Pathophysiology of Alcoholic Hepatitis: Inflammation to Liver Damage

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DESCRIPTION

Alcoholic hepatitis is a serious liver condition resulting from prolonged and excessive alcohol consumption. It represents a spectrum of liver damage ranging from mild inflammation to severe liver failure, with potentially life-threatening consequences.

Pathophysiology

Alcoholic hepatitis develops when prolonged alcohol abuse leads to inflammation and damage to liver cells. The liver plays a key role in metabolizing alcohol, but chronic exposure to ethanol disrupts normal liver function. This disruption triggers an inflammatory response, causing hepatocyte injury and activation of immune cells within the liver. Over time, ongoing inflammation can progress to fibrosis (scarring) and cirrhosis, further impairing liver function.

Clinical presentation and diagnosis

Individuals with alcoholic hepatitis may exhibit a variety of symptoms, such as jaundice (yellowing of the skin and eyes), abdominal pain or discomfort, ascites (fluid buildup in the abdomen), hepatic encephalopathy (confusion or changes in mental status due to liver dysfunction) and easy bruising or bleeding tendencies.

Diagnosing alcoholic hepatitis necessitates a comprehensive evaluation involving medical history, physical examination and laboratory tests. Blood tests may reveal elevated liver enzymes (such as Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT)), an elevated bilirubin level and abnormal coagulation parameters. Imaging studies, such as ultrasound or Computed Tomography (CT) scans, can help assess liver size, detect ascites or evaluate for complications like portal hypertension.

Treatment

The following are the treatment methods involved;

Abstinence from alcohol: The treatment for alcoholic hepatitis is complete abstinence from alcohol. Without cessation of drinking,

the liver damage will continue to progress, potentially leading to irreversible liver failure.

Nutritional support: Many patients with alcoholic hepatitis are malnourished due to poor dietary intake and impaired nutrient absorption. Nutritional support, including vitamin supplementation and high-calorie diets, can help improve liver function and promote healing.

Medications: In severe cases of alcoholic hepatitis, corticosteroids like prednisolone may be prescribed to reduce liver inflammation and improve short-term survival rates. Pentoxifylline medication has anti-inflammatory properties and may be considered as an alternative or adjunct therapy in patients who cannot tolerate steroids.

Management of complications: Treatment may also involve managing complications such as ascites with diuretics, addressing infections promptly with antibiotics and monitoring and managing hepatic encephalopathy.

Liver transplantation: In cases of severe alcoholic hepatitis that do not respond to medical therapy and where liver failure is imminent, liver transplantation may be considered as a life-saving option. However, strict criteria must be met, including abstinence from alcohol and comprehensive evaluation.

Prevention strategies

Preventing alcoholic hepatitis begins with education and awareness about the risks of excessive alcohol consumption. Public health initiatives aimed at reducing alcohol abuse and promoting responsible drinking habits are important. Encouraging early intervention and treatment for alcohol use disorder can prevent the progression to liver damage and alcoholic hepatitis.

CONCLUSION

Alcoholic hepatitis is a significant health burden worldwide, affecting individuals who engage in chronic alcohol abuse. Effective management requires a multidisciplinary approach,

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enclosing medical treatment, nutritional support and most importantly, cessation of alcohol consumption. Early diagnosis, prompt intervention and supportive care are essential in improving outcomes and preventing the progression to

advanced liver disease. By addressing both the medical and social aspects of alcoholic hepatitis, healthcare providers can mitigate its impact on patients' health and well-being.