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Profile of Bone Mass and Its Determining Factors in Type 2 Diabetes: Case-Control Study

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Abstract

Background: Type 2 diabetes mellitus, beyond its well-known cardiovascular and neurological complications, is now increasingly recognized as having deleterious effects on bone tissue. It's thus presented as an independent risk factor for bone fragility with a considerable fracture risk relating to many more or less intricate parameters. The general objective of our study is to assess bone mass during type 2 diabetes in Senegalese women. **Methodology:** We had carried out a cross-sectional and descriptive study. Socio-demographic characteristics were collected on the basis of a questionnaire. Then each of the subjects had undergone a complete clinical examination followed by a blood sample for a biological assessment of certain cardiovascular risk factors. Bone mass was measured using a bio-impedancemeter. **Results:** We recruited 88 women with type 2 diabetes and 83 healthy control women. The mean age of diabetic subjects was 52.7 years ± 6.8 (with extremes of 39 and 74 years). In control, the mean age was 51.0 ± 8.5 years (with extremes of 35 and 72 years). Among the diabetic subjects, 22 subjects or 25% practiced a regular walk against 27 (32.5%) in the control. Forty-three among the diabetic subjects (48.8%) were known hypertensive and followed. According to the body mass index, 71 patients (80.7%) were overweight compared to 59 (71.1%) controls. According to the waist size, 80 (90.9%) diabetic subjects had an elevated waist size compared to 69 control women (83.1%). Among diabetic subjects, 41 patients (46.5%) were hyperglycemic imbalance according to fasting blood glu-

cose and 59 patients (67%) according to glycated hemoglobin level. Thirty-seven diabetics (42%), had both high fasting blood glucose and elevated glycated hemoglobin. The mean duration of diabetes was 8.68 ± 7.18 years. We found significantly higher bone mass in type 2 diabetic subjects ($p = 0.03$). Among diabetics, 27.3% had low bone mass compared to 36.1% of control. It's noted that the subjects of the "low bone mass" group among the control subjects also have a significant drop in other anthropometric parameters (weight, body mass index, waist size, muscle mass). It should also be noted that the fat mass is significantly higher in diabetic subjects with normal or even high bone mass. In control subjects, bone mass was positively correlated with weight ($r = 0.36$; $p = 0.001$), muscle mass ($r = 0.93$; $p < 0.0001$) and fasting blood glucose ($r = 0.26$; $p = 0.02$); and negatively correlate with age ($r = -0.22$; $p = 0.04$). On the other hand, in type 2 diabetic subjects, bone mass is positively correlated with age ($r = 0.22$; $p = 0.04$), muscle mass ($r = 0.89$; $p < 0.0001$) and the diabetes duration ($r = 0.44$; $p = 0.001$). **Conclusion:** Bone mass is higher in type 2 diabetics compared to healthy controls. Chronic hyperglycemia and the diabetes duration are believed to be responsible for the increase in bone mass. In addition, an increase in muscle mass would lead to an increase in bone mass.

Keywords

Bone Mass, Bone Mineral Density, Type 2 Diabetes, Senegalese Women

1. Introduction

Beyond the well-known cardiovascular and neurological complications, type 2 diabetes mellitus (T2DM) is now increasingly recognized as having deleterious effects on bone tissue. Diabetic patients have multiple tissue damage, including bone damage, and these are risk factors for fragility bone fractures [1]. Many observational studies, such as Women's Health Initiative (WHI), demonstrated a 20% to 70% increased risk of fracture in diabetes mellitus [2] [3]. Diabetes mellitus is thus presented as an independent risk factor for bone fragility [4].

