ORIGINAL ARTICLE

A STUDY TO ASSESS THE BONE MASS IN CHRONIC ALCOHOLIC PATIENT

Madharam Bishnoi¹, Vikki Parikh¹

Authors' Affiliation: ¹Assistant Professor, Department of Orthopedics, GMERS, Dharpur, Patan Correspondence: Dr Vikki Parikh, Email:drvikkiprkh@gmail.com

ABSTRACT

Introduction: The excessive consumption of alcohol is an important risk factor for osteoporosis. The consumption of alcohol reduces bone mass by modifying bone formation and remodeling.

Methodology: A cross-sectional study was conducted to assess the alteration in bone mineral density (BMD) in alcoholic patients, under the age of 60 year and free of non-modifiable risk factors for osteoporosis. Complete blood examination and laboratory profile was conducted in all the cases. Liver function and bone mass density were also analyzed.

Results: Total 30 male patients were studied with an average age of 51 years. Pathological levels of bone mass (in the spinal column and hip) were detected in 57% of patients (41% with osteopenia and 16% with osteoporosis), a much higher percentage than that expected in a male population of such an age. Vertebral fractures were observed in six patients (16%) and hip fractures in four (11%).

Conclusion: The active treatment of the alcoholic patient depends upon the levels of addictive behaviour. The risk of fractures and pathological levels of bone mass in alcoholic cases should be taken into consideration and accordingly comprehensive treatment should be planned.

Keywords: Bone mineral density, Alcohol, Osteoporosis

INTRODUCTION

Osteoporosis (OP) is a systemic disease of the skeleton characterized by compromised bone resistance, which predisposed an increased risk of fracture¹. There are the two properties which play an important role in maintenance of bone resistance: the bone mineral density and its quality.

Bone mineral density (BMD) is expressed in grams of mineral by surface or volume. It can be estimated using different techniques, although Double Energy Axial Radiological Absorptionometry (DEXA) is considered the standard reference for this purpose.

Since 2008 we have had available a tool, based on the work of Kanis 2005⁴, which allows us to calculate the index of fracture. Among the risk factors included is alcohol intake, which makes this approach more useful for the patients. In Spain OP affects around 2 million women over 50 years of age and some 750,000 males⁵. However, it is an illness which is underestimated by the patients themselves, by the authorities, and by health professionals⁶.

The excessive consumption of alcohol is an important risk factor for osteoporosis, above all in the male population, and is included, as we have just seen, in the FRAX Index. The consumption of alcohol reduces bone mass by modifying bone formation and remodelling⁷⁻¹⁰. A high intake of alcohol is associated with pathological and dietetic changes which can have a negative impact on bone metabolism causing osteoporosis, such as: malnutrition, vitamin D deficiency and parathormone (PTH), hypoproteinemia, hepatopathia, hypomagesemia, deficiency in Group B vitamins and folic acid, excess of iron, diminution of testosterone¹¹⁻¹⁴. Other factors, such as a reduction in B12 and folates¹⁵, or hyperhomocisteinemia¹⁶, might also have a negative impact, although their importance is yet to be determined. These chronic changes will cause a loss of bone mass which will result in osteopenia and osteoporosis at a much earlier age of 17-21. The study was planned with an objective to assess the change in bone mass in male patients with alcohol dependency.

MATERIAL AND METHODS

The present study was designed as a cross-sectional study. The study was conducted on the patients who are chronic alcoholic and admitted in our hospital for detoxification, followed by treatment to get rid of alcohol dependency. The age of all the participants were less than 60 years and had extensive other non-modifiable risk factors for osteoporosis. All the participants informed about the study procedure and voluntary informed consent taken was taken from them. The detailed alcohol history in the form of duration of the dependence, type of consumption, episodic or continuous, quantity of alcohol, maximum period of abstention, personal medical history, psychiatric and bone fracture history, and body mass index (BMI), was taken from the participants. A standard analysis was carried out on all patients, which studied liver function, markers for hepatitis B & C & Mantoux viruses, also adding the factors to be assessed in our study calcium and phosphorus in blood and urine at 24 hours, PTH, vitamin D, osteocalcine, tartarate-resistant acid phosphatase (FATr), bone alkaline phosphatase, C-terminal telopeptide (CTX), magnesium, vitamin B12 and folic acid. In addition to a radiological study of the thorax and abdominal echography, we also carried out a radiological study of the dorso-lumbar spinal column in profile, to detect fractures, which we took as the reduction of its anterior, middle and posterior height, above 20% (Genant index), as well as densitometry of the spinal column and hip, by means of double photon absorptiometry (Lunar).

