The Egyptian Journal of Hospital Medicine (January 2023) Vol. 90 (2), Page 3551-3556

Obesity Impacts on Acute Myocardial Infarction Patients Relevant to Hospital Management and Outcomes – Single Center Experience

Heba Ahmed Ragab Ibrahim[1]*, Sheeren Khaled[2], Ghada Shalaby[3]

[1]Department of Cardiology, Faculty of Medicine, Helwan University, Egypt

[2]Department of Cardiology, Faculty of Medicine, Benha University, Egypt

[3]Department of Cardiology, Faculty of Medicine, Zagazig University, Egypt

***Corresponding author:** Heba Ahmed Ragab Ibrahim**, Mobile:** (+20) 01129718113**, E-Mail:** hebaibrahim881@yahoo.com

ABSTRACT

Background: Obesity is considered a hazard factor for ischemic heart disease (IHD). However, it has been proposed that obese people may have a good prognosis after cardiac events, a phenomenon identified as the "obesity paradox". **Objective:** The aim of the present study was to examine the effect of body mass index (BMI) on clinical manifestations and outcomes following acute myocardial infarction (AMI).

Patients and methods: A retrospective, single center research was conducted at KAMC, Makkah during 2015-2020. AMI cases were allocated into three groups; Group 1 included non-obese (BMI<25) patients, Group 2 included overweight (25≥BMI<30) and Group 3 included obese (BMI≥30). **Results:** A total of 3018 AMI cases were enrolled in our study, of whom 834 (28%) patients were obese. Obese cases came with AMI at a younger age than overweight and non-obese ones respectively (P<0.001). Overweight and obese groups were more diabetics, hypertensive, and dyslipidemic. Contrast volume used during coronary angiography (CAG) was much elevated in obese patients than other groups (P<0.001). Overweight and obese patient groups were more in need for thrombus aspiration during CAG than non-obese (40, 38 %, and 22% respectively, P<0.001), and tirofiban use post-procedure (46%, 31%, and 24% respectively, P=0.008) in comparison to the non-obese patient group. Obese patients were less common to have multivessel disease compared to overweight and non-obese groups (24%, 43%, and 33%, respectively, P=0.01). Post-MI ejection fraction has a tendency to be much higher in obese and overweight patients in comparison to non-obese group. **Conclusion:** Obese patients presented with AMI at a younger age and consequently less common to have multivessel disease.

Keywords: Obesity, Acute myocardial infarction, Hospital management, Hospital complications.

INTRODUCTION

Obesity and overweight represent increasing epidemic proportions globally [1]**.** Worryingly, morbid obese cases are elevated even though more than overweight and obese ones. Mostly major coronary artery disease (CAD) hazard factors, involving dyslipidemia (particularly raised triglycerides and reduced high-density lipoprotein (HDL) and cholesterol, glucose disorder, metabolic syndrome, hypertension (HTN), diabetes mellitus (DM), or left ventricular hypertrophy, and physical inactivity, are negatively impacted by overweight and obesity. Furthermore, overweight and obese cases might have an independent hazard factors for CAD and are subjected to other cardiovascular accidents (CVA) that may go along with CAD, like atrial fibrillation, heart failure (HF), and unexpected cardiac mortality [1].

In the United States, in the previous several decades, the incidence of obesity has risen considerably. Regarding recent estimates, two-thirds of individuals are overweight or obese according to their body mass index (BMI) [2]**.** Obesity is connected with several adverse outcomes and higher risk comorbidities such as cardiovascular disease (CVD) and death [3], among chronic illness cases but, this link might be overcome. Observational research of HF cases indicate that obese persons might have a better short- and long-term outcome [4].

The term "obesity paradox," has been mentioned in a lot of studies which showed either a U-shaped or inverse linear relationship among BMI or the all-cause death among HF cases [5,6]. The reason of this connection is not clear, but there were many explanations have been estimated like patient's clinical characteristics. Fairly fewer studies have discussed that association in acute coronary syndrome (ACS) cases or evaluated communications among BMI and the other demographics or health-related factors [7].

