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Research Article

Prevalence of Anemia and Variations of Hematological Parameters among Anemic Hemodialysis Patients in the Tripoli Region

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Abstract: Background: Prolonged decline in the ability of the kidney to regulate acid–base balance, eliminate waste products, and manage water homeostasis and entered chronic phase, toxic metabolic accumulates and erythropoietin secretion by the kidney is decreasing and causes hematological changes including decrease of HCT, MCV, RBCs and platelet counts. Hemodialysis became a practical treatment for kidney failure and is the most common method used to treat advanced and permanent kidney failure. Anemia is one of the most common complications in hemodialysis patients**. Objectives:** The study aimed to evaluate the prevalence of anemia among hemodialysis patients and investigate the variations of hematological parameters among anemic hemodialysis patients in the Tripoli region. **Materials and Methods:** The present study was conducted on 250 renal failure patients, attending Tripoli Center for dialysis and 100 normal healthy subjects. The study Ethical Committee of the medical centers and the Libyan Academy of graduate studies reviewed and approved the study design and patient consent statements were taken from each patient. Information's about the patients were recorded in a questionnaire. A blood sample of 5 ml was drawn by venous puncture from each normal healthy individual and hemodialysis patient. 2.5 ml of the blood sample was collected in K-EDTA tubes for the hematological examinations and another 2.5 ml of the blood sample was collected in a plain tubes for biochemical tests (serum urea, creatinine, and uric acid concentrations). The hematological parameters (RBCs count, Hb, HCT, MCV, MCH, MCHC, WBCs count, differential count of WBCs, and Platelets count) were determined using an automated hematology analyzer Sysmex (K- 4500) machine. The data were compared using GraphPad Prism version.9. The statistical significance of differences between groups was evaluated with the independent t-test. A P-value of <0.05 was considered significant for all statistical tests. **Results:** The results showed that the prevalence of anemia among hemodialysis patients was 89.8%. The degrees of anemia were 17% severe, 71.66% moderate, and 11.34% mild anemia. The types of anemia were 13.36% microcytic hypochromic, 82.59% normocytic hypochromic, and 4.05% macrocytic hypochromic anemia. RBCs, WBCs & platelets counts, Hct, MCHC, and Lymphocytes % showed a significant (*P<0.01*) decrease, and MCV was a significant (*P<0.01*) increase in the anemic hemodialysis patients compared with the healthy individuals. But, a significant (*P<0.05*) decrease in MCH was observed in the anemic hemodialysis patients when compared with the healthy individuals. A significant correlation was observed between RBCs and their indices with most of the hematological parameters. A significant (*P<0.01*) negative correlation was observed between serum urea with Hb, and RBCs count and Hct. While, a significant (*P<0.01*) positive correlation was recorded between uric acid with platelets count. A significant (*P<0.05*) positive correlation was observed between gender with platelets count, while, a significant negative correlation was recorded between gender with serum urea (*P<0.01*), creatinine, and uric acid, and Hb (*P<0.05*). A significant (*P<0.01*) negative correlation was observed between blood groups with serum uric acid. A significant (*P<0.01*) positive correlation was observed between durations of hemodialysis with RBCs count and Hb, while, a significant (*P<0.05*) negative correlation was

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recorded between durations of hemodialysis with body weight, and MCHC. **Conclusion**: It can be concluded that a higher prevalence of moderate, normocytic hypochromic anemia among hemodialysis patients. Also, results showed a significant variation in hematological parameters among the anemic hemodialysis patients. So, hemodialysis patients advice to examine the hematological parameters and treated from anemia if detected.

Keywords: Prevalence of Anemia, Hemodialysis Patients, Hematological Alterations, Tripoli Region

1. Introduction

When healthy, the kidney removes waste products and fluids from the blood stream and excreting them in the urine, also the kidneys maintain the body's internal equilibrium of water and minerals (sodium, potassium, chloride, calcium, phosphorus, magnesium and sulfate) [1]. Renal failure is a situation in which kidney fails to function adequately [2]. Chronic renal failure (CRF) is a syndrome characterized by progressive and irreversible deterioration of renal function due to slow destruction of renal parenchyma [3]. Symptoms develop slowly, diagnosis is based on laboratory testing of renal function, sometimes followed by renal biopsy [4]. Acidosis is the major problem in CRF with development of biochemical azotemia and clinical uremia syndrome [3]. Prolonged decline in the ability of the kidney to regulate acid–base balance, eliminate waste products, and manage water homeostasis and entered chronic phase, toxic metabolic accumulates and erythropoietin secretion by the kidney is decreasing and causes hematological changes include decrease of HCT, MCV, RBCs, and platelet counts [5]. Treatment is primary directed at the underlying condition but includes fluid and electrolyte management and after hemodialysis, peritoneal dialysis, or transplantation [2, 4]. The CRF refers to decline in the GFR caused by a variety of disease, such as diabetes, glomerulonephritis, hypertensive nephropathy, congenital and hereditary diseases [6], and polycystic kidney disease [6, 7].

Dialysis is a procedure that removes excess fluids and toxic end products of metabolism such as urea from the plasma and corrects electrolytes balance by dialyzing the patient's blood against fluid containing no urea but has levels of minerals like potassium and calcium that are similar to their natural concentration in healthy blood [8]. It may be used for those with an acute disturbance in kidney function or for those with progressive but chemically worsening kidney function a state know as chronic renal failure (CRF) [9]. Hemodialysis became a practical treatment for kidney failure and is the most common method used to treat advanced and permanent kidney failure [10].

End stage kidney disease (ESKD) is a major public health problem in Libya with diabetic kidney disease and chronic glomerulonephritis being the leading causes. The most frequent co-morbidities were hypertension, obesity and the metabolic syndrome [11]. The total number of patients with ESKD in the Middle East is almost 100 000, the mean prevalence being 430 per million population [12]. In 2003, the reported incidence of ESKD and prevalence of dialysis-treated ESKD in Libya were the same at 200 per million population (pmp). In 2007, the prevalence of dialysis-treated ESKD was 350 pmp, but the true incidence of ESKD was not available. The most recent published WHO data in 2012 showed the incidence of dialysis-treated ESKD had risen to 282 pmp and the prevalence of dialysis-treated ESKD had reached 624 pmp [11]. Between 2007 and 2009, the number of patients on dialysis in Libya increased from 2116 to 2417. It is projected that the number of patients on dialysis will increase from 2417 in 2009 to 7667 in 2024 [6]. To maintain a patient to dialysis machine ratio of 1: 3.4, the number of dialysis machines will need to grow from 1045 in 2014 to 2255 in 2024. The required number of general nephrology beds in 2014 is estimated around 468, with 59 additional regional transplant beds. These numbers are expected to be around 547 and 69 respectively by 2024[6] .

Anemia is a condition in which the number of RBCs (and consequently their oxygen carrying capacity) is insufficient to meet the body's physiologic needs [13]. It is one of the most common complications in hemodialysis patients [14]. The severity of anemia is related to the stage of kidney disease affecting nearly all patients with stage 5 CKD [15], and the deficiency or reduction in erythropoietin production which is the main cause. Also, inflammation, hyperparathyroidism, deficiency of folic acid, iron, and vitamin B_{12}, hemolysis, and blood loss are causes of anemia in among chronic kidney diseases patients [16, 17]. Anemia causes fatigue, reduced exercise capacity, reduced libido, and cognitive function, which ultimately have a negative impact on their quality of life [18, 19]. Anemia in CKD is associated with deprived general health, manifested as dizziness, fatigue, shortness of breath, reduced quality of life and exercise capacity, when Hb<10 g/dl [20]. In addition to being related to CVDs, heart failure are the leading causes of mortality in CKD [21]. Anemia in CKD is associated with cognitive impairment, sleep disturbances, CKD progression, cardiovascular comorbidities, and higher mortality [22, 23].

Most of the previous literatures studied the prevalence of anemia among hemodialysis patients among anemic patients were in other countries but, to our knowledge, the evidences reporting it in our country are very few. So, for this reason, we carried out the current study.

2. Objectives

The current study aimed to evaluate the prevalence of anemia among hemodialysis patients and investigate the variations of hematological parameters among anemic hemodialysis patients in the Tripoli region.

