

Chronic liver disease and diet

Abstract

Patients with chronic liver disease typically experience malnutrition, which may have an impact on their clinical outcomes in the short and long term. Therefore, dietary intervention may be crucial in the treatment of patients with chronic liver disease. The influence of malnutrition on the clinical outcome of chronic liver disease is reviewed and summarised, along with recent advancements in nutritional assessment, management approaches, and malnutrition. The management and prevention of malnutrition in these patients will be suggested.

The most prevalent liver disease in the world is non-alcoholic fatty liver disease (NAFLD), which is linked to other illnesses like obesity, metabolic syndrome, and diabetes mellitus. There is no agreement on the pharmaceutical treatment, and the processes of the underlying disease's genesis and progression are not well understood. Exercise, dietary therapy, and weight loss are all components of the gold standard NAFLD treatment. There is, however, scant scientific data on the relationship between nutrition, exercise, and NAFLD specifically. Numerous dietary strategies, like the Mediterranean and DASH diets, are utilised to treat various cardio metabolic risk factors, such as insulin resistance and type-2 diabetes mellitus (T2DM), although their role in NAFLD has been studied based on their constituent parts. With an emphasis on choosing the best non-pharmacological treatment to recommend for NAFLD, this review discusses the implications of current food and exercise regimens, including Brazilian and other guidelines.

Keywords: cirrhosis of the liver • chronic liver disease • malnutrition • nutrition • non-alcoholic liver disease • diet • sarcopenia

Introduction

Hepatic steatosis (HS), which is diagnosed when there is only fat accumulation, non-alcoholic steatohepatitis (NASH), which is characterized by inflammation, ballooning, and moderate fibrosis in addition to steatosis, and the progression from NASH to cirrhosis and hepatocellular carcinoma are all included under the umbrella term of non-alcoholic fatty liver disease (NAFLD) (HCC). NAFLD happens in people who don't drink much alcohol (20–40 g/day), while having identical morphological results to the alcohol-induced lesion. Parenteral nutrition (PN) is the intravenous administration of nutrients such vitamins, trace minerals, amino acids, glucose, lipids, and electrolytes [1]. Patients who cannot tolerate enteral feeding can get parenteral nourishment as a life-saving therapy. The long-term use of PN is however constrained by parenteral nutrition-associated liver disease (PNALD; also known as intestinal failure-associated liver disease) that often manifests in such patients, particularly infants and children [2].

Patients with type-2 diabetes (T2DM) are at a particularly high risk for developing the progressive forms of NAFLD, NASH, advanced liver fibrosis, hepatocellular carcinoma

Jacob Herring*

School of Medicine and Surgery, University of Milano-Bicocca, Lombardy, Italy

*Author for correspondence:
Jacob06@gmail.com

Received: 01-Aug-2022, Manuscript No. actvr-22-72118; **Editor assigned:** 04-Aug-2022, PreQC No. actvr-22-72118 (PQ); **Reviewed:** 18-Aug-2022, QC No. actvr-22-72118; **Revised:** 23-Aug-2022, Manuscript No. actvr-22-72118 (R); **Published:** 30-Aug-2022, **DOI:** 10.37532/actvr.2022.12(4).82-85

development, and liver-related mortality, according to growing evidence. NAFLD prevalence in these people ranges from 42.6% to 79%. It is important to note that these figures differ between nations and that roughly 13% of cases have already shown symptoms of cirrhosis. NAFLD and NASH were prevalent in a sample of American people with T2DM and normal serum levels of aminotransferases at 76% and 56%, respectively. Metabolomics, a global and all-encompassing analytical method for identifying a group of metabolites in biochemical and biological samples, has been used to find biomarkers and explain disease mechanisms because it provides the most downstream information about biochemical pathways by examining small organic molecules [3].

Due to their rising frequency, poor prognoses, and financial burden on the healthcare system, non-alcoholic fatty liver disease (NAFLD) and chronic kidney disease (CKD) are both major global public health issues. The most prevalent cause of chronic liver disease worldwide, particularly in poorer nations, is non-alcoholic fatty liver disease. NAFLD encompasses a broad spectrum of conditions, from benign steatosis (fat accumulation in >5% of hepatocytes, especially macro vesicular, without inflammation or fibrosis) to non-alcoholic steatohepatitis (NASH), which is characterized by liver inflammation with a high propensity to progress to advanced fibrosis, liver cirrhosis, and hepatocellular carcinoma [4]. Here, we used coupled ultrahigh-performance liquid chromatography with quadrupole time-of-flight mass spectrometry (UHPLC-QTOF-MS) platform-based metabolomics to compare the liver metabolic profiles of normal and PNAFLD models in order to identify which pathways are altered in the progression to PNAFLD and the molecular mechanisms involved in the development of PNAFLD [5].

