Journal of Education and Science (ISSN 1812-125X)

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Assessment of Foot Bone Quality in Patients with Type2 Diabetes Receiving Cortisone Treatment

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Article information

Article history:
Received January 30, 2024
Accepted March 12, 2024
Available online June 01, 2024

Keywords:
Quantitative ultrasound (QUS)
Dual-energy X-ray absorptiometry (DXA)
Type2 diabetes
Cortisone.

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Abstract

The aim of this study was to study the effects of tepy2 diabetes (T2DM) and treatment cortisone on the foot bones. A total of 123 Iraqi men and women participated in the study (18 males and 105 females), with type 2 diabetes receiving cortisone therapy. Quantitative Ultrasound (QUS) was used to evaluate osteoporosis, speed of sound (SOS), broadband ultrasound attenuation (BUA), and calcaneus bone quality index (BQI). A dual x-ray absorptiometry (DXA) was used to determine abdominal fat percentage. The results indicate that the correlation between T-score and heel bone mineral density (BMD) for cortisone, as well as Z-score and heel BMD, is a linear relationship that has statistical significance P-value<0.0001. When we examine the relationship between calcaneal BUA and T-score we found that it is exponational with a P-value<0.0001. The correlation between calcaneal SOS and BMI is not statistically significant (P-value=0.9). Osteopenia and osteoporosis appear at the age of 43-82 years, for T2DM patients receiving cortisone therapy so was a T-score (-2.0 to -3.1). It was found that the BQI is less for patients with T2DM (62.4, 58.2), for both genders .The body mass index was (BMI) for patients with T2DM (21, 45), whereas the abdominal fat % for patients with T2DM is high for males and females (32.7, 36.4). One of the risk factors for cortisone is high blood sugar, so we noticed an increasing number of T2DM patients receiving cortisone therapy (58).

DOI: 10.33899/edusj.2024.146466.1423,

1. Introduction

Osteoporosis is a skeletal disorder characterized by low bone mass and microstructural bone tissue deterioration, which increases bone fragility and fracture susceptibility [1]. It is considered one of the most serious diseases, often referred to as a silent disease because it is typically painless and shows no symptoms until a bone fracture. Osteoporosis fractures can occur in any bone, they most frequently occur in the vertebrae spine, proximal femur (hip), hummers (upper arm), distal forearm (wrist), and calcaneus bone (heel), and these sites where fractures commonly occur because they are containing a high ratio of trabecular bone, several factors that cause fragility fractures as shown Fig 1 [2]. It primarily affects older men and postmenopausal women, and its prevalence increases with age [2, 3]. In clinical practice, dual-energy X-ray absorptiometry (DXA) is the technique that is most frequently used to measure bone mineral density (BMD) accurately [4]. One of the disadvantages of DXA is the inability to distinguish between cortical and trabecular bone, as well as the possibility of measurement errors due to nearby soft tissues from the measurement site [5]. DXA also has disadvantages other than exposing the patient to ionizing radiation, is very expensive, and requires large equipment [6]. Quantitative ultrasound (QUS) is a different way to measure BMD; it is typically done at the calcaneus bone or other peripherals [7]. This method is widely

used because it is inexpensive, easy to use, portable, and produces no ionizing radiation [8]. The calcaneal bone is the only bone advised by the ISCD for measuring BMD with QUS. For several reasons, it has little soft tissue, making it easy to measure the bone, has relatively flat surfaces for bone, and contains 90% trabecular bone. The right calcaneal bone is used to measure BMD. BMD is higher for the right foot than the left foot, because it is dominant for the right foot [7]. Parameters measured by QUS broadband ultrasound attenuation (BUA, dB/MHz), which measures the scattering and absorption of ultrasound waves, reflects the BMD as well as its microarchitecture and elasticity, and the speed of sound (SOS, m/sec) measure the velocity of ultrasound passing through the water, bone, and surrounding soft tissues [9]. Diabetes is a group of metabolic disorders that are distinguished by a condition of chronic hyperglycemia that is brought on by inadequate insulin action. Type 2 diabetes was identified as a part of the metabolic syndrome. T2DM, which used to be called non-insulin-dependent DM, is the more common type of DM. It is marked by high blood sugar, increased insulin resistance, and a relative lack of insulin [10, 11]. T2DM is associated with an increased risk of fracture, despite that BMD is unaffected and higher in diabetic patients, confirmed by some studies [9,11]. The causes are likely a combination of factors, including the length of the illness, insufficient glycemic control, a higher risk of falling due to hypoglycemia, osteopenia, bone quality impairment, and medication side effects, which could increase the risk of bone fragility and fractures [12]. Some risk factors for T2DM include (old age (age>45 years), a history of gestational diabetes, cardiovascular diseases, hypertension, dyslipidemia, and irregular metabolism) [13]. Corticosteroids which include glucocorticoids and mineralocorticoids are a type of steroid hormone released by the adrenal cortex. The term corticosteroid is most commonly used to refer to glucocorticoids [14]. Glucocorticoids are commonly, for treating autoimmune and inflammatory diseases, such as asthma, allergies, rheumatoid arthritis, etc. It is the treatment used includes (cortisone, hydrocortisone, dexamethasone, prednisone, etc). The side effects that are associated with high doses and long-term lead to osteoporosis because of lost bone [14]. Cortisone is used to treat a variety of diseases as it reduces inflammation and pain, and it can be used to treat chest allergies. Long-term use of cortisone causes side effects such as obesity, osteoporosis, high blood sugar, and other diseases [15]. The most common type of secondary osteoporosis is bone loss brought on by corticosteroids. It typically occurs from an overusing corticosteroid, increasing the risk of pathological fracture while losing cortical and cancellous bone, as it affects bone metabolism, by changing the balance between osteoclasts and osteoblasts [16]. This study aimed to investigate the effects of T2DM and treatment cortisone on osteoporosis of the foot.

