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Research Article

The Impact of Serum Ghrelin on Body Mass Index in Children with Type1 Diabetes Mellitus

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Abstract:

Background: Insulin produced by pancreatic β cells ceases in people with type1 diabetes mellitus, an endocrine disease caused by autoimmune damage. It can cause weight loss. Energy balance controls the secretion of ghrelin, a peptide hormone made by entering endocrine cells found in the oxyntic glands of the stomach fundus.

Objectives: This study aimed to investigate the relationship between serum ghrelin and body mass index in newly diagnosed children with type 1 diabetes mellitus.

Methods: The study involved 120 pediatric participants aged 2-14 years conducted from February to May 2024. They were divided into four groups: group 1 contains 40 lean patients with type 1 diabetes mellitus and a body mass index ≥85 percentile, group 2: 40 underweight patients with type1 diabetes mellitus and a BMI ≥5 percentile, group 3: 22 healthy lean controls with a body mass index <85 percentile, group 4: 18 healthy underweight controls with a body mass index ≥5 percentile. The study measured fasting serum glucose, ghrelin, and HbA1c. **Results:** Diabetic patients had significantly lower ghrelin levels compared to controls. Underweight diabetics had higher mean HbA1c and fasting blood glucose compared to lean and underweight controls.

Conclusion: In type 1 diabetes mellitus patients, ghrelin levels are lower than in controls. Ghrelin correlates positively with FBG, HbA1c, weight, and height in healthy control participants (lean and underweight) and age in lean control subjects. No association was found between ghrelin and the patient (T1DM).

Keywords: Body mass index; Fasting blood glucose; Ghrelin; Glycated hemoglobin; Type 1 diabetes mellitus.

Introduction

Type 1 diabetes mellitus (T1DM) is a chronic metabolic condition. Genetically vulnerable people are caused by an autoimmune attack and the loss of functioning pancreatic β-cells that produce insulin(1). Weight loss, polyuria, thirst, and eyesight problems are among the signs of children with Type 1 diabetes mellitus who have diabetes. Certain cases of hyperosmola and more severe forms of diabetic ketoacidosis may cause coma. Regrettably, most of the symptoms are mild. Still, over time, they can cause harm to numerous organs or even their failure, resulting in irreversible impairments such as blindness, amputation, stroke, and, finally, death. Insulin-dependent diabetes, previously known as type 1 diabetes, can attack anyone at any age. Still, it most commonly affects children and adolescents (2). Ghrelin, often referred to as the "hunger hormone," plays a significant role in weight regulation by influencing appetite and energy balance. It acts primarily through the growth hormone secretagogue receptor type 1a (GHS-R1a), stimulating food intake and promoting adipogenesis. While ghrelin's orexigenic effects are well-documented, its role in weight control is complex and multifaceted, stimulating food intake and promoting adipogenesis. Ghrelin stimulates appetite by activating hypothalamic neurons that promote hunger while inhibiting those that suppress it (3). It also enhances dopaminergic signaling, which may increase the reward associated with food intake (3). The acylated form of ghrelin is crucial for its binding to GHS-R1a, linking calorie intake to energy homeostasis (4). The physiological significance of ghrelin in energy balance is debated; for instance, ghrelin-deficient rodents do not always exhibit anorexia or weight loss (4). Ghrelin, a 28-amino acid peptide, is predominantly produced in the small intestine and stomach, with very minor amounts produced by the kidneys, placenta, and pancreas. There are two main types of ghrelin in circulation: desacyl ghrelin (DAG) and acyl ghrelin (AG) (5). Ghrelin O-acyltransferase (GOAT), the membrane-bound enzyme, couples proghrelin with an octanoic acid at the serine-3 residue during posttranslational processing to generate AG(6). Growth hormone (GH) secretagogue receptor type 1a (GHS-R1a) is an

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endogenous ligand that increases GH secretion (7). Ghrelin is one of these ligands. A GPCR receptor is called GHS-R1a (8). Ghrelin, a peptide hormone predominantly produced in the stomach, plays a multifaceted role in energy balance and metabolism, particularly in glucose metabolism. The metabolic effects of serum ghrelin on glucose homeostasis are complex and can vary based on physiological context, including conditions such as obesity, diabetes, and metabolic syndrome. Key Metabolic Effects of Serum Ghrelin on Glucose Metabolism by Regulation of Insulin Secretion

