Comparison of early and late clinical outcomes in patients ≥80 versus <80 years of age after successful primary angioplasty for ST segment elevation myocardial infarction

Seksen yaş altı ve üstü hastalarda ST yükselmeli miyokart enfarktüsü için başarılı birincil anjiyoplasti sonrası erken ve geç dönem klinik sonuçların karşılaştırılması

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ABSTRACT

Objectives: We aimed to compare the efficacy of primary percutaneous coronary intervention (p-PCI) in patients ≥80 versus <80 years of age with ST-segment elevation myocardial infarction (STEMI).

Study design: We retrospectively enrolled 2213 patients with acute STEMI. The patients were prospectively followed up for a median of 42 months. Early and late clinical outcomes were compared according to age.

Results: One-hundred and seventy-nine (8.1%) of the 2213 patients were aged ≥80 years. Post-procedural TIMI grade 3 flow was significantly less frequent in the age ≥80 years patients (82.1% *vs.* 91.1%, p<0.001). Rates of mortality (14.5% *vs.* 3.4%, p<0.001), heart failure (20.7% *vs.* 10.5%, p<0.001), major hemorrhage (9.5% *vs.* 3.3%, p<0.001), secondary VT/VF (10.1% *vs.* 4.2%, p=0.002) and atrial fibrillation (12.8% *vs.* 4.3%, p<0.001) during the early hospitalization period were significantly higher in the age ≥80 years patient group. Overall rates of mortality (40% *vs.* 9.7%, p<0.001) and total stroke (5.6% *vs.* 1.1%, p=0.005) at long-term follow-up were also higher in the age ≥80 years patient group. However, there was no difference between the two groups with respect to the reinfarction/revascularization rates. Analysis, using the Cox proportional hazards model, revealed that age ≥80 to was an independent predictor of long-term mortality (hazard ratio 2.17, 95% CI 1.23-4.17, p=0.02).

Conclusion: Age is an independent predictor of mortality after p-PCI for STEMI. Although it seems to improve early outcomes, the efficacy of p-PCI at long-term follow-up is limited in elderly patients.

ÖZET

Amaç: Birincil (primer) perkütan koroner girişimin (p-PKG), ≥80 yaş ve <80 yaş ST yükselmeli miyokart enfarktüslü (STYME) hastalarda etkinliğini karşılaştırmayı amaçladık.

Çalışma planı: Akut STYME nedeniyle p-PKG uygulanan 2213 hasta geriye dönük olarak çalışmaya alındı. Hastalar ileriye dönük olarak (median süre 42 ay) takip edildi. Erken ve geç dönem klinik sonlanımlar yaşa göre karşılaştırıldı.

Bulgular: Hastaların 179'u 80 yaş ve üzerinde idi. İşlem sonrası TIMI 3 akım 80 yaş ve üzeri hastalarda anlamlı olarak daha nadirdi (%82.1 ve %91.1, p<0.001). Hastane içi erken dönemde mortalite (%14.5 ve %3.4, p<0.001), kalp yetersizliği (%20.7 ve %10.5, p<0.001), majör kanama (%9.5 ve %3.3, p<0.001), ikincil VT/VF (%10.1 ve %4.2, p=0.002) ve atriyum fibrilasyonu (%12.8 ve %4.3, p<0.001) oranları, 80 yaş ve üzeri grubu hastalarda anlamlı olarak daha yüksek idi. Toplam mortalite (%40 ve %9.7, p<0.001) ve inme (%5.6 ve %1.1, p=0.005) oranları uzun dönem takipte 80 yaş ve üzeri grubu hastalarda daha yüksek idi. Ancak, iki grup arasında tekrarlayan enfarktüs/revaskülarizasyon oranları açısından fark yoktu. Cox orantısal risk modeli ile yapılan analiz, 80 ve üzeri yaşın uzun dönem mortalite için bağımsız öngördürücü olduğunu gösterdi (risk oranı 2.17, %95 güven aralığı 1.23-4.17, p=0.02).

Sonuç: Yaş, STYME nedeniyle p-PKG yapılan hastalarda mortalitenin bağımsız öngördürücüsüdür. Erken dönem klinik sonuçları olumlu gözükse de, p-PKG'nin uzun dönem klinik etkinliği çok yaşlı hastalarda kısıtlı gözükmektedir.

