

Subcutaneous implantable cardioverter-defibrillator was inappropriate for use in a patient with aborted sudden cardiac death due to coronary spastic angina: a case report

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Background

Implantable cardioverter-defibrillator (ICD) is recommended for secondary prevention in patients with coronary spastic angina and aborted sudden cardiac death. The effectiveness of subcutaneous ICD (S-ICD) for patients with coronary artery spastic angina is controversial.

Case summary

A 54-year-old man presented with ventricular fibrillation. Emergent coronary angiography showed diffuse narrowing of the coronary arteries that was reversible with isosorbide dinitrate. He was diagnosed with coronary spastic angina. S-ICD was implanted after the administration of a calcium-channel blocker and nicorandil. Seven months after the implantation, he collapsed again due to sinus node dysfunction and atrioventricular block caused by cardiac ischaemia. He developed cardiac arrest at both admissions. Six hours after the admission, electrocardiogram showed transient right bundle branch block. Inappropriate shocks were delivered because of low R-wave amplitude and T-wave oversense. S-ICD was replaced with a transvenous device in order to manage these two arrhythmias and inappropriate shocks.

Discussion

Patients with coronary artery spasm and aborted sudden cardiac death are candidates for implantation of S-ICD, but there are risks of bradycardia and inappropriate shocks in other ischaemic events.

Keywords

Coronary spastic angina • Aborted sudden cardiac death • Subcutaneous implantable cardioverter-defibrillator • Inappropriate shock • Case report

Learning points

- Cardiac ischaemia due to vasospasm could cause both fatal bradycardia and malignant ventricular arrhythmias unpredictably.
- Because low R-wave amplitude and T-wave oversense during ischaemia result in inappropriate shocks, S-ICD may not be appropriate for coronary spastic angina.

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Introduction

Patients with coronary spastic angina could present with life-threatening ventricular arrhythmias.¹ Optimal medical therapy, including calcium-channel antagonists or nitrates, is effective in many cases, but concerns remain because medication may not be able to suppress spastic events completely.^{1,2} Many clinical reports showed that implantable cardioverter-defibrillator (ICD) was effective for the prevention of sudden cardiac death in patients with coronary spasm.^{3–5} However, these studies were conducted mainly before subcutaneous ICD (S-ICD) became available. This is a case report against S-ICD indication for these patients.

Timeline

Time	Events
Day 0	A 54-year-old male collapsed. An automated external defibrillator (AED) terminated ventricular fibrillation. He was transferred to an emergency room, and diagnosed with coronary spastic angina. Vasodilators were administered.
Day 10	A subcutaneous implantable cardioverter defibrillator(S-ICD) was implanted.
7 months	He collapsed again. An AED detected severe sinus bradycardia and advanced atrioventricular block. The arrhythmia was associated with coronary spastic angina again. No episode of ventricular arrhythmias were found, but 21 inappropriate shocks were delivered because of T wave oversense with smaller R wave during ischemia. Shock therapy was turned off.
10 months	S-ICD was replaced with transvenous ICD.

Case presentation

A 54-year-old Japanese male collapsed in his workplace at 8 a.m. An automated external defibrillator (AED) was used promptly to detect ventricular tachycardia and ventricular fibrillation (*Figure 1A*). The second electrical shock terminated the arrhythmia. His medical history was not remarkable. At presentation to the emergency room, he had a little impaired orientation with Glasgow coma scale score of 13/15 (E3, V4, M6). His blood pressure was 172/126 mmHg, heart rate was 80 b.p.m. His electrocardiogram (ECG) showed ST-elevation in the inferior leads and ST depression in the lateral leads (*Figure 1B*). Echocardiography showed hypokinesis of inferior left ventricular wall. Laboratory analysis revealed normal values: white blood cell count, $8.35 \times 10^9/L$ (normal: $3.58–8.15 \times 10^9/L$); haemoglobin, 161 g/L (133–166 g/L); platelet count, $150 \times 10^9/L$ ($172–359 \times 10^9/L$); sodium, 139 mmol/L (136–147 mmol/L); potassium, 3.7 mmol/L (3.6–5.0 mmol/L); chloride, 98 mmol/L (98–109 mmol/L); and creatine kinase (CK), 97 IU/L (56–244 IU/L). Emergent coronary angiography (CAG) showed diffuse narrowing of the coronary arteries (*Figure 2*). Intracoronary injection of isosorbide dinitrate (1 mg) fully dilated the arteries (*Figure 2*). He was diagnosed with coronary spastic angina which caused malignant ventricular

