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Correlation Between Cortisol Levels And BMI In Patients With Cushing's Syndrome

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دراسة العلاقة بين مستويات هرمون الكورتيزول ومؤشر كتلة الجسم لدى مرضى متلازمة كوشينغ

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ABSTRACT

Background:

Cushing's syndrome (CS) is a serious clinical condition caused by endogenous or exogenous cortisol excess, causing a cascade of severe metabolic complication such as fat accumulation. The objective is to study the level of cortisol in patients with Cushing's syndrome and its correlation with body mass index (BMI).

Materials and Methods:

This cross-sectional study was conducted at The Medical citythe Medical city, Baghdad, Iraq, from (January 2024- October 2025). The study comprised a total of forty patients diagnosed as having Cushing's syndrome, all of whom were confirmed to be cases. Details of age and BMI of the patients were retrieved directly from patients. Measurements of serum cortisol were obtained in the morning (8 AM) and afternoon (5 PM) twice daily.

Results:

The data confirmed the fact that the age of the majority of the study respondents (50%) falls between 18 and 32 years; within these ages, the later subgroup is between 33 and 47 years old (37.5%). More males were diagnosed than females (55% vs 45%). Most participants have no chronic diseases (57.5%). In Cushing's syndrome patients, cortisol levels ranged very widely and contrarily, weighed against the more narrowly distributed BMI values. Morning cortisol was significantly higher than afternoon ($p < 0.001$). Also, a strong positive correlation was seen between cortisol and BMI ($r = 0.786$, $p = 0.001$), implying that a higher BMI would be associated with higher levels of cortisol).

Conclusion:

The study suggested a strong positive correlation between levels of cortisol and BMI, indicating that that obesity in these patients is linked with higher cortisol levels.

Key words: Cortisol, BMI, Cushing's syndrome, Obesity.

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INTRODUCTION

Cushing's syndrome (CS) is a complicated type of hormone-related illness where the main problem is high levels of cortisol, causing a series of severe metabolic outcomes such as fat accumulation and heart problems. Cushing's syndrome may relate to obesity in several ways and frequently appears as one aspect of the overweight person's condition [1].

Cushing disease is an endocrine disorder characterized by excessive adrenocorticotropic hormone (ACTH) production by the anterior pituitary, which leads to the release of an excess of cortisol from the adrenal glands. The etiology of these disorders is rooted in the pathophysiological dysregulation of the HPA axis leading to both hypercortisolism as a nonphysiological output of this feedback loop. The deleterious impact of this is extensive as it leads to chronic elevation of cortisol in the circulation and disastrous metabolic disorders including obesity insulin resistance dyslipidemia and hypertension [2]. Another systematic review has even found that patients post-operative remained with cardiovascular risk factors suggesting the lipid effects of cortisol may persist following a period of hypersecretory it this further emphasizes the importance of understanding future metabolic outcomes in understanding the etiology of Cushing's syndrome.[3].

Obesity is the most characteristic feature of Cushing's syndrome; practically all patients with the disorder are overweight or obese, which subsequently intensifies cardiovascular risk. The relationship between glucocorticoids and fatness is characterized by the redistribution of fat induced by cortisol and central obesity with metabolic syndromes [4]. For example, Arnaldi et al. (2010) provides evidence on how cortisol acts on lipolysis and insulin sensitivity leading to dyslipidemia further worsening the metabolic profile of patients with CS disease. In addition, the increased prevalence of the different components of metabolic syndrome in a population of patients with CS, such as hypertension and dyslipidemia, calls for urgent intervention strategies [5]. Chanson and Salenave (2010) suggest that these should be followed up since in Cushing's syndrome obesity is linked with long-lasting metabolic derangements even after correction of hypercortisolism [6].

Cushing's syndrome is characterized by greatly disturbed metabolism and the end result could be dire in terms of health. According to studies, various patients with CS showed manifestations of obesity and dyslipidemia which are two critical factors in BMI assessments [7-8]. There exists a classic relationship between hypercortisolism and obesity. Metabolic processes promoting weight gain by way of metabolism catalyze action in the cortex are known. Elevation of BMI in patients with CS may heighten the morbidity associated with the disease and therefore there is a need for clear dynamics related to this problem [5].

The main aim of the current study is to evaluate the level of cortisol in patients with Cushing's syndrome and its correlation with BMI.

