Intramyocardial dissecting hematoma is an infrequent complication of subacute myocardial infarction. Pathological findings consist of a cavity filled with blood, the outer wall of which is the myocardium and pericardium and the inner wall, which faces the ventricular cavity, is part of the myocardium and endocardium. There is scarce information on the subject and the management of these patients continues to be debated. However, there is a certain preference for surgical repair of the defect. Cases in which conservative treatment is a therapeutic option have been reported. We report the case of a patient with an intramyocardial dissecting hematoma after acute anterolateral myocardial infarction who was treated conservatively and achieved a satisfactory outcome in the intermediate-to-long term period.

Key words: Intramyocardial dissection. Myocardial infarction.

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INTRODUCTION

Intramyocardial dissecting hematoma is an infrequent complication of the subacute phase of acute myocardial infarction (AMI) and can also arise as a secondary complication following severe chest trauma. This can be easily differentiated from pseudoaneurysm since it comprises a complete rupture of the myocardial wall contained by the pericardium, whereas an intramyocardial dissecting hematoma is a cavity filled with blood externally contained by part of the myocardium and pericardium. Controversy exists regarding whether surgical repair or conservative treatment is the best course to follow for this condition.

A 58-year-old man, with a history of cigarette smoking and chronic hepatic disease caused by hepatitis C virus (HCV), was admitted to a local hospital with angina-like prolonged chest pain. He was diagnosed as having anterolateral AMI of 12 hours’ evolution; therefore fibrinolysis was not done. A transthoracic echocardiogram which showed extensive anterolateral akinesis and slight mitral regurgitation. No other alterations were detected. He was discharged after 10 days without complications.

One month later, he was readmitted to the center for chest pain with some of the features of pleurocarditis and mild fever compatible with Dressler syndrome. At this time, cardiopulmonary exploration was normal, temperature was 37.8°C, blood pressure 110/75 mm Hg, heart rate 85 bpm, and leukocytosis with shift to the left was observed. The enzyme curve was normal and chest x-ray showed a slight left pleural hemorrhage. Serial
Electrocardiograms showed negative Q and T waves in V1–V4, I and aVL.

Transthoracic echocardiogram at 24 h showed anterolateral and apical akinesis. At the apex and toward the lateral wall we saw a tear in the endocardium and part of the parietal myocardium (Figure 1) which defined the limits of a cavity into which there was a low-velocity passage of fluid during systole from the left ventricle (Figure 2). The neocavity was partially clot-filled with areas of echodense fluid. There was no evidence of communication with the pericardial sac, and the apical myocardial wall appeared to be intact. There was no pericardial hemorrhage.

We interpreted these images as intramyocardial dissecting hematoma and underlying intramyocardial hematoma. Given the patient’s clinical and hemodynamic stability, we continued with serial echography, postponing possible surgery unless the clinical status deteriorated or the lesion got worse.

The serial echocardiographs showed a progressive decrease in mobility of the tear and in flow within the hematoma. At 15 days, a large apical intramyocardial hematoma was visualized (2 × 2.5 cm) without evidence of continuous or communicating flow to the left ventricle.

These findings were confirmed by contrast and non-contrast computed tomography (CT) and magnetic resonance imaging (MRI) which showed an intraparenchymatous hematoma at the apex (Figure 3). Coronary angiography showed occlusion of the middle portion of the anterior descending aorta with akinesis of the mid- and apical segments of the anterior wall. There was no angiographic evidence of significant stenosis in the rest of the coronary tree.

He was discharged 1 month later in a stable condition and had no clinical symptoms 26 months after treatment. Subsequent echocardiography showed no change compared to the last echocardiographic examination prior to discharge.

Fig. 1. Four-chamber apical view showing the endomyocardial tear at the apex of the left ventricle with a partially clotted neocavity.

Fig. 2. Four-chamber apical view with color Doppler showing flow in systole towards the interior of the neocavity.

Fig. 3. A: MRI. B: CT scan. One month after diagnosis an organized intramyocardial hematoma (arrows) was visualized at the apex of the left ventricle.
DISCUSSION

Following cardiogenic shock, cardiac rupture is the second most frequent cause of hospital death in AMI. There is a complete rupture of the myocardial wall in all these cases.

Partial rupture of the myocardial wall in the form of an intramyocardial dissection is infrequent and is generally associated with inferior Q-wave or transmural myocardial infarctions. Few cases have been reported of intramyocardial dissecting hematoma in the context of anterior AMI.

Before the 1980s, the only cases described were found during necropsy. After 1981 the first case was diagnosed in a living patient who underwent surgery. Since then this condition has almost always been diagnosed via a two-dimensional echocardiogram.

We decided on a conservative approach given the good clinical and hemodynamic tolerance, the limited experience in dealing surgically with these patients and the large size of the dissected area.

We believe that spontaneous reabsorption of the intramyocardial hematoma can occur not only in patients with an intact left ventricular endocardium, but also in the presence of an intramyocardial tear with passage of fluid from the left ventricle to the necocavity, as in our patient.

Pliam et al. reviewed 15 cases of intramyocardial dissecting hematoma. Eight were diagnosed post mortem after having received conservative treatment and dying a few weeks later. The remaining seven patients were diagnosed while alive: five were treated surgically with good short- to mid-term results, and the other two received medical treatment (one survived, one died). In 1998, Nilkanth reported a case of intramyocardial dissecting hematoma treated by surgical resection with good long-term survival.

Following this, Nakata et al., Jiménez et al. and Vargas-Barron et al. described three cases of intramyocardial dissection treated conservatively with good results and acceptable mid-to long-term survival.

Until now it was believed that the prognosis in intramyocardial dissection was fatal in the short- to mid-term in patients who did not undergo surgery. However, in the light of recent studies, and our own experience with the present case, we believe that given the lack of experience in most cardiac surgery services in treating this disorder, patients with clinical and hemodynamic stability can be treated conservatively, with echocardiography or CT/MRI monitoring to confirm the complete clotting of the dissecting hematoma and the absence of other mechanical complications.

Nevertheless, we believe that the form of therapy should be based on each patient’s condition and the amount of experience each surgical center has in managing this type of disorder.

REFERENCES