Type 2 diabetes is the most common form of the disease and accounts for about 90% of all cases [5] [6] [7]. Authors have reported that type 2 diabetes increases the risk of fractures due to many more or less intertwined contributing factors. Work has clearly shown an increased risk of hip fracture with an odds ratio (OR) varying between 1.2 and 1.7 [8]. Along with the increased fracture risk during T2DM, bone mineral density (BMD) is on average 5% to 10% higher in type 2 diabetics than in their matched controls [9]. Thus, for a given BMD, the incidence of fracture seems to be 50% to 90% in the diabetic individual. The reference method for measuring BMD is certainly DEXA, but this assessment tool isn't available to everyone. However, an increase in BMD may be reflected by an increase in bone mass.

It's with this in mind that we proposed to carry out this study on a diabetic

female population, the objective of which was to show that the evaluation of bone tissue should be required in the follow-up of the diabetic and in the absence of a measurement of the bone mineral density, measuring bone mass may be helpful.

2. Methodology

It was a prospective, cross-sectional and case-control study. It was carried out in the human physiology and functional exploration department of the Faculty of Medicine, Pharmacy and Odontology (FMPO) at the University of Cheikh Anta Diop (UCAD) in Dakar, Senegal. It took place over the period from November 2019 to September 2020.

2.1. Protocol

The study protocol is in line with the ethical principles set out in the 1975 Helsinki declaration and has been approved by UCAD's FMPO Ethics Committee.

The parameters required for this study were notified in a same day in the morning. Socio-demographic information and their diabetes history were collected using a questionnaire. After the interview, all patients underwent a complete clinical examination.

2.2. Subjects

We had recruited 83 healthy women control and 88 types 2 diabetic women. To determine the sample size we have fixed: the minimum detectable OR at 2; number of control per case at 1; margin of error for subjects with type 2 diabetes to 5; frequency of exposure to the risk factor studied in the control population at 50%;

The risk of the first kind alpha 5%; 80% power; A one-sided test. This gives us a total number of subjects at 171. All study subjects are at least 35 years old. We didn't include subjects whose diabetes was severely complicated (ischemia, gangrene, …), lactating or pregnant women and subjects with a disease predisposing to secondary osteoporosis (hyperparathyroidism, dysthyroidism, chronic digestive disease, chronic inflammatory disease, and chronic renal failure. Subjects under treatment predisposing to osteoporosis (long-term corticosteroid therapy, thyroid hormones, prolonged treatment with heparin, …) were also excluded.

The existence of cardiovascular risk factors was sought: treated or untreated arterial hypertension, history of obesity, treated or untreated hypercholesterolemia, active or weaned smoking.

All the subjects recruited were informed of the interest of this work and all gave their oral and written consent.

2.3. Clinical Evaluation

Each subject had a complete physical examination including taking anthropometric parameters and clinical constants necessary for the study.

Measurement of arterial pressure was performed by an Omron® electronic sphygmomanometer. The size, waist size (WS) and hip circumference (HC) were measured using a tape measure to the nearest cm. Then the Waist-Hip Ratio (WHR) was calculated. WC was considered high when it was greater than 80 cm and WHR was considered high when it was greater than 0.8. Measurement of weight to the nearest kg, the body mass index (BMI), the percentage of body water, the percentage of body fat and the bone mass were performed using a level 3 bioimpedancemeter (TANITA® brand model BC 601) that allows for a global and segmental evaluation of body composition (BC). Measurements of body composition parameters such as bone mass were performed according to the manufacturer's recommendations. It was taken at the same time of day at the morning during the study. All measurements were taken in fasting subjects. All of the subject's parameters must be recorded in the device (date, time, age, sex, height, athletic or not). We place the measuring platform on a hard, flat surface where there is no vibration to ensure safe and accurate measurement. To ensure accuracy, readings should be taken without clothing and under consistent conditions of hydration. We haven't undressed subjects, but we had always removed their heavy clothes, their socks or stockings. They step onto the platform before 30 seconds after "0.0" appears to the display Screen. We had been sure the soles of their feet are clean before stepping onto the measuring platform. Then the subject stands straight on the bio-impedancemeter with bare feet on the electrodes of the plate. Their heels were correctly aligned with the electrodes on the measuring platform and they hadn't bent their knees. We had been sure that all of their fingers contact electrodes and ensure that their arms are full extended and their elbows don't touch their body. They don't move until measurement is completed. After the measurement, the unit will display all the readings. They step off the scale and we had to press the set/result button and the other button to see the desired reading.