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MATERIALS AND METHODS

- **Subjects**

This cross-sectional study took place at The Medical City in Baghdad, Iraq, within the time frame of February 2024 to December 2024. A total of forty confirmed patients with Cushing's syndrome were included in the study. Information about age and body mass index (BMI) of the patients was collected directly from patients. BMI categories were classified according to the World Health Organization (WHO) classification [9].

- **Cortisol measurement**

Cortisol levels were measured using an ELISA kit from DRG International Inc., USA. When blood was collected, the samples were allowed to clot at room temperature for viscosity separation before being centrifuged to obtain serum; the serum was stored at −20°C until it was tested. The analysis was performed according to the manufacturer instructions from KITS and under his standard curve for concentration determination. The expected features of this method are high sensitivity and specificity in measurement of cortisol, which should be applicable for both clinical and research purposes. Serum cortisol was assessed twice daily at 8:00 AM and 5:00 PM.

- **Statistical Analysis**

Data was processed by SPSS statistics software version 25.0 (SPSS, Chicago). The normality test has been used to check the level of parametric data by Shapiro Wilk test; while the normally distributed data have been presented as (mean ± standard deviation) and applied by an independent t-test. A p-value of less than 0.05 was considered statistically significant.

Results and Discussion

Table 1 gives an overall view of the general characteristics of the study groups (patients) based on key demographic and health-related variables. The data underscore the fact that the age of the majority of the study respondents (50%) falls between 18 and 32 years; within these ages, the later subgroup is between 33 and 47 years old (37.5%). More males were diagnosed than females (55% vs 45%). Most participants have no chronic diseases (57.5%).

Table 1. Demographic data of patients' group

Demographic data		Patients' Group (No. = 40)	
		Freq.	%
Age/Years	18-32	20	50.0
	33-47	15	37.5
	48-62	5	12.5
Gender	Male	22	55.0
	Female	18	45.0
Chronic Diseases	Yes	17	42.5
	No	23	57.5

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The descriptive statistics for cortisol levels and BMI are shown in Table 2 for patients with Cushing's syndrome. The cortisol levels had an extremely wide range, from 104.57 to 7321, with a mean of 1194.53 and a standard deviation that is almost as high as the mean, 1751.76, indicating real differences between the people who have the condition. On the other hand, BMI values were much more tightly packed; they ranged from 17.2 to 38.1 with a mean of 25.83 and an SD of 37.61. These few numbers indicate that there was much more spread in the levels of cortisol than in BMI among these patients.

Table 2. Descriptive statistics of cortisol and BMI in patients with Cushing's syndrome

	Descriptive statistics			
	Min	**Max**	**Mean**	**SD**
Cortisol	104.57	7321	1194.53	1751.76
BMI	17.2	38.1	25.83	37.61

SD : Standard Deviation

As shown in Figure 1, cortisol levels were significantly high in the morning (mean = 2134.33, SD = 1657.22) and low in the afternoon (mean = 686.23, SD = 1231.5) with a p-value that is highly significant ($P < 0.001$).

