METABOLIC SYNDROME
FREQUENCY IN PATIENTS PRESENTING WITH ACUTE MYOCARDIAL INFARCTION

ORIGINAL
PROF-1774

DR. GHAZANFAR ALI SANDHU
FCPS, Medicine
Assistant Professor
Punjab Medical College, Faisalabad.

DR. SHAHID IQBAL
FCPS, Medicine
Allied Hospital Faisalabad.

PROF. DR. AHMED BILAL
FCPS, Medicine
Professor of Medicine
Allied Hospital Faisalabad

Dr. Mohammad Mohsin Rana
FCPS, Medicine.
Al-Shifa Medical Center,
Jail Road, Faisalabad.

Dr. Rehan Abdullah, MBBS
Medical Officer
Allied Hospital Faisalabad.

Dr. Faraz Saeed Qureshi
Senior Registrar
Allied Hospital Faisalabad.

ABSTRACT... Objective: To determine the frequency of metabolic syndrome in patients presenting with acute myocardial infarction (MI). **Design:** Cross –sectional, observational, multi center study. **Place and duration:** Allied Hospital Faisalabad from 01-01-2009 to 30-06-2010. **Materials and Method:** Any patient fulfilling the criteria of acute myocardial infarction were admitted and enrolled in the study during the study period. Demographic details, history and clinical examination of the patients were recorded on prescribed performa after securing an informed consent. Blood Pressure was recorded in lying posture from right arm and waist circumference measured at umbilical level in lying position. Blood sample was collected in fasting state for estimation of plasma glucose, serum HDL-cholesterol and serum triglycerides levels. **Results:** Out of 690 patients, 420(60.86%) were male and 270(39.14%) were females with average age 55.90±10.19. 40% males and 44% females had metabolic syndrome and incidence increased with age. Waist circumference was increased in 46.85% participants followed by increased fasting blood sugar (42%) levels. **Conclusions:** Frequency of metabolic syndrome was high among the patients with acute myocardial infarction. It supports the potential for preventive efforts in persons with high risk for acute myocardial infarction.

Key words: Acute myocardial infarction, metabolic syndrome.

INTRODUCTION

The Metabolic Syndrome is a cluster of metabolic abnormalities including centrally distributed obesity, decreased high density lipoprotein cholesterol (HDL-C), elevated triglycerides (Tg), hypertension (HTN), and hyperglycaemia[1]. Atherosclerotic disease is projected to become the leading cause of global morbidity and mortality by 2020 and is associated with the presence of the metabolic syndrome (MS)[2-4]. Incidence of coronary heart disease (CHD) is higher in Indian subcontinent indigenous population and is higher in the migrants to industrialized world than natives[5-7]. Third report of the National Cholesterol Education Program Expert Panel (ATP-III) draws attention to the importance of the metabolic syndrome, where the risk of Coronary Artery Disease (CAD) is increased by 7.3 times in males and 10.2 times in females[8,9].

OBJECTIVE

The objective of the study was to determine frequency of metabolic syndrome in patients with acute myocardial infarction in local population.

OPERATIONAL DEFINITIONS

STEMI, Acute myocardial infarction

All patients fulfilling the two of the following three criteria were labeled as having acute myocardial infarction i.e. classic history of ischemic cardiac pain>30 minutes, ECG findings (ST segment elevation>2mm in pericardial leads, >1mm in limb leads, or ST segment depression >1.2mm or T-wave inversions >6mm or new Left Bundle Branch Block (LBBB) and positive Cardiac Troponin T (cTrT).

NSTEMI, Acute myocardial infarction

Classic history of ischemic cardiac pain>30 minutes, ECG findings (ST segment depression >1.2mm or T-wave inversions >6mm) and positive Cardiac Troponin T (cTrT).

Metabolic Syndrome
Revised National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) definition was used to diagnose metabolic syndrome i.e. presence of three out of these five parameters. These are: Waist circumference >90cm for men and >80cm for women, Fasting plasma glucose >110mg/dl or previously diagnosed TYPE 2 diabetes, Hypertension i.e. Arterial Blood pressure >130/85mm Hg or on anti hypertensive medication, Triglyceride level (TG)>150mg/dl or on treatment for this abnormality, High density lipoprotein cholesterol (HDL-C) <50mg/dl for women and <40mg/dl for men or on treatment for this abnormality.

