Intramyocardial Spontaneous Hematoma Mimicking an Acute Myocardial Infarction

Leonarda Galiuto, MD, PhD; Luigi Natale, MD; Gabriella Locorotondo, MD; Sabrina Barchetta, MD; Maria Mastrantuono, MD; Antonio G. Rebuzzi, MD; Lorenzo Bonomo, MD; Filippo Crea, MD

An 80-year-old man affected by rheumatoid arthritis and chronic myeloid leukemia who was a smoker and hypertensive was admitted to the coronary care unit for first ST-elevation myocardial infarction. The diagnosis was suspected on the basis of the presence of chest pain associated with ST-segment elevation in leads III and aVF and a cardiac troponin T level of 0.30 ng/dL. At coronary angiography, performed 2 hours after pain onset, a 50% stenosis of the proximal right coronary artery with a translucent filling defect that suggested a parietal thrombus at the cardiac crux was found (see online-only Data Supplement Movie I); the left coronary artery had diffuse atherosclerosis in the absence of significant stenosis. Therefore, no interventional procedure was performed and the patient was treated with aspirin and clopidogrel. The echocardiogram performed soon after coronary catheterization showed an expansive lesion in the basal segment of the inferior wall characterized by echodensity intermediate between blood and tissue (Figure 1A). Myocardial contrast echocardiography documented that the expansive lesion was characterized by a reduced and irregular microvascular network compatible with both hemangioma[1] and intramyocardial hematoma[2] (Figures 1B and 2A; online-only Data Supplement Movies II and III). Magnetic Resonance Imaging further clarified the diagnosis. In fact, T2-weighted fat-suppressed images documented the presence of a hyperintense expansive lesion compatible with both hemangioma and intramyocardial hematoma (Figure 2B), but first-pass perfusion imaging showed marked signal loss in the lesion (Figure 3A), with no enhancement within the first minute (Figure 3B; online-only Data Supplement Movies IV and V). At late enhancement, a mild subendocardial perfusion defect remained only in the midventricular inferior wall. This pattern is typical of intramyocardial hemorrhage, whereas the contrast enhancement pattern of hemangioma is characterized by slow peripheral contrast uptake.[3]

When the diagnosis of intramyocardial hematoma was established, antiplatelet and heparin treatment was discontinued, but the next day, an echocardiogram documented a large intraventricular mural thrombus that dissolved rapidly within 2 days of heparin treatment. The patient was then screened for possible thrombotic and hemorrhagic disorders, and in fact, antiphospholipid antibodies were found to be elevated in his blood (cardiolipid antibody IgM 16 U/mL). These antibodies can be associated with both rheumatoid arthritis and chronic myeloid leukemia and are responsible for thrombosis and hemorrhagic disorders.[4] Thus, in this patient, antiphospholipid syndrome was a likely cause of both intramyocardial hemorrhage presenting as ST-segment elevation acute myocardial infarction and intraventricular mural thrombosis.

The patient has been discharged with aspirin and oral anticoagulant treatment (online-only Data Supplement Movies VI and VII). A control echocardiogram performed 6 months after the acute events documented that the expansive lesion remained confined to the basal segment of the inferior wall, in the absence of parietal thrombi. The patient is currently doing well.

Disclosures

None.

References

1. Ramasubbu K, Wheeler TM, Reardon MJ, Dokainish H. Visceral pericardial hemangioma: unusual location for a rare cardiac tumor. *J Am Soc Echocardiogr*. 2005;18:981.
2. Vargas-Barron J, Romero-Cardenas A, Roldan FJ, Molina-Carrion M, Avila-Casado C, Villavicencio R, Martinez-Sanchez C, Lupi-Herrera E, Zabalgoitia M. Long term follow-up of intramyocardial dissecting hematomas complicating acute myocardial infarction. *J Am Soc Echocardiogr*. 2005;18:1422.
3. Moniotte S, Geva T, Perez-Atayde A, Fulton DR, Pigula FA, Powell AJ. Cardiac hemangioma. *Circulation*. 2005;112:e103–e104.
4. Gomez-Puerta JA, Cervera R, Espinosa G, Aguilo S, Bucciarelli S, Ramos-Casals M, Ingelmo M, Asherson RA, Font J. Antiphospholipid antibodies associated with malignancies: clinical and pathological characteristics of 120 patients. *Semin Arthritis Rheum*. 2006;35:322–332.

From the Institute of Cardiology (L.G., G.L., S.B., M.M., A.G.R., F.C.) and Institute of Radiology (L.N., L.B.), Catholic University of the Sacred Heart, Rome, Italy.

The online-only Data Supplement, consisting of Movies I through VII, is available with this article at http://circ.ahajournals.org/cgi/content/full/116/15/e371/DC1.

Correspondence to Leonarda Galiuto, MD, PhD, FACC, Institute of Cardiology, Catholic University of the Sacred Heart, Policlinico A. Gemelli, Largo A. Gemelli, 8, 00168 Rome, Italy. E-mail lgaliuto@rm.unicatt.it

(*Circulation.* 2007;116:e371-e372.)