The BMI (kg/m^2) was classified according to WHO standards proposed in 2000; Leanness ≤18.49; Normal BMI from 18.50 to 24.99, overweight from 25 to 29.99 and obesity ≥30. The classification of bone mass is presented in Table 1.

2.4. Assessment of Glycemic Balance

All study subjects were called in at 8 a.m. for testing of fasting blood sugar and glycated hemoglobin after a 12-hour night-time fast. A fasting blood glucose ≥ 1.26 and/or a HbA1c ≥ 6.5% was considered to be abnormal, and therefore a state of hyperglycemia.

Table 1. Bone mass was classified according to the age of the subjects as follows.

Ages of women	Low	Normal
Under 50 years	<1.95	≥1.95
50 to 74 years	<2.40	≥2.40
75 years and over	<2.25	≥2.25

2.5. Statistical Analysis

All the variables were recorded in an Excel table. Quantitative variables were described using the mean ± standard deviation and qualitative variables using absolute values and percentages. Student's T test was used for comparison of the mean of quantitative variables. Pearson correlation and linear regression tests were performed to search for associations between bone mass and other clinical and biological parameters studied.

The results are considered significant for a p-value ≤ 5%. Data processing was performed using SPSS software version 23.0.

3. Results

3.1. Descriptive Results

3.1.1. Population Characteristics

In control, the mean age was 51.02 ± 8.49 years (with extremes of 35 years and 72 years). On the other hand, the mean age of the diabetic subjects was 52.72 ± 6.76 years (with extremes of 39 years and 74 years).

Among the diabetics, 23.8% of the subjects practiced a physical activity against 33.7% in the control. It was a regular walk in diabetics, while some control did gymnastic. There were no smokers or alcoholics in the study population.

3.1.2. Diabetes Characteristics

The mean duration of diabetes was 8.68 years ± 7.18. Forty-three of the diabetics (48.8%) were known to be hypertensive and monitored. Therapeutically, 43.18% of patients were taking oral antidiabetic drugs only, 22 of patients (25%) were taking insulin only. Eight patients (9%) combined the diet, oral antidiabetics and insulin at the same time.

3.2. Analytical Results

3.2.1. Comparison between the Two Study Groups

Table 2 shows that subjects with type 2 diabetes in this study were significantly different from control subjects only by their hyperglycaemic state (See Table 2).

As we see in Figure 1, the bone mass in type 2 diabetic subjects was significantly higher than in control women (p = 0.03) (See Figure 1).

3.2.2. Study of the Differences in Parameters According to Bone Mass in Each Group

In Table 3, we have divided each group (control and type 2 diabetic) into two sub-groups, namely subjects with low bone mass and subjects with normal or even high bone mass. In other words, subjects with low bone mass also have a significantly lower value of other anthropometric parameters and body composition. We noted that the other anthropometric and body composition parameters such as weight, Waist size, BMI, Muscular Mass and PBF were essentially determinants of bone mass in both controls subjects and type 2 diabetics sub-

jects (See Table 3).

3.2.3. Assessment of Correlations of Bone Mass with Other Study Parameters

As shown in Table 4, after Pearson correlation tests in each group, we found that, at control subjects, bone mass is positively correlated with body weight ($r = 0.36$; $p = 0.001$), muscle mass ($r = 0.93$; $p < 0.0001$), fasting blood glucose ($r = 0.26$; $p = 0.02$) and negatively correlated with age ($r = -0.22$; $p = 0.04$). In parallel, bone mass is positively correlated with age ($r = 0.22$; $p = 0.04$), muscle mass ($r = 0.89$; $p < 0.0001$) and the diabetes duration ($r = 0.44$; $p = 0.001$) at type 2 diabetes subjects (See Table 4).

