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Prevalence of Metabolic Syndrome in the Cases of Acute Myocardial Infarction and the Impact of its Components on in-Hospital Prognosis

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Introduction: All over the world, with the increasing risk factors like obesity and sedentary lifestyle, it is expected that incidence of Metabolic syndrome (MetS) will be increasing dramatically. It is well established that the five components of MetS also individually increase the risk of cardiovascular disease. *Aims and Objectives:* The aim of our study was to determine the incidence of MetS in the cases of acute myocardial ischemia; and to study the detrimental impact of syndrome and its components over the in-hospital outcome in these patients. *Methods:* A total of one hundred and fifty patients of acute myocardial infarction (AMI) were included in this study. *Results:* Among these 150 AMI patients (113 males, 37 females) one hundred and two (68%) met with ATP III criteria of MetS. The mean age for the entire cohort was 54.99±13.18 years. In our study high prevalence of MetS (68%) was observed in the patients of AMI and at least one risk factor of Mets was present in 98.67% of patients. Most common combination of MetS observed was a four combination of obesity+ hyperglycemia+elevated triglycerides+ low-HDl (OB+DM+HTG+low-HDL) in 16.67%. most common three components combination observed was OB+DM+HDL (8.82%). HT, DM and HDL showed significant correlation with adverse in-hospital outcome among AMI patients ($p<0.05$). *Conclusion:* The present study concludes that there is noticeable high occurrence of metabolic syndrome and its components in the cases of AMI. In our findings hypertension showed strong association with adverse outcome in these patients.

Keywords: Metabolic Syndrome; Acute myocardial infarction; Hyperglycemia; Elevated triglycerides.

Introduction

The clustering of metabolic and physiological abnormalities was first identified early in twentieth century. The metabolic syndrome (syndrome X/ Reaven's syndrome/insulin resistance syndrome) [1] consists of cluster of metabolic abnormalities which are also individually known to be associated with increased risk of coronary heart disease (CHD), type 2 diabetes, hypertension, stroke, polycystic ovarian disease and other chronic diseases [2]. There is also a genetic component in the etiology of this syndrome. The definition of metabolic syndrome (MetS) has differed slightly by different organizations. The one criteria by National Heart Lung and Blood Institute and the American Heart Association in the form of National Cholesterol Education Program's Adult Treatment Panel III (NCEP ATP III) report identifies presence of at least three of the five components, abdominal obesity, elevated triglycerides, reduced high-density lipoprotein cholesterol elevated blood pressure and elevated fasting blood sugar, for the diagnosis of metabolic syndrome [3].

ATP III identifies abdominal obesity as the key driver of the syndrome and whereas WHO identifies insulin resistance as the major underlying pathophysiological abnormality. Multiple studies have demonstrated a relationship between obesity and insulin resistance. Insulin resistance is clinically defined as inability of a known quantity of exogenous and endogenous insulin to increase to increase glucose uptake and utilization in an individual in similarity to normal individual [4]. Insulin, among its other actions, is a vasoactive hormone. Therefore in insulin resistance condition, in addition to diminished glucose uptake, there is impaired insulin-mediated vasodilatation and increased free fatty acids which altogether cause endothelial dysfunction [5]. In this hyperglycemic state, increased action of cytokines also occurs, cytokines cause a proinflammatory state in arteries and thereby play role in diabetes induced vascular damage. The release of plasminogen activator inhibitor-I, by excess adipose tissue, is thought to elicit a prothrombotic state. Hypertriglyceridaemia, apart from other severe consequences, causes widespread atherosclerosis in the body [6]. Small lipoproteins like HDL, LDL and VLDL continuously enter and leave the artery wall by diffusion and the rate of entry is in proportional to plasma concentration. A small amount of LDL and VLDL can be retained in subintimal space. In hypercholesterolaemia cholesterol deposition occur in arteries, by a rapid rate, which further cause inflammatory response and development of atherosclerosis [7]. Obesity and insulin resistance are linearly related with elevation in blood pressure. Investigators have shown epidemiological relationship of high blood pressure to coronary heart disease in both men and women [8]. Therefore inflammation is intimately associated with metabolic syndrome, development of atherosclerosis, and consequently to cardiovascular diseases (CVD) [9]. Hence investigators have studied and reported the relation of metabolic syndrome with incidence and outcome of myocardial ischemia [10-14]. The presence of MetS has resulted in almost three to four fold increase in CVD and also twofold increased morbidity risk in such cases [15,16].

