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ORIGINAL ARTICLE / KLİNİK ÇALIŞMA

Revascularization strategies in patients with infective endocarditis-related ST-elevation myocardial infarction: The STEMI-ENDO Registry

Enfektif endokarditle ilişkili ST yükselmeli miyokart enfarktüslü hastalarda revaskülarizasyon stratejileri: STEMI-ENDO Kayıt Çalışması

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ABSTRACT

Objective: Infective endocarditis (IE)-related ST elevation myocardial infarction (STEMI) is extremely rare. A clear clinical consensus is lacking regarding the management of this emergency. In this study, we aimed to describe the clinical outcomes of treatment strategies in this patient population.

Methods: The study population comprised 19 retrospectively evaluated patients (nine women; mean age 52±11.8 years) with a diagnosis of IE-related STEMI. Transesophageal echocardiography detected vegetation in all the patients. The study population was divided into two groups on the basis of in-hospital mortality.

Results: Major clinical manifestations included dyspnea (89.5%), fever (78.9%), and chest pain (63.2%). Catheter-based coronary angiography was performed in all the patients. The causative agent was isolated in all the cases, and *Staphylococcus aureus* was identified in seven (36.8%). The most common infarction was in the left anterior descending artery (n=12 [63.2%]). The treatment strategy consisted of mechanical thrombectomy (n=1), valve replacement following stent implantation (n=5), direct balloon angioplasty (n=4), valve replacement along with coronary artery bypass grafting (CABG; n=6), and medical follow-up (n=3). Moreover, thrombolysis in myocardial infarction III flow was significantly higher in the survival group (100% vs. 0%, p<0.001). All these patients preferred CABG or stent implantation for revascularization.

Conclusion: The current data suggest that a revascularization strategy with stent implantation or revascularization with CABG has a lower mortality rate in patients with IE-related STEMI.

ÖZET

Amaç: Enfektif endokardit (EE) ile ilişkili ST yükselmeli miyokart enfarktüsü (STYME) oldukça nadirdir. Bu acil durumun yönetimi konusunda net bir klinik fikir birliği yoktur. Bu çalışmada, bu hasta popülasyonunda tedavi stratejilerinin klinik sonuçlarını açıklamayı amaçladık.

Yöntemler: Çalışma popülasyonu, EE ile ilişkili STYME tanısı almış, geriye dönük olarak değerlendirilen 19 hastadan (dokuz kadın; ortalama yaş 52±11.8 yıl) oluşuyordu. Transözofageal ekokardiyografi ile tüm hastalarda vejetasyon tespit edildi. Çalışma popülasyonu hastane içi mortaliteye göre iki gruba ayrıldı.

Bulgular: Başlıca klinik belirtiler nefes darlığı (%89.5), ateş (%78.9) ve göğüs ağrısı (%63.2) idi. Tüm hastalara kateter bazlı koroner anjiyografi yapıldı. Tüm olgularda etken izole edildi ve yedi olguda (%36.8) *Staphylococcus aureus* belirlendi. En sık görülen enfarktüs sol ön inen arterdeydi (n=12 [63.2%]). Tedavi stratejileri mekanik trombektomi (n=1), stent implantasyonunu takiben kapak replasmanı (n=5), direkt balon anjiyoplasti (n=4), koroner arter baypas greftleme ile birlikte kapak replasmanı (KABG; n=6) ve tıbbi takip (n=3) idi. Ayrıca, miyokart enfarktüsünde tromboliz III akım, hayatta kalma grubunda anlamlı derecede daha yüksekti (%100'e karşı %0, p<0.001). Tüm bu hastalar revaskülarizasyon için KABG veya stent implantasyonunu tercih etti.

Sonuç: Mevcut veriler, stent implantasyonu veya KABG ile revaskülarizasyon içeren bir revaskülarizasyon stratejisinin, EE ile ilişkili STYME hastalarında daha düşük mortalite oranına sahip olduğunu göstermektedir.

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Infective endocarditis (IE) is a life-threatening disease.[1,2] Despite technological advances in imaging modalities and optimal therapy, IE continues to carry a high risk of mortality and morbidity, such as systemic/peripheral embolic events.[3-6] Although embolism most commonly occurs in the cerebral arteries, the incidence of coronary embolism is very low and rarely results in ST-segment elevation myocardial infarction (STEMI). Data on the treatment strategies for IE-related STEMI are limited.[5,6] Well-known thrombolytic and interventional revascularization therapies for the treatment of atherothrombotic STEMI may not be appropriate for IE and may even be detrimental in some patients.[7] Hence, a clear consensus is lacking regarding the management of this patient population. As only case reports,[8-11] a systematic review and meta-analysis,[12] and two major trials with a limited number of patients have been published to date in this field,[5,6] we decided to use our database to evaluate the characteristics of and treatment strategies for IE-related STEMI. In this study, we aimed to describe the clinical outcomes of treatment strategies (interventional or non-interventional) for IE-related STEMI.

