Prognostic value of transient conduction disturbance in out-of-hospital cardiac arrest

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Aim: A retrospective observational study to verify the impact of electrocardiograms (ECGs) following out-of-hospital cardiac arrest (OHCA) on mortality.

Methods: We retrospectively studied 101 OHCA patients who achieved a return of spontaneous circulation (ROSC) and survived for ≥3 h. Among them, 50 patients (66 ± 17 years; 22 male) were evaluated using 12-lead ECGs repeatedly and were included in the final analysis: immediately after ROSC (initial ECG) and after the initial evaluation in the emergency department (second ECG). Transient conduction disturbance (transient CD) was defined as a narrowing in QRS duration from the initial to second ECG of ≥18 ms. Multivariate Cox regression analyses were carried out to predict 90-day mortality following OHCA.

Results: Among 50 OHCA patients, 30 patients survived for 90 days. Thirty patients had initial ventricular fibrillation rhythm. Median emergency medical services response time and low-flow duration were 8 and 21 min, respectively. Multivariate analysis showed that the transient CD and low-flow duration were significant predictors of all-cause mortality (hazard ratio 16.55, 1.06; P = 0.001, 0.022, respectively).

Conclusion: Transient CD is a powerful predictor of 90-day mortality in patients who survived 3 h after ROSC from OHCA.

Key words: Bundle branch block, electrocardiogram, out-of-hospital cardiac arrest, resuscitation, transient conduction disturbance

INTRODUCTION

Patients experiencing out-of-hospital cardiac arrest (OHCA) potentially have a fatal prognosis.1,2 Despite advances in post-cardiac arrest care,3–5 only 24–44% of patients could achieve a return of spontaneous circulation (ROSC), and even a few patients could be discharged alive.6–8 To predict the prognosis in these challenging cases, physicians have repeatedly investigated the association between acute-phase variables and patient prognosis. Several variables, such as initial ventricular rhythm and bystander cardiopulmonary resuscitation (CPR) or the presence of a witness, contribute to achieving preferable outcomes.1,2,5–7 However, accurately predicting the clinical course remains difficult.

Electrocardiograms (ECGs) can be easily evaluated in patients who achieve ROSC following OHCA during the initial phase. Although the presence of bundle branch block had been reported to be associated with high mortality following OHCA, these investigations had been undertaken in selected populations, such as the subgroup of the hypothermia trial.9–13 Furthermore, the majority of cases of bundle branch block following OHCA resolve during the acute phase,9,10 but the prognostic value of these transient electrocardiographic changes has not been explored well.

We aimed to investigate the serial changes in ECGs following OHCA and the association between the presence of transient conduction disturbance (CD) following OHCA and mortality.

METHODS

Patient population

This retrospective observational study enrolled 1012 consecutive OHCA patients who were transferred to Yokohama Minami Kyosai Hospital (Yokohama, Japan) between March 2013 and November 2018.
Among 306 patients who achieved ROSC after OHCA, 145 survived for ≥3 h and underwent an initial evaluation at the emergency room (including prehospital data evaluation, blood gas analysis, and 12-lead ECG). We excluded 95 patients for reasons such as traumatic cardiac arrest, introduction of percutaneous cardiopulmonary support, and could not be evaluated ECGs repeatedly. Finally, 50 patients were evaluated in the present study (Fig. 1).

**Prehospital data**

Prehospital data, including witnessed cardiac arrest, bystander CPR, initial ventricular fibrillation (VF) rhythm, no-flow duration (NFD; from the cardiac arrest to the start of CPR), low-flow duration (LFD; from the start of CPR to ROSC), and emergency medical services response time (EMR; from call receipt to arrival at the patient’s site) were systematically collected at admission according to the Utstein guidelines. A presumed cardiac etiology was defined in accordance with the Utstein guidelines and identified in patients who did not fit in the more readily defined “cardiac arrest of non-cardiac etiology” category. The specific cause of the cardiac arrest (e.g., myocardial infarction, vasospastic angina, or idiopathic VF) and prearrest comorbidities were also assessed. Blood gas analyses were carried out on hospital arrival.

