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Bone aspect in long-standing liver and kidney diseases, part of physiology and clinical aspects

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ABSTRACT: Interest in focused treatments for different types of osteoporosis has increased as a result of growing awareness of the liver, kidney, and bone interaction. Injury to the liver and kidneys caused by various disorders can disrupt bone metabolism, suggesting a new regulatory relationship among the liver, kidneys, and bones. But little is known about how the liver-bone center contributes to primary and secondary osteoporosis. Thus, investigating the precise regulatory motion of the liver-bone center may provide new curative strategies for the treatment of liver and bone disorders. Chronic liver disease is a prevalent health issue worldwide, especially in East Asia. Osteoporosis (OP), a prevalent metabolic bone disorder, has garnered heightened attention in ageing populations. In recent years, a growing association between osteoporosis and chronic kidney disease (CKD), belonging to the larger category of CKD-mineral and bone disease (CKD-MBD), has been noted. Changes in parathyroid hormone (PTH), vitamin D, calcium, and phosphorus metabolism are characteristics of CKD-MBD, which greatly increases the risk of fracture. This review aims to examine the correlation between chronic renal and liver illness and their effects on bone throughout the patient's lifespan.

Keywords: Osteoporosis, chronic kidney disease, chronic liver disease, Hepatic osteodystrophy

1. INTRODUCTION

The liver is a vital metabolic organ, crucial for energy metabolism in the body. It acts as a metabolic hub, connecting various tissues, including adipose tissue and skeletal muscle [1]. Both the liver and bones are endocrine systems. The liver secretes hepatokines, and bone metabolites are derived from the bone. These metabolites regulate the activity of various organs and tissues [2]. Liver diseases, particularly liver failure, lead to changes in bone formation and breakdown processes, highlighting the complex relationship between these two physiological systems. Patients with liver failure are at increased risk of developing hepatic osteodystrophy (HOD), a disorder that causes bone loss and fractures. More than 50% of patients with chronic liver disease (CLD) develop HOD [3]. Chronic inflammation, imbalances in gut microbiota, and vitamin D deficiency are among the causes of non-alcoholic fatty liver disease (NAFLD), which is characterized by decreased bone mineral density, specifically reduced calcium and phosphorus deposition in bone tissue [4]. Among those with chronic liver disease, hepatic osteopathy is a metabolic bone condition under investigation. It includes both osteomalacia and osteoporosis [5].

As chronic kidney disease (CKD) worsens, irregularity inside bone structure and mineral metabolism become more common, resulting in a clinical condition called CKD-mineral and bone disorder (CKD-MBD). There are a variety of laboratory tests associated with this disease, such as measuring vitamin D levels in the body and thyroid hormones. (PTH) with fibroblast growth factor 23 (FGF23) levels, and mineral metabolism (calcium and phosphorus). Those anomalies are linked to several skeletal issues, including renal osteodystrophy (ROD))[6].

There are many symptoms of osteoporosis, such as decreased bone mass, altered bone microstructure, and deterioration of bone tissue, which weakens bones and increases the risk of fractures. [7].

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Primary and secondary osteoporosis are the traditional classifications for osteoporosis. Idiopathic, senile, and postmenopausal osteoporosis are the three types of primary osteoporosis [8]. The body uses bone, a dynamic, mineralised structure, for many essential purposes, such as calcium storage, bone marrow housing, support protection and movement facilitation[9].

Bone requires constant, dynamic remodeling during human growth and development in order to accommodate changes in the body [10]. Liver cirrhosis is the loss of liver function caused by factors such as viral infections, alcohol, and toxins[11]. This condition disrupts vitamin D metabolism, affecting calcium absorption and normal bone formation. It also impacts estrogen levels, a hormone crucial for postmenopausal women's health and essential for bone health[12]. Therefore, it has been found that patients with cirrhosis have increased bone resorption compared to bone formation, particularly in trabecular (spongy) bone, where decreased bone density (osteoporosis) occurs. This is a very common problem in patients with cirrhosis and can occur in both the early and late stages of the disease. Osteoporosis can lead to an increased incidence of fractures, hospital admissions, prolonged recovery time, higher morbidity and mortality rates, and increased healthcare costs[13]. Cirrhosis has been previously examined and acknowledged as a condition linked to osteoporosis; however, has not yet been realized as a distinct danger point for fragility fractures along with not included in breaking danger evaluation and calculators like FRAX, Garvan, and QFracture It is a measure (calculation tool) for estimating a person's risk of future bone fractures. (chronic liver illness, not cirrhosis, is a component of the QFracture score) [14].

