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ORIGINAL ARTICLE

Hormonal and Metabolic Alterations in Women with Endocrine Disorders in Southern Iraq

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ABSTRACT

Conditions such as hyperprolactinemia, obesity, and type 1 diabetes mellitus (T1DM) cause metabolic and endocrine alterations that affect the reproductive and metabolic functions of women. These conditions coexist with changes in sex hormones, glucose metabolism, vitamin D, and specific intracellular signaling pathways, indicating the necessity for integrated hormonal studies in these conditions. The purpose of this study was to evaluate and compare the hormonal, metabolic, and molecular biomarkers of women with hyperprolactinemia, obesity, and T1DM, and healthy controls, with particular focus on sex hormones, glucose levels, vitamin D, and the β-catenin pathway. A cross-sectional comparative study design was implemented with women with hyperprolactinemia, obesity, or T1DM, and age-matched healthy controls. The serum levels of prolactin, gonadotropins (FSH, LH), the hypothalamic releasing hormone GnRH, sex hormones (estradiol, progesterone, testosterone), fasting blood glucose, vitamin D, and β-catenin were measured using standard immunoassay and biochemical techniques. Statistical analysis was applied, and results were presented as mean ± standard deviation. Statistical significance was set at $p < 0.05$. Women with hyperprolactinemia had significantly altered sex steroid profiles compared with controls. Among women with obesity, the combination of hyperglycemia and altered sex steroid profiles contributed to obesity. The T1DM group exhibited significant hyperglycemia and estradiol deficiency that corresponded to impaired metabolic and reproductive hormone interactions. All groups confirmed a prevalent deficiency of vitamin D, but the T1DM group demonstrated the most severe deficiency. In the metabolically compromised groups, decreased serum β-catenin levels were observed, which may indicate reduced β-catenin signaling; however, serum β-catenin does not necessarily reflect intracellular Wnt/β-catenin activity. The study concludes that hyperprolactinemia, obesity, and T1DM in women are associated with several different but interrelated metabolic and hormonal changes. The interrelated changes in the metabolic and endocrine systems are illustrated by the changes in sex hormones, vitamin D, and β-catenin levels. There is a need for an integrated approach in the diagnostic evaluation of the metabolic and hormonal systems to improve the clinical management of women with hyperprolactinemia, obesity, and T1DM.

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1- INTRODUCTION

Endocrine disorders affect millions of people across the world. For women, the effects can be even more detrimental because of their interactions with the hormonal, metabolic, and reproductive systems. The endocrine system plays a pivotal role in maintaining physiological balance. It consists of numerous glands that release hormones directly into the bloodstream. These hormones control metabolism, growth, stress response, and reproduction [1]. Disruption of this system can lead to the development of severe chronic conditions. Endocrine system disorders are exacerbated in women because of the hormonal cycles they experience during puberty, menstruation, pregnancy, and menopause. These life phases often impact, uncover, or worsen endocrine disorders like polycystic ovary syndrome (PCOS), thyroid disorders, diabetes mellitus, and metabolic syndrome. In women, hormonal disorders can lead to a number of metabolic disorder consequences like insulin resistance, dyslipidemia, imbalanced adipokine secretion, and chronic low-grade inflammation. These effects illustrate how vital the close relationship between the endocrine and metabolic systems is [4,5]. Among women of reproductive age, polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine disorders, with worldwide prevalence estimates ranging between 6% and 20%, depending on population-based sample selection and diagnostic criteria [6,7]. In addition to obesity and insulin resistance, PCOS is defined by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Impaired glucose tolerance, type 2 diabetes mellitus, and heightened incidence of cardiovascular disease are some of the metabolic consequences of PCOS. Insulin resistance has been established as a fundamental cause of the pathophysiology of PCOS [8,9]. Another important group of endocrine dysfunctions in women is thyroid disorders, with hypothyroidism being the most common. Thyroid hormones are important regulators of the basal metabolic rate, lipid metabolism, and energy expenditure. Therefore, a small change in thyroid hormone levels can cause disturbances in the metabolic system, resulting in weight gain, increased concentration of lipids in blood, lethargy, and menstrual cycle irregularities. Hashimoto's thyroiditis is one of the most common forms of autoimmune thyroid disease and is more common in women [10,11]. In addition to thyroid disorders and PCOS, metabolic syndrome is becoming a major public health concern in women worldwide. Metabolic syndrome is comprised of a cluster of conditions linked to metabolism like obesity, insulin resistance, hypertension, hyperglycemia, and dyslipidemia. The hormonal changes associated with menopause, particularly the lower levels of estrogen, are accompanied by adverse changes in body fat distribution and increased cardiac and metabolic risks in women [12,13].

