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Immunomodulatory Pathways of Some Biomarkers in Individuals with Renal Failure

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***Abstract*—This study investigates the correlation between renal failure and immunoinflammatory marker pathways. Specimens were obtained from the Hemodialysis Center located in the cities of Ranya and QaladezeSulaymaniyah, KRG, Iraq. The investigation included a well-defined cohort of 120 individuals (60 patients and 60 control participants), comprising both sexes, during the period from October to December 2024. Participants ranged in age from 40 to 60 years. The concentrations of immunoglobulins (IgA, IgM, and IgG) were measured using the Cobas 6000 analyzer, while IgE levels were assessed with the Cobas e 411 analyzer. The enzyme-linked immunosorbent assay was utilized for detecting the variations of cytokines (Tumor necrosis factor-alpha, interleukin (IL)-6, and IL-10) and antinuclear antibody (ANA). Erythrocyte sedimentation rate (ESR) was determined using the automated ESR analyzer. Individuals diagnosed with end-stage renal disease (ESRD) exhibited significant immunological alterations, marked by a significant decrease in IgA, IgM, and IgG levels and accompanied by a substantial elevation in IgE levels. The current investigation revealed a markedly elevated ESR, tumor necrosis factor- alpha (TNF-α), IL-6, and IL-10 in reference to the healthy subjects. ANA titers exhibited a slight increase within the patient population. This immunological dysregulation is further associated with elevated levels of TNF-α, IL-6, and IL-10, indicating a persistent systemic inflammatory state. The synchronous augmentation of ESR and TNF-α and IL-6 confirms their potential utility as prognostic biomarkers for chronic inflammation in ESRD patients. Observation of elevated inflammatory markers in females indicates a potential gender-specific variation in immune response pathways within this clinical population.**

***Index Terms*—Antinuclear antibody, Chronic kidney disease, Immunoglobulin, Inflammatory and anti-inflammatory cytokines**

I. Introduction

Immunomodulators represent exogenous or endogenous substances that alter the magnitude, type, duration, or efficacy of the immune response (Singh, et al., 2022). Various types of renal disorders, independent of the precipitating factors, manifest inflammation and the activation of the immune system as a prevalent fundamental mechanism (Imig and Ryan, 2013). In the context of renal pathologies, the processes of inflammation, immune cell accumulation, and cellular apoptosis engender a detrimental feedback loop characterized by the deposition of fibrous matrices, which compromises the structural integrity of renal tissues and ultimately culminates in renal impairment (Petreski, et al., 2021). Immune cells possess the capability to sustain renal homeostasis and mitigate renal injury. Furthermore, agents that suppress immune responses were applied in the therapeutic renal disease management (Zhang, et al., 2012). The innate immunity cells play a pivotal role in renal immune mechanisms (Hao, et al., 2024). Impairment of renal function compromises the overall immune system, resulting in the deterioration of the intestinal barrier, the development of systemic inflammatory responses, and a state of immune deficiency; in contrast, the kidneys may also function as targets for harmful immune responses against renal auto-antigens and as locations for localized manifestations of systemic autoimmune disorders (Kurts, et al., 2013; Cohen, 2020). Acute renal failure is marked by a rapid decline in renal efficiency, as demonstrated by a fall in the glomerular filtration rate (Nazar, et al., 2015). A kidney disease signifies a progressive pathological state in both structural and functional changes within the renal system, resulting from numerous etiological influences (Kalantar-Zadeh, et al., 2021; Webster, et al., 2017). A notable deficiency for immunoglobulin (Ig) concentrations was identified in a significant proportion of individuals afflicted with uremia throughout the advancement of chronic renal impairment, indicating a potential suppression of their biosynthesis attributable to the uremic condition (Yarlagadda, et al., 2019). Increased levels of IgA in both serum and urine have been documented in patients, particularly among those exhibiting significant proteinuria (Nakayama, et al., 2008). However, similar to IgG and IgM, the concentrations of IgA did not demonstrate a statistically significant association with renal dysfunction (Anis, et al., 2016). The participation of IgE in nephropathy presents substantial challenges. Increased concentrations of IgE may indicate an alteration in the dynamics of the immune response (Tan, et al., 2011). The group exhibiting renal progression demonstrated a statistically significant increase in serum