MATERIALS AND METHODS

SETTINGS
The study was conducted at Allied Hospital Faisalabad.

DURATION
1st January 2009 to 30th June 2010.

SAMPLING TECHNIQUE
Non-probability purposive sampling.

INCLUSION CRITERIA
All patients of either sex with age more than 30 years fulfilling the criteria of acute myocardial infarction.

EXCLUSION CRITERIA
Patients having ascites due to any cause, Pregnancy, Familial hyperlipidemia, Renal failure, Hypothyroidism, Hepatobiliary disease and Nephrotic syndrome were excluded from study.

STUDY DESIGN
Cross –sectional, observational, multi center study

DATA COLLECTION
Patients fulfilling the above mentioned criteria of acute myocardial infarction were included in study after taking informed consent and relevant data was recorded on prescribed Performa.

DATA ANALYSIS
SPSS version 11 was used for data analysis. Descriptive statistics like mean with standard deviation (S.D.) was applied on age, fasting blood sugar, blood pressure and High density lipoprotein cholesterol (HDL-C, triglycerides (TG) and waist circumference. Male to female ratio was given. Frequency of metabolic syndrome was calculated in acute myocardial infarction.

RESULTS
Of total 690 patients, 420 (60.86%) males and 270 (39.14%) females met inclusion criteria with mean age 55.90±10.19 years. There were142(20.60%) patients in the age range of 40-50 years, 296 (42.9%) patients in the age range of 51-60 years, 154 (22.3%) patients in the age range of 61-70 years, , 68 (9.9%) patients in the age range of 71-80 years, 30(4.3%) patients in the age range of 81-90 years.

In this study the mean serum HDL-C was 42.65 ±6.13 in males and 50.39±4.99 in females, mean serum triglycerides was 148.07±27.23 in males and 146.69±20.12 in females, mean fasting blood sugar was 101.06±20.70 in males and 105.35±23.72 in females, mean waist circumference was 88.29±9.19 in males and 90.27±13.35 in females, mean systolic blood pressure was 126.81±11.27 in males and 124.25±10.77 in females and mean diastolic blood pressure was 83.64±7.01 in males and 82.47±6.18 in females (table I). Frequency in terms of distribution of individual components of metabolic syndrome in relation to acute myocardial infraction (Table 2) showed that increased waist circumference, 46.85% (males 47.84%, females 52.20%) was most frequent followed by increased fasting blood sugar ,42% (males 44.91% and females 38.%), low HDL, 38.55% (males 40.30%, females 36.47%), increased TG levels, 33.30%(males 33.33% and females 32.10%) and hypertension , 31.30% (males 33.33%, females 28.71%).

42% were diagnosed with metabolic syndrome, (40% male and 44% female patients). ST segment elevation myocardial infarction was more common, 65.79 %, than Non-ST segment elevation myocardial infarction, 34.21%.

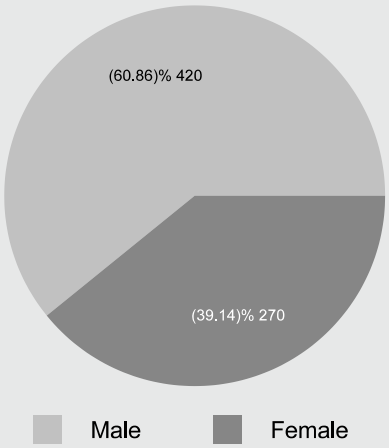
Table-I. Gender-wise distribution of variables. (N=690)

Variable	Males, N=420 Mean ± SD	Females, N=270 Mean ± SD	Total, N = 690 Mean ± SD
HDL-C	42.65±6.13	50.39±4.99	46.21±6.82
TG	148.07±27.23	146.69±20.12	147.43±24.19
FBS	101.06±20.70	105.35±23.72	103.04±22.21
Waist Circumference	88.29±9.19	90.27±13.35	89.20±11.33
Systolic blood pressure	126.81±11.27	124.25±10.17	125.63±11.10
Diastolic blood pressure	83.64±7.01	82.47±6.18	83.10±6.66

