Significance of electrocardiogram recording in high intercostal spaces in patients with early repolarization syndrome

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Aims
Published reports regarding inferolateral early repolarization (ER) syndrome (ERS) before 2013 possibly included patients with Brugada-pattern electrocardiogram (BrP-ECG) recorded only in the high intercostal spaces (HICS). We investigated the significance of HICS ECG recording in ERS patients.

Methods and results
Fifty-six patients showing inferolateral ER in the standard ECG and spontaneous ventricular fibrillation (VF) not linked to structural heart disease underwent drug provocation tests by sodium channel blockade with right precordial ECG (V1–V3) recording in the 2nd–4th intercostal spaces. The prevalence and long-term outcome of ERS patients with and without BrP-ECG in HICS were investigated. After 18 patients showing type 1 BrP-ECG in the standard ECG were excluded, 38 patients (34 males, mean age; 40.4 ± 13.6 years) were classified into four groups [group A (n = 6; 16%): patients with ER and type 1 BrP-ECG only in HICS, group B (n = 5; 13%): ERS with non-type 1 BrP-ECG only in HICS, group C (n = 8; 21%): ERS with non-type 1 BrP-ECG in the standard ECG, and group D (n = 19; 50%): ERS only, spontaneously or after drug provocation test]. During follow-up of 110.0 ± 55.4 months, the rate of VF recurrence including electrical storm was significantly higher in groups A (4/6: 67%), B (4/5: 80%), and C (4/8: 50%) compared with D (2/19: 11%) (A, B, and C vs. D, P < 0.05).

Conclusions
Approximately 30% of the patients with ERS who had been diagnosed with the previous criteria showed BrP-ECG only in HICS. Ventricular fibrillation mostly recurred in patients showing BrP-ECG in any precordial lead including HICS; these comprised 50% of the ERS cohort.

Keywords
Early repolarization • Ventricular fibrillation • Brugada syndrome • Electrocardiogram • High intercostal recording

Clinical perspective
This study shows evidence that ventricular fibrillation mostly recurs in patients with early repolarization syndrome (ERS) showing Brugada-pattern electrocardiogram (ECG) in any of the right precordial leads including high intercostal spaces (HICS), and that ~30% of them had Brugada-pattern ECG (BrP-ECG) only in the HICS. These results indicate the importance of a systematic search for BrP-ECG with high intercostal ECG recording for the risk stratification of patients with ERS.

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High intercostal ECG recordings in ERS

Introduction

Early repolarization syndrome (ERS) is diagnosed in patients with inferolateral early repolarization (ER) on the standard 12-lead electrocardiogram (ECG), who had been resuscitated from idiopathic ventricular fibrillation (VF) in the absence of other causes of cardiac arrest such as Brugada syndrome (BrS).1 It has been proposed that patients with ERS should not have type 1 Brugada-pattern ECG (BrP-ECG) in the right precordial leads on the standard ECG.1 So far, an additional ECG recording has not been required for the diagnosis of ERS. Even in the expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes, which was published in 2013, there is no mention with regard to recording sites in ERS.2 However, in patients with BrS, the new consensus statement recommends that ECG recordings be taken in the higher intercostal spaces, because it has been recognized since 2005 that the high intercostal recordings showed better sensitivity and specificity in the ECG diagnosis of BrS.3–6

Therefore, there is a possibility that patients with ERS in previous reports might have had a type 1 or non-type 1 BrP-ECG only in the high intercostal spaces (HICS). The purpose of this study was to investigate the prevalence of Brugada ECG pattern recorded in the HICS in patients with ERS and the prognosis of patients with and without BrP-ECG.

