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Original Research Article

ASSESSMENT OF COMPLETE BLOOD COUNT AND LIVER ENZYMES IN CHRONIC KIDNEY DISEASE PATIENT

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Abstract: *Chronic renal failure (CRF) poses a significant global health challenge, with chronic kidney disease (CKD) characterised by kidney damage or a glomerular filtration rate (GFR) persistently below 60 ml/min for over three months. This observational study, conducted in Faisalabad from January to July 2023, aimed to evaluate haematological variables and liver enzymes across different stages of CKD. Using non-probability purposive sampling, 144 CKD patient files and 48 healthy control files were collected, excluding specific conditions. CKD staging was determined using the Modification of Diet in Renal Disease (MDRD) algorithm, categorizing patients into mild (Group A), moderate (Group B), severe (Group C) CKD, and controls (Group D). Data analysis was performed using SPSS v23. Mean ± SD values were presented for each parameter across the four groups. Haemoglobin levels (Hb g/dl) exhibited a decreasing trend in CKD patients compared to the control group, with values of 13.7 ± 1.50 in controls, 11.7 ± 0.64 in Group 1, 9.7 ± 0.57 in Group 2, and 6.7 ± 1.5 in Group 3. Red blood cell count (RBCs per million/mm3) also decreased progressively in CKD patients compared to controls, with values of 5.48 ± 0.59, 4.62 ± 0.15, 3.57 ± 0.28, and 2.81 ± 0.23 in the respective groups. Conversely, white blood cell counts (WBC thousand/mm3) increased with CKD progression, showing values of 8406 ± 1383 in controls, 10674 ± 1006 in Group 1, 12028 ± 643.5 in Group 2, and 13564 ± 741.29 in Group 3. Similarly, platelet counts (lakh/ul) exhibited a decreasing trend in CKD patients compared to controls, with values of 324708 ± 112970, 235458 ± 63853, 144979 ± 6935.8, and 126333 ± 4768.3, respectively. Analysis of liver enzymes revealed a progressive decrease in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels as CKD advanced. AST levels (U/L) decreased from 29.104 ± 3.130 in controls to 7.5825 ± 1.543 in Group 3, while ALT levels (U/L) decreased from 48.145 ± 4.9679 in controls to 12.270 ± 3.3500 in Group 3. Our findings demonstrate that haemoglobin, red blood cell count, platelet count, and liver enzyme levels (AST and ALT) decrease as CKD progresses while white blood cell count increases. These insights into haematological and hepatic biomarkers underscore the dynamic nature of CKD pathology and may inform clinical management strategies.*

Keywords: Chronic Kidney Disease, Blood Cell Count, Liver Enzymes, Glomerular Filtration Rate

Introduction

A significant public health issue around the world is chronic renal failure (CRF) (Nahas, 2005). Damage to the kidney glomeruli on a structural and functional level that causes a progressive decline in the glomerular filtration rate is the hallmark of chronic kidney disease (CKD). The National Kidney Foundation defines chronic kidney disease (CKD) as either damage to the kidneys or a body surface area (BSA) glomerular filtration rate (GFR) of less than 60 ml/min for longer than three months (Levey et al., 2002a). Filtration occurs through clusters of capillaries known as glomeruli. Chronic renal failure (CRF) is indicated by a glomerular filtration rate (GFR) reduction to less than 60 mL/min/1.73m^2 or the presence of structural or functional renal abnormalities, even in individuals with a normal GFR (Prasad et al., 2012). One of the most significant and accurate measures of renal function is the glomerular filtration rate (De Broe & Delanaye, 2020).

According to the GFR, there are five stages of chronic kidney disease (CKD): renal damage with a standard or increased GFR (GFR >90), mildly reduced GFR (GFR between 60 and 89), moderately reduced GFR (GFR between 30 and 59), severely reduced GFR (GFR between 15 and 29), and renal failure (dialysis) with a GFR less than 15 (Levey et al., 2002b).

Kidney disease (CKD) is one of the primary causes of death and disability worldwide. CKD was the 27th most significant cause of death in 1990, but it grew to become the 18th leading cause of death in 2010 (Jha et al., 2013). Around 1 million people died in 2013 as a result of CKD-related causes (Abubakar, Tillmann, & Banerjee, 2015). Despite being a worldwide problem, CKD disproportionately affects people in developing countries. According to a 2015 systematic analysis, 109.9 million people in high-income countries had CKD (men: 48.3 million, women: 61.7 million), while 387.5 million lived in lower-middle-income countries (men: 177.4 million, women: 210.1 million) (Mills et al., 2015).

