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REVIEW ARTICLE

Role of Calcium, Vitamin D and Bisphosphonates in the Treatment of Osteoporosis in patients with Cirrhosis

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ABSTRACT

Osteoporosis is a significant and prevalent consequence of cirrhosis due to its detrimental influence on quality of life through persistent pain and immobility. Additionally, its significant morbidity necessitates early detection and treatment. BD is most frequently encountered in the elderly, smokers, postmenopausal women, alcoholics, malnourished individuals, and those with cholestatic liver disease. The prevalence of osteoporosis has been observed to range between 12--55% in patients with cirrhosis. Patients with primary biliary cirrhosis (PBC) have a higher prevalence due to persistent cholestasis and female gender. Prior to orthotopic liver transplantation, about 60% of cases with PBC and primary sclerosing cholangitis (PSC) have osteoporosis. It is critical to diagnose and treat osteoporosis prior to liver transplantation because of deterioration in early months i.e. 6—12 after transplantation. This resulted in much fractures due to trauma i.e. 25—35%. This osteoporosis may be due to toxic effect of immune compromised therapy after transplantation.
Keywords: Role of calcium, cirrhosis, vitamin D

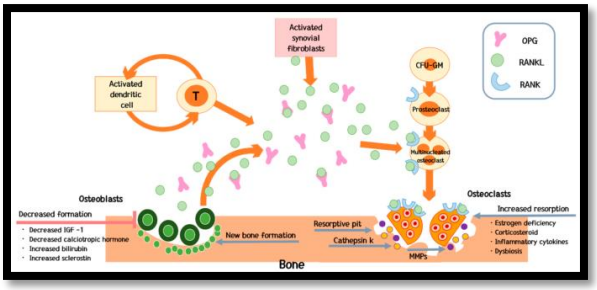
INTRODUCTION

Osteoporosis is clinically critical on the grounds that it is regularly determined in patients to have cirrhosis, bringing about spinal cracks that habitually go undetected clinically yet bring about extreme patient bleakness. Osteoporosis identification in these patients requires a high record of clinical doubt, as around 33% of spinal fractures are asymptomatic thus will be recognized just radiographically. In correlation, femoral neck cracks are remarkable in patients with cirrhosis since they happen around 10 years after the spinal fractures, beyond the future of most of patients with cirrhosis[1].

Osteoporosis is characterized as BMD under 2.5 SD beneath the ordinary pinnacle bone mass (T score of 2.5) as dictated by dual energy x-beam absorptiometry. While the danger of fracture increments fundamentally when BMD diminishes, other clinical danger factors for crack have been found that are disconnected to BMD. The most incredulous of these is a existing crack. After a spinal break, the danger of a resulting vertebral crack increments ten times, while the danger of an ensuing hip break increments 2.3 overlay[2].

Excessive alcohol use is a mediator for osteoporosis on its own and is connected with a twice increment in hip breakage. Reduced blood testosterone levels in active drinkers and individuals with liver disease almost certainly add to osteoporosis as well[3]. Vitamin D deficiency has also been noted in individuals with alcohol consumption with poor BMD[4].

Pathology of Osteoporosis: The underlying biological causes of osteoporosis in cirrhosis are multifaceted and scantily unspoken. Vitamin D receptor, collagen 1 alpha 1, low-density lipoprotein receptor binding protein 5, and oestrogen receptor are all candidates for bone mass-related genes, although polymorphisms in these genes have not yet been identified associated with a higher risk of fracture in patients who do not have disease of the liver.1 Polymorphisms in the vitamin D receptor and collagen 1 alpha 1 have been implicated in PBC. Genes do not appear to be associated with an increased incidence of osteoporosis[3].



