Clinical impact of ECG changes on oversensing of subcutaneous implantable cardioverter-defibrillators

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BACKGROUND Inappropriate shocks delivered by subcutaneous implantable cardioverter-defibrillators (S-ICDs) are most frequently caused by cardiac oversensing. However, the predictors for oversensing of S-ICD remain unclear.

OBJECTIVE We aimed to investigate the predictors for oversensing of S-ICD, especially clinical impact of an electrocardiographic (ECG) change.

METHODS We retrospectively enrolled 99 consecutive patients who underwent S-ICD implantation between 2013 and 2021. *Oversensing events* were defined as inappropriate charge of the capacitors induced by cardiac or noncardiac signals other than tachycardia.

RESULTS During a median follow-up period of 34 months (interquartile range 20–50 months), 11 of 99 patients (11%) experienced 34 oversensing events and 4 patients (4%) received inappropriate shocks during their events. Six patients exhibited ECG changes (bundle branch block, 3; ventricular pacing, 1; inverted T wave, 1; poor R-wave progression, 1) during the follow-up period. Oversensing events were observed in 4 of 6 patients with ECG change (67%), and 3 patients underwent S-ICD removal because of inevitable shock. Contrastingly, of the remaining patients without ECG change, all 7 patients who experienced oversensing events could continue using S-ICD with the reprogramming sensing vector and/or restriction of excessive exercise. Logistic regression analysis showed that lower voltage of Sokolow-Lyon ECG ($V_1S + V_5R$) was the predictor of oversensing in patients without ECG change. When the cutoff value was 2.1 mV, the sensitivity, specificity, positive predictive value, and negative predictive value were 85.7%, 62.7%, 15.7%, and 98.1%, respectively.

CONCLUSION Unavoidable oversensing resulting in S-ICD removal is caused by ECG change. Oversensing in patients without ECG change can be managed.

KEYWORDS Oversensing; Subcutaneous implantable cardioverter-defibrillator (S-ICD); Inappropriate shocks (IASs); ECG change

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Introduction

The subcutaneous implantable cardioverter-defibrillator (S-ICD) was developed as an alternative to the transvenous implantable cardioverter-defibrillator (TV-ICD).[1] Previous studies have demonstrated the safety and effectiveness of S-ICD.[2,3]

The novel S-ICD sensing filter (SMART Pass, Boston Scientific Corporation, Natick, MA) was reported to reduce the inappropriate shocks (IASs) delivered by S-ICD.[4] In the PRAETORIAN trial, the SMART Pass filter was not activated or was unavailable in 78% of patients who experienced IAS by S-ICD; however, S-ICD was shown to be noninferior to TV-ICD with respect to IAS.[5]

A previous study reported that IAS due to T-wave oversensing in S-ICD can be managed by reprogramming the sensing vector and/or the therapy zones of the device by using a template acquired during exercise screening.[6] However, there have been a few cases wherein a change from S-ICD to TV-ICD was necessary because IAS could not be regulated. Some of these cases were due to minor changes on electrocardiography (ECG).[7,8]

The impact of ECG change on sensing of S-ICD is still unknown. Additionally, the treatment for oversensing due to ECG change is still unknown. The purpose of this study was to clarify the impact of ECG change after implantation on oversensing and clinical outcomes in patients with S-ICD.

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Methods

Study population and definition

In this retrospective study, we included 105 consecutive patients who underwent S-ICD implantation at 2 institutions between 2013 and 2021.

As the primary objective, we determined the number of patients experiencing a change in ECG during the follow-up period and the number of patients experiencing oversensing events due to a change in ECG.

As the secondary objective, we estimated the number of patients who did not experience a change in ECG during the follow-up period but did experience oversensing events. In addition, we examined the risk factors for oversensing events in patients who did not experience a change in ECG. On the basis of the presence or absence of oversensing events during the follow-up period, patients were divided into 2 groups: oversensing and non-oversensing.

