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Pathology Section

# Histopathological Variants of Atrial Myxoma with Emphasis on Glandular Differentiation: A Cross-sectional Study

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## ABSTRACT

**Introduction:** Atrial myxomas are rare benign cardiac tumours with a variable clinical presentation, including obstructive symptoms, systemic embolisation, and constitutional manifestations. Echocardiography is the primary diagnostic modality, but histopathology remains the gold standard for confirmation. While most myxomas display a classic myxoid matrix with stellate cells, glandular differentiation is an uncommon variant that can mimic adenocarcinoma, necessitating immunohistochemical analysis. This study examines the histopathological diversity of atrial myxomas and its clinical implications.

**Aim:** The aim of the present study was to analyse the histopathological spectrum of atrial myxomas, identify rare variants, and differentiate them from potential mimickers to aid accurate diagnosis and clinical management.

**Materials and Methods:** The present retrospective cross-sectional study of 12 atrial myxoma cases was conducted at Jagjivan Ram Hospital, Mumbai, from January 2018 to December 2024. Gross and microscopic features were examined, supplemented by special stain (Alcian blue) and Immunohistochemistry (IHC) using markers such as (Cytokeratin 7 (CK7), Cytokeratin 20 (CK20), Caudal Type Homeobox 2 (CDX2), Carcinoembryonic Antigen (CEA), Calretinin, Cluster of Differentiation 31 (CD31), and Ki-67 protein (Ki-67) proliferation index. The findings were analysed for histopathological variations and their diagnostic significance.

**Results:** Eleven out of 12 cases involved the left atrium, and one case had bilateral atrial myxomas. Grossly, tumours were gelatinous and grey-white, with villous or lobulated surfaces. Microscopically, all cases showed myxoid stroma with stellate (lepidic) cells, haemosiderin-laden macrophages, and fibrosis. One case displayed glandular structures lined by columnar cells with mucin droplets, diagnosed as glandular myxoma. IHC confirmed CK7 and calretinin positivity in glandular areas, differentiating it from adenocarcinoma.

**Conclusion:** Atrial myxoma presents with diverse clinical manifestations and potential complications. Accurate histopathological evaluation is essential for diagnosis, differentiation from adenocarcinoma, and detection of lymphovascular emboli. Recognising histological variants and mimickers is crucial for guiding management and preventing misdiagnosis.

**Keywords:** Cardiac tumour, Echocardiography, Immunohistochemistry, Systemic embolisation

## INTRODUCTION

Cardiac myxomas are primary neoplasms of the heart that can involve any of the cardiac chambers, with the left atrium being the most common site. An unusual feature of cardiac myxomas is their biological potential to embolize and grow at the site of embolisation, potentially causing organ infarction [1]. Timely diagnosis and treatment are essential to prevent sometimes life-threatening complications. Although well-known, some aspects of cardiac myxoma are still evolving.

Immunohistochemical studies suggest that myxoma cells originate from multipotent mesenchymal cells, which have the capacity to differentiate into both neural and endothelial lineages [2]. Approximately 10% of myxomas are associated with Carney's complex, an inherited autosomal dominant disorder, while the remaining cases appear to be sporadic. The precise aetiology of atrial myxoma remains under investigation [3].

Cardiac myxoma is a rare tumour with an annual incidence of approximately 0.5 to 1 case per million individuals. Although it can occur at any age, it most commonly affects middle-aged individuals and shows a marked female predominance, with a female-to-male ratio of about 3:1 [4,5]. There are two epidemiological forms of cardiac myxoma: sporadic and familial. The sporadic form is significantly more common, constituting about 95% of all cases [6]. It is the most common primary cardiac tumour, accounting for 50% to 85% of benign cardiac tumours [6,7].

The recurrence rates are 1% to 3% in sporadic cases, 12% in familial cases, and 22% in complex atrial myxomas [8]. The prognosis for patients undergoing surgical resection of atrial myxomas is excellent; the operative mortality rate does not exceed five percent, with rapid postoperative recovery [8,9]. One study suggests that minimal tumour manipulation, excision with adequate margins, and careful inspection of all heart chambers are important measures to prevent the recurrence of tumours.

