



Case Report

Ventricular fibrillation survivor due to painless multiple spasm including left main trunk: is the subcutaneous implantable cardioverter-defibrillator necessary?



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ABSTRACT

A 52-year-old man was transferred to our hospital for ventricular fibrillation. He had no chest symptoms until then. After his full recovery, the administration of acetylcholine 20 μ g showed the multiple spasm (left main trunk/left anterior descending artery/right coronary artery) without any chest symptoms or ischemic electrocardiographic changes. Subcutaneous implantable cardioverter-defibrillator (S-ICD) was implanted because of no chest symptoms during his-daily-life. We performed the pharmacological spasm provocation tests under the abundant medications. Sequential spasm provocation test provoked just focal spasm at mid left anterior descending artery. We convinced that medications and S-ICD suppress the next fatal event.

< **Learning objective:** LMT spasm may cause aborted SCD. Aggressive spasm provocation tests under the abundant medical therapy provoked spasm just at mid LAD artery focally in patient with VF survivor due to multiple spasm including LMT, while these medications blocked the LMT and RCA spasm. S-ICD implantation may be one of the selections in aborted SCD patients with silent LMT spasm under the abundant vasodilators in the future.>

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Introduction

Coronary artery spasm may be involved in the genesis of various cardiac disorders. Serious fatal arrhythmia such as ventricular fibrillation (VF) or tachycardia is often observed in patients with aborted sudden cardiac death (SCD) due to coronary spasm. Implantable cardioverter-defibrillator (ICD) was effective in suppressing the next serious fatal event in patients with refractory spasm as well as structural heart disease [1]. However, some patients with pulseless electrical activity or cardiac arrest due to coronary spasm die after the implantation of ICD [2]. We sometimes encountered a case with silent coronary spastic angina in the clinic.

Case report

A 52-year-old man was transferred to our hospital because of VF. After 10 min later of his catastrophic event, he recovered from VF during jogging leisurely with the automated external defibrillator and cardiac massage by a bystander, as shown in Fig. 1-A. He was transferred to our hospital by the ambulance. He was under the intubation with hypothermia therapy. On day 4 of admission, he had total recovery without any irreversible complications. He had a smoking history for 32 years and quit smoking two years ago and had an untreated hypertension and dyslipidemia. During his daily life for 52 years, he had neither chest symptom nor history of syncope. He also had no SCD in his family.

His electrocardiogram on admission was normal as shown in Fig. 1-B. No abnormal left ventricular wall motion was detected on ultrasonography. Before the cardiac catheterization, angiotensin II receptor blocker (telmisartan 80 mg/day) was administered because of high blood pressure. Creatinine phosphokinase was 191 IU/l and Troponin-T was negative on admission.

