

Cirrhotic Cardiomyopathy

Sameh Emil, M.D , FSCAI



Introduction

- The heart and liver may interact in several different ways .
- Acute or chronic heart failure and especially right HF may lead to a spectrum of several liver manifestations, including cardiac cirrhosis or congestive liver disease.
- Chronic liver disease such as cirrhosis may affect the heart and the whole cardiovascular system, leading to a syndrome named ***Cirrhotic Cardiomyopathy***.
- Cirrhotic cardiomyopathy is independent of the aetiology of cirrhosis.

Cirrhotic Cardiomyopathy

- **Definition**

A 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 .

Prevalence

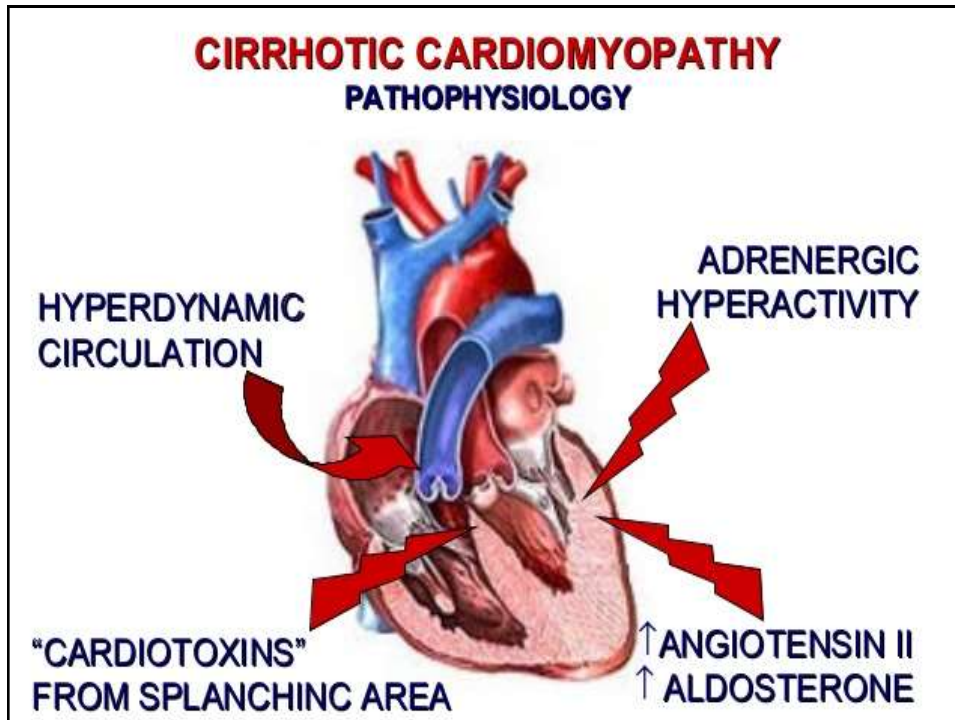
- The prevalence of cirrhotic cardiomyopathy is reported to be between 40 to 50% in cirrhotic patients independent of liver disease etiology.
- Studies on candidates with liver transplantation; features of cardiac dysfunction were reported in 50% of advanced cirrhosis patients and heart failure reported in 7% - 21% of post-operative deaths .

Pathophysiology

- Cardiac dysfunction is considered to be multifactorial in chronic liver disease patients .
- Liver cirrhosis is characterised by increased intrahepatic vascular resistance consecutive to fibrosis development and regeneration nodules formation, which result in "portal hypertension".
- Portal hypertension, in turn, is correlated to the production of vasodilators including carbon monoxide, NO, and TNF.
- In parallel, there is also a reduced degradation of these substances due to the metabolic hepatic dysfunction and portosystemic shunt .

Pathophysiology

- This contributes to splanchnic vasodilatation, which not only decreases global systemic vascular resistance but also creates splanchnic blood pooling.
- All these factors result in *hyperdynamic circulation* and *effective hypovolaemia* that induces baroreceptor and volume receptor activation of both (RAAS & SNS) with an increased secretion of ADH contributing to the development of cirrhotic cardiomyopathy.
- With progression of cirrhosis, there is a decrease in the thickness of the vessel walls as well as a decrease in total vascular wall area.



Clinical Presentation

- **Electrophysiologic changes:** QT-interval prolongation. At least 60% of end stage liver disease patients show this ECG abnormality.
- **Inotropic and chronotropic incompetence:** Blunted ability to increase heart rate or LVEF after appropriate stimulation with exercise, drug infusion, or postural challenge .
- **Systolic & Diastolic Dysfunction :** Due to volume overload & impaired myocardial relaxation.