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As a result of increased life expectancy, elderly individuals constitute an increasing proportion of patients admitted to hospitals for acute ST elevation myocardial infarction (STEMI). The selection of reperfusion strategy for elderly patients with acute STEMI bears great importance due to the high complication and low efficacy rates of fibrinolytic (FL) therapy.[1,2] Currently, primary percutaneous coronary intervention (p-PCI) is the most commonly preferred reperfusion strategy. Compared with FL therapy, mortality rates are significantly lower. However, there is still controversy regarding the efficacy of p-PCI in patients of advanced age due to limited outcome data in the literature.[2]

We aimed to compare the efficacy of primary percutaneous coronary intervention (p-PCI) in patients ≥80 versus <80 years of age with ST-segment elevation myocardial infarction (STEMI).

Abbreviations:

BNP	*B-type natriuretic peptide*
CBC	*Complete blood count*
CK	*Creatine kinase*
CRP	*C-reactive protein*
ECG	*Electrocardiography*
eGFR	*Estimated glomerular filtration rate*
FL	*Fibrinolytic*
LVEF	*Left ventricular ejection fraction*
MBG	*Myocardial blush grade*
p-PCI	*Primary percutaneous coronary intervention*
STEMI	*ST elevation myocardial infarction*
STR	*ST segment resolution*
TIMI	*Thrombolysis in myocardial infarction*

PATIENTS AND METHODS

Study population

Of the 2459 consecutive patients with acute STEMI who were admitted to our hospital between January 2006 and April 2009, 2213 subjects met the inclusion criteria and were retrospectively enrolled (Figure 1). Enrolled patients met the following criteria:[1] ST-segment elevation ≥0.1 mV in two or more leads (2 mm for V1-V3) or a new-onset left bundle branch block on an electrocardiogram, and[2] typical ongoing ischemic chest pain for longer than 30 minutes. Written, informed consent for the procedure was obtained from all patients. Clinical outcomes of enrolled patients were then prospectively followed for a median duration of 42 months. Local ethics committee approved this study.

Percutaneous coronary interventions

All patients received 300 mg of aspirin and a loading dose of clopidogrel (300 to 600 mg) upon admission