Cardiac tumours are rare, with an incidence ranging from 0.0017% to 0.03%, with atrial myxoma being the most prevalent. These benign neoplasms originate from multipotent mesenchymal cells [10]. The association between myxomas and hypercoagulability is intricate, involving various mechanisms that contribute to thrombotic risk. If left untreated, myxomas tend to progress relentlessly and can become life-threatening [11].

Echocardiography is the primary diagnostic tool for atrial myxomas, typically revealing a mobile, echogenic mass with a polypoid or papillary appearance, attached to the interatrial septum by a stalk. Polypoid myxomas often present with obstructive features, whereas papillary forms have a higher risk of embolisation. Cardiac Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) scans provide additional diagnostic value, helping to differentiate myxomas from other primary cardiac tumours and thrombi [12].

Surgical resection remains the definitive treatment. Antiplatelets and anticoagulants such as warfarin have traditionally been used for secondary stroke prevention but have not been effective in preventing recurrent embolic events [13].

The objective of this study is to analyse the histopathological spectrum of atrial myxoma. It aims to identify rare variants and distinguish them from potential mimickers, thereby aiding in accurate diagnosis and effective management.

## MATERIALS AND METHODS

The present observational, retrospective cross-sectional analysis of atrial myxomas at Jagjivan Ram Hospital, Mumbai, was conducted from January 2018 to December 2024. A total of 12 atrial myxoma cases were included in the study. Gross and microscopic features were examined, supplemented by special stain (Alcian blue) and IHC using markers such as CK7, calretinin, CK20, CDX2, CEA, CD31, and the Ki-67 proliferation index. The findings were analysed for histopathological variations and their diagnostic significance.

**Inclusion criteria:**

- Cases with histopathologically confirmed atrial myxoma.
- Availability of adequate Formalin-Fixed Paraffin-Embedded (FFPE) tissue blocks for further analysis.
- Detailed clinical records, including symptom presentation, imaging, and surgical outcomes.

**Exclusion criteria:**

- Cases with insufficient tissue samples for histopathological/ IHC evaluation.
- Patients with known metastatic adenocarcinoma to avoid confounding results.
- Cases with inadequate clinical follow-up data or incomplete records.

### Study Procedure

- The study aimed to provide a comprehensive analysis of tumour characteristics and clinical features of atrial myxoma.
- Histopathological features assessed included:
  - -Tumour architecture: classical, glandular, and inflammatory variants.
  - -Cell type: presence of spindle vs. stellate cells.
  - -Cell morphology and presence of nuclear atypia.
  - -Glandular differentiation mimicking adenocarcinoma.
  - -Inflammatory infiltrates and hemosiderin-laden macrophages.
- Clinical correlation included:
  - -Demographic data: age and sex.
  - -Presenting symptoms: chest pain, shortness of breath, dizziness, etc.
  - -Classification into embolic vs. non-embolic manifestations.
  - -Tumour size and location (left atrium, right atrium, or atypical sites).
- Haematological parameters studied:
  - -Platelet count, neutrophil count, Prothrombin Time/ International Normalised Ratio (PT/INR), and Mean Platelet Volume (MPV).
  - -Postoperative follow-up was conducted to evaluate for recurrence in available cases.

**Immunohistochemistry (IHC) analysis:** IHC was performed on FFPE sections using the Avidin-Biotin Complex (ABC) method. The markers analysed included CK7, CK20, CDX2, CEA, calretinin, CD31, and the Ki-67 proliferation index.

## STATISTICAL ANALYSIS

The mean age and tumour size were calculated. The percentage of histologic variants was determined, and comparative analysis was performed using crosstabulation.

## RESULTS

The mean age of patients with cardiac myxoma was 53.25 years. The mean tumour size was 3.7 cm. The percentage of males was 50%, and females were also 50%. Clinically, out of the 12 cases, 1 case presented with neurological symptoms such as weakness and syncope due to embolisation. Out of the 12 cases, 11 had myxomas located in the left atrium, while one case involved myxomas in both the right and left atrium. Grossly, four cases were larger than 5 cm (large myxomas), while the remaining eight cases were between 1 to 5 cm. Of the total cases, eight were globular, one was villous, and three were friable polypoid [Table/Fig-1].

