



Cardiomyopathies



Restrictive Cardiomyopathy (RCM)	Hypertrophic Cardiomyopathy (HCM)	Dilated Cardiomyopathy (DCM)	Feature
Stiff ventricles with ↓ compliance → impaired diastolic filling	Increased LV wall thickness (no dilation) with diastolic dysfunction	Dilated, poorly contractile LV with normal or thin walls	Definition
Severe diastolic dysfunction due to rigid walls	Diastolic dysfunction (impaired relaxation) ± LV outflow obstruction	Systolic dysfunction (ineffective contraction)	Primary Problem
Normal or slightly enlarged atria , normal ventricles	Small LV cavity (nondilated)	Dilated ventricles + atrial enlargement	Chamber Size
Normal or slightly thickened depending on cause	Markedly thickened (massive hypertrophy)	Normal or thin	Wall Thickness
Amyloidosis, endomyocardial fibrosis, radiation, hemochromatosis, sarcoidosis	Autosomal dominant mutations (β-myosin heavy chain, myosin-binding protein C, troponin T)	Genetic (20–50%), infections (coxsackie), alcohol, pregnancy, toxins (Adriamycin), nutritional deficiency	Common Causes
Firm, stiff myocardium; thick endocardium (fibrosis or amyloid)	Massive hypertrophy, no dilation	Globular/flabby heart, dilated chambers, mural thrombi	Pathology (Gross)
Amyloid deposits or dense subendocardial fibrosis	Myocyte disarray + interstitial fibrosis	Myocyte hypertrophy + interstitial fibrosis (nonspecific)	Histology
Signs of right/left heart failure, marked exercise intolerance	Exertional dyspnea, angina, syncope, sudden death in athletes	Slowly progressive CHF, dyspnea, fatigue, arrhythmias, emboli	Clinical Presentation
S4 common	Harsh systolic murmur (due to LVOT obstruction)	S3 common	Heart Sounds
Severe CHF, arrhythmias	Sudden cardiac death; A-fib; CHF; myocardial ischemia	CCF, secondary mitral regurgitation, arrhythmias, thromboembolism	Complications
Treat underlying cause, manage heart failure	Beta-blockers, septal reduction, ICD for sudden death	Heart transplant (definitive), manage CHF	Treatment
Stiff noncompliant heart + restrictive filling	Massive hypertrophy + diastolic failure + myocyte disarray	Dilated weak heart + systolic failure	Key Distinguishing Feature

DCM



- ◆ Dilated chambers
- ◆ Thin/normal walls
- ◆ Systolic dysfunction
- ◆ Causes: alcohol, viruses, pregnancy, Adriamycin
- ◆ Complication: mural thrombi

HCM

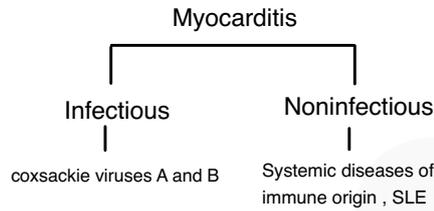
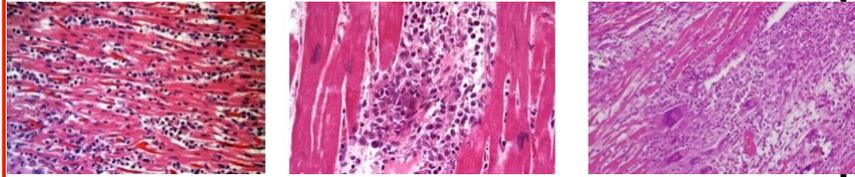
- ◆ Thick walls, small cavity
- ◆ Diastolic dysfunction
- ◆ Genetic (AD) – myosin heavy chain
- ◆ Myocyte disarray
- ◆ Sudden death in athletes

RCM

- ◆ Normal size ventricles but stiff
- ◆ Severe diastolic dysfunction
- ◆ Amyloidosis, endomyocardial fibrosis
- ◆ Impaired filling with normal systolic function (early)

Gross & Histological features

Myocarditis



Gross Morphology

- In acute myocarditis, the heart may appear normal or dilated.
- In advanced stages, the myocardium typically is flabby and pale and hemorrhagic areas.
- Mural thrombi may be present.