Ghrelin influences insulin secretion from the pancreas. It can inhibit insulin release, resulting in increased blood glucose levels. Studies suggest that elevated levels of ghrelin can contribute to insulin resistance, potentially exacerbating the glucose intolerance seen in metabolic disorders such as type 2 diabetes (21). Ghrelin has been linked to reduced glucose utilization by peripheral tissues. It appears to diminish the uptake of glucose by cells, which aligns with its overall function of conserving energy stores during fasting periods (22). While ghrelin is primarily known for its appetite-stimulating effects, it also acts on the hypothalamus and other brain centers to modulate energy expenditure. This modulation can indirectly influence glucose metabolism by altering overall energy balance and caloric needs (23). Ghrelin significantly influences glucose metabolism and insulin secretion in patients with type 1 diabetes mellitus. Its tendency to inhibit insulin secretion and promote higher blood glucose levels can pose challenges for glycemic control (24). Ghrelin levels in obese children have been associated with various components of metabolic syndrome, including insulin resistance and dyslipidemia. Studies demonstrate that the relationship between ghrelin levels and body mass index (BMI) in pediatric populations is relevant, with ghrelin acting as a potential biomarker for understanding obesity-related metabolic disorders in children (25).

A critical area of research understands the relationship between ghrelin levels and body weight which can help manage diabetes more effectively and improve overall health outcomes.

Investigating this relationship could reveal potential therapeutic targets for regulating appetite and metabolism in pediatric patients, ultimately leading to tailored interventions that promote healthier growth patterns and better glycemic control. Identifying specific ghrelin signaling pathways may also provide insights into the mechanisms underlying weight fluctuations in children with type 1 diabetes. Understanding how these pathways interact with other hormonal and metabolic factors will be essential for developing comprehensive management plans that address both diabetes control and weight maintenance in this vulnerable population. Such an integrated approach could not only improve clinical outcomes but also empower families with knowledge and tools to foster healthier lifestyles, ultimately contributing to the long-term well-being of children living with type 1 diabetes.

Therefore, the present study aimed to examine the relationship between weight (as measured by body mass index) and some metabolic hormones (Ghrelin) in newly diagnosed children with type 1 diabetes to shed light on the weight maintenance mechanism in these patients.

Patients and Methods

This case-control study included 120 child T1DM newly diagnosed aged between (2 and 14) years at Welfare Teaching Hospital, Medical City, and in addition to The Central Child Teaching Hospital of Pediatrics, Al-Karkh District, Baghdad, Iraq, from the first of February to the end of May 2024. Every individual provided an informed consent. Ethical Committee at the Department of Biochemistry, College of Medicine, University of Baghdad. Approved the study. The participants were classified into the following groups: the first group consisted of 40 pediatrics with lean T1DM, and the second group consisted of 40 children with underweight type 1 diabetes mellitus. The third group numbered 22 children with lean healthy as control. The fourth group consists of 18 children who are underweight and healthy as controls.

Inclusion criteri:

Patients with type 1 diabetes mellitus newly diagnosed

Exclusion criteria:

1) Diabetes type 2.
2) Patients are using dietary supplements.
3) Patients with Type 1 diabetes complications such as neuropathy, retinopathy, or nephropathy.
4) Thyroid disorders and celiac disease

Blood samples

Five milliliters of peripheral blood were drawn from the patient; two milliliters were placed in an EDTA (ethylenediaminetetraacetic acid) anticoagulant tube to measure HbA1c, and the remaining three milliliters were placed in a gel tube. Blood samples were allowed to coagulate for 15-30 minutes before centrifugation at 3000 rpm for 10 minutes. This is separated into two tubes. The separated serum samples were kept in aliquots at -20°C until the glucose and ghrelin levels were measured.

