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Overview on Cardiac Cirrhosis and Congestive Hepatopathy - A Review

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Review Article

ABSTRACT

Cardiac cirrhosis (congestive hepatopathy) refers to a group of hepatic abnormalities that develop as a result of right-sided heart failure. Cirrhosis of the liver can be induced by any right-sided pathology that leads to right-sided heart failure, which leads to increased venous congestion and

pressure in the hepatic sinusoids. Because cardiac cirrhosis might be asymptomatic or diagnosed incorrectly due to other types of liver disease, determining its prevalence is difficult. The underlying heart disease, rather than the hepatic congestion and damage, is usually the cause of death in cardiac cirrhosis. The control of the underlying cardiac disease, as well as the optimization of cardiac output, are the mainstays of congestive hepatopathy treatment. Diuresis can help with hepatic congestion, but it must be used with caution to avoid causing hepatic ischemia. Hemodynamic therapy may be able to reverse the early stages of congestive hepatitis. The widespread use of heart transplantation (HT) and considerable breakthroughs in medical and surgical treatments have drastically altered the profile of CH patients. In this overview we will be looking at the disease cause, epidemiology, diagnosis, and treatment.

1. INTRODUCTION

Cardiac cirrhosis (congestive hepatopathy) refers to a group of hepatic abnormalities that develop as a result of right-sided heart failure. The signs and symptoms of congestive heart failure (CHF) predominate in clinical practise. Unlike cirrhosis produced by persistent alcohol consumption or viral hepatitis, the impact of cardiac cirrhosis on overall prognosis is unknown. As a result, treatment focuses on addressing the underlying cause of heart failure. It's crucial to distinguish cardiac cirrhosis from ischemic hepatitis. Massive hepatocellular necrosis may result from rapid cardiogenic shock or other hemodynamic collapse in the latter case. It is usually discovered when serum hepatic transaminase levels rise suddenly and dramatically. Despite the fact that cardiac cirrhosis and ischemic hepatitis are caused by different underlying cardiac diseases (right-sided heart failure in the former and left-sided heart failure in the latter), they can appear simultaneously in clinical practise [1].

Cirrhosis of the liver can be induced by any right-sided pathology that leads to right-sided heart failure, which leads to increased venous congestion and pressure in the hepatic sinusoids. (1) valvular disease, (2) severe pulmonary hypertension, (3) cor pulmonale, (4) biventricular heart failure, (5) pericardial disorders, (6) cardiac tamponade, and (7) constrictive pericarditis are all common causes of cardiac cirrhosis [2].

For a long time, scientists have known about the interactions between the heart and the liver. However, in recent years, these cardio-hepatic interactions have attracted more attention, leading to a better understanding of their pathogenesis. They are commonly divided into three categories based on the role of each organ as the cause or victim of the other: (1) liver disease caused by heart illness; (2) heart disease caused by liver disease (e.g., cirrhotic cardiomyopathy); and (3) systemic diseases affecting both the heart and the liver (e.g., systemic amyloidosis). The former is commonly referred to as "cardiac hepatopathy" [3].

Congestion causes liver damage by reducing hepatic blood flow, decreasing arterial oxygen saturation, and increasing hepatic venous pressures. The hepatic veins and sinusoids are affected by elevated central venous pressure, which reduces portal venous input. Sinusoidal congestion, dilatation of sinusoidal fenestrae, and exudation of protein and fluid into the Disse space are all caused by increased hepatic venous pressure. Exudate builds up in the Disse space, obstructing oxygen and nutrient passage to hepatocytes. In conditions when arterial flow is compromised, such as hypotension, arrhythmias, or left-sided heart failure, which can cause ischemic hepatopathy, decreased hepatic blood flow increases vulnerability to injury [4-6].

In most situations, the presence of underlying cardiac or vascular illness determines the patient's prognosis. The goal of treatment is to address the underlying cause of CH. Diuretics may be used wisely to treat jaundice and ascites in patients with hepatic congestion caused by heart failure, keeping in mind that in individuals with low cardiac function, this may result in ischemia. Endovascular or surgical intervention to restore hepatic venous drainage or relieve portal hypertension may be used in individuals with Budd-Chiari syndrome, possibly in combination with anticoagulant therapy [7].

