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BRIEF REPORT
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Prevalence and prognostic impact of ST-segment elevation in lead aVR among patients with cardiac arrest

Soumya Banna[1†], Christopher Schenck[2†], Noah Kim[3], Tariq Ali[4], Emily J. Gilmore[3], David M. Greer[5], Rachel Beekman[3], and P. Elliott Miller [4*]

[1]Department of Internal Medicine, Yale School of Medicine, New Haven, CT, USA; [2]Department of Medicine, Massachusetts General Hospital, Boston, MA, USA; [3]Department of Neurology, Yale School of Medicine, New Haven, CT, USA; [4]Section of Cardiovascular Medicine, Yale School of Medicine, New Haven, CT 06517, USA; and [5]Department of Neurology, Boston University Chobanian and Avedisian Medical School, Boston Medical Center, Boston, MA, USA

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Aims In acute coronary syndrome, ST-segment elevation in lead aVR (STE-aVR) indicates global myocardial ischaemia, often related to multivessel or severe left main disease, and correlates with increased mortality. The prevalence and prognostic significance of STE-aVR in cardiac arrest (CA) patients is unknown.

Methods and results We identified patients (≥18 years) with CA between 2011 and 2022 who achieved return of spontaneous circulation (ROSC). The first electrocardiogram post-ROSC was assessed for STE-aVR, defined as ≥1 mm ST-segment elevation at the J point, measured by two trained assessors. Multivariable logistic regression was used to analyse the association between STE-aVR and outcomes (in-hospital mortality and poor neurologic outcome), adjusted for patient and arrest characteristics. Including 443 CA patients, the median (interquartile range) age was 61 years (50–72 years), with 60.5% ($n = 268$) male, 65.7% ($n = 291$) presenting with out-of-hospital CA (OHCA), and 29.8% ($n = 132$) with shockable rhythms. ST-segment elevation in lead aVR was observed in 18.3% ($n = 81$) of patients. Those with STE-aVR were more likely to present with OHCA and less likely to have a shockable rhythm (both, $P < 0.05$). ST-segment elevation in lead aVR was associated with higher in-hospital mortality (86.4% vs. 65.8%, $P < 0.001$) and poor neurologic outcomes (90.1% vs. 72.9%, $P = 0.001$). After multivariable adjustment, STE-aVR remained associated with higher in-hospital mortality [odds ratio (OR) 2.23; 95% confidence interval (CI): 1.02–4.84, $P = 0.04$], but not a poor neurologic outcome (OR 2.12; 95% CI: 0.90–4.98, $P = 0.09$).

Conclusion ST-segment elevation in lead aVR was present in one in five CA survivors and was independently associated with higher in-hospital mortality.

Keywords Cardiac arrest • Electrocardiogram

Key points

- ST-segment elevation in lead aVR is a common ECG finding among cardiac arrest survivors following ROSC.
- Those with STE-aVR on the post-ROSC ECG had higher in-hospital mortality.
- Post-ROSC ECG findings, such as STE-aVR, may be a practical tool for risk stratification in CA patients.

* Corresponding author. Tel: +1 203-737-6390, Email: Elliott.miller@yale.edu
† The first two authors contributed equally to the study.

Introduction

Cardiac arrest (CA) is a highly morbid condition, with in-hospital mortality rates exceeding 90% for out-of-hospital CA (OHCA) and 80% for in-hospital CA (IHCA).[1,2] Early risk stratification in CA patients is challenging due to a lack of reliable indicators of poor outcomes after return of spontaneous circulation (ROSC).[3] The post-resuscitative electrocardiogram (ECG) is a cost-effective tool for assessing electrical activity and ongoing ischaemia. Current guidelines recommend a 12-lead ECG after ROSC to assess the need for urgent coronary angiography.[4] While recent trials showed that immediate angiography in CA patients without ST-segment elevation (STE) myocardial infarction does not improve survival,[5,6] the significance of STE, particularly in lead aVR, remains unclear.

Although often overlooked, STE in lead aVR (STE-aVR) suggests global myocardial ischaemia, typically due to multivessel or critical left main disease.[7,8] Though debate exists on whether STE-aVR is a STEMI equivalent, guidelines recommend immediate coronary angiography.[9] In addition, STE-aVR has been found to be useful in distinguishing between myocardial ischaemia and pericarditis.[10] Given the clinical importance of STE-aVR, the lack of available literature in those with CA, and the need for improved early prognostication,[11] we aimed to describe its prevalence in CA patients and its impact on clinical outcomes.