2. PATHOPHYSIOLOGY OF OSTEOPOROSIS

Osteoporosis can significantly affect public health, resulting in temporary or permanent loss of mobility[15]. Numerous studies have demonstrated the pivotal role of bone as an organ that always exchanges and regulates many different tissues. As a consequence, it is reasonable that bone, in addition to its conventional roles in movement, controls mineral balance and protects internal organs [16]. Bone has many vital functions in the body. It is a dynamic tissue that makes preparations support, store calcium, form bone marrow, and ensure smooth movement [9] .Bones require constant dynamic rebuilding throughout human development and growth to cope with body changes [17]. There are lining cells that cover the surface of bones. When osteocytes are damaged, osteoclasts trigger processes that begin bone resorption. Osteoblasts are derived from mesenchymal stem cells (not hematopoietic stem cells). They are responsible for bone formation by producing the organic bone matrix (osteoid), which is later mineralized [10], as shown in figure1 .

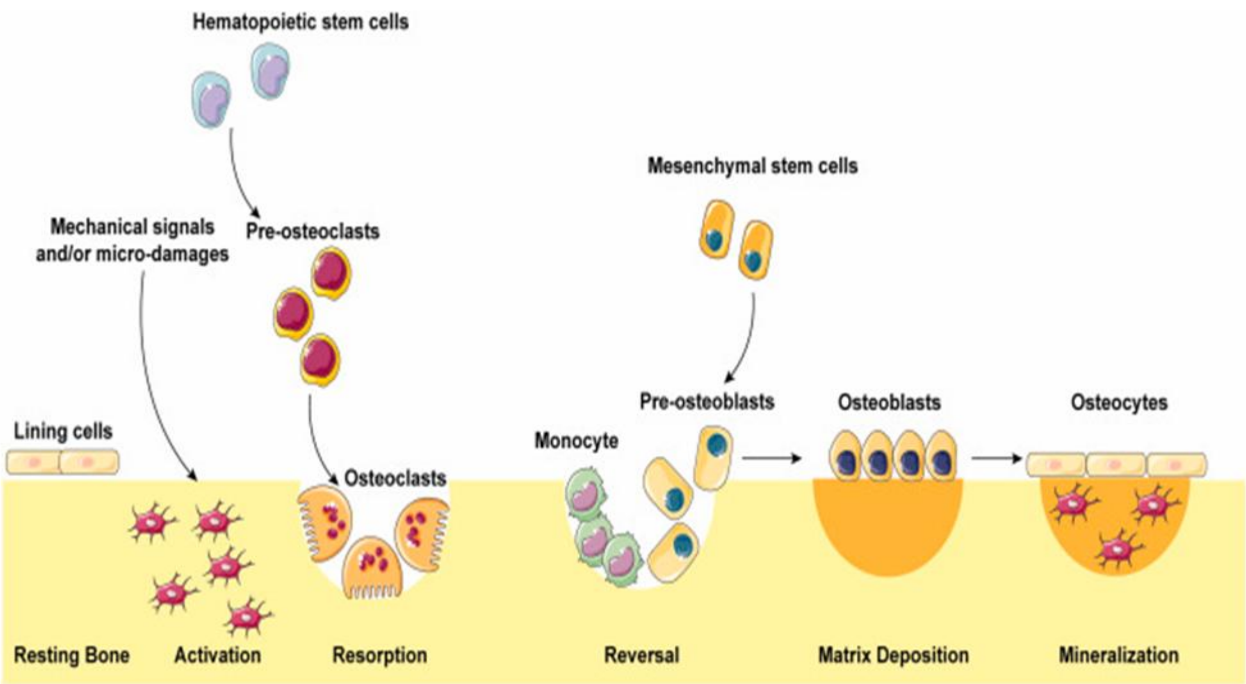

FIGURE 1. - Remodeling bones when they are damaged by maintaining bone turnover [17]

It results from a shift in differentiation potential that favours abiogenesis over ontogenesis and abnormalities in the bone remodeling process that favor mesenchymal stem cell (MSC) senescence. Bone loses its structural integrity and becomes more prone to fractures in this pathological state [18].

