Journal of Clinical and Basic Research
Online ISSN: 2538-3736

Assessment of serum interleukin-1 beta levels in hypothyroid patients: A comparative analysis with a control group and their correlation with biochemical markers of kidney function

Karrar Hani Mezaal Alrubaye [1,2], Somayeh Ghorbani [3], Safoura Khajeniazi [1,2]*

1. Metabolic Disorders Research Center, Golestan University of Medical Sciences, Gorgan, Iran
2. Department of Clinical Biochemistry, School of Medicine, Golestan University of Medical Sciences, Gorgan, Iran
3. Cancer Research Center Golestan University of Medical Sciences, Gorgan, Iran

* Correspondence: Safoura Khajeniazi. Department of Clinical Biochemistry, School of Medicine, Golestan University of Medical Sciences, Gorgan, Iran. Tel: +989112747344; Email: dr.khajeniazi@goums.ac.ir

Abstract

Background: Interleukin-1 beta (IL-1β), a prominent pro-inflammatory cytokine, initiates inflammatory responses implicated in various conditions, including thyroid disease and kidney dysfunction. However, the precise involvement of IL-1β in tissue damage remains undefined. This study aimed to investigate the relationship between kidney function and serum IL-1β levels in hypothyroid patients, in contrast to individuals with normal thyroid function.

Methods: Blood samples (5 cc) were collected from study participants and transferred into plain tubes (without anticoagulants). These tubes were immediately centrifuged to isolate the serum, which was then used for the detection of biochemical biomarkers. All markers were quantified using commercially available assay kits. Triiodothyronine (T3), thyroxine (T4), thyroid-stimulating hormone (TSH), and IL-1β levels were determined using the enzyme-linked immunosorbent assay (ELISA) method. Urea (Ur) and creatinine (Cr) concentrations were measured by enzymatic and Jaffe methods, respectively.

Results: Our findings indicated elevated IL-1β levels in hypothyroid patients compared to normal controls; however, this difference did not reach statistical significance ($P = 0.09$). Furthermore, IL-1β demonstrated a non-significant positive correlation with TSH, T3, T4, Ur, and Cr in the hypothyroid cohort ($P > 0.05$).

Conclusion: Our data indicate a positive correlation among TSH, IL-1β, Ur, and Cr, suggesting a relationship between hypothyroidism and kidney disease.

Article Type: Research Article

Article History

Received: 9 April 2025
Received in revised form: 28 April 2025
Accepted: 10 May 2025
Available online: 7 June 2025
DOI: 10.29252/JCBR.9.2.9

Keywords

Hypothyroidism
Kidney diseases
Interleukin-1beta
Urea
Creatinine

OPEN ACCESS
© The author(s)

Highlights

What is current knowledge?

Prior research has independently demonstrated relationships among IL-1β, thyroid hormone panels, and kidney disease in the context of hypothyroidism.

What is new here?

This study uniquely investigates the simultaneous interplay among thyroid hormone panels, IL-1β, and biomarkers of kidney dysfunction in hypothyroid individuals.

Introduction

Thyroid hormones are crucial for maintaining human physiological homeostasis. Their influence extends to various tissues, including the kidneys, where they can alter several renal factors. These alterations encompass changes in the glomerular filtration rate (GFR), renal blood flow, and tubular secretory and reabsorption processes (1). Furthermore, thyroid function itself can be modulated by various cytokines, such as interleukin-1 beta (IL-1β), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6). IL-1β, a member of the IL-1 cytokine family, significantly influences the growth and functional regulation of thyrocytes (2-7). Furthermore, IL-1 has been implicated in the pathogenesis of various human renal diseases (8-10). Notably, primary hypothyroidism in both adults and children often presents with a reversible increase in serum creatinine (Cr) levels, indicative of impaired kidney function associated with the hypothyroid state (11,12). Existing research indicates a positive correlation between compromised thyroid function and kidney impairment when compared to individuals with clinically normal thyroid function (13). Furthermore, there is growing evidence suggesting a link between inflammation and acute kidney injury (AKI), both in the general population and specifically in pregnant women. Sağlam et al. (2022) established a link between chronic kidney disease (CKD) and IL-1β. However, the specific involvement of IL-1β in CKD cases complicated by hypothyroidism remains unexamined (14,15).

