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# Evaluation of Bone Mineral Density in Children and Adolescents with Type 1 Diabetes

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## ABSTRACT

**Background**: Type 1 diabetes mellitus (T1DM) is an autoimmune disease characterized by pancreatic beta cell loss that typically affects children and teenagers. **Objective:** To evaluate bone mineral density ((BMD)) in children and adolescents with T1DM. **Methods**: This case-control study was carried out on 100 children who were attending diabetes clinics at Menoufia University and Benha Insurance Hospitals. Children divided into two groups: **Group 1** (n=50) children diagnosed T1DM, and **Group 2** (n=50) completely apparently normal children, matched with patient group in age, sex, ethnicity, and socioeconomic level as controls. All patients were subjected to the following: Full History Taking, complete Clinical Examination. Laboratory Tests Included (serum vitamin D level, Serum calcium, phosphorus, serum alkaline phosphatase). BMD was assessed by DEXA scan at the Spine (L2- L4) and the femur.

**Results**: Between T1DM patients and controls, there was a substantial difference in levels of vitamin D, BMD, and alkaline phosphatase (p-value <0.001). Phosphorus and calcium levels were substantially different between the control group and patients with type 1 diabetes (p=0.045 and p=0.007, respectively). There was a very significant correlation between serum alkaline phosphatase (p-value < 0.001) and DEXA scan (z-score).

**Conclusions**: BMD and Vitamin D level of diabetic patients is lower than the control group. There was a significant difference between patients and controls regarding serum calcium and phosphorus levels, their values are lower in patients than controls.

**Keywords:** Bone Mineral Density, Vitamin D, Type 1 Diabetes, DEXA scan.

## INTRODUCTION

Type 1 diabetes mellitus (T1DM) is an autoimmune disorder that results in the destruction of insulin-producing beta cells in the pancreas. It is mostly diagnosed in children and adolescents. Individuals with T1DM are prone to developing severe complications such as retinopathy, neuropathy, and nephropathy as the disease progresses. These complications can affect various systems, including the skeletal system, especially in adults. T1DM has been linked to a heightened risk of reduced bone density, even among younger patients like children and adolescents [(1)].

Glycaemic control, age at onset of diabetes, diabetic complications, and duration of disease may influence bone mass in type 1 diabetes. Disagreement exists on the crucial clinical variables that impact bone density. In addition, as insulin is believed to have anabolic effects on bone, decreased insulin production before diabetes symptoms appear may influence bone health [(1)]. A major public health problem, osteoporosis is associated with increased mortality and morbidity rates. Type 1 diabetics also have a higher risk of osteoporosis and fractures compared to the whole population, according to epidemiological studies [(2)].

There is an increased risk of bone fractures in adolescents with type 1 diabetes because they may not attain their optimum bone mass potential. Hypothesised but as-yet-unproven mechanisms for the phenomenon include hyperglycaemia, which significantly impairs osteoblast function and bone formation [(2)], and a decrease in circulating insulin-like growth factor 1 (IGF-1) levels, which in turn reduces their growth potential. Since evidence suggests that bone loss may be considered a late consequence of type 1 diabetes, evaluating bone health is an important issue in T1DM follow-up [(2)].

Bone mineral density (BMD) is best measured using a dual-energy X-ray absorption (DEXA) scan. DEXA typically checks the lumbar spine and proximal femur in children; however, it may check any part of the body. In addition to the reference population, the following factors should be considered: the child's sex, weight, height, radiographic bone age, pubertal staging, and ethnicity [(3)]. By comparing the measured BMD values with an essentially normal matched paediatric database, the Z score is generated, which is used to assess BMD in children. A value that is consistent across gender, race/ethnicity, and age is produced by this procedure. The widespread consensus is that Z scores of -2 or below fall outside of the range considered age appropriate [(3)]. Possible pathogenic pathways of T1D-related bone degradation include hyperglycaemia, an IGF-1 shortage, chronic inflammation, non-enzymatic glycosylation of type 1 collagen with advanced glycation end products, and vitamin D insufficiency [(4)]. The co-occurrence of low BMD and T1DM is a frequently contested subject in the literature and is considered a public health problem [(5)].

This is since the influence of T1DM on juvenile bone density is still up for discussion. This research aims to evaluate BMD in children and adolescents diagnosed with type 1 diabetes.

