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MINI-FOCUS ISSUE: INTERVENTIONAL COMPLICATIONS AND THEIR MANAGEMENT

BEGINNER

CASE REPORT: CLINICAL CASE

Vasopressor-Induced Generalized Coronary Vasospasm Presenting as Inferior ST-Segment Elevation in Post-Cardiopulmonary Resuscitation

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ABSTRACT

ST-segment elevation in post-return of spontaneous circulation after cardiac arrest is a major concern for underlying acute coronary syndrome. This case report presents a rare case of vasopressor-induced coronary vasospasm as an underlying cause for this ST-segment elevation with complete reversal of EKG changes after reducing the vasopressor dose. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2019;1:94–8) © 2019 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

This case describes a 47-year-old man with a history of malignant hypertension and end-stage renal disease who had an in-hospital cardiac arrest (CA) (pulseless electrical activity and ventricular fibrillation). The post-resuscitative period was complicated by hypotension requiring vasopressor support with norepinephrine, epinephrine, and vasopressin. Initial electrocardiogram (ECG) post-return of spontaneous circulation (ROSC) did not show acute ischemic changes (**Figure 1**), but on a later repeat, it showed ST-segment elevation in the inferior leads with ST-segment depression in the aVR lead consistent with possible inferior myocardial infarction (MI) (**Figure 2**). Creatine kinase level was elevated to 270 U/l, and troponin-T levels trended up as well, from 0.17 ng/ml (baseline) to 0.66 ng/ml.

LEARNING OBJECTIVES

- To recognize the possibility of vasopressor use during the post-cardiac resuscitation period as a possible cause of ST-segment elevation in post-RSOC ECG.
- To recognize the importance of cautious up-titration of the vasopressors, with recommended bedside titration to maintain the desirable goal and limiting side effects.

DIFFERENTIAL DIAGNOSIS

Post-ROSC ST-segment elevation is usually a sign of an urgent need for cardiac catheterization (CC), especially in the setting of shockable rhythm CA (1). Post-ROSC ST-segment elevation can be attributed to pericarditis, early repolarization, spontaneous subarachnoid hemorrhage, acute ST-segment elevation MI, or as a result of defibrillation electrical shock

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itself (2). CC is mandated in this situation to rule out acute coronary syndrome (1).

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INVESTIGATIONS AND MANAGEMENT. The patient was taken emergently to coronary angiogram, which demonstrated severe diffuse vasospasm (**Figure 3**) in all 3 coronary arteries that was completely reversed with intracoronary vasodilator injection and de-escalation of vasopressors' doses (**Figure 4**). No definite obstructive coronary artery disease was found. A bedside echocardiogram done after completion of the coronary angiography showed moderate global hypokinesis of the left ventricle (LV) and LV ejection fraction of <35% compared with a study done 2 days earlier that demonstrated normal LV wall motion and LV ejection fraction of 50% to 55%, likely stemming from myocardial stunning post-CA. A follow-up ECG demonstrated resolved ischemic changes (**Figure 5**), thus raising the question of an iatrogenic pseudo-MI due to the vasopressors.

In this case, the patient was initially started on norepinephrine, which was eventually up-titrated to 1.5 μg/kg/min, followed by vasopressin up-titrated to a maximum rate of 0.04 U/min and epinephrine up-titrated to a maximum rate of 0.5 μg/kg/min. CC was done approximately 2 h after the patient was on these maximized rates. Based on the CC findings, vasopressin and epinephrine were both discontinued and norepinephrine rate was titrated down over 1 h following CC with improvement in coronary spasm with subsequent ECG no longer showing an ST-segment elevation. Hemodynamic stability was maintained even after discontinuation of vasopressor support.

