



Left Atrial Myxoma presenting as Stroke- Case Report & Review of Literature

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Summary: Left Atrial (LA) Myxomas present as a wide variety of symptoms. We describe one such case which initially presented with hemiparesis and was diagnosed as cerebral infarction. On retrospective work up, the main cause was found to be a left atrial myxoma. The myxoma has embolised to give rise to cerebral infarction. The LA myxoma was then successfully operated under general anaesthesia and Cardiopulmonary bypass (CPB).

Key words: Hemiparesis, Cerebral infarction, Left Atrial (LA) Myxoma, Cardiopulmonary bypass

Case report: A 55 year old male presented to emergency ward with history of sudden weakness in right side of the body. He also gave history of breathlessness (NYHA-II) of six months duration. His past history was not suggestive of any significant illness. On physical examination, his heart rate was 80 beats per minute and blood pressure 130/80 mmHg. Neurological examination revealed a conscious patient with motor power 3/5 in both right upper & lower limb without sensory involvement. Urgent Computed tomography scan of head showed infarction of the left middle cerebral artery (MCA) territory. The patient shifted to ICU & started conservative management in the line of thrombotic cerebral infarction with physiotherapy, Aspirin, Clopidogrel & Phenytoin Sodium.

On the next day as cardiac evaluation for stroke, 2D transthoracic echocardiography was performed which showed a mass in the left atrium (LA) of about 6.5 cm X 4 cm, possibly LA myxoma with normal left ventricular systolic function. Once diagnosis was confirmed, the patient was



planned for excision of LA myxoma under general anaesthesia on CPB. All routine preoperative investigations were within normal limit & electrocardiography revealed normal sinus rhythm.

The patient was taken to the operation theatre, administered 100 % Oxygen via face mask while monitoring was started. Pulse oxymetry revealed O₂ saturation 95%, BP as recorded by non-invasive blood pressure measurement was 110/70 mm Hg & heart rate 100 beats/min. A right radial artery catheter & a large bore venous access was established using local anaesthesia. Anaesthesia was induced with Fentanyl 5 µgm/kg, Midazolam 0.02 mg/kg & sleep dose of Thiopentone sodium. Trachea secured with Rocuronium bromide 0.9 mg/kg. Post induction right internal jugular venous (IJV) triple lumen catheter was inserted. Arterial blood gases (ABG) following intubation revealed pH- 7.32, pO₂-95 mm Hg & SpO₂- 96%. The Oxygen saturation remained 94-96% until initiation of CPB.

After induction of general anaesthesia, a multiplane transesophageal echocardiography (TEE) probe was placed in the upper esophageal position. As the probe was positioned at the mid esophageal level, a mass (6 X 5 cm) was found to occupy most of the LA with to & fro movement through the mitral valve to the left ventricle during ventricular systole & diastole (Fig.1) without fixed dynamic obstruction. The mass was attached to the intra-atrial septum (IAS) inferiorly near the anterior mitral leaflet (Fig.2). Both mitral leaflets were free from the mass & the mass was homogenous in nature. No mass was seen in other cardiac structures. Anaesthesia was maintained with boluses of Fentanyl, Vecuronium, Midazolam and intermittent Sevoflurane.

Before initiation of CPB, Methylprednisolone 30 mg/kg and Epsilon amino caproic acid (EACA) 100 mg/kg were given with additional doses of EACA 100 mg/kg on CPB and after weaning from CPB as our institutional protocol to prevent inflammatory response to CPB and for antifibrinolytic properties respectively. Excision of LA myxoma was done including part IAS to which the myxoma was attached & the atrial septal defect created was closed with pericardial patch. The tumour was removed completely (Fig.3). Total CPB and cross clamp time was 50 and 35 minutes respectively. On post-operative TEE; LA was completely tumour free without any valvular pathology. Dopamine (5-10 µgm/kg/min) and Nitroglycerine (NTG) (0.5 µgm/kg/min) were started at re-warming. Heparin was reversed with Protamine and termination of CPB became possible with Dopamine support. At the termination of CPB, while breathing 100 % Oxygen, the pO₂ was 247 mm Hg, SpO₂-99%, pCO₂-30 mm Hg & pH- 7.45. Heart rate was 100 beats/min and BP 90-100/50-60 mm Hg. Post CPB red blood cells, platelet concentrate & fresh frozen plasma were started to maintain RA pressure 8-10 mm Hg.

Post-operatively the patient was shifted to ICU and mechanical ventilation continued with PRVC (Pressure Regulated Volume Control) mode. The patient was extubated after 6 hours & Oxygenation continued with ventury mask. Dopamine & NTG tapered from the next day & stopped after 48 hours. Patient was shifted to the surgical ward on 3rd postoperative day from where he was discharged uneventfully on 7th postoperative day with the advice for continued physiotherapy. The histopathology report of the tumour mass confirmed the diagnosis of myxoma.