Measurement of Biochemical Parameters: The following biomarkers were measured in the blood: Glucose measured by spectrophotometer and HbA1c on the Cobas c 111 system, and serum ghrelin was determined using an enzyme-linked immunosorbent assay (ELISA)and anthropometric measurements, including body mass index (BMI) by used (centers for disease control and prevention growth chart) CDC growth chart for children measured body mass index.

Statistical analysis

Data analysis was performed with SPSS software version 16.0. Data were presented as Mean ± SEM to compare serum levels based on analysis of (ANOVA) for each study. Pearson correlation

analysis was performed to see if there was a significant association between the parameters. The alpha level for statistical significance was established at a threshold of $p< 0.05$.

Results

For the lean control and underweight groups, the average age was 9.73 ± 0.80 years and 9.39 ± 0.62 years, respectively. The underweight DM group had a lower average age of 8.03±0.58 years. Lean DM has an average age of 9.33±0.31 years. ($P= 0.108$, meaning the age distribution was not statistically significant. Weights differ significantly between groups ($p< 0.0001$). The mean weight of the underweight and lean control groups is 34.55±2.67kg and 28.72±2.11kg, respectively. The weights of the underweight and lean T1DM groups were, (24.54± 1.75 kg) and (37.28± 1.47) kg, respectively, (P=0.05) indicating that height was not statistically significant. Table 1 shows that the groups' mean heights were very constant, with the lean and underweight control group measuring (133.23± 4.45cm) and (137.33± 4.12cm), respectively, and the lean and underweight T1DM group measuring (135.15± 2.10cm) and (125.97± 3.98cm), respectively.

Table 1: The mean (±SEM) values for weight and age of diabetes mellitus children and their controls

	Age/ year		
	Control n=40	Patient n=80	*P* value
Lean	9.73± 0.80	9.33± 0.31	
Underweight	9.39± 0.62	8.03± 0.58	*P*=0.108
Weigh/ Kg			
Lean	34.55± 2.67	37.28± 1.47	
Underweight	28.72± 2.11	24.54± 1.75	*P*<0.0001
Height/cm			
Lean	133.23± 4.45	135.15± 2.10	
Underweight	137.33± 4.12	125.97± 3.98	*P*=0.04

In the present study, there were substantial differences in BMI among the groups ($P< 0.001$) Tables 1&2. The lean and underweight control groups had mean percentile scores of (70.82± 2.23). They were shown to be significantly different from three groups: underweight control, underweight DM, and lean DM. The lean DM group had a significantly higher mean percentile score (85.67±1.53) than the underweight, underweight DM, and lean control groups. In the underweight control group's mean percentile, the results differed considerably from the lean control and DM groups. The underweight DM group had a substantially higher mean percentile result (19.15±3.68) than the lean control and lean DM groups.

Table 2: The mean (±SEM) body mass index values for control and diabetes patients

	BMI Percentile		
	Control n=40	Patient n=80	*P* value
Lean	70.82± 2.23	85.67± 1.53	
Underweight	21.44± 4.14	19.15± 3.68	*P*<0.001

In the present study, HbA1c in underweight patients has a mean (11.77± 0.32) %. In contrast, the lean control and underweight control groups had significantly lower mean HbA1c% values of (5.16±0.04) and (5.16± 0.05), respectively. The Lean DM group had a mean FBG of 214.33 ± 14.93 mg/dL. Similarly, the underweight DM group had an average FBG of 213.35 ±18.92 mg/dL. These values are considerably higher than those observed in the lean control and underweight control groups, with mean FBG levels of 86.63 ±1.70 and 86.99 ± 1.64 mg/dL, respectively. Ghrelin levels in the control group (lean and underweight) average (4.51± 0.38ng/ml) and (5.10± 0.47ng/ml), respectively. Patients with diabetes mellitus (lean and underweight) had significantly lower ghrelin levels (0.99±0.08 vs. 0.93±0.07 ng/ml). The difference in ghrelin levels was extremely significant (P<0.000001) as shown in Table 3. The research indicates significant differences in ghrelin levels among the four groups.

