



LETTER TO THE EDITOR

Primary prevention of sudden cardiac death in Prinzmetal angina: The role of electrophysiology study in risk stratification

**KEYWORDS**

Prinzmetal angina;
polymorphic ventricular
tachycardia;
ICD implantation;
sudden cardiac death;
electrophysiology study

Myocardial ischemia during coronary vasospasm may provoke polymorphic ventricular tachycardia (pVT), which, in some cases, may degenerate into ventricular fibrillation (VF) and cause sudden cardiac death (SCD)^{1,2}. Although spontaneous pVT during coronary vasospasm may well be explained as being triggered by ischemia, its induction during programmed ventricular stimulation (PVS) has not been adequately explored, and its association with the spontaneous occurrence of an arrhythmia remains dubious. We herein present a case of a patient with Prinzmetal angina who presented with spontaneous pVT, and who also had this arrhythmia reproducibly induced during an electrophysiology study (EPS), which was performed while the patient was under adequate medical therapy with no clinical or electrocardiographic evidence of ischemia.

A 74-year-old man with a history of smoking and hypertension presented to his local hospital with typical substernal chest pain, which had resolved spontaneously after 5 min. The patient described recurrent similar episodes of pain during the preceding two months. The pain was related to emotional stress and smoking. His ECG on admission revealed nonspecific ST-T wave abnormalities (Figure 1A), and his cardiac troponin was negative. His echocardiography results were normal. He was admitted to

the cardiology ward and was continuously monitored. Serial testing for cardiac enzymes was negative. During his hospital stay, he experienced recurrent episodes of angina, associated with transient ST-segment elevation followed by non-sustained pVT recorded on telemetry (Figure 1B and 1C).

The patient subsequently underwent coronary angiography, which demonstrated atherosclerotic coronary arteries without fixed critical stenoses. However, spontaneous vasospasm of the left anterior descending (LAD) and the left circumflex (LCx) coronary arteries was documented, which was resolved with intracoronary nitroglycerin infusion (Figure 2).

With the working diagnosis of vasospastic (Prinzmetal) angina, coronary vasodilatory therapy with nitrates and calcium channel blockers was initiated.¹ As he had experienced nonsustained VT, an EPS was recommended and performed while the patient was undergoing medical treatment and after he had been asymptomatic for five days. During the EPS, multiple episodes of non-sustained pVT were easily induced by PVS with use of two ventricular extrastimuli at the right ventricular apex. In addition, two of these episodes became sustained and degenerated into VF, which was electrically cardioverted (Figure 3) with no accompanying ST-T changes on the ECG; his electrolytes were normal. These findings prompted the recommendation for an implantable cardioverter-defibrillator (ICD) that was subsequently implanted. Six months later, the patient sustained another coronary event, albeit without the recurrence of arrhythmia, attributed to coronary vasospasm; a repeat coronary angiography was normal, and the medical therapy was intensified. At the latest follow-up of 18 months, runs of non-sustained VT that did not trigger the device and an episode of electromagnetic interference triggering an ICD shock were documented.

