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Original Article

A Review of the Association Between Demographic, Laboratory, and Angiographic Findings and Major Cardiovascular Events in Patients Undergoing Elective Percutaneous Coronary Interventions

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ABSTRACT

Background: Coronary artery disease is the leading cause of mortality worldwide. With the increasing number of elderly people and the development of stents, the tendency to perform percutaneous procedures has increased, leading to an increased risk of complications known as major adverse cardiovascular events (MACE). This study aimed to assess the association between demographic, laboratory, and angiographic findings and MACE in patients undergoing elective angiography.

Methods: This cross-sectional descriptive study enrolled 300 patients older than 18 scheduled for elective angiography in Rajaie Cardiovascular Medical and Research Center between 2015 and 2016. Those who did not undergo stenting or needed surgery initially were excluded, leaving 207 patients. The demographic, laboratory, and angiographic data of these patients were collected, and they were then followed for 2 years.

Results: During a mean follow-up of 24 months, MACE occurred in 20 (9.60%) patients, with 1 patient experiencing 2 events. There was significant relationships between older age (P = 0.01), the female gender (P < 0.0001), the body mass index (P < 0.0001), total cholesterol (P = 0.01), low-density lipoprotein (P = 0.001), high-density lipoprotein (P = 0.003), triglycerides (P = 0.014), hemoglobin (P = 0.004), lower glomerular filtration rates (P < 0.0001), higher post-angiography troponin I (P < 0.0001), and fasting blood sugar (P < 0.0001) and MACE. There were also relationships between the incidence of cardiovascular diseases and the number of diseased vessels (P = 0.047) and between the need for repeated revascularization and the number of vessels (P = 0.01).

Conclusions: As many MACE risk factors are modifiable, we suggest that patients with the aforementioned risk factors be monitored more closely after percutaneous coronary interventions to predict and prevent the incidence of MACE. ***(Iranian Heart Journal 2020; 21(4): 93-102)***

KEYWORDS: Major adverse coronary events, PCI

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Coronary artery disease (CAD) is currently the leading cause of morbidity and mortality in the world, with over 37.4% of deaths in the United States and almost half of the deaths in Iran being due to cardiovascular diseases (CVDs). [1] Other than traditional medications, newer therapies such as percutaneous coronary interventions (PCI) and coronary artery bypass graft surgery (CABG) have emerged during the last century. With the increasing number of elderly people and the development of stents and newer equipment, the tendency to perform procedures such as PCI has increased. [1-3]

However, although systematic stent placement has led to a decreased number of symptoms in patients, there is still the risk of dissection and restenosis in patients undergoing PCIs, leading to the incidence of major adverse cardiovascular events (MACE). MACE has been widely defined in various studies; it includes a combination of heart failure and nonfatal myocardial infarction (MI), nonfatal stroke, stable angina, readmission due to cardiovascular causes, repeated PCI, repeated CABG, and all-cause mortality. [4]

The risk factors related to the incidence of MACE after PCI have been studied in various studies. These risk factors include the demographic details and past medical history of the patients such as the female gender, higher age, obesity, hyperlipidemia, smoking, hypertension, and diabetes mellitus (DM). [5,6] Furthermore, in previous studies, the number of embedded stents, the estimated glomerular filtration rate (eGFR), anemia, high levels of high-sensitivity C-reactive protein (hs-CRP), high levels of uric acid, more extensive multivessel coronary involvement, left ventricular ejection fractions of less than 40%, and diastolic dysfunction have been identified as other MACE risk factors. [7-14]

Despite the many advances in the field of interventional cardiology, the rate of MACE is still high. The comparisons of MACE between different CAD presentations, including stable ischemic heart disease (IHD), non-ST elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI) after PCI, during 2-year follow-up studies, revealed a similar number in STEMI and stable angina groups (6.8% vs 6.5%) and a relatively higher number in the NSTEMI group of patients (10.3%). This was justified by the higher age of patients with NSTEMI and the greater presence of cardiovascular and non-cardiovascular comorbidities in them. [15]

Therefore, regarding the high rate of PCI, the higher probability of MACE, and the higher comorbidities of patients with NSTEMI based on previous studies, in the present study, we sought to assess the association between demographic, laboratory, and angiographic findings and MACE in patients who underwent elective PCI due to SIHD.

