Myocardial bridging occurs when a band of cardiac muscle overlies an intramural segment of a coronary artery, the intramural segment being referred to as a “tunneled” artery. We presented a case of rare localization of myocardial bridge in the right coronary artery in a patient who was admitted with angina pectoris.

**CASE REPORT**

A 38-year-old man was admitted to our center with exertional chest pain for two weeks. Smoking is the only risk factor for coronary artery disease in our patient. Blood pressure was 140/80 mm Hg and pulse rate was 70 beats/minute. The physical examination was completely normal. Total cholesterol, low density lipoprotein (LDL), cardiac enzymes and troponin-T were also found to be normal. ST segment depression was noted in leads II, III, aVF, V3-V6 on elec-
trocardiography (Figure 1). His echocardiogram showed normal wall motion. The patient underwent selective left and right coronary artery angiography. The left coronary angiography revealed free of critical lesion of the left main artery (LM), the left circumflex artery (LCx) and the left anterior descending (LAD) arteries. The right coronary angiography revealed the typical ‘milking effect’ for myocardial bridge in the right coronary artery (RCA), causing 95% stenosis during systole (Figure 2).

**DISCUSSION**

Myocardial bridge is usually a harmless pathology. Myocardial bridge is generally confined to the mid LAD artery; it is less frequently located in the Cx artery, and is occasionally seen in the RCA. Myocardial bridges are generally expressed clinically in young adult men, in whom typical or atypical chest pain appears, induced by physical exercise and appearing at rest.

Myocardial bridge have been considered to be a benign condition, but several recent studies have demonstrated that their clinical complications can be dangerous, including ischemia and acute coronary syndromes, coronary spasm, ventricular septal rupture, arrhythmias (including supraventricular tachycardia and ventricular tachycardia), exercise-induced atrioventricular conduction blocks, transient ventricular dysfunction and sudden death. Therefore, the prognosis of patients with myocardial bridge is not benign as it was believed to be in the past.

Obviously, medical treatment should be the first and principal strategy. Beta blockers are the suggest treatment of muscular bridge. Interventions should be limited to patients with refractory angina despite medical therapy. In our case, medical treatment was decided.
Consequently, clinical suspicion of a myocardial bridge should be considered in all cases of typical or atypical chest pain, particularly in young patients with a low probability of atherosclerosis who are free from traditional cardiovascular risk factors.

REFERENCES


