GMR
The Original

ENDOCRINE DISORDERS AND KIDNEY HEALTH: EXPLORING HORMONAL PATHWAYS IN RENAL DYSFUNCTION

Dr. Pranav Mallick[1*], Dr.Mohammad Shuaib[2], Nithi Doley [3], Dr. Himanshu Jha[4], Dr. Jwalit Jatinkumar Mehta[5], Dr. Rashi Sharma[6]

[1*] On Duty Medical Officer at New Life Diagnostics, Sri Vijayapuram, (MBBS) Pondicherry University, ORCID ID: 0009-0007-5940-7795, Email ID: pranavmallickpb@gmail.com
[2]Consultant Urology Diwan Hospital and Urology Centre G.T Road, Sujru Chungi, Muzaffarnagar, Uttar PradeshORCID ID: 0000-0002-7857-8481 Email ID: dr.mhdshuaib@gmail.com
[3]Associate Professor, Department of Pathology, MD Pathology, Takshashila Medical College ORCID ID : 0009-0003-8579-6553 Email ID: ndley47@gmail.com
[4]Resident Doctor, Department of Family Medicine, Down Town Hospital Guwahati, Specialization in Family Medicine Email- Drhj37@gmail.com, ORCID iD: 0009-0002-0985-5032
[5]Professor, Department of Physiology, Dr. N. D. Desai Faculty of Medical Science and Research, Dharmsinh Desai university, Nadiad, Gujarat, India Email ID: adms.medical@ddu.ac.in
[6]Associate Professor, Department of Kriya Sharir, Faculty of Ayurveda, IMS BHU Varanasi Email ID:drrashisharma21@gmail.com

ABSTRACT
Chronic kidney disease (CKD) is a progressive disease, which impacts on the functioning of the kidneys and usually ends up causing end-stage renal disease (ESRD). The CKD is often linked to endocrine diseases, such as thyroid dysfunction, diabetes, and adrenal malformations. It is believed that these hormonal imbalances have a role to play in the deterioration of the kidney functions. Still, the contribution of particular endocrine pathways to the development of CKD has not been studied thoroughly. Hormonal profiling was done to determine aldosterone and cortisol, thyroid hormones (T3, T4) and sex hormones (testosterone and estrogen). The renal functioning was measured with serum creatinine, glomerular filtration rate (GFR) and urinary albumin to creatinine ratio (UACR). The statistical analysis was conducted based on Pearson correlation model and multiple regression model in order to assess the relationship between hormonal imbalances and renal functioning. There were also negative correlations between GFR and thyroid hormones (T3, T4). The most common abnormalities in hormones were elevated aldosterone and cortisol, with 68 and 62% of CKD patients experiencing the abnormalities, respectively. Also, fifty % of male CKD patients had low testosterone levels. Endocrine dysfunction has a huge role to play in the development of CKD. An increase in aldosterone, cortisol and thyroid dysfunction is related to the progression of renal biopsy and increased incidence of complication. These results can be used to argue that hormonal imbalances need to be included among critical therapeutic areas in the management of CKD to enhance patient outcomes.

KEYWORDS: Chronic kidney disease, Endocrine dysfunction, Aldosterone, Cortisol, Testosterone