## PATIENTS AND METHODS

This case-control research involved 100 pediatric patients attending Diabetes Clinics at Menoufia University and Benha Insurance Hospitals from 2022 to 2024.



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All boys and girls between the ages of 6 and 18 were eligible to participate. Participants were not included if they were on corticosteroids or any other medication known to impair BMD, had a medical history of conditions including CKD, or had a history of bone fractures.

**Methodology:**

All participants underwent medical history review, clinical examination, anthropometric measurements (weight, height, and BMI, all adjusted for Z-scores), and laboratory tests for serum vitamin D, calcium, phosphorus, alkaline phosphatase, parathyroid hormone, and HbA1c. Bone mineral density (BMD) was assessed using DEXA scans at the lumbar spine (L2-L4) and femur. DEXA scan apparatus was produced by GE Health Care corporation in 2016. Standard deviation units (Z-values) were used for reporting the difference compared to the age-matched reference population. Z scores below -2 indicate poor BMD, BMD values between -2 and -1 suggest low normal BMD, and values more than or equal to -1 are considered normal.

**Ethical approval:**

**Signed informed consent forms were obtained from all participants. The study was authorized by the Faculty of Medicine Ethics Committee at Menoufia University with IRB No. 11/2022 PEDI 44. The study adhered to the Helsinki Declaration throughout its execution.**

***Statistical analysis***

Data was collected, tabulated, and analysed statistically using an IBM personal computer running SPSS version 20. Projects in Armonk, New York and Epi Info 2000 used the following data. We used the Shapiro-Wilk and Kolmogorov-Smirnov tests to check for normality and expressed the qualitative data as numbers and percentages (%). Mean ($\bar{X}$), standard deviation (SD), range, median, and interquartile range were the forms that quantitative data was presented in. Two groups may be compared using the Mann-Whitney test, which is a significance test for non-normally distributed quantitative variables, and the student's t-test, which is a test for parametric quantitative variables. Two qualitative variables were examined using the chi-squared test ($\chi 2$). The Spearman correlation coefficient test (r-test) is a powerful tool for investigating the connection between non-parametric quantitative data. Findings from an r-test, which measures the correlation coefficient, can indicate either positive (+) or negative (-) correlations. To find out how strongly two variables are related linearly, you may utilise it. A P-value below 0.05 was considered statistically significant.

**RESULTS**

There was no distinction between both groups regarding vital data. With p-values of 0.704, 0.746, and 0.306, respectively, for height (cm), BMI (kg/m2), and weight (kg), the T1DM patients and control group showed no statistically significant differences **(Table 1)**.

**Table (1): Sociodemographic and clinical data in studied groups.**

| | Items | Cases (No=50) | | Control (No=50) | | Test of sig. p-value |
|---|---|---|---|---|---|---|
| | | No | % | No | % | |
| **Sex** | - **Male** | 36 | 72.0 | 25 | 50.0 | |
| | - **Female** | 14 | 28.0 | 25 | 50.0 | |
| **Age (years)** | - **Mean ± SD** | 11.6±3.8 | | 11.8±3.6 | | U = 0.384 |
| | - **Min-Max** | 6-18 | | 6-18 | | P =0.701 (>0.05**)** |
| | - **Median (IQ)** | 12 (8-15) | | 11.5 (9-15) | | |
| **Residence** | - **Benha** | 27 | 54.0 | 27 | 54.0 | |
| | - **Menoufia** | 23 | 46.0 | 23 | 46.0 | |
| **Family history of diabetes** | - **Yes** | 22 | 44.0 | 22 | 44.0 | |
| | - **No** | 28 | 56.0 | 28 | 56.0 | |
| **Weight (kg)** | - **Mean ± SD** | 40.8±15.1 | | 39.5±10.7 | | U = 0.379 |
| | - **Min-Max** | 16-66 | | 20-60 | | P = 0.704 (>0.05) |
| | - **Median (IQ)** | 41.5 (27-52) | | 40.5 (29-49) | | |
| **Weight on Z score** | - **Mean** ± SD | 0.41 ± 0.32 | | 0.55 ± 0.66 | | U = 0.719 |
| | - **Median (Range)** | 0.74 (-1.65-2.76) | | 0.58 (-1.88-2.98) | | P = 0.524 (>0.05) |
| **Height (cm)** | - **Mean ± SD** | 140.9±22.5 | | 142.3±20.6 | | U = 0.324 |
| | - **Min-Max** | 104-185 | | 110-179 | | P = 0.746 (>0.05) |
| | - **Median (IQ)** | 139.5 (120-160) | | 143.5 (125-157) | | |
| **Height on Z score** | - **Mean** ± SD | 0.65 ± 1.11 | | 0.62 ± 0.89 | | U = 0.579 |
| | - **Median (Range)** | 0.98 (-2.33-2.55) | | 0.83 (-1.88-2.67) | | P = 0.844 (>0.05) |
| **Body mass index (kg/m2)** | - **Mean ± SD** | 21.4±7.6 | | 20.4±8.8 | | U = 1.02 |
| | - **Min-Max** | 10.4-46.6 | | 6-46 | | P = 0.306 (>0.05) |
| | - **Median (IQ)** | 20.1 (15-25.1) | | 17.5 (14-25) | | |
| **BMI on Z score** | - **Mean** ± SD | 0.83 ± 0.55 | | 0.67 ± 0.77 | | U = 0.906 |
| | - **Median (Range)** | 0.95 (-1.44-3.22) | | 1.11 (-2.22-3.15) | | P = 0.415 (>0.05) |

