Primary angioplasty in a high-volume tertiary center in Turkey: in-hospital clinical outcomes of 1625 patients

Türkiye'de yüksek hacimli üçüncü basamak bir merkezde primer anjiyoplasti: 1625 hastanın hastaneiçi klinik sonuçları

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ABSTRACT

Objectives: We evaluated in-hospital results of primary percutaneous coronary intervention (PCI) in a high-volume tertiary center.

Study design: We retrospectively evaluated 1625 patients (1323 males, 302 females; mean age 56.0±11.6 years) who underwent primary PCI for acute ST-elevation myocardial infarction between January 2006 and April 2008. All coronary angiography procedures were performed using the femoral artery route. In-hospital clinical and angiographic results were recorded.

Results: On admission, 23% of the patients had diabetes mellitus, 49.6% had anterior myocardial infarction, and 4.9% had cardiogenic shock. The mean duration of pain was 171.2±121.2 minutes, and the mean door-to-balloon time was 31.6±7.2 minutes. Infarct-related artery was the left anterior descending artery in 49.7%, multivessel disease was present in 40.9%, TIMI 2/3 flow was present in 23.6%, and high-grade thrombus was observed in 66.8%. Primary PCI involved balloon dilatation (5.7%) and stent implantation (94.3%). The incidence of angiographic no-reflow was 11.9%. The mean hospital stay was 5.2±3.3 days. All-cause mortality occurred in 71 patients (4.4%). Other in-hospital events were reinfarction (1.4%), target vessel revascularization (1.9%), hemorrhagic/ischemic stroke (0.6%), stent thrombosis (1.2%), major bleeding (3.8%), blood transfusion (4.8%), heart failure (10.5%), atrial fibrillation (4%), and ventricular tachycardia (3.9%).

Conclusion: Primary PCI is an effective method in achieving complete revascularization of the infarct-related artery. Successful in-hospital results not only depend on the experience and equipment of the center, but also on how rapidly reperfusion is achieved.

ÖZET

Amaç: Yüksek hacimli üçüncü basamak bir merkezde primer perkütan koroner girişim (PKG) uygulamalarının hastaneiçi sonuçları değerlendirildi.

Çalışma planı: Ocak 2006 - Nisan 2008 tarihleri arasında, akut ST yükselmeli miyokart enfarktüsü nedeniyle primer PKG uygulanan 1625 hasta (1323 erkek, 302 kadın; ort. yaş 56.0±11.6) geriye dönük olarak değerlendirildi. Tüm koroner anjiyografi işlemleri femoral yol ile yapıldı. Hastaların hastaneiçi klinik sonuçları ve anjiyografik bulguları kaydedildi.

Bulgular: Hastaların %23'ünde diyabet, %49.6'sında anteriyor miyokart enfarktüsü, %4.9'unda kardiyojenik şok vardı. Ortalama ağrı süresi 171.2±121.2 dakika, ortalama kapı-balon zamanı 31.6±7.2 dakika idi. Enfarktüsle ilişkili arter hastaların %49.7'sinde sol ön inen arter bulunurken, %40.9'unda çokdamar hastalığı, %23.6'sında TIMI 2/3 akım, %66.8'inde yüksek dereceli trombüs saptandı. Primer PKG olarak balonla genişletme (%5.7) ve stent yerleştirme (%94.3) uygulandı. İşlem sonrasında hastaların %11.9'unda anjiyografik olarak yeniden akım sağlanamadı. Hastanede yatış süresi ortalama 5.2±3.3 gün idi. Yatış sırasında tüm nedenlere bağlı ölüm 71 hastada (%4.4) görüldü. Hastaneiçi diğer olaylar şunlardı: Tekrarlayan enfarktüs (%1.4), hedef damar revaskülarizasyonu (%1.9), hemorajik/iskemik inme (%0.6), stent trombozu (%1.2), önemli kanama (%3.8), kan nakli (%4.8), kalp yetersizliği (%10.5), atriyal fibrilasyon (%4) ve ventrikül taşikardisi (%3.9).

