Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.234154.2873 Volume 30, Issue 7, Oct. 2024
Manuscript ID ZUMJ-2408-3509 DOI . 10.21608/ZUMJ.2024.310741.3509
REVIEW ARTICLE

Could Hematological parameters reflect inflammatory and nutritional state among dialysis patient?

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Submit date: 09-08-2024
Accept date: 13-08-2024

ABSTRACT

Background: When kidney function is consistently impaired, as in the case of chronic kidney disease (CKD) or chronic renal failure (CRF) or, diagnostic criteria include an unusually high blood creatinine level for longer than 3 months or the glomerular filtration rate (GFR) to be below 60 ml/min / 1.73 m^2. Loss of energy stores and body proteins is one indicator of a worsening nutritional condition in patients with poorly controlled chronic renal disease, especially in its latter stages.Cases who had end-stage kidney disease (ESKD) are already fragile due to muscular atrophy, sarcopenia, and cachexia; furthermore, malnutrition is a powerful predictor of illness and death in this population. We intended to provide an outline of hematological parameters that could reflect inflammatory and nutritional state in dialysis patient.

Conclusion: Cytokines like interleukin-6 (IL-6), interleukin-1 (IL-1), or tumor necrosis factor (TNF), gastric mediators like ghrelin as well as leptin, and adipokines may all play a role in mediating the anorexic process. More research into the identification and prev
ention of malnutrition, in particular, is necessary due to the complicated interaction between inflammatory and nutritional parameters during chronic kidney disease (CKD). We require additional studies on these mediators.

Keywords: Hematological parameters, inflammatory, nutritional state, dialysis

INTRODUCTION

When the kidneys stop filtering nitrogenous waste out of the blood in the usual way, it's called renal failure. ARF is a reversible condition characterized by an abrupt decrease in glomerular filtration rate that can happen from hours to days. One of the following may be used to diagnose The

Kidney Disease: Improving Global Outcomes (KDIGO) criteria is used to determine AKI: Instances when this may be applicable include a recent increase of 0.3 mg/dL in creatinine levels, an increase of 1.5 times your baseline level within the last seven days, or a continuous six-hour period in which your urine volume was less than 0.5 mL/kg. Acute renal failure (ARF) is no longer used; instead, the phrase acute kidney injury (AKI) encompasses the full range of symptoms, from a slight rise in blood creatinine to obvious renal failure [1].

An excessively high blood creatinine level for longer than3 months or a computed glomerular filtration rate (GFR) below 60 ml per minute / 1.73 m2 are the diagnostic criteria for chronic kidney disease (CKD), which is characterized by a continuous decline in kidney function. Dialysis or kidney transplantation may become necessary when kidney function gradually declines. The term "end-stage renal disease" (ESRD) describes a patient's need for renal replacement therapy [2].

One percent of patients admitted to hospitals experience acute kidney injury (AKI), two to five percent while hospitalized, one-third of patients in critical care units, and four to fifteen percent of patients after cardiac surgery [3].

Men are reported to have a higher prevalence of chronic renal failure (CRF) compared to women. There is a gender gap in ESRD as well. Racial disparities exist in the rates of end-stage renal disease (ESRD). Over three to four times as many Black people suffer from end-stage renal disease (ESRD) as white people do [3].

In this review article,we intended to provide an outline of hematological parameters that could reflect inflammatory and nutritional state in dialysis patient.

In the last 30 years, chronic kidney disease has become much more common. Dialysis alone provides treatment for 43.1% of patients undergoing kidney replacement therapy (KRT), and 77.5% of patients with end-stage kidney disease (ESKD) are on KRT. Worldwide, 89% of patients with ESKD receive therapy with hemodialysis (HD) [4].

In terms of dialyzer membrane biocompatibility, dialysis dosage, dialyzer reuse frequency, and dialysis duration, the current technology delivery of HD treatment to patients is deemed ideal according to medical practice guidelines. Malnutrition, which affects 28-54% of the world's population and puts this patient group at a higher risk of death (1.61-4.08), is a major concern [5].

Extensive research have focused on the prevalence of malnutrition in dialysis patients. Malnutrition can be defined in various ways depending on the degree to which inflammation, hypercatabolism, and elevated uremia are present[6].