METHODS

Rajaie Cardiovascular Medical and Research Center in Tehran, Iran, is a tertiary heart hospital and one of the largest cardiac centers in the Middle East. This center is affiliated to Iran University of Medical Sciences and serves patients with CVDs referred from all around the country. In this cross-sectional descriptive study, 300 consecutive patients who were admitted to this hospital for elective angiography between the years 2015 and 2016 were enrolled. The inclusion criteria consisted of being older than 18 years and undergoing elective angiography due to the symptoms of stable IHD.

Data were collected using a checklist including age, gender, the mean body mass index (BMI), the abdominal circumference, the hip circumference, past medical history

(DM, hypertension, hyperlipidemia, and smoking), hemoglobin (Hb) levels, eGFR, fasting blood sugar (FBS), the lipid profile, troponin I levels before and after PCI, and angiographic findings (vessel count and lesion location). Lesions with stenoses of between 50% and 70% and those over 70% were considered to be significant. Lesion locations were either ostial, proximal, middle, or distal. From these patients, those who did not undergo PCI or those who needed CABG initially were excluded. The study population was then followed for 2 years.

MACE was the endpoint of this study; it was assessed for a 24-month period after PCI. MACE was defined as death (whether cardiac or noncardiac), documented new MI defined as the symptoms of cardiac ischemia associated with either ST-elevation on electrocardiograms ($\geq$ 0.2 mV in leads V_1, V_2, and V_3 and $\geq$ 0.1 mV in the other leads) or without ST-elevation accompanied by a rise in cardiac enzymes (troponin or creatine kinase-MB), new coronary artery involvement defined as at least a new luminal stenosis of 50% or greater in an epicardial vessel, stroke (confirmed by a neurologist), or candidacy for PCI or CABG during the follow-up period. If a patient suffered more than 1 event, the event that occurred first was considered the endpoint.

The patients lost to follow-up were eventually excluded, and ultimately, 207 patients were studied.

The data analyses were conducted using the IBM SPSS software, version 25.0. Both descriptive and comparative statistics were used. The tests utilized were the χ^2 test, the Fisher exact test, the independent samples *t*-test, and the Mann–Whitney *U* test, with a significance level of 0.05.

RESULTS

The study population was comprised of 207 patients undergoing elective PCI. Of all the patients, 80.2% were male and the rest were female. The mean age of the patients was 58.0±8.35 years, BMI was 28.3±3.26 kg/m^2, and the mean abdominal-to-hip ratio was 1.0±0.06.

MACE occurred in 20 patients, with 1 patient experiencing 2 events. Two patients suffered a stroke during the 2 years of follow-up, 13 experienced an MI or a need for revascularization, and 6 died. One patient experienced both an MI and a stroke. No in-hospital mortality or emergency CABG was reported.

The baseline characteristics and laboratory data in the patients with MACE compared with the other patients and in different types of MACE are shown in Table 1. There were significant relationships between older age (P = 0.01), the female gender (P < 0.0001), BMI (P < 0.0001), hyperlipidemia (P = 0.029), higher total cholesterol (P = 0.01), lower low-density lipoprotein (LDL) (P = 0.001), lower high-density lipoprotein (HDL) (P = 0.003), higher triglycerides (P = 0.014), lower Hb (P = 0.004), lower GFR (P < 0.0001), and higher post-angiography troponin I (P < 0.0001) and MACE. Additionally, while no relationship was seen between the incidence of MACE and a previous history of DM, there was a significant relation between FBS and MACE (P < 0.0001). Furthermore, although there was no relationship between a history of hypertension or the mean arterial pressure and total MACE events (P = 0.499), CVDs, re-MI, and repeated need for revascularization were related to hypertension history (P = 0.007 and P = 0.029), while the incidence of stroke was not.

