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# Estimation Of Certain Heavy Metals, Oxidative Stress Markers, And Antioxidants In The Blood Serum Of Patients With Kidney Failure

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**Abstract:** This study focused on evaluating the levels of certain heavy metals, such as cadmium (Cd) and lead (Pb), as well as indicators of oxidative stress and antioxidants, namely interleukin-6 (IL-6) and superoxide dismutase (SOD). The study included 90 blood samples, divided into a control group of 60 patients with renal failure and a control group of 30 apparently healthy individuals. Blood samples were collected between January 2024 and March 2024. Serum cadmium (Cd) and lead (Pb) concentrations were estimated, and interleukin-6 (IL-6) and superoxide dismutase (SOD) levels were measured using standard laboratory methods.

The results showed a significant increase in the levels of cadmium ( $2.86 \pm 0.73$ ), lead ( $6.858 \pm 1.191$ ), and interleukin-6 ( $18.72 \pm 5.64$ ) in the patient group compared to the healthy control group ( $p \leq 0.001$ ). Conversely, a significant decrease in SOD enzyme concentration ( $1.91 \pm 0.54$ ) was observed in the renal failure patient group compared to the control group ( $p \leq 0.001$ ). These results indicate a disruption in the antioxidant capacity to combat inflammation and oxidative stress, as well as the role of heavy metals in the development and exacerbation of inflammation in renal failure patients. The study concludes that the role of heavy metals in causing inflammatory and oxidative imbalances may play a significant part in the development and progression of the pathological mechanisms associated with renal failure, making these variables important biomarkers for assessing, diagnosing, and monitoring the disease's progression.

**Keywords:** Kidney failure, heavy metals, interleukin-6, superoxide dismutase.

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## 1. Introduction

It is the pathological condition in which the kidneys are unable to eliminate and excrete metabolic waste products into the blood, and to regulate fluids, electrolytes, and acid-base balance in extracellular fluids [1]. Renal failure was formerly known as uremic poisoning, based on the most prominent pathological symptoms accompanying renal failure, which are elevated urea and creatinine levels in the blood. Starting in 1847, the term uremia was replaced to denote blood contamination with urine, until contemporary medical literature settled on the term renal failure [2]. Heavy metals are insoluble pollutants that enter the human body through various routes such as the mouth, skin, and inhalation. They negatively affect the body's organs as their concentration increases and they accumulate, including the kidneys, where they precipitate and are difficult to eliminate because most of them are not water-soluble. The accumulation of heavy metals in the body, particularly in the kidneys (which act as the body's filters), leads to decreased kidney function due to their high density and insolubility [3]. This can result in serious kidney complications such as kidney failure[4].

This study evaluated the levels of these heavy metals such as cadmium and lead, in a selected group and investigated the effects of high concentrations of these elements in the human body, compared to normal levels, on kidney function and efficiency.

Oxidative stress appears to be closely linked to kidney function and increases with the progression of chronic kidney disease. Both oxidative stress and inflammation play a significant role in disease progression and exacerbation [5, 6]. In patients on long-term dialysis, oxidative stress and inflammation are linked to higher mortality [7]. However, a scarcity of population research makes it difficult to determine the exact incidence of oxidative stress and inflammation among individuals with chronic renal disease [8]. Increased activity and neutrophil activity in patients may cause an increase in the production of reactive oxygen species compared to neutrophils in healthy individuals [9, 10]. Interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF- $\alpha$ ), interleukin-6 (IL-6), and interleukin-8 (IL-8), which may be produced when blood comes into contact with the washing membrane of patients, are examples of oxidative stress that may be higher in patients than in healthy individuals [11, 12].

Increased blood levels of free radicals and the antioxidant enzyme system's activation might worsen inflammation and hasten the development of tissue damage. Catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (GSH-Px) are some of these enzymes. These enzymes play a crucial role in protecting the body from excessive oxidative stress [13]. Catalase oxidizes compounds such methanol, ethanol, phenols, and nitrites and transforms hydrogen peroxide into oxygen and water [14].

