Constrictive Pericarditis

Introduction:
It is: Rigid adherent pericardial sac which inhibits ventricular diastolic filling
Results from fibrosis, scarring and loss of elasticity of pericardium.
Normal pericardial thickness-less than 3mm
Course: typically chronic
Variants- Subacute, transient (for 3mths ) and occult.

Causes;
- **Infective:**
  Post viral, tuberculous, fungal, parasitic infections
  In complete drainage of purulent pericarditis
- **Neoplastic:**
  pericardial infiltration
- **Autoimmune:**
  connective tissue disorders
- **Physical:**
  prior mediastinal radiation therapy
- **Ischemic**
  Following pericarditis in acute MI
  Dresslers syndrome (post MI)
- **Post surgical:**
  Following CABG
- **Others**
  Chronic renal failure treated with hemodialysis
  Following pulmonary asbestosis
- **Idiopathic** – in nearly half.

Common - idiopathic, infectious, ( viral pericarditis) radiation induced, post surgical, TB in developing countries and HIV patients

Stages in pathogenesis:
1. Initial sub clinical acute pericarditis-
2. sub acute stage of resorption of effusion plus organization-
3. chronic stage of thickening and fibrous scarring of pericardium; thickening heterogenous Calcification common – contributes to stiffness in addition to scarrings
  Serious Locations of thickening AV groove, SVC entrance, chest wall diaphragm

Pathogenesis:-
Post inflammatory organization and symmetrical rigidity leads to
1. Impaired cardiac filling in diastole esp mid & late diastole
2. So systemic and pulmonary venous return is impeded and hence
3. Systemic venous pressure is raised (not so much pulmonary venous pressure)
4. Cardiac output diminished secondary to impeded diastolic filling
5. Systolic function not affected till late.

Features of note:
1. There is uniform restriction of filling of all heart chambers
2. There is abnormal pattern of filling
   Ventricles get filled abnormally rapidly in early diastole itself
   Ventricular volume-time plot shows typical Dip and plateau waveform
   Early diastolic dip corresponds to excessive rapid filling period and
   Plateau corresponds to mid and late diastole with no additional volume filling
3. Early diastolic dip is reflected in JVP as prominent Y descent-Friedrich sign
4. In contrast in cardiac tamponade diastolic filling is impeded throughout diastole

Kusmauls’ sign:
Intrathoracic pressure changes during respiration fail to be transmitted to pericardium and cardiac chambers
During inspiration systemic venous pressure and Rt atrial pressure do not fall
Systemic venous pressure rises in inspiration causing Kussmauls sign.

Pulses paradoxus is less common in constrictive pericarditis than in Pericardial effusion

Clinical features:

1. Features of elevated systemic venous pressure:
   a. JVP distension, and deep y descent
   b. Hepatomegaly
   c. Ascites- unique feature is Ascites is out of proportion to pedal edema
   d. Peripheral edema
   Most impressive clinical findings are b and c
2. Features of reduced cardiac output
   Fatigue
   Tachycardia
   Hypotension
3. Features of pulmonary venous congestion
   Dyspnea, orthopnea, cough
4. Chest pain due to
   Reduced cardiac output
   Thickened pericardium compressing epicardial arteries

5. Important auscultatory feature:
   Characteristic “Pericardial Knock”

Pericardial Knock
   1. It is a diastolic sound
2. After opening snap and before S3 in timing
3. Heard along the left sternal border 3rd space
4. High pitched sound
5. Indicates sudden stopping of ventricular filling by rigid pericardium

**Variants of constrictive pericarditis**
1. Effusive-constrictive
2. Transient
3. Occult

**History:**
Careful history may reveal etiology in some cases/look for TB etiology
Can be idiopathic

**Diagnosis**

```
Constrictive pericarditis – features of heart failure - in the absence of signs of organic heart disease
```

**Differential diagnosis**
1. Pericardial effusion
2. Cardiac tamponade
3. Effusive-constrictive pericarditis
4. Restrictive cardiomyopathy
5. Right heart failure
6. Cirrhosis

**Differentiating features**

**Pericardial effusion**
- Accumulation of pericardial fluid under pressure
- Becks triad is diagnostic

**Cardiac tamponade**
- Life threatening emergency due to pericardial effusion; hemodynamic compromise
- Profound shock and Becks triad is diagnostic of cardiac tamponade

**Constrictive pericarditis**
- Is result of scarring and loss of elasticity of pericardial sac.

**Restrictive cardiomyopathy**
- In this there is increased myocardial thickness as against constrictive pericarditis with pericardial thickness
- In Echo-ventricular thickness seen in restrictive cardiomyopathy
- In Echo thickened pericardium seen with normal ventricular contraction in constrictive pericarditis

**Right heart failure**
- Shows signs of organic heart disease which is absent in constrictive pericarditis

**Cirrhosis**; shows features of portal hypertension plus liver cell failure

**Effusive constrictive pericarditis**
a. (Visceral) Constrictive physiology with coexisting pericardial effusion often with tamponade
b. Differentiation from isolated tamponade – After drainage of pericardial fluid – elevation of Rt atrial pressure and pulmonary wedge pressure persists
c. Amount of effusion may vary from moderate to large
   - JVP /arterial pressure may be within reference range – with or without signs of tamponade
e. Clinical importance:
   - Pericardiocentesis /pericardial window inadequate
   - Visceral pericardial constriction needs to be addressed with medical or surgical therapy
   - Close monitoring is required
   - Condition common in first months of chronic effusion
   - CT/MRI are investigation of choice

Investigations
1. ECG: Low voltage complexes in limb leads;
   - Generalized T wave inversion: atrial arrhythmias common especially atrial fib.
2. CXR: Heart silhouette – normal or mildly enlarged
   - Calcification in 50%
   - Calcific pericardium need not be necessarily constricted
   - Lateral view useful
   - Pleural effusion present in many, persistent unexplained pleural effusion may be presenting feature
3. Echocardiogram; if well imaged
   - Essential findings 1. Thickened pericardium, 2. Normal ventricular contraction
     - (Ventricular cavities small but contract vigorously.)
   - Pericardial fluid if present – seen as Echo free space between visceral and parietal pericardium
4. Doppler flow analysis: Diastolic flow ending abruptly in early diastole
5. CT/MRI: delineate thickened pericardium better
   - Note: Thickening/calcification do not always correlate with hemodynamic severity.
6. Cardiac catheterization:
   - Equalization of diastolic pressure in all 4 chambers
   - Ventricular pressure tracing shows diagnostic dip and plateau appearance
     - (Synonym-Square root sign)
7. Investigate for tuberculosis
8. Atrial natriuretic peptide is normal in constrictive pericarditis

Complications:
- Relentless progression (except very rarely)
- Cardiac cachexia

Drugs to be avoided:
- Beta blockers and calcium channel blockers;
- They slow down the mild compensatory tachycardia

Treatment
- Definitive treatment is stripping of pericardium
- Improvement may take several months
- During surgery must look for associated Rt atrial thrombus and remove it
Early surgery before the advent of cachexia and pericardial calcification – advisable
Operative technique is changed: median sternostomy or torocotomy not parasternal approach.
Operative mortality can be high

*Contraindications for surgery:*
Elderly patients with severe liver dysfunction
Severe cachexia
Dense calcification
Severe myocardial damage