Cirrhotic Cardiomyopathy

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Cardiac Output at rest in Laennec’s cirrhosis

Cardiac Syndromes in Liver Disease
I. Portopulmonary Arterial Hypertension
II. Hepatopulmonary Syndrome
III. Cirrhotic Cardiomyopathy

Porto-Pulmonary Arterial Hypertension

Porto-Pulmonary Arterial Hypertension
**Hepatopulmonary Syndrome**

**Hyperdynamic – High Output**

![Heart Images]  

**World Congress of Gastroenterology 2005**

**Definition of Cirrhotic Cardiomyopathy**

Cardiac dysfunction in patients with cirrhosis characterized by impaired contractile responsiveness to stress and/or altered diastolic relaxation with electrophysiological abnormalities in the absence of other known cardiac disease.

**Dobutamine Stress Echo**

Baseline EKG  

**Chronotropic Incompetence**

At peak dobutamine dose of 50 ug/kg/min
Diagnostic criteria

• Diastolic dysfunction
  – E/A ratio < 1.0 (age-corrected)
  – Prolonged deceleration time (>200 ms)
  – Prolonged isovolumetric relaxation time (>80 ms)

Additional and Supporting Criteria

• Abnormal chronotropic response
• Electromechanical uncoupling/dysynchrony
• Prolonged QTc interval
• Enlarged left atrium
• Increased myocardial mass
• Increased BNP and pro-BNP
• Increased troponin I

Cirrhotic Cardiomyopathy

• Limited data regarding true prevalence of cirrhotic cardiomyopathy because of near normal cardiac function at rest unless exposed to stress (vasodilation, pharmacologic or physiologic stress; bacterial infection, tips or transplantation)
• ~50% of patient undergoing OLT develop some signs of cardiac dysfunction
• Majority of patients with Child-Pugh class B and C have at least one feature of CCM i.e. QT prolongation

Concentric LVH

Algorithm for Diagnosis of Cirrhotic Cardiomyopathy

Is Cirrhotic Cardiomyopathy Intrinsic to Liver Disease?

• Other explanations for cardiac dysfunction
  – HTN (can be masked in advanced liver dysfunction)
  – Alcoholic cardiomyopathy
  – Viral myocarditis
  – Ischemic heart disease
  – Familial or other cardiomyopathies as in general population
  – Hemodynamic adaptation to low SVR, high output (HPS)
  – Response to pulmonary vasoconstrictors (PPH)
**Experimental Cirrhotic Cardiomyopathy**

- Down regulation of β receptors; impaired β adrenergic signaling and decreased cardiac contractility
  - Constant catecholaminergic stimulation
  - Decrease in cAMP production (impairment in cardiac contractility)
- Electrophysiological abnormalities
  - Abnormalities in K channels leading to QT prolongation
  - Abnormalities in SR leading to slower Ca recycling and slowed relaxation and increased ventricular stiffness
  - Chronotropic incompetence

**Autonomic Dysfunction**

- [Image of cardiac imaging with text: Camera imaging with mIBG reflects noradrenaline concentrations, storage, release and uptake]

**QT interval prolongation**

- 107 patients with cirrhosis and 42 controls examined.
- Prevalence of abnormal QTc:
- In control group QTc was longer in females, but no gender difference noted in patients

![Graph showing QT interval prolongation in cirrhosis and controls]

**Myocardial late gadolinium enhancement cardiovascular magnetic resonance in patients with cirrhosis**

- 20 consecutive patients with ELD with CMR scan
  - All had hyperdynamic LV
  - All showed evidence of LGE c/fibrosis – but to a greater extent in alcoholic liver/cardiac disease
  - Patterns similar to acute myocarditis
  - MELD score did not predict severity of LGE
  - No association between severity of LGE and EF.
  - Role in diagnosing, or prognosticating in ELD is unclear

**LV diastolic dysfunction is an independent predictor of mortality in patients who underwent liver transplantation.**

- [Graph showing survival rates post-LT]

**Future Challenges**

- To better identify latent cardiac dysfunction – systolic and diastolic as they appear to affect the clinical course in both the pre-OLT and post-OLT periods
- To differentiate reversible from irreversible changes in myocardial structure and function that occurs in association with liver disease - i.e. when is OLT curative
- To develop appropriate treatments for both latent and overt myocardial abnormalities that are present in association with liver disease