEP ATP III criteria (OR 2.19, 95% CI 1.30-3.67, $p < 0.05$) and IDF criteria (OR 1.90, 95% CI 1.16-3.12, $p < 0.05$). The WHO criteria marked out a much higher population for cardio vascular disease risk compared to NCEP ATP III and IDF criteria in males, but not in females.

At the same time, several other cardio vascular disease risk factors have been identified in the context of clinical and epidemiological studies in relation to cardio vascular disease outcomes, including Lp(a), homocysteine, fibrinogen and uric acid. The association of these newer risk factors with metabolic syndrome needs to be explored as it is likely to throw more light on the pathophysiology of metabolic syndrome as well as managing the cardio vascular disease risk in asymptomatic population. There have been reports on the prevalence of the novel risk factors in people with metabolic syndrome. Many of them are cross sectional studies from various parts of the world. Some prospective have also started coming up to get an insight into mechanistic relationship between these novel risk factors and development of metabolic syndrome. This may lead us in near future to a better understanding of the development of metabolic syndrome.

Risk Factors That Confer Greater Susceptibility to Coronary Artery Disease in Asian Indians

1. Greater predisposition to high lipoprotein (a)
2. Other components of Asian Indian dyslipidemia
    a. Small dense dysfunctional HDL-C
    b. High LDL-C
    c. High triglycerides and low HDL cholesterol
    d. High Apo B, low ApoA-1, increased Apo B/ Apo A ratio
    e. Low HDL-L and high TC/HDL-C ratio
    f. Abdominal obesity
    g. Metabolic syndrome
    h. Diabetes mellitus and pre-diabetes

Reports on cardio vascular disease in Indians from different parts of the world have shown that Asian Indians are at 3-4 times higher risk of cardio vascular disease than white Americans, 6 times higher than Chinese, 20 times higher than Japanese [66].

It must be emphasized that although the median age of presentation is higher in women (58 years for female, 54 years for male) they are known worldwide to have poor prognosis compared to men. Younger Asian women has worse survival at 28 days after acute myocardial infarction.

There is a strong correlation between urbanization and increase in coronary heart disease prevalence in urban subjects in India and it was hypothesized that coronary risk factors are more prevalent in urban subjects in India and it was hypothesized that coronary risk factors more prevalent in urban subjects should be important in atherosclerosis pathogenesis [67].

Padmavathi, *et al*. reported that the prevalence of coronary heart disease was significantly greater in the urban subjects in Delhi as compared to rural subjects around Delhi. She also reported that coronary risk factors like hypertension and cholesterol levels were more in the urban subjects. Similar urban-rural differences were reported by Gupta, Chadha, *et al.* in early 1990 s in Delhi and I CMR studies in Delhi and Vellore also reported similar urban- rural differences. All these studies reported that multiple life style factors (sedentary, dietary calories and fat intake) as well as physiological factors (weight, body mass index, waist circumference, blood pressure, total cholesterol and LDL, LDL/HDL ratio, triglycerides and diabetes mellitus) were significantly more in the urban population. All these studies noted that smoking which was an established risk factor was more in the rural subjects. The body mass index in urban Indians as compared to rural Indians is 24 versus 20 in males and 25 versus 20 in females [19,68].

Unfortunately, the ongoing urbanization of rural India is likely to narrow down these differences. New affluence is associated with sedentary life style and higher consumption of calories, saturated fats, salt, tobacco and alcohol. These factors contribute to obesity, dyslipidaemia, hypertension, type 2 diabetes mellitus. Therefore, there must be high index of suspicion for cardio vascular disease in Indians even in younger age group. The risk factor evaluation must start earlier. Investigations like tread mill, stress echo, thallium and coronary angiography should be more liberally recommended [69].

The Role of Risk Factors in CHD

There are multiple risk factors both modifiable and non-modifiable which can accelerate atherosclerosis independently or through an additive effect (Table 3).

Non-Modifiable risk factors	Modifiable risk factors	
a) Age b) Sex c) Genetic predisposition	**Major:** Hyper lipidemia Hypertension Cigarette smoking Psycho social tension Obesity Hyper homocysteinemia Metabolic syndrome X	**Minor:** Oral contraceptives Sedentary living Personality type Diabetes Mellitus

Table 3: Role of Risk Factors in CHD

Newer Risk factors

a) Homocysteine
b) Fibrinogen
c) Lipoprotein (a)

Diagnosis of Myocardial Infarction

The clinical diagnosis of myocardial infarction requires an integrated assessment of the history with some combination of indirect evidence of necrosis using biochemical, electrocardiographic and imaging modalities (Table 4). The sensitivity and specificity of the clinical tools for diagnosing myocardial infarction vary considerably and change at varying times after onset of the infarction.

