ORIGINAL ARTICLE

Evaluation of bone mineral density in type 2 diabetes mellitus patients before and after treatment

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ABSTRACT

BACKGROUND

The relationship between bone mineral density (BMD) and type 2 diabetes mellitus (T2DM) has been controversial. Recent studies have revealed adverse impact of antidiabetic drugs on BMD in type 2 diabetic patients. However, the influence of various antihyperglycaemic agents on BMD has not been well studied.

METHOD

A total of 200 patients with T2DM were screened initially for the study. Finally 67 patients (M:34, F:33) who satisfied the requirement of having been on one year of prescribed therapy were included for analysis.

RESULTS

Bone mineral density was lower in diabetic patients as compared to controls (hip $0.962 \pm 0.167\ g/cm^2$ vs $1.013 \pm 0.184\ g/cm^2$, $P=0.05$; spine $0.929 \pm 0.214\ g/cm^2$ vs $1.113 \pm 0.186\ g/cm^2$, $P<0.00001$). In males BMD was significantly lower at spine ($P<0.00001$) and in females BMD was significantly lower in both at the spine ($P<0.00001$) and hip ($P<0.032$). On multivariate analysis significant positive correlation was found between spine BMD and body mass index (BMI) ($r=0.372$, $P=0.002$), total cholesterol ($r=0.272$, $P=0.026$), low-density lipoprotein ($r=0.242$, $P=0.047$), and triglycerides ($r=0.282$, $P=0.021$). There was no correlation between BMD and glycosylated haemoglobin ($r=0.158$, $P=0.265$). A significant decrease in BMD at spine and hip was seen with the use of glitazones and metformin while increase was noted with sulphonylurea and its combination.

CONCLUSION

Men and women with T2DM have lower BMD. Bone mineral density did not have correlation to glycaemic control. Glitazones, metformin, and insulin are associated with decrease in BMD at spine, and hip, while sulphonylureas are associated with increase in BMD.

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Key Words: antihyperglycaemic drugs; bone mineral density; type 2 diabetes mellitus

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INTRODUCTION

The relationship between bone mineral density (BMD) and type 2 diabetes mellitus (T2DM) has been controversial. In some studies, patients with T2DM showed no significant difference either in BMD or prevalence of osteoporosis from nondiabetic patients,[1,2] while others have demonstrated either higher BMD in patients with T2DM compared to nondiabetics.[3,4]

Recently, there have been reports of adverse impact of antihyperglycaemic medications on BMD in T2DM patients[5,6] and increase risk of fractures with glitazones and protective effect from metformin.[6,7] However, there are limited data that address the impact of various drugs on BMD in diabetic patients. Hence, this prospective study was planned with the aim of assessing the effects of various antihyperglycaemic medications on BMD in type 2 diabetic patients in real world setting and also to compare BMD in T2DM patients with controls. We hypothesise that BMD will decrease with glitazone, increase with insulin, and will not change with sulphonylurea and metformin.

MATERIALS AND METHOD

This prospective study was conducted at the tertiary care from June 2008 to March 2010. Patients >40 years of age were selected from Endocrinology Outpatient Department at Army Hospital (R&R). All drug naïve newly diagnosed patients of T2DM (according to American Diabetes Association criteria), who consented for this study were recruited (called cases). Age- and sex-matched nondiabetic controls were recruited from the healthy population (relatives/serving soldier). All patients and controls underwent detailed evaluation about family history, past history of fracture, drug history specifically steroid use, hepatic, renal disease, thyroid and parathyroid disease, inflammatory conditions like rheumatoid arthritis, malabsorption, and menopausal status affecting BMD. Patients with renal failure, liver failure, malignant disease, on steroids or anti-epileptic drugs, and other endocrinopathies were excluded from the study. Each subject underwent measurement of BMD at hip and spine (L1–L4) using dual-energy radiograph absorptiometry (DXA) (Hologic QDR-4500 DOS Series bone densitometer).

After initial evaluation they were started on treatment (glimepiride, metformin, pioglitazone, insulin alone or in combination), which was modified monthly to achieve glycaemic control. Once stabilised, they were followed quarterly. Patients, who continued on same drugs, were compliant to treatment for one year, and maintained glycaemic control on the assigned

drugs were included in final analysis. Patients requiring modification of drugs for glycaemic control were excluded.

Of 200 patients who underwent initial screening, only 67 patients met the inclusion criteria. Three monthly clinical follow-up and yearly metabolic parameters were assessed. The BMD was measured at initial presentation and repeated after one year. The participants were divided into six groups (at the time of analysis and completion of one year of therapy) according to the therapy, viz. (1) sulphonylurea, (2) sulphonylurea and metformin, (3) sulphonylurea and glitazones, (4) metformin, (5) glitazones, and (6) insulin. According to cohort study with 95% power to detect differences with 95% exposure among cases; six cases and controls were required in each group.