#### **Objectives of the Study:**

- 1- To determine the serum levels of certain heavy metals, such as cadmium (Cd) and lead (Pb), in patients with renal failure and compare them with those of healthy individuals.
- 2- To evaluate the serum levels of certain biomarkers associated with inflammation and oxidative stress, such as interleukin-6 (IL-6) and superoxide dismutase (SOD), in patients with renal failure compared with the control group.
- 3- To reinforce the significant and influential role of heavy metals and inflammatory and oxidative disorders in renal failure by studying the changes in their levels and their relationship to the progression and worsening of the disease.

## **2. Materials and Methods**

### **2-1 Study Design and Participants:**

This study was conducted from January 2024 to March 2024 at Tikrit Teaching Hospital in Salah al-Din Governorate. The study included (90) participants who were divided into two groups. The first group comprised sixty patients who had been diagnosed with chronic kidney failure by specialist doctors using approved clinical criteria and laboratory tests; the second group consisted of thirty people who appeared to be in good health and were matched as closely as possible in terms of age and sex with the patients.

### **2-2 Sample Collection:**

Anticoagulant-free tubes were used to draw five milliliters of venous blood from each participant. After allowing the samples to coagulate for a reasonable amount of time, the serum was separated by centrifugation for ten minutes at 3000 rpm. After that, the serum samples were put into sterile tubes and kept at -20°C until they were examined in a lab.

### **2-3 Biochemical Measurements:**

Atomic absorption spectrophotometry (AAS) was used to measure the serum concentrations of the heavy metals lead (Pb) and cadmium (Cd) in accordance with conventional methods and the manufacturer's instructions. Superoxide dismutase (SOD) and interleukin-6 (IL-6) levels were also determined using enzyme-linked immunosorbent assay (ELISA) in accordance with the manufacturer's suggested procedures.

### 2-4 Statistical Analysis:

The Statistical Package for the Social Sciences (SPSS) version 26 was used to statistically analyze the data. The mean  $\pm$  standard deviation (Mean  $\pm$  SD) was used to express the results. The two groups were compared using the Independent Samples t-test, with a statistical significance threshold of  $P < 0.05$ .

### 3. Results

The patients with renal failure had levels of interleukin-6 (IL-6) and superoxide dismutase (SOD) of  $18.72 \pm 5.64$  and  $1.91 \pm 0.54$ , respectively, while the healthy control group had values of  $6.83 \pm 2.15$  and  $3.87 \pm 0.82$ , respectively. Table 1 and Figures 1 and 2 demonstrate that, at a probability threshold of ( $p < 0.001$ ), we found that the sick group had higher levels of IL-6 and lower levels of SOD when compared to the healthy control group. These findings align with earlier research [15, 16]. The results also showed a significant increase in the levels of heavy metals, specifically cadmium (Cd) and lead (Pb), in the patient group ( $2.86 \pm 0.73$ ) and ( $6.858 \pm 1.191$ ), respectively, compared to the healthy group ( $0.91 \pm 0.28$ ) and ( $1.450 \pm 0.426$ ), respectively, at a probability level of ( $p < 0.001$ ), as shown in Table 1 and Figures 3 and 4. These results are consistent with previous studies [17, 18].

