Cardiac Failure

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OVERVIEW

- Etiology
- Types
- Stages
- Pathophysiology
- Pathology
- Complications

HEART FAILURE

DEFINITION

It is a multisystem derangement that occurs when the heart is no longer able to eject blood……

- Delivered to it by the venous system or
- At a rate commensurate with the requirement of the metabolizing tissue.

Congestive Heart Failure

Common & recurrent condition with a poor prognosis.
Usually caused by……

- a slowly developing intrinsic deficit in myocardial contraction
- Increased volume load on the heart (fluid overload/ acute MI/ Acute valvular dysfunction)
- Impaired ventricular filling

COMPENSATORY MECHANISMS of CVS

To maintain arterial pressure perfusion of vital organs

- **FRANK – STARLING MECHANISM**
  - ↑ dilatation → enhanced contractility
- **MYOCARDIAL STRUCTURAL CHANGES**
  - Hypertrophy
- **ACTIVATION OF NEUROHUMORAL SYSTEMS**
  - Release of norepinephrine
  - Activation of R.A. system (Renin. Angiotensin)
  - Release of ANP (Atrial Natriuretic Peptide)

HEART FAILURE TYPES

- **FORWARD FAILURE**
  - following diminished cardiac output
- **BACKWARD FAILURE**
  - damming back of blood in the venous system.

FORWARD FAILURE

Causes

- Systolic Dysfunction
  - **Ischemic injury**
  - **Pressure/ volume overload**
  - **Dilated cardiomyopathy (eg. IHD)**

BACKWARD FAILURE

Causes

- Diastolic Dysfunction – Inability of the heart chambers to relax, expand and fill sufficiently during diastole, & accommodate adequate blood volume.

  - **Massive Left Ventricular Hypertrophy**
  - **Myocardial fibrosis**
  - **Deposition of amyloid**
  - **Constrictive pericarditis**

HEART FAILURE

Causes

- **FORWARD FAILURE IS ALMOST ALWAYS ACCOMPANIED BY BACKWARD FAILURE**

  - **↓ Ejection of blood from ventricle**

  - **↑ Blood in ventricles → ↑ end- diastotic pressure in ventricles → ↑ venous pressure**

CONGESTIVE HEART FAILURE

CHF may involve ……

- **Left Side of heart**
- **Right side of Heart**
- **Both Sides of Heart**

Left CCF- Causes

- IHD
- Hypertension
- Arotic & mitral Valualar Diseases
- Nonischemic Myocardial Diseases

Left CCF- Morphology

Morphology and clinical effects of left CCF result from ……

- progressive damming of blood in pulmonary circulation **(backward changes)**

- consequences of diminished blood pressure and flow **(reduced forward flow)**

Left CCF- MORPHOLOGY

Depends on the cause of disease process.

eg. in abnormalities like – MI/ Valvular deformities the changes are

- Hypertrophied and Dilated LV (except in mitral valve obstruction)
- Non- specific changes of Hypertrophy & fibrosis in the myocardium
- Secondary L.A. enlargement with Atrial Fibrillation

LVF EXTRACARDIAC CHANGES

Most commonly seen in….

- **Lungs**
- **Kidneys**
- **Brain**

LVF EXTRACARDIAC CHANGES LUNGS

PATHOGENESIS

- ↑ pressure in pulmonary veins → back pressure in the capillaries →↑ pressure in arteries.

- This causes pulmonary congestion and edema giving rise to **"Heavy, wet Lungs".**

LUNG, CHRONIC PASSIVE VENOUS CONGESTION - GROSS

Pooling of blood in the lung capillaries and associated microhemorrhages produce a dark brown discoloration.

Septal fibrosis causes the lung to become stiff. The fibrosis causes the lung to feel firm to the touch.