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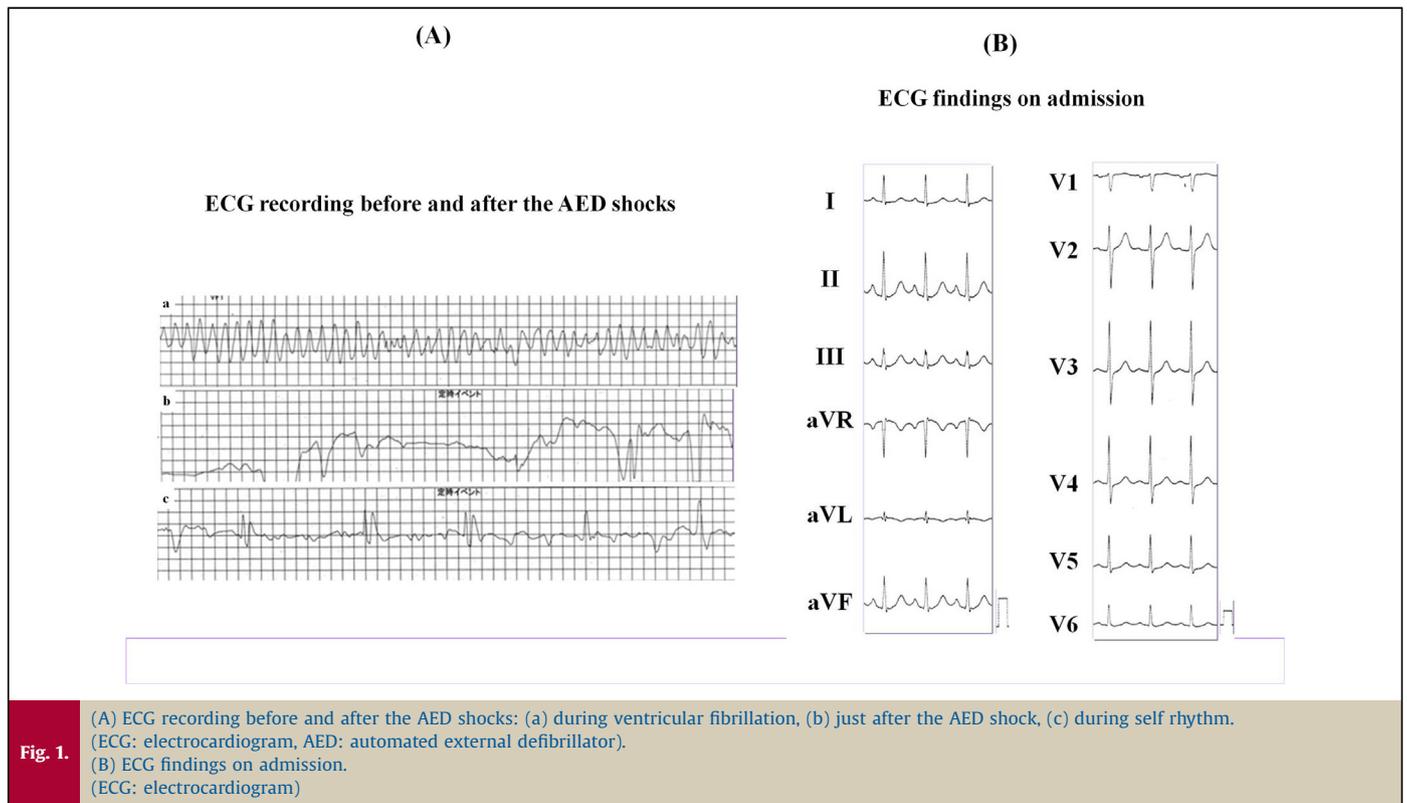


Fig. 1.

(A) ECG recording before and after the AED shocks: (a) during ventricular fibrillation, (b) just after the AED shock, (c) during self rhythm. (ECG: electrocardiogram, AED: automated external defibrillator).
 (B) ECG findings on admission. (ECG: electrocardiogram)

On day 7 of hospitalization, we performed the cardiac thallium adenosine stress scintigraphy. Redistribution on anterior and inferior portions was observed and washout rate was also decreased at the anterior and inferior portions. Washout rate was 24.7%.

On day 10 of admission, we performed coronary angiography at 15:00 without premedication. His left coronary artery showed the moderate stenosis at left main trunk (LMT) and mid left anterior descending (LAD) artery, as shown in Fig. 2-a. Intracoronary administration of 20 μ g acetylcholine (ACh) into the left coronary artery (LCA) disclosed transient 99% stenosis at LMT and mid LAD artery without any chest pain/discomfort or ischemic electrocardiographic changes (Fig. 2-c). After the spontaneous relief of spasm, we administered the 20 μ g ACh into the right coronary artery (RCA). Diffuse distal spasm at segment 4 was documented without any chest symptoms or ischemic electrocardiographic changes (Fig. 2-d). After the administration of nitrate, moderate stenosis (50%) was found at segment 7 (Fig. 2-e), while RCA had no stenosis (Fig. 2-f). We could not detect the coved and saddle back electrocardiographic changes irrespective of every day electrocardiographic recordings. We suspected him as a VF survivor due to silent severe multiple coronary spasm including LMT. We administered abundant vasodilators including two calcium channel antagonists (nifedipine CR 80 mg/day and diltiazem R 200 mg/day) and nitrates (isosorbide mononitrate 40 mg/day and nitrate tape 40 mg/day) instead of telmisartan.

We decided to implant the S-ICD in this patient because he had silent severe coronary spasm including LMT or some possibility of complicating with Brugada syndrome. S-ICD was implanted at Ehime University Hospital 17 days after the event.

