A comparative study of calcitonin and some minerals levels in type 2 diabetic patients

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Abstract: Calcitonin (CT) is a polypeptide hormone with 32 amino acids that is mostly secreted by the parafollicular "C" cells of the thyroid gland. Calcitonin inhibits bone resorption, which lowers blood calcium levels. Patients with type 2 diabetes are more susceptible to osteoporosis and decreased bone density because of poor calcitonin function or sensitivity, which may lead to abnormal calcium balance and bone metabolism. Determining the levels of calcitonin, calcium, phosphorus, and magnesium in individuals with type 2 diabetes and comparing them to a healthy control group was the aim of this study. A total eighty samples were collected involving forty samples from male patients with type 2 diabetes and forty samples from healthy men as the control group. Levels of calcitonin, calcium, magnesium, and phosphorus were measured. The study showed that while phosphorus and calcitonin levels were significantly lower in the diabetic group, calcium and magnesium levels were significantly greater in type 2 diabetes patients than in healthy people. This study demonstrated that diabetic patients exhibited decreased levels of calcitonin and phosphorus, along with increased levels of calcium and magnesium. These alterations may contribute to endocrine dysregulation associated with type 2 diabetes.

Keywords: Type 2 diabetes mellitus, Calcitonin, minerals.

1. Introduction

Chronic hyperglycemia is a symptom of type 2 diabetes mellitus (T2DM), a progressive disease marked by insulin resistance and pancreatic β-cell dysfunction. People who are genetically or acquired predisposed to insulin resistance and β-cell dysfunction are at risk for it, and environmental factors, sedentary lifestyles, and high calorie intake can make it worse [1]. When known as calcitonin. The C-cells of the thyroid gland release a hormone called CT, which has 32 amino acids [2]. By stimulating the release of calcitonin in response to an increase in blood calcium levels, hypercalcemia is avoided [3]. The body needs minerals and trace elements as vital micronutrients for proper operation [4]. These minerals such as calcium, phosphorous, magnesium, improve the action of insulin by activating insulin receptor sites. Particular roles are played by these trace elements in the

etiology and development of type 2 diabetes mellitus [5]. The body's mineral balance particularly that of calcium and phosphorus, is essential for maintaining healthy bones, neuron function, and muscular contraction [6]. Blood calcium and phosphorus levels are mostly controlled by the hormone calcitonin. By decreasing intestine absorption, increasing renal excretion, and blocking bone resorption, it lowers their levels [7]. Calcitonin disrupts insulin resistance in type 2 diabetics by modifying insulin signals in cells, reducing inflammation, controlling hepatic glucose synthesis, and enhancing muscular glucose uptake [8]. It also affects pancreatic function and insulin secretion by either raising the pancreatic beta cells' production of insulin, which impacts blood sugar regulation [9]. The aim of study, the relationship between calcitonin and some minerals in diabetic patients will be identified.

2. Methodology

a. Study design

A total eighty samples were collected between November 2024 and January 2025, involving forty samples from male patients with type 2 diabetes at Al-Hussein Teaching Hospital and forty samples from healthy men as the control group, all aged between 40 and 60 years. 5 mL of venous blood was collected from each participant. Using automated analyzers and standard laboratory kits, the levels of calcitonin, calcium, magnesium, and phosphorus were measured.

b. Blood sample

Following disinfection with 70% alcohol, 5 mL of venous blood were collected from both the patient and control groups using sterile syringes. The samples were divided into two portions: 2 mL were transferred into EDTA tubes for hematological analysis, while the remaining 3 mL were placed in gel tubes. The gel tubes were left to stand at room temperature for 15 minutes to allow clot formation, after which they were centrifuged at 3000 rpm for 10 minutes. The separated serum was collected and used for the evaluation of mineral content and hormone levels.

c. Hemoglobin A1c (HbA1c) parameter

A volume of 10µL from each sample was transferred using a disposable pipette into a sample diluent tube. The prepared mixture was then applied onto test and insert cards, which were subsequently loaded into the Getein1180 device for the measurement of HbA1c levels.

d. Hormone and Biochemical Parameter

A volume of 100µL of serum was transferred to the calcitonin kit (SNIB, China) using a sample transfer pipette for analysis with the MAGLUM 800 instrument. For the assessment of mineral parameters, a Semi-Auto Chemistry Analyzer

was used. Specifically, 10µL of serum was utilized to measure calcium using a calcium kit (Linear Chemical S.L., Spain), 10µL for magnesium using a magnesium kit, and another 10µL for phosphorus using a phosphorus kit (Linear Chemical S.L., Spain**).**

e. Statistical analysis

IBM SPSS Statistics version 20 (Statistical Package for the Social Sciences) was used to do the statistical analysis. To evaluate variations in rates between groups, the chi-square (χ^2) test was used. Statistical significance was established at a level of $p < 0.01$.

