Case Report

Atrial Myxomas Causing Severe Left and Right Ventricular Dysfunction

Abstract

Myxomas are the most common cardiac tumors, accounting for about 50% of benign primary cardiac tumors, with the majority located in the left atrium, and 80% of which originate in the interatrial septum. We report two cases with severe cachexia, neurological sequelae, and severe biventricle dysfunction secondary to atrial myxomas with marked early improvement after tumor excision.

Keywords: *Biatrial myxoma, intracardiac mass, left ventricular dysfunction, right ventricular dysfunction*

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Introduction

Cardiac myxoma is the most common type of primary cardiac neoplasm and accounts for 30%–50% of all primary tumors of the heart with an annual incidence of 0.5 per million populations.[1] Over 70% of all cardiac myxomas originate from the left atrium (LA) and 18% from the right atrium (RA). Biatrial myxomas account for <2.5% of all cardiac myxomas.[2] Most myxomas present with one or more effects of a triad of constitutional, embolic, and obstructive manifestations. We present two consecutive patients with atrial myxomas with very severe biventricular dysfunction.

Case Reports

Case report 1

A 38-year-old female patient, weighing 40 kg with a body mass index (BMI) of 15.8, was admitted to our hospital with chief complaints of dyspnea on exertion, New York Heart Association (NYHA) class III, occasionally associated with chest pain, relieved on resting in supine position. She had a history of an episode of cerebrovascular accident with the right-sided hemiparesis that recovered completely with conservative management. Electrocardiograph (ECG) was within normal limits. The patient had tender hepatomegaly and anemia with hemoglobin of 10.7 g%. Evaluation by transthoracic echocardiography (TTE) in the echo laboratory showed a large pedunculated mass (57 mm × 32 mm) in LA attached to interatrial septum moving in and out of mitral inflow and causing mild mitral regurgitation (MR). There was another mass (55 mm × 43 mm) in the RA, moving to and fro across the tricuspid valve and causing mild tricuspid regurgitation (TR). It was difficult to make out the exact attachment of RA mass. Both the ventricles were mildly dilated with severe dysfunction (left ventricular [LV] with ejection fraction [EF] of 36% and global hypokinesia). She was posted for urgent surgical removal of cardiac masses under cardiopulmonary bypass (CPB).

Standard monitoring and anesthesia induction were done according to department protocol but with the patient in Trendelenburg position. Intraoperative TEE confirmed the presence of two very large masses, one each in LA and RA [Figure 1 and Video 1] and also showed that mass in RA was attached to the eustachian valve near inferior vena cava insertion. Right ventricular (RV) function was grossly reduced (tricuspid annular plane systolic excursion [TAPSE] 10 mm; and S' 6 cm/s with tissue Doppler and fractional area change 28%) and LV showed severe global hypokinesia (EF 28%). RA mass was not obstructing coronary sinus and LA mass being quite big was not obstructing the LA appendage. There was mild TR and MR. Trendelenburg position resulted in central venous pressure increasing from 15 to 18 mmHg, but cardiac index improved from 2.6 to 3.2.

After median sternotomy, heparinization, and aortobicaval cannulation, CPB was

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established and cold antegrade blood cardioplegia was given. After arrest of the heart, RA was opened and a jelly-like myxomatous encapsulated mass originating from eustachian valve was removed. Next, through transseptal approach, another myxomatous mass with multiple projections and arising from the septum secundum was removed from LA [Figure 2]. The patient was given loading dose of milrinone (50 μg/kg) and aortic X-clamp removed. X-clamp time was 19 min and CPB time was 24 min. Weaning from CPB proved to be difficult (LV function measured by Simpsons method EF 27%) [Video 2]; therefore, CPB support was continued further, and only after 30 min, it was possible with the help of combination of injection dobutamine (2–4 μg/kg/min) and injection milrinone (0.5 μg/kg/min) along with injection adrenaline (0.01–0.05 μg/kg/min). TEE repeat examination showed the presence of mild TR and MR with normal valve structures. The patient was shifted to the Intensive Care Unit (ICU) with stable hemodynamics and in sinus rhythm. Postoperative course was uneventful and both the ventricles had regained normal function with mild mitral and TR (RV function: TAPSE 18 mm, S' 12 cm/s, FAC 55% and LV function EF 60%, teichholz method) in the next 48 h. Histopathologic reports of both the masses confirmed myxomas.

