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FREQUENCY OF CARDIOGENIC SHOCK IN PATIENTS PRESENTING WITH NSTEMI AND AGE LESS THAN 45 YEARS

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Abstract:

Introduction: *An inferior wall myocardial infarction also known as IWMI, or inferior MI, or inferior ST segment elevation MI, or inferior STEMI occurs when inferior myocardial tissue supplied by the right coronary artery, or RCA, is injured due to thrombosis of that vessel.* ***Objectives:*** *The main objective of the study is to analyse the Frequency of cardiogenic shock in patients presenting with NSTEMI and age less than 45 years.* ***Material and methods:*** *This descriptive study was conducted in Health Department, Pakistan during 2020 to 2021. Briefly, we analyzed the incidence rates by age groups, IHM, LOHS, and the use revascularization procedures, such as coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI), for men and women with an STEMI and NSTEMI.* ***Results:*** *The data was collected from 100 patients of both male and female. Patients with NSTEMI were older than those with STEMI, and presented more often history of hypertension, previous MI and coronary revascularization procedures, and clinical signs of metabolic syndrome.* ***Conclusion:*** *It is concluded that the long-term outcome for early survivors of CS is worse than that of patients without CS.*

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INTRODUCTION:

An inferior wall myocardial infarction also known as IWMI, or inferior MI, or inferior ST segment elevation MI, or inferior STEMI occurs when inferior myocardial tissue supplied by the right coronary artery, or RCA, is injured due to thrombosis of that vessel. When an inferior MI extends to posterior regions as well, an associated posterior wall MI may occur. Heart rate variability (HRV) has been known as a measurable parameter of the cardiac autonomic function. The cardiac autonomic innervation is heterogeneous and hence leads to different patterns of autonomic modulation [1]. The normal pattern of autonomic modulation is altered in the case of myocardial infarction, the pattern of alteration is not uniform, and it depends on the infarcted wall or region of the heart. This altered autonomic modulation starts within a few hours after the acute event. Cardiogenic shock is the most common cause of death in patients with acute myocardial infarction (AMI) and has a frequency of around 7-10%. It continues to cause significant mortality despite advances in pharmacological, mechanical and reperfusion endeavors [2].

Cardiogenic shock is defined as a systolic blood pressure of less than 90 mmHg for at least 30 minutes, which is secondary to myocardial dysfunction. It is associated with clinical signs of hypoperfusion, which include decreased urine output, altered mental status and peripheral vasoconstriction [3]. It is usually unresponsive to fluids, an important differentiating quality from other types of shock. However, it frequently responds to inotropes. The cardiac index (CI) and the pulmonary capillary wedge pressure (PCWP) are usually less than 2.2 l/min/m^2 and greater than 15 mmHg respectively [4].

Cardiogenic shock seems to occur with a greater frequency amongst patients with ST-segment elevation myocardial infarction (STEMI). It was observed that shock developed in 7.5% of patients with STEMI and in 2.5% of patients with non-ST-segment elevation myocardial infarction (NSTEMI). In another study, 4.2% of patients with STEMI and 2.5% of patients with NSTEMI had cardiogenic shock [5]. A significant delay precedes shock development in patients with NSTEMI. The underlying reason may be the rapid cell necrosis that takes place in STEMI contrasting with a slower cell loss in NSTEMI. Thus, the highest creatine kinase (CK) level is found in STEMI as compared to NSTEMI [6].

Objectives

The main objective of the study is to analyse the Frequency of cardiogenic shock in patients presenting with NSTEMI and age less than 45 years.

MATERIAL AND METHODS:

This descriptive study was conducted in MOMINABAD General hospital Karachi during 2020 to 2021. Briefly, we analyzed the incidence rates by age groups, IHM, LOHS, and the use revascularization procedures, such as coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI), for men and women with an STEMI and NSTEMI. The algorisms described by Sundararajan et al. were applied to identify the conditions and calculate the Charlson Comorbidity Index (CCI). The data was collected from 100 patients of both genders. All patients of anterior and inferior wall ST elevation myocardial infarction only who got thrombolytic therapy with age range 30-60 of both gender were included. Those patients who were not willing, autonomic neuropathy, thyrotoxicosis, hypothyroidism, diabetes mellitus, renal failure, previous history of MI, late presentation (after 24 hrs), cardiogenic shock, or known case of valvular heart disease, ventricular arrhythmia or atrial fibrillation, second- or third-degree AV nodal block, frequent PVCs (10/min), bigeminy or trigeminy and cerebrovascular accident were excluded.