Methods

Study population

The study population consisted of 56 consecutive patients with inferolateral ER and spontaneous VF who were admitted to National Cerebral and Cardiovascular Center, Suita, Japan, between 1996 and 2014 (52 men, mean age: 39.9 ± 13.0 years). None of the patients had structural heart disease, including arrhythmogenic right ventricular cardiomyopathy, which was confirmed by noninvasive studies (physical examination, 12-lead ECG, 87-lead body surface ECG, exercise stress test, signal-averaged electrocardiography, and cardiac magnetic resonance imaging or computed tomography), and invasive studies consisting of coronary angiography including ergonovine/acetylcholine injection and right or left ventricular cineangiography. Patients with coronary artery spasm, long QT syndrome, short QT syndrome, catecholaminergic polymorphic ventricular tachycardia, commotio cordis, drug-induced VF, and hypothermia were excluded. This study was approved by the Institutional Research Board of National Cerebral and Cardiovascular Center.

Classification of each group

Of the total of 56 patients, 18 patients showing type 1 BrP-ECG in the standard ECG spontaneously or after drug provocation tests by a sodium channel blocker were excluded, and 38 patients with inferolateral ER and a prior VF (34 males, mean age: 40.4 ± 13.6 years) were classified into four groups (A–D) based on ST-T morphology in the right precordial leads with the results of the drug provocation tests by a sodium channel blocking agent in at least one right precordial lead (V1 and V2), which was placed in a standard or a superior position (up to the second intercostal space).2 Type 1 ECG was defined as a coved-type J-point or ST elevation ≥2 mm followed by a negative T-wave.7

Non-type 1 BrP-ECG was defined as type 2 BrP-ECG, type 3 BrP-ECG or upward/downward notching or downward slurring with an amplitude ≥1 mm at the end of QRS to early ST segment in any of the anterior leads (V1, V2, and V3) in the baseline standard or high costal (second and third) ECG recordings or in those ECGs after drug provocation tests.9 The upward/downward notch in the anterior leads should have appeared between the late QRS and early ST period in the same timeframe as J-waves in other leads in the same 12-lead ECG.

Drug provocation test

Drug provocation tests were conducted with pilsicainide (up to 1 mg/kg body weight injected at a rate of 5–10 mg/min), disopyramide (1.5 mg/kg, 10 mg/min), or flecainide (2 mg/kg, 10 mg/min) during standard and high costal (second and third) ECG recordings. All ECGs were recorded at 25 mm/s and 10 mm/mV. First, the ECG recordings were independently analyzed by two cardiologists (T.K. and S.K.), and consensus was reached about the diagnosis. A third trained cardiologist (K.K.) independently evaluated all of the ECGs with no knowledge of the other observers’ judgment or the clinical information to test for inter-observer variability, and consensus was established.

Clinical data, electrocardiogram, and electrophysiological testing

Clinical data including age at the first episode of VF, sex, family history of sudden cardiac death at <45 years of age, patients’ activity at VF, prognosis, and drug therapy were collected on all patients. We defined the patients’ state at VF as sleep when VF occurred in a state of sleeping, as near sleep when VF occurred in a resting state just after waking,10 and as arousal at rest when VF occurred at rest in an awake state without active body movement. During follow-up, patients were considered to have an arrhythmic event if VF was documented by implantable cardioverter-defibrillator (ICD) interrogation. An electrical storm was defined as ≥3 VF episodes within 24 h. The beginning of the follow-up period was at the time of the first VF event. In patients with recurrent arrhythmias, the choice of antiarrhythmic drugs was decided by the patient’s physician.
Electrophysiological study (EPS) was conducted in 19 patients as previously described. Genetic testing for mutations in the SCN5A gene was performed in 22 patients (A: 3, B: 3, C: 4, D: 12), as previously described.

Clinical profiles, electrocardiographic characteristics, and VF recurrences during 110.0 ± 55.4 months of follow-up were compared among the four groups.

Statistical analysis
Data were analyzed with JMP10 software (SAS Institute Inc., Cary, NC, USA). Numeric values are presented as mean ± standard deviation. The \( \chi^2 \) test, Student’s t-test, or one-way analysis of variance was performed as appropriate to test for statistically significant differences. Survival curves were constructed by the Kaplan–Meier method and compared using the log-rank test. \( P \)-value of <0.05 was considered statistically significant.