| S. No. / Case No. | Age (years) | Sex | Clinical symptoms | Location | Gross | Diagnosis | Haematological Parameters -Neutrophil & Platelet Count -PT/INR -MPV | Follow-up For Recurrence |
|---|---|---|---|---|---|---|---|---|
| 1. | 70 | Female | Chest pain, syncope, weight loss | Right and left atrium | Soft-tissue -globular 8x7x3cm | Classical myxoma | Platelet-150000 Neutrophil -65 PT/INR-1.35 MPV-8.5 | Absent |
| 2. | 53 | Male | Dizziness, palpitation, anorexia | Left atrium | Blackish soft friable tissue with outer smooth irregular measures 3.5×3×2.5cm | Classical myxoma | Platelet-1450000 Neutrophil -66 PT/INR-1.12 MPV-8.0 | Absent |
| 3. | 76 | Male | Weakness in legs, difficulty in breathing when lying down | Left atrium | Soft to firm ,villous multiple tissue fragments aggregating, irregular measures 7×7×3 cm | Classical myxoma | Platelet-1,50,000 Neutrophil- 70 PT/INR -1.1 MPV-7.6 | Absent |
| 4. | 51 | Female | Chest pain, palpitations, fever | Left atrium | Two globular brown soft-tissue pieces 2×1.5×1 cm & 1.5×1×0.5 cm | Classical myxoma | Platelet-128000 Neutrophil -69 PT/INR-1.22 MPV-7.8 | Absent |
| 5. | 64 | Male | Tightness in chest, night sweat, palpitaions | Left atrium | Globular brown soft-tissue piece 6.5x5x3cm.gelatinous greyish brown areas mixed with greyish white areas noted. | Classical myxoma | Platelet -125000 Neutrophil- 73 PT/INR-1.25 MPV-7.6 | Absent |
| 6. | 30 | Female | Fainting ,dyspnea,palpitations | Left atrium | One soft-tissue 7×3×2.5 cm.external surface is irregular with papillary fronds. cut surface show variegated appearance with whitish and brown areas. | Glandular myxoma | Platelet-140000 Neutrophil - 71 PT/INR-1.14 MPV-8.4 | Follow-up not done |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 7. | 39 | Female | Dizziness, palpitations, anorexia | Left atrium | Soft spongy jelly like tissue pieces 3x3x0.5 cm. External surface is brown | Classical myxoma | Platelet - 1350000<br>Neutrophil -77<br>PT/INR-1.12<br>MPV-10.2 | Absent |
| 8. | 45 | Male | Chest pain, syncope,weight loss | Left atrium | Soft-tissue -globular 3x2.5x1cm | Classical myxoma | Platelet-156000<br>Neutrophil -80<br>PT/INR-1.22<br>MPV-9.2 | Absent |
| 9. | 67 | Female | Shortness of breathing,orthopnea, | Left atrium | Soft-tissue-globul 3x3x0.5 cm | Classical myxoma | Platelet-160000<br>Neutrophil -65<br>PT/INR-1.11<br>MPV-8.5 | Absent |
| 10. | 35 | Male | Chest pain, syncope,weight loss | Left atrium | Soft-tissue -globular 2.5x3cm | Classical myxoma | Platelet-180000<br>Neutrophil- 61<br>PT/INR-1.12<br>MPV-7.6 | Absent |
| 11. | 68 | Male | Fainting, dyspnea,palpitations | left atrium | Soft-tissue -globular 1.5x2x1cm | Classical myxoma | Platelet-178000<br>Neutrophil -74<br>PT/INR-1.13<br>MPV-10.2 | Absent |
| 12. | 41 | Female | Tightness in chest, nightsweat, palpitaions | Left atrium | Soft-tissue -globular 1x1x1cm | Classical myxoma | Platelet-156000<br>Neutrophil -79<br>PT/INR-1.24<br>MPV-8.0 | Absent |