ABBREVIATIONS AND ACRONYMS

ECG = electrocardiogram

CA = cardiac arrest

CC = cardiac catheterization

ROSC = return of spontaneous circulation

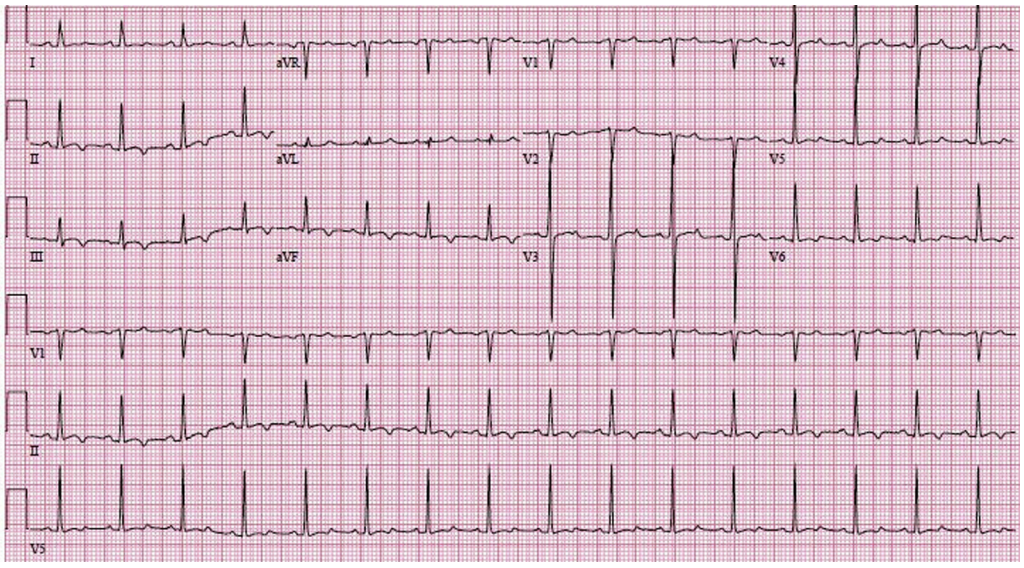
LVEF = left ventricular ejection fraction

MI = myocardial infarction

DISCUSSION

Management of patients post-ROSC covers a wide set of parameters. Obtaining an ECG is essential in forming a differential diagnosis as to the cause of CA (1,2). For patients in whom a cardiac cause is suspected and post-ROSC ECG shows ST-segment elevation (as it did in our patient), studies have shown that early invasive management with CC is needed with possible percutaneous coronary intervention, thus making it an integral part of the post-CA care guidelines (1). A recent study demonstrated that a culprit coronary lesion could be found in up to 81% of patients with ST-segment elevation on post-ROSC ECG (3). This is in contrast to non-ST-segment elevation MI patients in post-CA with no difference in survival with immediate versus delayed angiography (4). Transient ST-segment elevations can also be seen on post-ROSC ECG as a result of defibrillation electrical shock, with different explanations for this electrical phenomenon: direct and transient myocardial injury;

FIGURE 1 First ECG Immediately Post-ROSC With No ST-Segment Elevation



The first electrocardiogram (ECG) was performed immediately post return of spontaneous circulation (ROSC) with no ST-segment elevation.

