the AMI onset. Therefore, the presence or absence should be assessed in an ECG that is recorded before the AMI onset. If there is no ECG available before the AMI onset on hand, a previously recorded ECG should be collected whenever possible to clarify the risk for VF.

Study limitations

First, the approximately 10 % prevalence of VF in this study seemed to be higher than that of the previous studies.[18,20,22] The prevalence of ER before the onset of an AMI was 15%, which was comparable to that in a previous study from Japan.[32] However, it was also higher than that of the previous studies in the general population of Western countries.[11] The age, gender, and race-ethnic differences between this study and the previous studies might account for the differences in the prevalence of both VF and ER.[11,18,20,22] A recent study with Japanese patients demonstrated that the incidence of in-hospital VF/tachycardia during an AMI was 13.3%,[33] which was also higher than that of the previous studies conducted in Europe and the USA.[18,20,22] Another plausible reason might be due to the adequate management of VF before catheterization. The recent spread of the use of automated external defibrillators (AEDs) outside the hospital and by-stander cardio-pulmonary resuscitation in Japan may decrease the sudden cardiac death, but increase the prevalence of VF episodes before catheterization during the acute phase of an AMI. In this study, among 13 patients who had VF episodes before catheterization, 5 patients received an appropriate shock using AEDs before arrival at the hospital, which might contribute to the high prevalence of VF in this study. Second, the small sample size limits our power and is reflected in the broad confidence intervals, most notably in the adjusted statistical analyses regarding the incidence of ER. Third, because the presence of ER was assessed with only one 12-lead ECG obtained before the AMI onset, we could not evaluate the time course and reproducibility of the ER, and the prevalence of the ER might have been underestimated.[6,10]

Fourth, the information on hypokalemia, a lack of preinfarction angina, chronic kidney disease, and a family history of sudden cardiac death, which have been reported as independent predictors of VF occurrence in patients with an AMI,[18-22] were lacking in this study. Therefore, further prospective studies with a larger sample size, long-term follow-up, and the participation of many hospitals may be needed to resolve these limitations and to ensure and enhance our results.

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Table 1. Demographic and clinical characteristics of the patients with and without occurrences of ventricular fibrillation (VF).

	Total (n=220)	VF occurrence (n=21)	No VF occurrence (n=199)	*p value*
Age, years	69±11	67±9	69±12	0.382
Male gender, n (%)	163 (74%)	20 (95%)	143 (72%)	0.020
Cardiovascular risk factors				
Hypertension, n (%)	153 (70%)	16 (76%)	137 (69%)	0.487
Hyperlipidemia, n (%)	107 (49%)	11 (52%)	96 (48%)	0.718
Diabetes mellitus, n (%)	82 (37%)	9 (43%)	73 (37%)	0.578
Smoking, n (%)	110 (50%)	12 (57%)	98 (49%)	0.491
Culprit artery				
RCA, n (%)	73 (33%)	10 (48%)	63 (32%)	0.288
LAD, n (%)	97 (44%)	9 (43%)	88 (44%)	
LCx, n (%)	40 (18%)	1 (5%)	39 (20%)	
LMT, n (%)	10 (5%)	1 (5%)	9 (5%)	
Number of diseased coronary arteries, n	1.7±0.8	2.0±0.8	1.7±0.8	0.044
Time from the symptom onset to the ER, min	386±398	195±235	406±406	<0.001
Killip class on admission, n (%)				
I	156 (71%)	6 (29%)	150 (75%)*	<0.001
II	32 (14%)	4 (19%)	28 (14%)	
III	13 (6%)	2 (9%)	11 (6%)	
IV	19 (9%)	9 (43%)	10 (5%)*	
Peak creatine kinase levels, U/L	2,315±2,101	3,245±2,714	2,193±1,994	0.092
STEMI, n (%)	175 (80%)	20 (95%)	155 (78%)	0.085
Early repolarization, n (%)	34 (16%)	10 (48%)	24 (12%)	<0.001
Distribution				
Inferior leads, n (%)	26 (12%)	8 (38%)	18 (9%)	0.001
Amplitude				
≥0.2mV	17 (8%)	6 (29%)	11 (6%)	0.002
Morphology				
Notching	26 (12%)	8 (38%)	18 (9%)	0.001
Slurring	8 (4%)	2 (10%)	6 (3%)	0.171
ST-segment				
Upsloping	9 (4%)	1 (5%)	8 (4%)	0.602
Horizontal/descending	25 (12%)	9 (43%)	16 (8%)	<0.001

The values are reported as the mean±standard deviation or n (%). *p<0.001 vs. VF occurrence. ER=emergency room; LAD=left anterior descending artery; LCx=left circumflex artery; LMT=left main trunk; RCA=right coronary artery; STEMI=ST elevated myocardial infarction.