Despite earlier studies suggesting a reduction in Cr clearance in hypothyroid individuals (16-18), the precise regulatory role of thyroid function in governing the GFR remains a subject of ongoing debate. Previous research has established a connection between thyroid disorders and kidney disease, and has also explored the influence of thyroid hormones on renal development (19,20). However, these studies have not addressed the potential involvement of IL-1β. To bridge this knowledge gap, the current study aims to investigate the relationship between IL-1β and key indicators of kidney function, namely urea (Ur) and Cr, in hypothyroid patients.

Methods

Sample collection

This case-control study investigated 45 hypothyroid patients and 45 healthy controls. Data collection occurred between November 2022 and February 2023, with participant information directly documented to facilitate study management.

All participants meeting the inclusion criteria completed a consent form. To ensure an accurate assessment of the specified parameters, the case and control groups were matched for age and gender. From both

groups, 5 cc of blood was collected in tubes without anticoagulant. The blood samples were then immediately centrifuged to isolate the serum, which was subsequently used to measure the levels of IL-1β and other biochemical parameters.

All markers were quantified using commercially available kits. Triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) levels were determined via enzyme-linked immunosorbent assay (ELISA). Ur was measured using an enzymatic method, and Cr was assessed by the Jaffe method. The remaining serum samples were stored at -20°C for subsequent determination of serum IL-1β levels using a commercial ELISA kit, following the manufacturer's instructions.

Inclusion criteria

1. Participants, encompassing both hypothyroid individuals and control subjects, were required to be over 18 years of age.
2. All patients with a confirmed diagnosis of hypothyroidism;
3. All hypothyroid patients being newly diagnosed cases (Untreated hypothyroid).

Exclusion criteria

1. Individuals with chronic diseases, such as liver disease, diabetes, autoimmune disorders, malignancies, or infectious diseases;
2. Participants with a history of alcohol use.

Chemicals

TSH, T3, and T4 determination kits were acquired from PISHTAZTEB, while Ur and Cr detection kits were sourced from Pars Azmoon. IL-1β levels were subsequently measured using a kit from ZelBio.

Statistical analysis

Following the experimental procedures, statistical analysis was conducted using SPSS software, version 18. We compared the differences in parameters between the case and control groups. Normality of data distribution was assessed using the Shapiro-Wilk test. Additionally, various statistical tests were employed to determine relationships among the study parameters, including the Chi-square test, analysis of variance (ANOVA), Spearman's rho, and Pearson's correlation coefficient. To evaluate the relationships and differences in serum IL-1β levels and other measured parameters between the two groups, a general correlation analysis was conducted utilizing the Mann-Whitney U test and Spearman's rho correlation. All quantitative data are presented as the mean ± standard deviation. Statistical significance was defined as $P < 0.05$.

Results

This study investigated the potential association between IL-1β and hypothyroidism, specifically exploring its relationship with kidney dysfunction. The research included 90 participants, comprising 45 patients hypothyroid diagnosed and 45 healthy control subjects. Our findings revealed a statistically significant difference in thyroid hormone levels between the two groups, with TSH levels being markedly elevated in hypothyroid patients compared to the healthy controls. Hypothyroid subjects exhibited significantly lower T3 and T4 levels compared to the control group. Mean values for TSH, T3, and T4 were not reported in this study.

Table 1 indicates that Cr levels are significantly elevated in hypothyroid subjects compared to healthy individuals ($P = 0.017$). While Ur levels are also higher in hypothyroid subjects, this difference is not statistically significant ($P = 0.139$).

Table 2 illustrates the circulating IL-1β levels in both hypothyroid and healthy individuals. While the data indicate that IL-1β concentrations are elevated in hypothyroid subjects compared to the control group, this difference is not statistically significant.

Table 3 indicates a positive correlation between IL-1β and thyroid hormones (TSH, T3, and T4) in the case group. Conversely, a negative correlation was observed between the mentioned parameters in healthy subjects. It is important to note that none of these correlations were statistically significant ($P < 0.05$).

This table presents the correlation of IL-1β with Ur and Cr levels for both the case and control groups. A significance level of $P < 0.05$ was used for all reported results.

Table 4 indicates that in the case group, IL-1β demonstrates a non-significant positive correlation with both Ur and Cr levels. Conversely, within the control group, IL-1β exhibits a non-significant positive correlation with Ur but a negative correlation with Cr.

Table 5 presents the correlations of Cr and Ur with hypothyroidism profiles in hypothyroid patients. A significance level of $P < 0.05$ indicates a statistically significant correlation between these variables.