With or without IAS, an *oversensing event* was defined as inappropriate charge of the capacitors induced by cardiac or noncardiac signals other than tachycardia. *IAS* was defined as shock delivered due to (1) supraventricular tachycardia, (2) oversensing event (oversensing of cardiac or noncardiac signals), and (3) any other cause resulting in device shock in the absence of clinical arrhythmia.

Six patients with a follow-up period of <6 months were excluded. This study was approved by the institutional research board of Niigata University Medical & Dental Hospital. Niigata, Japan (2021-0100), and was conducted in accordance with the Declaration of Helsinki.

Clinical data

As the predictors of IAS by S-ICD and ineligibility for S-ICD screening ECG tests, previous studies reported height and weight, ECG characteristics, underlying heart disease, and S-ICD profile.[6,9,10] Therefore, clinical data collected from all patients included age, sex, type of underlying heart disease, medications, and ECG results including Sokolow-Lyon voltage before device implantation (Table 1). In all 3 vectors of S-ICD screening ECG, the amplitude of R and T waves and the R/T ratio were measured. The highest R/T ratio in the 3 leads were defined as the maximum R-wave amplitude, maximum T-wave amplitude, and maximum R/T ratio.

Table 1 Baseline characteristics and comorbidities of all included patients (N = 99)

Characteristic	Value
Female sex	16 (16)
Age (y)	52.5 ± 18.2
Height (cm)	166.6 ± 8.6
Weight (kg)	63.1 ± 13.6
BMI (kg/m^2)	22.6 ± 4.2
Underlying disease	
Coronary artery disease	39 (39)
Nonischemic cardiomyopathy	18 (18)
Brugada syndrome	24 (24)
Long QT syndrome	7 (7)
Others	11 (11)
Secondary prevention	56 (56)
Antiarrhythmic drugs class III	18 (18)
LV ejection fraction	54.7 ± 16.7
ECG findings at baseline	
Heart rate (beats/min)	67.5 ± 15.0
QT interval (ms)	420 ± 4.8
Corrected QT interval (ms)	440 ± 4.2
QRS duration (ms)	97.4 ± 25.3
$V_1S + V_5R$ (mV)	2.6 ± 1.3
Bundle branch block	3 (3)
Abnormal repolarization	2 (2)
Atrial fibrillation	15 (15)
AF paroxysmal	5 (5)
AF persistent/permanent	10 (10)
S-ICD screening ECG (n = 89)	
Max R-wave amplitude (mV)	1.8 ± 0.8
Max T-wave amplitude (mV)	0.26 ± 0.17
Max R/T ratio	9.1 ± 5.9
Programmed setting	
Dual-zone programming	98 (99)
SMART Pass enabled	90 (91)
Programmed vector	
Primary	46 (46)
Secondary	42 (42)
Alternate	11 (11)

Values are presented as mean ± SD or n (%).

AF = atrial fibrillation; BMI = body mass index; ECG = electrocardiographic; LV = left ventricular; S-ICD = subcutaneous implantable cardioverter-defibrillator.

Implantation procedure and device programming

All patients eligible for S-ICD implantation were screened using the recommended tool provided by the manufacturer. Screening was performed in the supine and standing/sitting position. Patients were required to have at least 1 screening vector to pass the screening test.

All implantation procedures were performed using local anesthesia and conscious sedation with infusion of dexmedetomidine and propofol. In the majority of cases, the pulse generator was placed between the anterior aspect of the serratus muscle and the posterior aspect of the latissimus dorsi muscle. Further, all patients underwent successful perioperative defibrillation testing. Sensing vectors were automatically determined at the end of the implantation procedure.

Within 7 days of the procedure, exercise screening of appropriate QRS/T wave detection was performed. In patients with comorbidities or inability to undergo the procedure, exercise screening was not performed. Sensing vectors were manually modified in several cases after exercise screening to obtain a better QRS/T wave ratio on the basis of the judgment of the operator.