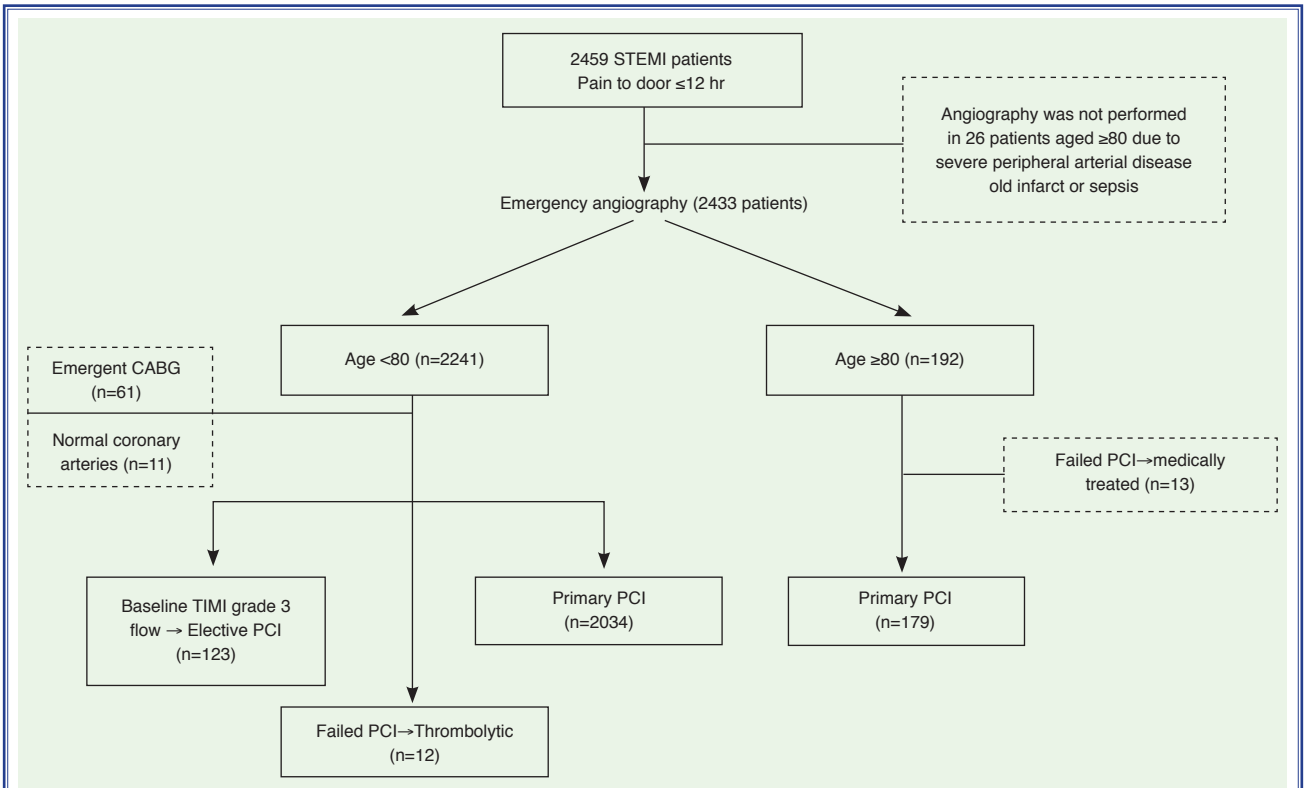

Figure 1. Flow chart. Enrollment and follow-up of study patients.

as well as intravenous standard heparin 70 U/kg (60 U/kg in patients receiving glycoprotein IIb/IIIa inhibitor) before the procedure. The use of a glycoprotein IIb/IIIa inhibitor (tirofiban) was left to the primary operator's discretion. All p-PCI procedures were performed by experienced interventional cardiologists using a femoral approach. Depending on the coronary anatomy and lesion characteristics, patients underwent direct stenting, conventional stenting, or solely balloon dilation procedures. All patients were transferred to a coronary intensive care unit after undergoing p-PCI. After the intervention, all patients were given 1 mg/kg of subcutaneous enoxaparin twice daily (dosages were adjusted according to the patient's estimated glomerular filtration rates [eGFR]), 150 mg/day of aspirin, and 75 mg/day of clopidogrel.

Data collection and clinical follow-up

In addition to coronary risk factors, data on demographic and clinical characteristics of the patients, such as age and gender, were obtained from hospital files and records. On admission, baseline complete blood count (CBC), urea, creatinine, glucose, troponin I, cholesterol profile, B-type natriuretic peptide (BNP) and C-reactive protein (CRP) serum studies were obtained. Serum studies were repeated for cardiac enzymes every 6 hours until peak levels were attained, then repeated daily. CBC studies were also repeated daily. BNP was measured with the immunoassay method using ADVIA Centaur BNP assay kits (Bayer, Tarrytown, New York). The limits of detection by this method are values between 2 pg/ml and 5000 pg/ml. CRP was measured by the nephelometric method (Beckman Coulter, Dublin, Ireland) with a cut-off value of 8 mg/l and lowest detectable level of 1 mg/l. Post-procedural left ventricular ejection fraction (LVEF) and development of mechanical complications were evaluated by transthoracic echocardiography (Vivid 3 or 5, GE, Horten, Norway). ST segment resolution (STR) was evaluated by obtaining electrocardiography (ECG) on admission, immediately [prior to intervention], 60 minutes after the intervention, and then twice daily. All coronary hemodynamic data were recorded, stored off-line, and analyzed by two independent investigators. Coronary lesions were evaluated in at least two non-foreshortened angiographic views at the end-diastolic phase. Lesions >50% were identified and labeled as hemodynamically significant. Pre- and post-procedural thrombolysis in myocardial infarction (TIMI) grade flows, collateral flow (Rentrop), infarct-related artery, severity of identified lesions, and the number of diseased vessels were noted.

In-hospital outcomes of all study subjects were obtained from the hospital record system. The long-term follow-up data of the patients were obtained during follow-up visits, or through telephone calls, review of polyclinic records of the re-hospitalized patients and archive and computer records. The patients who could not be reached were investigated via the Statistical Institute and Birth Registration Office to determine whether or not mortality occurred.