In this study, Cr demonstrated a positive correlation with TSH and negative correlations with T3 and T4. However, these correlations were not statistically significant ($P > 0.05$). Similarly, Ur exhibited negative correlations with TSH and T3, and a positive correlation with T4, but none of these relationships were statistically significant ($P > 0.05$).

Table 1. Creatinine and urea levels in hypothyroid and healthy subjects

Variables	N	Case Median (Inter-Quartile)	Control	Test statistic	P-Value
Cr	45	1.05 (0.955, 1.125)	0.94 (0.9, 1.08)	-2.392	0.017
Ur	45	29 (24, 33.5)	23 (20, 32.5)	-1.479	0.139

Cr: Creatinine, Ur: Urea

Table 2. Interleukin-1 beta levels in hypothyroid and healthy subjects

Variables	N	Case Median (Inter-Quartile)	Control	Test statistic	P-Value
IL-1β	45	56.03 (52.45, 60.71)	55.72 (50.25, 59.80)	-0.048	0.961

IL-1β: Interleukin-1 Beta

Table 3. The correlation between interleukin-1 beta and thyroid hormones in case and control groups

Group	Spearman's Rho		T3	T4	TSH
Case	IL-1β	r	0.048	0.028	0.228
		P-Value	0.755	0.856	0.132
		N	45	45	45
Control	IL-1β	r	-0.270	-0.134	-0.017
		P-Value	0.073	0.380	0.914
		N	45	45	45

IL-1β: Interleukin-1 Beta, T3: Triiodothyronine, T4: Thyroxine, TSH: Thyroid-Stimulating Hormone

Table 4. The correlation of interleukin-1 beta with creatinine and urea in case and control groups

Group	Parameter	Spearman's Rho	Cr	Ur
Case	IL-1β	r	0.148	0.132
		P-Value	0.347	0.387
		N	45	45
Control	IL-1β	r	-0.117	0.117
		P-Value	0.444	0.445
		N	45	45

IL-1β: Interleukin-1 Beta, Cr: Creatinine, Ur: Urea

Table 5. The correlations of creatinine and urea with hypothyroidism profiles in the case group

Group	Spearman's Rho	T3	T4	TSH
Cr	r	0.182	-0.088	0.080
	P-Value	0.231	0.565	0.602
	N	45	45	45
Ur	r	-0.68	0.034	-0.125
	P-Value	0.655	0.827	0.412
	N	45	45	45

Cr: Creatinine, Ur: Urea, T3: Triiodothyronine, T4: Thyroxine, TSH: Thyroid-Stimulating Hormone

Discussion

Hypothyroidism is a prevalent global disorder, often linked to inflammation and insufficient iodine absorption (15). IL-1β, an inflammatory mediator, is implicated in the pathogenesis of hypothyroidism and has been shown to induce apoptosis and tissue damage in thyroid follicular cells (16). Furthermore, both hypothyroidism and hyperthyroidism can directly impact kidney function, in addition to exerting systemic hemodynamic, metabolic, and cardiovascular effects (17).

In this study, we assessed kidney function and serum IL-1β levels in hypothyroid patients and compared them to individuals with normal thyroid function. Our findings revealed that IL-1β levels were insignificantly higher in hypothyroid patients. Statistical analysis further revealed that Cr levels were significantly elevated in hypothyroid subjects compared to healthy controls, while Ur levels were insignificantly higher.

As shown in Kwakkel et al. in 2007, IL-1β significantly modulates endogenous thyroid hormone receptors, which are crucial for gene transcription in liver cells. According to their research, conducted both in vivo and in vitro, elevated IL-1β levels culminate in a reduction of TRα1 and TRα2 messenger ribonucleic acid (mRNA) (19).

Mikos et al. (2014) investigated the diagnostic utility of IL-1β, TNF-α, and IL-6 in pediatric patients with autoimmune thyroid disease (AITD). Their study compared levels of these cytokines in children experiencing hypothyroidism and hyperthyroidism against those in healthy controls. The research findings demonstrated a significantly elevated level of IL-1β in the hypothyroid group compared to both the hyperthyroid and control groups. Furthermore, the authors conducted a receiver operating characteristic (ROC) analysis, concluding that IL-1β holds potential as a valuable biomarker for both the diagnosis and follow-up of AITD in children. Our findings align with Mikos's observations, revealing a positive correlation between IL-1β and TSH in hypothyroid patients. Conversely, a negative correlation was noted in normal subjects. However, it is important to note that the correlations in both groups were not statistically significant (21). These results may suggest that elevated IL-1β levels could lead to increased TSH, potentially contributing to a higher incidence of hypothyroidism. Furthermore, our study assessed the correlation between IL-1β and thyroid hormone levels. We found that IL-1β was positively correlated with both T3 and T4 in hypothyroid patients. Conversely, an insignificantly negative correlation was observed in the control group.

