

Infective Endocarditis of a Left Ventricular Myxoma in a Heroin User

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Infected cardiac myxomas are rare and can have disastrous sequelae; urgent surgical resection is typically indicated. We report the case of a 43-year-old user of intravenous heroin who presented with weakness and dyspnea. He was diagnosed with infective endocarditis of a myxoma attached to the left ventricular lateral wall. The patient underwent successful surgical resection of the myxoma and then completed 4 weeks of antibiotic therapy. In addition to discussing this patient's case, we briefly review the relevant medical literature, in which we found only 4 previous reports of left ventricular myxoma associated with infective endocarditis. (Tex Heart Inst J 2019;46(3):215-8)

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Infected cardiac myxomas pose a severe threat of sepsis, septic emboli, disseminated intravascular coagulation, and even sudden cardiac death. Observational evidence suggests that diagnosis can be made based on results of transesophageal echocardiography (TEE) 5 to 7 days after the onset of bacteremia. Class I indications for urgent surgery in cases of infective endocarditis (IE) include expanding vegetation, fistula formation, abscess formation, pseudoaneurysm, or persistent fever with positive blood cultures for at least 7 to 10 days. However, these indications all presume that the IE is localized to a native valve.

We report the case of an intravenous heroin user who had IE of a myxoma attached to the left ventricular (LV) lateral wall. His initial transthoracic echocardiographic (TTE) results were negative for IE; however, our high clinical suspicion and the patient's history of intravenous drug use prompted us to perform TTE early. In addition to reporting our case, we briefly review the relevant medical literature, which to our knowledge includes only 4 other reports of LV myxoma associated with IE.

Case Report

In October 2016, a 43-year-old man presented at our emergency department with a 2-day history of shortness of breath and weakness. The patient's dyspnea had occurred initially at rest but then also on exertion. His exercise tolerance had decreased from walking 10 or 12 blocks to half that distance. He reported no paroxysmal nocturnal dyspnea, chest pain, palpitations, lower-extremity swelling, headaches, changes in vision, or other systemic symptoms. His medical history included hepatitis C, multiple inpatient treatments for cellulitis, abdominal surgery, and an open reduction with internal fixation of his right ankle. He was actively enrolled in a methadone maintenance program for opiate addiction. His last heroin use was one week before the current presentation, and his last methadone dose had been earlier in the day.

On presentation, the patient's temperature was 37.8 °C; blood pressure, 129/79 mmHg; heart rate, 116 beats/min; respiratory rate, 20 breaths/min; and oxygen saturation, 99% on room air. Physical examination revealed blanket whiteness over his tongue and multiple papules around his uvula. An electrocardiogram indicated sinus tachycardia, but otherwise the results were normal. Laboratory results included hemoglobin, 10.6 g/dL (microcytic anemia); white blood cell count, 5,600/μL; aspartate aminotransferase, 43 U/L (slightly elevated); cardiac troponin, 0.169 ng/mL (elevated); and creatine kinase, 878 U/L. The patient was admitted to the hospital.

The patient's troponin level peaked at 0.509 ng/mL on day 3 of admission. Initial blood cultures grew *Staphylococcus aureus*, so therapy with intravenous vancomycin and piperacillin-tazobactam was initiated. When the bacteria were found to be methicillin-sensitive, the antibiotic therapy was changed to nafcillin. Given the patient's

history of intravenous drug use, TTE was performed to rule out IE. The size of his heart chambers, systolic function, and pulmonary artery pressures were normal. However, the patient's bacteremia was persistent: blood cultures were positive for methicillin-sensitive *S. aureus* for 6 consecutive days. When TEE was ordered to reevaluate possible endocarditis on day 4, it revealed a 4-cm mass with mobile extensions under the mitral valve annulus that protruded into the LV outflow tract (Fig. 1).

The location and size of the LV mass raised concern that the patient might have an underlying myxoma that had become infected by *S. aureus*, with possible embolic sequelae. Magnetic resonance images (MRI) of the brain revealed multiple acute or subacute infarcts in the frontal and parietal lobes, the medulla, and the cerebellum (Fig. 2). Consequently, cardiothoracic surgeons were urgently consulted about possible excision of the LV mass. The interim risk was deemed too high to evaluate whether a mycotic aneurysm was present, as suggested by our neurologists.