The prevalence of CKD is increasing in developing countries, including Pakistan (Alebiosu, Ayodele, & disease, 2005). It is compounded by a lack of community awareness, a disproportionately more significant burden of

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known CKD risk factors, and limited access to renal replacement therapy (Martins et al., 2006). The total prevalence of CKD in Pakistani adults was 21.2%. According to high-quality studies, the most significant CKD prevalence in Pakistan was 29.9% (Jafar, Schmid, & Levey, 2005), while the lowest was 12.5% (Jessani, Bux, & Jafar, 2014).

Even though each trial included people of similar ages, using different equations to define CKD could explain the disparity. Prevalence by age group: Only Alam et al. found an age-specific prevalence of CKD among Pakistani studies. The study discovered the highest incidence of CKD among senior participants over the age of 50 (43.6%) and the lowest prevalence among younger participants under the age of 30 (10.5%) (Alam, Amanullah, Baig-Ansari, Lotia-Farrukh, & Khan, 2014). Gender-specific prevalence: The gender-specific prevalence of CKD was identified in all four Pakistani investigations (Alam et al., 2014; Imran et al., 2015; Jafar et al., 2005; Jessani et al., 2014). Although Alam et al. and Imran et al. found that men had a greater frequency of CKD (Alam et al., 2014; Imran et al., 2015), Jessani et al. and Jafar et al. found that women had CKD more frequently than men (Jafar et al., 2005; Jessani et al., 2014). In a high-quality study that employed a country-specific equation to determine CKD, female participants had a slightly more significant proportion than male participants (male-11.6%, female-13.3%) (Jessani et al., 2014). The incidence and prevalence of CKD vary by ethnicity, social variables, and genetic factors. CKD is expected to impact 10% of the general population and more than 50% of the high-risk group (C. George, T. E. Matsha, R. T. Erasmus, & A. P. Kengne, 2018a; Webster, Nagler, Morton, & Masson, 2017).

Chronic renal disease is a significant, life-threatening medical condition. It is also known as chronic kidney failure since it is a process of permanent nephron loss that eventually leads to renal failure (De Broe & Delanaye, 2020). CKD is caused by a combination of factors and chronic diseases, including hypertension, diabetes, hypercholesterolemia, ageing, smoking, obesity, urinary infections, glomerulonephritis, pyelonephritis, polycystic kidney disease, urinary tract obstruction, renal stones, chronic use of particular drugs (lithium, NSAIDs, opium), and severe renal trauma (Chen, Knicely, & Grams, 2019; George et al., 2018a; HADIAN, ANBARI, & HEIDARI, 2014; Lu et al., 2017; Pizzorno, 2015; Webster et al., 2017; Zhang, Zhang, Li, & He, 2015).

The kidneys' primary functions are to regulate blood parameters and eliminate waste from the circulation. The kidneys also play a vital role in maintaining electrolyte balance and water levels. Another role of the kidneys is to create hormones such as erythropoietin and vitamin D, which, while not hormones, behave similarly in the human body (De Broe & Delanaye, 2020). The kidneys generate erythropoietin to stimulate bone marrow synthesis of red blood cells. During severe kidney impairment, erythropoietin levels fall, resulting in a drop in RBC levels. Following CKD, hematologic parameters, particularly RBC, are the most affected markers (C. George, T. E. Matsha, R. T. Erasmus, & A. P. J. B. O. Kengne, 2018b).

Liver enzymes such as ALT, AST, and GGT indicate hepatocyte aggressiveness. However, in CKD patients with renal failure receiving hemodialysis, serum levels of liver enzymes can be low. Such patients could be misdiagnosed with liver disease (Kheradmand et al., 2019; Sette & de Almeida Lopes, 2014). Lower pyridoxine serum levels, greater homocysteine levels, and hemodilution due to fluid retention are linked to lower AST and ALT serum levels in patients with CKD (Lopes et al., 2009). Other factors that contribute to the decrease in aminotransferase in HD patients infected with the hepatitis B (HBV) or hepatitis C viruses (HCV) include lower viremia caused by dialysis through virus sequestration by the dialyser, increased production of hepatocyte growth factor (HGF), which is induced via dialysis and accelerates liver regeneration; and increased endogenous interferon serum levels and lymphocyte activation (Maia et al., 2009). Indeed, AST and ALT serum levels' behaviour during CKD development is poorly understood (Moosazadeh, Espahbodi, Afshari, & Eslami, 2023).