Definitions

Apart from the standart procedural success definition, unsuccessful p-PCI was defined as an inability to perform p-PCI due to any factor such as advanced tortuosity, severe calcification or lesion angulation. Anemia was diagnosed according to World Health Organization criteria (baseline hemoglobin level <13 g/dl for men and <12 g/dl for women).[3] eGFR was estimated by the Modification of Diet in Renal Disease (MDRD) Study formula.[4] Contrast-induced nephropathy was defined as either an increase in serum creatinine greater than 25% or an absolute increase in serum creatinine of 0.5 mg/dL. Pre-infarction angina was defined as the presence of chest pain lasting less than 20 minutes that occurred <48 hours before the onset of acute myocardial infarction (AMI). Collateral circulation was graded according to Rentrop classification with grade 2/3 collateral flow being accepted as well-developed collateral flow.[5] Multivessel disease was diagnosed in the presence of >50% diameter stenosis in two or more major epicardial arteries. The complete STR was defined as ≥70% STR at 60-minutes by post-procedural ECG.[6] Myocardial blush grade (MBG) was evaluated according to established standart methods.[7] Major hemorrhage was diagnosed, in accordance to TIMI bleeding classification, by the presence of more than 5 g/dl decrease in hemoglobin, >15% decrease in hematocrit level, or the presence of intracranial bleeding.[8] Myocardial re-infarction was diagnosed in the presence of either 1) new, pathologic Q waves or 2) a new ST segment elevation lasting for at least 30 minutes together with at least a two-fold increase in creatine kinase (CK) level or more than 50% increase in CK levels for a patient whose cardiac enzymes were already elevated.

Revascularization was defined as repeated p-PCI or CABG for not only IRA but also other vessels (including re-stenosis, de-novo lesion and re-infarction).

Statistical analysis

Continuous variables are expressed as median (interquartile range) while categorical variables are expressed as numbers and percentages. The difference between categorical variables was analyzed either by chi-square or Fisher's exact test, while the difference between the continous variables was analyzed using the Mann-Whitney U-test. Survival rates of both groups were tested by the life-table method, whereas the difference between the survival curves was analyzed by the Wilcoxon signed-rank test. The Cox proportional hazard model was applied for the prediction of mortality. All variables showing significance values $p<0.05$ (age, gender, diabetes, hypertension, smoking status, pre-infarction angina, reperfusion time, history of CABG, previous p-PCI, previous stroke, chronic obstructive pulmonary disease, cognitive disorder, peripheral artery disease, cardiogenic shock, baseline CRP, BNP, glucose, baseline anemia, eGFR <60 ml/min/1.73m², LVEF, LDL-cholesterol and triglyceride, STR, multi-vessel disease, right coronary artery (RCA) infarct, rentrop 2/3, final TIMI flow, MBG, major hemorrhage, blood transfusion required, intra-aortic balloon pump (IABP) use and alterations in the treatment) were included in the model. In order to show the effect of a 10-year increase in age over mortality, age was added to the model separately. This was done both as a dichotomized and continous variable, and the model was repeated. In all statistical analyses, two tailed p values of less than 0.05 were considered to indicate statistical significance. The study's findings were statistically analyzed using SPSS 11.5 (SPSS Inc, Chicago, IL, USA).

RESULTS

One-hundred and seventy-nine (8.1%) of the 2213 patients were aged ≥80 years (median age 82, 93 female). P-PCI was unsuccessful in 13 patients (6.8%) aged ≥80 years and 14 patients (0.6%) aged <80 years, $p<0.001$. Female gender, diabetes, hypertension, renal failure, anemia, presentation with cardiogenic shock, cognitive dysfunction, peripheral artery disease, and inferior myocardial infarction were significantly more common in the patients aged ≥80 years. Although there was no significant difference between the two groups with regard to the average door-to-balloon time, pain-to-door times were significantly higher in the patients aged ≥80 years (median [IQR] 140 [80-230] *vs.* 190 [100-290], $p<0.001$). Additionally, several baseline laboratory parameters, such as BNP, CRP, and glucose levels, were significantly higher in the patients aged ≥80 years (Table 1).

