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Associated Cardiac and Hepatic Disorders

- Heart disease affecting the liver
 - Mild alterations of liver function test in heart failure
 - Cardiogenic ischemic hepatitis and its variants
 - Congestive liver fibrosis and congestive cirrhosis (cardiac cirrhosis)
- Liver diseases affecting the heart
 - Hepatopulmonary syndrome
 - Portopulmonary hypertension
 - Pericardial effusion in cirrhosis
 - Cirrhotic cardiomyopathy
 - High output failure caused by intrahepatic arteriovenous fistula in the noncirrhotic liver
- Cardiac and hepatic disorder with joint etiology
 - Infectious and parasitic
 - Metabolic
 - Immune and vasculitic
 - toxic

Mild alteration of liver function tests in CHF

- The congested liver is usually enlarged and firm, often associated with slight enlargement of the spleen
- Modest elevations of ALT, AST, LDH, r-GT,ALP, T-Bil; small decreases in albumin levels
- Liver function abnormalities are most commonly seen in patients with a cardiac index < 1.5 L/min per m² (up to 80 % of cases)
- In general, these enzyme abnormalities are not associated with clinically apparent hepatic disease, are fluctuating, and resolve with compensation of heart failure
- Mild jaundice occurs on one-third of the patients and increases with prolonged and repeated bouts of CHF

Clinical presentation

- Occurs during the course of MI complicated by cardiogenic shock
- Typically has a protracted course of CHF (NYHA class 3 or 4)
- After recovery from an episode of pulmonary edema, the IH revealed after a latency period of 2-24 hrs
- Symptoms at the onset: weakness and apathy; in a minority of cases, mental confusion, jaundice, oliguria, flapping tremor, and hepatic coma may present
- Lab:sharp elevations of ALT, AST, and LDH (usually > 10×), elevation of bilirubin, prolongation of PT
- A minority has consumption coagulopathy: prolonged PTT and PT, low fibrinogen levels, elevated FDPs, thrombocytopenia
- Occasionally a functional renal impairment appears: abrupt increase in BUN, Cre, K, low Una, normal urinary sediment
- In the survivors, the abnormalities of the hepatic, coagulation, and renal function tests reach their peak 1-3 days after the onset of the cardiogenic IH and return to normal within 5-10 days

D/D of hepatitis in patient with CHF

- 1. Mild, asymptomatic: reversible increase of one or several liver function tests -- AST, ALT, bilirubin, ALP
- 2. Cardiogenic IH, mainly a "laboratory syndrome": abnormalities within a few hours of an acute cardiac event, sharp increase in AST and ALT to 10- to 20-fold normal levels followed by a >50% decrease within 72 hours
- 3. Shock liver--similar to IH, complicates severe arterial hypotension of various causes
- 4. Common variants of hepatitis: drug-induced hepatotoxicity, alcoholic hepatitis, viral hepatitis
- 5. Jaundice after cardiac surgery: very high levels of AST and ALT may occur by the second postoperative day

IH and drug-induced hepatotoxicity

- Currently, there is no specific test to differentiate IH from drug-induced liver damage.
- It may have practical importance that the ALT/LDH ratio in IH and viral hepatitis is significantly less than for acute acetaminophen hepatitis.
- When the fold increase (fi) of the enzymes was calculated, an ALT_{fi}/LDH_{fi} of 11.25 or more was characteristic for acetaminophen hepatitis, with a sensitivity of 75% and specificity of 76% versus viral hepatitis and IH.

Treatment

- Identify and remove precipitating cause
 - medications with negative inotropic or hypotensive effects (certain antiarrhythmic drugs, calcium channel blockers, and vasodilators)
 - Medications likely to cause impairment of renal function (ACEIs, angiotensin receptor-1 blockers)
 - Medications likely to accumulate with evolving renal failure (Digoxin)
- Low-dose iv Dopamine to augment splanchnic perfusion

- Theoretic treatment
 - Dobutamine
 - L-arginine
 - Acetylcystein
 - Antioxidants
 - Antibiotics
 - Oxygenation of the intestinal lumen

- Prognosis depends on
 - Cardiovascular status
 - Drug regimen at the time the disorder is developing
 - Mortality rate: 83% among those taking antiarrhythmic drugs with cardiodepressant side effects; but only 18% among those not taking such medication
- The ischemic liver injury is usually self-limiting when it affects the normal liver, but more serious changes may occur when the liver has been previously damaged

- CLF: clinically silent disorder characterized by a spectrum of morphologic alterations from mild deposition of sinusoidal collagen to emergence of broad fibrous septa
- CC: The presence of extensive fibrosis in association with the formation of regenerative nodules is called cirrhosis
- variants: focal, incomplete, complete
- Chief causes:
 - Ischemic heart disease (31%)
 - Cardiomyopathy (23%)
 - Valvaular heart disease (23%)
 - Restrictive lung disease (15%)
 - Pericardial disease(8%)

Pathogenesis-thrombosis

- Clinical presentation
 - usually masked by s/s of right-sided heart failure
 - In the majority of patients, ALT, AST, ALP and bilirubin are within normal range
 - Hepatic synthetic function is usually preserved with normal plasma albumin and prothrombin time
 - Occurrence of cardiac ascites is the hallmark of CC
 - High ascitic fluid protein : $\geq 2.5 \text{ g/dL}$
 - High serum ascites albumin gradient: $\geq 1.1 \text{ g/dL}$
 - The ascitic fluid LDH and red cell counts are significantly higher than in cirrhotic ascites of other causes

D/D of ascites in CHF

High serum ascites albumin gradient (1.1 g/dL)

Cardiac ascites Infected cardiac ascites Cirrhosis Budd-Chiari syndrome Alcoholic hepatitis Fulminant hepatic failure Hepatic veno-occlusive disease Massive liver metastases Myxedema High ascitic protein level (2.5 g/dL) Cardiac ascites Cirrhotic ascites after diuretic treatment Malignant ascites Peritonitis Infected ascites occasionally

High serum ascites albumin gradient and high ascitic protein

Cardiac ascites

Cirrhotic ascites after diuretic treatment

Infected cirrhotic ascites occasionally

- Diagnostic testing
 - Clinical Triad:
 - 1.Right heart failure
 - 2.Hepatomegaly
 - 2.Ascites with high protein content, and high serum ascites albumin gradient, along with refractoriness of ascites to diuretic treatment that contrasts with resolution of peripheral edema with diuretics
 - Esophageal varices and splenomegaly may also be present
 - Portal flow studies
 - Liver biopsy

■Treatment – no prospective studies

- Similar to heart failure
- Paracentesis ; no need to regularly replace the albumin lost
- peritoneovenous shunts
- ## Transjugular portosystemic shunt is contraindicated in cardiac ascites

Prognosis

- No evidence that CC worsens the prognosis of patents with CHF
- The mortality rate is determined by the severity of the underlying cardiac disease

Hepatic acinus

Zone 1: periportal region Zone 2 Zone 3: perivenular region