Numerous haematological and biochemical markers can alter as a result of kidney disorders. Haematological and biochemical profiles are frequently impacted by chronic kidney disease (CKD). As the illness worsens, this becomes more evident, further exacerbating the patient's condition and increasing their risk of cardiovascular problems (Habib, Ahmad, & Rehman, 2017). This study aims to assess the haematological parameters and liver enzymes in individuals with chronic kidney disease (CKD).

Methodology

This study aimed to observe chronic kidney disease (CKD) patients and was conducted in the Nephrology departments of Allied Hospitals 1 and 2 in Faisalabad from January 2023 to July 2023. The researchers used non-probability purposive sampling, where they preferred convenience sampling over random sampling. They collected 144 files of CKD patients who attended the nephrology outpatient department (OPD) and 48 files of healthy controls who attended the hospital for routine health check-ups during the same period.

Participants with diagnosed aplastic anaemia, liver disease, haematological malignancy, a history of erythropoietin hormone therapy, and blood transfusion during the last three months were excluded from the study. Data such as age, sex, serum creatinine, haematological parameters, and liver enzymes were collected from all the files. Haematological and serum parameters were measured using the hospital laboratory's automated haematology and routine chemistry analyser. CKD was diagnosed based on the glomerular filtration rate (GFR), estimated using the MDRD algorithm (Levey et al., 2002b). The formula used was: GFR = 186 × (Scr)-1.154× (age)-0.203× (0.742 if female) × (1.212 if black). Based on GFR, all the collected patient files were divided into four groups: Group A (mild CKD), Group B (moderate CKD), Group C (severe CKD) and Group D (control group). Patients in Group A and B did not need dialysis, while patients in Group C needed dialysis. There were 48 patient files in each group. The researchers used SPSS version 23 to handle and analyse the data.

Results

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This study aimed to assess patients' complete blood count and liver enzymes in different stages of chronic kidney disease. The study involved 192 participants with comparable gender and age distribution. The complete blood count assessment included haemoglobin (Hb), red blood cells (RBCs), white blood cells (WBCs), and platelets. The liver enzyme assessment included AST and ALT. The means and standard deviations of haematological parameters and serum aminotransferase levels were calculated in all four groups, and the mean of different parameters in each group was compared.

The haemoglobin level in the control group was 13.7±1.50. In group 1, the haemoglobin level was 11.7±0.64; in group 2, it was 9.7±0.57; in group 3, it was 6.7 ± 1.5. The haemoglobin level was found to be lower in CKD patients than in the control group. The red blood cell count in the control group was 5.48 ± 0.59; in group 1, it was 4.62 ± 0.15; in group 2, it was 3.57 ± 0.28; and in group 3, it was 2.81 ± 0.23. The red blood cell count decreased in chronic kidney disease patients compared to the control group.

The white blood cell count in the control group was 8406 ± 1383; in group 1, it was 10674 ± 1006; in group 2, it was 12028 ± 643.5; and in group 3, it was 13564 ± 741.29. The white blood cell count increased in chronic kidney disease patients compared to the control group. The platelet count in the control group was 324708 ± 112970; in group 1, it was 235458 ± 63853; in group 2, it was 144979 ± 6935.8; and in group 3, it was 126333 ± 4768.3. The platelet count decreased in chronic kidney disease patients compared to the control group. (Table 1)

Table 1: Distribution of blood components in different groups of the study population

Parameter	Control	Group 1	Group 2	Group 3
Haemoglobin (g/dl)	13.7 ± 1.50	11.7 ± 0.64	9.7 ± 0.57	6.7 ± 1.5
Red Blood Cell Count (million/µL)	5.48 ± 0.59	4.62 ± 0.15	3.57 ± 0.28	2.81 ± 0.23
White Blood Cell Count (cells/µL)	8406 ± 1383	10674 ± 1006	12028 ± 643.5	13564 ± 741.29
Platelet Count (cells/µL)	324708 ± 112970	235458 ± 63853	144979 ± 6935.8	126333 ± 4768.3

The level of AST enzyme in the control group (mean ± SD, 29.104 ± 3.130). The level of AST enzyme in group 1 (mean ± SD, 18.479 ± 1.031). The level of AST enzyme in group 2 (mean ± SD, 12.812 ± 1.315). The level of AST enzyme in group 3 (mean ± SD, 7.5825 ± 1.543). The level of AST enzyme decreased in chronic kidney disease patients compared to the control group. (Figure 1)

