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CARDIAC MYXOMA CLINICAL PRESENTATION AND ITS ANAESTHETIC MANAGEMENT; A SYSTEMIC REVIEW.

Anaesthesiology

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ABSTRACT

Cardiac myxomas are the most common primary tumours of the heart. Although they are benign, they are considered "functionally malignant" due to their high potential for embolisation. These tumours primarily originate in the left atrium, specifically from the limbus of the fossa ovalis in the interatrial septum, but they can occur in any chamber of the heart. While most cardiac myxomas are sporadic, some cases are familial and associated with the Carney complex. Morphologically, they can be either pedunculated or sessile. Clinically, they present with a triad of symptoms: intracardiac obstruction, systemic embolisation, and constitutional symptoms. Echocardiography is the preferred diagnostic tool for assessing cardiac myxomas. Genetic testing can identify mutations in the PRKAR1A gene in familial cases of the condition. Surgical excision is the treatment of choice, and the prognosis after cardiac myxoma resection surgery is excellent, with a very low rate of recurrence. Despite their benign nature, cardiac myxomas pose significant challenges for anesthesiologists due to their variable presentation. This review summarizes the diverse clinical presentations of cardiac myxomas, their implications, the anesthetic challenges they present, and strategies for anesthetic management.

KEYWORDS

Cardiac Tumour, Cardiac Myxoma, Carney Complex.

INTRODUCTION

"HEART is a noble organ to be affected by a neoplasm", but whenever it gets affected mostly is metastatic. Primary Neoplasm of the heart is rare, majority of them are benign (almost three-quarters of total cardiac neoplasm). Cardiac myxoma is the most common primary cardiac tumour, comprising almost 50-85% of total cardiac neoplasm[1]. Papillary fibroelastoma which is the common primary neoplasm of the heart, earlier it was classified as growth, in the WHO cardiac tumours classification[2].

Table 1 ICD-9-O Topographic Coding of Heart Tumors[3]

Benign Tumors	Malignant tumors		
Papillary fibroelastoma	8820/0	Angiosarcoma	9120/3
Myxoma	8840/0	Leiomyosarcoma	8890/3
Fibroma	8810/0	Pleomorphic sarcoma	8802/3
Rhabdomyoma	8900/0	Metastatic neoplasm	8000/6
Adult cellular rhabdomyoma	8904/0	Hematolymphoid tumors	
Lipoma	8850/0	Diffuse large B-cell lymphoma	9680/3
Lipomatous hypertrophy of the atrial septum Lipomatous hamartoma of atrioventricular valve		Fibrin-associated diffuse large B-cell lymphoma	9680/3
Hamartoma			
Hemangioma	9120/0		
Venous Hemangioma	9122/0		
Capillary Hemangioma	9131/0		
Arteriovenous Hemangioma	9123/0		
Cavernous Hemangioma	9121/0		
Cystic tumour of atrioventricular node	8454/0		

MATERIALS AND METHODS

This systemic review was performed in adherence to the preferred reporting items for systemic review guidelines (PRISMA), and the Cochrane Handbook for Systematic Reviews of Interventions.

The language was restricted to English, and relevant search terms were selected from Medical Subject Headings, and Embase subject headings. A comprehensive search was made on the PUBMED search engine from January 2000 to December 2024 for articles having the following keywords 'cardiac myxoma', 'carney complex', 'atrial myxoma', 'clinical feature of cardiac myxoma', 'anaesthetic management of cardiac myxoma'. A total of 2853 Articles were searched and 56 were critically reviewed.

DISCUSSION

Epidemiology/Demography:

Almost all age groups can be affected by myxoma, but it has a greater predilection for the middle-aged population (age group between 30-60 years, mean age 50 years), with female predominance[4] (female: male ratio is 3:1). The Prevalence is 0.03% among the general population.