In the past two decades, the Middle East and North Africa (MENA) region has seen rapid urbanization, and the most significant increase in the world concerning endocrine and metabolic diseases. An increase in the consumption of severely processed, calorically dense, and nutritionally void food, coupled with decreased physical activity, has led to increased obesity and diabetes in the MENA region. Very few studies have investigated endocrine and metabolic diseases in women in Southern Iraq [14]. Lack of access to adequate secondary and tertiary healthcare, food availability, and the psychological stress of living in a war zone are likely to contribute to a haphazard dysregulation of the hypothalamus/pituitary/adrenal axis, leading to altered levels of hormones and metabolic derangement [15]. Another factor for decline in endocrine health in southern Iraq is toxic exposures due to industries, oil extraction, and inefficient waste disposal systems. There is a high presence of many heavy metals and other endocrine disrupting chemicals (EDCs) such as lead, cadmium, and mercury, which impact the biosynthesis and secretion of hormones, as well as their receptors and signaling pathways. EDCs can cause dysregulation of the endocrine system by disrupting thyroids; in addition, they can alter levels of estrogens and androgens by impairing insulin signaling, which then leads to metabolic dysfunction [16,17].

The occurrence of vitamin D deficiency among women in Iraq seems paradoxical, considering that there are abundant opportunities for solar exposure. Deficiencies in vitamin D can impact the secretion of insulin, alteration of immune responses, and the regulation of some reproductive hormones. Several studies have confirmed low levels of vitamin D as a contributing factor for the development of PCOS, insulin resistance, and metabolic syndrome, indicating the connection of certain micronutrients and endocrine disorders [18]. The balance between hormonal and metabolic issues is critical to the reproductive health of women. Disruptions to the menstrual cycle or complications in achieving/conceiving pregnancy may occur due to endocrine ailments such as diabetes mellitus and thyroid disorders. The prevalence of gestational diabetes mellitus (GDM) has substantially increased in recent years. GDM has adverse health consequences for both the mother and the child. Mothers with GDM are at increased risk for developing type 2 diabetes, and children of mothers with GDM are at increased risk for developing obesity and metabolic disorders as a result of epigenetic influences [19,20].

An increase in endocrine and metabolic disorders has been noted in women in southern Iraq, while clinical and epidemiological information is lacking. Previous studies do not have comprehensive metabolic and hormonal profiling and are restricted by site and sample size. The absence of relevant literature is a major factor in the formulation of prevention and management of diseases in the region. To implement effective public health policies, it is essential to understand the disease burden and the distributions of the public health problems in different regions [21]. Women face sociocultural barriers that affect health-seeking and disease management. Stigma around reproductive and hormonal disorders, diagnosis, and treatments is prevalent. The absence of subspecialty endocrine care and financial constraints exacerbate the situation. Integrated clinical, health systems, and education approaches will be essential to overcome the barriers to care [22]. Fertility and metabolic health are managed with hormonal therapy and improved lifestyle such as diet, physical activity, and weight management. Health systems and prompt, early diagnosis and treatment improve the outcomes of lifestyle modifications, and in some cases use of insulin sensitizers and hormone replacement therapy [23]. For women, endocrine disorders can lead to significant changes in metabolism that impact their reproductive health. In southern Iraq, multiple environmental, dietary, economic, and health variables further complicate the situation. We urgently need to develop structured studies to assess the range of hormonal, metabolic, and regional negative profiles to provide targeted improvements for women in this area.

