ORIGINAL ARTICLE

Relationship between the extent of coronary artery disease and in-stent restenosis in patients with acute ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention

Primer perkütan koroner girişim ile tedavi edilen ST yükselmeli miyokart enfarktüslü hastalarda stent restenozu ile koroner arter hastalığının yaygınlığı arasındaki ilişkinin değerlendirilmesi

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ABSTRACT

Objective: The pathophysiological mechanism of in-stent restenosis (ISR) is different from atherosclerosis of native coronary arteries. The aim of this study was to evaluate the relationship between ISR and the extent of coronary artery disease (CAD), and to identify other risk factors associated with ISR in ST-segment elevation myocardial infarction (STEMI) patients.

Methods: A total of 372 consecutive patients presenting with first acute STEMI who were successfully treated with primary percutaneous coronary intervention within 12 hours from the onset of symptoms and who had an angiographic follow-up at 3 months were included in the study. The extent of CAD was calculated using the Gensini score.

Results: The incidence of ISR observed in our group of patients was 23.4% (n=87). The mean Gensini score was significantly higher in patients with ISR when compared with group without restenosis (69 [range: 51–90] vs 42 [range: 32–61]; p<0.001). The presence of diabetes mellitus, left ventricular ejection fraction (LVEF), and low-density lipoprotein cholesterol (LDL-C) level differed significantly between the 2 groups (p<0.05 for all). Stent diameter and stent length were found to be significantly different between the ISR group and the no-restenosis group (p<0.05 for both). In multivariate logistic regression analysis, the Gensini score, stent diameter, stent length, LVEF, and LDL-C were independently associated with ISR.

Conclusion: Despite the differences in the underlying pathophysiological mechanism of ISR and native coronary atherosclerosis, patients with a greater extent of CAD should be considered candidates for future stent restenosis.

ÖZET

Amaç: Stent içi restenoz (SİR) ile koroner aterosklerozu arasında altta yatan patofizyolojik mekanizmalar açısından önemli farklılıklar vardır. Çalışmamızda ST yükselmeli miyokart enfarktüsünde (STYME) koroner arter hastalığı (KAH) yaygınlığı ile SİR arasındaki ilişkinin değerlendirilmesini ve SİR ile ilişkili olabilecek diğer faktörleri tespit etmeyi amaçladık.

Yöntemler: Çalışmamıza ilk kez STYME ile başvuran, ilk 12 saatte primer perkütan koroner girişim ile başarılı bir şekilde tedavi edilen ve üçüncü ayda kontrol anjiyografisi yapılan 372 hasta dahil edildi. KAH yaygınlığı Gensini skoru ile tespit edildi.

Bulgular: Çalışmamızda SİR oranı %23.4 (87 hasta) olarak tespit edildi. Ortalama Gensini skoru SİR olan grupta belirgin derecede yüksek bulundu (69 [dağılım, 51–90] ve 42 [dağılım, 32–61], p<0.001). Diabetes mellitus varlığı, sol ventrikül ejeksiyon fraksiyonu (SVEF) ve LDL-C seviyeleri SİR olan ve olmayan grupta anlamlı bir şekilde farklı bulundu (tümü için, p<0.05). Ayrıca stent çapı ve stent uzunluğu da SİR olan ve olmayan grupta anlamlı düzeyde farklı bulundu (iki grupta da, p<0.05). Çok değişkenli regresyon analizi sonucunda Gensini skoru, stent çapı, stent uzunluğu, LVEF ve LDL-C ile SİR arasındaki ilişkinin bağımsız olduğu tespit edildi.

Sonuç: Stent içi restenoz ile koroner ateroskleroz için altta yatan patofizyolojik mekanizmalar farklı olsa da, koroner arter hastalığı yaygın olan hastalar SİR açısından riskli kabul edilmelidir.

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ST-segment elevation myocardial infarction (STEMI) is one of the leading causes of mortality and morbidity all over the world. The mortality rate has decreased in recent years due to the widespread use of primary percutaneous coronary interventions (p-PCI) rather than thrombolytic drug administration.[1,2] Nonetheless, in-stent restenosis (ISR) reduces the long-term efficacy of p-PCI in patients with STEMI and leads to recurrent coronary interventions.[3–5] Several demographic, clinical, and coronary angiographic variables have been shown to be associated with restenosis in either percutaneous transluminal coronary angioplasty (PTCA) or stent populations.[6–10] But most of these predictors were defined in studies with elective patients. STEMI is an acute condition and several different factors could account for ISR.