Statistical analysis was carried out using EPI3.5.1 programme of Communicable Diseases Clinics (CDC), Atlanta. Data were presented as mean ± standard deviation (SD) or number (%) unless specified. Statistical analysis was done using paired '*t*' test for paired data. All unpaired parametric data were analysed by student's *t*-test and non-parametric data by χ^2-test. Pearson's correlation was calculated to assess the strength of relationship between BMD and other parameters. A *P* value of <0.05 was considered statistically significant.

RESULTS

In this study 67 patients with T2DM and 134 controls were studied. Diabetic patients had significantly lower BMD at hip ($P=0.059$) and spine ($P<0.00001$) compared to controls (Table 1). A T-score ($P=0.02$) and Z-score ($P=0.045$) at spine was significantly lower in diabetics compared to control. The T-scores and Z-scores at hip were not significantly different from controls.

Diabetic males had significantly lower BMD ($P<0.00001$) and T-scores ($P=0.0098$) at the spine than healthy controls, however, BMD at hip was comparable to controls (Table 2). Diabetic females had significantly lower BMD at both the hip ($P=0.03$) and spine ($P<0.00001$), but T-scores and Z-scores were not statistically different from the controls (Table 3).

Serum calcium was significantly higher ($P=0.0009$) and alkaline phosphatase (ALP) lower ($P<0.00001$) in diabetic population at baseline compared to controls (Table 1). Bone mineral density at spine was positively correlated with body mass index (BMI), cholesterol, low-density lipoprotein (LDL), and triglyceride, which was statistically significant in multivariate analysis; however BMD at hip was negatively correlated with age and positively with BMI (Table 4). Calcium, phosphorus, ALP, and haemoglobulin A_{1c} (HbA_{1c}) showed no relation to BMD at both sites.

Blood glucose, triglycerides (TG), LDL, and HbA_{1c} decreased with all modalities of treatment at one year. Body mass index, high-density lipoprotein (HDL), and alkaline phosphatase (ALP) increased after one year of antihyperglycaemic treatment (Table 3).

After one year of treatment when subgroup analysis was done according to treatment modalities spine BMD decreased significantly in glitazones ($P<0.009$) and metformin ($P<0.002$) groups, while it increased significantly with sulphonylurea and its combination with metformin ($P=0.006$) (Table 5). Insulin–sulphonylurea and sulphonylurea–glitazone combination produced no significant change in spine BMD. Insulin use was associated with mild decrease in BMD at hip.

Table 1 Anthropometric and bone mineral density data among cases and controls.

	Cases	Control	*P* value
Number	67	134	
Sex (M:F)	34:33	68:66	
Age (yr)	53.1±12.8	53.1±12.7	0.97
(median, range)	(48, 40–66)	(48, 40–66)	
BMI (Kg/m^2)	24.89±4.37	28.46±7.35	0.0008
Calcium (mg/dL)	9.7±0.5 9	5±0.5	0.0009
Phosphorus (mg/dL)	3.6±0.5 3	6±0.7	0.89
ALP (U/L)	88.5±33.3	214.7±59.7	<0.00001
Hip BMD	0.962±0.167	1.013±0.184	0.0596
Hip T	−0.31±1.11	−0.12±0.15	0.56
Hip Z	0.41±1.095	0.42±1.17	0.9661
Spine BMD	0.929±0.214	1.113±0.186	<0.00001
Spine T	−1.18±1.65	−0.64±1.51	0.022
Spine Z	−0.58±1.64	−0.14±1.36	0.0454

ALP: alkaline phosphatase, BMD: bone mineral density, BMI: body mass index.

DISCUSSION

The relation between diabetes and BMD is a complex one. In type 1 diabetes mellitus BMD has been shown to be reduced in several studies.[8,9] Bone mineral density has been reported to be elevated,[3,4] decreased,[8,9] and unaltered[1,2] in T2DM. Similarly, there are conflicting reports about risk of fracture among diabetic populations.[10,11]

Diabetic patients in the present study showed lower BMD at hip and spine. T-score and Z-scores at spine were lower showing loss of trabecular bone, whereas normal T-score and Z-score at hip suggesting preservation of cancellous bone. Diabetic males in the present study showed significantly lower BMD at the spine, but comparable BMD at hip with controls. T-scores at the spine were significantly lower in those above 50 years of age in the study group implying an increase in prevalence of osteoporosis among them. Z-scores were not significantly different underscoring the fact that those below 50 years of age were not likely to have increased prevalence of osteoporosis despite a lower BMD (consistent with the position statement of the International Society for Clinical Densitometry, Z-scores are recommended in evaluating BMD in individuals <50 years of age).[12] Diabetic females had significantly lower BMD at both hip and spine, however, T-score and Z-score were not different from the control group highlighting that the prevalence of osteoporosis in study group was not likely to be different from the normal female population.[12]

Table 3 Bone densitometry measurements according to sexes among cases and controls.