An increase in all-cause mortality was found to be independently associated with moderate to advanced stages of CKD in patients with ultrasound-detected NAFLD during a mean follow-up period of 19 years in a recent analysis of the Third National Health and Nutrition Survey database (which included approximately 11,700 American subjects). Numerous studies conducted over the past 10 years have demonstrated the link between

NAFLD and CKD, regardless of the presence or absence of established risk factors for illnesses such as obesity, hypertension, type 2 diabetes (T2DM) or metabolic syndrome [6].

Materials and Methods

We followed the guidelines outlined in the Helsinki Declaration when conducting our research. Since all of the treatments were carried out in clinical settings, a special Ethical Committee permission was not necessary. Every patient did, however, sign an informed consent form allowing their data to be distributed anonymously for research proposals. From January 2010 to December 2011, the Department of Radiology at the AKUH conducted this cross-sectional analytical investigation. Through the use of a nonprobability purposive sampling technique, the data was obtained prospectively [7]. All patients who were sent to the Aga Khan University Hospital's Radiology Department for an ultrasound-guided liver biopsy were included. Patients were disqualified if the liver biopsy's histopathology report was unavailable and the biopsy was carried out due to localized lesions or autoimmune liver disease. Patients, who couldn't get a liver biopsy because of ascites, jaundice, or abnormal blood profiles were also eliminated. During the investigation, patient confidentiality was ensured and upheld [8]. The on-call radiologist wearing the ultrasound interventional suit had at least three years of expertise doing abdominal sonography when he performed real-time ultrasound on all patients prior to the biopsy utilizing the Toshiba Nemio XG and a 3.5-5.0 MHz convex transducer. A liver ultrasound was done, both liver lobes were assessed, and a combined impression was created. Additionally, the dimensions of the liver, spleen, and portal vein were measured and recorded. Before the operation, the examining radiologist received an explanation of the ultrasonography parameters and the scoring system, and the results were recorded on a typical. The ultrasonic parameters/variables and their corresponding scoring system, which was adapted from published literature are shown [9].

The biopsy specimen was graded and staged according to the histopathology reports, which were checked through the hospital

information system and evaluated using the Bats and Ludwig scoring system. In the biopsy specimen, stage and grade were used to assess the extent and area of fibrosis and the degree and location of inflammation, respectively. The histopathological grading method defined stage 0 as having no fibrosis, and with increasing fibrosis, cirrhosis received a score of 4. Score 0 in the grading system denoted merely portal inflammation, and score 4 indicated significant widespread hepatocellular damage with bridging necrosis. The ultrasound scoring system was also divided into two categories: "A" for the evaluation of the liver's morphology, which includes the liver's surface, parenchymal echo texture, and edge, and "B" for the combined score of the liver's morphology, which includes the measurements of the liver, spleen, and portal vein [10].

Discussion

Physicians frequently underestimate the impact of pruritus on the quality of life of patients with chronic liver failure. Although the degree of pruritus varies from patient to patient, it can significantly affect a patient's psychological and mental health. Because there is so little clinical research on pruritus in liver patients, it is challenging for doctors to treat pruritic patients who are resistant to therapy. This challenge is highlighted by the dearth of clinical literature on the subject. Uncertainty regarding the underlying pathophysiology suggests that it is caused by a variety of interconnected complicated pathways with multiple etiologies. Guidelines for the initial clinical assessment, investigation, and management of pruritus in cholestasis liver disorders have been created by the European Association for the Study of the Liver (EASL). Starting with the above-mentioned straightforward medicines, the management strategy should be stepwise before progressing to more experimental therapies in resistant instances. An appropriate strategy would be to begin with UDCA, move on to cholestyramine, rifampicin, and naltrexone, and if symptoms persisted, add in treatments like sertraline. For patients who are resistant to conventional therapy, experimental therapies like UVA/B light therapy or other experimental pharmacological therapies can be used [11].

This review was limited to works that were

published in the English language and adhered to the qualifications outlined in the methodology section. A quantitative analysis was not possible due to the variety of the outcomes measured in the literature and the broad scope of this evaluation [12]. However, the broad conclusions drawn from the available evidence have been provided. New drugs for the treatment of pruritus will be the main focus of experimental research in this area in the future, but fundamental study into the underlying causes and signals of pruritus is crucial for pharmacological advancement in this area. To strengthen the evidence base on which clinical guidelines can be developed, however, there is also a clear need for focused work in phase III and IV studies comparing the clinical effectiveness of established agents and their combinations in various etiologies of liver disease and various patient subgroups [13].