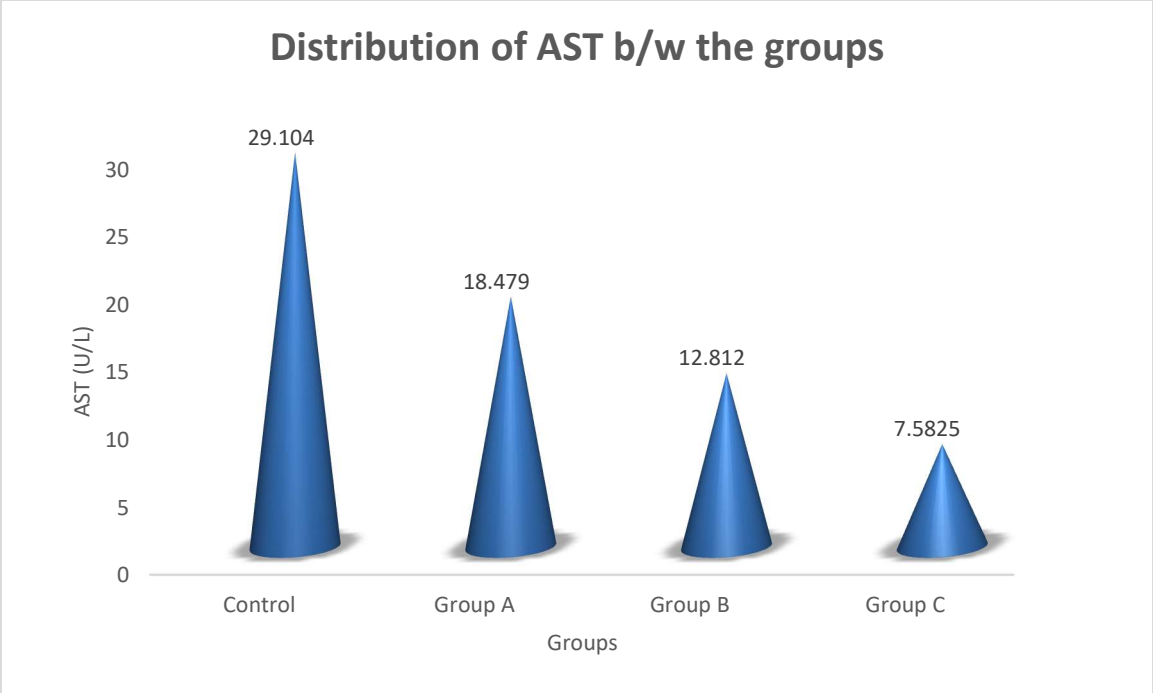

Figure 1: Distribution of AST levels between the groups of the study population

The level of the ALT enzyme in the control group (mean ± SD, 48.145 ± 4.9679). The level of the ALT enzyme in group 1 (mean ± SD, 33 ± 2.6174). The level of the ALT enzyme in group 2 (mean ± SD, 24.187 ± 3.3047). The level of ALT enzyme in group 3 (mean ± SD, 12.270 ± 3.3500). The level of ALT enzyme decreased in chronic kidney disease patients compared to the control group. (Figure 2)

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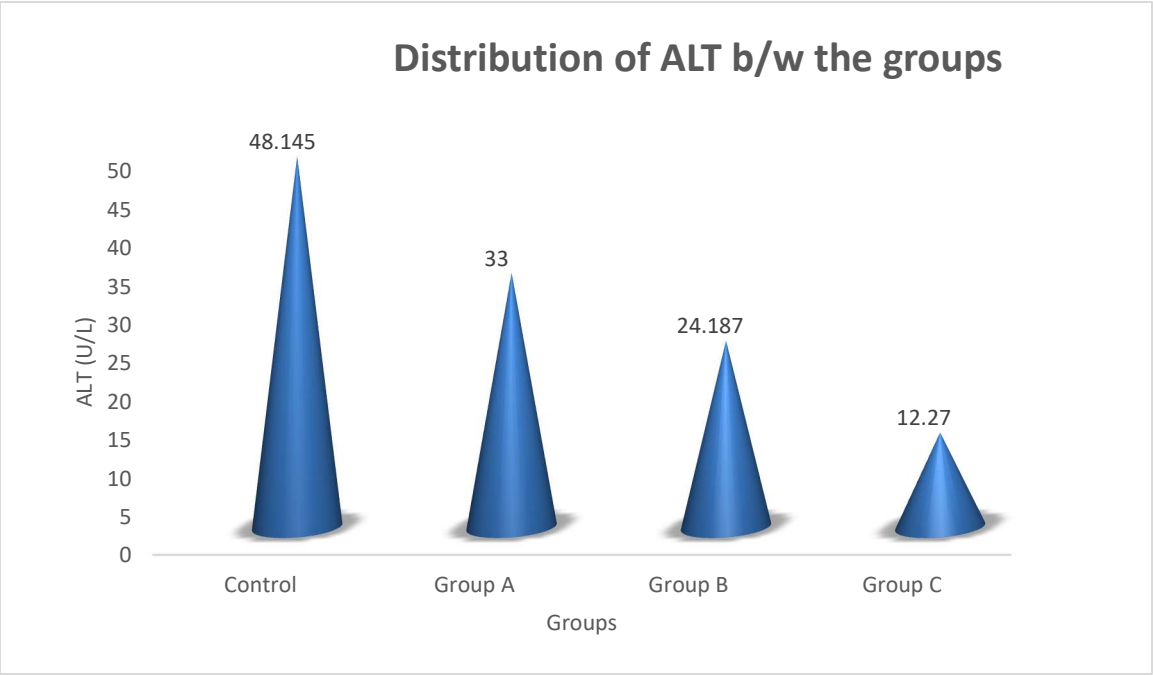