2. MATERIALS AND METHODS

This study used a cross-sectional design at a single point in time over a duration of 9 months, ranging from June 2022 to February 2023, in the Dhi Qar Governorate of Iraq. Participants were selected from different health facilities, including Al-Sadr Teaching Hospital, Maternity & Children's Hospital, Specialized Center for Diabetes & Endocrinology, in addition to a number of private clinics and health centers. Two hundred women in the study were aged between 20 and 40 years. Participants were divided into four equal groups of 50 women based on clinical parameters. Group I (Control) consisted of healthy women with regular menstrual cycles and no history of hormonal disorders. Group II (Hyperprolactinemia) comprised women who had been diagnosed with hyperprolactinemia by fertility and gynecology specialists. Group III (Obesity) consisted of women who were classified as obese with a body mass index (BMI) over 30 kg/m². Group IV (Type 1 Diabetes Mellitus, T1DM) comprised women who had been diagnosed with T1DM by endocrinologists based on clinical history and examination. Some participants were receiving prescribed hormonal therapy; this was recorded and considered as a potential confounder in the analysis.

2.1 Inclusion criteria

Hyperprolactinemia was defined as serum levels of prolactin > 25 ng/mL. Obesity was classified as a BMI ≥ 30 kg/m². Diagnosis of T1DM was made based on the American Diabetes Association (ADA) criteria.

2.2 Sample collection

All blood samples were drawn in the morning, after an overnight fast (8 hours or more) to minimize circadian and postprandial effects. Premenopausal women: Samples were taken in the early follicular phase (days 2–5 of the menstrual cycle).

2.3 Assay validation and reference ranges

All hormone assays were done with commercial immunoassay kits per manufacturer instructions and validated, and all reference ranges were provided by the manufacturer. The reference ranges were: prolactin 4.8–23.3 ng/mL; FSH 3.5–12.5 mIU/mL; LH 2.4–12.6 mIU/mL; estradiol 12.5–166 pg/mL; progesterone 0.2–1.5 ng/mL; testosterone 0.08–0.48 ng/mL; vitamin D 30–100 ng/mL; and β-catenin as specified by the manufacturer of the kit.

2.4 Sample size

The sample size was calculated using software G*Power (version 3.1) with $\alpha = 0.05$, power = 0.80, and an expected effect size of 0.25. To ensure that sufficient numbers of participants were available, 50 participants were recruited per group, with a minimum of 45 participants required per group. The exclusion criteria removed individuals from the control group with polycystic ovary syndrome (PCOS), irregular menstrual cycles, thyroid disorders, and BMI >25 kg/m². Standardized blood samples were taken from all participants in the morning after an overnight fast to minimize circadian and postprandial effects. These samples were used to determine the serum levels of the hormones and metabolic markers: kisspeptin, gonadotropin-releasing hormone (GnRH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, estradiol, progesterone, testosterone, and glucose.

Hormone concentrations were expressed as ng/mL or mIU/mL, as applicable, and glucose levels as mg/dL. For the four groups, the data collected were summarized as means ± standard error. One-way ANOVA was performed to compare group means, followed by the Least Significant Difference (LSD) post-hoc test where ANOVA was significant. Multivariate analysis (ANCOVA) was used to adjust for potential confounders including age, BMI, and duration of disease. Correction for multiple comparisons was applied using the Bonferroni method. Effect sizes were reported as partial eta squared (η^2). Statistical significance was set at $p < 0.05$.

3. RESULTS AND DISCUSSION

Table 1 and Figures 1-6 demonstrate the hormonal and metabolic factors present for the four study groups. Findings indicate the presence of endocrine and metabolic factors associated with hyperprolactinemia, obesity, and type 1 diabetes mellitus, which are distinctive from the healthy control group.

Table(1): Analysis of Different Sample Groups and Their Relative Hormonal Concentrations (ng/mL)

