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SITAGLIPTIN-INDUCED HYPERTHYROIDISM: A CASE REPORT

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Ключові слова: *ситагліптин, цукровий діабет, гіпертиреоз, автоімунні захворювання щитоподібної залози, карбімазол*

Abstract. Sitagliptin-induced hyperthyroidism: a case report. Almarshad Feras M., Jamal Yusuf, Ram Dushad, Arif Jamal, Usman Kauser. *Sitagliptin, a dipeptidyl peptidase-4 (DPP-4) inhibitor, is commonly used to manage type 2 diabetes mellitus (T2DM). Although generally considered safe, recent studies suggest a potential role of DPP-4 inhibitors in modulating immune responses, potentially leading to autoimmune conditions, including thyroid dysfunction. This case report aims to investigate the potential association between sitagliptin use and hyperthyroidism, emphasizing the significance of regular thyroid function monitoring in patients treated with DPP-4 inhibitors. A 54-year-old man with a history of hypertension and a family history of diabetes and hypothyroidism was initiated on sitagliptin and metformin for diabetes (HbA1c=8.1%) with normal thyroid function. Three months after initiating treatment, the patient noted weight loss and other initial symptoms of hyperthyroidism; however, nine months later, the patient developed full symptoms of hyperthyroidism, including significant weight loss, palpitations, tremors, and fatigue. Laboratory findings confirmed elevated total T3 (260 ng/dL) and total T4 (20 µg/dL) levels, suppressed TSH (<0.05 µIU/mL) levels, and increased anti-thyroid peroxidase (anti-TPO) antibodies (548 IU/mL). Although thyroid-stimulating hormone receptor antibody (TRAb) testing, ultrasonography, and scintigraphy were not performed, the temporal relationship between sitagliptin initiation and symptom onset, followed by resolution upon drug discontinuation, strongly suggests a probable association. Sitagliptin was discontinued, and Carbimazole was initiated, resulting in clinical and biochemical improvement. The patient's thyroid function normalized within six months, further supporting the association between sitagliptin and hyperthyroidism. Carbimazole was continued for 15-months resulting in Hb1Ac (6.6%), total T3 (140 ng/dL), total T4 (8.85 µg/dL), and TSH (1.81 µIU/mL) levels. Five months after discontinuation of Carbimazole, laboratory investigations revealed HbA1c (6.6%), fasting blood glucose (112.33 mg/dL), vitamin D (29.7 ng/mL), vitamin B12 (231 pg/mL), total T3 (151 ng/dL), total T4 (9.84 µg/dL), and TSH (1.18 µIU/mL) levels. In conclusion, this case suggests a potential association between sitagliptin and hyperthyroidism, possibly via immunomodulation. While routine thyroid function monitoring in patients on DPP-4 inhibitors may be considered, larger studies are needed to confirm this association.*

Реферат. Гіпертиреоз, індукований ситагліптином: клінічний випадок. Альмаршад Ферас М., Джамал Юсуф, Рам Душад, Аріф Джамал, Усман Каузер. *Ситагліптин, інгібітор дипептидилпептидази-4 (DPP-4), зазвичай застосовується для лікування цукрового діабету 2 типу (ЦД2). Хоча його вважають загалом безпечним, останні дослідження свідчать про можливу роль інгібіторів DPP-4 у модуляції імунних реакцій, що потенційно може призводити до автоімунних станів, включно з дисфункцією щитоподібної залози. Цей клінічний випадок має на меті дослідити можливий зв'язок між застосуванням ситагліптину та розвитком*