Statistical analysis

The data was collected and analysed using SPSS version20.0. All the values were expressed in mean and standard deviation.

RESULTS:

The data was collected from 100 patients of both male and female. Patients with NSTEMI were older than those with STEMI, and presented more often history of hypertension, previous MI and coronary revascularization procedures, and clinical signs of metabolic syndrome. Patients with NSTEMI had greater number of critical coronary stenoses, revascularization was more often incomplete, and such patients presented more often with symptoms of heart failure on initial admission to the coronary care unit.

Table 1: Main data of patients, reported for the whole group and for patients with ST-elevation myocardial infarction and non-ST-elevation myocardial infarction

	All patients	**STEMI**	**NSTEMI**	**P[1]**	**P[2]**
Age, yr	63.5 ± 12.1	61.3 ± 12.5	67.4 ± 10.4	< 0.001	
Previous AMI, n (%)	60 (18)	21 (10)	39 (33)		< 0.001
Previous stroke, n (%)	11 (3)	5 (2)	6 (5)		0.193
Total cholesterol (under treatment), mg/dL	124.3 ± 26.0	123.4 ± 26.3	125.8 ± 25.5	0.424	
Metabolic syndrome, n (%)	204 (62)	124 (60)	80 (68)		0.011
BMI	27.2 ± 4.3	26.9 ± 3.7	27.9 ± 5.2	0.090	
AMI characteristics					
Anterior, n (%)	171 (52)	138 (66)	33 (28)		< 0.001
Inferior, n (%)	86 (26)	66 (32)	20 (17)		0.003
Other, n (%)	69 (21)	4 (2)	65 (55)		< 0.001
Coronary vessels with critical lesions, n	2.05 ± 0.85	1.94 ± 0.84	2.25 ± 0.85	0.002	
Incomplete revascularization, n (%)	151 (46)	87 (42)	64 (54)		0.031
Left ventricle ejection fraction, %	47.2 ± 10.3	47.8 ± 9.2	46.4 ± 12.0	0.222	
Patients with LVEF < 40%, n (%)	85 (26)	43 (21)	41 (35)		0.006
Patient with heart failure at initial admission, n(%)	37 (11)	15 (7)	22 (19)		0.002
Time before Holter, d	16.2 ± 9.6	15.6 ± 9.5	17.4 ± 9.8	0.117	
Therapy at time of discharge from hospital (number of cases, %)					
Aspirin	314 (96)	202 (97)	112 (95)		0.469
Clopidogrel	302 (93)	192 (92)	110 (93)		0.458
Warfarin	38 (12)	22 (11)	16 (14)		0.399
β-blocker	290 (89)	189 (91)	101 (86)		0.198
Ca-antagonist	38 (12)	18 (9)	20 (17)		0.022
ACE-inhibitor	264 (81)	180 (86)	84 (71)		0.001
AT-II-antagonist	43 (13)	16 (8)	27 (23)		< 0.001
Statin	314 (96)	201 (97)	113 (96)		0.893
Diuretic(s)	140 (43)	75 (36)	65 (55)		0.001
HRV parameters					
Mean heart rate, bpm	68.1 ± 10.0	69.1 ± 10.1	66.2 ± 9.7	0.016	

DISCUSSION:

HRV is accepted as clinical test when it was 13 confirmed that it is one of the strong and independent risk factor for cardiac arrhythmias and sudden death especially after acute myocardial infarction [7]. Sympathetic surge after 14 acute myocardial infarction is strong predictor of malignant arrhythmias and sudden death while parasympathetic activity has protective effect. Low heart rate variability shows sympathetic over activity which is one the powerful and independent risk for malignant arrhythmias and sudden death after acute myocardial infarction [8].

