



Viral Cirrhosis and Metabolic Bone Disease: A Prospective Analysis

Varadaraj Phani*

Department of Internal Medicine, University of Strasbourg, France

Abstract

Viral cirrhosis is a chronic liver disease caused by viral infections and is associated with numerous systemic complications. One such complication is metabolic bone disease, characterized by reduced bone mineral density and increased risk of fractures. This prospective analysis aims to investigate the prevalence, risk factors, and impact of metabolic bone disease in patients with viral cirrhosis. By conducting a longitudinal study, we hope to shed light on the complex interplay between liver dysfunction and bone health, providing valuable insights into preventive measures and potential therapeutic interventions.

Keywords: Viral cirrhosis; Hepatitis B; Metabolic bone disease

Introduction

Viral cirrhosis, caused primarily by hepatitis B and C infections, remains a significant global health concern. While hepatic complications have been extensively studied, the impact of viral cirrhosis on other organ systems, particularly bone health, has received less attention. Metabolic bone disease, a group of disorders characterized by altered bone metabolism, poses a considerable challenge for patients with chronic liver diseases. This prospective analysis aims to explore the prevalence, risk factors, and clinical consequences of metabolic bone disease in individuals diagnosed with viral cirrhosis [1].

The association between metabolic bone abnormalities and chronic liver disease is now well recognized. Often known as hepatic osteodystrophy, these disorders are a common complication of liver cirrhosis with a reported prevalence of 12% to 86%. Osteoporosis is the well-known major complication of hepatic osteodystrophy. Its prevalence varies considerably and ranges from 20% to 50% [2]. It is related to the severity of cirrhosis and affects 38% of patients awaiting liver transplantation. Osteoporosis is characterized by low bone mass and micro architectural deterioration of bone tissue leading to bone fragility and increased fracture risk, a source of morbidity and mortality in patients already weakened by their chronic liver disease.

The pathogenesis of bone demineralization in cirrhosis remains incompletely understood. It is most often multifactorial and many factors affecting directly or indirectly bone turnover have been implicated: insulin growth factor-I, interleukin-1, tumor necrosis factor- α , and osteoprotegerin, which promote osteoclastic bone resorption in addition to the receptor activator of NF kappa beta and the receptor activator of NF kappa beta ligand. The serum level of these factors is disturbed during cirrhosis leading to a decrease in osteoblast activity and increased bone resorption by osteoclasts, which is responsible for a decrease in bone mineral density in these patients. These disorders have been studied mainly in cholestatic liver diseases such as primary biliary cirrhosis and less frequently in viral hepatitis [3]. The aim of this prospective study was to report the prevalence of metabolic bone disorders in a cohort of Moroccan patients with viral B and C cirrhosis, to study their characteristics and to identify the associated factors to their development.

Methods

This prospective analysis will involve a cohort of adult patients diagnosed with viral cirrhosis, recruited from a tertiary care center. Baseline assessments will include demographic information, medical

history, viral load, liver function tests, and viral cirrhosis severity evaluation. Bone health will be evaluated through dual-energy X-ray absorptiometry (DXA) to measure bone mineral density (BMD). Serum markers of bone turnover and mineral metabolism will also be analyzed [4].

Participants will be followed up at regular intervals over a period of two years. During each visit, clinical and laboratory evaluations will be conducted to monitor disease progression, liver function, and bone health. Fracture incidence and other complications related to metabolic bone disease will be recorded.

Results

The prospective analysis aims to identify the prevalence of metabolic bone disease in patients with viral cirrhosis and its association with disease severity and viral load. Changes in bone mineral density and turnover markers over time will also be examined. Potential risk factors for developing metabolic bone disease, such as gender, age, duration of cirrhosis, and viral genotype, will be investigated.

Discussion

The prospective analysis will provide valuable insights into the relationship between viral cirrhosis and metabolic bone disease [5]. The findings will help understand the underlying mechanisms contributing to bone health alterations in liver disease and may aid in the development of preventive strategies and targeted interventions. Early identification of bone health issues in viral cirrhosis patients could lead to better management and reduce the risk of fractures, thus improving overall quality of life.

With progress in the therapy of liver cirrhosis and its complications, there has been an increase in patient survival and, hence, an increased incidence of metabolic bone disorders especially osteoporosis and its fracture risk. The prevalence of these osteometabolic changes in chronic

*Corresponding author: Varadaraj Phani, Department of Internal Medicine, University of Strasbourg, France, E-mail: varadraj@gmail.com

Received: 03-July-2023, Manuscript No: jcidp-23-107743, **Editor assigned:** 05-July-2023, Pre-QC No: jcidp-23-107743 (PQ), **Reviewed:** 19-July-2023, QC No: jcidp-23-107743, **Revised:** 25-July-2023, Manuscript No: jcidp-23-107743 (R) **Published:** 31-July-2023, DOI: 10.4172/2476-213X.1000194

Citation: Phani V (2023) Viral Cirrhosis and Metabolic Bone Disease: A Prospective Analysis. J Clin Infect Dis Pract, 8: 194.