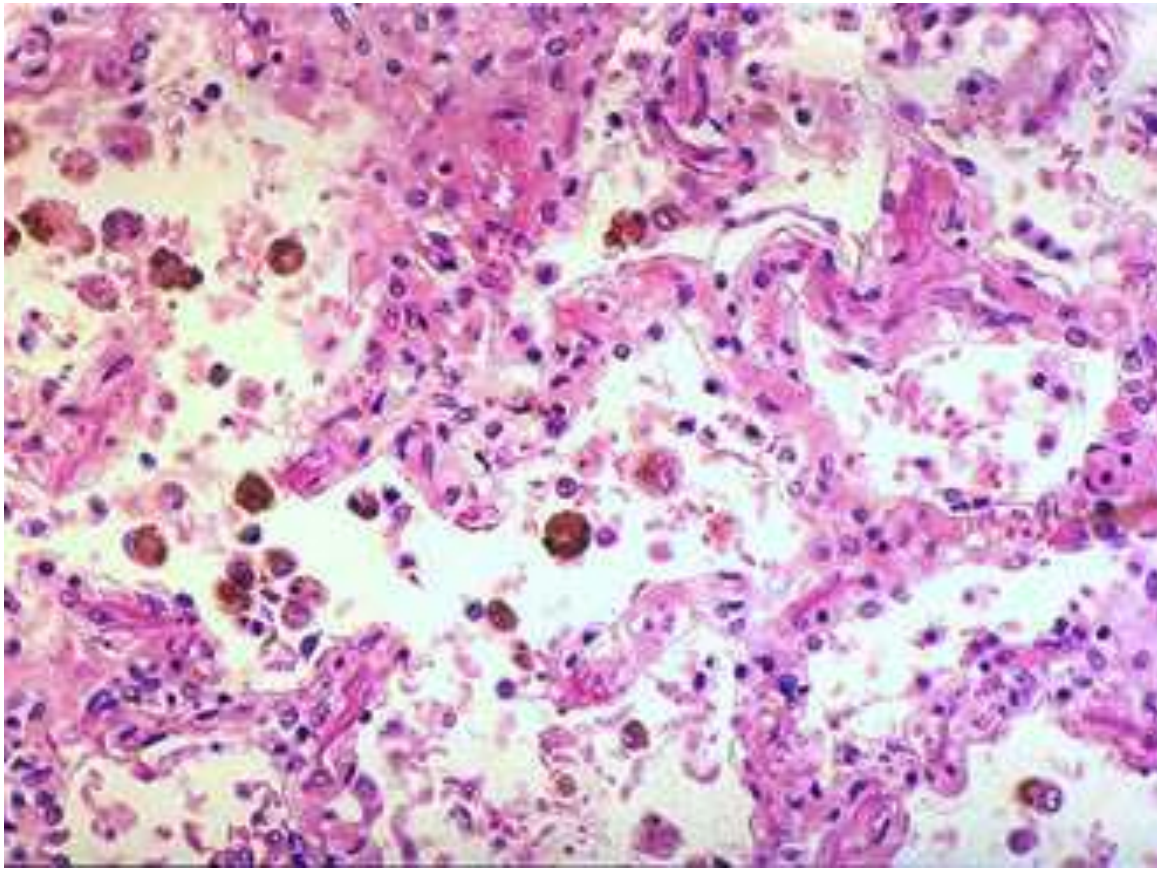
This gross appearance is also called **BROWN INDURATION OF THE LUNG.**

LVF EXTRACARDIAC CHANGES LUNGS

MORPHOLOGY

- Perivascular/ Interstitial transudate mainly present in interlobular septae **(Kerley's B-lines on X-ray)**
- Edematous widening of alveolar septae is seen.
- There is accumulation of edema fluid in the alveolar spaces.
- **SIDEROPHAGES/ HF CELLS** There are hemosiderin containing macrophages in the alveoli. caused due to ingestion by the macrophages of iron – containing proteins in the edema fluid, & hemoglobin from the erythrocytes which leak from congested capillaries.

LUNG, CHRONIC PASSIVE VENOUS CONGESTION - HIGH POWER



SIDEROPHAGES/ HF CELLS

These are hemosiderin containing macrophages in the alveoli.

LVF EXTRACARDIAC CHANGES LUNGS

Clinical Features

- Dyspnea
  - earliest & cardinal complaint.
- Orthopnea – dyspnea on lying down
  - relieved by sitting/ standing
- Paroxysmal Nocturnal Dyspnea (PND) – extention of orthopnea
  - There are attacks of extreme dyspnea bordering on suffocation; usually occurring at night.