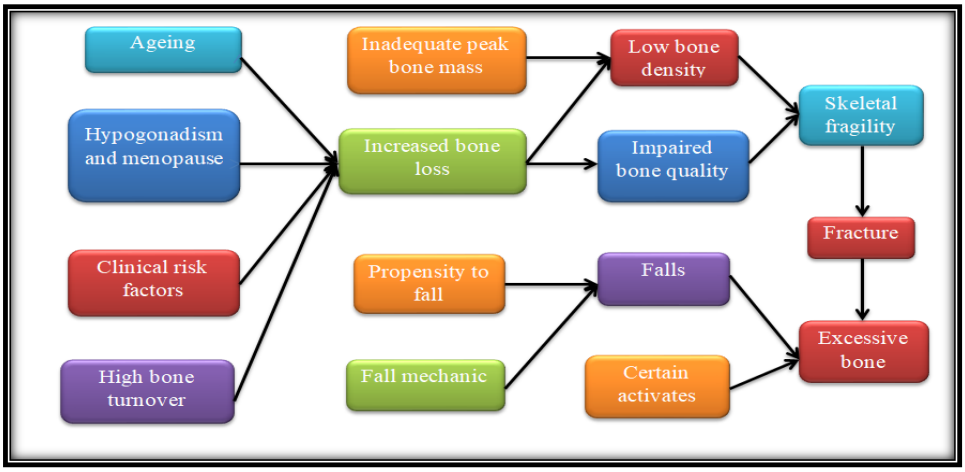

Figure 1: Show factors causing fractures associated with osteoporosis [17].

2. Material and Method

The number of patient participants was 123 of both genders (105 females and 18 males) aged (23-82) years, with $mean \pm SD$ male ages (54.28 ± 14.79) years, and female (56.76 ± 10.88)years. QUS technique (SONOST 3000 OsteoSys) was used to measure osteoporosis for the right foot at the calcaneus (heel) for both genders for patients with T2DM, and cortisone treatment. DXA was used to measure abdominal fat %.

At the start anthropometric data measurements including height, weight, and body mass index (BMI), as in the following Equ.1. The body weight (kg) was measured by a highly sensitive digital scale. A stadiometer was used to measure the height (m). The World Health Organization (WHO) can classify BMI, as shown in Table 1.

Using the following equation: $BMI = (weight/height^2)\ to\ (Kg/m^2)$ (1)

Table 1: Classification of BMI (Underweight, Normal, Overweight, and Obesity) according to WHO [18].

Weight Status	BMI (kg/m^2)
Underweight	< 18.5
Normal	18.5-24.9
Overweight	25.0-29.9
Class**I** Obesity	30.0-34.9
Class**II** Obesity	35.0-39.9
Class**III** Obesity	≥ 40

Patients were asked when they got diabetes and no diabetes which period of age, and if they take cortisone treatment. A calibrated device was used to calibrate the QUS device before the testing procedure was started, as shown in Fig.2 (B). At the start of the testing, where a gel was put at the calcaneal (heel), and the foot was put inside the QUS device, as shown in Fig. (A)2. The room temperature the patient's age, height, and weight, and the patient's gender, were recorded (males-females). The QUS data include SOS, BUA, BQI, T-score, and Z-score. The testing time was between 5 to 10 minutes. By using the DXA device can be a measure of abdominal fat percentage. The T-score, which has been used to identify osteoporosis in old people, postmenopausal women, and men over 50 years, is inversely correlated with fracture risk. The Z-score was used for children and young adults with low bone mass. The T-score can be classified according to WHO guidelines as shown in Table 2 [19]. Several other measures were derived from these measurements to include (SOS, and BUA), and estimated BMD.

The (BMD) at the heel, by combining between SOS and BUA, as in the following Equ.2 [20]:

$$Est, heelBMD = 0.002592 \times (BUA + SOS) - 3.687\ g/cm^2 \qquad (2)$$

Table 2: T-score for osteoporosis diagnosis according to WHO criteria suitable for men and women after menopause [21].

Status	Criteria
Normal	T-score$\geq$-1.0
Osteopenia	T-score between -1 and -2.5
Osteoporosis	T-score$\leq$-2.5
Severe osteoporosis	T-score$\leq$-2.5 in the presence of one or more fragility fractures

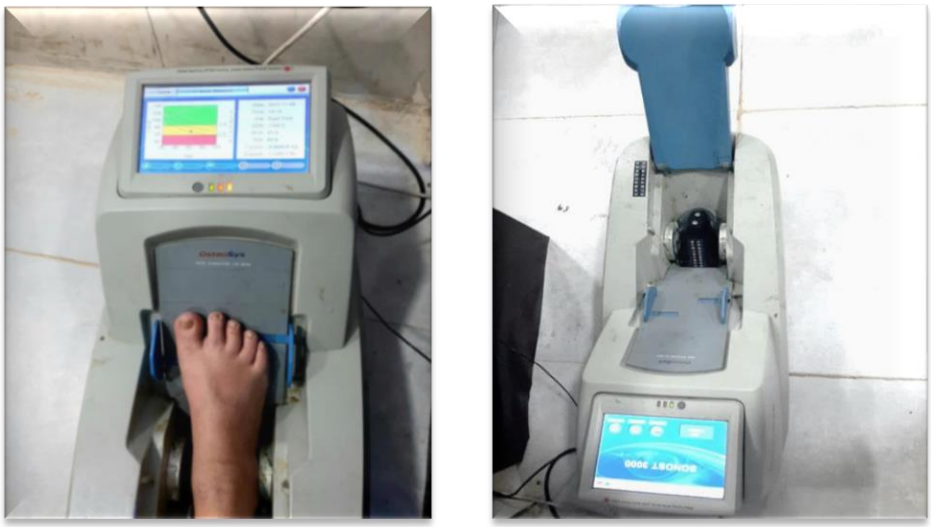

Figure 2: (A) QUS was the measurement of osteoporosis at the heel of the right foot, (B) Quantitative ultrasound device calibration.

3. Results

Table 3 shows height measurements for the studied sample for both genders according to the ages 23-82 years. Males had the highest (1.80±0.10 m) for the age 23-32 years and the highest for females (1.62±0.011 m) in the same age period for males. Females had the lowest height (1.52±0.02 m) for the ages 73-82 years and the lowest male (1.70±0.07 m) for the ages 63-72 years. Minimum for males was (1.61 m) for the age range 43-52 and 53-62 years, whereas the maximum for males (1.90 m) for the ages 23-32 years. Maximum height for females (1.76 m) for the age 53-62 years, whereas the minimum height for females (1.49 m) for the ages 23-32 and 73-82 years.

Table 3: Height measurements for patients according to age.