2. PATHOPHYSIOLOGY

Hepatic damage is caused by a variety of factors that disrupt the circulatory system's homeostasis. Increased ventricular filling pressures or decreased cardiac output and poor perfusion are the main mechanisms producing hepatic dysfunction as a result of heart failure. Due to increased retrograde pressure to the venous and capillaries into the liver, an increase in preload or central venous pressure caused by right ventricular dysfunction may induce direct liver injury.This causes an increase in liver enzymes [2].

Elevated right atrial pressure is transmitted to the liver via the inferior vena cava and hepatic veins in decompensated right ventricular or biventricular heart failure. Venous congestion obstructs the effective drainage of sinusoidal blood flow into terminal hepatic venules at the cellular level. Sinusoidal stasis causes deoxygenated blood to accumulate, parenchymal atrophy, necrosis, collagen deposition, and, eventually, fibrosis [1].

Impaired perfusion and tissue hypoxia from decreased cardiac output are another mechanism that can cause hepatic damage, especially when the left side of the heart is compromised. This may be linked to acute hepatocellular necrosis, which is characterised by elevated serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), and Lactate dehydrogenase (LDH), as well as thrombin and prothrombin time prolongation [2].

3. ETIOLOGY

Cirrhosis of the liver has many of the same causes as right-sided congestive heart failure (CHF), such as congenital heart disease. Despite the fact that inferior vena caval thrombosis and Budd-Chiari syndrome have a similar pathogenesis, Budd-Chiari syndrome is a congestive hepatopathy caused by obstruction of the hepatic veins. This syndrome occurs in 1/100,000 in the general population.

The initial pathophysiological event in BCS is obstruction of venous outflow between the hepatic veins and the suprahepatic portion of the IVC. Obstruction of one hepatic vein is not sufficient for the onset of the syndrome: at least two veins must be blocked for its clinical manifestation. The consequence of this obstruction is a complex alteration of the circulatory system with an increase in hydrostatic pressure in the portal capillaries resulting in altered vascular pressure gradients (Fig. 1).
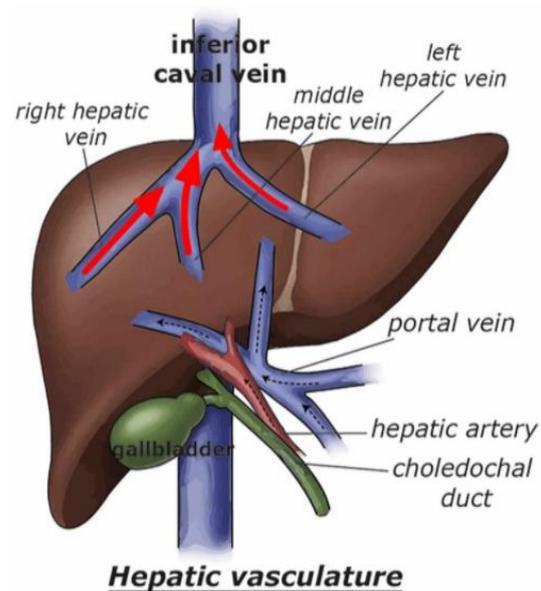

Fig. 1. Hepatic vasculature

They are classified separately and are not considered causes of cardiac cirrhosis.