The primary angiographic findings in the 2 groups are depicted in Table 2 and Table 3. The number of involved vessels was not statistically different between the 2 groups (Table 2); nonetheless, when each type of MACE was compared individually, a

relationship was seen between them and the incidence of CVDs (P = 0.047) and also the incidence of re-MI or the need for repeated revascularization (P = 0.01). This was not true with respect to stroke. Moreover, there was a relationship between MACE and lesions located in the proximal portion of the left anterior descending coronary artery (LAD) (P = 0.005) and the ostial portion of the right coronary artery (RCA) (P = 0.05), and the patients with first diagonal involvements were less likely (P = 0.02) to experience MACE (Table 3).

Table 1: Baseline characteristics in the patients with and without MACE

	With MACE	Without MACE	*P*-value
Age (y)	58.5±7.90	52.6±10.76	*0.01*
Gender (male/female)	0.81	5.23	*<0.0001*
BMI (kg/m^2)	31.0±4.07	28.1±3.03	*<0.0001*
A/H ratio	1.0±0.04	1.0±0.06	0.806
Smoker	40%	20.3%	0.14
Hyperlipidemia	35.3%	15%	*0.029*
Hypertension	60%	55.1%	0.499
MAP	107.00±97.62	98.20±9.50	0.71
DM	10%	23.5%	0.256
Cholesterol	147.5±38.72	119.9±22.49	*0.01*
Triglycerides	157.90±48.19	123.4±43.58	*0.014*
HDL	37.5±5.88	42.2±15.28	*0.003*
LDL	57.2±18.44	80.7±34.69	*0.001*
FBS	112.4±5.66	99.3±9.17	*<0.0001*
GFR	105.9±30.17	120.7±27.65	*<0.0001*
Hemoglobin	13.1±1.77	14.5±1.68	*0.004*
Hs-CRP	4.8±0.96	5.6±4.96	*0.114*
Positive TnI (pre-angiography)	0	0	-
Positive TnI (post-angiography)	33.3%	27.30%	*<0.0001*
Total: 207	20	187	

MACE, Major adverse cardiovascular events; BMI, Body mass index; A/H ratio, Abdomen-to-hip ratio; MAP, Mean arterial pressure; DM, Diabetes mellitus; HDL, High-density lipoprotein; LDL, Low-density lipoprotein; FBS, Fasting blood sugar; eGFR, Estimated glomerular filtration rate; hs-CRP, High-sensitivity C-reactive protein; TnI, Troponin I

Table 2: Number of involved vessels in the patients with MACE

	With MACE			Total	*P*-value
	Stroke	MI/re-PCI	Death		
1VD	1 (1.2%)	1 (1.2%)	6 (7.06%)	8/85 (9.4%)	0.192
2VD	1 (3.5%)	11 (41.4%)	0	12/29 (41.4%)	
3VD	0	0	0	0	
	2 (0.10%)	12 (5.80%)	6 (30%)	20/207	
Total	20			20/207	

MACE, Major adverse cardiovascular events; VD, Vessel disease; MI, Myocardial infarction; PCI, Percutaneous coronary intervention

Table 3: Location of the lesions

Lesion location	With MACE	Without MACE	*P*-value
LAD ostial	0	28 (13.7%)	-
LAD proximal	7(35%)	20 (9. 7%)	0.005
LAD D1	1 (5%)	56 (27.1%)	0.02
LAD Mid	13 (65%)	128 (61.8%)	0.782
LAD distal	0	0	-
LCX ostial	0	0	-
LCX proximal	0	29 (14.0%)	-
LCX mid	0	29 (14.01%)	-
LCX distal	12 (60%)	129 (62.3%)	0.833
LCX OM1	1 (5%)	9 (4.4%)	0.607
RCA ostial	6 (30%)	26 (12.6%)	*0.05*
RCA proximal	4 (20%)	21 (10.1%)	0.175
RCA mid	12 (60%)	100 (48.3%)	0.231
RCA distal	4 (20%)	51 (24.6%)	0.435
Total: 207	20	187	

