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Clinical paper

Diagnostic value of lead aVR in electrocardiography for identifying acute coronary lesions in patients with out-of-hospital cardiac arrest

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Abstract

Aim: There is no simple clinical tool that reliably indicates the presence of acute coronary lesions in out-of-hospital cardiac arrest (OHCA) patients without typical ST-segment elevations. ST-segment elevation in electrocardiographic lead aVR suggests global subendocardial ischemia. This study aimed to evaluate the diagnostic value of lead aVR for identifying acute coronary lesions following resuscitation from OHCA.

Methods: A total of 74 patients without evidence of ST-segment elevations, who were resuscitated from OHCA, were examined. The degree of ST-segment elevation in lead aVR was measured directly after return of spontaneous circulation (ROSC) and at early follow-up. Coronary angiograms were retrospectively reviewed.

Results: Acute coronary lesions were detected in 20 patients (27%). No difference in ST-segment elevation in lead aVR directly after ROSC was observed between patients with or without acute coronary lesions. However, ST-segment elevation values significantly decreased at early follow-up (median, 137 min) in patients without acute coronary lesions. An ST-segment elevation ≥ 0.5 mm in lead aVR at early follow-up was associated with a higher prevalence of multivessel coronary artery disease and was an independent indicator of the presence of acute coronary lesions (odds ratio, 4.41; 95% confidence interval, 1.12–17.4; $p=0.034$).

Conclusion: ST-segment elevation in lead aVR at early follow-up was associated with the presence of acute lesions accompanied by severe coronary artery disease in post-cardiac arrest patients without other ST-segment elevations. The analysis of ST-segment elevation in lead aVR may aid in the identification of patients who will benefit from further invasive coronary diagnostic procedures.

Keywords: Out-of-hospital cardiac arrest, Acute coronary lesion, Lead aVR

Introduction

The majority of out-of-hospital cardiac arrests (OHCA) are related to obstructive coronary artery disease.^{1,2} Although current guidelines recommend emergency coronary angiography for patients with ST-segment elevation on the electrocardiogram (ECG) after return of spontaneous circulation (ROSC), its role in those without ST-segment elevation remains controversial.^{3,4} Acute coronary culprit lesions are angiographically detected in approximately

26–33% of patients after ROSC, even in the absence of ST-segment elevation.^{5,6} A recent meta-analysis indicated that the performance of early coronary angiography and adjunctive percutaneous coronary intervention (PCI) in cardiac arrest patients without ST-segment elevation was associated with reduced mortality and improved neurological outcomes.⁷ However, the performance of routine emergency coronary angiography in all patients without evidence of ST-segment elevation, particularly in patients with unfavourable comorbidities and a low probability of neurological recovery, raises ethical and cost-effectiveness issues.

Abbreviations: ECG, electrocardiogram; OHCA, out-of-hospital cardiac arrest; PCI, percutaneous coronary intervention; ROSC, return of spontaneous circulation; SYNTAX, SYnergy between percutaneous coronary intervention with TAXus and cardiac surgery.

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Finding a method to rapidly and accurately identify patients with acute coronary lesions after ROSC is of major interest, as this would lead to earlier revascularization for those who require it. Previous studies have reported a higher prevalence of multivessel coronary artery disease in resuscitated patients without ST-segment elevation than in those with ST-segment elevation.^{5,6} An ST-segment elevation in electrocardiographic lead aVR, in combination with widespread ST-segment depressions in other leads, is a key feature of left main or proximal left anterior descending coronary artery occlusion or of severe triple-vessel coronary artery disease that can result in global subendocardial ischemia.⁸ Several studies have shown that an ST-segment elevation in lead aVR can indicate left main coronary artery disease and/or triple-vessel disease in non-ST-segment elevation acute coronary syndrome.^{9,10} However, its diagnostic value in patients resuscitated from OHCA has not been investigated to date.

Accordingly, the present study aimed to investigate whether a singular ST-segment elevation in lead aVR following ROSC in OHCA patients can identify patients with acute coronary lesions in the absence of ST-segment elevations in other leads.

