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SERUM LEVELS OF IL-6, TNF-A, AND IL-10 IN PATIENTS WITH CHRONIC KIDNEY DISEASE: A COMPARATIVE STUDY OF PRE- AND POST-DIALYSIS CONCENTRATIONS

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ABSTRACT

Background: Chronic kidney disease and renal failure are associated with persistent systemic inflammation, particularly involving pro-inflammatory and anti-inflammatory cytokines. Hemodialysis (HD), although essential for the management of renal failure, may further influence inflammatory responses through interactions between blood and the dialysis membrane and other dialysis-related factors.

Aim of the Study: This study aimed to evaluate serum levels of interleukin-6 (IL-6), interleukin-10 (IL-10), and tumor necrosis factor-alpha (TNF- α) in renal failure patients undergoing hemodialysis and to compare cytokine concentrations before and after hemodialysis with those of healthy individuals.

Materials and Methods: The study was conducted from March 2026 to August 2026 at Amal Center in Kirkuk. A total of 90 participants were included and divided into three groups: 30 renal failure patients evaluated before hemodialysis (Pre-HD), 30 patients evaluated after hemodialysis (Post-HD), and 30 healthy individuals serving as controls. The control participants were comparable in age and had normal renal parameters with no previous history of renal impairment. Blood samples were collected for determination of serum/plasma IL-6, IL-10, and TNF- α concentrations. Statistical analysis was performed to assess differences among the study groups, with differences considered statistically significant at $P < 0.001$.

Results: The results showed a significant increase in IL-6 levels among hemodialysis patients. The mean IL-6 concentration increased from 13.63 ± 2.10 pg/dl before hemodialysis to 28.72 ± 9.02 pg/dl after hemodialysis, compared with 3.90 ± 3.03 pg/dl in the control group ($P < 0.001$). In contrast, IL-10 levels decreased from 1.23 ± 0.74 pg/dl in the control group to 0.75 ± 0.57 pg/dl before hemodialysis, followed by a significant increase to 3.12 ± 0.52 pg/dl after hemodialysis ($P < 0.001$). Similarly, TNF- α levels demonstrated a progressive increase, from 0.53 ± 0.28 pg/dl in controls to 1.11 ± 0.41 pg/dl before hemodialysis and 1.59 ± 0.57 pg/dl after hemodialysis ($P < 0.001$).

Conclusion: Hemodialysis was associated with significant alterations in circulating inflammatory cytokines. The marked elevation of IL-6 and TNF- α in hemodialysis patients indicates an enhanced pro-inflammatory state, particularly following the dialysis session. The increase in IL-10 after hemodialysis may reflect activation of compensatory anti-inflammatory mechanisms in response to the inflammatory stimulus.

Keywords: Chronic renal failure; Hemodialysis; Interleukin-6; Interleukin-10; Tumor necrosis.

1 INTRODUCTION

Chronic kidney disease (CKD) is a progressive disorder characterized by persistent abnormalities in kidney structure or function and represents an important global health problem. The progression of CKD is accompanied by metabolic disturbances, accumulation of uremic toxins, oxidative stress, and alterations in immune function. These changes contribute to the development of a persistent low-grade inflammatory state, particularly in patients with advanced CKD and end-stage kidney disease (ESKD) receiving renal replacement therapy (1, 2). Chronic inflammation in CKD is clinically important because it is associated with cardiovascular disease, protein-energy wasting, anemia, and increased morbidity and mortality among patients undergoing dialysis(2-4).

The inflammatory environment associated with CKD involves a complex imbalance between pro-inflammatory and anti-inflammatory cytokines. Among the most important pro-inflammatory mediators are interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α), whereas interleukin-10 (IL-10) is considered an important anti-inflammatory cytokine. Alterations in the production, clearance, and biological activity of these cytokines contribute to the persistent inflammatory state observed in uremia. The kidney also plays an important role in the metabolism and elimination of several cytokines; therefore, declining renal function may contribute to their accumulation in the circulation (3).