METHODS

Study population

A total of 19 patients with IE-related STEMI in three tertiary centers between February 2012 and March 2021 were included in this retrospective study. IE-related STEMI was defined according to the following criteria: clinically, imaging, and microbiologically definite IE, and STEMI diagnosed as chest pain with persistent elevation in consecutive ST segments. Patients who did not meet both criteria were excluded from the study. This retrospective study was conducted in accordance with the principles of the Helsinki Declaration and approved by the Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital Institutional Ethics Comittee (Approval Date: July 12, 2021; Approval Number: 2021/60). The following data were collected from the patients at admission: age; sex; blood pressure; rhythm status; and history of diabetes mellitus, dyslipidemia, tobacco use, previous coronary artery disease, cancer, chronic kidney disease, amyloidosis, and lung diseases. Routine laboratory tests and blood cultures were performed at hospital admission and during treatment. The study was divided into two groups on the basis of in-hospital mortality.

Abbreviations:

AKI	*Acute kidney injury*
CABG	*Coronary artery bypass grafting*
CS	*Cardiogenic shock*
IE	*Infective endocarditis*
STEMI S	*T-segment elevation myocardial infarction*
TEE	*Transesophageal echocardiography*
TIA	*Transient ischemic attack*
TIMI	*Thrombolysis in myocardial infarction*
VAs	*Ventricular arrhythmias*

Three sets of blood culture samples were collected at one-hour intervals starting at admission to the hospital. Any other tissue (valves, vegetation) or foreign body samples (pacemaker leads, device) removed during surgery were additionally used to isolate the microorganisms. Routine laboratory investigations periodically recorded the complete blood count, C-reactive protein, procalcitonin levels, and serum chemistry. At presentation, the majority of patients were administered empiric broad-spectrum antibiotics. If a patient was referred from another hospital, antibiotic treatment was administered according to the previous culture results. Subsequently, the patients were switched over to suitable antibiotics in accordance to their antibiotic susceptibility reports and standard recommendations. These recommendations were based on the European Society of Cardiology guidelines.[2]

Echocardiography

All the patients underwent comprehensive transthoracic echocardiography using a Vingmed CFM 800 Vivid 5 (GE Vingmed Ultrasound AS, Horten, Norway), Vivid 7 Dimension® (GE Vingmed Ultrasound AS), or Philips iE33 (Philips Medical Systems, Andover, MA, USA) echocardiography device. Transesophageal echocardiography (TEE) studies were performed in all the patients using a 5-MHz multiplane transducer connected to a GE Vingmed CFM 800 ultrasound machine or an X7-2t transducer connected to an iE33 ultrasound machine (Philips Medical Systems). TEE examinations were performed at hospital admission and within 24 h of the STEMI diagnosis with particular focus on the presence and size of the vegetation and the presence of perivalvular complications, including abscesses, fistulas, or pseudoaneurysms. Vegetation and abscesses were defined in accordance with previous studies.[2-5] In all the patients, left atrial diameter and left ventricular end-systolic and end-diastolic diameters were measured on the parasternal long-axis view in M-mode. Tricuspid an-

nular plane systolic excursion was acquired by placing an M-mode cursor through the lateral tricuspid annulus and measuring the amount of longitudinal motion of the annulus at peak systole in the standard apical four-chamber view. Left ventricular ejection fraction was calculated using the biplane Simpson's method.

Definitions

STEMI was diagnosed according to European Society of Cardiology recommendations, that is, chest pain with persistent ST elevation in consecutive ST segments.[7] Events causing ST-segment elevation but not associated with acute coronary syndrome, such as myocarditis, severe sepsis, electrolyte disturbances, and pericarditis, were not considered as STEMI. Moreover, an occluded (thrombolysis in myocardial infarction [TIMI] flow 0/I) coronary artery was angiographically documented. The 2015 European Society of Cardiology modified criteria were used to diagnose IE.[2] The main outcome measure was the occurrence of in-hospital death. Complications accompanying IE-related STEMI include cerebral embolism (including ischemic stroke and transient ischemic attack [TIA]), peripheral and/or splenic embolism, fatal ventricular arrhythmias (VAs), and cardiogenic shock (CS). TIA was defined as a brief episode of neurological dysfunction resulting from focal cerebral ischemia not associated with a permanent cerebral infarction.[13] Ischemic stroke was defined as an episode of neurological dysfunction caused by focal cerebral, spinal, or retinal infarction.[14] Acute peripheral arterial thromboembolism was defined as signs of ischemia (pain, pulselessness, pallor, paresthesia, and paralysis) caused by a sudden decrease in peripheral arterial perfusion threatening limb viability that requires urgent evaluation and treatment.[15] Acute kidney injury (AKI) diagnosis was made according to the following three major criteria: increase in serum creatinine ≥1.5 times than the baseline, increase of ≥0.3 mg/dL (≥26.4 mmol/L), or urine output <0.5 mL/kg/h for >6 h. An embolic event was assessed by the presence of clinical signs and verification using at least one of the following diagnostic tools: diffusion-weighted magnetic resonance imaging (neurological events) or coronary/peripheral angiography and/or duplex ultrasound (coronary and peripheral artery events). The clinical diagnosis of embolic TIA or stroke was made by a neurologist. Acute limb ischemia was diagnosed by a cardiologist or a cardiovascular surgeon after a detailed evaluation of coronary and peripheral angiographs. Sustained ventricular tachycardia or fibrillation was defined as a fatal VA. CS was defined in accordance with the literature.[7]