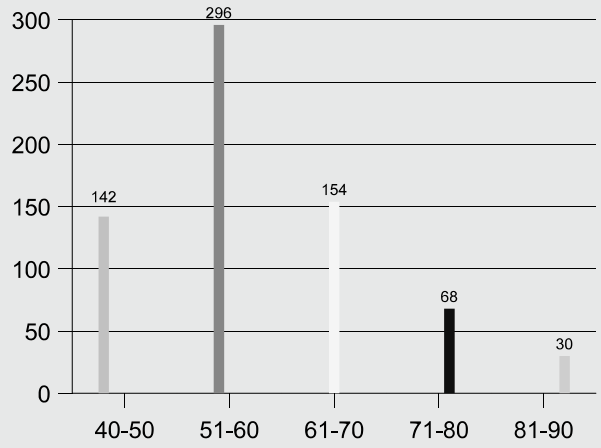
Table-II. Distribution by Frequency of components of metabolic syndrome in acute myocardial infarcation. (N=690)

Variable	Males (N=420)	%age	Female (N=270)	%age	Total (N=690)	%age
Serum HDL <40 in males and <50 in females	150	40.32	108	36.47	266	38.55
Serum Triglycerides >150	126	33.9	102	32.10	228	33.04
Fasting blood sugar >110	152	40.91	122	38.4	274	42.85
Blood pressure >130/85	124	33.33	98	28.71	216	31.30
Waist circumference >90cm in males and >80cm in females	188	47.84	166	52.20	276	46.85

Total Cases = 690



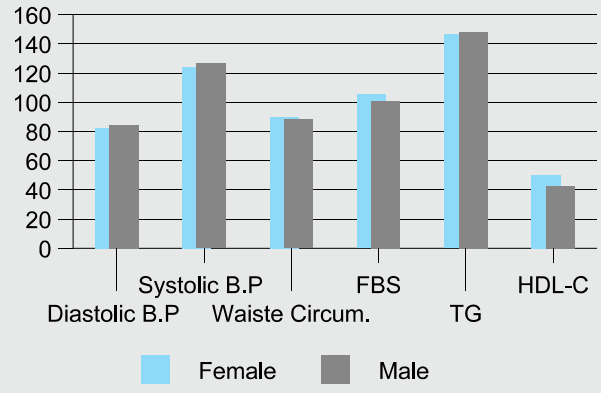
Age ranges of patients



DISCUSSION

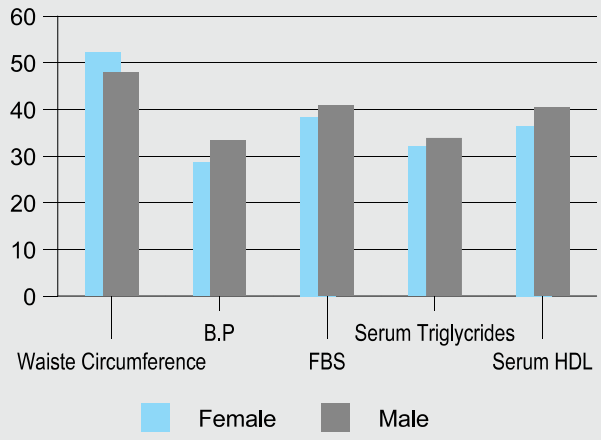
The term "metabolic syndrome" dates back to at least the late 1950s, but came into common usage in the late 1970s to describe various associations of risk factors with diabetes that had been noted as early as the 1920s. The Centers of Disease Control and Prevention (CDC) estimate that 20% of US adults have this syndrome. The prevalence of metabolic syndrome and increased risk for CHD in Pakistani adults is 35.2%. This study will help in generating awareness about modifiable risk factors of this syndrome.