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IgE concentrations in comparison to the control group (Lee, et al., 2016). Tumor necrosis factor alpha serves as a modulator within the inflammatory signaling pathways and is acknowledged for its participation in the pathophysiological mechanisms that define numerous inflammatory and autoimmune diseases. This cytokine is produced by a diverse array of immune cells (Jang, et al., 2021; Brierly, et al., 2023; Samimi, et al., 2020). The fundamental function of interleukin (IL)-10 is to act as an anti-inflammatory mediator, thereby safeguarding the organism against an excessive immune response. Moreover, IL-10 has the potential to demonstrate immunostimulatory characteristics (Carlini, et al., 2023). The role of IL-6 in various renal pathologies is increasingly recognized within scholarly discussions (Su, Lei and Zhang, 2017). IL-6 is integral to the pathophysiological mechanisms that underlie renal dysfunction, and it holds considerable prognostic value concerning the extent of such dysfunction (González-Lafuente, et al., 2024). The antinuclear antibody assay is utilized as a preliminary diagnostic instrument for the detection of autoimmune diseases (Ge, et al., 2022). Moreover, the detection of antinuclear antibodies has been reported in patients suffering from acute and chronic infectious diseases, neoplastic conditions, adverse effects of therapeutic agents, as well as in individuals who present with no apparent health issues (Sur, et al., 2018). The erythrocyte sedimentation rate (ESR) serves as an essential biomarker relevant to systemic inflammatory responses, particularly in evaluating its clinical implications in individuals experiencing renal insufficiency (Buckenmayer, et al., 2022). Consequently, the current study aims to explore the correlation between renal insufficiency and the pathways associated with immunological markers.

II. Materials And Methods

A. Materials and Instruments

Table I illustrates the various chemical agents, instruments, and suppliers employed for the evaluation of immunoinflammatory biomarkers within the scope of the research.

B. Study Design and Methods

This research was systematically organized to shed light on immune variations, involving immunoglobulin concentrations as well as proinflammatory and anti-inflammatory indicators, in those afflicted by renal failure. This research included two separate cohorts. The initial cohort, known as the patient group, consisted of individuals of both genders who were diagnosed with end-stage renal disease (ESRD) (stage V) and were aged between 40 and 60 years. Individuals at earlier stages of the condition, along with minors and adolescents, were systematically excluded from the analysis. The second cohort, identified as the control group, was made up of 60 healthy individuals, both male and female, aged 40–60 years, who showed no signs of renal pathologies. The subjects constituting the patient cohort and the control group demonstrated congruencies in socioeconomic status, social background, and living conditions. All participants engaged in the study were sourced from neighboring areas within the Raparin District of the Sulaymaniyah Governorate. Specimens were procured under rigorously regulated and standardized experimental conditions, subsequently leading to the clear division of the samples into three distinct tubes. The initial tube was allocated for the evaluation of Ig concentrations (IgA, IgM, IgG, and IgE), while the second tube was designated for the quantification of cytokine levels (tumor necrosis factor-alpha [TNF-α], IL-10, human IL-6), and antinuclear antibody [ANA]); the third tube was reserved for the determination of ESR. The protocol for serum extraction from whole blood was conducted through centrifugation at a centrifugal force of 1000 g for a duration of 30 min at ambient temperature. Before the execution of any analytical methodologies, aliquots of the samples were assiduously prepared and preserved at a temperature of −20°C. The enzyme-linked immunosorbent assay was utilized to accurately measure the cytokines and ANA. The levels of Igs were determined using the Cobas 6000 analyzer, with the exception that IgE was measured employing the Cobas e 411 analyzer. The ESR was evaluated through the utilization of the automatic ESR analyzer.

C. Ethical Consideration

Each of the suggested protocols and methodologies detailed in this investigation was subjected to a thorough evaluation and attained approval from the scientific committee of the biology department, alongside the head of the research institution at the University of Raparin-Rania/Raparin district of the Kurdistan Region of Iraq.

TABLE I
Chemicals and Equipment in the Current Study

No.	Chemical material (Kits)	Instrument	Supplier and company	Country
1	Immunoglobulin A	Cobas 6000 analyzer	Roche diagnostics	Germany
2	Immunoglobulin M	Cobas 6000 analyzer	Roche diagnostics	Germany
3	Immunoglobulin G	Cobas 6000 analyzer	Roche diagnostics	Germany
	Immunoglobulin E	Cobas e 411 analyzer	Roche diagnostics	Germany
4	Tumor necrosis factor alpha (Human)	ELISA-based test system	Fine biotech	China
5	Human Interleukin 10	ELISA-based test system	Fine biotech	China
6	Human interleukin-6	ELISA-based test system	Fine biotech	China
7	Antinuclear antibody	ELISA-based test system	Fine biotech	China
8	Erythrocyte sedimentation rate	Automatic ESR analyzer smart rate 10	JOKOH	Japan

ELISA: Enzyme-linked immunosorbent assay, ESR: Erythrocyte sedimentation rate

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D. Statistical Analysis

Statistical assessments and graphical illustrations were executed with GraphPad Prism version 10.5.0 (774) to assure a high degree of precision and the production of outputs of professional quality. A significance level of 0.05 for the p-value was established as the standard for determining statistical significance for all results acquired.