The associated processes include malabsorption because of swelling in the digestive tract, loss of appetite because of the release of cytokines, and problems with swallowing and food preparation because of exhaustion and trouble breathing. Despite this, it is known that malnutrition increases the risk of heart failure in chronic kidney disease (CKD) patients; however, in heart failure (HD) patients, evaluating malnutrition does not correlate with echocardiographic

findings or the risk of cardiovascular disease [7].

Signs of Poor Nutritional Status and the Progress of Malnutrition During HD Initiation:

Early mortality is most prevalent at the beginning of dialysis and can reach 80% in the first two months of beginning HD. There are a number of controllable risk factors for early mortality, including dietary status, catheter vascular access, and pre-dialysis treatment. Clearly, pre-existing malnutrition begins in progressive CKD stages 3–5, when the patient is most vulnerable due to metabolic abnormalities caused by a decline in glomerular filtration rate, delayed access to nephrology services, and inadequate dietary care prior to dialysis [8].

Patients whose protein prescriptions were more liberal before dialysis are likely to have an improvement in their nutritional condition throughout treatment. Patients who have just begun dialyzing are at increased risk for early mortality due to malnutrition if they exhibit certain characteristics, such as a low albumin level, low cholesterol levels, a low mid-arm muscle circumference (MAMC), a low body mass index (BMI), and reduced food consumption [5].

Iatrogenic Factors of Malnutrition:

Maintenance dialysis patients with ESKD who also suffer from pre-existing malnutrition are at increased risk for death and morbidity because to the catabolic consequences of the treatment. The main worry is that dialysis patients are more likely to die if they have a poor nutritional state [9].

A condition known as "physician-induced malnutrition" or iatrogenic malnutrition can occur as a result of certain medical operations, medications, extended hospital stays, infections contracted while in the hospital, or slowed healing of wounds. Malnutrition is another side effect of dialysis that can't be avoided because it's an inherent feature of the treatment [10].

Dialysis-Induced Nutrient Losses:

The persistent loss of nutrients, particularly protein and amino acids, is exacerbated by dialysis. Hypoalbuminemia can occur as a result of the loss of about 6-12 g of amino acids and 7-8 g of protein after each dialysis session.. This condition is a strong indicator of malnutrition and death. A possible way to restore depleted plasma amino acids is by optimal dietary protein intake (DPI) [11,12].

However, between 32% and 81% of HD patients around the world suffer from DPI insufficiency. The increased proteolysis of proteins throughout the body and in muscles is a result of protein catabolism caused by suboptimal dialysis protein intake and dialysis-induced amino acid losses. [7].

The amount of nutrients lost during dialysis is controlled by the dialyzer membrane's pore size, which regulates the clearance of solutes. In order to improve the removal of intermediate molecules, dialyzer membranes are made larger, leading to an increase in involuntary albumin losses. These losses range from 2 to 14 g, depending on the degree of membrane permeability [6].

Molecular weights below 0.5 kDa were readily removed by low-flux membranes during dialysis in the 1960s; however, intermediate molecules spanning from 0.5 to 60 kDa could not be processed. Membranes with medium and high cutoffs (MCO and HCO) have made dialysis much easier by combining bigger pore diameters with

enhanced HF and HDF processes. In the past, higher concentrations of uremic solutes could be removed using high-flux membranes [4].

Improvements in nutritional health, as shown by increases in body mass index (BMI), dry weight (DW), and hunger, are documented in patients who undergo HF and HDF procedures or use highly permeable membranes, leading to greater intradialytic and hemodynamic stability. The reason for the improvements is that HDF is able to remove more intermediate molecules [13].

Dialysis-Induced Inflammation:

Inflammation can occur in HD patients due to many variables, including dialyzer membrane biocompatibility, infection associated with dialysis access, and the presence of endotoxins in impure dialysate. No evidence has been found to directly link dialysis availability to malnutrition. Low normalized protein catabolic rate (nPCR) levels and high cholesterol are the only indicators of poor nutritional status that are associated with arteriovenous fistula (AVF) failures [12].

Serum albumin levels were lower and malnutrition-inflammation score (MIS) was considerably greater in HD patients with catheter access as compared to those with fistula or transplant. Patients on central venous catheters (CVCs) had a 2% worse survival rate than those with AVF, but regardless of nutritional status, AVF patients had a 52% higher survival rate [11].