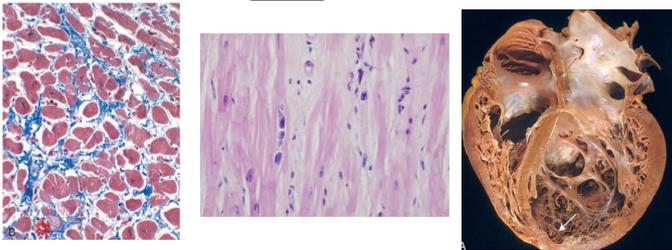
Histological features

myocarditis is characterized by:

- edema and myocyte injury.
- interstitial inflammatory infiltrates:
- Lymphocytic type: numerous lymphocytes.
- hypersensitivity myocarditis: abundant eosinophils.
- Giant cell myocarditis: containing multinucleate giant cells

Gross & Histological features

DCM



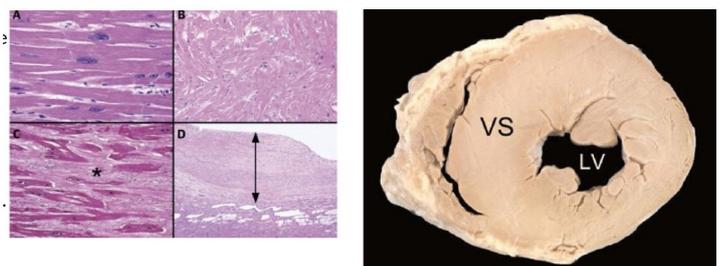
Gross Morphology

- The heart assumes a globular shape.
- ventricular chamber dilatation.
- Atrial enlargement.
- Mural thrombi are often present and may be a source of thromboemboli

Histological features

- The characteristic histologic abnormalities in DCM are nonspecific.
- Myocytes exhibit hypertrophy with enlarged nuclei.
- Interstitial and endocardial fibrosis.

Hypertrophic Cardiomyopathy



Histological features

- The characteristic histologic features in HCM are marked myocyte hypertrophy, haphazard myocyte (and myofiber) disarray, and interstitial fibrosis
- A) Myocyte hypertrophy.
- (B) myocyte disarray.
- (C) interstitial (pericellular-type) fibrosis (asterisk).
- (D) endocardial fibrosis (double-headed arrow).