The patient underwent surgical resection of the LV mass, with use of cardiopulmonary bypass and the antegrade administration of cardioplegic solution. The aorta was opened transversely, 3 to 4 mm above the right coronary artery. After the aortic valve leaflets were removed, the anterior mitral valve leaflet was moved laterally for a direct view of the amorphous mass attached to the LV lateral wall (Fig. 3). Multiple vegetations were noted on the mass, and débridement was performed down to the level of the endocardium. There were no complications.

In the cardiothoracic surgical intensive care unit, the patient briefly needed vasopressor support with norepinephrine and epinephrine for postoperative cardiogenic shock. Histopathologic analysis indicated that the mass was a myxoma with neutrophilic infiltration, confirming infection (Fig. 4). The patient recovered uneventfully

and was discharged to a skilled nursing facility for an additional 4 weeks of intravenous nafcillin therapy, after which he was to follow up with our neurology department about the possible presence of a mycotic aneurysm.

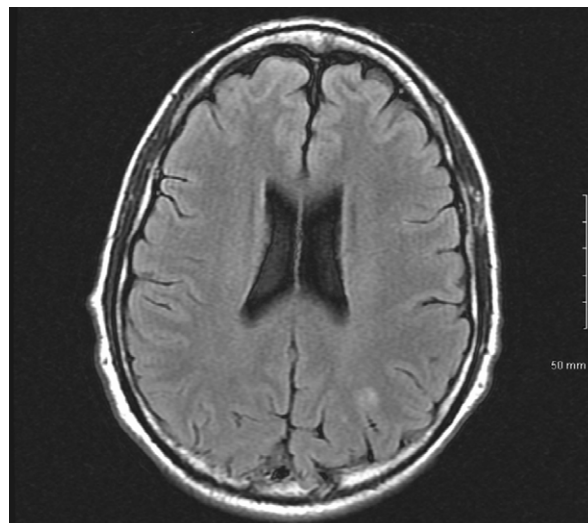


Fig. 2 Magnetic resonance image (T2-weighted) of the brain shows multiple hypointense lesions consistent with septal emboli.

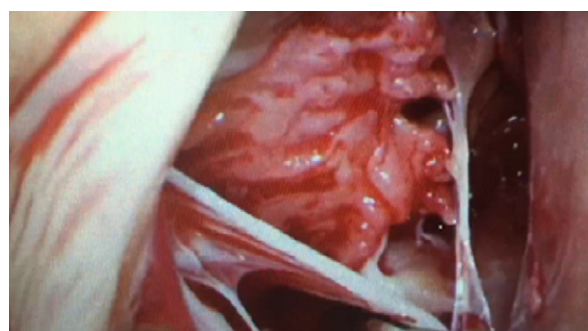


Fig. 3 Intraoperative photograph shows the left ventricular myxoma.



Fig. 1 Transesophageal echocardiogram shows a valve-sparing intracardiac mass in the left ventricle, extending into the left ventricular outflow tract.

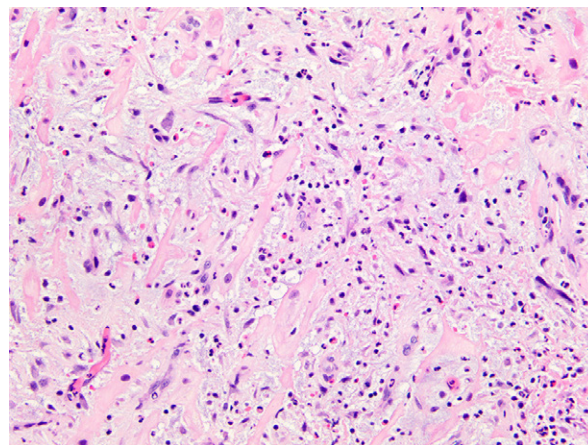


Fig. 4 Photomicrograph shows myxoma cells in a fibromyxoid stroma infiltrated with neutrophils (H & E, orig. $\times 200$).

