Left Ventricular Thrombosis in Ulcerative Colitis

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Key Words

Left ventricular thrombosis · Ulcerative colitis

Abstract

Left ventricular thrombi usually occur in the setting of an acute myocardial infarction, left ventricular aneurysm, or dilated cardiomyopathy. In the absence of ventricular wall motion abnormalities, they are rare. This report describes a patient with ulcerative colitis in whom two-dimensional echocardiography revealed a left intraventricular mass. Thrombosis in ulcerative colitis is a serious condition and can occur in a very young population. This case also shows that left ventricular thrombi can occur in the active setting of ulcerative colitis.

Introduction

Left ventricular thrombi usually occur in the setting of an acute myocardial infarction, left ventricular aneurysm, or dilated cardiomyopathy. In the absence of ventricular wall motion abnormalities, they are rare. This case report describes a patient with ulcerative colitis in whom two-dimensional echocardiography revealed a left intraventricular mass.

Case Report

A 39-year-old Caucasian male patient with a history of ulcerative colitis presented to the hospital with a complaint of nausea, vomiting, abdominal pain and bloody diarrhea. He had been taking prednisone and mesalamine but had been noncompliant. On physical exam, he was alert and oriented with a temperature of 97.3°F, a pulse of 108 bpm and blood pressure of 115/64 mm Hg. Cardiac exam was normal with regular rate and rhythm, normal heart sounds and no murmurs. The patient had some diffuse tenderness in the lower abdomen and the rest of the physical exam was unremarkable. Laboratory data included a hemoglobin of 6.9 g/dl, hematocrit of 20.6%, white blood count of 18,000 cells/mm³ and platelets of 256,000 cells/mm³. The patient was transfused with 2 units of packed red blood cells and was started on intravenous metronidazole. Chest X-ray film did not show any acute process. EKG was done and showed normal sinus rhythm and no ST or T wave changes. On the second day of admission, the patient experienced a transient episode of dysarthria, slurred speech, right mouth drooping and right-sided weakness. CT scan of the head was done and did not show any acute process.

For further evaluation, he had two-dimensional echocardiogram which showed a large apical thrombus in the left ventricle, about 3.5 cm in size ([fig. 1](#)). Ventricular size and function were normal, and no segmental wall motion abnormalities were observed. Hypercoagulable workup was also done. Factor V Leiden, cardiolipin antibodies, lupus antibodies, protein C and protein S were all negative. The patient was started on intravenous heparin and a heart catheterization was done on the third day. Heart catheterization did not show any coronary artery disease ([fig. 2](#)). After one week of hospitalization, his hemoglobin was stable and his abdominal pain subsided. He was discharged home in stable condition on aspirin, coumadin, prednisone and mesalamine.

Discussion

The formation of a left ventricular thrombus with normal ventricular function is rare. Few case reports have hypothesized that the presence of a hypercoagulable state may be an important causative mechanism of left ventricular thrombus. Schmaier and Denenberg described a patient with polycystic disease of multiple organs and a left ventricular mural thrombus that was ascribed to a hypercoagulable state caused by giant, hyperaggregable platelets [1]. Toto et al. described an apical left ventricular thrombus in a patient with thrombocytosis due to myeloproliferative disease [2]. DeGroat et al. reported left ventricular mural thrombi with systemic emboli in two patients with normal ventricular function and underlying malignancies [3]. Chin et al. reported left ventricular thrombus with systemic emboli in a patient with ulcerative colitis and cocaine abuse [4]. The etiology of thromboembolic complications in patients with inflammatory bowel disease (IBD) is thought to be multifactorial and related to acquired factors as well as inherited risk factors [5]. All components of the coagulation cascade are thought to be involved in pathogenesis. The increased levels of tumor necrosis factor, interleukin-6, factor V, factor VIII, fibrinogen, fibrinopeptide A and enhanced expression of CD40 ligand on platelets are thought to play an important role by promoting a prothrombotic state. In addition, platelet leukocyte aggregates, which cause microinfarction and thrombus formation by enhancing the production of tissue factor, have also been implicated, and they are increased in patients with IBD [6, 7]. Patients with IBD are three times more likely to experience thromboembolic complications than the general population, and such events complicate the conditions of 1–8% of patients with IBD. They are most often venous in origin rather than arterial, with deep venous thrombosis/pulmonary embolism being most common. More than half of these cases occur during disease exacerbations [7].

In conclusion, thrombosis in ulcerative colitis is a very serious condition and can occur in a very young population. This case also shows that left ventricular thrombi can occur in the active setting of ulcerative colitis.

Fig. 1. Large apical thrombus in the left ventricle.

Fig. 2. Normal coronary arteries.

References

- 1 Schmaier AH, Denenberg B: Left ventricular thrombus with normal left ventricular function and hyperaggregable platelets in a patient with polycystic disease of multiple organs. *Am J Med Sci* 1984;288:223–227.
- 2 Toto AS, Parameswaran R, Kotler MN, Parry W: Rapid development of left ventricular thrombus in a patient with myeloproliferative disorder. *Am Heart J* 1987;114:436–437.
- 3 DeGroat TS, Parameswaran R, Popper PM, Kotler MN: Left ventricular thrombi in association with normal left ventricular wall motion in patients with malignancy. *Am J Cardiol* 1985;56:827–828.
- 4 Chin WW, Van Tosh A, Hecht SR, Berger M: Left ventricular thrombus with normal left ventricular function in ulcerative colitis. *Am Heart J* 1988;116:562–563.
- 5 Saleh T, Matta F, Yaekoub AY, Danescu SC, Stein PD: Risk of venous thromboembolism with inflammatory bowel disease. *Clin Appl Thromb Hemost* 2010, Epub ahead of print.
- 6 Jackson LM, O’Gorman PJ, O’Connell J, Cronin CC, Cotter KP, Shanahan F: Thrombosis in inflammatory bowel disease: clinical setting, procoagulant profile and factor V Leiden. *QJM* 1997;90:183–188.
- 7 Khan AB, Bhat N, Hassan A, Al Saied G: Aortic thrombosis complicating Crohn disease: an unusual complication. *Can J Surg* 2009;52:E182–E184.