Statistical analysis

The statistical analysis was performed using SPSS for Windows version 19.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was tested using the Shapiro–Wilk test. Normally distributed continuous variables were expressed as mean±standard deviation or median (25^{th}-75^{th} percentiles). Categorical variables were expressed as frequencies and percentages. Continuous variables were compared using student's t-test or the Mann-Whitney U test as appropriate. The chi-squared or Fisher's exact test was used to compare categorical variables as appropriate. Two-sided values of $p<0.05$ were considered significant.

RESULTS

This retrospective study included 19 patients (nine women; mean age 52±11.8 years) with a diagnosis of IE-related STEMI. Nine of the 19 patients diagnosed with IE-related STEMI died during the follow-up period. The causes of death in these patients were as follows: CS plus fatal VA in four, CS plus AKI in one, fatal VA plus major ischemic stroke in one, sepsis plus systemic/peripheral embolism plus AKI in one, sepsis in one, and sepsis plus severe paravalvular leak in one. The baseline clinical characteristics of patients who died in hospital and those who survived are shown in Table 1. There was no significant intergroup difference in demographic findings including age, sex, or the frequencies of hypertension, chronic obstructive pulmonary disease, diabetes mellitus, chronic kidney disease, malignancy, cigarette smoking, or history of coronary artery disease. The clinical manifestations included dyspnea (89.5%), fever (78.9%), syncope (10.5%), palpitations (52.6%), and chest pain (63.2%) (Table 1). There was no intergroup difference in clinical findings at the time of the STEMI diagnosis.

Table 2 summarizes the echocardiographic, laboratory, and microbiological characteristics of patients who died in hospital and those who survived. When the main laboratory findings were examined, there

Table 1. Baseline characteristics of patients with infective endocarditis-related ST-elevation myocardial infarction

Variables	All patients (n=19)	Survivors (n=10)	Non-survivors (in-hospital death) (n=9)	*p*
Age (years)	52±11.8	50±6	56±16	0.273
Gender, female, n (%)	9 (47.4)	3 (30.0)	6 (66.7)	0.128
Diabetes Mellitus, n (%)	6 (31.6)	2 (20.0)	4 (44.4)	0.259
Hypertension, n (%)	7 (36.8)	3 (30.0)	4 (44.4)	0.430
Amyloidosis n (%)	1 (5.3)	1 (10.0)	0 (0)	0.526
Chronic kidney disease, n (%)	3 (15.8)	2 (20.0)	1 (11.1)	0.542
COPD, n (%)	2 (10.5)	2 (20.0)	0 (0)	0.263
Smoker, n (%)	6 (31.6)	5 (50.0)	1 (11.1)	0.091
Prior CAD, n (%)	5 (26.3)	3 (30.0)	2 (22.2)	0.556
Malignancy, n (%)	1 (5.3)	0 (0)	1 (11.1)	0.474
Atrial fibrillation, n (%)	4 (21.1)	1 (10.0)	3 (33.3)	0.249
NYHA, n (%)				
NYHA I/II	10 (52.6)	6 (60.0)	4 (44.4)	0.414
NYHA III/IV	9 (47.4)	4 (40.0)	5 (55.6)	
Clinical presentation, n (%)				
Chest pain	12 (63.2)	7 (70.0)	5 (55.6)	0.430
Dyspnea	17 (89.5)	9 (90.0)	8 (88.9)	0.737
Palpitation	10 (52.6)	5 (50.0)	5 (55.6)	0.586
Fever	15 (78.9)	8 (80.0)	7 (77.8)	0.667
Syncope	2 (10.5)	0 (0)	2 (22.2)	0.211

CAD: coronary artery disease; COPD: chronic obstructive pulmonary disease; NYHA: New York Heart Association; STEMI: ST-segment elevation myocardial infarction.

