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ALCOHOLIC CIRRHOSIS:ETIOLOGY, DIAGNOSIS, TREATMENT

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Annotation: This article provides an overview of alcoholic cirrhosis, the most advanced form of alcohol-related liver disease (ARLD). It examines the etiology, emphasizing chronic alcohol consumption and its metabolic consequences leading to hepatic fibrosis. The paper explains the pathophysiology, including the biochemical mechanisms of ethanol metabolism and resultant oxidative stress. Clinical manifestations, diagnostic approaches, and treatment strategies-ranging from alcohol abstinence to liver transplantation-are discussed. The study also highlights the importance of prevention, nutritional rehabilitation, and patient education to reduce global morbidity and mortality associated with alcoholic cirrhosis.

Keywords: Alcoholic cirrhosis; Alcohol-related liver disease (ARLD); Ethanol metabolism; Hepatic fibrosis; Liver biopsy.

Introduction

Alcoholic cirrhosis is the final and most severe stage of alcohol-related liver disease (ARLD). It results from chronic and excessive alcohol consumption leading to irreversible scarring (fibrosis) of the liver tissue. This condition causes progressive liver failure and is a major cause of morbidity and mortality worldwide.

Etiology

The primary cause of alcoholic cirrhosis is long-term excessive alcohol intake. Ethanol metabolism in the liver generates toxic metabolites, mainly acetaldehyde, which induces oxidative stress, inflammation, and hepatocellular injury. Over time, continuous liver cell death and regeneration lead to fibrosis and architectural distortion of the hepatic lobules.
Risk factors include:
• Chronic alcohol consumption (more than 80 g/day in men, 40 g/day in women for over 10 years)
• Genetic predisposition (e.g., variants in PNPLA3 gene)
• Coexisting hepatitis B or C infection
• Malnutrition and vitamin deficiency (especially folate and thiamine)
• Female gender (greater susceptibility to alcohol toxicity)

Pathophysiology

Alcohol metabolism increases $NADH/NAD^+$ ratio, causing fatty liver (hepatic steatosis). Continued alcohol exposure triggers inflammatory reactions (alcoholic hepatitis), and prolonged inflammation leads to fibrotic changes and cirrhosis. Fibrosis replaces normal hepatic

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parenchyma with nodules, leading to portal hypertension and impaired detoxification function.

Clinical Manifestations

Symptoms develop gradually and include fatigue, weakness, anorexia, jaundice, ascites, spider angiomas, palmar erythema, hepatomegaly, hepatic encephalopathy, peripheral edema, and weight loss.

Diagnosis

Diagnosis is based on clinical history, laboratory findings, and imaging studies.
1. Laboratory tests: Elevated AST > ALT (2:1 ratio), increased bilirubin, prolonged PT/INR, low albumin.
2. Imaging: Ultrasound and CT/MRI show irregular liver surface, nodularity, and splenomegaly.
3. Liver biopsy: Confirms micronodular cirrhosis with fibrosis and fatty change.

Treatment

There is no cure for alcoholic cirrhosis except liver transplantation, but treatment aims to slow progression and manage complications.
1. Lifestyle modification: Complete alcohol abstinence and nutritional support.
2. Medical management: Corticosteroids, diuretics, lactulose, beta-blockers, and antibiotics.
3. Liver transplantation: The only definitive treatment for end-stage liver disease.

Prognosis

Prognosis depends on the stage of cirrhosis and continued alcohol use. Abstinent patients have a significantly better survival rate. Child-Pugh and MELD scores are used to assess disease severity and transplant priority.

Prevention

Prevention includes avoiding alcohol, regular liver function testing, vaccination against hepatitis A and B, and public education on alcohol-related harm.

Conclusion. Alcoholic cirrhosis is a preventable but potentially fatal disease. Early diagnosis, strict alcohol abstinence, nutritional rehabilitation, and appropriate management of complications can improve survival and quality of life. Public awareness and preventive strategies remain the cornerstone in reducing its global burden.

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