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SPOTLIGHT

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"Spasms in Silence": A case of coronary vasospasm-induced ventricular fibrillation

Deepti Ranganathan MBChB[1] | **Mussa Saad MD**[1] | **Sheldon M. Singh MD**[1,2]

[1]Schulich Heart Center, Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada

[2]Department of Medicine, Temetry Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada

Correspondence
Sheldon M. Singh, Room A222, 2075 Bayview Ave, Toronto, ON M3N 3M5, Canada.
Email: sheldon.singh@sunnybrook.ca

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1 | CASE PRESENTATION

A 56-year-old man presented after an aborted out-of-hospital cardiac arrest. The patient was well before the event, with no reported symptoms of chest pain, palpitations, presyncope, or syncope. His presenting ECG showed episodes of transient repolarization abnormalities (Figure 1). ECGs (Figure 2) and continuous telemetry monitoring (Figure 3), during in-hospital, revealed a Shark fin sign ST-segment elevation commonly associated with multivessel coronary vasospasm,[1] with an initiating premature ventricular complex at 600 ms and runs of nonsustained polymorphic ventricular tachycardia.

The patient had no significant background history or family history of sudden cardiac death. Workup including a toxicology screen, transthoracic echocardiogram, cardiac MR, and procainamide challenge were all normal. A coronary angiography, which was performed demonstrated the absence of flow-limiting coronary artery disease, however

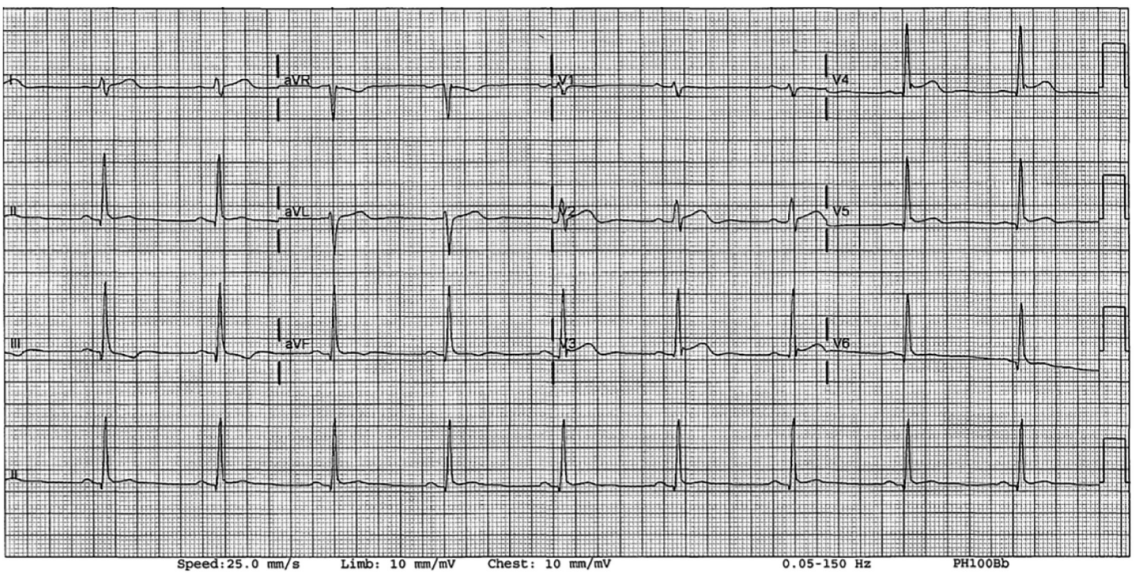

FIGURE 1 Baseline ECG, on admission, showed repolarization changes, most notably in the inferior lead.