Table 3: The mean (±SEM) glycated hemoglobin and glucose values in controls and diabetic patients

			HbA1C%
	Control n=40	Patient n=80	*P* value
Lean	5.16± 0.04	11.40± 0.25	
Underweight	5.16± 0.05	11.80± 0.32	*P*<0.0001
Glucose (mg/dl)			
Lean	86.63± 1.70	214.33± 14.93	
Underweight	86.99±1.64	213.35± 18.92	*P*<0.0001
Ghrelin (ng/ml)			
Lean	4.51± 0.38	0.99± 0.08	
Underweight	5.10± 0.47	0.93± 0.07	*P*<0.0001

There is no association between ghrelin and T1DM subjects, but there is a correlation between it and underweight as well as lean controls. Positive correlations between ghrelin and FBG (r = 0.648, p = 0.0037) and HbA1c (r = 0.696, p = 0.0013) indicated that underweight control people with higher ghrelin levels also have higher FBG and HbA1c levels. Other significant relationships

between ghrelin and factors like height and weight include positive correlations in lean and underweight controls (r = 0.582, p = 0.0045) and weight (r = 0.572, p = 0.0054). Figure 1 showed a substantial positive correlation (r=0.65) between ghrelin and serum fasting glucose (P<0.004) in the underweight control group of subjects.

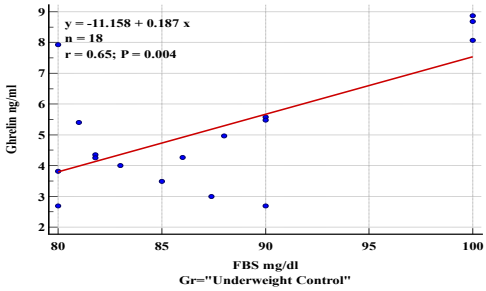

Figure 1: The connection between ghrelin and fasting glucose serum (FBG) in the underweight control group.

Figure 2 demonstrated a substantial positive connection (r = 0.70) between Ghrelin and HbA1C in the underweight control group (p<0.001).

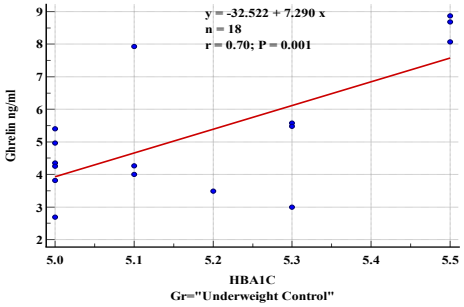

Figure 2: Association of ghrelin and HbAlc in the underweight control group.

Figure 3 demonstrated a substantial positive connection (r=0.72) between ghrelin and weight in the underweight control group (P<0.001).

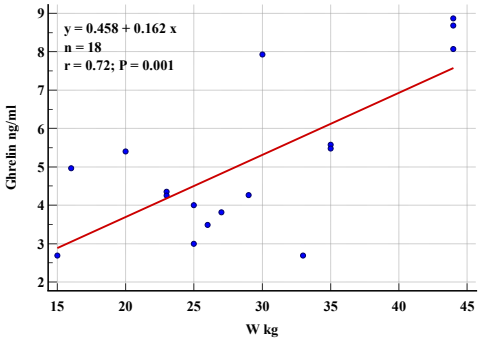

Figure 3: The association between Gherlin and weight in the underweight control group

Discussion

In the current investigation, the influence of age was minimized by selecting control group ages that were similar to those of the patients. It was supported by the non-significant results obtained. This observation matched the findings of Marcovecchio et al. (2024), who discovered no significant variations in age (9). Also, in the current study, there was a statistically significant difference (P<0.0001) in weight across the groups (lean DM and lean healthy) versus (underweight DM and underweight healthy).