The primary and secondary osteoporosis are the two principal classification of osteoporosis. There are bone problems without a medical cause; this is known as primary osteoporosis. These consist of involution and idiopathic osteoporosis. The etiopathogenesis of idiopathic osteoporosis is yet unknown, and it primarily have affect on children and young people [19].

Most people over 75 are affected by type II osteoporosis which affects some women after menopause, as well as men, due to endocrine dysfunction or genetic factors. involution osteoporosis, sometimes known as "senile osteoporosis." The pattern of bone loss in this disorder is mostly trabecular and cortical [20]. Less than 5% of all occurrences of osteoporosis are secondary, which is guided by an underlying disease or drug use [19].

3. THE BONE-LIVER CONNECTION

The liver metabolizes and stores three primary nutrients: carbs, lipids, and proteins. The liver also facilitates the metabolism and elimination of alcohol, narcotics, and toxins, as well as the synthesis and release of bile[21]. There is no direct physical contact between the liver and bone because of their physical separation. The liver secretes proteins, enzymes, and cytokines that play a significant role in regulating bone metabolism[22].

Nonetheless, diverse hepatic lesions can disrupt the liver's secretory and biosynthetic processes, subsequently resulting in aberrant bone metabolism. Simultaneously, irregularities in osteokine secretion during pathological conditions are crucial for hepatic control[17].

Compared to patients with other causes of liver disease and cirrhosis, those with cholesteric liver disease who do not have severe fibrosis are also more likely to develop osteoporosis [23].

The intricate pathophysiology of bone disease with chronic liver illness that linked to dietary deficits, inflammatory cytokines, and abnormalities in bone remodeling. [24]. Other risk factors for osteoporosis in people with cirrhosis include alcohol usage, tobacco use, and a sedentary lifestyle [25].

4. OSTEOPOROSIS COMBINED TO LIVER DISEASE

A hallmark to chronic liver disease (CLD) be that a steady decline in liver function. Fibrosis and cirrhosis are brought on by an ongoing sequence of inflammation, liver parenchymal damage, and regeneration [26]. A primary contributor to chronic liver disease is NAFLD (nonalcoholic fatty liver disease, who presently impacts one at four adults globally. Chronic liver illness can lead to numerous serious consequences, as well as bone problems like bone fractures and osteopenia who often keep undetected till advanced stages [27].

Increased prevalence of osteoporosis (10–40%) compared to the general population without liver illness[28].

Hepatic osteodystrophy is the appellation used to describe the collection from modification on bone mineral metabolism identified of CLD cases [29].Another common complication of CLD is osteoporosis, which is more prevalent in sick persons with cirrhosis and cholesteric liver disease. Numerous investigations have identified a significant decrease in bone production, indicating that osteoporosis in individuals with cirrhosis is a complex condition wherein various processes collaboratively diminish bone mass, ultimately leading to skeletal fragility[24].

According to a meta-analysis, osteoporosis is more common in cirrhosis patients than in controls (oddsratio [OR], 2.52)[12]. Patients with CLD are more likely than those without to suffer osteoporotic fractures (pooled OR, 2.13), according to another meta-analysis.[30].

Hyperbilirubinemia, a variation in the nuclear factor receptor activator (kappa-B/osteoprotegerin) ratio, low insulin-like growth factor 1 levels, vitamin D deficiency, and raised pro-inflammatory cytokines like IL-6 (interleukin), and TNF-α (tumor necrosis factor alpha), with an increase in sclerosis levels, are some of the causes of such conditions, as shown in Figure 2 [31].

Growth hormone (GH) causes hepatocytes to release IGF-1 (insulin-like growth factor) suppresses apoptosis and increases osteoblast formation through the stability of the Wnt/β-catenin pathway. This found an effect on the anabolic bone formation [32].