The underlying pathophysiological mechanism in ISR is quite different from atherosclerosis of native coronary arteries. The leading mechanism is accelerated neointimal hyperplasia in ISR. It has been speculated that although the underlying pathophysiological mechanism is different, patients with more severe coronary artery disease (CAD) might be at greater risk for stent restenosis. Therefore, the aim of this study was to evaluate the relationship between ISR and the extent of CAD and to identify other risk factors associated with ISR in STEMI patients undergoing p-PCI.

METHODS

Study design and population

This was a retrospective, single-center study. A total of 372 consecutive patients presenting with first acute STEMI, who were successfully treated with a bare metal stent (BMS) in a p-PCI within 12 hours of the onset of symptoms, and who had an angiographic follow-up 3 months later were included in the study. Due to the reinvestment policy of the state social security institution, STEMI patients were treated with BMS at our center until 2013. Outside the setting of clinical trials, follow-up angiography has mostly been restricted to patients with recurrent symptoms or positive functional testing. There have been some reports in the literature supporting the routine use of angiographic follow-up, but there is no internationally recognized clinical practice guideline supporting such routine follow-up. Only a few interventional centers have installed a routine angio follow-up protocol for their patients. At our institution, only a low-quality BMS that had relatively thicker struts was used during p-PCI until 2013. Due to concerns about the possible greater risk of restenosis with these stents, a routine angiography follow-up protocol was used at our center. Since the change in the reinvestment policy of the social security institution, high-quality BMS and drug-eluting stents (DES) can be used in PCI, and therefore, the follow-up angiography protocol is no longer in use. Only a small number of patients (n=18) were treated with DES and follow-up angiography was not performed. These patients were excluded from the present study.

Abbreviations:

BMS	*Bare metal stent*
CABG	*Coronary artery bypass graft*
CAD	*Coronary artery disease*
DES	*Drug-eluting stents*
DM	*Diabetes mellitus*
ISR	*In-stent restenosis*
IVUS	*Intravascular ultrasound*
LDL-C	*Low-density lipoprotein cholesterol*
LVEF	*Left ventricular ejection fraction*
p-PCI	*Primary percutaneous coronary intervention*
PTCA	*Percutaneous transluminal coronary angioplasty*
STEMI	*ST-segment elevation myocardial infarction*

Briefly, the diagnosis of STEMI was made using the criteria of the classic symptoms of coronary ischemia (chest pain lasting >30 minutes), detection of >1-mm ST-segment elevation in at least 2 contiguous leads, and elevation in cardiac biomarkers as defined in the guidelines of the American College of Cardiology and the European Society of Cardiology.[11] Since our institute is a tertiary center, p-PCI was the preferred reperfusion strategy in most STEMI cases. According to the follow-up protocol of the center, an angiographic follow-up at 3 months was routinely recommended for nearly all patients treated with stents. Previous coronary artery bypass graft (CABG) operation, previous STEMI or PCI, thrombolytic drug administration, DES implantation, failure of procedure during p-PCI, lack of angiographic control at 3 months, or inadequate data from the clinical recordings were all defined as exclusion criteria.

The extent of CAD was calculated using the Gensini scoring system[12] based on a recording made before p-PCI and incorporating the culprit lesion. Data of patient demographic variables, medical history, and clinical features, as well as major in-hospital adverse events were obtained from the hospital computer system. All angiograms were analyzed by visual estimation in a random sequence by 2 experienced

observers blinded to the patient's clinical data. A successful procedure was defined as residual stenosis <30% associated with Thrombolysis In Myocardial Infarction grade III flow. Stent length was the sum of the total length when more than 1 stent was used. Angiographically assessed ISR was defined as luminal narrowing of 50% or more occurring in the segment with the stent.[13,14]

The local ethics committee approved the study protocol.