LVF EXTRACARDIAC CHANGES

Kidneys

PATHOGENESIS

- ↓ cardiac output → ↓ renal perfusion → activation of rennin–angiotensin–aldosterone system → retention of salt and water with expansion of interstitial fluid and blood volumes → ↑ pulmonary edema (CHF) → release of ANP (Atrial Natriuretic Peptide) through atrial dilatation → reduction in excessive blood volume.
- Severe perfusion deficit → leads to impaired excretion of nitrogenous products leading to development of ***PRE – RENAL ASOTEMIA.***

LVF EXTRACARDIAC CHANGES
Brain

HYPOXIC ENCEPHALOPATHY

- Seen following cerebral hypoxia leading to features of irritability, ↓ ed attention, restlessness, stupor and coma.

RIGHT SIDED HEART FAILURE

Isolated right sided failure is rare

- Usually occurs as a secondary consequence of left – sided heart failure.

- PURE RIGHT SIDED HEART FAILURE

  Associated with chronic severe pulmonary hypertension. **(COR – PULMONALE)**

RIGHT SIDED HEART FAILURE
PATHOGENESIS

- Right ventricle is burdened by pressure workload due to increased resistance in pulmonary circulation.

- This results in minimal pulmonary congestion with pronounced engorgement of systemic and portal venous systems.

