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ASSESSMENT OF THYROID HORMONES AND LIVER ENZYMES IN PATIENTS WITH CHRONIC KIDNEY DISEASE UNDERGOING MAINTENANCE HEMODIALYSIS IN AL-QAIM GENERAL HOSPITAL, IRAQ

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ABSTRACT

Chronic kidney disease (CKD) is associated with endocrine and metabolic disturbances that extend beyond classical renal biomarkers and may influence both thyroid homeostasis and liver-related biochemical indices. The present case-control study evaluated serum thyroid hormones and liver enzymes in 60 patients with CKD receiving maintenance hemodialysis at Al-Qaim General Hospital, Al-Anbar Governorate, Iraq, and compared them with 30 apparently healthy controls. Blood samples were obtained before the dialysis session. Serum thyroid-stimulating hormone (TSH), triiodothyronine (T3), thyroxine (T4), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP) were measured by standard immunoassay and colorimetric methods. CKD patients showed significantly higher mean levels of TSH, AST, ALT, and ALP and significantly lower mean levels of T3 and T4 than controls ($P<0.001$ for all comparisons). Within the patient group, TSH was significantly higher in females than males ($P=0.032$), whereas T3, T4, AST, ALT, and ALP did not differ significantly by sex. Patients younger than 50 years had significantly higher TSH and ALP values than older patients ($P=0.010$ and $P=0.035$, respectively). No significant differences were observed according to monthly dialysis frequency or disease duration. ROC analysis showed strong discriminatory performance within this dataset for AST (AUC=0.988), TSH (AUC=0.916), and ALT (AUC=0.871). These findings indicate that CKD patients on maintenance hemodialysis in Al-Qaim exhibit a distinct biochemical pattern characterized by altered thyroid function and increased liver enzyme activity, with additional variation related to sex and age in selected markers.

KEYWORDS: chronic kidney disease, hemodialysis, thyroid hormones, liver enzymes, ROC analysis, Al-Qaim.

INTRODUCTION

Chronic kidney disease is a progressive disorder characterized by persistent abnormalities in kidney structure or function and by a substantial burden of cardiovascular, endocrine, metabolic, and inflammatory complications.[1] At the population level, CKD has become a major global health problem, affecting hundreds of millions of individuals and imposing a disproportionate burden on low- and middle-income settings where access to long-term renal care remains limited.[2] Contemporary clinical practice guidelines emphasize that advanced CKD should be viewed as a multisystem condition rather than an isolated renal disorder, because deterioration in kidney function is accompanied by broad biochemical and hormonal disturbances that may alter several organ systems simultaneously.[3] Among the endocrine abnormalities associated with CKD, disturbances of the hypothalamic-pituitary-thyroid axis are particularly relevant. Systemic inflammation, altered hormone binding, impaired peripheral deiodination, metabolic acidosis, protein-energy wasting, and reduced renal clearance of hormones and metabolites may all contribute to abnormal thyroid profiles in patients with renal impairment.[4]

The interaction between thyroid function and kidney function is bidirectional. Thyroid hormones influence renal blood flow, glomerular filtration, tubular transport,

and electrolyte handling, while renal dysfunction may modify the metabolism, degradation, and circulating concentrations of thyroid hormones.[5] Recent reviews have underlined that euthyroid patients with CKD may still show relatively high-normal TSH, low T3, and low-normal or reduced T4, particularly in more advanced stages of disease or in the setting of hemodialysis.[6] Cross-sectional clinical studies have likewise reported that thyroid dysfunction is not uncommon in CKD populations and that subclinical hypothyroidism and low T3 states may coexist with worsening renal function.[7] More recent observational data have continued to document a substantial burden of thyroid abnormalities among CKD patients, reinforcing the clinical value of thyroid function assessment in this population.[8]

Liver-related biochemical markers may also be altered in CKD, but the published pattern is less uniform than that of thyroid dysfunction. A comprehensive review of hemodialysis populations described generally lower aminotransferase activities than in individuals with preserved renal function, whereas other markers, particularly ALP, may increase depending on bone turnover, mineral metabolism, inflammation, and hepatobiliary status.[9] Similarly, comparative clinical work has suggested that AST and ALT may decline with CKD severity in some cohorts, whereas ALP can move in the opposite direction, highlighting the need to interpret liver enzymes within the specific metabolic context of renal disease.[10] ALP deserves special attention in CKD because it is not only a liver-associated enzyme but also a marker linked to bone remodeling and chronic kidney disease-mineral and bone disorder. Recent guidance on bone turnover markers supports the clinical relevance of ALP-related measurements in CKD and CKD-associated osteoporosis.[11] Updated Asia-Pacific guidance has also emphasized the usefulness of bone-turnover markers, including alkaline phosphatase-related markers, in the biochemical evaluation of patients with advanced kidney disease.[12] In hemodialysis cohorts, higher serum ALP has additionally been linked with adverse outcomes and mortality, which increases its interpretive importance beyond routine liver function testing alone.[13]

