Scientific Research Publishing

Open Journal of Endocrine and Metabolic Diseases, 2025, 15(11), 227-236
https://www.scirp.org/journal/ojemd
ISSN Online: 2165-7432
ISSN Print: 2165-7424

Prevalence of Hypercortisolism in Patients with Adrenal Adenomas and Type 2 Diabetes Mellitus: A Mixed Retrospective-Prospective Study

Sanna Salam[1*], Nimra Memon[2], Masoud Amini[1], Isaac Sachmechi[1]

[1]Department of Internal Medicine, Icahn School of Medicine at Mount Sinai Queens, New York, NY, USA
[2]Department of Internal Medicine, Peoples University of Medical and Health Sciences for Women, Nawabshah, Pakistan
Email: *Drsanama_leo@yahoo.com

How to cite this paper: Salam, S., Memon, N., Amini, M. and Sachmechi, I. (2025) Prevalence of Hypercortisolism in Patients with Adrenal Adenomas and Type 2 Diabetes Mellitus: A Mixed Retrospective-Prospective Study. *Open Journal of Endocrine and Metabolic Diseases*, **15**, 227-236.
https://doi.org/10.4236/ojemd.2025.1511021

Received: September 2, 2025
Accepted: November 11, 2025
Published: November 14, 2025

Abstract

Objective: Adrenal adenomas are often discovered incidentally, and some demonstrate autonomous cortisol secretion that can worsen metabolic and cardiovascular outcomes. Patients with type 2 diabetes mellitus (T2DM) may be at higher risk, yet the prevalence and clinical significance of mild hypercortisolism in this group are not well defined. Design: Mixed retrospective-prospective review of patient records. **Methods:** Electronic medical records from NYC Health + Hospitals/Queens Diabetes Center (2014-2020) were reviewed. Adults (≥18 years) with radiologically confirmed adrenal adenomas and T2DM were included. Patients on corticosteroids, psychiatric medications, or with life-threatening illness were excluded. Hypercortisolism was defined by a 1-mg dexamethasone suppression test (DST) with a cortisol cutoff ≥ 1.8 μg/dL. Clinical and biochemical parameters, medication use, and imaging follow-up were assessed over one year. **Results:** Among 500 screened patients, 30 had adrenal adenomas. Thirteen (43.3%) demonstrated abnormal DST cortisol levels (≥1.8 μg/dL). Compared with patients with normal DST results, those with abnormal values were more likely to require escalation of diabetes (77%) and antihypertensive (77%) therapy during follow-up. Five patients also had an increase in adenoma size, while most with normal DST values showed stable adenoma size and fewer medication changes. **Conclusions:** Nearly half of patients with T2DM and adrenal incidentalomas showed biochemical evidence of hypercortisolism, which was associated with worsening glycemic and blood pressure control. These findings highlight the need for careful endocrine and metabolic monitoring in this population.

Keywords

Hypercortisolism, Type 2 Diabetes Mellitus, Adrenal Adenomas, Autonomous Cortisol Secretion, Metabolic Complications

1. Introduction

Mild hypercortisolism (MH) is a clinically asymptomatic, mild irregularity in the HPA axis, and it rarely becomes vigorously active over time. Therefore, it is referred to as subclinical hypercortisolism. However, slow release of excessive cortisol levels can cause osteopenia, central obesity, diabetes, hypertension, atherosclerotic disease, and an increased risk of death. Cortisol-secreting adrenal adenomas, which are found incidentally during imaging, occur in about 5% of the general population, with their incidence increasing with age; these account for 5% to 30% of all adrenal adenomas discovered incidentally [1]. MH occurs in approximately 0.2% to 2% of the general population. Most patients with uncontrolled metabolic syndromes are clinically misdiagnosed and equivocally treated due to MH [2]. Therefore, the aim of our study is to determine the prevalence of MH in type 2 diabetic patients with adrenal incidentaloma.