Cardiac Myxoma can be sporadic or familial. The sporadic form is more common in almost 95% of cases. Sporadic myxomas are usually single. Familial myxoma may be multiple, multicentric, can arise from atypical sites, and could be a part of a "carney complex"

Carney complex [5-6] (CNC) is a group of disorders that present as Lentiginosis (hyperpigmented skin spots), myxoma (mainly cardiac myxoma, but it may involve skin, mouth, breast, and other organs), endocrine tumours (primary pigmented nodular adrenocortical disease, MEN Syndrome, Pituitary gland tumour, schwannomas, etc.). A familial type of cardiac myxoma presents at an early age (mean age 24 years) and is inherited as an autosomal dominant pattern caused by a mutation in protein kinase A regulatory subunit 1 alpha (PRKAR1A) gene on chromosome 17q22-24[7].

Diagnostic Criteria for CNC[8]

Major Criteria
1. Spotty skin pigmentation with typical distribution (lips, conjunctiva and inner or outer canthi, vaginal and penile mucosal) 2. Myxoma** (cutaneous and mucosal) or cardiac myxoma** 3. Breast myxomatosis** or fat-suppressed magnetic resonance imaging findings suggestive of this diagnosis 4. PPNAD** or paradoxical positive response of urinary glucocorticosteroid excretion to dexamethasone administration during Liddle's test

5. Acromegaly as a result of growth hormone (GH)-producing adenoma*
6. LCCSCT** or characteristic calcification on testicular ultrasound
7. Thyroid carcinoma*(at any age) or multiple hypoechoic nodules on thyroid ultrasound in prepubertal child
8. Psammomatous melanotic schwannomas (PMS)**
9. Blue nevus, epithelioid blue nevus (multiple)**
10. Breast ductal adenoma (multiple)**
11. Osteochondromyxoma**
Supplemental criteria
1. Affected first-degree relative
2. Activating pathogenic variants of PRKACA (single base substitutions and copy number variation) and PRKACB (Beuschlein, Fassnacht et al. 2014, Forlino, Vetro et al. 2014)
3. Inactivating mutation of the PRKAR1A gene (Bossis, Voutetakis et al. 2004)
Findings suggestive of or possibly associated with CNC, but not diagnostic for the disease (minor criteria)
1. Intense freckling (without darkly pigmented spots or typical distribution)
2. Blue nevus, common type (if multiple)
3. Café-au-lait spots or other 'birthmarks'
4. Elevated IGF-I levels, abnormal glucose tolerance test (GTT), or paradoxical GH response to TRH (thyrotropin-releasing hormone) testing in the absence of clinical acromegaly
5. Cardiomyopathy
6. History of Cushing's syndrome, acromegaly, or sudden death in extended family
7. Pilonidal sinus
8. Colonic polyps (usually in association with acromegaly)
9. Multiple skin tags or other skin lesions; lipomas
10. Hyperprolactinemia (usually mild and almost always combined with clinical or subclinical acromegaly)
11. Single, benign thyroid nodule in a child younger than age 18 years; multiple thyroid nodules in an individual older than age 18 years (detected on ultrasound examination)
12. Family history of carcinoma, in particular of the thyroid, colon, pancreas, and ovary; other multiple benign or malignant tumours

*To make the diagnosis of CNC, a patient must either: (1) exhibit two of the major criteria confirmed by histology, imaging or biochemical testing or meet (2) one major criterion and one supplemental one **with histologic confirmation

Embryology

Embryologically its origin is thought to be entrapped embryonic foregut, derived from multipotent mesenchymal cells [9]. These cells are able to differentiate into endothelial cells, smooth muscle cells, angioblasts, fibroblasts, cartilage cells, myoblasts neural or epithelial lining.

