

# Immunological Study of Some Pathogenic Bacteria Isolated from Patients with Chronic Kidney Failure

Areej Hamad Hassan<sup>1</sup>, Muthana Badeaa Farhan<sup>2</sup>

<sup>1</sup>Postgraduate, College of Women Education, University of Anbar, Ramadi, IRAQ,

<sup>2</sup>Assistant Professor, College of Women Education, University of Anbar, Ramadi, IRAQ

## Abstract

Renal failure is one of kidney diseases in which the kidney work in less than 15% of normal, the chronic kidney failure may develop slowly and its irreversible. Many bacterial infection can associate with kidney failure due to low immunity and insufficient kidney infiltration. This study was aimed to investigate the immunological parameters in kidney failure patients. Sixty patients with chronic renal failure reviewed to one hemodialysis center was evaluated, 50 samples with bacterial infection and 10 samples without bacteria in diuresis. The results show significant decrease in IgA, IgM, IgG concentrations for patients with bacterial infection 15.24 pg/ml 60.52, 29.9, in compared with uninfected patients which show 59.1, 33.8, 18.3 pg/ml, the IgE was increased due to the presence of bacteria by 206.6pg/ml. comparing with 186.04 pg/mL in uninfected patients,also IL-1 $\beta$  increased significantly 72.74 pg/ml compared with controls 5.38 pg/ml and a significant decrease in complement C3 in the presence of bacteria at a concentration of 112.45 pg/ml compared with concentration of 155.4 pg/ml in healthy people. This study conclude that the immunological parameters different significantly in patients with chronic kidney failure infected with different bacterial infection in compared with uninfected patients.

**Keywords:** *Chronic kidney failure, immunoglobulins,IL-1 $\beta$ ,complement system*

## Introduction

That chronic kidney failure (Renal failure) is a deterioration in the excretory secretory function of the kidney as a result of accumulation of nitrogenous waste and harmful substances produced by various metabolic reactions<sup>(1,2)</sup> as they are unable to remove the final metabolic products from the blood, which leads to a disturbance in the value balance pH and balance of bodily fluids<sup>(3)</sup>. Chronic kidney failure is a common disease in the modern era, especially in cases of simultaneous infection with multiple bacterial infections, where E. coli bacteria are the most common and cause urinary tract infection in patients with kidney failure followed by bacteria Others<sup>(4)</sup>. People with kidney failure have an

acute systemic immunity deficiency that is responsible for around 20-30% of deaths of patients with kidney failure and those who continue with hemodialysis<sup>(5)</sup> that may cause failure Renal to an increase in inflammatory responses, which includes a number of mechanisms that include the reaction of the immune system, which is an increase in the concentration of Proinflammatory Interleukins.<sup>(6)</sup> Interleukins act as inflammatory indicators that stimulate or inhibit immune response cells, as the production of Interleukins is considered as a primary response of the mucous layer when Bacterial invasion, where the concentration of interleukin increases when infection occurs<sup>(7)</sup>. Most of these bacterial factors may weaken the systemic immunity of patients with chronic renal failure.

The study was aimed to Identification of some pathogenic bacteria accompanying patients with chronic renal failure from clinical samples. Study of some immunological indicators,include measurement of immunoglobulins IgG IgM, IgA, IgE and complement C3 and IL- 1 $\beta$ .

---

### Corresponding Author:

**Areej Hamad Hassan**

Postgraduate, College of Women Education, University of Anbar, Ramadi, IRAQ

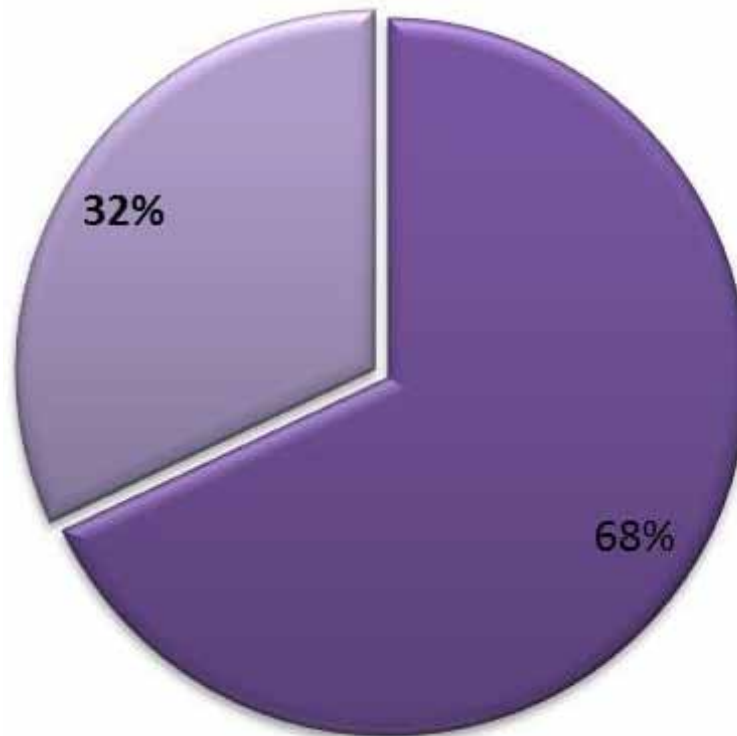
**Materials and method of work:** 110 blood and diarrhea samples were collected at the same time from chronic kidney failure patients attending the Kidney Dialysis Unit at Ramadi General Teaching Hospital in Anbar Governorate for the period from October 2018 to August 2019 and 60 samples were divided into 50 blood and urine samples containing bacteria and 10 samples Blood and urine do not contain bacteria. The ages of patients ranged between (15-85) years and of both sexes. Also, 24 control samples were collected.