Case report 2

A 38-year-old adult male weighing 48 kg and BMI of 17.4 kg/m^2 reported with a history of dyspnea on minimal exertion for 8 months, pedal edema, ascites, and puffiness over face for 2 months with exacerbation of dyspnea (NYHA IV). Examination revealed pallor, a mid-diastolic murmur in mitral area, tender hepatomegaly with shifting dullness. ECG showed normal sinus rhythm with the RV strain pattern.

TTE revealed a LA mass measuring 51 mm × 37 mm attached to interatrial septum. RA and right ventricle were dilated with severe biventricular dysfunction and mild TR and MR. Blood investigations revealed hemoglobin of 9.8 g/dl and brain natriuretic peptide of 906 pg/ml.

In intraoperative period, standard monitoring and anesthesia induction were done according to our department protocol but with the patient in Trendelenburg position. TEE confirmed the TTE findings with severe biventricular dysfunction (TAPSE 4.0 mm, S' 3.2 cm/s, and RV FAC 12% and LV EF 20%) [Figure 3 and Video 3]. The myxomatous mass arising from septum secundum in the LA was excised through transseptal approach through the RA. Milrinone bolus (50 μg/kg) was given on pump. X-clamp was released in 32 min and the patient was weaned off bypass after prolonged CPB support in next 60 min with infusions of injection milrinone (0.5 μg/kg/min), injection dobutamine (1–2 μg/kg/min), and injection adrenaline (0.01–0.05 μg/kg/min). Postbypass TEE showed improvement in LV function (EF = 36%, Simpson's method) and there was mild MR but with preserved mitral valve anatomical structure. The patient was shifted to the postoperative ICU in stable condition.

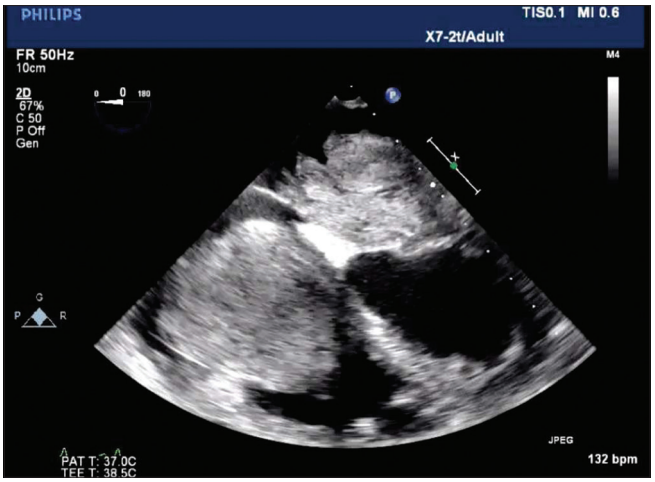

Figure 1: Big mass in right and left atrium (case 1)

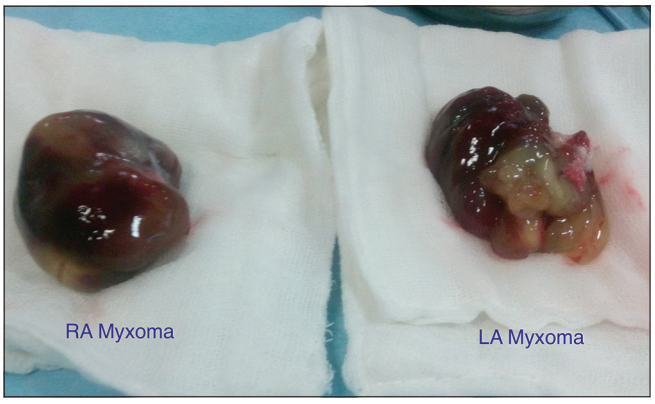

Figure 2: Left and right atrial masses after excision (case 1)