1. INTRODUCTION
The kidneys remain vital organs that ensure the body is in a state of homeostasis, owing to their role in balancing fluids, electrolytes, blood pressure in addition to excretion of metabolic waste. Chronic kidney disease (CKD) is a progressive disease that is described by the deterioration of the renal functionality, and the prevalence rates of the condition continue to rise worldwide [1]. In CKD, due to the failure of the kidney, one will experience metabolic wastes that include urea and creatinine, which remain usually released in the urine. This gradual deterioration in kidney functioning may result in the end-stage renal disease (ESRD) that will demand either dialysis or kidney transplantation to survive [2]. Also, CKD is usually linked to a number of metabolic and endocrine conditions, which complicates its clinical treatment even more [3]. The pathophysiology of CKD implies the complex interactions of genetic, environmental, and clinical factors. Another important fact about the CKD progression is the lack of control of metabolic functions, such as glucose metabolism, lipid homeostasis, and blood pressure regulation, which remain also conditioned by endocrine disruptions [4]. With the course of the disease, cardiovascular disease, obesity and diabetes remain some of the comorbid conditions that patients develop with and this has made their kidney damage worse in addition to the overall deterioration of the kidney function [5].
The endocrine disorders substances contribute to the pathogenesis and development of CKD. Renal dysfunction is predisposed to hormonal imbalances, the kidneys and endocrine system is also bidirectionally related, which in turn may worsen the performance of kidneys. The hormonal pathways that remain affected in CKD remain the hormonal pathways that control the fluid balance, glucose metabolism and the levels of sex hormones. Some of the most frequent endocrine disruptions in CKD include dysfunctions of the hypothalamic-pituitary-thyroid axis, gonadal hormones and renin-angiotensin-aldosterone system (RAAS) [6]. Thyroid dysfunction has been specifically high in CKD and both hypo- and hyperthyroid dysfunction have a negative impact on renal functioning. Thyroid hormones affect the hemodynamics of the kidney, the glomerular filtration rate (GFR) and the tubular activity, and their disproportions may worsen kidney disease

[7]. Additional, dysfunction of the gonads, particularly those in men, is also a frequent endocrine defect in CKD, causing reduction in testosterone levels, which can also result in the deterioration of renal and general health [8]. Aldosterone is one of the main hormonal regulators that play a role in the health of the kidney. In CKD, sodium retention, hypertension, and kidney fibrosis remain increased due to excessive secretion of aldosterone, which aggravates the course of the disease [9]. The dysregulation of cortisol, commonly seen in CKD caused by a defective glucocorticoid metabolism leads to heightened sodium retention, fluid overload, and hypertension, which only worsens the renal damage [10]. Adipose tissue also has an effect on the relationship between endocrine disorders and renal dysfunction. Adipose tissue, which is regarded as one of the major endocrine organs, releases a number of hormones, such as leptin, adiponectin, and resistin, which remain demonstrated to influence the functioning of kidneys. Obesity is a comorbid disease that is closely related to CKD and involves enhanced adipose-derived hormone secretion that may exacerbate kidney damage by regulating inflammatory and fibrosis signaling [11]. Also, recent evidence reports that gut dysbiosis of CKD patients could affect neurohormonal pathways, which subsequently affect kidney performance due to their involvement in complicated effects of adipokines and inflammatory signatures [12].

The hormonal pathways remain important in the control of many facets of kidney operations such as sodium equilibrium, glomerular filtration and renal blood flow. There remain various hormones that maintain kidney homeostasis, these include mineralocorticoids, glucocorticoids in addition to sex hormones [13]. The mineralo corticoids like aldosterone controls the sodium and potassium levels by determining the water retention and blood pressure by acting on the renal tubules. Overproduction of aldosterone in CKD increases the retention of the fluid, which increases blood pressure and enhances kidney fibrosis [14]. The effect of glucocorticoids on kidney is also well-reported. The adverse effect of the excessive levels of glucocorticoids, caused by stress or the side effect of corticosteroid treatment, is the retention of sodium and the encouragement of hypertension that leads kidney damage in CKD patients [15]. Conversely, lack of adequate cortisol activity may hinder kidney functioning such as witnessed in diseases such as Addison disease [16]. It is also important to know the exact processes through which glucocorticoids influence the functioning of kidney to facilitate the use of the best treatment plans in CKD. Kidney also functions significantly of sex hormones. Some of these effects include protection of the kidney to estrogen, which has been depicted to curb inflammation and improve blood flow to the kidney, and similarly, excess testosterone may worsen kidney damage via inflammatory and fibrotic processes [17]. The sex differences in hormonal action on the kidney activity exemplify the necessity of individual approaches to treatment, particularly in patients with CKD who have hormonal disproportions [18].