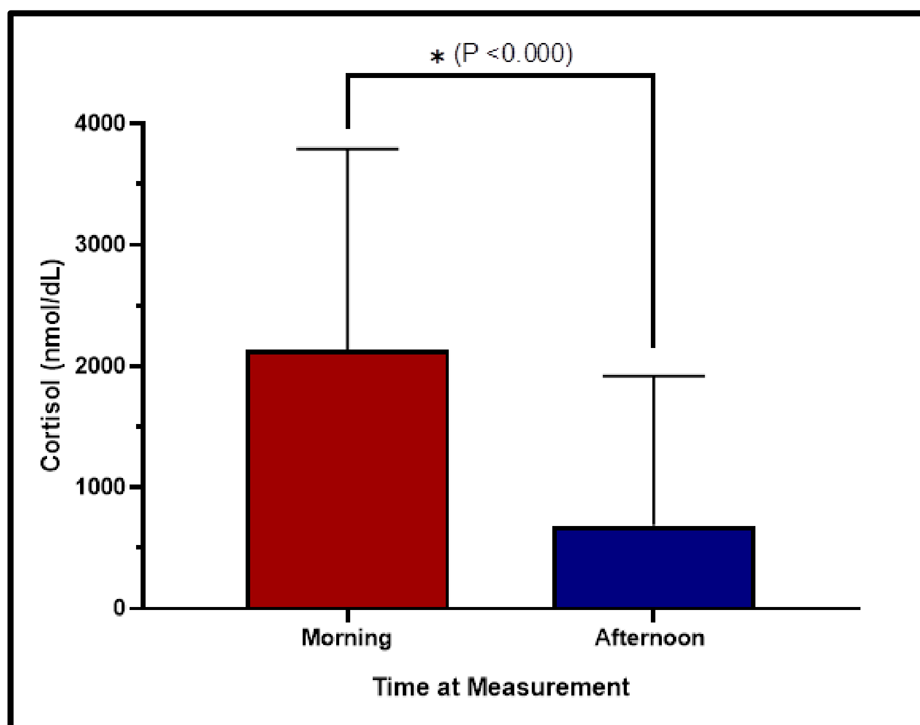

Figure 1. Measurement and differences in cortisol level between morning and afternoon

Concerning Pearson correlation coefficients between cortisol and BMI. Current results suggest a strong positive correlation between cortisol and BMI (0.786, p = 0.001), as shown also in figure 2.

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Table 3. Descriptive statistics of cortisol and BMI in patients with Cushing's syndrome

Marker	Correlation Coefficient	P value
BMI	0.785	0.001

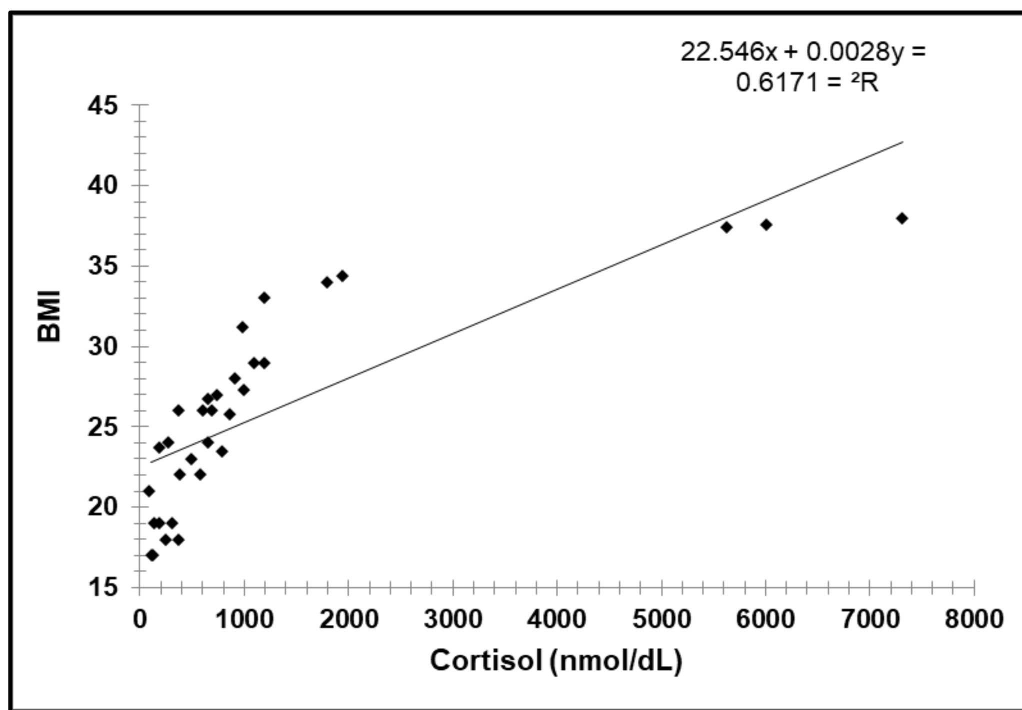

Figure 2. Scatter plot and regression equation for the correlation between cortisol and BMI