2. Materials and Methods

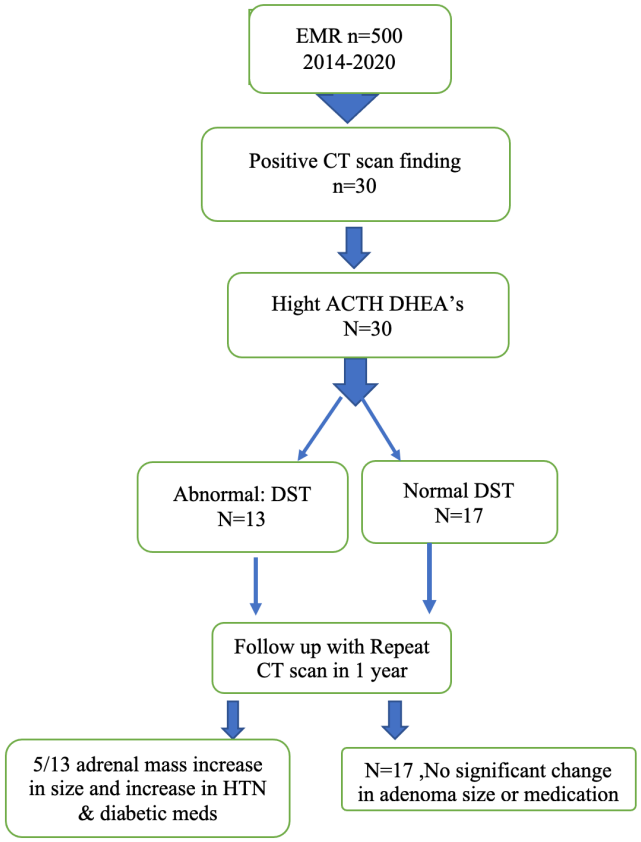
This mixed retrospective-prospective study reviewed electronic medical records (EMR) from 2014 to 2020 to identify patients aged ≥ 18 years with radiologically confirmed adrenal adenoma(s) and type 2 diabetes mellitus (T2DM). ICD-10-CM codes used were: T2DM (E11.9) and adrenal mass/nodule (E27.9). Patients on systemic corticosteroids, psychiatric medications, pregnant individuals, and those with life-threatening conditions such as metastatic cancer were excluded.

A total of 500 EMR records were screened, and 30 patients had positive CT findings of adrenal adenoma. The study flow and patient selection process are illustrated in **Figure 1**. After an incidental finding of an adrenal mass on CT imaging, a 1-mg dexamethasone suppression test (DST) was performed to confirm hypercortisolism using a diagnostic threshold of ≥1.8 μg/dL, either retrospectively (if available in EMR) or prospectively after informed consent. The distribution of patients with abnormal and normal DST cortisol values is summarized in **Table 1** and **Table 2**.

All participants underwent physical examination, blood pressure and BMI measurement, and biochemical assessments, including adrenocorticotropic hormone (ACTH), dehydroepiandrosterone sulfate (DHEA-S), HbA1c, and lipid profile.

Two different ACTH assays were used because the hospital laboratory implemented a new testing platform during the study period. The Cobas ACTH assay (Roche) was used for samples analyzed earlier in the study, and the Immulite ACTH assay (Siemens) was introduced as the new method midway through the study. To address potential assay variability, ACTH results were interpreted ac-

cording to the reference range specific to each platform. Patients were classified as having adrenal (ACTH-independent) hypercortisolism if ACTH levels were below the lower limit of normal for that assay. Values near the cutoff were interpreted in conjunction with other biochemical and imaging findings.



Note: DST: Dexamethasone Suppression Test; EMR: Electronic Medical Records; T2DM: Type 2 Diabetes Mellitus; Meds: Medication; HTN: Hypertension.

Figure 1. Patient enrollment and follow-up flowchart.

Table 1. Glycosylated hemoglobin A1C.

Characteristic	Patients (N = 30)
Male/female, n (%)	12 (40)/18 (60)
Age, years, mean (range)	64.9 (46 - 89)
HbA1c, %, mean (range)	7.3 (5.8 - 13.4)
Adrenal nodule size, cm, mean (range)	1.8 (1.1 to 5.1)
Systolic/diastolic blood pressure, mmHg, mean (range)	140 (209 - 125)/ 78 (101 - 79)

Each patient had two CT scans, one year apart, to assess for changes in adenoma size. Follow-up imaging findings for normal and abnormal DST groups are detailed in **Tables 2-4**.

Table 2. Patients with abnormal DST (cortisol ≥ 1.8 μg/dL).