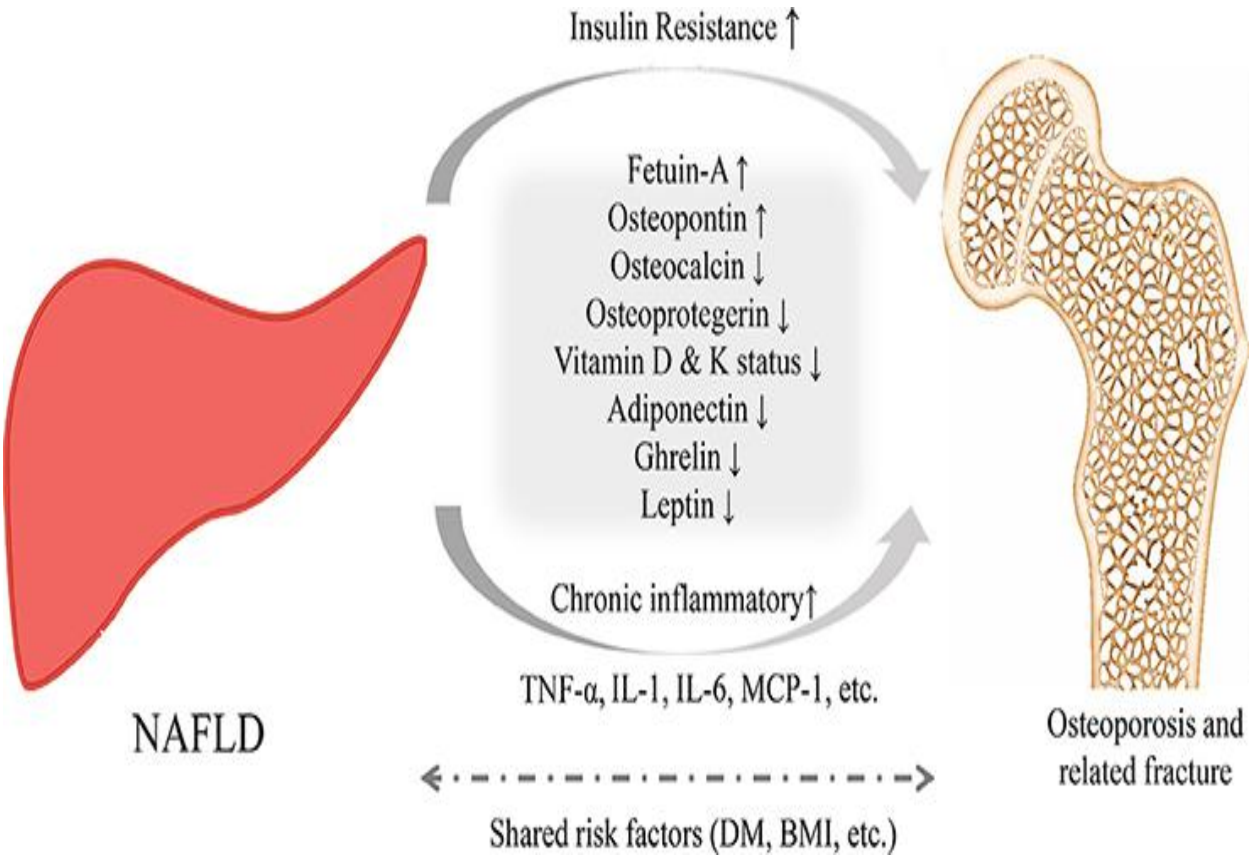

FIGURE 2. - The effect of low insulin-like growth factor 1 levels, vitamin D deficiency, and elevated pro-inflammatory cytokines in patients with non-alcoholic fatty liver disease on bone [33]

Hepatocellular dysfunction and decreased GH receptors in end-stage liver illness result in low serum IGF-1 levels, which in turn cause osteoporosis [34]. By promoting the synthesis of collagen with the generation of osteoblasts and regeneration, insulin, a crucial hormone linked to NAFLD, also influences bone remodeling [28].

A study with mean age of 70 years sought to examine the clinical features and the connection between osteoporosis and crispness in 291 individuals with chronic liver disease (CLD), 137 of whom were men and 154 of whom were women. The Japanese Society of Hepatology's criteria were used to diagnose osteoporosis. They used an absorption device (Dual-energy x-ray) to evaluate the density of bone minerals. Of the 291 patients, 81 (27.8%) had frailty and 49 (16.8%) had bone loss. Patients with osteoporosis were more likely than those without to experience frailty and vertebral fractures (79.6% vs. 17.4% and 59.2% vs. 20.2%, respectively; $P < 0.001$ for both) [35].