Multiple studies have reported incidence of metabolic syndrome in various ethnic groups [17,18,19,20,21]. All over the world, with the increasing risk factors like obesity and sedentary lifestyle, it is expected that incidence of MetS will be increasing dramatically. The Covantry Diabetes Study, in London, found the prevalence to be 11.2%in Asian men and 8.9% in Asian women whereas it was 2.8% in white men and 4.3% in white women [22]. The age specific prevalence of metabolic syndrome has also been reported in most populations of the world [23,24,25]. Although some Indian investigators have reported the incidence of MetS in different regions of India but still that there is dearth in the data. There is need for larger studies on prevalence of MetS in Indian population and its influence on CVD among Indian population.

Our objective was to assess the incidence of acute myocardial infarction associated with the metabolic syndrome according to the NCEP: ATP III guidelines, with modified ethnic criteria to define central obesity in Indians (waist circumference male ≥ 90cm, female ≥ 80cm). An attempt is made to study the impact of metabolic syndrome on hospital outcomes and to assess the influence of individual components of MetS on the prognosis with reference to death and ventricular tachycardia in the patients of acute myocardial infarction.

Materials and Methods

An analysis on incidence of Mets, overall mortality and Cardio-vascular complication was carried out on one hundred and fifty patients of acute myocardial infarction (AMI). This study was conducted with the consent of patient and college ethical committee at the Department of Medicine, GMC, Kota. AMI was defined as the presence of at least 2 of the following 3 conditions: 1. typical chest pain for at least 30min, 2. typical electrocardiogram changes (at least 1 mm ST elevation in at least 2 adjacent leads, with subsequent evolution of the changes on electrocardiography (ECG) ST depression, T inversion and new pathological Q waves) and 3. diagnostic cardiac enzyme changes: elevation of serum creatine kinase level to more than twice the upper normal limit. Data was collected prospectively on demographics, cardiovascular risk factors, previous history of heart disease, any other pervious or ongoing medical treatment, any relevant clinical data and electrocardiogram.

Exclusion Criteria

We excluded patients who are diagnosed with septicemia, acute/chronic renal failure, any neoplastic disease, cardiomyopathies, or any other chronic or degenerative disease with life expectancy <2 years.

Metabolic Syndrome was characterized by the presence of three or more of the following five abnormalities set by the NCEP:ATP-III report with modifications:

Blood pressure was measured using a mercury sphygmomanometer. Waist circumference (WC)

Risk factor	Defining level
Abdominal obesity, given as waist circumference(OB)	
Male	≥90
female	≥80
Elevated triglycerides level	≥150
Reduced high-density lipoprotein cholesterol	
Male	40
female	50
Elevated blood pressure	≥130/≥85mmHg
Elevated fasting plasma glucose	≥100 mg/dL

was measured at the widest diameter between the xiphoid process of the sternum and the iliac crest. Lipid profiles and blood sugar were done, within 24 hrs of admission in hospital, in the blood sample drawn after overnight fasting. Patients were divided into two groups on the presence or metabolic syndrome 1. Mets and 2. Non-MetS. The adverse hospital outcome of AMI patients in reference to ventricular tachycardia/fibrillation , recurrent myocardial infarction, heart failure and death was noted and statistically analyzed.

Statistics

Where ever appropriate the data was calculated in mean±SD and in percentages. Data was analyzed for normal distribution using the Kolmogorov-Smirnov test. We performed comparisons between groups by unpaired t test. An analysis of categorical data was done by chi square test. A multiple logistic regression model was performed to examine the association between the metabolic syndrome and the adverse hospital outcome. Odds Ratio and Relative risk was calculated in the cases of AMI with reference to individual components of Mets.

Results

A total of one hundred and fifty patients of acute myocardial infarction were included in the study. Among these 150 AMI patients (113 males, 37 females) one hundred and two (68%) met with ATP III criteria of MetS. The mean age for the entire cohort (range 25-95 years) was 54.99±13.18 years. The mean age in male patients (range 25-95 years) was 54.41±12.96 years and the female patient (range 26-90 years) was 56.76±13.87 years, 54.42 years and 56.29 years respectively. The demographic and clinical data is shown in Table 1. ST segment elevation myocardial infarction (STEMI) was noted in majority (70.7%) and Non-ST segment elevation was observed in 29.3% of cases.