The aim of the present study was to examine the effect of BMI on clinical manifestations and outcomes following acute myocardial infarction (AMI).

PATIENTS AND METHODS

This research was conducted as a part of STEMI registry approved and conducted in our center and still ongoing until now. A retrospective, single center research was conducted at KAMC, Makkah during 2015-2020.

Inclusion criteria: All AMI cases diagnosed between 2016 and 2019 were involved.

Exclusion criteria: AMI cases who did not undergo CAG because of case refusal, restrictions, or contraindications.

Demographics (age, gender, BMI, Arabic speaking, south Asian and pilgrims,), clinical presentation (DM, HTN, dyslipidemia, smoking, ischemic heart diseases (IHD), positive family history of CAD, history of cerebrovascular stroke (CVA), ECHO findings (left ventricular ejection fraction (LVEF) and LV thrombus),

Received: 24/10/2022
Accepted: 27/12/2022

laboratory investigations such as hemoglobin (Hb) on admission and on discharge, Hb drop, HBA1c, mean platelet volume (MPV), random blood sugar (RBS), serum sodium, serum potassium, creatinine on admission and discharge, blood urea nitrogen (BUN), blood HDL, LDL, total cholesterol and triglyceride) and CAG findings (AMI type, contrast volume, thrombolysis, tirofiban usage, fluro-time, left main or three vessel disease). During the hospitalization, problems (Pulmonary edema, ventilation, cardiogenic shock, duration of stay, cardiac arrest, and in-hospital mortality) were acquired and reported from the patient's medical records.

Group definition: BMI definition includes an individual's body weight divided by the square of their height. In this study, AMI cases were allocated into three groups; Group 1 included non-obese (BMI<25) patients, Group 2 included overweight (25≥BMI<30) and Group 3 included obese (BMI≥30). The classifications for BMI used in the current study were validated by the NIH and the World Health Organization (WHO) for all White, Hispanic, and Black individuals [8,9].

Ethical Consideration:

This study was ethically approved by the Institutional Review Board of the Faculty of Medicine, Mansoura University. Written informed consent was obtained from all participants. This study was executed according to the code of ethics of the World Medical Association (Declaration of Helsinki) for studies on humans.

Statistical Analysis

The collected data were introduced and statistically analyzed by utilizing the Statistical Package for Social Sciences (SPSS Inc.; Chicago, Ill), version 21.0 for windows. Qualitative data were defined as numbers and percentages. Chi-Square test, Fisher's exact test were used for comparison between categorical variables as appropriate. Quantitative data were tested for normality by Kolmogorov-Smirnov test. Normal distribution of variables was described as mean and standard deviation (SD), while non-parametric data was described as median and range. Independent sample t-test/ANOVA test was used for comparison of quantitative variables between groups. Linear regression analysis was also applied. P-value ≤0.05 was considered to be statistically significant.

RESULTS

Out of 3018 AMI cases entered our center, 871 (28%) patients were non-obese (Group 1), 1313 (44%) were overweight (Group 2) and 834 (28%) were obese (Group 3).

This study is a retrospective follow up study and it included 3018 AMI cases with a mean BMI of 27.9 (SD 5.2). A proportion (n=834, 28%) were considered obese with BMI>30. The obese cases presented with AMI at a relatively younger age (57.7±11.9 vs. 55.8±11.8 vs. 54.8±11.8 years, for non-obese, overweight and obese, respectively; P<0.001). Obese and overweight groups were more diabetics, hypertensive, dyslipidemic and smoker. AMI pilgrim patients were recorded less in obese patients compared to non-obese and overweight groups (33%, 45% and 22% for non-obese, overweight and obese, respectively; P<0.001). Overweight and obese cases had an elevated prevalence of Arabic speaking population but lower percentage of south Asian population. No significant variance was observed in both family history and history of IHD among the 3 groups (**Table 1**).