Epidemiological reports from the World Health Organization and American Heart Association beginning in the late 1950s required the presence of at least two of the following for the diagnosis of myocardial infarction: characteristic symptoms, electrocardiographic changes, and a typical rise and fall in the biochemical markers. The main features of revised definition of myocardial infarction are summarized. The revised definition of myocardial infarction has important implications not only for clinical care of patients but also for tracking epidemiological trends, public policy, and clinical trials.

Pathology	Aspect of Diagnosis of Myocardial Infarction by Different Techniques
Biochemistry	Myocardial cell death
Electrocardiography	Markers of myocardial cell death recovered from blood samples
	Evidence of myocardial ischemia (ST and T wave abnormalities) Evidence of loss of electrically functioning cardiac tissue (Q waves)
Imaging	Reduction or loss of tissue perfusion Cardiac wall motion abnormalities

Table 4: Aspect of Diagnosis of Myocardial Infarction by Different Techniques

ACC/AHA Definition of Myocardial Infarction

The term myocardial infarction should be used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischemia. Under these conditions any one of the following criteria meets the diagnosis for myocardial infarction.

Criteria for Acute Myocardial infarction

1. Typical rise and/or fall of biochemical markers of myocardial necrosis with at least one of the following:
a. Ischemic symptoms
   b. Development of pathological Q waves in ECG
   c. ECG changes indicative of ischemia (ST elevation or depression)
   d. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
2. Sudden, unexpected cardiac death involving cardiac arrest often with symptoms suggestive of myocardial ischemia and accompanied by presumably new ST elevation, and /or evidence of fresh thrombus by coronary angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood.
3. For percutaneous coronary interventions in patients with normal base line troponin values, elevations of cardiac biomarker above the 99th percentile URL are indicative of periprocedural myocardial necrosis. By convention, increases of biomarkers greater than 3 times 99th URL are indicative of periprocedural myocardial necrosis. By convention, increases of biomarkers greater than 3 times 99th percentile URL have been designated as defining PCI-related myocardial infarction.
4. For coronary artery bypass grafting (CABG) in the patients with normal baseline troponin values, elevations of cardiac biomarkers above 99th percentile URL are indicative of peri-procedural myocardial necrosis. By convention increase of biomarkers greater than 5 times 99th percentile URL plus either new pathological q waves or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium have been designated as defining CABG related myocardial infarction.

The incidence, prevalence, hospitalization, morbidity, and mortality from Asian Indians have been 50% to 300% higher than Europeans, Americans, and other Asians irrespective of whether they live in India or immigrated.

Clinical Features

Symptoms: The classic symptoms of acute myocardial infarction involve chest discomfort that is commonly retrosternal or precordial in location that is described as pressure, aching, burning, squeezing, and heaviness in quality. The pain radiates over the anterior chest and frequently in to the left arm or both arms and or into the neck and jaw. Rarely can it radiate into the interscapular area. The duration of pain is prolonged. The intensity of pain is usually steady following an initial crescendo. The combination of substernal chest pain persisting for >30 min and diaphoresis strongly suggests STEMI associated symptoms may include dysnoea,

diaphoresis, nausea and vomiting. Occasionally presenting symptoms include syncope, acute confusion, agitation, stroke or palpitations. An acute myocardial infarction may also masquerade as the development or worsening of congestive cardiac failure, the appearance of an arrythmia, acute indigestion, pericarditis or peripheral embolism. Presentation with painless myocardial infarction is more common in the elderly than non-elderly.

Physical Findings

General Examination: The heart rate and rhythm arc very important indicators of cardiac function in the initial hours of acute myocardial infarction. A normal rate indicates no significant hemodynamic compromise. Persistent sinus tachycardia beyond the initial 12-24 hrs is predictive of a very high mortality. Acute inferior wall myocardial infarction might have bradycardia which is usually transient. Low pulse volume with cool extremities may indicate cardiac failure. All peripheral pulses should be examined to observe their presence, to exclude current occlusion and to provide a baseline in case of future embolic events. The rhythm of the pulse is very important because of the frequency of ectopic atrial and ventricular beats (large **'a'** wave in JVP indicating ventricular ectopics). Examination of jugular venous pulse is important in-patients with an inferior infarction (The right ventricle is commonly involved). JVP is usually raised with a prominent **'a'** wave and kussmaul's sign may also be positive. Indicators of hyperlipidaemia should be looked for (Xanthelasma, eruptive xanthomas, tendinous xanthomas). There is also an association between oblique earlobe crease with premature atherosclerosis [70].

