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Mitral Valve Aneurysm in Mitral Valve Endocarditis:A Case Report

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Abstract

Mitral valve aneurysm (MVA) is an ominous complication of infective endocarditis (IE), with worse outcomes seen among patients with preexisting valvular disease or intravenous drug use. Valve aneurysms can perforate or lead to rupture of the chordae tendineae, with the consequent development of severe mitral regurgitation and acute pulmonary edema. We present a case of a 54-year-old woman with hypertension, obesity, diabetes mellitus, hyperlipidemia, chronic obstructive pulmonary disease, peptic ulcer disease, obstructive sleep apnea, gastroesophageal reflux disease, intravenous drug abuse and bipolar disorder who developed MVA one month after being discharged for IE. Decline in the clinical status of patients with IE is a troubling sign that may indicate an IE complication such as MVA. Physicians should diligently monitor patients with IE for changes in signs and symptoms, as early recognition and surgical intervention are key to prevent further morbidity and mortality.

Keywords

mitral valve aneurysm; infective endocarditis; infective endocarditis risk factors; surgical indications in infective endocarditis; post-pericardiotomy syndrome

1. Introduction

Infective endocarditis (IE) is an infection of the inner lining of the heart that commonly affects the atrioventricular valves [1]. Valve involvement by IE can cause new onset heart failure, embolic events, myocardial abscess, and valvular aneurysms. Mitral valve aneurysm (MVA), a seldom reported complication of IE, appears to occur in the setting of aortic valve endocarditis leading to valvular insufficiency, with regurgitant blood striking the mitral valve leading to valvular infection which is followed by aneurysm formation [2]. Alternatively,

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granulation and scarring occurring during the recovery period of the valvular infection may also precipitate aneurysm formation [3]. The diagnosis of MVA relies on a recognition of changes in clinical status and repeat echocardiography. Our case illustrates MVA developing after IE treatment course possibly due to scarring changes to the valve.

2. Case Report

A 54-year-old woman with a past medical history of hypertension, diabetes mellitus, hyperlipidemia, chronic obstructive pulmonary disease, peptic ulcer disease, obstructive sleep apnea, gastroesophageal reflux disease, intravenous drug abuse and bipolar disorder presented with a four day-history of bloody emesis associated with epigastric pain and malaise. The patient had been admitted two weeks before for hematochezia. No endoscopic procedures were performed at that time, as the gastrointestinal bleed did not recur. Home medications included insulin glargine, insulin aspart, nifedipine, lisinopril, tiotropium, montelukast, atorvastatin, folic acid, vitamin D supplements and pantoprazole.

On initial examination, her blood pressure was 149/58 mmHg, pulse 107 beats per minute, and temperature 100.2°F. The patient appeared restless, but no abnormal cardiac or pulmonary findings were noted. Additionally, the abdominal exam revealed epigastric tenderness without any distension, rebound or guarding. Initial blood work was significant for leukocytosis of 19.9 K/uL; however, the chest x-ray showed no acute cardiopulmonary changes and EKG revealed normal sinus rhythm with left ventricular hypertrophy. Complete laboratory test results are shown in Table 1. Given the clinical presentation, consistent with systemic inflammatory response syndrome (SIRS), she underwent an infectious work-up including blood and urine cultures; vancomycin and ceftriaxone daily were commenced to cover for a possible healthcare-associated infection. On hospital day 2, blood cultures grew *Staphylococcus aureus*. A transthoracic echocardiogram (TTE) was equivocal for valvular vegetations. A transesophageal echocardiogram (TEE) revealed a medium-sized (4.35 mm x 9.17 mm) mobile echodensity on the mitral valve consistent with a vegetation. Cefazolin at a dose of 2 grams every 8 hours was initiated after sensitivities demonstrated methicillin-sensitive *S. aureus*. Following antibiotic treatment, intensive insulin adjustment and supportive measures, the patient improved clinically with repeat blood cultures reported negative for growth. Four weeks from admission and prior to discharge, a peripheral inserted-central catheter was placed, and the patient was to continue cefazolin intravenously at the same dose of 2 grams every 8 hours for two additional weeks. Home infusion arrangements were made as well as follow-up appointments with Cardiology and Infectious Disease clinics.