Methods

Patients

This is a retrospective, single-centre observational study. The study flow chart is presented in Fig. 1. A total of 286 OHCA patients who had achieved ROSC were consecutively screened from January 2012 to December 2017 at the National Center for Global Health and Medicine in Tokyo, Japan. Medical histories were reviewed, and 110 patients with obvious extracardiac aetiologies including brain stroke, hemorrhage, sepsis, metabolic disorder, trauma, suffocation, or attempted suicide were excluded. Of the remaining 176 patients with presumed cardiac causes of cardiopulmonary arrest, 65 were excluded and did

not undergo coronary angiography because of severe hypoxic brain damage or unfavourable comorbidities, as assessed by the physician in charge. ECG analysis was performed at the emergency department for 111 patients directly after ROSC. Furthermore, 37 patients were excluded from the analysis owing to ST-segment elevation myocardial infarction, left bundle branch block, or left ventricular hypertrophy on ECG, which interfered with the accurate evaluation of ST segments.

A diagnosis of ST-segment elevation myocardial infarction was established if ST-segment elevations were observed in two contiguous leads and measured ≥ 2.0 mm at the J-point in leads V_2 – V_3 and/or ≥ 1.0 mm in all other leads. Left bundle branch block was defined as QRS duration > 120 ms with QS or rS pattern in lead V_1 and broad R waves in leads I, V_5 , and V_6 . Left ventricular hypertrophy was defined as a sum of R waves in lead V_5 or V_6 and S wave in lead V_1 ≥ 40 mm. Finally, 74 patients without significant ST-segment elevation were included.

The study was approved by the institutional review board of our hospital (reference number 3123). The requirement for acquisition of informed consent was waived owing to the retrospective nature of the study.

Clinical data collection

Data on demographics, cardiovascular risk factors, and resuscitation status including witnessed status (bystander or emergency medical services), bystander cardiopulmonary resuscitation, automated external defibrillator use, initial cardiac rhythm, and time until ROSC were collected from medical records. Troponin I levels at hospital arrival and the use of targeted temperature management and coronary revascularization procedures were also documented for all patients. Troponin I was measured using a quantitative assay (Abbott Japan Co. Ltd., Chiba, Japan), with troponin I level > 0.026 ng/mL (99th percentile cutoff value) considered to be positive.

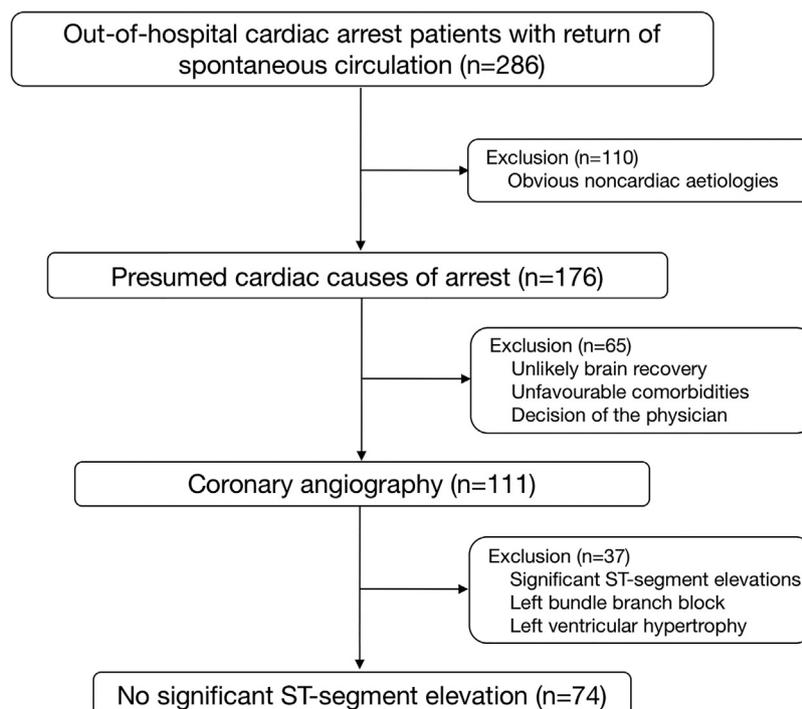


Fig. 1 – Study flow chart.

Electrocardiographic interpretation

Initial and early follow-up ECGs obtained at the emergency department after ROSC were retrospectively analysed by one cardiologist blinded to patients' clinical data and coronary angiography results. ECGs were recorded in standard 12-lead format at a paper speed of 25 mm/s and calibration of 10 mm/mV (Nihon Kohden Corporation, Tokyo, Japan). ST-segment deviations were automatically measured at one-sixteenth of RR interval (in ms) after the J-point, which was determined in leads V₄–V₆, for ST-segment depression and at the J-point for ST-segment elevation using the preceding PR segment as a baseline. The timing of early follow-up ECG was left to the discretion of emergency physician.