**[Table/Fig-1]:** Summary of clinical, gross features, haematological parameters and histopathology variant of myxoma.

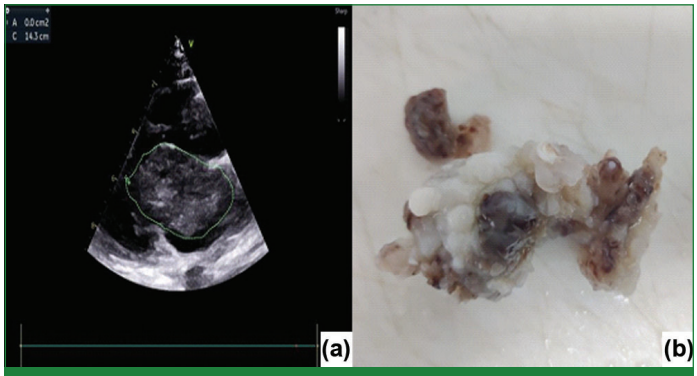
Most tumours (11/12) exhibited classical architecture, comprising stellate and spindle-shaped myxoma cells embedded in a myxoid matrix. One case showed glandular differentiation, characterised by gland-like spaces with mucin production, raising a differential diagnosis of adenocarcinoma. Among the tumour cells, stellate morphology was predominant (8 cases), while spindle-shaped cells were seen in four cases. Nuclear atypia with variation in cell size and shape was noted in only one case. Inflammatory infiltrates, composed of lymphocytes and macrophages, along with hemosiderin-laden macrophages, were observed in 11 cases, representing a common secondary feature [Table/Fig-2].

| Features | Description | Total cases |
|---|---|---|
| Tumour architecture | Classical<br>Glandular | 11<br>1 |
| Morphology of tumour cell | spindle<br>stellate | 4<br>8 |
| Cell morphology & nuclear atypia | Variation in cell shape, size, and presence of nuclear atypia | 1 |
| Glandular differentiation | Glandular architecture with mucin | 1 |
| Inflammatory infiltrates & hemosiderin deposits | Presence of lymphocytes, macrophages, and haemosiderin-laden deposits | 11 |

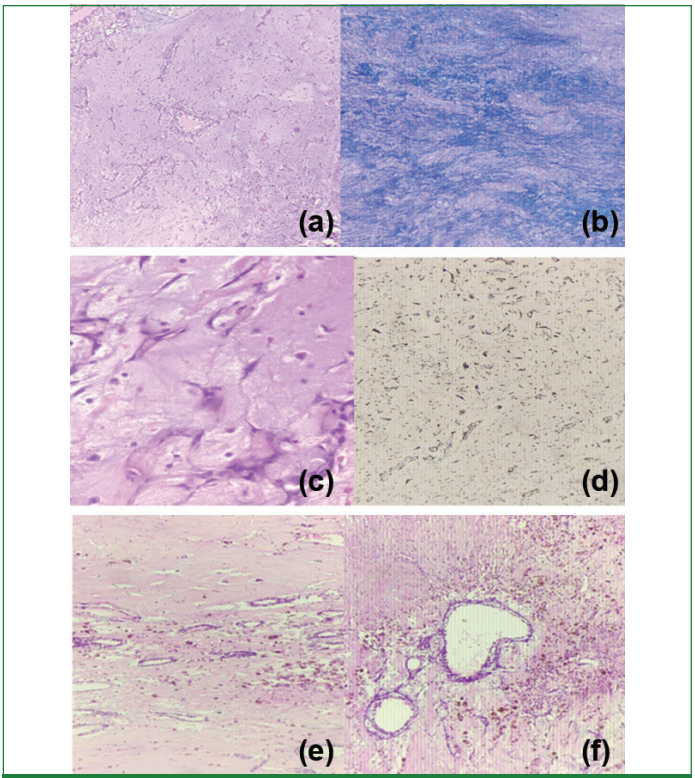
**[Table/Fig-2]:** Summarising the key histopathological features of cardiac myxomas.

A Two Dimensional (2D) echocardiogram shows a well-defined, mobile, pedunculated mass in the left atrium, attached to the interatrial septum, often prolapsing into the mitral valve during diastole. The mass appears heterogeneous in echotexture, with possible areas of cystic change or calcification [Table/Fig-3a]. The received tissue was grossly fragmented, varying in size, with a greyish-white, shiny appearance and areas of haemorrhage [Table/Fig-3b].