Table (1): Comparison of demographic data and risk factors between the two groups.

Variable	Non-obese 871 (28%)	Overweight 1313 (44%)	Obese 834 (28%)	P value
Age (Mean ± SD)	57.7 ± 11.9	55.8 ± 11.8	54.8 ±11.8	<0.001
Male	400 (29%)	400 (45%)	400 (66%)	<0.001
BMI (kg/m2) (Mean ± SD)	22.5 ± 1.8	27.2 ± 1.4	34.2 ± 4.6	<0.001
DM	400 (27%)	400 (43%)	400 (30%)	0.02
HTN	632 (27%)	632 (43%)	632 (31%)	0.002
Smoking	196 (27%)	196 (45%)	196 (28%)	0.5
Dyslipidemia	22 (23%)	22 (45%)	22 (31%)	0.03
CVA	26(3%)	39(3%)	17(2%)	0.8
Old IHD	17 (29%)	17 (43%)	17 (28%)	0.9
Positive family history of CAD	97 (4%)	97 (5%)	97 (5%)	0.4
Pilgrims	7 (33%)	7 (45%)	7 (22%)	<0.001
Arabic speaking	15 (24%)	15 (42%)	15 (34%)	<0.001
South Asian	400 (35%)	400 (46%)	400 (19%)	<0.001

BMI: Body Mass Index; DM: Diabetes Mellitus; HTN: Hypertension; CVA: Cerebro-Vascular Accidents; IHD: Ischemic Heart Disease; CAD: Coronary Artery Disease

Obese patients had elevated Hb on admission (P<0.001 for non-obese, overweight and obese respectively) and on discharge (P<0.001 for non-obese, overweight and obese respectively) however, Hb drop was not significantly variant among the three groups. Obese cases had reduced HDL (P=0.05 for non-obese, overweight and obese, respectively), whereas higher level of both triglycerides and LDL (P=0.04 for non-obese, overweight and obese, respectively) Even though no significant variance among the groups as regard creatinine on presentation, it was higher among obese patients on discharge (P=0.05 for non-obese, overweight and obese, respectively) (**Table 2**).

Table (2): Comparison of laboratory data between the studied groups.

Variable	Non-obese 839 (28 %)	Overweight 1310 (44%)	Obese 834 (28%)	P-value
Hb on admission (mg\dl)	13.5 ± 2	13.9 ± 2	14 ± 2	<0.001
Hb on discharge (mg\dl)	13 ± 2.1	13.4 ± 2	13.6 ± 2	<0.001
Hb drop ≥3 mg\dl	12 (5%)	444 (6%)	123 (5%)	0.5
HBA1c	7.7 ± 1.4	7.6 ± 1.3	7.7 ± 1.1	0.7
Creatinine on admission (mg\dl)	1.3 ± 0.3	1.2 ± 0.2	1.4 ± 0.3	0.1
Creatinine on discharge (mg\dl)	1.1 ± 0.2	1.2 ± 0.1	1.2 ± 0.1	0.05
HDL (mg\dl)	39.4 ± 9.4	37.7 ± 6.1	37.7 ± 9.1	0.05
LDL (mg\dl)	112 ± 27.3	115 ± 28.2	119 ± 28.3	0.04
TG (mg\dl)	120 ± 28.1	135 ± 31.1	135.6 ± 30.3	0.04
TC (mg\dl)	174.6 ± 43.2	177 ± 42.4	178 ± 42.3	0.6

Hb: Hemoglobin; MCV: Mean Corpuscular Volume; HBA1c: Glycosylated Hemoglobin; BUN: Blood Urea Nitrogen; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein; TG: Triglyceride; TC: total Cholesterol.