While multi-vessel disease and the culprit right coronary artery occlusion were significantly more common in the patients aged ≥80 years, pre-procedural use of tirofiban was less frequent in this patient group. The rate of well-developed collateral circulation on the baseline angiography was also significantly lower in patients aged ≥80 years (6.7% *vs.* 12%, $p=0.035$). The preprocedural TIMI flow grades were similar in both age groups. Post-procedural TIMI grade 3 flow rate was less common in the patients aged ≥80 years. The frequency of complete STR (47.8% *vs.* 62.3%, $p=0.003$), MBG 3 (27.0% *vs.* 45.2%, $p<0.001$) and acute LVEF (median [IQR] 44 [35-52] *vs.* 48 [42-55], % $p<0.001$) was significantly lower in the patients aged ≥80 years. The use of in-hospital tirofiban, statin, and β blocker therapy was significantly less common while diuretic treatment was more frequent in the elderly patient population. Duration of hospitalization was also higher in the age ≥80 group (median [IQR] 5 [4-8] *vs.* 4 [3-6] days, $p<0.001$) (Table 2).

In-hospital events

Among patients ≥80 years of age, rates of in-hospital mortality (14.5% *vs.* 3.5%, $p<0.001$), heart failure (20.7% *vs.* 10.5%, $p<0.001$), major hemorrhage (9.5% *vs.* 3.3%, $p<0.001$), mechanical complication (3.4% *vs.* 0.7%, $p=0.005$), contrast induced nephropathy (31.8% *vs.* 12.2%, $p<0.001$), requirement of blood transfusion, and arrhythmic complications (ventricular tachycardia, atrial fibrillation, high-degree AV block) were significantly higher. While the rate of stroke events was 3.4-fold higher in the patients aged ≥80 years, this difference was not statistically significant (Table 3).

Long-term events

Despite all efforts, 68 patients could not be reached for outcome follow-up and were excluded from the study (9 [5%] patients aged ≥80 years, 59 [2.9%] patients aged <80 years $p=0.315$). While 1-year mortality (29.7% *vs.* 7.0%, $p<0.001$) and stroke (4.1% *vs.*

Table 1. Baseline demographic and clinical characteristics of enrolled patients

	Age <80, p-PCI (n=2034)		Age ≥80, p-PCI (n=179)		*p*
	n	%	n	%	
Age	56	48-63	82	81-85.3	**<0.001**
Gender (female)	365	17.9	93	52.0	**<0.001**
Hypertension	810	39.8	123	68.7	**<0.001**
Diabetes mellitus	466	22.9	62	34.6	**<0.001**
Hypercholesterolemia	840	41.3	58	32.4	0.020
Current smoker	1125	55.5	39	21.8	**<0.001**
Prior MI	108	5.3	16	8.9	0.043
Prior PCI	181	8.9	26	14.5	0.019
Prior CABG	65	3.2	17	9.5	**0.002**
Prior stroke	43	2.1	24	13.4	**<0.001**
COPD	73	3.6	30	16.8	**<0.001**
Cognitive impairment	12	0.6	19	10.6	**<0.001**
Peripheral arterial disease	170	8.4	37	20.7	**<0.001**
Pre-infarction angina	525	25.8	27	15.1	**0.003**
Pain to door time (min)	140	80-230	190	100-290	**<0.001**
Door to balloon time (min)	29.5	23.5-35	30	25-36.5	0.135
SBP (mmHg)	130	116-140	134	100-145	0.650
DBP (mmHg)	78	68-87	80	56-89	0.913
Baseline heart rate (bpm)	77	69-86	76	66-87	0.076
Killip class >1	324	15.9	53	28.6	**<0.001**
Cardiogenic shock	83	4.1	26	14.5	**<0.001**
Baseline creatinine (mg/dl)	0.86	0.76-1.00	1.01	0.80-1.30	**<0.001**
eGFR <60 ml/min/1.73 m²	199	9.8	90	50.3	**<0.001**
Baseline BNP (pg/ml)	70	37-132	165	87-300	**<0.001**
Baseline glucose (mg/dl)	125	104-165	148	120-192	**0.007**
Baseline CRP (mg/l)	9.7	5.6-16.6	14.3	8.7-19.9	**<0.001**
LDL-cholesterol (mg/dl)	110	84-138	104	74.5-128.5	0.012
HDL-cholesterol (mg/dl)	38	32-45	37	30-46	0.870
Triglyceride (mg/dl)	118	83-168	89	56-126	**<0.001**
Baseline anemia	432	21.2	92	51.4	**<0.001**
Anterior infarct location	1003	49.3	73	40.8	0.029
Previous medication					
Aspirin	195	9.6	33	18.4	**<0.001**
Statin	434	21.3	33	18.4	0.362
-blocker	243	11.9	23	12.8	0.518
ACE-I	413	20.3	63	35.2	**<0.001**

Data are expressed as median (interquartile range) or as frequency (percentage).

p-PCI: Primary percutaneous coronary intervention; MI: Myocardial infarction; PCI: Percutaneous coronary intervention; CABG: Coronary artery by-pass graft; COPD: Chronic obstructive pulmonary disease; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; eGFR: Estimated glomerular filtration rate; BNP: B type natriuretic peptide; CRP: C-reactive protein; LDL: Light density lipoprotein; HDL: High density lipoprotein; ACE–I: Angiotensin converting enzyme inhibitor.