Follow-up of patients and data collection

Data on arrhythmic episodes and IASs were collected during follow-up. Patients were routinely followed up every 3–6 months at the outpatient clinic. At each visit, device examinations were performed, and patients were asked if they experienced adverse events or IASs. Additional follow-up visits occurred in case of shock or if there were individual

device-related complications. A new significant change in the QRS and T-wave morphology that may affect S-ICD sensing was defined as a change in ECG (>50% decrease in QRS amplitude in the limb or chest leads, complete right or left bundle branch block, inversion of the T wave in ≥2 leads, ventricular pacing, QT prolongation, and abnormal Q wave).

Statistical analysis

The intergroup differences in clinical characteristics and clinical course were determined using the unpaired *t* test for continuous variables and the χ^2 test for categorical variables. Quantitative data were presented as mean ± SD, median and interquartile range, and ranges depending on the distribution of data, and these parameters were compared using the Student *t* test for normally distributed data and the Mann-Whitney *U* test for nonparametric data. In patients without ECG change, logistic regression was used to evaluate the association between the oversensing event and the characteristics of the patients, and the odds ratios together with their 95% confidence intervals were reported. A *P* value of <.05 indicated statistical significance between the groups (oversensing group vs non-oversensing group). Categorical data were displayed as frequency and percentage. Continuous data were expressed as mean ± SD. S-ICD screening ECG was missed in 10 patients; therefore, we used the full information maximum likelihood method to account for missing data. All analyses were performed using JMP 14 software (SAS Institute Inc, Cary, NC).

Results

Clinical characteristics and outcomes

The clinical characteristics of the patients are summarized in Table 1. In total, 98 patients (99%) had dual-zone programming, the most frequent setting being a conditional zone of 200 beats/min and a shock zone of 220 beats/min in 87 of 99 (88%). The SMART Pass filter was available in 90 patients (91%). The primary vector was programmed in 46 (46%), the secondary vector in 42 (42%), and the alternate vector in 11 patients (11%).

During a median follow-up period of 34 months (interquartile range 20–50 months), 11 of 99 patients experienced 34 oversensing events (11%) (2 [2–3] events per patient). Of the 11 patients, 7 patients experienced inappropriate charge without a shock (7%) and 4 patients experienced an IAS during their events (4%). Table 2 presents the demographic and oversensing event data of the patients. S-ICD had to be explanted in 3 patients (3%) because of refractory oversensing events without programming options. Their clinical history of oversensing events and vector setting are summarized in Figure 1. The duration until the first oversensing event was 103 (48–362) days.

Because of supraventricular tachycardia, 4 patients experienced IAS without oversensing (paroxysmal atrial fibrillation, 3; paroxysmal supraventricular tachycardia, 1) (Figure 2). After a change in the detection zone or rate control by oral administration of medications or rhythm control by ablation, patients experienced no IAS.

Correlation between ECG change and oversensing events

During the follow-up period, a change in ECG was confirmed in 6 patients (bundle branch block, 3; ventricular pacing, 1; inverted T wave, 1; poor R-wave progression, 1) (Table 3). Of these 6 patients, 4 experienced oversensing events, including 3 patients who underwent TV-ICD implantation after removing the S-ICD, since it was determined that an IAS was unavoidable. For 7 patients who experienced oversensing events without a change in ECG, continuing the use of S-ICD was possible by changing the sensing vectors

Table 2 Summary of 11 patients who experienced oversensing events

Patient no.	Age (y)/ sex	Underlying disease	ECG change	Potential oversensed	Event time after implantation (d)	IAS	Sensing vector		Exchange to TV-ICD (d)
							At the event	After the event	
1	66/M	VSA	Inverted T wave	TWOS + noncardiac noise	32	+	Secondary	Secondary	48
2	63/M	DCM	Poor R-wave progression	Noncardiac noise	546	+	Secondary	Primary	762
3	33/M	HCM	CRBBB	TWOS	103	+	Secondary	Alternate	311
4	76/M	HCM	Ventricular pacing	TWOS + noncardiac noise	520	–	Primary	Secondary	–
5	56/M	ICM	–	Noncardiac noise	1	+	Alternate	Alternate	–
6	39/M	Brs	–	Noncardiac noise	64	–	Primary	Alternate	–
7	13/F	LQT	–	Noncardiac noise	805	–	Primary	Primary	–
8	31/F	HCM	–	TWOS	204	–	Secondary	Secondary	–
9	56/M	Brs	–	Noncardiac noise	69	–	Secondary	Secondary	–
10	49/M	Brs	–	Noncardiac noise	3	–	Primary	Alternate	–
11	70/M	ICM	–	Noncardiac noise	201	–	Primary	Primary	–

+ = presence, – = none.