The first week of type 1 diabetes in children is marked by a loss in both total body and muscle weight. This occurs because the body cannot produce insulin, which is required for the body's cells to convert glucose into energy. As a result, there is a decrease in ATP production and a breakdown of protein and fat (10). In contrast, there was no significant difference in height across the four groups (P = 0.05). This conclusion is consistent with an earlier work by Polkowska et al. (2016). They observed that children with newly diagnosed diabetes had considerably lower body weight but no significant differences in height (11). Another study that did not agree discovered significant variations in height (12). The body mass index percentile scores in (the lean healthy and lean DM) groups are considerably higher than (the underweight healthy and underweight DM groups). This is consistent with the earlier study of (13). Furthermore, diabetes patients who are skinny and underweight have considerably higher serum HbA1c readings than healthy people who are similarly lean and underweight. Similarly, lean and underweight diabetics had significantly higher serum glucose levels than healthy lean and underweight individuals. These results are comparable to results from another study which discovered that the lack of glycemic control was the cause of the significantly increased HbA1c levels. HbA1c is an accurate measure of long-term glucose exposure and is required for determining the patient's prediagnostic state of hyperglycemia (14). Also, another study, found that individuals who had just received their diagnosis had the greatest HbA1C and blood glucose values (15). The enteroendocrine cells in the stomach are the main source of the peptide hormone ghrelin, whereas the pancreas, brain, and small intestine also emit tiny amounts of this hormone. Many refer to it as the "hunger hormone" (16).

Also, the current investigation demonstrated increased serum ghrelin levels in healthy participants compared to diabetes mellitus patients, which accords with previous findings by (17) which found that plasma ghrelin was lower in diabetic individuals compared to individuals with good long-term glycemic control. According to Lopes et al. in2023, ghrelin may influence glucose homeostasis by promoting stomach emptying and inhibiting insulin synthesis stimulated by glucose. This phenomenon may assist in explaining how a decline in ghrelin after a meal leads to a rise in insulin levels (18). Ghrelin influences insulin release from pancreatic beta cells. Insulin secretion decreases, and blood glucose levels rise. Ghrelin suppresses insulin production in β cells by inhibiting Ca^{2+}andK^{+} channels that activate during glucose metabolism.

People with diabetes and obese people have lower plasma ghrelin levels than lean people. It is plausible to assume that this is a controlled reaction to decreased GHR. Inactivity, which promotes fat.
Further research showed that ghrelin levels can be adjusted to combat diabetes and obesity. Techniques for reducing insulin levels are under investigation because they drop when ghrelin is present (19). Ghrelin is positively associated with increasing FBG and HbA1c levels, indicating a potential role in the glucose metabolic process; however, no correlation was found between Ghrelin and T1DM patients. Ghrelin's production of GLP-1 reduces β-cell function and impairs glucose tolerance, as demonstrated by ghrelin infusion studies (20).

Limitation

limitations including small sample sizes, lack of non-diabetic controls, confounding insulin therapy effects, age/disease duration variability, and short-term follow-up.

Conclusion

In children with Type I diabetes mellitus, ghrelin levels were considerably lower than healthy controls. Ghrelin was also positively associated with FBG, HbA1c, weight, and height in healthy control participants (lean and underweight) and age in lean control subjects. No link was found between ghrelin and the patient (T1DM) because of a defect in insulin.

Authors' declaration:

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for re-publication attached to the manuscript. Authors sign on ethical consideration's approval-Ethical Clearance: The project was approved by the local ethical committee (at the Department of Biochemistry, College of Medicine, University of Baghdad) according to code number (278) on (29/ 9/ 2024).

Conflict of interest: None

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Authors' contributions:

Study conception & design: Safa Ahmed Ayash & Hedef Dhafir El-Yaseen. Literature search: Hedef Dhafir El-Yaseen & Safa Ahmed Ayash). Data acquisition: Safa Ahmed Ayash) Data analysis & interpretation: (Safa Ahmed Ayash, Hedef Dhafir El-Yaseen & Munib A. Kadhim Alzubaidi). Manuscript preparation: (Safa Ahmed Ayash). Manuscript editing & review: (Safa Ahmed Ayash, Hedef Dhafir El-Yaseen & Munib A. Kadhim Alzubaidi).

References:

1. Nekoua MP, Alidjinou EK, Hober D. Persistent coxsackievirus B infection and pathogenesis of type 1 diabetes mellitus. Nat Rev Endocrinol. 2022;18(8):503–16. https://doi.org/10.1038/s41574-022-00688-1 . Epub 2022 Jun 1. PMID: 35650334; PMCID: PMC9157043.