Coronary angiography evaluation

Coronary angiography was directly or electively performed during index hospitalization at the discretion of the treating physician. An angiogram was retrospectively reviewed by two experienced interventional cardiologists without knowledge of the electrocardiographic findings. A $\geq 50\%$ diameter stenosis in the major epicardial

vessels or their main branches by visual estimation was considered clinically significant. An acute culprit lesion was defined as follows: (1) a lesion with a local thrombus or ruptured plaque, (2) a vulnerable plaque with a lipid core on intravascular ultrasound, or (3) an occlusion easily recanalized using a guide wire during the subsequent PCI. The κ value for inter-observer variability with respect to the acute culprit lesion was 0.79. In cases of disagreement about acute culprit lesions, two readers consulted with each other and reached a final decision. The coronary status of all patients was scored for anatomic complexity using the SYnergy between percutaneous coronary intervention with TAXus and cardiac surgery (SYNTAX) score algorithm, which is available on the SYNTAX website (www.syntaxscore.com).

PCI was attempted immediately after angiography if an acute culprit lesion that could likely lead to cardiac arrest was detected.

Statistical analysis

Continuous variables with normal distribution were compared using mean (standard deviation). Median (interquartile range) was used for variables with skewed distribution. Continuous variables were analyzed using the Wilcoxon signed-rank test or Mann-Whitney U

Table 1 – Clinical characteristics, coronary angiography results, and in-hospital revascularization.

	Overall (n=74)	Acute coronary lesion		p-value
		No (n=54)	Yes (n=20)	
Demographics				
Age, years	58 (16)	54 (17)	66 (11)	0.001
Male	63 (85%)	46 (85%)	17 (85%)	0.62
Cardiovascular risk factors				
Hypertension	36 (49%)	24 (44%)	12 (60%)	0.23
Diabetes mellitus	16 (22%)	8 (15%)	8 (40%)	0.025
Dyslipidemia	39 (53%)	26 (48%)	13 (65%)	0.20
Current smoker	23 (31%)	19 (35%)	4 (20%)	0.21
Resuscitation status				
Witnessed arrest				
Bystander	56 (76%)	40 (74%)	16 (80%)	0.42
Emergency medical services	9 (12%)	6 (11%)	3 (15%)	0.46
Bystander CPR	54 (73%)	38 (70%)	16 (80%)	0.41
Automated external defibrillator use	61 (82%)	44 (81%)	17 (85%)	0.51
Initial rhythm				
VT/VF	54 (73%)	40 (74%)	14 (70%)	0.73
PEA/asystole	20 (27%)	14 (26%)	6 (30%)	
Time until ROSC, min	13 (8–20)	14 (9–20)	11 (6–20)	0.67
Ongoing CPR at hospital arrival	8 (11%)	5 (9.3%)	3 (15%)	0.37
Cardiac biomarker at hospital arrival				
Troponin I, ng/mL	0.029 (0.010–0.115)	0.020 (0.008–0.093)	0.045 (0.019–2.478)	0.013
Targeted temperature management (34 °C)	26 (35%)	17 (31%)	9 (45%)	0.28
Coronary angiography findings				
Performance within the first 24 h				
Acute culprit lesion	20 (27%)	–	20 (100%)	–
Ruptured plaque or thrombus	–	–	11 (55%)	–
Vulnerable plaque with lipid core	–	–	9 (45%)	–
Stable CAD (diameter stenosis $\geq 50\%$)	23 (31%)	23 (43%)	–	–
No CAD	31 (42%)	31 (57%)	–	–
In-hospital revascularization				
PCI	28 (38%)	9 (17%)	19 (95%)	<0.001
CABG	2 (2.7%)	1 (1.9%)	1 (5.0%)	0.47

Data are expressed as number (%), mean (SD), or median (25th to 75th percentiles). CABG, coronary artery bypass grafting; CAD, coronary artery disease; CPR, cardiopulmonary resuscitation; PEA, pulseless electrical activity; PCI, percutaneous coronary intervention; ROSC, return of spontaneous circulation; VF, ventricular fibrillation; VT, ventricular tachycardia.