Gender-wise distribution of variables (N=690)



Female	82.47	124.25	90.27	105.35	146.69	50.39
Male	84.63	126.81	88.29	101.06	148.07	42.65

Distribution by frequency of components of metabolic syndrome in acute myocardial infarction (N=690)



Female	52.2	28.71	38.4	32.1	36.47
Male	47.84	33.33	40.91	33.9	40.32

In this study, among 690 patients presenting as myocardial infraction, 288 (41.7%) were diagnosed with metabolic syndrome. Samra Yasmin, Nadeem Hayat Malik[10] et al reported the frequency of metabolic syndrome in cases of acute MI as 32% in men & 28% women whereas our study showed that 42% having metabolic syndrome with female preponderance, (40%male and 44 %female) and incidence was increasing with passing years. Onat A et al[11] reported a similar incidence in Turkish population. Wierzbicki AS, et al[12] reported a similar incidence in patients at tertiary referral cardiology unit in UK. In a meta-analysis of 21 studies, prevalence of the metabolic syndrome ranged from 23% to 46% with female preponderance among the general population with different levels of cardiovascular risk factors in majority of the studies. Identifying and preventing these at early stage at screening clinics is desirable[13].

An important feature of this syndrome is insulin resistance and hyperinsulinemia, suggesting that insulin itself is atherogenic[14]. DM may become apparent for the first time in susceptible patient during an acute MI and is associated with a poorer prognosis. Among all component of Metabolic Syndrome DM has the strongest association with CHD[15-16] However, individuals with diabetes but without metabolic syndrome had about the same frequency of IHD as those with neither[17].

The worldwide epidemic of type 2 diabetes is fuelled in large part by a parallel epidemic of obesity and physical inactivity[18]. Hypertension, diabetes, obesity and subsequent IHD spares no socioeconomic group and geographical region thus requiring priority preventive intervention, early diagnosis and intervention at primary health care level[19].

Obesity, particularly abdominal obesity, is associated with insulin resistance and fatty acid utilization, resulting in hyperinsulinemia and possibility hyperglycemia in genetically susceptible persons, adipocyte cytokines (adipokines) leading to vascular endothelial dysfunction, an abnormal lipid profile, hypertension, and vascular inflammation, all of which promote the development of atherosclerotic cardiovascular disease (CVD)[20,21,22].

Physical inactivity is a predictor of CVD events and related mortality. Many components of the metabolic syndrome are associated with a sedentary lifestyle including increased adipose tissue (predominantly central); reduced HDL cholesterol; and a trend toward increased triglycerides, blood pressure, and glucose in the genetically susceptible individuals and the incidence increases with age[23].

In addition to age, stress postmenopausal status,

smoking, low household income, high carbohydrate diet, no alcohol consumption, and physical inactivity are associated with an increased risk of metabolic syndrome in the presence of family history[24,25]. Polycystic ovary syndrome have an increased prevalence of metabolic syndrome, insulin resistance with compensatory hyperinsulinemia characterizes this syndrome[26].

Elevated triglyceride and low HDL cholesterol levels were as strong predictor of vascular events as the presence of other components of metabolic syndrome in a prospective study of a population of patients with angiographically determined coronary artery disease[27].

Elevated serum triglycerides, increased small LDL particles and a reduced level of HDL cholesterol (HDL-C) consist of atherogenic dyslipidemia[28]. Insulin resistance is central patho physiological process along with acquired factors such as excess body fat and physical inactivity[29,30]. Effective lifestyle change or if required relevant pharmacological intervention can reduce the risk. Aspirin with or without clopidogrel, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers and statins are the mainstays of intervention[31]. Drugs that target insulin resistance like metformin and the thiazolidinediones have demonstrated favorable effects on plasma lipids and the progression of intima-media thickness of the carotid arteries in patients with DM. In addition, rosiglitazone has demonstrated a benefit in endothelial parameters in patients with proven coronary disease but without DM[32].

Patients with unstable angina who have elevated bio markers of necrosis, such as CK-MB and cTrT (a much more specific and sensitive marker of myocardial necrosis), are at increased risk for death or recurrent MI. Elevated levels of these markers distinguish patients with NSTEMI from those with unstable angina. There is a direct relationship between the degree of troponin elevation and mortality. However, in patients without a clear clinical history of myocardial ischemia, minor troponin elevations have been reported and can be caused by congestive heart failure, myocarditis, or pulmonary embolism, or they may be false-positive readings[33].

The risk of renal disease and micro albuminuria appears to increase with the number of metabolic syndrome elements[34]. Hyperurecemia and gout is also associated with metabolic syndrome[35]. Sleep-related breathing disordered like obstructive sleep apnea suggest a relationship with insulin resistance as well as obesity[36].