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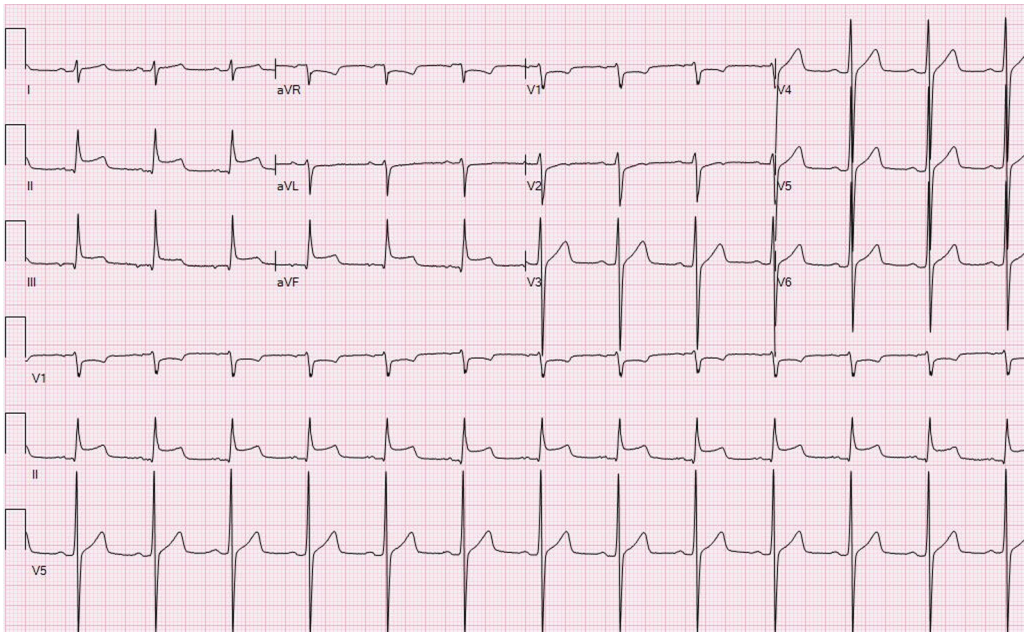
FIGURE 2 Second ECG Post-ROSC With ST-Segment Elevation in Inferior Leads

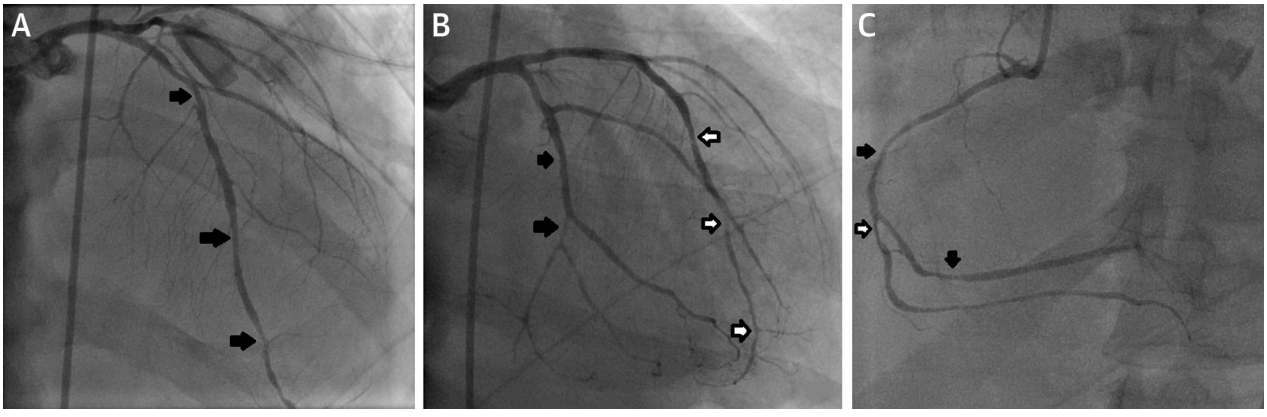


The second ECG was performed post-ROSC with ST-segment elevation in the inferior leads. Abbreviations as in Figure 1.

electrical-induced coronary vasospasm; or sustained depolarization due to microscopic breaks in the cardiomyocyte's membrane (5). However, this phenomenon is likely to occur in the immediate post cardiopulmonary resuscitation episode, whereas our patient had the ST-segment elevation changes show up on the ECG almost 5 h post cardiopulmonary resuscitation.