гіпертиреозу, підкреслюючи важливість регулярного контролю функції щитоподібної залози в пацієнтів, які отримують терапію інгібіторами DPP-4. Чоловікові 54 років з артеріальною гіпертензією в анамнезі та сімейним анамнезом цукрового діабету й гіпотиреозу було призначено ситагліптин та метформін для лікування діабету (HbA1c=8,1%) при нормальній функції щитоподібної залози. Через три місяці після початку лікування пацієнт відзначив зниження маси тіла та інші початкові симптоми гіпертиреозу; однак через дев'ять місяців у нього розвинулися повні клінічні прояви гіпертиреозу, включно зі значною втратою маси тіла, серцебиттям, тремтінням і втомлюваністю. Лабораторні дослідження підтвердили підвищені рівні загального Т3 (260 нг/дл) і загального Т4 (20 мкг/дл), пригнічений рівень ТТГ (<0,05 мкМО/мл) та підвищений титр антитіл до тиреоїдної пероксидази (anti-TPO) (548 МО/мл). Хоча визначення антитіл до рецептора ТТГ (TRAb), ультразвукове дослідження та сцинтиграфія не проводилися, часовий зв'язок між початком терапії ситагліптином і появою симптомів, а також їх регрес після відміни препарату сильно свідчить про ймовірну асоціацію. Ситагліптин було відмінено, а розпочато терапію карбімазолом, що привело до клінічного та біохімічного покращення. Протягом шести місяців функція щитоподібної залози нормалізувалася, що додатково підтвердило можливий зв'язок між ситагліптином і гіпертиреозом. Лікування карбімазолом продовжувалося протягом 15 місяців, у результаті чого рівні HbA1c становили 6,6%, загальний Т3 – 140 нг/дл, загальний Т4 – 8,85 мкг/дл і ТТГ – 1,81 мкМО/мл. Через п'ять місяців після відміни карбімазолу лабораторні показники виявили HbA1c – 6,6%, рівень глюкози натще – 112.33 мг/дл, вітаміну D – 29,7 нг/мл, вітаміну B12 – 231 нг/мл, загального Т3 – 151 нг/дл, загального Т4 – 9,84 мкг/дл та ТТГ – 1,18 мкМО/мл. У підсумку, цей клінічний випадок свідчить про можливу асоціацію між застосуванням ситагліптину та розвитком гіпертиреозу, ймовірно через імуномодулюючий механізм. Хоча може бути доцільним рутинний контроль функції щитоподібної залози в пацієнтів, які отримують терапію інгібіторами DPP-4, потрібні масштабніші дослідження для підтвердження цього зв'язку.

Hyperthyroidism is characterized by increased synthesis and secretion of thyroid hormones, resulting in systemic hypermetabolism. The primary etiologies of hyperthyroidism include Graves' disease, toxic multinodular goiter, and subacute thyroiditis [1]. Additional contributors may include primary and secondary factors, such as pharmacological agents that influence thyroid function [2]. Several medications, notably anti-obesity drugs, have been implicated in disrupting endogenous thyroid homeostasis through such mechanisms as altered hypothalamic-pituitary axis regulation, enhanced hormone release, increased thyroid hormone production, destructive thyroiditis, and exacerbation of thyroid autoimmunity [3]. Conversely, other anti-obesity agents, such as naltrexone + bupropion, appear to lack significant thyroid-modulating effects on TSH levels. Furthermore, no documented interactions between setmelanotide or sitagliptin and thyroid function have been reported to precipitate clinical thyroid disorders in humans.

Sitagliptin, a dipeptidyl peptidase-4 (DPP-4) inhibitor, is extensively employed in the management of type 2 diabetes mellitus (T2DM) by augmenting incretin hormone levels, thereby enhancing glucose-dependent insulin secretion and suppressing glucagon release [2]. Although DPP-4 inhibitors are generally well tolerated, emerging evidence suggests potential immune-mediated effects [4]. Reports of immune-related adverse events have prompted scrutiny of the immunomodulatory properties of DPP-4 inhibitors [5,6,7]. Given the established association between immune dysregulation and thyroid disorders, investigating the potential link between sitagliptin and hyperthyroidism is critical for elucidating the underlying pathophysiology of the latter [8]. Notably, Sekizaki et al. reported a potential association between DPP-4 inhibitor use and exacerbation of Grave's disease in a retrospective multicenter study [9].

This case report aims to investigate the incidence of sitagliptin-associated hyperthyroidism, analyzing the patient's clinical manifestations, laboratory findings, and therapeutic outcomes to elucidate potential immune-mediated mechanisms underlying this adverse event. The findings help to understand the safety profile of DPP-4 inhibitors in type 2 diabetes mellitus management, emphasizing the need for vigilant monitoring of immune-related adverse effects.

MATERIALS AND METHODS OF RESEARCH

This case report adhered to the ethical codes outlined in the WMA Declaration of Helsinki – "Ethical principles for medical research involving human participants". Written informed consent was obtained from the patient prior to their inclusion in the study. Ethical approval was deemed unnecessary because this study constituted an observational analysis of a single patient's clinical course.

The research was conducted in accordance with the principles of bioethics set out in the WMA Declaration of Helsinki – "Ethical principles for medical research involving human participants" and "Universal Declaration on Bioethics and Human Rights" (UNESCO).

