Interventricular Septal Aneurysm With Fistulas Developing Into the Right Cavities

To the Editor,

The formation of an aneurysm in the ventricular wall, due to thinning and dilatation, is a relatively common complication after transmural myocardial infarction. It can be associated with the formation of an adjacent intraventricular thrombus, form the substrate of ventricular arrhythmias, or encourage remodeling and dilatation of the left ventricular chamber. On rare occasions, a rupture may occur outside the acute phase of the infarction.

We present the case of a 77-year-old man with an inferior infarction not treated with fibrinolytic therapy in 2006, who subsequently underwent percutaneous revascularization. The patient was admitted to our center due to intermittent thoracic pain episodes lasting several hours and with atypical characteristics which had begun 1 month before. The ECG showed inferior Q-wave infarction, and physical examination revealed systolic-diastolic murmur with thrill more audible at the left sternal border. A transthoracic echocardiogram was performed, which detected an echo-free cavity (An) communicating with the left ventricle (LV) at the basal level of the posterior

Figure 1. A: 2D-echocardiography (4-chamber apical view) where thin interventricular septum can be observed protruding toward the ventricle right (RV) forming an aneurysm (An). B: 2D color Doppler echocardiography (4-chamber apical view) shows the flow from the left ventricle (LV) toward the aneurysm at protosystole (blue), and a turbulent flow between the aneurysm and the right ventricle (RV) (color mosaic).
Letters to the Editor

Figures 2. Intraoperative photograph showing a thin and necrotic interventricular septum, through the paraseptal left ventriculotomy. Note that the aspirator (white cylinder in the upper part of the photograph) is introduced through the interventricular shunt.

a chronic aneurysm. Scarring of the necrotic wall involves the replacement of myocardioocytes by fibrous tissue. This scar tends to be rigid and resistant, at the expense of losing elasticity, which favors dilatation in response to the increase in intracavitary pressures. In this case, the thin wall of the aneurysm had given way and opened toward another cardiac cavity years after the infarction, thus leading to an acquired interventricular shunt. We only know of one other case reported in the literature which described late rupture of the interventricular septum 2 months after an myocardial infarction. Since presentation was slow and progressive, it was possible to administer surgical treatment before the shunt was large enough to produce cardiac failure, shock, or death.

M. del Mar Martínez Quesada,a M. Carmen Durán Torralba,a Ignacio Muñoz Carvajal,b and Gustavo Aníbal Cortez Quirogaa

aUnidad de Cardiología, Hospital Alto Guadalquivir, Andújar, Jaén, Spain
bServicio de Cirugía Cardiovascular, Hospital Reina Sofia, Córdoba, Spain

REFERENCES


Rupture of the myocardial wall may occur in the context of acute infarction, an inflammatory process of the wall (eg, due to a septal abscess or complicated endocarditis on the aortic valve), or after a non-penetrating thoracic injury, but rarely in the scar of