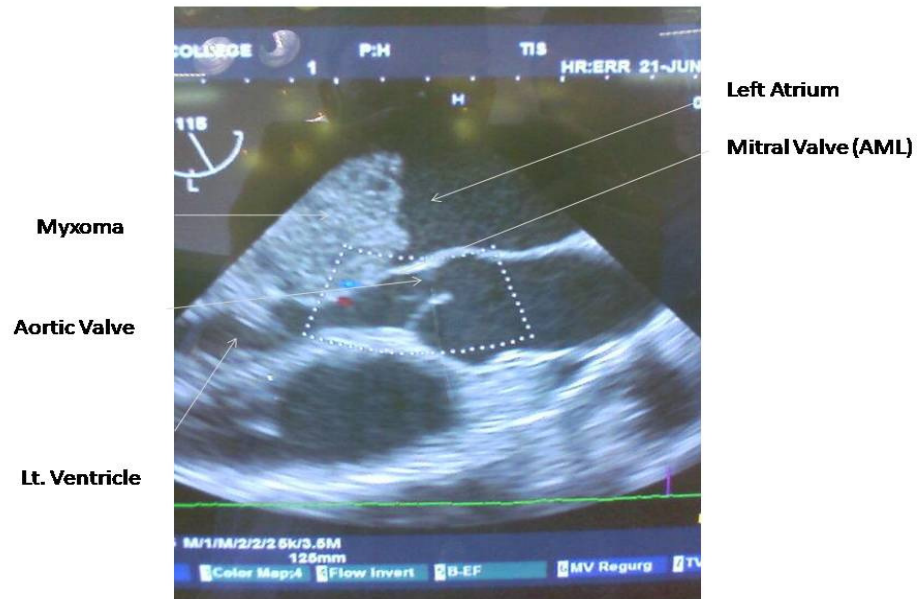


Fig.1: TEE midesophageal Aortic long axis view showing free movement of the myxoma across the mitral valve.

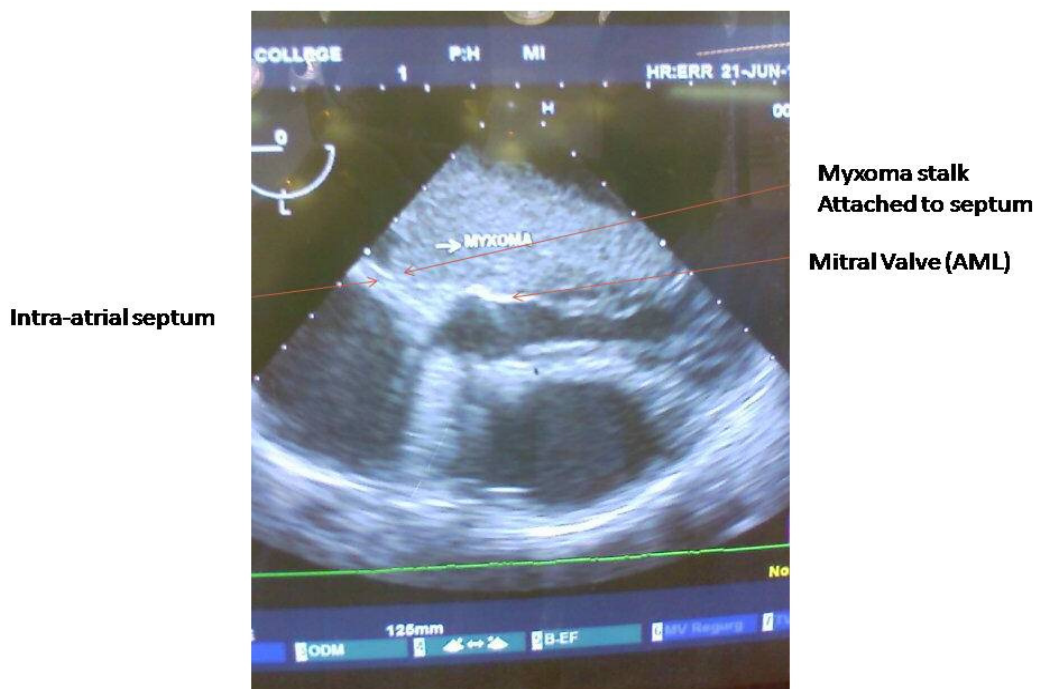


Fig.2: TEE midesophageal four chamber view showing myxoma at left atrium attached to the intra-atrial septum inferiorly near the anterior mitral leaflet (AML).



Fig.3: Postoperative specimen of LA myxoma

Discussion: Myxomas are the commonest primary benign cardiac tumours with the incidence of 0.5 per million population¹. Myxomas account for 0.3% of all cardiac surgeries performed². Although any age group can be affected, it predominates in the age group of 30-60 years of age with more than 75% of the affected being women³. Although most cases occur sporadically, 7-10% of atrial myxomas occur in a familial pattern, with an autosomal dominant transmission⁴. The occurrence of myxomas in left & right atrium are 75 % & 20% respectively. However, it appears in other locations of the heart, even occupying more than one chamber⁵.

