Dobutamine stress induced severe prolonged left ventricular dysfunction

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Received 8 February 2008; received in revised form 17 June 2008; accepted 17 June 2008
Available online 15 August 2008

Case presentation

A 36-year-old diabetic woman with terminal renal insufficiency, awaiting renal transplantation, was scheduled for dobutamine stress echocardiography. Arterial hypertension, hyperlipidemia and current smoking status were other cardiovascular risk factors. Rest images were normal (Fig. 1, panel A). Peak stress images showed rapidly extending wall motion abnormalities with akinesia in the left anterior descending (LAD) and right coronary artery (RCA) distributions (Fig. 1, panel B, arrows), severe hypokinesia of the other regions and marked ventricular enlargement suggestive of multivessel disease. The patient had intensive chest pain and the ECG showed a new incomplete right bundle branch block and frequent premature ventricular contractions (PVC) but no significant deviation of the ST segment. Peak blood pressure was 190/130 mmHg. Wall motion abnormalities persisted partially for up to 40 min, essentially in the LAD distribution, despite intravenous administration of 25 mg metoprolol and sublingual nitroglycerine (Fig. 1, panel B, arrows). Blood pressure normalized at recovery (110/75 mmHg). An emergent coronary angiogram revealed no significant lesions (Fig. 1, panel D). Cardiac troponin I increased to 2.1 µg/L without a significant rise in creatine kinase. At one-year follow-up, pharmacological 201-thallium myocardial perfusion scintigraphy with dipyridamole showed normal global and regional ventricular function.

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Discussion

This report illustrates a rare case of prolonged ischemia induced by dobutamine stress in the absence of significant epicardial coronary lesions, possibly due to severely impaired microvascular function and endothelial dysfunction in a diabetic patient with multiple other cardiovascular risk factors. In diabetic patients, myocardial vasodilatory capacity is reduced by these mechanisms, resulting in reduced myocardial flow reserve (MFR) in response to dipyridamole challenge. This is probably the underlying mechanism for ischemia in this case; the reduced MFR cannot offset the augmented oxygen demand induced by the dobutamine stress. Furthermore, the negative perfusion scintigraphy with dipyridamole at follow-up supports this hypothesis (no significant augmented oxygen demand with this pharmacological agent in contrast to dobutamine). Nevertheless the patient did not undergo microvascular testing such as coronary reserve measurements to document reduced MFR.

Other potential, but less probable, mechanisms involved could be coronary spasm of the epicardial coronary arteries (given the diffuse and extensive wall motion abnormalities, several arteries had to be involved and one would expect ST-segment elevation on the ECG), diffuse subendocardial ischemia induced by hypertension (but peak blood pressure was not excessively high and wall motion abnormalities persisted despite rapid normalization of blood pressure), or a Takotsubo cardiomyopathy-like mechanism (but there was no typical apical ballooning).

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.acvd.2008.06.005.

Conflicts of interest

The author does not have any conflict of interest.