Hormones	Control	Hyperprolactinemia	Obesity	Type one diabetes	LSD
Kisspeptin(ng/ml)	12٥.١5±2.١٨	12٩.١9±1.٩٠	12٢.50±2.٢٢	12١.٦8±2.٦2	0.72
GnRH(ng/ml)	0.6٣±0.01	0.٧١±0.02	0.6٥±0.0٢	0.٦٩±0.0٢	0.07
FSH (mIU/ml)	6.٧3±0.3٠	6.٥9±0.3٠	6.٣5±0.3٣	6.٦5±0.3٢	0.04
LH (mIU/ml)	5.٢٢±0.4٠	5.١١±0.3٢	4.٣٢±0.٢7	٤.٨٩±0.32	0.56
Prolactin(ng/ml)	11.٤٥±0.9٢	4٦.٩٩±4.١٠	1٢.٠١±0.8٨	1٤.٦6±1.6٥	1.78
Estradiol(pg/ml)	2٨.٠5±1.83	٥٢.٩7±6.32	٤٠.74±4.18	2٤.٠1±1.98	6.23
Progesterone(pg/ml)	0.٣1±0.0٢	1.٣2±0.07	0.9٨±0.0٨	0.9٩±0.0٧	0.52
Testosterone(ng/ml)	0.1٢±0.02	0.2٢±0.03	0.3٤±0.03	0.2٣±0.03	0.24
Glucose(mg/dl)	9٥.٩4 ±2.٢3	94.٨8±2.٣8	9٩.٠1±٤.٩٩	28١.2١±2٣.47	3.98
vitamin D(ng/mL)	32.51 ± 2.54	16.45 ± 3.88	15.95 ± 3.94	13.41 ± 3.04	2.31
β-catenin (ng/mL)	0.35 ± 0.08	0.31 ± 0.07	0.29 ± 0.06	0.23± 0.05	0.01