Sonuç: Primer PKG, enfarkt ile ilişkili arterde tam reperfüzyon sağlanmasında etkili bir tedavi yöntemidir. Hastaneiçi sonuçların başarısı sadece merkezin deneyimi ve donanımına değil, aynı zamanda reperfüzyonun ne kadar kısa sürede sağlandığına da bağlıdır.

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Despite the advances in pharmacoinvasive strategies, acute myocardial infarction is still a serious health care problem with high mortality and morbidity rates. In parallel to the advances in percutaneous coronary intervention and anticoagulant-antiaggregant therapies, the frequency of major adverse cardiovascular events including death, reinfarction, and re-revascularization has decreased both in-hospital and long-term follow-up.[1] Primary PCI is the treatment of choice in the treatment of ST-elevation myocardial infarction.

We aimed to evaluate the results of primary PCI procedures of patients admitted to our hospital, which is a high-volume center among primary PCI facilities.

PATIENTS AND METHODS

Study population

We retrospectively evaluated 1781 patients who were admitted to our emergency department with STEMI between January 2006 and April 2008, and underwent emergent cardiac catheterization. Inclusion criteria were admission within 12 hours of onset of chest pain (18 hours for cardiogenic shock), and ≥1 mm ST-segment elevation in at least two consecutive leads (2 mm for V1-3) or new or presumed-new left bundle branch block accompanying chest pain. After baseline evaluations, 156 patients were excluded from the study with the following reasons: presence of TIMI 3 flow on coronary angiography and absence of chest pain (n=77), decision for bypass surgery (n=51), angiographic normal coronary arteries (n=5), less than 50% stenosis in infarct-related artery (n=14), and administration of thrombolytic therapy (n=9) because of failure to cross the target lesion due to peripheral arterial tortuosity, peripheral arterial disease, coronary tortuosity, severe angulation, or absence of coronary flow after balloon dilatation. The remaining 1625 patients (1323 males, 302 females; mean age 56.0±11.6 years) formed the study group. The study was approved by the ethical committee of our center and written informed consent was obtained from all patients before PCI.

Data collection

Demographic variables and clinical properties of the patients including age, sex, history of hypertension, diabetes mellitus, tobacco use, chronic obstructive pulmonary disease, previous coronary intervention, previous myocardial infarction, drug use, dyslipidemia, and duration of chest pain were derived from hospital records. Door-to-balloon time, angina-to-door time, presence of preinfarction angina, and Killip class were also recorded.

Abbreviations:

DTB *Door-to-balloon*
ECG *Electrocardiography*
MACE *Major adverse cardiovascular events*
PCI *Percutaneous coronary intervention*
STEMI *ST-elevation myocardial infarction*

Baseline hemogram, and the levels of cardiac enzyme-troponin, urea, creatinine, cholesterol, blood glucose, and electrolytes were obtained from all patients, and creatine kinase, creatine kinase-MB, and troponin levels were measured every six hours until peak levels were determined and every 24 hours thereafter. Daily hemogram measurements were also obtained. Estimated glomerular filtration rate was calculated using the MDRD (Modification of Diet in Renal Disease) formula.[2] Baseline, postprocedural, and 60-minute electrocardiograms were obtained and ECG follow-up was continued twice daily until discharge. All patients were monitored in the coronary care unit until clinical stabilization was achieved after the procedure. Besides medical treatment and hemodynamic monitoring, transthoracic echocardiography (Vivid system 3, GE, Hortan, Norway) was performed to evaluate mechanical complications and left ventricular ejection fraction with the modified Simpson method.