Reactive oxygen species (ROS) generation is significantly increased and antioxidant levels are significantly decreased in dialysis patients, making them susceptible to oxidative stress. ROS triggers nuclear factor kappa B (NF-κB) activation, which leads to cytokine production stimulation and inflammation after being translocated to the cell nucleus. It is true that NF-κB gene expression is significantly higher in HD patients compared to the healthy population [4].

Hematological parameters reflect inflammatory/nutrition state in dialysis patient:

Patients with poorly managed chronic renal disease may show signs of decreasing nutritional status, including decreased protein and energy reserves, particularly in the later stages of the disease. Muscle atrophy, sarcopenia, and cachexia make this group already fragile, and among the strongest indicators of mortality and morbidity in patients with end-stage renal disease is the existence of malnutrition [14].

Loss of muscle, visceral fat, and body fat is the hallmark of protein-energy wasting (PEW), a disorder of catabolism brought on by uraemia. There is substantial evidence linking PEW to mortality in CKD and ESKD from retrospective epidemiological research. At present time, there are no established indicators for the bidirectional evaluation of nutritional-inflammatory factors in this group, and it is also unclear which metrics provide consistently better results [15].

Malnutrition and the Inflammatory Processes in Chronic Kidney Disease:

The chronic inflammatory state in chronic kidney disease (CKD) is caused by many things. The inflammatory cascade begins with the activation of monocytes as well as macrophages, which leads to an increase in the production of proinflammatory cytokines and oxidative stress. A poor prognosis and severe cardiovascular outcomes (such as atheromatosis and atherosclerosis) are related

with chronic inflammation, which in turn increases mortality and morbidity in patients with chronic kidney disease [16].

The malnutrition-inflammation-cachexia syndrome (MICS) is associated with malnutrition–PEW. Body mass index, muscle mass loss, and inadequate dietary intake are the diagnostic criteria for PEW; specialized grading systems have also been created to help evaluate nutritional status. Subjective Global Assessment and Kalantar score, commonly called Malnutrition Inflammation Score (MIS), are the two most noteworthy grading methods that are now accessible [17].

A number of factors contribute to the inflammatory and malnutritive processes in CKD and ESKD. Anorexia is brought on by proinflammatory cytokines, which also cause chronic exhaustion and the breakdown of muscle proteins, leading to atrophy. Gastric mediators (e.g., leptin and ghrelin), adipokines, or cytokines (e.g.or interleukin-1 (IL-1), interleukin-6 (IL-6), ortumor necrosis factor (TNF), may orchestrate this anorexic process. [14].

Additional investigation into these mediators, though, is required. Further symptoms and health conditions, like IL-6-induced depression, can cause a decrease in dietary intake, an increase in resting energy expenditure, a decrease in anabolic hormones (like testosterone, insulin-like growth factor (IGF)-1, and growth hormone), or even a resistance to hormonal factors [18].

Lastly, adipokines' involvement in chronic kidney disease (CKD) has just lately been recognized. One such adipokine is leptin, which is known to promote oxidative stress, inflammation, and lipid abnormalities in addition to having a possible inflammatory function. Elevated plasma C-reactive protein levels and metabolic syndrome scores are associated with elevated leptin levels in chronic kidney disease (CKD) stages G2–5, and they have also been seen in hypertensive individuals experiencing cachexia [19].

Malnourished chronic kidney disease patients have been found to have additional circulating biomarkers. As chronic kidney disease (CKD) advances, several nutrients, including vitamin D, magnesium, and zinc, are less active, which may contribute to their anti-inflammatory benefits. Other features of chronic renal illness, such as mineral and bone status, phosphate, ionized calcium, and alkaline phosphatase, have also been associated with nutritional variables. Omega-3 polyunsaturated fatty acids are known to have a potent anti-inflammatory effect; however, individuals undergoing renal replacement treatment have lower levels of these fatty acids compared to the general population [16].

All of these things contribute to an inflammatory condition, which affects the patient's nutritional status, which in turn affects their quality of life and prognosis in the long run. Patients with end-stage renal disease (ESRD) require better dietary habits. Thus, to effectively avoid cachexia, PEW, and the loss of lean muscle mass, intraperitoneal parenteral nutrition, intradialytic feeding, and oral meal supplements may be useful [20].

