Spontaneous Retraction of an Intramyocardial Dissecting Hemorrhage and Multiple Left Ventricular Thrombus Formations in Subacute Myocardial Infarction and Antiphospholipid Syndrome: A Case Report with Long-term Follow-up

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This report describes a 68-year-old patient with a subacute myocardial infarction and antiphospholipid syndrome. He developed an intramyocardial dissecting hemorrhage involving the left ventricular apex and multiple left ventricular thrombus formations, documented by contrast echocardiography and magnetic resonance imaging. By use of transthoracic echocardiography, spontaneous retraction of the dissecting hemorrhage could be detected. Severe coronary 3-vessel disease was successfully treated by coronary artery bypass grafting. During follow-up of 16 months, the dissecting hematoma could not be detected. Under initiation of anticoagulant treatment with Coumadin, the patient was in stable clinical condition and improved in New York Heart Association class from III to II. The pathophysiology, diagnosis, and management of this potentially highly lethal complication is reviewed. (J Am Soc Echocardiogr 2006;19:578.e5-e8.)

A 68-year-old man was admitted in June 2004 to our department of cardiology with progressive rest chest discomfort and severe dyspnea during the last week. Since 1996 he had had severe left ventricular (LV) dysfunction; he was symptomatic only for exertional dyspnea (New York Heart Association [NYHA] class III) in optimized medical therapy for heart failure. He did not agree to undergo heart angiography and the genesis of heart failure remained unclear. Former echocardiograms were also unavailable. In the last month he had been admitted to a neurologic department with a generalized epileptic crisis. Brain magnetic resonance imaging demonstrated multiple older ischemic lesions in the region of the right medial cerebral artery.

On admission, clinical evaluation revealed a noncooperative patient with bilateral basal pulmonary edema, a systolic heart murmur (2/6), gallop rhythm, apical lift, an enlarged liver with hard inferior border, and no peripheral edema. The patient’s systolic blood pressure was 100/70 mm Hg with a regular pulse of 96/min.

Electrocardiogram showed sinus rhythm and complete left bundle branch block. Multiple ventricular extrasystoles and couplets were seen on Holter recording.

Laboratory evaluation was notable for a positive antiphospholipid syndrome (anticardiolipin antibodies and lupus anticoagulants positive), and revealed also the following pathologic findings: platelet 62,000/mm³, international normalized ratio 1.7, creatine kinase 461 U/L, creatine kinase-MB 115 U/L, troponin T 0.03 µg/L, aspartate aminotransferase 515 UI/L, alanine aminotransferase 550 UI/L, lactate dehydrogenase 906 U/L, y-glutamyltransferase 309 UI/L, total bilirubin 4.9 mg/dL, and factor VIII 399%. Chest radiograph demonstrated heart enlargement and biphasal pleural effusion.

Transthoracic echocardiography (TTE) was performed and showed a severe LV enlargement, severe dysfunction, and spontaneous echocontrast in the LV and left atrium. Ejection fraction (EF) was 10% (biplane Simpson’s method). TTE revealed also a large echo-free neocavitation (50 × 35 mm) involving the LV apex, clearly delimited by endocardium toward the middle portion of ventricular cavity. In the basal inferior wall a thrombus formation was detected (Figure 1, and video 1 A). Real-time myocardial contrast echocardiography with Sonovue
demonstrated no opacification in the apical neocavitation with a noncomplete perfusion of the endomyocardial border (Figure 1 and video 1, B). A filling defect was also shown for the basal thrombus formation.

There was right ventricle failure with systolic pulmonary pressure of 58 mm Hg.

These findings suggested an intramyocardial dissecting hemorrhage formed after subacute myocardial infarction. Cardiac magnetic resonance imaging revealed an apical intramyocardial hematoma that involved the major part of the LV apex, clearly delimitated by endocardium toward LV cavity confirming the diagnosis of a dissecting hemorrhage (Figure 2 and video 2).

In the following week, clinical and laboratory signs of right heart failure ameliorated with medical therapy and the patient was asymptomatic for rest dyspnea and chest pain.