After collecting blood samples, the serum was separated in a tube by centrifugation at a speed of 3.000 rpm for 10 minutes and then the tests were done. The test was based on the Sandwich-ELISA method to conduct the examination where a specialized antibody dish was

installed on the surface of the microprocessing plate, which is particularly associated with standard solutions or With serum when added to the microplate pits then washed with distilled water and read on the ELISA device with a wavelength of 450 nm .

## Results

The isolation results showed 50 urine samples with positive growth of bacterial culture out of 110 samples collected from patients with chronic renal failure. The results showed that 50 samples gave bacterial growth (for both negative and positive bacteria) as in figure (1) the number of negative bacteria was (68%)The number of positive bacteria (32%).



**Figure (1): The percentage of bacterial culture isolated from patients with chronic renal failure**

The level of concentrations of the final rates in the measurement of immunoglobulins for patients with chronic kidney failure in the presence of bacteria when compared with patients with chronic kidney failure without bacteria and healthy people, the level of IgG in the patient sample decreased kidney failure in the presence of bacteria and the absence of bacteria compared to a sample Control. The decrease in the

presence of bacteria was higher than 15.24 pg/ml, when bacteria were not found pg/ml 18.3 Comparing them with healthy subjects pg/ml 39.0. A similar decrease occurred in the serum IgM level in the patient sample with the presence of bacteria pg/ml 60.52 and lack of it pg/ml. 59.1 compared to the control sample 81.83 pg/ml and provides for a decrease in the level of Both IgG and IgM in patients with renal failure,

**Table 1: Levels of concentrations of immunoassay among patients with chronic renal failure without bacteria and with bacteria and healthy subjects**

Studied totals Immunohistochemical replications	Healthy people	Bacterial patients	Patients without bacteria	L.S.D.
IgG	39.0	15.24	18.3	0.012**
IgM	81.83	60.52	59.1	0.064**
IgA	43.62	29.9	33.8	0.079**
IgE	123.37	186.04	206.6	0.278**
IL-1 $\beta$	36.64	72.74	57.43	4.79 **
Complement C3	147.38	112.45	155.4	6.48 **

As for the concentration of IgA shown in Table(1), it was found that its serum concentration levels in patients with kidney failure in the presence of bacteria pg/ml 29.9 and its absence 33.8 pg/ml decreased compared to pg/ml 43.62 in the control sample. The results showed a high concentration of immunoglobulin IgE for chronic renal failure patients as shown in table (1) with bacteria 186.04 pg/ml and lack of it 206.6pg/ml compared to healthy subjects 123.37 pg/ml where high levels of IgE in the blood were considered an indication of the disease.

Whereas, bacterial infections stimulate macrophages cells to release inflammatory media such as  $\beta$  IL-1 (18), as the results shown in Table (1) show the complement concentrations of Complement C3, an increase in patients with chronic renal failure without bacteria 155.4pg/ml and a decrease in the presence of bacteria. 112.45pg/ml compared to healthy subjects 147.38pg/ml,

### Discussion

The lack of growth in negative samples of bacterial culture may be attributed to the fact that the cause may be viral or from anaerobic bacteria that cannot be isolated by the usual culture method used in this study Or due to patients taking doses of antibiotics, or perhaps inappropriate use of the antibiotics that cause the disappearance of bacteria that cause urinary tract infection. The cause of the spread of negative bacteria more than positive bacteria is due to the presence of lipid polysaccharide (LPS), the main component of the outer membrane of bacteria that is negative for Gram stain. LPS helps protect bacterial cells from their surroundings<sup>(8)</sup>.