The other significant factor that affects renal health is the connection between obesity and kidney function. The endocrine processes of the adipose tissue such as the leptin and adiponectin secretions have been suggested in regulating the renal functioning. These hormones affect renal vascularity, glomerular filtration and sodium. Obesity has also been demonstrated to increase kidney damage due to its elevated fat and adipocyte-derived hormones which contribute to cancer development, thus making obesity a significant risk factor in CKD development [19]. Well-compounded hormonal control in kidney disease is becoming more obvious. Hormonal defects including imbalances of aldosterone, cortisol, and sex hormones remain also involved in the pathogenesis of CKD and CKD complications. Moreover, participation of adipose tissue and microbiota in gut in adjusting the functioning of kidneys underlines the necessity of extensive approaches that consider various endocrine pathways in management of CKD. Innovations in the study of these hormonal processes have a future in the creation of specific treatments to reduce kidney damage and patient outcome. Finally, endocrine diseases play an important role in the dysfunction of kidneys and development of CKD [20]. Such hormonal alterations as the Renin-Angiotensin-Aldosterone System (RAAS), thyroid axis, gonadal hormones cause changes in kidney functioning, such as fibrosis, hypertension, and fluid retention. A mutual dependence of the endocrine system and kidneys highlights why combined treatment regimens should be used to correct renal and endocrine problems. It is necessary to learn more about these complicated interactions to create more effective therapeutic interventions to treat CKD patients.

Objectives of the study

1. To examine the role of endocrine disorders in the progression of chronic kidney disease (CKD) through key hormonal pathways.
2. To assess the impact of hormonal imbalances on renal function and complications in CKD patients.

2. MATERIALS AND METHODS

2.1 Study Design and Population

The study has used a cross-sectional design to examine the connection between endocrine conditions and chronic kidney disease (CKD). One hundred and fifty patients who were diagnosed with different levels of CKD were recruited in a tertiary care hospital. The partakers had been chosen on the basis of medical history and clinical diagnosis of renal dysfunction, which was established by the nephrologists. The sample size was 26 of both sexes who had the age of 18-75 years of age and represented different ethnic groups and economic statuses. All subjects were informed and gave informed consent before they were included in the study and the institutional review board approved the study.

2.2 Inclusion and Exclusion Criteria

They included adults between 18-75 years that had a confirmed CKD diagnosis on the basis of a GFR of less than 60 mL/min/1.73m2 at least three months. Endocrine patients such as thyroid disorders, diabetes and adrenal disorders were taken to determine in what way hormonal imbalance affects renal functioning. The exclusion criteria were patients with acute kidney injury, malignancies, autoimmune disorders or individuals who remain receiving active cancer treatment.

Severely psychiatric patients and pregnant women were also eliminated, to eliminate confounding factors that may interfere with hormonal and renal measurements.

2.3 Diagnostic and Measurement Tools

Renal functioning was evaluated by serum creatinine, glomerular filtration rate (GFR) and urinary albumin-to-creatinine ratio (UACR), which remain conventional diagnostic parameters of CKD severity. The levels of aldosterone, cortisol, thyroid hormones (T3, T4, and TSH), and sex hormones (testosterone and estrogen) were measured on the basis of blood samples to conduct hormonal profiling. High sensitivity enzyme-linked immunosorbent assays (ELISA) were used to analyze these biomarkers. Also, blood pressure, weight and renal ultrasound imaging were used to determine the existence of hypertension, obesity and structural changes of kidneys which remain typical complications of CKD.