Brs = Brugada syndrome; CRBBB = complete right bundle branch block; DCM = dilated cardiomyopathy; ECG = electrocardiographic; F = female; HCM = hypertrophic cardiomyopathy; IAS = inappropriate shock; ICM = ischemic cardiomyopathy; LQT = long QT syndrome; M = male; TV-ICD = transvenous implantable cardioverter-defibrillator; TWOS = T-wave oversensing; VSA = vasospasm angina.

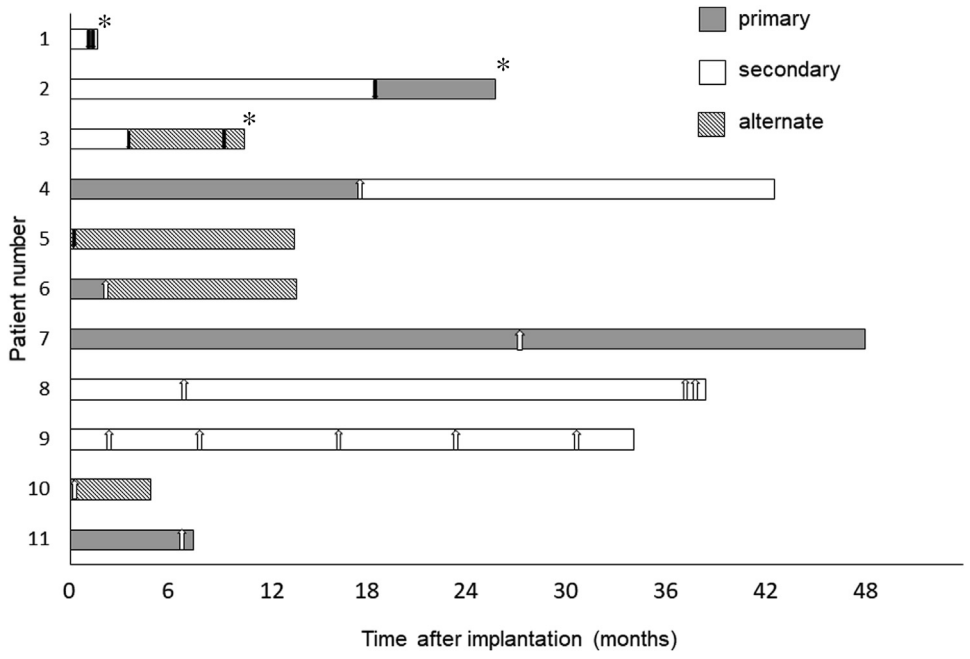

Figure 1 Temporal relation of oversensing events with regard to implantation. The *arrows* denote inappropriate charge of the capacitors due to oversensing (*black arrows*, with inappropriate shock; *white arrows*, without shock). The *vertical bars* indicate time since implantation. *Subcutaneous implantable cardioverter-defibrillator removal because of an inevitable shock due to an oversensing event (patients 1–3).

(2 patients) and restricting excessive exercises (5 patients). Oversensing events were significantly more common in patients with ECG change than in those without ECG change (with ECG change: 4 of 6; without ECG change: 7 of 93; $P = .001$) (Figure 2).

