CJC Open 6 (2024) 1351–1354

Case Report

Cardiac Arrests Due to Medically Refractory Coronary Vasospasm in the Acute Cardiac Care Unit

Skyler Eastman, MD,[a] Michael McGregor, MD,[a,b] David Allen, MD, FRCPC,[a,b] Allan Schaffer, MD, FRCPC,[a,b] Kunal Minhas, MD, FRCPC,[a,b] and Malek Kass, MD, FRCPC[a,b]

[a] *Department of Internal Medicine, University of Manitoba, Winnipeg, Manitoba, Canada*

[b] *Section of Cardiology, University of Manitoba, Winnipeg, Manitoba, Canada*

We present a case of medically refractory coronary vasospasm in the right coronary artery (RCA) region that precipitated 5 separate cardiac arrests, in which 2 mechanisms of arrest occurred—ventricular tachyarrhythmia and profound atrioventricular block (AVB) with bradycardia. Traditional interventions, such as high-dose nitroprusside and/or nitroglycerin infusions, and high-dose use of calcium-channel blockers, were tried, with limited benefit. Transient improvement was seen with stellate ganglion block. Ultimately, the vasospasm resolution likely was a result of both the passage of time allowing for healing of injuries, and careful titration of analgesia, sedation, and coronary vasodilation.

A male patient, aged 54 years, with coronary risk factors, including smoking and hypertension, presented to the hospital following a 10-minute, witnessed, out-of-hospital cardiac arrest. Return of spontaneous circulation (ROSC) was achieved with the first defibrillation attempt, via emergency medical services, and the initial rhythm was charted as being "shockable." He was brought to our emergency room, where a post-ROSC electrocardiogram indicated that he had inferior ST-segment elevation (STE), and the cardiac catheterization laboratory was activated.

Coronary angiography was performed, which revealed obstructive epicardial coronary artery disease, with diffuse, 80% stenosis in the second obtuse marginal artery (OM2; Fig. 1A), and with 70% stenosis in the mid-to-distal RCA (Fig. 1B); the prior initially was thought to be the culprit. The patient underwent percutaneous coronary intervention (PCI), with placement of 2 drug-eluting stents to OM2. The patient was admitted to the acute cardiac care unit for further care.

At 24 hours post-PCI, the patient developed frequent recurrent transient significant STE in the inferior leads, with prominent lateral and anterior reciprocal change, which provoked progressive AVB and frequent ventricular ectopy. The pattern of STE was identical to that in the initial arrest. This situation led to ventricular fibrillation–mediated cardiac arrest (Fig. 1D). ROSC was achieved with defibrillation following provision of 1 round of advanced, cardiac life-support.

Lightening of propofol sedation consistently resulted in transient inferior STE and bradycardia. The following day, sedation weaning led to inferior STE, which subsequently provoked cascading AVB and decompensation into transient complete heart block, which resolved with administration of intravenous nitroglycerin (Fig. 2). Unfortunately, the patient continued to have STE in conjunction with any attempts to wean him from sedation in the subsequent days, which was interpreted to represent the occurrence of diffuse coronary vasospasm within the RCA (see Fig. 2). The sympathetic suppressive effects of deep propofol sedation mitigated the incidence of vasospastic episodes.

Despite the escalation of doses of nitrates, the initiation of high-dose calcium-channel blockade, and trials of both nitroglycerin and nitroprusside infusions, the patient continued to have transient STE that ultimately led to a third cardiac arrest, in which the rhythm was pulseless electrical activity in the setting of profound bradycardia (Bezold–Jarisch reflex). ROSC again was achieved after provision of 2 rounds of advanced, cardiac life-support. Another cardiac arrest occurred several hours later, in which ROSC again was achieved. Ultimately, medically refractory coronary vasospasm was the mechanism proposed to be mediating these arrests. Anaesthesia services was consulted for a stellate ganglion nerve block, which was performed successfully and led to transient ~60-hour resolution of vasospasm. The sympathetic modulatory effects of stellate ganglion blocks are known to sometimes be relatively short-lived.[1]

Received for publication January 30, 2024. Accepted August 5, 2024.