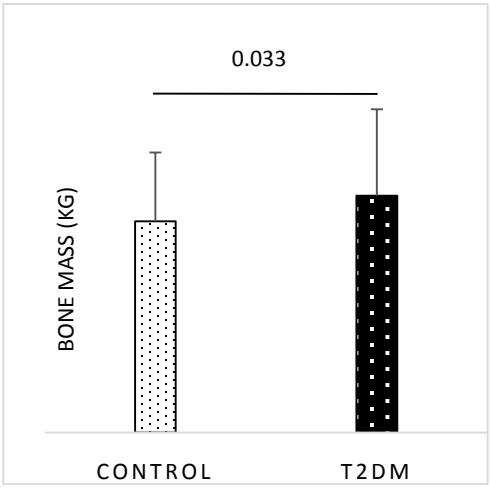

Figure 1. Comparison of bone mass between control and diabetic.

Table 2. Comparison of socio-demographic and clinical-biological data between control and diabetic.

Variables	Control	Diabetic	p-value
	Means ± SD	Mean ± SD	
Age (years)	51.02 ± 8.49	57.72 ± 6.76	NS
Weight (kg)	79.47 ± 15.64	81.34 ± 15.65	NS
Height (m)	163.53 ± 19.38	165.15 ± 6.46	NS
BMI (kg/m^2)	28.97 ± 5.65	29.74 ± 5.58	NS
Waist size (cm)	94.28 ± 12.74	96.74 ± 12.62	NS
Waist-Hip ratio	0.86 ± 0.08	0.88 ± 0.08	NS
SBP (mmHg)	135.54 ± 24.67	141.58 ± 29.69	NS
DBP (mmHg)	95.27 ± 21.95	92.80 ± 17.91	NS
Fasting blood glucose (g/l)	0.84 ± 0.12	1.46 ± 0.81	<0.0001
Glycated hemoglobin (%)	5.29 ± 0.57	7.75 ± 2.20	<0.0001

SBP: systolic blood pressure, DBP: diastolic blood pressure.

Table 3. Comparison of the different parameters studied according to bone mass in controls and diabetics.

Variables	Control		p-value	Type 2 diabetic mellitus		p-value
	Bone mass			Bone mass		
	Normal n = 53	Low n = 30		Normal n = 64	Low n = 24	
Age (years)	47.66 ± 7.47	56.97 ± 6.83	<0.0001	52.11 ± 7.31	54.33 ± 4.75	0.10
Height (cm)	167.04 ± 7.08	163.30 ± 7.63	0.92	165.78 ± 6.74	163.46 ± 5.39	0.10
Weight (kg)	85.15 ± 14.38	69.45 ± 12.58	<0.0001	86.19 ± 14.77	68.40 ± 9.43	<0.0001
Waist size (cm)	97.34 ± 12.30	88.87 ± 11.86	0.003	98.59 ± 12.42	89.08 ± 10.51	0.001
Waist-Hip ratio	0.87 ± 0.08	0.86 ± 0.07	0.53	0.88 ± 0.08	0.89 ± 0.08	0.55
BMI (kg/m^2)	30.51 ± 5.42	26.27 ± 5.07	0.001	31.33 ± 5.47	25.48 ± 3.10	<0.0001
PBF (%)	42.25 ± 6.50	39.84 ± 7.48	0.15	42.05 ± 6.99	38.08 ± 5.32	0.006
Muscular Mass (kg)	48.10 ± 10.7	38.80 ± 3.60	<0.0001	52.97 ± 10.96	40.03 ± 2.97	<0.0001
Fasting blood sugar (g/l)	0.84 ± 0.12	0.83 ± 0.12	0.77	1.94 ± 0.88	1.38 ± 0.63	0.50
Glycated hemoglobin (%)	5.30 ± 0.58	5.27 ± 0.55	0.82	7.60 ± 2.14	8.15 ± 2.36	0.33
Diabetes duration (years)	-	-	-	10.42 ± 7.45	8.05 ± 2.36	0.19

BMI: body mass index, PBF: percentage of body fat.