Statistical analysis

The data were processed using SPSS for Windows, Version 16.0 statistical package (SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was applied to determine if the distribution of continuous variables was normal or not. Continuous variables with normal distribution were expressed as mean±SD and compared using Student's T-test. Non-normally distributed data were presented as median (25th-75th) percentiles and compared using the Mann-Whitney U test. Categorical variables were presented as percentages and compared using Pearson's chi-square or Fisher's exact test (when expected frequencies were less than or equal to 5). Multiple logistic regression using the backward LR method was performed to determine the best predictor(s) of ISR. Any variable with univariable test p value less than 0.25 was accepted as a candidate for the multivariable model, along with all variables of known clinical importance. A cut-off P value of <0.05 was set for results to be considered statistically significant.

RESULTS

The study population consisted of 372 patients with first acute STEMI. Of these 372 patients, 294 (79%) were male and 78 (20.9%) were female. The mean age of the study population was 58.40±12.52 years (range: 25–89 years). The baseline demographic characteristics are presented in Table 1. Laboratory findings, including complete blood count and standard biochemical parameters, are provided in Table 2. The incidence of ISR observed in our group of patients was 23.4% (87 patients), which is consistent with previous studies that have reported a range from 20% to 30%. The status of diabetes mellitus (DM), left ventricular ejection fraction (LVEF), and low-density lipoprotein cholesterol (LDL-C) level were significantly different between the 2 groups of those with ISR and those without restenosis. The mean Gensini score was significantly higher in patients with ISR than it was in those with no restenosis (69 [range: 51–90] vs 42 [range: 32–61]; p<0.001). Furthermore, stent diameter and stent length were significantly different between the ISR group and the no-restenosis group (p<0.05 for all) (Table 3).

Table 1. Basal demographic and clinical data

	In-stent restenosis (n=87)	No restenosis (n=285)	*p*
Age (years)	58 (47–66)	58 (50–68)	0.996
Male, n (%)	70 (80.5)	224 (78.6)	0.709
Smoker, n (%)	42 (48.3)	132 (46.3)	0.748
Heart rate (beats/minute)	80.73±14.85	79.95±17.55	0.105
Systolic blood pressure (mmHg)	130 (115–148)	130 (114–147)	0.947
Diastolic blood pressure (mmHg)	73 (66–86)	77 (70–89)	0.172
Left ventricular ejection fraction (%)	47 (42–50)	51 (45–56)	0.001
Diabetes mellitus, n (%)	29 (33.3)	55 (19.3)	0.006
Hypertension, n (%)	39 (44.8)	119 (41.8)	0.612
Hyperlipidemia, n (%)	22 (25.3)	59 (20.7)	0.364
Chronic renal disease, n (%)	4 (4.6)	10 (3.5)	0.747
History of coronary artery disease, n (%)	20 (23)	42 (14.7)	0.071

Continuous variables are presented as median (interquartile range) or mean (standard deviation); categorical variables are presented as number (percentage).

Table 2. Laboratory findings

	In-stent restenosis (n=87)	No restenosis (n=285)	*p*
Glucose, (mg/dL)	126 (150–188)	150 (108–161)	0.763
Urea (mg/dL)	35 (30–45)	33 (28–43)	0.241
Creatinine (mg/dL)	1.03 (0.90–1.13)	1.05 (0.86–1.10)	0.114
Uric acid (mg/dL)	5.0 (4.1–6.1)	5.53 (4.05–6.34)	0.374
High-density lipoprotein cholesterol (mg/dL)	41.02±8.79	43.10±29.54	0.637
Low-density lipoprotein cholesterol (mg/dL)	112.95±39.60	110.15±32.45	0.017
Triglycerides (mg/dL)	105 (77–146)	115 (79–166)	0.178
Albumin (g/dL)	3.35 (3.14–4.43)	3.49 (3.29–4.21)	0.776
White blood cell count ($10^3/\mu L$)	10.90 (9.10–13.30)	11.25 (9.30–13.50)	0.563
Hemoglobin (g/dL)	13.86±2.00	13.76±1.90	0.670
Hematocrit (%)	41.23±5.17	41.06±5.05	0.926
Platelet count ($10^3/\mu L$)	243 (195–286)	242 (197–290)	0.653
Neutrophil count ($10^3/\mu L$)	7.10 (5.23–10.1)	7.22 (5.20–10.0)	0.948
Lymphocyte count ($10^3/\mu L$)	2.10 (1.80–3.00)	2.00 (1.83–3.10)	0.749

Data are presented as median (interquartile range) or mean (standard deviation).

