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Acute myocardial infarction in cardiogenic shock: beyond coronary anatomy

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Topic(s):
Coronary Artery Disease – Pathophysiology and Mechanisms

Citation:
We present the case of a 76 year-old male patient with a history of arterial hypertension, dyslipidemia, non-insulin-treated type II diabetes mellitus, non-ST-segment elevation acute myocardial infarction (NSTEMI) in 2001 submitted to percutaneous coronary intervention (PCI) of the right coronary artery, and Parkinson's syndrome.

He presented to the Emergency Department due to chest pain with two hours of evolution, associated with elevated blood pressure (BP), vomiting and headache.

On admission he was hypertensive with a blood pressure (BP) of 185/89 mmHg and with an heart rate of 93 bpm. The remaining physical exam was unremarkable.

The electrocardiogram documented sinus rhythm, with a frequency of 92/minute and ST-segment depression in the precordial leads. Laboratory evaluation showed high-sensitivity troponin T of 600 ng/l.

Transthoracic echocardiography (TTE) documented left ventricle with hypokinesia of the infero-lateral wall and of the medium and apical segment of the anterior wall; global systolic function was at the lower limit of normality.

He was admitted with a diagnosis of NSTEMI in Killip-Kimball class I. In the following hours he became hypotensive and with signs of systemic hypoperfusion and repeated TTE documented severe biventricular dysfunction.

In this setting the patient was submitted to emergent coronary angiography that revealed a left anterior descending (LAD) artery with a proximal lesion of 90%; first diagonal of small caliber with an ostial lesion of 80%; right coronary artery with anomalous origin in the LAD, with non-significant restenosis of the stent in the proximal segment, diffuse disease of the mid segment with maximum stenosis of 50 to 70% and distal occlusion; there was contralateral filling of the posterior descending artery. PCI was performed in the LAD, with implantation of a drug-eluting stent.

After the intervention the patient presented with marked blood pressure variation, alternating periods of shock requiring vasopressor support with noradrenaline, with hypertensive peaks (maximum BP of 270/130 mmHg), accompanied by vomiting and headache, requiring nitrate infusion. For this reason, the diagnosis of pheochromocytoma was considered, and abdominal computed tomography (CT) was performed, which documented a right retroperitoneal hypervascular mass suggestive of paraganglioma, with a dimension of 65 x 35 mm. The remaining study also showed a positive urinary metanephrine assay, and PET demonstrated hypercaptation at the localization previously described, compatible with secretory paraganglioma. Alpha-adrenergic blocking therapy was initiated with tansulosin and amlodipine, with control of the hypertensive spikes and remission of paroxysms.

TTE at discharge documented recovery of biventricular function.

Four months after hospitalization the patient underwent excision of the paraganglioma, without complications.
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