Site of Origin & Location;

Cardiac myxomas can develop in any chamber of the heart. They most commonly occur in the left atrium (75%), followed by the right atrium (15-20%), and then in the left ventricle (3-4%) and right ventricle (3-4%). Myxomas can be bi-atrial or multi-chambered, and, although rare, they can also arise from heart valves. In the left atrium, myxomas typically form at the limbus of the fossa ovalis, which is part of the interatrial septum. They can also originate from the posterior atrial wall, the anterior atrial wall, or the atrial appendage.

Cardiac Myxoma (Intracardiac location & Its percentage)	
Left atrium	75%
Right atrium	15-20%
Left ventricle	3-4 %
Right ventricle	3-4%

Macroscopic Appearance

Cardiac myxoma is typically smooth, oval, or lobulated in shape, and can be either sessile or pedunculated. In approximately two-thirds of cases, they appear polypoidal, while in one-third of cases, they are villous or papillary. Polypoidal myxomas are usually pedunculated, more compact, and have a lower tendency to fragment and embolize. In contrast, papillary myxomas are generally sessile, gelatinous, less compact, and more fragile, resulting in a higher likelihood of embolizing to areas such as the central nervous system (CNS), sternum, spine, and pelvis.

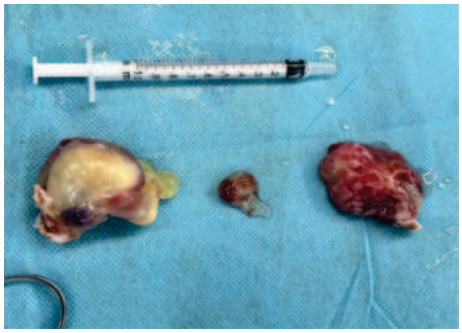


Figure 1 Morphological Appearance of Myxoma

Clinical Presentations

The clinical signs, manifestations, and symptoms of cardiac myxoma (CM) are non-specific and depend on factors such as the size, location, and mobility of the tumour, as well as any associated anomalies and comorbidities. CM is a benign tumour that progresses slowly and does not invade the myocardium. However, due to its abnormal location and the risk of embolization, it can lead to serious complications. Therefore, it is clinically considered to be "functionally malignant".

Most of the patients present with one or more components of its classical Triad of intra-cardiac obstruction, embolism, and constitutional symptoms[10].

Intracardiac Obstruction

The impact of a cardiac mass (CM) depends on its size, mobility, and location. In cases of left-sided atrial myxoma, the mass often obstructs the opening of the mitral valve[11-12], which can present as mitral stenosis. Additionally, it may act as a space-occupying lesion that decreases the filling of the left atrium. If the myxoma is pedunculated, it can move back and forth across the mitral valve, leading to mitral insufficiency[13]. As a result, patients may display symptoms of mitral stenosis (MS) or mitral regurgitation (MR), or both. Symptoms can include dyspnea, pulmonary hypertension, recurrent pulmonary edema, and left heart failure. In some cases, if the cardiac mass is large enough, it can completely obstruct the mitral valve, potentially causing syncope or cardiac arrest.

In right-sided atrial myxoma, the tumour can obstruct the tricuspid valve, presenting as tricuspid stenosis. It may also mimic constrictive pericarditis, leading to functional tricuspid stenosis with elevated right atrial pressure (RAP). If the myxoma is large and pedunculated, it can result in tricuspid valve insufficiency. Additionally, if the foramen ovale is patent, it may lead to a right-to-left shunt, resulting in signs of central cyanosis while obscuring signs of right heart failure [14-15]. Other symptoms may include peripheral edema and fatigue.

In ventricular myxoma, the tumour can obstruct either the left ventricular outflow tract (LVOT) or the right ventricular outflow tract (RVOT), depending on its location within the left or right ventricle. This obstruction may cause syncope.

Embolism

Atypical tumour locations, irregular macroscopic appearances (such as irregular surfaces, fragility, and low collagen content), along with a high platelet count, are significant risk factors for embolization. The papillary type of myxoma has a loose consistency and is particularly fragile, making it more prone to embolization. Overall, embolism occurs in 30% to 40% of cases.