Figure 2: Distribution of ALT levels between the groups of the study population

The average levels of haemoglobin (HB), red blood cells (RBCs), platelets, and liver enzymes are lower in mild, moderate, and severe chronic kidney disease (CKD) patients compared to healthy individuals. On the other hand, the mean white blood cell (WBC) count is higher in mild, moderate, and severe CKD patients than in healthy individuals. As the disease progresses, the levels of HB, RBC count, platelet count, and liver enzymes (AST and ALT) continue to decrease while the WBC counts increase.

Discussion

The current epidemiological investigation aimed to assess patients' complete blood count and liver enzymes in different stages of chronic renal disease. The current study found that as the disease advances, the levels of haematological parameters (HB, RBC, and platelet count) and liver enzymes (AST and ALT) decline while the WBC count rises. This finding is in agreement with other authors' work (Chandrasekhar).

Patients with chronic renal disease have reduced levels of haemoglobin, red blood cells, and platelets. These findings are consistent with those of Suresh et al., Naghmi Asif et al., and Afshan Zeeshan Wasti et al., who found decreased RBC counts, Hb, and hematocrit values in CKD patients. Suresh M. et al. showed that individuals with chronic renal failure have reduced haematological indices due to poor erythropoietin production and other variables such as increased hemolysis, suppression of bone marrow erythropoiesis, hematuria, and gastrointestinal blood loss. All haematological markers have a negative connection with serum creatinine levels. The extent of the modifications is determined by the severity of the renal failure (Suresh et al., 2012).

Reduced synthesis of erythropoietin, together with other variables that inhibit marrow erythropoiesis and reduce red cell survival, is the leading cause of the decline in these red blood cell characteristics (Hsu, Bates, Kuperman, & Curhan, 2001; Katz, 2005; Locatelli, Pozzoni, Vecchio, & management, 2007). The hormone erythropoietin controls the synthesis of red blood cells and preserves their viability by delaying DNA cleavage. Without erythropoietin, DNA cleavage occurs rapidly, resulting in cell death. In CKD, due to impaired production of erythropoietin and death of red cells, Hb and Hct concentrations fall, which is seen even in individuals with mild to severe renal insufficiency (Gallagher, Glader, & Wilkins, 2009). The most prevalent, reliable, and severe type of the several haematological disorders is anaemia (BROWN & Roth, 1922). There is a substantial association between the occurrence of anaemia and the severity of CKD, even though anaemia can be seen at different stages of the disease (McClellan et al., 2004).

Haematological markers are reported to be decreased in CKD. Erythrocyte indices are the most frequently impacted. Most erythropoietin is synthesised in the juxtaglomerular apparatus, except 10% in the liver and other organs. Red blood cell (RBC) indices can be affected by reduced erythropoietin levels as well as vitamin B12, iron, and folic acid shortages, which can result from diet inadequacies, blood loss, or shortened erythrocyte life spans (Eschbach Jr et al., 1967; Locatelli et al., 2007).