Contrast volume was much higher in the obese patients (P<0.001 for non-obese, overweight and obese, respectively). No significant variance among the three groups (P>0.05 for all) in complications post AMI such as pulmonary edema, cardiogenic shock and cardiac arrest. However, Post-MI ejection fraction has tendency to be elevated in obese and overweight cases in comparison to non-obese group (P=0.08 for non-obese, overweight and obese, respectively) (**Table 3**).

Table (3): Comparison of coronary angiographic results, in-hospital outcomes and mortality between the two groups

Variable	Non-obese 839 (28 %)	Overweight 1310 (44%)	Obese 834 (28%)	P-value
Thrombolysis	534 (19 %)	442 (18%)	2 (19%)	0.6
AWMI	318 (54 %)	1375 (55%)	1375 (55%)	0.7
LVEF	40 ±11.4	41 ± 10.8	41 ± 10.9	0.08
Contrast(ml)	122 ± 72.9	129 ± 79	138 ± 70	<0.001
Fluro-time(minute)	12.8 ± 27.9	21 ± 79	15.8 ± 85.5	0.5
Thrombus Aspiration	185 (22%)	524 (40%)	317 (38%)	<0.001
Tirofiban	201 (24%)	602 (46%)	259 (31%)	0.008
3VD	276 (33%)	563(43%)	200 (24%)	0.01
LM disease	25 (3%)	65 (5%)	17 (2%)	0.3
Number of stents	1.3 ± 0.9	1.3 ± 0.8	1.3 ± 0.8	0.7
LV thrombus	7 (0.9%)	10 (0.8%)	8 (1%)	0.8
Pulmonary edema	33 (4%)	39 (3%)	17 (2%)	0.1
Cardiogenic shock	50 (6%)	52 (4%)	25 (3%)	0.06
Ventilation	50 (6%)	65 (5%)	33 (4%)	0.3
Cardiac Arrest	50 (6%)	65 (5%)	33 (4%)	0.6
Length of stay (days)	6.4 ± 8.5	5.5 ± 8.7	5.2 ± 10.5	0.06
In-hospital Mortality	8 (1%)	9 (0.7%)	4 (0.5%)	0.1

AWMI: Anterior Wall Myocardial Infarction; LV: Left Ventricular; EF: Ejection Fraction; LM: Left Main; 3VD:3 Vessel Disease.

Independent factors such as age, female and DM were shown to be predictors for in-hospital death (P value 0.002, 0.04 and 0.05, respectively) (**Table 4**).

Table (4): Binary regression analysis for risk factors

Variable	B	S.E	Wald	df	Sig.	Exp(b)
Age	0.033	0.011	8.876	1	0.003	1.034
Female	-0.570	0.291	3.845	1	0.050	0.565
BMI	0.002	0.025	0.004	1	0.951	1.002
DM	0.530	0.313	2.861	1	0.050	1.698
Smoking	-0.269	0.0323	0.693	1	0.405	0.764
HTN	0.129	0.284	0.205	1	0.651	1.137
HBA1C	-0.047	0.058	0.651	1	0.420	0.954
BMI: Body Mass Index; DM: Diabetes Mellitus; HBA1C: Glycosylated Hemoglobin.						

DISCUSSION

The present study included 3018 patients, a sizable portion of them (834) of them were obese (BMI>30) presenting with AMI and all were subjected to CAG. It is a hard estimation of AMI incidence among obese people being about 28% that is considered a close agreement with other research [13]. Male gender is identified as one of the hazard factors CAD which goes with our results as males were preponderant in obese groups. This might be due to male dominance, smoking which is more prevalent among males and female estrogen protective effects [10].

Because high obese cases with ACS are younger, they might be directed to experts for secondary prevention, aggressive lifestyle adjustment for co-morbidities, and medical treatment optimization. This could be a possible cause to the greater survival of these people. In our research also higher BMI cases had a much younger population that may have contributed to their better result [11,12].