Methods

Data source and study population

We conducted a retrospective analysis of patients enrolled in the Multimodal Outcome CHAracterization in Comatose Cardiac Arrest Patients Registry at Yale New Haven Hospital. We included adults aged ≥18 years admitted between 2011 and 2022 with CA who achieved ROSC and had an ECG. Patients both with IHCA and OHCA were included. This study was approved by the Yale University IRB.

Electrocardiogram analysis

The first 12-lead ECG following ROSC in CA patients was evaluated for STE-aVR, defined as STE at the J point of ≥1 mm, measured manually by two trained assessors (S.B. and C.S.). A third reviewer (P.E.M.) was used in case of discrepancy ($n = 4$, 0.9%). All readers were blinded to the other readers' interpretations.

Outcomes

The primary outcome was in-hospital mortality, and the secondary outcome was poor neurologic outcome at hospital discharge, defined as a Cerebral Performance Category score of 3–5.

Statistical analysis

Baseline characteristics were compared between patients with and without STE-aVR on the first ECG after ROSC. Continuous variables were described with median and interquartile range (IQR) and categorical variables as frequencies and percentages. The Kruskal–Wallis and χ^2 tests were used for continuous and categorical variables, respectively. We performed hierarchical logistic regression: unadjusted (Model 1), demographics (age, sex, race, Model 2), comorbidities (coronary artery disease, heart failure, chronic kidney disease, hypertension, diabetes, Model 3), and CA characteristics (arrest location, initial rhythm, witnessed status, and bystander cardiopulmonary resuscitation) and initial lactate (Model 4). Interobserver variability was assessed using Cohen's kappa statistic. All analyses were performed on STATA 16.0 (Stata Corp, College Station, TX, USA) with statistical significance considered at a two-tailed $P < 0.05$.

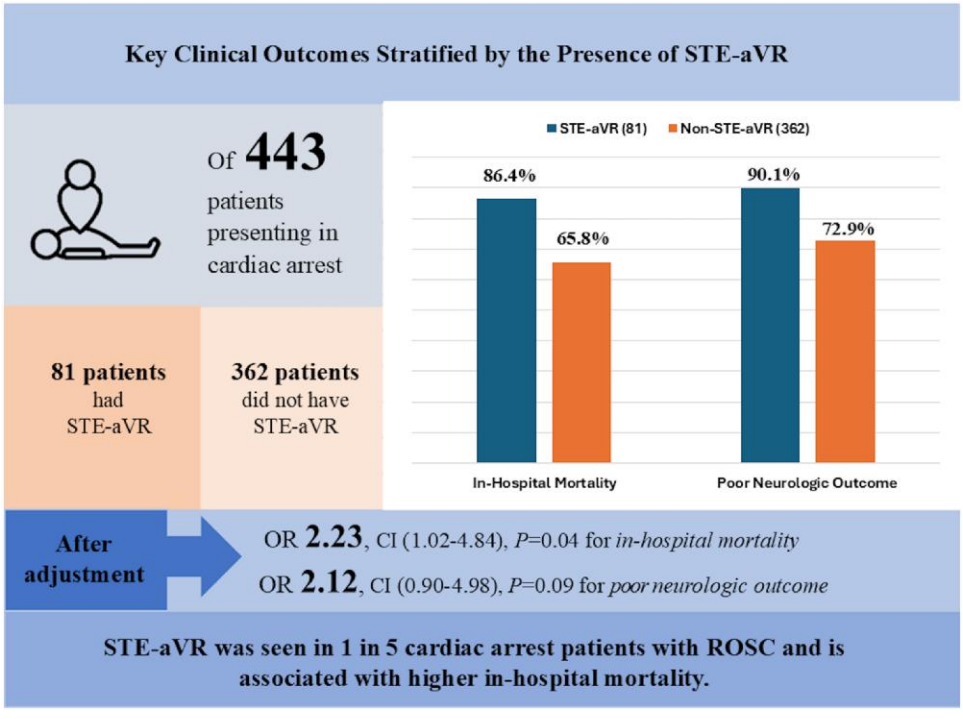

Figure 1 Key clinical outcomes stratified by the presence of ST-segment elevation in lead aVR. STE-aVR, ST-segment elevation in lead aVR; OR, odds ratio; CI, confidence interval; ROSC, return of spontaneous circulation.