Ines Zaaber et al. (2016) investigated the relationship between IL-1β and interleukin-4 (IL-4) gene variants and AITDs within the Tunisian population. Their findings indicated an association between the IL-1β +3953C/T variable number of tandem repeat (VNTR) polymorphism and the presence of anti-thyroid peroxidase (TPO) antibodies, which are key factors in the progression of Hashimoto's thyroiditis (HT). Previous research has indicated a higher frequency of the T allele of IL-1β in HT patients who test negative for TPO antibodies compared to those who test positive (3). Furthermore, a study by Sandra A. Rebuffat et al. (2012) examined the distribution and integrity of IL-1β and TSH within the thyroid epithelium of individuals with AITDs. Their findings demonstrated that both HT and Graves' disease (GD) exhibited distinct patterns of junction protein expression, suggesting a compromise in the integrity of the thyroid epithelium in these conditions. IL-1β has been shown to disrupt the stability of human thyroid epithelial cells by altering the expression and localization of junction proteins. These findings suggest that IL-1β plays a role in the pathogenic changes observed in thyroid autoimmunity (2). In a separate study conducted in 2021, Zahra Heidari et al. investigated the association of polymorphisms in the IL-1β, nucleotide-binding oligomerization domain (NOD)-like receptor family pyrin domain-containing 3 (NLRP3), and cyclooxygenase-2 (COX-2) genes with the risk of AITD. Their research demonstrated a significant association between IL-1β single nucleotide polymorphisms (SNPs) and TSH levels in patients with GD (22).

Studies on pro-inflammatory cytokines, such as IL-1β, have demonstrated that IL-1β is able to activate the canonical nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) pathway in various cell types. In renal cells, including mesangial, endothelial, and tubuloepithelial cells, this activation by IL-1β can lead to kidney injury (23-25).

Lei et al. (2019) investigated the impact of IL-1β inhibition on CKD in diabetic obese mice. Their research, utilizing an anti-IL-1β immunoglobulin G (IgG) antibody, demonstrated a significant capacity to preserve the GFR by approximately 50 µL/min (9). In a separate study, Stephen J. et al. (2021) compiled data concerning the role of inflammation in the complexity of clinical signs associated with AKI. They identified inflammation, specifically involving IL-1β, as a common mechanism contributing to AKI, acting either as an initiating factor or in the progression of kidney injury (26). Saini et al. (2012) explored the relationship between serum Cr and TSH levels in individuals with overt hypothyroidism. Their findings indicated that serum Ur, Cr, and uric acid levels were notably elevated in overtly hypothyroid subjects when compared to healthy controls. However, in patients with subclinical hypothyroidism, only serum Ur and Cr levels showed a significant increase. The study also revealed a significant

positive correlation between TSH and both Cr and uric acid levels, while free T4 (FT4) demonstrated a negative correlation with uric acid in overt hypothyroidism (11). Previous research has demonstrated a reversible elevation of serum Cr in individuals with primary hypothyroidism, establishing an association between Cr levels and this condition in humans (15,16). Our current findings align with these prior observations. Our investigation revealed significantly higher serum Cr levels in hypothyroid patients compared to healthy controls. While serum Ur levels were also elevated in the patient group, this increase was not statistically significant.

Matsuoka-Uchiyama et al. (2023) investigated the relationship between urinary Cr excretion and hypothyroidism in patients with CKD. Their findings suggest that increased Cr production is unlikely to be a primary factor in hypothyroidism. Instead, the elevated serum Cr levels observed in hypothyroid individuals appear to be predominantly linked to a decrease in kidney function. While the association between hypothyroidism and elevated Cr levels is well-established in the literature (17,18), the precise underlying mechanisms remain to be fully elucidated. Huang et al. (2016) explored the relationship between thyroid hormones and the risk of developing CKD and renal failure in a cohort of middle-aged and elderly Chinese individuals. Their research indicated that elevated FT4 levels were associated with an increased risk, while TSH levels showed no significant change. Furthermore, their findings suggested no association between free T3 (FT3) levels and a heightened risk of CKD incidence or a rapid decline in estimated GFR (eGFR) within this demographic (1). Asvold et al. (2011) investigated the relationship between thyroid function and the eGFR. Their research indicated a significant inverse correlation between TSH levels and eGFR; individuals with lower TSH values exhibited higher eGFRs. Notably, eGFR was observed to be lowest in individuals with mid-range TSH levels, and highest among those experiencing either subclinical or overt hypothyroidism. A higher prevalence of CKD was observed in individuals with mid-range TSH levels when compared to normal subjects. Furthermore, CKD was found to be more prevalent in patients diagnosed with either subclinical or overt hypothyroidism (14).