2.4 Hormonal Profiling and Renal Function Assessment

Aldosterone, cortisol, thyroid hormones, and gonadal hormones (testosterone, estrogen) levels were taken and highlighted as hormonal imbalances. This was accompanied by a detailed hormonal profile that was used to estimate the correlation between these imbalances and renal function. The samples of blood were taken in the morning time following a fast of an overnight period to reduce the effects of diurnal variation in the level of hormones. The renal functions were examined via the calculation of GFR using the level of serum creatinine and the albumin excretion was measured using the urine samples. The correlations between the hormonal markers and GFR were performed to identify the effect of the hormonal markers on CKD progression.

2.5 Statistical Analysis

The analysis of data was done with the help of SPSS. Demographic and clinical variables were used in calculating descriptive statistics, such as means and standard deviations and frequencies. Correlation coefficient of Pearson and linear regression were used to analyze the relationship between hormonal imbalances and renal functioning. Several regression models were used to adjust the variables that could confound the research (e.g., age, sex, comorbid conditions e.g., diabetes and hypertension). The statistical significance was $p = 0.05$. The findings were reported with 95 % confidence intervals and relevant post hoc tests were conducted in order to evaluate pair wise comparisons where they were essential.

3. RESULTS

3.1 Endocrine Biomarkers and Kidney Function Correlations

Relationship between the major endocrine biomarkers and glomerular filtration rate (GFR) in chronic kidney disease (CKD) patients. There were significant negative relationships found with aldosterone (-0.45, $p = 0.01$) and cortisol (-0.38, $p = 0.05$) which suggests that increased levels of these hormones correlate with poorer functioning of the kidneys as presented in Table 1. There were also negative correlations between thyroid hormones, T3 ($r = -0.42$, $p < 0.01$) and T4 ($r = -0.41$, $p < 0.01$) and GFR, indicating that thyroid failure may be a contributing factor of renal failure. Male patients also gave similar results when their levels of testosterone were correlated ($r = -0.32$, $p < 0.05$).

Table 1: Correlation Between Endocrine Biomarkers and GFR

Endocrine Biomarkers	GFR Correlation (r)	p-value
Aldosterone	-0.45	< 0.01
Cortisol	-0.38	< 0.05
T3 (Triiodothyronine)	-0.42	< 0.01
T4 (Thyroxine)	-0.41	< 0.01
Testosterone	-0.32	< 0.05