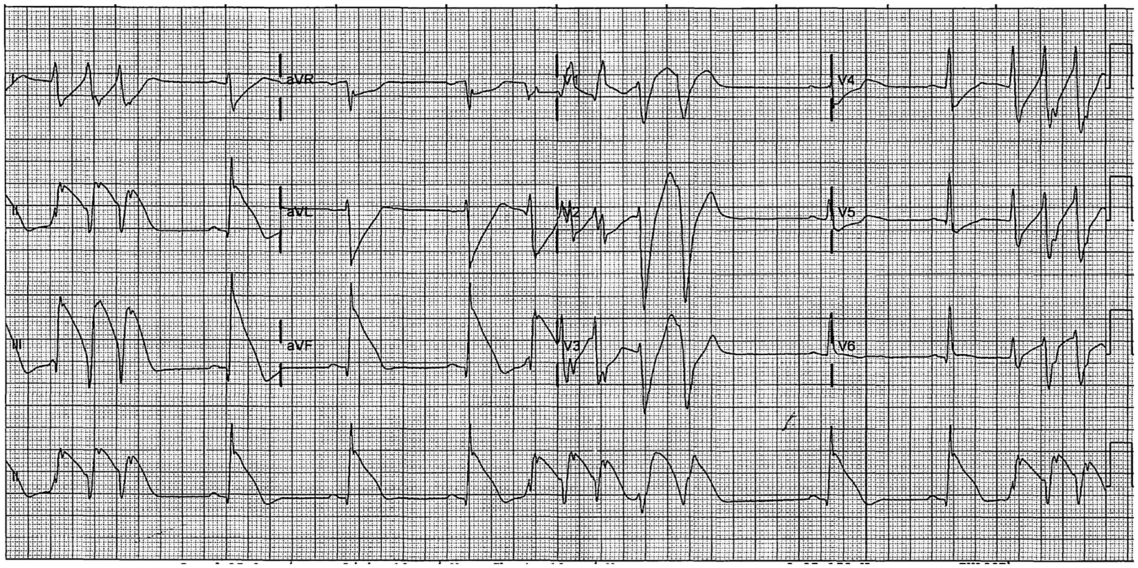

FIGURE 2 ECG showing an episode of transient nonsustained ventricular tachycardia with associated ST changes ("shark fin" pattern, associated with multivessel coronary vasospasm).

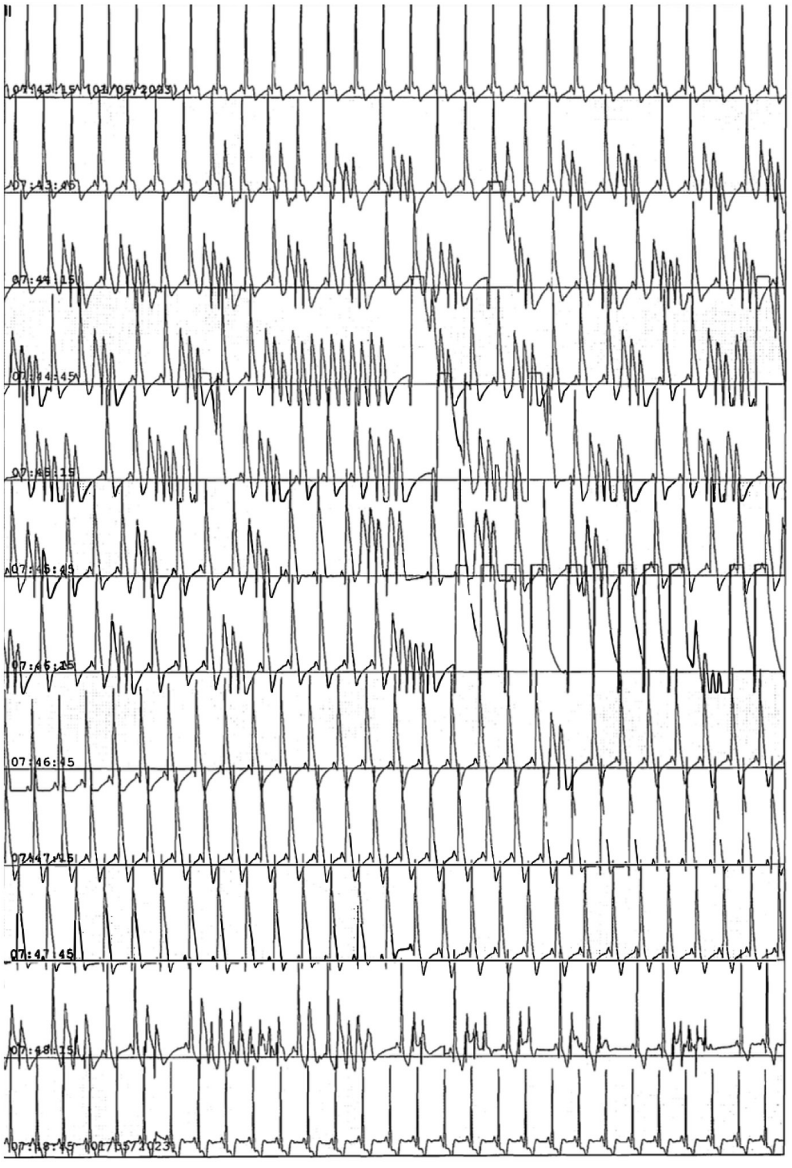

FIGURE 3 Continuous telemetry monitoring in the intensive care unit showed episodes of nonsustained polymorphic ventricular tachycardia associated with repolarization changes.