Sağlam et al. (2022) explored the connection between kidney injury molecule-1 (KIM-1) and IL-1β levels and hepatic and renal functions in pregnant women with thyroid disorders. Their research indicated that hypothyroid pregnant women exhibited significantly elevated mean levels of Cr, aspartate aminotransferase (AST), and procalcitonin (PCT) compared to those with hyperthyroidism. Additionally, the study found that IL-1β levels were higher in hypothyroid patients than in euthyroid individuals. These results suggest a greater prevalence of renal damage in hypothyroid pregnant women diagnosed (15). In 2012, Saini et al. explored the relationship between serum Cr and TSH levels in individuals with overt hypothyroidism. Their investigation revealed significantly elevated serum Ur, Cr, and uric acid levels in subjects with overt hypothyroidism when compared to healthy controls. Conversely, in patients with subclinical hypothyroidism, only serum Ur and Cr levels demonstrated a significant increase. Furthermore, the study identified a significant positive correlation between TSH and both Cr and uric acid values in overt hypothyroidism, while FT4 showed a negative correlation with uric acid in the same group (11). Saini's research, aligned with our observations, demonstrated a positive correlation between TSH and Cr in both hypothyroid patients and healthy controls. However, the correlation between Ur and TSH was negative in hypothyroid patients but positive in healthy subjects. Moreover, our study also investigated the correlations of Ur and Cr with thyroid hormones. Statistical analysis revealed a negative correlation between Cr and both T3 and T4. In contrast, Ur exhibited a negative correlation with T3 but a positive correlation with T4 (11).

Other research has also indicated a positive correlation between Cr and TSH levels in individuals with overt hypothyroidism. Furthermore, the hypothyroid state is significantly linked to alterations in biochemical parameters of kidney function. It has also been established that primary hypothyroidism is associated with a reversible elevation of serum Cr levels in both adult and pediatric populations (27). In the present study, we investigated the correlation of IL-1β with Ur and Cr in both case and control groups. Our findings revealed that IL-1β had non-significant positive correlations with both Ur and Cr levels in hypothyroid patients. These data suggest that elevated IL-1β may contribute to increased Ur and Cr levels in hypothyroidism.

Limited research has been conducted on hypothyroidism, and consequently, there is a scarcity of information regarding the impact of IL-1β on kidney function and the progression to kidney failure in hypothyroid human subjects.

Conclusion

Hypothyroidism has been established as a contributing factor to kidney dysfunction; however, the precise mechanisms linking these two conditions remain undefined. IL-1β is recognized as a key mediator in the pathogenesis of numerous diseases. Our preliminary data indicate a positive correlation among TSH, IL-1β, Ur, and Cr levels. It is important to note that not all observed correlations were statistically significant, which may be attributable to the limited sample size of our study. Consequently, we are currently unable to definitively conclude a direct reciprocal relationship between hypothyroidism and kidney disease based on these findings.

Acknowledgement

The authors would like to thank Mrs. Nahid Poursharifi for her contributions to the experimental procedures of this study.

Funding sources

This project, officially registered with the Vice-Chancellor of Research and Technology, Golestan University of Medical Sciences (Gorgan, Iran), under approval number 113123, was funded by Mr. Karrar Hani Mezaal Alrubaye, a master's student at Golestan University of Medical Sciences.

Ethical statement

This study was approved by the Vice-Chancellor of Research and Technology and the Ethics Committee, Golestan University of Medical Sciences (Code of ethics: IR.GOUMS.REC.1401.561).

Conflicts of interest

No conflicts of interest.

Author contributions

Karrar Hani Mezaal Alrubaye: Writing the proposal and conducting the experimental aspects of the study. Somayeh Ghorbani: Data analysis. Safoura Khajeniazi: Supervising the thesis, interpreting the results, and writing the manuscript.

