Spontaneous Echocardiographic Wall Motion Abnormalities in Variant Angina

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Variant angina, defined as spontaneous angina pectoris associated with transient ST-segment elevation, is usually caused by episodic coronary spasm. At present, coronary artery spasm is a reversible coronary stenosis that limits coronary blood flow under resting conditions. Prinzmetal first described this type of angina pectoris as a distinct entity in 1959.1 Although several hypotheses have been suggested, the precise mechanism for coronary vasospastic disease remains unclear. Natural history of variant angina is heterogeneous. In

Figure 1. Top, 12-lead ECG performed before coronary angiography showing sinus rhythm, left axis deviation and marked ST-segment elevation. Bottom, 12-lead ECG performed after coronary angiography and nitroglycerin infusion. It reveals complete regression of ST-segment elevation.
most of the cases, the prognosis is good; however, it can lead to myocardial infarction (MI), life-threatening ventricular arrhythmias, and sudden death.

A 51-year-old man, a smoker, who had no history of cardiac disease and who was affected by hepatitis C positive-related cryoglobulinemic glomerulonephritis with mild renal failure, was admitted to the emergency department for sudden, typical chest pain that occurred during a routine nephrologic visit. The 12-lead ECG showed a significant ST-segment elevation in leads D1, D2, aVL, and V2 through V6 (Figure 1, top). The physical examination was unremarkable. The patient was referred for urgent coronary angiography, which showed a subcritical stenosis in the middle left anterior descending coronary artery (Figure 2, top, and Movie I), which disappeared after intracoronary nitroglycerin injection (Figure 2, bottom, and Movie II). No coronary angioplasty was performed. After this procedure was completed, the patient’s symptoms were relieved, and the ECG changed with regression of the ST elevation (Figure 1, bottom). Serially obtained blood samples showed a significant increase in cardiac troponin I (maximum, 1.6 ng/mL) and normal electrolyte levels. After normalization of cardiac biomarkers, the patient was discharged from the hospital with instructions for a daily low oral dose of acetylsalicylic acid and 20 mg/day mononitrate isosorbide. The patient underwent a follow-up echocardiographic examination 20 days later. Ventricular function was initially normal without regional wall motion abnormalities (Movies III and IV). Suddenly, during the echocardiogram, the patient complained of spontaneous typical chest pain, which was associated with development of extensive regional wall motion abnormalities involving the apical and septal left ventricular segments (Figure 3, top, and Movies V and VI). The ECG showed ST-segment elevation similar to that observed before coronary angiography. Five minutes after administration of sublingual dinitrate isosorbide, the patient’s chest pain subsided, and the ECG and echocardiographic abnormalities regressed completely (Figure 3, bottom, and Movies VII and VIII). No increase in cardiac biomarkers was detected. The patient was discharged the next day with verapamil (60 mg, 3 times per day) in addition to the previous therapy. He remains free of symptoms after 6 months of this regimen.

This report is the first to detail transient and spontaneous left ventricular systolic dysfunction detected by echocardiogram during vasospastic angina. The mechanism for coronary vasospastic disease is unclear. Coronary artery spasm appears to be related to the presence of atherosclerotic intimal disease.
in the coronary artery because intravascular ultrasound studies revealed atherosclerotic plaques in almost any spastic segment. Furthermore, deficient endothelial nitric oxide production may predispose certain patients to higher arterial basal tone and coronary vasospasm because of unopposed constrictor effects.

Cigarette smoking is the only established risk factor for coronary vasospasm, likely because it affects nitric oxide-mediated regulation of coronary artery tone. The patient had subcritical coronary stenosis and he was a smoker. He also was affected by hepatitis C positive-related cryoglobulinemic glomerulonephritis. Cryoglobulinemia is a systemic vasculitis involving small and, less frequently, medium-sized vessels that can induce endothelial dysfunction with subsequent coronary atherosclerosis. In this patient, smoking, coronary atherosclerosis, and cryoglobulinemia may provide an explanation for the recurrent vasospastic episodes with transient but extensive left ventricular ischemia.

Disclosures

None.

References

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