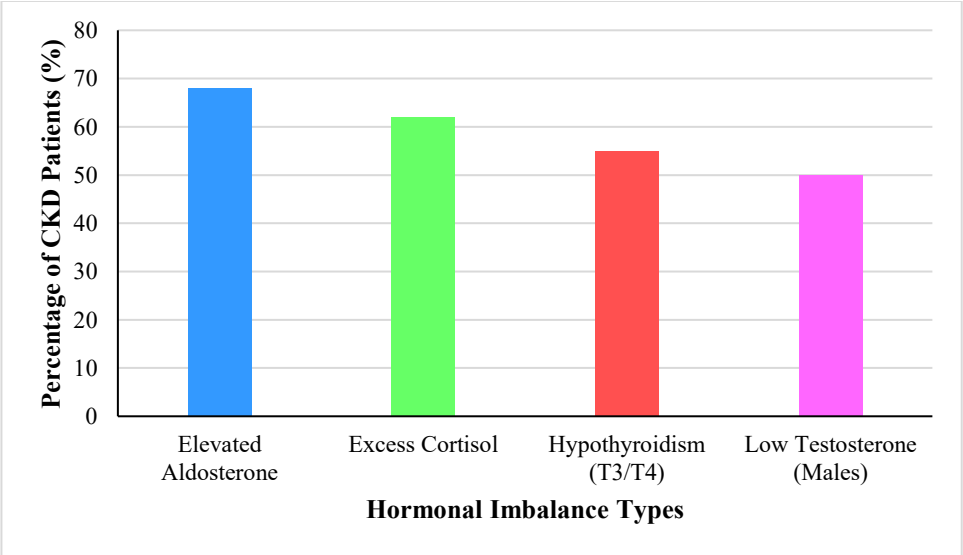

Figure 1: Distribution of Hormonal Imbalances in CKD Patients

The prevalence of different hormonal dystrophy in CKD victims. The chart will be a comparison of the four most common hormonal imbalances in male patients which remain: high aldosterone, high cortisol, hypothyroidism (T3/T4), and low testosterone as illustrated in Figure 1. The most common imbalances were high aldosterone and elevated cortisol which were then replaced by the hypothyroidism and low testosterone. The chart also adds much importance to the idea of endocrine dysfunction role in the development of CKD, where hormonal monitoring and combined approaches to treatment should be encouraged to control not only the functioning of the kidney but also the condition of the endocrine system in such patients.

3.2 Hormonal Imbalances in Renal Dysfunction Cases

The research was able to note some major hormonal disbalances in CKD patients. It was found that 68 % of them had high levels of aldosterone and 62 % of the patients had high levels of cortisol. Table 2 shows that hypothyroidism which was characterized by a low level of T3 and T4 was common in 55 % of the cohort. Low testosterone was observed in half of male CKD patients with a significantly lower median testosterone of patients with CKD (220 ng/dl) than in controls (450ng/dl, p= 0.01). These results have revealed the universal endocrine dysfunction in CKD that deteriorates as the disease advances and relates to worse clinical outcomes and kidney performance.

Table 2: Hormonal Imbalances in Renal Dysfunction Cases

Hormonal Imbalance	Percentage of CKD Patients (%)	p-value
Elevated Aldosterone	68%	< 0.01
Excess Cortisol	62%	< 0.05
Hypothyroidism (T3/T4)	55%	< 0.05
Low Testosterone (Males)	50%	< 0.01

3.3 Clinical and Experimental Findings

The clinical observations indicated that CKD patients with high levels of aldosterone and cortisol levels differed significantly with those having normal levels of the hormones. The patients who had a high level of aldosterone and cortisol had significantly higher blood pressure having a mean of 160/95 mmHg versus 140/85 mmHg in the patients with normal levels of these hormones ($p < 0.01$) as demonstrated in Table 3. The Renal ultrasound images showed that more persons in the high aldosterone group had moderate to severe renal fibrosis (60 %) as compared to the normal hormone level group (20 %) ($p < 0.05$). The urinary albumin was found to be significantly greater in the high hormone group with a mean of 250 +- 150mg/g versus 120-80mg/g of the control group ($p < 0.01$). These results highlight the adverse effect of hormonal disequilibrium on the health of kidneys.

Table 3: Clinical and Experimental Findings

Clinical Measure	Elevated Aldosterone/Cortisol (n=90)	Normal Hormone Levels (n=60)	p-value
Mean Blood Pressure (mmHg)	160/95	140/85	< 0.01
Renal Fibrosis (US Imaging)	60% with moderate/severe fibrosis	20% with moderate/severe fibrosis	< 0.05
Urinary Albumin (mg/g)	250 ± 150	120 ± 80	< 0.01

4. DISCUSSION

The results of the study highlight the high level of endocrine dysfunction on chronic kidney disease (CKD). The relationship between hormonal imbalances with increased aldosterone and cortisol concentration and the kidney functionality was observed to be negative, as indicated in Table 1 ($r = -0.45$, $p < 0.01$ and $r = -0.38$, $p < 0.05$, respectively). These disequilibriumes were correlated to decrease in glomerular filtration rate (GFR). Moreover, there was also a similar negative correlation between thyroid hormones (T3 and T4) and kidney functioning, implying that thyroid functioning is a cause of renal impairment as revealed by their correlation with GFR ($r = -0.42$, $p < 0.01$ in the case of T3 and $r = -0.41$, $p < 0.01$ in the case of T4). Figure 1 shows hormonal imbalances in CKD patients with high values of aldosterone and excess cortisol being the most common with a value of 68 and 62 % in the study population respectively. Such observations confirm the decisive role of aldosterone and cortisol in the pathophysiology of CKD, both hormones with regard to the sodium retention, hypertension, and renal fibrosis, thus deteriorating the kidney functioning. The fact that CKD patients have a high prevalence of hypothyroidism and low levels of testosterone as evidenced by Table 2 is another indication of the multifactorial nature of hormonal involvement in CKD. The research also noted that testosterone depletion among male CKD was also quite common with half of the respondents recording low levels of testosterone, which were indeed very low as compared to healthy controls as indicated in Table 3. This is consistent with prior studies which point out the relationship between testosterone deficiency and deteriorated kidney outcome in male CKD patients. On the whole, the results of this study indicate that the development of hormonal dysregulation is one of the key issues that precondition the progression of CKD and patient outcomes.