Table 2. Procedural characteristics and treatment data

	Age <80, p-PCI		Age ≥80, p-PCI		*p*
	n	%	n	%	
Multi-vessel disease	815	40.1	97	54.2	**<0.001**
Infract related artery					
Left anterior descending artery	1005	49.4	76	42.5	0.074
Circumflex artery	283	13.9	25	14.0	0.984
Right coronary artery	706	34.7	76	42.5	0.038
Left main/diagonal/saphenous	41	2.0	2	1.1	0.404
Baseline TIMI 0/1 flow	1551	76.3	136	76.0	0.921
Good collateral channel	243	12.0	12	6.7	0.035
Stent use	1925	94.7	166	92.7	0.271
Multi-vessel intervention	322	15.8	33	18.4	0.176
Final TIMI flow					**<0.001**
0/1	65	3.2	15	8.4	
2	117	5.8	17	9.5	
3	1852	91.1	147	82.1	
Myocardial blush grade 3	761	45.2	41	27.0	**<0.001**
Complete ST-segment resolution (>70%)	1221	62.3	85	47.8	**0.003**
Peak troponin I (ng/ml)	78	35-169	87	45-211	**0.002**
Acute LVEF (%)	48	42-55	44	35-52	**<0.001**
Intra-aortic balloon pump use	105	5.2	27	15.1	**<0.001**
In-hospital medications					
Aspirin	1999	98.3	172	96.1	0.337
Clopidogrel	2025	99.6	177	98.9	0.901
Statin	1847	90.8	150	83.8	**0.004**
□-blocker	1738	85.4	135	75.4	**0.002**
ACE-I	1754	86.2	146	81.6	0.086
Diuretic	236	11.6	44	24.6	**<0.001**
Glycoprotein IIb/IIIa inhibitors	885	43.5	40	22.3	**<0.001**
Medication at discharge					
Aspirin	1901	96.9	144	94.7	0.138
Clopidogrel	1951	99.5	150	98.7	0.203
Statin	1776	90.6	129	84.9	0.023
□-blocker	1765	90.0	131	86.2	0.135
ACE-I	1576	80.4	132	86.8	0.054
Hospitalization duration (day)	4	3-6	5	4-8	**0.009**

Data are expressed as median (interquartile range) or as number (percentage). p-PCI: Primary percutaneous coronary intervention; TIMI: Thrombolysis in myocardial infarction; LVEF: Left ventricular ejection fraction; ACE–I: Angiotensin converting enzyme inhibitor.

0.7%, p=0.006) were significantly more common in the patients aged ≥80 years, there was no difference between the two age groups with respect to the rates of re-infarction or total revascularization. With long-term follow-up (median 42 months, IQR 37-51 month), all cause mortality (40% *vs.* 9.7%, p<0.001) and stroke (5.6% *vs.* 1.1%, p=0.003) rates were found to be significantly more common in the patients aged

≥80 years. Long-term cardiac mortality was also 3-fold higher, while non-cardiac mortality was approximately 7-fold higher, in the patients aged ≥80 years. There was no significant difference observed between the two age groups with regard to the total revascularization and re-infarction rates (Table 3). Life table analysis revealed a significant difference between the long-term survival curves of the two age groups (Wilcoxon signed-rank, p<0.001, Figure 2).