The most common risk factor found was abdominal obesity both in Mets and non-MetS. One or more component of Mets was present in 98.67% of all AMI cases. The prevalence of single and double components was 33% and 36% respectively. The prevalence of three or more components, the ATP III criteria for MetS, is given in Table 3.

Table 1: Demographic and clinical data in150 cases of AMI

Characteristic	Mets (n=102)	Non-Mets (n=48)	p-value	All cases (n=150)
Average Age	55.61	53.92	0.26	54.99±13.18
Male (n=109)	55.07	53.42		
Female (n=51)	55.08	55.17		
Smoker	41	14	0.19	55
Alcoholic	27	15	0.55	42
Mean systolic pressure	135±20.03	128.33±14.89	0.04	132.87±18.75
Mean diastolic pressure	84.4±8.51	80.75±6.02	0.008	83.23±7.97
Mean waist circumference	97.13±10.29	92.83±14.21	0.037	95.75±11.81
Mean fasting sugar	146.07±38.23	103.69±32.78	6.25E-10	132.51±41.51
Mean HTG	187.17±53.53	143.46±36.43	9.29E-07	173.18±52.72
Mean HDL	38.27±10.04	44.48±10.5	0.0007	40.26±10.56

Table 2: Individual incidence of risk factors of MetS in AMI cases

Mets components	MetS	Non-Mets	Total	
High Blood pressure (HT)	61	15	76	16.56%
Abdominal Obesity (OB)	82	26	108	23.53%
Diabetes mellitus (DM)	76	12	88	19.17%
Elevated triglycerides (HTG)	78	12	90	19.61%
Reduced HDL cholesterol (HDL)	77	20	97	21.13%

There are 16 different combinations possible from five MetS components. We found that the most common combination of three components was OB+DM+HDL. The most common overall combination was a four-component combination OB+DM+HTG+HDL. This result shows the significance between obesity and raised triglycerides.

Table 4 shows the adverse in hospital outcome during hospital in AMI cases. In these cases MetS appear to have an impact on ventricular fibrillation/ tachycardia ($p<0.05$).

There was a significant difference in the development of ventricular fibrillation/ tachycardia in hospital stay outcome of the patient. The outcome of logistic regression analysis of MetS risk factors with the adverse cardiovascular outcomes including fatality is shown in Table 5. Although in our results HT, DM and HDL showed significant correlation with adverse in-hospital outcome; but MetS did not appear to have an impact more than its independent components.

Table 3: Incidence of combinations in 102 cases of MetS

Number of components	Components of the metabolic syndrome	Incidence (n)	%
Three	HT+OB+DM	3	2.94%
	HT+OB+HTG	5	4.90%
	HT+OB+HDL	5	4.90%
	HT+DM+HTG	4	3.92%
	HT+DM+HDL	3	2.94%
	HT+HTG+HDL	3	2.94%
	OB+DM+HTG	6	5.88%
	OB+DM+HDL	9	8.82%
	OB+HTG+HDL	6	5.88%
	DM+HTG+HDL	3	2.94%
	Total	47	46.08%
Four	HT+OB+DM+HDL	3	2.94%
	HT+OB+DM+HTG	7	6.86%
	HT+OB+HTG+HDL	7	6.86%
	HT+DM+HTG+HDL	7	6.86%
	OB+DM+HTG+HDL	17	16.67%
	Total	41	40.20%
Five	HT+OB+DM+HDL+HDL	14	13.73%
	Grand Total	102	100%

Table 4: In hospital outcomes in AMI cases

Complication	MI with Mets (n=102)		MI without Mets (n=48)		Total	p-value
Heart failure	37	36.27%	15	31.25%	52	0.895537
Case fatality	23	22.55%	7	14.58%	30	0.2582
VT/VF	13	12.75%	1	2.08%	14	**0.036453**
Recurrent MI	4	3.92%	1	2.08%	5	0.561581
Stroke	1	0.98%	-	-	1	0.49456

Table 5: Logistic regression outcome between individual components of metabolic syndrome and occurrence of adverse cardiovascular outcomes including mortality

Components	ODDS Ratio (OR)	95%CI	p-value
Hyperglycemia	3.12	1.16 to 8.42	0.025
Low HDL	3.08	1.19 to 7.95	0.020
Hypertension	4.48	1.70 to 11.79	0.002
High triglyceride level	1.56	0.61 to 3.98	0.36
MetS	0.69	0.18 to 2.59	0.58
Obesity	1.82	0.71 to 4.68	0.21