Cirrhosis is the final stage of a range of chronic liver diseases. Since treatment and management options are currently being developed and improved, it is important to diagnose CLD quickly in order to detect asymptomatic patients in high-risk populations, such as those with a high frequency of viral hepatitis, and thereby improve patient outcomes. For therapy, prognosis, and surveillance, an accurate assessment of the degree of hepatic damage in fibrosis or cirrhosis before the compensation becomes clinically evident is essential. Serologic fibrosis markers such as the fibro test, the aspartate aminotransferase-to-platelet ratio index (APRI), and radiologic imaging are noninvasive techniques to evaluate the characteristics of CLD. These tests are only considered ideal and perfect if they are straightforward, affordable, and display great accuracy. The study has several restrictions [14]. The study's findings indicate high sensitivity but low specificity, necessitating more investigation to improve diagnostic accuracy. This can be done by paying attention to things like intra- and inter-observer variability, ultrasound method and equipment quality assurance, and so on. The likelihood of sampling mistakes and inter- and intraobserver variability in assessment of biopsy samples cannot be ruled out and may have also affected our results because liver histology.

Hepatic steatosis has a considerable impact

on the appearance of the liver parenchyma, although this observation was not considered in the US evaluation of the study group [15].

Conclusion

Pruritus is a persistent problem for people with liver disease, and although being a typical symptom, it can be challenging to treat. Pruritus is a disorder that many patients continue to suffer from since its cause and treatment are still poorly understood, despite a substantial amount of study on the subject. What is currently known has been summarized in this review, but more basic science research is still needed to help us understand the causes of pruritus, as well as clinical research to help individuals with chronic liver disease live better lives.

Acknowledgments

None

Conflicts of Interest

None

References

1. Rische E, Azarm A, Bergasa NV *et al.* Itch in primary biliary cirrhosis. *Acta Dermato-Venereologica*. 88, 34-37 (2008).
2. Montagnese S, Nsemi LM, Cazzagon N *et al.* Sleep-Wake profiles in patients with primary biliary cirrhosis. *Liver International*, 33, 203-209 (2013).
3. Chapman R, Fevery J, Kalloo A *et al.* Diagnosis and management of primary sclerosing cholangitis. *Hepatology*. 51, 660-678 (2010).
4. Boots AW, Wilms LC, Swennen ELR *et al.* *In vitro* and *ex vivo* anti-inflammatory activity of quercetin in healthy volunteers. *Nutrition*. 24, 703-710 (2008).
5. Nickel T, Hanssen H, Susic Z *et al.* Immunoregulatory effects of the flavonol quercetin *in vitro* and *in vivo*. *European Journal of Nutrition*. 50, 163-172 (2011).
6. Moro T, Shimoyama Y, Kushida M *et al.* Glycyrrhizin and its metabolite inhibit Smad3-mediated type I collagen gene transcription and suppress experimental murine liver fibrosis. *Life Sciences*. 83, 531-539 (2008).
7. Kolasa KM, Rickett K. Barriers to providing nutrition counselling cited by physicians. *Nutrition in Clinical Practice*. 25, 502-509 (2010).
8. Glanz K. Review of nutritional attitudes and counselling practices of primary care physicians. *American Journal of Clinical Nutrition*. 65, 2016-2019 (1997).
9. Holeček M. Nutritional modulation of liver regeneration by carbohydrates, lipids, and amino acids. *Nutrition*. 15, 784-788 (1999).
10. Nishiura T, Watanabe H, Ito M *et al.* Ultrasound evaluation of the fibrosis stage in chronic liver disease by the simultaneous use of low and high frequency probes. *British Journal of Radiology*. 78, 189-197 (2005).
11. Xu Y, Wang B, Cao H *et al.* An ultrasound scoring system for the diagnosis of liver fibrosis and cirrhosis. *Chinese Medical Journal*. 112, 1125-1128 (1999).
12. Colli A, Fraquelli M, Andreoletti M *et al.* Severe liver fibrosis or cirrhosis. *Radiology*. 227, 89-94 (2003).
13. Møller S, Henriksen JH. Cardiovascular complications of cirrhosis. *Gut*. 57, 268-278 (2008).
14. Bernardi M, Calandra S, Colantoni A *et al.* relationship with severity and etiology of the disease and possible pathogenetic factors. *Hepatology*. 27, 28-34 (1998).
15. Lédinghen de V, Vergniol J, Foucher J *et al.* Non-invasive diagnosis of liver steatosis using controlled attenuation parameter (CAP) and transient elastography. *Liver International*. 32, 911-918 (2012).