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EDITORIAL COMMENT

Are Vasopressors an Enemy of the Coronary Arteries?*

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In patients presenting with ST-segment elevation after cardiac arrest, current guidelines recommend invasive evaluation with cardiac catheterization (1,2).

In this issue of *JACC: Case Reports,* Elkaryoni et al. (3) present the case of a complex medical patient who developed new electrocardiographic (ECG) changes mimicking inferior ST-segment elevation myocardial infarction several hours following an in-hospital cardiac arrest, probably due to iatrogenic coronary spasm. The suggested hypothesis for the ECG changes was the introduction of inotropic and vasopressor agents to treat hypotension after cardiac arrest.

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The use of inotropes and vasopressor agents after cardiac arrest to maintain tissue perfusion is an important component of post-arrest care (1). Coronary spasm as a response to these agents is unusual, because norepinephrine and epinephrine normally produce vasodilation of the coronary arteries through their β-adrenergic actions (4). Coronary spasm is more frequently associated with selective α-adrenergic agents such as ephedrine or phenylephrine (5). In the current case (3), the patient was receiving substantial doses of all these drugs, which were carefully down-titrated after diagnosing coronary spasm. Additionally, the patient was described as suffering from malignant hypertension and end-stage renal impairment. We wonder whether use of a nonselective β-blocker such as propranolol prior to the cardiac arrest could have left the norepinephrine providing only α-adrenergic stimulation. In this case, inotropes such as dobutamine, which have a predominantly β-adrenergic action, may be less likely to precipitate this response.

Guidelines for the management of patients with ST-segment elevation on their post-arrest ECG are broadly written with out-of-hospital cardiac arrest in mind (1,2). Context is always important when making decisions around cardiac catheterization in complex patients after cardiac arrest. In this case, there was no history of chest pain preceding the arrest, and the initial post-arrest ECG did not show any convincing ischemic changes. On the basis of this report, the development of ST-segment elevation on repeat ECG many hours later should prompt a reduction in vasopressor therapy as a first response. A cautious approach should be taken regarding transfer to the cardiac catheterization laboratory. Nevertheless, it is important to consider acute angiography if the ECG injury current persists.

Elkaryoni et al. (3) quickly realized the cause of the coronary narrowing and administered appropriate treatment, confirming the diagnosis of coronary spasm. The case serves to highlight the importance of context in the interpretation of ECG findings. The unusual response of the patient to vasopressors was unexpected, but early adjustment of the therapy de-escalated the situation.

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