Coronary angiography and angioplasty procedures

All patients received 300 mg aspirin, a loading dose of clopidogrel (300 or 600 mg), and 10,000 unit intravenous heparin before the procedure. Use of pre-procedural glycoprotein IIb/IIIa inhibitor (tirofiban) was left to the operator's discretion and was applied as 10 μg/kg bolus followed by 0.15 μg/kg/min intravenous infusion. Intravenous or oral nitroglycerine was given unless contraindicated.

All coronary angiography procedures were performed using the femoral artery route. Selective left and right coronary angiography was performed and the lesion severity and coronary anatomy were evaluated. Coronary lesions were evaluated from at least two non-foreshortened angiographic views. More than 50% stenosis was labeled as hemodynamically significant. Stenosis percentage, preprocedural TIMI (Thrombolysis in Myocardial Infarction) flow, presence of collateral flow, and angiographic morphologic findings were recorded. Lesion localization and angiographic thrombus load were also noted. Length of lesion, reference vessel diameter, and postprocedural minimal luminal diameter measurements were made by quantitative coronary angiographic analysis. The lesions were crossed by 0.014 wires and percutaneous transluminal coronary angioplasty or intracoro-

nary stent implantation were performed with standard methods. In patients with preprocedural TIMI 0 flow, angiographic measurements were made after the lesion had been crossed with the wire. Intracoronary 150-300 mcq nitroglycerine was administered after balloon dilatation unless contraindicated. Measurements were made after nitroglycerine administration. Post-procedural TIMI flow grade and myocardial blush grade were assessed. Nonionic contrast media was used. After percutaneous coronary intervention, all patients were followed-up in the intensive care unit and 1 mg/kg enoxaparin (twice daily), 150 mg/day aspirin, 75 mg clopidogrel and, if required, tirofiban infusion were applied.

Definitions

ST-elevation myocardial infarction was defined as the presence of chest pain lasting for at least 30 minutes with ≥1 mm ST-segment elevation in at least two contiguous derivations or typical chest pain together with new or presumed-new left bundle branch block. Door-to-balloon time was defined as time from the first admission of the patient to the emergency department to the first intracoronary balloon inflation. Reperfusion time was defined as time from the onset of chest pain to the first balloon inflation.

Anemia was defined according to the World Health Organization definition (admission hemoglobin <13 g/dl in men and <12 g/dl in women).[3] Hyperlipidemia was defined as a history of hyperlipidemia diagnosed and/or treated by a physician, documentation of total cholesterol >200 mg/dl, low-density lipoprotein cholesterol ≥130 mg/dl, high-density lipoprotein cholesterol <30 mg/dl or admission cholesterol >200 mg/dl. Cardiogenic shock was defined as the presence of peripheral hypoperfusion signs (cold shivering, paleness, oliguria, loss of consciousness, etc.) accompanied by low systemic blood pressure (<90 mmHg) that were resistant to fluid administration and required inotropic therapy and/or intra-aortic balloon pump.

Multivessel disease was defined as the presence of at least 50% stenosis involving two or more major epicardial coronary arteries. Good collateral flow was defined as Rentrop grade 2-3 collateral flow. Thrombus burden was defined according to the TIMI thrombus classification.[4] No-reflow was defined as the presence of TIMI ≤2 flow after the procedure, without residual stenosis, spasm, dissection, or distal embolization. Myocardial blush grade was evaluated based on the standard methods.[5] The severity of bleeding was evaluated according to the TIMI bleeding classification.[6]

Table 1. Baseline clinical characteristics (n=1625)

	n	%	Mean±SD
Age (years)			56.0±11.6
Age ≥70 years	246	15.1	
Male	1323	81.4	
Female	302	18.6	
Diabetes mellitus	373	23.0	
Hypertension	646	39.8	
Hyperlipidemia	638	39.3	
Family history of coronary artery disease	333	20.5	
Current smoker	871	53.6	
Previous percutaneous coronary intervention	127	7.8	
Previous coronary artery bypass grafting	48	3.0	
Previous myocardial infarction	91	5.6	
Estimated glomerular filtration rate <60 ml/min/1.73 m^2	113	7.0	
Dialysis history	13	0.8	
Anemia	395	24.3	
Killip class ≥2	252	15.5	
Cardiogenic shock on admission	79	4.9	
Preinfarction angina	399	24.6	
Cardiac arrest before admission	38	2.3	
Anterior wall infarction	806	49.6	
Stent thrombosis on admission	45	2.8	
Time from symptom onset to hospital arrival (min)			171.2±121.2
Door-to-balloon time (min)			31.6±7.2
Reperfusion time >4 hours	481	29.6	