Obesity is a recognized risk factor for HTN, DM, dyslipidemia, and CAD. This was denoted in our research as overweight and obese case had greater incidence of both DM and HTN. Dyslipidemia was also observed to be widespread in overweight and obese AMI cases based on their greater triglycerides and LDL and lower levels of HDL than non-obese cases. Several earlier researches indicated an elevated LDL and triglyceride among obese cases compared to non-obese ones. These results were consistent with these ones [9,13].

With regards to ventricular systolic function post AMI, in our study LVEF was found to be less affected in overweight and obese population. Similarly, **Haridasan *et al.*** [14] stated that obese and overweight groups were significantly younger, with better LVEF and they were mostly males with an elevated HTN, DM, and hyperlipidemia prevalence.

Moreover, Obesity is known to be associated with impaired fibrinolysis and elevated platelet aggregation; thus interestingly, less Hb drop especially post-procedure was observed and this was mostly due to fewer procedurally related bleeding events. This also was agreed by **Mehta *et al.*** [15] in a previous study.

Regarding CAG findings, it was demonstrated that obese AMI cases had a preponderance of single vessel disease (SVD) and low prevalent three-vessel disease and this is observed in the present study. Therefore, suggesting that massive coronary involvement is rare in obese AMI patients as they presented at a younger age. On the other hand, A study was done in 2007 findings suggested that obese cases underwent CAG have a paradoxically decreased CAD burden (Duke myocardial jeopardy scores) and incidence of high-risk coronary anatomy (significant left main or triple vessel disease) in comparison to non-obese although an elevated DM, HTN, and dyslipidemia incidence denoting the occurrence of an obvious “obesity paradox”. But, after adjustment of additional important cardiovascular hazard factors, BMI was not considered an independent predictor of CAD burden or severity. It was detected that a negative correlation among BMI and age demonstrating higher BMI cases had been indicated for CAG at a younger age [16-19].

Interestingly, the incidence of coronary thrombus necessitating thrombus aspiration was elevated in overweight and obese cases than in non-obese ones. This conclusion may be attributable to a lower plaque load and a greater propensity for thrombus formation in obese AMI cases. This might be explained with a theory; as plasminogen activator inhibitor-1 is a recognized biochemical indicator of obesity which works for inhibition of endogenous fibrinolysis [20].

In transgenic mice with overexpression of plasminogen activator inhibitor 1, the incidence of coronary artery thrombi increases irrespective of established cardiac risk factors such as hyperlipidemia and HTN. Moreover, it is acknowledged that urokinase-type plasminogen activator, thrombomodulin and tissue plasminogen activator, are molecules which definitely obstruct procoagulant states within heart tissue [21,22]. It is convincing that these processes might be the explanation for high thrombus burden in obese population. In concordance with our study **Bakirci *et al.*** [23] who stated that BMI and a waist circumference were considered to be significantly elevated in coronary thrombus cases. Another study also commented that all coronary artery thrombus patient’s variables were positively associated with BMI, which indicates an increased possibility to thrombin formation [24].

Reaching to hospital outcome measures,

overweight and obese cases revealed reduced in-hospital mortality prevalence than non-obese. This is explained by presentation at younger age, less incidence of CVA and extensive CAD (triple vessel disease) in comparison to non-obese group of patients. Otherwise, there were observed no significant variance among the three groups as regard post AMI complications and this highlights the fact that all of our cases get the same level of care. Regarding to mortality issue, despite of BMI was not identified to be an independent predictor for the in-hospital deaths, but when the cases are allocated into BMI subgroups (normal weight, overweight, and obese), it was difficult to prove with significance obese cases have reduced in hospital death though that propensity has been detected (obesity paradox). Our data has been consistent with another data which was published [25], including SYNERGY [26], Merlin-TIMI 36 [27], and CRUSADE [28], that also refer to lower deaths in obese cases diagnosed as ACS. Romero-**Corral *et al.*** [27] detected that overweight CAD cases may have a reduced hazard of cardiovascular and total death compared to low and normal weight ones. Nevertheless, in morbid obese cases, who have an elevated risk of cardiovascular death, the trend vanishes.