3. Results and Discussion

a. Glycated Hemoglobin A1c (HbA1c)

As shown in Figure (1), hemoglobin A1c (HbA1c) levels were significantly higher in the diabetic group compared to the healthy group ($p < 0.05$). The healthy group recorded a mean HbA1c level of 4.90 whereas diabetic patients exhibited a markedly elevated mean level of 10.77.

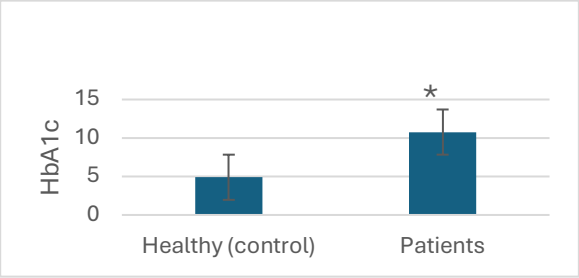

Figure 1: The variation in HbA1c levels healthy and diabetic groups. P value (0.0001).

b. Calcitonin

As shown in Figure (2), calcitonin levels were significantly lower in the diabetic group compared to the healthy group ($p < 0.05$). The healthy group recorded a mean calcitonin level of 6.12, while the diabetic patients exhibited a markedly reduced level of 1.48.

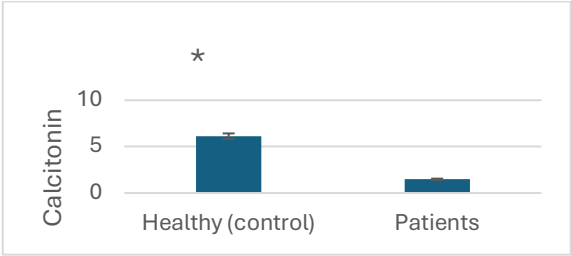

Figure 2: The variation in calcitonin levels between healthy and diabetic groups. P value (0.0001).

c. Calcium

As shown in Figure (3), calcium levels were significantly higher in the diabetic group compared to the healthy group ($p < 0.05$). The highest recorded calcium level was 11.88 in diabetic patients, whereas the lowest level was 9.21 in healthy group**.**

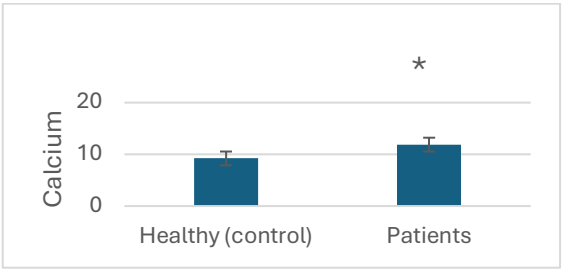

Figure 3: The variation in calcium levels between healthy and diabetic groups. P value (0.0003)

d. Phosphorus

As shown in Figure (4), phosphorus levels were significantly lower in the diabetic group compared to the healthy group ($p < 0.05$). The diabetic group recorded a mean phosphorus level of 1.64, indicating a deficiency, while the healthy group showed a higher level of 3.12.

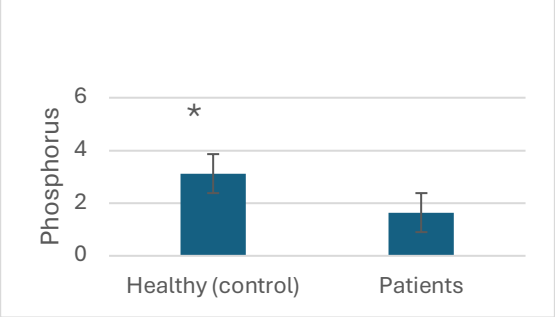

Figure 4: The variation in phosphorus levels between healthy and diabetic groups. P value (0.0001)

e. Magnesium

As shown in Figure (5), magnesium levels were significantly higher in the diabetic group compared to the healthy group ($p < 0.05$). The healthy group recorded a mean magnesium level of 2.18, whereas the diabetic group exhibited a markedly elevated level of 3.76.