The patient underwent coronary angiography that demonstrated severe 3 coronary vessel disease with indication to coronary aortic bypass graft. The patient was considered too sick, as a result of very low LV EF, to tolerate operation for evacuation of the hematoma.

Coronary artery bypass grafting (CABG) intervention was programmed based on coronary angiography findings without additional tests to prove viability. Follow-up TTE 2 weeks after admission showed spontaneous retraction of the dissecting hematoma and persistent basal thrombus formation (Figure 3 and video 3).

After another week, on the fourth week of hospitalization, a repeated transthoracic study was performed that demonstrated, despite heparin therapy, 3 masses in LV, compatible with recent thrombus formations, one affixed on the interventricular septum, one in the LV apex, and the third in the basal inferior wall (Figure 4 and video 4).

Heart surgery intervention (CABG) was performed successfully with CABGs placed to the right coronary artery and left anterior descending artery. The patient was hemodynamically stable throughout his postoperative recovery. Coumadin therapy was initiated and the patient could be discharged. At 1 year and 4 months after hospital dismissal the patient was in a stable condition in NYHA functional class II.
DISCUSSION

The intramyocardial dissecting hematoma is an unusual form of cardiac rupture of the LV wall. A review of 106 cases of cardiac rupture described a hemorrhagic dissection in 9% of them. It has been reported as an infrequent complication with poor prognosis after chest trauma, myocardial infarction, heart operation, and even spontaneously. Diagnosis is often difficult and in most of the cases by operation or postmortem. Prompt recognition, therefore, is important. To our knowledge, about 20 cases are described in the literature with a very poor survival (approximately 10%) in the medically treated group compared with a good prognosis (100% survival) in the group of patients who were treated surgically. Differential diagnosis must be made with pseudoaneurysm by establishing integrity of epicardium and with intracavitary thrombosis by identifying the endomyocardial layer surrounding the neoformation and associated wall movement.

This report describes an unusual intramyocardial dissecting hemorrhage and multiple LV thrombus formations complicating subacute myocardial infarction and ischemic cardiomyopathy with a very low EF in a patient with an antiphospholipid syndrome. Original images of TTE and myocardial contrast echocardiography and cardiac magnetic resonance imaging of the LV are demonstrated and could determine the diagnosis. Severe 3-vessel disease indicated CABG intervention and due to high risk for cardiac operation because of very low LV EF, conservative treatment of the intramyocardial dissecting hematoma was planned. Preoperative echocardio-

Figure 3 Transthoracic echocardiography follow-up 4-chamber (A) and 2-chamber (B) views: severe left ventricular dysfunction and spontaneous echocontrast, no evidence of intramyocardial dissecting hemorrhage. B, Residual basal thrombus formation. Video sequences of all displayed images are available online.

Figure 4 Transthoracic echocardiography follow-up 5-chamber (A), 2-chamber (B), and parasternal long- (C) and short- (D) axis views. Multiple thrombus formations: apical, anteroseptal, and inferobasal. Video sequences of all displayed images are available online.
raphy showed retraction of the dissecting hematoma. This was interpreted as spontaneous drainage of the dissecting hematoma through the endomyocardial layer into the LV cavity. The combination of severe LV dysfunction with development of spontaneous echocast and an unfavorable thrombogenic status of an antiphospholipid syndrome resulted in multiple LV thrombus formations. These were obtained Initially and developed progressively after spontaneous retraction of the dissecting hematoma. With initiation of Coumadin therapy after CAGB intervention the patient was in stable clinical condition and could progress from NYHA classification III to II over 16 months of follow-up. This favorable evolution with a demonstrated spontaneous retraction and healing of an intramyocardial dissecting hemorrhage is discordant with the majority of previous observations and case reports.

Spontaneous resolution of intramyocardial hematoma of the LV has been reported in 3 cases. One case has been reported that partially resolved spontaneously. The first successful surgical correction of this condition was reported by Stewart et al in 1981. Other reports followed. The only reported long-term survival of postinfarction intramyocardial dissecting hematoma treated nonsurgically has been that of Drozdz et al and in an intermediate long-term period that of Galache Osuna et al.

REFERENCES