were no significant intergroup differences, except for platelet count (270 [179-321] 10^9/L vs. 175 [129-211] 10^9/L, p=0.043). Vegetation was visualized using TEE in all the patients. In 10 patients, the vegetation was previously detected by transthoracic echocardiography. In our series, the mitral valve was the most common site of infection (11 patients [57.9%]) (Figure 1A). Ten patients had native valve involvement, eight had mechanical prosthetic valve involvement, and one had bioprosthetic valve involvement. There was no significant intergroup difference in infected valve localization or type (Table 2). In our series, only one patient presented with aortic periannular complication (abscess); no pseudoaneurysms or fistulas were encountered. Eleven patients had moderate valve regurgitation because of IE. Of these, five had native mitral valve regurgitation, two had native aortic regurgitation, and four had para-prosthetic leakage. None of the patients showed signs of valve obstruction. Native mitral valve regurgitation owing to IE was significantly more common in the survivors group (50% vs. 0%, p=0.022). The causative agent was isolated in 19 patients (100%). The following microorganisms were identified: *Staphylococcus aureus* in seven, *Staphylococcus epidermidis* in five, other coagulase-negative staphylococci *in three, Group B Streptococcus* in two, *Candida albicans* in one, and *Brucella melitensis* in one. There was no statistically significant intergroup difference in microbiological characteristics (Table 2).

ECG revealed anterior STEMI in 11 patients, inferior STEMI in five, inferolateral STEMI in two, and lateral STEMI in one. Table 3 shows the complications and management of patients diagnosed with IE-related STEMI. The timing of IE-related STEMI was six (range four to 11) days after admission. There was no statistically significant intergroup difference (six [range four to 12] days vs. seven [three to 10] days; p=0.780). Moreover, cerebral and peripheral embolism

Table 2. Baseline echocardiographic and main laboratory findings of patients with infective endocarditis-related ST-elevation myocardial infarction

Variables	All patients (n=19)	Survivors (n=10)	Non-survivors (in-hospital death) (n=9)	*p*
White blood cell count, (10^9/L)	16.5 (13.9-18.2)	15.5 (13.9-17.18)	16.9 (15.5-18.2)	0.400
Hemoglobin, (g/dL)	10.5 (9.2-11.9)	10.5 (8.6-11.0)	11.5 (9.7-12.0)	0.278
Platelet (10^9/L)	211 (132-285)	270 (179-321)	175 (129-211)	0.043
CRP, (mg/dL)	27.8 (13.1-71.9)	45.9 (21.0-115.0)	20.7 (13.1-29.0)	0.356
ESR, (mm/h)	95 (72-122)	119 (88-124)	80 (72-102)	0.156
Troponin T, (ng/mL)*	1.05 (0.64-2.08)	1.08 (0.78-1.9)	1.05 (0.45-2.08)	0.661
Procalcitonin, (ng/mL)	6.76 (4.45-9.26)	5.88 (3.2-7.6)	6.76 (5.27-9.26)	0.497
Creatinine, (mg/dL)	1.02 (0.88-1.3)	1.15 (1.0-1.3)	0.98 (0.88-1.15)	0.356
LV ejection fraction (%)	55 (40-55)	52.5 (40-55)	55 (50-55)	0.842
LV end-diastolic diameter (mm)	49 (45-55)	46.5 (45-55)	50 (45-51)	0.842
LV end-systolic diameter (mm)	35 (29-38)	34 (29-40)	36 (35-37)	0.780
Left atrium diameter (mm)	39 (35-45)	36 (35-41)	42 (37-45)	0.182
Moderate to severe TR, n (%)	6 (31.6)	4 (40.0)	2 (22.2)	0.370
TAPSE (mm)	18.5 (17-21)	17.8 (17-21)	19 (18-20)	0.604
Infected valve location, n (%)				
Aortic	8 (42.1)	4 (40.0)	4 (44.4)	0.605
Mitral	11 (57.9)	6 (60.0)	5 (55.6)	0.605
Infected valve type, n (%)				
Native	10 (52.6)	7 (70.0)	3 (33.3)	0.128
Bioprosthetic	1 (5.3)	0 (0)	1 (11.1)	0.474
Mechanical prosthetic	8 (42.1)	3 (30.0)	5 (55.6)	0.255
Size of vegetation, (mm)	15 (12-24)	16 (12-24)	15 (13-19)	1.000
Abscess, n (%)	1 (5.3)	0 (0)	1 (11.1)	0.474
Pseudoaneurysms or fistula, n(%)	0 (0)	0 (0)	0 (0)	-
Newly developed moderate or severe regurgitation, n (%)				
Native aortic vale	2 (10.5)	1 (10.0)	1 (11.1)	0.737
Native mitral valve	5 (26.3)	5 (50.0)	0 (0)	0.022
Paravalvular leakage	4 (21.1)	1 (10.0)	3 (33.3)	0.249
Causative organism, n (%)				
Staphylococcus aureus	7 (36.8)	2 (20.0)	5 (55.6)	0.556
Staphylococcus epidermidis	5 (26.3)	3 (30.0)	2 (22.2)	0.263
Other coagulase-negative staphylococci	3 (15.8)	2 (20.0)	1 (11.1)	0.542
Group B streptococcus	2 (10.5)	2 (20.0)	0 (0)	0.526
Brucella melitensis	1 (5.3)	1 (10.0)	0 (0)	0.474
Candida albicans	1 (5.3)	0 (0)	1 (11.1)	0.474

*Normal range: 0-0.014 ng/mL.

CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; LV: left ventricle; TAPSE: Tricuspid Annular Plane Systolic Excursion; TR: tricuspid regurgitation.

Table 3. Complication and clinical outcomes of the patients with infective endocarditis-related ST-elevation myocardial infarction

Variables	All patients (n=19)	Survivors (n=10)	Non-survivors (in-hospital death) (n=9)	*p*
Timing from admission to SCE, days	6 (4-11)	6 (4-12)	7 (3-10)	0.780
ICU stay (days)	15 (11-22)	13 (10-21)	18 (11-29)	0.278
Total hospital stay (days)	32 (21-42)	40.5 (32-43)	22 (12-32)	0.022
Cardiogenic shock, n (%)	6 (31.6)	1 (10.0)	5 (55.6)	0.050
Fatal ventricular arrhythmia, n (%)	5 (26.3)	0 (0)	5 (55.6)	0.011
Mechanical complication associated with STEMI, n (%)	0 (0)	0 (0)	0 (0)	-
Localization of SCE, n (%)				
LMCA	1 (5.3)	0 (0)	1 (11.1)	0.474
LAD	12 (63.2)	5 (50.0)	7 (77.8)	0.220
LCX	3 (15.8)	3 (30.0)	0 (0)	0.124
RCA	4 (21.1)	2 (20.0)	2 (22.2)	0.667
Cerebral embolism concurrent with SCE, n (%)	6 (31.6)	3 (30.0)	3 (33.3)	0.630
Splenic embolism concurrent with SCE, n (%)	1 (5.3)	0 (0)	1 (11.1)	0.474
Peripheral embolism concurrent with SCE, n (%)	3 (15.8)	1 (10.0)	2 (22.2)	0.458
TIMI 0/I flow before treatment, n (%)	19 (100)	10 (100)	9 (100)	-
TIMI flow after treatment, n (%)				
0	5 (26.3)	0 (0)	5 (55.6)	<0.001
I	2 (10.5)	0 (0)	2 (22.2)	
II	1 (5.3)	0 (0)	1 (11.1)	
III	11 (57.9)	10 (100.0)	1 (11.1)	
Door to wire time, (min)	15.5 (15-18)	15 (15-18)	16 (15-17)	0.690
Treatment strategy				
CABG + valve replacement, n (%)	6 (31.6)	5 (50.0)	1 (11.1)	-
Stent implantation + valve replacement, n (%)	5 (26.3)	5 (50.0)	0 (0)	-
Only PTCA, n (%)	4 (21.1)	0 (0)	4 (44.4)	-
Conservative therapy, n (%)	3 (15.8)	0 (0)	3 (33.3)	-
Manual thrombectomy, n (%)	1 (5.3)	0 (0)	1 (11.1)	-

CABG: coronary artery by-pass grafting; ICU: intensive care unit; LMCA: left main coronary artery; LAD: left anterior descending; LCX: left circumflex artery; PTCA: percutaneous transluminal coronary angioplasty; RCA: right coronary artery; SCE: septic coronary embolism; STEMI: ST-segment elevation myocardial infarction.

developed simultaneously in 10 patients with IE-related STEMI. Of these, six were cerebral, one was splenic, and three were peripheral arterial embolisms. No statistically significant intergroup difference was found in these embolic events (Table 3). Six (31.6%) patients had CS requiring vasopressors; of them, three were treated with an intra-aortic balloon pump. Table 3 presents the clinical outcomes and complications. Although the intensive care unit duration of stay did not differ significantly between groups, the total hospital stay was significantly longer in the survival group (40.5 [range 32-43] vs. 22 [range 12-32] days, p=0.022). Catheter-based coronary angiography was performed in all the patients. The most common localization of the infarct-related artery (in 12 patients [63.2%]) was the left anterior descending artery, followed by the right coronary artery in four (Figure 1B) and the left circumflex artery in three (Table 3). Before treatment, all the patients had a TIMI flow of 0/I. The major treatment strategies for the patients included thrombus aspiration

Table 4. PubMed database search summary for revascularization strategies of case reports of patients who developed infective endocarditis-related ST-segment elevation myocardial infarction between 1991 and 2021