MACE, Major adverse cardiovascular events; LAD, Left anterior descending; LCX, Left circumflex; RCA, Right coronary artery

DISCUSSION

CAD is a huge cause of morbidity and mortality and financial burden, with almost 50% of deaths being related to CAD in Iran. PCI is currently a desirable alternative to CABG in many situations in that it is safer, more effective, and less invasive. Indeed, PCI is currently performed at an annual rate of 600 000 cases in Iran. Despite the efficacy and high feasibility of this procedure, PCI does not lead to the stoppage of the atherosclerosis process and unfortunately, stenosis is progressive and leads to many cases of MACE worldwide. [16] In the present study, we attempted to investigate the risk factors of MACE in patients with SIHD undergoing PCI at 2 years' follow-up in Rajaie Cardiovascular Medical and Research Center between 2015 and 2016.

The prevalence of MACE in our study was 9.7% and comprised 2 (0.1%) cases of stroke; 13 (5.80%) cases of revascularization (due to new symptoms or MI), which were mostly in patients with double-vessel; and 6 (5.80%) cases of death among patients with single-vessel disease, which is in contrast with other recent studies [12] but could be due to the fact that, in our study, we could not differentiate the causes of cardiovascular mortality from all-cause mortality in some cases.

The risk factors associated with MACE were older age, the female gender, high BMI, hyperlipidemia, high cholesterol, anemia, low eGFR, and high troponin I after angiography.

In studies by Aghajani et al [17] and Nakazato et al, [18] older age was identified as one of the causes of higher MACE rates, which is similar to our study. In the study by Nakazato et al, older patients had higher MACE event rates for nonobstructive, single-vessel, double-vessel, triple-vessel, and left main diseases. In another study by Xu et al, [19] the female gender was recognized as a risk factor for MACE in elderly patients, which was also confirmed in our study. Guo et al [20] reported that the prognosis of their male patients with CAD after PCI was better than that of their female patients, with the exception of long-term revascularization.

In the present study, we observed a significant relationship between high BMI and MACE but not with the abdominal-to-hip ratio. In contrast, Choi [21] concluded that abdominal obesity was associated with a

higher risk of MACE than general obesity without abdominal obesity. This could be justified by the fact that BMI cannot represent the distribution of body fat, and markers such as the wrist circumference and abdominal circumference are stronger predictors for this matter. The fact that our study was a retrospective study based on charted nursing data and measurements for each patient were performed by a different person could have had an effect on the results and led to measurement errors.

Further, we found a significant relationship between high cholesterol, high triglycerides, and low HDL and an increased risk of MACE. However, there was no relationship between MACE and the LDL level. In an investigation by Su et al, [22] given the widespread use of statins in the present age, despite decreased LDL levels, MACE levels did not fall significantly and non-LDL cholesterol measurements were superior in predicting cardiovascular risk to LDL measurements. Their finding is concordant with our study inasmuch as our patients were also mainly on statin therapy; therefore, the LDL level was not significant in predicting MACE rates.

Anemia was a marker of an increased MACE risk in this research. In other studies, including those by Wang et al [10] and Hosseini et al,[23] anemia was also shown to increase the risk of IHD. In a study by Jiang et al, [24] anemia was divided into 2 groups: anemia before and anemia after PCI. Anemia after PCI was proven to have a role in MACE in the mentioned study, while in our study, only pre-PCI anemia was assessed and proven to have a relationship with MACE.

