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CASE REPORT

BEGINNER

EDUCATIONAL CORNER

Vasospastic Arrest

A Heart-Stopping Case of Prinzmetal Angina

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ABSTRACT

Vasospastic angina is an uncommon cause of cardiac arrest. We describe a patient who presented with sudden cardiac arrest due to severe coronary vasospasm. Telemetry during the event revealed ventricular arrhythmias and asystole followed by spontaneous self-conversion back to normal sinus rhythm. The patient underwent implantable cardioverter-defibrillator therapy. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:611–4)

PRESENTATION

A 60-year-old man presented to the emergency department after being found unresponsive at work. On arrival, he was agitated and hypertensive and was intubated. Physical examination showed a laceration over his right eyebrow. A head computed tomography (CT) scan showed a small subdural hematoma.

LEARNING OBJECTIVES

- Recognize that coronary vasospasm is a cause of transient ST-segment changes, episodes of ventricular arrhythmia, and sudden cardiac arrest.
- Note the supportive role ambulatory cardiac monitoring may play in establishing the diagnosis of coronary vasospasm.
- Implantable cardioverter-defibrillator therapy may be appropriate in vasospastic angina patients who present with life-threatening arrhythmia or sudden cardiac arrest.

While in the emergency department, telemetry demonstrated abrupt QRS-complex widening. An initial electrocardiogram (ECG) showed normal sinus rhythm, a wide QRS, and lateral ST-segment elevations with massive reciprocal ST-segment depressions in leads II, III, aVF, and V_1 to V_4 (**Figure 1A**). A follow-up ECG obtained minutes later showed resolution of these ST changes with only residual lateral T-wave inversions (**Figure 1B**). The admission troponin concentration was 0.08 mg/dl, which peaked 3 h later at 0.12 mg/dl. Admission magnesium level was 2.4 mg/dl, and urine drug screen was negative. An echocardiogram demonstrated normal left and right ventricular size and function without focal wall motion abnormalities. Emergent coronary angiography was deferred due to the subdural hematoma.

MEDICAL HISTORY. The present patient had a history of hypertension and a 40-pack-year smoking history. Further history from the patient's wife revealed that he had had a similar episode 3 weeks previously when

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ABBREVIATIONS AND ACRONYMS

CT = computed tomography

ECG = electrocardiogram

ICD = implantable cardioverter-defibrillator

CMR = cardiac magnetic resonance

RCA = right coronary artery

SCA = sudden cardiac arrest

VF = ventricular fibrillation

VSA = vasospastic angina

VT = ventricular tachycardia

she found him unresponsive at home. He spontaneously recovered from that episode within 15 min. An outside hospital evaluation was reportedly unrevealing for the cause. He was discharged with an outpatient telemetry monitor (MCT 3-lead model, BioTel Heart, Malvern, Pennsylvania), which he was wearing on presentation to the authors' hospital.

DIFFERENTIAL DIAGNOSIS. The differential diagnosis for this patient included acute coronary syndrome, coronary vasospasm, or a primary neurologic process with cardiac manifestations.

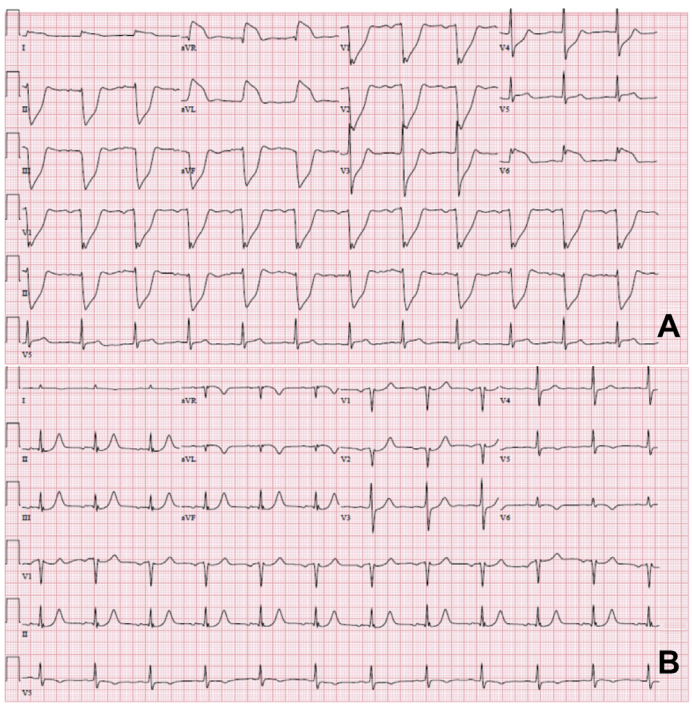
INVESTIGATIONS. Review of the patient's ambulatory telemetry strips correlating with his episode of unresponsiveness revealed sinus rhythm with a wide QRS complex consistent with the morphology seen on the initial admission 12-lead ECG (Figure 2A). This suggested coronary vasospasm and ensuing myocardial ischemia as the inciting event. This was followed by polymorphic ventricular tachycardia (VT) lasting 90 s (Figure 2B), which degenerated into ventricular fibrillation (VF) for 140 s (Figure 2C). VF then organized back to VT (Figure 2D), followed by asystole for 20 s (Figure 2E) before resumption of sinus rhythm (Figure 2F). The timing of his ventricular arrhythmia episodes coincided with the time he was found unresponsive.

