CASE REPORT

Acute myocardial infarction after a negative dobutamine stress echocardiogram

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Abstract  Acute myocardial infarction is a rare complication of dobutamine stress echocardiography (DSE). We described angiographic findings of a patient who developed acute inferior ST segment elevation myocardial infarction 2 h after a normal dobutamine stress echocardiogram. The patient failed thrombolysis and underwent coronary angiography, which showed 60% stenosis of proximal right coronary artery with a complex ulcerated lesion and intracoronary thrombus. These findings suggest that myocardial infarction following DSE does not necessarily occur in patients with severe obstructive coronary artery disease. High shear stress may result in destabilization of a complex plaque with subsequent thrombotic occlusion, despite the absence of a flow-limiting lesion at the time of DSE.

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Case report

A 52-year-old man with a history of hypertension, fibromyalgia and rheumatoid arthritis was referred for DSE for evaluation of atypical chest pain. The patient was receiving a diuretic but no statins, aspirin or any cardiac medication. The patient had a normal baseline ECG and normal left ventricular systolic function on baseline echocardiogram. Dobutamine was infused up to 50 μg/kg/min. The patient developed paradoxical sinus node deceleration at a dose 30 μg/kg/min of dobutamine and required 2 mg of atropine intravenously to achieve the target heart rate. The patient had a hypercontractile response to dobutamine in all myocardial segments. His ejection fraction increased from 60% to 75%. There were no wall motion abnormalities, symptoms or ECG changes at peak stress or at recovery. The patient drove back home but developed acute severe chest pain 2 h after completion of DSE. At the emergency department, his ECG showed ST segment elevation in the inferior leads and he received intravenous thrombolysis immediately. Subsequently, he developed ventricular fibrillation and received electrical defibrillation. Chest pain continued and ST segment elevation did not resolve. Therefore, coronary angiography was performed and revealed a complex proximal right coronary artery lesion with 60% luminal narrowing (Fig. 1A). The lesion was ulcerated.
with an overlying thrombus. Left ventriculography revealed inferior wall hypokinesis. The patient underwent angioplasty with successful stent placement in the right coronary artery and no residual stenosis (Fig. 1B). His course was uncomplicated thereafter and was discharged in a stable condition.

Discussion

Acute myocardial infarction is a rare complication of dobutamine stress echocardiography (DSE).\textsuperscript{1,2} Little is known about coronary angiographic findings in patients with this complication. The incidence of acute myocardial infarction during or shortly after DSE is 1 per 2000 according to pooled safety data.\textsuperscript{1} The maximal reported time onset after the study was 20 min. In our case, the onset was 2 h after the study, which is probably related to the time required for an occlusive thrombus to develop over a complicated plaque. The angiographic finding of non-obstructive complicated plaque with an overlying thrombus suggests that the shear stress during high dose DSE may have resulted in destabilization of the plaque with subsequent triggering of the thrombotic cascade. Nevertheless, the occurrence of plaque rupture in this patient may have been coincidental after the DSE. Interestingly, the patient had no signs or symptoms of ischemia during or shortly after DSE. Previous studies have shown that the sensitivity of DSE for diagnosis of single vessel right coronary artery stenosis greater than 50% is as low as 40%.\textsuperscript{3} Although DSE was shown to predict cardiac death, the ability to predict myocardial infarction is suboptimal. Furthermore, most of the myocardial infarctions during follow up do not occur in the vascular distribution that demonstrated ischemia during stress echocardiography.\textsuperscript{4} This was explained by the fact that myocardial infarction often occur due to acute change in an unstable intermediate lesion that may have not been severe enough to cause flow limitation at the time of stress test. This case report demonstrates a clear distinction between anatomical and functional coronary artery disease since the anatomic stenosis of this patient was not functionally significant. The occurrence of myocardial infarction immediately after a normal DSE represents a scenario similar to this reported rarely after exercise or other forms of stress testing. Another possible explanation of our finding is that the DSE test was false negative. Although the inferior wall was well visualized in this study with good imaging quality, it is possible that the concomitant use of a perfusion technique may have detected perfusion defects\textsuperscript{5} since these occur earlier in the ischemic cascade and may be inducible with less severe stenosis. Paradoxical sinus node deceleration at high dose dobutamine has been attributed to increased vagal tone particularly in young adults, or activation of Bezold Jarisch reflex due to inferior wall hypercontractility or ischemia.\textsuperscript{6,7} The proximal location of right coronary artery disease in this case suggests that ischemia in the sinoatrial nodal artery distribution (but not necessarily in the inferior wall) may have been the underlying mechanism of sinus node deceleration. Therefore, high-risk patients who develop sinus node deceleration with
dobutamine may require closer follow up even without demonstrable ischemia in the inferior wall.

References