**Electrocardiographic findings**

An ECAPS 12c (Nihon Kohden Co., Tokyo, Japan) 12-lead ECG system was used to record the ECGs after ROSC (initial ECG) and after the initial evaluation in the emergency department (second ECG). Any ECGs obtained before the present cardiac arrest were also evaluated (previous ECG). The ECG variables were automatically calculated and confirmed manually by two expert cardiologists. Electrocardiogram morphologies were divided into four groups: (i) complete right bundle branch block (CRBBB): QRS duration of ≥120 ms, secondary R wave in V1 or V2, and wide slurred S wave in leads I, V5, and V6; (ii) complete left bundle branch block (CLBBB): QRS duration of ≥120 ms, QS or rS complex in V1–V2, and monophasic R wave with no Q waves in leads V6 or I; (iii) intraventricular conduction delay (IVCD): non-specific wide QRS complex and QRS duration of ≥120 ms without obvious CRBBB or CLBBB; (iv) normal morphology: QRS duration of <120 ms. The serial change in morphology was calculated as the difference from the QRS duration of the initial ECG to the second ECG (ΔQRS duration). Transient CD was defined based on receiver operating characteristic (ROC) curve and Youden’s J statistical analyses.

![Study flow diagram](image-url)

**Fig. 1.** Study flow diagram. A total of 1,012 patients who experienced out-of-hospital cardiac arrest were transferred to our hospital between March 2013 and November 2018. Among the 306 patients who achieved return of spontaneous circulation after out-of-hospital cardiac arrest, 145 survived for ≥3 h and underwent an initial evaluation at the emergency room (including prehospital data evaluation, blood gas analysis, and 12-lead electrocardiogram [ECG]). The following patients were excluded: 18 who experienced traumatic cardiopulmonary arrest, 12 who required percutaneous cardiopulmonary support, nine with intracranial bleeding, two with aneurysm rupture, one with a pacing rhythm, one with ECG findings of Brugada syndrome, and one who was younger than 18 years. Among them, 50 patients could be evaluated by repeated ECGs and were included in the final analysis.
### Table 1. Baseline characteristics of patients who achieved return of spontaneous circulation following out-of-hospital cardiac arrest (OHCA), grouped into those who survived for 90 days after OHCA (group S) and those who died within 90 days after OHCA (group D)

|                          | Total (n = 50) | Group S (n = 30) | Group D (n = 20) | P-value |
|--------------------------|---------------|-----------------|-----------------|---------|
| Age (years)              |               |                 |                 |         |
|                          | 66 ± 17       | 59 ± 17         | 76 ± 11         | <0.001* ** |
| Male, n (%)              |               |                 |                 | 1.000   |
| Initial VF, n (%)        |               |                 |                 |         |
|                          | 22 (44)       | 13 (43)         | 9 (45)          |         |
| EMS response time (min)  | 8 (7, 11)     | 8 (7, 10)       | 8 (7, 13)       | 0.236   |
| NFD (min)                | 2 (0.9)       | 2 (0.6)         | 10 (0.13)       | 0.031*  |
| LFD (min)                | 21 (11, 31)   | 15 (7, 24)      | 30 (25, 43)     | <0.001* ** |
| Witness, n (%)           |               |                 |                 | 0.171   |
| Bystander, n (%)         | 32 (64)       | 23 (77)         | 9 (45)          | 0.035*  |
| Presumed cardiac etiology, n (%) | 39 (78)       | 26 (87)         | 13 (65)         | 0.090   |
| AMI, n (%)               |               |                 |                 |         |
|                          | 12 (24)       | 11 (35)         | 1 (5)           | 0.006*  |
| VSA, n (%)               | 5 (10)        | 4 (13)          | 1 (5)           | 0.299   |
| Idiopathic VF, n (%)     | 7 (14)        | 6 (19)          | 1 (5)           | 0.403   |
| Other, n (%)             | 10 (20)       | 5 (16)          | 5 (26)          | 0.232   |
| Unknown, n (%)           | 5 (10)        | 0 (0)           | 5 (26)          | 0.001*  |
| Prearrest comorbidities, n (%) |             |                 |                 |         |
| DM                       | 9 (18)        | 3 (10)          | 6 (30)          | 0.130   |
| HTN                      | 16 (32)       | 10 (33)         | 6 (30)          | 1.000   |
| DLP                      | 9 (18)        | 6 (20)          | 3 (15)          | 0.724   |
| CHF                      | 10 (20)       | 5 (17)          | 5 (25)          | 0.494   |
| CKD                      | 6 (12)        | 1 (3)           | 5 (25)          | 0.032   |
| HD                       | 3 (6)         | 1 (3)           | 2 (10)          | 0.556   |
| CAD                      | 7 (14)        | 5 (17)          | 2 (10)          | 0.687   |
| MI                       | 5 (10)        | 4 (13)          | 1 (5)           | 0.636   |
| Af                       | 5 (10)        | 3 (10)          | 2 (10)          | 1.000   |
| CPA                      | 1 (2)         | 0 (0)           | 1 (5)           | 0.400   |
| CK (IU/L)                | 131 (105, 220)| 162 (118, 350) | 104 (96, 124)  | 0.185   |
| CKMB (IU/L)              | 39 (25, 52)  | 36 (24, 57)    | 42 (26, 47)    | 0.378   |
| Creatinine (mg/dL)       | 1.19 (0.99, 1.44) | 1.07 (0.96, 1.20) | 1.39 (1.24, 1.96) | 0.053 |
| Lactate (mmol/L)         | 10.3 ± 3.6    | 8.5 ± 2.9      | 12.8 ± 2.9     | <0.001* ** |
| Glucose (g/dL)           | 292 ± 216     | 252 ± 135      | 356 ± 297      | 0.104   |
| EF (%)                   | 48 ± 13       | 48 ± 14        | 47 ± 12        | 0.883   |
| CAG, n (%)               | 29 (58)       | 26 (87)        | 3 (15)         | <0.001* ** |
| PCI, n (%)               | 13 (26)       | 12 (40)        | 1 (5)          | 0.008* ** |
| TTM, n (%)               | 19 (38)       | 15 (50)        | 4 (20)         | 0.041*  |
| ΔQRS, n (%)              | 23 ± 25       | 10 ± 15        | 42 ± 26        | <0.001* ** |
| Transient CD, n (%)      | 24 (48)       | 6 (20)         | 18 (90)        | <0.001* ** |