The association between low eGFR and MACE was also of significance in our study, which chimes in with other studies, including those by Currie et al [25] and Huang et al. [26] Thomsen et al [27] concluded that chronic kidney disease (especially GFR <75 mL/kg/min) was a morbidity risk factor in their patients with CAD. The proposed mechanism for increased MACE following PCI in patients with low GFR is the presence of baseline risk factors such as DM, hypertension, mineral and bone abnormalities, anemia, and inflammation, and oxidative stress, as well as factors related to dialysis and sub-intima and media calcification. Coronary plaques in patients suffering from chronic kidney disease are frequently seen with inflammation in comparison with coronary plaques in patients without kidney disease. [28]

All the patients in our study were elective patients with SIHD and, thus, all had a negative baseline troponin I. Our research revealed that those with increased levels of troponin I after PCI had an increased 2-year risk of MACE. Previous studies, including those performed by Nageh et al [29] and Leutner et al, [30] have also shown a significant relationship between MACE and troponin I increase after PCI.

In our study, a previous history of DM was not associated with an increased risk of MACE, whereas high FBS was associated with a higher MACE risk. This means that patients with controlled DM are at a lower risk for MACE. In another study, [12] similar to our research, DM did not predict MACE risk; nevertheless, other studies, [31,32] have reported contradictory results regarding the relationship between MACE and DM. This difference may have been due to differences in the clinical diagnosis of DM and the variations in FBS levels and whether or not patients had a controlled FBS level with appropriate medication.

Torresani et al [33] assessed 135 patients and found no association between smoking and other background diseases and MACE. This finding is in line with our study, displaying that despite the higher prevalence of smoking in patients who experienced MACE compared with others (40% vs 20%), the

association between MACE and smoking was not significant. Other studies, including the INTERHEART study, [34] have also referred to smoking as a MACE risk factor. This may be in consequence of the fact that in our study, current smokers and ex-smokers were all classified as a smoker.

In most previous studies, hypertension was known as a risk factor for MACE after PCI. The thrombotic event leading to the incidence of MACE is induced by the influence of high blood pressure on the endothelial surface, leading to vascular dysfunction and the disruption of the communication between platelets and the arterial wall surface, as well as by the alteration in blood rheology and the increase in the number of hemostatic factors against fibrinolysis. [34,35] However, in our study, there was no relationship between a history of hypertension or the patients' mean arterial pressure and total MACE events (P = 0.499), while CVDs and re-MI or repeated need for revascularization were related to hypertension history (P = 0.007 and P = 0.029), although the incidence of stroke was not.

Alidoosti et al [36] concluded that MACE in PCI on the proximal portion of the LAD was not significantly different from PCI on the lesions in the non-proximal portions of the LAD. In contrast, in our study, PCI on the lesions in the proximal portions of the LAD and the RCA was significantly associated with the increased incidence of MACE. This finding does not tally with some previous studies [37,38] that reported that although thrombosis was usually found in the proximal part of the epicardial vessels, this relationship did not increase the risk of MACE. On the other hand, in the PROTECT study, [39] a total of 8709 patients were treated with drug-eluting stents via PCI, and although MACE, mortality, and the target vessel failure did not differ between the 2 groups of proximal LAD and non-proximal LAD lesions, high MI incidence rates following proximal LAD stenting by comparison with non-proximal LAD stenting were reported.

There are several limitations to the current study. First, this investigation is a retrospective cross-sectional study based on previously charted data, which may have skewed the results. Second, we could not differentiate cardiac mortality from all-cause mortality in some cases of death. Third, this is an observational study in a single center; hence, further research is required to confirm these findings in different populations.

CONCLUSIONS

The results of the present study indicated that higher age, the female gender, high BMI, a history of hyperlipidemia, higher total cholesterol, lower LDL, lower HDL, higher triglycerides, anemia, lower GFR, higher post-angiography troponin I, and higher FBS were associated with a higher risk of MACE. There were also relationships between the incidence of death and a lower number of diseased vessels and between the need for repeated revascularization and a higher number of diseased vessels. Moreover, the relationship between MACE and lesions located in the proximal portion of the LAD and the ostial portion of the RCA was of great significance, and patients with first diagonal involvements were less likely to experience MACE.

Fortunately, many of these factors are modifiable and should be identified and monitored early. We suggest that patients with the aforementioned risk factors be monitored more closely after PCI to predict and prevent the incidence of MACE.

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