



Association of Liver Fibrosis with Impaired Bone Mineralization

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DESCRIPTION

The most prevalent chronic liver ailment in the world, Non-Alcoholic Fatty Liver Disease (NAFLD) is defined by aberrant intrahepatic fat buildup [1]. It can also include varying degrees of inflammation and fibrosis and has insulin resistance as its underlying cause. Due to the close connection between aberrant fat liver content and the presence of metabolic disorder, a recent reclassification and alternative definition for NAFLD have been proposed, introducing the expression "Metabolic (Dysfunction) Associated Fatty Liver Disease" (MAFLD). Hepatosteatosis causes an increase in insulin resistance. It is well known that there is a bidirectional relationship between the presence of fatty liver disease and Abnormal Glucose Metabolism (AGM), which also explains why complications of diabetes are more common and severe when NAFLD is present [2]. NAFLD is still regarded as an independent predictor of cardiovascular and all-cause mortality, independent of conventional risk factors. NAFLD can have a deleterious effect on the bone in addition to its potential effects on metabolic outcomes. In chronic liver diseases, or viral-induced chronic hepatopathies, such as cirrhosis and advanced disease stages, there has been evidence of increased bone fragility and fracture risk. This is because reduced synthesis of certain growth factors derived from the liver, like the Insulin-Like Growth Factor 1 (IGF-1), may have a negative effect on bone metabolism. There is some evidence that both adults and children with NAFLD have lower IGF-1 levels [3].

NAFLD is linked to overweight and obesity, where a high body mass index and fat content prevent a Dual-Energy X-Ray Absorptiometry (DXA) scan, the gold standard method for diagnosing osteoporosis, from accurately estimating bone mineralization. Additionally, both type 1 and type 2 diabetes mellitus are associated with increased bone fragility. In people with diabetes, deteriorated microarchitecture and qualitative bone structure impairment enhance the risk of fracture even in the presence of normal to high bone mineral density. Therefore, although evaluating bone structure quality rather than mineral density in dysmetabolic patients may reveal greater fragility and fracture risk, information in this regard is limited and not

accessible in NAFLD groups. IGF-1, a hepato-derived growth hormone that has anabolic effects on bone by blocking osteoblast apoptosis and promoting osteoclast generation, may have a role to play in this situation. It has been demonstrated that the decline in growth hormone receptors linked to hepatocyte malfunction in patients with severe liver illness contributes to the onset of osteoporosis. We investigated serum IGF-1 alterations in relation to estimated liver fibrosis as a potential relationship between NAFLD and bone fragility after discovering decreased IGF-1 levels in study participants with osteopenia and osteoporosis. We discovered a linear inverse correlation between IGF-1 and FIB-4. Following consideration of age, sex, BMI, and the presence of MS components in multivariable regression models, we finally showed the existence of an independent connection between FIB-4 and decreased IGF-1. In fact, IGF-1 reduction may be the link between NAFLD and altered bone metabolism, especially when there is more extensive liver impairment [4].

As indicated for dysmetabolic disorders such as T2DM, the interaction between poor bone metabolism and NAFLD may also be bidirectional and may be mediated by circulating substances other than insulin that may have an impact on both bone and liver metabolism. Osteocalcin might be one of them. Osteocalcin is a non-collagen protein that is taken from bone and is made by osteoblasts; its carboxylated form only operates in the bone, promoting mineralization processes; the uncarboxylated form is detectable in blood circulation and exhibits many extra-skeletal metabolic functions. In experimental animals, osteocalcin prevents the development of NASH and hepatosteatosis through binding to the GPRC6A hepatic receptor. Our work shown that in obese people with NAFLD, there was a linear negative connection between osteocalcin levels and the FIB-4. Longitudinal studies are recommended to explore the possible effects of altered bone metabolism on liver pathophysiology in the clinical environment [5].

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Received: 06-Sep-2022, Manuscript No. JLR-22-18301; **Editor assigned:** 09-Sep-2022, Pre QC No. JLR-22-18301 (PQ); **Reviewed:** 23-Sep-2022, QC No JLR-22-18301; **Revised:** 30-Sep-2022, Manuscript No. JLR-22-18301 (R); **Published:** 10-Oct-2022, DOI: 10.35248/2167-0889.22.11.145.

Citation: Chen P (2022) Association of Liver Fibrosis with Impaired Bone Mineralization. J Liver. 11:145.

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