Parametric variables are shown as mean ± standard deviation or median (interquartile range), and non-parametric variables as median (25%, 75% value). ΔQRS, difference in QRS duration between initial and second electrocardiogram; Af, atrial fibrillation; AMI, acute myocardial infarction; Bystander, cardiopulmonary resuscitation by bystander; CAD, coronary artery disease; CAG, coronary angiography; CD, conduction disturbance; CHF, congestive heart failure; CK, creatine kinase; CKD, chronic kidney disease; CPA, history of cardiopulmonary arrest; DLP, dyslipidemia; DM, diabetes mellitus; EF, ejection fraction; EMS response time, duration from call receipt to emergency medical services arrival at the patient’s site; HD, hemodialysis; HTN, hypertension; Idiopathic VF, patients with documented ventricular fibrillation without any specific etiology; Initial VF, initial ventricular fibrillation rhythm; LFD, low-flow duration defined as the duration from start of cardiopulmonary resuscitation to return of spontaneous circulation; MI, myocardial infarction; NFD, no-flow duration defined as the duration from the cardiac arrest to the start of cardiopulmonary resuscitation; Other, including congestive heart failure, Takotsubo syndrome, hypertrophic myopathy, and aortic valve stenosis; PCI, percutaneous coronary intervention during hospitalization; Presumed cardiac etiology, defined by Utstein guidelines; TTM, targeted temperature management after return of spontaneous circulation; VSA, vasospastic angina; Witness, witnessed cardiac arrest.

*P < 0.05 considered as significant.

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Percutaneous coronary intervention and targeted temperature management

Emergent percutaneous coronary intervention (PCI) was considered if the cardiac arrest seemed to be caused by a myocardial infarction according to the initial evaluation with the consensus of the cardiac team including the physician who resuscitated the patient.\(^\text{17}\) Targeted temperature management was considered if the patient did not have a contraindication, such as infection or bleeding. Targeted temperature management was initiated at 34°C for 24 h using a combination of ice packs, cooled fluids, and active surface cooling blankets, followed by rewarming to 36°C for 24 h.\(^\text{5}\)

Data collection, follow-up, and outcomes

Clinical characteristics and outcomes were collected from hospital charts by independent researchers.

The all-cause death was recorded over the observation period. The primary end-point of the present study was 90-day mortality. Predictors for 90-day mortality were analyzed by using the Cox proportional hazards regression models.

Statistical analysis

We divided the overall population into two groups: S group, those surviving for 90 days \((n = 30)\), and D group, those dying within 90 days \((n = 20)\) (Figure). The baseline characteristics between these groups were examined. We compared the variables of the previous, initial, and second ECGs in the S and D groups.