Although the available literature confirms that thyroid hormones and liver enzymes may be altered in CKD, local evidence from western Iraq remains limited, especially in patients managed in Al-Qaim district. Accordingly, the present study was conducted to evaluate thyroid hormones and liver enzymes in CKD patients undergoing maintenance hemodialysis in Al-Qaim General Hospital and to compare the findings with those of apparently healthy controls.

MATERIALS AND METHODS

This case-control study was conducted in Al-Qaim General Hospital, Al-Qaim District, Al-Anbar Governorate, Iraq. The study included 60 patients with chronic kidney disease receiving regular maintenance hemodialysis and 30 apparently healthy individuals used as controls. The patient group comprised 30 males and 30 females, whereas the control group included 15 males and 15 females.

Venous blood samples were collected under aseptic conditions from all participants. In the patient group, blood was drawn immediately before the dialysis session to avoid the acute influence of the procedure on the measured biochemical variables. After clotting, samples were centrifuged and serum was separated for biochemical and hormonal assays.

Serum TSH was measured using commercially available kits based on the enzyme-linked immunosorbent assay principle.[14] The analytical framework for T3 and T4 assessment followed established solid-phase and two-site enzyme immunoassay concepts used in clinical hormone measurement.[15]

Serum AST and ALT activities were determined by the classical colorimetric method of Reitman and Frankel, which depends on the formation of oxaloacetate or pyruvate followed by derivatization with 2,4-dinitrophenylhydrazine and spectrophotometric reading.[16]

Serum ALP activity was measured by the method of Kind and King using phenyl phosphate substrate with color development based on amino-antipyrine chemistry.[17]

Data were analyzed using SPSS version 23. Quantitative variables are presented as mean ± standard deviation. Independent-samples t test was used for two-group comparisons. ROC analysis was used to evaluate the discriminatory performance of the studied markers, and interpretation of AUC values followed established principles of ROC methodology.[18] Pearson correlation analysis was used to examine selected relationships between continuous variables. A P value of less than 0.05 was considered statistically significant.

RESULTS

The demographic and clinical characteristics of the study groups are shown in Table 1. The CKD group was older than the control group, while sex distribution was balanced by design. Among patients, 41.7% underwent 8 dialysis sessions per month and 58.3% underwent 12 sessions per month. The mean disease duration was 5.02 ± 2.94 years.

Comparison between CKD patients and controls demonstrated a clearly altered biochemical profile in the patient group (Table 2). Mean TSH was significantly higher in CKD patients, whereas T3 and T4 were significantly lower. At the same time, AST, ALT, and ALP were all significantly elevated in patients relative to controls ($P<0.001$ for all studied markers).

Sex-based comparison within the CKD group revealed a significant increase in TSH among female patients compared with male patients (4.45 ± 1.29 vs 3.77 ± 1.11 µIU/mL, P=0.032). No significant sex-related differences were observed for T3, T4, AST, ALT, or ALP (Table 3).

Age-based analysis showed that patients younger than 50 years had significantly higher TSH and ALP values than those aged 50 years or older. The differences in T3, T4, AST, and ALT according to age group did not reach statistical significance (Table 4).

When patients were classified according to monthly dialysis frequency, no significant differences were detected in any of the studied thyroid or liver markers between those receiving 8 sessions per month and those receiving 12 sessions per month (Table 5).

Similarly, stratification according to disease duration (≤4 years versus >4 years) did not demonstrate significant differences in thyroid hormones or liver enzyme activities (Table 6).

ROC analysis indicated that AST had the highest discriminatory performance within this dataset for distinguishing CKD patients from controls (AUC=0.988), followed by TSH (AUC=0.916) and ALT (AUC=0.871). T3, T4, and ALP showed moderate to fair discriminatory performance (Table 7 and Figure 1).