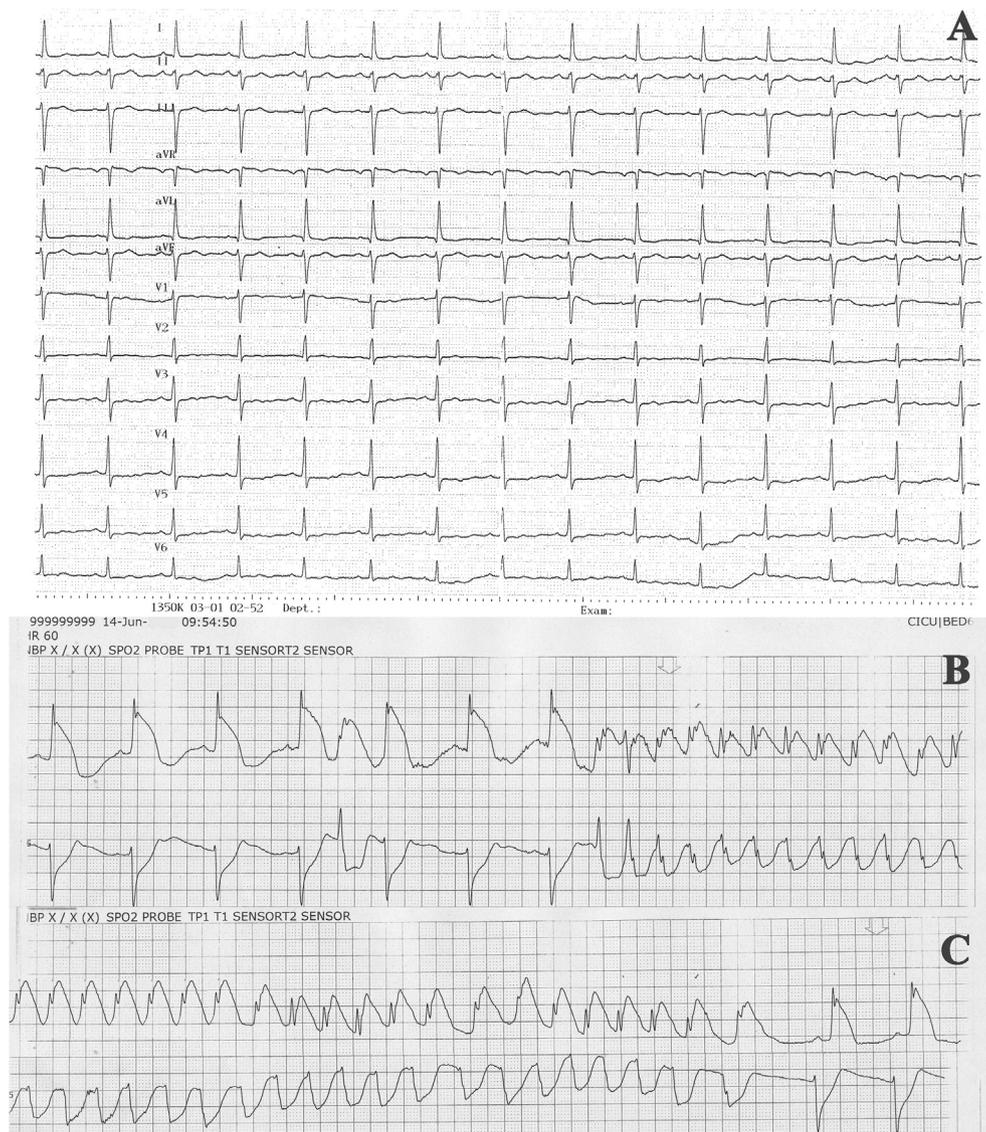


Figure 1 A) Resting 12-lead ECG on admission. B) Rhythm strip showing transient ST-segment elevation during an episode of angina, followed by non-sustained ventricular tachycardia (VT). C) Spontaneous termination of the same VT episode.

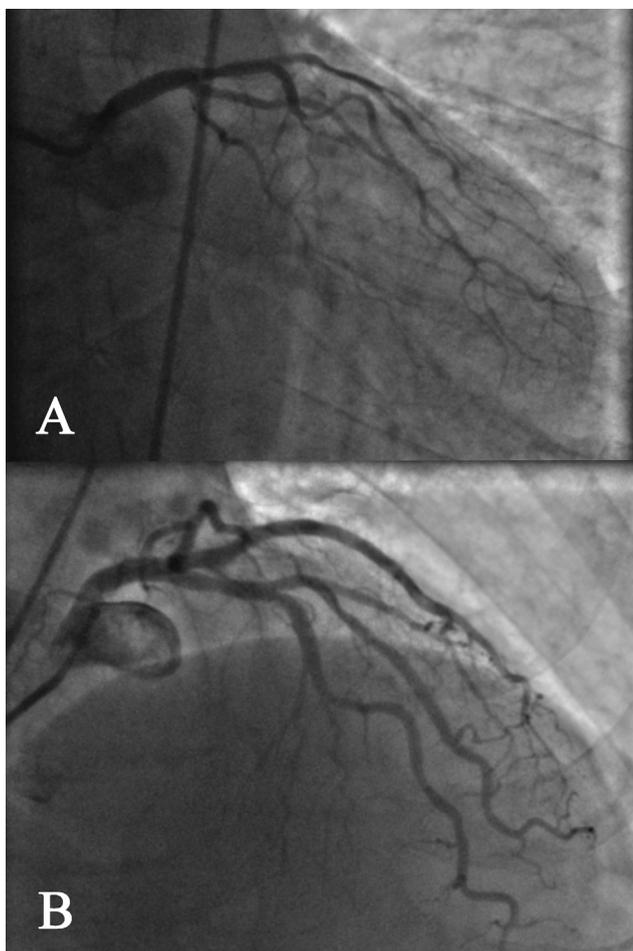


Figure 2 Coronary angiography demonstrating A) vasospasm of the LAD and the LCx (paucity of vessels), B) resolution with intracoronary nitrate infusion. LAD = left anterior descending; LCx = left circumflex.