There was a very significant disparity between the serum levels of vitamin D and alkaline phosphatase in T1DM patients compared to controls (p-value <0.001). Blood calcium and phosphorus levels were substantially different between the control group and patients with type 1 diabetes (p-values of 0.045 and 0.007, respectively). The levels of serum parathyroid hormone in both the control group and the individuals with type 1 diabetes are identical **(Table 2)**.

**Table (2): Comparison between laboratory investigations in studied groups**

| Items | Cases (No=50) | Control (No=50) | Test of sig. p-value |
|---|---|---|---|
| **Serum vitamin D (ng/ml)** | 19.7±4.3 | 31.2±1.8 | **U = 5.89 P =0.00**(≤0.001)** |
| **Serum calcium (mg/dl)** | 9.4±0.95 | 8.9±0.78 | **t test = 2.73 P =0.007*(≤0.05)** |
| **Serum phosphorus (mg/dl)** | 5.04±0.88 | 5.6±1.2 | **U = 1.99 P =0.046*(≤0.05)** |
| **Serum ALP (IU/L)** | 255.3±8.4 | 193.9±8.6 | **U = 3.92 P =0.00**(≤0.001)** |
| **Serum PTH level(pg/ml)** | 36.4±5.6 | 35.4±6.3 | U = 0.314 P =0.754 (>0.05) |
| **HbA1c** | 8.3±1.1 | 4.5±0.51 | **t test = 24.02 P =0.00**(≤0.001)** |

*: Significant; **: High Significant.

There was a very significant difference (p-value <0.001) in the DEXA scan results between the two groups (**Table 3**). There was no statistically significant relationship between the results of DEXA scans and the anthropometric measures taken from the study populations. Furthermore, the DEXA scan (z-score) does not seem to have any correlation with the levels of calcium, phosphorus, and parathyroid hormone in either group. In the patient group, there was a strong correlation between serum alkaline phosphatase and DEXA scan results (p-value <0.001), while this is not the case in the control group. The results of the DEXA scan are strongly correlated with the blood vitamin D level of the patient group (P-value <0.05) (**Table 4)**.

**Table (3): Comparison between the two studied groups according to DEXA scan results (Z scores)**

| DEXA scan SD results | Patients (n=50) (n (%)) | Controls (n=50) (n (%)) | P |
|---|---|---|---|
| **DEXA scan Low (< −2 SD)** | 15 (30) | 4 (8) | **0.01*** |
| **Low normal (−1 to −2 SD)** | 20 (40) | 15 (30) | |
| **Normal (>−1 SD)** | 15 (30) | 27 (62) | |

DEXA, dual-energy X-ray absorptiometry. *: Significant.