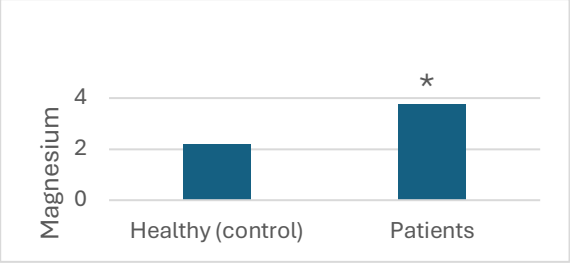

Figure 5: The variation in magnesium levels between healthy and diabetic groups. P value (0.0002**)**

The hallmark of type 2 diabetes mellitus (T2DM) is consistently high blood glucose levels brought on by peripheral insulin resistance and decreased insulin synthesis [10]. High blood sugar interferes with the hormone calcitonin, which affects insulin secretion and results in insulin resistance and glucose intolerance [11]. Furthermore In type 2 diabetes, oxidative stress, fat-derived inflammatory cytokines, adipokines, and hyperglycemia all work together to impair osteocyte function and mineral imbalance [12]. Patients with type 2 diabetes mellitus in the current study had significantly higher HbA1c values than those in the control group. Because of insulin resistance or β-cell malfunction, T2DM patients frequently have chronic hyperglycemia and poor glycemic control, which is reflected in this increase [13].

According to recent research, persons with type 2diabetes may have much lower calcitonin levels. Thyroid C-cells release calcitonin, which is crucial for increasing renal calcium excretion and preventing bone resorption. According to Kapitza[14] and Sgambato[15], reduced calcitonin levels in type 2 diabetes have been linked to autonomic dysfunction and chronic inflammation, which may further compromise the hormonal regulation of mineral metabolism. Lower calcitonin may be an early sign of endocrine dysregulation in diabetic patients, according to Faloona & Unger [16] who showed that decrease might worsen hypercalcemia and lead to bone fragility. Studies have shown that

long-term hyperglycemia may impair thyroid C-cell function, which would decrease the release of calcitonin, a hormone that regulates phosphorus metabolism and reduces blood calcium levels.

Based on current evidence, patients with type 2 diabetes also have increased serum calcium levels. Increased parathyroid hormone (PTH) activity and diminished calcitonin inhibitory regulation, together with poor renal clearance from diabetic nephropathy, may all contribute to this hypercalcemia. Doe [17] and Smith [18] concur that recent data indicate that calcium metabolism abnormalities are multifactorial, resulting from hormonal imbalances and renal dysfunction, and may put patients at risk for vascular calcification and bone demineralization.

The current findings revealed that the diabetic group exhibited significantly higher serum magnesium levels compared to the healthy control group. This unexpected elevation may be attributed to reduced renal clearance associated with diabetic nephropathy. In addition, studies by Kim [19] and Wang [20] indicated that chronic hyperglycemia and the accompanying inflammatory milieu may interfere with intracellular magnesium transport, thereby increasing extracellular magnesium concentrations. These observations underscore the complexity of magnesium regulation in T2DM and highlight the necessity for individualized mineral assessment in diabetic patients. There is a tendency for reduced serum phosphorus levels in T2DM, according to research on phosphorus metabolism. The action of insulin, which encourages phosphate's intracellular uptake and reduces its extracellular content, is one contributing element. Furthermore, because diabetic nephropathy affects tubular reabsorption, it might result in renal phosphate wasting , this result was agreed with Garcia [21] and Martinez [22], who investigated these changes in phosphate homeostasis have an impact on energy metabolism as well as bone health. AlBayati found that long-term hyperglycemia may impair thyroid C-cell function, which would decrease the release of calcitonin, a hormone that regulates

phosphorus metabolism and reduces blood calcium levels. Patients with type 2diabetes may have changes in their calcium and phosphorus balance due to a decrease in calcitonin. [23].

These results clearly show that type 2 diabetes disrupts the mineral and hormonal balance**.**

4. Conclusion

According to this study, low calcitonin may result in higher calcium and lower phosphorus levels, which could impact insulin sensitivity and raise glycated hemoglobin. Another possible

explanation for elevated magnesium is a compensatory reaction to other diabetes-related metabolic abnormalities

Credit Author Contributions Statement:

A.M.M. designed the study, participated in data collection, and drafted the manuscript.

W.A.N. contributed to the data interpretation, reviewed the literature, and revised the manuscript critically for important intellectual content.

M.A.M. performed statistical analysis, assisted in methodology validation, and contributed to the final approval of the version to be published.

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Data availability statement:

This study does not include new data. All data used in this research are available in previously published articles cited in the references.

Conflict of Interest Statement:

The authors declare that there is no conflict of interest regarding the publication of this paper**.**

Ethical Approval (for studies in humans/animals)

This study involving human participants was approved by the Health Directorate of Al-Muthanna, Approval No. [3347]. All participants provided informed consent prior to sample collection.

Informed Consent

Written informed consent was obtained from all individual participants included in the study.

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