Biomarkers reflect inflammatory/nutrition state in dialysis patient:

Nutritional indicators tend to decline with time in individuals with ESKD who undergo HD, according to multiple studies. Malnutrition and inflammation contribute to

the increased mortality rate among HD patients [19].

Creatinine, C-reactive protein, and hematocrit levels were substantially linked with nutritional assessment done using MIS. Also, among hemodialysis patients, MIS was significantly associated with death. The MIS score is the most reliable independent predictor of mortality in HD patients over the course of four years, according to recent research. Surprisingly, MIS mortality forecasting is on par with or higher than numerous other intricate assessments of inflammatory and nutritional condition [14].

Hypoalbuminemia has been the nutritional indicator most often studied in dialysis patients due to its strong association with increased mortality from all causes and cardiovascular disease. Noteworthy, maximizing the S-albumin level still leads to a decrease in mortality in the first year of HD, even though serum albumin's (S-) ability to distinguish malnutrition is restricted **(Figure 1)** [17].

Inflammation is thought to play a role in the well-known correlation between hypoalbuminemia and mortality in dialysis patients. Albumin concentration, synthesis, and half-life are all negatively correlated with inflammation in this population, and S-albumin levels are already low. Not only in HD patients, but also in chronic kidney disease (CKD) generally, prealbumin (or transthyretin) is a marker for nutritional evaluation [21].

According to the new 2020 Kidney Disease Outcomes Quality Initiative (KDOQI) recommendations for nutrition in chronic kidney disease (CKD), additional biomarkers such as albumin and serum prealbumin (if available) can be used to assess nutritional status. They are not sufficient on their own to assess nutritional status due to the impact of non-nutritional variables [17].

Because of its association with nutrition, serum creatinine—an inexpensive and commonly accessible nutrient—is sometimes disregarded as a surrogate measure of skeletal muscle mass and food protein consumption. There is strong evidence that lower serum creatinine levels are related with an increased mortality risk, and systemic inflammation can also influence creatinine levels. Creatinine levels in HD patients are affected by factors such as endogenous degradation, dialysis dosage, and residual renal function [14].

In terms of the possible advantages of reducing inflammatory molecules, a meta-analysis demonstrated that persons undergoing various stages of chronic kidney disease (CKD) who were subjected to a diet devoid of animal products experienced a decrease in inflammatory markers. Patients whose protein intake came from animals had greater C-reactive protein levels compared to those whose protein intake came from other sources. This marker has a lot of room for error because to its high variability; for example, it could give an inaccurate reading of protein consumption when the body is in a catabolic or anabolic state, respectively [19].

In contrast, patients whose diets were heavy in animal proteins had much higher levels of interleukin-6 than those whose diets were heavy in vegetable proteins. This finding is likely associated with the severity of renal insufficiency and the inflammation that characterizes chronic kidney disease (CKD), as well as the specific protein types consumed and broken down by the body [20].

Inflammatory muscle protein losses and potential changes to IGF-1 signaling due to elevated glucocorticoid levels have been linked to elevated IL-6 levels in the blood, according to a number of studies. An further mechanism by which IL-6 contributes to uremic skeletal muscle protein loss is by increasing caspase-3 activation [18].

The TNF superfamily member TWEAK stands out because it binds to its receptor Fn14 and triggers cell death in sensitive tissues. An association between TWEAK and the signaling pathways that control NF-κB, myogenesis, and apoptotic cascades has been established [16].

The last hormone is leptin, which is produced by adipocytes, skeletal muscle, and bone cells. It has multiple roles, including controlling hunger and fullness. Additionally, leptin is associated with inflammation in obese individuals and has been found to be a proinflammatory cytokine [21].

The structural and functional features of leptin and its receptor are similar to those of other cytokines. Levels of leptin are inversely related to spontaneous food energy intake. Many recent investigations have shown that most CKD patients have abnormally high levels of leptin, which has led to the hypothesis that this hormone is one of the causes of anorexia and wasting in this patient group [15].

When we consider all of the evidence, we conclude that chronic inflammation is directly associated with the onset of malnutrition in ESKD patients. Nevertheless, it should be noted that evaluating nutritional status in HD patients based on a single biomarker is likely inadequate. Instead, a combination of markers, including normalized protein catabolic rate, S-albumin and/or prealbumin, C-reactive protein, creatinine, and possibly others, could provide a more accurate prediction [17].