Finally, BMI was not considered an independent predictor for the in-hospital mortality, and this was in agreement with research acknowledged significantly fewer individuals died in the hospital and overall while obese. But obesity appeared not to be an independent predictor of overall mortality [29,30].

STUDY LIMITATIONS

This is cross-sectional research without a control group; thus, risk variables and their statistical significance cannot be assessed. We did not study long-term outcomes since a significant proportion were pilgrims (returning to their nations with no further action taken). Additionally, the reality of a single center highly suggested is validation in a multicenter, large population with prolonged follow-up.

We noticed a significantly increased number of males in this research group in comparison to females. This may have altered the outcomes. Moreover, there were significant variance among the study groups regarding to age, HTN, dyslipidemia and EF, that may have participated as confusing factors. There was no data on cause-specific death primarily because of fairly limited sample size and relatively short follow-up period.

Many limitations to the clinical evaluation of obesity have been found, and in this sample, the united use of additional anthropometric parameters like waist circumference was investigated as a possible alternative. Also reviewing of abdominal obesity and the physical state of the cases within the designated categories may play a crucial role in shedding light on this conundrum.

CONCLUSION

Obese patients had multiple cardiovascular risk factors, presented with AMI at a younger age and consequently less common to have multivessel disease. They had more frequent intracoronary thrombus formation that requires aspiration during CAG and tirofiban infusion post procedure. Females, age and DM were all appeared as independent predictors for in-hospital death however, BMI was not considered as independent predictor for the in-hospital mortality. These all might reflect the BMI related clinical characters, management and outcomes in AMI patients and hence increase our awareness by those disparities to be taken into considerations.

Supporting and sponsoring financially: Nil.
Competing interests: Nil.

REFERENCES

1. **Lavie C, Milani R, Ventura H (2009):** Obesity and cardiovascular disease: risk factor, paradox, and impact of weight loss. J Am Coll Cardiol., 53:1925-32.
2. **Ogden C, Carroll M, Curtin L *et al.* (2006):** Prevalence of overweight and obesity in the United States, 1999-2004. JAMA., 295:1549-55.
3. **Peeters A, Barendregt J, Willekens F *et al.* (2003):** Obesity in adulthood and its consequences for life expectancy: a life-table analysis. Ann Intern Med., 138:24-32.
4. **Morse S, Gulati R, Reisin E (2010):** The obesity paradox and cardiovascular disease. Curr Hypertens Rep., 12:120-6.
5. **Fonarow G, Srikanthan P, Costanzo M *et al.* (2007):** An obesity paradox in acute heart failure: analysis of body mass index and inhospital mortality for 108,927 patients in the Acute Decompensated Heart Failure National Registry. Am Heart J., 153:74-81.
6. **Wu A, Eagle K, Montgomery D *et al.* (2009):** Relation of body mass index to mortality after development of heart failure due to acute coronary syndrome. Am J Cardiol., 103:1736-40.
7. **Cicoira M, Maggioni A, Latini R *et al.* (2007):** Body mass index, prognosis and mode of death in chronic heart failure: results from the Valsartan Heart Failure Trial. Eur J Heart Fail., 9:397-402.
8. **Michigan Medicine (1998):** Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. Obes Res., 6:51-209.
9. **WHO (2000):** Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser., 894:1-253. Available at: https://apps.who.int/iris/handle/10665/42330
10. **Sinha S, Krishna V, Thakur R *et al.* (2017):** Acute myocardial infarction in very young adults: A clinical presentation, risk factors, hospital outcome index, and their angiographic characteristics in North India-AMIYA Study. ARYA Atheroscler., 13:79-87.
11. **Buettner H, Mueller C, Gick M *et al.* (2007):** The impact of obesity on mortality in UA/non-ST-segment elevation myocardial infarction. Eur Heart J., 28:1694-701.

