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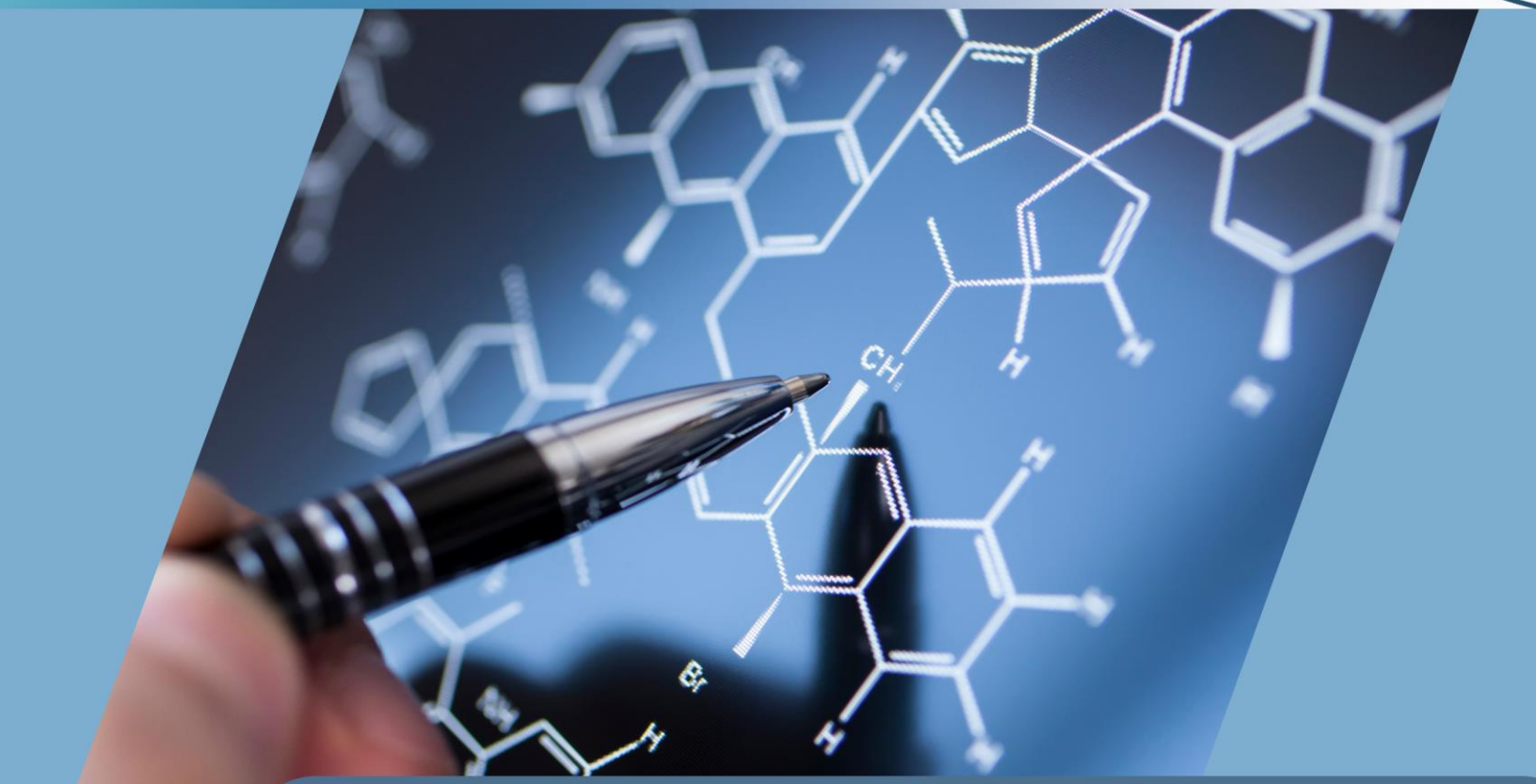

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Comparative Study of Hematological and Biochemical Parameters in Patients with Renal Failure depending on gender

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A B S T R A C T

The research aimed to evaluate the hematological and biochemical parameters in patients with kidney failure who are being treated at the nephrology department. This study was performed on 100 samples (50 males and 50 females) of patients who attended the nephrology department at Ibn Sina University Hospital. The survey of these samples was carried out among patients attending from January to May 2023. This study depended on the analysis of individual samples obtained from patient records, expressed as complete blood count (CBC) as well as biochemical changes in kidney function. It was found that most patients suffered from a decrease in hemoglobin, WBCs, and RBCs levels. According to the obtained results in this study, we found a significant decrease in the concentrations of hemoglobin, platelets, and leukocytes in both genders, while we found a significant increase in the concentrations of urea and creatinine. The results revealed no decline in potassium ions in patients, but sodium ions slightly decreased somewhat in males alone and not in females.