Stent thrombosis was defined according to the Academic Research Consortium criteria.[7] Reinfarction was defined as progression of new pathologic Q waves and at least two-fold elevation in creatine kinase level in patients whose cardiac enzymes returned to normal and more than 50% elevation in creatine kinase level in patients whose cardiac enzymes remained elevated. Major adverse cardiovascular events were defined as all-cause mortality, reinfarction, target vessel revascularization, and stroke (hemorrhagic and ischemic). ST resolution was evaluated on the ECG obtained at postprocedural 60 minutes.[8] The percent resolution of the sum of ST-segment elevation in the infarct leads was classified as complete (≥70%), partial (30%–70%), or no resolution (<30%).

RESULTS

Demographic and clinical characteristics of the patients are given in Table 1. Localization of myocardial infarction was anterior in 806 patients (49.6%) and 252 patients (15.5%) were admitted with a Killip class of ≥2. Cardiogenic shock was present on admission in 79 patients (4.9%). The mean time from symptom onset to hospital arrival was 171.2±121.2 minutes and the mean DTB time was 31.6±7.2 minutes.

Angiographic data

Angiographic findings and procedural data are presented in Table 2. The infarct-related artery was left anterior descending artery in 807 patients (49.7%) and left main coronary artery in six patients (0.4%). Multivessel disease was present in 665 patients (40.9%). The target lesion was located in the proximal segment in 900 (55.4%) patients. TIMI 2/3 flow was present in 384 patients (23.6%). High-grade thrombus (TIMI thrombus score ≥4) was observed in 1085 patients (66.8%).

All patients received a loading dose of clopidogrel before the procedure, being 600 mg in 1598 patients (98.3%) and 300 mg in 27 patients (1.7%). Tirofiban was administered to a total of 1328 patients (81.7%) before or after PCI. Intracoronary stent implantation was performed in 1533 patients (94.3%); of these, 300 patients (18.5%) underwent direct stenting. Bare metal stents were used in 1457 patients, and drug-eluting stents were used in 76 patients (4.7%). The mean stent diameter was 3.1±0.4 mm and the mean stent length was 21.4±9.0 mm. Two or more stents were implanted for infarct-related artery in 236 patients (15.4%), and 231 patients (14.2%) underwent multivessel PCI, of which, 26 patients (1.6%) underwent noninfarct-related artery intervention at the same session. The incidence of angiographic no-reflow was 11.9%. Myocardial blush grade 3 was observed in 532 patients (41.9%).

Postprocedural echocardiographic findings

Postprocedural mean left ventricular ejection fraction of the patients was measured as 46.9±8.2% (Table 3). Mechanical complications were observed in 12 patients (0.7%). Two patients (0.1%) had free wall ruptures. Postprocedural 60-minute ECG showed complete ST resolution in 937 patients (57.7%).

In-hospital events

In-hospital events are summarized in Table 4. All-cause mortality was 4.4% (n=71). Non-shock mortality occurred in 33 patients (2.0%). Reinfarction was observed in 23 patients (1.4%) and target vessel revascularization was required in 30 patients (1.9%). In-hospital stent thrombosis was seen in 19 patients (1.2%), being acute stent thrombosis in 10 patients (0.6%).