III. Results and Discussion

A. Results

The assessment of Ig levels (IgA, IgM, IgG, IgE), ESR, ANA, TNF-α, IL-6, and IL-10 was comprehensively conducted in both individuals afflicted with renal failure and control subjects. The results are delineated by gender and overall mean, with standard errors as indicated in (Tables II-IV).

The Shapiro–Wilk test reveals a significant prevalence of non-normality in immunological markers, especially within patient cohorts and across both genders. This implication underscores the necessity for employing non-parametric statistical approaches for forthcoming analyses concerning these variables. Biomarkers yielding a $p < 0.05$ are considered to represent a non-normal distribution, as shown in Tables V and VI.

A preponderance of immunological biomarkers reveals deviations from normative values within the patient cohort, in contrast to numerous biomarkers that exhibit a normal distribution within the control group. Significant exceptions include IgG and IgE, which reveal a normal distribution among patients yet not within controls, alongside IgM and ANA, both of which demonstrate non-normal distributions in each cohort. The ensuing table articulates the findings resulting from the Shapiro–Wilk assessment for normality, systematically categorized by clinical classification.

The preponderance of biomarkers demonstrates non-normative distributions across both genders. Significant deviations are represented by ESR (which conforms to normality in males) and IgE (which adheres to normality in females), underscoring the possibility of distributional discrepancies based on sex.

To further exp through graphical representation, as indicated in (Figs. 1-9). This study thoroughly examines the serum levels of IgA, IgM, IgG, IgE, ESR, ANA, TNF-α, IL-6, and IL-10 in individuals recognized as having renal impairment relative to healthy control subjects. Box plot analysis was employed to visually clarify the distribution, central tendency, and variability of these biomarkers. The results highlight potential immunological alterations and inflammatory responses that are linked to renal pathology. These findings suggest significant immunological changes and inflammatory processes associated with kidney dysfunction.This investigation assesses the interrelationships among IgA, IgM, IgG, and IgE, ESR, cytokines markers (TNF-α, IL-6, and IL-10), and autoimmune indicators (ANA) utilizing Spearman's rho statistical method. Robust correlations identified between particular markers imply interrelated pathways of immune system activation and systemic inflammatory responses. These results may elucidate mechanisms of immune dysregulation pertinent to inflammatory or autoimmune disorders, as given in Table VII.

IV. Discussion

The individuals diagnosed with renal failure exhibited markedly reduced mean concentrations of IgA (1.671 g/L

TABLE II
Means±Standard Error of Parameters in Control Participants

Gender	IgA G/L	IgM G/L	IgG G/L	IgE IU/mL	ESR mm/h	ANA IU/mL	TNF-α pg/mL	IL-6 pg/mL	IL-10 pg/mL
Female	2.641±0.073	1.624±0.067	10.828±0.389	57.752±4.093	42.500±0.686	0.782±0.042	6.903±0.342	1.059±0.065	6.820±0.351
Male	2.760±0.094	1.261±0.093	10.759±0.461	30.447±4.712	30.333±1.020	0.584±0.041	4.167±0.409	1.377±0.077	6.693±0.487
Total	2.701±0.060	1.443±0.061	10.794±0.299	44.100±3.568	36.417±0.999	0.683±0.032	5.535±0.319	1.218±0.054	6.757±0.297

Ig: Immunoglobulin, ESR: Erythrocyte sedimentation rate, ANA: Antinuclear antibody, TNF-α: Tumor necrosis factor- alpha, IL: Interleukin

TABLE III
Mean±Standard Error of Parameters in Patient Participants

Gender	IgA G/L	IgM G/L	IgG G/L	IgE IU/mL	ESR mm/h	ANA IU/mL	TNF-α pg/mL	IL-6 pg/mL	IL-10 pg/mL
Female	1.719±0.096	1.985±0.047	7.994±0.080	68.333±2.495	61.833±0.871	0.741±0.075	47.487±18.644	6.747±2.291	15.433±2.735
Male	1.622±0.046	1.560±0.072	7.546±0.093	66.367±3.708	45.567±0.895	0.733±0.103	22.170±2.461	5.280±1.665	14.560±2.060
Total	1.671±0.053	1.773±0.051	7.770±0.067	67.350±2.219	53.700±1.227	0.737±0.063	34.828±9.467	6.013±1.407	14.997±1.698