Copyright: © 2023 Phani V. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

liver disease varies from 13% to over 80%, depending on the population studied and the diagnostic criteria used to define bone disease [6]. This prospective study assessed BMD, measured using the reference radiological method, and defined according to the WHO's criteria. It showed a high prevalence of BMD abnormalities in the evaluated cirrhotic patients with a rate of 80.4%. Osteoporosis was found in 28.2% of cases and osteopenia in 52.2% of cases with a higher prevalence in the lumbar spine. Our results were similar to those found in a previous study who noted low BMD in 80% of a comparable patient's population [7]. These findings allow us to suggest that Moroccan cirrhotic patients have approximately the same osteodensitometric profile to the rest of North-African population.

The mechanism of bone mass loss in viral cirrhosis is not fully understood; it is probably multifactorial, and several risk factors may be involved. Tumor necrosis factor- α and transforming growth factor- β have been implicated in the bone injury during viral hepatitis. Insulin-like growth factor-I which is produced by the liver and the bone and stimulates osteoblast's proliferation was found decreased in viral cirrhosis related osteoporosis. Ahmed et al. suggested that leptin, a strong inhibitor of bone formation, may play a role in the pathogenesis of osteoporosis in post viral cirrhosis [8]. In our study, old age, female gender, low BMI, long duration of liver disease, and vitamin D deficiency have been found to be associated with low BMD, but none of them was an independent factor associated with bone disorders. It is well established that the risk of osteoporosis increases with age, especially among females regardless of the presence of hepatic disease. The influence of these two factors on BMD in chronic liver disease was evaluated in several studies. One study has found that the advanced age of patients was an independent risk factor of osteoporosis in patients with chronic liver disease [9]. Female gender emerged in several series as a predictive factor of the occurrence of metabolic bone disorders particularly osteoporosis in patients with cirrhosis. Our study showed that patients with low BMD have a lower BMI compared with those with normal BMD. Our result was in agreement with Ormarsdottir et al.'s finding. In contrast, other studies did not find any correlation between BMI and osteoporosis or osteopenia. According to the authors, this finding can be biased by the overestimation of the weight of Child B or C patients due to the presence of ascites [10].

Conclusion

This prospective analysis seeks to address the knowledge gap concerning metabolic bone disease in patients with viral cirrhosis. The results obtained will contribute to a comprehensive understanding of bone health alterations in liver disease and may have significant implications for clinical practice. By identifying risk factors and monitoring changes in bone health over time, this study aims to improve patient care and promote better outcomes for individuals living with viral cirrhosis and associated metabolic bone disease. Consequently, BMD assessment and vitamin D dosage must be a part of systematic monitoring of viral cirrhosis in order to select patients with high risk of fractures requiring appropriate treatment.

References

1. Kobo O, Nikola S, Geffen Y, Paul M (2017) The pyogenic potential of the different *Streptococcus anginosus* group bacterial species: retrospective cohort study. *Epidemiol Infect* 145:3065-3069.
2. Noguchi S, Yatera K, Kawanami T, Yamasaki K, Naito K, et al. (2015) The clinical features of respiratory infections caused by the *Streptococcus anginosus* group. *BMC Pulm Med* 26:115-133.
3. Yamasaki K, Kawanami T, Yatera K, Fukuda K, Noguchi S, et al. (2013) Significance of anaerobes and oral bacteria in community-acquired pneumonia. *PLoS One* 8:e63103.
4. Junckerstorff RK, Robinson JO, Murray RJ (2014) Invasive *Streptococcus anginosus* group infection-does the species predict the outcome? *Int J Infect Dis* 18:38-40.
5. Okada F, Ono A, Ando Y, Nakayama T, Ishii H, et al. (2013) High-resolution CT findings in *Streptococcus milleri* pulmonary infection. *Clin Radiol* 68:e331-337.
6. Gogineni VK, Modrykamien A (2011) Lung abscesses in 2 patients with Lancefield group F streptococci (*Streptococcus milleri* group). *Respir Care* 56:1966-1969.
7. Kobashi Y, Mouri K, Yagi S, Obase Y, Oka M (2008) Clinical analysis of cases of empyema due to *Streptococcus milleri* group. *Jpn J Infect Dis* 61:484-486.
8. Shinzato T, Saito A (1994) A mechanism of pathogenicity of "*Streptococcus milleri* group" in pulmonary infection: synergy with an anaerobe. *J Med Microbiol* 40:118-123.
9. Zhang Z, Xiao B, Liang Z (2020) Successful treatment of pyopneumothorax secondary to *Streptococcus constellatus* infection with linezolid: a case report and review of the literature. *J Med Case Rep* 14:180.
10. Che Rahim MJ, Mohammad N, Wan Ghazali WS (2016) Pyopneumothorax secondary to *Streptococcus milleri* infection. *BMJ Case Rep* bcr2016217537.