3. Subjects and Methods

3.1. Experimental design

The present study was conducted on 250 renal failure patients, attending Tripoli Center for dialysis and 100 normal healthy subjects. The study design was reviewed and approved by the Ethical Committee of the medical centers and the Libyan Academy of graduate studies. Ethical approval and patient consent statements were taken from every patient. Personal information's regarding hemodialysis were collected through face-to-face interviews, using a structured questionnaire. Demographic and anthropometric data were included file number, gender, age, and duration of dialysis.

3.2. Blood sampling

A blood sample of 5 ml was drawn by venous puncture from each normal healthy individual and hemodialysis patient. 2.5 ml of the blood sample was collected in K-EDTA tubes for the hematological examinations and another 2.5 ml of the blood sample was collected in plain tubes for biochemical tests (serum urea, creatinine, and uric acid).

The blood samples of all the study subjects were sent to the hospital laboratory for estimation of CBC and concentrations of serum urea, creatinine, and uric acid.

3.3. Determination of a complete blood count (CBC)

The complete blood count (RBCs count, Hb, HCT, MCV, MCH, MCHC, WBCs count, differential count of WBCs, and Platelets count) were determined using an automated hematology analyzer Sysmex (K- 4500) machine.

3.4. Determination of anemia among hemodialysis patients

According to the World Health Organization [24] criteria for anemia in men is Hb<13 gm/dL and women is Hb<12 gm/dL. The degrees of anemia were mild, moderate, and severe in male>14 years, when hemoglobin concentrations were (11–12.9), (8–10.9), and <8g/dL and in female>14 years, when hemoglobin concentrations were (11–11.9), (8–10.9), and <8 g/dL, respectively [25].

3.5. Determination of serum urea, creatinine, and uric acid concentrations

2.5 ml of the blood sample was collected in a plain tubes for estimation of serum urea, creatinine, and uric acid concentrations. After clotting of blood in the plain vial, serum was separated, within an hour; by centrifugation at 3000 - 5000 g for 5 min. Serum was used for measurements of the levels of serum urea, creatinine, and uric acid. Biochemical studies were performed using commercially available kits from Biomeriux (France), and serum parameters were quantified according to the manufacturer's instructions.

3.6. Data analysis

The data of blood parameters of both healthy individuals and hemodialysis patients were performed on a computer excel sheath and captured from excel for further analysis on GraphPad Prism 9.0 software. The Kolmogorov-Smirnov test was used to assess the normality of distribution of continuous variables. The statistical significance of differences between groups was evaluated with the independent t-test. A P-value of <0.05 was considered significant for all statistical test.

4. Results

4.1. The prevalence of anemia among hemodialysis patients

Table 1 and Figure 1 show that anemic hemodialysis patients were 247 patients (98.8%) and non-anemic patients were 03 patients (1.2%).

Table 1. The prevalence of anemia among hemodialysis patients

Groups	Frequency	Percent (%)
Anemic Patients	247	98.8
Non-anemic Patients	03	1.2

4.2. Distribution of anemic hemodialysis patients according to the degrees of anemia

The distribution of anemic hemodialysis patients according to the degrees of anemia was 42 patients (17%) with severe anemia, 177 patients (71.66%) with moderate anemia, and 28 patients (11.34%) with mild anemia (Table 2& Figure 2).

Table 2. Distribution of anemic hemodialysis patients according to the degrees of anemia

Degrees of anemia	Frequency	Percent (%)
Mild anemia	28	11.34
Moderate anemia	177	71.66
Severe anemia	42	17

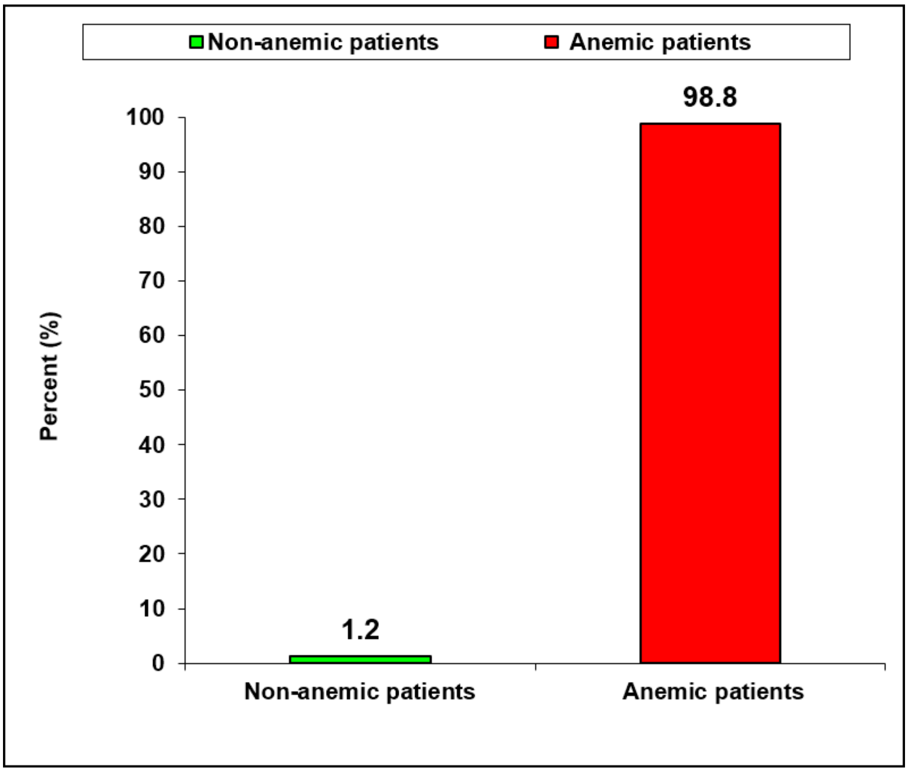

Figure 1. Distribution of anemia among hemodialysis patients according to gender

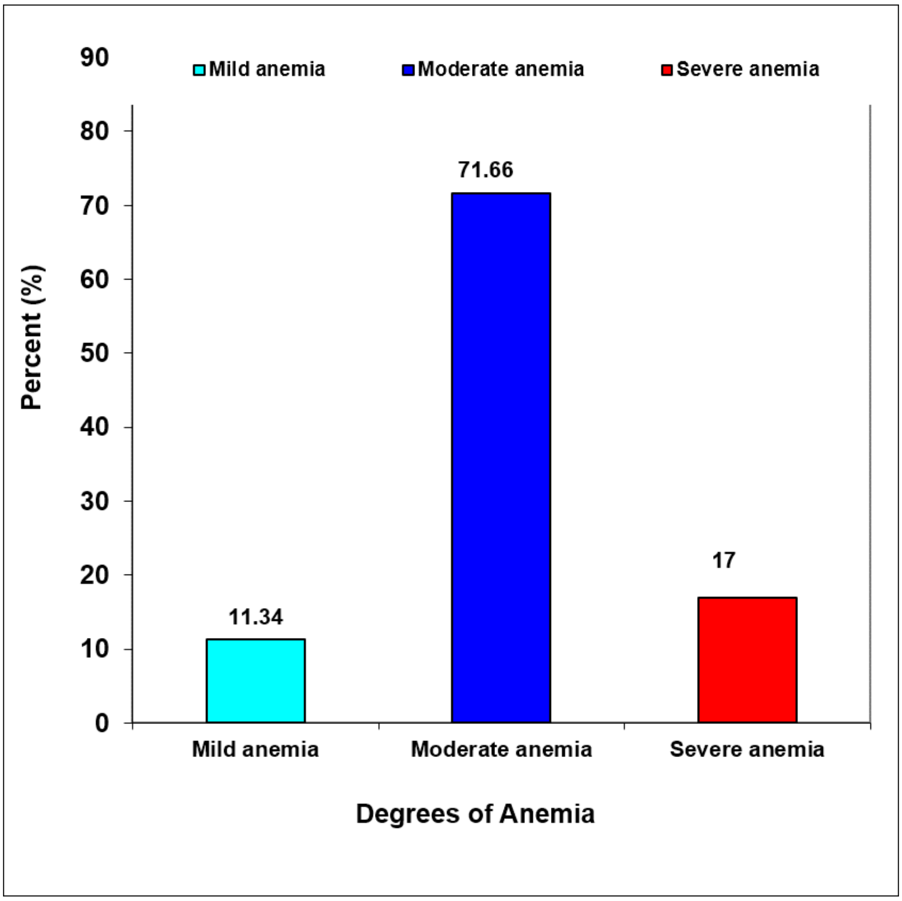

Figure 2. Distribution of anemic hemodialysis patients according to the degrees of anemia

4.3. Distribution of anemic hemodialysis patients according to the type of anemia

The distribution of anemic hemodialysis patients according to the types of anemia was 33 patients (13.36%) with Microcytic hypochromic, 204 patients (82.59%) with normocytic hypochromic, and 10 patients (4.05%) with macrocytic hypochromic anemia (Table 3 & Figure 3).

