DOI: https://doi.org/10.22516/25007440.991

Editorial

Fatty Liver: A Heterogeneous Disorder with a High Global Prevalence

Mauricio Orrego.[1*]

OPEN ACCESS

Citation:
Orrego M. Fatty Liver: A Heterogeneous Disorder with a High Global Prevalence. Revista. colomb. Gastroenterol. 2022;37(4):353-354. https://doi.org/10.22516/25007440.991

[1] Gastroenterologist and Hepatologist. Clínica Las Américas AUNA and Clínica Las Vegas Quirón Group. Ex-president of the ACH. Medellín, Colombia.

***Correspondence:** Mauricio Orrego.
mauricioorrego@hotmail.com

Received: 20/11/2022
Accepted: 22/11/2022

Non-alcoholic fatty liver disease (NAFLD) is the most prevalent liver disease globally and represents a real public health problem. NAFLD includes a spectrum of conditions, including hepatic steatosis, non-alcoholic steatohepatitis (NASH), and liver fibrosis, with the risk of progressing to cirrhosis, hepatocellular carcinoma (HCC), and eventually the need for liver transplantation[(1)]. The global prevalence of NAFLD in the adult population is estimated to be 23%-25% and varies by region; the areas with the highest prevalence are the Middle East (32%) and South America (30%), while Africa has the lowest (13%). The prevalence of NAFLD in 1990–2017 has increased from 8.2% to 10.9%[(2)], which has to do with the increased prevalence of obesity and type 2 diabetes mellitus worldwide[(3)]. Up to 20% of people with NAFLD may develop NASH. The development of NASH is the most critical stage in the pathogenesis of liver damage in patients with NAFLD.

Insulin resistance and obesity result in metabolic injury to hepatocytes, with activation of lipogenesis, lipid accumulation, and lipotoxicity exacerbating hepatocyte damage. Adipose tissue contributes to insulin resistance by secreting adipokines and cytokines (e.g., leptin and adiponectin). Endoplasmic reticulum (ER) stress causes the secretion of inflammatory, fibrogenic, and chemokine cytokines (e.g., interleukin-6 [IL-6], tumor necrosis factor-alpha [TNF-α], interleukin-1 beta [IL-1β], and transforming growth factor-beta 1 [TGF-β1]). ER stress is associated with changes in the microbiota (prevalence of *Firmicutes* over *Bacteroidetes* 13), increased gut permeability, the release of bacterial products such as lipopolysaccharides into the circulation, activation of Toll-like receptor-dependent signaling pathways (specifically TLR-4), and the recruitment and activation of inflammatory cells and myofibroblasts in the compromised liver[(4, 5)]. Liver fibrosis is the most crucial determining factor of liver-related and non-liver-related outcomes in patients with NAFLD[(6)].

Written by Dr. Prieto et al.[(7)], the article "Non-alcoholic fatty liver disease part 1: general aspects, epidemiology, pathophysiology, and natural history" in this issue provides a clear and concise review of general, epidemiological, pathophysiology, and natural history features of fatty liver.

A critical aspect of the review is the pathophysiology, emphasizing that NAFLD is a complex and very heterogeneous disorder derived from the interaction of multiple genetic, epigenetic, environmental, and cultural factors. All these elements combined lead to an accumulation of liver fat, insulin resistance, and hormonal and intestinal microbiota alterations, causing hepatocellular damage by forming oxygen-free radicals and activating liver fibrogenesis.

Another crucial part of the review is a forecast of what could happen in Colombia based on international data. It is estimated that, of 15 million people with fatty liver in Colombia, three million would have NASH. In three years, some 600,000 people could have stage 1-3 fibrosis, and of the patients with stage 3 fibrosis, 20% could develop cirrhosis with the consequent complications of liver failure and HCC. This analysis reveals that NAFLD is an actual public health issue; therefore, primary care physicians must be trained to timely detect fatty liver and make an adequate stratification to determine when to refer the patient to a specialist based on non-invasive fibrosis tests[8].

REFERENCES

1. El-Kassas M, Cabezas J, Coz PI, Zheng MH, Arab JP, Awad A. Nonalcoholic Fatty Liver Disease: Current Global Burden. Semin Liver Dis. 2022;42(3):401-412. https://doi.org/10.1055/a-1862-9088
2. Ge X, Zheng L, Wang M, Du Y, Jiang J. Prevalence trends in nonalcoholic fatty liver disease at the global, regional and national levels, 1990-2017: a population-based observational study. BMJ Open. 2020;10(8):e036663. https://doi.org/10.1136/bmjopen-2019-036663
3. Younossi ZM. Non-alcoholic fatty liver disease - a global public health perspective. J Hepatol 2019;70(03):531-544. https://doi.org/10.1016/j.jhep.2018.10.033
4. Kobayashi T, Iwaki M, Nakajima A, Nogami A, Yoneda, M. Current Research on the Pathogenesis of NAFLD/NASH and the Gut-Liver Axis: Gut Microbiota, Dysbiosis, and Leaky-Gut Syndrome. Int J Mol Sci. 2022;23(19):11689. https://doi.org/10.3390/ijms231911689
5. Carvalho-Gontijo R, Han C, Zhang L, Zhang V, Hosseini M, Mekeel K, et al. Metabolic Injury of Hepatocytes Promotes Progression of NAFLD and AALD. Semin Liver Dis. 2022;42(3):233-249. https://doi.org/10.1055/s-0042-1755316
6. Kanwal F, Shubrook JH, Adams LA, Pfotenhauer K, Wai-Sun Wong V, Wright E, et al. Clinical Care Pathway for the Risk Stratification and Management of Patients With Nonalcoholic Fatty Liver Disease. Gastroenterology. 2021;161(5):1657-1669. https://doi.org/10.1053/j.gastro.2021.07.049
7. Prieto-Ortiz JE, Sánchez-Luque CB, Ortega-Quiróz R. Hígado graso (parte 1): aspectos generales, epidemiología, fisiopatología e historia natural. Rev Colomb Gastroenterol. 2022;37(4):420-433. https://doi.org/10.22516/25007440.952
8. Castera L, Boursier J. Noninvasive Algorithms for the Case Finding of "At-Risk" Patients with NAFLD. Semin Liver Dis. 2022;42(3):313-326. https://doi.org/10.1055/s-0042-1751081