Correlation analysis demonstrated weak but significant negative correlations between age and both TSH and ALP. In addition, T3 showed an almost perfect positive correlation with T4, and AST showed a strong positive correlation with ALT (Table 8 and Figure 2).

Table 1: Demographic and clinical characteristics of the study groups.

Variable	CKD patients (n=60)	Controls (n=30)	P value
Age (years)	48.80 ± 16.01	34.77 ± 8.84	<0.001
Male/Female	30/30	15/15	Matched
Monthly dialysis sessions	8 sessions: 25 (41.7%); 12 sessions: 35 (58.3%)	Not applicable	-
Disease duration (years)	5.02 ± 2.94	Not applicable	-

Table 2: Comparison of thyroid hormones and liver enzymes between CKD patients and controls.

Marker	CKD patients (mean ± SD)	Controls (mean ± SD)	P value
TSH (µIU/mL)	4.11 ± 1.24	1.85 ± 1.10	<0.001
T3 (ng/mL)	0.76 ± 0.34	1.10 ± 0.37	<0.001
T4 (µg/dL)	5.84 ± 1.56	7.49 ± 1.81	<0.001
AST (U/L)	13.73 ± 3.65	8.06 ± 1.23	<0.001
ALT (U/L)	11.84 ± 3.39	8.33 ± 1.14	<0.001
ALP (U/L)	83.35 ± 34.54	65.08 ± 9.54	<0.001

Table 3: Sex-based comparison of thyroid hormones and liver enzymes within the CKD group.

Marker	Males (n=30)	Females (n=30)	P value
TSH (µIU/mL)	3.77 ± 1.11	4.45 ± 1.29	0.032
T3 (ng/mL)	0.83 ± 0.37	0.69 ± 0.28	0.103
T4 (µg/dL)	6.18 ± 1.74	5.50 ± 1.30	0.094
AST (U/L)	12.97 ± 2.64	14.49 ± 4.36	0.109
ALT (U/L)	11.34 ± 2.68	12.33 ± 3.95	0.262
ALP (U/L)	85.68 ± 35.61	81.03 ± 33.89	0.606

Table 4: Age-group comparison of thyroid hormones and liver enzymes within the CKD group.

Marker	<50 years (n=30)	≥50 years (n=30)	P value
TSH (µIU/mL)	4.52 ± 1.25	3.70 ± 1.10	0.010
T3 (ng/mL)	0.77 ± 0.32	0.75 ± 0.36	0.805
T4 (µg/dL)	5.89 ± 1.45	5.79 ± 1.69	0.818
AST (U/L)	14.46 ± 4.29	13.00 ± 2.76	0.124
ALT (U/L)	12.66 ± 3.94	11.02 ± 2.52	0.060
ALP (U/L)	92.73 ± 37.87	73.98 ± 28.49	0.035

Table 5: Comparison according to monthly dialysis frequency within the CKD group.

Marker	8 sessions/month (n=25)	12 sessions/month (n=35)	P value
TSH (µIU/mL)	4.18 ± 1.30	4.06 ± 1.21	0.704
T3 (ng/mL)	0.71 ± 0.26	0.79 ± 0.38	0.357
T4 (µg/dL)	5.63 ± 1.20	6.00 ± 1.77	0.338
AST (U/L)	13.04 ± 2.48	14.21 ± 4.27	0.187
ALT (U/L)	11.52 ± 3.08	12.06 ± 3.62	0.532
ALP (U/L)	83.99 ± 29.46	82.90 ± 38.18	0.902

Table 6: Comparison according to disease duration within the CKD group.

Marker	≤4 years (n=32)	>4 years (n=28)	P value
TSH (µIU/mL)	4.00 ± 1.13	4.23 ± 1.36	0.482
T3 (ng/mL)	0.74 ± 0.31	0.78 ± 0.36	0.581
T4 (µg/dL)	5.73 ± 1.46	5.97 ± 1.69	0.554
AST (U/L)	13.42 ± 2.58	14.08 ± 4.61	0.510
ALT (U/L)	11.60 ± 3.11	12.11 ± 3.72	0.574
ALP (U/L)	81.97 ± 33.23	84.93 ± 36.54	0.746

Table 7: ROC analysis of thyroid hormones and liver enzymes.