Among the 93 patients without ECG change, the oversensing group (7 patients) was compared with the non-oversensing group (86 patients). Baseline and follow-up characteristics are presented in Table 4. ECG findings at baseline showed that the Sokolow-Lyon voltage (adding

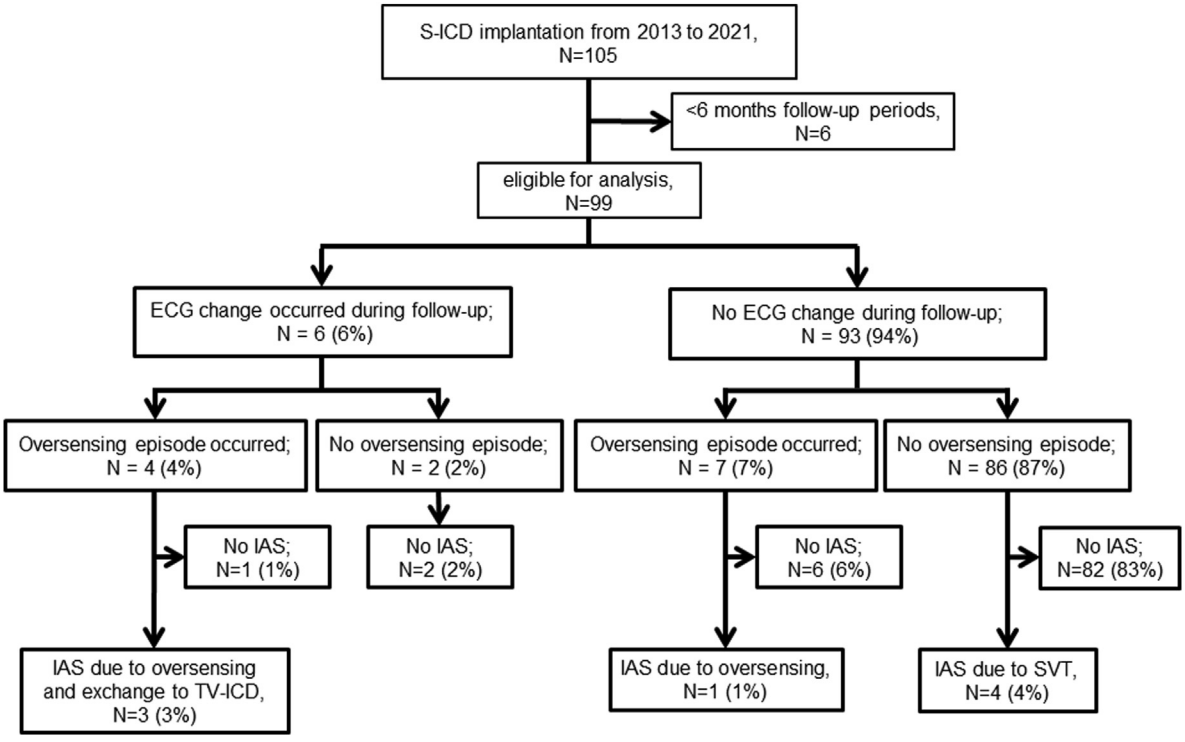

Figure 2 Flowchart of the clinical course in this study. ECG = electrocardiographic; IAS = inappropriate shock; S-ICD = subcutaneous implantable cardioverter-defibrillator; SVT = supraventricular tachycardia; TV-ICD = transvenous implantable cardioverter-defibrillator.

Table 3 Summary of 6 patients with ECG change during the follow-up period

Patient no.	Age (y)/sex	Underlying disease	ECG change	Event time after implantation (d)	Potential oversensed	IAS	Exchange to TV-ICD (d)	Follow-up period (d)
1	66/M	VSA	Inverted T wave	32	TWOS + noncardiac noise	+	48	–
2	63/M	DCM	Poor R-wave progression	546	Noncardiac noise	+	762	–
3	33/M	HCM	CRBBB	103	TWOS	+	311	–
4	76/M	HCM	Ventricular pacing	520	TWOS + noncardiac noise	–	–	1261
5	63/M	IVF	CRBBB	–	–	–	–	865
6	75/M	ICM	First-degree AVB + CLBBB	–	–	–	–	1774

+ = presence; – = none; AVB = atrioventricular block; CLBBB = complete left bundle branch block; CRBBB = complete right bundle branch block; DCM = dilated cardiomyopathy; ECG = electrocardiographic; F = female; HCM = hypertrophic cardiomyopathy; IAS = inappropriate shock; ICM = ischemic cardiomyopathy; IVF = idiopathic ventricular fibrillation; M = male; TV-ICD = transvenous implantable cardioverter-defibrillator; TWOS = T-wave oversensing; VSA = vasospasm angina.