Table 2. Angiographic and procedural findings

	n	%	Mean±SD
Multivessel disease	665	40.9	
Infarct-related artery			
Left anterior descending	807	49.7	
Left circumflex	213	13.1	
Right coronary	568	35.0	
Saphenous-vein graft	8	0.5	
Left main/diagonal	29	1.8	
Proximal lesion	900	55.4	
Baseline TIMI flow grade			
0-1	1241	76.4	
2	224	13.8	
3	160	9.9	
TIMI thrombus score ≥4	1085	66.8	
Lesion length (mm)			15.2±5.8
Reference vessel diameter (mm)			3.1±0.4
Good collateral channel	86	5.3	
Clopidogrel loading dose (600 mg)	1598	98.3	
Tirofiban use			
Before PCI	717	44.1	
After PCI	611	37.6	
Stent diameter (mm)			3.1±0.4
Stent length (mm)			21.5±9.1
Number of stents implanted			
Per infarct-related artery			1.2±0.4
Per patient			1.3±0.6
Maximal balloon inflation pressure (atm)			14.6±2.2
Drug-eluting stent	76	4.7	
Final TIMI flow grade			
0-1	64	3.9	
2	129	7.9	
3	1432	88.1	
Myocardial blush grade 3 (n=1269)	532	41.9	
Final luminal diameter (mm)			3.2±0.4
Method of reperfusion			
Balloon angioplasty	92	5.7	
Stenting with predilation	1233	75.9	
Direct stenting	300	18.5	
Maximum creatine kinase (IU/l)			2277±1756
Complete ST resolution (>70%)	937	57.7	

Table 3. Postprocedural echocardiographic findings

	n	%	Mean±SD
Left ventricular ejection fraction (%)			46.9±8.2
Left ventricular thrombus	20	1.2	
Severe mitral regurgitation	21	1.3	
Mechanical complications	12	0.7	
Rupture of chordae tendineae and/or papillary muscle	6	0.4	
Ventricular septal rupture	4	0.3	
Free wall rupture	2	0.1	

Nine patients (0.6%) suffered from stroke, which was hemorrhagic in three (0.2%). Cumulative MACE was observed in 101 patients (6.2%). Major bleeding was seen in 62 patients (3.8%). Blood transfusion was required in 78 patients (4.8%), which arose from non-bleeding causes in 19 patients (1.2%). Clinical heart failure was observed in 171 patients (10.5%), requiring mechanical ventilation in 103 patients (6.3%). Atrial fibrillation developed in 65 patients (4.0%) and sustained and/or non-sustained ventricular tachycardia was seen in 63 patients (3.9%). The mean hospital stay was 5.2±3.3 days.

Table 4. In-hospital events

	n	%
Mortality	71	4.4
Cardiac causes (including shock)	63	3.9
Noncardiac causes	8	0.5
Reinfarction	23	1.4
Target vessel revascularization	30	1.9
Stroke (Hemorrhagic/ischemic)	9	0.6
Major adverse cardiovascular events	101	6.2
Non-shock mortality	33	2.0
Stent thrombosis	19	1.2
Bleeding complications		
TIMI major bleeding	62	3.8
TIMI minor bleeding	45	2.8
Blood transfusion	78	4.8
Arrhythmic complications		
Ventricular tachycardia/fibrillation		
Primary	88	5.4
Secondary (sustained/non-sustained)	63	3.9
High-degree atrioventricular block	79	4.9
New-onset atrial fibrillation	65	4.0
Intra-aortic balloon pump	96	5.9
Heart failure	171	10.5

DISCUSSION

In this retrospective analysis, we aimed to present the results of primary PCI performed during a 2.5-year period in a high-volume tertiary center. We evaluated demographic, angiographic, and procedural characteristics of the patients and in-hospital adverse cardiovascular events.