Patient	1-mg DST (μg/dL)	Baseline HbA1C (%)	After 1 Year HbA1C (%)	Baseline BP (mmHg)	BP after 1 Year (mmHg)	Baseline CT Scan	CT Scan after 1 Year	ACTH (pg/mL)	Lab Assay	DHEA-S (μg/dL)
1	8.0	13.4	10.4	137/87	138/94	1.0-cm left adrenal nodule	No change	33.6	Immulite	27
2	3.9	6.6	8.8	110/70	114/73	1.3-cm right adrenal adenoma	1.8-cm right adrenal adenoma	36.1	Immulite	90.1
3	3.6	6.5	6.3	138/84	136/78	1.5-cm left adrenal nodule	1.8-cm left adrenal nodule	31.5	Immulite	87.7
4	3.4	6.2	6.3	133/87	138/74	1.1-cm left adrenal nodule	2.1-cm left adrenal nodule	20	Immulite	162
5	3.3	7.2	6.3	156/82	125/78	1.2 × 1.1-cm right adrenal nodule	1.3-cm right adrenal nodule	10	COBAS	25
6	3.0	5.0	6.1	146/72	136/56	1.0-cm left adrenal nodule	No change	14.8	COBAS	171
7	2.7	8.6	8.7	153/69	148/76	3.4-cm hypodense mass	3.3 × 2.4-cm right adrenal nodule	7	COBAS	<15
8	2.7	5.8	5.5	124/85	132/89	2.1-cm left adrenal nodule	No change	15	COBAS	182
9	2.2	7.2	7.1	130/70	138/80	2.4 × 1.7-cm nodule	2.4-cm nodule	10	COBAS	20
10	2.2	6.9	-	120/70	110/75	1.2-cm left adrenal nodule	No change	19	Immulite	18
11	2.0	7.7	6.9	145/89	134/78	Indeterminate right adrenal adenoma	Determinant right adrenal adenoma	8.9	COBAS	18
12	2.0	7.6	6.4	148/75	123/71	1.8-cm right adrenal nodule	2-cm right adrenal nodule	15.1	COBAS	46.7
13	1.9	7.8	8.0	130/86	167/90	2.1-cm left adrenal nodule	No change	1.6	COBAS	72

Table 3. Patients with normal DST (cortisol < 1.8 μg/dL).

Patient	1-mg DST (μg/dL)	Baseline HbA1C (%)	HbA1C after 1 Year (%)	Baseline BP (mmHg)	BP after 1 Year (mmHg)	Baseline CT Scan	CT Scan after 1 Year	ACTH (pg/mL)	Lab Assay	DHEA-S (μg/dL)
1	1.7	7.0	7.5	140/80	145/95	1.1 cm right	No change	10.9	Immulite	20.2
2	1.6	7.4	7.4	139/92	129/81	1.0 cm left	No change	15	Immulite	182
3	1.5	7.8	6.5	132/68	128/59	2.5 cm left	No change	32	Immulite	65.8
4	1.5	7.1	7.2	138/86	136/78	Left adrenal	No change	9.4	Immulite	85.6
5	1.4	6.3	7.1	142/88	170/100	1.1 × 1.0 cm left	No change	8.2	Immulite	49
6	1.3	7.2	7.1	132/70	137/79	1.3 cm left	No change	20.2	Immulite	26.8
7	1.2	7.4	6.2	231/109	149/74	1.9 cm left	No change	0.9	Immulite	198
8	1.2	6.0	6.5	135/87	127/76	2.5 × 1.8 cm left	No change	7.3	Immulite	46.7
9	1.1	6.2	4.9	180/101	209/107	1.2 cm right	No change	18	Immulite	87.7
10	0.9	6.3	6.3	126/87	143/75	1.4 × 2.2 cm left	No change	<15	Immulite	15
11	0.8	6.3	4.2	139/85	118/84	1.9 × 1.0 cm left	1.9 × 1.6 cm left	19.4	Immulite	0.9

Continued

12	0.8	5.8	6.3	126/73	143/76	1.5 × 1.4 cm right	No change	19.4	Immulite	103
13	0.7	7.4	6.8	168/80	166/97	1.5 cm left	1.4 cm left	45	Immulite	100
14	0.7	7.2	7.4	145/71	179/84	1.3 × 3.5 cm right	No change	68	Immulite	103
15	0.6	6.9	6.5	159/77	140/74	1.0 cm adrenal	No change	44	Immulite	121
16	0.6	7.7	6.7	114/78	115/75	1.2 × 1.3 cm right	0.8 cm right	9.6	Immulite	20
17	0.1	7.0	6.4	114/78	115/75	1.2 × 1.3 cm right	0.8 cm right	9.6	Immulite	20

Normal values: ACTH: Adrenocorticotropic Hormone; BP: Blood Pressure; CT: Computed Tomography; DHEA-S: Dehydroepiandrosterone-Sulfate; DST: Dexamethasone Suppression Test; Hb: Hemoglobin.