Corresponding author: Dr. Malek Kass, University of Manitoba, Y3517-409 Tache Avenue, Winnipeg, Manitoba R2H 2A6, Canada. Tel.: 204-258-1266

E-mail: MKASS@sbgh.mb.ca

Twitter: @SkylerEastman, @kunal_minhas, @KassMalek

See page 1354 for disclosure information.

https://doi.org/10.1016/j.cjco.2024.08.010

Novel Teaching Points

This complex case provides important lessons for critical care cardiology physicians, including the following:

- rare clinical syndromes always should be considered when the clinical picture is dynamic and/or changing;
- coronary v
  asospasm can cause severe cardiac dysfunction and is important to keep in the differential diagnosis;
- a multidisciplinary approach often is required to provide the best care to a complex, critically ill patient; and
- nontraditional measures, such as stellate ganglion block, should be considered for handling vasospastic storm.

Vasospasm reccurred, and a second stellate ganglion nerve block was less successful at preventing STE. Therefore, the patient again underwent coronary angiography at 10 days postadmission, which revealed no suitable PCI targets in the RCA. The 70% stenotic lesion within the RCA was reported to have been reduced to a $< 40\%$ lesion within the 10-day interval (Fig. 1C). Coronary vasospasm, unfortunately, was not stimulated during the angiogram, given the concern that it might cause an arrest.

Approximately 2 weeks later, the patient tolerated cessation of sedation and was extubated. Remarkably, he had excellent neurologic recovery, despite having had 1 out-of-hospital and 4 in-hospital cardiac arrests (Fig. 2C). His vasospasm was treated using long-acting diltiazem, as well as isosorbide dinitrate 3 times daily. An cardioverter defibrillator was implanted for secondary prevention. After 1 month, the patient was discharged home, with excellent physical and neurologic recovery. Transthoracic echocardiography indicated that he had normal biventricular function with no abnormalities.

We present a case of medically refractory coronary vasospasm in the RCA region that precipitated 5 separate cardiac arrests, in which 2 mechanisms of arrest occurred—ventricular tachyarrhythmia and pulseless electrical activity and/or bradycardia. The adrenergic surge caused by the pain associated with post—cardiopulmonary resuscitation injuries is hypothesized to have provoked clinically significant vasospasm in a coronary distribution that was already predisposed to smooth-muscle hypercontractility and vascular-wall hypertonicity, leading to a 2-week period of medically refractory vasospasm.

Coronary vasospasm is defined by severe, reversible, diffuse or focal vasoconstriction that is driven by the effect of vascular smooth-muscle hypercontractility and vascular-wall hypertonicity of narrowing the lumen of normal or atherosclerotic