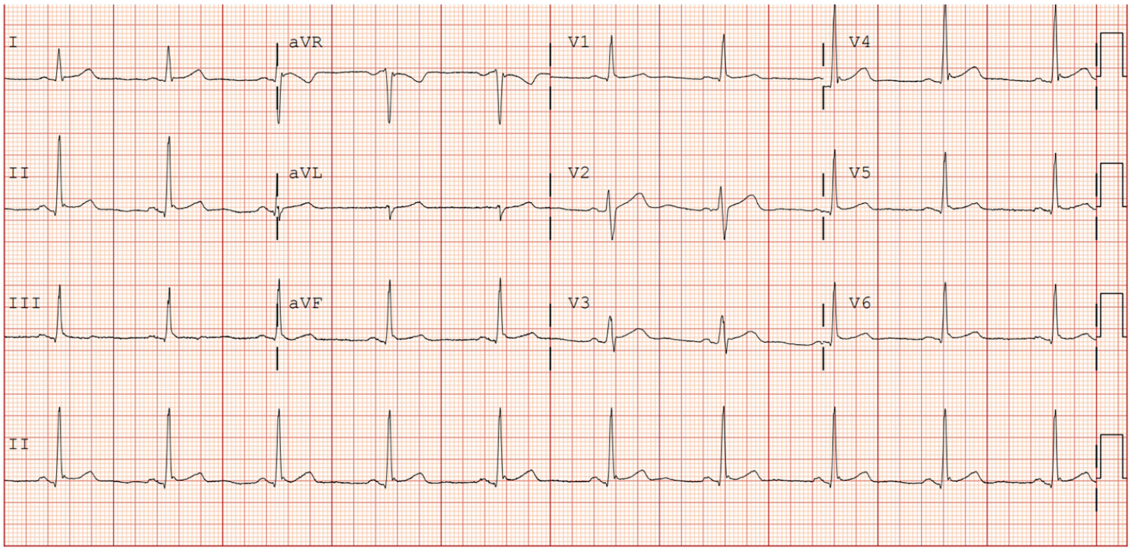

FIGURE 4 ECG on follow-up showed normal intervals similar to the repolarization changes, as initial presenting ECG.

on several coronary injections, showed slow distal flow and raised the possibility of vasospasm. We did not perform provocation testing to confirm the diagnosis, given the lack of intracoronary ergonovine or acetylcholine and the associated complications of repeated ventricular fibrillation in our patient who had full neurological recovery.[2]

The presumptive diagnosis was coronary vasospasm resulting in ventricular fibrillation. Treatment was commenced with a nitroglycerine patch and oral calcium channel blockers (CCB). Given his presentation, we elected to implant a subcutaneous defibrillation for secondary prevention. 9-months postdischarge, he has remained free of recurrent ventricular arrhythmia with a similar ECG as the initial presentation (Figure 4). Additionally, given the possibility of early repolarization syndrome, he underwent a broad sudden death genetic panel, which did not reveal any pathologic genetic abnormalities.

Coronary vasospasm has been reported as the precipitant for ventricular arrhythmia in 7% of patients who suffered an aborted out-of-hospital cardiac arrest.[3] The exact mechanism of coronary artery vasospasm is not fully understood; however, several theories have been elucidated. Vascular smooth muscle hyper-reactivity secondary to Ca^{2+}-mediated myosin light-chain phosphorylation, nitric oxide deficiency because of endothelial dysfunction, oxidative stress, and lifestyle factors, such as smoking, have been implicated. The risk of recurrent arrhythmia in this population is unclear. A prior paper has quoted an annual incidence of sudden death of 6% in patients with coronary vasospasm associated with VF,[3] and experts have suggested that an ICD implantation may be reasonable.[4] To date, there have been no randomized controlled trials comparing ICD and medical therapy with medical therapy alone in survivors of sudden death presumed to be because of coronary vasospasm.

Though rare, coronary vasospasm can result in fatal ventricular arrhythmias, and patients should be assessed for recurrence and risk of sudden death. In a recent study of 34 patients with inferolateral J point elevation, 38% of patients were asymptomatic preceding a VF event and were deemed to have a silent coronary artery vasospasm,[5] and this might be the case in our patient.

Our case highlights that vasospasm may occur in the absence of symptoms and demonstrates the utility of continuous ECG monitoring to assist in the diagnosis of this phenomenon. Although CCB can reduce the risk of recurrence of episodes, it does not eliminate the risk of ventricular arrhythmia, as such in high-risk patients, an implantable defibrillator may be appropriate.

CONFLICT OF INTEREST STATEMENT

Authors declare no conflict of interests for this article.

ORCID

Deepti Ranganathan https://orcid.org/0000-0001-6888-4154
Mussa Saad https://orcid.org/0009-0007-1913-8615

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