

CASE REPORT

Left ventricular outflow tract thrombus detected by echocardiography in a young man with a structurally normal heart: Case report and literature review

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Abstract

Left ventricular outflow tract (LVOT) thrombi are extremely rare and only two previous cases have been published in the medical literature. We present the third case of LVOT thrombus from Sinai Hospital of Baltimore. This thrombus was an incidental finding in a patient who was status post motor vehicle accident. The patient was a 24 year old man who was found to have a mobile mass in the LVOT by echocardiogram. His heart was otherwise structurally normal. After surgical excision and pathologic examination, the mass was diagnosed as being a fibrin thrombus.

Keywords

Left ventricular outflow tract thrombus, Echocardiography, Motor vehicle accident

1 Introduction

Mass formation on the basal left ventricular septum at the left ventricular outflow tract (LVOT) is a rare phenomenon. Possible etiologies of a cardiac mass include tumor^[1-3], thrombus, and vegetation^[4]. Left ventricular (LV) thrombi usually form in association with impaired LV function as is seen with myocardial infarction^[5-6], LV aneurysm^[7], dilated cardiomyopathy^[8], and ventricular assist devices^[9]. LV thrombi can also be seen in association with cardiac trauma^[10], myeloproliferative disorders, antiphospholipid antibody syndrome^[11] and hypereosinophilic syndrome^[12]. Cases of LV thrombi in normal hearts have also been reported^[13-15]. Thrombi formation on the basal left ventricular septum at the LVOT is extremely rare as it is a region of high velocity blood flow. On our review of the literature, 2 previous cases were reported. One reported case of LVOT thrombus was attributed to transient eosinophilia^[16]. In the other case, thrombus formation in the LVOT was discovered during surgery in a patient on biventricular mechanical support^[17]. Here we report a case of LVOT thrombus in a young man with a structurally normal heart incidentally detected by echocardiography after a motor vehicle accident.

2 Case report

A 24 year old African American man was admitted to our hospital after a motor vehicle accident. On admission, he had some superficial facial lacerations, as well as CT evidence of Grade II liver laceration with subcapsular hematoma. He had no fixed neurologic defects. There was no evidence of intrathoracic injury seen on the CT scan.

On 2nd day of admission, he complained of mid-sternal chest pain and shortness of breath. Upon further questioning, he admitted to a previous history of exertional chest pain radiating down to his left arm when doing power walks at work. He had no other symptoms or significant medical history. In hospital, his pulse varied from 50-90 bpm and blood pressure ranged from 94/54 -118/78 mmHg. Cardiac examination revealed regular heart sounds and no murmurs/gallops/rubs. Chest X-ray was within normal limits. ECG showed sinus bradycardia but was otherwise within normal limits. Laboratory examination on admission showed elevated AST and ALT of 953 unit/L and 939 unit/L respectively. Total bilirubin of 1.2 mg/dl. Alcohol level of 212 mg/dl. White blood cell count of 10 k/mm^3 and hemoglobin of 15 g/dl. Blood cultures were negative. Other laboratory results were within normal limits including: troponin 0.069 ng/ml, Prothrombin time 11.1 sec, Platelet Count 224 k/mm^3 , activated partial thromboplastin time 18.4 sec. Transthoracic echocardiography revealed a mobile mass attached to basal LVseptum at the left ventricular outflow tract that measured $1.5 \text{ cm} \times 0.5 \text{ cm}$. LV function was normal (ejection fraction 55%-65%).

The LV was otherwise structurally normal and there were no valvular abnormalities. The patient was then referred for Transesophageal echocardiography (TEE). That study confirmed the presence of an echogenic mobile mass attached to the LV septum at the hinge point of the right coronary cusp of the aortic valve (see Figure 1). Trace aortic and mitral regurgitation was noted. Considering the possibility that this mass was high risk for embolism, the patient was referred for urgent surgical intervention. Prior to surgery, the patient underwent cardiac catheterization that showed his coronary arteries were normal.



Figure 1. Transesophageal echocardiogram 5 chamber view showing mass in LVOT.

Intraoperatively, the echocardiographic findings were confirmed. A pedunculated mass measuring $1.5 \text{ cm} \times 0.5 \text{ cm}$ was found attached to the basal LV septum in the LVOT just below the right coronary leaflet hinge point. There were no other valvular abnormalities. The LV function was normal. The mass was carefully excised and sent for pathologic examination (see Figure 2).



Figure 2. 1.5 cm × 0.5 cm mass resected from LVOT.

A postoperative echocardiogram showed no residual mass. The patient's postoperative course was unremarkable. The final pathologic diagnosis of the mass was fibrin thrombus (see Figure 2). Postoperatively, the patient has been asymptomatic and has had no recurrence of ventricular thrombi as confirmed by yearly echocardiograms over the last 4 years.

3 Discussion

Thrombi formation on the basal left ventricular septum at the LVOT in a structurally normal heart is extremely rare as it is a region of high velocity blood flow. The etiology of the thrombus in our case is unknown. In a previous case of LV thrombus in normal heart it was hypothesized that "Patchy fibrosis and myofibrillar hypertrophy resulting from diffuse small coronary vessel disease and pathologic coronary spasm causing transient myocardial ischemia may predispose LV thrombus" [18]. The patient in our case was involved in a motor vehicle accident so it is possible that cardiac trauma may have played a role in this finding (although no significant thoracic traumatic injuries were demonstrated by chest CT scan or clinically). Regarding the patient's history of exertional chest pain, the patient had a cardiac catheterization prior to surgery which ruled out coronary artery disease. It's possible that his symptoms could have been caused by emboli or intermittent obstruction from the cardiac thrombus. Postoperatively, he has been asymptomatic over the last 4 years. Yearly echocardiograms have been negative for recurrent cardiac thrombi.

4 Conclusions

This case report clearly demonstrates the rare occurrence of LV outflow tract thrombus formation using echocardiography confirmed by pathologic examination.

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