Univariate and multivariate Cox regression analyses were undertaken to predict the 90-day mortality after OHCA. Hazard ratios for 90-day mortality with corresponding 95% confidence intervals were reported. The multivariate model included transient CD, along with age, initial VF rhythm, EMS response time, and LFD; traditional predictor for mortality among patients with OHCA.\(^\text{18–20}\)

Parametric continuous variables were shown as means ± standard deviations and non-parametric variables as medians (25%–75%). Parametric variables were analyzed using a two-tailed \(t\)-test and non-parametric variables using the Mann–Whitney test. \(P\)-values of <0.05 were considered statistically significant.

All statistical analyses were undertaken with EZR (Saitama Medical Center, Jichi Medical University, Saitama, Japan),\(^\text{21}\) which is a graphical user interface for R (The R Foundation for Statistical Computing, Vienna, Austria).\(^\text{22}\)

RESULTS

The baseline characteristics of the 50 patients (mean age, 66 ± 17 years; 22 men) are summarized in Table 1. Thirty patients (60%) survived for 90 days. Transient CD was defined as a \(\Delta\)QRS duration of ≥18 ms based on the cut-off value of ROC curve analysis. Several clinical characteristics, including age, initial VF rhythm, NFD, LFD, bystander CPR, and lactate level \((P < 0.05)\), were significantly different between the S and D groups. Acute myocardial infarction occurred more frequently in the S group than in the D group (35% vs. 5%, \(P = 0.006)\). There was no intergroup difference in the pre-arrest comorbidities (e.g., diabetes mellitus, coronary artery disease, and atrial fibrillation) except chronic kidney disease.

Percutaneous coronary intervention (40% vs. 5%, \(P = 0.008)\) and targeted temperature management (TTM) (50% vs. 20%, \(P = 0.041)\) were undertaken more frequently in the S group than in the D group.

The variables of the initial and second ECGs were evaluated (Table 2). The median duration from ROSC to ECG evaluation was 15 min (initial ECG) and 178 min (second ECG), respectively. The previous ECG was evaluated 139 days (median) before OHCA (Fig. S1). The ECGs were divided according to the presence of 90-day survival (group S and group D). Comparing groups S and D, several variables of initial ECG had significant difference (QRS duration, QRS duration ≥120 ms, and QRS morphology). Among variables of the second ECG, QRS morphology and QRS duration ≥120 ms had significant difference between group S and group D, but QRS duration had no significant difference between groups (Table 2).

In the multivariate analysis including transient CD, age, initial VF, EMS response time, and LFD, transient CD and LFD were found to be the significant and independent predictors of all-cause mortality within 90 days (hazard ratio = 16.55, 1.06; \(P = 0.001, 0.022\), respectively; Table 3).

Receiver operating characteristic curve analysis showed that a cut-off value of 18 ms for the \(\Delta\)QRS duration had the highest accuracy for predicting 90-day mortality (specificity, 80.0%; sensitivity, 90.0%; area under the ROC curve, 0.886; Table 4).

DISCUSSION

To the best of our knowledge, this is the first study to evaluate the serial changes in ECGs after OHCA and verify the prognostic value of transient CD. The main findings of the present study are as follows: ECG findings drastically change in the first 3 h after OHCA, and transient CD is...
a significant predictor of 90-day mortality among patients who experience OHCA, the accuracy of which is comparable to that of LFD.23,24

Pathophysiology of transient CD
The present study showed that transient CD could predict 90-day mortality after OHCA (Table 3). The pathophysiology of transient CD can be explained by the changes in QRS morphology during the acute phase after OHCA. Global myocardial ischemia, caused by hypoperfusion associated with cardiac arrest, causes changes in QRS morphology in patients who experience OHCA.9,10 We also detected an association between the presence of transient CD and hypoperfusion duration measured by LFD (Fig. S2). Furthermore, Table 2 reveals that the acute phase after OHCA frequently included CRBBB and IVCD (initial ECG), and most of these morphologies normalized in the second ECG.

Table 2. Electrocardiogram (ECG) variables evaluated in patients with return of spontaneous circulation following out-of-hospital cardiac arrest (OHCA), grouped into those who survived for 90 days after OHCA (group S) and those who died within 90 days after OHCA (group D)