Hormonal imbalances reported in this study remain in line with the accumulating evidence on the connection between endocrine malfunctions and CKD. It has been widely reported that patients with CKD experience elevated levels of aldosterone, and the literature indicates that aldosterone is a contributor of hypertension, in addition to worsens kidney fibrosis and leads to the formation of end-stage renal disease (ESRD) [21]. Equally, cortisol overload is also a proven risk factor to CKD progression especially in individuals having comorbidities such as diabetes and high blood pressure. One

of the findings of this study that established a negative correlation between cortisol and GFR corroborates earlier studies that established high levels of cortisol hormone with poor kidney functionality and amplified proteinuria [22]. Another proven factor of CKD progression is thyroid dysfunction and, specifically, hypothyroidism. The works have also established thyroid hormone imbalances to aggravate renal hemodynamics and play a role in the deterioration of kidney functioning. These findings remain similar to the findings in this study whereby hypothyroidism was observed in 55 % of CKD patients. The observed negative correlation between lower levels of T3/T4 and the renal functioning is consistent with the other reports that indicate that thyroid dysfunction is a contributing factor to CKD through elevated renal vascular resistance and reduced GFR [23]. Another acute problem is testosterone deficiency of male CKD patients, and past research suggests that low testosterone levels remain the causes of cardiovascular comorbidities, reduced muscle mass, and poorer prognosis in CKD patients [24]. The discovery of much lower testosterone levels in the male CKD patients in the comparison with healthy control suggests the necessity of hormonal assessment and control of male CKD patients to enhance the clinical results and quality of life. The findings of this study also indicate the relevance of hormonal imbalances among CKD patients, as a result of the clinical outcomes. High levels of aldosterone and cortisol were correlated with strongly increased blood pressure and worse renal fibrosis and this is consistent with other studies that have also related these hormones to cardiovascular risk and renal damage. It is important to note that moderate to severe renal fibrosis was found in 60% of patients with high levels of aldosterone and cortisol in the blood and it is clear that these hormones play a critical role in the progression of CKD and the onset of ESRD [25].

The results of this research represent the significance of endocrine dysfunction management in CKD treatment. The high levels of aldosterone and cortisol, specifically, must be viewed as one of the primary therapeutic objectives in the management of CKD. Aldosterone blocking mineralocorticoid receptor antagonists have demonstrated potential in lessening proteinuria and decreasing clinical deterioration of kidney disease in CKD patients. Also, cortisol blockers have the potential to counteract the adverse outcomes of elevated glucocorticoid functions in CKD. Future studies must address the effectiveness of hormonal replacement therapies especially thyroid hormones and testosterone on enhancing the kidney functioning among CKD patients. Research that focuses on the advantages of thyroid hormone replacement therapy in patients with hypothyroidism induced by CKD and testosterone therapy among male CKD patients should be encouraged to evaluate whether these treatment methods can be used to enhance kidney outcomes and alleviate the comorbidity load in CKD. Moreover, the correlation between the hormonal disturbances and the cardiovascular outcomes in patients with CKD is also the subject of the future research. Since cardiovascular mortality is high among CKD patients, learning in what way hormones such as aldosterone, cortisol, and testosterone affect cardiovascular health may result into more thorough management approaches to CKD. To sum it up, the present work has revealed that endocrine dysfunction has a considerable impact on the development of CKD. Hormonal imbalances especially aldosterone, cortisol, thyroid hormones, and testosterone remain common among CKD patients and highly linked to deteriorated kidney function and clinical results. The findings highlight the importance of combined strategies that should consider the endocrine and renal health when managing CKD. The effectiveness of hormonal therapies to enhance the outcomes and quality of life of CKD patients should also be evaluated in future research.