Age Classes years	Height (m)									P-value
	Female			Male			Total			
	Min	Max	$Mean \pm SD$	Min	Max	$Mean \pm SD$	Min	Max	$Mean \pm SD$	
23-32	1.49	1.71	1.62±0.11	1.70	1.90	1.80 ±0.10	1.49	1.90	1.70±0.13	0.1
33-42	1.51	1.70	1.59±0.06	1.78	1.78	1.78±0.00	1.51	1.78	1.61±0.08	0.03
43-52	1.50	1.71	1.61±0.05	1.61	1.76	1.72±0.07	1.50	1.76	1.63±0.06	0.003
53-62	1.50	1.76	1.58±0.05	1.61	1.80	1.71±0.08	1.50	1.80	1.59±0.07	P<0.0001
63-72	1.48	1.72	1.56±0.05	1.63	1.76	1.70±0.07	1.48	1.76	1.58±0.07	0.001
73-82	1.49	1.55	1.52±0.02	1.72	1.74	1.73±0.014	1.49	1.74	1.56±0.09	P<0.0001

Table 4: shows weight measurements for both genders in the age group 23-82 years. Males had the highest (97±0.00 kg) for the ages 53-62 years, and females had the highest (86.65±13.47 kg) for the ages 43-52 years. Females had the lowest (59±10.53 kg) for the ages 23-32 years, and males had the lowest (78±8.88 kg) for the ages 63-72 years. The minimum is for

males (72 kg) for the ages 23-82 years, and the maximum is for males (97 kg) aged 33-42 years. Maximum females (145 kg) for the ages 63-72 years and the minimum females (42 kg) for the ages 53-62 years.

Table 4: Weight measurements for patients according to age.

Age Class year	Weight (kg)									P-value
	Female			Male			Total			
	Min	Max	$Mean \pm SD$	Min	Max	$Mean \pm SD$	Min	Max	$Mean \pm SD$	
23-32	48	69	59±10.53	72	88	82±8.71	48	88	70.50±15.28	0.04
33-42	64	123	91.13±21.97	97	97	97±0.00	64	123	91.78±20.651	0.8
43-52	68	113	86.65±13.47	81	95	89±6.68	68	113	87±12.63	0.2
53-62	42	112	79.71±15.29	77	117	95.60±17.74	42	117	81.56±16.20	P<0.0001
63-72	62	145	84.15±17.52	68	85	78±8.88	62	145	83.52±16.83	0.001
73-82	74	85	79.57±4.39	80	81	80.50±0.70	74	85	79.78±3.83	P<0.0001

The SOS measurements according to age for both genders are shown in Table 5. The minimum value for females was (1459.8 m/sec) for the ages 53-62 years, whereas the minimum value for males (1474.2 m/sec) for the ages 43-52 years. The maximum SOS for females was (1555.9 m/sec) aged 43-52 years, whereas the maximum for males (1564.8 m/sec) aged 33-42. Females had the highest (1510.62±13.60 m/sec) for the ages 33-42 years, and males had the highest (1564.8±0.00 m/sec) for the ages 33-42 years. Males had the lowest value (1491.27±13.41 m/sec) for the ages 43-52 years, and females had the lowest value (1478. 97±17.01 m/sec) for the ages 73-82 years.

Table 5: SOS measurements for patients according to age.

Age Classes year	SOS (m/sec)									P-value
	Female			Male			Total			
	Min	Max	$Mean \pm SD$	Min	Max	$Mean \pm SD$	Min	Max	$Mean \pm SD$	
23-32	1486.4	1509.1	1497±11.42	1482	1511.1	1495.53±14.65	1482	1511.1	1496.26±11.78	0.9
33-42	1488	1534.5	1510.62±13.60	1564.8	1564.8	1564.8±0.00	1488	1564.8	1516.64±22.09	0.007
43-52	1459.9	1555.9	1500.55±18.48	1474.2	1506.9	1491.27±13.41	1459.9	1555.9	1499.17±17.92	0.3
53-62	1459.8	1546.4	1496.18±18.23	1476.4	1534.3	1505.3±24.54	1459.8	1546.4	1497.24±18.94	0.3
63-72	1464.5	1516.5	1486.98±13.03	1480.2	1498.1	1491.66±9.95	1464.5	1507.9	1487.46±12.68	0.6
73-82	1466.5	1516.5	1478.97±17.01	1479.4	1481.8	1480.6±1.69	1466.5	1516.5	1479.33±14.76	0.1

Bone quality index measurements according to age for both genders are shown in Table 6. The minimum for females was (33.4) for the ages 53-62 years, and the minimum BQI for males was (33.4) for the ages 63-72 years. The maximum for females was (139.9) for the ages 43-52 years, and the maximum BQI for males was (147.9) for the ages 33-42 years. The highest value for females was (85.87± 10.48) for the ages 33-42 years, and the highest value for males was (147.9±0.00) at the

same age for females. The lowest value for males was (53.30±3.67) for the ages 73-82 years, and the lowest value for females was (49.68± 17.37) the same age as males.

Table 6: BQI measurements according to age.

Age Classes year	BQI Female Min	Female Max	Female $Mean \pm SD$	Male Min	Male Max	Male $Mean \pm SD$	Total Min	Total Max	Total $Mean \pm SD$	P-vale
23-32	66.1	80.4	74.27± 7.36	57	88.9	72.73±15.95	57	88.9	73.50± 11.14	0.9
33-42	64.6	99.4	85.87± 10.48	147.9	147.9	147.9±0.00	64.6	147.9	92.76± 22.88	0.8
43-52	37.7	139.9	75.63± 21.26	45.3	78.1	64.80±13.88	37.7	139.9	74.03± 20.50	0.2
53-62	33.4	114.2	69.49± 17.91	60.3	125.7	89.22±27.53	33.4	125.7	71.78± 19.89	0.4
63-72	35.2	86.1	61.54± 15.17	33.4	76.2	60.60±23.64	33.4	86.1	61.44± 15.67	0.3
73-82	38.1	87.7	49.68± 17.37	50.7	55.9	53.30±3.67	38.1	87.7	50.48± 15.18	0.7

As we mentioned earlier, bone loss or low BMD causes osteoporosis. The results show a good correlation between T-score and heel BMD for cortisone, and a correlation between Z-score and heel BMD, as shown in Fig. 3 & Fig. 4. The determined, strength of the correlation between the two variables is established using the Pearson correlation coefficient (r) by way of correlation analysis, and find determination coefficient (r^2). Table 7 shows a strong positive correlation coefficient (r=0.993, 0.948), and a determination coefficient (r^2=0.986, 0.899). The between T-score and heel BMD, Z-score, and heel BMD was seen to be linear and has a significance of (P<0.0001). Linear regression prediction equations were used to plot the variation in T-score and heel BMD, Z-score, and heel BMD, as follows in Equ. 3&4.

$$T - score = -6.76 + 11.46 \times heel\ BMD \quad (3)$$

$$Z - score = -7.72 + 15.45 \times heel\ BMD \quad (4)$$

Table 7: Correlation analysis of ultrasound T-score Z-score and heel BMD.