| Initial ECG | Group S (n = 30) | Group D (n = 20) | P-value | Second ECG | Group S (n = 30) | Group D (n = 20) | P-value |
|-------------|------------------|------------------|---------|------------|------------------|------------------|---------|
| Heart rate (b.p.m.) | 110 [91,119] | 108 [77,119] | 0.267 | 87 [76,101] | 94 [79,106] | 0.390 |
| Atrial fibrillation, n (%) | 9 (30) | 10 (50) | 0.235 | 3 (10) | 5 (25) | 0.240 |
| QRS duration (ms) | 111 ± 18 | 154 ± 37 | <0.001* | 101 ± 16 | 112 ± 29 | 0.099 |
| QRS ≥ 120 ms, n (%) | 8 (27) | 17 (85) | <0.001* | 3 (10) | 9 (45) | 0.007* |
| QRS morphology, normal/CRBBB/CLBBB/IVCD | 22 (73)/3 (10)/1 (3)/4 (13) | 10 (50)/11 (55)/0/7 (35) | 0.001* | 27 (90)/3 (10)/0/5 (15) | 11 (55)/5 (25)/1 (5) | 0.010* |
| QTc (ms) | 442 ± 52 | 465 ± 40 | 0.102 | 425 ± 29 | 439 ± 42 | 0.180 |
| ST elevation, n (%) | 9 (30) | 3 (15) | 0.323 | 6 (20) | 1 (5) | 0.219 |
| T wave inversion, n (%) | 20 (67) | 17 (85) | 0.197 | 11 (37) | 12 (60) | 0.149 |
| J wave, n (%) | 5 (17) | 3 (15) | 1.000 | 6 (20) | 6 (30) | 0.506 |

Electrocardiograms obtained before the cardiopulmonary arrest were also evaluated as previous ECG. CRBBB, complete left bundle branch block; CLBBB, complete right bundle branch block; IVCD, unspecific interventricular conduction delay; J wave, presence of end-QRS notching/slurring ≥1 mV in two or more continuous leads (II IIIaVf or IaVlV5,V6); QTc, corrected QT interval; ST elevation, presence in two or more contiguous ECG leads with an amplitude ≥1 mV; T wave inversion, presence of T wave inversion in two or more contiguous ECG leads. *P < 0.05 considered as significant.

Table 3. Multivariate analysis of predictive factors of 90-day mortality among patients who achieved return of spontaneous circulation following out-of-hospital cardiac arrest

| Univariate analysis | Multivariate analysis |
|---------------------|-----------------------|
| Variable            | HR CI P-value         | HR CI P-value         |
| Age                 | 1.06 1.03–1.10 0.001* | 1.04 0.97–1.10 0.251 |
| VF                  | 0.20 0.08–0.53 0.001* | 0.44 0.12–1.56 0.203 |
| EMS response time   | 1.12 0.95–1.33 0.179  | 1.06 0.90–1.24 0.505  |
| LFD                 | 1.05 1.02–1.08 <0.001* | 1.06 1.01–1.10 0.022* |
| Transient CD        | 17.18 3.93–74.99 <0.001* | 16.55 2.95–92.80 0.001* |

CD, conduction disturbance; CI, confidence interval; EMS, emergency medical services; HR, hazard ratio; LFD, low-flow duration; VF, ventricular fibrillation. *P < 0.05 was considered as significant.
Therefore, the progression of CRBBB and IVCD could contribute to the presence of transient CD. In contrast, CLBBB rarely progressed in our population (Table 2). We speculate that CLBBB should occur at the presence of a localized injury of the left bundle branch, such as myocardial infarction.\(^{13,16}\) Therefore, the presence of CLBBB might indicate a limited injury of the myocardium rather than the severity of hypoperfusion associated with the cardiac arrest itself. Our study cohort included a relatively small number of patients with myocardial infarction compared with previous reports,\(^{9,13}\) which could contribute to the low presence of CLBBB in the present study.

On the other hand, the present study includes patients who had been documented bundle branch block before the event; however, the presence of persistent bundle branch block is not associated with the burden of cardiac arrest itself. We then evaluated the ECGs before OHCA (previous ECG), which has not been explored in previous studies.\(^{9,13}\) Although we could obtain data in a relatively small number of cases (\(n = 15\)), the rate of a bundle branch block in previous ECG was almost the same as in healthy subjects,\(^{25,26}\) indicating that the morphologic changes in the initial ECG were largely affected by the cardiac arrest itself (Table 2). Furthermore, as the prevalence of bundle branch block is comparable in previous ECGs and second ECGs, the “transient CD” could rule out the previous CD (Table 2, Fig. S1). Therefore, transient CD could predict mortality following OHCA more precisely compared with the sole morphology following OHCA, such as bundle branch block of initial ECG. Particularly, among patients for whom previous, initial, and second ECGs were available, transient CD could predict survival (except one patient with PCI); conversely, morphology at one time point (either initial or second) could not predict survival so accurately (Fig. S3).