Interleukin-6 is a multifunctional cytokine involved in acute-phase responses, immune regulation, and inflammatory signaling. Increased IL-6 concentrations have been frequently reported in patients with CKD and particularly in those receiving hemodialysis. Recent evidence confirms that IL-6 is among the important inflammatory biomarkers associated with CKD and dialysis-related inflammation(1). In addition, a systematic review and meta-analysis of randomized controlled trials in hemodialysis patients identified IL-6 and TNF- α among the principal inflammatory biomarkers investigated in this population, emphasizing their relevance to the inflammatory burden associated with maintenance hemodialysis(5).

Tumor necrosis factor-alpha is another major pro-inflammatory cytokine involved in immune-cell activation, endothelial dysfunction, oxidative stress, and amplification of inflammatory responses. Increased TNF- α activity has been associated with the inflammatory milieu of uremia and may contribute to several complications of advanced kidney disease. The altered cytokine network in uremia reflects an imbalance between inflammatory mediators and their regulatory mechanisms, with TNF- α and IL-6 representing important components of this response(3, 6).

In contrast, IL-10 is an important anti-inflammatory cytokine that helps control excessive inflammatory activation by suppressing the production of several pro-inflammatory mediators. The balance between pro-inflammatory cytokines and anti-inflammatory mechanisms is particularly relevant in patients with ESKD because chronic uremic inflammation may coexist with impaired immune regulation. Previous research has suggested that variation in IL-10 production may influence the degree of systemic inflammation and clinical outcomes in patients undergoing hemodialysis (7-9).

Hemodialysis itself may influence the circulating concentrations of inflammatory cytokines. During treatment, blood is exposed to the extracorporeal circuit and dialysis membrane, potentially resulting in complement activation and stimulation of immune cells. The inflammatory response may also be influenced by membrane biocompatibility, dialysate quality, vascular access, and other dialysis-related factors(10-12). Consequently, cytokine concentrations measured before and after a dialysis session may reflect not only the underlying inflammatory status of CKD but also the acute biological response to the dialysis procedure.

However, studies evaluating the acute effect of hemodialysis on cytokine concentrations have produced variable findings. A study by (13)evaluated 21 patients receiving chronic hemodialysis and compared serum concentrations of IL-6 and TNF- α before and after a dialysis session. No significant differences were observed in IL-6 or TNF- α concentrations following dialysis, suggesting that a single hemodialysis session does not necessarily produce an immediate alteration in these pro-inflammatory cytokines.

Similarly, studies investigating cytokine production during hemodialysis have demonstrated that the response may differ among individual cytokines. An increase in IL-6 and IL-10 during hemodialysis, whereas TNF- α did not show a significant increase. These findings suggest that the dialysis procedure may affect pro-inflammatory and anti-inflammatory pathways differently and that the timing of blood sampling may be important when evaluating cytokine concentrations(14).

Further evidence comes from studies examining cytokine production in relation to skeletal muscle and oxidative stress during hemodialysis. Muscular tissue was shown to participate in the production and regulation of inflammatory mediators, including IL-6, IL-10, and TNF- α , in patients with ESKD. This supports the concept that the inflammatory

response during dialysis is complex and involves multiple tissues and biological pathways rather than being determined solely by renal clearance of cytokines(15).

The type of dialysis treatment and dialysis-related exposure may also influence cytokine responses. In a prospective study comparing different hemodialysis strategies, TNF- α decreased significantly during dialysis, whereas IL-6 and IL-10 did not show significant intradialytic changes. These findings further demonstrate that cytokine responses are not uniform and may depend on the characteristics of the dialysis procedure and the inflammatory status of the patient (12).

Accordingly, the present study was designed to determine and compare serum concentrations of IL-6, TNF- α , and IL-10 in patients with chronic kidney disease before and immediately after hemodialysis. The study aims to assess whether a single dialysis session is associated with measurable changes in these pro-inflammatory and anti-inflammatory cytokines and to provide further information regarding the inflammatory response of patients undergoing maintenance hemodialysis.

2 MATERIALS AND METHODS

2.1 Sample collection

The present study was conducted in renal failure patients who were on dialysis From March 2026 to August 2026 at Amal center in Kirkuk. The first group (n:30), for patients before hemodialysis, the second groups(n:30), for patients with after hemodialysis, and third groups (control) (n:30), for normal individuals whose renal parameters were within normal limits and with no history of renal impairment in the past were also selected between the comparable age group,

About 5 ml of venous blood without any anticoagulant was collected into sterile clean and dry polypropylene tubes to avoid contamination. Haemolysis is avoided, blood was allowed to clot. The serum after separation was centrifuged and transferred into sterile, clean and dry polypropylene tubes. The biochemical tests were done on the same day.