Most data available on the long-term outcome of CS patients surviving the early phase of AMI were obtained before major changes in AMI treatment were widely implemented [5]. They are derived from populations selected for inclusion into randomized trials, or less frequently from real-life populations. Patient inclusion was usually before the year 2000, at a time when outcomes were notably poorer than nowadays [9].

Despite improved early management, early mortality in CS patients remains considerably higher than that of patients without CS [2]. In prior studies the reported overall long-term survival of CS patients surviving the early period varies widely from 12% to 73% at 5 years. In the large GUSTO-1 population of STEMI patients, mortality at 11 years in early survivors was 45% in patients with CS, compared with 31% in patients without CS. Patients included in the trial were relatively young and had to fulfill the trial inclusion criteria, which included early presentation after symptom onset, and all received intravenous fibrinolytic therapy [10].

CONCLUSION:

It is concluded that the long-term outcome for early survivors of CS is worse than that of patients without CS. Cardiogenic shock is higher in patients with younger age group when they are diabetic, had underlying chronic kidney disease, and increased duration of hospital stay.

REFERENCES:

1. Erdogan A, Coch M, Bilgin M, Parahuleva M, Tillmanns H, Waldecker B, Soydan N. Prognostic value of heart rate variability after acute myocardial infarction in the era of immediate reperfusion. Herzschrittmacherther Elektrophysiol. 2008;19:161–168.
2. Wiliński J, Sondej T, Kusiak A, Wiliński B, Kameczura T, Bacior B, Czarnecka D. Heart rate variability in the course of ST--segment elevation myocardial infarction treated with primary percutaneous transluminal coronary angioplasty in elderly and younger patients. Przegl Lek. 2014;71:61–65.
3. Larosa C, Sgueglia GA, Sestito A, Infusino F, Niccoli G, Lamendola P, Mariani L, Santangeli P, Lombardo A, Crea F, et al. Predictors of impaired heart rate variability and clinical outcome in patients with acute myocardial infarction treated by primary angioplasty. J Cardiovasc Med (Hagerstown) 2008;9:76–80.
4. Brateanu A. Heart rate variability after myocardial infarction: what we know and what we still need to find out. Curr Med Res Opin. 2015;31:1855–1860.
5. Huikuri HV, Raatikainen MJ, Moerch-Joergensen R, Hartikainen J, Virtanen V, Boland J, Anttonen O, Hoest N, Boersma LV, Platou ES, et al. Prediction of fatal or near-fatal cardiac arrhythmia events in patients with depressed left ventricular function after an acute myocardial infarction. Eur Heart J. 2009;30:689–698.
6. Stein PK, Domitrovich PP, Huikuri HV, Kleiger RE. Traditional and nonlinear heart rate variability are each independently associated with mortality after myocardial infarction. J Cardiovasc Electrophysiol. 2005;16:13–20.
7. Steg PG, James SK, Atar D, Badano LP, Blömstrom-Lundqvist C, Borger MA, Di Mario C, Dickstein K, Ducrocq G, Fernandez-Aviles F, et al. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Eur Heart J. 2012;33:2569–2619.
8. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, Bax JJ, Borger MA, Brotons C, Chew DP, et al. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC) Eur Heart J. 2016;37:267–315.
9. Gan, S.C.; Beaver, S.K.; Houck, P.M.; MacLehose, R.F.; Lawson, H.W.; Chan, L. Treatment of Acute Myocardial Infarction and 30-Day Mortality among Women and Men. N. Engl. J. Med. 2000, 343, 8–15
10. Jánosi, A.; Ferenci, T.; Ofner, P.; Lupkovics, G.; Becker, D.; Faluközy, J.; Polgár, P.; K″oszegi, Z.; Horváth, I.; Jambrik, Z.; et al. Does Gender Have Prognostic Value Among Patients with Myocardial Infarction? Analysis of the Data from

the Hungarian Myocardial Infarction Registry. J. Women's Health 2018, 27, 1491–1498.