Patients with Prinzmetal angina usually have a good prognosis with medical treatment.^{1,2} However, some patients experience SCD due to pVT.^{2,3} ICD implantation is a proven therapy for the prevention of SCD.^{4,5} However, little research has been done regarding primary prevention of SCD in patients with vasospastic angina and ventricular arrhythmias and the efficacy of ICD implantation in such patients is currently under investigation.⁶ Most studies have dealt with patients who are cardiac arrest survivors, whereby an ICD seems beneficial (Class I recommendation by the current guidelines),² but with no reference to patients with vasospastic angina.⁵ Regarding the primary prevention of SCD, there is a Class IIa indication for an ICD in patients with an uncommon arrhythmogenic condition whose annual risk of SCD is $\geq 3\text{--}4\%$.⁵ Moreover, inducible pVT in patients without structural heart disease is not included as indication for ICD.⁷

Patients with Prinzmetal angina have high ventricular vulnerability.⁸ However, no study has been done to estimate the precise annual risk for cardiac arrest for these patients, except for retrospective studies with patients already having an ICD. Nevertheless, the risk stratification

of such patients is crucial. Takagi et al. have developed a clinical scoring system as an effort to provide risk assessments and prognostic stratifications for these patients.⁹ However, it is still difficult to distinguish patients who may respond to medical treatment from those at high risk for SCD despite optimal medical treatment, as this distinction cannot be based on the presence or absence of symptoms.¹⁰

We considered our patient as being at high risk for SCD, as he had experienced symptomatic non-sustained VT and had many other independent risk factors for adverse cardiac events.^{6,10} Therefore, to further evaluate him, we performed an EPS. During EPS his arrhythmia was easily inducible without an aggressive protocol and despite undergoing an optimal medical treatment and without developing clinically and electrocardiographically apparent coronary vasospasm-induced ischemia, as evidenced by the fact that the patient experienced no angina during the EPS and there were no ECG signs of ischemia. Based on all these results and findings, we implanted an ICD for prevention of SCD.⁸ The patient had runs of non-sustained VT at follow-up but no sustained arrhythmic episodes to date.

To the best of our knowledge, this is the first reported Prinzmetal angina patient with clinically recurrent episodes of pVT during coronary vasospasm-induced myocardial ischemia, who subsequently underwent an EPS while being asymptomatic on an optimal medical therapy to evaluate for ventricular vulnerability and the need for an ICD. Furthermore, this is the first patient with documented vasospastic angina who during EPS had easily and reproducibly inducible sustained episodes of pVT without an aggressive protocol, despite being clinically asymptomatic under medical therapy and with no ECG signs of silent ischemia, finally having an ICD for primary SCD prevention based on the results of EPS. It appears that some patients with vasospastic angina continue having ventricular vulnerability even when their symptoms are well controlled with medical therapy and an EPS can uncover such patients and guide further therapy. Although inducible pVT has been considered as a nonspecific arrhythmia, especially when induced with an aggressive protocol (e.g., three extrastimuli at the right ventricular outflow tract), a reproducibly inducible pVT with only two ventricular extrastimuli at the right ventricular apex and coupling intervals > 200 ms could not have been lightly discarded as a nonspecific finding in this particular case, as EPS reproduced the clinical arrhythmia, albeit in the absence of ischemia or vasospasm. This case highlights a gap in the literature regarding the management of patients with Prinzmetal angina, as most patients with acute coronary syndrome and sudden death commonly have underlying fixed coronary disease,^{11,12} whereby an EPS may determine arrhythmia vulnerability and predict the subsequent need for ICD therapies.^{13,14} In conclusion, the important role of EPS in risk stratification and the indication for an ICD implantation in patients with Prinzmetal angina needs to be further addressed in future studies.