arrhythmia. Benidipine hydrochloride (8 mg/day) and nicorandil (15 mg/day) were administered. He recovered without neurological defect and his left ventricular wall motion was completely restored. S-ICD (EMBLEM MRI A219, Boston Scientific, Marlborough, MA, USA) was implanted after he passed standard ECG screening algorithm in all vectors in both supine and standing positions. The primary sensing vector was set for the detection and SMART PASS function was activated. Dual-zone was programmed (conditional shock zone >200 b.p.m.; shock zone >240 b.p.m.).

Seven months after the implantation, he felt chest pain in his sleep, and call for ambulance at 3 a.m. Ambulance crews found him lying unconscious when they arrived. An AED detected severe sinus bradycardia and advanced atrioventricular block (*Figure 1C*). After successful cardiopulmonary resuscitation, he was brought to our hos-

pital. On presentation to the emergency room, he was unconscious with Glasgow coma scale score of 6/15 (E2, V2, M2). His blood pressure was 144/105 mmHg, heart rate was 94 b.p.m. His ECG showed abnormal Q wave and ST-segment elevation in the inferior leads, and ST depression in the lateral leads (*Figure 1D*). Echocardiography showed inferior wall motion of his left ventricle was impaired. Laboratory analysis revealed normal values of complete blood cell count and electrolytes, and elevated cardiac enzymes (CK 765 IU/L). Because isosorbide dinitrate was injected in the coronary artery before CAG, emergent CAG was normal. At this time of admission, a device interrogation found no episode of ventricular arrhythmias. In the next 6 hours, 21 shocks were delivered without discernible arrhythmia events. Device interrogation revealed eight episodes of ventricular arrhythmia that were recorded incorrectly. The amplitude of sensed ECG was markedly decreased (*Figure 3*). Shock therapy was then turned off to avoid inappropriate sensing and shock delivery. Surface ECG, sensed ECG amplitude, and ventricular wall motion were gradually normalized. Because there were risks of recurrent bradycardia and inappropriate shock in the next event, we explanted S-ICD and switched to transvenous ICD (TV-ICD). In subsequent follow-up of 7 months, TV-ICD interrogation revealed no significant events.