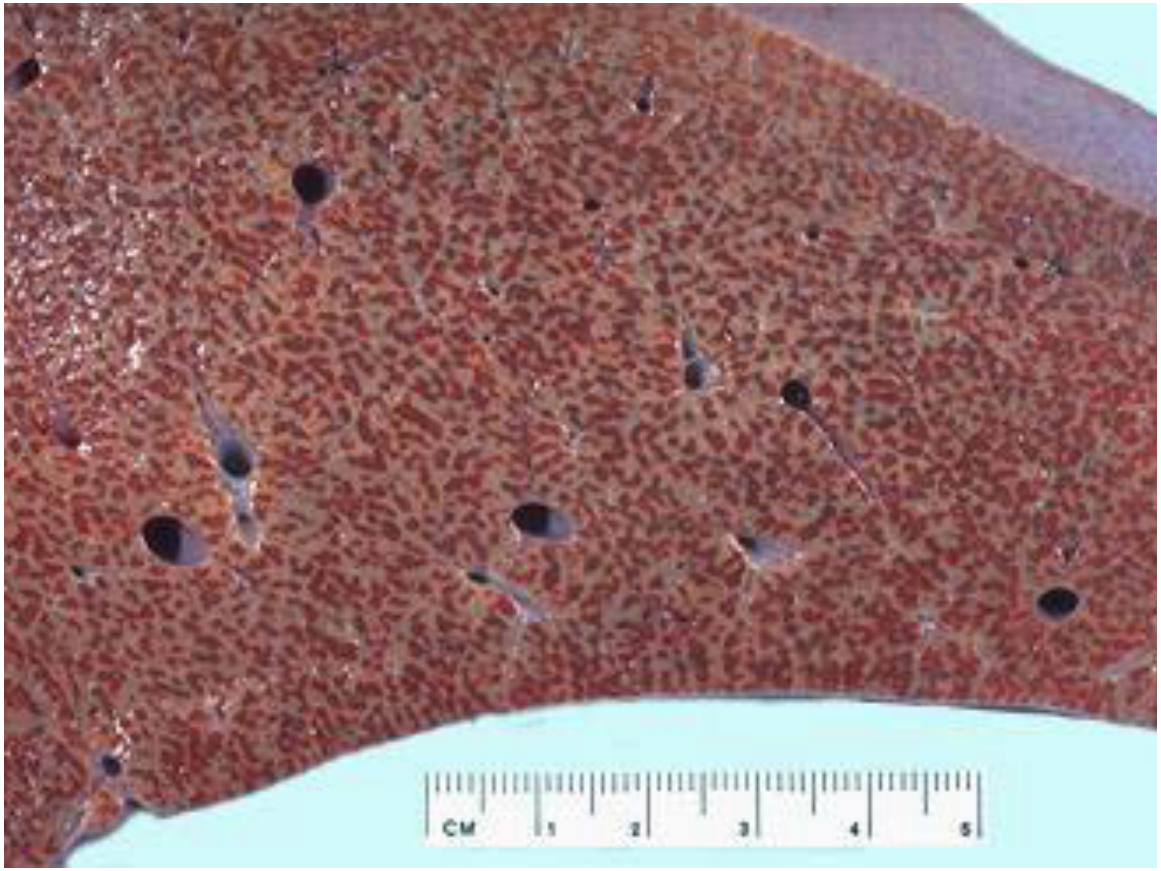
RIGHT SIDED HEART FAILURE
MORPHOLOGY

LIVER & PORTAL SYSTEMS

- There is increased size and weight of liver. **(Congestive Hepatomegaly.)**
- On C/s → Features of passive congestion are seen. **(Congested red centres surrounded by paler peripheral regions)**
- **CENTRILOBULAR NECROSIS :** Seen along with sinusoidal congestion (in cases associated with LVF)
- **CARDIAC CIRRHOSIS/ CARDIAC SCLEROSIS**

Refers to fibrotic central areas seen with long standing severe right – sided heart failure

LIVER, CHRONIC PASSIVE VENOUS CONGESTION - GROSS, CUT SURFACE



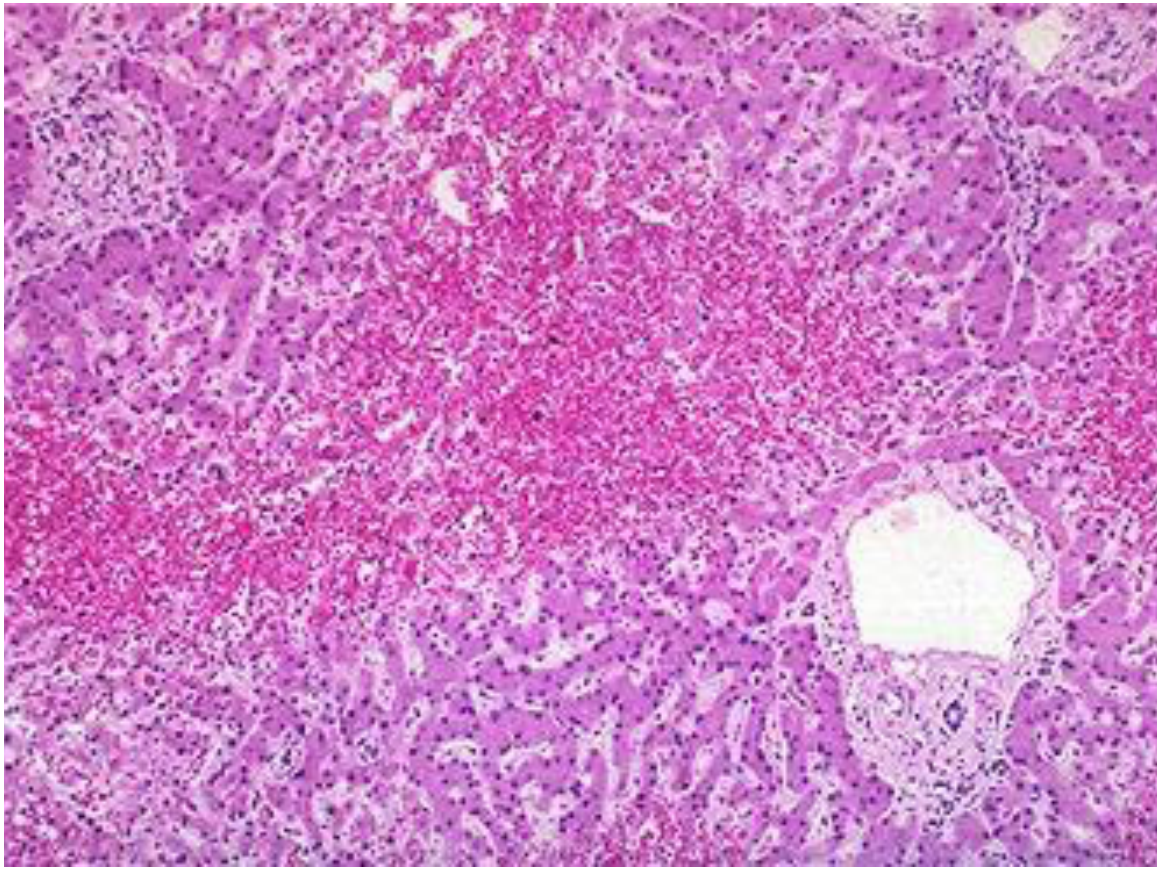
Hepatocytes in the central zone may even become necrotic due to hypoxia.