Variable	Correlation coefficient (r)	Determination coefficient (r^2)	P-value
T-score and heel BMD	0.993	0.986	P<0.0001
Z-score and heel BMD	0.948	0.899	P<0.0001

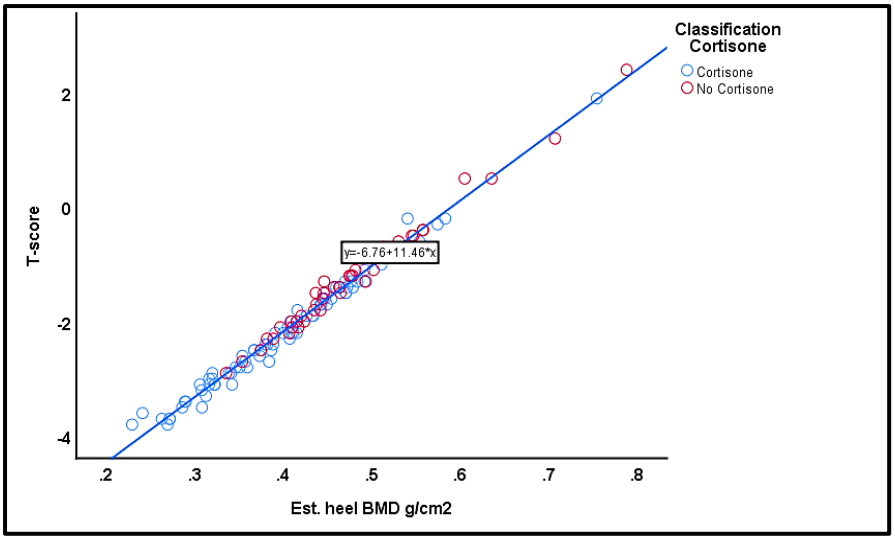

Figure 3: Correlation between T-score and Est. heel BMD for classification cortisone.

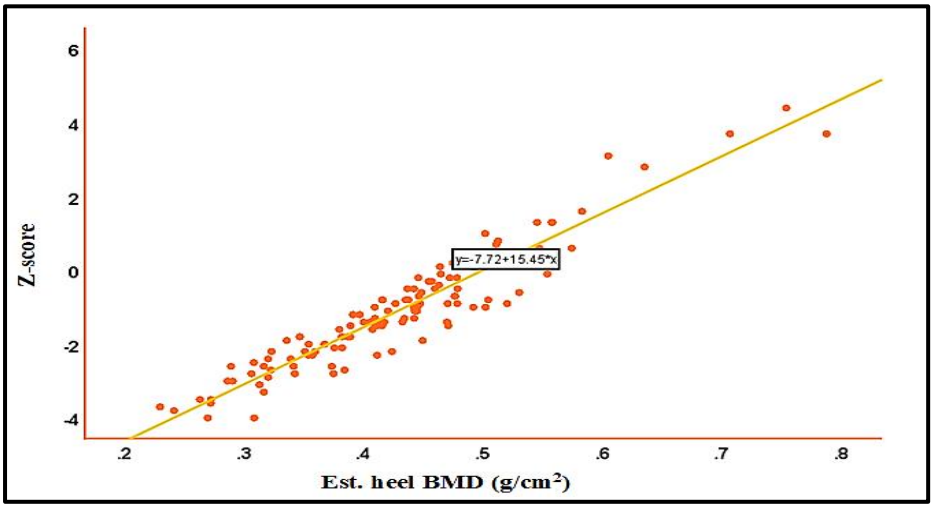

Figure 4: Correlation between Z-score and Est. heel BMD.

Fig. 5 shows the relationship between BUA and T-score, which is established using Pearson's correlation coefficient (r), and determination coefficient (r^2). By using correlation and regression analysis, an exponential regression model can be obtained. The relationship between calcaneal BUA and T-score increases exponentially, and the increase in BUA leads to an increase in T-score. Table 8 shows the perfect and strong positive correlation coefficient (r=0.820), and the determination coefficient (r^2= 0.673). It was found this relationship has a statistical significance at (P<0.0001), the exponential regression model as follows Equ. 5:

$$BUA = 127 \times (exp(\ 0.19 \times T - score)) \qquad (5)$$

Table 8: Correlation analysis calcaneal BUA, BQI, and T-score.

Variable	Correlation coefficient (r)	Determination coefficient (r^2)	P-value
BUA and T-score	0.820	0.673	P<0.0001

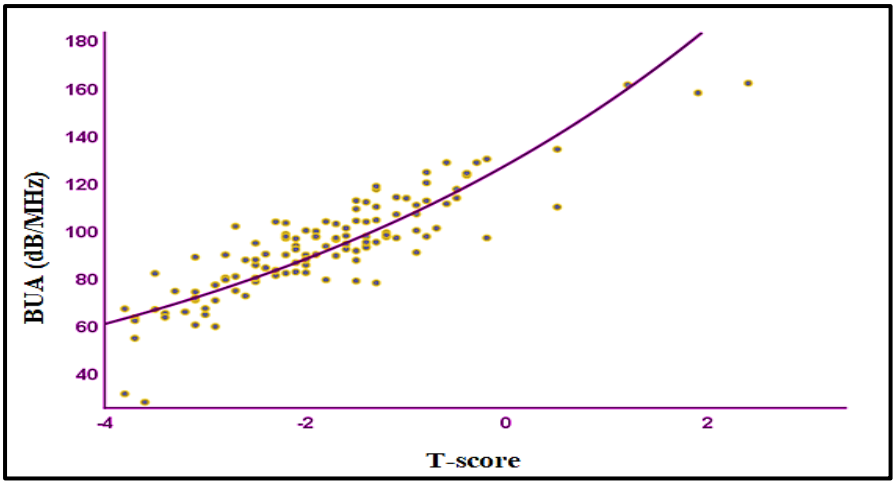

Figure 5: Correlation between calcaneal BUA and T-score.