2. Mobasseri M, Shirmohammadi M, Amiri T, Vahed N, Fard HH, Ghojazadeh M. Prevalence and incidence of type 1 diabetes in the world: a systematic review and meta-analysis. Heal Promot Perspect. 2020;10(2):98. https://doi.org/10.34172/hpp.43143. PMID: 32296622; PMCID: PMC7146037.

3. Mihalache L, Gherasim A, Niţă O, Ungureanu MC, Pădureanu SS, Gavril RS, et al. Effects of ghrelin in energy balance and body weight homeostasis. Hormones [Internet]. 2016;15(2):186–96. Available from: https://doi.org/10.14310/horm.2002.1672

4. Solomou S, Korbonits M. The role of ghrelin in weight-regulation disorders: Implications in clinical practice. Hormones. 2014;13(4):458–75. https://doi.org/10.14310/horm.2002.1551 . PMID: 25555181.

5. NoorAldeen ZE, EL-Yassin HD, Al Naddawi MN. Suppression of Insulin Secretion by Ghrelin and The Deterioration of Glucose Tolerance in Healthy Children. J Fac Med Baghdad [Internet]. 2013 Jul 1;55(3 SE-Articles):254–7. Available from: https://doi.org/10.32007/jfacmedbagdad.553626

6. Rhea EM, Salameh TS, Gray S, Niu J, Banks WA, Tong J. Ghrelin transport across the blood–brain barrier can occur independently of the growth hormone secretagogue receptor. Mol Metab. 2018;18:88–96 https://doi.org/10.1016/j.molmet.2018.09.007 . Epub 2018 Sep 24. PMID: 30293893; PMCID: PMC6308033.

7. Razak Abdl Ghani ZA, Mukhtar RS, Fadhel MA, Turki KM. Impact of weight loss achieved through gastric sleeve surgery with circulating level of ghrelin hormone in obese Iraqi subjects. J Fac Med Baghdad [Internet]. 2015 Apr 5;57(1 SE-Articles):50–3. https://doi.org/10.32007/jfacmedbagdad.571308

8. Ramasamy I. Physiological Appetite Regulation and Bariatric Surgery. J Clin Med. 2024;13(5):1347. https://doi.org/10.3390/jcm13051347 . PMID: 38546831; PMCID: PMC10932430.

9. Marcovecchio ML, Hendriks AEJ, Delfin C, Battelino T, Danne T, Evans ML, et al. The INNODIA Type 1 Diabetes Natural History Study: a European cohort of newly diagnosed children, adolescents and adults. Diabetologia. 2024;67(6):995–1008. https://doi.org/10.1007/s00125-024-06124-5 . Epub 2024 Mar 22. PMID: 38517484; PMCID: PMC11058619.

10. Keşim DA, Kelle M, Aşır F, Kaya HK, Diken H, Gökdemir GŞ, et al. Investigation of the relationship between plasma ghrelin levels and muscle atrophy in experimental diabetic rats. Pol J Vet Sci. 2024;279–88. Polish Journal of Veterinary Sciences https://doi.org/10.24425/pjvs.2024.149358