Table 4. Correlationship between bone mass with the others parameters.

Variables	Bone mass			
	Control		Type 2 diabetes mellitus	
Age (years)	r = −0.22	p = 0.04	r = 0.22	p = 0.04
Height (cm)	r = 0.13	p = 0.24	r = 0.10	p = 0.33
Weight (kg)	r = 0.36	p = 0.001	r = 0.18	p = 0.09
Waist size (cm)	r = 0.15	p = 0.19	r = 0.19	p = 0.07
Waist-Hip ratio	r = −0.06	p = 0.61	r = 0.02	p = 0.83
BMI (kg/m^2)	r = 0.13	p = 0.23	r = 0.12	p = 0.26
Percentage body fat (%)	r = −0.07	p = 0.55	r = −0.01	p = 0.90
Muscle mass (kg)	r = 0.93	p < 0.0001	r = 0.89	p < 0.0001
Fasting blood glucose (g/l)	r = 0.26	p = 0.02	r = 0.30	p = 0.11
Glycated hemoglobin (%)	r = 0.14	p = 0.20	r = 0.60	p = 0.06
Diabetes duration (years)	-	-	r = 0.44	p = 0.001

4. Discussion

Diabetic patients have multiple tissue damage, including bone damage with risk factors for bone fragility fractures [1]. Diabetes mellitus is a chronic endocrinopathy whose metabolic disturbances observed in all its forms interfere with bone metabolism and is accompanied by a moderate but significant increase in bone fragility with an increased risk of pathological fractures [2] [10] [11]. We there-

fore conducted this cross-sectional case-control study using a population of healthy control women and type 2 diabetic patients. At the end of this work, we noted a significantly higher bone mass in type 2 diabetics subjects compared to healthy controls ($p = 0.03$), see **Figure 1**. We didn't find any studies that directly address bone mass in diabetes mellitus, however data from the literature reports that bone mineral density (BMD) in subjects with type 2 diabetes mellitus is higher than the general population [8], but for a given age and T-score the fracture risk is higher in diabetic patients [12]. The increase in BMD [13] [14] and deterioration of bone quality [15] would be multifactorial.

We found that the control subjects in the low bone mass category were significantly older and also had a significant decrease in other anthropometric and body composition parameters namely body weight ($p < 0.0001$), BMI ($p = 0.001$), waist size ($p = 0.003$) and muscle mass ($p < 0.0001$) when they were compared to control subjects of the normal bone mass category, see **Table 3**. Also in the control subjects, bone mass was negatively correlated with age ($r = -0.22$; $p = 0.04$) and positively correlated with body weight ($r = 0.36$; $p = 0.001$) and muscle mass ($r = 0.93$; $p < 0.0001$), see **Table 4**. Likewise, diabetic subjects in the low bone mass group also have a significant decrease in other parameters namely weight, BMI, WS and muscle mass, see **Table 4** yet. Authors have reported that high body weight and/or high BMI were positively correlated with increased BMD and a risk of reduced bone fragility in healthy subjects regardless of gender [16]. In addition, in healthy women of old age, decreased body weight led to bone loss [17] unlike diabetic subjects. In view of these constants, the other anthropometric parameters and body composition would be determinants of bone mass both in control subjects and in type 2 diabetic subjects.