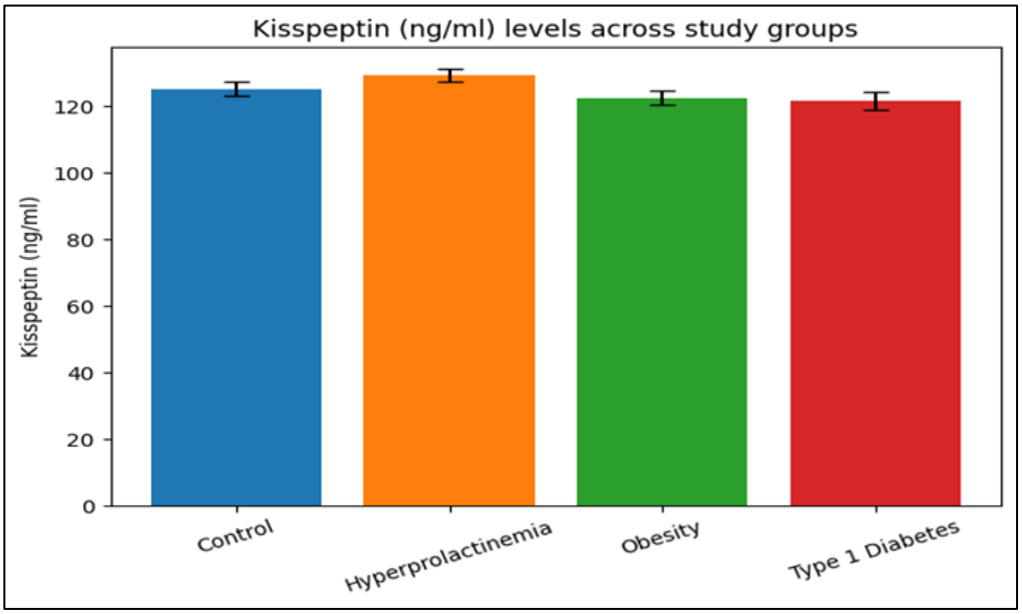

Fig (1): Levels of Kisspeptin in serum for control and endocrine disorder groups

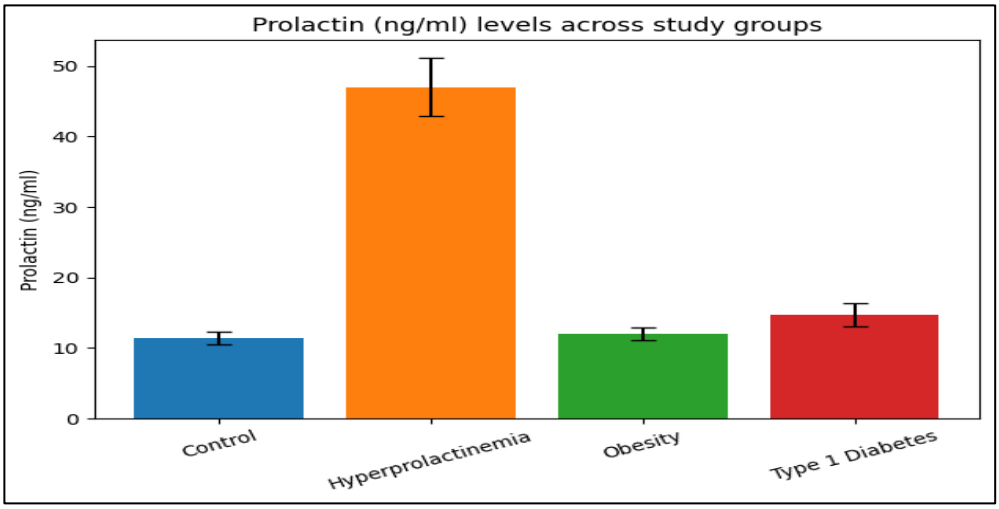

Fig (2): Levels of Prolactin in Serum of Various Study Groups

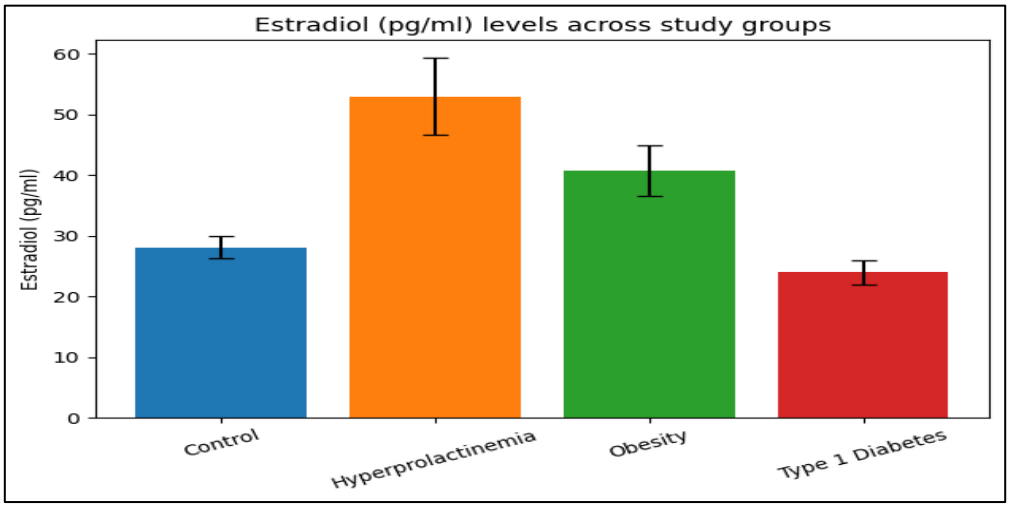

Fig (3): Levels of estradiol (pg/ml) in females with endocrine disorders

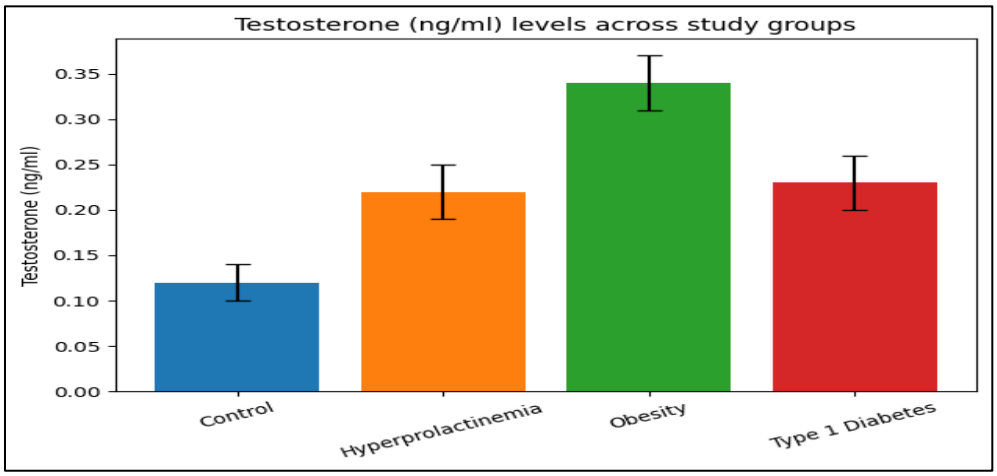

Fig (4): Changes in Testosterone levels in control vs. patient groups

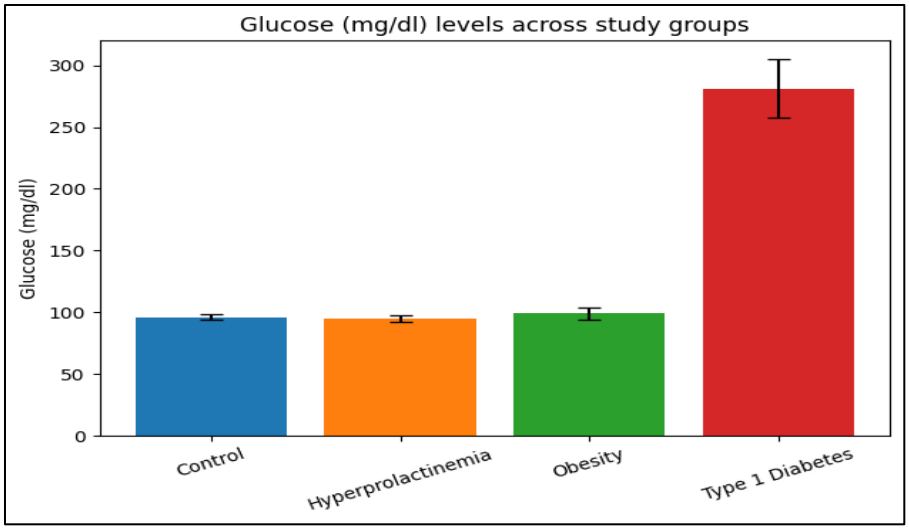

Fig (5): Blood glucose levels after fasting in control women and women with Type 1 diabetes

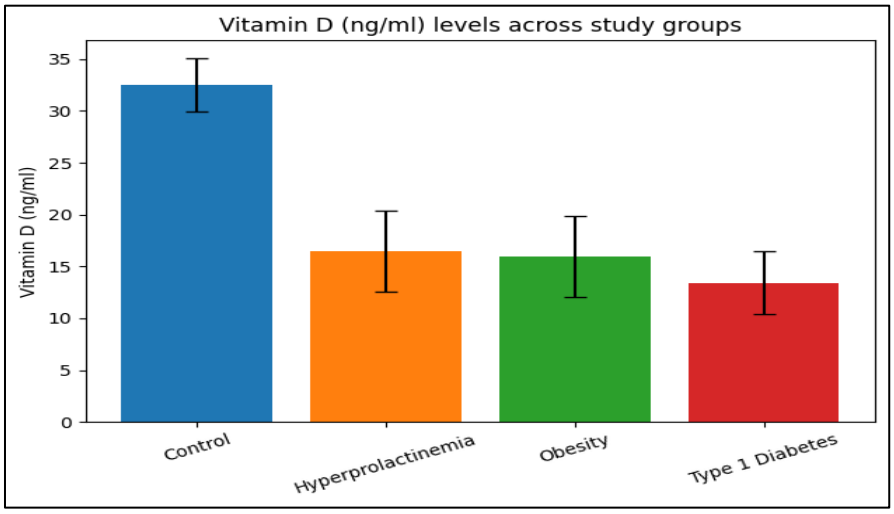

Fig (6): Study group classification versus concentrations of Vitamin D

Differences in kisspeptin levels may explain some discrepancies among groups (Figure 1). Obese and type 1 diabetic women have more pronounced kisspeptin losses, while hyperprolactinemic women showed a small increase in kisspeptin concentration compared to control women. However, due to the narrow range, variations suggest reductions in kisspeptin signaling in all the studied endocrine disorders. The concentration of GnRH also showed variability among the groups, but the groups of hyperprolactinemia and type1 diabetes have a slightly higher concentration than their control counterparts, whereas the levels of GnRH in the obese women are comparable to the levels of GnRH in the healthy control women. Peripheral GnRH levels showed limited variability, suggesting minimal differences in overall secretion among the groups of the population under study. The levels of FSH and LH showed a marked variation compared to GnRH variation, but the magnitude of FSH and LH variance was low. LH levels in the obesity group were more pronounced than in controls. For the patients with hyperprolactinemia, serum prolactin levels increased (Figure 2) relative to other patients. Prolactin levels in obese and type 1 diabetic women, however, resembled the levels in the control women. Despite this, hyperprolactinemia in Group II was indicated by prolactin levels that were within a narrow range. Estradiol levels across the different groups were not uniform (Figure 3). Of the hyperprolactinemic women, obese women had the highest estradiol levels, whereas type 1 diabetic women had lower levels in comparison to control women. This suggests variability in ovarian steroidogenic activity due to a combination of changes in the endocrine and metabolic systems. In terms of progesterone, the control groups had the lowest levels of progesterone, with hyperprolactinemic women having the highest levels and most significant increase. Obese women and type 1 diabetic women had similar levels of progesterone, which suggests altered dynamics of luteal hormones in the endocrine and metabolic disorders.