Table 3. Distribution of anemic hemodialysis patients according to the type of anemia

Types of anemia	Frequency	Percent (%)
Microcytic hypochromic (MCV<80)	33	13.36
Normocytic hypochromic [MCV(80-98)]	204	82.59
Macrocytic hypochromic (MCV>98)	10	4.05

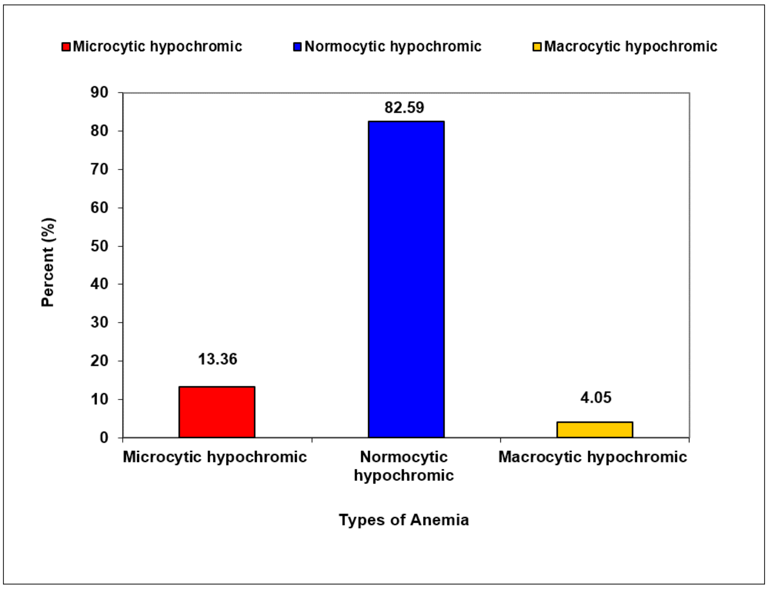

Figure 3. Distribution of anemic hemodialysis patients according to the type of anemia.

4.4. Haematological parameters in healthy individuals and hemodialysis patients

4.4.1. RBCs count and its indices in healthy individuals and hemodialysis patients

The data shown in Table 4 and Figure 4 showed a significant (*P<0.01*) decrease in RBCs counts (3.24 ± 0.04) x10^6 cell/µl in the hemodialysis patients compared with the healthy individuals (4.81 ± 0.04) x10^6 cell/µl.

Table 4. RBCs count and its indices in healthy individuals and hemodialysis patients

Groups Parameters	Healthy Individuals Mean ± SE	Hemodialysis Patients Mean ± SE
RBCs Count (x10^6)	4.81 ± 0.04	3.24 ± 0.04**
Hb (g/dl)	14.21 ± 0.11	9.27 ± 0.10**
Hct (%)	41.33 ± 0.30	27.97 ± 0.35**
MCV (fl)	85.11 ± 0.60	87.36 ± 0.41**
MCH (Pg)	29.40 ± 0.14	28.84 ± 0.16*
MCHC (g/dl)	34.32 ± 0.10	32.90 ± 0.11**

Healthy Individuals (n=100); Hemodialysis Patients (n=250); *: Significant at P<0.05 compared with the healthy individuals; **: Significant at P<0.01 compared with the healthy individuals.

A significant (*P<0.01*) decreased in hemoglobin concentrations (9.27 ± 0.10) g/dl were recorded in the hemodialysis patients when compared with the healthy individuals (14.21 ± 0.11) g/dl (Table 4 & Figure 5).

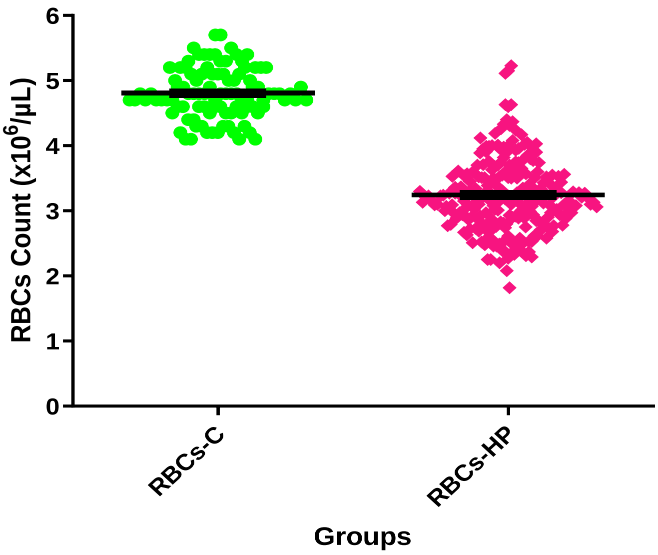

Figure 4. RBCs count in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients)

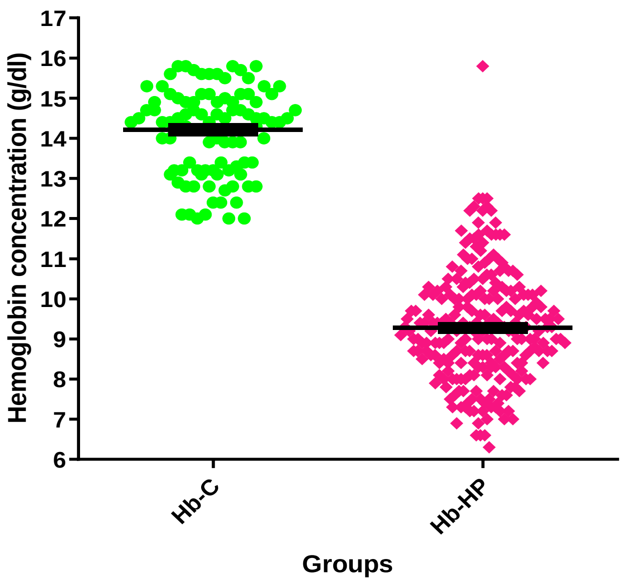

Figure 5. Hemoglobin concentration in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients)

Hematocrit values were a significantly (*P<0.01*) decreased (27.97 ± 0.35%) in the hemodialysis patients when compared to the healthy individuals (41.33 ± 0.30%) (Table 4 & Figure 6).

A significant ((*P<0.01*) increase was observed in mean corpuscular volume (MCV) (87.36 ± 0.41fl) of hemodialysis patients compared with the healthy individuals (85.11 ± 0.60fl) (Table 4 & Figure 7).