Data availability statement

All data were included in the manuscript.

References

1. Huang X, Ding L, Peng K, Lin l, Wang T, Zhou Z, et al., Thyroid hormones associate with risk of incident chronic kidney disease and rapid decline in renal function: a prospective investigation. J Transl Med .2016;14(1):336. [View at Publisher] [DOI] [PMID] [Google Scholar]
2. Rebuffat SA, Kammoun-Krichen M, Charfeddine I, Ayadi H, Bougacha-Elleuch N, Peraldi-Roux S. IL-1B and TSH disturb thyroid epithelium integrity in autoimmune thyroid Diseases. Immunobiology.2013;218(3):285-91. [View at Publisher] [DOI] [PMID] [Google Scholar]
3. Zaaber I, Mestiri S, Hammedi H, Marmouch H, Mahjoub S, Jrad Tensaout BBH,et al. Association of Interleukin-1B and Interleukin-4 Gene Variants with Autoimmune Thyroid Diseases in Tunisian Population. Immunol Invest. 2016;45(4):284-97. [View at Publisher] [DOI] [PMID] [Google Scholar]
4. Gianoukakis AG, Smith TJ. The role of cytokines in the pathogenesis of endocrine disease. Can J Diabetes. 2004;28(1):30-42. [View at Publisher] [Google Scholar]
5. Rasmussen AK. Cytokine actions on the thyroid gland. Dan Med Bull. 2000;47(2):94-114. [View at Publisher] [PMID] [Google Scholar]