Fig. 6 & Fig. 7 show the relationship between heel BMD and BMI, SOS, and age, which is established using Pearson's correlation coefficient (r), and determination coefficient (r^2). By using correlation and regression analysis, a linear regression model can be obtained. Table 9 shows the weak negative correlation coefficient (r=-0.008,-0.329), and the determination coefficient (r^2= 0.00006, 0.108). The inverse correlation between SOS and age. The relationship between heel BMD and BMI has no significance (P-value=0.9), but the relationship between SOS and age has significance (P-value=0.0002). Linear regression prediction equations are used to plot the variation in heel BMD and BMI, (SOS and age), as follows in Equ. 6&7.

$$heel\ BMD = 0.43 - 0.000112 \times BMI \quad (6)$$

$$SOS = 1530 - 0.54 \times age \quad (7)$$

Table 9: Correlation analysis of heel BMD and BMI, calcaneal SOS, and age.

Variable	Correlation coefficient (r)	Determination coefficient (r^2)	P-value
heel BMD and BMI	-0.008	0.00006	0.9
SOS and age	-0.329	0.108	0.0002

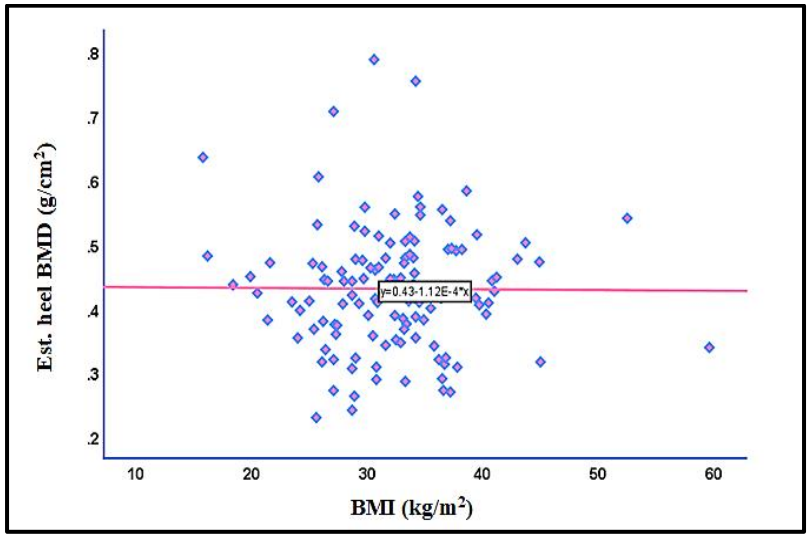

Figure 6: Correlation between heel BMD and BMI

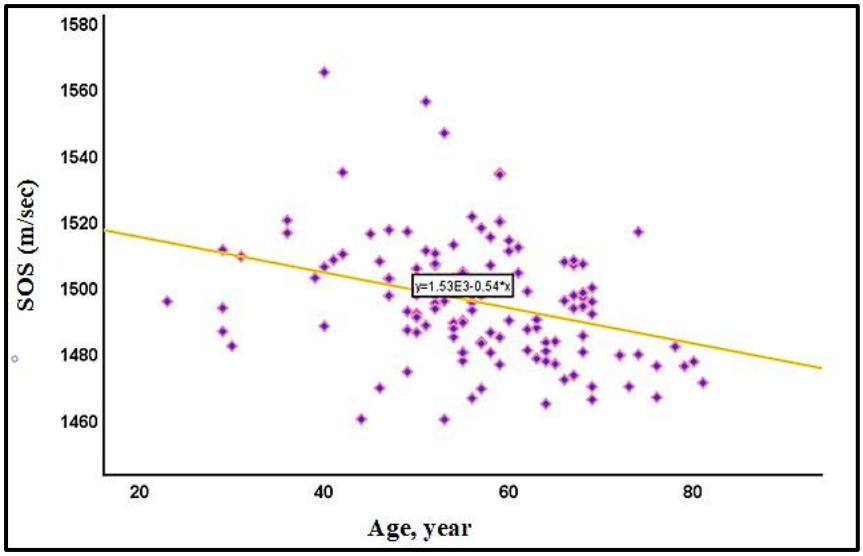

Figure 7: Correlation between calcaneal SOS and age.

This study provides an evaluation of the measurements of the T-score at the foot calcaneus (heel) area and knows its amounts according to patients undergoing therapy by cortisone. In the following Fig. 8, the T-score (normal, osteopenia, and osteoporosis) was higher in the patients receiving cortisone treatment than in the patients not receiving cortisone treatment.

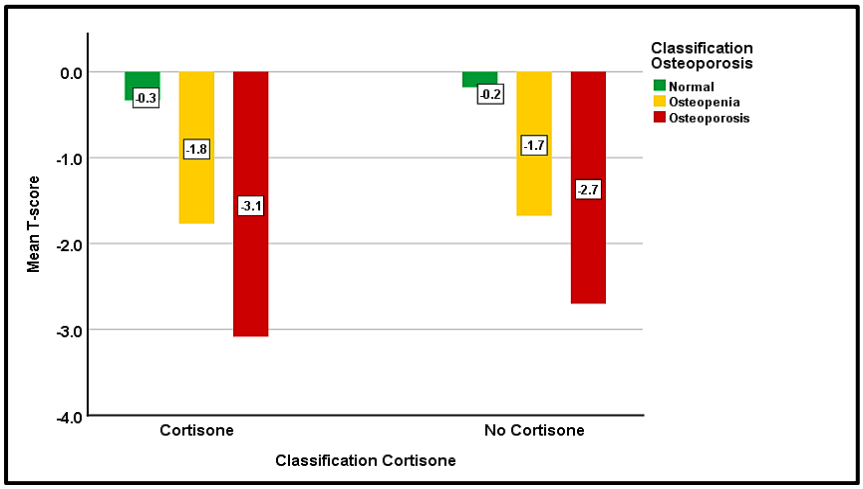

Figure 8: The mean T-score calcaneal bone according to cortisone.

Fig. 9 shows the distribution T-score of the calcaneus of patients undergoing cortisone treatment and not undergoing cortisone according to age 23-82 years. The T-score was for patients undergoing cortisone treatment (-1.5) and not undergoing cortisone (-1.8) at the age of 23-32 years. In the age period 33-42 years, the value was T-score for patients undergoing cortisone (-1.0) and not undergoing cortisone (-0.3). The T-score was for patients aged 43-52 years undergoing cortisone (-1.7) and not undergoing cortisone (-1.1). The patients undergoing cortisone T-score (-2.2) for the ages 53-62 years and the patients not undergoing cortisone T-score (-1.2). The T-score for patients undergoing cortisone (-2.5) and not undergoing cortisone (-1.7) for the age of 63-72 years. In the age period 73-82 years, the value T-score for patients undergoing cortisone (-3.1) and not undergoing cortisone (-1.9). Therefore, these results, show older age and receiving cortisone treatment, lead to osteoporosis.