Hematological parameters reflect inflammatory/nutrition state in dialysis patient:

A high rate of morbidity is associated with chronic renal illness, which commonly causes anemia as a consequence. The symptoms of renal failure anemia, which manifest in the early stages of kidney disease, progressively intensify as the kidneys continue to decline. Reduced erythropoiesis due to inadequate erythropoietin (EPO) production is the primary problem, although there may be other factors at play as well [21].

Consequences of chronic renal disease often include normocytic normochromic anemia. Hypochromic and microcytic blood could indicate an iron deficiency or aluminum poisoning. Macrocytic anemia is caused by a lack of vitamin B12 and folate, which are the most common deficiencies. Anemia of any kind can show up in people with chronic renal disease [19].

Patients diagnosed with chronic kidney disease had normal total white blood cell counts, whereas those with acute renal failure had increased counts. Neutrophil leucocytosis and lymphopenia are common in both conditions. They reduce lymphopoiesis and increase granulopoiesis, however it is unclear which chemicals stayed in the body after renal failure (Figure 2)[14].

This illness is characterized by an increased risk of bleeding due to platelet dysfunction caused by abnormal platelet activation and adhesion. Electrolyte imbalances can be corrected and urea and other toxic substances

removed from the bloodstream through dialysis [15].

Of the two forms of dialysis, hemodialyzation is the more common one. To do this, the dialysate is introduced to the bloodstream through an extracorporeal circuit that pumps the blood through semi-permeable artificial membranes [18].

It is possible for blood components to be damaged by either type of dialysis. Age, sex, ethnicity, muscle activity, posture, dialysis duration and type, and other physiological and non-physiological factors may all influence the severity of these effects [20].

There is evidence that hemostatic indicators such as mean platelet volume (MPV) and red cell distribution width (RDW) can be used to assess chronic inflammation, cardiovascular disease (CVD), anemia, and poor renal outcome in patients with chronic kidney disease (CKD). Thus, it may be prudent to utilize them as markers for follow-up in a subset of our CKD patients [21].

Variation in erythrocyte size is quantified by RDW. High RDW values on blood count measurements indicate increased size variability of the erythrocytes, which is known as anisocytosis. Cardiovascular disease death, and unfavorable renal outcomes were all linked to high RDW in chronic kidney disease patients [15].

One way to gauge platelet size in the blood is with the multi-platelet volume (MPV) test. In order to trigger adhesion and aggregation, large platelets often have more reactive granules and are younger. In order to measure platelet reactivity, atherosclerosis, and inflammatory state, elevated MPV was introduced. However, it has been shown that patients with advanced chronic kidney disease (CKD) and those with significant inflammation often have low MPV levels [14].

Alterations in hematological parameters are more noticeable as chronic kidney disease (CKD) advances. Hematological markers such as hemoglobin concentration, RBC count, leukocyte count, and platelet count might become abnormal in chronic kidney disease (CKD). Elevated blood creatinine and urea levels are additional prognostic indicators [19].

The elimination of metabolic waste and extra toxic fluids is facilitated by hemodialysis, which is also a less expensive alternative to kidney transplantation. A large body of research has examined how hemodialysis affects several biochemical and hemodynamic markers [16].

The Neutrophil-Lymphocyte Ratio (NLR) is an important new biomarker that has come out recently. In the past, hemodialysis patients' risk of cardiovascular disease and death from any cause could be predicted using a straightforward inflammatory biomarker known as NLR. Also, many studies have linked it to inflammatory factors and nutritional status in HD patients. The systemic immune-inflammation index (SII) is a measure of the host immune inflammatory reactions that is calculated by combining the lymphocyte, neutrophil, and platelet counts [17].