Table 4. Summary of patients with abnormal and normal DST cortisol results.

Groups	Description
1	Thirteen patients (13/30, 43.3%) had an abnormal DST cortisol value ≥ 1.8 μg/dL (range: 1.9 - 8.0 μg/dL) ■ Eight patients had validated ACTH results (range: 1.6-15.1 pg/mL) that supported the adrenal etiology, while 5 patients had ACTH values above the normal range using a different assay (Immulite) ■ Among the 13 patients with abnormal DST results, the adrenal nodule size ranged from 1.0 to 3.4 cm (Table 2)
2	Seventeen patients (17/30, 56.7%) had a normal DST cortisol value (range 0.7 - 1.7 μg/dL) ■ Among these patients, the adrenal nodule size ranged from 1.0 to 5.1 cm (Table 3)

Medication use was reviewed throughout the study and reconciled at its conclusion. Patterns of medication adjustment during follow-up are presented in Table 5, and baseline medication distribution is shown in Figure 2.

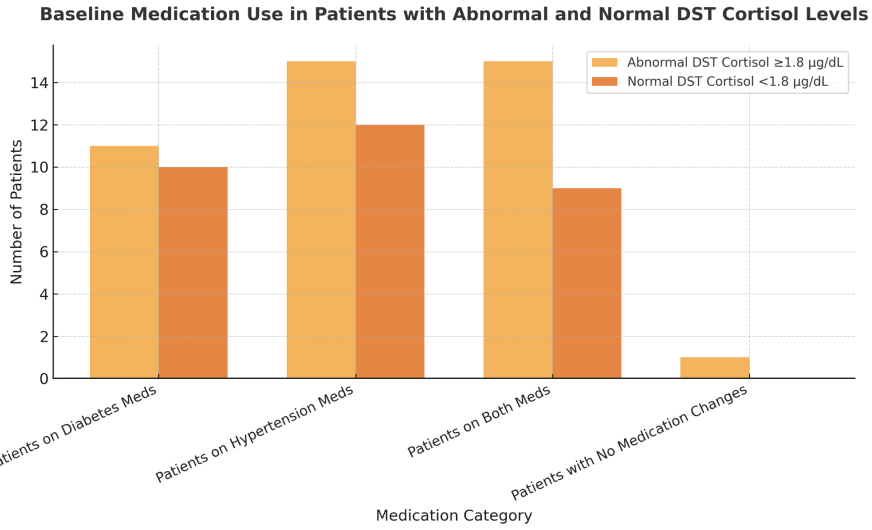


Figure 2. Comparison of medication use between patients with abnormal and normal DST cortisol results.

"Medication intensification" was defined as an increase in the dose or number of antihyperglycemic or antihypertensive agents in response to suboptimal disease

control, indicated by either a rise in HbA1c of ≥0.5% from baseline or systolic blood pressure > 140 mmHg on two consecutive visits. "Medication tapering" was defined as a reduction in dose or discontinuation of these medications following improvement in control, defined by a decrease in HbA1c of ≥0.5% or sustained blood pressure < 120/80 mmHg over two visits.

The study protocol was approved by the Institutional Ethics Committee of NYC Health + Hospitals/Queens, and written informed consent was obtained from all participants enrolled in the prospective evaluations.

2.1. Study Participants Characteristics

Medication Changes Compared with baseline medication use (**Figure 1**), there were more medication dosage increases, and new medications were added to improve metabolic control of both diabetes and hypertension in the group with abnormal DST baseline values.

2.2. Patterns of Medication Adjustment for Diabetes and Hypertension during Follow-Up Are Summarized

Table 5. Patterns of medication adjustment for diabetes and hypertension during follow-up in patients with abnormal and normal DST cortisol levels.