Table 1 Patient demographics and clinical characteristics for patients with cardiac arrest

Patient characteristics	Overall ($n = 443$)	STE-aVR ($n = 81$)	Non-STE-aVR ($n = 362$)	*P*-value
Demographics				
Age (years)	61 (50–72)	60 (45–73)	61 (52–72)	0.35
Sex				
Male	268 (60.5%)	45 (55.6%)	223 (61.6%)	0.31
Female	175 (39.5%)	36 (44.4%)	139 (38.4%)	
Race and ethnicity				
White	276 (62.3%)	45 (55.6%)	231 (63.8%)	0.53
Black	106 (23.9%)	22 (27.2%)	84 (23.2%)	
Hispanic	50 (11.3%)	11 (13.6%)	39 (10.8%)	
Other	11 (2.5%)	3 (3.7%)	8 (2.2%)	
Medical comorbidities				
CAD	115 (26.0%)	22 (27.2%)	93 (25.7%)	0.79
Heart failure	79 (17.8%)	12 (14.8%)	67 (18.5%)	0.43
CKD	75 (16.9%)	12 (14.8%)	63 (17.4%)	0.57
Hypertension	279 (63.0%)	49 (60.5%)	230 (63.5%)	0.61
Diabetes	141 (31.8%)	26 (32.1%)	115 (31.8%)	0.95
Procedures				
LHC	49 (11.1%)	11 (13.6%)	38 (10.5%)	0.42
PCI	35 (7.9%)	5 (6.2%)	30 (8.3%)	0.52
Temperature control	258 (58.2%)	50 (61.7%)	208 (57.5%)	0.48
Laboratory data				
Initial lactate, mmol/L	8.2 (5.0–11.4)	10.4 (8.4–13.7)	7.4 (4.4–10.9)	<0.001
Initial pH	7.28 (7.16–7.37)	7.30 (7.14–7.39)	7.28 (7.17–7.37)	0.80
Arrest characteristics				
OHCA	291 (65.7%)	64 (79.0%)	227 (62.7%)	0.005
Initial shockable rhythm	132 (29.8%)	15 (18.5%)	117 (32.3%)	0.01
Witnessed arrest	327 (73.8%)	51 (63.0%)	276 (76.2%)	0.01
Bystander CPR	234 (52.8%)	42 (51.9%)	192 (53.0%)	0.85
PCAC				
1	52 (12.6%)	4 (5.1%)	48 (14.3%)	0.005
2	31 (7.5%)	4 (5.1%)	27 (8.0%)	
3	75 (18.1%)	8 (10.3%)	67 (19.9%)	
4	256 (61.8%)	62 (79.5%)	194 (57.7%)	
Cardiac characteristics				
MAP[a], mmHg	81 (75–89)	81 (75–91)	81 (74–89)	0.84
Peak NEE[a], mg/kg/min	0.68 (1.07)	0.85 (1.4)	0.64 (0.99)	0.11
LVEF, %	51 (35–62)	51 (30–61)	51 (35–62)	0.46

CAD, coronary artery disease; CKD, chronic kidney disease; LHC, left heart catheterization; PCI, percutaneous coronary intervention; OHCA, out-of-hospital cardiac arrest; CPR, cardiopulmonary resuscitation; MAP, mean arterial pressure; NEE, norepinephrine equivalents; LVEF, left ventricular ejection fraction; PCAC, Pittsburgh Cardiac Arrest Category[15] [Level 1: awake; Level 2: coma with mild cardiopulmonary failure (minimal ventilator support, norepinephrine ≤0.1 μg/kg/min); Level 3: coma with severe cardiopulmonary failure (high ventilator support, norepinephrine >0.1 μg/kd/min); Level 4: deep coma (absent pupil and/or corneal responses, no movement)].
[a]Average over the first 24 h.

Results

Patient and arrest characteristics

Of 471 patients, 443 patients (94.1%) had an ECG obtained within 24 h, with a median time to ECG of 52 min (IQR: 30–139 min). Eighty-one patients (18.3%) had STE-aVR on their post-ROSC ECG (*Figure 1*). Interobserver ECG variability was excellent (kappa = 0.97). Baseline characteristics by STE-aVR status are shown in *Table 1*. Demographics and comorbidities were similar between groups (all, $P > 0.05$). Rates of percutaneous coronary intervention and temperature control were also similar (both, $P > 0.05$). ST-segment elevation in lead aVR was more common among OHCA patients (79.0% vs. 62.7%,

Table 2 Associations between ST-segment elevation in lead aVR and clinical outcomes

Model	Odds ratio (95% CI)	*P*-value
In-hospital mortality		
Model 1: unadjusted	3.32 (1.69–6.49)	<0.001
Model 2: +demographics	3.51 (1.78–6.92)	<0.001
Model 3: +comorbidities	3.55 (1.79–7.02)	<0.001
Model 4: +arrest characteristics	2.23 (1.02–4.84)	0.04
Poor neurologic outcome[a]		
Model 1: unadjusted	3.39 (1.57–7.29)	0.002
Model 2: +demographics	3.49 (1.61–7.55)	0.002
Model 3: +comorbidities	3.61 (1.66–7.86)	0.001
Model 4: +arrest characteristics	2.12 (0.90–4.98)	0.09

Model 1: unadjusted; Model 2: adjusted for age, sex, and race and ethnicity; Model 3: additionally adjusted for coronary artery disease, heart failure, chronic kidney disease, hypertension, and diabetes; Model 4: additionally adjusted for arrest location, initial rhythm, witnessed status, bystander cardiopulmonary resuscitation, and initial lactate.
[a]Poor neurologic outcome was defined as Cerebral Performance Category score of 3–5.