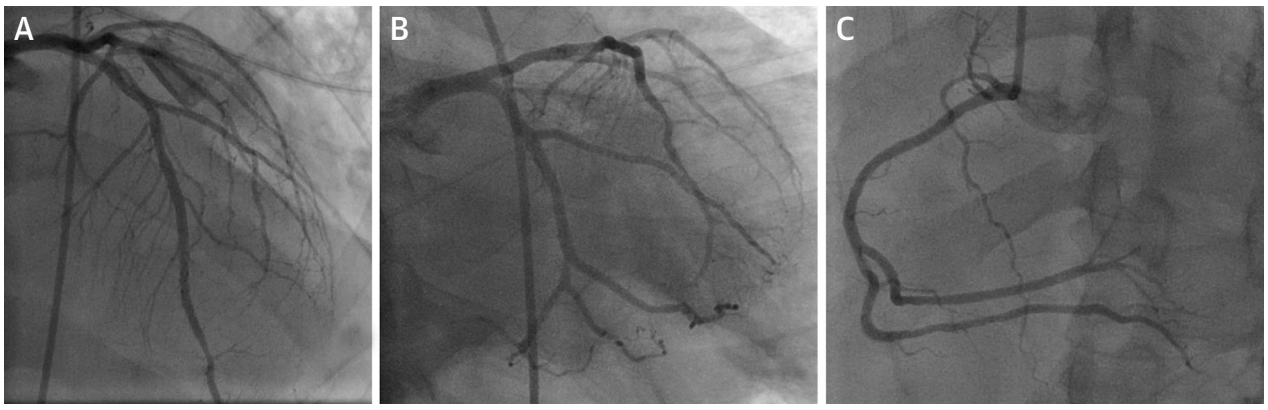
Another aspect of post-ROSC management is maintaining hemodynamic stability and volume resuscitation, which in some cases requires use of vasopressor and/or inotropic agents to combat the

FIGURE 3 Coronary Angiogram Showing Diffuse Coronary Vasospasm Before Injecting Vasodilators or De-Escalating the Vasopressor Dose



(A) Coronary angiogram anteroposterior-cranial view showing diffuse coronary vasospasm in proximal, mid, and distal segments of left anterior descending artery **(black arrows). (B)** Coronary angiogram anteroposterior-caudal view showing coronary vasospasm mid and distal segments of left anterior descending artery **(white arrows),** as well as mid and distal segments of left circumflex artery **(black arrows). (C)** Coronary angiogram left anterior oblique artery-30° view showing proximal and mid-segment vasospasm of the right coronary artery **(black arrows)** and posterior descending artery **(white arrow).**

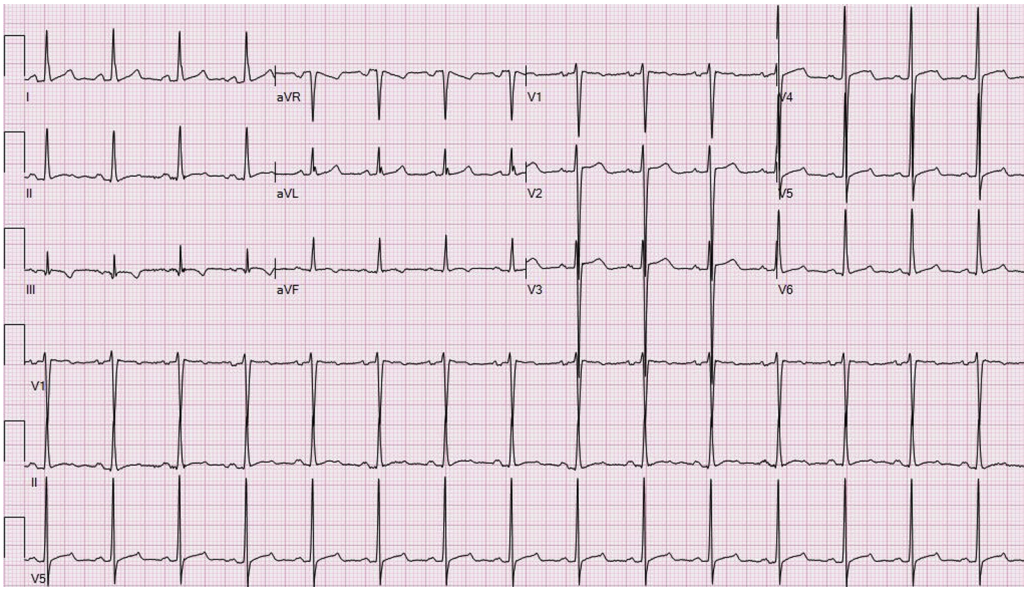
FIGURE 4 Coronary Angiogram After Injecting Vasodilators and De-Escalating the Vasopressors Showing Normal Coronary Arteries