the S-wave amplitude in lead V_1 and the R-wave amplitude in lead V_5) was significantly lower in the oversensing group than in the non-oversensing group (oversensing group: 1.5 [1.0–2.1] mV vs non-oversensing group: 2.5 [1.7–3.2] mV; $P = .03$). The amplitude of $V_1S + V_5R$ in patients without ECG change was significantly lower at follow-up than at baseline (baseline: 2.5 ± 1.3 mV vs follow-up: 2.3 ± 0.9 mV; $P = .001$), and ECG findings at follow-up also showed that the amplitude of $V_1S + V_5R$ was significantly lower in the oversensing group than in the non-oversensing group (oversensing group: 1.4 [1.0–1.8] mV vs non-oversensing group: 2.2 [1.7–2.7] mV; $P = .005$).

Univariate logistic regression analyses showed that the lower voltage of Sokolow-Lyon was the only predictor of an oversensing event (Table 5). Receiver operating characteristic curve analysis showed that 2.1 mV was the optimal cut-off value for prediction (area under the curve 0.75; 95% confidence interval 0.57–0.93; $P = .03$), and its sensitivity, specificity, positive predictive value, and negative predictive value were 85.7%, 62.7%, 15.7%, and 98.1%, respectively.

Table 4 Differences between patients with oversensing and non-oversensing among 93 patients without ECG change

Variable	Oversensing (n = 7)	Non-oversensing (n = 86)	*P*
Female sex	2 (29)	14 (16)	.35
Age (y)	44.9 ± 17.5	52.5 ± 18.3	.29
BMI (kg/m^2)	22.2 (19.7–23.8)	22.0 (19.6–24.4)	.93
Underlying disease			
Coronary artery disease	2 (29)	35 (41)	.70
Nonischemic cardiomyopathy	1 (14)	14 (16)	>.99
Brugada syndrome	3 (43)	21 (24)	.37
Long QT syndrome	1 (14)	6 (7)	.43
Antiarrhythmic drugs, class III	0 (0)	18 (21)	.34
LV ejection fraction (%)	54.7 ± 18.2	55.0 ± 16.3	.96
ECG findings at baseline			
Corrected QT duration (ms)	433 (409–460)	417 (410–458)	.92
QRS duration (ms)	105 (88–110)	96 (80–108)	.42
$V_1S + V_5R$ (mV)	1.5 (1.0–2.1)	2.5 (1.7–3.2)	.03
$V_1S + V_5R$ in follow-up ECG (mV)	1.4 (1.0–1.8)	2.2 (1.7–2.7)	.005
Decreasing voltage of $V_1S + V_5R$	5 (71)	60 (70)	>.99
History of atrial fibrillation	0 (0)	13 (15)	.59
S-ICD screening ECG (n = 83)			
Max R-wave amplitude (per 1 mV)	1.5 (1.1–1.8)	1.5 (1.2–2.2)	.29
Max T-wave amplitude (per 1 mV)	0.27 (0.2–0.3)	0.2 (0.12–0.37)	.50
Max R/T ratio	6 (3–8)	7.8 (5–12)	.19
Programmed vector			
Primary	2 (29)	41 (48)	.44
Secondary	3 (43)	36 (42)	.96
Alternate	2 (29)	9 (10)	.19

Values are presented as mean ± SD, median (interquartile range), or n (%).

BMI = body mass index; ECG = electrocardiographic; LV = left ventricular; S-ICD = subcutaneous implantable cardioverter-defibrillator.