The following are the most common causes of heart cirrhosis: [1]

- Ischemic heart disease (31 percent)
- Cardiomyopathy (23 percent)
- Valvular heart disease (23 percent)
- Primary pulmonary disease (15 percent)
- Pericardial illness (8 percent)

4. EPIDEMIOLOGY

Because cardiac cirrhosis might be asymptomatic or diagnosed incorrectly due to other types of liver disease, determining its prevalence is difficult. The underlying heart disease, rather than the hepatic congestion and damage, is usually the cause of death in cardiac cirrhosis. The age at which cardiac cirrhosis manifests is unknown because it is dependent on the beginning of decompensated heart failure. When it comes to heart disease, the risk of cardiac cirrhosis rises with age. It is also more common in males, who have a higher risk of developing heart failure and, as a result, cardiac cirrhosis [2].
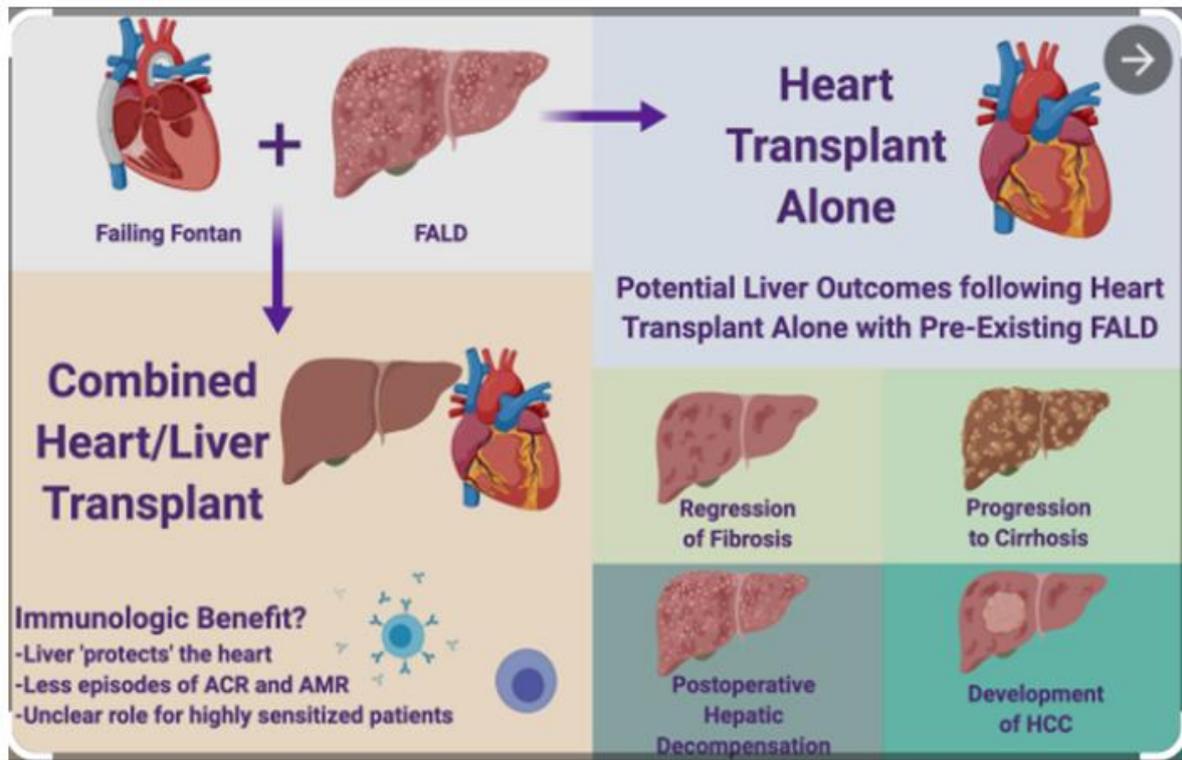
Fig. 2. Liver outcomes following heart transplant

The widespread use of heart transplantation (HT) and considerable breakthroughs in medical and surgical treatments have drastically altered the profile of CH patients. As a result, compared to previous reports, cardiac cirrhosis due to non-congenital HF is decreasing, ischemic cardiomyopathy has superseded rheumatic HF as the primary cause of HF, and CH following Fontan surgery is increasing [3].