The current study reflects the normal circadian rhythm of cortisol secretion; however, the wide standard deviations indicate that there is considerable variability among patients and perhaps even according to disease severity or individual physiological differences. Cortisol is characterized by its circadian pattern of basal secretion from the adrenal glands, under central regulation by corticotrophin-releasing hormone (CRH) and arginine vasopressin (AVP) from the paraventricular nucleus (PVN) of the hypothalamus [10]. Both CRH and AVP are transported via the vasculature to the pituitary where they activate pituitary corticotrophs. Here, they stimulate the release of corticotrophin (ACTH), which in turn is released into the general systemic circulation and stimulates the adrenal cortex to produce CORT. For instance, changes in cortisol secretion can be accurately measured when these circadian rhythms are seriously out of whack, as commonly occurs in mood disorders. The keywords compared to the aberrant morning hypercortisolism which statistically directly indicate a disruption of circadian rhythm for example patients afflicted by mental illness. [11]. Intervention that alters these disrupted circadian rhythms well, may alter the relationship of cortisol levels influencing mood regulation and its associated health outcomes. We need the timing and patterns of feedback when we think through cortisol dynamics. Awakening and

late afternoon would be the times that one would want to test for dysfunction within an HPA system because feedback inhibition occurs rapidly at these points in time [12]. It emphasizes the critical role of timing in cortisol measures to properly document its physiological impacts. In so doing, it also highlights the significance of its circadian rhythm for the cardiovascular system. A natural peak of cortisosis is found just after waking up, providing tools for the daily chores of a person and by itself transforms with cardiovascular system; apparently any disturbance in this rhythm leads to serious health damages like heart diseases. Therefore, its regulation during the daytime is regarded as more than prevalent sweetness. [13].

In Cushing syndrome patients, there is an increased level of cortisol that is produced in excessive amounts, and this high level of cortisol regulates your weight, leading to the observed relationship of patiemts with BMI. In this condition, as the levels of cortisol are often elevated, fat accumulation was originally expressed with a rise in BMI. Although we might therefore expect an inverse correlation so that as the concentration of cortisol falls mine rises, but patient to patient differences, severity of illness and other metabolic factors can alter (even inconsistently) the magnitude and most importantly significance of such a priori expectation. [14].

A more powerful statistic—such as Pearson's correlation—is required to appropriately evaluate this relationship in the population of patients being studied. Diagnosing Cushing's syndrome can be especially challenging because if a patient does not exhibit classic characteristics, but instead presents with obesity and associated metabolic disturbances. Both adipose tissue and obesity have been implicated as triggers of cortisol release from the adrenal glands. Cortisol can accumulate in the bloodstream and in body tissues, particularly in fat tissue, and when it does, it binds to receptors (such as the GR and the MR) to initiate pathways involved in a number of chronic diseases [15]. Therefore, in this case, the delayed diagnosis period worsens metabolic health, reflected by an increase in BMI and related comorbidities over time [3]. Accurate diagnostic measures are needed—these include late-night salivary cortisol and urinary free cortisol measurements—for this group of patients with high BMI since such conditions may alter their metabolism of cortisol and hence the interpretation of results as per diagnosis [16].

Management strategies for Cushing's syndrome should be planned considering the very complex interaction between CS and BMI, together with all the comorbidities. The clinical presentation is highly variable and makes treatment approaches extremely complicated, therefore a multidisciplinary approach is warranted and must be tailored to each patient's individual needs [17]. Surgical and medical treatment options should take into consideration the patient's BMI as well as metabolic status so that outcomes can be maximized and complications minimized [8].

There is substantial evidence now that shows sleep deprivation has a significant effect on the secretion of cortisol and, hence, BMI. Leproult and van Cauter (2010) explain that chronic insufficiency of sleep can eventually raise levels of cortisol and, thus, drive possible weight gain [18]. In support of this, Aldabal and Bahammam (2011) also reported that sleeplessness raises not just the levels of cortisol but also brings about metabolic changes that could culminate in insulin resistance and obesity. The relationship between the quality of sleep, cortisol level, and BMI provides a basis for the proposition that any intervention which

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improves sleep might simultaneously improve obesity. Stressing the complex interactions between stress, sleep, and cortisol rhythms lately in 2019 by Russell & Lightman to explain their hypothesis; in poor sleepers exacerbating stress reactivity drives persistently higher evening salivary cortisol which could translate into weight gain plus metabolic dysregulation; maligned sleep issues would be sustainable as one primary preventive measure as well as treatment intervention for obesity [19].