Ig: Immunoglobulin, ESR: Erythrocyte sedimentation rate, ANA: Antinuclear antibody, TNF-α: Tumor necrosis factor- alpha, IL: Interleukin

TABLE IV
Combined Summary Of All Participants

Gender	IgA G/L	IgM G/L	IgG G/L	IgE IU/mL	ESR mm/h	ANA IU/mL	TNF-α pg/mL	IL-6 pg/mL	IL-10 pg/mL
Female	2.180±0.085	1.804±0.047	9.411±0.270	63.043±2.474	52.167±1.373	0.762±0.043	27.195±9.614	3.903±1.195	11.127±1.478
Male	2.191±0.090	1.411±0.061	9.153±0.313	48.407±3.782	37.950±1.198	0.659±0.056	13.168±1.704	3.329±0.865	10.627±1.168
Total	2.186±0.062	1.608±0.042	9.282±0.206	55.725±2.348	45.058±1.117	0.710±0.035	20.182±4.904	3.616±0.735	10.877±0.938

Ig: Immunoglobulin, ESR: Erythrocyte sedimentation rate, ANA: Antinuclear antibody, TNF-α: Tumor necrosis factor- alpha, IL: Interleukin

TABLE V
The Normality of Immunological Marker Distributions Across Clinical Groups

Marker	Control (p-value)	Patient (p-value)	Normality interpretation
IgA	0.148	0.000	Standard in the control subject; abnormal in the patient group
IgM	0.005	0.014	Non-standard in the control subject and patients group
IgG	0.000	0.298	Non-standard in the control subject; normal in patient group
IgE	0.006	0.275	Non-standard in the control subject; normal in patient group
ESR	0.080	0.013	Standard in the control subject; non-normal in patients group
ANA	0.005	0.000	Non-standard in the control subject and patient group
TNF-α	0.286	0.000	Standard in the control subject; non-normal in patients group
IL-6	0.246	0.000	Standard in the control subject; non-normal in patient group
IL-10	0.141	0.000	Standard in the control subject; non-normal in patient group

Ig: Immunoglobulin, ESR: Erythrocyte sedimentation rate, ANA: Antinuclear antibody, TNF-α: Tumor necrosis factor- alpha, IL: Interleukin

TABLE VI
The Normality of Immunological Marker Distributions Based on Participant Sex

Marker	Female (p-value)	Male (p-value)	Normality interpretation
IgA	0.024	0.017	Non-normal in both sexes
IgM	0.050	0.003	Non-normal in both sexes
IgG	0.000	0.000	Non-normal in both sexes
IgE	0.122	0.004	Normal in female; non-normal in male
ESR	0.000	0.273	Non-normal in female; normal in male
ANA	0.003	0.000	Non-normal in both sexes
TNF-α	0.000	0.000	Non-normal in both sexes
IL-6	0.000	0.000	Non-normal in both sexes
IL-10	0.000	0.000	Non-normal in both sexes

Ig: Immunoglobulin, ESR: Erythrocyte sedimentation rate, ANA: Antinuclear antibody, TNF-α: Tumor necrosis factor- alpha, IL: Interleukin

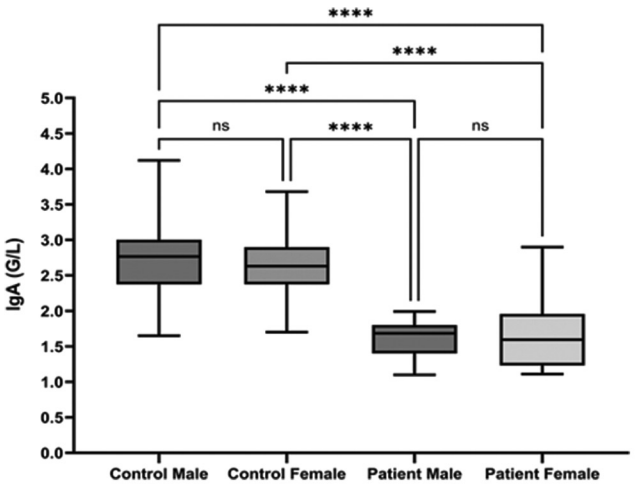

Fig. 1. Serum immunoglobulin A in both (patients and control) groups.