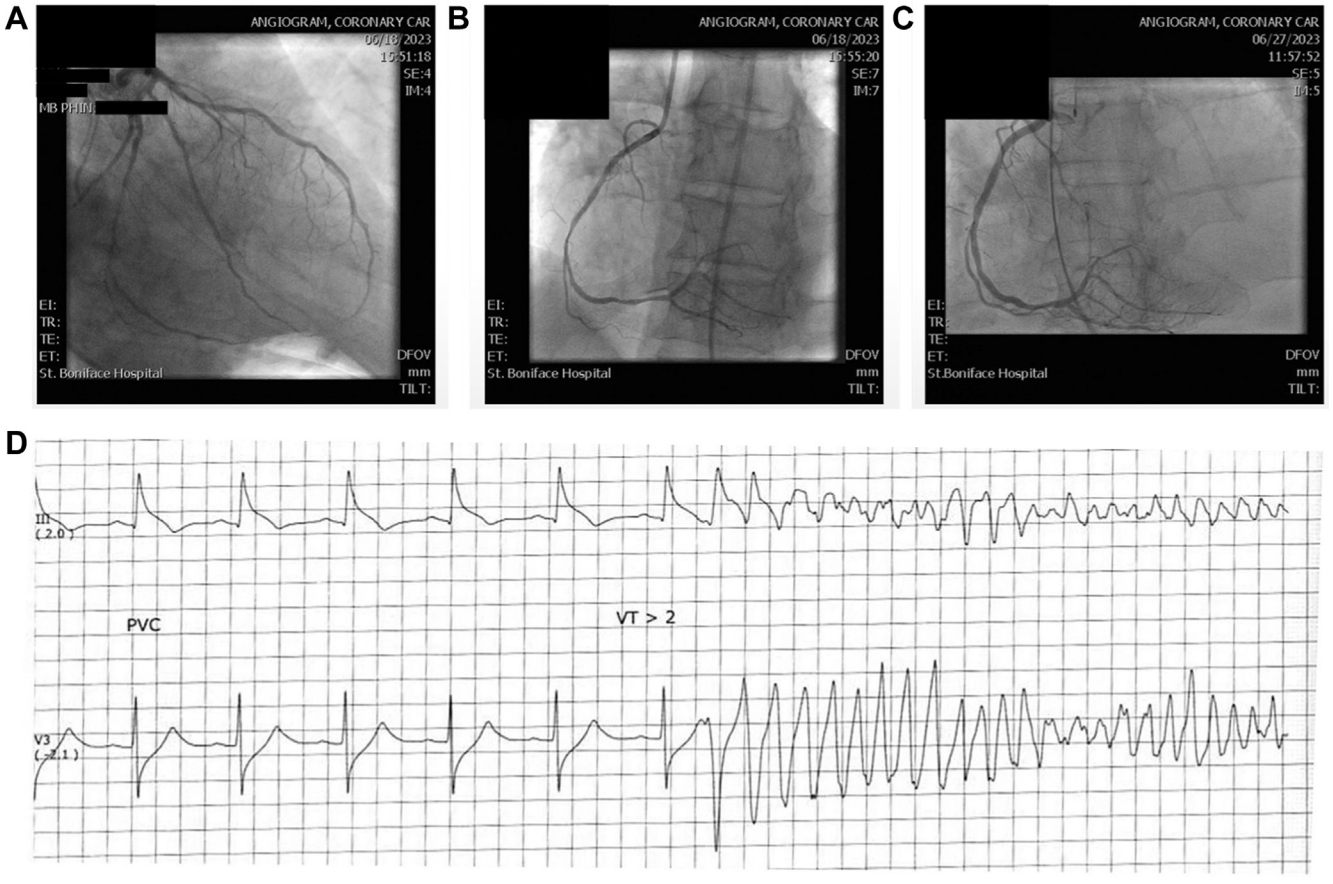

Figure 1. (**A**) Left circulation with emphasis of left circumflex, obtuse marginals (right anterior oblique caudal view). (**B**) Right coronary artery (RCA; 1st angiogram). (**C**) RCA (2nd angiogram, 10 days after the 1st). (**D**) ST-segment elevation induced from RCA-region coronary vasospasm leading to ventricular fibrillation. PVC, premature ventricular contraction; VT, ventricular tachycardia.