6. Afsar B, Covic A, Ortiz A, Afsar RE, Kanbay M. The Future of IL 1 Targeting in Kidney Disease. Drugs. 2018;78(11):1073-83. [View at Publisher] [DOI] [PMID] [Google Scholar]
7. de Torre-Minguela C, Mesa Del Castillo P, Pelegrin P. The NLRP3 and Pyrin Inflammasomes: Implications in the Pathophysiology of Autoinflammatory Diseases. Front Immunol. 2017;8:43. [View at Publisher] [DOI] [PMID] [Google Scholar]
8. Akash MSH, Rehman K, Sun H, Chen S. Sustained delivery of IL-1Ra from PF127-gel reduces hyperglycemia in diabetic GK-rats. PLoS One. 2013;8(2):e55925. [View at Publisher] [DOI] [PMID] [Google Scholar]
9. Lei Y, Devarapu SK, Motrapu M, Cohen CD, Lindenmeyer MT, Moll S. et al: Interleukin-1b Inhibition for Chronic Kidney Disease in Obese Mice With Type 2 Diabetes. Front Immunol. 2019:10:1223. [View at Publisher] [DOI] [PMID] [Google Scholar]
10. Alique M, Sanchez-Lopez E, Rayego-Mateos S, Egido J, Ortiz A, Ruiz-Ortega M. Angiotensin II, via angiotensin receptor type1/nuclear factor-kappaB activation, causes a synergistic effect on interleukin-1-beta-induced inflammatory responses in cultured mesangial cells. J Renin Angiotensin Aldosterone Syst. 2015;16(1):23-32. [View at Publisher] [DOI] [PMID] [Google Scholar]
11. Saini V, Yadav A, Arora MK, Arora S, Singh R, Bhattacharjee J. Correlation of creatinine with TSH levels in overt hypothyroidism - a requirement for monitoring of renal function in hypothyroid patients. Clin Biochem. 2012;45(3):212-4. [View at Publisher] [DOI] [PMID] [Google Scholar]
12. Kreisman SH, Hennessey JV. Consistent Reversible Elevations of Serum Creatinine levels in Severe Hypothyroidism. Arch Intern Med. 1999;159(1):79-82. [View at Publisher] [DOI] [PMID] [Google Scholar]
13. Akagunduz B, Akcakaya M. Evaluation of the Correlation of Urea, Creatine, and Uric Acid Levels with TSH in Patients with Newly Diagnosed Overt and Subclinic Hypothyroidism. EJMI. 2021;5(3):317-21 [View at Publisher] [DOI] [Google Scholar]
14. Asvold BO, Bjøro T, Vatten LJ. Association of thyroid function with estimated glomerular filtration rat in a population-based study: the HUNT study. Eur J Endocrinol. 2011;164(1):101-5. [View at Publisher] [DOI] [PMID] [Google Scholar]
15. Sağlam E, Esen B, Pilten S, Gözütok V, Atay AE. The Relationship of Kidney Injury Molecule-1 and Interleukin-1 Beta with Hepatic and Renal Functions in Pregnant Women with Thyroid Disease. İstanbul Med J. 2022;23(2):85-9 [View at Publisher] [DOI] [Google Scholar]
16. Szczepanski J, Griffin A, Novotny S, Wallace K. Acute kidney injury in pregnancies complicated with preeclampsia or HELLP syndrome. Front Med (Lausanne). 2020;7:22. [View at Publisher] [DOI] [PMID] [Google Scholar]
17. Matsuoka-Uchiyama N, Tsuji K, Takahashi K, Fukushima K, Takeuchi H, Kitamura S, et al: Association between Urinary Creatinine Excretion and Hypothyroidism in Patients with Chronic Kidney Disease. Diagnostics. 2023;13(4):669. [View at Publisher] [DOI] [PMID] [Google Scholar]
18. Montenegro J, Gonzalez O, Saracho R, Aguirre R, Gonzalez O, Martinez I. Changes in renal function in primary hypothyroidism. Am J Kidney Dis. 1996;27(2):195-8. [View at Publisher] [DOI] [PMID] [Google Scholar]
19. Kwakkel J, Wiersinga WM, Boelen A. Interleukin-1 modulates endogenous thyroid hormone receptor a gene transcription in liver cells. J Endocrinol. 2007;194(2):257-65. [View at Publisher] [DOI] [PMID] [Google Scholar]
20. Mohamedali M, Maddika SR, Vyas A, Iyer V, Cheriyath P. Thyroid Disorders and Chronic Kidney Disease .Int J Nephrol. 2014;520281:1-6. [View at Publisher] [DOI] [PMID] [Google Scholar]
21. Mikos H, Mikos M, Rabska-Pietrzak B, Niedziela M. The clinical role of serum concentrations of selected cytokines: IL-1b,TNF-a and IL-6 in diagnosis of autoimmune thyroid disease (AITD) in Children. Autoimmunity. 2014;47(7):466-72. [View at Publisher] [DOI] [PMID] [Google Scholar]
22. Heidari Z, Salimi S, Rokni M, Rezaei M, Khalafi N, Jafari Shahroudi M, et al. Association of IL-1β, NLRP3, and COX-2 Gene Polymorphisms with Autoimmune Thyroid Disease Risk and Clinical Features in the Iranian Population. Biomed Res Int. 2021:7729238. [View at Publisher] [DOI] [PMID] [Google Scholar]
23. Dinarello CA. Reduction of inflammation by decreasing production of interleukin-1 or by specific receptor antagonism. Int J Tissue React. 1992;14(2):65-75. [View at Publisher] [DOI] [PMID] [Google Scholar]
24. Carter DB, Deibel MR Jr, Dunn CJ, Tomich CS, Laborde AL, Slightom JL, et al. Purification, cloning, expression and biological characterization of an interleukin-1 receptor antagonist protein. Nature. 1990;344(6267):633-8. [View at Publisher] [DOI] [PMID] [Google Scholar]
25. Dripps DJ, Brandhuber BJ, Thompson RC, Eisenberg SP. Interleukin- 1 (IL-1) receptor antagonist binds to the 80-kDa IL-1 receptor but does not initiate IL-1 signal transduction.J Biol Chem. 1991;266(16):10331-6. [View at Publisher] [PMID] [Google Scholar]
26. McWilliam SJ, Wright RD. Welsh GI, Tuffin J, Budge KL, Swan L,et al. The complex interplay between kidney injury and inflammation. Clin Kidney J. 2020;14(3):780-8. [View at Publisher] [DOI] [PMID] [Google Scholar]
27. Simeoni M, Cerantonio A, Pastore I, LiguoriR, Greco M, Foti D, et al. The correct renal function evaluation in patients with thyroid dysfunction. J Endocrinol Invest. 2016;39(5):495-507. [View at Publisher] [DOI] [PMID] [Google Scholar]

How to Cite:

Hani Mezaal Alrubaye K, Ghorbani S, Khajeniazi S. Assessment of serum interleukin-1 beta levels in hypothyroid patients: A comparative analysis with a control group and their correlation with biochemical markers of kidney function. *JCBR*. 2025;9(2):9-13. http://dx.doi.org/10.29252/JCBR.9.2.9