

The Study of Bone Density in Type 2 Diabetic Patients and Comparison with Non-Diabetic Patients Referred to Two Hospitals in Tehran , Iran

MOHSEN SOROUSH*, MOHSEN ABBASZADEH and SOOSAN SOROOSH

Rheumatology section, Department of Internal Medicine,
AJA University of Medical Sciences, Tehran, Iran.

Corresponding author E-mail : mohsensoroosh@yahoo.com

<http://dx.doi.org/10.13005/bpj/528>

(Received: October 09, 2014; accepted: November 04, 2014)

ABSTRACT

At present, the prevalence of diabetes type 1 and 2 has been increasing; on the other hand, osteoporosis is the most common metabolic bone disease in the world. The dominant view is the increased risk of osteoporosis caused by diabetes type 1. At present, scientists are seeking answers to questions about the relationship between diabetes type 2 and osteoporosis. The aim of this study is to assess the relationship between these two diseases together. If yes, researches should be done as integrated to cure the disease. This study is a case-control one. Our study population was postmenopausal women referred to densitometry sector in Imam Reza and Milad Hospitals. Study variables were collected by questionnaire and bone density was measured with Norland device. Data was statistically analyzed using SPSS software. 148 cases were studied including 69 diabetic and 79 nondiabetic patients that were matched in terms of underlying and confounding variables ($P>0.05$). Bone density in lumbar spine had no significant difference in both groups, but bone density at the lumbar spine was higher in diabetics than non-diabetics ($P<0.05$). It seems that diabetes type 2 not only doesn't cause osteoporosis, but also has a protective effect. However, given that this study did not examine the effects of anti-diabetic drugs, this result can be justified influenced by antidiabetic drugs.

Key words: Diabetes, Osteoporosis, Bone densitometry.

INTRODUCTION

Diabetes is a metabolic disorder of metabolism in the body. In this disease, the body loses the ability to produce insulin or the body becomes resistant to the effects of insulin. Thus, the produced insulin cannot do its normal function. Insulin lowers blood sugar levels by several mechanisms. There are two forms of diabetes. In diabetes type 1, the destruction of beta cells in the pancreas leads to defects in insulin production and in diabetes type 2, there is a progressive resistance to insulin in the body that ultimately may lead to the destruction of pancreatic beta cells and complete defects in insulin production. It is characterized in

diabetes type 2 that genetic factors, obesity and lack of physical activity have an important role in its development¹. There are two ways: Diabetes type 1, once known as juvenile diabetes or insulin-dependent diabetes before, is a chronic disease that occurs when pancreas (pancreatitis) secretes a small amount of insulin (A hormone needed to enter sugar into cells to produce energy) or does not secrete insulin at all. Several factors, including genetic factors and infection with certain viruses may cause diabetes type. Although diabetes type 1 usually occurs in childhood and adolescence, adults are also susceptible to this disease²⁻³. Diabetes type 2 (adult-onset or noninsulin-dependent diabetes), is one of the most common types of diabetes and

constitutes approximately 90% of patients with diabetes. Unlike diabetes type 1, the body doesn't produce insulin in diabetes type 2; but either the amount of insulin produced by the pancreas is not sufficient or body cannot use the produced insulin⁴⁻⁵. When there is no enough insulin in the body or the body does not use insulin, glucose (sugar) existing in the body cannot enter the body's cells and causes an accumulation of glucose in the body and results in problem and insufficiency. Unfortunately, there is no complete cure for this disease, but it can be improved with a healthy diet, exercise and fitness. If diet and exercise are not enough, you need to initiate medication or insulin therapy⁶⁻⁷. Osteoporosis is another very common disease in which the density of bone is reduced and then, bone strength decreases and bone becomes fragile that results in greater risk of fractures⁸. Osteoporosis usually progresses slowly and no symptoms occur until a fracture occurs. Osteoporosis can occur in any bone in the body, but it is more often in spine, pelvic, wrist and ribs. Bone is a living tissue and has blood vessels and living cells. It is being alive that lets it grow and self-repair. Over a lifetime, body bone is absorbed regularly by feeder cells in which it is (called osteoclasts) and at the same time, it is replaced with new bone by osteoblast cells. Thus, all the bones in the body are constantly being new [9-10]. Building is more than absorbing in early childhood