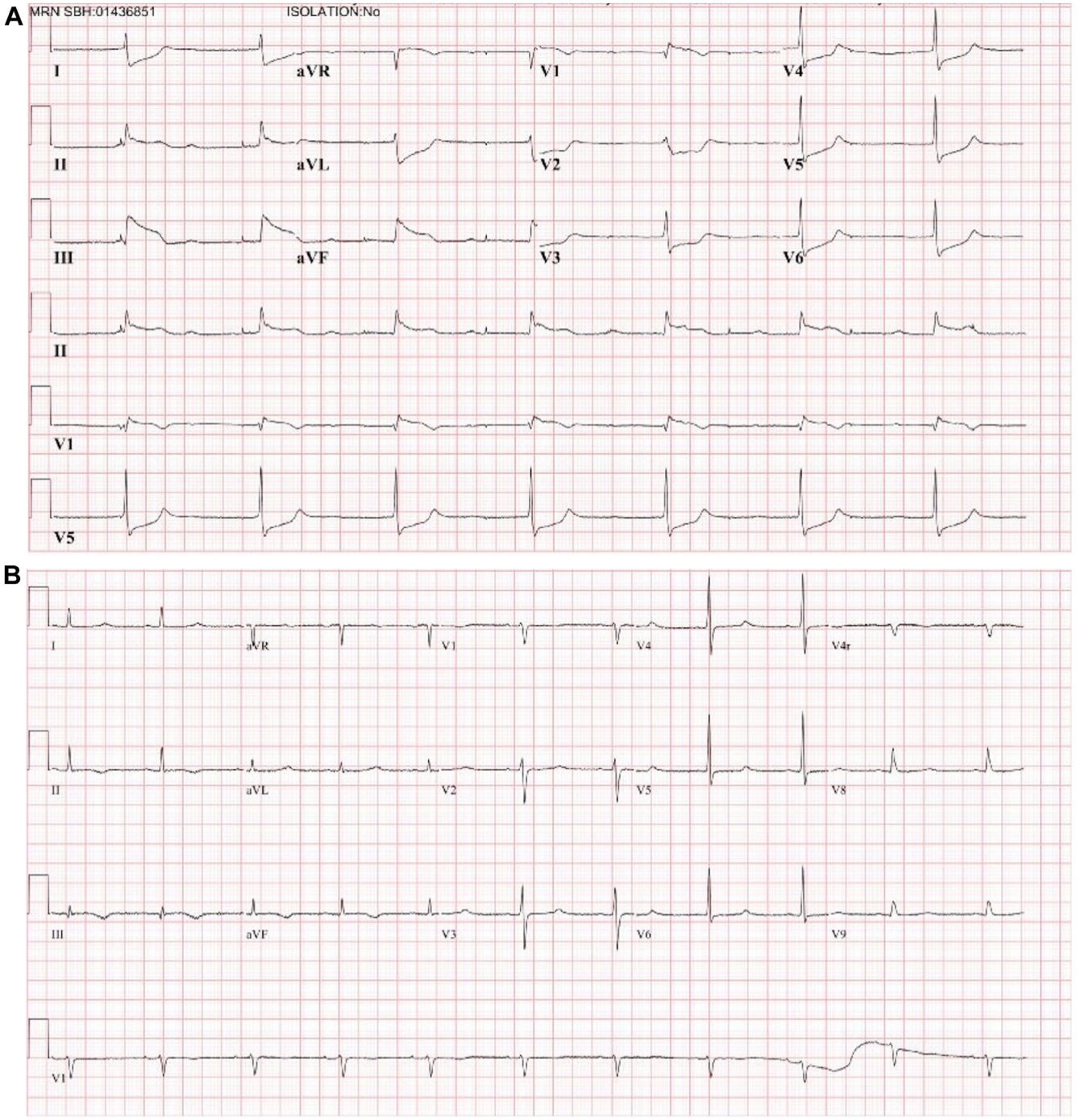

Figure 2. (**A**) Right coronary artery—region coronary vasospasm resulting in ischemia of the atrioventricular node and sinus bradycardia with third-degree atrioventricular block and atria—ventricle (A—V) dissociation. (**B**) Resolution of ST-segment elevation (STE) and atrioventricular block 4 minutes later, with administration of escalating doses of intravenous nitroglycerin. (**C**) Timeline of vasospastic events in hospital. amio, amiodarone; ARDS, acute respiratory distress syndrome; aVF, augmented vector foot; aVL, augmented vector left; aVR, augmented vector right; CCU, cardiac care unit; ICD, implantable cardioverter defibrillator; ISDN, isosorbide dinitrate; NDP-CCB, nondyhydropyridine calcium-channel blocker; PAD, peripheral artery disease; PEA, pulseless electrical activity; PO, per oral; VF, ventricular fibrillation.

coronary arteries, thereby compromising myocardial blood flow and oxygen delivery.[2,3] This cause is recognized as the most common for myocardial infarction with nonobstructive epicardial coronary artery disease.[2,3] Clinically, coronary vasospasm often presents with an anginal chest-pain syndrome. Rarely, coronary vasospasm is severe enough to