Table 2. Univariate and multivariate logistic regression analyses of ventricular fibrillation occurrence.

Variables	Univariate Odds ratio (95% confidence interval)	*p value*	Multivariate* Odds ratio (95% confidence interval)	*p value*
Age per year	0.983 (0.945–1.022)	0.381	0.964 (0.912–1.019)	0.198
Male gender	7.832 (1.027–59.754)	0.047	7.353 (0.663–81.538)	0.104
Time from the symptom onset to ER of < 180 min	2.468 (0.978–6.227)	0.056	3.767 (1.127–12.587)	0.031
A Killip class > I	7.653 (2.815–20.807)	<0.001	13.598 (3.425–53.990)	<0.001
Peak creatine kinase levels > 3000 U/L	2.495 (0.989–6.291)	0.053	0.691 (0.212–2.252)	0.540
Number of diseased coronary arteries >1	2.629 (0.980–7.052)	0.055	3.257 (0.926–11.460)	0.066
ST elevated myocardial infarction	5.677 (0.741–43.492)	0.095	2.574 (0.223–29.695)	0.449
Hypertension	1.448 (0.508–4.130)	0.489	0.636 (0.176–2.305)	0.491
Diabetes mellitus	1.295 (0.521–3.219)	0.579	0.752 (0.231–2.454)	0.637
Smoking	1.374 (0.554–3.406)	0.493	0.937 (0.307–2.861)	0.908
Early repolarization	6.629 (2.546–17.256)	<0.001	7.305 (2.210–24.144)	0.001

ER=emergency room. * All the variables in the columns were used in the multivariate model.

Table 3. Univariate and multivariate logistic regression analyses of ventricular fibrillation (VF), according to the J-point pattern in the inferior leads.*

Variables	No of VFs n (%)	Univariate Odds ratio (95% confidence interval)	*p value*	Multivariate* Odds ratio (95% confidence interval)	*p value*
No J-point elevation (N=186)	11 (6%)	1.000		1.000	
J-point elevation of ≥0.1 mV in the inferior leads (N=26)	8 (31%)	6.188 (2.265–16.908)	<0.001	6.849 (2.006–23.385)	0.002
J-point elevation of ≥0.2 mV in the inferior leads (N=14)	6 (43%)	9.550 (2.929–31.135)	<0.001	10.649 (2.346–48.343)	0.002
J-point notched elevation of ≥0.1 mV in the inferior leads (N=22)	7 (32%)	6.133 (2.149–17.507)	0.001	4.883 (1.357–17.565)	0.015
Inferior J-point elevation with a horizontal/descending ST-segment (N=21)	8 (38%)	8.805 (3.097–25.033)	<0.001	8.049 (2.181–29.696)	0.002

*The variables that were included in the multivariate analyses were the age, sex, time from symptom onset to arrival in the emergency room of less than 180 min, Killip class of greater than I, peak creatine kinase levels greater than 3000 U/L, number of diseased coronary arteries greater than 1, ST elevated myocardial infarction, hypertension, diabetes mellitus, and smoking.

Figure Legends:

Figure 1. Baseline electrocardiograms from patients with early repolarization. This shows two patients with a J-point elevation of more than 0.2 mV. Panel A has a notched elevation (arrows) in the inferior leads and Panel B has a slurred elevation (arrows) in the inferior and lateral leads.

Figure 2. A representative case of preserved early repolarization. (**A**) A baseline 12-lead ECG which was obtained 6 months before the AMI onset shows a notched early repolarization with an upsloping ST-segment in the inferior leads. (**B**) A 12-lead ECG during the onset of an anterior AMI showing preserved early repolarization in the inferior leads. AMI=acute myocardial infarction.

Figure 3. A representative case with serial 12-lead ECGs who consecutively suffered from anterior and inferior AMIs within 3 months of each other. (**A**) A baseline 12-lead ECG which was obtained 1 month before the AMI onset demonstrating a notched early repolarization (ER) with a horizontal/descending ST-segment in the inferior leads (horizontal ST-segment in leads II and aVF; descending ST-segment in lead III). **(B)** An ECG which was obtained at the onset of an anterior AMI showing the lack of ER because of reciprocal ST-T changes in the inferior leads. **(C)** At 2 weeks after the onset of the anterior AMI, the J point was re-elevated in the inferior leads. **(D)** Three months after an anterior AMI onset, the case suffered from an inferior AMI, and the ECG at that time demonstrated no ER. **(E)** The ER had still vanished 2 weeks after the inferior AMI onset. AMI=acute myocardial infarction.