Discussion

Primary cardiac tumors are rare, with a prevalence of 0.0017% to 0.19%.¹ Of these, myxomas are the most abundant, with 75% occurring in the left atrium.^{2,3} Left ventricular myxomas are exceptionally infrequent, constituting only 2.5% to 4% of all myxoma cases.⁴ In a case report and literature review, Revankar and Clark³ proposed diagnostic criteria for infected myxomas. The patient whose case they discussed had an LV myxoma, persistent bacteremia with *S. aureus*, and documented inflammation on pathologic analysis, which, by their criteria, was consistent with infected myxoma. Although many cases of LV myxoma have been documented, theirs appears to be the first reported case of an infected LV myxoma.

According to Yuan's comprehensive review of infected myxomas,² *Streptococcus* was the predominant infective cause (41.5% of cases); however, methicillin-sensitive *S. aureus* caused only 9.7% of cases. Most of the infected myxomas were in the left atrium (27 of 39, 69.2%), 5 (12.8%) were in the right atrium, 5 (12.8%) were on the mitral valve, 1 (2.6%) was in the right ventricle, and the location of 1 was unspecified; none were in the LV.² Thus, Yuan's findings can be only partially applied to our case. Our patient's infected myxoma was on the LV lateral wall, completely sparing all valves. Also of note in our case was the method of diagnosis. Thirty-one (77.5%) of the myxomas in Yuan's review were diagnosed with use of TTE and were confirmed by using TEE; conversely, only 9 (22.5%) were diagnosed solely by using TEE, as in our patient. Finally, whereas most of the studied patients were severely ill and hemodynamically unstable, our patient was hemodynamically stable throughout his course and did not need intensive care preoperatively.

Only a few authors have described cases of LV myxoma, and only some of these myxomas were infected. Yoshioka and colleagues⁵ discussed a rare case of an infected pedunculated LV fibroma that resulted in central and peripheral embolization. Mahmoud and Moursi⁶ described the case of a 35-year-old man who presented with a 2.5 × 5-cm grapelike LV mass that substantially obstructed the LV outflow tract (peak systolic pressure gradient, 35 mmHg). Of note, the patient presented with an acute cerebrovascular stroke. Although our patient was diagnosed with multiple emboli within the infratentorial and supratentorial regions of the brain, his neurologic examination revealed no noticeable deficits in his cranial nerve function, motor function, sensation, coordination, gait, or reflexes.

The meta-analysis by Yuan² confirmed that the complications resulting from an infected myxoma are severe. In 39 cases of infected myxoma, 12 patients had one or more medical complications: 3 had disseminated intravascular coagulation, 4 had sepsis, and 10 had septic

emboli. In addition to the risk of embolization, some patients have an elevated risk of sudden cardiac death.^{4,5,7} Our patient's brain MRI showed multiple small hypoattenuating lesions throughout all lobes bilaterally, the medulla, and the left cerebellum. Nevertheless, throughout his course, the patient remained hemodynamically stable and had no neurologic consequences from his septic emboli.

The commonality in all the authors' meta-analyses and case reports is that after patients were diagnosed with an infected myxoma, all underwent immediate surgical intervention. Of the 39 patients who had an infected cardiac myxoma in Yuan's analysis,² 38 underwent surgical resection. Although their preoperative antibiotic regimen, timing of surgical procedure (emergency vs urgent), and type of procedure all varied, the primary endpoint of survival was 92.6% at a mean duration of 11.1 ± 14.5 months. The secondary endpoint, survival free of medical or surgical complications from septic emboli, was 94.6%.

Intravenous drug use is a distinct risk factor for endocarditis. Chao and colleagues⁷ studied drug users who had endocarditis and compared them with nonusers who had endocarditis to find possible prognostic indicators for their hospital course. The authors found that the most important prognostic factor for death when intravenous-drug users have endocarditis is whether they go into noncardiac shock (odds ratio, 15.5). However, this study had substantial limitations, and its findings may be applicable only to endocarditis.

Our patient presented atypically. He was hemodynamically stable with no cardiac murmur or abnormal findings on TTE, despite the high risk of endocarditis associated with his infected myxoma, as well as its unusual location. The septic emboli in his brain caused no neurologic sequelae. Although observational evidence suggests that TEE is most sensitive 5 to 7 days after the onset of bacteremia, we opted to perform TEE earlier, given our patient's history of drug use and our strong clinical suspicion of endocarditis. Our experience with this patient suggests that high suspicion must be maintained in patients who have pertinent risk factors and toxic behaviors. We conclude that early detection and a multidisciplinary approach can help to prevent severe complications from this disease.

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