test, as appropriate. Categorical variables are presented as percentages, and proportions were compared using χ^2 test or Fisher's exact test, as appropriate. ST-segment elevation in lead aVR was considered significant if it measured ≥ 0.5 mm, as previously described in prior studies that evaluated left main coronary artery disease and/or triple-vessel disease in non-ST-segment elevation acute coronary syndrome.^{9,10} Previous studies reported that approximately 30% of resuscitated patients have acute coronary culprit lesions in the absence of ST-segment elevations. Owing to our small study population, we could not exclude confounding factors in order to evaluate the diagnostic value of lead aVR. Therefore, inverse probability weighting using the propensity score, calculated by logistic regression analysis, was performed to adjust for confounding factors such as age, male sex, hypertension, diabetes mellitus, dyslipidemia, and smoking. Subsequently, we modified the final case number using robust estimation. In addition, sensitivity, specificity, positive predictive value, and negative predictive value for diagnosing an acute coronary lesion were calculated. A two-tailed p -value of <0.05 was considered significant in all analyses. Statistical analysis was performed using SPSS version 25.0 (IBM Japan Ltd., Tokyo, Japan).

Results

Clinical characteristics, coronary angiography findings, and in-hospital revascularizations

The baseline characteristics of 74 patients without evidence of ST-segment elevations who were resuscitated from OHCA are presented in Table 1. The mean age was 58 years, and 63 patients (85%) were male. At the time of resuscitation, 65 patients (88%) had a witnessed arrest (bystander-witnessed arrest, $n=56$; emergency medical service-witnessed arrest, $n=9$), whereas 54 patients (73%) had confirmed initial rhythm of ventricular tachycardia or fibrillation; furthermore, bystanders performed cardiopulmonary resuscitation in 54 patients (73%), and 61 patients (82%) underwent defibrillation using an automated external defibrillator. The median time from cardiac arrest to ROSC was 13 (8–20) min. Cardiopulmonary resuscitation was ongoing at hospital arrival in eight patients (11%). The median troponin I level was 0.029 (0.010–0.115) ng/mL.

Patients with an acute coronary lesion were significantly older, had a higher frequency of diabetes mellitus, and had higher troponin I level.

Coronary angiography was performed within 24 h after admission in 37 patients (50%). Acute culprit lesions were detected in 20 patients (27%); of these patients, 11 had ruptured plaques or thrombi, whereas the remaining 9 had lipid-rich vulnerable plaques that were visualized using intravascular ultrasound. Stable coronary artery disease with $\geq 50\%$ diameter stenosis was detected in 23 patients (31%), and no significant stenosis was observed in 31 patients (42%). Coronary revascularization was performed in 30 patients (41%) during index hospitalization. Moreover, 28 patients underwent PCI and 2 underwent coronary artery bypass grafting.

Initial and early follow-up electrocardiographic features

Initial ECG was obtained within 10 min of hospital arrival or after ROSC in the case of ongoing cardiopulmonary resuscitation at hospital arrival. Early follow-up ECG was acquired at a median time of 137 (53–287) min after hospital arrival. Initial ECG showed comparable ST-segment elevations in lead aVR as well as a similar sum of ST-segment depressions and number of leads with ST-segment depressions irrespective of the presence of an acute coronary lesion. The ST-segment elevation in lead aVR at early follow-up decreased in patients without an acute coronary lesion. However, the ST-segment elevation in lead aVR on early follow-up ECG did not decrease in patients with an acute coronary lesion (0.1 mm vs. 0.5 mm; $p=0.01$) (Table 2). Fig. 2 shows representative cases of initial and early follow-up ECGs along with respective angiographic findings. Patients treated with epinephrine prior to the performance of ECG analysis had higher ST-segment elevations in lead aVR on initial ECG than those who were not (0.8 mm vs. 0.2 mm; $p<0.001$). However, no ST-segment differences were observed among patients treated with antiarrhythmic drugs (amiodarone or lidocaine), defibrillator, and targeted temperature management. These results were confirmed by early follow-up ECG.

Correlation between coronary artery disease complexity and ST-segment elevation in lead aVR on early follow-up ECG

At early follow-up, the location of acute culprit lesions was similar in patients with ST-segment elevation ≥ 0.5 mm in lead aVR and patients

Table 2 – Initial and early follow-up electrocardiographic features after return of spontaneous circulation with respect to the presence of acute coronary lesions.