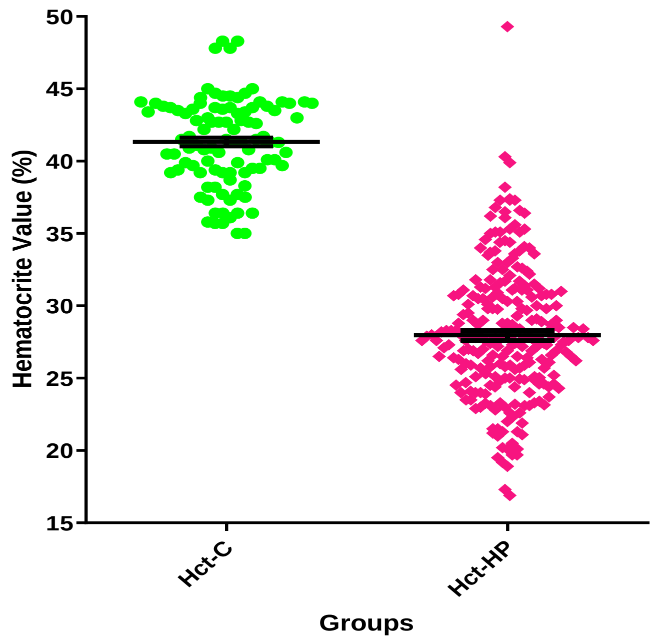

Figure 6. Hematocrit value in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients)

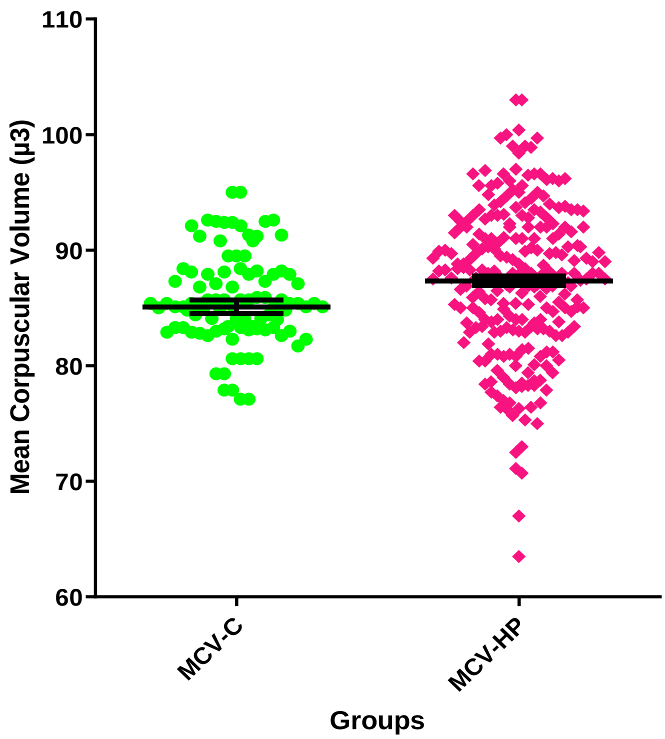

Figure 7. Mean corpuscular volume in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients).

A significant (*P<0.05*) decrease in MCH (28.84 ± 0.16) was observed in the hemodialysis patients when compared with the healthy individuals (29.40 ± 0.14) (Table 4 & Figure 8).

Table 4 & Figure 9 were showed a significant (*P<0.01*) decrease in MCHC (32.90 ± 0.11) in the hemodialysis patients when compared with the healthy individuals (34.32 ± 0.10).

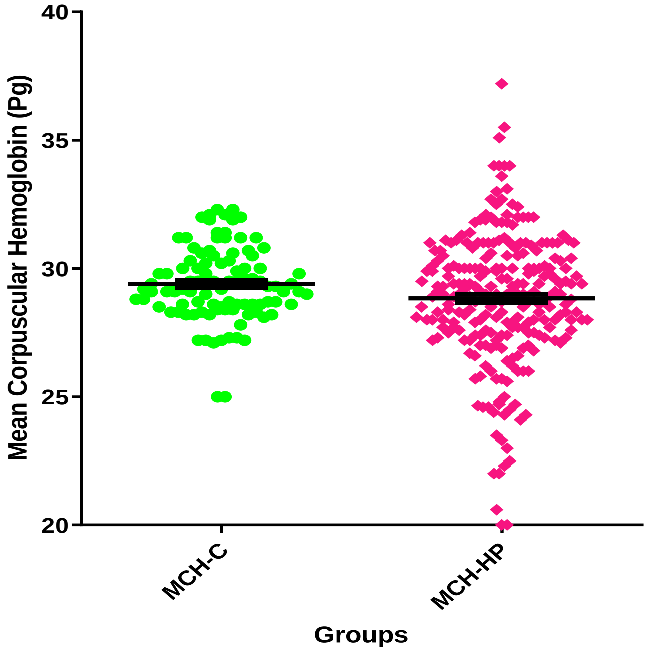

Figure 8. Mean corpuscular hemoglobin in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients).

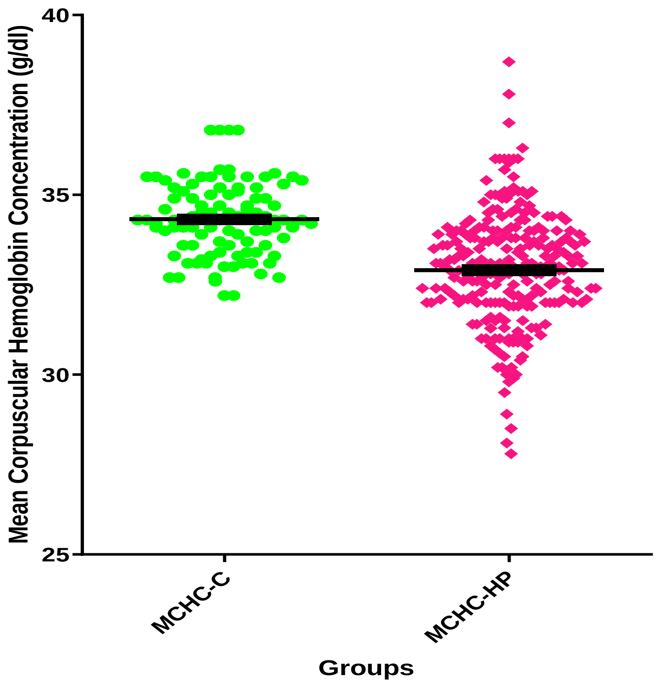

Figure 9. Mean corpuscular hemoglobin concentration in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients).

4.4.2. WBCs count, Neutrophils %, Lymphocytes%, and Platelets Count in healthy individuals and hemodialysis patients

WBCs count was showed a significant (*P<0.01*) decrease (6.39 ± 0.13) x10³ cell/µl in the hemodialysis patients as compared to the healthy individuals (7.80 ± 0.18) x10³ cell/µl (Table 5 & Figure 10).

Table 5. WBCs count, Neutrophils %, Lymphocytes%, and Platelets Count in healthy individuals and hemodialysis patients

Groups Parameters	Healthy Individuals Mean ± SE	Hemodialysis Patients Mean ± SE
WBCs Count (x10³)	7.80 ± 0.18	6.39 ± 0.13**
Neutrophils %	63.63 ± 1.48	63.79± 0.77
Lymphocytes %	29.43 ± 0.81	24.05 ± 0.69**
Platelets Count (x10³)	241.50 ± 5.55	203.06 ± 4.03**

Healthy Individuals (n=100); Hemodialysis Patients (n=250); **: Significant at P<0.01 compared with the healthy individuals.

The data recorded in Table 5 and Figure 11 indicated a none significant (*P>0.05*) changes was observed in neutrophils % (63.79± 0.77) in the hemodialysis patients as compared with the healthy individuals (63.63 ± 1.48).

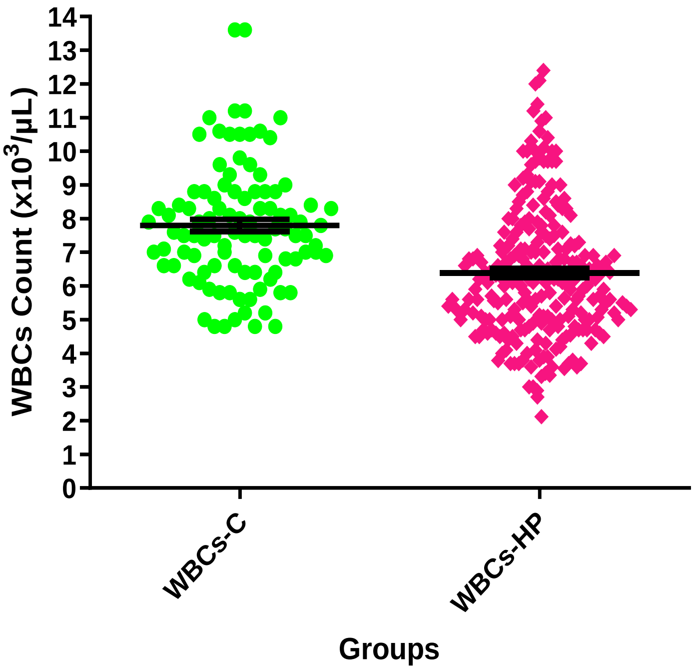

Figure 10. WBCs count in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients).