Prevalence of Osteoporosis in Cirrhosis: Cirrhosis almost doubles the opportunity of cracks. Various examinations led in the course of the most recent twenty years have uncovered a predominance of osteoporosis of somewhere in the range of 12% and 55% in patients with cirrhosis. The disparity between contemplates is doubtlessly because of contrasts in age, etiology of liver illness, dietary status, hypogonadism, and seriousness of liver infection[3]. The danger of osteoporosis was discovered to be connected with the seriousness of cirrhosis in one examination of 58 individuals with viral cirrhosis. The mediating factors for osteoporosis in an investigation of 243 patients with blended end-stage liver sickness who required transplantation were a lower weight record in ladies and expanding age. Cholestasis was not related with an expanded danger of bone mass thickness[5].

Assessment and Indications for Bone Mineral Density: There has been huge conversation in regards to when to

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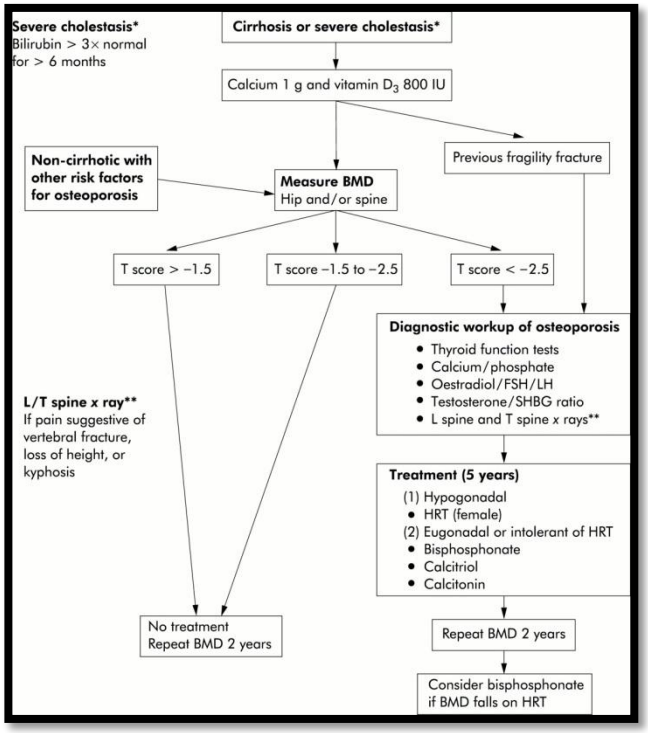
assess bone thickness in patients with persistent liver ailment, and this highlights the troubles of determining a person's danger of osteoporosis and fracture[6]. The easy pickings is to assess BMD in all patients with persistent liver infection. There is inescapable understanding that BMD screening ought to be performed on each and every individual who has had a delicacy crack or is taking long haul corticosteroids, and that treatment ought to be started if osteoporosis is analyzed. Also, BMD ought to be assessed before to liver transplantation and, probably, in all patients with cirrhosis[7].

Diagnosis and management:



Persistent liver sickness patients, especially those with set up cirrhosis, ought to be assessed for osteoporosis, given the disease's disturbing pervasiveness[8]. On the off chance that the underlying assessment is typical, resulting bone density ought to be done every year. In people at high danger of quick bone loss, for example, those with cholestatic liver sickness or who have various danger factors for osteoporosis, reconnaissance for bone density ought to be investigated at more limited stretches, even, every year. Undoubtedly, osteoporosis screening ought to be remembered for the typical preoperative assessment for liver transplantation[9]. Alongside bone densitometry, an intensive assessment of hazard factors and conditions related with bone density ought to be directed.[8]