The patient was a 54-year-old male biochemistry professor with a sedentary lifestyle. His medical history included an eight-year diagnosis of hypertension, managed with aspirin (Ecosprin, 75 mg) and telmisartan (40 mg). No additional comorbidities, allergies, psychiatric conditions, or surgical histories were reported. A family history of diabetes mellitus

and hypothyroidism prompted an annual comprehensive health evaluation of the patient.

During a routine evaluation in July 2022, the patient reported no symptomatic complaints. Physical examination revealed normal vital signs and a body weight of 75.5 kg. Laboratory investigations were conducted by accredited laboratories (Thyrocare Technolgies Limited, Navi Mumbai, India) using standardized, advanced and approved protocols indicated an HbA1c of 8.1% (equivalent to an average blood glucose level of 186 mg/dL). Other parameters, including vitamin D and B12 levels, lipid profile, and hepatic and renal function test results, were within normal limits.

Following consultation with an endocrinologist and confirmation of elevated HbA1c, fasting glucose (130 mg/dL), and postprandial glucose (180 mg/dL) levels, a diagnosis of prediabetes was established. The patient was initiated on a combination therapy of sitagliptin (50 mg) and metformin (500 mg) twice daily, complemented by lifestyle modifications, including dietary adjustments and increased physical activity. No immediate adverse reactions were noted.

Several weeks after treatment initiation, the patient experienced unexpected weight loss despite fasting glucose levels stabilizing within the normal range (100±10 mg/dL). Over the subsequent months, additional symptoms emerged, including palpitations, finger tremors, fatigue, anxiety, and generalized weakness of the body. Due to non-medical constraints, the patient delayed follow-up with the endocrinologist for nine months. By April 2023, his weight had decreased to 64 kg, reflecting an 11 kg loss. Clinical examination revealed a pulse rate of 137 beats/min, fine tremors, and brisk deep tendon reflexes at the knees and elbows.

Diagnostic investigations in April 2023 revealed marked thyroid dysfunction: total triiodothyronine (T3) at 260 ng/dL, total thyroxine (T4) at 20 µg/dL, thyroid-stimulating hormone (TSH) at <0.05 µIU/mL, and thyroid peroxidase antibodies (anti-TPO) at 548 IU/mL. Concurrently, HbA1c (5.5%), vitamin D (81.53 ng/mL), and vitamin B12 (204 pg/mL) levels remained within the normal range.

RESULTS AND DISCUSSION

The sitagliptin-metformin combination was discontinued, and metformin (500 mg) was continued alongside aspirin (Ecospirin, 75 mg), atorvastatin (10 mg), and telmisartan (40 mg). Antithyroid therapy with carbimazole (10 mg) was initiated thrice daily. The patient maintained this dose for three months with monthly thyroid function monitoring. Significant symptomatic improvement was observed, permitting a dose reduction of carbimazole to 10 mg twice daily.

The World Health Organization-Uppsala Monitoring Center (WHO-UMC) causality assessment framework was applied, yielding the following observations.

- **Temporal Association**: Hyperthyroid symptoms manifested 3-6 months after sitagliptin initiation.
- **Dechallenge Evidence**: Symptom resolution and normalization of thyroid hormone levels occurred after sitagliptin withdrawal.
- **Alternative Causes**: No prior history of thyroid dysfunction or other precipitating factors was identified.
- **Objective Evidence**: Laboratory confirmation of hyperthyroidism was supported by elevated anti-TPO antibody levels in the present case.

Based on the WHO-UMC framework, the association between sitagliptin and hyperthyroidism was classified as “probable.”

Monthly evaluations guided subsequent management of the patient. Based on the improvement in thyroid function, the regimen was adjusted to metformin (500 mg) daily and carbimazole (5 mg) daily for one month, followed by carbimazole on alternate days. Adherence to lifestyle recommendations resulted in sustained symptom resolution and normalization of thyroid function. In October 2023, laboratory results indicated free T3 (3.38 pg/mL), free T4 (14.78 pmol/L), and TSH (5.01 µIU/mL, slightly above the reference range of 0.3-4.5 µIU/mL). Investigations conducted between November and December 2023 revealed HbA1c (6.3%), near-normal anti-TPO (54.3 IU/mL, reference <50 IU/mL), free T3 (4.79 pmol/L), free T4 (1.41 ng/dL), and TSH (3.91 µIU/mL) levels within normal limits. Carbimazole (5 mg) was administered on alternate days alongside metformin (500 mg) until June 2024.