	Acute coronary lesion					
	No ($n=54$)			Yes ($n=20$)		
	Initial ECG (<10 min)	Early follow-up ECG (median, 137 min)	p -value	Initial ECG (<10 min)	Early follow-up ECG (median, 137 min)	p -value
Maximal ST-segment depression, mm	0.6 (0.2–1.5)	0.4 (0.2–1.0)	0.002	1.2 (0.6–3.0) [†]	1.0 (0.2–1.6) [†]	0.008
Sum of ST-segment depressions, mm	1.9 (0.2–5.2)	0.8 (0.2–3.4)	0.002	3.7 (1.6–12.5)	2.9 (0.5–6.2)	0.014
Number of leads with ST-segment depressions ≥ 0.5 mm	2 (0–5)	0 (0–3)	0.001	5 (2–7)	4 (1–5) [†]	0.058
ST-segment elevation in lead aVR, mm	0.2 (0.0–0.5)	0.1 (0.0–0.3)	0.001	0.4 (0.0–0.7)	0.5 (0.0–0.9) [†]	0.60
ST-segment elevation ≥ 0.5 mm in lead aVR	18 (33%)	8 (15%)	0.002	9 (45%)	10 (50%) [†]	1.0

Data are expressed as number (%) or median (25th to 75th percentiles).

[†] $p<0.05$ compared with no acute coronary lesion. ECG, electrocardiogram.

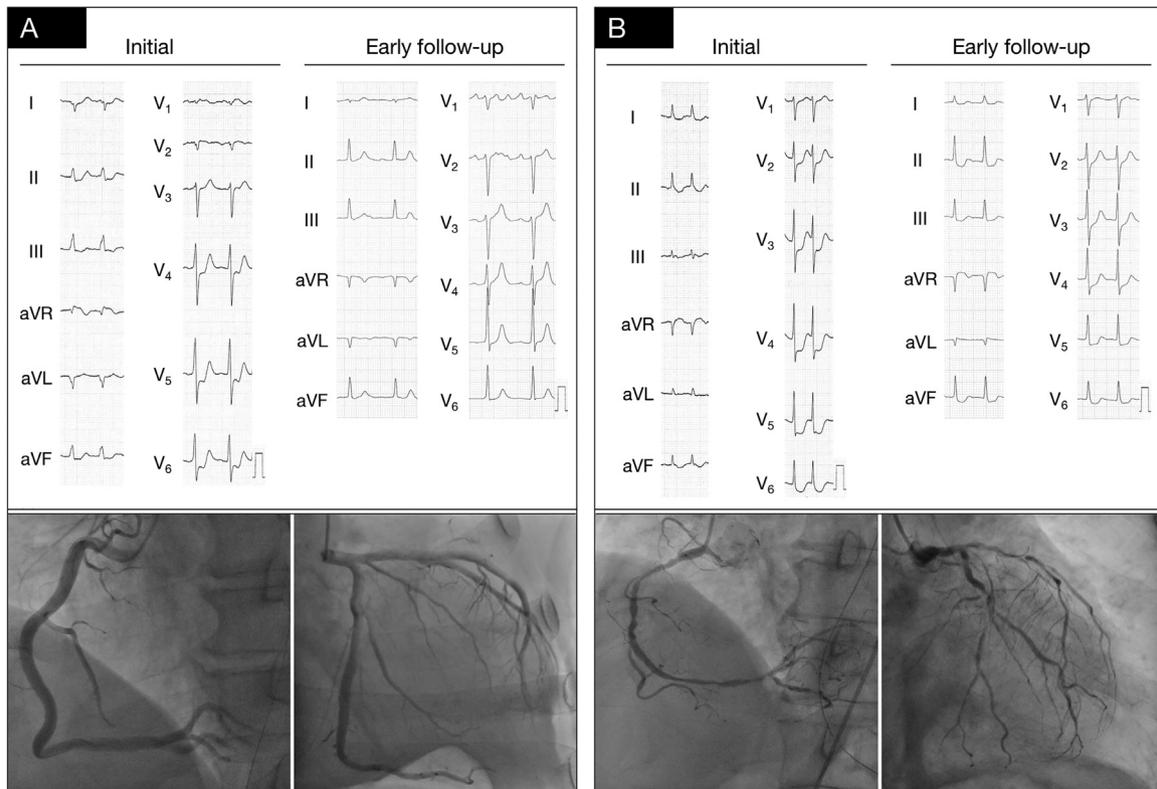


Fig. 2 – Representative electrocardiogram and coronary angiography after return of spontaneous circulation in a patient who did not have coronary artery disease (A) and a patient who had severe coronary artery disease with an acute lesion (B). ST-segment elevation in lead aVR resolved at early follow-up in patient A but persisted in patient B.

with ST-segment elevation <0.5 mm in lead aVR. However, patients with ST-segment elevation ≥ 0.5 mm in lead aVR had a significantly higher prevalence of multivessel coronary artery disease ($p < 0.001$) and chronic total occlusion (50% vs. 7.1%; $p < 0.001$) and a higher SYNTAX score (34 vs. 0.0; $p < 0.001$) than patients with ST-segment elevation <0.5 mm in lead aVR (Table 3).