2.2 Human IL-6 ELISA test

The ELISA method was employed to evaluate the serum concentrations. The measurement of IL-6 levels was conducted in accordance by using ELISA technique. The plate was pre-coated with an antibody specific to human IL-6. The sample is supplemented with IL-6 content. The concentration of human IL-6 exhibited a positive correlation with the emergence of color in the substrate solution. The cessation of the process is achieved by the introduction of an acidic stop solution, followed by the quantification of absorbance at a wavelength of 450 nm.

2.3 F-2: Human IL-10 ELISA test

The serum concentrations were evaluated by ELISA. The plate was pre-coated with an antibody specific to human IL-10. The IL-10 concentration of the sample is introduced. The concentration of human IL-10 exhibited a positive correlation with the emergence of color in the substrate solution. The process is halted by the introduction of an acidic stop solution, after which the absorbance is quantified at a wavelength of 450 nm.

2.4 F-3: Human IL-4 ELISA test

The concentration of TNF-a was determined using ELISA. The plate has been pre-coated with a human anti-IL-4 antibody. Upon the introduction of TNF-a into the sample, it forms a binding interaction with the antibodies that have been immobilized on the wells. Next, the immune complex is treated with a human biotinylated conjugated IL-4 antibody, which is subsequently introduced to the substrate. The process is terminated by the introduction of an acidic stop solution, after which the absorbance is quantified at a wavelength of 450 nm.

2.5 Statistical Analysis

Data are given as means $\pm$ SE. α Value was set at 0.05. Paired and unpaired *t*-tests were used when applicable. Repeated measures of analysis of variance (ANOVA) were used, with pre- and post-HD vs. control as the grouping factor, with a post hoc Tukey test. Linear regression analysis was used to identify relations between variables.

3 RESULT

The present study demonstrated a significant elevation in plasma interleukin-6 (IL-6) levels during hemodialysis (HD). Plasma IL-6 increased from 13.63 ± 2.1 pg/dl before HD to 28.72 ± 9.02 pg/dl after HD, whereas the control group showed a substantially lower level of 3.9 ± 3.03 pg/dl. The different superscript letters indicate a statistically significant difference (< 0.001) among the study groups. As shown in Table (1).

Table 1: Serum IL-6 levels in control subjects and hemodialysis patients before and after hemodialysis.

Study groups	IL-6, pg/dl	P- value
Control	3.9±3.03c	< 0.001
Pre-HD	13.63±2.1b	
Post HD	28.72±9.02a	

The present study demonstrated a significant alteration in serum interleukin-10 (IL-10) levels among hemodialysis patients. The mean IL-10 concentration decreased from 1.23±0.74 pg/dl in the control group to 0.75±0.57 pg/dl before hemodialysis (Pre-HD), whereas it increased significantly to 3.12±0.52 pg/dl after hemodialysis (Post-HD). The different superscript letters indicate a statistically significant difference (< 0.001) among the study groups. As shown in Table (2).

Table 2: Serum IL-10 levels in control subjects and hemodialysis patients before and after hemodialysis.

Study groups	IL- 10, pg/dl	P- value
Control	1.23±0.74 a	< 0.001
Pre-HD	0.75±0.57 c	
Post HD	3.12±0.52 a	

The present study demonstrated a significant progressive increase in serum tumor necrosis factor-alpha (TNF-α) concentrations among hemodialysis patients. The mean TNF-α concentration increased from 0.53±0.28 pg/dl in the control group to 1.11±0.41 pg/dl before hemodialysis (Pre-HD) and further increased to 1.59±0.57 pg/dl after hemodialysis (Post-HD). The different superscript letters indicate a statistically significant difference (< 0.001) among the study groups. As shown in Table (3).

Table 3: Serum TNF-α levels in control subjects and hemodialysis patients before and after hemodialysis.