**Table 1. Comparison of serum levels of IL-6, SOD, cadmium (CD) and lead (Pb) between kidney failure patients and healthy individuals.**

Parameters	Mean $\pm$ SD		P-value
	Control	Patients	
IL-6	$6.83 \pm 2.15$	$18.72 \pm 5.64$	$<0.001^{**}$
SOD	$3.87 \pm 0.82$	$1.91 \pm 0.54$	$<0.001^{**}$
CD	$0.91 \pm 0.28$	$2.86 \pm 0.73$	$<0.001^{**}$
Pb	$1.450 \pm 0.426$	$6.858 \pm 1.191$	$<0.001^{**}$

\*  $P \leq 0.05$ , \*\*  $P \leq 0.01$

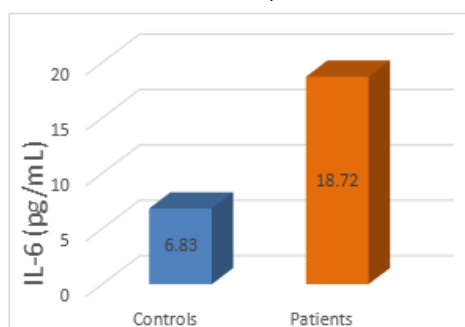


Figure 1. Shows the level of IL-6 in the patient Group compared to the control group.

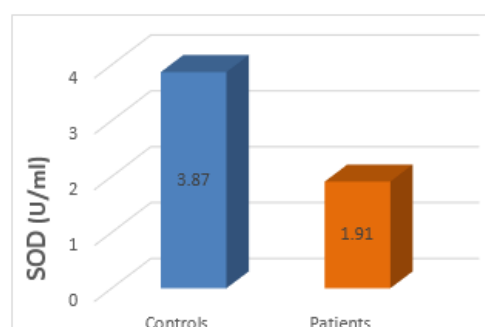


Figure 2. Shows the level of SOD in the patient Group compared to the control group.

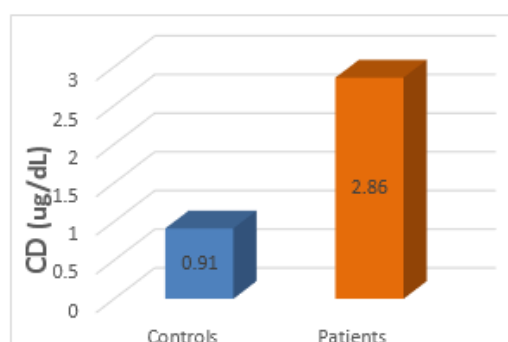


Figure 3. Shows the level of CD in the patient Group compared to the control group.

4.

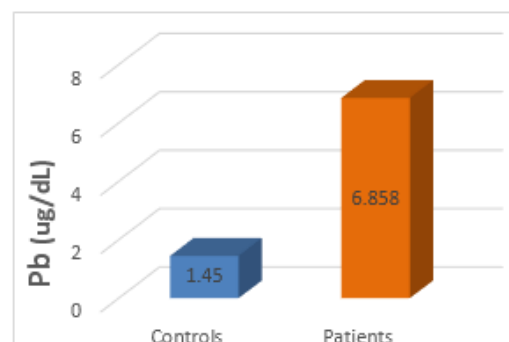


Figure 4. Shows the level of Pb in the patient Group compared to the control group.

4. 4.

#### 4. Discussion

Previous studies have shown that kidney failure patients are exposed to several inflammatory factors, which may be linked to kidney disease or immunodeficiency. Elevated levels of interleukin-6 (IL-6), secreted by T lymphocytes, may contribute to the development of oxidative stress and inflammation [19, 20]. The current study revealed a significant increase in IL-6 levels in kidney failure patients compared to the control group, and these findings are consistent with previous research [21]. Studies indicate that the main source of elevated IL-6 levels in the blood serum of patients was due to uremia factors, and that the dialysis procedure leads to increased IL-6 production [22]. Some studies have found an increase in the level of interleukin-6 in kidney failure patients compared to healthy individuals [23]. An automatic increase in the production of interleukin-6 and tumor necrosis factor-alpha (TNF- $\alpha$ ) by peripheral white blood cells has also been observed in dialysis patients, and an increase in interleukin-6 secretion during a dialysis session [24]. Previous studies have shown a clear decrease in SOD levels in the kidney failure patient group ( $p < 0.05$ ) compared to the control group [25]. Oxidative stress impairs endothelial function, leading to decreased SOD levels. Therefore, several studies have shown reduced levels of this enzyme and other antioxidant enzymes in preclinical acute kidney injury. The genetic predisposition to SOD increases the risk factors for acute kidney injury in cases of ischemia and hypoxia [26]. One factor that leads to increased superoxide dismutase activity is the overgrowth of oxygen-secreting macrophages. Reduced antioxidant activity in the plasma of patients with chronic kidney disease may contribute to increased oxidative damage and the development of kidney problems [27].

