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Evaluation of Inflammatory state among Sudanese with Renal Disorders patients

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Abstract

Sudan has increased number of renal disorders patients every year, no risk factors detected rather than area of living, malaria endemics and somehow hereditary related kidney issues, as consanguinity mirage is a life role for most of population, so accumulation of genetic variants occur during life. Inflammation one of the accompanied issue to renal disorders, so this study aimed to evaluate health state of kidney disease patients through certain measurements (white blood cell, serum iron and ferritin). Using hematology analyzer to measure TWBC and chemical analyzer for ferritin and iron, data obtained analyzed via statistical package of social science (SPSS version 20).

Result: Comparing measured parameters; TWBC, serum iron and ferritin between patients with chronic kidney disease, acute kidney disease and normal control showed significant increased in serum ferritin, especially amomg chronic kidney disease (p value < 0.05).

In contrast when these parameters were compared between patients with chronic kidney disease and acute kidney disease revealed significance difference regarding both serum iron and ferritin (p value < 0.05), while TWBCs showed insignificant differences as (p value > 0.05).

Conclusion: ferritin as acute phase reactant has significant role in state of inflammation of kidney disease patients.

1. Introduction

One of the few African nations that has offered dialysis and transplants under universal health coverage for a number of decades is Sudan. Approximately 4500 patients were living with kidney transplants prior to the war, and about 8000 patients needed continuous dialysis (appendix). In order to accommodate more patients within financial limits, hemodialysis was offered twice a week rather than three times [1].

In comparison to other essential organs, the kidneys are an extremely delicate organ with limited regeneration efficiency. Renal problems rank among the fastest-growing health conditions worldwide, along with diabetes, hypertension, and cardiovascular disease. Thus, a new area of research focusing on

different regenerative alternatives has been sparked by the study of renal tissue repair and regeneration. Because repairing damaged cells is just as important as stopping the advancement of end-stage renal illness in order to prevent kidney damage. Therefore, stem cells are being studied as a novel therapeutic strategy with the goal of lessening the burden of various kidney diseases[2].

Kidney disease: A diverse collection of conditions affecting the structure and function of the kidneys is referred to as kidney disease. It is now known that even slight deviations in kidney structure and function measurements are linked to a higher chance of complications in other organ systems and death, all of which happen much more frequently than renal failure. A length of three months or less is referred to as acute, but a duration of more than three months is considered chronic. Within a week, alterations in kidney function occur in acute kidney injury (AKI), a subtype of acute kidney illnesses and disorders (AKD). AKI and CKD have a complicated relationship; CKD raises the risk of AKI, while AKI can cause CKD[3].

Acute Kidney Injury (AKI)

Acute kidney injury, also known as acute renal failure, is defined as an unanticipated decrease in urine production followed by an increase in serum creatinine concentration, along with an inability to eliminate waste, maintain electrolyte balance, and maintain water balance. An arbitrary increase in serum creatinine levels of at least 26.5 mol/L with urine output less than 0.5 mL/kg/h for more than 6 hours is used as a marker for a patient to be classified under stage 1, or the initial stage of AKI, according to the AKI Network criteria. AKI is divided into multiple stages. Additionally, for stage 2, urine production should be < 0.5 mL/kg/h for more than 12 hours, and serum creatinine levels should be 2.0–3.0 times higher than the reference line.This continues until the third stage, when the urine output is less than 0.3 mL/kg/h for the entire day and the serum creatinine level is higher than 354 mol/L. In some cases, patients who initiated renal replacement therapy are directly classified under stage 3 AKI[4].

Chronic kidney disease (CKD)

Is characterized by progressive loss of kidney function, ultimately leading to end-stage kidney disease (ESKD), necessitating long-term kidney replacement therapy such as transplantation or hemodialysis/peritoneal dialysis. As kidney function declines, mortality and comorbidities, particularly cardiovascular complications, rise steadily.CKD is a significant global public health challenge, particularly affecting the elderly population, with nearly half of CKD patients aged over 70 years. However, while younger patients with CKD typically experience progressive loss of kidney function, 30% of patients over 65 years of age with CKD have stable disease (2–7). Currently, CKD affects 10–15% of the global population, significantly impacting overall health. The surge in CKD prevalence worldwide is primarily attributed to the escalating prevalence of traditional risk factors, such as obesity, hypertension, and diabetes mellitus. Additionally, metabolic factors, including insulin resistance, dyslipidemia, and hyperuricemia, have been associated with CKD development and progression. Some studies indicate a higher prevalence of CKD among men, with African Americans exhibiting a higher predisposition to kidney damage than Caucasians[5].