The reason for the decrease in the level of immunoglobulins for patients with chronic kidney failure

may be due to several reasons, including accumulated uremic toxins, which inhibit the manufacture of immunoglobulins in patients with chronic kidney failure and a decrease in the numbers of B cells B-cell, which is considered the basis for the manufacture of cloplines as a response against different antigens<sup>(9,10)</sup> and address some types of antibiotics and some types of steroids contribute to the process of low concentrations of Clobiolins in the serum of patients with chronic renal failure by inhibiting their recycling<sup>(11)</sup> and it can be known if the previous infection in the body through IgG antibodies and a recent infection through antibody type IgM<sup>(12)</sup>. IgA due to decreased immunoglobulin synthesis in these patients which resulted from the patient's malnutrition status As a result of his deterioration A decrease in IgA causes frequent infections in the urinary system, as this non-inflammatory antibody specializes in protecting the mucous membrane. Whereas, the prevalence of IgA deficiency in a uremic patient may be due to the neutralization of IgA antibodies and previous destruction leading to a decrease in the number of cells B and IgA Secreting B in a uremic patient as the decrease of this antagonist by the presence of bacteria was one of the main causes of death and disease in kidney patients, usually This is explained by the negative effects of the uremia on the immune defense, for both humoral and cellular immunity and the specific antibody response was low<sup>(13)</sup>. If an increase in the concentration of clopiolines is demonstrated by the presence of bacteria in the response of the intestinal immune system to bacterial colonization by acquiring a state of a deficiency response in the blood defense process against reproductive and active readiness against pathogens, this dialogue leads to the production of large amounts of IgA<sup>(14)</sup>.

The total IgE level in the blood was found to be

clinically relevant in nephrotic syndrome and this is consistent with<sup>(15)</sup>, it showed that the serum IgE level was significantly higher in the renal group compared to the control group from IgE to increased risk of hypersensitivity, resulting from dialysis in patients with chronic renal failure with itching<sup>(16)</sup>. The results of the current study shown in Table (1-1) showed a significant increase in the concentration of interleukin IL-6 in patients with kidney failure. Chronic presence of bacteria is higher than the absence of bacteria in patients Chronic kidney failure and comparison with the control sample, as patients with kidney failure with bacteria reached 72.74 pg/ml Also we note there is a rise in patients with chronic kidney failure not having bacteria pg/ml 57.43 compared to the control group 5.58 pg/ml) and explained that immunodeficiency in renal failure patients is conservative The changes in immune-dependent antibodies, as a result of these changes the proportion of immune stimuli such as interleukin and my increase in patients with renal failure to increase excretion and lack of exit from the kidneys. The lack of antibody production is a reflection of the deficiency of T-lymphocyte function<sup>(17)</sup>. The reason for the high concentration in this interleukin is due to the fact that it is from the IL-1 $\beta$  group, as the pro-inflammatory cytokine kinases increase their production rates during cases of immunopathy and infections. And different infections. renin in the kidneys can lead to supplemental activation It is an effect that prevents the use of eskirine. Because renin concentrations are higher in the renal tissues, this may explain the renal tendency of complementary diseases in the presence of complementary mutations or autoantibodies<sup>(18)</sup>. These results are consistent with the researcher's findings<sup>(19)</sup> and his group, which indicated a decrease in complement protein C3 in patients with chronic renal failure Comparing it with the healthy people, the reason for the decrease in the complement protein in bacteria may be due to several reasons<sup>(20)</sup> including the higher loss of the complement parts by urinary tract method, an increase in its consumption of immune reactions in the circulatory system or by mucous surfaces and interference with the complement-anti-complementary substance And the activation taking place In the complement system of patients with chronic renal failure by complications and a decrease in immune production<sup>(21)</sup>.

**Ethical Clearance:** The Research Ethical Committee at scientific research by ethical approval of both environmental and health and higher education and

scientific research ministries in Iraq.