	Males			Females		
	Case (34)	Control (68)	*P* value	Case (33)	Control (66)	*P* value
Hip						
BMD	1.035±0.15	1.072±0.199	0.351	0.89±0.15	0.96±0.15	0.0321
Z	0.71±1.18	0.64±1.39	0.80	0.11±0.92	0.21±0.38	0.60
T	–0.06±1.19	0.19±1.45	0.37	–0.57±0.98	–0.42±1.18	0.52
Spine						
BMD	0.980±0.18	1.182±0.194	<0.00001	0.88±0.23	1.05±0.15	<0.00001
Z	–0.69±2.17	–0.24±1.55	0.144	–0.46±0.82	–0.51±1.03	0.82
T	–1.07±1.79	–1.14±1.59	0.0098	–1.29±1.51	–1.12±1.25	0.55

BMD: bone mineral density.

Table 2 Pre- and post-treatment parameters in patients (cases).

	Pretreatment	Post-treatment	*P*
BMI (Kg/m^2)	24.39±4.37	24.99±4.61	0.003
Glucose F (mg/dL)	166±32	96±10	<0.001
Glucose PP (mg/dL)	235.4±30.0	140.37±9.30	<0.001
Cholesterol (mg/dL)	181.29±29	167±23	<0.001
TG (mg/dL)	132±32	118±20	<0.001
LDL (mg/dL)	121.045±25.79	108.26±20.2	<0.001
HDL (mg/dL)	33±3	35±2	<0.001
HbA_{1c} (%)	8.497±0.65	7.69±0.07	<0.001
Calcium (mg/dL)	9.72±0.48	9.73±0.41	0.945
Phosphate (mg/dL)	3.65±0.52	3.62±0.42	0.569
ALP (U/L)	88.187±31.32	100.16±30.18	<0.001

ALP: alkaline phosphatase, BMI: body mass index, Glucose F: glucose fasting, Glucose PP: glucose postprandial, HbA_{1c}: haemoglobulin A_{1c}, HDL: high-density lipoprotein, LDL: low-density lipoprotein, TG: triglycerides.

Table 4 Correlation of spinal and hip bone mineral density with various parameters.*

	Spine (BMD)	Hip (BMD)
Age	–0.016 (0.895)	–0.0276 (0.0083)
BMI	0.372 (0.002)	0.320 (0.0084)
Sex	0.08 (0.13)	0.134 (0.0013)
ALP	–0.028 (0.819)	0.236 (0.0546)
Calcium	–0.072 (0.561)	0.043 (0.728)
Cholesterol	0.272 (0.026)	0.212 (0.08)
TG	0.282 (0.021)	0.207 (0.09)
HDL	0.437 (0.725)	0.0752 (0.533)
LDL	0.243 (0.047)	0.160 (0.195)
HbA_{1c}	0.158 (0.265)	0.091 (0.458)
Phosphates	0.015 (0.902)	0.034 (0.784)
Plasma glucose		
Fasting	0.048 (0.697)	0.052 (0.673)
Postprandial	0.02 (0.865)	0.083 (0.478)

ALP: alkaline phosphatase, BMD: bone mineral density, BMI: body mass index, HbA_{1c}: haemoglobulin A_{1c}, HDL: high-density lipoprotein, LDL: low-density lipoprotein, TG: triglycerides.
*Results expressed in *r* value (*P* value).

Table 5 Bone mineral density changes with treatment.

		L1–L4			Hip		
Drug	No.	Pretreatment	Post-treatment	*P*	Pretreatment	Post-treatment	*P*
Sulphonylurea	12	0.981±0.196	0.981±0.155	0.728	1.018±0.15	1.024±0.142	<0.001
Sulphonylurea + Metformin	13	0.927±0.19	0.937±0.185	0.006	0.959±0.172	0.964±0.03	0.048
Sulphonylurea + Glitazone	12	0.956±0.188	0.954±0.189	0.109	0.967±0.128	0.961±0.027	0.012
Metformin	12	0.866±0.097	0.817±0.307	0.002	0.939±0.193	0.897±0.203	0.013
Glitazone	10	0.938±0.214	0.898±0.21	0.009	0.966±0.172	0.944±0.173	<0.001
Insulin	8	0.900±0.162	0.836±0.162	0.055	0.903±0.151	0.881±0.142	0.02

A recent study from northern India reported lower BMD measured by osteosonography in T2DM patients after adjusting for age, BMI, and waist hip ratio.[13] Other studies have also documented similar findings.[8,9] The Rotterdam study[14] and others[3,4] have recorded a higher BMD in T2DM patients, however, BMI in these studies were higher than the present study. Body mass index (BMI) of the diabetic population was significantly lower than the control population in the present study, possibly contributing to the low BMD. Body mass index has strong positive correlation with BMD[3,4] which has also been observed in the present study. Some studies have noted no change in BMD in T2DM patients[1,2]; however, all studies had small number of patients, and were on treatment for variable duration confounding the result.