One month after hospital discharge, the patient presented to her cardiologist office due to new onset of chest pain and shortness of breath. Given the recent history of endocarditis as provided by the patient, an echocardiogram was immediately obtained, which revealed abnormal results that prompted a referral to the emergency department (ED).

The patient reported stopping antibiotics after the first week of the intended two-week course due to a rash. The patient had also developed increasing shortness of breath and decreased exercise tolerance which prompted her visit to her cardiologist. Further

history elicited non-radiating, left-sided, pleuritic chest pain, orthopnea, and leg swelling; however, she denied fever, chills, or night sweats. On examination, her blood pressure was 148/80 mmHg, pulse 93 beats per minute and temperature 97.8°F. Physical exam revealed no cardiac murmurs, rubs, or gallops, or pulmonary abnormalities; initial labs were significant for a troponin elevation of 0.47 ng/dl. Laboratory test results obtained on re-admission are included in Table 1. Her electrocardiogram showed normal sinus rhythm and her labs showed no leukocytosis. Cardiology recommended to obtain a transesophageal echocardiography (TEE) and repeat blood cultures. The TEE showed a new 10 mm mitral valve aneurysm (Image 1 and Image 2), with prolapse of the medial and distal segments of the anterior leaflet of the mitral valve, mobile echo densities thickly coating the atrial aspect of the outpouching, and small, strand-like vegetations on the posterior leaflet. Two separate blood culture sets showed no growth. Infectious diseases recommended no further antibiotics, since echo densities were likely due to post-infectious changes and her endocarditis seemed to have been adequately treated. A diagnostic cardiac catheterization demonstrated an elevated pulmonary artery systolic pressure of 51 mmHg and non-obstructed coronary arteries.

Due to the extensive valvular damage, a decision was made to perform a robotic-assisted mitral valve repair with ring annuloplasty. Intraoperatively, the patient received three units of packed red blood cells, two units of fresh frozen plasma, and one unit of platelets. Her bypass time was 194 minutes and her cross-clamp time was 105 minutes. The procedure was uncomplicated except for difficult grafting the left saphenous vein. Labs following the procedure revealed a hemoglobin/hematocrit of 6.9g/dL/19% and two units of packed red blood cells were administered. Oxycodone, low-dose metoprolol and aspirin were added to the patient's medical regimen. The immediate post-operative period was otherwise uneventful. She was discharged on post-operative day six with instructions to follow-up with the cardiothoracic surgery clinic one week following discharge.

The patient presented to the emergency department five days following discharge, on post-operative day 11, endorsing pleuritic right-sided chest pain and dyspnea. A chest x-ray showed a partial opacification of the right hemithorax consistent with a large pleural effusion and atelectasis. Interventional radiology drained 1800 ml of straw-color pleural fluid and placed a drain in the pleural space. The drain was removed after 48 hours given minimal drainage. The fluid studies revealed an exudate and the cultures and cytology were reported as negative. Over the course of the next few days, the patient's pain and dyspnea diminished gradually. She was discharged home given her improved clinical status. One week later, she returned for a regular clinic visit later and offered no complaints.

3. Discussion

MVA is a rare entity, appearing in only 0.204% of all TEEs in a large, single-center study [5]. Untreated MVA can lead to severe mitral regurgitation and acute pulmonary edema through multiple mechanisms, including perforation of the aneurysm and rupture of the chordae tendineae [6,7]. The mainstay of treatment for IE is antibiotics. Surgery is indicated for heart failure, complicated IE manifested with uncontrolled infection, paravalvular abscess involving the aortic root, resistant microorganisms and the presence of

large vegetations with high risk of embolization [8]. Despite MVA not being counted among the surgical indications, few cases of MVA have been treated without valve replacement surgery as reported in the current literature [9,10]. In the present case, the decision to replace the valve was based on the degree of structural damage of the mitral valve, with both the anterior and posterior leaflets being affected.

While mortality in MVA has not been quantified outside of small case series, IE alone has up to 22% in-hospital and 40% five-year mortality rate [11,12,13]. As IE is the most common precursor for MVA, early detection and treatment of IE is a reasonable strategy for prevention of MVA. Certain populations, such as intravenous drug users or those with preexisting valve damage secondary to conditions like rheumatic heart disease, are at increased risk for IE [14]. In the United States, *Staphylococcus aureus*-associated IE has more than doubled in the past 50 years, in part due to increased prevalence of intravenous drug use [15]. To prevent IE and its dangerous sequelae, the American College of Cardiology and American Heart Association recommends antibiotic prophylaxis to those with a high risk such as past history of IE, prosthetic heart valves or cardiac valve repair with prosthetic material, a number of repaired or unrepaired congenital heart diseases, and cardiac transplant patients with cardiac valve abnormalities [16].