Since cardiac myxomas are most commonly found in the left atrium, systemic embolization is more frequent[16]; it embolizes to the brain[17-18], causing ischemic stroke[19] and possibly leading to cerebral aneurysm formation. Additionally, embolization in the central retinal ophthalmic artery can sometimes result in vision loss[20-21]. Other arteries that may be affected include the coronary, renal, and limb arteries[22-24]. In rare cases, emboli can obstruct coronary artery flow, leading to acute myocardial infarction[25-27].

Right-sided myxomas rarely embolize; however, when they do, it is more commonly into the pulmonary circulation, which may cause pulmonary embolism [28]. There is also a risk of paradoxical embolism if there is a right-to-left shunt present. It may cause right

ventricular outflow tract obstruction [29], pulmonary hypertension, systemic congestion, and right heart failure. Lu C et al [30] report a rare case of Giant right ventricular myxoma presenting as right heart failure with systemic congestion.

Constitutional Symptoms

It presents with a variety of symptoms like anorexia, malaise, fever [31], joint pain (arthralgia), fatigue, weakness (adynamia), myalgia, generalized weakness, weight loss, etc. Recent data suggest that these symptoms are related to the overproduction of interleukin-6 (IL-6) by the tumour and it depends upon the size of the tumour. May sometimes also mimic asthma, or pulmonary tuberculosis.

On examination signs of cachexia, poor weight gain, cyanosis, clubbing, rash, and other endocrine abnormality may be observed. On auscultation, a systolic or diastolic murmur may be heard, depending on the size, location, and mobility of the tumour. When it hampers the filling of ventricles, it produces a diastolic murmur, and when it obstructs the closure of the atrioventricular valve it causes a systolic murmur. Additionally Left atrial myxoma also has a widely split 1st heart sound due to delayed closure of the mitral valve. "Tumor polyps" may be heard in one-third of patients.

Due to the atypical presentation of CM, it is also known as "great imitators" in cardiology[32-33].

Symptoms Due to obstruction
Dyspnoea
Palpitation
Chest pain
Syncope/ dizziness
Orthopnoea
Symptoms Due to embolization
Cerebral
Peripheral
Coronary artery
Retinal artery
Constitutional symptoms
Fever
Appetite/weight loss
Joint Pains
Pedal oedema
Anaemia
Fatigue

Lab Findings

Anaemia (normochromic or hypochromic), leukocytosis, and thrombocytopenia may be present, along with raised interleukin-6 levels. There is an elevated erythrocyte sedimentation rate (ESR), increased C-reactive protein, and serum globulins. The serum tests positive for antinuclear antibodies and IgM rheumatoid factor, which may confuse clinicians regarding autoimmune pathology.

Investigations

ECG findings are non-specific and may reflect the long-term hemodynamic alteration caused by CM manifested as atrial or ventricular hypertrophy. The Rhythm is usually sinus, which contrasts to Mitral stenosis where AF is common. However atrial myxoma may lead to supra ventricular arrhythmia, while ventricular myxoma can result in ventricular arrhythmias.

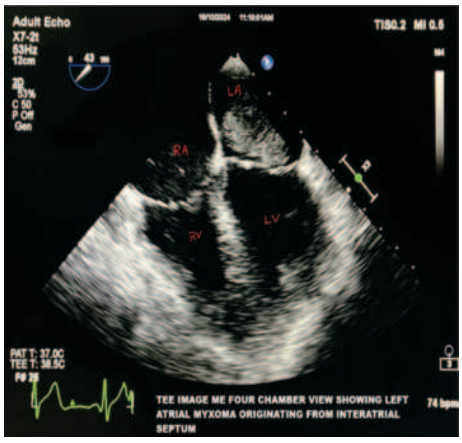
Imaging Modality[34]