The ATP III definition has been most widely adopted because of its clinical simplicity. The American Heart Association, the National Heart, Lung, and Blood Institute the US National Cholesterol Education Program Adult Treatment Panel III, the World Health Organization, the European Group for the Study of Insulin Resistance and the International Diabetes Federation (IDF) are other commonly practiced guidelines. The relative value of different metabolic syndrome definitions in terms of prognosis and management appears to be similar[37,38,39].

Therapeutic goals for management of metabolic syndrome include weight loss of 10% from baseline in 6-12 months, physical exercise of 30-60min per day, reduced intake of saturated fat and trans-fatty acids, LDL cholesterol <130mg/dL for moderate-risk patients and <70-100mg/dL for high-risk patients, lifestyle modification and pharmacotherapy, if necessary for type 2 diabetes mellitus, should be used to achieve near-normal HbA1C (<7%). For IFG, weight reduction and increased physical activity shall be encouraged. Blood pressure target should be lower than 130/85 mmHg (lower in high-risk patients, e.g. those with diabetes mellitus). If TG is 500 mg/dL, fibrate or nicotinic acid shall be initiated[40].

Weight reduction is optimally achieved with a multi modality approach including diet, exercise, and possible pharmacologic therapy, as with orlistat[41]. Dyslipidemia may be successfully treated through lifestyle modifications alone.

HMG-CoA reductase inhibitors (statins) are the drugs of first-choice. Ezetimibe is another lipid-lowering agent that may be used alone or in combination with a statin. Most patients with metabolic syndrome can tolerate low doses of nicotinic acid, although some find it difficult to take long term, because of flushing. In high-risk patients,

fibrates or nicotinic acid may be prescribed in addition to statins to further raise low HDL and reduce triglyceride levels[41,42].

The endocannabinoid system through endocannabinoid (CB1) receptor play newly emergent roles in the regulation of energy balance and body composition. Inhibition of CB1. Rimonabant is associated with weight loss, reduced waist circumference, improvements in atherogenic dyslipidemia, hypertension, hyperglycemia, reduced and levels of adiponectin thus a reduced prevalence of metabolic syndrome. Rimonabant in not yet available for use in the United States and its clinical role remains undefined[33,43].

Blood pressure is often reduced by lifestyle modifications, such as weight loss and a low-fat, low-salt diet, anti hypertensive medications may be required if hypertension persists.

CONCLUSIONS

The frequency of metabolic syndrome in acute myocardial infarction is significantly high in our population and compares well to other studies with female preponderance. The two most common components of metabolic syndrome were increased waist circumference and increased fasting blood glucose levels .The study showed that metabolic syndrome is an important risk factor for cardiovascular disease incidence. Detection, prevention, and treatment of the underlying risk factor of the metabolic syndrome should become an important approach for the reduction of cardiovascular disease in general population.

REFERENCES

1. Hu R, Ma CS, Nie SP, Lu Q, Kang GP, Du X, et al. **Effect of metabolic syndrome on prognosis and clinical characteristics in patients with coronary artey disease.** Chin Med J 2006;119:1871-6.
2. Yusuf S, Reddy S, Ounpuu S, Anand S. **Global burden of cardiovascular diseases.Part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization.** Circulation 2001;104:2746–53.
3. Ounpuu S, Anand S, Yusuf S. **The impending global epidemic of cardiovascular diseases.** Eur Heart J 2000;21:880–3.
4. Yusuf S, Ounpuu S. **Tackling the growing epidemic of cardiovascular disease in South Asia.** J Am Coll Cardiol 2001;38:688–9.
5. Yusuf S, Reddy S, Ounpuu S, Anand S. **Global burden of cardiovascular diseases. Part II: variations in cardiovascular disease by specific ethnic groups and geographic regions and prevention strategies.** Circulation 2001;104:2855–64.
6. Enas EA, Garg A, Davidson MA, Nair VM, Huet BA, Yusuf S. **Coronary heart disease and its risk factors in first-generation immigrant Asian Indians to the United States of America.** Indian Heart J 1996;48:343–53.
7. Nishtar S. **Prevention of coronary heart disease in south Asia.** Lancet2002;360: 1015–8.
8. **Executive summary of third report of National Cholesterol Education Program, expert panel on detection, evaluation and treatment of high blood cholesterol in adults (adult treatment panel-III of National Cholesterol Education Program.** JAMA 2001; 285 : 2486-97.
9. Chaudhary GM. **Metabolic syndrome X in diabetic patients-experience in 3275 diabetic patients at Jinnah Hospital, Lahore.** J Coll Physicians Surg Pak 2000; 10: 278-80.
10. Yasmin S, Mallik NH, Naveed T, Ali M, Noman A, Shakoor T. **Metabolic syndrome in patients with ischemic heart disease.** J Coll Physicians Surg Pak 2008;18:605-7.
11. Onat A, Ceyhan K, Ba A, Yar O, Erer B, Toprak S, et al. **Metabolic syndrome: major impact on coronary risk in a population with low cholesterol levels--a prospective and cross-sectional evaluation.** Atherosclerosis 2002; 165: 285-92.
12. Wierzbicki AS, Nishtar S, Lumb PJ, Lambert-Hammill M, Turner CN, Crook MA, et al. **Metabolic syndrome and risk of coronary heart disease in a Pakistani cohort.** Heart 2005;91;1003-7.
13. Galassi A, Reynolds K, He J. **Metabolic syndrome and risk of cardiovascular disease: a meta-analysis.** Am J Med 2006;119:812-9.
14. Stout RW. **Hyperinsulinemia and atherosclerosis.** Diabetes 1996; 45:45-6.