11. Polkowska A, Szczepaniak I, Bossowski A. Assessment of serum concentrations of ghrelin, obestatin, omentin-1, and apelin in children with type 1 diabetes. Biomed Res Int. 2016;2016(1):8379294. https://doi.org/10.1155/2016/8379294 . Epub 2016 Jan 19. PMID: 26904686; PMCID: PMC4745415.
12. Shaikh I, Ibrahim MN, Laghari T, Chachar S, Riaz M, Ahmed SH. Comparison of Body Composition Bio Electrical Impedance Analysis of Type-1 Diabetes vs. Non-Diabetes in Children and Adolescent. Liaquat Natl J Prim Care. 2024; https://doi.org/10.37184/lnjpc.2707-3521.6.9
13. De Keukelaere M, Fieuws S, Reynaert N, Vandoorne E, Kerckhove K Vande, Asscherickx W, et al. Evolution of body mass index in children with type 1 diabetes mellitus. Eur J Pediatr. 2018;177:1661–6. https://doi.org/10.1007/s00431-018-3224-9 . Epub 2018 Aug 9. PMID: 30091111.
14. Rahman MS, Hossain KS, Das S, Kundu S, Adegoke EO, Rahman MA, et al. Role of insulin in health and disease: an update. Int J Mol Sci. 2021;22(12):6403. https://doi.org/10.3390/ijms22126403 . PMID: 34203830; PMCID: PMC8232639.
15. Ochocińska A, Wysocka-Mincewicz M, Świderska J, Cukrowska B. Selected serum markers associated with pathogenesis and clinical course of type 1 diabetes in pediatric patients—The effect of disease duration. J Clin Med. 2023;12(6):2151. https://doi.org/10.3390/jcm12062151 . PMID: 36983153; PMCID: PMC10051659.
16. Reed J, Bain SC, Kanamarlapudi V. The Regulation of Metabolic Homeostasis by Incretins and the Metabolic Hormones Produced by Pancreatic Islets. Diabetes, Metab Syndr Obes. 2024;2419–56. https://doi.org/10.2147/DMSO.S415934 . PMID: 38894706; PMCID: PMC11184168.
17. Li J, Huang P, Xiong J, Liang X, Li M, Ke H, Chen C, Han Y, Huang Y, Zhou Y, Luo Z, Feng D, Chen C. Serum levels of ghrelin and LEAP2 in patients with type 2 diabetes mellitus: correlation with circulating glucose and lipids. Endocr Connect. 2022 May 27;11(5):e220012. https://doi.org/10.1530/EC-22-0012 . PMID: 35521798; PMCID: PMC9175609.
18. Lopes KG, Silva VL da, Lopes F de AM, Bouskela E, Souza M das GC de, Kraemer-Aguiar LG. Ghrelin and glucagon-like peptide-1 according to body adiposity and glucose homeostasis. Arch Endocrinol Metab. 2023;67(4):e000611. https://doi.org/10.20945/2359-3997000000611 . PMID: 37252699; PMCID: PMC10665067.
19. Picciotto S. Regulation of Ghrelin: A Possible Treatment Option for Obesity and Diabetes. Sci J Lander Coll Arts Sci. 2015;8(2):8. Retrieved from https://touroscholar.touro.edu/sjlcas/vol8/iss2/8
20. Özcan B, Delhanty PJD, Huisman M, Visser JA, Neggers SJ, van der Lely AJ. Overweight and obesity in type 1 diabetes is not associated with higher ghrelin concentrations. Diabetol Metab Syndr. 2021;13:1–7. https://doi.org/10.1186/s13098-021-00699-4 . PMID: 34294136; PMCID: PMC8296697.
21. Leinonen T, Kesäniemi YA, Hedberg P, Ukkola O. Serum ghrelin and prediction of metabolic parameters in over 20-year follow-up. Peptides. 2016;76:51–6. https://doi.org/10.1016/j.peptides.2015.12.002 . Epub 2015 Dec 22. PMID: 26721207.
22. Young ER, Jialal I. Biochemistry, ghrelin. 2019; In: StatPearls. StatPearls Publishing, Treasure Island (FL); 2023. PMID: 31613472.
23. Lv Y, Liang T, Wang G, Li Z. Ghrelin, a gastrointestinal hormone, regulates energy balance and lipid metabolism. Biosci Rep. 2018 Oct;38(5). https://doi.org/10.1042/BSR20181061 . PMID: 30177523; PMCID: PMC6153372.
24. Mani BK, Shankar K, Zigman JM. Ghrelin's Relationship to Blood Glucose. Endocrinology [Internet]. 2019 May 1;160(5):1247–61. Available from: https://doi.org/10.1210/en.2019-00074
25. Haddawi KH, Al-Ziaydi AG, Al-Kathem Al-Khalidi FA. The role of adipokines and ghrelin in interactions and clinical implications in childhood obesity. J Educ Health Promot. 2024;13:40. https://doi.org/10.4103/jehp.jehp_972_23 . PMID: 38545313; PMCID: PMC10968273.