and adolescence, but it is contrariwise at older ages. Of course, body builds new bone even in the most severe cases of osteoporosis; just it is in small amounts. The highest bone density is at about the age of twenty. Hormone levels have an impact on the continuous absorption and production of bone. The most important hormones influential in this process are the female hormone of estrogen and the male hormone testosterone and parathyroid hormone. Among these, estrogen has the greatest impact. Genetic backgrounds and individual differences affect both in bone absorption and production and in its final result, namely, the strength and density of bone¹¹⁻¹³. Most bones in the body are formed of a main body that is perforated and porous like a sponge and it is called cancellous bone on which a rigid layer of bone is covered with no holes and no porosity, called cortical bone. In cortical area, bone is very dense and layered. In some parts of body, cancellous bone is more and in some parts cortical area. Lattice and three-dimensional structure of bones is formed due to being light, while being strong too¹⁴⁻¹⁵. Cancellous bone porosity increases in osteoporosis, so that its pores become larger and the space between the rods of scaffold increases and rods of scaffold become thinner. Also cortical part on the bone becomes thinner. The relationship between the two diseases can be very important. In a study conducted by scientists, the results indicate the decrease in BMD at the lumbar

Table. 1: Evaluation of femoral BMD in two groups (quantitatively)

Total	Femoral BMD				
	Osteoporosis	Osteopenia	Normal		
69	14	30	25	Positive	Diabetes
100%	20.3%	43.5%	36.2%		
79	26	42	11	Negative	
100%	32.9%	53.2%	13.9%		

Table. 2: Evaluation of lumbar spine BMD in entire population (quantitatively)

Total	Lumbar Spine BMD				
	Osteoporosis	Osteopenia	Normal		
69	7	39	23	Positive	Diabetes
100%	10.1%	56.5%	33.3%		
79	27	48	27	Negative	
100%	34.2%	60.8%	5.1%		

spine and normal hip BMD in type 1 diabetic patients. In another study performed on patients in Spanish, the results show a decrease in bone density at all sites in diabetic patients and in another study, no association between diabetes and bone density was emphasized. In some cases, bone density at the femoral neck was higher even in diabetic patients¹⁶⁻¹⁷.

The aim of this study is to assess the relationship between diabetes, which is a very common today, and osteoporosis. On the one hand, osteoporosis is one of the most common diseases throughout the world. In case of being any relationship between these two diseases, researches should be focused as an integrated of cross-examination of both diseases to cure.

MATERIAL AND METHODS

This study is a case-control one. Our study population was 148 postmenopausal women referred to Imam Reza and Milad Hospitals in 2011. At first, the survey questionnaire, including check list of demographic information and questions relevant to main variables, was designed and after completion by the researcher from two densitometry centers in Imam Reza hospital (501) and Milad hospital and recording bone density by Norland device, data were extracted and recorded. Then, subjects were divided into two diabetic and non-diabetic groups and patients were examined in terms of confounding variables include male

gender, smoking, history of surgery, hyperthyroidism and non-menopausal women and were excluded, if they had above items. Data collected from the patients were entered into SPSS software. In the study of quantitative variables, the distribution of variables was studied using Kolmogorov - Smirnov (KS) and P Value > 0.05 was regarded as a normal distribution. According to their distribution, parametric and non-parametric tests were used to compare the mean of each group. Descriptive data was expressed as mean, median and percentage.

