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Left Ventricular Thrombus Leading to Embolic Stroke in a Patient without Typical Risk Factors

Bradley Tennant
United Memorial Medical Center, btennant@ummc.org

Riley Fernan
United Memorial Medical Center, rfernan@ummc.org

Michael Maggiulli
United Memorial Medical Center, mmaggiulli@ummc.org

Elizabeth Loomis
United Memorial Medical Center, elizabeth.loomis@ummc.org

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Left Ventricular Thrombus Leading to Embolic Stroke in a Patient without Typical Risk Factors

Abstract

Left ventricular thrombi are most commonly observed in patients after a myocardial infarction with left ventricular aneurysm. We present a case of a 60 year old male who developed an embolic cerebrovascular accident (CVA) secondary to a left ventricular thrombus without evidence of an anteroapical aneurysm or diagnosed myocardial infarction. Outpatient investigation after hospitalization for the acute CVA led to an eventual diagnosis of multiple myeloma. This case reinforces the importance of considering rare etiologies including malignancy in patients who present without typical risk factors for embolic disease.

Introduction

The most common event leading to a left ventricular thrombus is an anterior ST elevation myocardial infarction (MI) with anteroapical left ventricle aneurysm.[1] Once developed, a left ventricle thrombus then poses a significant risk of embolizing and leading to an embolic stroke.[2] With advances in rapid revascularization following an MI, left ventricular thrombi have become less common over time. Left ventricular thrombi used to occur in up to 60% of patients prior to widespread use of percutaneous coronary intervention (PCI) however with PCI, prevalence of left ventricular thrombi after an MI now occurs in about 5-15% of cases.[3] Other common causes of left ventricle thrombus include dilated cardiomyopathy, rheumatic heart disease, and peripartum cardiomyopathy.[3]

Case Presentation

A 60 year old male who had not previously engaged in routine healthcare for the past several decades presented to the emergency department with right sided weakness. He had no known medical history and did not take any medications. He had no history of chest pain or dyspnea on exertion. He did have a 45 pack per year smoking history. Family history included a mother deceased from emphysema and father deceased for unknown reasons. Initial workup during his hospitalization revealed a left middle cerebral artery thrombus and infarction of left caudate nucleus and left insular cortex. The patient received tissue plasminogen activator and his symptoms of right sided weakness gradually resolved over the course of a few hours. Standard stroke workup included a transthoracic echocardiogram that demonstrated a left ventricular thrombus with apical hypokinesis near the left ventricle apex. The patient reported no chest pain or palpitations and his initial troponin level was normal but not followed up. An electrocardiogram (EKG) showed sinus rhythm with heart rate of 80 bpm, T-wave inversions in anterolateral and inferior leads. Prior EKG's were unavailable for comparison and coronary angiogram was not performed during the hospitalization. The patient was started on metoprolol, atorvastatin, and warfarin with an enoxaparin bridge and discharged to home.

The patient received routine outpatient follow up care however at two months post hospitalization, he reported a new concern of low back pain. The pain was right sided and the patient reported that it started after a long day of work as a mechanic at a bowling alley. On exam, the patient demonstrated a right lumbar paraspinal muscle spasm and he was treated with a short course of cyclobenzaprine. The back pain persisted so further work up was performed including labwork to assess baseline liver and kidney function. This lab work included a total protein was shown to be elevated at 9.1 g/dL and a decreased albumin:globulin ratio of 0.7. The patient had normal hemoglobin, hematocrit, and calcium levels. Given the overall picture, which included an unexplained left ventricular thrombus and low back pain, a serum protein electrophoresis (SPEP) level was obtained due to concern for possible multiple myeloma. The SPEP resulted with an IgG lambda monoclonal spike. The patient was referred to oncology and a bone marrow biopsy confirmed a diagnosis of multiple myeloma. He was started on chemotherapy treatment shortly after, including zoledronic acid, carfilzomib, lenalidomide, and dexamethasone.

Discussion

With this patient, we suspect that an underlying diagnosis of multiple myeloma contributed to his LV thrombus. While MI with anteroapical left ventricle aneurysm is the most common cause of LV thrombus, there are documented cases of patients developing LV thrombus related to Takotsubo cardiomyopathy,[4] Heparin-induced thrombocytopenia,[5] Behcet disease,[6] and disseminated intravascular coagulation.[7] The lack of chest pain and negative troponin levels, and no ischemic EKG changes would argue against a MI. It is possible that a silent MI could have resulted in the T wave inversion and left ventricular hypokinesis seen on echocardiogram, however in the absence of an anteroapical left ventricle aneurysm it was appropriate to be open to other possible causes of thrombus formation.

While we were unable to find another reported case of left ventricular thrombus related to multiple myeloma, it is known that patients with multiple myeloma and IgG/IgA MGUS do show a significantly increased risk of both arterial and venous thrombosis.[8] The mechanism of hypercoagulability in patients with multiple myeloma remains unknown, but it has been suggested that ongoing clonal plasma cell activity is a potential cause.[8] Despite the elevated risk of arterial and venous thrombosis it is not common practice to place patients with multiple myeloma on thromboprophylaxis outside of the treatment course described below. Our patient however, was continued on warfarin given the known LV thrombus.

Preferred treatment for patients with symptomatic multiple myeloma is hematopoietic cell transplantation (HCT) however this patient declined the procedure as he did not want to have it done during the COVID 19 pandemic. For patients who are ineligible for HCT, triplet therapy is the preferred initial treatment. Triplet therapy consists of a Thalidomide agent, steroid, and an alkylating agent or an anthracycline. Some patients who are deemed too frail for triplet therapy will often be treated with thalidomide and a steroid. Interestingly people on this treatment have reported frequency of thrombotic events as high as 26%.[9] All multiple myeloma patients receiving combination therapy are recommended to be on routine VTE prophylaxis per the American Society of Clinical Oncology.

Conclusion

Left ventricular thrombi have become rarer over time due to increased use of percutaneous coronary angiography after MI. Other risk factors continue to exist for left ventricular thrombi and thrombi in general. When patients present without typical risk factors for left ventricular thrombi, it is important to consider some of the less common causes of thrombi including malignancy.

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