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هرمون الغريلين في مصل الأطفال المصابين بمرض السكري من النوع الأول

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[2]فرع طب الاطفال،كلية الطب، جامعة بغداد، بغداد ، العراق.

ملخص: يتوقف إنتاج الأنسولين بواسطة خلايا بيتا البنكرياسية عند الأشخاص المصابين بداء السكري من النوع الأول، وهو مرض غدي صماء يحدث بسبب تلف المناعة الذاتية. يمكن أن يسبب فقدان الوزن. يتحكم توازن الطاقة في إفراز هرمون الغريلين، وهو هرمون ببتيدي يتم تصنيعه عن طريق دخول الخلايا الصماء الموجودة في الغدد المؤكسدة لقاع المعدة.
الهدف: تهدف هذه الدراسة إلى التحقيق في العلاقة بين هرمون الغريلين في المصل ومؤشر كتلة الجسم لدى الأطفال الذين تم تشخيصهم حديثًا بمرض السكري من النوع الأول.
الطريقة: شملت هذه الدراسة 120 فردًا من الأطفال تتراوح أعمارهم بين (2-14) عامًا في مستشفى الرعاية التعليمي بمدينة الطب، بالإضافة إلى مستشفى الأطفال التعليمي المركزي بمنطقة الكرخ، بغداد، العراق، من الأول من فبراير إلى نهاية مايو 2024. تم تقسيمهم إلى أربع مجموعات. تم قياس مؤشر كتلة الجسم بالاعتماد على مخطط نمو مراكز السيطرة على الأمراض والوقاية منها المستخدم للأطفال؛ تحتوي المجموعة الأولى على 40 مريضًا نحيفين مصابين بداء السكري من النوع الأول مع مؤشر كتلة الجسم ≤ 85، وتحتوي المجموعة الثانية على 40 مريضًا يعانون من نقص الوزن مصابين بداء السكري من النوع الأول مع مؤشر كتلة الجسم ≤ 5. المجموعة تحتوي على 40 طفل سليم وأعمارهم تتناسب مع المرضى مقسمة إلى (المجموعة الثالثة، ضوابط نحيفة مع مؤشر كتلة الجسم أقل من 85 مئوية. المجموعة رقم 22، والمجموعة الرابعة تتكون من 18 طفل سليم نحيف، ضوابط نحيفة مع مؤشر كتلة الجسم ≤ 5 مئوية. تم تقييمهم على سبيل المثال من حيث الجلوكوز في المصل الصائم، والجريلين، والهيموجلوبين السكري.
النتائج: كان متوسط مستوى الجريلين في المجموعة الضابطة 4.51±0.38 (5.10±0.47) نانوجرام/مل. أظهر مرضى السكري مستويات جريلين أقل بشكل ملحوظ (0.99±0.08) (0.93±0.07) نانوجرام/مل. تباينت مستويات الغريلين بشكل كبير
.علاوة على ذلك، تم العثور على نسبة الجلوكوز في الدم الصائم (FBG) في مجموعة مرضى السكري النحيفين. كان لدى مرضى السكري الذين يعانون من نقص الوزن معدلات أعلى من الأشخاص النحيفين وذوي الوزن الناقص. كان لدى مرضى السكري الذين يعانون من نقص الوزن متوسط أعلى لـ .HbA1c في المقابل، كان لدى مجموعات التحكم النحيفة وذوي الوزن الناقص متوسط قيم HbA1c% أقل بكثير.
الاستنتاج: في المرضى المصابين بداء السكري من النوع الأول، كانت مستويات الغريلين أقل بكثير من الضوابط الصحية. ارتبط الغريلين أيضًا بشكل إيجابي بـ
FBGو HbA1cوالوزن والطول لدى المشاركين الأصحاء (النحيفين وذوي الوزن الناقص) والعمر لدى الأشخاص النحيفين. لم يتم العثور على أي ارتباط بين الغريلين والمريض.(T1DM)
الكلمات المفتاحية: مؤشر كتلة الجسم، نسبة الجلوكوز في الدم أثناء الصيام، الغريلين، الهيموجلوبين السكري (HbA1c) ، داء السكري من النوع الأول.(T1DM)