$P = 0.005$) and less common in those with initial shockable rhythms (18.5% vs. 32.5%, $P = 0.01$).

Outcomes

Overall, in-hospital mortality was 69.5% ($n = 308$) and significantly higher in patients with STE-aVR (86.4% vs. 65.8%, $P < 0.001$). Withdrawal of life-sustaining therapy (WLST) occurred more frequently among the STE-aVR group (70.4% vs. 55.8%, $P = 0.02$), but a similar proportion of those undergoing WLST died (80.0% vs. 84.0%, $P = 0.43$). After multivariable adjustment, STE-aVR remained significantly associated with higher in-hospital mortality [Model 4: odds ratio (OR) 2.12; 95% confidence interval (CI): 1.02–4.84, $P = 0.04$] (*Table 2*). Poor neurologic outcome was more frequent in STE-aVR patients (90.1% vs. 72.9%, $P = 0.001$), which did not persist after multivariate adjustment (Model 4: OR 2.12; 95% CI: 0.90–4.98, $P = 0.09$).

Discussion

In this retrospective, single-centre analysis of patients with CA who attained ROSC, we found several important findings. First, STE-aVR was relatively common and identified in approximately one in five patients with CA. Second, STE-aVR was independently associated with higher in-hospital mortality. Third, STE-aVR was not associated with poor neurologic outcome after multivariable adjustment. These findings suggest STE-aVR is a relatively common post-ROSC ECG finding that is associated with mortality in patients with CA.

To our knowledge, this is the first study to report the prevalence of STE-aVR in patients with CA and its association with clinical outcomes. A smaller ($n = 74$) study of OHCA patients found that STE-aVR was associated with multivessel disease and acute lesions on coronary angiogram but did not report clinical outcomes.[12] Another study reported that STE in >1 segment was seen in >60% of patients with OHCA and was associated with increased 30-day mortality[13]; however, they did not report which lead had STE. In addition to being larger, our study is additive to previous studies by including the impact of ECG changes on clinical outcomes, notably mortality and neurologic outcomes.

Although the prognostic utility of STE-aVR in CA has not previously been described, it has been linked to poor outcomes in patients with acute coronary syndrome (ACS).[14] A meta-analysis of over 7000 ACS patients reported STE-aVR was associated with higher in-hospital and 90-day mortality.[14] A shared mechanism may involve pump failure exacerbating subendocardial ischaemia, which is reflected reciprocally as STE-aVR. Overall, beyond global myocardial ischaemia and dysfunction, the mechanism for STE-aVR and poorer outcomes in CA is unclear. Regardless of the presence or absence of plaque rupture as the arrest aetiology, STE-aVR may be a marker of extended low-flow time, akin to lactate and associated metabolic disarray, which would impact all organ systems, including the neurologic system. While early mortality following ROSC is related to circulatory collapse, later deaths are often due to WLST due to poor predicted neurological recovery from anoxic brain injury. Our study found a correlation between STE-aVR and unfavourable neurologic outcomes, but after adjusting for arrest characteristics and illness severity, STE-aVR was no longer an independent predictor.

Our results should be interpreted considering several limitations, including our retrospective, single-centre study design, and lack of some important data including coronary anatomy and mode of death. Single-centre institutional practice may have influenced a lower than expected rate of coronary angiography. Second, we did not compare post-ROSC ECGs with baseline ECGs and therefore cannot confirm if certain ECG findings were present pre-arrest. However, even if there was STE-aVR pre-arrest, our findings still provide post-arrest clinical utility. Third, while some interobserver variability in ECG analysis is possible, agreement between reviewers in our study was excellent. Fourth, given the observational nature of CA studies, the impact of WLST leading to a self-fulfilling prophecy is incompletely accounted for.

Conclusions

In conclusion, STE-aVR is present in nearly 20% of CA patients and is independently associated with higher in-hospital mortality. Incorporating STE-aVR into risk stratification models may enhance early prognostication and post-arrest care. Further, longitudinal studies are needed to identify potential therapeutic interventions for patients with STE-aVR.

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Conflict of interest: none declared.

Data availability

The data underlying this article are available in the article.

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