Medication Adjustment	Abnormal DST > 1.8 (n = 13)	Normal DST < 1.8 (n = 17)
Intensification of DM Medication	10/11 (90.91%)	5/15 (33.3%)
Intensification of HTN Medication	9/12 (75%)	2/15 (13.3%)
Tapering of DM + HTN Meds	1 /13 (7.69%)	01 (5.88)
Tapering of HTN Meds Only	1/13 (7.69)	2 (11.76)
Reduction in DM Meds	0	4 (23.53)
No Changes in Medication	1(7.69%)	0

Abnormal DST cortisol ≥ 1.8 μg/dL group (n = 13):

- 10/11 (90.1%) patients had an increase in the dosage of their diabetes medications and/or were prescribed a new medication for diabetes. 9/12 (75%) patients had an increase in blood pressure medications and/or were prescribed a new medication
- 1 patient had a reduction in the number and/or dosage of diabetes and blood pressure medications, 1 patient had a reduction in blood pressure medication, and 1 patient had no medication changes at all

Normal DST cortisol < 1.8 μg/dL group (n = 17):

- 5/15 (33.3%) patients had an increase in diabetes medication dosages and/or a new medication added
- 2/15 (13.3%) patients had a blood pressure medication dose increase and/or a new medication added
- No patients had an increase or new medication for both diabetes and hyper-

tension

- 4 patients had a decrease in dosage or discontinuation of diabetes medications, 2 patients had a decrease in dosage or discontinuation of blood pressure medications, and 1 patient had a dosage decrease or discontinuation of both diabetes and blood pressure medications.

3. Results

Thirteen of thirty patients (43.3%) had an abnormal 1-mg DST cortisol value ≥ 1.8 μg/dL (range 1.9 - 8.0 μg/dL).

Eight of these patients had validated ACTH results (range 1.6 - 15.1 pg/mL) consistent with adrenal etiology, while five had ACTH values above the normal range using a different assay (Immulite).

Among patients with abnormal DST results, adrenal nodule size ranged from 1.0 to 3.4 cm (**Table 2**).

Seventeen patients (56.7%) had a normal DST cortisol value < 1.8 μg/dL (range 0.7 - 1.7 μg/dL).

In this group, adrenal nodule size ranged from 1.0 to 5.1 cm (**Table 3**).

Results are presented descriptively for the abnormal DST and normal DST groups, and no inferential statistical tests were performed.

4. Discussion

Autonomous glucocorticoid hypersecretion (AGH) has been reported in 5% to 20% of patients with adrenal incidentalomas in several studies using different diagnostic criteria [3]-[7]. In our mixed retrospective-prospective study, subclinical or incidentally detected adrenal adenomas were found predominantly in female patients, with a mean age of 60 years. Similar findings have been reported in previous studies showing a higher prevalence among women [8] [9].

In our cohort, tumor involvement of the left adrenal gland was more common in the abnormal DST group compared with the normal DST group, and adenoma size in all patients was less than 6 cm. Earlier studies have reported up to a 9% incidence of hypercortisolism in diabetic patients [10]. Due to limited screening for cortisol excess, many patients with diabetes are managed as idiopathic or insulin-resistant diabetes mellitus. Consistent with prior research [11], our patients with subclinical hypercortisolism also demonstrated a higher prevalence of hyperlipidemia, insulin resistance, and hypertension.

Glucocorticoids impair peripheral glucose utilization and increase hepatic glucose output, leading to elevated blood glucose levels [12]. The mechanisms underlying this increase include enhanced lipolysis and proteolysis, which provide substrates for gluconeogenesis, as well as increased hepatic extraction and metabolism of glucose precursors and induction of gluconeogenic enzymes [13] [14]. Cortisol also has a permissive effect on glucagon and epinephrine, which may further worsen hyperglycemia in poorly controlled diabetes [15].

Subclinical hypercortisolism has been reported to be more common in diabetic,

obese, and hypertensive patients. Between 5% and 30% of adrenal adenomas discovered incidentally are reported to be cortisol-secreting [2].

In this study, we aimed to determine the prevalence of AGH in patients with T2DM and adrenal incidentalomas. We found that 43.3% of patients had cortisol-secreting adenomas, which is higher than previously reported rates [16] [17]. This excess cortisol likely contributes to poor glycemic and blood pressure control.

As suggested in multiple studies, hypercortisolemia is associated with increased mortality, predominantly from cardiovascular disease and infections such as pneumonia [18] [19]. Interestingly, another study also demonstrated that malignancy, rather than cardiovascular disease, was the leading cause of mortality among patients with adrenal incidentalomas and autonomous cortisol secretion [19].