Up to 4 grams of iron are typically found in the body, mostly as hemoglobin. Iron intake from food or other sources, such as blood transfusions, and iron loss, such as monthly bleeding or the shedding of epithelial cells, are balanced to determine the total iron content of the body. The human body does not,

however, have a natural mechanism for excreting excess iron. Patients with CKD experience marked alterations in iron balance and distribution, when some cells and tissues are iron deficient and some iron loaded. It leads to dysregulation of physiological crosstalk between iron, oxygen, and erythropoiesis[6].

Inflammation: A complex web of interactions between renal parenchymal cells and local immune cells, including dendritic and macrophage cells, as well as the recruitment of circulating monocytes, lymphocytes, and neutrophils, characterizes inflammation, a condition closely associated with renal illness[7].More than 800 million people worldwide suffer from chronic kidney disease (CKD), a complicated and multivariate pathological illness that has become a major global health concern and a leading cause of death due to a rise in deaths over the previous 20 years. Hypertension, diabetes, glomerulonephritis, tubulointerstitial disease, immune-mediated disorders, and hereditary kidney illnesses are the primary causes of chronic kidney disease (CKD) [8].

Inflammation is one of the main factors contributing to poor outcomes for individuals with renal failure, according to mounting clinical evidence. For example, a high level of C-reactive protein is now commonly recognized as a sign of atherosclerosis and is suggestive of an inflammatory response. Furthermore, prior research has demonstrated a strong correlation between the white blood cell (WBC) count and unfavorable outcomes in dialysis patients. The WBC count is also a conventional biomarker of inflammation and infection responses. As a clinical indicator of iron storage, ferritin is frequently impacted by inflammation. Ferritin promotes the production of reactive oxygen species (ROS) and causes macrophage accumulation during inflammation. In patients with renal failure, ferritin is substantially linked to cardiovascular prognosis and mortality[9].

2. Material and method

This study involved 42 patients diagnosed with chronic kidney disease. 20 (47.6%) and 22 (52.4%) diagnosed with acute kidney disease. They were recruited in order to measure total white blood cell count (TWBC), which conducted via automated hematology analyzer (Mindray BC3000 plus), S iron and s ferritin, which measured via BTS350 plus (Biosystem). Patients were Khartoum state relocated population, as war occurred, people had to changes directions of living due to conflicts. Control data of normal population (30 subjects) with no obvious disorders and complains.

3. Result

This cross sectional case control study was conducted among renal diagnosed patients in order to evaluate the state of inflammation in their systems regarding to white blood cell %count and iron with ferritin levels. They were 42 patients, they were 66.7 % males and 33.3% females as in figure 1, their age ranged from 20 to 77 years, their duration of illness with renal issues ranged from 0.3 to 10 years, measured parameters were TWBC, serum iron and ferritin as in table 1.

Table 1: Descriptive Statisticsfor chronic kidney disease patients

	N	Minimum	Maximum	Mean	Std. Deviation
Age	42	20	77	44.23	16.289
Duration/y	42	.30	10.00	4.4591	3.17545

TWBC	42	2.6	17.0	5.738	2.2910
S. iron	42	7.6	1009.0	102.980	153.7361
Ferritin	42	22	10000	1362.14	1585.147