Table 3. Angiographic data

	In-stent restenosis (n=87)	No restenosis (n=285)	*p*
Coronary artery involvement			
Left anterior descending coronary artery, n (%)	41 (47.1)	117 (41.1)	0.547
Circumflex coronary artery, n (%)	17 (19.5)	56 (19.6)	
Right coronary artery, n (%)	29 (33.3)	112 (39.3)	
Stent diameter (mm)	2.5 (2.5–3.0)	3.0 (3.0–3.5)	<0.001
Stent length (mm)	23 (23–23)	18 (15–18)	<0.001
Gensini score	69 (51–90)	42 (32–61)	<0.001

Continuous variables are presented as median (interquartile range); categorical variables are presented as number (percentage).

In multivariate logistic regression analysis using the Gensini score, stent diameter, stent length, status of DM, history of CAD, LVEF, triglycerides, LDL-C, heart rate, diastolic blood pressure, creatinine (variables with p<0.25), the model revealed that the Gensini score (odds ratio [OR]: 1.029; 95% confidence interval [CI], 1.012–1.046), stent diameter (OR: 0.041; 95% CI, 0.013–0.125), stent length (OR: 1.255; 95% CI, 1.144–1.376), LVEF (OR: 0.928; 95% CI, 0.875–0.985) and LDL-C (OR: 1.015; 95% CI, 1.002–1.028) were independently associated with ISR in STEMI patients undergoing p-PCI (Table 4).

DISCUSSION

In our study, we found that STEMI patients with more severe CAD as evidenced by Gensini score were at greater risk for ISR at 3-month follow-up. In addition, stent length, stent diameter, LDL-C, and LVEF were all found to be independently associated with ISR.

Table 4. Variables associated with in-stent restenosis by the multivariable regression analysis

	B	OR	95% CI	*p*
Step 1				
Creatinine	-0.176	0.838	0.339–2.071	0.702
History of coronary artery disease	-0.394	1.483	0.487–4.512	0.488
Triglycerides	-0.002	0.998	0.993–1.004	0.534
Heart rate	0.010	1.011	0.984–1.038	0.438
Diastolic blood pressure	-0.016	0.985	0.957–1.013	0.284
Diabetes mellitus	0.960	0.383	0.138–1.060	0.065
Left ventricular ejection fraction	-0.078	0.925	0.871–0.984	0.013
Low-density lipoprotein cholesterol	0.016	1.016	1.002–1.031	0.025
Gensini score	0.031	1.032	1.014–1.050	<0.001
Stent length	0.231	1.260	1.148–1.383	<0.001
Stent diameter	-3.156	0.043	0.013–0.136	<0.001
Final Step				
Diabetes mellitus	0.871	0.419	0.163–1.075	0.070
Left ventricular ejection fraction	-0.074	0.928	0.875–0.985	0.015
Low-density lipoprotein cholesterol	0.014	1.015	1.002–1.028	0.027
Gensini score	0.029	1.029	1.012–1.046	0.001
Stent length	0.227	1.255	1.144–1.376	<0.001
Stent diameter	-3.205	0.041	0.013–0.125	<0.001

The variables included were the Gensini score, stent diameter, stent length, presence of diabetes mellitus, history of coronary artery disease, left ventricular ejection fraction, triglycerides, low-density lipoprotein cholesterol, heart rate, diastolic blood pressure, and creatinine ($p<0.25$).