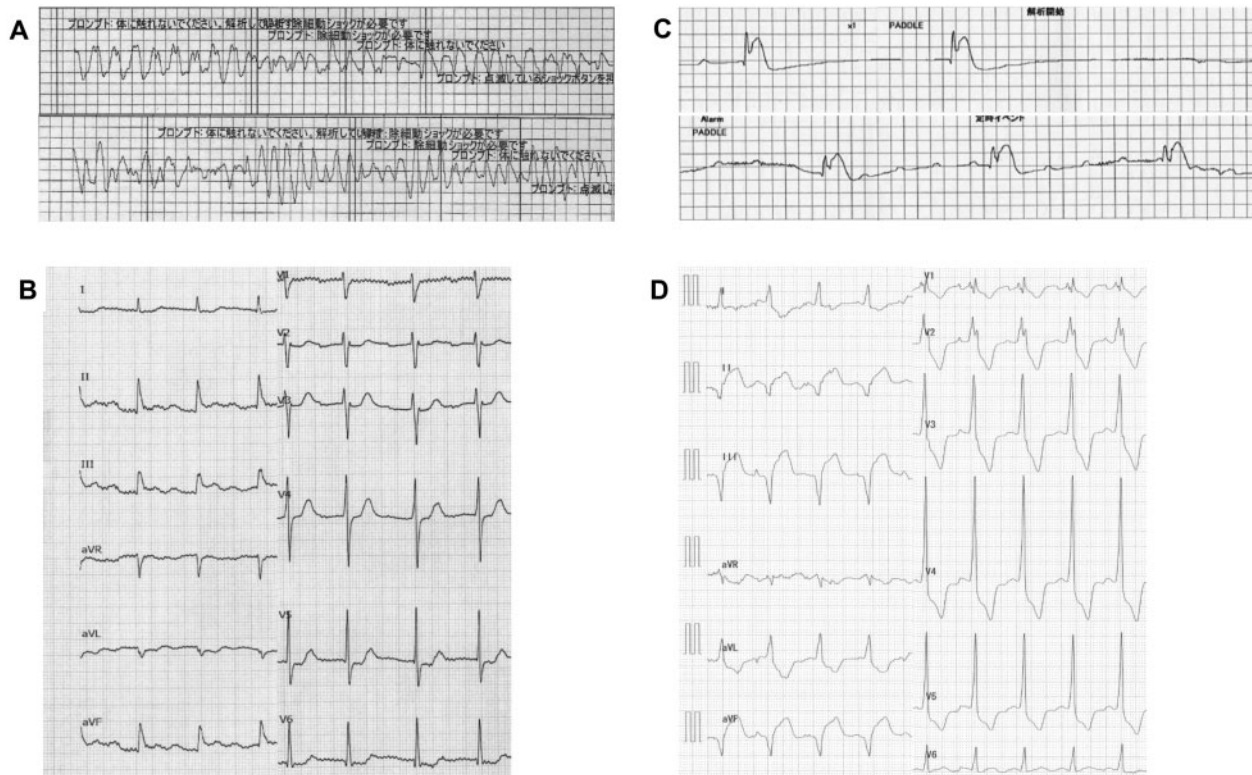


Figure 1 Electrocardiogram. (A) Ventricular tachycardia and fibrillation recorded by automated external defibrillator in the first episode. (B) Twelve-lead electrocardiogram recorded on the first admission showed ST-segment elevation in the inferior leads and ST-segment depression in the lateral leads. (C) An automated external defibrillator record showed severe sinus bradycardia and atrioventricular block, and ST-segment elevation in the second episode. (D) Twelve-lead electrocardiogram recorded on the second admission showed abnormal Q wave, wide QRS morphology, ST-segment elevation in the inferior leads and ST-segment depression in the lateral leads.

Discussion

We report here a fatal case of coronary spastic angina whose S-ICD was replaced to TV-ICD because of severe bradycardia and inappropriate shocks during transient cardiac ischaemia. ICD implantation is recommended for secondary prevention in patients with coronary spastic angina, and aborted sudden cardiac death (ASCD).³⁻⁵ This recommendation is based on the studies conducted before the introduction of S-ICD, thus TV-ICD was used. Recently, the number of implantations of S-ICD is gradually increasing for patients with coronary spastic angina.^{6,7} However, their effectiveness has not been clearly elucidated. Although S-ICD is useful to resume sinus rhythm, electrical shock may have deleterious effects in this situation, resulting in electrical storm. Moreover, atrioventricular block and sinus pause are often observed. In such situations, pacing function is necessary to prevent collapse.

In our patient, abrupt decrease in sensed ECG was observed during ischaemia. Such a phenomenon was reported in a case of ablation for ventricular tachycardia and in the forward flexion position, but was not reported in cases of cardiac ischaemia.^{8,9} It was reported that complete right bundle branch block and QRS-T discordance of surface ECG was associated with S-ICD ineligibility when three sensing vectors were checked as a screening test

before implantation.¹⁰ A prolonged QRS duration may be associated with ineligibility.¹¹ Our case had these ECG characteristics during the second admission when inappropriate electrical shocks were observed. The previous observation pointed out the characteristic surface ECG pattern, but not necessarily the low amplitude of sensed ECG. On the other hand, we found that the inappropriate function of S-ICD was related to very low amplitude of sensed ECG that occurred temporarily. Setting sensitivity automatically higher, both R and T waves were sensed as ventricular events, and recognized as ventricular tachycardia to deliver shocks inappropriately. Although we could change the sensing vector to secondary or alternate, there was a risk of R-wave decrease in other ischaemic events. Once bradycardia is documented, we consider it is appropriate to replace the device to TV-ICD because of pacing function and larger amplitude of intracardiac R wave. Because the decrease of sensed ECG amplitude coincided with ventricular ischaemia, cardiac muscular injury may be related to the low amplitude of sensed ECG.