15. Khwaja AK, Rafique G, White F, Azam I. **Macrovascular complications and their associated factors among persons with type 2 diabetes in Karachi, Pakistan.** J Pak Med Assoc 2004; 54:60-6.

16. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. **The continuing epidemics of obesity and diabetes in the United States.** JAMA 2001; 286:1195-200.

17. Grundy SM, Cleman JI, Danels SR, Donato KA, Eckel RH, Franklin BA, et al. **Diagnosis and management of the metabolic syndrome.** Circulation 2005;112: 2735-52.

18. Ito H. Nakasuga K, Ohshima A, Maruyama T, Kaji Y, Harada M, et al. **Detection of cardiovascular risk factors by indices of obesity obtained from anthropometry and dual-energy X-ray absorptiometry in Japanese individuals.** Int J Obest Relat Metab Disord 2003; 27:232-7.

19. Qureishi R. **Metabolic syndrome: a silent killer.** Med Today 2005; 3: 3.

20. Reaven GM. **Banting lecture 1988. Role of insulin resistance in human disease.** Diabetes 1988;37:1595-607.

21. Rosenson RS. **Assessing risk across the spectrum of patients with the metabolic syndrome.** Am J Cardiol. 2005;96:8E–10E.

22. Tamsma JT, Jazet IM, Beishuizen ED, Fogteloo AJ, Meinders AE, Huisman MV. **The metabolic syndrome: A vascular perspective.** Eur J Intern Med. 2005;16:314–20.

23. Liu S, Manson JE. **Dietary carbohydrates, physical inactivity, obesity, and the metabolic syndrome as predictors of coronary heart disease.** Curr Opin Lipidol 2001;12: 395-404.

24. Szapary PO, Hark LA, Burke FM. **The metabolic syndrome: a new focus for lifestyle modification.** Patient Care 2002; 36: 75-88.

25. Avogadro A, Crepaldi G, Enzi G, Tiengo A. **Associazione di iperlipidemia, diabete mellito e obesit di medio grado.** Acta Diabetol Lat 1967;4:572-90.

26. Deugarte CM, Bartolucci AA, Azziz R. **Prevalence of insulin resistance in the polycystic ovary syndrome using the homeostasis model assessment.** Fertil Steril 2005; 83: 1454-60.

27. Joslin EP. **The prevention of diabetes mellitus.** JAMA 1921;76:79–84.

28. Haller H. **Epidemiology and associated risk factors of hyperlipoproteinemia (German).** Z Gesamte Inn Med 1977;32:124-8.

29. Ishaq M, Beg MS, Ansari SA, Hakeem A, Ali S. **CAD risk profiles at a specialized tertiary care center in Pakistan.** Pak J Cardiol 2003;14:61-8.