Predictors of long-term mortality

When the Cox proportional hazard model was used, with multivariate correction relative to the baseline values, age ≥80 years was determined to be an independent predictor for long-term mortality (Hazard ratio [HR] 2.17, 95% Confidence Interval [CI] 1.23-4.17, p=0.02). By repeating the model with age as a continous variable, increasing age was also found to be independently predicting mortality and HR for every 10-years of age was calculated (HR 1.40, 95% CI 1.17-1.63, p<0.001). Other independent predic-

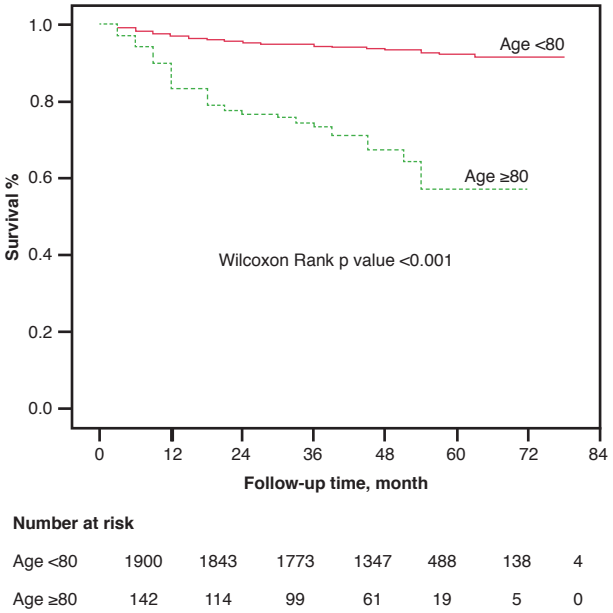

Figure 2. Kaplan-Meier survival analysis demonstrating the survival rates at long-term follow-up (median 42 months).

Table 3. In-hospital and long-term outcomes

	Age <80, p-PCI		Age ≥80, p-PCI		*p*
	n	%	n	%	
In-hospital events					
Death	70	3.4	26	14.5	**<0.001**
Heart failure	214	10.5	37	20.7	**<0.001**
Re-infarction	25	1.2	3	1.7	0.608
Stroke*	10	0.5	3	1.7	0.066
Major bleeding	68	3.3	17	9.5	**<0.001**
Blood transfusion	95	4.7	34	19.0	**<0.001**
Atrial fibrillation	87	4.3	23	12.8	**<0.001**
Secondary VT/VF	85	4.2	18	10.1	**0.002**
Mechanical complications	15	0.7	6	3.4	**0.005**
Contrast induced nephropathy	245	12.2	57	31.8	**<0.001**
Long-term outcomes†‡					
Death	191	9.7	70	40.0	**<0.001**
Cardiac causes	156	7.9	45	25.7	**<0.001**
Non-cardiac causes	35	1.8	25	14.3	**<0.001**
Re-infarction	142	7.4	16	11.0	0.125
Revascularization	432	22.7	27	18.6	0.261
Stroke*	20	1.1	9	5.6	**0.003**

*Hemorrhagic and ischemic; †Including in-hospital events; ‡Median follow-up time 42 month; p-PCI: Primary percutaneous coronary intervention; VT: Ventricular tachycardia; VF: Ventricular fibrillation.

Table 4. Independent predictors of long-term mortality - Cox proportional hazard model

	Hazard ratio	95% CI	*p*
Age (for each 10 year increment)	1.40	1.17-1.63	**<0.001**
Age ≥80	2.17	1.23-4.17	**0.02**
Gender (female)	2.03	1.23-3.47	0.021
Baseline anemia	1.77	1.05-2.97	0.032
Baseline BNP (for each 10 pg/ml increment)	1.02	1.01-1.04	**<0.001**
Major bleeding	4.56	1.02-20.53	0.048
eGFR <60 ml/min/1.73 m²	3.12	1.47-6.59	**0.003**
Incomplete ST-segment resolution (<70%)	2.08	1.02-4.24	0.043
Post-procedural LVEF <40%	3.71	1.88-7.35	**<0.001**

CI: Confidence interval; BNP: B type natriuretic peptide; eGFR: Estimated glomerular filtration rate; LVEF: Left ventricular ejection fraction.

tors for long-term mortality identified by this study were female gender, baseline anemia, major hemorrhage, eGFR <60 ml/min/1.73 m², incomplete STR, post-procedural LVEF <40%, and baseline BNP level (Table 4).

DISCUSSION

This retrospective study illustrated that, despite a slight improvement in early outcomes, the clinical benefit of p-PCI on long-term patient outcomes is limited in elderly patient populations when compared to younger individuals. However, total revascularization and re-infarction rates between age groups were not significantly different. In addition to advanced age, female gender, baseline anemia, major hemorrhage, renal failure, incomplete STR, post-procedural left ventricular systolic dysfunction, and baseline BNP level were found to independently predict long-term mortality in elderly patients.