Study groups	TNF-α, pg/dl	P- value
Control	0.53±0.28 c	< 0.001
Pre-HD	1.11±0.41 b	
Post HD	1.59±0.57 a	

4 DISCUSSION

The higher pre-HD IL-6 concentration compared with the control group may reflect the chronic inflammatory state associated with advanced kidney disease. Chronic kidney disease is characterized by persistent low-grade inflammation resulting from uremic toxin accumulation, oxidative stress, impaired immune regulation, comorbid conditions, and reduced renal clearance of inflammatory mediators. IL-6 is one of the major pro-inflammatory cytokines involved in this process and is considered an important marker of systemic inflammation in patients receiving dialysis. Previous evidence has demonstrated that increased circulating IL-6 concentrations are associated with adverse outcomes in dialysis populations(16, 17).

The significant increase in IL-6 after HD observed in the present study is consistent with the findings of(14). The authors suggested that the intradialytic increase in IL-6 was associated with changes in protein metabolism during dialysis. They also demonstrated a significant positive relationship between plasma IL-6 concentrations and the fractional synthesis rates of albumin, fibrinogen, and muscle protein, suggesting that IL-6 may participate in the acute metabolic response to HD.

The acute elevation of IL-6 during HD may be related to activation of inflammatory pathways by the extracorporeal circulation. During dialysis, blood comes into contact with the artificial surface of the dialyzer membrane and the extracorporeal circuit, which can stimulate leukocytes and other components of the innate immune system. This process may promote the release of inflammatory mediators and consequently increase circulating IL-6. Experimental and clinical studies have demonstrated that HD can induce changes in cytokine production and inflammatory gene expression during the dialysis session, supporting the possibility that the dialysis procedure itself contributes to

transient inflammatory activation(13). A study by (18) demonstrated an increased net efflux of IL-6 from skeletal muscle during HD and reported a positive association between IL-6 release and muscle protein catabolism. These observations suggest that skeletal muscle may represent an important source of IL-6 during the dialysis procedure. However, not all studies have demonstrated a significant increase in circulating IL-6 after HD. A meta-analysis including 28 studies and 8,370 dialysis patients reported that higher IL-6 concentrations were associated with increased cardiovascular and all-cause mortality, with a significant association also observed specifically among patients receiving HD(16). Similarly, a study by (17), in a systematic review and meta-analysis, confirmed an adverse association between systemic IL-6 concentrations and survival among people treated with dialysis.

The findings are in agreement with(14), while the contrasting results reported by (13)emphasize that intradialytic cytokine responses may depend on patient characteristics and dialysis-related factors.

The reduction in IL-10 observed before HD may reflect an imbalance in the anti-inflammatory response in patients with advanced chronic kidney disease (CKD). IL-10 is an important anti-inflammatory cytokine that contributes to the regulation of immune responses and limits excessive production of pro-inflammatory mediators. In patients with kidney disease, persistent uremic toxicity, oxidative stress, and chronic immune activation may disturb the balance between pro-inflammatory and anti-inflammatory mechanisms. A study by (19) emphasized the regulatory and protective role of IL-10 in kidney disease and its involvement in controlling inflammatory responses. Therefore, the lower IL-10 concentration observed in the Pre-HD group may indicate inadequate anti-inflammatory regulation during the uremic state.

The present finding is further supported by previous research demonstrating impaired IL-10 production in patients receiving maintenance HD. A study by (20) reported defective IL-10 synthesis by peripheral blood mononuclear cells from HD patients compared with healthy individuals. The authors suggested that impaired IL-10 production may reduce feedback inhibition of pro-inflammatory cytokine synthesis and consequently contribute to the chronic inflammatory state observed in HD patients. This mechanism may partly explain the lower IL-10 concentration observed before dialysis in the current study.

Similarly, A study by (21)demonstrated that metabolic disturbances in HD patients can influence the secretion of anti-inflammatory cytokines. They reported significantly lower IL-10 secretion in HD patients with metabolic acidosis compared with those without acidosis, suggesting that the uremic environment and associated metabolic abnormalities may affect anti-inflammatory cytokine responses. Thus, the reduced IL-10 concentration before HD in the present study may be related to the accumulation of uremic and metabolic factors during the interdialytic period.