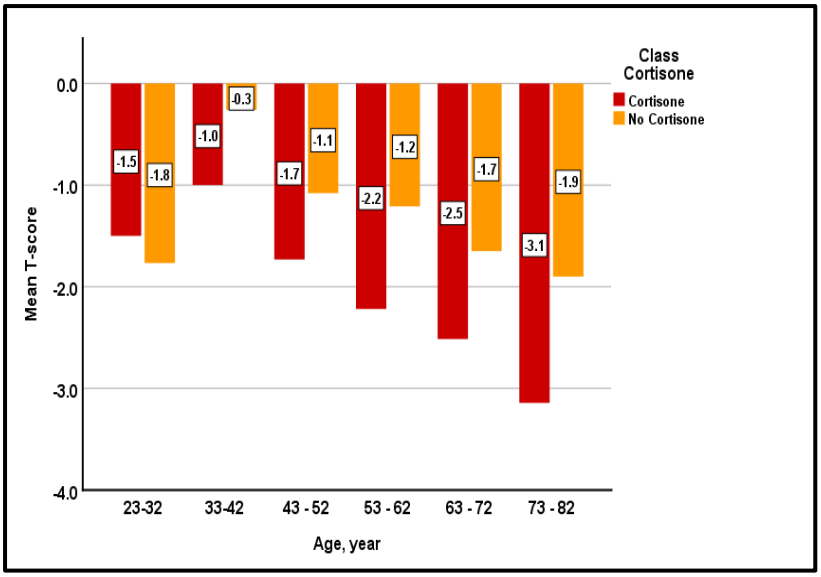

Figure 9: The mean T-score calcaneus bone according to age for cortisone.

In this study, one can know the effect of T2DM on the BQI. Fig. 10 shows that patients with T2DM had BQI less than patients with no T2DM for females-males. Where BQI was females (58.2), less than the males (62.4) for T2DM because females are more likely to have diseases that affect bones.

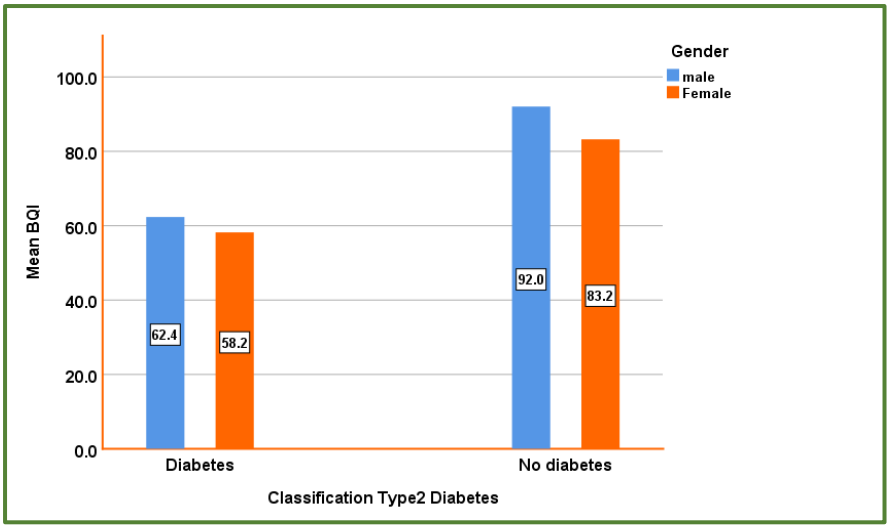

Figure 10: The mean BQI calcaneus bone according to type2 diabetes for both genders.

Fig. 11 shows the T-score for the foot calcaneus (heel) according to the age of patients with T2DM. The start of T2DM occurs between the age of 43-82 years, and during this age period, patients with T2DM are more exposed risk to osteopenia and osteoporosis than patients with no T2DM.

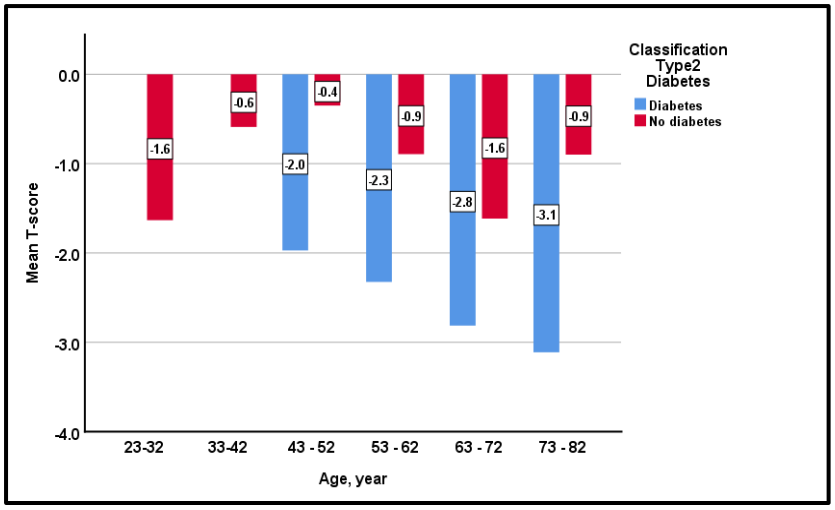

Figure 11: The mean T-score calcaneus bone according to age, for patient's type2 diabetes.

The results give the evaluation of the measurements of the abdominal fat % for patients with T2DM for females and males. Fig.12 shows that abdominal fat % was high in the patients with T2DM for both genders (32.7, 36.4) compared to the patients without T2DM (32.1, 33.8).