5. CHRONIC RENAL DISEASE ACCOMPANIED BY OSTEOPOROSIS

More than 800 million individuals globally suffer from CKD (chronic kidney disease, a degenerative illness which affects more than 10% of the population [36]. The clinical illness famous as CKD-MBD (CKD-mineral and bone disease) is brought on by alterations in mineral metabolism and disturbances in bone morphology that become more common as CKD worsens. [6]. The most common issue among patients accompanied by CKD (chronic kidney disease) is MBD (mineral and bone disorder), which has a major effect on their general health and well-being. The consequences of MBD linked to CKD are numerous and include poor clinical outcomes such fractures, cardiovascular events, and, in extreme situations, death.[37].

Changes in PTH (parathyroid hormone) with FGF23 (fibroblast growth factor 23) levels, vitamin D3 pathway, and mineral metabolism (calcium and phosphorus) are among the laboratory abnormalities linked to chronic kidney disease (CKD) [38]. As shown in figure3.

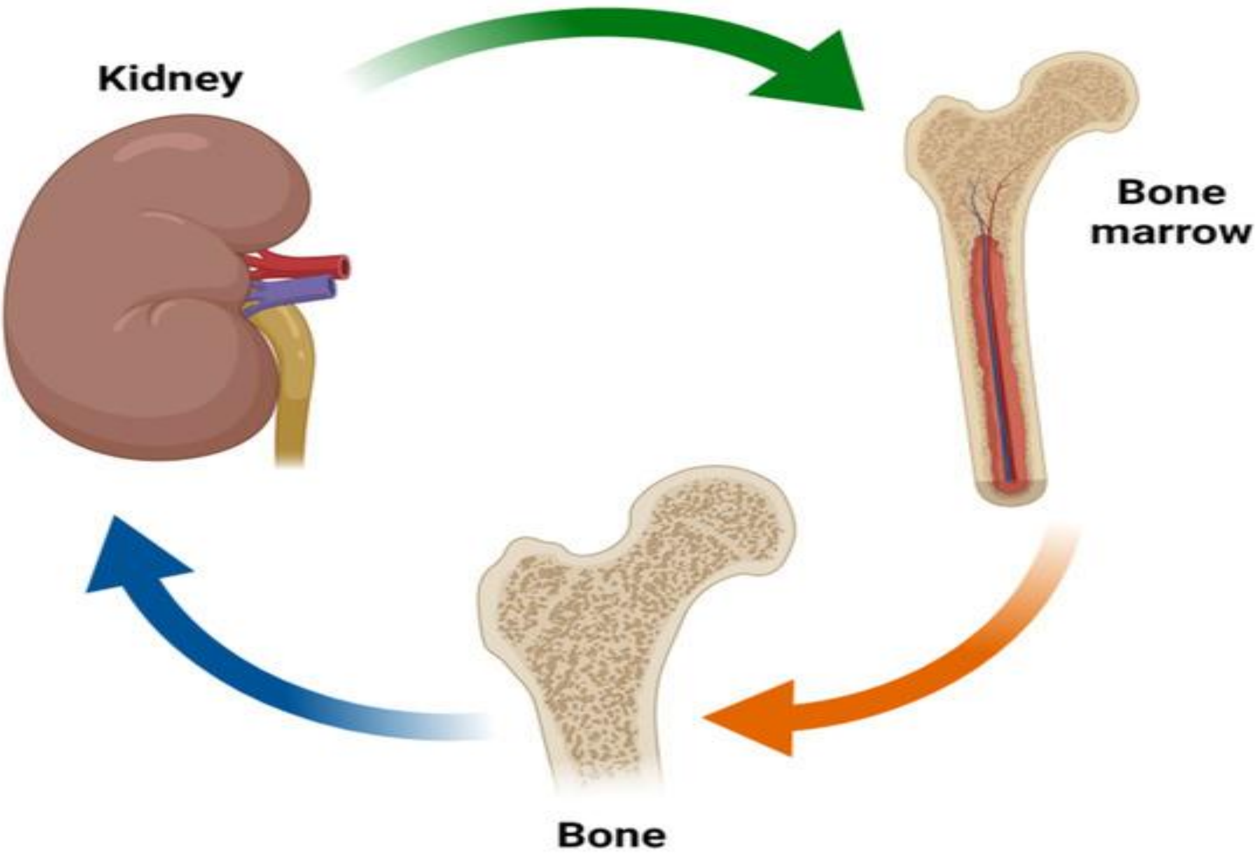

FIGURE 3. - Complex hormonal systems, including as FGF-23, vitamin D3, and some components that regulate the iron pathway are included in the kidney-bone marrow-bone center.[39]

These anomalies are linked to extraskeletal calcifications, renal osteodystrophy (ROD), osteoporosis, and other skeletal disorders that are all related to a higher risk of cardiovascular morbidity and death[40].