Marker	AUC	Cut-off	Rule	Sensitivity (%)	Specificity (%)
TSH (µIU/mL)	0.916	2.07	Patient if value >= cut-off	98.3	73.3
T3 (ng/mL)	0.782	0.59	Patient if value <= cut-off	55.0	93.3
T4 (µg/dL)	0.796	5.17	Patient if value <= cut-off	56.7	96.7
AST (U/L)	0.988	10.40	Patient if value >= cut-off	93.3	96.7
ALT (U/L)	0.871	9.00	Patient if value >= cut-off	81.7	80.0
ALP (U/L)	0.658	78.30	Patient if value >= cut-off	51.7	96.7

Table 8: Selected Pearson correlations among the studied variables in CKD patients.

Variable 1	Variable 2	r	P value
Age	TSH (µIU/mL)	-0.255	0.049
Age	ALP (U/L)	-0.262	0.043
T3 (ng/mL)	T4 (µg/dL)	0.999	<0.001
AST (U/L)	ALT (U/L)	0.666	<0.001

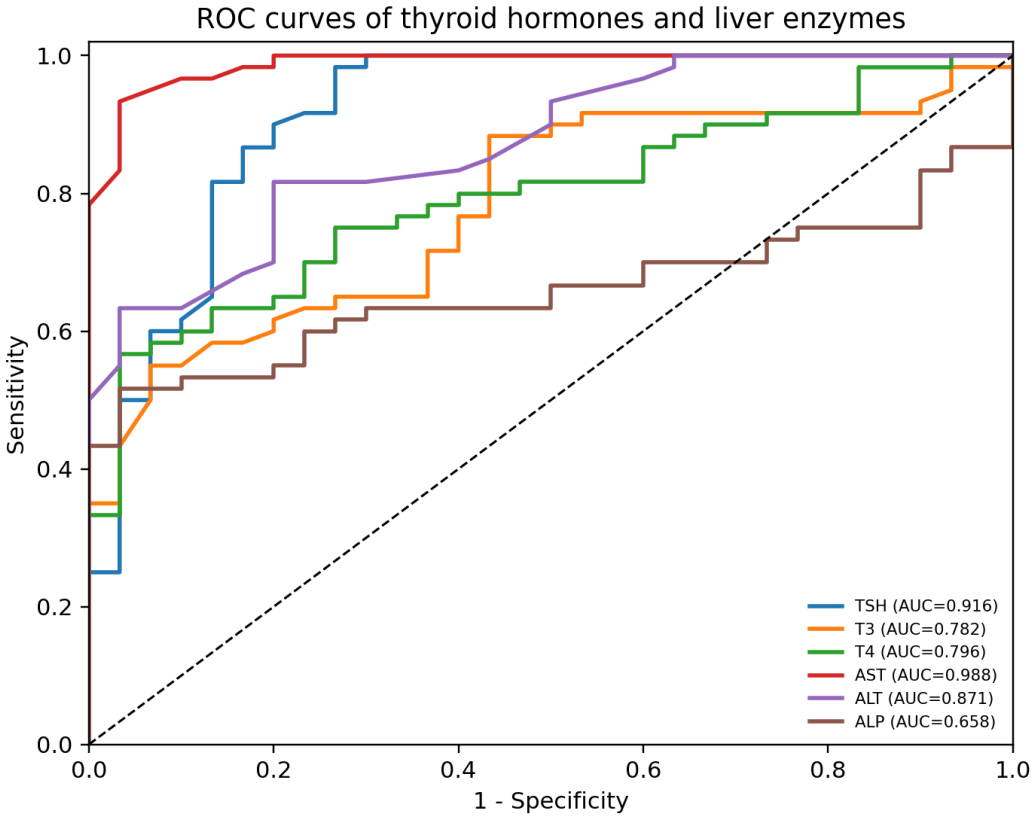

Figure 1: ROC curves for thyroid hormones and liver enzymes.