Caution will be needed when one selects S-ICD for the patients with coronary spasm and ASCD because severe bradycardia may occur in another ischaemic event. There is also a risk of unpredictable change in R-wave amplitude that may cause inappropriate shock. Further studies including large numbers of patients are needed for

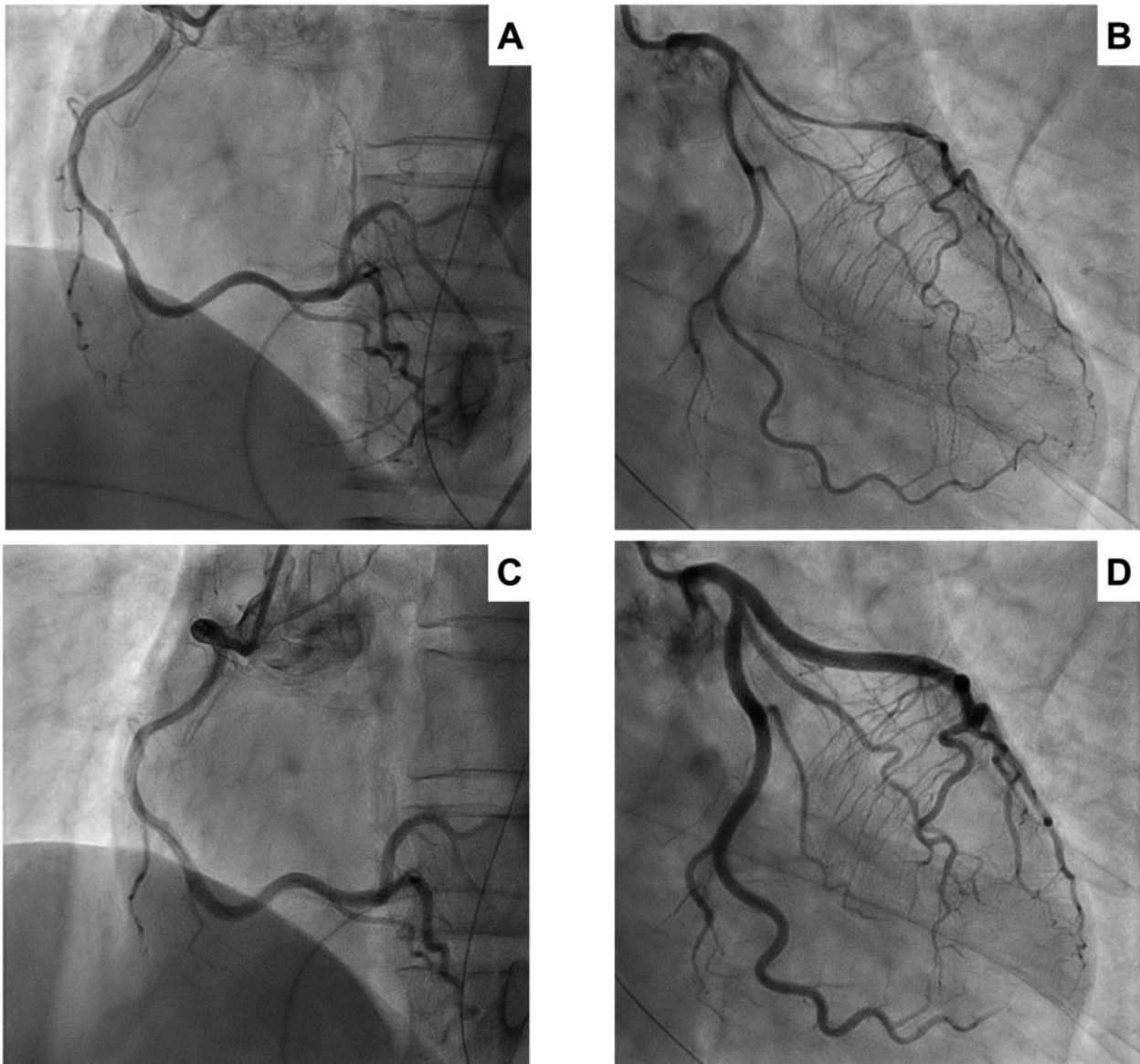


Figure 2 Coronary angiography (CAG). Right (A) and left (B) CAG at baseline. Right (C) and left (D) CAG after intracoronary injection of isosorbide dinitrate.

QRS morphology alterations and inappropriate shocks during acute ischaemia.

Conclusion

We reported a case of coronary spastic angina in a patient who developed two cardiac arrests with ventricular arrhythmia on the first admission and bradycardia on the second admission. S-ICD delivered shocks inappropriately during ischaemia and was replaced with TV-ICD. Caution will be needed when one selects S-ICD for the patients with coronary spasm and ASCD.