1 Introduction

Kidney failure is defined as a disorder in which the kidneys lose their ability to remove the waste product from the bloodstream (Prasad, 2014). Kidney ailment is a serious and widespread health problem. The manifestations of this disease are changes that affect the ability of the kidneys to remove toxins, dysregulation of salt, and water balance (Perco et al., 2006). Impairment of normal kidney function affects the metabolic secretion of many endocrine hormones, including thyroid hormones, insulin, and sex hormones (Kaka et al., 2022). The physiological spectrum of kidney disease is divided into two general aspects, which are “acute kidney injury (AKI) and chronic kidney disease (CKD)” (Khan et al., 2005).

AKI is a quick deterioration in kidney function that worsens over days or weeks and is usually accompanied by smaller volumes of urine and increase creatinine concentrations in the bloodstream (Figure 1). Acute kidney disease coincides with a significant increase in mortality by 40-80% and thus also contributes to fatal acute anemia (Lafrance & Miller, 2010).

CKD is a worldwide public health problem that confuses many health institutions around the world (McClellan & Powe 2009). It can be explained as a significant decrease in kidney excretory function or evidence that the kidneys have suffered significant damage (Genovese et al., 2010). More than 200 papers were analyzed to better recognize the disease that exterminates more than 30,000 people each year (Priyadarshani et al., 2023). CKD is a clinically silent disease in up to 90% of individuals until it reaches a high-risk condition (Chadban et al., 2003; John et al., 2004).

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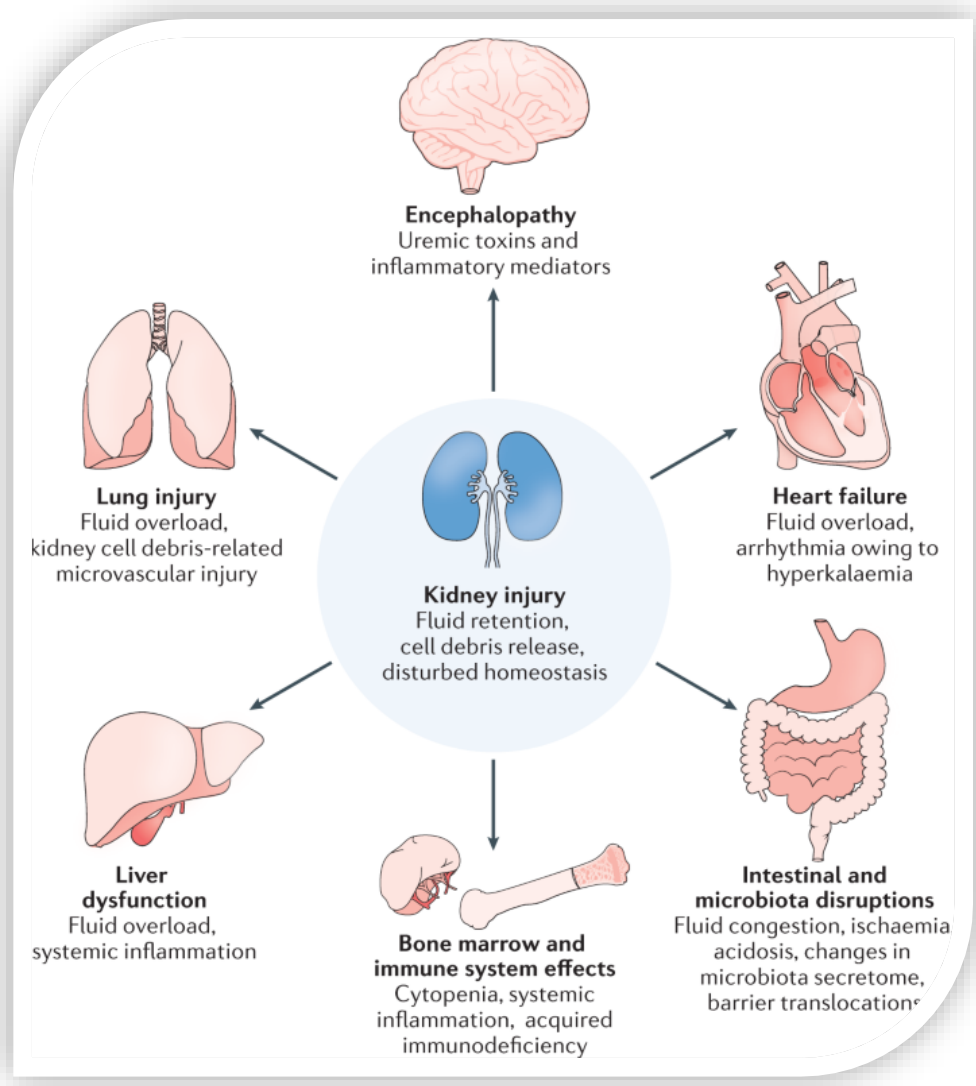