Cortisol plays a key role in pituitary adenomas, according to Molitch (2017). Most of these patients with such tumors will present with hypercortisolism and subsequent weight gain and obesity. This finding shows the need for monitoring cortisol levels in patients with ACTH-secreting tumors, suggesting BMI and cortisol have a direct relationship within this clinical population [20]. Bringing in some new data, Muscogiuri et al. (2019) connected subclinical hypercortisolism to adrenal tumors, again linking elevated levels of cortisol with obesity. It would thus seem from their findings that increased metabolic diseases depend on high levels of metabolic-type disorders linked to obesity that should lead researchers into greater involvement regarding the role of cortisol in common types of endocrine diseases related to obesity [21]. Antonelli et al. (2019) would review literature on how such molecules as glucocorticoids and 11β-hydroxysteroid dehydrogenase type 1 act in the matter by which one hormone regulates the metabolism imposing it over an area where its activation contributes significantly toward promoting fat deposition and hence malfunctioning adipose tissue. This highlights a possible treatment path—focusing on cortisol metabolism might give us new ways to deal with obesity and metabolic syndrome [22]. Kumari et al. (2010) do a study on how cortisol secretion patterns relate to general and central obesity. Their findings reported a nonlinear association, which suggests that the complexities of cortisol dynamics require further exploration to fully understand its impact on BMI [23].

Conclusions:

The findings of the current study have showed a significant variation in cortisol levels within patients with Cushing's syndrome, whilst BMI values were overall less variable. Difference between morning and afternoon cortisol levels was more in the morning with high significant difference. In Addition, there was a positive correlation between the levels of cortisol and BMI ($p < 0.05$), which suggest that higher BMIs in these patients were associated with higher layers of cortisol.

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Conflict of interests.

There are non-conflicts of interest.

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الخلاصة

مقدمة:

تُعد متلازمة كوشينغ (Cushing's syndrome) من الاضطرابات الهرمونية المعقدة التي تتمثل بارتفاع مستويات هرمون الكورتيزول، مما يؤدي إلى سلسلة من التأثيرات الأيضية الشديدة، من أبرزها تراكم الدهون وزيادة الوزن . تهدف هذه الدراسة إلى تقييم مستوى هرمون الكورتيزول لدى المرضى المصابين بمتلازمة كوشينغ، ودراسة علاقته بمؤشر كتلة الجسم

طرق العمل:

أُجريت هذه الدراسة المقطعية في مدينة الطب بمدينة بغداد – العراق، خلال الفترة الممتدة من شباط 2024 إلى كانون الأول 2024. شملت الدراسة أربعين مريضاً مشخصين بمتلازمة كوشينغ. تم جمع المعلومات المتعلقة بالعمر ومؤشر كتلة الجسم (BMI) مباشرة من المرضى. كما تم قياس مستويات الكورتيزول في مصل الدم مرتين يومياً، صباحاً في الساعة 8:00 صباحاً، ومساءً في الساعة 5:00 مساءً.

النتائج:

: أظهرت النتائج أن أغلب أفراد العينة (50%) كانت أعمارهم تتراوح بين 18–32 سنة، بينما بلغت نسبة الفئة العمرية 33–47 سنة نحو 37.5%. كما تبين أن نسبة الذكور المصابين كانت أعلى من الإناث (55% مقابل 45%). وأظهرت الدراسة أن معظم المشاركين لا يعانون من أمراض مزمنة بنسبة 57.5%. تراوحت مستويات الكورتيزول لدى مرضى متلازمة كوشينغ ضمن مدى واسع مقارنة بالقيم الأقل تشتتاً لمؤشر كتلة الجسم. كذلك، كان مستوى الكورتيزول الصباحي أعلى معنوياً من المستوى المسائي ($p < 0.001$). كما وُجد ارتباط إيجابي قوي بين مستويات الكورتيزول ومؤشر كتلة الجسم ($r = 0.786$, $p = 0.001$)، مما يشير إلى أن ارتفاع مؤشر كتلة الجسم يرتبط بارتفاع مستويات الكورتيزول.

الاستنتاجات:

تشير نتائج الدراسة إلى وجود علاقة ارتباط إيجابية قوية بين مستويات الكورتيزول ومؤشر كتلة الجسم، مما يدل على أن السمنة لدى مرضى متلازمة كوشينغ ترتبط بارتفاع مستويات الكورتيزول.

الكلمات المفتاحية :**الكورتيزول، مؤشر كتلة الجسم، متلازمة كوشينغ، السمنة.**