These centrilobular areas are seen as the dark red spots on the cut surface.

The alternating pale areas represent the periportal hepatocytes, which have sustained a lesser degree of hypoxia.

This gross appearance is also called **NUTMEG LIVER.**

LIVER, CHRONIC PASSIVE VENOUS CONGESTION



The congestion and accompanying sinusoidal dilatation are maximum in and around central veins and decrease progressively toward portal triads.

RIGHT SIDED HEART FAILURE

MORPHOLOGY

Spleen

- Elevated pressure in the portal vein and its tributaries lead to Tense, enlarged spleen.

  (congestive spleenomegaly)

- **MICROSCOPY:** There is marked sinusoidal dilatation and congestion.

- In long standing congestion – splenic weight may reach 300 – 500 gms.

RIGHT SIDED HEART FAILURE
MORPHOLOGY

Bowel

- There is chronic edema of the bowel wall.
- This leads to impaired absorption of nutrients.
- **ASCITES** - accumulation of transudate in the peritoneal cavity is seen.

RIGHT SIDED HEART FAILURE
MORPHOLOGY

Kidneys

- Congestion is more marked in RHF (than LHF)
- Fluid Retention, Peripheral edema & azotemia is seen.

RIGHT SIDED HEART FAILURE
MORPHOLOGY

Brain

- Symptoms are identical to those in LHF ***(including venous congestion & hypoxia of CNS.)***

RIGHT SIDED HEART FAILURE
MORPHOLOGY

Pleural & Pericardial Spaces

- Accumulation of fluid in pleural space (particularly right side) & pericardial space (effusions) may appear.

- Pleural effusions may range from 100ml – 1lit, with partial atelectasis of lung

  (LHF causes pulmonary Edema)

RIGHT SIDED HEART FAILURE
MORPHOLOGY

Subcutaneous Tissues

- There is peripheral edema of the dependant portions of the body. (eg. ankle, pretibial & presacral (*in bed -ridden patients*.)

- **ANASARCA :** Development of generalized massive edema.

RIGHT SIDED HEART FAILURE
Clinical features

- Systemic & Portal Congestion resulting in distended neck veins with enlarged tender liver.
- Hepatic and splenic enlargement
- Increased frequency of DVT (Deep Vein Thrombosis) and Pulmonary Embolism.
- Peripheral Edema.
- Pleural Effusion.
- Ascites.

CCF-Progression

- Most Patients Present With Biventricular CCF- With Clinical Syndromes Of Both Right & Left Sides Failure.

- With Progression - Patients become Cyanotic/ Acidotic Due to Reduced Tissue Perfusion → results in ventricular Arrythmias due to over activity of the sympathetic nervous sudden → leading to **SUDDEN DEATH**.

CARDIAC CAUSES of SUDDEN DEATH

CORONARY ARTERY DISEASES

- Coronary Atherosclerosis
- Developmental Abnormalities
- Coronary artery embolism
- Other (Vasculitis, dissection)

MYOCARDIAL DISEASES

- Cardiomyopathies
- Myocarditis and other infiltrative processes
- Right Ventricular Dysplasia.

VALVULAR DISEASES

- Mitral Valve Prolapse
- Aortic stenosis & other forms of left ventricular outflow obstruction
- Endocarditis

CONDUCTION SYSTEM ABNORMALITIES

CHRONIC IHD

DEFINITION

It is defined as the development of progressive CHF as a consequence of long – term ischemic myocardial injury.

Also called – Ischemic Cardiomyopathy.

Chronic IHD-Morphology

- Coronary arteries – Usually contain moderate to severe atherosclerosis.
- Moderate to marked cardiac enlargement is present following dilatation of all cardiac chambers.
- Areas of myocardial fibrosis or myocardial atrophy may be present.
- Endocardium: It is thick and opaque with thrombi in varying stages of organization.

Chronic IHD-Microscopy

- Extensive Myocardial fibrosis
- Atrophic/ Hypertrophic myocytes
- Myocytolysis

Chronic IHD- Clinical Features

- There is development of severe, progressive heart failure punctuated by episodes of angina /MI.
- Patient has Arrythmias.
- Heart failure with intercurrant MI may account for many deaths.
- It may be difficult to distinguish from dilated cardiomyopathy.

- THANK YOU