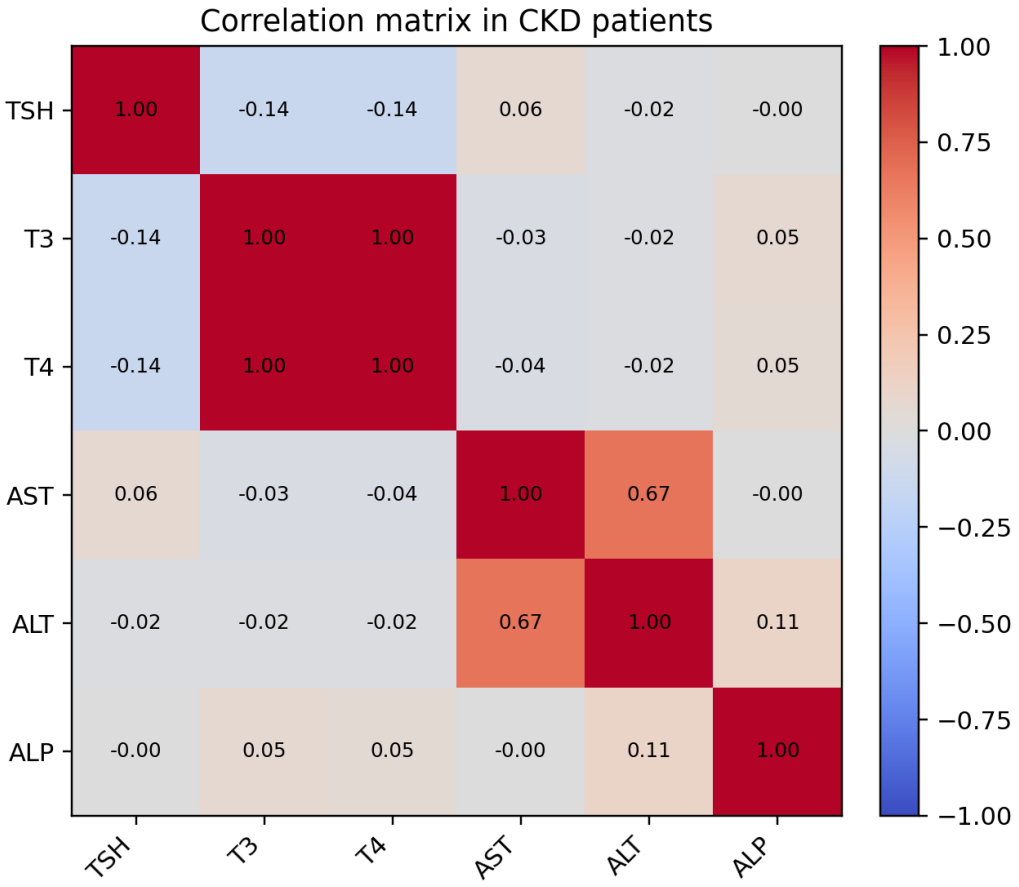

Figure 2: Correlation heatmap of thyroid hormones and liver enzymes in CKD patients.

DISCUSSION

The present study demonstrated that CKD patients undergoing maintenance hemodialysis in Al-Qaim General Hospital had a distinct pattern of thyroid and liver biochemical alterations. Relative to controls, the patients showed significantly higher TSH and lower T3 and T4, together with significantly higher AST, ALT, and ALP. Taken together, these findings support the concept that advanced CKD is accompanied by clinically relevant endocrine and metabolic disturbances extending beyond traditional renal indices.[4] The thyroid findings are biologically plausible. Reduced renal clearance of inflammatory mediators, altered protein binding, impaired peripheral conversion of T4 to T3, and the catabolic environment of uremia all contribute to a thyroid profile that tends to shift toward low T3 and compensatory changes in TSH as kidney disease progresses.[5] Our overall hormonal pattern is in keeping with more recent nephro-endocrine reviews that describe low T3 and altered TSH regulation as frequent abnormalities in CKD and hemodialysis populations.[6] The present results also agree with recent cross-sectional clinical studies reporting a considerable burden of thyroid dysfunction in CKD patients and supporting the need for routine thyroid evaluation in this setting.[19] A further point of agreement comes from recent work in Indian CKD cohorts showing that thyroid abnormalities remain common across different clinical settings and disease stages, which strengthens the clinical significance of our observations.[20]

Additional contemporary evidence has also shown that thyroid dysfunction becomes more apparent across advancing CKD stages, supporting the interpretation that the abnormalities observed in our patients are part of a broader and reproducible CKD-related endocrine pattern.[21] Within the patient group, female patients had significantly higher TSH values than males, while T3 and T4 did not differ significantly by sex. This suggests that sex-related variation in pituitary-thyroid axis response may persist even when the dominant metabolic effect of CKD is shared by both sexes. Although our sample size was modest, this difference is still noteworthy and should be interpreted in parallel with the broader tendency for thyroid dysfunction to be more frequent among women in many clinical populations.[7] The age-based findings in our study showed significantly higher TSH and ALP in patients younger than 50 years. Because the absolute sample size in each age stratum was limited, and because the patient and control groups were not age-matched, this result should be interpreted cautiously. Nevertheless, the correlation analysis supported a weak inverse association between age and both markers. The age effect observed here may reflect differences in disease phenotype, nutritional status,

inflammatory burden, or unmeasured treatment variables rather than age alone.