Elevated levels of both lead (Pb) and cadmium (Cd) may be caused by environmental pollutants such as agricultural sources and fertilizer use, which contaminate soil and crops, leading to food contamination (vegetables, grains, and seafood). Water and air pollution also contribute. Air pollution from burning fossil fuels is a likely cause [28]. Older buildings may be more susceptible to lead poisoning, as deteriorating paints can produce lead dust. In buildings near roads, water contamination from lead pipes is another significant source. Lead poisoning can also result from the use of cosmetics and traditional medicines [29]. Trace and heavy metals are of great importance and have vital functions in the human body despite their low concentrations. Due to pollutants from food and other sources, the concentrations of these elements can be disrupted by endotoxemia, kidney disease, dietary patterns, and medical treatments. Acute or subacute exposure to these elements at low concentrations poses a risk of kidney toxicity. Due to numerous factors, such as food, water, and gas contaminants, the health risks may appear suddenly (acute) or develop into long-term damage [30]. Dietary and environmental factors, in addition to tobacco smoking, play a major role in the imbalance of heavy metals in patients with kidney failure [31], while other factors and metabolic disorders resulting from drug contamination with these elements also contribute. Scientific reports indicate that the lipid peroxidation observed in patients may result from the disruption in the levels and concentrations of heavy metals, with a correlation between their deficiency and impaired antioxidant efficiency [32]. The antioxidant system in patients with chronic kidney disease is negatively affected due to the dysfunction of enzyme pathways responsible for detoxifying free radicals, paving the way for the unchecked release of reactive oxygen species targeting cell membranes, resulting in the formation of lipid peroxidation derivatives [33]. In a related context, laboratory and animal studies show that oxidative damage resulting from lead poisoning contributes to erythropoiesis [34], a conclusion supported by human research demonstrating an imbalance between oxidizing and antioxidant factors in workers exposed to cadmium and/or lead [35, 36].

#### 5. Conclusion

The renal failure patient group had significantly higher levels of interleukin-6 (IL-6), lead (Pb), and cadmium (Cd) than the healthy control group, according to our study's findings. On the other hand, the sick group's superoxide dismutase (SOD) enzyme level was significantly lower than that of the healthy control group, according to the data. These results imply that heavy metal buildup and elevated levels are detrimental to renal failing patients. Additionally, the findings showed that oxidative stress, inflammation, and a reduction in antioxidant capacity occur in these individuals. The elevated levels of inflammation and oxidative stress, coupled with the reduced

antioxidant capacity of SOD, may be a result of the accumulation of heavy metals in chronic kidney failure patients, potentially contributing to disease progression and complications. Therefore, these variables can be considered important biomarkers that aid in understanding the pathophysiological mechanisms associated with chronic kidney failure and in monitoring the health status of patients.

#### 6. Recommendations:

1. Carry out a comprehensive and in-depth inquiry to ascertain the connection between oxidative stress indicators, inflammation, and heavy metals, as well as to comprehend their involvement in the development of renal failure and other disorders.
2. Explore and enhance the role of heavy metals and oxidative stress markers in monitoring patients' health, both diagnostically and in disease progression.
3. Conduct future studies to explore the use of heavy metals and oxidative stress markers as diagnostic tools for other diseases, such as diabetes and rheumatoid arthritis.

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