According to the BMI, 71 patients (80.7%) were obese against 59 (71.1%) subjects among the control women. In addition, bone mass is positively correlated with muscle mass in both control and diabetic subjects. More interestingly, fat mass was significantly higher in diabetic subjects with normal or even high bone mass. In fact, type 2 diabetes is often associated with obesity or an abundant fat mass could have positive effects on bone tissue; in particular on the mechanical load which could stimulate bone formation by reducing apoptosis and increasing the proliferation and differentiation of osteoblasts and osteocytes via the Wnt β-catenin signaling pathway [18]. This mechanical explanation was the basis of the hypothesis that obese people could be protected from bone loss and osteoporosis. In addition, large fatty tissue is considered a source of estrogen production by increasing the aromatization of androgens to estrogen, and may therefore contribute to an increase in BMD. However, these assumptions are controversial as more recent studies have reported that people with a high percentage of body fat have low BMD and a higher prevalence of osteoporosis [19] [20]. In addition, increased adipose tissue in type 2 diabetes is a source of production of adipokines such as leptin which exerts negative effects on trabecular bone [21], adiponectin which was also negatively correlated with total bone den-

sity [22]. On the other hand, these adipocytokines could increase the number and activity of osteoclasts, in particular by an increased production of RANK ligand [23]. Locally, increased intramedullary adiposity has been correlated with decreased BMD and increased fractures [4]. The increase in bone marrow adipogenesis was accompanied by a reduction in the number of osteoblasts, confirming an inverse relationship between osteoblastic and adipocyte differentiation. The brown phenotype of bone marrow fat, which secretes bone anabolic factors, is attenuated in diabetic [24]. Activation of the transcription factor PPAR—directs mesenchymal cells towards adipocyte differentiation to the detriment of osteoblast differentiation. At the same time, sarcopenia is a risk factor for falls in the general population. This appears particularly important in diabetic subjects, especially if they are obese, this is the notion of sarcopenic obesity [25]. This is a plausible pathophysiogenic hypothesis because apart from diabetes, sarcopenia is a risk factor for loss of bone mass.

According to the fasting blood glucose level, 41 diabetic patients (46.5%) were in a hyperglycemia state, whereas if we consider the Glycated hemoglobin (HbA1c) this imbalance concerned 59 (67%) diabetic patients. In addition, bone mass being positively correlated with fasting blood glucose ($r = 0.26$; $p = 0.02$) in the control subjects and with the diabetes duration ($r = 0.44$; $p = 0.001$) in the diabetic subjects, this would indicate that long-term chronic hyperglycemia is a factor in both increased bone mass and bone fragility. This finding is in line with certain data in the literature which has shown that the diabetes duration and poor glycemic control seem to be associated with an increased risk of fractures [26]. This risk is even higher the older the diabetes even if it's treated [27]. T2DM is characterized by insulin resistance and it has been shown that alterations in insulin metabolism have a negative impact on bone remodeling. Insulin has bone anabolic properties, it stimulates osteoblastic proliferation and differentiation. But it also stimulates the activity of osteoclasts, responsible for bone resorption. A duration of type 2 diabetes beyond 10 years is associated with an increased risk of major osteoporotic fracture. Moreover, the risk of hip fracture is increased regardless of the diabetes duration, but especially since the diabetes has been evolving for a long time [28]. At the same time, a high concentration of glucose in vitro has a deleterious effect on bone forming cells, osteoblasts, and may promote bone resorption [29]. In humans, an increased risk of fracture is observed for an HbA1c level ≥ 8% [8]. Chronic hyperglycemia also has an indirect effect through the production of advanced glycation end products (AGEs) which will accumulate in the bone matrix [30]. Chronic hyperglycemia induces the production of AGEs through a non-enzymatic glycation process, modifies intracellular signaling cascades and increases oxidative stress. All of these mechanisms interact and lead to numerous structural and functional changes in the tissues of the body, especially bone tissue [31] and the vascular wall inducing atherosclerosis [32]. Atherosclerosis is a microvascular complication of diabetic mellitus which is itself associated with an increased risk of fractures [8] by de-

creased bone quality [33].

In addition, chronic inflammation could have a role in the development of bone fragility in diabetic subject. In the literature it has been shown that the production of pro-inflammatory cytokines is stimulated by chronic hyperglycemia but also by the activation of RAGEs expressed by bone cells [29].