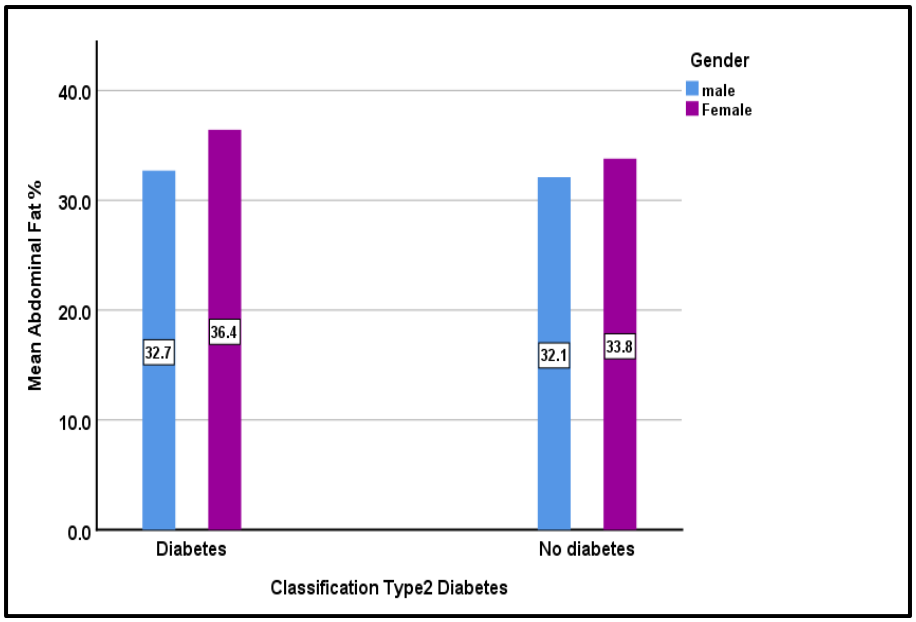

Figure 12: Abdominal fat % according to type2 diabetes for both genders.

Fig.13 shows the number of patients with T2DM according to BMI (Underweight, Normal, Overweight, and Obesity). It shows the highest number of patients with T2DM and those that do not have T2DM in the overweight classes (21, 14), and obesity classes (45, 33). The results of this study, that people overweight, obese, and with high abdominal fat % is high, are more likely to have T2DM because considered that one of the risk factors main for T2DM is obesity and high abdominal fat %. On the other hand, there is a relationship between T2DM and the treatment of cortisone as in Fig. 14. The number of patients with diabetes undergoing cortisone treatment (58) was high compared to no diabetes and not receiving cortisone (21). This means undergoing cortisone therapy leads to high blood sugar.

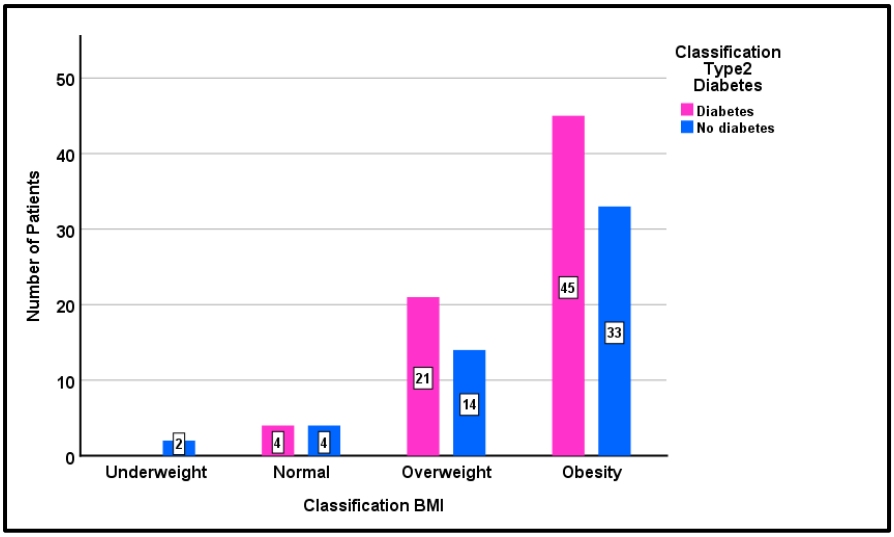

Figure 13: The number of patients according to body mass index for type2 diabetes.

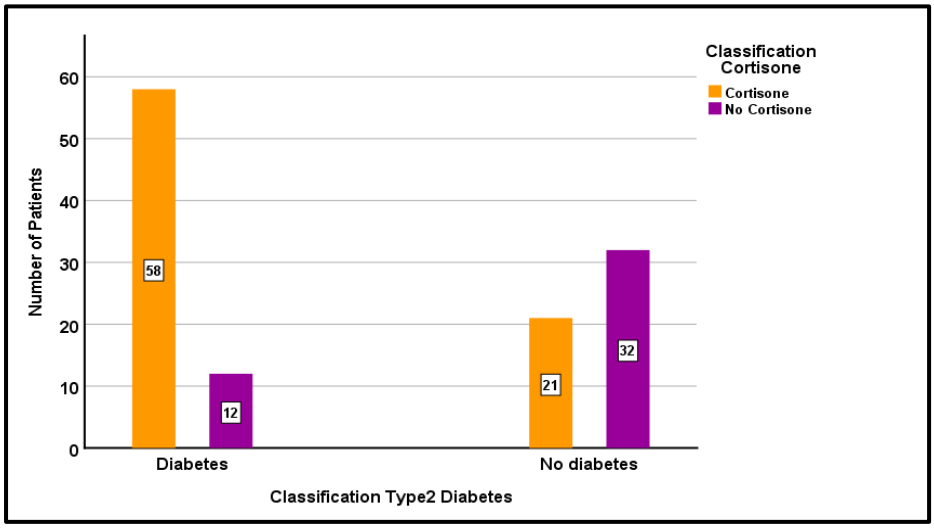

Figure 14: The number of patients with type2 diabetes for cortisone

4. Discussion

This study used the QUS technique measurement of osteoporosis of the right foot at the heel (calcaneal) for patients with T2DM and receiving cortisone therapy for the male and female Iraqi populations. Previous studies have used the technique of DXA, quantitative computed tomography (QCT), and peripheral quantitative computed tomography (pQCT) [22, 23]. Tables 5 and 6 show the decreases in SOS and BQI with age for females aged 43-82 years, because of entering females in menopause, and estrogen levels drop this comes in agreement with the study Rivas-Ruiz et al [24], whereas the males had different measurements. In previous studies, women had a much higher risk of developing osteoporosis and osteoporotic fractures than men, because of larger bone size for men, and stronger bone structure compared to women. Therefore osteoporosis is common in postmenopausal women because of the sudden drop in estrogen levels [25]. BMD of the calcaneus is dependent not on BMI, and this agrees with Damilakis et al. [26], but the SOS is dependent on age this study we observed their linear decreases with age in the measurement of SOS, which agrees with Moris et al[27]. Although, it was found there is a linear correlation between body weight and bone density in the scales Roberts et al [28]. Diabetes is a metabolic disorder characterized by hyperglycemia. An etiology includes defects in insulin secretion, action, or both, considered T2DM to be one of the most common metabolic disorders [29]. This study shows people aged 43-82 years, suffering T2DM, and is more likely osteoporosis. Therefore, the prevalence of osteoporosis is associated with T2DM and aging, and these chronic diseases are commonly associated with the elderly, has been confirmed by Russo et al [25]. T2DM causes abnormal bone cell function and matrix structure, which leads to more osteoblasts dying. It increases osteoclasts' work and makes osteoblasts work less. This