Rarely discovered by incidental echocardiography examination, it may manifest a variety of symptoms. The classic triad includes embolism, intracardiac obstruction & constitutional symptoms (Goodwin's triad)⁶. Approximately 80% of individuals present⁶ with one component of the triad, yet upto 10% may be asymptomatic even with mitral myxomas, arising from both atrial & ventricular sides of the anterior mitral leaflet^{5,7}. The most common initial symptom, dyspnoea on exertion, reflects mitral valve obstruction usually present with LA myxoma³. Persistence of sinus rhythm in the presence of such symptoms helps distinguish atrial myxomas from mitral stenosis. Severe pulmonary hypertension without significant mitral valve involvement suggests tricuspid valve obstruction & recurrent pulmonary embolism known to occur with a RA or RV myxoma. Cardiogenic embolisation occurs in 15% of all ischemic infarcts, commonly because of atrial fibrillation, valvular heart disease, cardiomyopathy, prosthetic valves in around 90%, but 10% of such embolisation occurs because of mitral valve prolapse, paradoxical emboli, endocarditis, and cardiac myxoma⁸. Recurrent fragmentation & embolization of the gelatine like tumour mass usually appear with systemic



manifestations & are characteristics of myxoma and our patient presented with such cerebral stroke due to embolization of the gelatine like material.

LA myxomas most likely cause mitral valve obstruction, often in conjunction with pulmonary artery and venous hypertension. Anaesthetic management closely resembles that for a patient with mitral stenosis. In contrast, RA myxomas may produce signs of right sided heart failure corresponding to tricuspid valve obstruction. Positioning of the patient for surgery must be carefully performed to detect severe restriction of venous return that is often followed quickly by profound hypotension and arrhythmias. A large tumour increases the likelihood of hemodynamic instability.

Fever, malaise, weight loss, fatigue, anaemia, and raised erythrocyte sedimentation rate are common constitutional symptoms which occur in around 90% patients with myxomas^{6,8}. These features resolve immediately after surgery and are believed to be due to release of inflammatory mediators from tumour cells^{8,9}.

Findings on a chest radiography of a myxoma may be absent in one third of patients. Calcifications on the chest radiography is more diagnostic of RA myxoma but is rarely present in LA myxoma. In our patient, also there was no such calcifications on chest radiography. CT & Magnetic resonance imaging can help delineate the extent of the tumour & its relationships to surrounding cardiac & thoracic structures¹⁰. TEE is 100% sensitive for diagnosis of myxoma. Specifically, it yields morphologic detail in the evaluation of cardiac tumours, including points of tumour attachment & degree of mobility. Intraoperative TEE monitoring can aid in recognizing & avoiding tumour embolization⁸. We used TEE in our patient for preoperative confirmation of diagnosis & postoperative assessment of adequacy of tumour resection.

Pulmonary artery (PA) & central venous catheterization in the head & neck is avoided in RA myxoma due to the high risk of disturbing the mass & showering tumour emboli into the pulmonary circulation. If required, the placement of the jugular catheter can be done under direct echocardiographic visualization of the mass¹¹. But in LA myxoma, this is not a problem. We used right IJV for central venous cannulation. Usually PAC is not necessary unless there are specific indications for it.

Surgical management is the treatment of choice for myxomas but open heart surgery immediately after cerebral embolisation is risky. One school of thought considers immediate surgery as the treatment, as recurrent embolisation can be fatal¹². The recurrence of myxoma has been reported to be less than 2% on most series⁸.

Tumour location has a strong influence on anaesthetic management. LA myxomas most likely cause mitral valve obstruction, in contrast to RA myxomas which produce signs of right sided heart failure corresponding to tricuspid valve obstruction. Position of the patient for surgery must be carefully performed to detect severe restriction of venous return that is often followed quickly by profound hypotension & arrhythmias.



The anaesthetic regimen for myxoma excision is not different from anaesthesia for any other cardiac surgery, and a balanced anaesthetic approach is the preferred method^{11,13}. But the main challenge in our patient was the associated central nervous system disorders (history of recent stroke). So, in our patient we carefully titrated all drugs intraoperatively as well as postoperatively & drugs having neuroprotective effects were used. Bispectral index (BIS) monitoring helps us guiding depth of anaesthesia. Opioids, along with volatile anaesthetic agents, which have additional advantage of inducing ischemic preconditioning (in patients likely to have ischemic myocardial insults), and any of the commonly used muscle relaxants can be combined for the balanced approach¹⁴. Benzodiazepines, forms a core component of the balanced approach and midazolam in particular is preferred for minimal effect on coronary blood flow autoregulation. However induction with propofol is not advised because of action causing significant depression of myocardium, and hypotension owing to decrease systemic vascular resistance^{11,13}. In our patient, we used Thiopentone for induction for its neuroprotective effects.

Conclusion: TEE has important role in patients with embolic stroke to exclude cardiac causes & should be done as early as possible. Surgical excision of myxoma is the treatment of choice to prevent further embolic events which can be fatal. Balanced anaesthesia technique is the technique of choice for anaesthetic management of myxoma associated with central nervous system illnesses like cerebral stroke. Careful titration of drugs along with advanced intraoperative TEE monitoring results in excellent postoperative outcome.

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