Another possible explanation for the low Pre-HD IL-10 concentration is the accumulation of inflammatory stimuli between dialysis sessions. A study by (22) demonstrated that dialysis adequacy was associated with circulating inflammatory and anti-inflammatory mediators in patients with end-stage renal disease. In particular, inadequate dialysis was associated with higher concentrations of several inflammatory mediators and alterations in IL-10 levels. The authors suggested that insufficient removal of uremic toxins may contribute to systemic inflammatory stress and disturbances in anti-inflammatory mechanisms. A recent study by (23) demonstrated abnormalities in IL-10 signaling among dialysis patients and reported alterations in STAT3 phosphorylation and miR-142-3p expression. These findings suggest that the anti-inflammatory IL-10 pathway may be substantially influenced by the physiological conditions associated with dialysis and fluid status.

The significantly higher TNF- α concentration in the Pre-HD group compared with the controls may reflect the persistent pro-inflammatory state associated with advanced chronic kidney disease (CKD). TNF- α is a major pro-inflammatory cytokine involved in the activation of immune cells and the amplification of inflammatory responses. In CKD, chronic inflammation can result from the accumulation of uremic toxins, oxidative stress, metabolic abnormalities, recurrent infections, and impaired immune regulation. TNF- α is among the inflammatory mediators commonly increased in patients with CKD and dialysis-dependent kidney failure(24). Therefore, the elevated Pre-HD TNF- α concentration observed in the present study may reflect the chronic inflammatory environment associated with renal dysfunction.

The increased TNF- α concentration before dialysis may also be related to the reduced renal clearance of inflammatory mediators and the continuous stimulation of immune cells by the uremic environment. Chronic kidney disease is increasingly recognized as a systemic inflammatory condition in which increased production and impaired clearance of pro-inflammatory cytokines contribute to persistent inflammation(25). TNF- α , together with IL-1 β and IL-6, participates in this inflammatory network and may contribute to several complications of CKD, including endothelial dysfunction, cardiovascular disease, malnutrition, and oxidative stress.

Importantly, the present study showed a further increase in TNF- α after the HD session. This increase suggests that the dialysis procedure itself may contribute to acute inflammatory activation. Hemodialysis exposes blood to an extracorporeal circuit and a dialysis membrane, which can activate monocytes, complement pathways, and other

components of the innate immune system. A study by (26) reported that hemodialysis is associated with persistent immune activation and systemic inflammation, with dialysis-related factors such as membrane biocompatibility, complement activation, vascular access, and dialysate quality potentially contributing to inflammatory responses.

The increase in TNF- α following HD is biologically plausible because interaction between blood components and the dialysis membrane can stimulate monocytes and promote cytokine production. An experimental study examining cytokine production during HD demonstrated that monocyte production of TNF- α increased during dialysis and was associated with complement activation. The investigators suggested that membrane–blood interaction and complement activation could contribute to enhanced cytokine production during the dialysis procedure(27). Thus, the increase in Post-HD TNF- α observed in the present study may reflect acute activation of inflammatory pathways during HD rather than simply reflecting the underlying CKD.

The present finding is also consistent with evidence demonstrating that TNF- α gene expression can increase during HD. In a study examining intradialytic cytokine gene expression, an increase in TNF- α mRNA was detected as early as 15 minutes after initiation of dialysis in a proportion of patients. This increase was accompanied by changes in other inflammatory cytokines, supporting the concept that the dialysis procedure can rapidly activate cytokine-related pathways(28). Such activation may provide a mechanistic explanation for the higher TNF- α concentration observed after dialysis in the current study.

Similarly, (29) reported that peripheral blood mononuclear cells from HD patients produced substantially more TNF- α than cells from healthy controls and that TNF- α production was significantly greater after HD than before the dialysis session. The authors suggested that chronic stimulation of mononuclear cells in HD patients, together with additional activation during the dialysis procedure, may contribute to the inflammatory complications associated with HD. This observation is consistent with the progressive increase in TNF- α detected in the current study.

5 CONCLUSION

Hemodialysis was associated with significant alterations in circulating inflammatory cytokines. The marked elevation of IL-6 and TNF- α in hemodialysis patients indicates an enhanced pro-inflammatory state, particularly following the dialysis session. The increase in IL-10 after hemodialysis may reflect activation of compensatory anti-inflammatory mechanisms in response to the inflammatory stimulus. These findings suggest that hemodialysis may significantly influence the balance between pro-inflammatory and anti-inflammatory cytokines in patients with renal failure.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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