Platelet count was lower in chronic renal failure patients, according to Yassein et al. (Yassein et al., 2016). Erythropoietin enhances the effects of megakaryocyte colony-stimulating factors (PAF-AH) and paraoxonase (PON1). Platelet count decreases in chronic renal illness due to reduced erythropoietin production (Gouva et al., 2006). The finding of erythropoietin receptors in megakaryocytes is reasonable since erythropoietin levels can alter platelet levels. Because erythropoietin and thrombopoietin have substantial similarities, erythropoietin acts as the principal humoral regulator of platelet mass. Thrombocytopenia is

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regarded as a consequence of hemodialysis. Patients undergoing hemodialysis or predialysis typically have lower platelet counts (Gafter et al., 1987). However, it is uncommon among patients receiving hemodialysis with biocompatible membranes (Katz, 2005). Numerous investigations revealed that patients with CKD had a statistically significant decrease in their platelet count (Alghythan & Alsaeed, 2012; Suresh et al., 2012; Yassein et al., 2016).

A recent study showed increased WBC count in patients with chronic kidney disease. WBC counts rise as a result of inflammation-induced immune system activation (Turkmen, Guney, Yerlikaya, & Tonbul, 2012); this indicates the potential use of WBC indices as a proxy indicator of inflammation in chronic kidney disease (CKD) (Okyay et al., 2013). TNF-α and IL-6, two cytokines present in blood and contribute to chronic inflammation in the uremic state, cause an inflammatory response marked by a prominent presence of pro-inflammatory cytokines. In our study, the count of white blood cells (WBCs) was slightly elevated in CRF patients, and the dialysis process further increased WBC count (Tbahriti et al., 2013).

In our study, serum aminotransferase levels decreased in patients with chronic kidney disease. According to specific research, serum AST and ALT levels in CKD patients without ESRD and CKD patients with ESRD were significantly lower than in controls. Furthermore, CKD patients with ESRD had significantly lower levels of these two enzymes than those without ESRD. Several recent investigations have also shown that serum ALT levels in CKD patients are lower than in those with normal renal function (Al-Wakeel et al., 1996; Cotler et al., 2002; Hung et al., 1997; Lopes et al., 2009; Trevizoli et al., 2008; Yuki et al., 2000). Even though fewer authors have discussed AST levels in CKD patients, the results of those studies also revealed a lower AST level in CKD patients compared to controls (Mustafa, Al-Abachi, & Khalaf, 2008; Yasuda et al., 1995). Furthermore, an Italian study found lower AST and ALT levels in dialysis patients compared to predialysis patients with CKD and lower levels of aminotransferases in CKD compared to healthy individuals (Fabrizi et al., 2001). Our findings broadly agree with those of the studies cited above. Only a few studies, meanwhile, have combined the analysis of AST and ALT levels.

The pathophysiological cause for this decrease in serum aminotransferase levels in CKD patients is unknown. Possible explanations include a decrease in pyridoxal-5-phosphate, an aminotransferase coenzyme, ultraviolet-absorbing materials, and high amounts of uremic toxins. Other possibilities include reduced synthesis and inhibition of AST and ALT release from hepatocytes, as well as faster clearance from serum (Fabrizi et al., 2001; Mustafa et al., 2008; K. Ono, T. Ono, & T. J. C. n. Matsumata, 1995b; Rej, Fasce Jr, & Vanderlinde, 1973; Wolf, Williams, Coplon, & Coulson, 1972). A low serum aminotransferase level in CKD patients could be attributable to water retention and hemodilution (Lopes et al., 2009).

Several hypotheses have been proposed to explain the reason why serum aminotransferase levels are lower in CKD patients with HD, including pyridoxine insufficiency, hemodilution, and hyperhomocysteinemia (Badalamenti et al., 2003; Lopes et al., 2009; Ono et al., 1995b). Ono et al. found that pyridoxine-deficient CKD patients with HD had lower blood aminotransferase levels than non-pyridoxine-deficient individuals (K. Ono, T. Ono, & T. Matsumata, 1995a).

The main limitations of the current investigation were the reduced sample size and the absence of assessments of ALP and GGT enzymes in individuals suffering from chronic renal disease. Future research should gather larger samples of CKD patients to analyse ALP and GGT enzymes and determine which factors contribute to ALP and GGT change.

Conclusion

As the disease progresses, the Hb, RBC count, platelet count, and liver enzymes (AST and ALT) decrease, but the WBC count increases. In CKD patients, it is imperative to assess these markers regularly. The morbidity and mortality associated with CKD decrease when these anomalies are corrected.

Declarations

Data Availability statement
All data generated or analyzed during the study are included in the manuscript.
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Conflict of interest

The authors declared absence of conflict of interest.

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