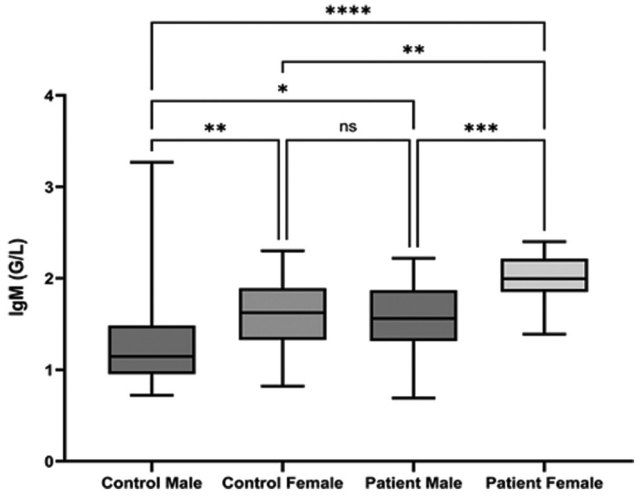

Fig. 2. Serum immunoglobulin M in both (patients and control) groups.

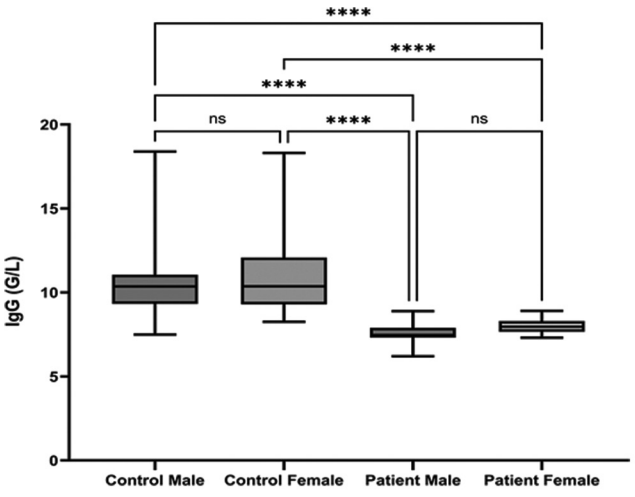

Fig. 3. Serum immunoglobulin G in both (patients and control) groups.

compared to 2.701 g/L), IgM (1.773 g/L vs. 1.443 g/L), and IgG (7.770 g/L as opposed to 10.794 g/L) relative to healthy control subjects. Conversely, IgE levels were found to be elevated in the patient cohort (67.35 IU/mL compared to 44.1 IU/mL). The observed reduction in IgA, IgG, and IgM levels indicates a potential compromise in humoral immunity among individuals with renal failure, which may be attributable to the uremic suppression of B-cell functionality or the loss of proteins through dialysis. Augmented IgE levels could potentially represent a significant biomarker for systemic inflammatory activities or allergic phenomena commonly documented in individuals afflicted by uremia and those in dialysis treatment (Kato, et al., 2019; Carrero and Stenvinkel, 2018). Participants demonstrated a notably heightened ESR of 53.7 mm/h relative to the control cohort, which exhibited an ESR of 36.42 mm/h, thereby suggesting the possible presence of chronic inflammatory conditions. Moderate inflammatory responses, which could be correlated with the presence of uremic toxins, oxidative stress, and the application of dialysis membranes (Stenvinkel, Pecoits-Filho, and Lindholm, 2020). The titers of ANA observed in the patients (0.737) were identified as being marginally elevated when compared to those detected in the control group (0.683),

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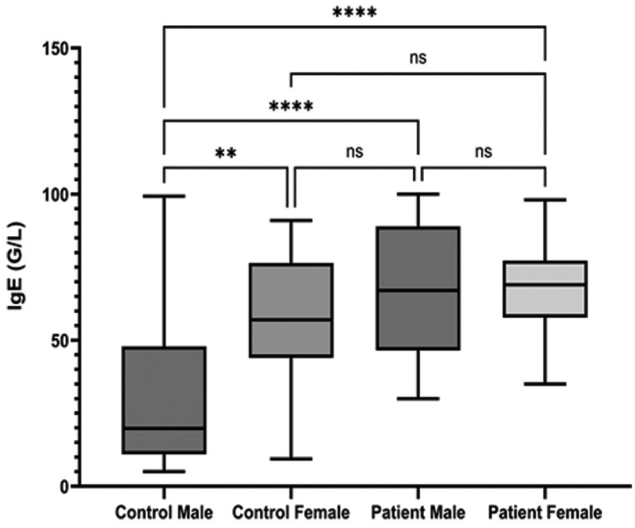

Fig. 4. Serum immunoglobulin E in both (patients and control) groups.