With a functional single ventricle, the latter operation is utilized to treat a variety of difficult congenital heart disorders (e.g., tricuspid or mitral atresia and hypoplastic left or right heart syndrome). It is frequently done on children aged 2 to 5 years old who have already had a superior cavopulmonary link made through the Glenn operation. By implanting a surgical shunt to divert blood from the inferior and superior vena cava to the pulmonary arteries, which passively deliver the blood to the single ventricular chamber, the Fontan procedure produces a total cavopulmonary link. Due to high-pressure nonpulsatile flow in the inferior vena cava, this bypass causes chronic hepatic venous congestion. Due to the lack of a subpulmonary ventricle, the systemic ventricle's cardiac preload is reduced, resulting in chronically low cardiac output. The damage that can impact practically all organs is caused by these hemodynamic abnormalities combined with the typical mild low arterial blood oxygen saturation. Fontan-associated liver disease is the term used to describe the functional and anatomical changes in the liver that occur as a result of this procedure. Its natural history is unknown, and we are unable to forecast and correctly identify patients who will develop clinically severe advanced liver disease at this time, Patients with congenital heart disease with single ventricular anomalies, who need to establish a Fontan circulation, are particularly vulnerable because they have high venous filling pressure with chronic hepatic congestion. Development of cirrhosis and eventually cirrhosis of the liver may occur, with associated risks of liver failure and hepatocellular carcinoma. This risk is likely to increase over the course of the patient's life [3,8-10] (Fig. 2).

5. DIAGNOSIS

The aetiology of liver injury and cardiac decompensation must be determined at the time of presentation. To rule out potential causes of liver injury such as hepatitis, medicines, gallbladder disease, and others, a thorough medical history and physical examination should

be performed. A preliminary assessment of liver function should also be performed. The most prevalent finding is an increase in total bilirubin as a result of increased indirect bilirubin. This condition has been discovered in 70% of individuals with cardiac cirrhosis, with bilirubin levels rarely exceeding 3 mg/dl. Prolonged coagulation times are a symptom of a change in the liver's synthetic activity. Despite this, rises in the international normalized ratio are uncommon, though they can occur in severe ischemia injury. In 40% of individuals, serum albumin levels can be reduced. Hypoalbuminemia is defined as a level of albumin in the blood that is less than 2.5 g/dL. In individuals with acute or chronic heart failure, this component is an independent predictor of death. The degree of serum albumin loss is unrelated to the severity of liver injury. This is because albumin loss is more likely to occur as a result of intestinal edema caused by heart failure, resulting in protein-losing enteropathy and malnutrition. Perfusion damage can be distinguished from other causes of acute hepatitis by a significant increase in LDH and an ALT/LDH ratio of less than 1.5 [2,11-14].

Radiographs play a minor role in the diagnosis of CH. Cardiac chamber enlargement due to heart failure, pulmonary artery enlargement due to cor pulmonale, or pericardial calcifications in constrictive pericarditis can all be seen on chest radiographs. It's possible that the vena cava and azygos veins are engorged [7].

These are useful physical examination findings:

Edema: Edema is defined as a palpable swelling produced by expansion of the interstitial fluid volume; when massive and generalized, the excess fluid accumulation is called anasarca. A variety of clinical conditions are associated with the development of edema, including heart failure, cirrhosis, and the nephrotic syndrome, as well as local conditions such as venous and lymphatic disease or malignant ascites.

Edema usually affects the lower extremities and dependent areas, and in cases of advanced and untreated heart failure, it can proceed to anasarca. Chronic edoema is linked to pigmentation, induration, and cellulitis in the lower extremities [1].

Jugular venous pressure:

- The jugular venous pressure is high. Applying pressure to the right upper quadrant for up to 1 minute can cause further distention of the neck veins (ie, hepatojugular reflux).
- Constrictive pericarditis, right ventricular heart failure, tricuspid stenosis, or cor pulmonale can all cause a paradoxical rise in jugular venous pressure on inspiration (the Kussmaul sign).
- Large a waves can be seen on right atrial pressure recordings, indicating increased right atrial pressure that can be mistaken for presystolic liver pulsations.
- Trichuspid regurgitation is indicated by prominent v waves and a fast y decline. In severe tricuspid insufficiency, a systolic, or c-v, wave develops, which might manifest as systolic liver pulsations. [1]

On a lung examination, rales indicate biventricular CHF. Pleural effusion can also cause a reduction in basilar breath sounds [1].