Cardiac examination: Palpation of precordium is helpful in detecting the dyskinetic impulses. First heart sound (Sl) is usually muffled due to decreased contractility. Heart sound **(S2)** may be paradoxically split. Heart sound **(S4)** is a rule rather than exception due to decreased LV compliance. Heart sound **(S3)** occurs in 10-15% of cases. Pericardial friction rub is present in 10% of cases after initial 48-72 hrs. A Pansystolic murmur can be heard because of mitral regurgatation due to papillary muscle dysfunction or because of rupture of interventricular septum leading to acquired ventricular septal defect.

Investigations

Electro cardiogram: Terminology has changed from transmural/non-transmural to Q wave/non-Q wave myocardial infarction because of discrepancy at autopsy studies. More recently ST elevation/Non-ST elevation are the terms used keeping in view of the difference in management [71].

ECG Criteria for Acute MI: a) **ST** elevation of > 1mm in limb leads and > 2mm in chest leads which should be present in at least two contiguous leads measured at 0.02s after j point.

Electrocardiographic Evolution of Acute Myocardial Infarction: The infarction process evolves through three easily recognizable electrocardiographic phases. These are:

1. The Hyperacute phase.
2. The Fully evolved phase.
3. The chronic stabilized phase.

Cardiac Biomarkers

Recommended approach is

- Measure serum troponin-I or troponin-Tat first presentation.
- If the troponin is not elevated, repeat at six to nine hours. It is not uncommon to measure a second troponin earlier than six hours in patients who are highly suspected of having ongoing NSTEMI, since 80 percent of patients who rule in will do so in two to three hours. In an occasional patient in whom the index of suspicion for acute myocardial infarction is high, but the first two troponin measurements are not elevated, a repeat measurement at 12 to 24 hours may be necessary.
- Creatine kinase -MB is measured when a troponin assay is not available. Previously, creatine kinase-MB was advocated to help diagnose reinfarction, but now troponin has subsumed that role. Reinfarction is diagnosed if there is a ≥20 percent increase of the value in the second sample.

Troponin elevations persist for one to two weeks after acute myocardial infarction, but values are usually not rising or falling rapidly currently, allowing one to distinguish acute from more chronic events

Conclusion

- Prevalence of Metabolic Syndrome was 33% in acute myocardial infarction patients which was significantly higher than controls, after excluding known hypertensives and diabetics.
- Incidence of acute myocardial infarction was more between 51-60 yrs.
- Prevalence of acute myocardial infarction was more in males compared to females.
- Diabetes mellitus, elevation of systolic blood pressure & diastolic blood pressure, and central obesity were more prevalent in acute myocardial infarction patients.
- Serum total cholesterol, triglycerides, low density lipo protein (LDL-C), very low density lipo protein (VLDL-C), non-high density

lipo protein (NON-HDL-C), were significantly higher and high density lipo protein (HDL-C) was lower in acute myocardial infarction patients.

• Ratio of serum total cholesterol / high density lipo protein (TC/HDL-C), serum triglycerides / high density lipo protein (TG/HDL-C) were significantly higher in acute myocardial infarction (AMI).

These observations suggest that prevalence of Metabolic Syndrome was higher in patients with acute myocardial infarction and early recognition and treatment might reduce the burden [72].

Acknowledgement

I express my profound gratitude to my revered teacher **Dr. K. Dileep Kumar, M.D., D.M.,** Professor and Head of the Department of Endocrinology, Andhra Medical College, Visakhapatnam. It is with his constant guidance and sincere advice that I was able to complete this oceanic task. Working under his guidance, has been a matter of pride.

I am thankful to **Dr. K.A.V. Subrahmanyam** Professor of Endocrinology, Gandhi Medical College, Hyderabad for his thoughtful suggestions and constant support.

I am indebted, to my teacher **Dr. A. Mythili** Associate Professor of Endocrinology, Andhra. Medical College, Visakhapatnam for her personal interest, invaluable guidance and encouragement.

I am thankful to my Assistant Professor **Dr. B.G.V. Giridhar**, and my colleagues **Dr. B. Vivekanand, Dr. A. Kiranmai, Dr. Arun**, **Dr. Brahmanandam for their constant encouragement**. The help rendered by **Dr. A Sridevi** is specially acknowledged for sparing her invaluable time in the completion of this work.

I would be doing great injustice to myself, if I do not acknowledge the Cardiology Department for their cooperation.

I am thankful to all the patients and their caretakers for their willingness and co-operation in the conduct of this study.

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