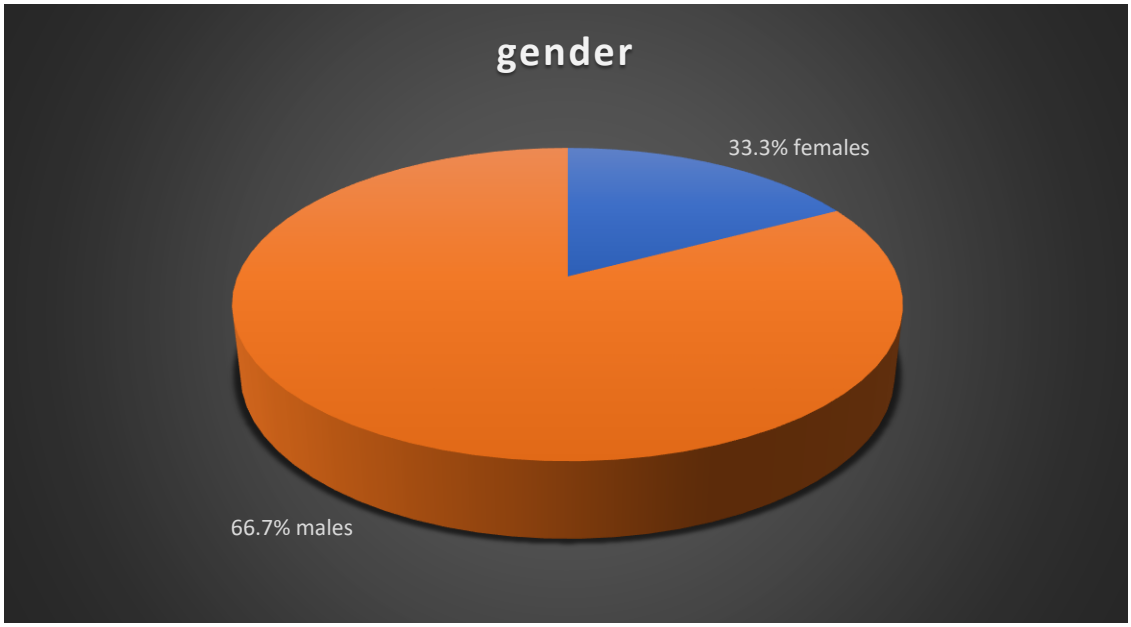

Figure 1: Distribution of gender among patients

Testing normality of data by means of the Shapiro-Wilk test, which is a hypothesis test that is applied to a data sample with a null hypothesis that the sample has a normal distribution. In this test, a high p-value indicates the data set has a normal distribution, while a low p-value indicates that it does not have a normal distribution, so the data of this study showed significant variations for measured parameters as in table 2.

Table 2: Tests of Normality

	Shapiro-Wilk	**P. value**
TWBC	.753	.000*
S. iron	.439	.000*
Ferritin	.599	.000*

Due to abnormal distribution of data, for comparison, the Mann-Whitney U test is used to compare differences between two groups, data of chronic with data of acute kidney disease, then each data of chronic and acute kidney disease with control group to find significant differences, comparing data of chronic kidney disease with control group's data, it revealed that the significant difference obtained by ferritin only as it was highly increased among patients CKD and AKD than control (p value 0.000),

while no significant difference for TWBC, and serum iron, as each has p value >0.05 as in table 3 and the same for comparing data of acute kidney disease with control as in table 3.

Table 3: Compassion of TWBCs, S. iron and Ferritin between Chronic and Acute kidney disease and Control

Parameters		N	Mean Rank	Median	P. value
TWBCs	Chronic kidney disease	22	32.05	5.9	0.097
	Acute kidney disease	20	23.98	4.8	
	Control	18	35.86	7.0	
S. iron	Chronic kidney disease	22	23.95	61.7	0.075
	Acute kidney disease	20	35.73	93.5	
	Control	18	32.69	82.5	
Ferritin	Chronic kidney disease	22	43.84	1367.0	0.000*
	Acute kidney disease	20	34.08	969.0	
	Control	18	10.22	29.0	

Comparing measured parameters, TWBC, serum iron and ferritin between patients with chronic kidney disease and acute kidney disease showed insignificant difference in TWBC level has p value > 0.05, while serum iron and ferritin showed significant differences as each has (p value <0.05) as in table 5.

Table 5: Compassion of TWBCs, S. iron andFerritin between Chronic and Acute kidney disease

Parameters		N	Mean Rank	Median	U-value	P. value
TWBCs	Chronic kidney disease	22	24.89	5.9	145.5	0.060
	Acute kidney disease	20	17.78	4.8		
S. iron	Chronic kidney disease	22	17.82	61.7	139.0	0.041*
	Acute kidney disease	20	25.55	93.5		
Ferritin	Chronic kidney disease	22	25.84	1367.0	124.5	0.016*
	Acute kidney disease	20	16.73	969.0		

Considering genders, comparing data of females with data of males, there was no significant difference for each of measured parameters (p value>0.05) as in table 6.