12. **Kadakia M, Fox C, Scirica B *et al.* (2011):** Central obesity and cardiovascular outcomes in patients with acute coronary syndrome: observations from the MERLIN-TIMI 36 trial. Heart, 97:1782-7.
13. **Cretu D, Udroiu C, Stoicescu C *et al.* (2015):** Predictors of in-Hospital Mortality of ST-Segment Elevation Myocardial Infarction Patients Undergoing Interventional Treatment. An Analysis of Data from the RO-STEMI Registry. Maedica (Bucur), 10:295-303.
14. **Haridasan V, Rajesh K, Sajeev C *et al.* (2015):** Study on correlation of obesity with short-term prognosis in acute myocardial infarction. Indian Heart J., 68:306-10.
15. **Mehta L, Devlin W, McCullough P *et al.* (2007):** Impact of body mass index on outcomes after percutaneous coronary intervention in patients with acute myocardial infarction. Am J Cardiol., 99:906-10.
16. **Gregory A, Lester K, Gregory D *et al.* (2017):** The Relationship between Body Mass Index and the Severity of Coronary Artery Disease in Patients Referred for Coronary Angiography. Cardiol Res Pract., 17:5481671. doi: 10.1155/2017/5481671.
17. **Niraj A, Pradhan J, Fakhry H *et al.* (2007):** Severity of coronary artery disease in obese patients undergoing coronary angiography: "obesity paradox" revisited. Clin Cardiol., 30:391-6.
18. **Rubinshtein R, Halon D, Jaffe R *et al.* (2006):** Relation between obesity and severity of coronary artery disease in patients undergoing coronary angiography. Am J Cardiol., 97:1277-80.
19. **Phillips S, Roberts W.** Comparison of body mass index among patients with versus without angiographic coronary artery disease. Am J Cardiol., 100:18-22.
20. **De Taeye B, Smith L, Vaughan D (2005):** Plasminogen activator inhibitor-1: a common denominator in obesity, diabetes and cardiovascular disease. Curr Opin Pharmacol., 5:149-54.
21. **Rosenberg R, Aird W (1999):** Vascular-bed--specific hemostasis and hypercoagulable states. N Engl J Med., 340:1555-64.
22. **Robinson S, Ludlam C, Boon N *et al.* (2007):** Endothelial fibrinolytic capacity predicts future adverse cardiovascular events in patients with coronary heart disease. Arterioscler Thromb Vasc Biol., 27:1651-6.
23. **Bakirci E, Degirmenci H, Duman H *et al.* (2015):** Increased Epicardial Adipose Tissue Thickness is Associated With Angiographic Thrombus Burden in the Patients With Non-ST-Segment Elevation Myocardial Infarction. Clin Appl Thromb Hemost., 21:612-8.
24. **WHO Expert Consultation (2004):** Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet., 363:157-63.
25. **Diercks D, Roe M, Mulgund J *et al.* (2006):** The obesity paradox in non-ST-segment elevation acute coronary syndromes: results from the Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the American College of Cardiology/American Heart Association Guidelines Quality Improvement Initiative. Am Heart J., 152:140-8.
26. **Mahaffey K, Tonev S, Spinler S *et al.* (2010):** Obesity in patients with non-ST-segment elevation acute coronary syndromes: results from the SYNERGY trial. Int J Cardiol., 139:123-33.
27. **Romero-Corral A, Montori V, Somers V *et al.* (2006):** Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. Lancet., 368:666-78.
28. **Madala M, Franklin B, Chen A *et al.* (2008):** Obesity and age of first non-ST-segment elevation myocardial infarction. J Am Coll Cardiol., 52:979-85.
29. **Hoit B, Gilpin E, Maisel A *et al.* (1987):** Influence of obesity on morbidity and mortality after acute myocardial infarction. Am Heart J., 114:1334-41.
30. **Kosuge M, Kimura K, Kojima S *et al.* (2008):** Impact of body mass index on in-hospital outcomes after percutaneous coronary intervention for ST segment elevation acute myocardial infarction. Circ J., 72:521-5.