Gross MORPHOLOGY

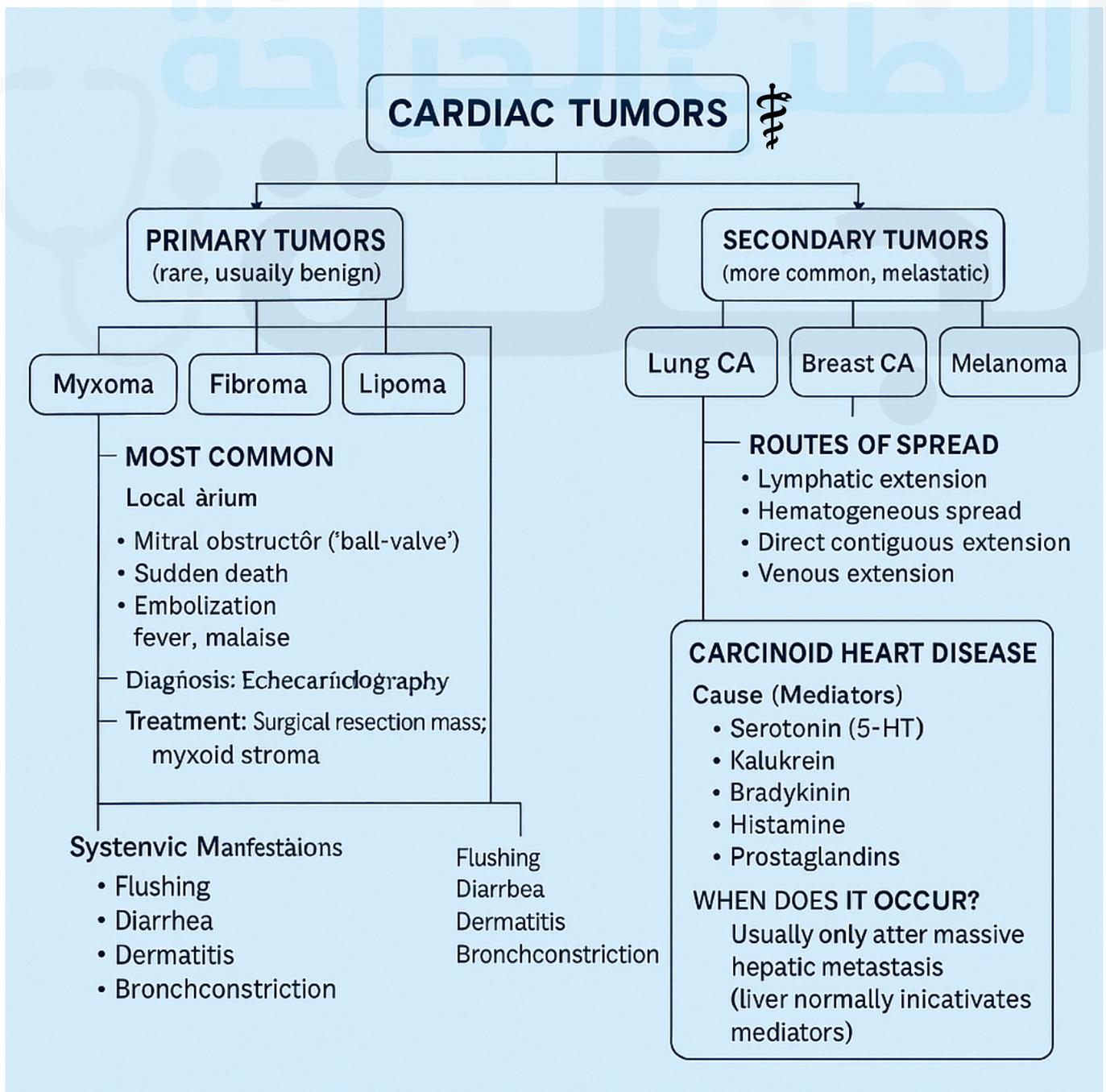
- Hypertrophic cardiomyopathy is marked by massive myocardial hypertrophy without ventricular dilation.

HFrEF (Heart Failure with Reduced Ejection Fraction)

- The heart cannot contract well (weak pumping).
- Systolic dysfunction.
- Ejection fraction is low (<40%).
- Common cause: Dilated cardiomyopathy.

HFpEF (Heart Failure with Preserved Ejection Fraction)

- The heart contracts normally, but cannot relax and fill properly.
- Diastolic dysfunction.
- Ejection fraction is normal or preserved ($\geq 50\%$).
- Common causes: Hypertrophic and restrictive cardiomyopathy.



Cardiac Tumors (Reorganized & Clarified)



Overview

Cardiac tumors are rare and include both primary and secondary (metastatic) tumors.

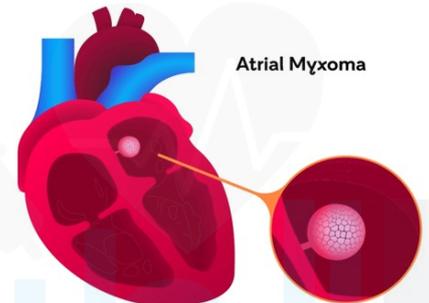
1. Primary Cardiac Tumors

Primary cardiac tumors are uncommon and are usually benign. Their frequency (from most to least common):

1. Myxomas – most common primary cardiac tumor.
 2. Fibromas
 3. Lipomas
 4. Papillary fibroelastomas
 5. Rhabdomyomas – most frequent in infants and children; often regress spontaneously.
- Most common primary malignant tumor: Angiosarcoma

Myxoma

Most common primary tumor of the heart.
Usually single in sporadic cases.
Most commonly located in the left atrium.