Table 6: Compassion of TWBCs, S. iron and Ferritinaccording to gender of patients

Parameters	Gender	N	Mean Rank	Median	U-value	P. value
TWBCs	Male	28	22.20	5.5	176.5	0.602
	Female	14	20.11	4.8		
S. iron	Male	28	21.61	85.5	193.0	0.936
	Female	14	21.29	70.5		
Ferritin	Male	28	20.88	1000.0	178.5	0.639
	Female	14	22.75	1000.0		

Pearson's correlation of measured parameters with age and duration of disease brought ferritin has negative correlation with age with no significant difference (p value>0.05) and positive correlations of age with iron and TWBC and duration also has positive correlations with WBC, iron and ferritin with no significant differences as in table 7.

Table 7: Correlationsof Age and duration withTWBCs, S. iron and Ferritin

		TWBC	S. iron	Ferritin
Age	Pearson Correlation	.262	.080	-.177
	P. value	.093	.616	.263
Duration/y	Pearson Correlation	.030	-.195	.115
	P. value	.850	.217	.467

No association of TWBC with type of kidney disease and control group data, no significant difference obtained as in table 8.

Table 8: Association between TWBCs category and type of kidney disease

		Chronic kidney disease	Acute kidney disease	Control	Chi-Square	P. value
TWBCs category	Normal	22 (100.0%)	19 (95.0%)	15 (83.3%)	4.554	0.103
	High	0 (0.0%)	1 (5.0%)	3 (16.7%)		

4. Discussion

persistent, low-grade inflammation in chronic kidney disease (CKD), which raises the risk of cardiovascular events in both CKD patients and healthy people and is frequently a sign of atherosclerosis. However, because of functional deficiencies in several immune system components, people with chronic kidney disease (CKD) are also more susceptible to infections. Because this immune response frequently results in a similar inflammatory profile, it can be difficult to differentiate between inflammation brought on by infection and CKD[10].

In hospitalized patients, acute kidney damage (AKI) is a common and serious consequence that is linked to longer hospital stays, higher rates of morbidity, and death. Thus, with a reported prevalence of 0.25% in the general population and 18% in hospitalized patients, AKI is a serious public health concern. Those who have survived AKI are more likely to develop chronic kidney disease (CKD), which can lead to end-stage renal disease (ESRD)[11].

Ferritin: Our study's finding of elevated ferritin in CKD and AKD patients is consistent with prior research. Elevated ferritin is often associated with inflammation and altered iron metabolism in kidney diseases, these findings have agreement with outcomes of study conducted by Kalantar-Zadeh et al. (2001)[12] observed that ferritin is an acute-phase reactant and tends to be elevated in CKD patients due to chronic inflammation, even in the absence of iron overload as well as outcomes of Drüeke et al. (2006)[13] in the CHOIR study also noted elevated ferritin in CKD patients, associated with both iron stores and inflammatory response, while Chung et al. (2015) [14]found ferritin significantly higher in AKD patients, especially during the acute phase of illness, suggesting inflammatory activation.

Serum Iron: Our findings showed no significant differences in serum iron between patient groups and controls, an agreement obtained by A Pfeffe M et al (2009) [15]reported that serum iron may not reliably reflect iron stores in CKD due to variability with inflammation and diurnal changes.The lack of significance in your study may be due to variations in iron supplementation, diet, or inflammation masking serum iron levels.

Total White Blood Cell Count (TWBC): Our study found no significant changes in TWBC across groups, including between males and females or with disease duration.In contrast, Sarnak et al. (2003)[16] reported increased TWBC in patients with CKD associated with higher cardiovascular risk, but these findings were more prominent in end-stage renal disease (ESRD), which may not reflect your study population.

Correlation with Age and Disease Duration: This study finding showed no significant correlations between age/duration and measured parameters, although trends were observed (e.g., a non-significant negative correlation of ferritin with age).KDOQI (2006)[17] guidelines suggest disease duration may affect iron metabolism, but this is often confounded by treatment regimens.

5. Conclusion

Serum ferritin was strong inflammatory markers for patients with kidney disease, especially for chronic state. While the serum irons state in this study did not bind with inflammatory state for patients with kidney disease. White blood cell has no significant role as well.

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