This patient, who was without any known heart valve defect, presented with bacteremia and mitral valve endocarditis during her first admission. Although right-sided cardiac catheterization revealed significantly elevated pulmonary artery pressures, likely secondary to mitral insufficiency, it is unknown if MVA was an effect or a contributory cause without previous studies. Additionally, most cases of MVA occur in the setting of aortic valve endocarditis with aortic regurgitation [17], which was not present in our patient.

The patient's poorly controlled diabetes, as evidenced by her initial hemoglobin A1c level of 11.4%, can be hypothesized as a risk factor for this patient. A review analyzing predictors of in-hospital mortality in IE patients showed that patients with diabetes mellitus had a higher in-hospital mortality rate compared with nondiabetic patients (OR, 3.33; 95% CI, 1.77 to 6.27) [18], while another study identified diabetes mellitus as an independent predictor of mortality (OR 2.49; 95% CI 1.15-5.62) [19]. Prognosis aside, most data support the notion that diabetes increases the likelihood of developing infections, and IE is no exception. There are two large-scale studies illustrating the association between diabetes and IE [20,21].

Besides the patient's metabolic comorbidities, our patient's history of intravenous drug use (IVDU) seems to have been another risk factor for developing IE and, by extension, MVA. If this patient's IE did develop in association with her intravenous drug use, however, her presentation would be atypical, since in IVDUs, IE is normally right-sided with the tricuspid valve most commonly affected in 46%–78% of cases [22], whereas our patient's tricuspid valve remained unaffected. Although the patient did prematurely stop her antibiotic course, completing roughly five out of the intended six weeks, evidence seemed to suggest that the IE was adequately treated, and no antibiotics were deemed necessary during her second admission.

Interestingly, this patient developed yet another complication, post-pericardiotomy syndrome (PPS), after the mitral valve replacement surgery. PPS can develop with a frequency of 9-50% after cardiothoracic surgery [23,24]. Proposed pathophysiologic mechanisms for the development of PPS include heightened inflammatory response, chemokine level derangements, complement cascade products, intra-operative usage of red cell transfusions and patient's age [25]. Our patient required drainage of the large pleural effusion given the respiratory compromise; however, she did not require anti-inflammatory drugs for the management.

While caring for patients with IE patients, physicians must bear in mind the complications that these patients can develop during the course of care or after discharge. Appropriate follow-up, antibiotic therapy monitoring, and patient education can aid in early detection and intervention of IE complications.

4. Conclusion

MVA is a rare complication of IE that can lead to severe morbidity and mortality when is not promptly recognized and not appropriately managed. Patients with IE who are stable enough to be discharged on home antibiotics must still be monitored for worsening or new symptoms, as early surgical intervention may prevent perforation of a MVA aneurysm and flash pulmonary edema.

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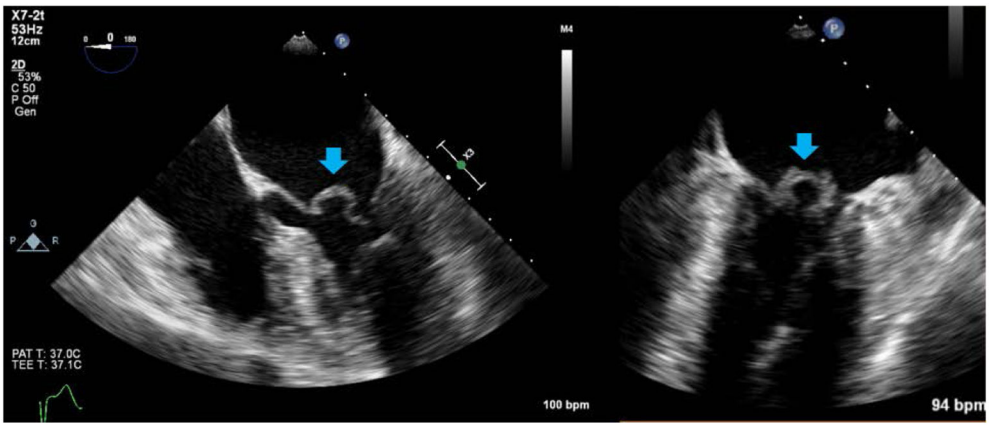

Image 1.