Study	A/G	Causative organism	IRA	Vegetation valve localization/type	Concomitant SE/PE	Revascularization strategy	Outcome
Herzog et al.[22]							
Patient 1	21/M	S. viridans	LAD	Mitral/native	-	PTCA	Alive
Patient 2	38/M	Not reported	LAD	Mitral/native	-	t-PA	Alive
Beldner et al.[23]	31/F	Lactobacillus	LAD	Mitral/native	-	Stent	Alive
DeKam et al.[24]	85/M	CNS	Diagonal	Mitral/native	-	Thrombectomy	Alive
Casazza et al.[25]	47/F	-	RCA	Aort+Mitral/native	-	CABG	Alive
Yeoh et al.[26]	71/F	S. aureus	LAD/PDA	Aort/native	-	Thrombectomy	Died
Voss et al.[28]	39/F	E. faecalis	LAD	Mitral/native		PTCA	Alive
Dhawan et al.[29]	53/F	Not reported	LAD	Aort/native	+	CABG	Alive
Donal et al.[30]	-	-	LAD	Mitral/native	-	PTCA	Alive
Ural et al.[18]	59/F	-	LAD	Aort/native	-	PTCA	Alive
Di Salvo et al.[31]	49/M	S. viridans	LAD	Aort+mitral/native	-	t-PA	Died
Ortega-Carnicer et al.[32]	73/M	-	RCA	Mitral/native	-	Tenecteplase	Alive
Esen et al.[8]	22/F	-	LMCA	Aort/MPV	-	CABG	Died
Glazier et al.[33]	37/M	S. bovis	LAD	Aort/native	-	Stent	Alive
Gultekin et al.[34]	40/M	CNS	LAD	Aort+mitral/MPV	-	Medical therapy	Died
Hibbert et al.[40]	53/M	S. aureus	LAD	Aort/native	-	Thrombectomy+PTCA+Stent	Died
Hohmann et al.[35]	16/F	S. mitis	LAD	Aort/native	-	Stent	Alive
Okai et al.[36]	53/M	S. sangius	LAD	Mitral/native	-	CABG	Alive
Perera et al.[21]	54/F	S. aureus	LCx	Aort/native	-	t-PA	Died
Roxas et al.[37]							
Patient 1	39/F	Staphylococci	LAD	Mitral/Native	-	Fibrinolytic	Alive
Patient 2	56/M	S. bovis	LAD	Mitral/native		Fibrinolytic	Alive
Singh et al.[11]	70/M	S. aureus	RCA	Aort/native	-	Stent	Died
Winkler et al.[44]	67/M	Gamella	LAD+D1	Mitral/native	-	Medical therapy	Died
Llaó-Ferrando et al.[45]	70/M	S.epidermidis	LAD	Aort/native	-	Thrombectomy	Alive
Wojciuk et al.[27]	70/M	Streptococcus group G	LCx	Mitral/native	+	Thrombectomy	Alive
Baek et al.[19]	27/M	S. viridans	LAD	Mitral/native	-	CABG	Alive
Chen et al.[39]	39/M	E. faecalis	RCA	Mitral/native	-	Tenectaplase	Alive
Luther et al.[38]	73/M	Not reported	LAD	Aort/BPV	-	Medical therapy	Died
Kleczyński et al.[41]	55/F	-	RCA	Mitral/native	-	Medical therapy	Died
Seo et al.[42]	53/M	Aspergillus	LAD	Mitral/native	-	Thrombectomy	Died
Maqsood et al.[43]	40/M	C. albicans	LAD	Aort/MPV	-	Thrombectomy	Died
Sugi et al.[20]	73/M	S. oralis	LMCA	Aort/native	+	Thrombectomy+Stent	Alive
Açar et al.[16]	54/M	Brucella	LAD	Aort/native	-	CABG	Alive
Oestreich et al.[10]	27/M	E. faecalis	RCA	Mitral/BPV	-	Thrombectomy	Alive
Thompson et al.[46]	22/F	Not reported	RCA	Mitral/native	+	Thrombectomy	Died
Murtaza et al.[48]	56/M	Enterococci	LAD	Aort/native	-	Stent	Alive

Table 4. PubMed database search summary for revascularization strategies of case reports of patients who developed infective endocarditis-related ST-segment elevation myocardial infarction between 1991 and 2021 (Continue)