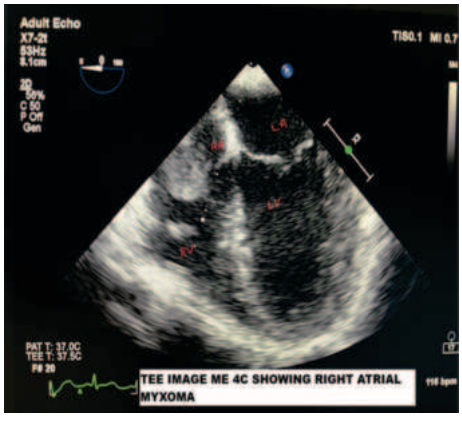
Chest X-ray- Depending on the location and size of the myxoma, cardiac chamber enlargement may be seen. Signs of pulmonary congestion, or pulmonary hypertension might also be present. Fluoroscopic examination allows for better visualisation of calcification and the motion of the tumour.

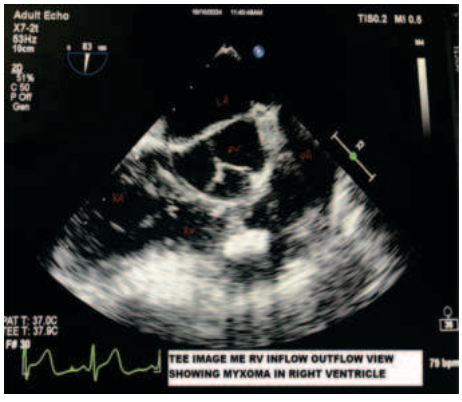
Echocardiography

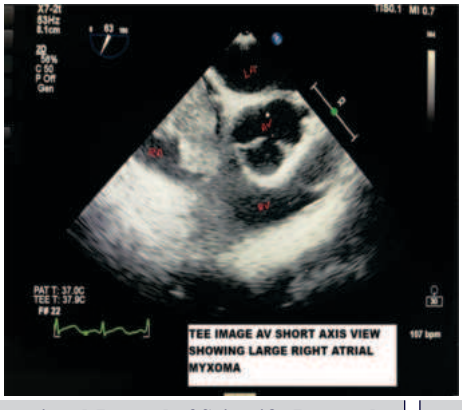
Transthoracic echocardiography (TTE) is the initial and most practical non-invasive investigation; however, its sensitivity in diagnosing myxoma is lower than transesophageal echocardiography (TEE) with a value of 95% for TTE compared to 99-100% for TEE [35-36]. Echocardiography can quantify the shape, size, location, sessile or pedunculated, origin of myxoma. TEE can detect tumours of small size (1mm-3mm), and tumours in abnormal locations [37-38]. Additionally, Doppler echo can also quantify hemodynamic alteration caused by either obstruction in filling the chamber or in closing the atrioventricular valve.

Intracardiac thrombus mimics cardiac myxoma. The gold standard for the diagnosis is a biopsy, although differentiation from thrombus can be done by echocardiography. A thrombus typically appears free-floating in the posterior surface of the atrium, especially in the left atrial appendage. It has a layered appearance and is seen with mitral stenosis, spontaneous echogenic contrast, and atrial fibrillation. In contrast, a myxoma usually has a stalk that mostly originates from the fossa ovalis of interatrial septa, characteristic to and fro mobility.









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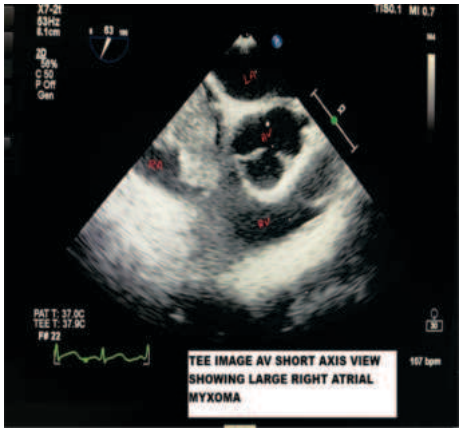