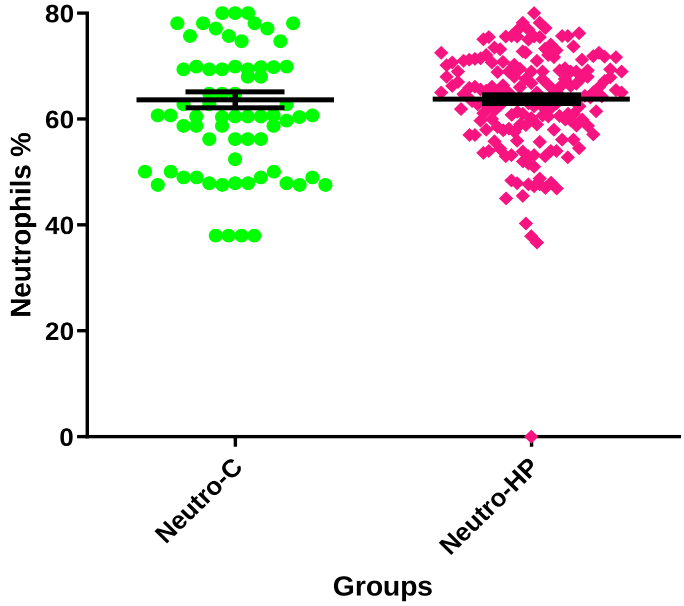

Figure 11. Neutrophils % in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients).

Lymphocytes % was significantly (*P<0.01*) decreased (24.05 ± 0.69) in the hemodialysis patients when compared with the healthy individuals (29.43 ± 0.81) (Table 5 & Figure 12).

The blood platelets count was significantly (*P<0.01*) decreased (203.06 ± 4.03)x 10^3/µl in the hemodialysis patients when compared with the healthy individuals (241.50 ± 5.55) x 10^3/µl (Table 5 & Figure 13).

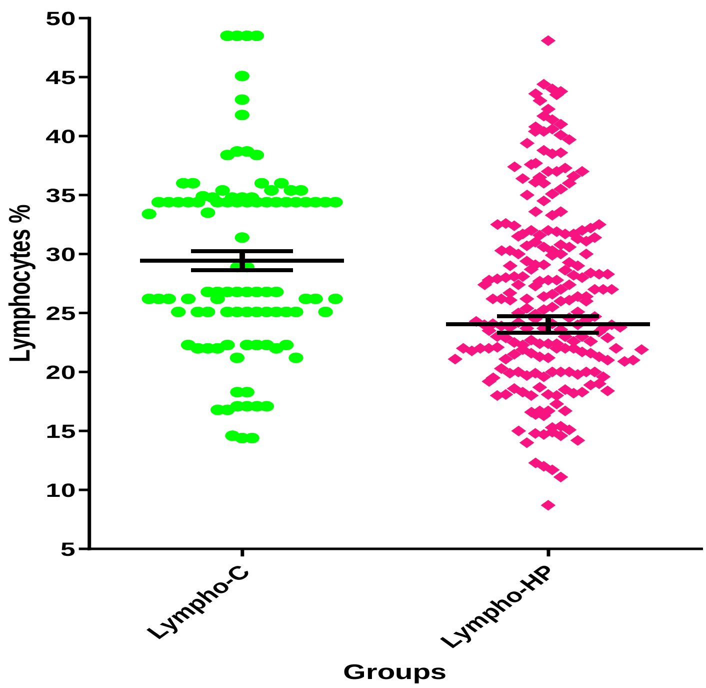

Figure 12. Lymphocytes % in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients).

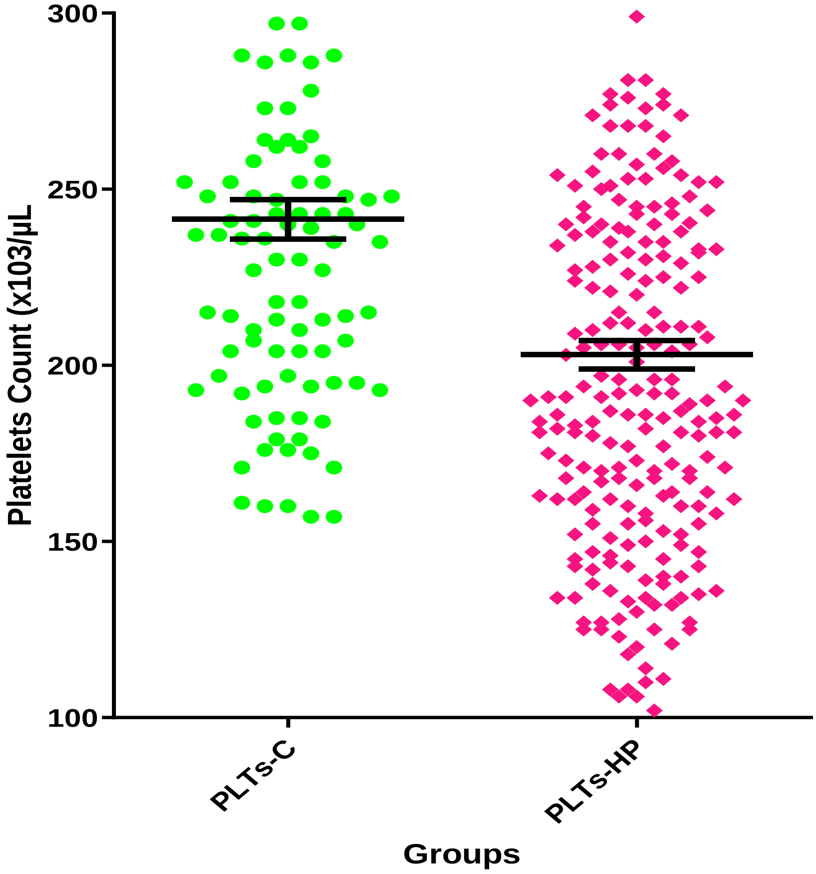

Figure 13. Platelets Count in healthy individuals and hemodialysis patients. (C: Healthy individuals; HP: Hemodialysis patients).

4.5. Correlation between RBCs count, Hb, Hct, and MCV & hematological parameters

Data in Table 6 show the correlation between RBCs count, Hb, Hct, and MCV with hematological parameters. A significant (*P<0.01*) positive correlation was observed between RBCs count with Hb, Hct, and WBCs (r = 0.851, 0.838, and 0.236, respectively), and lymphocytes% (*P= 0.042*, r = 0.129). While, a significant (*P<0.01*) negative correlation was recorded between RBCs count with MCV, MCH, and MCHC (r = -0.296, -0.401, and -0.265, respectively).

A significant (*P<0.01*) positive correlation was observed between Hb concentration with RBCs count, Hct, and WBCs (r = 0.851, 0.831, and 0.177, respectively), and MCV, and lymphocytes% (P= 0.020& 0.037, r = 0.148, 0.32, respectively).

A significant (*P<0.01*) positive correlation was observed between Hct value with RBCs count, Hb, WBCs, and lymphocytes% (r = 838, 0.831, 0.174, and 0.224, respectively). While, a significant (*P<0.01*) negative correlation was recorded between Hct value with MCHC (r = - 0.279).

Also, a significant (*P<0.01& P<0.05*) positive correlation was observed between mean corpuscular volume with MCH and Hb (r = 0.834 & 0.148), respectively. While, a significant (*P<0.01*) negative correlation was recorded between mean corpuscular volume with RBCs count (r = - 0.296).