Figure (1) Systemic consequences of AKI (Kellum et al., 2021).

As for Libya, in 2003 the occurrence of the ailment was 200 patients per million people, and in 2007 the occurrence of the disease increased to 350 patients per million people. "Between 2007 and 2009, the number of patients undergoing dialysis in Libya increased from 2116 patients to 2417 patients, and the number of dialysis patients is expected to increase from 2417 patients in 2009 to 7667 patients in 2024" (Akkari 2013). Data published by the World Health Organization in 2012 revealed that the global occurrence of kidney failure was 282 individuals per million people, and the occurrence of the ailment increased to 624 individuals per million people (Goleg et al., 2014).

Chronic kidney failure is an irreversible destruction of the kidneys' nephrons that occurs slowly; the disease process is advanced and continuous until most of the kidney's nephrons are destroyed and replaced by fibrous tissue (Vaidya & Aeddula 2022). This failure may occur slowly in patients with normal diseases or kidney vesicular ailments, or it may result from injury to the kidneys (Figure 2). In chronic kidney failure, the kidneys lose their functions in filtering and maintaining blood balance due to inflammation of the kidney cells or nephrons (Kelly, et al, 1996).

Anemia is indicated by a low erythrocyte count or an inadequate quantity of hemoglobin (Hb) within erythrocytes (Begum & Latunde-Dada, 2019). It is estimated that more than half of those with CKD suffer from anemia (Bolignano et al., 2015). The lack of iron results when there is a need for more iron than can be released from iron reserves to promote Hb production (Nairz et al., 2016). Anemia can cause the heart muscles

to enlarge, increase the heart rate, and lead to cardiac failure in individuals with acute or chronic renal failure; this does not mean the heart stops functioning completely (Somvanshi et al., 2012).

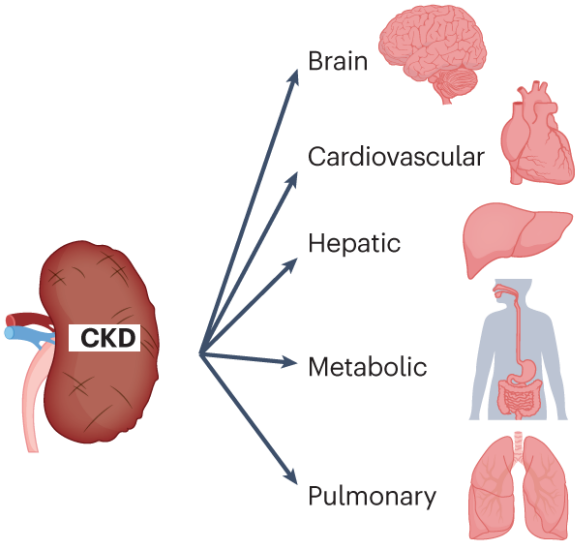

Figure (2) Systemic consequences of CKD (Benzing & Schumacher, 2023).