Table 6: Association between components of MetS and adverse cardiovascular outcome including mortality

Components	Relative risk (RR)	95%CI
Hyperglycemia	1.35	1.05 to 1.73
Low HDL	1.25	0.97 to 1.62
Hypertension	1.39	1.10 to 1.75
High triglyceride level	1.17	0.92 to 1.48
Obesity	1.14	0.87 to 1.48

Analysis of the association between individual MetS components and the risk of developing future adverse prognosis is shown in table 6. In our results high blood pressure showed highest RR of 1.39 (95%CI 1.10-1.75) closely followed by hyperglycemia (RR-1.35) and low HDL (RR-1.25).

Discussion

A number of studies have evaluated the inter-relationship between individual characteristics of MetS as well as their effect on development of CVD. The presence of MetS is associated with an increased cardiovascular morbidity and mortality.

The major findings in our study are that firstly high prevalence of MetS (68%) was observed in the patients of acute myocardial ischemia and at least one risk factor of Mets was present in 98.67% of all AMI cases. Secondly we observed significant difference in all the five risk components between MetS group and Non-MetS group. Similar findings were reported by other authors [13,26,27]. Thirdly the most common combination observed in MetS was a four combination of OB+DM+HTG+low-HDL (16.67%). Whereas, in remigijus study it was OB+HT+HTG+low-HDL. Thirdly HT, DM and low HDL showed significant impact on the adverse in-hospital outcome in the AMI cases.

We obtained strong association between hypertension, hyperglycemia and low HDL with the overall adverse in-hospital outcome in AMI cases. Zeller et al obtained greater risk between hyperglycemia and low HDL cholesterol level association with severe heart failure [26]. Isomaa and associates, in a cohort study, reported that presence of the metabolic syndrome is strongly associated with an increased risk of CHD and MI as 2.96 and 2.63 respectively [16]. In similarity to our findings they reported that hypertension is associated with increased risk for CHD (RR 2.33; P <0.001).

A cohort study on 1209 Finnish men followed for almost 11.4 years, Kuopio Ischaemic Heart Disease Risk Factor Study, estimated prevalence of metabolic syndrome according to ATP III and WHO as 8.8% to 14.3% respectively. And the presence of metabolic syndrome in these men showed corresponding RR of 3.6 and 3.2 to die of CHD or CVD, respectively [15]. A cohort study on 1108 patients with CAD reported a MetS prevalence up to 51% [28]. And these patients who met MetS criteria along with severe CAD had a poorer prognosis compared with patients without MetS [28].

These findings suggest that MetS is associated with CAD and its prevalence is high in cases of both stable and unstable ischemic coronary syndromes. Whereas a retrospective study of 385 of AMI showed no statistical difference in the severity and or prognosis between patients with and without MetS [29].

In this study statistical significance impact of MetS was observed only in rhythm abnormality ($p < 0.05$). According to some authors controversy still exists, whether the impact of presence MeS is more than the sum of its individual risk components [30]. Inference driven from the available data is that a study on a larger scale and over a longer period is warranted in different population.

The prevalence of the metabolic syndrome is dependent on the definition of the different components of the syndrome. Adjusting the obesity criteria for Asian Indian has resulted in inclusion of more cases in MetS in our study. Thereafter, prevalence of MetS observed in patients of AMI was 68%. Zeller et al. reported a prevalence of 46% of MetS, according to NCEP ATP III criteria, in patients diagnosed with ST-segment elevation AMI. Their data revealed prognosis in terms of mortality was higher in patients of AMI with MetS than in patients of AMI without MetS [11].

Alejandra Madrid et al reported similar results with a MetS prevalence of 43.4% in patients, according to NCEP ATPIII criteria, in 971 patients diagnosed with acute coronary syndrome. Also prevalence of individual metabolic characteristics was significantly higher in patients with MetS than in the group of patients without MetS [13].

In this study the ST-segment elevation myocardial infarction (STEMI) was the most common diagnosis observed at the time of admission in 71.7% of all cases and this is comparable to the similar finding of 63.5% [13].

In our study three and four components were present in 46.08% and 40.20% respectively. This is in similarity to other findings; Remigijus et al reported three, four and five components in 48.99%, 38.45% and 12.55% of AMI cases respectively [31]. In further similarity to Remigijus [32] three components were more frequently detected in males (51.22%) than in females (25%), whereas four components were more common in females than males (60% vs 35.37%) and (15% vs 13.41%) respectively.