After approximately 15 months of carbimazole therapy, laboratory assessments demonstrated HbA1c (6.6%), fasting blood glucose (104.35 mg/dL), vitamin D (36 ng/mL), vitamin B12 (240 pg/mL), total T3 (140 ng/dL), total T4 (8.85 µg/dL), and TSH (1.81 µIU/mL) levels. Following consultation, carbimazole was discontinued, while metformin (500 mg), aspirin (Ecosprin, 75 mg), atorvastatin (10 mg), and telmisartan (40 mg) were maintained along with ongoing lifestyle interventions. Five months after carbimazole cessation (November 2024), laboratory results indicated HbA1c (6.6%), fasting blood glucose (112.33 mg/dL), vitamin D (29.7 ng/mL), vitamin B12 (231 pg/mL), total T3 (151 ng/dL), total T4 (9.84 µg/dL), and TSH (1.18 µIU/mL). A follow-up evaluation in June 2025 showed normal thyroid functions [total T3 (122 ng/dL); total T4 (11.2 µg/dL); TSH (1.615 µIU/mL)], fasting blood glucose (90.6 mg/dL) and HbA1c (6.9%).

This case report presents a probable association between sitagliptin, a dipeptidyl peptidase-4 (DPP-4)

inhibitor, and the onset of hyperthyroidism in a 54-year-old male patient with type 2 diabetes mellitus (T2DM). The patient's clinical progression, marked by the emergence of hyperthyroid symptoms 3-6 months after commencing sitagliptin, resolution of symptoms upon cessation, and normalization of thyroid function following antithyroid treatment, strongly indicates a drug-induced cause. The World Health Organization-Uppsala Monitoring Center (WHO-UMC) causality assessment classified this association as "probable," based on the temporal relationship, dechallenge evidence, absence of alternative causes, and objective laboratory findings, including elevated anti-thyroid peroxidase (anti-TPO) antibodies (548 IU/mL).

The potential mechanisms responsible for sitagliptin-induced hyperthyroidism require further investigation. DPP-4 inhibitors, such as sitagliptin, are recognized for their ability to modulate immune responses by preventing the enzymatic degradation of incretin hormones and affecting immune cell function [4]. Several hypotheses may explain the observed thyroid dysfunction:

1. Autoantibody Induction: Sitagliptin may induce an immune response in individuals with a genetic predisposition, resulting in the production of autoantibodies such as anti-TPO, which was notably elevated in this instance. This observation is consistent with reports indicating that DPP-4 inhibitors may exacerbate autoimmune conditions, such as Graves' disease, as documented in a multicenter study where 41% of cases associated DPP-4 inhibitors with aggravated thyroid autoimmunity [6, 9].

2. T-Cell Dysregulation: DPP-4 is expressed on T-cells, and its inhibition may disrupt immune homeostasis, potentially fostering autoimmune responses against thyroid tissue. Studies suggest that DPP-4 inhibition alters T-cell signaling, which could precipitate or exacerbate thyroid autoimmunity in predisposed individuals [4, 6].

3. Molecular Mimicry: The structural similarities between sitagliptin or its metabolites and thyroid antigens may trigger an inappropriate immune response, potentially resulting in thyroid dysfunction. This mechanism has been suggested in other autoimmune disorders associated with DPP-4 inhibitors [8].

4. Immune Tolerance Disruption: Sitagliptin may compromise immune tolerance mechanisms, thereby increasing the risk of autoimmune thyroid disorders. This consideration is particularly pertinent given the patient's family history of hypothyroidism, which may suggest a genetic predisposition to thyroid autoimmunity [6].

5. Cytokine Dysregulation: The modulation of cytokine profiles resulting from DPP-4 inhibition may play a role in the development of thyroid autoimmunity. Specifically, an increase in pro-inflammatory cytokines could enhance the presentation of thyroid antigens, potentially initiating an autoimmune response [10].

6. Antigen Presentation: DPP-4 inhibitors have the potential to alter antigen presentation by immune cells, which may result in inappropriate immune responses directed at thyroid tissue. This mechanism has been associated with other immune-mediated adverse events linked to DPP-4 inhibitors [8].