**Table 4. Correlation between DEXA scan results and anthropometric and laboratory data in patient group.**

| Parameter | Patients with T1DM, N=50 | |
|---|---|---|
| | DEXA Scan | |
| | r | P value |
| **Weight** | -0.075 | 0.606 |
| **Height** | -0.135 | 0.345 |
| **Body mass index (kg/m2)** | 0.188 | 0.195 |
| **Vitamin D** | 0.137 | 0.016* |
| **Calcium** | -0.173 | 0.235 |
| **Phosphorus** | 0.019 | 0.899 |
| **Alkaline phosphatase** | 0.453 | 0.001** |
| **Parathyroid hormone** | 0.138 | 0.346 |
| **HbA1c** | 0.219 | 0.130 |

*:Significant; **: High Significant.

There was no statistically significant relationship between anthropometric, clinical, or blood vitamin levels in patients who have type 1 diabetes **(Table 5)**. The results of a DEXA scan are inversely connected to the duration of a patient's diabetes. Vitamin D levels in the blood have a favourable correlation with DEXA scan results**.**

**Table (5): Correlation of Vitamin D3 level, anthropometric, clinical measures and radiological measures in patient group.**

| | Patients with Type 1 diabetes | |
|---|---|---|
| **Parameter** | **Correlation with Serum Vitamin D Level** | **P-value** |
| **Clinical data** | | |
| **Weight (Kg)** | -0.084 | 0.794 |
| **Height (cm)** | -0.065 | 0.840 |
| **Body Mass Index (BMI)** | -0.346 | 0.269 |
| **Laboratory data** | | |
| **Serum Calcium(mg/dl)** | 0.395 | 0.202 |
| **Serum Phosphors (mg/dl)** | -0.135 | 0.674 |
| **Serum Alkaline Phosphatase (IU)** | 0.373 | 0.232 |
| **Serum Parathyroid Hormone Level (pg/ml)** | -0.162 | 0.614 |
| **HBA1c** | -0.065 | 0.840 |
| **Radiological data** | | |
| **DEXA scan results** | 0.241 | 0.016* |

## DISCUSSION

Possible reasons of reduced BMD connected to diabetes include vitamin D insufficiency, advanced glycation end products in bone collagen, inflammatory cytokines, hypercalciuria linked to glycosuria, diabetic microangiopathy with decreased blood flow to bone, and others. Actively managing diabetes mellitus, however, may protect bone health [(6)].

Among the children in our control group, 64% had normal levels of vitamin D, 20% had insufficiency, and 20% had a deficiency. The average blood vitamin D level was 31.2 (15-50) ng/ml. Vitamin D insufficiency was seen in 22% of the diabetic group, acceptable levels in 22%, and a mean blood vitamin D level of 19.7 (9-25) ng/ml in 56%.

In a similar vein, **Litchfield *et al.*** [(6)] discovered that the average vitamin D level of 74% of type 1 diabetic patients was a very low 20.02 ng/ml. Sixty percent of the participants had insufficient vitamin D levels, 14% had a deficit, and 26% had suitable levels; Furthermore, 58% of the type 1 diabetic patients had low blood vitamin D levels, with 40% having an inadequate level, 18% a deficiency, and 42% an adequate level. According to **Al-Zubeidi *et al.*** [(7)]. Nevertheless, **Azab *et al.*** [(8)] found that both the diabetes and control groups had low average vitamin D levels, measuring 24.7±5.6, and that there was no difference between the two groups.

In our research, the average calcium level in the healthy group was 9.4±0.95 mg/dl, whereas in the ill group it was 8.9±0.78 mg/dl. Furthermore, this confirmed our study's findings of statistically significant differences between the two categories. The control group had mean phosphorus levels of 5.6±1.2 mg/dl, while the sick group had 5.04±0.88 mg/dl.

**Hamed *et al.*** [(9)] reported that the average calcium level in the control group was 9.85±1.01 mg/dl, whereas in their patients it was 8.81±1.22 mg/dl. A statistically significant difference existed between the two sets of participants.

Twenty patients, or 40% of the total, had low normal BMD, whereas fifteen controls, or 30% of the total, had normal BMD; twenty-seven controls, or 62% of the total, had normal BMD. Within the patient group, the median DEXA score was -1.34±1.22 with an interquartile range of -1.4 ((-2.2) - (-0.3)).

Despite this, **Mosso *et al.*** [(10)] and **Onder *et al.*** [(11)] both found that 10% and 25% of the participants had low normal BMD, respectively. Twenty percent of children with type 1 diabetes had low BMD, whereas eighty percent had normal density. Results from DEXA scans showed significant differences between the control group and the T1DM patients in our study.