Conflict of Interest

None.

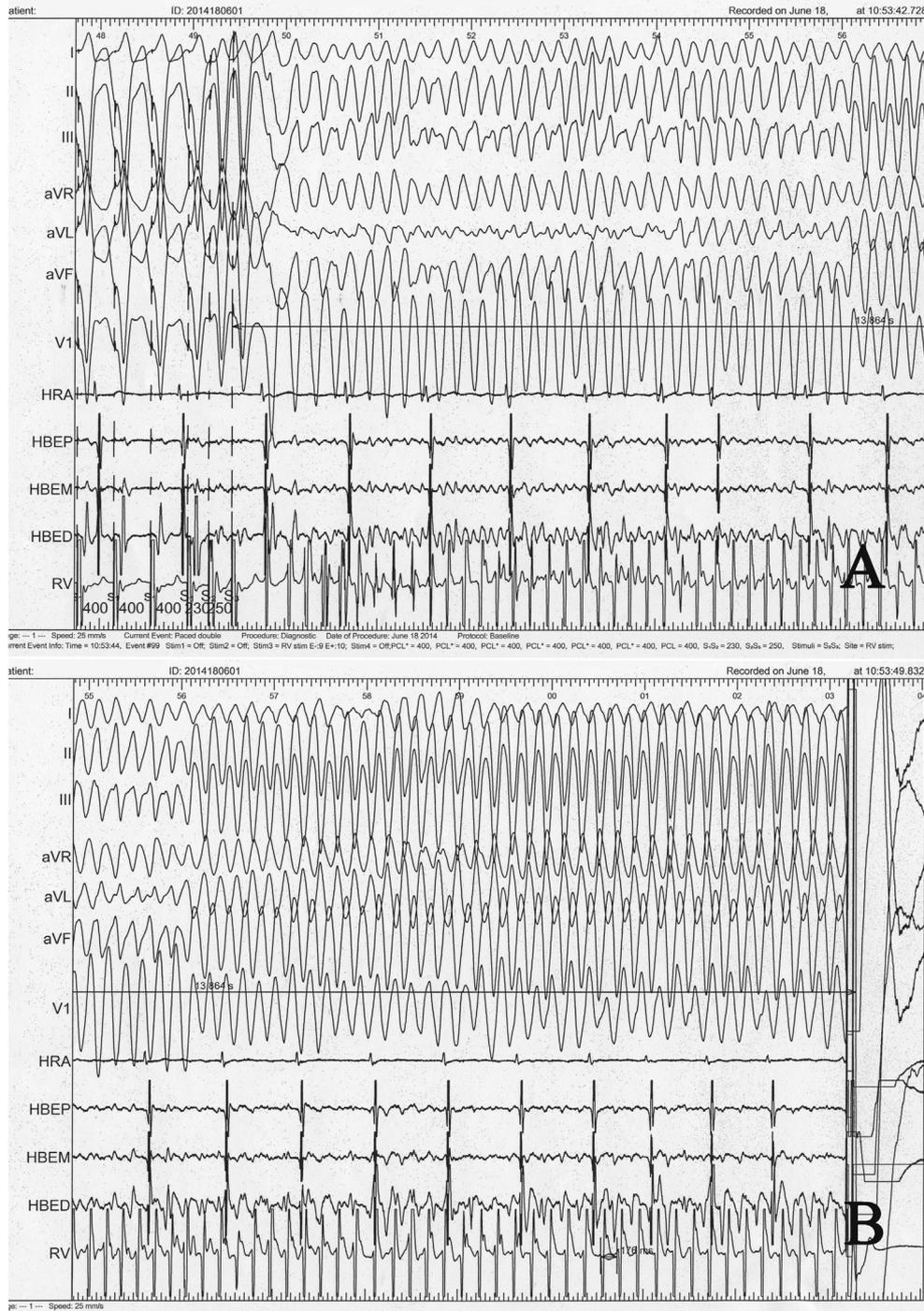


Figure 3 A) Hemodynamically unstable, sustained polymorphic VT, induced during programmed ventricular stimulation at the right ventricular apex with 2 ventricular extrastimuli at a drive cycle length of 400 ms. B) The same VT episode terminated with DC shock.

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