Diabetic patients had lower serum alkaline phosphatase and higher serum calcium than controls probably reflecting low bone turnover stat[15,16] and increased osteclastogenensis.[17] It has been suggested that increased oxidative stress in diabetic patients have detrimental effect on osteoblast and may contribute to diabetic osteopenia.[18] However, calcium, phosphorus, and ALP showed no correlation to BMD in multivariate analysis, which have also been observed by others.[24,25] Total cholesterol, LDL cholesterol and triglycerides were positively correlated with BMD at spine also been reported earlier.[19,20] Haemoglobin A_{1c} showed no correlation to BMD in the present study, which has been seen by investigators from India.[13]

Bone mineral density at hip and spine showed a decrease after one year of treatment possibly a consequence of glitazones use and also insulin—both of which in earlier studies have been associated with decreased BMD.[21,22] Glitazone use being associated with failure of commitment of mesenchymal stem cell precursors to differentiation into osteoblast series.[21,22] Exogenous insulin therapy removes the impact of endogenous insulin as an anabolic agent on bone.[23] After one year of treatment when subgroup analysis was done, spine BMD decreased significantly in glitazone and metformin groups, while it increased significantly with combination of sulphonylurea and metformin. Insulin–sulphonylurea and sulphonylurea–glitazone combination produced no significant change in spine BMD indicating neutralisation of effect by each other. Our findings were consistent with those of previous studies with glitazones which have shown significant decrease in BMD, increased fracture rate at hip and spine, increased bone resorption markers in patients of T2DM.[21,22] A significant association between BMD and C-peptide levels has been reported.[24,25] C-peptide has been shown to stimulate proliferation of chondrocytes thereby implying that the peptide has growth factor activity.[26] Since, sulphonylurea increase endogenous C-peptide and proinsulin levels it is prudent to expect BMD to increase with therapy, whereas exogenous insulin will inhibit C-peptide release hence may have adverse impact.

A variance from existing evidence noted in the present study was a decrease in BMD at both spine and hip with metformin therapy, contrary to earlier reports of increased BMD.[24] Metformin has been shown to decrease cellular proliferation by causing cell cycle arrest by activation of adenosine monophosphate (AMP) kinase and down regulation of cyclin D1.[27] Markers of bone formation have also been reported to be reduced with metformin use.[7] Such an inhibitory influence on cellular proliferation could, theoretically; also effect bone progenitor cells thus decreased BMD with metformin as seen in the present study. Further studies would however be required to clarify this issue.

CONCLUSION

Patients with T2DM have lower BMD at spine and hip compared to healthy controls. In males with lower T-score than controls may translates to increase prevalence of osteoporosis in males above 50 years of age, but not in females. Also, BMD in T2DM decreased with use of glitazones, metformin, and insulin, but increased with sulphonylurea in any combination. Further studies are required to ascertain its effect on fracture risk.

Intellectual Contributions of Authors

Study concept: Col MK Garg, Col R Pakhetra
Drafting and manuscript revision: Lt Col MK Dutta, Col MK Garg, Col R Pakhetra
Statistical analysis: Col MK Garg
Study supervision: Lt Col MK Dutta, Col MK Garg, Col R Pakhetra

CONFLICTS OF INTEREST

None identified.

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Book review

Clinical review of oral and maxillofacial surgery. Shahrokh C Bagheri, Chris Jo. Publishers: Mosby Elsevier, St Louis, USA. Publication: 2007. Price: $110 (Paperback). Pages: 464. ISBN: 9780323045742.

This book reflects the classic presentation format of each disease process. It also helps residents learn and review 'high yield' material that is commonly found in oral and maxillofacial training and on board examinations. The book highlights clinical information that is commonly asked during rounds, in the operating room, and during examinations. It includes an overview of the most common clinical presentation, physical exam findings, diagnostic tools, complications, treatment, and a discussion of any controversial issues related to the cases of maxillofacial surgery. The book includes 95 clinical cases focusing on essential information regarding each disease process. The detailed illustrations including radiographs and clinical photographs or drawings provide a visual guide to conditions, techniques, diagnoses, and key concepts. Contributing authors include recent graduates or senior residents in oral and maxillofacial surgery, so they are fully cognizant of students' and residents' needs as they prepare for cases, exams, and surgical procedures. It complements other Elsevier books such as *Peterson: Contemporary Oral and Maxillofacial Surgery, Fonseca: Oral and Maxillofacial Surgery, and Ward Booth: Maxillofacial Surgery*.

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