When the body lacks iron, it can result in a reduction in the formation of hemoglobin in red blood cells (RBCs), which can cause hypochromic anemia Srinivasan et al., 2016). Chronic anemia can be caused by a significant decrease in erythropoietin (EPO) hormone levels, as EPO hormone is responsible for the formation and increase of RBCs. Certain factors, such as signals sent from the bone marrow, can affect its production and release (Besarab et al., 2000).

2 Materials and Methods

This study aimed to investigate the hematological and biochemical variables in adult patients suffering from kidney failure. The variables evaluated included total white blood cell count (WBCs), estimated Hb concentration, total platelet count (PLT), and RBCs count, as well as creatinine, urea, sodium, and potassium. A total of 100 samples were analyzed, with an equal representation of both genders (50 males, 50 females) from the adult group.

A Chemistry AccentT 200 analyzer was utilized to measure various biochemical parameters. In addition, CBC Sysmex analyzer was employed to measure hematological parameters.

2.1 Data source and patients

100 patients have admitted to the Ibn Sina Teaching Hospital at Sirte (Libya) had been selected with age 40 years or above, who agreed to participate in the study. All of the selected patients (50 males and 50 females) had chronic kidney disease. A retrospective chart review was performed of those patients who attended *the nephrology department* between *January* 2023 and *May 2023*.

2.2 Statistical analysis

The analysis of the data was conducted through the utilization of IBM's Statistical Package for the Social Sciences (SPSS) software version 23.0 for windows (Armonk, New York: IBM Crops). Variables are compared using (cross tab) and Fisher's exact test, and differences between two means are used with (independent t-test). P value ≤ 0.05 was taken as level for statistical significance.

3 Results

The measurements of hematological and biochemical variables are summarized in Tables 1 and 2. There was a significant decrease (P-value ≤ 0.05) in haemoglobin concentration in both genders. This indicated the existence of anaemia in patients with kidney failure. There is a slight increase in the number of WBCs in the blood of patients with kidney failure compared to the normal range for this variable. The current results showed a significant decrease (P-value ≤0.05) in the number of platelets in the patients with kidney failure compared to the normal range of platelets.

The results of this study showed that the number of RBCs decreased significantly in patients with kidney failure, whether male or female, compared to normal levels, and that anaemia was noticeable in patients with chronic kidney failure. The results showed significant increases in both urea and creatinine compared to the normal levels of these variables.

The results in this study also indicated that there was a slight decrease in the concentration levels of sodium ions among patients with kidney failure compared to the normal levels of these variables. The results revealed no decline in potassium in patients with CKD.

Table (1) shows the hematological parameters (Hb level, WBC, red blood cell distribution width (RBC), and platelet counts) of the patients.

Hematological parameters	unit	Males		Females	
		Normal range*	**Mean ± S.D**	Normal range	**Mean ± S.D**
Hb	**g/dl**	14–18	9.6 ± 1.4	12–16	9.6 ± 1.4
WBCs	**$\times 10^9$/L**	4.8–10.8	6.3 ± 1.7	4.8–10.8	6.5 ± 2.1
RBCs	**$\times 10^{12}$/L**	4.5–6.0	3.1 ± 0.5	4.1–5.1	3.2 ± 0.5
PLT	**$\times 10^9$/L**	175–450	203.1 ± 77.3	175–450	218.4 ± 74.1
*from the references: (Billett, 1990; Marshall, 2022).					

Table (2) shows the biochemical parameters (urea, creatinine, sodium, and potassium) of the patients.

Biochemical parameters	SI Unit	Gender	Normal range*	Mean ± S.D
Creatinine	µmol/L	Male	61.9 - 114.9	131.75 ± 26.42
		Female	53 - 97.2	117.25 ± 37.14
Urea	mmol/L	Male	2.9 - 7.1	9.05 ± 1.86
		Female	2.9 - 7.1	8.099 ± 2.76
Sodium	mmol/L	Male	136 - 145	135.99 ± 3.04
		Female	136 - 145	136.595 ± 2.57
Potassium	mmol/L	Male	3.5 - 5.0	4.899 ± 0.74
		Female	3.5 - 5.0	4.366 ± 0.98
*from the references: (Lee, 2009 & Tyagi, 2023).				