One typical mineral anomaly in patients with CKD is a low calcium content. Because of increased phosphate binding to calcium, decreasing 1,25D levels, and skeletal resistance to PTH effects, total calcium concentration decreases as chronic kidney disease (CKD) worsens. Increased PTH secretion and bone remodelling are caused by hypocalcaemia [41].

There is a special membrane receptor known as CaSR (Calcium-sensing receptor) found on the face of chief cells in the parathyroid gland. It can tell when calcium levels drop [6].Recent research revealed that the parathyroid gland's expression of Klotho was downregulated. Klotho serves as a FGF23 cofactor.

Thus, resistance to the FGF23-lowering effect on PTH is probably caused by negative regulation of klotho in the parathyroid gland, which leads to further advancement of SHPT [41].

Increased dietary calcitriol, PTH, phosphate load, and calcium all induce the production of the hormone FGF-23 by osteoblasts and osteocytes in bone. The bone, kidney, heart, parathyroid, and maybe other organs are all impacted by FGF-23[42].

Fractures and mortality are associated with uncontrolled SHPT, a significant manifestation of CKD-BMD. Additionally, FGF-23 hold block WNT pathways, which results in bone degradation and a corresponding increase in fracture susceptibility [6]. An important mediator that triggers phosphate retention in response to deteriorating renal function is FGF-23. FGF-23, independent of PTH, increases renal phosphate excretion and inhibits the synthesis of 1-alpha hydroxylase, worsening calcitriol insufficiency [43].

D vitamin balance is affected by the loss of vitamin D-linked to protein in the urine, as well as by some immunosuppressive medications like corticosteroids, and the actions of immune cells, leading to bone disorders with glomerular diseases in patients, particularly with nephrotic syndrome.

They demonstrated that patients suffer from nephrotic syndrome with high osteomalacia, which is probably caused by lower vitamin D levels and may or may not be linked to fast bone turnover [39].

There is no clear consensus despite of many studies on the relationship between osteoarthritis (OP) and FGF-23 (fibroblast growth factor-23) and FGFR1 (FGF receptor1). Serum FGF23 levels and the bone mineral mass at the heel and neck of femoral were shown to be strongly correlated in a 2024 investigation; the posterior odds ratios were0.919 (95% CI:0.860–0.938, P=0.014) and 0.751 (95%CI: 0.587–0.962; P=0.023), respectively.

The femoral necks of OP patients had considerably higher levels of FGF23 expression than those of the control group (p<0.0001).

An elevated risk of OP is causally linked to elevated FGF23 levels. Similarly, human bone marrow cells' ability to differentiate osteogenically is substantially inhibited by overexpression of FGF23, which may exacerbate the pathological process of OP[44].

6. CONCLUSION

In addition to underlining highlighting the challenges of assessing and treating bone health in liver disease and chronic renal kidney disease, this review attempts aims to provide a thorough comprehensive overview of the pathophysiology, challenges with diagnostic techniques challenges, and management options of for osteoporosis. It should be noted that the research from which this information was drawn was based on human samples. It was also found that measuring growth hormone, insulin-like growth factor, and severe vitamin D deficiency in the body raised pro-inflammatory cytokines, indicating liver dysfunction that led to osteoporosis.

Furthermore, abnormalities in parathyroid hormones and fibroblast growth factor 23 levels suggest a link between kidney disease and osteoporosis.

The next step for this manuscript is to examine the impact of bone density in patients with kidney and liver disease through comprehensive clinical studies that evaluate hormones and biomarkers and their effects on bone, with the aim of improving and accelerating the early detection of osteoporosis in these patients.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest

REFERENCES

[1] W.-J. Zhang et al., “Liver function linked to bone health: A bibliometric of the liver–bone axis,” World J. Hepatol., vol. 17, no. 2, p. 103016, 2025.

[2] J. Zhao, H. Lei, T. Wang, and X. Xiong, “Liver–bone crosstalk in non-alcoholic fatty liver disease: Clinical implications and underlying pathophysiology,” Front. Endocrinol., vol. 14, p. 1161402, 2023.

