INTRODUCTION

Chronic liver disease implies a long-standing irreversible change in the structure of the liver, which may develop asymptomatically, insidiously and with no abnormal physical signs. So the aim must be to identify the condition as early as possible so that further liver damage may be minimized by appropriate treatment ***(Bisset, 1998).***

Chronic hepatitis is a continuous inflammatory hepatopathy capable of progression to cirrhosis, liver failure and death. This process can be initiated by viral infections, defective metabolism and unknown factors that initiate overreaction of immune responsiveness ***(Gupta and Fitzgerald, 1996).***

Therefore, whereas previously a six months duration of disease was required for a diagnosis of chronic hepatitis to be made, this is no longer mandatory, as the onset of illness is often uncertain, which may needlessly delay therapy ***(Thio et al., 2000).***

Better insight into the etiology and pathogenesis of chronic hepatitis also caused a shift in emphasis for the classification from purely histological features to a combination of histological, clinical and serological factors. A comprehensive and clinically useful categorization of a patient with chronic hepatitis has to consider: (i) etiology, (ii) disease activity, (iii) stage of the disease, and also additional items such as super infection with other viruses, viral mutants, immunosuppression (congenital, drug-induced, or HIV induced), and life style of the patient ***(Desmet and Roskams, 1999).***

Bone is a dynamic organ capable of rapid turnover, weight bearing and withstanding the stresses of a variety of physical activities. It is constantly being formed (Modeling) and reformed (Remodeling) ***(Casella et al., 1994).***

Bone turnover, the product of bone resorption and formation, is a tightly coupled process, with the net balance between the two will determine the bone mass and the serum calcium level. Regulation of bone turnover requires the input of a large number of hormones, growth factors, and cytokines ***(Mary et al., 2002).***

Hepatic osteodystrophy (HO) is a generic definition for the metabolic bone disease that may occur in individuals with chronic liver disease. Two distinct bone metabolic processes, *osteoporosis* (OP) and *osteomalacia* (OM) are combined together in various proportions in HO syndromes. HO is a common complication among individuals with long time lasting hepatic disease, particularly those with cholestasis ***(Guichelaar et al., 2002).***

Osteoporosis accounts for the majority of cases whereas osteomalacia is rare in the absence of advanced liver disease and severe malabsorption. The reported prevalence of osteoporosis among patient with chronic liver disease ranges from 20% to 100% depending on patient selection and diagnostic criteria ***(Vedi, 1999).***

Several studies suggest that reduced bone formation in patients with chronic liver disease is the primary abnormality (low turnover osteoporosis), whereas other report reduced or normal formation coupled

with increased resorption (high turnover osteoporosis) ***(Idliman et al., 1997).***

Osteoporosis is a disease characterized by low bone mass and architectural deterioration of bone tissue. Osteomalacia is characterized by defective mineralization of bone. Both of which are related to abnormalities of bone turnover. Biochemical markers of bone turnover reflect the degree of increase in over all bone turnovers. The rate of bone formation and resorption can be combined to assess remodeling imbalance. The rate of bone remodeling can be assessed either by measuring an enzymatic activity of the osteoblastic or osteoclastic cells, such as alkaline and acid phosphatase activity, or by measuring components of the bone matrix released into the circulation during formation or resorption, such as osteocalcin and pyridinoline cross links ***(Rosen, 1996).***

A large number of studies have shown a relationship between the level of various bone markers and the associated prediction of the Future bone mineral density measurements. In a practical sense, the higher the bone marker i.e. urine NTX value, the greater will be the risk of bone loss over the next year ***(Ross and knowlton, 1998).***

Bone markers have also been shown to predict fracture risk. The risk of fracture is greater in states of high bone turnover. Interestingly, the risk of fracture is similar in patients with low vertebral mass and in those with high bone turnover ***(Ross et al., 1997).***

The only sure way to determine bone density and fracture risk for osteoporosis is to have a bone mass measurement (also called bone mineral density or BMD test) ***(Gilsanz, 1998).***

There are several ways to measure (BMD), all are painless, non invasive and safe and are becoming more readily available ***(Bachrach et al., 1999).***

Dual-energy X-ray absorptiometry is widely accepted as a quantitative measurement technique for assessing skeletal status in adults. The world Health organization criteria for the diagnosis of osteoporosis in adults are based on the comparison of measured BMD result with the average BMD of young adults at the time of peak bone mass (PBM), defined as a T-score ***(Tothill and Avenel, 1998).***

The clinical significance of impaired bone mineralization in childhood lies in the occurrence of fractures and in the long-term impact of decreased PBM on the risk for osteoporosis later in life. Bone mineral density testing is indicated for any one at risk of osteoporosis. Axial DXA is considered the gold standard for measuring bone mineral density. It has a precision of +/-3% and is used for diagnosis and to follow treatment ***(Martin, 1999).***

Quantitative measurements of bone density by DXA should be considered in any child with one or more risk factors for osteoporosis. Identifying those at risk for reduced bone mineral density and implementing appropriate measures to modify those risks will help to ensure bone mineral health in the present as well as the future (***Orlent and Fevery, 2003).***