4 Discussion

Hb levels in patients with kidney failure: "Many studies have indicated that anaemia represents one of the most important complications that accompany kidney failure" (Costa et al., 2008 & Hsu et al., 2001).This may be attributed to a deficiency in the secretion of the hormone EPO which is responsible for stimulating the process of forming RBCs (Erythropoiesis in the bone marrow) according to (Brunelli et al., 2009 & Erslev 1997). Additionally, the deficiency in the iron element in the patients with kidney failure suffer from (Kausz 2000). "Another reason that leads to the occurrence of anaemia in patients with kidney failure is the accumulation of nitrogenous wastes in the blood that inhibit the production of cells that generate RBCs in the bone marrow" (Besarab 2000).

WBCs count in patients with kidney failure: it found that there is a slight increase in the number of WBCs. This result did not reach agreement with what was mentioned by Kralova (2009). A notable increasing in WBCs is considered an inflammatory condition that is accompanied by uraemia, while it agreed with what was mentioned in the same study, as the slight increase is attributed to haemodialysis treatment, as neutrophils and monocytes are at the forefront of phagocytic cells whose number increases in response to any opportunistic stimulation.

PLTs count in patients with kidney failure: it found that there is a slight decline in the number of PLTs. This result agreed with Remuzzi (1989). It is believed that the decrease observed can be caused by the accumulation of nitrogenous compounds in the bloodstream. Nitrogenous wastes inhibited the formation of PLTs in the bone marrow. Moreover, the blood acidity was related to the decrease in PLT counts (Kopple et al., 2005). One of the important factors is the decrease in the level of the hormone thrombopoietin, which is responsible for regulating the formation process of PLTs and kidney excretion in the patients may be attributed to the accumulation of nitrogenous compounds in blood (Altun et al., 1999). Nitrogenous wastes inhibited the process of platelet formation in the bone marrow. Moreover, the blood acidity in patients with kidney failure was related to the decrease in platelet counts (Kopple et al., 2005). One of the important factors is the decrease in the level of the hormone thrombopoietin, which is responsible for regulating the formation process of platelets and kidney excretion (Altun et al. 1999).

RBCs count in patients with kidney failure: the number of RBCs decreased may be due to a lack of

secretion of the hormone (EPO), which is responsible for stimulating the process of Formation of RBCs in the bone marrow (Brunelli & Berns 2009; Erslev & Besarab 1997). Additionally, deficiencies in vitamin B12 and folate, and changes in red cell volume brought on by dialysis, might cause patients to exhibit macrocytic anemia (Shastry & Belurkar 2019). These patients have a lower hematocrit because of hemodilution.

There were noticeable similarities between both genders (Fig. 4) which suffering from kidney failure according to their levels of HB, WBCs, RBCs, sodium, potassium, and creatinine. Males exhibited higher mean PLT and urea levels than females. The outcomes point out that there are high connections between some parameters and gender differences that should be taken into account in future analyses.

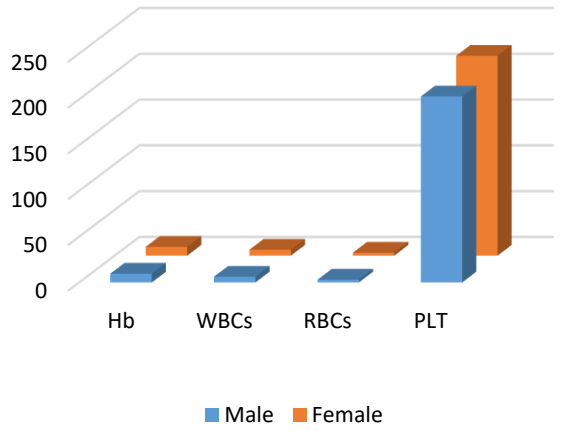

Figure (4) shows the hematological parameters of the patients

Creatinine and urea concentration in patients with kidney failure: It is known that urea and creatinine in blood serum are used as diagnostic tests for kidney function. The significant increases in urea and creatinine values are consistent with some previous studies (Zilve et al., 1989; Meri et al., 2022). The high concentration of urea and creatinine in the blood serum of patients with kidney failure may be attributed to the fact that they are metabolic wastes that are naturally excreted through urine, and in the case of kidney failure, a defect and decrease in kidney function occurs, leading to decreased excretion. These wastes accumulate and accumulate, increasing their concentration in the blood serum (Zilva et al., 1989). For the reason that urea is a substance that is reabsorbed again, it is not an indicator of kidney dysfunction. However, some studies have suggested that both serum creatinine and blood urea are the primary signs of kidney failure in both acute and chronic cases (Chhetri et al., 2008).