Arrow indicating mitral valve aneurysm in 4 chamber view (left) and 2 chamber view (right) of TEE

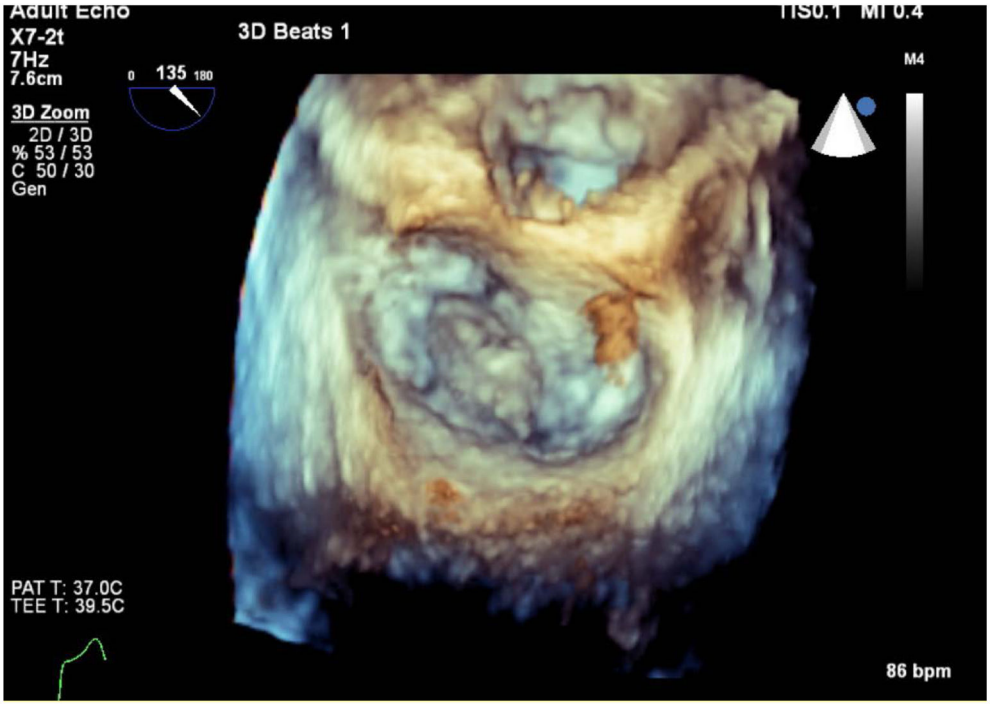

Image 2.
3D view of mitral valve aneurysm as seen from left atrial side in TEE

Table 1.

Laboratory Data

Serum	First admission	On discharge	Readmission	Reference Range
WBC (K/uL)	19.9	8.44	9.40	4.5-10.9
RBC (M/uL)	3.89	3.06	3.29	4.2-5.4
Hemoglobin (g/dL)	9.9	8.0	8.7	12.0-16.0
Hematocrit (%)	30	25.9	27.5	37.0-47.0
Platelets (K/uL)	365	290	225	130-400
Sodium (mmol/L)	127	136	143	136-146
Potassium (mmol/L)	2.8	4.1	3	3.5-5.0
Chloride (mmol/L)	84	101	106	98-106
BUN (mg/dL)	20	17	28	6-20
Creatinine (mg/dL)	1.53	1.08	0.94	0.4-1.2
Calcium (mg/dL)	7.8	8.6	8.7	8.4-10.3
Total Protein (g/dL)	7.1	6.2	6.2	6.0-8.5
Albumin (g/dL)	2.3	2.2	2.85	2.8-5.7
AST (U/L)	59	31	15	10-35
ALT (U/L)	29	14	9	0-31
Alk. Phos (U/L)	307	393	236	25-125
Total Bilirubin	1.82	0.43	0.30	0.0-1.2
Glucose (mg/dL)	442	260	131	70-99
Hemoglobin A1c (%)	11.4	Not obtained	Not obtained	< 6