Further studies conducted by de **Souza *et al.*** [(12)] and **Joshi *et al.*** [(13)] shown that BMD was lower in the general population compared to children and adolescents with type 1 diabetes. Furthermore, in a study conducted by **Gunczler *et al.*** [(14)], 23 children in Venezuela who were not yet fully grown and had type 1 diabetes were examined. The children's average age was 9.5±2.2 years, which is similar to our results. Diabetic children had a noticeably decreased BMD of the lumbar spine compared to healthy controls. The average Z score for the lumbar spine in the patients was −0.89±1.2, whereas in the controls it was −0.27±0.68. While we found a statistically significant difference between healthy individuals and those with type 1 diabetes in BMD values. **Maggio *et al.*** [(15)] found no such difference.

Several factors, such as different BMD measuring methods, research designs, and patient selection criteria, are likely to blame for these contradictory findings. We found no statistically significant relationship between the sex of the patients and the results of the DEXA scans. Also, in Tehran, Iran, **Sayarifard *et al.*** [(16)] looked at 112 diabetic youngsters, 55 of them were boys and 57 women. In age, they ranged from four to fourteen years old. They agreed with our conclusions on this point. Our results are at odds with those of cross-sectional research in Paris, France, which examined 127 white children (73 males and 54 girls) with type 1 diabetes (ranging in age from 6 to 20) and found that although the BMD of control females was identical, it was much greater for diabetic girls.

**Heilman *et al.*** [(17)] found that lesser BMD was associated with male sex in children with diabetes, which contradicts our results. Since their sample consisted mostly of prepubertal males rather than girls, this makes sense. Since peak bone mass growth occurs

during puberty, the delayed pubertal pattern may account for the reduced BMD in diabetic boys.

Our study Concurring with **Heap *et al.*** [18], our patients had an average duration of diabetes of 7.91 2.36 years. The results of a DEXA scan demonstrated a substantial negative correlation with the duration of the illness. Although glucose toxicity may lead to net bone loss by enhancing osteoclast activity and impairing osteoblast function, research by **Gregory *et al.*** [19] shows that bone remodelling can be negatively affected by persistent hyperglycaemia and insufficient metabolic control.

Specifically, we found that average insulin dose was negatively related to average vitamin D and DEXA scan scores. Low BMD was associated with greater insulin dose, as shown by Torres-**Costoso *et al.*** [20] and **Eller-Vainicher *et al.*** [21]**,** as low mean vitamin D is associated with increased insulin demand and reduced insulin sensitivity.

In addition, the study found a strong correlation ($p < 0.001$) between the BMD of T1DM patients and blood alkaline phosphatase, a marker of bone turnover, suggesting that bone resorption is accelerated due to an imbalance in calcium and phosphorus. Consistent with previous studies, our findings show that bone turnover indices are elevated in type 1 diabetics [9].

The control group had an average HbA1c of 4.5% compared to 8.3% in the T1DM group, indicating considerably better glucose management ($p < 0.001$). Nonetheless, we found no statistically significant relationship between HbA1c and BMD. In a similar vein, **Hamed *et al.*** [9] discovered that 22 patients (61.1%) had poorly controlled HbA1c, with an average level of 9.14±1.9%. Furthermore, they could not uncover any correlation between the results of the DEXA scan and the control of blood sugar levels.

The studies done by **Sayarifard *et al.*** [16] and **Abd El Dayem *et al.*** [22] showed the exact opposite, a substantial negative correlation. A lack of relationships between HbA1c and BMD in certain studies may be explained by relatively strong metabolic control and a shorter duration of the condition. This is because HbA1c values only represent a short-term glycaemic control phase and are unlikely to reflect cumulative bone damage as determined by BMD.

We found a substantial association between vitamin D level and DEXA scan results, which is in agreement with **Ortiz *et al.*** [23] and **Mosso *et al.*** [10] who also found a positive relationship between DEXA scan findings and blood calcium and vitamin D.

## CONCLUSIONS

Significantly reduced BMD and lower vitamin D levels were observed in children and adolescents with type 1 diabetes compared to healthy controls, highlighting the detrimental impact of diabetes on skeletal health. Early routine monitoring of BMD and vitamin D supplementation are strongly recommended for diabetic pediatric patients to prevent long-term bone complications.

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