Testosterone levels progressively increased in hyperprolactinemic and control women, with obese women having the highest levels (Figure 4). In comparison to control women, type1 diabetic women had lower levels of testosterone. These findings suggest increased activity of androgens, especially in association with obesity. Having the highest levels of fasting blood glucose in comparison to the other groups was type 1 diabetes (Figure 5). For hyperprolactinemic and obese women, we recorded the same blood glucose levels as in the control group, which confirms the proper glycemic discrimination within the groups in this study. The markedly elevated fasting glucose in the T1DM group (281.21 mg/dL) indicates poor glycemic control and is discussed as a limitation of the study. Control, type 1 diabetic women, and the obese and hyperprolactinemic women have been documented to have the lowest serum vitamin D concentrations (Figure 6). This pattern shows a greater vitamin D deficiency across all endocrine and metabolic disorders. In terms of the progressive decrease in the concentration of β-catenin, the women with type 1 diabetes have the lowest of all groups. The fact that all groups have a gradual decline indicates metabolic dysregulation and a reduction in β-catenin signaling. The study demonstrates the distinct consequences of hormonal and metabolic changes associated with hyperprolactinemia, obesity, and type 1 diabetes mellitus, given the changes in levels of β-catenin, vitamin D, glucose, prolactin, and sex steroid hormones compared to the healthy controls.

Recent findings demonstrate the close intersection between endocrine and metabolic disorders and their profound effects on the reproductive and metabolic homeostasis of women. This study examines and details the hormonal and metabolic alterations in women suffering from hyperprolactinemia, obesity, and type 1 diabetes, and the changes involved in contrast to those of healthy individuals. This study supports the concept of hyperprolactinemia, obesity, and type 1 diabetes as forms of endocrine dysregulation, encompassing more than hormonal alterations, but rather demonstrating disrupted homeostasis [24,25]. There was hyperprolactinemia in the study, and it was explained by the presence of increased serum prolactin. This demonstrates the clinical picture and how it corresponds to the loss of inhibitory influence of dopamine at the level of the anterior pituitary lactotrophs. It is a fact that hyperprolactinemia is associated with inhibition of the pulsatile secretion of GnRH, and as a consequence, is the cause of subsequent changes in the downstream gonadotropins and sex steroids [26,27]. Although the current cohort showed only marginal changes in peripheral gonadotropin levels, the marked increase in progesterone and estradiol indicates changes in ovarian steroidogenesis, most probably resulting from prolactin-driven increases in ovarian sensitivity to gonadotropins. Clinical and experimental studies report similar endocrine patterns in hyperprolactinemia [28,29]. When associated with obesity, the most notable alterations are observed in the sex steroid hormones, especially with regard to the increase in testosterone and estradiol. Adipose tissue produces mediators and adipokines that promote inflammation and also modulate the HPG axis and peripheral steroid metabolism [30,31]. In women with obesity, the higher levels of estradiol are caused by higher rates of aromatization in the adipose tissue, which converts androgens into estrogens. Obesity-associated endocrine dysfunction results in less steroid clearance and increased androgens [32,33]. The impact of increased testosterone levels in women with obesity is significant. Insulin resistance, reproductive dysfunction, and dyslipidemia are all conditions that are associated with excess androgens. Hyperandrogenism, i.e., having excessive levels of androgens, has been clinically observed to decrease insulin sensitivity and increase visceral obesity, which worsens the metabolic disorder. This results in a cycle of metabolic and hormonal dysregulation [34,35]. Women with type 1 diabetes mellitus (T1DM) showed greater metabolic disturbances, demonstrated by markedly elevated fasting glucose levels. Insulin deficiency not only disrupts glucose homeostasis but also negatively influences ovarian activity and the metabolism of sex hormones. Insulin is a co-gonadotropin in the process of ovarian steroidogenesis, and its absence and/or dysregulation can lead to dysregulated synthesis of estrogens and androgens [36,37]. The lower estradiol (E2) levels in the T1DM group may reflect the lower ovarian response as well as the altered hepatic metabolism of sex steroid hormones.