Sodium and potassium concentration in patients with kidney failure: The results revealed no decline in potassium in patients (Fig. 5), which differed with (Kumar 2004), as his study demonstrated that increased potassium can be attributed to causes many of them include the role of the kidney in excreting approximately (90-95%) of the potassium entering the body. When chronic kidney failure occurs, the efficiency of this process diminishes. However, the study of (Al-Abachi et al., 2012), in which the results of their study indicated that there is a decrease in the level of sodium and potassium concentration in the blood serum, and the deficiency of sodium and potassium ions in the blood serum among patients with kidney failure may be due to a deficiency of the hormone aldosterone, which it increases the loss of sodium and potassium ions in the urine (Al-Abachi et al., 2012).

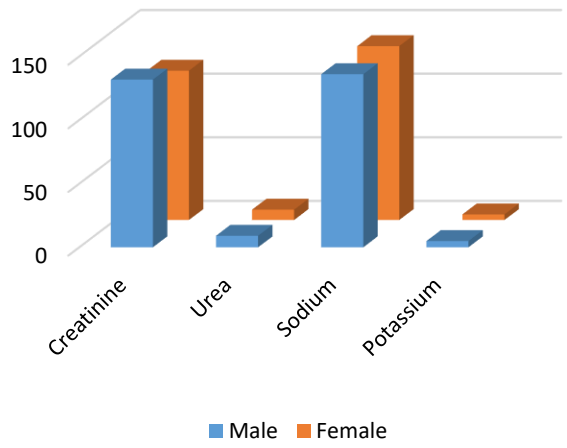

Figure (5) shows the biochemical parameters (urea, creatinine, sodium, and potassium) of the patients

5 Conclusions

Based on the results of this study, a significant decrease was observed in the concentration levels of haemoglobin, platelets, and RBCs in patients with kidney failure compared to the normal levels of these variables. Even though there was a slight decrease in the number of WBCs in the patients with kidney failure compared to normal levels for this variable. Furthermore, urea and creatinine were found to have significantly increased. While the results indicated a little decrease in sodium ions in the male group only and no decrease in the female group. There was no decrease in potassium ions in patients. Therefore, the results

revealed significant changes in both haematological parameters and some biochemical parameters, suggesting that kidney failure effects on the blood cell formation, and electrolyte balance.

Conflict of interest: The authors affirm that there are no conflicts of interest.

References

Akkari, K. (2022). Projecting requirements for end stage renal disease services in Libya 2014-2024. *Ibnosina Journal of Medicine and Biomedical Sciences*, *5*(06), 354-362.

Al-Abachi, S. Z., Mustafa, L. A., Hassan, D. S. K., & Al-Hadidi, A. A. (2012). Study of some biochemical changes in serum of patients with chronic renal failure. *Iraqi National Journal of Chemistry*, *12*(46), 270-280.

Begum, S., & Latunde-Dada, G. O. (2019). Anemia of Inflammation with An Emphasis on Chronic Kidney Disease. *Nutrients*, *11*(10), 2424. https://doi.org/10.3390/nu11102424

Benzing, T., & Schumacher, B. (2023). Chronic kidney disease promotes ageing in a multiorgan disease network. *Nature Reviews Nephrology*, *19*(9), 542–543. https://doi.org/10.1038/s41581-023-00729-6

Besarab, A., Amin, N., Ahsan, M., Vogel, S.E., Zazuwa, G., Frinak, S., Zazra, J.J., Anandan, J.V. & Gupta, A. (2000). Optimization of epoetin therapy with intravenous iron therapy in hemodialysis patients. *Journal of the American Society of Nephrology*, *11*(3), 530-538.