Mechanical reperfusion interventions have significantly reduced the rate of mortality for patients with STEMI when compared with FL therapy.[1,8] Patients aged ≥80 years represent an increasing proportion of STEMI victims, with early mortality rates as high as 30%.[9] However, there is a paucity of research directly comparing FL and p-PCI interventions in elderly patient populations. The SENIOR-PAMI study reported that p-PCI was superior to FL therapy among patients aged ≥70 years; however, the same findings were not observed in patients aged >80 years.[8] de Boer et al.[10] published a meta-analysis reporting lower mortality rates, even in very old individuals, for those treated by p-PCI as compared to those receiving FL therapy. Moreover, it was noted that advanced age should not be among the exclusion criteria for p-PCI. Nonetheless, an additional study found no difference between the p-PCI and FL with respect to long-term outcomes in patients aged ≥75.[11] It has been previously reported that p-PCI reduced the in-hospital mortality rate from 29% to 16% in patients aged ≥80 years. The 5-year mortality rate in the p-PCI group was 45% in this same study.[12] The TRIANA study showed that despite similar 30-day and 1-year mortality rates between FL and p-PCI groups, recurrent ischemia was less frequent in those treated by p-PCI among STEMI patients aged ≥75 years.[13] In our study, among patients aged ≥80 years, in-hospital mortality rate was 14.5% while 30-day and long-term mortalities were 20% and 40%, respectively. Although there is an improvement in the early outcomes, long-term mortality is still very high in this patient group. For every 10 years of age, a 1.4-fold increase in the long-term mortality rate was observed.

In our study, age ≥80 years was found to be an independent predictor of long-term mortality. The higher incidence of comorbidities and the more severe presentation in this age group may be responsible for this association. Further, it may be associated with lower incidence of pre-infarction angina in the octogenarians. Ischemic pre-conditioning is also known to be associated with smaller infarction areas and reduced mortality.[14,15] Abete et al.[16] showed that advanced age was associated with loss of pre-conditioning. Late presentation to the hospital and prolonged reperfusion time may be another contributing

factor to increased mortality. This delay could be due to alterations in pain perception and higher rates of silent ischemia at advanced ages.[17] In accordance with previous reports, collateral flow was less developed in these elderly patients and this is known to be associated with poorer prognosis.[18] Moreover, the higher incidence of multi-vessel disease, angiographic no-reflow phenomenon, clinical heart failure, mechanical and arrhythmic complications, contrast induced nephropathy, worse STR, and lower LVEF may all have contributed to increased mortality in these elderly patients.

Despite its association with lower rates of intracerebral hemorrhage, as compared to the FL therapy, p-PCI is also known to be associated with non-cerebral major hemorrhages.[19] In our study, despite less aggressive anti-aggregant therapy, non-cerebral major hemorrhages were 2.8-fold more common in the patients aged ≥80 years. These incidences were generally in the form of access site and gastrointestinal hemorrhages. Thus, transfusion of blood products was significantly more common in this elderly patient group. While the total in-hospital stroke rates were not statistically different between age groups, 1-year and long-term rates were significantly higher in the aged ≥80 years group. Major hemorrhage[20] and transfusion of blood products[21] are known to be associated with poor prognosis in the course of acute STEMI. We also found that major hemorrhage was an independent predictor of long-term mortality in the patients aged ≥80 years.

There were several limitations of our study. Despite a large total volume of enrolled patients, the number of patients aged ≥80 years was relatively limited. The study was also conducted retrospectivally and at a single medical center. The most important limitations of retrospective studies are the reliability of data and researcher bias. In order to mitigate researcher bias, the study data, including the long-term results, were acquired by different investigators.

Age has been found to be an independent predictor for mortality after p-PCI. Although it appears to improve early outcomes, the efficacy of p-PCI on long-term clinical outcomes is limited in patients aged ≥80 years. Future prospective and retrospective studies including elderly patients with STEMI are needed in order to compare the effectiveness and safety of p-PCI among this patient population.

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Key words: Age factors; myocardial infarction/etiology/therapy; percutaneous coronary intervention; treatment outcome.

Anahtar sözcükler: Yaş faktörü; miyokart enfarktüsü/etyoloji/tedavi; perkütan koroner girişim; tedavi sonucu.