RESULTS

Demographic and underlying information: In this study, a total of 148 subjects were studied, of these, 69 patients were diabetic and 79 non-diabetic. The mean age of diabetic and non-diabetic patients participating in the study was 59 and 57 years old, respectively. BMI in diabetic and non-diabetic was 28.98 and 28.36, respectively. Based on the parametric test of independent-samples t-test, both groups were matched in terms of BMI. The average age of menopause in diabetic and non-diabetic groups was 47.83 and 48.06, respectively, which indicates that both were matched on this variable. Family history of diabetes was studied in two groups. According to the results, 37.3% of diabetic and 35.9% of non-diabetic patients had a family history of diabetes. Chi-square test results indicated consistency between the two groups on this variable. 17.4% of diabetics

Table 3: Evaluation of femoral BMD in diabetic group (quantitatively)

Variable	Number	Mean	SD	Standard error of the mean	Min.	Max.
Femoral BM in diabetic group	69	0.79	0.13	0.016	0.52	1.18

Table 4: Evaluation of femoral BMD in control group (quantitatively)

Variable	Number	Mean	SD	Standard error of the mean	Min.	Max.
Femoral BM in diabetic group	79	0.74	0.10	0.012	0.49	1.14

and 17.8% of non-diabetic patients has a history of bone fractures. Calcium intake was also similar in both studied groups. The average duration of diabetes was 7 years in diabetic group, varying between 1 to 20 years.

The results of qualitative study of bone densitometry variable

A statistically significant difference can be seen using chi-square test in both diabetic and non-diabetic groups for femoral BMD.

No statistically significant difference can be seen using chi-square test in both diabetic and non-diabetic groups for spine BMD.

The results of quantitative study of bone densitometry variable, Kolmograph Smirnov test was used to assess this variable, with $P < 0.05$. Distribution of variable in the study population is reported to be normal. About femur; using parametric test of independent-samples t-test $P < 0.05$, so there was significant differences in terms of densitometry in two groups.

About spine densitometry, the results of parametric test of independent-samples t-test showed that there is no significant relationship between diabetes and spine density ($p > 0.05$).

DISCUSSION

In our study, two diabetic and non-diabetic groups were similar in terms of age, jobs, BMI, menopause age, menarche age, family history of

osteoporosis, history of fractures in themselves or first-degree family, calcium intake, history of chronic disease involved in osteoporosis and use of drugs involved in osteoporosis. In addition, smokers and people with the history of gastric surgery and hyperthyroid were exclude in the study group due to the small numbers. we concluded that lumbar spine bone density was not significant in both groups. The result was the same in the study of the effect of diabetes type 2 on osteoporosis¹⁸ and in the study of femoral neck and lumbar spine bone density¹⁹. In the study of bone turnover in postmenopausal women with diabetes type 2 using biochemical markers and assessment of bone mineral density (Turkey Study), density was higher at this location. In the study of bone mineral density in type 2 diabetic patients in Sanandaj, the bone density was lower at this location, but the difference of femoral neck bone density was significant in this study. In this case, the density was higher in diabetics. There was no significant relationship in the study of the effect of diabetes type 2 on osteoporosis¹⁸ and in the study of femoral neck and lumbar spine bone density¹⁹.

CONCLUSION

Based on results from our study it seems that diabetes type 2 not only doesn't cause osteoporosis, but also has a protective effect. The results of our research can be caused by the effects of antidiabetic drugs. Therefore it is recommended that a more comprehensive study to be done to investigate the effects of antidiabetic drugs on bone density.

REFERENCES

1. Janghorbani, M, Van Dam, R M, Willett, W C and Hu, F B. Systematic review of type 1 and type 2 diabetes mellitus and risk of fracture. *American journal of epidemiology*; **166**: 495-505 (2007)
2. Trial, C. Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. *The New England journal of medicine*; **353**: 2643 (2005)
3. Karvonen, M, Tuomilehto, J, Libman, I and LaPorte, R. A review of the recent epidemiological data on the worldwide incidence of type 1 (insulin-dependent) diabetes mellitus. *Diabetologia*; **36**: 883-892 (1993)
4. Nouwen, A, Winkley, K, Twisk, J, Lloyd, C, Peyrot, M, Ismail, K, et al. Type 2 diabetes mellitus as a risk factor for the onset of depression: a systematic review and meta-analysis. *Diabetologia*; **53**: 2480-2486 (2010)
5. Group, U P D S. Tight blood pressure control