30. Samad A. **Coronary artery disease in Pakistan preventive aspects.** Pak J Cardiol 2003;14:59-60.

31. Sabatine MS, Cannon CP, Gibson CM, López-Sendón JL, **Montalescot G, Theroux P, et al. Addition of clopidogrel to aspirin and fibrinolytic therapy for myocardial infarction with ST-segment elevation.** N Engl J Med 2005;352:1179- 89.

32. Nathan DM, Buse JB, Davidson MB, Heine RJ, Holman RR Sherwin R, et al. **Management of hyperglycemia in type 2 diabetes: a consensus algorithm for the initiation and adjustment of therapy.** Diabetes Care 2006; 29: 1963-72.

33. Koh, KK, Han SH, Quon MJ. Inflammatory markers and the metabolic syndrome insights from therapeutic interventions. J Am Coll Cardiol 2005; 46:1978-85.

34. Zhang L, Zuo L, Wang F, Wang M, Wang S, Liu L, et al. **Metabolic syndrome and chronic kidney disease in a chinese population aged 40 years and older.** Mayo Clin Proc 2007; 82: 822-727. Phillips GB. **Relationship between serum sex hormones and glucose, insulin, and lipid abnormalities in men with myocardial infarction.** Proc Natl Acad Sci U S A 1977;74:1729-33

35. Choi HK, Ford ES. **Prevalence of the metabolic syndrome in individuals with hyperuricemia.** Am J Med 2007; 120: 442-7.

36. Young T, Skatrud J, Peppard PE. **Risk factors for obstructive sleep apnea in adults.** JAMA 2004; 291: 2013-6.

37. Saely CH, Koch L, Schmid F, Marte T, Aczel S, Langer P, et al. **Adult treatment Panel III 2001 but Not International Diabetes Federation 2005 Criteria of the Metabolic syndrome predict clinical cardiovascular events in subjects who underwent coronary angiography.** Diabetes Care 2006; 29: 901-7.

38. Grundy SM, Hansen B, Smith SC Jr, Cleeman JI, Kahn RA. **Clinical management of metabolic syndrome: report of the American Heart Association/National**

Heart, Lung, and Blood Institute/American Diabetes Association conference on scientific issues related to management. Circulation 2004; 109: 551-6.

39. Lorenzo C, Williams K, Hunt KJ, Haffner SM. **The National Cholesterol Education Program - Adult Treatment Panel III, International Diabetes Federation, and World Health Organization definitions of the metabolic syndrome as predictors of incident cardiovascular disease and diabetes.** Diabetes Care 2007; 30:8-13.

40. Grundy SM, Brewer HB, Cleeman JI, Smith SC, Lenfant C. **Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition.** Circulation 2004; 109:433-8.

41. Deedwania P, Barter P, Carmena R, Fruchart JC, Grundy SM, Haffner S, et al. **Reduction of low-density lipoprotein cholesterol in patients with coronary heart disease and metabolic syndrome: analysis of the Treating to New Targets study.** Lancet 2006; 368: 919-28.

42. Vega GL, Ma PT, Cater NB, Filipchuk N, Meguro S, Garcia-Garcia AB et al. **Effects of adding fenofibrate (200 mg/day) to simvastatin (10 mg/day) in patients with combined hyperlipidemia and metabolic syndrome.** Am J Cardiol 2003;91:956–60.Pi-Sunyer FX, Aronne LJ, Heshmati HM, Devin J, Rosenstock J.

43. **Effect of rimonabant, a cannabinoid-1 receptor blocker, on weight and cardiometabolic risk factors in overweight or obese patients: RIO-North America: a randomized controlled trial.** JAMA 2006; 295: 761-75.

Article received on: 18/04/2011 Accepted for Publication: 14/06/2011 Received after proof reading: 12/08/2011

Correspondence Address:
Dr. Ghazanfar Ali Sandhu
FCPS Medicine
Assistant Professor
Punjab Medical College
Faisalabad.
dr_sindhu@hotmail.com

Article Citation:
Sandhu GA, Iqbal S, Bilal A, Rana MM, Abdullah R, Qureshi FS. The frequency of metabolic syndrome in patients presenting with acute myocardial infarction. Professional Med J Sep 2011;18(3): 454-461.

An idea is salvation by imagination.

(Frank Lloyd Wright 1869 - 1959)