Coronary angiogram after injecting vasodilators or de-escalating the vasopressors showing: **(A)** relief of vasospasm in left anterior descending artery shown in anteroposterior-cranial view; **(B)** anteroposterior-caudal view with the relief of the vasospasm in both left anterior descending artery and left circumflex artery; **(C)** left anterior oblique artery-30° view with relieved spasm in both right coronary artery proper and pulmonary descending artery.

shock. There is no evidence demonstrating the superiority of any vasopressor or inotropic in the post-CA. Norepinephrine is an adrenergic agent that is usually the agent of choice especially in undifferentiated type of shock, with vasopressin, phenylephrine, dopamine, milrinone, or epinephrine being used as secondary agents for refractory shock (6). Although in general, studies have demonstrated improved outcomes with better control of blood pressure post-resuscitation, target goals for mean arterial pressure (MAP) and oxygen saturation have been difficult to define; however, a MAP ≥65 mm Hg and O_2 saturation ≥70% are generally reasonable targets (6). In our patient, the post-ROSC shock state required support with 3 vasopressors to maintain a MAP goal of >65 mm Hg. The overall effects of adrenergic

FIGURE 5 ECG Post Coronary Angiogram Showing Resolved Previous ST-Segment Elevation



Electrocardiogram (ECG) performed post coronary angiogram showing resolved previous ST-segment elevation.

vasopressors on the coronary arteries have usually been found to be vasodilatory and hyperemic in prior studies (7). However, there have been case reports where the use of vasopressor support with adrenergic agents to correct hypotension occurring with anesthesia has been associated with coronary spasm (8). Coronary vasospasm leading to MI has also been seen to occur in a patient receiving pseudoephedrine for sinus congestion (9). However, there is no reported case showing the occurrence of coronary vasospasm and ST-segment elevation due to vasopressor use in post-resuscitative efforts.

A stepwise approach is recommended in the management of post-CA hypotension. First, volume replacement is important to maintain central venous pressure of 8 to 12 mm Hg. Then inotropic and vasopressor agents should be tailored for each patient based on the possible underlying cause for the shock (e.g., cardiogenic vs. distributive) and undesirable adverse effects of medications (e.g., arrhythmia, increased afterload). Medications have to be titrated at the bedside to ensure the intended MAP goal and limiting the side effects (6).

FOLLOW-UP. A follow-up echocardiogram done 2 weeks post code showed a hyperdynamic LV with LV ejection fraction >70% and normal LV wall motion. The patient was discharged from the hospital in stable condition once his other major comorbidities were adequately addressed.

CONCLUSIONS

Post-ROSC ST-segment elevation ECG changes can be attributed to vasopressor-induced coronary vasospasm without underlying obstructive coronary artery disease. Although per guidelines, it is reasonable to rapidly correct hypotension post-ROSC, it is imperative to de-escalate vasopressor use whenever and as soon as possible (6).

The question arises whether an ST-segment elevation on ECG in this setting of high vasopressor dose post-ROSC would lend credence to initially monitoring and de-escalating vasopressor support with serial monitoring of ECG to prevent unnecessary invasive interventions. However, the evidence has shown that in the majority of such cases, a culprit coronary lesion is usually found. In such situations, it may be life-threatening to wait and watch while reducing vasopressor dose, thus putting the onus on early invasive management with CC despite suspicion of vasospasm in all cases with post-ROSC ST-segment elevation as recommended in the guidelines.

AMENDMENT

1. The first ECG was just after ROSC and was not yet on the 3 agents but in the process of uptitration as his mean blood pressure readings were low.
2. ST-segment elevation around 15 min after being max on the 3 pressors.
3. The patient did not receive propranolol.

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KEY WORDS cardiac arrest, coronary vasospasm, return of spontaneous circulation, ST-segment elevation myocardial infarction, vasopressor