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Non-Alcoholic Fatty Liver Disease (NAFLD): A growing metabolic epidemic in youth and adults.

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Introduction

Over the past two decades, NAFLD has transformed from a relatively obscure liver condition into a major global health burden. It now affects approximately 25% of the world's population, with rising trends observed in both high-income and low-income nations. Worryingly, NAFLD is no longer limited to middle-aged adults—it is now being diagnosed in adolescents and even young children, often as a result of poor diet and sedentary lifestyles. NAFLD encompasses a spectrum of liver conditions, ranging from simple steatosis (fat accumulation without inflammation) to Non-Alcoholic Steatohepatitis (NASH), which involves inflammation and can progress to fibrosis, cirrhosis, or Hepatocellular Carcinoma (HCC) [1].

In adults, the global prevalence of NAFLD is closely aligned with obesity and type 2 diabetes rates. The Middle East and South America report the highest prevalence (over 30%), while Southeast Asia is experiencing a sharp increase, largely due to urbanization and westernized dietary habits. Alarming, NAFLD in children has become a major concern. Recent studies estimate that 10-20% of children in Western countries now exhibit hepatic steatosis. Among obese children, the prevalence may exceed 50%. Pediatric NAFLD is often underdiagnosed and, if left unmanaged, may predispose individuals to advanced liver disease in early adulthood [2].

The development of NAFLD is a multifactorial process. The most widely accepted explanation is the “two-hit” hypothesis. Accumulation of triglycerides in liver cells due to insulin resistance and increased lipolysis. Oxidative stress,

mitochondrial dysfunction, and inflammatory cytokines lead to hepatocellular injury and fibrosis. However, more recent models suggest a “multiple parallel hits” theory, incorporating genetic predisposition, gut microbiota imbalances, dietary factors (e.g., high fructose intake), and environmental toxins. Genetics also play a significant role, with certain gene variants (such as PNPLA3 and TM6SF2) associated with increased risk and progression. NAFLD is often asymptomatic and discovered incidentally through elevated liver enzymes or imaging studies. Common symptoms, if present, include fatigue, malaise, and mild abdominal discomfort [3].

Diagnosis involves a combination of Elevated ALT and AST (though not always). Ultrasound is commonly used for initial detection; MRI and CT offer greater sensitivity. The gold standard for distinguishing simple steatosis from NASH and assessing fibrosis stage. Non-invasive alternatives like transient elastography (FibroScan) and serum fibrosis scores (e.g., FIB-4, NAFLD fibrosis score) are increasingly used in routine practice. NAFLD rarely occurs in isolation. It is strongly associated with components of metabolic syndrome. Moreover, NAFLD significantly increases the risk of cardiovascular disease, which remains the leading cause of death in these patients, even more than liver-related complications. It is also associated with chronic kidney disease, polycystic ovary syndrome (PCOS), and sleep apnea [4].

Currently, there is no FDA-approved pharmacological treatment specifically for NAFLD. The cornerstone of therapy remains lifestyle modification. Reducing body weight by 7–10% can lead to histological improvements. A

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Mediterranean diet rich in fiber, whole grains, fruits, and healthy fats has shown benefit. At least 150 minutes of moderate-intensity exercise per week is recommended. Emerging drugs, such as obeticholic acid, GLP-1 receptor agonists (e.g., liraglutide), and metabolic regulators, are under investigation. Vitamin E and pioglitazone may be considered in selected non-diabetic patients with biopsy-proven NASH. NAFLD represents a major healthcare challenge, not just for hepatologists but for all sectors of medicine. Its progression can be silent yet devastating, leading to end-stage liver disease and costly complications. Moreover, its strong links with lifestyle and socioeconomic factors make it a crucial target for preventive strategies. Early screening, particularly in high-risk groups (obese, diabetic, or family history), must be integrated into primary care. Education campaigns promoting healthy eating and active lifestyles, especially among youth, are essential in curbing the epidemic [5].

Conclusion

NAFLD is more than a liver condition it is a metabolic syndrome manifesting in the liver. As its prevalence rises in both adults and youth, particularly in urbanized societies, there is an urgent need for early diagnosis, lifestyle-focused management, and public health intervention. Tackling NAFLD effectively requires a multidisciplinary approach that addresses the root

causes of metabolic dysfunction at both individual and societal levels.

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