As in most studies BMD is normal or even increased in type 2 diabetic subjects, bone fragility is strongly suspected to be due to an alteration in bone quality, and it could be explained at least in part by an alteration of the bone quality and bone remodeling [29]. Studies have reported an alteration of bone microarchitecture during T2DM with increased cortical porosity compared to control subjects [34] [35], decreased cortical bone density and strength, decreased total bone area [36].

On the other hand, similar conclusions have been made by other authors with data that support a decrease in bone material strength index (BMSi) during type 2 diabetes mellitus [37] [38] [39]. Results from the literature have shown in larger studies that HbA1c was inversely correlated with the value of BMSi [38] [39]. This index measures the resistance to penetration at the periosteum of the tibia's upper end. It's an index of bone resistance that is reduced in situations of bone fragility.

The pathophysiology of diabetic bone has been unclear, but the recent development of new tools has provided evidence for a particular bone metabolism in type 2 diabetes mellitus.

Abnormalities of several metabolic pathways have been suggested such as deregulation of oxidative stress, accumulation of advanced glycation end products of bone matrix components that impair bone quality and defective acquisition of bone mass [40].

The deterioration in bone quality linked to disturbances in carbohydrate metabolism and changes in BMD can be indirectly estimated by evaluating bone mass which has been possible using a bio-impedancemeter. It's a simple, non-invasive, inexpensive method with a portable equipment. Its handling is simple with a highly acceptable methodology [41].

The limitation of this study lies in the fact that the sample of the study population is small and the non-inclusion of men; which can constitute a selection bias. A study on a larger population would allow us to better establish our results.

5. Conclusion

An increase of bone mineral density during T2DM has been reported by many authors. The increase in bone mass that we saw in this study would be a reflection of the increase in BMD which seems to be a predictive sign of bone fragility. The increase in bone mass can be assessed by bio-impedancemetry which is a rapid, reliable, reproducible, inexpensive and highly acceptable method. Thus, in the absence of a BMD measurement, regular assessment of bone mass should be integrated into the follow-up of the diabetic to watch for possible bone fragility.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Data Sheet for Bone Mass in Type 2 Diabetes

Date of the survey …… Patient Index ……

Order number …… Address ……

Tel ……

I) Socio-demographic characteristics

First name (s): ……Last name: ……

Age (years): ……Sex: ……...…….…….

Ethnic group: ……Profession: …….

Civil servant ☐ Self-employed ☐ Volunteer ☐ Housewife ☐

Unemployed ☐ Retired ☐ Student ☐ Others ☐

Schooling: Yes ☐ No ☐

Level of study: ……Language of study: ……

II) Personal background, defect and lifestyle

➢ Medical:

HTA ☐ Obesity ☐ Dyslipidemia ☐

Sedentary ☐ Heart disease ☐ which ……..

Others:……..……

Others illness: Yes ☐ No ☐ which……..……..

➢ Surgical

Yes ☐ No ☐

Type of surgery: ……..……...……

➢ Gyno-Obstetrics:

Pregnancy in progress: ……..……

Others: ……..……

➢ Lifestyle

Smoking: Yes ☐ No ☐

If yes, number of packages/Day: ……..……..…

Duration of smoking: ……

If weaned, give the weaning date: ……..……...……

Alcohol: Yes ☐ No ☐

Physical activity: Yes ☐ No ☐ Frequency per week: ……..

III) Clinical characteristics

1) Constants

Height: ……m Weight: ……kg Waist size:……cm

Hip circumference: ……cm BMI: …….kg /m^2 DBP: ……...…mmHg

SBP: …….mmHg Heart rate: ……..……..bpm

2) Characteristics of diabetes:

Age of onset: ……years Diabetes duration: ……years

Presence of diabetes complications: ……..…….

Others: ……

IV) Biological parameters

Fasting blood glucose……g/l Glycated hemoglobin ……..……..…%

V) Treatment:

Diet: Monitoring ☐ Not monitoring ☐
Antidiabetic drugs: yes ☐ No ☐
Others: ……