The patient's clinical presentation, characterized by significant weight loss (11 kg), palpitations, tremors, and fatigue, in conjunction with laboratory findings of elevated total T3 (260 ng/dL), total T4 (20 μg/dL), suppressed TSH (<0.05 μIU/mL), and elevated anti-TPO antibodies, aligns with a diagnosis of autoimmune hyperthyroidism. The lack of thyroid-stimulating hormone receptor antibody (TRAb) testing, ultrasonography, or scintigraphy constrains the ability to definitively confirm Graves' disease or rule out alternative etiologies, such as toxic multinodular goiter. Nevertheless, the temporal correlation between the initiation of sitagliptin and the onset of symptoms, along with the resolution of symptoms and normalization of thyroid function within six months following the discontinuation of sitagliptin, strongly suggests a drug-induced etiology. The sustained normalization of thyroid function five months after the cessation of carbimazole (total T3: 151 ng/dL, total T4: 9.84 μg/dL, TSH: 1.18 μIU/mL) further supports this hypothesis.

Comparative analysis with existing literature indicates similar associations between DPP-4 inhibitors and thyroid dysfunction. Sekizaki et al. reported that DPP-4 inhibitors, particularly sitagliptin, were associated with the exacerbation of Graves' disease in a multicenter case-control study [9]. Furthermore, instances of painless thyroiditis linked to the cessation of DPP-4 inhibitors have been documented, suggesting a complex interaction between DPP-4 inhibition and thyroid function [7]. Unlike liraglutide, a glucagon-like peptide-1 receptor agonist implicated in altering thyroid function, the effects of sitagliptin appear to be mediated through immune dysregulation rather than direct hormonal interference [3].

The patient's family history of hypothyroidism and diabetes mellitus may suggest a genetic predisposition to autoimmune thyroid disease, potentially exacerbated by sitagliptin. The elevated anti-TPO antibody levels indicate an autoimmune mechanism, possibly triggered or amplified by DPP-4 inhibition. The lack of baseline anti-TPO or TRAb testing prior to the initiation of sitagliptin limits the ability to confirm whether subclinical autoimmune thyroid disease pre-existed. However, the patient's

normal thyroid function at baseline and the rapid onset of symptoms following the initiation of sitagliptin argue against a pre-existing condition.

The clinical implications of this case are noteworthy. The American Thyroid Association advises periodic thyroid function screening for at-risk populations, while Apollo Pharmacy, India, recommends annual TSH monitoring for patients on sitagliptin [11]. Considering the potential of DPP-4 inhibitors to induce or worsen thyroid dysfunction, it is advisable for clinicians to conduct routine thyroid function tests, especially in patients with a family history of thyroid disease or other autoimmune conditions. The patient's effective management with carbimazole and metformin, in conjunction with lifestyle modifications, highlights the importance of timely recognition and intervention in cases of drug-induced hyperthyroidism.

This case report is subject to several limitations. Firstly, the absence of baseline anti-TPO and TRAb testing prevents the definitive exclusion of pre-existing subclinical autoimmune thyroid disease. Secondly, the lack of thyroid imaging restricts the ability to rule out structural causes of hyperthyroidism. Thirdly, ethical considerations precluded a rechallenge with sitagliptin, which would have offered stronger evidence of causality. Lastly, as a single-case study, the findings lack generalizability and necessitate validation through larger, controlled studies.

In conclusion, this case underscores a potential association between sitagliptin and hyperthyroidism, likely facilitated by immune dysregulation. The temporal correlation, resolution upon cessation of the drug, and lack of alternative explanations support this hypothesis. It is imperative for clinicians to remain alert to indications of thyroid dysfunction in patients receiving DPP-4 inhibitors, especially those with predisposing risk factors. Comprehensive studies are necessary to validate this association and inform guidelines for routine thyroid monitoring in patients treated with sitagliptin.

CONCLUSION

1. This case highlights the potential association between sitagliptin and hyperthyroidism.
2. Given the limitations of this study, the findings remain hypothesis-driven, and definitive conclusions require further validation.
3. Large-scale comprehensive studies are imperative to elucidate the risk of thyroid dysfunction associated with dipeptidyl peptidase-4 inhibitors.
4. Until such evidence is available, clinicians should remain vigilant for signs of thyroid dysfunction in patients receiving sitagliptin and consider individualized periodic thyroid function testing based on patient-specific risk factors.

Contributors:

Almarshad Feras – methodology, investigation, writing – original draft;

Jamal Yusuf – data curation, formal analysis, resources;

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Arif Jamal – resources, formal analysis;

Usman Kauser – writing – review & editing.

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