RESULTS

30 male patients with an average age of 51 years were included in the study. The average body mass index was 25.50. The patients had an average duration of alcohol dependency of 26 years, with a continuous pattern of consumption in the majority of cases (74%) and an average daily consumption of 200 ml, with periods of abstinence of a maximum of 8 months. 26% of patients had psychiatric histories, most of which were anxietydepressive disorder (32%). In terms of known medical history, predominant in order of frequency were: alcoholic hepatitis 52% (cirrhosis 22.0%), ulcer 34%, diabetes 22%, pancreatitis 12%, poly-neuropathy 15% and encephalopathy 8%. It was found that 90% of patients combined tobacco smoking with their drinking habit, and 10% consumed other addictive products. Liver affectation was also found with the echographic study finding hepatic enlargement in 27% of patients and signs of portal hypertension in 46%. It was observed that 46% of patients had altered coagulation. Among the liver enzymes, GOT was high in 42% of cases, with an average of 86 U/L (normal interval 10-35 U/L), GPT was high in 64% of patients, with an average of 63U/L (10-45 U/L), FA was normal in 88% of cases, GGT high in 92%, with 373 U/L as the average (8-55 U/L). Bilirubin was high in 47%, and amylase and lipase normal in 24%.

In terms of indices of nutritional profile, the following findings stand out: anemia in 62% of patients, macrocytosis in 45%, B12 deficiency in 18%, reduction in folic acid in 21% and of magnesium in 47%. 16% of patients had hypoalbuminemia. Ferritin was high in 53% of cases. Study of lipids: hypocholesterolemia in 16% and hypoglyceridemia in 15%; hypercholesterolemia in 42% and hyperglyceridemia in 19%. Bone metabolism study: no changes in values of calcium or phosphorus were detected. PTH was high in 9% of cases, with values 14% higher than normal in these cases. Osteocalcine and bone fraction of alkaline phosphatases were normal. FATr was high in 54% of cases. CTX was increased in 39.0% of cases.

Table 1: Concurrent disease present in the participants

Disease	Percentage
Psychiatric histories	26%
Alcoholic hepatitis	52%
Ulcer	34%
Diabetes	22%
Pancreatitis	12%
Polyneuropathy	15%
Encephalopathy	8%
Hepatic enlargement	27%
Altered coagulation	46%
Anemia	62%

Table 2: Laboratory parameter of the participants

Laboratory parameters	Percentage
Hypo-albuminemia	16%
Hypercholesterolemia	42%
Folic acid deficiency	21%
B12 deficiency	18%
Increased SGOT	42%
Increased SGPT	64%
Increased GGT	92%
Increased Bilirubin	47%
Increased FATr (tartarate-resistant acid	54%
phosphatase)	
Increased CTX (C-terminal telopeptide)	39.0%

According to the OMS' densitometric criteria, 41 percent cases had osteopenia and 16 percent have osteoporosis. Of the twelve patients with osteopenia, in six it was present in the spinal column and hip, in three only in the spinal column and hip. Of the four patients with osteoporosis, in three cases this was detected in the spinal column and in one case, in the hip. The three patients with densitometric osteoporosis in the spinal column had osteopenia in the hip, and the case of osteoporosis in the hip had osteopenia in the spinal column. We found vertebral fractures in 6 patients and hip fractures in 4 patients. We did not find other extravertebral fractures. The existence of costal fractures, so prevalent in alcoholic patients, was not evaluated, since they frequently pass unnoticed clinically and radiologically, we would have required other complementary investigations (gammagraphy) to identify them with certainty.