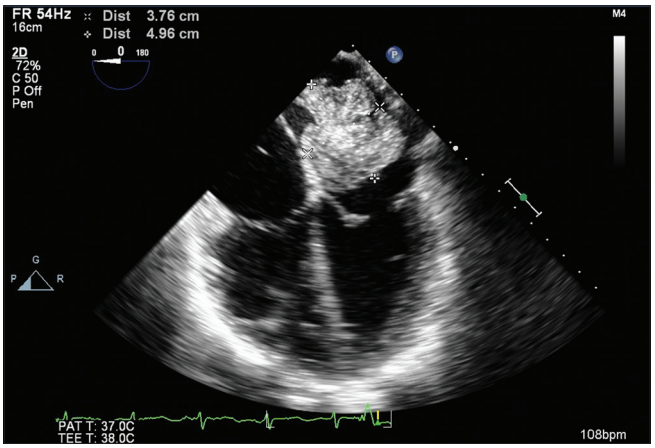

Figure 3: Mass in left atrium in (case 2)

Vasoactive agents were tapered over 8 h and the patient was extubated after 16 h with stable hemodynamics. TTE evaluation after 72 h showed improvement in LV (EF 48% teichholz method) and RV function (TAPSE 10 mm, and FAC 38%).

Discussion

Cardiac myxomas provoke systemic manifestations in 90% of the patients, characterized by weight loss, fatigue, fever, anemia (often hemolytic), and elevated sedimentation rate. These are shown to be related to enhanced interleukin-6 (IL-6) levels that are associated with myxomas.[3] Neurologic events are common in patients with myxoma and can occur in approximately one-third of these patients and association of cerebral aneurysm has also been reported.[4] The mechanism of stroke in atrial myxoma is said to be either due to embolic showers from tumor or implication of enhanced levels of IL-6. Chockalingam *et al*. suggested cardio-depressant effect due to the presence of LA myxoma through unclear mechanisms that reversed only after myxoma removal.[5] Boutayeb *et al*. described RA myxoma in patient with severe LV dysfunction with probable reason being occlusion of coronary sinus (CS) by prolapse of right atrial myxoma during diastole.[6] However, this hypothesis cannot be true because in our first case, RA mass did not occlude the CS as we could register normal flow from CS on color flow Doppler. Very few studies suggest that overproduction of IL-6 by cardiac myxoma could be a possible cause of its association with LV dysfunction.[7] Yan *et al*. confirmed the finding that IL-6 is not only a biomarker predicting the onset of heart failure but also potentially linked in the pathophysiological cascade, possibly through nitroso–redox imbalance and other direct mechanisms especially affecting the septal and inferior wall of left ventricle.[8] In our patients, there was severe septal, inferior, and inferolateral hypokinesia probably due to large biventricular myxomas and in patient with only LA myxoma, global hypokinesia was present and they might have been associated with enhanced IL-6 levels. Although we could not measure the levels of IL-6 in both the patients, regional wall motion abnormalities reduced significantly after excision of myxomas and such early response points toward the removal of a causative element. This biventricular dysfunction can be troublesome in handling these patients in perioperative period and it is still uncertain how to control enhanced levels of IL-6 if this is the real cause of biventricular dysfunction. Chockalingam *et al*. tried steroid therapy for this, but it did not work.[5] More insight in the mechanism of biventricular dysfunction associated with myxomas is needed.

Conclusion

This series of case reports of atrial myxomas highlights the problems associated with handling such cases where severe biventricular dysfunction can lead to difficult weaning of the patient from CPB support. Further insight into the definite cause of biventricular dysfunction is needed.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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