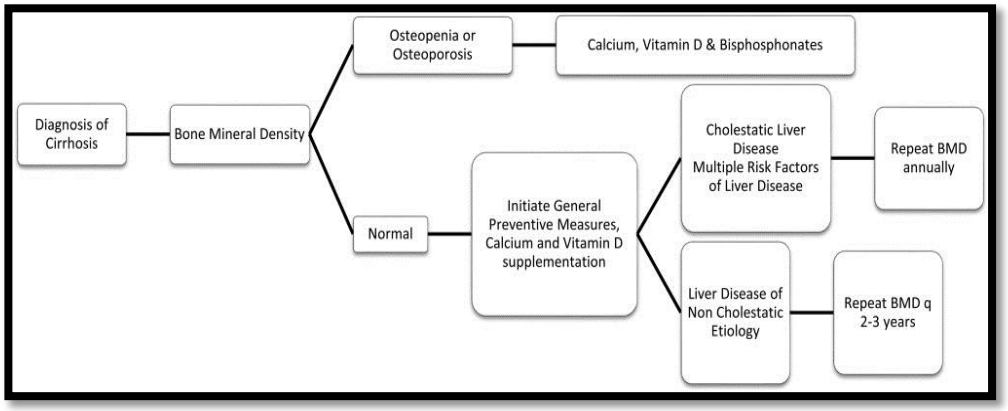
Risk factors in FRAX tool are:

- Previously fractured fragility
- Glucocorticoid orally for 12 weeks
- (19 kg/m2) BMI
- Consumption of alcohol i.e. 3 units per day
- Hip fracture in the maternal lineage

Treatment: The key advice for bone disease prevention is to abstain from alcohol and tobacco and to eat a well-balanced diet. In addition, these interventions should be considered:

- Home safety evaluation
- Restriction of CNS depressants
- Dose modification of antihypertensive drugs
- Calcium and vitamin D supplementation
- To lower corticosteroids and immunosuppressive drugs to avoid future BMD reduction[9].

Vit. D and Calcium: Calcium supplementation is still used to treat osteoporosis. Calcium consumption should be between 1.0 and 1.5 gm per day, depending on age and other factors. Calcium from diet should be preferred, as this will facilitate patient compliance. Furthermore, evidence on the cardiovascular risk associated with calcium supplementation is currently lacking[10]. Nonetheless, because this risk was not examined in specific populations, it should not disqualify these supplements from use. Calcium carbonate is the most frequently used supplement by patients, however it must be taken with food to maximize absorption. Calcium citrate is preferable for patients with achlorhydria or other gastrointestinal disorders that may limit absorption. Additionally, calcium should never be taken with fluoroquinolones, tetracycline, bisphosphonates, phenythoin, or levothyroxine, as the calcium hinder their absorption[9].



Oral 25-hydroxyvitamin D supplementation at a dose of 260 gm every two weeks may be given. Calcitriol (1,25-dihydroxycholecalciferol) is vitamin D's active metabolite, it appears to be a more effective treatment for these patients. Calcitriol is typically administered as an 800 U daily oral dose but can alternatively be taken as a 5000 U weekly dose[10]. The authors demonstrated that treatment with calcitriol (0.5 mg twice day) was the only factor substantially associated with improving BMD in 38 cirrhotic individuals over a 12-month period. Although calcium and vitamin D are frequently prescribed to osteoporotic patients, the data supporting their ability to reverse or prevent osteoporosis is uncertain[11].

Bisphosphonates: Bisphosphonates are antiresorptive medicines that have been seen in many studies to enhance bone mass and decrease the risk of fractures in postmenopausal osteoporosis[13]. Etidronate and alendronate both reduce bone loss in patients with PBC after one and two years of treatment, respectively. Bisphosphonates are generally well tolerated when used weekly. In patients with esophageal varices, the parenteral form is preferable because of ulcers of oesophagus[12].

CONCLUSION

Osteoporosis is the most common clinical appearance of hepatic osteodystrophy and is moderately easy to miss clinically. It is basic to identify such people before to the beginning of cracks, as this raises the danger of post transplant breaks, and lumbar spine cracks can bring about extreme morbidity even in non transplant patients with cirrhosis. While BMD is as yet used to recognize individuals with persistent liver ailment in danger of osteoporosis, clinical danger factors, for example, corticosteroid use and hypogonadism ought to be viewed as when risk is assessed. Because of the shortage of studies on ongoing liver sickness, the proof for treating osteoporosis in constant liver infection is still essentially on huge investigations of postmenopausal ladies. Bisphosphonates do improve bone mineral thickness in cholestatic patients and after transplantation.

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Conflict of interest: Nil

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