***Circulation* is available at http://circ.ahajournals.org** **DOI: 10.1161/CIRCULATIONAHA.107.699793**

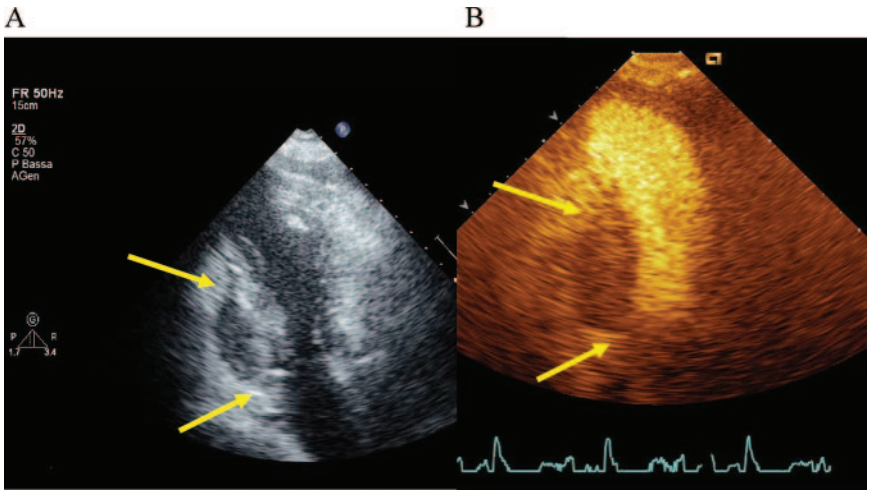

Figure 1. Two-dimensional (A) and myocardial contrast echocardiography (B) images in 2-chamber view. An expansive lesion of the basal segment of the inferior wall is evident in both images between arrows. A, The lesion appears to be of reduced echodensity with tissue characteristics intermediate between blood and tissue. B, The lesion appears darker than normal adjacent myocardium and thus significantly hypoperfused.

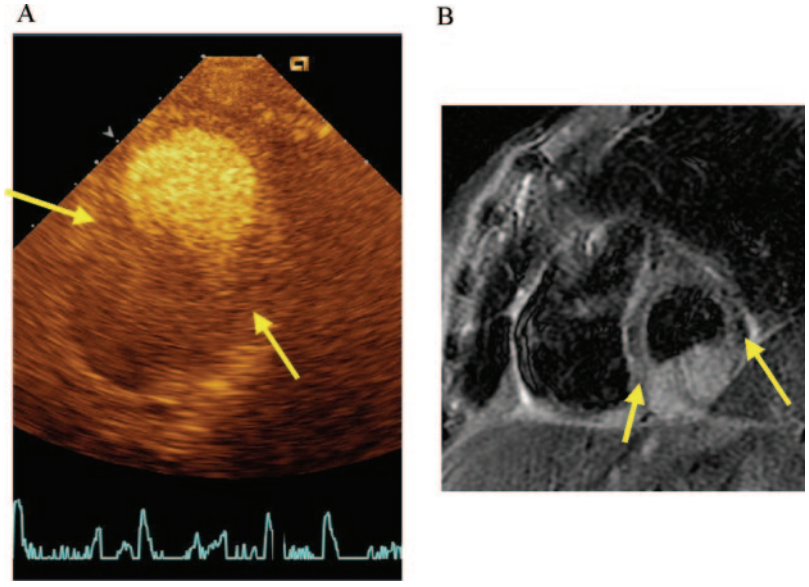

Figure 2. Comparison between short-axis view obtained with myocardial contrast echocardiography (A) and ECG-gated short-T1 inversion-recovery (STIR) short-axis magnetic resonance imaging (B). In the region corresponding to the hypoperfused expansive lesion of the inferobasal myocardial segment (A), magnetic resonance imaging (B) showed a large hyperintense expansive lesion in the same left ventricular wall.

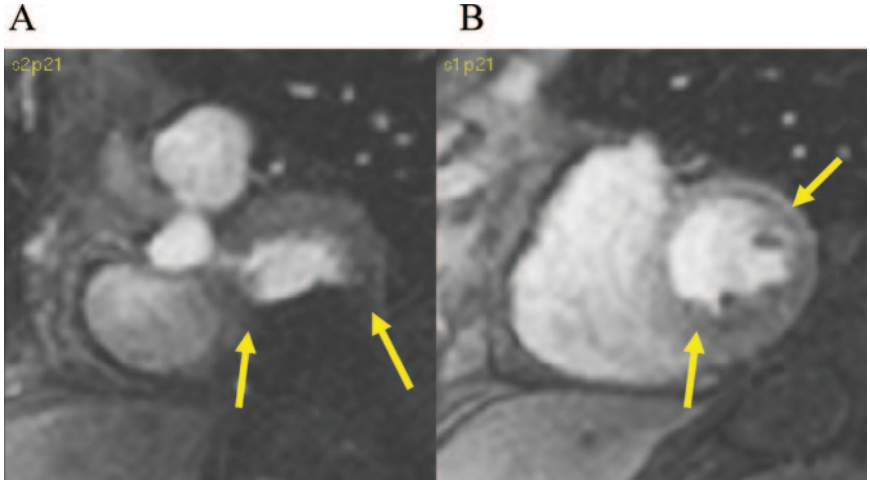

Figure 3. Magnetic resonance imaging rest perfusion study at basal (A) and midventricular (B) level showing a large dark expanding area in the left ventricular inferobasal wall (A, between arrows) and a mild subendocardial perfusion defect in the midventricular inferior wall (B, between arrows). Dark area is due to accumulation of hemosiderin.

Intramyocardial Spontaneous Hematoma Mimicking an Acute Myocardial Infarction

Leonarda Galiuto, Luigi Natale, Gabriella Locorotondo, Sabrina Barchetta, Maria Mastrantuono, Antonio G. Rebuzzi, Lorenzo Bonomo and Filippo Crea

Circulation. 2007;116:e371-e372
doi: 10.1161/CIRCULATIONAHA.107.699793
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539