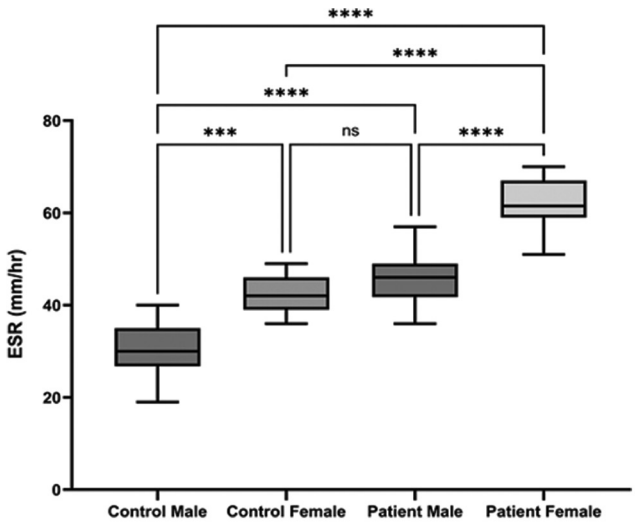

Fig. 5. Erythrocyte sedimentation rate in both (patients and control) groups.

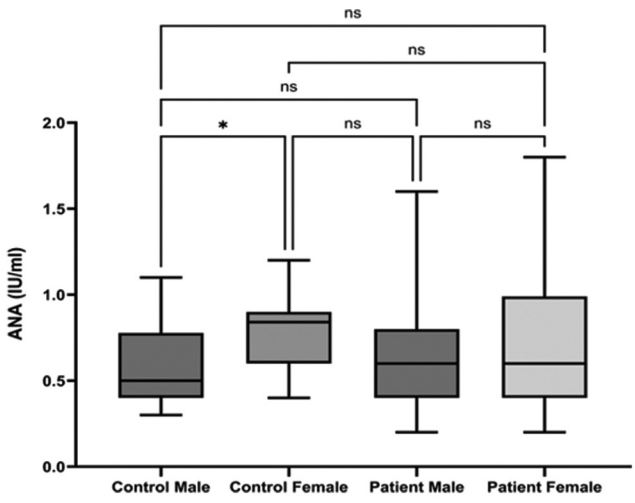

Fig. 6. Antinuclear antibody in both (patients and control) groups.

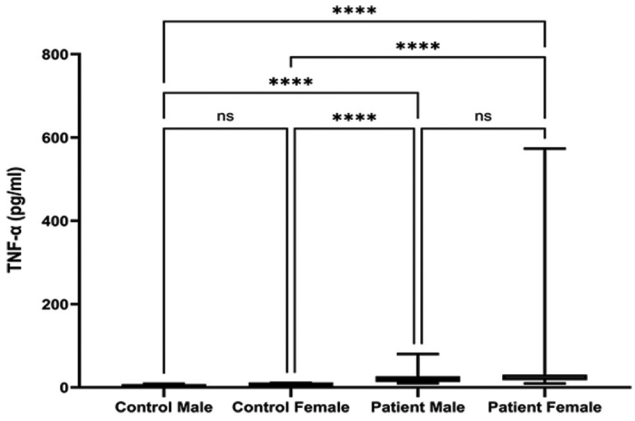

Fig. 7. Tumor necrosis factor-alpha in both (patients and control) groups

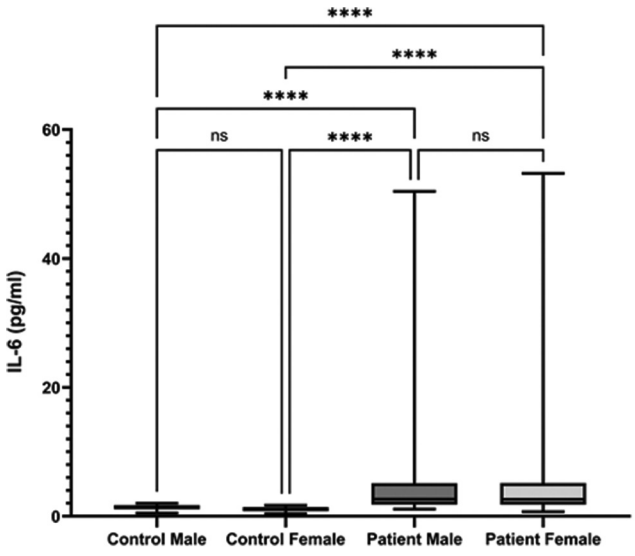

Fig. 8. Interleukin-6 in both (patients and control) groups.