Results
Electrocardiogram findings in groups A, B, C, and D
Figure 1 shows typical ECGs at baseline and after the drug provocation test, respectively, in patients in each group. Assignment to groups was performed according to ECG findings at screening: 6 patients (16%) were assigned to group A (Figure 1A), 5 patients (13%) to group B (Figure 1B), 8 patients (21%) to group C (Figure 1C), and 19 patients (50%) to group D (Figure 1D). Therefore, 16% of the

**Figure 1** (A) At baseline, electrocardiograms of a 26-year-old male exhibited J-waves (arrows) followed by near horizontal ST segments in leads II, III, aVF, and V6 and ascending ST segments in V4 and V5. There was no sign of coved or saddleback ST elevation in all chest leads. After injection of 50 mg pilsicainide, type 1 ST elevation (broad arrow) was noted in V2 only in the second intercostal space. He experienced VF recurrence 4 years after implantable cardioverter-defibrillator implantation. (B) At baseline, electrocardiograms of a 42-year-old male exhibited J-waves (arrows) followed by ascending ST segments in leads I, aVL, V4, and V5 in the standard (4th) recording and in lead V2 in the 2nd intercostal recording. After pilsicainide injection, all J-waves in limb lead disappeared with appearance of s-waves and an R-wave in lead aVR. Saddleback ST elevation with slightly augmented J-waves (broad arrows) was also noted in V2 in the high (2nd and 3rd) intercostal spaces. He experienced an electrical storm 4 years after ICD implantation. (C) At baseline, electrocardiograms of a 27-year-old male exhibited J-waves followed by ascending ST segments in leads I, aVL, V4, and V5 in the standard (4th) recording and in lead V2 in the 2nd intercostal recording. After pilsicainide injection, saddleback ST elevation with slightly augmented J-waves (broad arrows) was also noted in lead V2 in the standard recording and in lead V3 in the high intercostal spaces. (D) At baseline, electrocardiograms of 63-year-old male showed J-waves in leads II, III, aVF, V5 and V6 (arrows). After pilsicainide injection, they disappeared or attenuated with appearance of s-waves. Electrocardiograms in leads V1 – V3 during standard and high costal recordings remained normal even after pilsicainide injection.
patients who had been diagnosed with ERS under the previous criteria were actually BrS patients with inferolateral ER. Nineteen patients (50%) had BrP-ECG in any of the right precordial leads and 11 patients (29%) had type 1 or non-type 1 BrP-ECG only in the HICS.

In group A, type 1 BrP-ECG was observed in the high intercostal position spontaneously \((n = 3)\) or only after the drug provocation test \((n = 3)\). In group B, non-type 1 BrP-ECG was observed before \((n = 4)\) or after \((n = 1)\) the drug provocation test only in the HICS.
Clinical profiles in each group

Clinical characteristics of the patients in each group are shown in Table 1. Ventricular fibrillation was observed during sleep or near sleep in three patients (50%) in group A, five patients (100%) in group B, and six patients (75%) in group C; in contrast, only two patients (11%) in group D had VF during sleep or near sleep (B, and C vs. D, \( P < 0.05 \)). Most patients in group D had VF in an awake state. The VF inducibility among the four groups was similar. Mutations of SCN5A were identified in one patient in group C, but in none of the patients in the other groups.

Clinical outcome

Mean follow-up period of group A, B, C, and D was 120 ± 51, 109 ± 61, 106 ± 67, and 108 ± 54 months, respectively. Thirty-seven of the 38 patients received an ICD. One patient in group D was followed without ICD implantation. No patients died during the follow-up period.