In all the patient groups studied, and particularly in women with T1DM, the levels of vitamin D were found to be very low. The nuclear vitamin D receptor (VDR) is expressed in numerous tissues, including pancreatic β-cells, ovaries, and the hypothalamus, where it modulates endocrine and metabolic functions [38,39]. These deficiencies have been proved to be associated with insulin resistance, impaired glucose tolerance, and altered reproductive hormones. Vitamin D deficiency is very common in this cohort, and in all the studies conducted both locally and in distant locations. Deficiency of vitamin D is likely to contribute to the endocrine and metabolic disorders [40, 41]. In this study, a clear and stepwise decline in the levels of β-catenin was observed across the different categories of endocrine disorders, especially in women with T1DM. β-catenin is a key player in the Wnt signaling pathway, which is crucial for metabolism, insulin signaling, and reproductive tissue homeostasis. There is growing recognition of the disruption of the Wnt/β-catenin pathway as a cause of metabolic disorders and the disruption of the endocrine system in the existing scientific literature. The literature, however, has not fully addressed this issue.

The decline in β-catenin could suggest a more severe metabolic stress and disturbance of intracellular signaling. However, serum β-catenin does not necessarily reflect intracellular Wnt signaling activity, and this finding should be interpreted with caution. Most patient groups also show some degree of preservation of GnRH, FSH, and LH, which indicates that pituitary function, is intact and the endocrine dysfunctions are attributed to changes in the hypothalamus and/or shifts in the oxidation of peripheral hormones. These findings highlight the complexity of endocrine dysfunctions and how the slightest change in regulation can have far-reaching consequences on metabolism and reproduction. This research shows that hyperprolactinemia, obesity, and type 1 diabetes mellitus are associated with distinct hormonal configurations which ultimately produce the same adverse metabolic outcomes in women, negatively impacting reproductive health, increasing metabolic risk, and exacerbating chronic disease. Moreover, understanding these processes is crucial for developing appropriate diagnostic and therapeutic strategies for women with endocrine disorders.

4. CONCLUSION

Hormonal and metabolic dysregulation are present in hyperprolactinemia, obesity, and type 1 diabetes mellitus (T1DM). The hyperprolactinemia condition has an increase in prolactin and an altered balance in sex steroids. Obesity is associated with increased peripheral endocrine activity and elevated circulating testosterone and estradiol. For T1DM, there is hyperglycemia and decreased estradiol, which indicates an issue with the cross-talk between the metabolic and reproductive systems. Women participants in the study demonstrated vitamin D deficiency. Women with T1DM had the greatest deficiency in vitamin D. β-catenin deficiency may indicate involvement of the Wnt/β-catenin signaling pathway in the pathology of endocrine disorders of the participants. The results demonstrate the complexity of intertwined conditions in metabolism, hormones, and signaling pathways in the studied participants with endocrine disorders. These results support an integrated assessment approach encompassing metabolic, hormonal, and sub-cellular parameters. There are a number of limitations with this study. The first problem with the cross sectional design is inference of causality. Secondly, some of the participants in this study were treated with hormonal therapy, which may have affected the measured hormonal parameters. Third, fasting glucose was significantly higher in the T1DM group (281.21 mg/dL) suggesting poor glycemic control and potentially affecting other parameters studied. Fourth, the serum level of β-catenin was measured, but this might not reflect the activity of intracellular Wnt/β-catenin signaling. Fifth, the measurement of GnRH was done peripherally and not necessarily a reflection of hypothalamic secretion. Sixth, the number of samples was relatively small and multiple comparisons were carried out, but the Bonferroni method was used to correct for multiple comparisons. Seventh, control of lifestyle factors, such as dietary habits, physical activity and smoking, was not achieved. Lastly, the results might not be applicable to other areas outside of Southern Iraq.

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