The basic reperfusion strategies in the management of STEMI are mechanical (PCI) and pharmacologic reperfusion. Beyond 1990's, particularly in the last decade, primary PCI has become the emerging acute reperfusion method throughout the world. In the ACC/AHA and ESC guidelines of STEMI management, primary PCI is recommended as a class I indication.[9,10] It is emphasized that primary PCI should be the treatment of choice in the presence of experienced staff and operator (>75 PCIs/year and ≥11 primary PCIs/year), <90 minutes DTB time, and preferentially <30 minutes door-to-needle time. The guidelines also suggest that ECG evaluation be performed during transportation and, before hospital admission, emergency department, catheterization laboratory, and the laboratory staff be prepared in order to decrease the delay time. In the light of the guidelines, DTB time should be <90 minutes. Thrombolytic treatment should be preferred if DTB time exceeds this period, especially when the duration of chest pain is <2 hours.[9-11]

Our hospital is a tertiary cardiology center with 24 hours/7 days PCI facility and experienced interventional cardiology staff. Premedication is given immediately at the emergency department, and the patients are admitted to the catheterization laboratory, which is located on the upper floor of the emergency department. The mean DTB time of our center was 31 minutes, which is shorter than many reference centers,[12,13] and in none of our patients, DTB time exceeded 90 minutes as suggested in the guidelines. McNamara et al.[13] showed a close relationship between in-hospital mortality and DTB time, mortality rates being 3%, 4.2%, 5.7%, and 7.4% with DTB times of <90 minutes, 91-120 minutes, 121-150 minutes, and >150 minutes, respectively. Every 15-minute decrease in the treatment time between 150 and 90 minutes was associated with a decrease of 6.3 mortalities in 1000 deaths.

Although the effectiveness of primary PCI depends on variables such as DTB time and total ischemic time, experience of the center and operator is also important. Srinivas et al.[14] examined the relationship between in-hospital mortality and hospital and operator volume. In high-volume hospitals, mortality was found 3.4% compared to 5.4% in low-volume centers. Moreover, in a high-volume center, mortality of high-volume operators was significantly lower than that of low-volume operators (3.5% *vs*. 7.9%, p=0.01). All the interventional cardiologists of our center are high-volume operators with an experience of over 300 PCI procedures a year and the total number of elective and primary PCI procedures of our center exceeds 5000/year. The mortality rates of our center are similar to the reported numbers of high-volume centers.

In the German MIR and MITRA trials, a progressive significant decrease was reported in in-hospital mortality rates of the primary PCI group from 1994 to 1998 (13.9% *vs*. 3.9%).[15] Similarly, in the NRMI study, a significant decrease was reported in mortality rates from 1990 to 2005 (8.6% *vs*. 3.7%).[11] In-hospital mortality of our center including shock- and noncardiac-related mortality was 4.4%. The causes of mortality included renal failure, stroke, and major bleeding in eight patients. In-hospital cardiac mortality including cardiogenic shock was 3.9%, and nonshock-related mortality including cardiac and noncardiac causes was 2.1%. Decrease in mortality rates results from the advances in mechanical reperfusion, increased experience of interventional cardiologists, improvements in acute cardiac care, decreased transfer and DTB times, and advances in anticoagulant and antiaggregant treatments. Our center also closely follows and applies evolving strategies and advances in the management of acute myocardial infarction.