Table 6. Correlation between RBCs count, Hb, Hct, and MCV & hematological parameters

Parameters		RBCs	Hb	Hct	MCV	MCH	MCHC	WBCs	NEUT	LYM	PLT
Correlation of RBCs with	Correlation Coefficient (r)	-	0.851**	0.838**	-0.296	-0.401**	-0.265**	0.236**	0.005	0.129*	0.011
	P Value	-	0.000	0.000	0.000	0.000	0.000	0.000	0.933	0.042	0.868
Correlation of Hb with	Correlation Coefficient (r)	0.851**	-	0.831**	0.148*	0.119	-0.026	0.177**	0.009	0.132*	-0.031
	P Value	0.000	-	0.000	0.020	0.061	0.682	0.005	0.887	0.037	0.629
Correlation of Hct with	Correlation Coefficient (r)	0.838**	0.831**	-	0.077	-0.092	-0.279**	0.174**	0.086	0.224**	-0.037
	P Value	0.000	0.000	-	0.225	0.147	0.000	0.006	0.176	0.000	0.565
Correlation of MCV with	Correlation Coefficient (r)	-0.296**	0.148*	0.077	-	0.843**	-0.004	-0.102	0.035	0.003	-0.012
	P Value	0.000	0.020	0.225	-	0.000	0.953	0.108	0.586	0.958	0.850

: Significant correlation at P<0.05; **: Significant correlation at P<0.01

4.6. Correlation between serum urea, creatinine and uric acid concentrations & hematological parameters

Data in Table 7 show the correlation between serum urea, creatinine, and uric acid with hematological parameters. A significant (*P<0.01*) negative correlation was observed between serum urea with Hb (r = 0.174), and RBCs count and Hct (*P= 0.010& 0.027*, r = 0.172& 0.146, respectively). While, a significant (*P<0.01*) positive correlation was recorded between uric acid with platelets count (r = 0.232).

Table 7. Correlation between serum urea, creatinine and uric acid concentrations & hematological parameters

Parameters		RBC	Hb	Hct	MCV	MCH	MCHC	WBC	NEUT	LYM	PLT
Correlation of Urea with	Correlation Coefficient (r)	-0.172*	-0.174**	-0.146*	-0.008	0.032	-0.023	0.081	0.018	-0.048	0.030
	P Value	0.010	0.008	0.027	0.906	0.628	0.730	0.224	0.789	0.468	0.647
Correlation of Creatinine with	Correlation Coefficient (r)	-0.066	-0.095	-0.081	0.012	-0.015	-0.005	-0.032	0.053	-0.096	-0.004
	P Value	0.322	0.151	0.222	0.854	0.820	0.937	0.634	0.424	0.148	0.947
Correlation of Uric acid with	Correlation Coefficient (r)	-0.020	-0.040	-0.036	-0.050	-0.038	0.092	0.006	0.085	0.022	0.232**
	P Value	0.808	0.622	0.654	0.538	0.640	0.256	0.941	0.297	0.787	0.004

: Significant correlation at P<0.05; **: Significant correlation at P<0.01

4.7. Correlation between gender and blood groups with hematological data.

Data in Table 8 show the correlation between gender and blood groups with hematological data. A significant (*P<0.05*) positive correlation was observed between gender with platelets count (r = 0.163), while, a significant negative correlation was recorded between gender with serum urea (*P<0.01*, r =-0.220), creatinine (r = -0.391), and uric acid (*P<0.05*, r=-0.180), and Hb (*P<0.05*, r=-0.160).

A significant (*P<0.01*) negative correlation was observed between blood groups with serum uric acid (r=-0.225).

Table 8. Correlation between gender and blood groups with hematological data

Parameters		WBC	RBC	HB	HCT	MCV	MCH	MCHC	PLT	LYM	NEUT	Urea	Creat	Uric acid
Correlation of gender with	Correlation Coefficient (r)	-0.106	-0.122	-.160-*	-0.109	0.047	-0.051	-0.116	0.16**	0.111	-0.144	-0.220**	-0.391-**	-0.180*
	P- Value	0.095	0.054	0.011	0.086	0.460	0.425	0.067	0.010	0.080	0.055	0.001	0.000	0.026
Correlation of blood group with	Correlation Coefficient (r)	-0.039	-0.017	-0.058	-0.094	-0.101	-0.062	0.038	-0.108	0.014	-0.024	-0.055	-0.071	-0.225**
	P- Value	0.536	0.785	0.360	0.139	0.113	0.333	0.553	0.089	0.821	0.746	0.409	0.285	0.005

****: Significant correlation at P<0.05; **: Significant correlation at P<0.01***

4.8. Correlation between body weight and durations of hemodialysis with hematological data

Data in Table 9 show the correlation between body weight and durations of hemodialysis with hematological data. A significant positive correlation was observed between body weight with WBCs count (*P<0.01*, r = 0.236), and serum uric acid (*P<0.05*, r = 0.162).

A significant (*P<0.01*) positive correlation was observed between durations of hemodialysis with RBCs count (r = 0.206) and Hb (r = 0.184), while, a significant (*P<0.05*) negative correlation was recorded between durations of hemodialysis with body weight (r =-0.157), and MCHC (r=-0.131).

Table 9. Correlation between body weight and duration of dialysis with and hematological data

Parameters		Weight	WBC	RBC	HB	HCT	MCV	MCH	MCHC	PLT	LYM	NEUT	Urea	Creat	Uric acid
Correlation of weight with	Correlation Coefficient (r)	-	0.236**	0.070	0.031	0.032	-0.082	-0.077	0.011	0.089	-0.022	0.051	0.093	0.067	0.162*
	P-Value	-	0.000	0.272	0.628	0.618	0.198	0.228	0.861	0.161	0.724	0.496	0.160	0.312	0.046
Correlation of Duration of dialysis with	Correlation Coefficient (r)	-.157-*	-0.110	.206**	.184**	-0.032	0.016	-0.052	-.131-*	0.014	0.091	-0.108	-0.124	0.052	-0.053
	P-Value	0.013	0.082	0.001	0.003	0.610	0.800	0.414	0.038	0.820	0.152	0.151	0.061	0.433	0.515

****: Significant correlation at P<0.05; **: Significant correlation at P<0.01***

5. Discussion

The present study aimed to evaluate the prevalence of anemia and investigate the variations of hematological parameters among hemodialysis patients in Tripoli region

In chronic kidney disease patients, RBCs count, hemoglobin concentration, hematocrit value, MCHC, and MCH are changed compared with the healthy individuals [26]. The kidneys are the main site of erythropoietin synthesis which is the primary regulator of erythropoiesis [27], that produce approximately 90% of erythropoietin by the Juxta glomerular apparatus (JGA), while the liver and other organs are produce about 10% of it [26].

The results of the current study showed a significant decreased in RBCs counts, Hb, Hct, MCH, and MCHC, and increase in MCV in the hemodialysis patients compared with the healthy individuals. These results run parallel with those obtained by the previous studies [27-31]. Olasunkanmi *et al.* [26] and Singh and Bhatta [30] reported that hemoglobin concentration, Hct value, and RBCs count were significantly changes in chronic kidney disease patients compared with the healthy individuals.

The present study showed that the prevalence of anemia among hemodialysis patients was 89.8%. The degrees of anemia were 17% severe, 71.66% moderate, and 11.34% mild anemia. The types of anemia were 13.36% microcytic hypochromic, 82.59% normocytic hypochromic, and 4.05% macrocytic hypochromic anemia. These results are in concordance with those of the previous studies [26, 32].

The moderate and normocytic normochromic anemia were the most prevalent [26]. Juma, [32] mention that the prevalence of anemia was 97% (26% mild, 31% severe, and 40% moderate and 47.4% normocytic normochromic, 28.9% microcytic hypochromic, 16.5% normocytic normochromic and microcytic hypochromic, 5.2% macrocytic, and 2% microcytic normochromic) in patients with chronic kidney disease at Muhimbili national hospital Dar Essalaam. The severity of anemia is directly proportional to the degree of renal function [33]. Rathod *et al.*, [34] mentioned that the most common anemia in chronic renal failure is normochromic normocytic, which can be correlated with the severity of renal failure. A severe anemia was observed in 18% of renal failure patients [35], mild, and moderate anemias were recorded in up to 69% and 100% of patients [36] and Normochromic normocytic blood picture is commonly seen in chronic disorders specially in renal failure patients, which is seen in all patients in Nigeria [26, 36], but a low percent (30%) was recorded in India, macrocytic anemia is seen in 5% of patients, and microcytic hypochromic is seen in 65% of patients [37].