After the implantation of S-ICD, we performed the pharmacological spasm provocation tests under the above medications, because we checked whether these medications suppress the LMT spasm or not. Intracoronary administration of ACh 20/50/100/200 μ g into the LCA or 80 μ g into the RCA did not disclose the spasm

(Fig. 3-c and d). Furthermore, intracoronary injection of ergonovine (ER) 64 μ g into the LCA or 40 μ g into the RCA did not show the coronary vasoconstriction (Fig. 3-e and f). Adding ACh 200 μ g after the ER 64 μ g into the LCA disclosed the focal spasm at segment 7 with ischemic ST segment depression in I aVL leads (1.0 mm) and without any chest pain or oppression, as shown in Fig. 3-g. Adding ACh 80 μ g after the ER 40 μ g into the RCA did not show the spasm (Fig. 3-h). After the administration of nitrate, moderate stenosis was recognized at segment 7 (Fig. 3-a and b). We did not perform the percutaneous coronary intervention at segment 7 because of 50% stenosis. No ischemic findings or no chest symptom was observed during the treadmill exercise test (maximal heart rate; 145/min) under the medications. He was discharged in good condition on day 28. We also could not detect the coved and saddle back electrocardiographic changes after the implantation of S-ICD. We performed the pilsicainide test five month after the event. However, neither coved nor saddle back type electrocardiographic changes were documented. We could again perform the cardiac thallium adenosine stress scintigraphy approximately 5 months later. Early up take was improved and redistribution was not found. Washout rate was improved to 33.5% and just decrease at the anterior portion was recognized.

Discussion

In this article, we reported a case of VF survivor due to severe coronary spasm including LMT who had any chest symptom during his daily life. After the implantation of S-ICD, aggressive pharmacological spasm provocation tests disclosed focal spasm at mid LAD artery under the medications. Full medication totally blocked the LMT and RCA spasm. We selected these medications to suppress the next fatal attacks after the implantation of S-ICD. S-ICD implantation is one of the selections in patients with VF survivor due to silent coronary spasm including LMT spasm and multiple spasm. Furthermore, we decided to implant the S-ICD,

Angiographic findings during the acetylcholine testing before medications

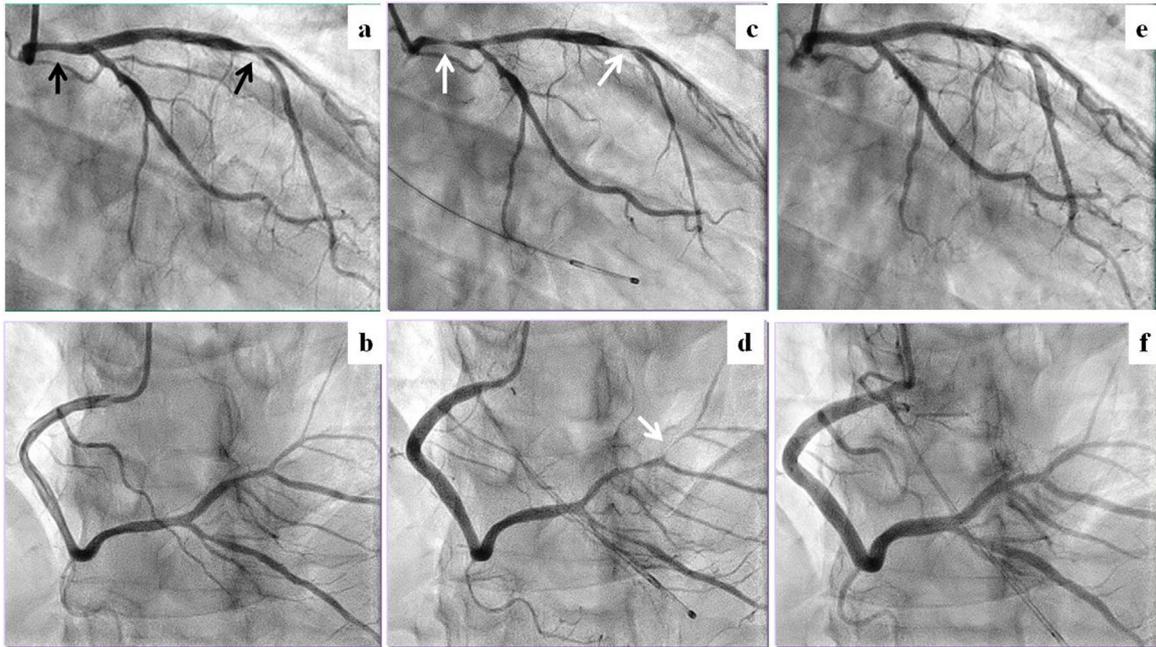


Fig. 2.