Hepatomegaly:

- Hepatomegaly is a frequent condition that manifests as a firm, rigid, and possibly pulsatile liver.
- Cardiac ascites is caused by an increase in hydrostatic pressure inside the hepatic veins and the peritoneal venous drainage system. Ascites can also be exacerbated by protein-losing enteropathy, which results in a drop in plasma oncotic pressure.
- Splenomegaly is a condition that can be find [1].

CT and magnetic Resonance Imaging (MRI): In CH, the kinetics of IV contrast enhancement are often aberrant. An IV contrast bolus injection delivered into the upper extremities in the setting of right heart failure may transiently reflux into the hepatic veins. Reduced portal venous flow is caused by stasis in the hepatic veins as a result of congestion. Due to decreased portal venous flow and low cardiac output, bolus arrival to the liver is abnormally delayed and transit through the parenchyma is poor. Slow systemic circulation is indicated by an abnormally early phase of enhancement despite conventional contrast bolus timing (a late arterial phase of enhancement when utilizing standard portal venous timing). The hepatic veins and Inferior vena cava (IVC) are commonly dilated, and these can be seen on Computed Tomography (CT) or MRI, as well as with ultrasound. Atypical hepatic venous to hepatic venous shunts can

arise in extreme situations. Even when both hepatic morphological signs indicating cirrhosis and indications suggesting portal hypertension, such as splenomegaly and ascites, portosystemic shunts are frequently missing. Cirrhosis and a high transhepatic pressure gradient are both indicated by the existence of portosystemic shunts. Periportal edema is detected on CT and MRI when the perivascular lymphatics are engorged. The edema may be observed extending all the way around the portal tracts.In the delayed phase, extracellular contrast media may diffuse into the periportal lymphatic region, resulting in a ring of enhancement that might be mistaken for a luminal thrombus [7,15-19].

It's crucial to look into the possible causes of heart failure aggravation. Cardiac enzymes and brain peptide natriuretic peptide (BNP) levels could be abnormally high. Arrhythmias, ischemia changes, and right ventricular hypertrophy can all be seen on an ECG. An echocardiography is a crucial test for determining the aetiology of congestive hepatitis. Valvular disease, wall motion abnormalities, pulmonary artery systolic pressure, mitral inflow, atrial and ventricular size, and pericardial effusion or constrictive pericarditis are all assessed by an echo. The examination of the inferior vena cava (IVC) would aid in determining the volume status and pressure between the right atrium and the hepatic circulation. Right-sided cardiac illness with elevated right atrial filling pressures is indicated by IVC respiratory fluctuation (usually greater than or equal to 50% constriction during inspiration) or IVC diameter larger than or equal to 2.3 cm. The aetiology of the clinical appearance can also be determined by CT and MRI examinations of heart and hepatic problems. Imaging evaluation should be explored due to the prevalent signs and symptoms of biliary illness. Abdominal ultrasound can rule out acute obstructive illness or hepatic vein thrombosis by evaluating the biliary system and gallbladder. Paracentesis should be performed if ascites is present. This fluid has a high protein concentration, usually greater than 2.5 g/dl. This rise in protein levels could be the result of a hepatic lymphatic rupture resulting in the leakage of protein-rich fluid. Portal hypertension is indicated by a serum to ascites albumin gradient serum-ascites albumin gradient (SAAG) greater than 1.1 [2].

A moderate hyperbilirubinemia, which can be seen in up to 70% of individuals with chronic CH, is the most common laboratory abnormality. Total bilirubin levels rarely reach 3 mg/dL, and serum bilirubin levels have been demonstrated to correlate with right atrial pressures. Aspartate amino transferase (AST) levels in the blood may be somewhat high (2–3 times normal). AST values may be considerably high in abrupt cardiac decompensation, indicating acute liver injury. The source of AST rise in these patients is most likely ischemia damage, and AST levels are related to the amount of zone 3 hepatocyte necrosis. In CH, the liver's synthetic function is usually retained. Nutritional inadequacy, edoema, or protein-losing enteropathy can all cause mild hypoalbuminemia (rarely less than 2.5 g/dL). The levels of brain natriuretic peptide are usually high [7].