The predominant histopathological feature was a rich myxoid background with stellate myxoma (lepidic) cells. These cells exhibited abundant eosinophilic cytoplasm, unclear cell borders, and oval nuclei, observed in approximately 90% of cases. There was no mitosis. Both thin and thick-walled blood vessels were present [Table/Fig-4a-d]. One case displayed lymphovascular emboli. Hemorrhagic areas, hemosiderin-laden macrophages, and areas of fibrosis were noted. An inflammatory infiltrate comprising lymphocytes and plasma cells was seen in the perivascular area [Table/Fig-4e,f].

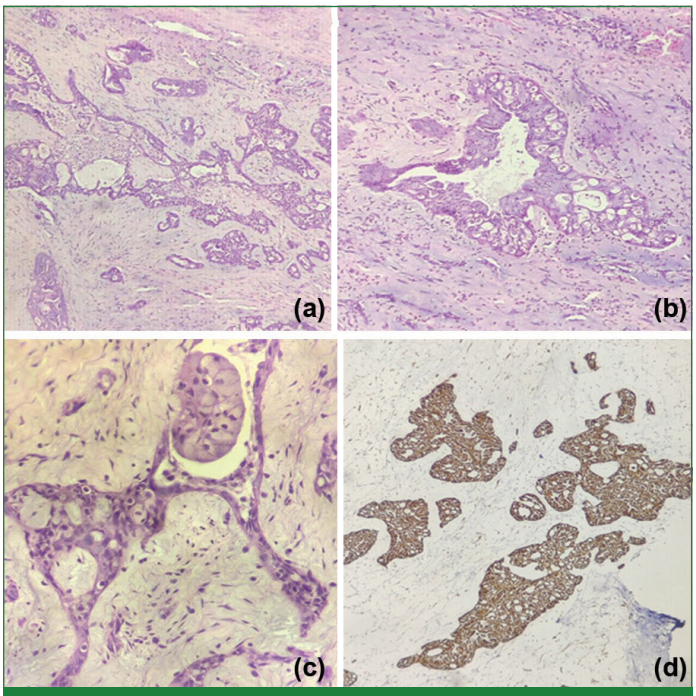



**[Table/Fig-3]:** a) A 2D Echo of cardiac myxoma, pedunculated mass; b) Gross soft globular friable tissue greyish white to brown.




**[Table/Fig-4]:** a) (H&E, 100X) myxoid stroma; b) Alcian blue, (100X) staining acidic mucopolysaccharide; c) (H&E, 400X) stellate cells; d) (100X) IHC calretinin positive in stellate cells; E) (HE, 100X) Fibrosis; F) (HE, 400X) Hemosiderin laden macrophages, inflammatory cells.
H&E: Haematoxylin and Eosin

One case (8.3%) demonstrated irregular glandular formations, trabecular patterns, and epithelial cell islands with mucin-containing lumina. The glands were lined by columnar cells showing abundant eosinophilic cytoplasm and irregular, hyperchromatic nuclei, consistent with a diagnosis of the glandular variant of cardiac myxoma a rare subtype accounting for 2-5% of all myxomas. IHC confirmed the diagnosis, with the glandular components staining positive for CK7 and calretinin [Table/Fig-5a-d], while being negative for CK20, CDX2, CEA, and CD31, helping to differentiate it from metastatic adenocarcinoma. The Ki-67 proliferation index was less than 1%.




**[Table/Fig-5]:** a) (H&E, 100X) Infiltrative appearance of epithelial cells; b) (H&E, 400X) Intraluminal mucin in glandular structure. c) (H&E, 400X) Cuboidal to columnar cells with mucin droplets; d) (400x) IHC positive for CK7.

## DISCUSSION

Cardiac myxomas are rare primary heart tumours, and their exact prevalence is not well established. However, it is reported to be 0.03% in the general population, making up 50% to 85% of all benign cardiac tumours. Epidemiologically, atrial myxomas are more commonly seen in females, particularly between the ages of 30 and 70. While the majority of cases arise sporadically, some are linked to genetic disorders such as Carney's complex, which is associated with specific mutations [11].

Although benign in nature, their clinical behaviour can be regarded as malignant due to the risk of cardioembolic complications. Surgical excision is the standard treatment, offering favourable outcomes when performed without delay [14,15].