DISCUSSION

In the present study the major organic damage was found to be hepatopathy and ulcers. A high percentage presented with nutritional deficiencies like anemia, hypoalbuminemia and vitamin deficiencies. These were accompanied by active tobacco smoking in most of the patients almost 90% of the cases. Only 10% had an addiction to other addictive substances.

Pathological levels of bone mass was much higher than would be expected in a population of males of the same age^{23,24}. This increase in oseopenia/osteoporosis concurs with that described in other studies of alcoholic patients²⁵.

In the parameters related to bone metabolism, we only detected an increase in the markers for bone resorption, both in FATr and CTX, as a manifestation of an increase in bone resorption in these patients. We detected vertebral fractures in 6 patients and of the hip in 4 patients. Four patients with vertebral fractures had osteopenia in the spinal column and hip and two of those had osteoporosis in the spinal column. Of the four patients with hip fractures, one had osteoporosis in the hip (the only case), while the three remaining had osteopenia in the hip. We only considered fractures considered to be osteoporotic, that is, produced by low impact trauma or without known cause, discarding those caused by significant trauma. Other extra-vertebral fractures were not found and costal fractures were not studied.

The present study reveals that the care of alcoholic patients needs to be comprehensive and we must study the impact of alcohol on the different organs and systems, whether the patient is admitted for detoxification, or due to alcohol-related secondary pathologies. The nature of this integrated approach will depend on the state of their addictive pathology, with the active treatment of the alcoholism being essential. However, given the importance of fractures associated with osteoporosis in the alcoholic patient, which diminishes their quality of life and increases mortality, above all through fracture of the hip, we believe it essential to assess the use of anti-fracture treatment in these patients. The application of the FRAX index could help us in this, since it already includes alcohol as a risk factor, and could help us take decisions in light of a prediction of a fracture in the next 10 years. In treating patients with digestive intolerances, and possibly with little adherence to treatment, the current availability of new drugs such as zoledronic acid, which can be given intravenously and with an annual dose, could contribute to a reduction in fractures in these patients, as well as reducing their mortality, which is increased by fractures^{26,27}. Since they are often in poor health, we must ensure that these patients do not have a septic mouth, to reduce the possibility of mandibular osteonecrosis28.

CONCLUSION

The active treatment of the alcoholic patient depends upon the levels of addictive behaviour. The risk of fractures and pathological levels of bone mass in alcoholic cases should be taken into consideration and accordingly comprehensive treatment should be planned.