Complications

Can cause sudden death, usually due to mitral valve obstruction.

Clinical Manifestations

- Valvular “ball-valve” obstruction
- Embolization
- Fever and malaise

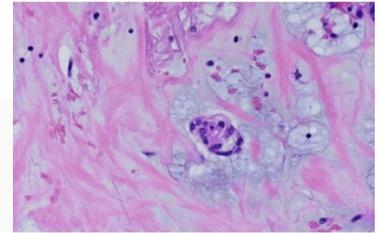
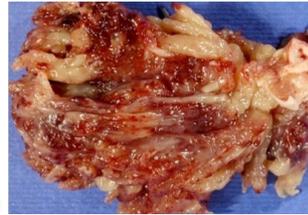
Diagnosis

Echocardiography is the diagnostic modality of choice.

Treatment

Surgical resection is almost always curative.

Morphology



Gross: sessile or pedunculated mass.

Microscopic: neoplastic cells within myxoid stroma.

2. Secondary Cardiac Tumors (Metastatic Tumors)

These are more common than primary tumors.

Most Frequent Metastatic Sources

- Lung carcinoma
- Breast carcinoma
- Melanoma
- Leukemias & lymphomas

Routes of Metastatic Spread to the Heart

- Lymphatic extension
- Hematogenous seeding
- Direct contiguous extension
- Venous extension

Carcinoid Heart Disease

Caused by bioactive compounds (e.g., serotonin) released by carcinoid tumors (neuroendocrine tumors).

Important Concept

Cardiac lesions usually do not develop until there is massive hepatic metastasis, because the liver normally inactivates the mediators before they reach the heart.

Areas Most Affected

Mainly involve right-sided heart valves and endocardium, because they are the first tissues exposed to circulating mediators.

Mediators Produced by Carcinoid Tumors

- Serotonin (5-HT)
- Kallikrein
- Bradykinin
- Histamine
- Prostaglandins
- Tachykinins

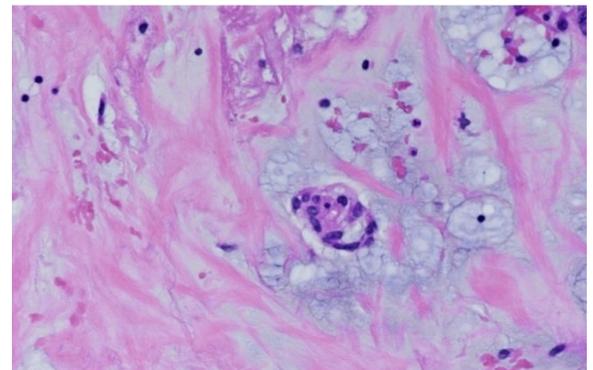
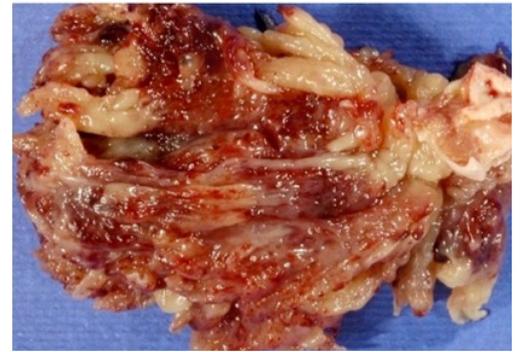
Systemic Manifestations

- Flushing
- Diarrhea
- Dermatitis
- Bronchoconstriction

Myxoma

Grossly : appear as sessile or pedunculated mass.

Microscopic: neoplastic cells within myxoid stroma



«إله أدت اليسر منه الله فيسّر علي خلقه ما استطعت كما أذ تسبّرك عليهم سبب لعسر أدرك
وإله نصوصه الشريعة قد نظافت علي أه الجراء الذي تلقاه يكونه منه جنبه عملك
فليسر لأهل اليسر، والعسر لأهل العسر...»

♥ (مساعدة لك لجميلك له تقصه منك شيئاً)



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