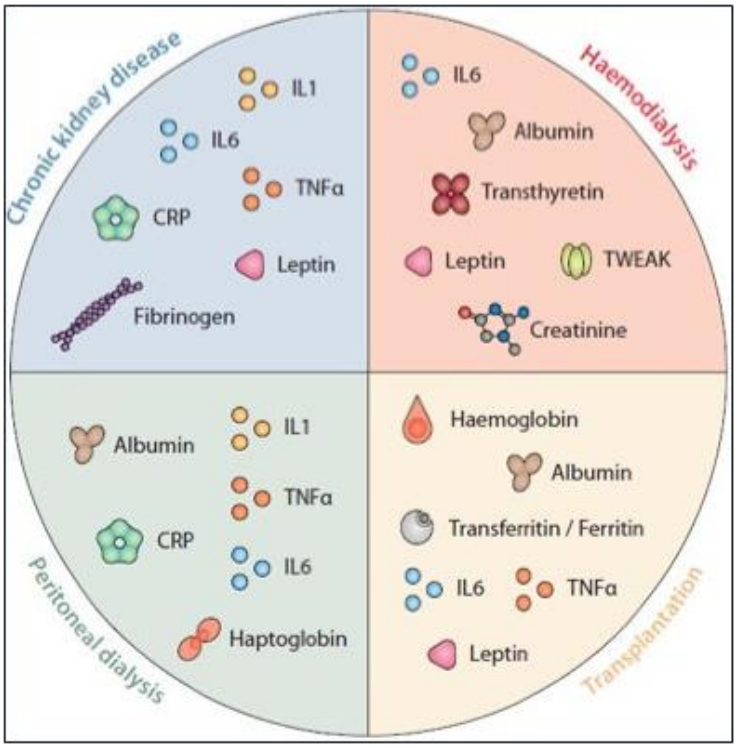

Figure (1): Biomarkers of nutritional status and inflammation in Renal Failure patients [14].

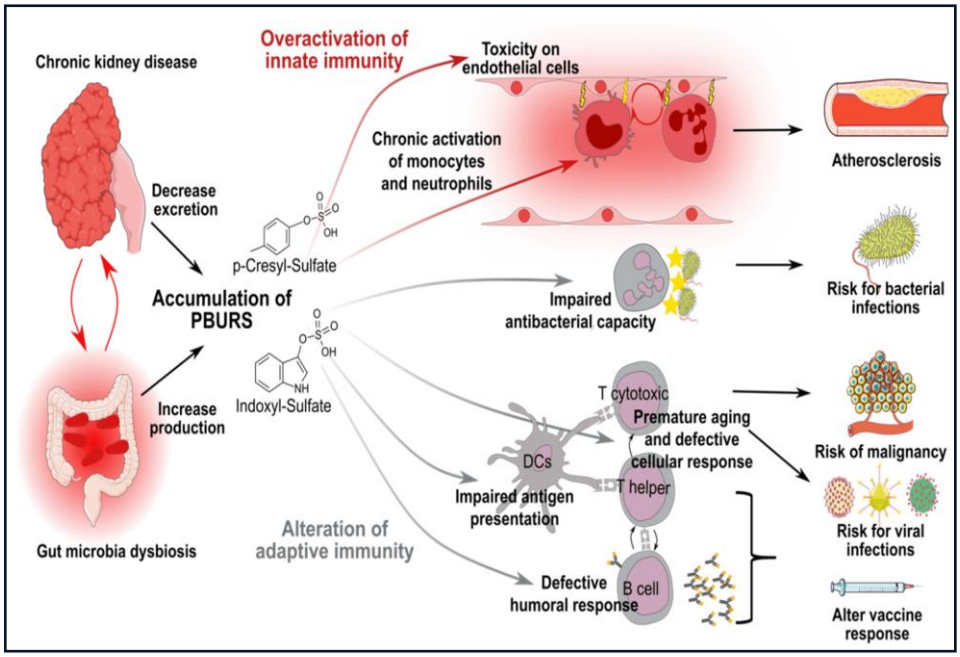

Figure (2): Neutrophils in Renal Failure [14].

DISCUSSION

Cytokines like interleukin-6 (IL-6), interleukin-1 (IL-1), or tumor necrosis factor (TNF), gastric mediators like ghrelin as well as leptin, and adipokines may all play a role in mediating the anorexic process. More research into the identification and prevention of malnutrition, in particular, is necessary due to the complicated interaction between inflammatory and nutritional parameters

during chronic kidney disease (CKD). We require additional studies on these mediators.

No potential conflict of interest was reported by the authors.

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Figure legends

Figure (1): Biomarkers of nutritional status and inflammation in Renal Failure patients

Figure (2): Neutrophils Renal Failure.

Citation:

Abdelmoneem, S., Arafa, A., shoieb, M., Easa, M., Metwally, E. Could Hematological parameters reflect inflammatory and nutritional state among dialysis patient?. Zagazig University Medical Journal, 2024; (3644-3654): -. doi: 10.21608/zumj.2024.310741.3509