REFERENCES

- NIH Consensus Development Panel on Osteoporosis Prevention, Diagnosis, and Therapy. Osteoporosis prevention, diagnosis, and therapy. JAMA. 2001;285:785-95.
- WHO Study Group. WHO Technical Report Series, 843. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Ginebra: World Health Organization, 1994.
- González Macías J, Guañabens Gay N, Gómez Alonso C, del Río Barquero L, Muñoz Torres M, Delgado M, et al. Guías de práctica clínica en la osteoporosis posmenopáusica, glucocorticoidea y del varón. Sociedad española de Investigación Ósea y Metabolismo Mineral. Rev Clin Esp 2008;208 Supl 1:1-24.
- Kanis JA, Borgstrom F, De Late C, Johansson H, Johnell O, Jonson B et al. Assessment of fracture risk. Osteoporosis Int 2005;16:581-9.
- Díaz Curiel M. Prevalencia de la osteoporosis densitométrica en la población española. En: Rhöne-Poulenc Rorer, Ed. Edimsa. Madrid, 1996;95-117.
- Calvo Catalá J, García-Borrás JJ, Campos Fernández C y grupo COSMIS: Conocimiento de la osteoporosis en los servicios de medicina interna. Resultados del proyecto COSMIS. Rev Esp Enf Metab Óseas 2004;13:1-4.
- Grazio S, Korsic M, Jajic I. Effects of smoking and alcohol consumption on vertebral deformity in the elderly an epidemiological study. Coll Antropol 2005;29:567-72.
- Naves Diaz M, O'Neill TW, Silman AJ. The influence of alcohol consumption on the risk of vertebral deformity. European Vertebral Osteoporosis Study Group. Osteoporos Int 1997;7:65-71.
- 9. Chakkalakal DA. Alcohol-induced bone loss and deficient bone repair. Alcohol Clin Exp Res. 2005;29:2077-90.
- Turner RT. Skeletal response to alcohol. Alcohol Clin Exp Res. 2000;24:1693-701.
- Kim MJ, Shim MS, Kim MK, Lee Y, Shing YG, Churg CH et al. Effect of chronic alcohol ingestión on bone mineral density in males without liver cirrosis. Korean J Intern Med. 2003;18:174-80.
- Laitinen K, Lamberg-Allardt C, Tunninen R, Harkonen M, Valimaki M. Bone mineral density and abstention-induced changes in bone and mineral metabolism in noncirrohotic male alcoholics. Am J Med. 1992;93:642-50.
- Klein RF. Alcohol-induced bone disease: impact of etanol on osteoblast proliferation. Alcohol Clin Exp Res. 1997;21:392-9.
- Sampson HW. Alcohol's harmful effects on bone. Alcohol Health Res World. 1998; 22:190-4.
- Herrmann M, Wildemann B, Wagner A, Wolny M, Schorr H, Taban-Shomal O et al. Experimental folate and vitamin B12 deficiency does not alter bone quality in rats. J Bone Miner Res 2009;24:589-96.
- Herrmann M, Tami A, Wildemann B, Wolny M, Wagner A, Schorr H et al. Hyperhomocysteinemia induces a tissue specific accumulation of homocysteine in bone by collagen binding and adversely affects bone. J Bone Miner Res 2009;44:467-75.
- Rider KM, Shorr RI, Bush AJ, Kritchevsky SB, Harris T, Stone K et al. Magnesium intake from food and supplements is associated with bone mineral density in healthy older white subjects. J Am Geriatr Soc. 2005;53:1875-80.
- Ilich JZ, Brownbill RA, Tamborini L, Crncevic-Orlic Z. To drink or not to drink: how are alcohol, caffeine and past smoking related to bone mineral density in elderly women?. J Am Coll Nutr. 2002;21:536-44.

- Kanis JA, Johansson H, Johnell O, Oden A, De Laet C, Eisman JA et al. Alcohol intake as a risk factor for fracture. Osteoporos Int. 2005;16:737-42.
- Tucker KL, Hannan MT, Qiao N, Jacques PF, Selhub J, Cupples LA et al. Low plasma vitamin B12 is associated with lower BMD: the Framingham Osteoporosis Study. J Bone Min Res 2005;20:152-8.
- Schnitzler CM, MacPhail A, Shires R, Schnaid E, Mesquita J, Robson H. Osteoporosis in African hemosiderosis: role of alcohol and iron. J Bone Miner Res. 1994;9:1865-73.
- 22. DSM-IV. American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington, DC.
- Ebeling PR. Osteoporosis in men. N Eng J Med 2008;358:1474-82.

- Calabuig E, Muñoz ML, Valero JL, García–Borrás JJ. Osteoporosis en el varón. En: Calvo Catalá. Osteoporosis. Ed. Aguilar. Valencia 2008.59-79.
- Kathleen M, Sowers MFR, Dekordi F, Nichols S. Osteoporosis Int 2003;14:396-403.
- Black DM, Delmas PD, Eastell R, Reid IR, Boonen S, Cauley JA et al. HORIZON Pivotal Fracture Trial. Once-yearly zoledronic acid for treatment of postmenopausal osteoporosis. N Engl J Med 2007;357:1799-809.
- Lyles KW, Colón-Emeric CS, Magaziner JS, Adachi JD, Pieper CF, Mautalen C et al, for the HORIZON Recurrent Fracture Trial. Zoledronic Acid in Reducing Clinical Fracture and Mortality after Hip Fracture. N Engl J Med. 2007;357:1799-809.
- Bagan JV, Poveda R, Murillo J, Díaz Fernández JM, Carbonell E, Sanchis JM et al. Bifosfonatos y osteonecrosis de los maxilares. En: Calvo Catalá. Osteoporosis. Eds. Aguilar, Valencia 2008, pág 203-13.