Erythropoietin hormone regulates RBCs production and preserve the viability of RBCs by retarding the cleavage of deoxyribonucleic acid. When the EPO is absent, the cleavage of deoxyribonucleic acid is rapid which leads to the death of cells [30, 38]. Also, EPO excites the effect of plasma platelet activating factor-acetylhydrolase (PAF-AH), paraoxonase 1 (PON 1), and megakaryocyte colony activating factors [30, 39].

The main causes of the decrease in red blood cell count, hemoglobin concentration, and hematocrit in chronic kidney disease patients are decrease in the production of erythropoietin, factors increase hemolysis, low levels of serum iron, suppression of bone marrow erythropoiesis, hematuria and gastrointestinal blood loss, and decrease of RBCs life span [26, 30, 40-46]. Also, the affinity of hemoglobin to oxygen decreases and the availability of O_2/unit of Hb circulating through the kidney increases [34]. In addition, iron and folate deficiency, and chronic disease with endogenous erythropoietin resistance, heavy metal toxicity, blood loss, and a reduction in red cell survival induced by toxic radicals are from the contributing factors [47]. Iron replacement is required in the treatment of renal failure anemia [40].

Proinflammatory cytokines antagonize the action of erythropoietin by inhibiting erythroid progenitor cells and disrupting iron metabolism in renal failure patients. Erythropoietin resistance may be induced by inflammation. Also, it could be caused by inflammation-induced changes in erythropoietin-receptor properties, recycling, and assembly and by interference with post-receptor signaling routes. In addition, Neocytolysis might have a role in erythropoietin resistance [48], which is a physiological process initiated by a drop in erythropoietin levels, leads to selective hemolysis of young circulating red blood cells and subsequent down regulation of red cell mass when it is excessive [49].

Arogundade *et al.* [36], and Akinsola *et al.* [50] reported that the degree of anemia was worsened with the progression of chronic kidney disease.

The present study showed a significant decrease in WBCs count and lymphocytes % in the hemodialysis patients compared with the healthy individuals. These results are in agreement with the result of the previous studies [30, 33]. Olasunkanmi *et al,* [26] and Singh and Bhatta, [30] recorded that WBCs count was significantly changes in chronic kidney disease patients compared with the healthy individuals. Wasti *et al.[33]* mentioned that the lymphocytes count was decreased in 16.3% of chronic kidney disease patients which may be due to severe stress, kidney failure, chronic infections, and prolonged use of cortisone injection.

The decline of WBCs count in chronic kidney disease patients during dialysis is because of complement activation which causes the aggregation of neutrophils and adherence to the internal wall of the blood vessels [30, 51]. Suresh *et al.* [28] reported that WBCs count showed a non-significant decreased in CKD patients.

On the other hand, Kadhim *et al. [31]* observed an increase in WBCs count in CKD patients compared to the control group, which caused by up regulation, presence of interleukin-6 (IL-6) and TNF-α in blood, that contribute to inflammation in renal failure patients [31, 52].

The current study appeared a significant decrease in platelets count in the hemodialysis patients compared with the healthy individuals. This result is compatible with the result of Abdu *et al.* [37] who reported that the prevalence of thrombocytopaenia in chronic kidney disease patients was 52%. Mohamed, [53] confirmed that the patients with renal failure are at high risk of bleeding due to decrease of platelets count and platelet dysfunction. Also, Dorgalaleh *et al.* [5] found that platelet count was a significant decreased in chronic renal failure patients. In addition, Olasunkanmi *et al.,* [26] and Singh and Bhatta, [30] mentioned that platelet count was significantly changes in chronic kidney disease patients compared with the healthy individuals. In patients with CKD the decrease of EPO leads to fall in platelets count [30, 39].

On the other hand, Kadhim *et al. [31]* recorded a significant increase in platelet count among CKD patients compared to the control group.

Previous studies were showed that a negative correlation was observed between serum creatinine concentration and hematological parameters in renal failure patients and the degree of changes depends on the severity of the disease (Suresh *et al.,* 2012, and George *et al.,* 2015).

6. Conclusion

It can be concluded that a higher prevalence of moderate, normocytic hypochromic anemia among hemodialysis patients. In addition, results showed a significant variation in hematological parameters among the anemic hemodialysis patients. So, hemodialysis patients advice to examine the hematological parameters and treated from anemia if detected.

References

[1] Clase C., Gary A. and Kiberd B., (2004). Classifying kidney problems: can we avoid framing risks as diseases? BMJ, 329 (7471): 912–915.

[2] Alghythan A. K. and Alsaeed A. H. (2012). Hematological changes before and after hemodialysis. Sci. Res. Essays, 7(4): 490-497.

[3] Alpers, C. E.(2004). The Kidney. In: Vinay Kumar, Abul K. Abbas and Nelson Fausto. Robbins "pathologic basis of disease", Seventh edition, Elsevier Inc., 20: 960-965.

[4] Meyer, T. W., and Hostetter, T. H. (2007). Uremia. New England Journal of Medicine, 357(13), 1316-1325.

[5] Dorgalaleh A, Mahmudi M, Tabibian S, Khatib ZK, Tamaddon GH, Moghaddam ES, Bamedi T, Alizadeh S, and Moradi E. (2013). Anemia and thrombocytopenia in acute and chronic renal failure. Inter J Hematol Oncol Stem Cell Res., 7(4): 34-39.