Study	A/G	Causative organism	IRA	Vegetation valve localization/type	Concomitant SE/PE	Revascularization strategy	Outcome
Regmi et al.[49]	69/M	S. aureus	LAD	Mitral/native	+	Medical therapy	Died
Aron et al.[47]	57/F	C. glabrata	RCA	Aort/native	-	Thrombectomy	Alive
García-Izquierdo et al.[50]	55/M	S. faecalis	LAD	Aort+mitral/native	-	Thrombectomy	Alive
Calero-Núñez et al.[51]							
Patient 1	71/M	-	LCx	Mitral/native	+	Stent	Alive
Patient 2	67/M	S. sanguinis	LCx	Mitral/native	-	Thrombectomy+PTCA	Alive
Patient 3	59/M	S.epidermidis	RCA	Aort/native	-	Medical therapy	Alive
Campanile et al.[52]	82/F	S. aureus	LMCA	Aort/MPV	-	Stent	Died
Karaarslan et al.[53]	64/M	CNS	RCA	Mitral/native	+	Medical therapy	Died
Joy et al.[54]	63/M	E. coli	LAD	Aort/MPV	-	Stent	Alive
Panagides et al.[17]	48/F	S. oralis	LCx	Mitral/BPV	-	Thrombectomy+PTCA	Alive
Bolton et al.[55]	53/F	S. aureus	RCA	Aort/native	-	Coronary repair	Alive
Khiatah et al.[56]	22/M	A. defectiva	RCA	Aort/native	+	Stent	Alive
Ghazzal. et al.[57]	38/M	C. albicans	LAD	Aort/native	-	Medical therapy	Alive
Prashar et al.[58]	54/M	S. agalactiae	Septal	Aort/native	-	Medical therapy	Alive
Cho et al.[59]	80/M	S.epidermidis	RCA	Aort/BPV	-	Thrombectomy	Alive
Denegri et al.[60]	77/M	S. aureus	LMCA	Aort/MPV	+	Stent	Died
Fujito et al.[61]	79/F	S. agalactiae	LAD	Mitral/native	-	Medical therapy	Died

A. defectiva: *abiotrophia defectiva*; A/G: age/gender; C. glabrata: candida glabrata; C. albicans: *candida albicans*; CNS: coagulase negative Staphylococcus; E. faecalis: *enterococcus faecalis*; E. coli: *escherichia coli*; LAD: left anterior descending artery; LCx: left circumflex artery; LMCA: left main coronary artery; IRA: infarct related artery; MPV: mechanical prosthetic valve; BPV: bioprosthetic valve; RCA: right coronary artery; S. aureus: *staphylococcus aureus*; S. bovis: *streptococcus bovis*; S. epidermidis: *staphylococcus epidermidis*; SE/PE: systemic/peripheral embolism; S. mitis: *streptococcus mitis*; S. viridans: *streptococcus viridans*; S. agalactiae: *streptococcus agalactiae*; S. fecalis: *streptococcus fecalis*; S. sanguinis: *streptococcus sanguinis*; S. oralis: *streptococcus oralis*; t-PA: recombinant tissue plasminogen activator.

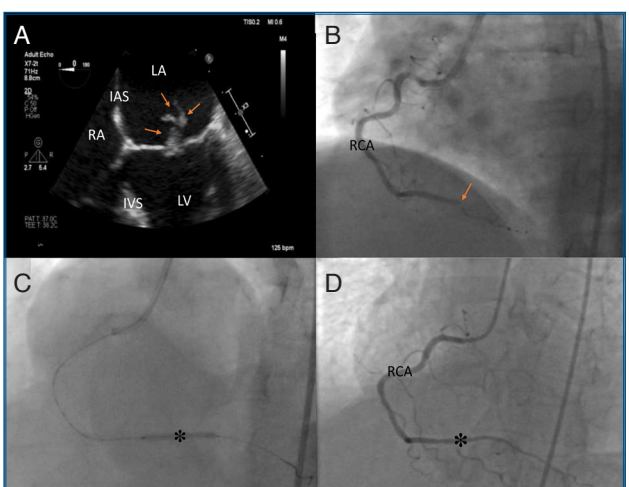

Figure 1. (A) Two-dimensional transesophageal echocardiography indicates a vegetation on the mitral valve (arrowhead). **(B-D)** Coronary angiography demonstrates a total occlusion of the distal right coronary artery and successful revascularization with stent implantation (asterisk).

(n=1), valve replacement following stent implantation (n=5) (Figures 1C, 1D), direct balloon angioplasty (n=4), valve replacement and coronary artery bypass grafting (CABG) (n=6), and conservative therapy (n=3). After intervention therapy, TIMI-III flow was significantly more common in the survival group (100% vs. 0%; p<0.001) (Table 3). All these patients preferred CABG or stent implantation for revascularization. All the patients who underwent direct balloon angioplasty, manual thrombectomy, and conservative treatment died. Moreover, fatal VAs and CS were significantly less common in the survivors group (0% vs. 55.6%, p=0.011; 10% vs. 55.6%, p=0.05, respectively) (Table 3).