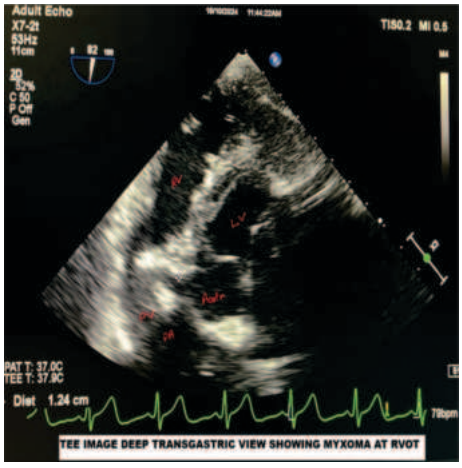

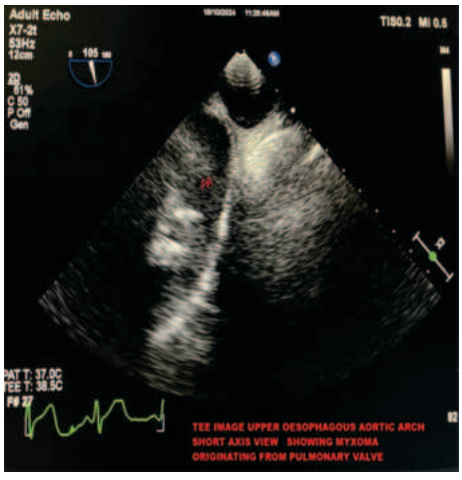

MRI[39]

Magnetic resonance imaging (MRI) can detect its shape, size, surface characteristics, origin, stalk attachment even mobility of myxoma. It can differentiate myxoma from thrombus by tissue characteristics. In MRI, T1 weighted imaging shows isointense mass, but on T2 weighted imaging, it shows hyperintense mass.

CT Scan

It is of limited value in myxoma, as it can't reliably differentiate myxoma from thrombus. However, it can detect calcification in myxoma.

Angiocardiography

Because of the availability of better non-invasive diagnostic modalities i.e. echocardiography, and more complication rate during the procedure, as it may embolize the myxoma, angiocardiography is not routinely done. Cardiac myxoma presents with a filling defect in angiocardiography.

Genetic Testing

Familial myxoma, which is associated with the Careny complex has autosomal dominant inheritance due to mutation in the PRKAR1A gene [40-42], so genetic testing for mutation in the PRKAR1A gene can be done.

Anaesthetic Consideration and Treatment

There is currently no effective medical treatment for cardiac myxoma. The preferred treatment is surgical excision with adequate margins [43]. Once a diagnosis of myxoma is confirmed, it should be removed urgently through either a mini-thoracotomy [44], mini-sternotomy, or median sternotomy [45] while the patient is on cardiopulmonary bypass. This urgency is necessary due to the high risk of embolization associated with the condition.

Intraoperative challenges associated with cardiac myxoma are influenced by several factors, including the tumour's size, shape, location, fragility, and the symptoms it produces, such as obstruction to blood flow in the heart chamber or atrioventricular valve insufficiency. Right-sided myxomas carry a higher risk of fragmentation and embolization, particularly during procedures like central line or pulmonary artery catheter insertion. Patients with a history of pre-operative stroke or pulmonary embolism are at greater risk as well. Additionally, acute coronary syndrome may arise if a myxoma embolizes into the right or left main coronary artery ostium. Therefore, it is essential to conduct a thorough pre-operative assessment to improve postoperative outcomes.

When familial myxoma is linked to the Carney complex, it can lead to additional challenges due to its associated features. Patients with this condition often have multiple intracardiac lesions and are at a higher risk of recurrence even after undergoing adequate surgical resection. The presence of various endocrinopathies also significantly influences intraoperative management, particularly with Primary Pigmented Nodular Adrenocortical Disease, which is the most common endocrinopathy associated with the Carney complex, and Growth Hormone-Producing Pituitary Adenoma[46].