In our study, patients with abnormal DST results (cortisol ≥ 1.8 μg/dL) required more frequent medication adjustments for diabetes and hypertension, reflecting worsening metabolic control. These findings are consistent with results from previous meta-analyses showing that patients with autonomous cortisol secretion have higher rates of diabetes and hypertension—both risk factors for cardiovascular disease [1].

During the 12-month follow-up period, there was a mild but statistically non-significant increase in adrenal nodule size among patients with post-DST cortisol > 1.8 μg/dL. In a large multicenter cohort followed for at least 5 years, 8.2% of patients with initially nonfunctioning adenomas developed autonomous cortisol secretion over time, particularly when adenoma size was ≥2.4 cm [20]. Cardiovascular events were more frequent in AGH patients compared with those who had non-cortisol-secreting adenomas, regardless of age or presence of type 2 diabetes. These findings emphasize the importance of long-term follow-up and appropriate management for all patients with adrenal incidentalomas [2] [20].

This study has several limitations. It was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. The follow-up period was short, and long-term outcomes such as cardiovascular events or mortality could not be fully assessed. In addition, potential confounding factors, including medication adherence, duration of diabetes, and comorbid conditions, were not adjusted for in the analysis. These limitations should be considered when interpreting the results, and larger multicenter studies with extended follow-up are warranted to confirm these observations.

5. Conclusion

Patients with type 2 diabetes and adrenal incidentalomas (AI) have a high prevalence of autonomous cortisol secretion, which contributes to worsening glycemic and blood pressure control—both key risk factors for cardiovascular disease. Early identification and management of hypercortisolism in these populations are crucial for mitigating these risks. The integration of electronic medical records (EMR) with clinical markers of hypercortisolism, particularly in patients with diabetes, can improve the detection of autonomous cortisol secretion. This approach supports

timely interventions, potentially improving metabolic and cardiovascular outcomes in affected individuals.

Authors' Contributions

All authors had full access to the data and contributed significantly to the writing and revision of the manuscript.

Acknowledgements

The authors gratefully acknowledge the contributions of Corcept pharmaceuticals whose efforts made this study possible.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Elhassan, Y.S., Alahdab, F., Prete, A., Delivanis, D.A., Khanna, A., Prokop, L., *et al.* (2019) Natural History of Adrenal Incidentalomas with and without Mild Autonomous Cortisol Excess: A Systematic Review and Meta-Analysis. *Annals of Internal Medicine*, **171**, 107-116. https://doi.org/10.7326/m18-3630

[2] Chiodini, I. (2011) Diagnosis and Treatment of Subclinical Hypercortisolism. *The Journal of Clinical Endocrinology & Metabolism*, **96**, 1223-1236. https://doi.org/10.1210/jc.2010-2722

[3] Reincke, M., Nieke, J., Krestin, G.P., Saeger, W., Allolio, B. and Winkelmann, W. (1992) Preclinical Cushing's Syndrome in Adrenal "Incidentalomas": Comparison with Adrenal Cushing's Syndrome. *The Journal of Clinical Endocrinology & Metabolism*, **75**, 826-832. https://doi.org/10.1210/jcem.75.3.1517373

[4] Terzolo, M., Osella, G., Alì, A., Borretta, G., Cesario, F., Paccotti, P. and Angeli, A. (1998) Subclinical Cushing's Syndrome in Adrenal Incidentaloma. *Clinical Endocrinology*, **48**, 89-97. https://doi.org/10.1046/j.1365-2265.1998.00357.x

[5] Barzon, L. and Boscaro, M. (2000) Diagnosis and Management of Adrenal Incidentalomas. *Journal of Urology*, **163**, 398-407. https://doi.org/10.1016/s0022-5347(05)67888-3

[6] Reincke, M. (2000) Subclinical Cushing's Syndrome. *Endocrinology and Metabolism Clinics of North America*, **29**, 43-56. https://doi.org/10.1016/s0889-8529(05)70115-8

[7] Rossi, R., Tauchmanova, L., Luciano, A., Di Martino, M., Battista, C., Del Viscovo, L., *et al.* (2000) Subclinical Cushing's Syndrome in Patients with Adrenal Incidentaloma: Clinical and Biochemical Features. *The Journal of Clinical Endocrinology & Metabolism*, **85**, 1440-1448. https://doi.org/10.1210/jcem.85.4.6515

[8] Angeli, A., Osella, G., Ali, A. and Terzolo, M. (1997) Adrenal Incidentaloma: An Overview of Clinical and Epidemiological Data from the National Italian Study Group. *Hormone Research*, **47**, 279-283. https://doi.org/10.1159/000185477