A repeated CT brain scan suggested the previously observed subdural hematoma was actually an engorged cerebral vein.

MANAGEMENT. The patient's mental status improved with conservative management, and he was extubated. He was empirically started on oral verapamil, 40 mg twice daily. The following day, he had anginal chest pain. Telemetry during this event demonstrated transient ST-segment elevations in the inferior leads (Figure 3). His symptoms and telemetry changes resolved within minutes, and verapamil was increased to 3 times daily.

Coronary angiography demonstrated a 90% mid right coronary artery (RCA) fixed lesion unresponsive to nitroglycerin and mild, left-sided, nonobstructive disease. A drug-eluting stent was placed in the RCA, and he was started on dual-antiplatelet therapy with aspirin and clopidogrel. Cardiac magnetic resonance (CMR) showed no scar or late gadolinium enhancement. An implantable cardioverter-defibrillator (ICD) was placed prior to discharge for secondary prevention of sudden cardiac arrest (SCA).

FIGURE 1 Initial Electrocardiograms



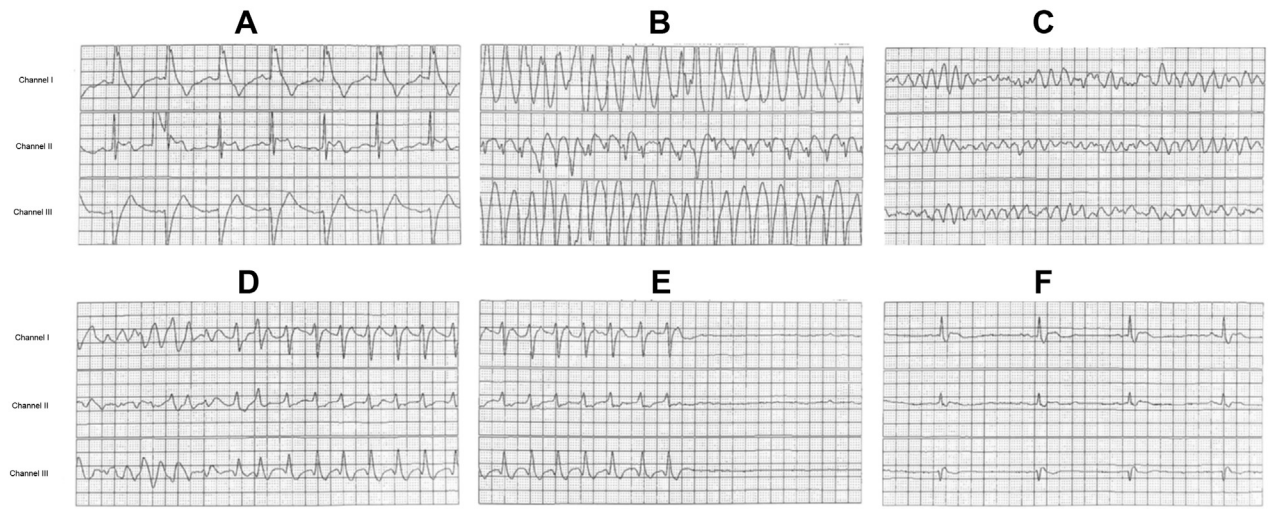
(A) Lateral ST-segment elevations with massive reciprocal ST-segment depressions in leads II, III, aVF, and V_1 to V_4. **(B)** ST-segment changes resolved within minutes of the first electrocardiogram.