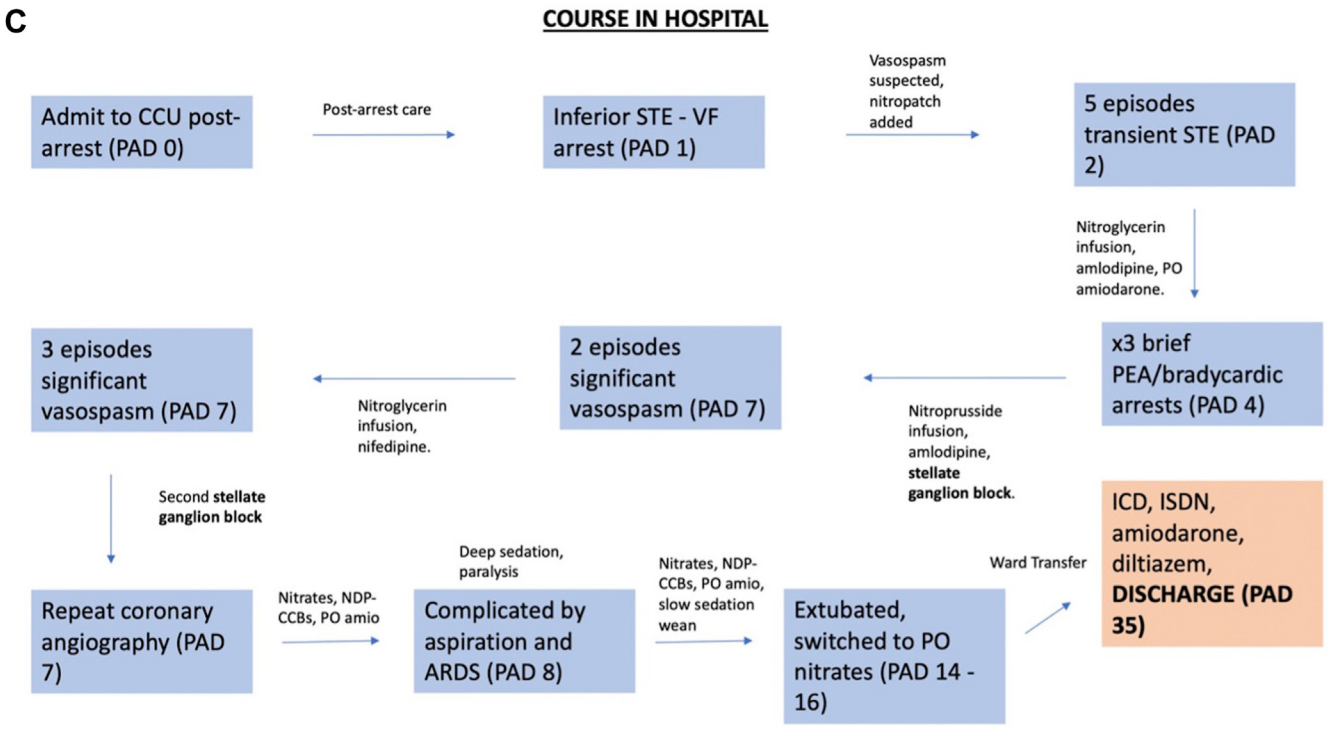

Figure 2. (*continued*).

precipitate cardiac arrest. Here, we present a case of medically refractory coronary vasospasm that resulted in 5 separate cardiac arrests.

The mainstay of therapy is preferential coronary vasodilation with nitrates and calcium-channel blockers. In this case, careful consideration of all treatment modalities, including sedation, stellate ganglion block, and time for healing of injury, was required to ultimately deliver a favourable outcome for the patient. In summary, the arrests were thought to be mediated by vasospasm, and the 80% OM2 lesion that initially was thought to be the culprit lesion was in fact a "red herring."

Ethics Statement

The Research Reported has adhered to the relevant ethical guidelines.

Patient Consent

The authors confirm that patient consent is not applicable to this article, as all the information used is deidentified.

Funding Sources

The authors have no funding sources to declare.

Disclosures

The authors have no conflicts of interest to disclose.

References

1. Fudim M, Boortz-Marx R, Patel CB, Sun AY, Piccini JP. Autonomic modulation for the treatment of ventricular arrhythmias: therapeutic use of percutaneous stellate ganglion blocks. J Cardiovasc Electrophysiol 2017;28:446-9.

2. Pasupathy S, Tavella R, Beltrame JF. Myocardial infarction with non-obstructive coronary arteries (MINOCA). Circulation 2017;135:1490-3.

3. Ford TJ, Rocchiccioli P, Good R, et al. Systemic microvascular dysfunction in microvascular and vasospastic angina. Eur Heart J 2018;39:4086-97.