Table 5 Oversensing risk factors at baseline in 93 patients without ECG change

Variable	Univariate		
	OR	CI	*P*
Female sex	2.06	0.3611.7	.44
Age	0.98	0.94–1.02	.30
BMI	0.96	0.78–1.18	.67
LV ejection fraction	0.998	0.953–1.05	.96
ECG findings at baseline			
Corrected QT duration (per 10 ms)	1.01	0.84–1.21	.96
QRS duration (per 10 ms)	1.05	0.79–1.39	.74
$V_1S + V_5R$ (per 1 mV)	0.34	0.12–0.97	.02
S-ICD screening ECG			
Max R-wave amplitude (per 1 mV)	0.89	0.76–1.06	.12
Max T-wave amplitude (per 1 mV)	1.01	0.63–1.61	.97
Max R/T ratio	0.82	0.63–1.06	.08
Programmed vector			
Primary	0.42	0.08–2.28	.29
Secondary	1.04	0.23–4.94	.96
Alternate	3.90	0.65–23.5	.17

CI = confidence interval; OR = odds ratio; other abbreviations as in Table 1.

Discussion

Main findings

The major findings of the present study are as follows: (1) Oversensing events were significantly more common in patients with ECG change than in those without ECG change (with ECG change: 4 of 6; without ECG change: 7 of 93; $P = .001$). (2) Of the 6 patients who exhibited ECG changes, 3 underwent S-ICD removal because of inevitable shock. (3) Of the 93 patients without ECG change, all 7 patients who experienced oversensing events could continue using S-ICD. (4) In patients without ECG change, a lower Sokolow-Lyon voltage ($V_1S + V_5R$) at baseline was associated with a higher likelihood of oversensing and the optimal cutoff value was 2.1 mV for predicting oversensing events (sensitivity and negative predictive value reached 85.7% and 98.2%, respectively).

Cause of IAS in S-ICD

IAS remain a relevant S-ICD complication and are different from TV-ICD in which programming optimization has led to a clear reduction of oversensing-related IAS over the years.[11] Basu-Ray et al[12] reported that inappropriate therapies in patients with TV-ICD were primarily due to supraventricular tachycardia whereas IAS in patients with S-ICD were mostly episodes of oversensing (sensing of noise and T-wave oversensing, among others). In the European multicenter study assessing long-term complications in patients undergoing S-ICD implantation (ELISIR project),[13] despite the availability of the SMART Pass filter in 85% of patients, 118 patients (8.9%) experienced IAS during the first 2 years of implantation and oversensing was one of the most common triggers. IAS caused by supraventricular tachycardia present treatment options such as programming optimization, rate control by oral administration of medications, and rhythm control by catheter ablation[14]; thus, it is unlikely that the use of TV-ICD needs to be discontinued because of IAS. In contrast, the only response to oversensing, a major cause of IAS in S-ICD, is reprogramming of the sensing vector and/or the therapy zones of the device.

A retrospective study that examined all patients at a facility with S-ICD implantation observed that during the follow-up period (mean duration ~4 years), 18 of 351 patients (5%) presented with T- or P-wave oversensing resulting in IAS. Reprogramming of the ICD could not avert oversensing, and 3 of 18 patients concerned underwent extraction of the S-ICD and implantation of a TV-ICD.[15]

In this study, because of supraventricular tachycardia, 4 patients experienced IAS without oversensing; however, after ablation or a change in the detection zone, these patients did not experience IAS. In contrast, 4 of 11 patients who experienced oversensing events suffered IAS. Three of these patients had no option but to change to TV-ICD.

In several case reports, it has been mentioned that generator repositioning can also be considered to reduce myopotential oversensing that cause IAS; however, since the follow-up period was short, it is unclear whether generator repositioning can avert IAS in the future.[16,17] IAS in S-ICD are different from those in TV-ICD, which leads to shock in all cases. It has been reported that this can cause posttraumatic stress disorder; and thus, appropriate assessment of and response to oversensing is necessary.[8]

Change in ECG and oversensing

There are several reported cases in which iatrogenic change in ECG had led to IAS by oversensing. Alcohol septal ablation or myectomy in the patient with hypertrophic obstructive cardiomyopathy was reported to cause bundle branch block, resulting in IAS due to T-wave oversensing.[18,19] The former case required a change to TV-ICD; however, in the latter case, a change to S-ICD equipped with a SMART Pass filter was adopted. By changing the sensing vector from primary to alternate, it was possible to continue using S-ICD.