**Conflict of Interest:** The authors declare that they have no conflict of interest.

**Funding:** Self-funding

## References

1. Davidson's, Principles & practice of medicine. 21st edition. New York. Churchill Livingstone: 482-492 dialysis.; *Pediatr. Nephrol.* 2010; 22(10):1689–1702
2. Júnior, O., de Paula Sabino, A., Carvalho, R., Romana, D., & Rios, A. Inflammation and poor response to treatment with erythropoietin in chronic kidney disease. *CEP.* 2015; 35501, 296.
- 3- Ricci, Z., & Ronco, C. New insights in acute kidney failure in the critically ill. *Swiss medical weekly.* 2012; 142(3334).
4. Al-Salamy, M. H., Darweesh, M. F., & Almousawi, A. N. Detection of erm genes in *Staphylococcus lentus* Erythromycin resistance isolated from Renal failure patients in Najaf province. *Al-Qadisiyah Journal Of Pure Science.* 2017; 22(3), 647-656.
5. United States Renal Data System. *USRDS. Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States.* Bethesda, MD:2013; National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases.
6. Pecoits-Filho, R., Heimbürger, O., Bárány, P., Suliman, M., Fehrman-Ekholm, I., Lindholm, B., & Stenvinkel, P. Associations between circulating inflammatory markers and residual renal function in CRF patients. *American journal of kidney diseases.* 2003; 41(6), 1212-1218.
7. Diepold, M., Noellke, P., Duffner, U., Kontny, U., & Berner, R. Performance of interleukin-6 and interleukin-8 serum levels in pediatric oncology patients with neutropenia and fever for the assessment of low-risk. *BMC infectious diseases.* 2008; 8(1), 28.
8. Loreen R. Stromberg, Heather M. Mendez and Harshini Mukundan *Reviewed Detection Method for Lipopolysaccharides:2016; Past and Present.*
9. Nasr, S. H., Satoskar, A., Markowitz, G. S., Valeri, A. M., Appel, G. B., Stokes, M. B., ... & D'Agati, V. D. Proliferative glomerulonephritis with monoclonal IgG deposits. *Journal of the American*

- Society of Nephrology. 2006; 20(9), 2055-2064.
10. Wagner, F. B.; Gustavo, A. D. and Sell, et al. The role of the immune system in idiopathic nephrotic syndrome. Immunology Laboratory of Integrated Center for Health Research, Brazil. 2013; Pp; 3-6.
  11. Panzer, E. (2015). Algorithms for the symbolic integration of hyperlogarithms with applications to Feynman integrals. *Computer Physics Communications*, 188, 148-166.
  12. Pinlaor, S., Ieamviteevanich, K., Pinlaor, P., Maleewong, W., & Pipitgool, V. Seroprevalence of specific total immunoglobulin (Ig), IgG and IgM antibodies to *Toxoplasma gondii* in blood donors from Loei Province, Northeast Thailand. *Southeast Asian journal of tropical medicine and public health*. 2000;31(1), 123-127.
  13. Descamps-Latscha, B., Witko-Sarsat, V., Nguyen-Khoa, T., Nguyen, A. T., Gausson, V., Mothu, N., ... & Jungers, P. Early prediction of IgA nephropathy progression: proteinuria and AOPP are strong prognostic markers. *Kidney international*. 2004; 66(4), 1606-1612.
  14. Le Bourgot, C., Ferret-Bernard, S., Le Normand, L., Savary, G., Menendez-Aparicio, E., Blat, S., ... & Le Huërou-Luron, I. Maternal short-chain fructooligosaccharide supplementation influences intestinal immune system maturation in piglets. *PLoS One*. 2014; 9(9), e107508.
  15. Shin, Y. H., Yu, J., Ahn, K., Kim, K. W., Suh, D. I., Yu, H. S., ... & Lee, E. The interaction between prenatal exposure to home renovation and reactive oxygen species genes in cord blood IgE response is modified by maternal atopy. *Allergy, asthma & immunology research*. 2016; 8(1), 41-48.
  16. Mettang, T. Uremic itch management. In *Itch-Management in Clinical Practice*. 2016; Vol. 50, pp. 133-141 Karger Publishers.
  17. Ahmed, N.; Dawson, M.; Smith, G. and Wood, E. *Biology of Disease*. Taylor Francis group. 2007; pp: 25-41.
  18. Zivile D. Békássy Ann-Charlotte Kristoffersson Johan Rebetz Ramesh Tati Anders I. Olin Diana Karpman. Aliskiren inhibits renin-mediated complement activation. *Kidney international*. 2018; 94(4), 689-700
  19. Saeki, T., Saito, A., Yamazaki, H., Emura, I., Imai, N., Ueno, M.,... & Gejyo, F. Tubulointerstitial nephritis associated with IgG4-related systemic disease. *Clinical and experimental nephrology*. 2007; 11(2), 168-173.
  20. Mohamed Abdel-Dayem Saleh, & Alaa Mohamed Hamid Al-Bayati. The inflammatory role of some cellular kinetics, immunoglobulins and complement proteins in immunological pathology of kidney failure in a sample of patients in Diyala province. *Baghdad Journal of Science*. 2017;14 (2), 247-253.
  21. Reis, E. S., DeAngelis, R. A., Chen, H., Resuello, R. R., Ricklin, D., & Lambris, J. D. Therapeutic C3 inhibitor Cp40 abrogates complement activation induced by modern hemodialysis filters. *Immunobiology*. 2015; 220(4), 476-482.