6. HISTOPATHOLOGY

The congested liver is described as having dark centrilobular zones reflecting sinusoidal congestion alternating with pale periportal zones with normal or fatty liver tissue. Passive hepatic congestion is characterised by sinusoidal dilatation, congestion, and hepatocyte atrophy on histological examination. Extravasation of red blood cells into the Disse space may be precipitated by elevated hepatic venous pressures. Right atrial and hepatic pressures have been linked to the amount of necrosis, inflammation, and dilatation, while ischemia can also cause necrosis. Perivenular fibrosis and, eventually, cirrhosis are caused by chronic congestion [4,20-24].

Traditional fibrosis severity scores, such as METAVIR, may not be precise enough in the context of CH, particularly in intermediate phases of fibrosis. Because they do not sufficiently depict the inverted lobulation pattern of fibrosis seen in CH, this is the case. The Congestive Hepatic Fibrosis Score, developed by Dai et al., is a four-tiered approach for histologically assessing liver fibrosis in CH patients. Despite the fact that this scoring method has been progressively used in recent clinical outcome studies and has been tested for repeatability among pathologists, it is still not generally used in clinical practise. Furthermore, liver biopsies may not be a reliable predictor of post-HT outcomes. Louie et al. showed that the presence of bridging fibrosis was not substantially related with post-operative survival or post-operative liver failure in a retrospective research, leading them to infer that patients with bridging fibrosis may still be viable candidates for isolated HT. Dhall et al. described

similar findings. Despite these drawbacks, liver biopsy is still useful in assessing the stage of liver disease, ruling out hepatocarcinoma and other etiologies of liver disease, and determining candidacy for solo HT or combination heart-liver transplantation [3,25-27].

7. MANAGEMENT

The control of the underlying cardiac disease, as well as the optimization of cardiac output, are the mainstays of congestive hepatopathy treatment. Diuresis can help with hepatic congestion, but it must be used with caution to avoid causing hepatic ischemia. Hemodynamic therapy may be able to reverse the early stages of congestive hepatitis. Improvement in liver injury has been observed after the implantation of a left ventricular assist device (LVAD) or cardiac transplantation in severe instances that have failed to respond to medical treatment. Because studies show that patients with higher Model for End-Stage Liver Disease (MELD) and modified MELD scores have worse outcomes 30 days after surgery and lower 10-year survival rates, liver function is an important variable when considering cardiac transplantation in patients with heart failure [4].

The only proven treatment for ACLI in people with underlying disorders is to control the underlying conditions. Despite the lack of evidence, some specialists advise utilising N-acetylcysteine, avoiding excessive vascular filling to avoid passive liver congestion, and recommending the use of dobutamine in patients with a low cardiac index because of its inotropic and vasodilating properties [3].

Familial amyloid polyneuropathy (FAP), HF with cardiac cirrhosis (including congenital heart abnormalities requiring Fontan operation), and HF with associated noncardiac cirrhosis are the three main indications for CHLT (combined heart-liver transplantation). The liver is transplanted in FAP to protect the cardiac allograft from further deterioration. The fundamental motivator of mixed organ transplant is cardiac failure, regardless of the rationale. Until recently, FAP was the most common sign of CHLT in the United States [3,28-30].

8. CONCLUSION

Cirrhosis of the liver can be induced by any right-sided pathology that leads to right-sided heart failure, which leads to increased venous congestion and pressure in the hepatic sinusoids, there are many reasons to that and thus the treatment of cardiac cirrhosis (congestive hepatopathy) begins with diagnosis and thus identifying the underlying cause of the disease. And thus, treatment of the disease is treatment of the underlying cause itself which is the cardiac disease. Diuresis can help with hepatic congestion, but it must be used with caution to avoid causing hepatic ischemia. In severe cases transplantation may be needed. We hope in the future in presence of better diagnostic and treatment techniques.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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