Billett, H. H. (1990). *Hemoglobin and hematocrit*. Clinical Methods - NCBI Bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK259/

Bolignano, D., Lennartz, S., Leonardis, D., D'arrigo, G., Tripepi, R., Emrich, I.E., Mallamaci, F., Fliser, D., Heine, G. & Zoccali, C. (2015). High estimated pulmonary artery systolic pressure predicts adverse cardiovascular outcomes in stage 2–4 chronic kidney disease. *Kidney international*, *88*(1), 130-136.

Brunelli, S. M., & Berns, J. S. (2009). Anemia in chronic kidney disease and end-stage renal disease. *Nephrology Rounds*, *7*(8), 1-6.

Chadban, S. J., Briganti, E. M., Kerr, P. G., Dunstan, D. W., Welborn, T. A., Zimmet, P. Z., & Atkins, R. C. (2003). Prevalence of kidney damage in Australian adults: The AusDiab kidney study. *Journal of the American Society of Nephrology*, *14*(suppl_2), S131-S138.

Chhetri, P. K., Manandhar, D. N., Bhattarai, S. P., Pahari, L. R., & Shrestha, R. (2008). Chronic kidney disease 5 on hemodialysis in Nepal medical college teaching hospital. *Nepal Med Coll J*, *10*(1), 8-10.

Costa, E., Rocha, C., Rocha-Pereira, P., Castro, E., Miranda, V., Faria, M. D. S., Quntannilha, A.; Belo, L. & Santos-Silva, A. (2008). Band 3 profile as a marker of erythrocyte changes in chronic kidney disease patients. *The Open Clinical Chemistry Journal*, *1*(1): 57-63.

Erslev, A. J., & Besarab, A. (1997). Erythropoietin in the pathogenesis and treatment of the anemia of chronic renal failure. *Kidney International*, *51*(3), 622-630.

Genovese, G., Friedman, D.J., Ross, M.D., Lecordier, L., Uzureau, P., Freedman, B.I., Bowden, D.W., Langefeld, C.D., Oleksyk, T.K., Uscinski Knob, A.L. and Bernhardy, A.J. (2010). Association of trypanolytic ApoL1 variants with kidney disease in African Americans. *Science*, *329*(5993), 841-845.

Goleg, F. A., Kong, N. C. T., & Sahathevan, R. (2014). Dialysis-treated end-stage kidney disease in Libya: Epidemiology and risk factors. *International urology and nephrology*, *46*, 1581-1587.

Hsu, C. Y., Bates, D. W., Kuperman, G. J., & Curhan, G. C. (2001). Relationship between hematocrit and renal function in men and women. *Kidney international*, *59*(2), 725-731.

John, R., Webb, M., Young, A., & Stevens, P. E. (2004). Unreferred chronic kidney disease: a longitudinal study. *American Journal of Kidney Diseases*, *43*(5), 825-835.

Kaka, N., Sethi, Y., Patel, N., Kaiwan, O., Al-Inaya, Y., Manchanda, K., & Uniyal, N. (2022). Endocrine manifestations of chronic kidney disease and their evolving management: A systematic review. *Disease-A-Month*, *68*(12), 101466. https://doi.org/10.1016/j.disamonth.2022.101466

Kausz, A. T., Obrador, G. T., & Pereira, B. J. (2000). Anemia management in patients with chronic renal insufficiency. *American journal of kidney diseases*, *36*(6), S39-S51.

Kellum, J. A., Romagnani, P., Ashuntantang, G., Ronco, C., Zarbock, A., & Anders, H. (2021). Acute kidney injury. *Nature Reviews Disease Primers*, *7*(1). https://doi.org/10.1038/s41572-021-00284-z

Kelly, F.J., Anderson, S., Thompson, M.M., Oyama, T.T., Kennefick, T.M., Corless, C.L., Roman, R.J., Kurtzberg, L., Pratt, B.M. & Ledbetter, S. R. (1999). Acute and chronic renal effects of recombinant human TGF-β2 in the rat. *Journal of the American Society of Nephrology*, *10*(6), 1264-1273.

Khan, R. N., Vohra, E. A., & Suleman, W. (2005). Factors determining outcome of acute renal failure patients. *Journal-Pakistan Medical Association*, *55*(12), 526.

Kopple, J. D., Kalantar-Zadeh, K., & Mehrotra, R. (2005). Risks of chronic metabolic acidosis in patients with

chronic kidney disease. *Kidney International*, *67*(95), S21-S27.