(a) coronary spastic portion was found at left main trunk and mid left anterior descending artery (black arrow), (b) no stenosis was observed in the RCA, (c) intracoronary 20 μg ACh disclosed focal spasm at segment 5 and segment 7 (white arrow), (d) diffuse spasm at segment 4 was observed after the administration of ACh 20 μg (white arrow), (e) no significant stenosis was recognized in the LCA, (f) no stenosis was found in the RCA. (ACh: acetylcholine, RCA: right coronary artery, LCA: left coronary artery)

Angiographic findings during pharmacological spasm provocation tests under the full medications

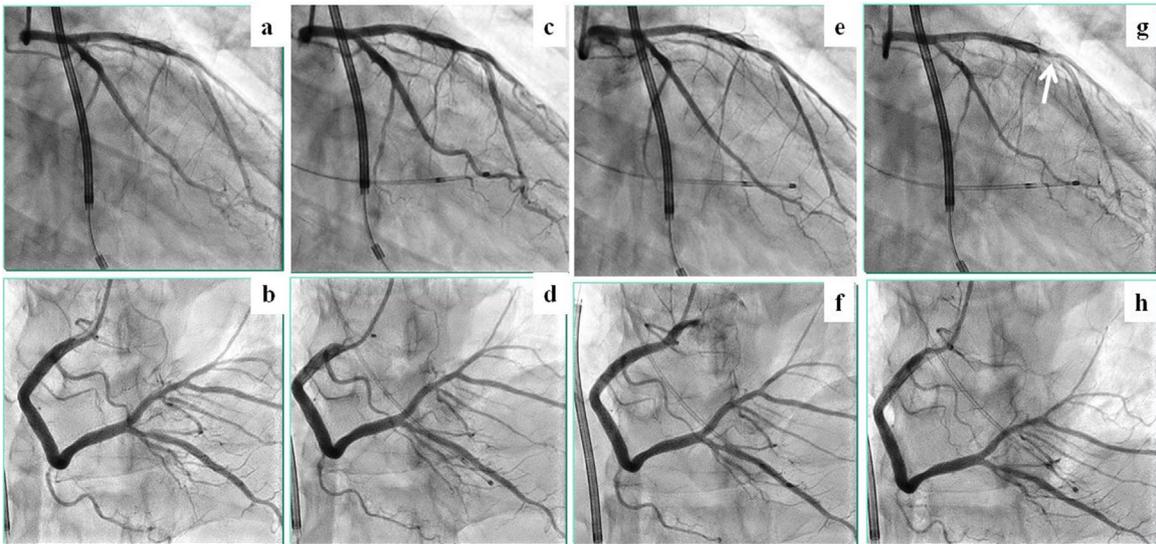


Fig. 3.

Angiographic findings during pharmacological spasm provocation tests under the full medication: (a) no significant stenosis was found in the LCA after the nitrate, (b) no stenosis was observed in the RCA after the nitrate, (c) intracoronary ACh 200 μg into the LCA disclosed no spasm, (d) intracoronary ACh 80 μg into the RCA showed no spasm, (e) intracoronary ER 64 μg into the LCA found no spasm, (f) intracoronary ER 40 μg into the RCA showed no spasm, (g) adding ACh 200 μg after the ER 64 μg into the LCA disclosed focal spastic portion at segment 7 without chest symptom. (white arrow), (h) adding ACh 80 μg after the ER 40 μg into the RCA showed no spasm. (ACh: acetylcholine, ER: ergonovine, RCA: right coronary artery, LCA: left coronary artery)

because we could not totally differentiate the complication with Brugada syndrome irrespective of no documentation of the coved and saddle back electrocardiographic changes or negative pilsincainide test.

According to the Japanese Circulation Society (JCS) guidelines [3], no detailed recommendation was found when we implanted the ICD in patients with VF survivor due to coronary spasm. JCS guidelines for non-pharmacotherapy on cardiac arrhythmias said that ICD implantation in patients with coronary spasm was defined as class IIa (refractory coronary spasm) or IIb (non-resistant coronary spasm) [4]. Appropriate ICD shocks were observed in a quarter of patients with aborted SCD due to coronary spasm after the ICD implantation [5]. Recently, S-ICD demonstrated high efficacy for VF and ventricular tachycardia as well as the intravenous ICD [6]. If the intravenous lead of ICD was implanted in patients with VF survivor due to coronary spasm, it was difficult to retrieve the lead of ICD. In contrast, when we encountered the VF survivor patients due to coronary spasm who had no appropriate ICD shocks under the medications for a long time, we can retrieve the S-ICD device. However, we should select the intravenous ICD but not S-ICD in patients with pulseless electrical activity due to coronary spasm [7].