Lead author biography



Dr. Akiteru Kojima was trained in electrophysiology at Kyoto prefectural University of Medicine in Japan. He works at Japanese Red Cross Kyoto Daiichi Hospital as cardiologist and specializes in arrhythmia. His field of interest is a role of arrhythmias in the progression of heart failure.

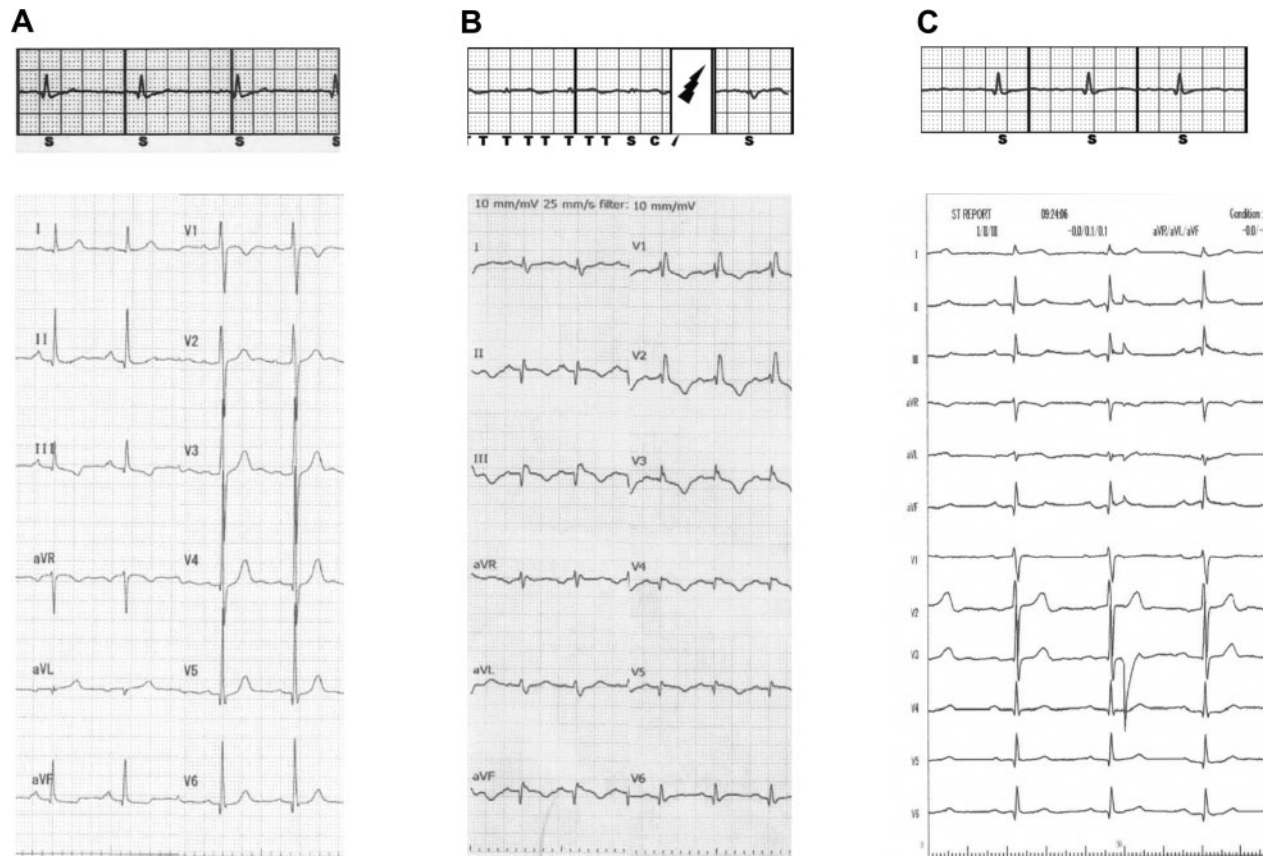


Figure 3 Sensed electrocardiogram (upper panels) and surface electrocardiogram (lower panels). (A) Electrocardiogram at implantation. (B) Electrocardiogram when inappropriate shocks were delivered. (C) Electrocardiogram after recovery.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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