Regarding liver enzymes, our findings differ from the classical pattern described in many hemodialysis studies, where AST and ALT were often lower than in individuals with preserved renal function.[9,10] This contrast is important because aminotransferase behavior in CKD is not uniform and may vary according to dialysis timing, hydration status, hemoconcentration, nutritional status, comorbidities, viral hepatitis burden, and local laboratory context.[9,10] In the present work, both AST and ALT were significantly higher in patients than controls, suggesting that the biochemical profile of our cohort may reflect combined effects of uremic metabolic stress, oxidative injury, medication exposure, subclinical hepatic involvement, and local case-mix characteristics rather than a single mechanism. Supportive evidence for higher enzyme profiles in selected dialysis cohorts does exist. Liberato et al. demonstrated that aminotransferase levels increased significantly after the hemodialysis session and interpreted this pattern in light of hemodilution before dialysis and hemoconcentration afterward, indicating that the timing of blood sampling can materially influence the observed AST and ALT values.[22] More recently, Gad-Allah et al. reported significantly increased AST and ALP in ESRD patients relative to healthy controls and concluded that post-dialysis liver enzyme levels may be elevated in specific ESRD populations, particularly when diabetes coexists.[23] Thus, although reduced aminotransferase activity remains the traditional pattern in many reports, the present findings are still biologically and clinically plausible within the broader heterogeneity of dialysis populations.

ALP was also significantly elevated in CKD patients. This is a plausible and clinically meaningful finding because ALP in advanced CKD may reflect bone remodeling and CKD-mineral and bone disorder in addition to possible hepatobiliary contributions.[11,12] Recent work has further shown that the liver fraction of circulating ALP is elevated in CKD and is associated with mortality among patients treated with hemodialysis, highlighting that increased ALP may capture inflammatory and hepatic as well as skeletal pathways in this population.[24] The clinical importance of ALP is also reinforced by earlier reports linking higher serum ALP to worse outcomes in maintenance hemodialysis populations.[13] The ROC analysis further strengthened the interpretive value of the study, but these findings should be viewed as discriminatory performance within this dataset rather than as stand-alone diagnostic tools for CKD itself.[18] AST showed excellent discrimination, while TSH and ALT also demonstrated strong performance. The correlation results also showed good internal coherence. The very strong positive correlation between T3 and T4 is physiologically expected because both hormones arise from the same thyroid axis, although its near-perfect magnitude should still be interpreted with caution. Likewise, the strong positive relationship between AST and ALT is consistent with their shared origin as aminotransferases and supports the internal reliability of the biochemical dataset. No significant differences were found according to monthly dialysis frequency or disease duration. This may indicate that, within the ranges represented in our cohort, the biochemical abnormalities were more closely linked to the presence of established hemodialysis-dependent CKD itself than to modest variation in exposure time or treatment frequency. However, a larger sample could reveal subtler associations that were not detectable here. The present study has practical value because it provides local data from Al-Qaim district, an area for which published biochemical information on CKD patients remains limited. At the same time, several limitations should be acknowledged. The study was single-center, cross-sectional, and based on a relatively modest sample size. In addition, the patient and control groups were not age-matched. Important clinical variables such as viral hepatitis status, nutritional markers, bone-specific ALP, medication history, and detailed thyroid autoantibody data were not available. Consequently, the findings should be interpreted as clinically informative but not mechanistically definitive.

CONCLUSION

Patients with chronic kidney disease undergoing maintenance hemodialysis in Al-Qaim General Hospital exhibited significantly increased TSH, AST, ALT, and ALP together with significantly decreased T3 and T4 compared with apparently healthy controls. Female patients had higher TSH than male patients, whereas younger patients showed higher TSH and ALP than older patients. No clear effect of monthly dialysis frequency or disease duration was detected. AST, TSH, and ALT showed the strongest discriminatory performance within this dataset. These results support the value of integrated thyroid and liver biochemical assessment in hemodialysis patients and provide locally relevant data from western Iraq.

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