Kralova, S., Leva, L., & Toman, M. (2009). Polymorphonuclear function in naturally occurring renal failure in dogs. *Veterinární medicína*, *54*(5), 236-243.

Kumar, A. (2003). Animal physiology. Discovery Publishing House.

Lafrance, J. P., & Miller, D. R. (2010). Acute kidney injury associates with increased long-term mortality. *Journal of the American Society of Nephrology: JASN*, *21*(2), 345.

Lee, M. (Ed.). (2009). *Basic skills in interpreting laboratory data*. ASHP.

Macdougall, I., Bock, A. H., Carrera, F., Eckardt, K. U., Gaillard, C. A., Van Wyck, D. B., ... & Roger, S. D. (2016). CHRONIC KIDNEY DISEASE ANAEMIA. *Nephrology Dialysis Transplantation*, *31*(1), i193-i199.

Marshall, A. L. (2022). Williams Manual of Hematology. Mcgraw-Hill Education.

McClellan, W. M., & Powe, N. R. (2009). Introduction to the Proceedings of a Centers for Disease Control and Prevention Expert Panel Workshop: Developing a comprehensive public health strategy for preventing the development, progression, and complications of CKD. *American Journal of Kidney Diseases*, *53*(3), S1-S3.

Meri, M. A., Al-Hakeem, A. H., Al-Abeadi, R. S., & Mahdi, D. M. (2022). Study of the changes of some biochemical parameters of patients with renal failure. *Bulletin of National Institute of Health Sciences*, *140*(3), 2925-2933.

Nairz, M., Theurl, I., Wolf, D., & Weiss, G. (2016). Iron deficiency or anemia of inflammation? Differential diagnosis and mechanisms of anemia of inflammation. *Wiener Medizinische Wochenschrift*, *166*(13-14), 411-423.

Perco, P., Pleban, C., Kainz, A., Lukas, A., Mayer, G., Mayer, B., & Oberbauer, R. (2006). Protein biomarkers associated with acute renal failure and chronic kidney disease. *European journal of clinical investigation*, *36*(11), 753-763.

Prasad, G. R. (2014). Metabolic syndrome and chronic kidney disease: Current status and future directions. *World journal of nephrology*, *3*(4), 210.

Priyadarshani, W. V. D., de Namor, A. F. D., & Silva, S. R. P. (2023). Rising of a global silent killer: critical analysis of chronic kidney disease of uncertain aetiology (CKDu) worldwide and mitigation steps. *Environmental geochemistry and health*, *45*(6), 2647-2662.

Remuzzi, G. (1989). Bleeding disorders in uremia: pathophysiology and treatment. *Advances in nephrology from the Necker Hospital*, *18*, 171-186.

Saxena, R., Sharma, G., & Gulati, N. (2018). Iron-deficiency Anemia and Chronic Kidney Disease: An Overview. *World Journal of Anemia*, *2*(3and4), 85–89. https://doi.org/10.5005/jp-journals-10065-0037

Shastry, I., & Belurkar, S. (2019). The spectrum of red blood cell parameters in chronic kidney disease: A study of 300 cases. *Journal of Applied Hematology*, *10*(2), 61-66.

Somvanshi, S., Khan, N. Z., & Ahmad, M. (2012). Anemia in chronic kidney disease patients. *Clinical Queries: Nephrology*, *1*(3), 198-204.

Srinivasan, R., Fredy, I. C., Chandrashekar, S., Saravanan, J., Mohanta, G. P., & Manna, P. K. (2016). Assessment of erythropoietin for treatment of anemia in chronic kidney failure-ESRD patients. *Biomedicine & Pharmacotherapy*, *82*, 44-48.

Tyagi, N. (2023) Serum Creatinine Test: Means, Normal Range, Price, Procedure, retrieved from: https://www.hexahealth.com/blog/serum-creatinine-test

Vaidya, S. R., & Aeddula, N. R. (2022, October 24). *Chronic kidney disease*. StatPearls - NCBI Bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK535404/

Zilva, J. F., & Pannall, P. R. (1988). Clinical chemistry in diagnosis and treatment. 5th ed., Edward Arnold, London, UK.