There are also reports of IAS after ablation and oversensing caused by combined use with cardiac resynchronization therapy.[7,20] It is still unknown what change in ECG are likely to cause abnormal sensing. Caution is necessary in a treatment that could lead to iatrogenic change in ECG, and the necessity for vector reprogramming should be noted.

There is no past study that discussed the change in ECG during the follow-up period. In this study, a change in ECG was confirmed in 6 of 99 patients, 3 of whom required a change to TV-ICD because oversensing events could not be averted with reprogramming. In 1 case, preoperative transient right branch bundle block was confirmed (patient 3). In another case, a negative T wave after resuscitation in acute coronary syndrome was not completely improved when the screening test was performed (patient 1). Indications for S-ICD implantation should be determined after considering whether the patient is out of the acute phase with a potential change in ECG and whether the disease is progressive with

potential for future change in ECG. It is important to routinely monitor 12-lead ECG in patients with S-ICD and consider the need for repeat exercise testing and/or device reprogramming if a significant ECG change occurs.

Oversensing without a change in ECG

There are prior reports of S-ICD oversensing without a change in 12-lead ECG; in 1 case, a change to TV-ICD was required.[8,21] In our analysis of patients without ECG change during the follow-up period, a lower Sokolow-Lyon voltage ($V_1S + V_5R$) at baseline was associated with a higher likelihood of oversensing. With use of 2.1 mV as a cutoff value for Sokolow-Lyon voltage, the sensitivity and negative predictive value for oversensing events reached 85.7% and 98.2%, respectively. Because of its high negative predictive value, higher $V_1S + V_5R$ ($\geqq$2.1 mV) suggests that the risk of future oversensing events is negligible in the absence of ECG change. Although the exact reason why $V_1S + V_5R$ is the factor associated with oversensing event is unclear, we propose the following hypothesis: The Sokolow-Lyon voltage ($V_1S + V_5R$) in patients without ECG change was significantly lower at follow-up than at baseline. In ECG at follow-up and at baseline, the Sokolow-Lyon voltage was significantly lower in patients with oversensing than in those without oversensing. Therefore, patients with an initially low Sokolow-Lyon voltage might have been on the edge of suffering oversensing, and disease progression might have caused oversensing through a decrease of R-wave amplitude in S-ICD. In this study, no risk factors were identified that would cause a decrease in Sokolow-Lyon voltage (Supplementary Table 1), and further prospective studies that include a larger number of patients with S-ICD are warranted to confirm these findings.

This study did not identify the R/T ratio in screening ECG before S-ICD implantation as a predictive factor for a postoperative oversensing event. A previous study suggested that too anterior generator position causes the current to shunt via the anterior chest wall instead of traversing the critical mass of the heart and that fat tissue under the generator has isolating properties, thereby leading to resistance in the electrical circuit.[22] For preventing these factors resulting in conversion failure, the generator was placed deeply in a more posterior position than the one in which screening ECG was performed. Thus, the Sokolow-Lyon voltage value might have predicted the height of the R wave after implantation more accurately than did screening ECG.

Limitations

This study has several limitations. First, this study is a retrospective study with a small sample size, which might have affected the results. Second, we could not eliminate the potential bias caused by the missing value with regard to S-ICD screening ECG. Complete case analysis performed by including only the 89 patients without the missing value showed almost the same results. We used the full information maximum likelihood method to handle the missing data of S-ICD screening ECG; however, this analytical method might have affected the result, especially the predictor of an oversensing event in logistic regression analyses. However, no other studies have evaluated the significance of an ECG change in terms of clinical outcomes, and this study is the first to report the Sokolow-Lyon voltage value as a predictive factor for an oversensing event.

Conclusion

S-ICD oversensing events associated with a significant change in ECG from the time of initial preimplant screening are more likely to necessitate device removal than oversensing events in the absence of an ECG change, which can often be managed with reprogramming. The lower Sokolow-Lyon voltage at baseline is associated with a higher likelihood of later oversensing events.

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The authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2022.05.037.

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