[9] Sconfienza, E., Tetti, M., Forestiero, V., Veglio, F., Mulatero, P. and Monticone, S. (2023) Prevalence of Functioning Adrenal Incidentalomas: A Systematic Review and Meta-Analysis. *The Journal of Clinical Endocrinology & Metabolism*, **108**, 1813-1823. https://doi.org/10.1210/clinem/dgad044

[10] Steffensen, C., Dekkers, O., Lyhne, J., Pedersen, B., Rasmussen, F., Rungby, J., *et al.*

(2018) Hypercortisolism in Newly Diagnosed Type 2 Diabetes: A Prospective Study of 384 Newly Diagnosed Patients. *Hormone and Metabolic Research*, **51**, 62-68. https://doi.org/10.1055/a-0809-3647

[11] Costa, D.S., Conceição, F.L., Leite, N.C., Ferreira, M.T., Salles, G.F. and Cardoso, C.R.L. (2016) Prevalence of Subclinical Hypercortisolism in Type 2 Diabetic Patients from the Rio De Janeiro Type 2 Diabetes Cohort Study. *Journal of Diabetes and its Complications*, **30**, 1032-1038. https://doi.org/10.1016/j.jdiacomp.2016.05.006

[12] Shamoon, H., Jacob, R. and Sherwin, R.S. (1980) Epinephrine-Induced Hypoaminoacidemia in Normal and Diabetic Human Subjects: Effect of β Blockade. *Diabetes*, **29**, 875-881. https://doi.org/10.2337/diab.29.11.875

[13] Kraus-Friedmann, N. (1984) Hormonal Regulation of Hepatic Gluconeogenesis. *Physiological Reviews*, **64**, 170-259. https://doi.org/10.1152/physrev.1984.64.1.170

[14] Simmons, P.S., Miles, J.M., Gerich, J.E. and Haymond, M.W. (1984) Increased Proteolysis. An Effect of Increases in Plasma Cortisol within the Physiologic Range. *Journal of Clinical Investigation*, **73**, 412-420. https://doi.org/10.1172/jci111227

[15] Lecavalier, L., Bolli, G. and Gerich, J. (1990) Glucagon-Cortisol Interactions on Glucose Turnover and Lactate Gluconeogenesis in Normal Humans. *American Journal of Physiology-Endocrinology and Metabolism*, **258**, E569-E575. https://doi.org/10.1152/ajpendo.1990.258.4.e569

[16] Leibowitz, G., Tsur, A., Chayen, S.D., Salameh, M., Raz, I., Cerasi, E., *et al.* (1996) Pre-Clinical Cushing's Syndrome: An Unexpected Frequent Cause of Poor Glycaemic Control in Obese Diabetic Patients. *Clinical Endocrinology*, **44**, 717-722. https://doi.org/10.1046/j.1365-2265.1996.737558.x

[17] Catargi, B., Rigalleau, V., Poussin, A., Ronci-Chaix, N., Bex, V., Vergnot, V., *et al.* (2003) Occult Cushing's Syndrome in Type-2 Diabetes. *The Journal of Clinical Endocrinology & Metabolism*, **88**, 5808-5813. https://doi.org/10.1210/jc.2003-030254

[18] Debono, M., Bradburn, M., Bull, M., Harrison, B., Ross, R.J. and Newell-Price, J. (2014) Cortisol as a Marker for Increased Mortality in Patients with Incidental Adrenocortical Adenomas. *The Journal of Clinical Endocrinology & Metabolism*, **99**, 4462-4470. https://doi.org/10.1210/jc.2014-3007

[19] Patrova, J., Kjellman, M., Wahrenberg, H. and Falhammar, H. (2017) Increased Mortality in Patients with Adrenal Incidentalomas and Autonomous Cortisol Secretion: A 13-Year Retrospective Study from One Center. *Endocrine*, **58**, 267-275. https://doi.org/10.1007/s12020-017-1400-8

[20] Morelli, V., Reimondo, G., Giordano, R., Della Casa, S., Policola, C., Palmieri, S., *et al.* (2014) Long-Term Follow-Up in Adrenal Incidentalomas: An Italian Multicenter Study. *The Journal of Clinical Endocrinology & Metabolism*, **99**, 827-834. https://doi.org/10.1210/jc.2013-3527