Primary PCI has a reported incidence of 7% for major bleeding, including entrance-site bleeding.[1,16] A significant decrease was observed in the amount of bleeding with controlled anticoagulation regimens, early sheath removal, use of smaller-diameter cannulation, and increased experience of interventional cardiologists.[16] It has been reported that use of clopidogrel and glycoprotein IIb/IIIa receptor blocker (tirofiban) together with heparin was associated with an increased tendency to bleeding. In some studies, however, it was shown that this combination was not associated with major bleeding,[17,18] but enoxaparin use resulted in a slightly increased rate of major bleeding compared to standard heparin.[19] In a recent meta-analysis of 31 studies, Valgimigli et al.[18] reported that the rate of minor bleeding significantly increased, whereas the rate of major bleeding did not increase in patients with acute coronary syndrome and receiving tirofiban. In the On-TIME 2 trial, tirofiban infusion before primary PCI was compared to placebo in patients with STEMI, and the rates of overall MACE and in-hospital mortality were found to be significantly lower in the tirofiban group (5.8% *vs*. 8.6%, p=0.043 and 2.2% *vs*. 4.1%, p=0.051, respectively).[17] There was no difference between the two groups with respect to major bleeding (3.4% *vs*. 2.9%, p=0.58). However, in this study, patients with refractory cardiogenic shock, end-stage renal failure, and tendency to bleeding were excluded. In our study, the rate of overall in-hospital MACE (death, reinfarction, revascularization, and stroke) was 6.2%, and the rate of major bleeding was 3.8%. One-third of the bleedings were related to the entrance site. Although the rate of major bleeding is increased in patients undergoing primary PCI, the incidence of intracranial bleeding is significantly lower compared to patients receiving thrombolytic therapy.[1,20] In our study, intracranial hemorrhage was observed in 0.2% of the patients and 0.4% of the patients had ischemic stroke. The overall stroke rate was 0.6%.

An important complication responsible for early reinfarction and mortality is stent thrombosis. Most cases of stent thrombosis are observed in the early, particularly in-hospital period.[21] The reported incidence of early stent thrombosis is 1.5% and the rate of this complication is higher in STEMI patients compared to elective procedures, as stent implantation during acute myocardial infarction is a risk factor for stent thrombosis.[21,22] In our study, in-hospital stent thrombosis was observed in 19 patients (1.2%). Ten patients developed acute, and nine patients developed subacute stent thrombosis. The incidence of stent thrombosis was similar to the reported rates in the literature.

Another complication that decreases the effectiveness of primary PCI is no-reflow phenomenon. Although restoration of angiographic TIMI 3 flow is the main target in the management of STEMI, microvascular reperfusion is more important.[16,23] The rate of this complication varies from 5% to 40% in different series, depending on the method to evaluate successful reperfusion.[23,24] No-reflow is associated with high short- and long-term mortality and morbidity rates. In the GUSTO-IIb trial, 30-day mortality was 1.6% in patients with TIMI 3 flow, 19.9% in patients with TIMI 2 flow, and 20% in patients with TIMI 0-1 flow.[25] In our study, angiographic no-reflow rate was 11.8% and myocardial blush grade 3, which is a pre-

dictor of microvascular perfusion, was observed in 41.9% of the patients. These results are similar to the rates of high-volume, multicenter trials.[18]

Mechanical complications are also responsible for in-hospital mortality and morbidity in patients with acute myocardial infarction. The frequency of mechanical complications has progressively decreased with the advances in and increased application rate of mechanical reperfusion, compared to thrombolytic therapy.[26-28] In the large patient population of the APEX-AMI study, the total mechanical complication rate (free wall rupture, chordae/papillary muscle rupture, ventricular septal rupture) was 0.91%.[28] In our study, the overall mechanical complication rate was 0.7%.

Study limitations

The main limitation of our study is its retrospective design, despite high-patient volume. In retrospective studies, the reliability of data depends on regular and accurate recording of patients' data by responsible physicians and hospital staff. In addition, some data could not be obtained from the archive. Another limitation is the lack of long-term cardiovascular events, which is planned to be included in a future study.

In conclusion, primary PCI outcomes of our high-volume tertiary center are similar to those of other reference cardiovascular centers with similar characteristics. Optimal use of adjunctive antiplatelet treatment and decreases in hospital delay will result in better in-hospital outcomes, as shown in our center.

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Key words: Angioplasty, balloon, coronary; hospital mortality; myocardial infarction/therapy/mortality; treatment outcome.

Anahtar sözcükler: Anjiyoplasti, balon, koroner; hastane mortalitesi; miyokart enfarktüsü/tedavi/mortalite; tedavi sonucu.