### Definition of transient CD

In the present study, we defined transient CD as a \(\Delta QRS\) duration of \(\geq 18\) ms. The \(\Delta QRS\) duration might approximate the prolongation of QRS duration in the setting of cardiac arrest; the QRS prolongation between previous ECG and initial ECG was close to the \(\Delta QRS\) duration in the present study (Table 2, Fig. S1). Previously, Attin et al.\(^{27}\) reported that the QRS prolongation just before cardiac arrest was associated with poor outcomes. The definition of QRS prolongation was 20 ms in that report. It was also repeatedly indicated in other studies that prolongation of QRS duration during cardiac arrest might be associated with poor outcomes.\(^{28,29}\) Considering these previous reports, we presumed the definition of the transient CD (\(\Delta QRS\) duration of \(\geq 18\) ms) could be close to the preferable cut-off, then the definition might be reasonable.

### Repolarization abnormality from ECGs after OHCA

We detected fewer changes in repolarization abnormalities than in depolarization abnormalities among the patients who experienced OHCA. The changes in the corrected QT interval could have largely been explained by changes in the QRS duration (Table 2), indicating the effect of the depolarization phase. T wave inversion tended to disappear in the initial 3 h (Table 2); however, the presence of a bundle branch block could affect the presence of the T wave inversion. The presence of J waves could not explain the impact of the repolarization phase in the ECG after OHCA. Therefore, we emphasized that the serial ECG changes after OHCA were largely explained by a depolarization, not repolarization, abnormality.
Association between clinical management and ECG changes

In this study, clinical management, including TTM and PCI, influenced the ECG changes. Although the patients who received TTM tended to have a narrow QRS duration and less commonly had transient CD, the association between the QRS duration or transient CD and 28-day mortality is still relevant despite the TTM status (Fig. S4). The presence of PCI was also associated with ECG changes and mortality. Furthermore, the Cox regression analysis including TTM and PCI indicated that transient CD is still a significant predictor of 90-day mortality. Therefore, we emphasize that ECG changes, especially transient CD, can predict mortality after OHCA regardless of the use of these consecutive procedures after OHCA.

Generalizability

The present study indicated that transient CD might be a predictor of mortality in patients following OHCA. It is a strength of transient CD that the predictor could be easily evaluated in every patient who achieved ROSC following OHCA during the initial phase. Although the present study could not evaluate the neurological outcome, the early prediction of mortality is crucial to determine the optimal course of care.\(^\text{19}\) As the present study was retrospective and observational and had few patients, further study is needed to establish the efficacy of transient CD.

Limitations

This study was limited by its retrospective design, single-center location, and relatively small number of patients. As this was a retrospective study, the baseline characteristics differed widely between the S and D groups. However, several ECG parameters were significantly associated with a poor prognosis among the patients who experienced OHCA in the multivariate Cox regression analyses.

The change in ECG findings during the acute phase after OHCA, and transient CD in particular, is a powerful predictor of 90-day mortality among patients who experience OHCA.

As the prediction of mortality was substantially different from the prediction of neurological outcomes among patients following OHCA, we could not discuss the neurological outcomes in the present study.

CONCLUSION

THE CHANGE IN ECG findings in the acute phase following OHCA, and transient CD in particular, is a powerful predictor for 90-day mortality in patients who survived 3 h after ROSC from OHCA.

DISCLOSURE

Approval of the research protocol: The protocol for this research project has been approved by a suitably constituted Ethics Committee of the institution and it conforms to the provisions of the Declaration of Helsinki.

Informed consent: Informed consent was obtained from the subjects or guardians.

Registry and the registration no. of the study/trial: N/A.

Animal studies: N/A.

Conflict of interest: None.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher’s web-site:

Fig. S1. Electrocardiogram (ECG) variables before out-of-hospital cardiac arrest (OHCA). The ECGs were evaluated according to 90-day survival following OHCA (group S versus group D).

Fig. S2. Association between low-flow duration (LFD) and the presence of transient conduction disturbance (CD) in patients with out-of-hospital cardiac arrest. Patients with transient CD had higher LFD compared with patients without (P = 0.028).

Fig. S3. Patients with out-of-hospital cardiac arrest (OHCA) who underwent previous, initial, and second electrocardiogram (ECG). All patients who died within 90 days following OHCA had transient conduction disturbance (CD).

Fig. S4. Comparison of electrocardiographic variables (ΔQRS duration and transient conduction disturbance [CD]) and clinical management (targeted temperature management and percutaneous coronary intervention) in patients with out-of-hospital cardiac arrest.