In this case, provoked LMT spasm was blocked after the abundant medications. If this patient had provoked coronary spasm in the LMT irrespective of aggressive medications, we should dose up medications more such as nicorandil or short acting calcium channel antagonist.

We already reported the usefulness of the pharmacological spasm provocation test under the medications in patients with refractory spasm including aborted SCD [8]. We also reported the clinical usefulness of the sequential spasm provocation tests in patients with coronary spasm [9]. If patients with VF survivor due to coronary spasm had no provoked spasm under the medications, we can select the medical therapy without implantation of ICD in patients with aborted SCD due to coronary spasm. However, if VF survivor patients with coronary spasm had provoked typical spasm by a single ACh or ER test, we should dose up vasodilators and select the implantation of ICD positively.

We selected the S-ICD implantation in this patients because he had spontaneous vasoconstriction of LMT and low dose ACh 20 μ g caused the focal spasm at LMT and mid LAD artery. Furthermore, this patient had silent coronary spasm because he had no chest pain/chest oppression/discomfort until the catastrophic event. Additionally, we could not totally discriminate the complication with Brugada syndrome. In the real world, there may be some patients with silent coronary spastic angina [10]. He is completely

helpless against the aura of proceeding serious fatal event. Appropriate sublingual nitroglycerine use can suppress the serious attacks due to coronary spasm in some patients. However, just sublingual nitroglycerine use did not share in benefit in this case because he had no chest symptom during his daily life. If this case had appropriate ICD shocks here after, we should perform the coronary intervention at mid left anterior descending artery.

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None.

Conflict of interest

The authors declare that they have no conflicts of interest.

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References

- [1] Meisel SR, Mazur A, Chetboun I, Epshtein M, Canetti M, Gallimidi J, et al. Usefulness of implantable cardioverter-defibrillator in refractory variant angina pectoris complicated by ventricular fibrillation in patients with angiographically normal coronary arteries. *Am J Cardiol* 2002;89:1114–6.
- [2] Ahn JM, Lee KH, Yoo SY, Cho YP, Suh J, Shin ES, et al. Prognosis of variant angina manifesting as aborted sudden cardiac death. *J Am Coll Cardiol* 2016;68:137–45.
- [3] JCS Joint Working Group. Guidelines for diagnosis and treatment of patients with vasospastic angina (coronary spastic angina) (JCS 2013). *Circ J* 2014;78:2779–801.
- [4] JCS/JHRS Guidelines on non-pharmacotherapy of cardiac arrhythmias. www.j-circ.or.jp/guideline/pdf/JCS2018_kurita_nogami.pdf.
- [5] Sueda S, Kohno H. Optimal medications and appropriate implantable cardioverter-defibrillator shocks in aborted sudden cardiac death due to coronary spasm. *Intern Med* 2018;57:1361–9.
- [6] Brouwer TF, Yilmaz D, Lindeboom R, Buiten MS, Nordkamp LRAO, Schalij MJ, et al. Long-term clinical outcomes of subcutaneous versus transvenous implantable defibrillator therapy. *J Am Coll Cardiol* 2016;68:2047–55.
- [7] Sueda S, Fujimoto K, Sasaki Y, Habara H, Kohno H. Cardiogenic shock due to pulseless electrical activity arrest associated with severe coronary spasm. *Intern Med* 2018;57:2853–7.
- [8] Sueda S, Kohno H, Miyoshi T, Sasaki Y, Sakaue T, Habara H. Spasm provocation tests performed under medical therapy: a new approach for treating patients with refractory coronary spastic angina on emergency admission. *Intern Med* 2014;53:1739–47.
- [9] Sueda S, Miyoshi T, Sasaki Y, Sakaue T, Habara H, Kohno H. Sequential spasm provocation tests might overcome a limitation of the standard spasm provocation tests. *Coron Artery Dis* 2015;26:490–4.
- [10] Sueda S, Sasaki Y, Habara H, Kohno H. Silent coronary spastic angina: a report of a case. *J Cardiol Cases* 2015;11:166–8.