The most common MetS component among patients of AMI was abdominal obesity in our study, whereas in study by Anderson et al. [33] it was hypertension. To determine obesity criteria we used the adjusted values for Asian Indians (WS ≥ 90 in males and ≥ 80 in females) and this could the reason for inclusion of more obesity cases in our study.

Limitations of the study

Influence of sedentary life style, behavioral risk factors namely alcohol/smoking and a small study group is the limitation of this study. Acute metabolic stress derangement during acute MI may affect blood glucose and lipid levels and thereby can give alter the prevalence of MetS in AMI cases. In this study we evaluated the short period of in hospital adverse outcome in patients of AMI but a longer period follow up study will give better information on overall impact of MetsS on the prognosis of these patients.

Conclusion

The present study concludes that there is high prevalence of MetS (68%) among the AMI cases. At least one risk criteria was present in over 98% of AMI cases. There was significant difference in the five components of Metabolic syndrome between two groups- Mets and non-MetS. In the MI with MetS group the most common combination observed was OB+DM+HTG+HDL, this indicates interrelationship between these factors and development of CVD. Although no statistically significant detrimental impact of MetS over in-hospital adverse outcome, among AMI cases, was observed in this study; but considering the increasing prevalence of metabolic syndrome worldwide, our findings has important clinical implications.

References

1. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes. 1988;37(12): 1595-1607.
2. Qiao Q, Gao W, Zhang L, Nyamdorj R, Tuomilehto J. Review Article Metabolic syndrome and cardiovascular disease. Ann Clin Biochem. 2007;44:232-263.
3. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA. 2001;285(19): 2486-2497.
4. Folli F, Saad MJ, Velloso L, et al. Crosstalk between insulin and angiotensin II signalling systems. Exp Clin Endocrinol Diabetes. 1999;107(2):133-39. doi:10.1055/s-0029-1212088.
5. Ueda S, Petrie JR, Cleland SJ, Elliott HL, Connell JM. Insulin vasodilatation and the "arginine paradox". Lancet (London, England). 1998;351(9107):959-60. doi:10.1016/S0140-6736(05)60615-0.
6. Ference BA, Ginsberg HN, Graham I, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. A consensus statement fromthe European Atherosclerosis Society Consensus Panel. Eur Heart J. 2017;38(32):2459-72. doi:10.1093/eurheartj/ehx144.
7. Pentikainen MO, Oksjoki R, Oorni K, Kovanen PT. Lipoprotein lipase in the arterial wall: linking LDL to the arterial extracellular matrix and much more. Arterioscler Thromb Vasc Biol. 2002;22(2):211-217.
8. Franklin SS, Wong ND. Hypertension and cardiovascular disease: contributions of the framingham heart study. Glob Heart. 2013;8(1):49-57. doi:10.1016/j.gheart.2012.12.004.
9. Mente A, Yusuf S, Islam S, et al. Metabolic Syndrome and Risk of Acute Myocardial Infarction. A Case-Control Study of 26,903 Subjects From 52 Countries. J Am Coll Cardiol. 2010;55(21):2390-98. doi:10. 1016/ j.jacc.2009.12.053.
10. Kazemi T, Sharifzadeh G, Zarban A, Fesharakinia A. Comparison of components of metabolic syndrome in premature myocardial infarction in an Iranian population: A case -control study. Int J Prev Med. 2013;4(1):110-14.
11. Zeller M, Steg PG, Ravisy J, et al. Prevalence and Impact of Metabolic Syndrome on Hospital Outcomes in Acute Myocardial Infarction. Arch Intern Med. 2005;165(10):1192-98. doi:10.1001/archinte. 165.10. 1192.
12. Prasad SB, Fahrtash F, Malaiapan Y, Meredith IT, Cameron J. Prevalence, detection, and management of the metabolic syndrome in patients with acute

myocardial infarction: role of an obesity-centric definition. Cardiol Res Pract. 2010;2010. doi:10.4061/2010/814561.