DISCUSSION

Vasospastic angina (VSA), first described by Prinzmetal et al. (1) in 1959, due to coronary vasospasm can lead to a number of clinical presentations, including no symptoms or SCA (2,3). Precipitating factors for vasospasm include smoking, magnesium deficiency, physical/emotional stress, hyperlipidemia, sympathomimetic agents, and hyperventilation (2). Classic transient ischemic changes on ECG due to coronary vasospasm include ST-segment elevation, ST-segment depression, and a negative U-wave (3). ST-segment elevations are due to transient transmural ischemia from total occlusion of 1 or more vessels by spasm and are less common than ST-segment depressions. Life-threatening episodes of arrhythmia associated with vasospasm can result from myocardial ischemia (2,4).

The electrocardiographic features captured in this case are notable. The ambulatory recording of an episode of VF organizing into VT followed by asystole with spontaneous return to sinus rhythm has not been featured in the medical literature to date. Our patient's dynamic ECG changes shortly after arrival initially raised concern for a lateral ST-segment elevation myocardial infarction. Coronary angiography later

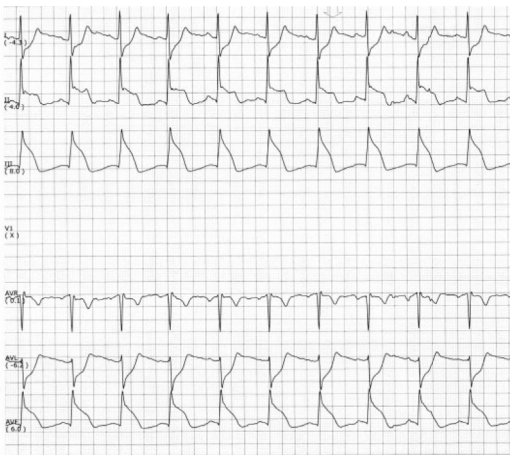
FIGURE 2 Selected Outpatient Telemetry Strips



(A) Sinus rhythm with wide QRS complex. **(B)** Polymorphic VT. **(C)** Ventricular fibrillation. **(D)** Ventricular fibrillation organizing into VT. **(E)** Onset of asystole. **(F)** Spontaneous return to sinus rhythm. VT = ventricular tachycardia.

demonstrated a significant fixed, obstructive lesion in the RCA, but transient ischemic ECG changes, mild cardiac biomarker elevation, normal cardiac CMR, and lack of wall motion abnormalities on echocardiography argued against plaque rupture. Our patient's cardiac monitor findings were helpful in correlating his episodes of altered consciousness with the suspected diagnosis, suggesting SCA due to vasospasm. This helps highlight the supportive role that cardiac monitoring can play in diagnosis (5).

FIGURE 3 Inpatient Telemetry



Transient ST-segment elevations inferiorly.

VSA is generally thought to carry a favorable prognosis (3). However, this patient's presentation with VT/VF and SCA placed him in a high-risk group (4,6). In a large retrospective, observational study, Ahn et al. (6) enrolled VSA patients both with and without SCA across 13 centers in South Korea, examining long-term mortality and VT recurrence risk (6). They demonstrated that the incidence of cardiac death (adjusted hazard ratio: 7.26; 95% confidence interval [CI]: 4.21 to 12.50; $p < 0.001$) and all-cause mortality (adjusted hazard ratio: 3.00; 95% CI: 1.92 to 4.67; $p < 0.001$) was higher in VSA patients with SCA than in those without SCA over a mean follow-up period of 7.5 years. Predictors of SCA included family history of sudden cardiac death, multivessel spasm, and left anterior descending coronary artery spasm. Interestingly, increased age, hypertension, and hyperlipidemia were associated with a lower SCA risk. Our patient had no pertinent family history, but his observed ECG findings and presentation with SCA raised the possibility of multivessel spasm, including involvement of the left anterior descending artery.

Management of vasospasm includes avoidance of precipitating factors such as smoking and application of pharmacologic therapy with calcium-channel blockers. The aforementioned study by Ahn et al. (6) demonstrated a high rate of recurrent VT in patients who presented with SCA despite intensive medical

therapy (32.4 per 1,000 patient-years). They observed a nonsignificant trend limited by small sample size toward lower cardiac mortality in patients who received ICD therapy. Contemporary guidelines support ICD therapy in addition to medical therapy in patients who survive SCA due to vasospasm with a Class IIb recommendation (7). Although more definitive recommendations for ICD therapy in this patient population require further investigation, current medical literature suggests that ICD implantation may be of benefit (6,8,9).

FOLLOW-UP. The patient remained free of anginal episodes or ICD therapy at a follow-up of 3 months after discharge.

CONCLUSIONS

VSA has a wide spectrum of clinical presentations and ECG manifestations and should be considered when transient ischemic ECG changes are observed. Patients with VSA who present with SCA are at higher risk and may benefit from ICD placement in addition to smoking cessation and initiation of calcium-channel blockers.

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