Cirrhotic Cardiomyopathy

Diagnostic criteria

Systolic dysfunction

- Blunted increase in cardiac output with exercise, volume challenge or pharmacological stimuli
- Resting EF <55%

Diastolic dysfunction

- E/A ratio <1.0 (age-corrected)
- Prolonged deceleration time (>200 msec)
- Prolonged isovolumetric relaxation time (>80 msec)

Supportive criteria

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

Journal of Hepatology 2010 vol. 53 | 179–190

Diagnosis of cirrhotic cardiomyopathy

• **Clinical symptoms :**

The diagnosis is difficult as cardiac function is nearly normal at rest. In most cases this condition is well tolerated and may be asymptomatic for a long period of time.




Most of the patients are diagnosed during the worsening of the liver disease when the main clinical characteristics of heart failure (diastolic and high-output) usually appear.

Diagnosis of cirrhotic cardiomyopathy

- **Echocardiography:**
To assess both systolic and diastolic abnormalities.
- **Electrocardiography :**
To assess QT interval changes.
- **Cardiac magnetic resonance:**
To assess volumes; ejection fraction; response to pharmacological stress.

Diagnosis of cirrhotic cardiomyopathy

- **Biomarkers :**
Increased levels of circulating cardiac biomarkers might reflect the presence of cardiac dysfunction.
 - B-type natriuretic peptide (**BNP**) and **proBNP** have been reported to be elevated.
 - **Troponin I** is increased in some patients with cirrhosis.
 - Atrial natriuretic peptide (**ANP**) levels are often increased in patients with decompensated cirrhosis.

	Reduced Cardiac function	Compensatory phase	CARDIAC FAILURE
Stages of Cirrhotic Cardiomyopathy			
CLINICAL Findings	Hyperdynamic State	Hyperdynamic State ↑↑↑ Palpitations Tachycardia	Hypotension Cardiac Failure sign and symptoms Pulmonary edema
ECG abnormalities	QT prolongation	Multiple Extrasystoles QT prolongation ↑↑↑	Bundle branch block ST-segment depression Electrical and mechanical dyssynergy
Echocardiographic Findings	prolonged isovolumetric relaxation time	prolonged isovolumetric relaxation time (>80ms) decreased pattern of contractility Diastolic dysfunction	- enlarged left atrium - decreased wall motion - increased wall thickness Systolic Dysfunction

Prognosis

- The patient with cirrhosis is a severely ill patient with an overall unfavorable prognosis if liver transplant is not safely performed.
- When there is a rapid hemodynamic change (e.g., after transjugular intrahepatic portosystemic shunt (TIPS) or liver transplantation), the increased filling pressure may favor the development of congestive heart failure.
- An improvement after liver transplant is expected and validates the concept that the cardiomyopathy is truly cirrhotic in origin.

Prognosis

- Patients with cirrhosis undergoing liver transplantation reported the disappearance of left ventricular hypertrophy and diastolic dysfunction as well as normalization of systolic response and exercise capacity during stress.

Treatment approach

- Until now, no evidence exists of any benefit of a specific pharmacological therapy for cirrhotic cardiomyopathy.
- Routine heart failure management is applied.
- Vasodilators, like ACE-I, should not be used due to the risk of further aggravation of the systemic vasodilatory state.

Treatment approach

- Aldosterone antagonists may have beneficial effects in terms of a reduction in left ventricular dilatation and wall thickness and improvement of diastolic function.
- Cardiac glycosides do not seem to improve cardiac contractility in cirrhotic cardiomyopathy.
- Nonselective β -blockers have been shown to improve the prolonged QT interval and might reduce the hyperdynamic load in patients with cirrhosis.

Treatment approach

- At present, investigations on the gene expression pattern of the cardiomyocyte adrenergic pathway in animal models of cirrhosis are an attempt to better understand the causes of the blunted cardiac response.

Take home message

- Don't be impressed by normal resting LV function in liver cirrhosis patients because this ventricle might fail under stress.
- Strict diagnostic criteria for cirrhotic cardiomyopathy are still lacking.
- No specific treatment or management strategies has been tested for patients with cirrhotic cardiomyopathy.
- In the meantime ,management of cirrhotic cardiomyopathy should follow the recommendation of the ACC/AHA guidelines for the treatment of patients with heart failure.

THANK YOU