5. CONCLUSION

The study presents strong factors that indicate that hormonal imbalance is very critical in the development of chronic kidney disease (CKD). High levels of aldosterone, excessive cortisol and thyroid dysfunction were also all observed to have a negative association with kidney functioning as shown by the level of glomerular filtration rate (GFR). The deficiency of testosterone in the male patients with CKD was also very common, making the effect of the disease even more pronounced. Clinical implications of such findings remain high. High levels of aldosterone and cortisol remain known to play a role in impaired kidney performance, systolic hypertension and renal fibrosis. Therefore, mineralocorticoid receptor antagonists or cortisol inhibitors would be potential treatment methods to delay the CKD progression and enhance the patient outcomes. Also, treating thyroid dysfunction and testosterone deficiency with proper hormone replacement treatment can play a major role in enhancing kidney functioning and quality of life especially in men with CKD. This combined method has the potential to transform the management of CKD since hormonal imbalances remain the cause of rapid advancement of the disease. Moreover, the research concentrated on a particular category of patients and this can restrict the extrapolation of the findings. Research on the long-term outcomes of hormonal imbalances on kidney health should be conducted in future studies through longitudinal research studies. Also, more, multi-center studies remain required to assess the effectiveness of hormonal therapies, especially in different populations with different stages of CKD.

REFERENCES

1. Mahmoud T, Borgi L. The interplay between nutrition, metabolic, and endocrine disorders in chronic kidney disease. InSeminars in Nephrology 2021 Mar 1 (Vol. 41, No. 2, pp. 180-188). WB Saunders.
2. Sobolewska J, Żak Z, Monia-Tutur K, Wojciechowska-Luźniak A, Witek P, Niemczyk S. Endocrine disorders in chronic kidney disease. Paediatr Fam Med. 2022;18(3):209-18.
3. Zhang X, Flaws JA, Spinella MJ, Irudayaraj J. The relationship between typical environmental endocrine disruptors and kidney disease. Toxics. 2022 Dec 29;11(1):32.
4. Nagami GT, Kraut JA. The role of the endocrine system in the regulation of acid–base balance by the kidney and the progression of chronic kidney disease. International Journal of Molecular Sciences. 2024 Feb 19;25(4):2420.