- and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. *BMJ: British Medical Journal* ; 317: 703 (1998)
6. Amori, R E, Lau, J and Pittas, A G. Efficacy and safety of incretin therapy in type 2 diabetes: systematic review and meta-analysis. *Jama* ; **298**: 194-206 (2007)
 7. Setter, S M, Iltz, J L, Thams, J and Campbell, R K. Metformin hydrochloride in the treatment of type 2 diabetes mellitus: a clinical review with a focus on dual therapy. *Clinical therapeutics*; **25**: 2991-3026 (2003)
 8. Homik, J, Suarez-Almazor, M E, Shea, B, Cranney, A, Wells, G and Tugwell, P. Calcium and vitamin D for corticosteroid-induced osteoporosis. *Cochrane Database Syst Rev* ; **2**: (1998)
 9. Kanis, J A, Melton, L J, Christiansen, C, Johnston, C C and Khaltaev, N. The diagnosis of osteoporosis. *Journal of Bone and Mineral Research* ; **9**: 1137-1141 (1994)
 10. Cummings, S R, Kelsey, J L, Nevitt, M C and O'DOWD, K J. Epidemiology of osteoporosis and osteoporotic fractures. *Epidemiologic reviews* ; **7**: 178-208 (1985)
 11. Fink, H A, Ewing, S K, Ensrud, K E, Barrett-Connor, E, Taylor, B C, Cauley, J A, et al. Association of testosterone and estradiol deficiency with osteoporosis and rapid bone loss in older men. *The Journal of Clinical Endocrinology & Metabolism*; **91**: 3908-3915 (2006)
 12. Lufkin, E G, Wahner, H W, O'Fallon, W M, Hodgson, S F, Kotowicz, M A, Lane, A W, et al. Treatment of postmenopausal osteoporosis with transdermal estrogen. *Annals of Internal Medicine* ; **117**: 1-9 (1992)
 13. Neer, R M, Arnaud, C D, Zanchetta, J R, Prince, R, Gaich, G A, Reginster, J-Y, et al. Effect of parathyroid hormone (1-34) on fractures and bone mineral density in postmenopausal women with osteoporosis. *New England journal of medicine* ; **344**: 1434-1441 (2001)
 14. Horsman, A, Nordin, B, Aaron, J and Marshall, D. Cortical and trabecular osteoporosis and their relation to fractures in the elderly. *Osteoporosis: recent advances in pathogenesis and treatment*. University Park Press, *Baltimore* ; 175-185 (1981)
 15. Hodzman, A B, Bauer, D C, Dempster, D W, Dian, L, Hanley, D A, Harris, S T, et al. Parathyroid hormone and teriparatide for the treatment of osteoporosis: a review of the evidence and suggested guidelines for its use. *Endocrine reviews* ; **26**: 688-703 (2005)
 16. Miazgowski, T, Pynka, S, Noworyta-Zielińska, M, Krzyzanowska-Œwiniarska, B and Pikul, R. Bone mineral density and hip structural analysis in type 1 diabetic men. *European journal of endocrinology* ; **156**: 123-127 (2007)
 17. Munoz-Torres, M, Jodar, E, Escobar-Jimenez, F, Lopez-Ibarra, P and Luna, J. Bone mineral density measured by dual X-ray absorptiometry in Spanish patients with insulin-dependent diabetes mellitus. *Calcified tissue international* ; **58**: 316-319 (1996)
 18. Sharifi, F, Ahmadi Moghaddam, N and Mousavi-Nasab, N. The effects of type ii diabetes on bone density in menopause women. *Iranian Journal of Diabetes and Metabolism* ; **5**: 135-142 (2005)
 19. Hosseini-Nezhad, A, Larijani, B, Pajouhi, M, Adibi, H and Maghbouli, J. Type 2 diabetes mellitus and the effects of lifestyle on bone mineral density in pre-and postmenopausal women. *Iranian Journal of Diabetes and Metabolism* ; **3**: 13-23 (2004)