Coronary ISR is a complex and multifactorial process.[15,16] Despite all the advances in stent technology in recent years, there has been no significant improvement in the rate of restenosis. In addition to the technical and mechanical factors associated with the procedure, inflammatory status before and after stent implantation is a significant risk factor for ISR.[6] However, the underlying physiopathological mechanism of intra-stent restenosis should specifically be addressed in patients with stable CAD and STEMI patients. As assessed by intravascular ultrasound (IVUS) studies, the leading mechanism in ISR is accelerated neointimal hyperplasia.[17] To date, the available data for ISR in STEMI patients is limited when compared with the stable condition.[18–20] STEMI is an emergency situation and several factors should be taken into account in terms of ISR. The infarct-related artery vessel diameter can be underestimated immediately after reopening, which may increase ISR risk due to implantation of undersized stents.[21] The possibility of technical mistakes during the intervention is also increased due to the emergency setting of STEMI. In addition, late stent malapposition is more common in patients treated with a stent.

Several demographic and clinical-based scoring systems have been developed to define ISR. But there is no established angiography-based scoring system to identify patients at higher risk for ISR. Nonetheless, some coronary angiographic variables, such as minimal lumen diameter, have been shown to be associated with restenosis in PTCA and stent populations.[6–10] However, the individual ability of these angiographic variables to predict outcomes is uncertain. The Gensini score was originally developed to quantify the severity of CAD; however, subsequent studies have demonstrated its ability to identify patients treated by PCI who are at high risk of adverse events.[22,23] The current study has demonstrated that patients with a higher Gensini score are at increased risk of ISR irrespective of other clinical variables. According to our study, each 1-point increase in the Gensini score was associated with a 3% increase in ISR risk.

However, this finding should be improved through a study combination that includes clinical and procedural features.

Because our purpose in the study was to assess the extent of CAD, we preferred to use the Gensini score instead of the SYNTAX score. The SYNTAX score is widely used for patients with CAD, but it focuses on complexity rather than the extent of CAD. It also has some limitations regarding the extent of CAD when compared with Gensini score. For instance, the percent diameter stenosis is not considered in scoring and a distinction is made only between occlusive (100%) and non-occlusive (50 99% stenosis) disease. Furthermore, vessels under 1.5 mm are not considered in the scoring algorithm.

In our population, increased stent length, reduced stent diameter, and reduced LVEF were also found to be associated with restenosis in multivariate analysis. Consistent with previous studies,[24,25] our data indicated that ISR was more frequent in patients with longer stents. This finding can be explained with several mechanisms: longer stent use is associated with a marked inflammatory reaction that can cause an increase in neointimal proliferation. In addition, long stents may not fully expand and some portions may not come into contact with the vascular walls, which results in an increased risk of both thrombosis and restenosis.[26] Reduced stent diameter was also independently associated with ISR in our study. This finding was similar to previous reports that revealed a greater risk for ISR when the diameter of the target vessels is <3 mm.[27,28] It is a well-known fact that patients with reduced LVEF are at high risk for mortality and mortality. But the relationship between reduced LVEF and ISR is unknown, as most ISR studies excluded this group of patients. Gioia et al.[29] demonstrated improved survival with DES implantation in patients with severe left ventricular dysfunction compared with BMS implantation. Our study revealed an independent association between ISR and reduced LVEF in STEMI patients treated with BMS during p-PCI. This finding may support the use of DES in STEMI patients with reduced LVEF. Finally, it was determined in the present study that LDL-C was also independently associated with ISR. LDL-C is a well-known risk factor for coronary atherosclerosis. This finding can be accepted as supporting our results regarding the relationship between the extent of CAD and ISR.

Study limitations

Our study has some inherit limitations. First and foremost, it has a retrospective and single-center design. Patients with a history of CABG, previous PCI, or STEMI and reperfusion with thrombolytic drug administration were excluded. Thus, the results of this study may not be extrapolated to all patients. In clinical practice, angiographic scores are calculated by visual lesion assessment (rather than laboratory determination), which would likely lead to greater intra-observer and inter-observer variability. Furthermore, quantitative computerized analysis and IVUS were not performed. Despite these limitations, we believe that our results indicate a need for further studies.

Conclusion

Despite differences in the underlying pathophysiological mechanism between ISR and native vessel atherosclerosis, patients with a greater extent of CAD should be considered candidates for future stent restenosis. Therefore, physicians should be alert during the follow-up of those patients with more severe CAD. However, prospective randomized studies with a large sample and multicenter participation are required to support our findings.

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