5. Ahsan A, Khan A, Farooq MA, Naveed M, Baig MM, Tian WX. Physiology of endocrine system and related metabolic disorders. InEndocrine Disrupting Chemicals-induced Metabolic Disorders and Treatment Strategies 2020 Aug 5 (pp. 3-41). Cham: Springer International Publishing.
6. Pasupulati AK, Kilari S, Sahay M. Endocrine abnormalities and renal complications. Frontiers in Endocrinology. 2023 Aug 21;14:1274669.
7. Zhu Q, Scherer PE. Immunologic and endocrine functions of adipose tissue: implications for kidney disease. Nature Reviews Nephrology. 2018 Feb;14(2):105-20.
8. Conte C, Antonelli G, Melica ME, Tarocchi M, Romagnani P, Peired AJ. Role of sex hormones in prevalent kidney diseases. International Journal of Molecular Sciences. 2023 May 4;24(9):8244.
9. Kumar M, Dev S, Khalid MU, Siddenthi SM, Noman M, John C, Akubuiro C, Haider A, Rani R, Kashif M, Varrassi G. The bidirectional link between diabetes and kidney disease: mechanisms and management. Cureus. 2023 Sep 20;15(9):e45615.
10. Palmer BF, Clegg DJ. Gonadal dysfunction in chronic kidney disease. Reviews in Endocrine and Metabolic Disorders. 2017 Mar;18(1):117-30.
11. Iglesias P, Bajo MA, Selgas R, Díez JJ. Thyroid dysfunction and kidney disease: An update. Reviews in Endocrine and Metabolic Disorders. 2017 Mar;18(1):131-44.
12. Jazani NH, Savoj J, Lustgarten M, Lau WL, Vaziri ND. Impact of gut dysbiosis on neurohormonal pathways in chronic kidney disease. Diseases. 2019 Feb 13;7(1):21.
13. Haffner D, Grund A, Leifheit-Nestler M. Renal effects of growth hormone in health and in kidney disease. Pediatric Nephrology. 2021 Aug;36(8):2511-30.
14. Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity, kidney dysfunction and hypertension: mechanistic links. Nature reviews nephrology. 2019 Jun;15(6):367-85.
15. Czarnecka AM, Niedzwiedzka M, Porta C, Szczylik C. Hormone signaling pathways as treatment targets in renal cell cancer. International Journal of Oncology. 2016 Jun 1;48(6):2221-35.
16. Bertoldo E, Adami G, Rossini M, Giollo A, Orsolini G, Viapiana O, Gatti D, Fassio A. The emerging roles of endocrine hormones in different arthritic disorders. Frontiers in endocrinology. 2021 May 21;12:620920.
17. Giandalia A, Giuffrida AE, Gembillo G, Cucinotta D, Squadrito G, Santoro D, Russo GT. Gender differences in diabetic kidney disease: focus on hormonal, genetic and clinical factors. International journal of molecular sciences. 2021 May 28;22(11):5808.
18. Sagmeister MS, Taylor AE, Fenton A, Wall NA, Chanouzas D, Nightingale PG, Ferro CJ, Arlt W, Cockwell P, Hardy RS, Harper L. Glucocorticoid activation by 11β-hydroxysteroid dehydrogenase enzymes in relation to inflammation and glycaemic control in chronic kidney disease: A cross-sectional study. Clinical endocrinology. 2019 Jan;90(1):241-9.
19. Chow J, Rahman J, Achermann JC, Dattani MT, Rahman S. Mitochondrial disease and endocrine dysfunction. Nature Reviews Endocrinology. 2017 Feb;13(2):92-104.
20. Sagmeister MS, Harper L, Hardy RS. Cortisol excess in chronic kidney disease–A review of changes and impact on mortality. Frontiers in Endocrinology. 2023 Jan 17;13:1075809.
21. Shen Y, Cai R, Sun J, Dong X, Huang R, Tian S, Wang S. Diabetes mellitus as a risk factor for incident chronic kidney disease and end-stage renal disease in women compared with men: a systematic review and meta-analysis. Endocrine. 2017 Jan;55(1):66-76.
22. Edey MM. Male sexual dysfunction and chronic kidney disease. Frontiers in medicine. 2017 Mar 22;4:32.
23. Sheikh AM, Yano S, Tabassum S, Nagai A. The role of the vascular system in degenerative diseases: mechanisms and implications. International Journal of Molecular Sciences. 2024 Feb 11;25(4):2169.
24. Zoccali C, Mallamaci F, Adamczak M, de Oliveira RB, Massy ZA, Sarafidis P, Agarwal R, Mark PB, Kotanko P, Ferro CJ, Wanner C. Cardiovascular complications in chronic kidney disease: a review from the European Renal and Cardiovascular Medicine Working Group of the European Renal Association. Cardiovascular research. 2023 Aug;119(11):2017-32.
25. Cobo G, Hecking M, Port FK, Exner I, Lindholm B, Stenvinkel P, Carrero JJ. Sex and gender differences in chronic kidney disease: progression to end-stage renal disease and haemodialysis. Clinical science. 2016 Jul 1;130(14):1147-63.