Indicators of the presence of acute coronary lesions

In univariate logistic regression analysis, age, diabetes mellitus, positive troponin I level at hospital arrival, and ST-segment elevation ≥ 0.5 mm in lead aVR at early follow-up were positively correlated with the presence of acute coronary lesions, whereas

Table 3 – Correlation between coronary artery disease complexity and ST-segment elevation in lead aVR on early follow-up electrocardiogram.

	ST-segment elevation in lead aVR		p-value
	<0.5 mm	≥ 0.5 mm	
Location of acute culprit lesion			0.11
Left main	0/10	0/10	
Left anterior descending artery	8/10	5/10	
Left circumflex artery	1/10	1/10	
Right coronary artery	1/10	4/10	
Number of diseased vessels (diameter stenosis $\geq 50\%$)			<0.001
None	32/56 (57%)	1/18 (5.6%)	
1-vessel disease	6/56 (11%)	2/18 (11%)	
2-vessel disease	6/56 (11%)	3/18 (17%)	
3-vessel disease	12/56 (21%)	12/18 (67%)	
Chronic total occlusion	4/56 (7.1%)	9/18 (50%)	<0.001
SYNTAX score	0.0 (0.0–20.3)	34.0 (18.5–38.0)	<0.001

Data are expressed as number (%) or median (25th to 75th percentiles). SYNTAX, SYnergy between percutaneous coronary intervention with TAXus and cardiac surgery.

ST-segment elevation ≥ 0.5 mm in lead aVR directly after ROSC exhibited no significant association. After adjustments for cardiovascular risk factors, ST-segment elevation ≥ 0.5 mm in lead aVR at early follow-up was the only independent indicator of the presence of acute coronary lesions (odds ratio, 4.41; 95% confidence interval, 1.12–17.4; $p=0.034$) (Table 4). When the ST-segment elevation in lead aVR at early follow-up was ≥ 0.5 mm, the sensitivity, specificity, positive predictive value, and negative predictive value for diagnosing an acute coronary lesion were 50%, 85%, 55%, and 82%, respectively.

Discussion

This study investigated the diagnostic implications of ST-segment elevation in lead aVR after ROSC to identify patients with acute coronary lesions after OHCA without other ST-segment elevations. The results indicated that ST-segment elevations in lead aVR at early follow-up were associated with a higher prevalence of multivessel coronary artery disease and with an increased odds ratio for acute coronary lesions independent of the presence of documented cardiovascular risk factors.

The valuable information provided by lead aVR has not been previously explored in a series of post-cardiac arrest patients. Post-resuscitation ECG is considered less useful for diagnosing acute coronary lesions because of ongoing ischemia, preceding defibrillation, electrolyte abnormalities, and use of intravenous drugs such as epinephrine.¹¹ In fact, ST-segment elevation ≥ 0.5 mm in lead aVR at early follow-up, but not in lead aVR on initial ECG directly after ROSC, was observed to be a significant indicator of the presence of acute coronary lesions in our study. This finding may be explained by hindered subendocardial reperfusion after ROSC due to acute lesions in addition to pre-existing severe coronary artery disease, leading to maintained ischemia and ST-segment elevation in lead aVR.

The optimal timing for early follow-up ECG remains unclear. A previous study showed that, compared with an initially conservative approach, urgent coronary angiography performed earlier, within 2 h from admission, was not associated with better clinical and neurological outcomes in comatose cardiac arrest survivors without ST-segment elevations.¹² The median time of 137 min for early follow-up ECG in the present study suggests that follow-up ECG should be

obtained at approximately 2 h after ROSC in patients without ST-segment elevation myocardial infarction or other clear indicators of acute coronary syndrome.