VF recurrence rates including electrical storm were significantly higher in group A (4/6:67%), group B (4/5:80%), and group C (4/8:50%), compared with group D (2/19:11%) (A, B, and C vs. D, \( P < 0.05 \)). Kaplan–Meier curves of the four groups are illustrated in Figure 2. Patients in group A, B, and C exhibited significantly higher rates of arrhythmic events than those in group D (log rank, \( P = 0.0019 \)). Type 1 and non-type 1 BrP-ECG were observed only in the HICS in B (type 1:4, non-type 1:4) of the 14 patients with VF recurrence. The incidence of BrP-ECG in any of the right precordial leads was significantly higher in patients with VF recurrence than in those without (12/14; 86% vs. 7/24; 29%, \( P = 0.0019 \)) and the presence of BrP-ECG showed 86% sensitivity, 71% specificity, and 63% positive predictive value to identify VF recurrence in patients with ERS. Single or combination drug therapy consisting of isoproterenol, quinidine, cilostazol, and bepridil was effective in five patients with a VF recurrence in group A, B, and C.

Discussion

Main findings

This is the first study in which high intercostal ECGs of all ERS patients were evaluated on a long-term basis. Results showed thatVF mostly recurred in patients showing BrP-ECG in any right precordial lead including HICS, who comprised 50% of the cohort diagnosed with ERS under the previous criteria, in contrast to favorable prognosis in the remaining 50% of ERS patients without BrP-ECG in both the standard and HICS. A systematic search of the BrP-ECG with high intercostal ECG recordings and drug challenge test is considered requisite not only to exclude BrS but also to stratify the risk of ERS.

Diagnosis of early repolarization syndrome

Early repolarization syndrome is diagnosed when structural and nonstructural heart diseases including BrS are excluded as a cause of VF. In the diagnostic criteria of BrS proposed in the consensus reports that were published in 2002 and 2005, a diagnostic Brugada ECG was defined as the presence of a coved-type ST segment elevation ≥2 mm followed by a negative T wave in at least two right
precordial leads on the standard 12-lead ECG. Regarding the high intercostal ECG recordings, it was stated that it could increase sensitivity, but there were not enough data at that time to exclude the possibility of false positives. However, some studies addressed the diagnostic value of the high intercostal ECG recordings in the following years, leading to revision of the diagnostic criteria for BrS in the expert consensus statement in 2013. The new diagnostic criteria allow diagnosis of BrS when a type 1 ST-segment elevation is observed either spontaneously or after drug provocation tests in at least 1 lead among the right precordial leads (V1 and V2) positioned in the second, third, or fourth intercostal spaces. These criteria proposed in 2013 have been reported to increase diagnostic sensitivity without increasing specificity.

Since inferolateral ER was reported in association with idiopathic VF in 2008 by Haïssaguerre et al., numerous studies on ERS have been published, leading to revision of the diagnostic criteria for BrS in the expert consensus statement in 2013. The new diagnostic criteria allow diagnosis of BrS when a type 1 ST-segment elevation is observed either spontaneously or after drug provocation tests in at least 1 lead among the right precordial leads (V1 and V2) positioned in the second, third, or fourth intercostal spaces. These criteria proposed in 2013 have been reported to increase diagnostic sensitivity without increasing specificity.