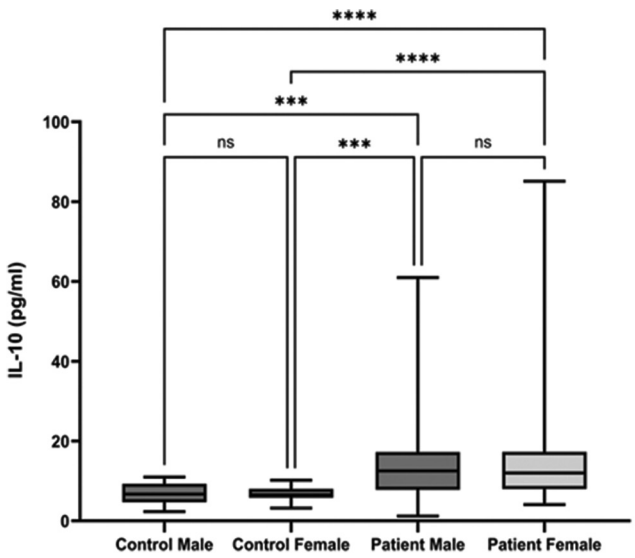

Fig. 9. Interleukin-10 in both (patients and control) groups.

TABLE VII
Correlation Matrix of Immunoglobulins and Inflammatory Biomarkers in Renal Failure Patients

Markers	IgA	IgM	IgG	IgE	ESR	ANA	TNF-α	IL-6	IL-10
IgA	1.000	−0.284**	0.639**	−0.347**	−0.541**	0.005	−0.648**	−0.557**	−0.458**
		0.002	0.000	0.000	0.000	0.956	0.000	0.000	0.000
IgM		1.000	−0.238**	0.267**	0.551**	0.126	0.453**	0.270**	0.261**
			0.009	0.003	0.000	0.169	0.000	0.003	0.004
IgG			1.000	−0.368**	−0.494**	−0.048	−0.661**	−0.650**	−0.511**
				0.000	0.000	0.602	0.000	0.000	0.000
IgE				1.000	0.452**	0.084	0.430**	0.325**	0.212*
					0.000	0.360	0.000	0.000	0.020
ESR					1.000	0.120	0.722**	0.487**	0.372**
						0.191	0.000	0.000	0.000
ANA						1.000	0.040	−0.043	0.053
							0.661	0.638	0.564
TNF-α							1.000	0.566**	0.567**
								0.000	0.000
IL-6								1.000	0.379**
									0.000
IL-10									1.000

The correlation coefficients according to Spearman's rank (ρ) with respect to the serum immunoglobulin classes (IgA, IgM, IgG, and IgE) and ESR, ANA, TNF-α, IL-6, and IL-10 have been described. A P value limit of *p<0.05 and **p<0.01 was designated as the reference point for assessing the correlation test
Ig: Immunoglobulin, ESR: Erythrocyte sedimentation rate, ANA: Antinuclear antibody, TNF-α: Tumor necrosis factor- alpha, IL: Interleukin