[3] Z. Li et al., “The roles of hepatokine and osteokine in liver–bone crosstalk: Advance in basic and clinical aspects,” Front. Endocrinol., vol. 14, p. 1149233, 2023.

[4] O. M. Drapkina, A. Y. Elkina, A. F. Sheptulina, and A. R. Kiselev, “Non-alcoholic fatty liver disease and bone tissue metabolism: Current findings and future perspectives,” Int. J. Mol. Sci., vol. 24, no. 9, p. 8445, 2023.

[5] M. Turkeli et al., “Effects of cirrhosis on bone mineral density and bone metabolism,” Eurasian J. Med., vol. 40, no. 1, p. 18, 2008.

[6] O. T. Iyidir, “Navigating bone health in chronic kidney disease: A comprehensive review of CKD-MBD and osteoporosis,” Endocrinol. Res. Pract., vol. 29, no. 1, 2025.

[7] T. Sözen, L. Özışık, and N. Ç. Başaran, “An overview and management of osteoporosis,” Eur. J. Rheumatol., vol. 4, no. 1, p. 46, 2016.

[8] M. M. Sobh et al., “Secondary osteoporosis and metabolic bone diseases,” J. Clin. Med., vol. 11, no. 9, p. 2382, 2022.

[9] P. Deng et al., “Loss of KDM4B impairs osteogenic differentiation of OMSCs and promotes oral bone aging,” Int. J. Oral Sci., vol. 14, no. 1, p. 24, 2022.

[10] R. Florencio-Silva, G. R. S. Sasso, E. Sasso-Cerri, M. J. Simões, and P. S. Cerri, “Biology of bone tissue: Structure, function, and factors that influence bone cells,” BioMed Res. Int., vol. 2015, p. 421746, 2015.

[11] S. G. Sepanlou et al., “The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990–2017,” Lancet Gastroenterol. Hepatol., vol. 5, no. 3, pp. 245–266, 2020.

[12] R. Lupoli et al., “The risk of osteoporosis in patients with liver cirrhosis: A meta-analysis,” Clin. Endocrinol., vol. 84, no. 1, pp. 30–38, 2016.

[13] A. S. Nazrun, M. N. Tzar, S. A. Mokhtar, and I. N. Mohamed, “Outcomes of osteoporotic fracture patients after hospital discharge: A systematic review,” Ther. Clin. Risk Manag., pp. 937–948, 2014.

[14] J. Kang, H. Gopakumar, S. R. Puli, and S. Puli, “Prevalence of osteoporosis in cirrhosis: A systematic review and meta-analysis,” Cureus, vol. 15, no. 1, 2023.

[15] M. S. LeBoff et al., “The clinician’s guide to prevention and treatment of osteoporosis,” Osteoporos. Int., vol. 33, no. 10, pp. 2049–2102, 2022.

[16] U. Föger-Samwald, P. Dovjak, U. Azizi-Semrad, K. Kerschan-Schindl, and P. Pietschmann, “Osteoporosis: Pathophysiology and therapeutic options,” EXCLI J., vol. 19, p. 1017, 2020.

[17] H. Gao et al., “Emerging role of liver–bone axis in osteoporosis,” J. Orthop. Transl., vol. 48, pp. 217–231, 2024.

[18] T.-L. Yang et al., “A roadmap for understanding molecular and genetic determinants of osteoporosis,” Nat. Rev. Endocrinol., vol. 16, no. 2, pp. 91–103, 2020.

[19] A. Aibar-Almazán et al., “Current status of the diagnosis and management of osteoporosis,” Int. J. Mol. Sci., vol. 23, no. 16, p. 9465, 2022.

[20] T. Sozen, L. Ozisik, and N. C. Basaran, “An overview and management of osteoporosis,” Eur. J. Rheumatol., vol. 4, no. 1, pp. 46–57, 2017.

[21] M. Woo, J. S. Noh, M. J. Kim, Y. O. Song, and H. Lee, “Magma seawater inhibits hepatic lipid accumulation through suppression of lipogenic enzymes regulated by SREBPs,” Mar. Drugs, vol. 17, no. 6, p. 317, 2019.

[22] W. Liang et al., “An integrated multi-omics analysis reveals osteokines involved in global regulation,” Cell Metab., vol. 36, no. 5, pp. 1144–1163.e7, 2024.