### Table 1 Patient characteristics of the four groups

<table>
<thead>
<tr>
<th></th>
<th>A (n = 6)</th>
<th>B (n = 5)</th>
<th>C (n = 8)</th>
<th>D (n = 19)</th>
<th>P-value comparing four groups</th>
<th>Total (n = 38)</th>
</tr>
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<tbody>
<tr>
<td><strong>Clinical characterisitics</strong></td>
<td></td>
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<tr>
<td>Age at diagnosis (year)</td>
<td>35.2 ± 6.9</td>
<td>38.2 ± 8.7</td>
<td>40.8 ± 14.4</td>
<td>42.5 ± 15.9</td>
<td>0.83</td>
<td>40.4 ± 13.6</td>
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<tr>
<td>Men (100%)</td>
<td>6 (100%)</td>
<td>5 (100%)</td>
<td>5 (63%)</td>
<td>18(95%)</td>
<td>0.045</td>
<td>34 (89%)</td>
</tr>
<tr>
<td>FH of SCD (%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>1 (13%)</td>
<td>0 (0%)</td>
<td>0.28</td>
<td>1 (3%)</td>
</tr>
<tr>
<td><strong>Activity at the time of initial SCA</strong></td>
<td></td>
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<tr>
<td>Sleep, near sleep (%)</td>
<td>3 (3, 0) (50%)</td>
<td>5 (4, 1) (100%)</td>
<td>6 (4, 2) (75%)</td>
<td>2 (2, 0) (11%)</td>
<td>0.0023</td>
<td>16 (13, 3) (42%)</td>
</tr>
<tr>
<td>Physical activity (%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
<td>6 (31%)</td>
<td>0.0023</td>
<td>6 (16%)</td>
</tr>
<tr>
<td>Arousal at rest (%)</td>
<td>3 (50%)</td>
<td>0 (0%)</td>
<td>2 (25%)</td>
<td>11 (58%)</td>
<td>0.0023</td>
<td>16 (42%)</td>
</tr>
<tr>
<td><strong>Occurrence of initial VF</strong></td>
<td></td>
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<tr>
<td>Out of hospital (%)</td>
<td>5 (83%)</td>
<td>4 (80%)</td>
<td>7 (87%)</td>
<td>18 (95%)</td>
<td>0.73</td>
<td>34 (89%)</td>
</tr>
<tr>
<td>In hospital for syncope or other reason (%)</td>
<td>1 (17%)</td>
<td>1 (20%)</td>
<td>1 (13%)</td>
<td>1 (5%)</td>
<td>0.12</td>
<td>4 (11%)</td>
</tr>
<tr>
<td>Induction of VF by EPS (%)</td>
<td>2/3 (67%)</td>
<td>1/1 (100%)</td>
<td>2/5 (40%)</td>
<td>3/10 (30%)</td>
<td>0.44</td>
<td>8/19 (42%)</td>
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<tr>
<td><strong>Clinical outcome</strong></td>
<td></td>
<td></td>
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<tr>
<td>Follow-up period (months)</td>
<td>120 ± 51</td>
<td>109 ± 61</td>
<td>106 ± 67</td>
<td>108 ± 54</td>
<td>0.95</td>
<td>110 ± 55</td>
</tr>
<tr>
<td>VF recurrence (%)</td>
<td>4 (67%)</td>
<td>4 (80%)</td>
<td>4 (50%)</td>
<td>2 (11%)</td>
<td>0.0057</td>
<td>14 (37%)</td>
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<tr>
<td>Electrical storm (%)</td>
<td>0 (0%)</td>
<td>2 (40%)</td>
<td>3 (38%)</td>
<td>0 (0%)</td>
<td>0.011</td>
<td>5 (13%)</td>
</tr>
<tr>
<td>ICD implantation</td>
<td>6 (100%)</td>
<td>5 (100%)</td>
<td>8 (100%)</td>
<td>18 (95%)</td>
<td>0.79</td>
<td>37 (97%)</td>
</tr>
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</table>

Numeric values are expressed as mean ± standard deviation. FH of SCD: family history of sudden cardiac death before 45 years of age. SCA, sudden cardiac arrest; EPS, electrophysiological study; VF, ventricular fibrillation; ICD, implantable cardioverter defibrillator.

A: Patients with ER and type 1 BrP-ECG only in HICS; B: ERS with non-type 1 BrP-ECG only in HICS; C: ERS with non-type 1 BrP-ECG in the standard ECG; D: ERS without BrP-ECG.

**Figure 2** Kaplan–Meier analysis of freedom from lethal arrhythmic events (documented ventricular fibrillation) during follow-up in the four groups of patients with inferolateral early repolarization and a prior ventricular fibrillation. The incidence of ventricular fibrillation during follow-up was significantly higher in early repolarization syndrome patients with BrP-ECG (groups A, B, and C) than in those without BrP-ECG (group D).
patients could have been misdiagnosed as having ERS under the previous criteria.

**Significance of Brugada-pattern electrocardiogram in the right precordial leads in early repolarization syndrome**

Higher intercostal recordings in leads V1 and V2 were reported to show similar prognostic value as standard recordings in BrS.3,5,14 Even in this study, six patients with type 1 BrP-ECG and five patients with non-type 1 BrP-ECG only in the high costal spaces showed a similar recurrence rate of arrhythmia as BrS patients in previous studies who were diagnosed by the standard ECG.