means that it affects bone metabolism, which speeds up bone loss and causes osteopenia and osteoporosis [12]. Some studies by Majumdar et al show T2DM leads to high BMD, but in our study, it was found the bone quality of patients with T2DM is less than those without T2DM for both genders, but females show less than males as a result of female exposure to more diseases, as well as pregnancy and childbearing. Therefore this study shows that T2DM affects bone quality and works to weaken the bone. A substantial body of prior research demonstrates that diabetes is more severe or long-term and is associated with decreases in BMD [30]. Furthermore, common diseases including cardiovascular diseases also have low BMD and lead to osteoporosis. The association between osteoporosis and cardiovascular diseases is partially due to conventional risk factors such as diabetes, estrogen deficiency in women, smoking, and low physical activity, this agrees with Michel et al [31]. Several studies by Russo et al show that related complications to diabetes affect bone metabolism and low bone quality, complications include especially nephropathy or peripheral neuropathy, which is one of the most common problems affecting the foot for diabetes, and it leads to inflammation in the soft tissue and bone. Additionally, medications used in the treatment of T2DM may affect bone metabolism [25]. Studies have proven that patients with T2DM are often distinguished by their obesity or increased body fat percentage, which is typically found in the abdominal area [31], this is in agreement with our study that people with T2DM suffer from being overweight and obese, which also increases the abdominal fat for both genders, especially females because T2DM start at the age of 43-82 years, therefore, at this period females enter menopause and the decreases in estrogen, as a result, adipocytes increases in the abdominal area. The main causes of the T2DM epidemic are the rise of obesity, sedentary lifestyles, high-calorie diets, and the aging population [31]. Glucocorticoids are used in medicine to treat many diseases caused by an overactive immune system, such as asthma, allergies, and autoimmune diseases [32]. Cortisone is one of the glucocorticoid medicines used in the treatment of asthma and allergies, so alterations in ratio bone turnover were related to variations in the amount of cortisone in the blood. Nevertheless, studies have confirmed patients receiving long-term therapy glucocorticoids, lead to happen harmful effects on bone, including osteopenia, osteoporosis, and osteonecrosis [33]. The current study shows that people on medication cortisone are more likely to have osteopenia and osteoporosis than people not on medication cortisone. A body of previous evidence shows that the effects of glucocorticoid (cortisone) medication on bone metabolism are causing death cells osteoblasts, as well as extending the lifespan of osteoclasts [34]. Fig. 14 shows that there is a relationship between cortisone and diabetes, so people the take medications cortisone are found to suffer from diabetes at a greater than people no take cortisone, this means the glucocorticoid medications (cortisone) lead to a ratio of high blood sugar, this agreement with Lansang and Hustak [35]. The cortisone risk factor is obesity, so the results the current study show that people with obesity have T2DM as a result of receiving medication cortisone, and metabolic irregularities. Cortisone increases blood sugar because cortisone considers is a hormone that is anti-insulin, it faces the insulin normally secreted by the pancreas and stops its work inside the body.

5. Conclusion

This study found that diabetic patients receiving cortisone had a high risk of infection osteopenia and osteoporosis, resulting in the impact of T2DM and cortisone on bone metabolism. A study found a reduction in bone quality for patients with T2DM in the foot for both genders, but females more than males, and these decreases occur due to age, low estrogen in women, in addition, the long-term use of for medication diabetes, and cortisone. Patients with T2DM are distinguished by their obesity and increased abdominal fat percentage in women more than in men. Long-term use of cortisone therapy leads to overweight/obesity, weakness in the tendons and nerves, and high blood sugar. So, we found there is a relationship between cortisone and T2DM. The linear relationships between T-score and heel BMD for therapy cortisone, addition T-score, and calcaneal BQI for T2DM. The relationship between Z-score and heel BMD is also linear. The relationship between calcaneal BUA and T-score is exponential. There is a low inverse relationship between heel (BMD) and BMI, in addition to calcaneal SOS and age.

6. Acknowledgements

The authors would like to thank the University of Mosul / College of Education for Pure Sciences for their facilities, which have helped to enhance the quality of this work.

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تقييم جودة العظام القدم لدى المرضى المصابين بداء السكري من النوع الثاني والذين يتلقون علاج الكورتيزون

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المستخلص

كان الهدف من هذه الدراسة هو دراسة تأثير مرض السكري النوع الثاني T2DM والعلاج بالكورتيزون على عظام القدم. شارك في الدراسة 123 رجلاً وامرأة عراقيين (18 ذكراً و105 اناث)، من مرضى السكري من النوع الثاني الذين يتلقون العلاج بالكورتيزون. تم استخدام الموجات فوق الصوتية الكمية (QUS) لتقييم هشاشة العظام، وسرعة الصوت (SOS)، والتوهين بالموجات فوق الصوتية ذات النطاق العريض (BUA)، ومؤشر جودة العظام العقبي (BQI). تم استخدام قياس امتصاص الأشعة السينية المزدوج (DXA) لتحديد نسبة الدهون البطن. تشير النتائج الى أن العلاقة بين T-score وheel BMD كثافة المعادن في عظم اكعب للكورتيزون، وكذلك Z-score وheel BMD هي علاقة خطية لها دلالة احصائية P-value<0.0001. عندما نفحص العلاقة بين calcaneal BUA و T-score وجدنا انها اسية P-value تبلغ قيمتها P<0.0001. العلاقة بين calcaneal SOS و BMI ليست ذات دلالة احصائية (P-value=0.9). تظهر قلة العظام وهشاشة العظام في سن 82-43 بالنسبة لمرضى T2DM الذين يتلقون العلاج بالكورتيزون كانت T-score (-3.1 to -2.0). وقد وجد أن BQI اقل لدى المرضى T2DM (62.4, 58.2) لكلا الجنسين. كان مؤشر كتلة الجسم (BMI) الذين يعانون من T2DM (21, 54)، في حين أن نسبة الدهون في البطن للمرضى الذين يعانون من T2DM مرتفعة بالنسبة للذكور والأناث (32.7, 36.4). احد عوامل الخطر الكورتيزون هو ارتفاع نسبة السكر في الدم، لذلك لاحظنا عددًا متزايدًا من مرضى T2DM الذين يتلقون العلاج بالكورتيزون (58).