13. Miller AM, Alcaraz Ruiz A, Borrayo Sanchez G, Almeida Gutierrez E, Vargas Guzman RM, Jauregui Aguilar R. Metabolic syndrome: clinical and angiographic impact on patients with acute coronary syndrome. Cir Cir. 2010;78(2):113-20.
14. Levantesi G, Macchia A, Marfisi RM, et al. Metabolic syndrome and risk of cardiovascular events after myocardial infarction. J Am Coll Cardiol. 2005; 46(2):277-83. doi:10.1016/j.jacc.2005.03.062.
15. Lakka H-M, Laaksonen DE, Lakka TA, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA. 2002;288(21):2709-16.
16. Isomaa B, Almgren P, Tuomi T, et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care. 2001;24(4):683-89. doi:10. 2337/diacare.24.4.683.
17. Lin CC, Liu CS, Lai MM, et al. Metabolic syndrome in a Taiwanese metropolitan adult population. BMC Public Health. 2007;7:1-5. doi:10.1186/1471-2458-7-239.
18. Ahmed AM, Elabid BEH, Elhassan KEH, Waggiallah HA. Metabolic syndrome among undergraduate students attending medical clinics for obligatory medical screening. Trop J Pharm Res. 2015;14(2):317-21. doi:10.4314/tjpr.v14i2.18.
19. Aguilar M, Bhuket T, Torres S, Liu B WRJ. Prevalence of the Metabolic Syndrome in the United States, 2003-2012. Jama. 2015;313(19):1973-74. doi:10.1001/jama.2015.4260.
20. Sawant A, Mankeshwar R, Shah S, et al. Prevalence of metabolic syndrome in Urban India. Cholesterol. 2011;2011. doi:10.1155/2011/920983.
21. Marjani A. Prevalence of Metabolic Syndrome in Turkmen Ethnic Groups in Gorgan. J Clin Diagnostic Res. 2013;7(9):1849-51. doi:10.7860/JCDR/2013/6035.3331.
22. Simmons D, Williams DR, Powell MJ. Prevalence of diabetes in a predominantly Asian community: preliminary findings of the Coventry diabetes study. BMJ. 1989;298(6665):18-21.
23. Hildrum B, Mykletun A, Hole T, Midthjell K, Dahl AA. Age-specific prevalence of the metabolic syndrome defined by the International Diabetes Federation and the National Cholesterol Education Program: the Norwegian HUNT 2 study. BMC Public Health. 2007;7:220. doi:10.1186/1471-2458-7-220.
24. Thiruvagounder M, Khan S, Sherif DS. The prevalence of metabolic syndrome in a local population in India. Biochem Medica. 2010;20(2):249-52.
25. Mozaffarian D, Kamineni A, Prineas RJ, Siscovick DS. Metabolic Syndrome and Mortality in Older Adults. Arch Intern Med. 2008;168(9):969. doi:10.1001/archinte.168.9.969.
26. Zeller M, Steg PG, Ravisy J, et al. Prevalence and impact of metabolic syndrome on hospital outcomes in acute myocardial infarction. Arch Intern Med. 2005;165(10):1192-98. doi:10.1001/archinte.165.10.1192.
27. Kazemi T, Sharifzadeh G, Zarban A, Fesharakinia A. Comparison of Components of Metabolic Syndrome in Premature Myocardial Infarction in an Iranian Population/: A Case -Control Study. 2018;4(1):110-14.
28. Solymoss BC, Bourassa MG, Campeau L, et al. Effect of increasing metabolic syndrome score on atherosclerotic risk profile and coronary artery disease angiographic severity. Am J Cardiol. 2004;93(2):159-164.
29. Babiæ Z, Pavlov M, Bulj N, et al. metabolic syndrome and outcome in patients with acute myocardial infarction. 2011;50(2):193-99.
30. Takeno M, Yasuda S, Otsuka Y. Impact of Metabolic Syndrome on the Long-Term Survival of Patients With Acute Myocardial Infarction. 2008;72(March):415-19.
31. Zaliunas R, Slapikas R, Babarskiene R, et al. The prevalence of the metabolic syndrome components and their combinations in men and women with acute ischemic syndromes. Medicina (Kaunas). 2008;44(7):521-28.
32. Remigijus Ž, Rimvydas Š, Luk D, Milvidait I, Laukaitien J. The prevalence of the metabolic syndrome components and their combinations in men and women with acute ischemic syndromes. 2008;44(7):521-28.
33. Anderson JL, Horne BD, Jones HU, et al. Which features of the metabolic syndrome predict the prevalence and clinical outcomes of angiographic coronary artery disease? Cardiology. 2004;101(4):185-193. doi:10.1159/000076695.