although this differentiation was considered to exhibit negligible variation. Although ANA is largely correlated with autoimmune diseases, it may also appear as non-specifically elevated in chronic conditions, including ESRD. The slight variation detected in this context indicates a constrained relevance of ANA in differentiating individuals undergoing renal failure from those classified as healthy (Pisetsky, 2021). A marked increase in TNF-α concentrations is noted among patients (34.83 pg/mL) relative to control subjects (5.54 pg/mL), with female patients exhibiting even more substantial levels. TNF-α functions as a principal mediator of inflammatory processes and is integral to the pathophysiology of renal failure by promoting mechanisms such as fibrosis, apoptosis, and inflammation. Elevated TNF-α levels have been correlated with poor prognoses in patients receiving dialysis and an increased likelihood of cardiovascular events (Wang and Mitch, 2020). Both IL-6 (6.01 pg/mL compared to 1.22 pg/mL) and IL-10 (14.99 pg/mL compared to 6.76 pg/mL) revealed statistically significant elevations in individuals afflicted with renal failure. IL-6 operates as an inflammatory marker. Raised levels of IL-10, even with its established anti-inflammatory functions, propose a refined regulatory influence in the alteration of immune responses. This imbalance in cytokine levels represents a crucial reflection of the pathological processes involved in ESRD (Kalantar-Zadeh, et al., 2021). The finding that female patients show increased concentrations of TNF-α, IL-6, and IL-10 when compared to their male counterparts indicates a likely gender-specific variation in immune responses. These results support the well-established theory that estrogen might regulate immune processes and intensify inflammatory responses, which could play a role in clarifying these differences (Klein and Flanagan, 2016). The IgA exhibited robust positive correlations with IgG ($\rho = 0.639$, $p < 0.01$), alongside significant negative correlations with proinflammatory cytokines, including TNF-α (−0.648), IL-6 (−0.557), IL-10 (−0.458), and ESR (−0.541). The presence of an inverse correlation might indicate a prospective regulatory capacity of IgA or its decline in the setting of intensified inflammatory reactions. Elevated IgA levels are commonly recorded in mucosal and renal ailments, as illustrated by the pathology identified as IgA nephropathy, distinguished by immune system dysfunction (Suzuki, et al., 2011). IgM demonstrated a noteworthy positive correlation with ESR (0.551), TNF-α (0.453), IL-6 (0.270), and IL-10 (0.261). The elucidation of this association underscores the pivotal significance of IgM during the initial phases of the immune response and within the mechanisms that regulate acute-phase inflammatory processes. As the primary antibody produced in response to an immunological stimulus, IgM is critical for the initiation of complement activation pathways and the modulation of macrophage activity (Boes, 2000). These correlations imply a potential role of IgM in the preliminary phases of immune responses and in processes associated with acute-phase inflammation. The findings presented underscore the importance of IgM in facilitating the initial immune response and in the dynamics of acute-phase inflammatory reactions. IgG demonstrated significant negative associations with TNF-α (−0.661), IL-6 (−0.650), and IL-10 (−0.511). The existence of such inverse relationships indicates that increased systemic inflammation might disrupt IgG synthesis or encourage its degradation, a phenomenon commonly noted in chronic inflammatory disorders (Van der Meide and Schellekens, 1996). IgE exhibited moderate positive correlations with ESR (0.452), TNF-α (0.430), and IL-6 ($\rho = 0.325$), while illustrating a negative correlation with IgG (−0.368). The data indicate the occurrence of an allergic response or a dominant inflammatory pathway typified by T-helper cell Type 2 activities. IgE assumes a crucial function in facilitating systemic inflammation within the framework of chronic allergic and autoimmune conditions. Such observations underscore the significance of IgE in the

context of inflammatory responses associated with allergic and autoimmune pathologies (Galli and Tsai, 2012). The ESR exhibited a significant positive correlation with TNF-α (0.722), IL-6 (0.487), IL-10 (0.372), and IgM (0.551), while demonstrating an inverse relationship with IgA and IgG. These results substantiate the role of ESR as a reliable biomarker for systemic inflammation (Brigden, 1999). The ANA did not reveal statistically significant links with various biomarkers, comprising inflammatory cytokines. The quantification of ANA is crucial in autoimmune diagnostics; however, it may lack dynamic alterations in parallel with cytokines (Pisetsky, et al., 2011). TNF-α exhibited a statistically significant positive correlation with both IL-6 and IL-10, thereby suggesting a concurrent up-regulation of these cytokines. The elevation of IL-10 in conjunction with TNF-α serves to attenuate immune-mediated tissue damage (Moore, et al., 2001). The identified negative correlations between TNF-α and IL-6 with IgG and IgA suggest possible immunosuppressive effects during inflammatory processes. These findings underscore the interconnected role of TNF-α and IL-10 in the modulation of immune responses.

V. Conclusion

Subjects suffering from renal insufficiency presented distinct markers of immune system dysfunction, which were defined by diminished Ig levels (IgA, IgM, and IgG) and augmented activity of IgE. The level of ESR was elevated in patients in comparison to the control group. The amounts of ANA detected in the patient cohort were appraised as being somewhat increased relative to the titers manifested in the control group. The finding that female patients exhibit elevated concentrations of (TNF-α, IL-6, IL-10) compared to their male counterparts suggests a possible gender-specific variation in immune responses. The data gathered affirm the systemic inflammatory context that defines ESRD and confirm its relevance in the prognostic analysis of individuals suffering from this condition. The acknowledgment of these inflammatory biomarkers is significant for grasping the profound repercussions of chronic inflammation in relation to cardiovascular morbidity among individuals affected by ESRD.

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