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ALCOHOLIC HEPATITIS AND ITS ASSESSMENT USING THE GLASGOW ALCOHOLIC HEPATITIS SCORE

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Abstract. Alcoholic hepatitis is an inflammatory liver disease caused by excessive and prolonged alcohol consumption. It represents a significant health burden worldwide due to its association with liver failure, cirrhosis, and increased mortality. The pathophysiology of alcoholic hepatitis involves hepatocellular injury, oxidative stress, inflammatory cytokine release, and progressive fibrosis. Clinical manifestations range from mild symptoms to severe presentations, including jaundice, abdominal pain, hepatomegaly, ascites, and hepatic encephalopathy. Assessment of disease severity and prognosis is essential for patient management. The Glasgow Alcoholic Hepatitis Score (GAHS) is a useful tool for evaluating disease severity and predicting outcomes. Understanding the etiology, pathophysiology, clinical features, and prognostic factors of alcoholic hepatitis is important for early diagnosis and appropriate treatment, ultimately improving patient outcomes.

Keywords: Alcoholic hepatitis, alcohol-related liver disease, hepatomegaly, jaundice, liver inflammation, Glasgow Alcoholic Hepatitis Score, prognosis, liver cirrhosis.

Introduction. Excessive alcohol consumption could result in fatty liver disease or steatosis, alcoholic hepatitis (AH), and eventually cirrhosis. Alcoholic hepatitis is a severe syndrome of alcoholic liver disease (ALD), characterized by rapid onset of jaundice, malaise, tender hepatomegaly, and subtle features of systemic inflammatory response. Alcoholic hepatitis usually progresses to cirrhosis if drinking is continued. For those who discontinue alcohol, hepatitis returns to normal within a few months but the cirrhosis that has already occurred does not reverse

Etiology. The onset of jaundice within 60 days of heavy alcohol consumption (more than 50 g/day) for a minimum of 6 months. Serum bilirubin more than 3 mg/dL. Elevated aspartate aminotransferase (AST) to 50 U/L to 400 U/L AST:ALT (alanine aminotransferase) ratio of more than 1.5 No other cause of acute hepatitis. While using the terminology alcoholic hepatitis, it is important to understand the difference between alcoholic steatohepatitis (ASH) and alcoholic hepatitis (AH). About 20% to 40% of those who drink alcohol in heavy amounts and have fatty liver eventually develop liver inflammation, which is known as ASH. ASH is a diagnosis based on liver histology, while AH is a clinical diagnosis. The typical features of ASH on liver biopsy are steatosis, hepatocyte ballooning, infiltration of neutrophils, Mallory-Denk hyaline inclusions, and zone 3 perivenular injury with pericellular fibrosis or chicken-wire pattern of fibrosis. AH, on the other hand, is characterized by a history of chronic heavy alcohol consumption until at least 3 to 4 weeks before the onset of jaundice, fever, tachycardia, tachypnea, hepatomegaly, leukocytosis with neutrophilia, and an AST:ALT elevation greater than 1.5:1 with the absolute value of AST/ALT typically never exceeding 500 U/L. AH can occur in patients with any stage of alcoholic liver disease. Although the amount of alcohol ingested is the most important risk factor for the development of chronic liver disease, the progression

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to alcohol-induced chronic liver disease is neither dose-dependent nor is the correlation with the quantity of alcohol consumed and liver injury linear. Even shorter durations of alcohol abuse could lead to AH. A typical patient would be between 40 to 60 years of age with a history of more than 100 g/day of alcohol consumption for a decade, in whom you have ruled out other causes of acute hepatitis. Risk factors include a high BMI (body mass index), female sex, and having a genetic variant of patatin-like phospholipase domain-containing protein 3 (PNPLA3). Clinical jaundice is a poor prognostic factor. Acute binge drinking is likely the trigger for AH in patients with a history of chronic, heavy alcohol abuse

Pathophysiology. Alcohol undergoes an oxidative metabolic pathway in the hepatocytes, leading to a reduced ratio of the nicotinamide adenine dinucleotide (NAD) to NADH. This promotes lipogenesis by inhibiting the oxidation of triglycerides and fatty acids. Another known mechanism of alcohol-induced liver injury is the translocation of endotoxins in the form of lipopolysaccharides (LPS), from the intestines into the hepatocytes. In the hepatic Kupffer cells, the LPS binds to CD 14 and toll-like receptor 4 to release a barrage of reactive oxygen species (ROS). The ROS activates the release of cytokines such as tumor necrosis factor-alpha (TNF alpha), interleukin-8, monocyte chemotactic protein 1 (MCP-1), and platelet-derived growth factor (PDGF), all of which leads to the accumulation of neutrophils, macrophages, and systemic clinical features of alcohol injury. Recent studies indicate that patients with specific intestinal dysbiosis have been increasingly susceptible to alcoholic liver disease and AH

Clinical Manifestations. Some authors distinguish four major clinical features of alcoholic hepatitis: *Pseudo-infectious syndrome:* persistent low-grade fever (38–38.5°C), accompanied by polymorphonuclear leukocytosis and an elevated erythrocyte sedimentation rate (ESR). Repeated bacteriological examinations are usually negative. *Painful abdominal syndrome*: constant pain in the right upper quadrant or epigastric region, sometimes presenting as severe episodic attacks. *Hepatargia syndrome:* characterized by jaundice, ascites, hemorrhagic diathesis, and bleeding manifestations from various organs.
Hepatomegaly: enlargement of the liver. The presence of asterixis (flapping tremor) is generally considered a sign of poor prognosis in alcoholic hepatitis. In the terminal stage of the disease, hepatorenal syndrome may develop.

Prognostic Assessment. The severity and prognosis of acute alcoholic hepatitis can be evaluated using the Glasgow Alcoholic Hepatitis Score (GAHS).

Glasgow Alcoholic Hepatitis Score (GAHS)

Score given	1	2	3
Age	<50	≥50	–
WCC (109/L)	<15	≥15	–
Urea (mmol/L)	<5	≥5	
PTr/INR	<1.5	1.5-2.0	>2.0
Bilirubin (µmol/L)	<125	125-250	>250

Picture1. *Glasgow Alcaholic Hepatitis Score*

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Interpretation: A total score of 9 or higher indicates a poor prognosis and increased risk of mortality.

Conclusion. Alcoholic hepatitis is a serious liver disorder resulting from chronic excessive alcohol consumption. The disease is characterized by complex pathophysiological mechanisms leading to liver inflammation and progressive hepatic damage. Clinical manifestations such as jaundice, abdominal pain, hepatomegaly, and complications including hepatorenal syndrome significantly affect patient prognosis. Early recognition of symptoms and accurate assessment of disease severity using prognostic tools such as the Glasgow Alcoholic Hepatitis Score are essential for effective management. Increased awareness of risk factors, timely diagnosis, and appropriate therapeutic interventions can reduce complications and improve the quality of life and survival of affected patients.

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