Clinically, myxomas manifest with diverse symptoms, including blood flow obstruction, systemic embolism, and constitutional signs like fever and weight loss. Systemic embolisation occurs in approximately 30-40% of cases, affecting cerebral, peripheral, or visceral circulation [16]. This can lead to serious complications such as transient ischemic attacks or pulmonary embolism. Routine laboratory tests may reveal anaemia, leukocytosis, thrombocytopenia, elevated erythrocyte sedimentation rates, and increased gamma globulin levels [17].

These tumours vary in size and shape but are generally soft and pedunculated. Their fragile nature makes them prone to fragmentation and embolisation, especially during handling or surgical intervention. The risk of post-resection embolism is higher in cases with larger tumour size, pedunculated attachment, multiple tumours, or a history of embolic events. Therefore, diligent postoperative surveillance using echocardiography and multimodal cardiac MRI is essential.

Histologically, atrial myxomas are characterised by neoplastic (lepidic) cells dispersed within a myxoid stroma. These cells are typically polygonal to stellate in shape, with abundant eosinophilic cytoplasm, ill-defined borders, and possess an oval nucleus with open chromatin and a small or inconspicuous nucleolus. The glandular variant of cardiac myxoma, seen in about 2-5% of cases, features glands lined by cuboidal to columnar cells with nuclear atypia and mucin, closely resembling adenocarcinoma. If not recognised, it may be mistaken for metastatic carcinoma, especially in patients with a known primary, leading to unnecessary imaging, IHC, extended work-up, patient anxiety, and increased healthcare costs.

IHC plays a crucial role in confirming glandular atrial myxoma and differentiating it from primary adenocarcinoma, with the glandular areas typically showing cytokeratin positivity. The main differential diagnosis includes mural thrombus with myxoid changes. Mural thrombi with myxoid stroma can mimic atrial myxomas histologically, making them an important differential. Calretinin, a myxoma-specific marker, aids in distinguishing the two. Other malignancies such as cardiac sarcomas and primary cardiac or large B-cell lymphomas can also resemble myxomas, further complicating the diagnosis. Although echocardiography aids in detecting cardiac masses, a definitive diagnosis relies on histopathological evaluation [18].

Studies have been conducted correlating histopathological features with clinical symptoms. Sotoudeh Anvari M et al., found that anaemia was more common in papillary tumours (75%) than in solid tumours (13.3%) (p=0.037) [18]. Neutrophils were observed in nine solid tumours and one papillary tumour, with three solid tumours showing Gamma-Gandy bodies. Neutrophil counts in tumour tissue showed no significant difference between patients with and without chest pain (p=0.057).

Further research integrating histopathology with molecular markers is needed to enhance our understanding of pathogenesis and guide treatment planning. Postoperative follow-up is crucial for detecting recurrence and evaluating embolisation risk. Routine monitoring through echocardiography and haematological parameters, such as MPV and platelet count, can aid in identifying potential thromboembolic events.

### Limitation(s)

- The sample size in this study is limited to 12 cases, restricting the generalizability of findings.
- Clinicopathological correlation with individual symptoms, such as chest pain, dizziness, and shortness of breath, was not assessed.
- Postoperative follow-up data were not available for two patients, and in one case, tumour recurrence could not be assessed due to loss to follow-up.
- IHC was performed in only one case (Case 6), which showed glandular differentiation. The remaining 11 cases displayed classical gross and histopathological features of atrial myxoma, and thus IHC was not deemed necessary. According to established diagnostic criteria, IHC is not routinely required for morphologically typical myxomas without atypia or diagnostic ambiguity. In Case 6, IHC was warranted to rule out metastatic adenocarcinoma and other gland-forming neoplasms due to the presence of glandular structures with mucin. This selective and indication-based use of IHC aligns with standard pathology practices, ensuring judicious application of ancillary studies.

## CONCLUSION(S)

Atrial myxoma presents a wide range of clinical manifestations and potential complications. An interdisciplinary approach is crucial for optimising patient outcomes. Histopathological examination plays a key role in confirming the diagnosis, identifying rare variants of glandular myxoma that may resemble adenocarcinoma, detecting lymphovascular emboli, and ruling out the presence of a thrombus.

When examining emboli histopathologically, atrial myxoma and its mimickers should be considered.

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