Regarding the significance of non-type 1 BrP-ECG in inferolateral ERS, we previously reported that such ERS patients had clinical profiles similar to BrS patients with high recurrence rates of VF and electrical storm.10 In this study, in which 30% of ERS patients had BrP-ECG only in the HICS, VF mostly recurred in patients showing BrP-ECG in any of the right precordial leads including HICS and these patients comprised 50% of our ERS cohort diagnosed under the previous criteria for ERS, although the remaining ERS patients without BrP-ECG exhibited a favorable outcome and clinical profiles dissimilar to BrS. Furthermore, the presence of BrP-ECG showed a good positive predictive value (63%) to identify VF recurrence during ~110 months of follow-up. This indicates that BrP-ECG can be a reliable marker of poor outcome in patients with ER or ERS. So far, useful predictors for sudden death due to VF have not been identified by both retrospective and prospective studies on ERS, although wider distribution of J-waves in inferolateral leads and horizontal/descending ST segment following J-waves were reported to be weak predictors for sudden death in patients with ER by retrospective studies in which the cause of sudden death was never identified.18–20 On the other hand, the registry of BrS patients in Japan,25 in which the prognosis of individuals with non-type 1 BrP-ECG was investigated prospecively in addition to the prognosis of those with type 1 ECG, contains records that 1 of 7 individuals with inferolateral J-waves and non-type 1 BrP-ECG on the standard ECG, who were not included in this study, developed VF during 40.7 ± 16.4 months of follow-up.

Early repolarization syndrome has been considered a clinical entity different from BrS, although they share a similar genetic background and represent a continuous spectrum of phenotypic expression.20 Quinidine and isoproterenol were reported to be the first-line therapy for suppressing VF even in ERS patients, the exact reasons for which are unknown.1,21 This study clarified that 12 of 14 ERS patients with VF recurrence had BrP-ECG in any of the right precordial leads. This means that many of the previously reported ERS patients with poor prognosis might have included patients with type 1 BrP-ECG only in the high costal ECG recordings or non-type 1 BrP-ECG in any lead, which could account for the clinical similarity of half of the ERS patients to BrS patients and the effectiveness of quinidine and isoproterenol in suppression of VF storm due to ERS. In contrast, most of the ERS patients without BrP-ECG in any of the right precordial leads exhibited a favorable outcome and clinical profiles dissimilar to BrS. We previously reported that ERS consisted of two heterogeneous subtypes with or without non-type 1 BrP-ECG.10 Provided that half of the ERS cases are nearly identical to BrS, the remaining ERS cases without BrP-ECG may be considered as true ERS possibly caused by a different mechanism. A systematic search for BrP-ECG with high intercostal ECG recordings and drug challenge test is required not only to exclude BrS but also to classify the subgroups of ERS.

**Study limitations**

This study was conducted at a single center using retrospective analysis. The small number of patients might limit the interpretation of the results; nevertheless, it should be pointed out that the number of patients with ERS is comparable to that in previous multicenter studies. Further prospective multicenter studies with larger numbers of patients will be needed to confirm these results.

**Conclusions**

This study showed that 16% of the patients diagnosed with ERS under the previous criteria were actually BrS patients with inferolateral ER and a type 1 BrP-ECG only in HICS that would not have been previously recognized unless appropriate recordings were performed. Thirty-four percent of the ERS patients with non-type 1 BrP-ECG in any of the right precordial leads including HICS were at high risk for arrhythmic events, in contrast to the 50% of ERS patients without BrP-ECG who were at very low risk for recurrent events despite a previous VF episode. These results also indicated that the presence of BrP-ECG can be a reliable marker of poor outcome in patients with ER or ERS. High intercostal ECG recording may be considered essential not only to exclude BrS but also to stratify the risk of ERS.

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**Conflict of interest:** none declared.

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