[23] L. A. A. Santos and F. G. Romeiro, “Diagnosis and management of cirrhosis-related osteoporosis,” BioMed Res. Int., vol. 2016, p. 1423462, 2016.

[24] H. M. Jeong and D. J. Kim, “Bone diseases in patients with chronic liver disease,” Int. J. Mol. Sci., vol. 20, no. 17, p. 4270, 2019.

[25] M. Thomson et al., “Low screening rates and high prevalence of osteoporosis in cirrhosis,” Aliment. Pharmacol. Ther., vol. 59, no. 4, pp. 535–546, 2024.

[26] S. A. Alnemari et al., “The relationship between chronic liver disease and osteoporosis: A retrospective single-center experience,” 2024.

[27] H. Gokcan et al., “Prevalence and characteristics of bone disease in cirrhotic patients,” Hepatol. Forum, vol. 1, no. 2, p. 48, 2020.

[28] Y. J. Yang and D. J. Kim, “Molecular mechanisms contributing to musculoskeletal disorders in chronic liver disease,” Int. J. Mol. Sci., vol. 22, no. 5, p. 2604, 2021.

[29] R. Ranjan et al., “Common musculoskeletal disorders in chronic liver disease patients,” Joint Dis. Relat. Surg., vol. 32, no. 3, p. 818, 2021.

[30] D. F. Hidalgo, B. Boonpheng, S. Sikandar, L. Nasr, and J. Hidalgo, “Chronic liver disease and the risk of osteoporotic fractures: A meta-analysis,” Cureus, vol. 12, no. 9, 2020.

[31] C. Saeki, M. Saito, and A. Tsubota, “Association of chronic liver disease with bone diseases and muscle weakness,” J. Bone Miner. Metab., vol. 42, no. 4, pp. 399–412, 2024.

[32] Y. Kobayashi, S. Uehara, N. Udagawa, and N. Takahashi, “Regulation of bone metabolism by Wnt signals,” J. Biochem., vol. 159, no. 4, pp. 387–392, 2016.

[33] Y. Wang et al., “Association of nonalcoholic fatty liver disease with osteoporotic fractures,” Front. Endocrinol., vol. 9, p. 408, 2018.

[34] R. G. de la Garza, L. A. Morales-Garza, I. Martin-Estal, and I. Castilla-Cortazar, “Insulin-like growth factor-1 deficiency and cirrhosis establishment,” J. Clin. Med. Res., vol. 9, no. 4, p. 233, 2017.

[35] C. Saeki et al., “Relationship between osteosarcopenia and frailty in patients with chronic liver disease,” J. Clin. Med., vol. 9, no. 8, p. 2381, 2020.

[36] A. K. Bello et al., “Global disparities in kidney disease burden and care,” Lancet Glob. Health, vol. 12, no. 3, pp. e382–e395, 2024.

[37] P. Simic, “Bone and bone-derived factors in kidney disease,” Front. Physiol., vol. 15, p. 1356069, 2024.

[38] M.-H. Lafage-Proust, “Bone and chronic kidney disease,” in Semin. Musculoskelet. Radiol., vol. 27, no. 4. New York, NY, USA: Thieme, 2023, pp. 463–470.

[39] N. Carullo et al., “Anemia and mineral bone disorder in kidney disease patients,” Int. J. Mol. Sci., vol. 25, no. 23, p. 12838, 2024.

[40] S. Moe et al., “Definition, evaluation, and classification of renal osteodystrophy,” Kidney Int., vol. 69, no. 11, pp. 1945–1953, 2006.

[41] H. Komaba and M. Fukagawa, “FGF23–parathyroid interaction in chronic kidney disease,” Kidney Int., vol. 77, no. 4, pp. 292–298, 2010.

[42] S. K. Murali et al., “FGF23 regulates bone mineralization independent of $1,25(OH)_2D_3$ and klotho,” J. Bone Miner. Res., vol. 31, no. 1, pp. 129–142, 2016.

[43] E. Bellorin-Font, E. Rojas, and K. J. Martin, “Bone disease in chronic kidney disease and kidney transplant,” Nutrients, vol. 15, no. 1, p. 167, 2022.
[44] X. Zhang and J. Xu, “FGF23-mediated regulation of osteoporosis,” J. Cell. Mol. Med., vol. 28, no. 14, p. e18551, 2024.