Cushing syndrome is associated with primary pigmented nodular adrenocortical disease (PPNAD), so a stress dose of steroids is administered in cases where adrenalectomy has occurred. Patients with growth hormone-secreting pituitary adenomas present with acromegaly[47], which can lead to anticipated challenges in airway management. Additionally, these patients often have a higher prevalence of hypertension, impaired blood glucose levels, and altered fluid balance, all of which can complicate anaesthetic management.

Identifying structural heart disease and comorbidities in patients during the pre-anesthetic assessment is crucial for effective peri-operative management. It is important to consider the risk of embolization to the brain and abdominal organs, such as the spleen, liver, and kidneys. If there is infected CM, the risk of dislodging infected thrombi is significant, which may lead to abscess formation in target organs like the brain, spleen, liver, and kidneys. To prevent infective endocarditis, pre-operative blood cultures and appropriate antibiotic therapy should be administered.

Cardiac myxoma often results in impaired filling of the left or right ventricle due to its space-occupying nature. This condition can present with symptoms such as dyspnea, recurrent pulmonary edema, congestive heart failure, and even death. These signs can mimic those of atrioventricular stenosis, specifically either mitral stenosis or tricuspid stenosis. Consequently, the management plan for cardiac myxoma is similar to that of mitral stenosis, focusing on avoiding tachycardia, optimizing preload, and maintaining atrial rhythm. Tachycardia can reduce diastolic time, which in turn decreases cardiac filling and increases pulmonary artery pressure, potentially leading to pulmonary edema. Preoperatively, a beta-blocker such as metoprolol is often considered before induction[48-49].

In right atrial myxoma, patients exhibit symptoms of hypoxemia and a low cardiac output[50]. There may be a left-to-right shunt and paradoxical embolization[51]. Pulmonary artery catheterization is contraindicated, and central venous line placement should be performed cautiously[52-54].

All ASA-recommended monitors, such as continuous ECG, SpO2, invasive arterial blood pressure, central venous pressure monitoring, temperature monitoring, and NIRS, should be attached.

Goals during the induction of anaesthesia include:

1. Constant monitoring of systemic blood pressure.
2. Ensuring adequate preloading.
3. Avoiding tachycardia.
4. Maintaining sinus rhythm.
5. Judicious use of vasoactive drugs to optimize blood pressure.

To address hemodynamic instability, the following methods can be employed:

- Infusion of 100-200 ml of crystalloid solution preoperatively.
- Administration of intermittent bolus doses of phenylephrine (50 micrograms) or methoxamine (1-2 mg) until adequate preload and hemodynamic stability are achieved.
- Positioning the patient in the Trendelenburg position, which redistributes pooled venous blood from the lower limbs into the central circulation. This position can also mobilize cardiac myxoma and help relieve valvular obstruction, thereby optimizing preload and increasing cardiac output.

Opioid based Induction is typically performed, after giving an adequate dose of muscle relaxant, three minutes of intermittent positive pressure ventilation (IPPV) with 100% oxygen should be given. The Airway should then be secured with a cuff endotracheal tube of appropriate size. TEE should be placed after intubation, and USG guided CVC line should be inserted. The usual protocol of cardiac anaesthesia should be followed. After heparinisation and achieving adequate ACT i.e. 420 or more patient can be put on CPB. Cardiac myxoma should be excised with sufficient margin, which is curative in most cases. However familial type of myxoma have a higher tendency to relapse.

Postoperative complications are few, including rhythm disturbances (most commonly atrial fibrillation), neurological disorders, and myocardial infarction. The overall motility during and within 30 days after surgery ranges from 0% to 10%, the relapse rate spans from 0% to 7%, and long-term survival rates are between 86% and 95%[55-56]

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