DISCUSSION

The major findings of this study were as follows: the mortality rate of IE-related STEMI is quite high;

successful revascularization with stent implantation or CABG enables short-term survival in this patient group; and survival after isolated balloon angioplasty or non-interventional treatment, where successful revascularization cannot be achieved, is unlikely. The in-hospital mortality rate was 47.3%, that of CS was 31.6%, and those of concurrent cerebral embolism and peripheral and splenic embolisms were 31.6%, 31.6%, and 5.3%, respectively. This study represents the largest retrospective series published in the literature evaluating the clinical outcomes of revascularization strategies in patients with IE-related STEMI.

STEMI is a very rare complication, occurring in 0.8%-1% of patients with IE.[5,6,12,16,17] Despite numerous case reports and one meta-analysis and systematic review,[12] the optimal treatment strategy for IE-related STEMI remains controversial. Several revascularization strategies have been reported in the literature, such as mechanical thrombectomy, direct balloon angioplasty, bypass surgery or surgical embolectomy, stent implantation, and even fibrinolytic therapy, with inconsistent results (Table 4).[5,6,9-12,17-62] In the era of revascularization, stent implantation is the first-line treatment for STEMI because of atherosclerosis.[7] However, despite the concern that this treatment strategy may lead to the formation of mycotic coronary aneurysms, long-term data are not available in the literature; it should also be noted that this concern is based on a limited number of case reports.[11,63] These aneurysms can also occur when the angioplasty balloon strikes the septic embolism vessel wall. However, continuation of antibiotic therapy after the revascularization period can eliminate this concern. In addition, owing to valve pathology following stent implantation, surgical intervention may be important for optimizing treatment. Moreover, the surgical option has the advantage of allowing tissue debridement, valve replacement, and coronary artery bypass. However, emergency surgical revascularization is difficult in the case of STEMI, which is not possible in a hospital without cardiac surgery. It must be favored in cases of incomplete occlusion of the vessel or in cases of spontaneous restoration of the distal blood flow after initial complete occlusion. In this study, stent implantation was the first-line treatment strategy in five patients with STEMI, followed by surgical intervention for valve pathology. TIMI-III flow was achieved in all the patients, and the survival rate was 100%.

Thromboaspiration with a large catheter may be the optimal technique for restoring complete blood flow.[64] Unfortunately, when adequate retrieval of the septal debris is impossible using aspiration alone, balloon angioplasty with stent implantation must be considered. However, the large burden of embolic material can necessitate alternative techniques for restoring epicardial coronary artery flow, including the use of a larger catheter such as the GuideLiner to ensure efficient thrombectomy.[10] Moreover, the manual thrombectomy technique presents its own complications with an increased risk of stroke.[64]

It is well known that mortality rates are high in patients with STEMI who cannot be adequately revascularized (TIMI 0/I).[7] The conservative management of patients with IE-related STEMI has also been associated with high mortality rates in some case reports. In our study, all the patients who were followed up with medical treatment died. In addition, adjuvant therapy such as heparin and antiplatelet agents, is a major concern. There are no data on this strategy; however, it can be chosen with consideration that surgical valve replacement will be required in most patients. In addition, many patients are at risk of hemorrhagic complications, making the choice even more difficult. However, pharmacological treatment must consider both the bleeding and thrombotic risks because of the acute presentation.

Despite advances in revascularization strategies, CS and fatal VAs remain the most common causes of death in patients with STEMI.[7] Robust evidence has been provided regarding the early mortality risk associated with STEMI complicated by CS and fatal Vas.[7,65]

Limitations

It is important to emphasize the limitations pertinent to the methods of this study. First, this was a retrospective study and included a relatively small patient population. However, it should be noted that IE-related STEMI is a very rare complication of IE. Second, the current data covered short-term (in-hospital or 30-day) mortality and morbidity. Finally, the absence of pathological confirmation of embolic material was another limitation.

Conclusion

Consensus is lacking regarding the most appropriate revascularization strategy for patients with IE-relat-

ed STEMI. Stent implantation or revascularization with CABG has a lower mortality rate in IE-related STEMI and may be considered in this patient population. Manual thromboaspiration or direct balloon angioplasty does not seem appropriate as complete blood flow restoration is impossible. Moreover, treatment must be individualized based on angiographic features and cardiac surgery center availability.

Ethics Committee Approval: Ethics committee approval was received for this study from the Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital Institutional Ethics Comittee (Approval Date: 12/07/2021; Approval Number: 2021/60).

Informed Consent: Informed consent was obtained from the patients who participated in this study.

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Keywords: Acute myocardial infarction/STEMI; coronary artery disease; endocarditis; primary percutaneous coronary intervention

Anahtar Kelimeler: Akut miyokart enfarktüsü/STYME; koroner arter hastalığı; endokardit; primer perkütan koroner girişim