Coronary angiography revealed significant coronary artery stenoses in 58% of patients and acute culprit lesions in 27% of resuscitated patients without an ST-segment elevation pattern. These findings were consistent with prior study results.^{5,6,13,14}

A previous study verified the diagnostic value of troponin I on admission for acute myocardial infarction after OHCA.¹⁵ However, in our study, troponin I level at hospital arrival could not reliably indicate the presence of an acute coronary lesion after adjustments. This negative result could be most likely elucidated by the exclusion of patients presenting with ST-segment elevation. Hence, thrombotic coronary occlusions were rare and only observed in three patients (4.1%).

In our study on OHCA patients with presumed underlying cardiac causes but without typical ST-segment deviations, an ST-segment elevation in lead aVR at early follow-up had a positive predictive value of 55% and a negative predictive value of 82% for the detection of acute coronary lesions. The poor positive predictive value may be explained by the large variability in cardiopulmonary resuscitation procedures that may be applied after cardiac arrest. However, the high negative predictive value aids in the identification of resuscitated patients who will not benefit from emergency coronary angiography.

The results of the present study offer a potential clinical implication for the evaluation of ST-segment elevation in lead aVR at early follow-up after approximately 2 h rather than directly after ROSC. Furthermore, an absence of ST-segment elevation in lead aVR at early follow-up points toward a low possibility of acute coronary lesions in post-cardiac arrest patients. These patients may benefit from elective coronary angiography instead of emergency coronary angiography after hemodynamic stabilization and thorough assessment of comorbidities and severity of hypoxic encephalopathy. Routine emergency coronary angiography after ROSC may increase the number of adjunctive PCI unnecessarily performed for chronic coronary lesion. Moreover, the use of heparin and dual antiplatelet therapy after stenting may worsen occult bleeding, which often occurs as a result of fall injuries and resuscitative chest compressions after cardiac arrest.

Several limitations of this study should be noted. First, this is a single-centre study with a relatively small study population (i.e., 74

Table 4 – Indicators for identifying patients with acute coronary lesion.

	Unadjusted odds ratio (95% CI)	<i>p</i> -value	Adjusted odds ratio (95% CI) [†]	<i>p</i> -value
Age, 1-year increment	1.05 (1.01–1.10)	0.008		
Male	0.99 (0.23–4.16)	0.98		
Hypertension	1.88 (0.66–5.32)	0.24		
Diabetes mellitus	3.83 (1.19–12.3)	0.024		
Dyslipidemia	2.00 (0.69–5.79)	0.20		
Current smoker	0.46 (0.14–1.58)	0.22		
Shockable initial rhythm	0.82 (0.26–2.54)	0.73		
Positive troponin I level at hospital arrival	3.04 (1.01–9.14)	0.047	1.52 (0.47–4.92)	0.48
ST-segment elevation ≥ 0.5 mm in lead aVR on initial ECG	1.64 (0.57–4.66)	0.36		
ST-segment elevation ≥ 0.5 mm in lead aVR on early follow-up ECG	5.75 (1.81–18.2)	0.003	4.41 (1.12–17.4)	0.034

[†] Adjustments for age, male sex, hypertension, diabetes mellitus, dyslipidemia, and current smoking status. CI, confidence interval; ECG, electrocardiogram.

patients). Biases from unobserved confounding factors possibly remain. This study targeted patients with presumed cardiac causes of arrest only, and nearly one-third of OHCA patients with ROSC were excluded owing to non-cardiac aetiologies. Moreover, the study patients had a high probability of an acute coronary lesion because of the high incidence of ventricular tachycardia or fibrillation and bystander interventions. This might have led to higher sensitivity and specificity. Second, ST segments of patients with left bundle branch block and left ventricular hypertrophy in lead aVR could not be evaluated. Third, the time between ROSC and initial ECG in the hospital may have differed, depending on whether the patient experienced ROSC farther away from the hospital or in the hospital. This suggests that initial ECG was performed at different time points among patients, which may have led to inconsistency in analysis, as ST segments dynamically change during re-flow. However, the maximum time from ROSC to hospital arrival was only 15 min. Therefore, further studies with larger patient populations are required to verify our results.

Conclusions

Persistent ST-segment elevation ≥ 0.5 mm in lead aVR on early follow-up ECG after resuscitation was associated with a high prevalence of severe coronary artery disease with acute lesions in OHCA survivors without other ST-segment elevations. These findings suggest that the analysis of ST-segment elevation in lead aVR may aid in the identification of patients after ROSC who will benefit from further invasive coronary diagnostic procedures.

Conflict of interest

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