[6] Akkari K. B. 2013)). Projecting Requirements for End Stage Renal Disease Services in Libya 2014-2024. Ibnosina J Med BS 2013;5(6):354-362.
[7] Schnackenberg, C. G. (2002). Physiological and pathophysiological roles of oxygen radicals in the renal microvasculature. Am. J. physiol. Regul. Integr. Comp., 282(2), R335-R342.
[8] Ahmad S, Misra M, Hoenich N, and Daugirdas J.(2008) Hemodialysis Apparatus In: Handbook of Dialysis. 4th ed. New York, NY;:59-78.
[9] Pendse S, Singh A, and Zawada E. (2008). Initiation of dialysis in : Handbook of dialysis 4th ed. New York, NY. PP: 14-21.
[10] Foote, E.F., and Manley, H.J. (2008). Hemodialysis and peritoneal dialysis. Pharmacotherapy, A Pathophysiologic Approach, PP. 793-810.
[11] George SV, Pullockara JK, Sailesh KS, and Mukkadan JK. (2015). A study to assess changes in the hematological profile in chronic kidney disease. Pharm Innov J., 4(6): 1-3.
[12] Najafi I (2009). Peritoneal dialysis in Iran and the Midde East. Peritoneal Dial. Int., 29: 217-221.
[13] World Health Organization. (2011). Haemoglobin concentrations for the diagnosis of anemia and assessment of severity.
[14] Mc Cullough PA, and Lepor NE. (2005). The deadly triangle of anemia, renal insufficiency, and cardiovascular disease: Implications for prognosis and treatment. Rev Cardiovasc Med., 6(1): 1-10.
[15] Astor, B. C., Muntner, P., Levin, A., Eustace, J. A., and Coresh, J. (2002). Association of kidney function with anemia: the third national health and nutrition examination survey (1988-1994). Arch Inter Med., 162 (12): 1401-1408.
[16] Abensur H. (2004). Anemia of chronic kidney disease. J Bras Nephrol., 26: 26-28.
[17] Abensur H, Bastos MG, and Canziani ME. (2006). Current aspects of anemia in chronic kidney disease. J Bras Nephrol., 28:104-107.
[18] Valderrábano F, Jofre R, and Lopez-Gomez JM. (2001). Quality of life in end-stage renal disease patients. Am J Kidney Dis., 38(3): 443-464.
[19] Bastos MG, Bregman R, and Kirsztajn GM. (2010). Chronic kidney diseases: Common and harmful, but also preventable and treatable. Rev Assoc Med Bras., 56(2): 248-253.
[20] Lankhorst, C. E., and Wish, J. B. (2010). Anemia in renal disease: diagnosis and management. Blood Rev., 24(1): 39-47.
[21] Abensur H. (2010). Iron deficiency in chronic kidney disease.. Rev Bras Hematol Hemo., 32: 95-98.
[22] Mehdi U, and Toto RD. (2009). Anemia, diabetes, and chronic kidney disease. Diabetes Care 32(7):1320-1326.
[23] Smith RE Jr. (2010). The clinical and economic burden of anemia. Am J Manag Care, 16: 59-66.
[24] World Health Organization (WHO). (2001). Prevalence and diagnosis of iron deficiency anemia: a guide for program manager. Geneva: WHO, UNICEF;52. 447-501.
[25] Qureshi NA, Chauhan MAZ, Goswami AP, and Suri SK. (2015). Study of anemia and its correlation with hematological parameters in patient of various age group. IOSR J Dent Med Sci., 14(9): 29-35
[26] Olasunkanmi, S. A., Olasunkanmi, S. A., Adindu, C., Sikiru, B., Mohammed, M., Mohammed, S., and Mohammed, A. A. (2012). Haematological Profile of Patients with Chronic Kidney Disease in Nigeria. J Nephrol Renal Transpl., 5(1): 2-10.
[27] Sabry, A., El-Dahshan, K., Mahmoud, K., El-Husseini, A., Sheashaa, H., and Abo-Zenah, H. (2007). Effect of hepatitis C virus infection on haematocrit and haemoglobin levels in Egyptian hemodialysis patients. Eur J Gen Med, 4(1), 9-15.
[28] Suresh M , Mallikarjuna reddy N, Sharan B.S. M, Bandi H.K.,Shravya k. G, and Chandrasekhar M. (2012). Hematological changes in chronic renal failure . Inter. J. Sci. Res. Publ., 2 (9): 1-4.
[29] Fouad, M., Ismail, M. I., Mahmoud, A. A., Fathy, H., Zidan, A., and Mostafa, E. (2015). Influence of chronic hepatitis B and C infections on anemia in hemodialysis patients. Enliven: Nephrol Renal Stud, 2(1): 1-6.
[30] Singh, S., and Bhatta, S. (2018). Biochemical and hematological parameters in chronic kidney disease. J Manmohan Memor Inst Health Sci., 4(1): 4-11.
[31] Kadhim, H. M., Al-Ghanimi, H. H., and Al-Dedah, R. M. (2020). Haematological parameters and biochemical indices in patients with chronic kidney disease before haemodialysis Al-Furat Al-Awsat Governorates/Iraq. In AIP Conference Proceedings, AIP Publishing LLC., 2290(020004): 1-13.
[32] Juma, A. (2012). Prevalence of Anemia and its associated factors in patients with Chronic Kidney Disease at Muhimbili National Hospital Dar es Salaam (Doctoral dissertation, Muhimbili University of health and Allied Sciences).
[33] Wasti AZ, Iqbal S, Fatima N and Haider S. (2013). Hematological disturbances associated with chronic kidney disease and kidney transplant patients. Inter J Advan Res., 1(10): 48-54.
[34] Rathod SG, Ade AK, and Shekokar PP. (2014). A study of haematological changes in chronic renal failure. Sch. J App Med Sci., 2(4A): 1232-1234.
[35] Oluboyede OA, and Williams AIO. (1995). Serum ferritin and other iron indices in adult Nigerians with chronic renal failure: Review of management of anaemia. Afr J. Med. Med. Sci., 24: 231-237.
[36] Arogundade FA, Bappa A, Sanusi AA., Akinola NO, Adediran JA. and Akinsola A. (2006). ;Haematologic indices and the response to erytheropoeitin in Chronic Renal Failure. Trop. J. Nephrol., 1: 13-20.
[37] Abdu A, Arogundade F. Adamu B, Dutse AI, Sanusi A, Sani MU, Mijinyawa MS, Nalado A, Akinsola A and Borodo MM. (2009). Anaemia and its response to treatment with recombinant human erythropoietin in chronic kidney disease patients. W. Af. J. Med., 28 (5): 295-299.

[38] Dessypris EN, and Sawyer ST. (2009). Erythropoiesis. In: Greer JP, Foerster J, Rodgers GM, Paraskevas F, Glader B, and Arber DA. Wintrobe's Clinical Hematology. 12th ed. Philadelphia: Lippincott Williams and Wilkins. PP.120.
[39] Papavasiliou EC, Gouva C, Siamopoulos KC, and Tselepis AD. (2006). PAF-acetylhydrolase activity in plasma of patients with chronic kidney disease. Effect of long-term therapy with erythropoietin. Nephrol Dial Trans., 21: 1270-1277.
[40] Kaufman, J. S., Reda, D. J., Fye, C. L., Goldfarb, D. S., Henderson, W. G., Kleinman, J. G., and Department of Veterans Affairs Cooperative Study Group on Erythropoietin in Hemodialysis Patients. (2001). Diagnostic value of iron indices in hemodialysis patients receiving epoetin. Kidney Inter., 60(1): 300-308.
[41] Cohen, G., Glorieux, G., Thornalley, P., Schepers, E., Meert, N., Jankowski, J., and Horl, W. H. (2007). Review on uraemic toxins III: recommendations for handling uraemic retention solutes in vitro—towards a standardized approach for research on uraemia. Nephrology Dialysis Transplantation, 22(12), 3381-3390.
[42] Locatelli F, Pozzoni P, and Del VL. (2007). Recombinant Human Epoietin beta in the treatment of renal anemia. Ther Clin Risk Manag., 3: 433-439.
[43] Piroddi, M., Depunzio, I., Calabrese, V., Mancuso, C., Aisa, C. M., Binaglia, L., and Galli, F. (2007). Oxidatively-modified and glycated proteins as candidate pro-inflammatory toxins in uremia and dialysis patients. Amino Acids, 32(4): 573-592.
[44] Vanholder, R., Meert, N., Schepers, E., Glorieux, G., Argiles, A., Brunet, P., and European Uremic Toxin Work Group (EUTox). (2007). Review on uraemic solutes II—variability in reported concentrations: causes and consequences. Nephrol Dial Transpl., 22(11): 3115-3121.
[45] Floris, M., Marchionna, N., Clementi, A., Kim, J. C., Cruz, D. N., Nalesso, F., and Ronco, C. (2011). Evaluation of a new polysulfone hemofilter for continuous renal replacement therapy. Blood Purific., 32(2): 133-138.
[46] Nasouti, M. A., Halili, S. A., Mousavi, Z. B., Nasouti, A., and Tamadon, M. R. (2017). Anemia among hemodialysis patients; an updated mini-review. Annf Res Dial., 2(1): 1-4.
[47] Scheiber-Mojdehkar, B., Lutzky, B., Schaufler, R., Sturm, B., and Goldenberg, H. (2004). Non–transferrin-bound iron in the serum of hemodialysis patients who receive ferric saccharate: No correlation to peroxide generation. J Amer Soc Nephrol., 15(6): 1648-1655.
[48] van der Putten K, Braam B, Jie KE, and Gaillard CAJM. (2008). Mechanisms of disease: Erythropoietin resistance in patients with both heart and kidney failure. Nat Clin Pract Nephr., 4(1): 47-57.
[49] Rice L, Ruiz W, Driscoll T, Whitley CE, Tapia R, Hachey DL, Gonzales GF, and Alfrey CP. (2001). Neocytolysis on descent from altitude: a newly recognized mechanism for the control of red cell mass. Ann Inter Med., 134(8): 652-656
[50] Akinsola A, Durosinmi MO, and Akinola NO, (2009). The haemoatological profile of Nigerians with chronic renal failure. Afr. J. Med. Med. Sci., 29: 13-16.
[51] Watts RG. (2009). Neutropenia. In: Greer JP, Foerster J, Rodgers GM, Paraskevas F, Glader B, Arber DA. Wintrobe's Clinical Hematology. 12th ed. Philadelphia: Lippincott Williams and Wilkins. PP. 1532.
[52] Tbahriti, H. F., Meknassi, D., Moussaoui, R., Messaoudi, A., Zemour, L., Kaddous, A., and Mekki, K. (2013). Inflammatory status in chronic renal failure: The role of homocysteinemia and pro-inflammatory cytokines. World J Nephrol., 2(2): 31-37.
[53] Mohamed, N. M. B. (2010). Evaluation of hemostatic mechanism in Sudanese renal failure patients attending Khartoum teaching hospital (Doctoral dissertation, Sudan University of Science and Technology).