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The Correlation Between Kidney Function Tests and Complete Blood Count in Iraqi Patients with Chronic Hypertension

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Abstract

Hypertension, also known as the silent killer, is a condition that typically remains asymptomatic until it causes significant harm to the cardiovascular system. Unmanaged hypertension can lead to renal failure, myocardial infarction, and even mortality. Kidney and blood pressure (BP) have a significant correlation with renal failure, particularly renal disease, leads to an increase in blood pressure. On the other hand, high blood pressure worsens the decrease in kidney function in those with kidney disease. This study aims to examine the correlation between kidney function tests (KFTs) and complete blood count (CBC) in Iraqi patients with chronic hypertension. The average disparity in kidney function tests (KFTs) between individuals with chronic hypertension and the control group. Urea, creatinine, and uric acid showed statistically significant increases ($p<0.01$). The findings indicated a noteworthy reduction ($p<0.01$) in HGB, HCT, MCH, and MCV concentrations among individuals with chronic hypertension. There is a strong and statistically significant association ($p<0.01$) between the average increase in urea and creatinine levels and the average reduction in MCH and MCV levels. Additionally, a significant correlation ($p<0.01$) exists between the uric acid level and deficiencies in HGB, HCT, MCH, and MCV. However, no statistically significant connection ($p>0.01$) was seen between HGB and HCT levels and urea and creatinine levels. Conclusions: Hypertension individuals with low levels of HGB, HCT, MCH, and MCV tend to have elevated levels of urea, creatinine, and uric acid. Thus, anemia may be advised for people suffering from hypertension and renal illness.

Keywords: Erythropoietin, Complete Blood Count, Hypertension and Kidney Functions

Introduction

Researchers recommend diagnosing hypertension when a patient's systolic blood pressure (SBP) in a medical environment is 140 mm Hg or higher, and their diastolic blood pressure (DBP) is 90 mm Hg or higher following many exams, according to a major study of Muntner et al. (2019). It is fairly uncommon for people to have hypertension and not notice any symptoms. However, more testing is necessary to rule out secondary hypertension or problems associated with hypertension if certain symptoms are present. You should gather all the necessary information about your health and family history, including: A personal history of cardiovascular disease (CVD) including myocardial infarction (MI), heart failure (HF), stroke (TIA), diabetes, and dyslipidemia is part of the individual's medical background.

According to Williams et al. (2018), additional crucial characteristics include whether or not they smoke, their eating habits, how much alcohol they drink, how active they are, psychological issues, and whether or not they have a history of depression or chronic kidney disease (CKD). CKD is a non-communicable disease (NCD) that accounts for 41 million deaths per year, or 71% of all deaths in the world, according to the World Health Organization (Balasubramaniyan et al., 2020). The kidney is essential for regulating the volume of bodily fluids, maintaining fluid osmolality, managing acid-base balance, controlling electrolyte concentrations, eliminating toxins, converting a precursor of vitamin D into calcitriol, and producing erythropoietin hormone in response to tissue-level hypoxia (Lv & Zhang, 2019). The molecular weight of human erythropoietin is 30.4 kilo Dalton. The production of this substance occurs in the kidney, namely by interstitial fibroblasts, which are closely associated with the peritubular capillary and tubular epithelial tubule.

Erythropoietin serves a vital role as a hormone necessary for producing red blood cells. Erythropoietin primarily affects the progenitors and precursors of red blood cells by promoting their lifespan through the prevention of apoptosis. Various disease occurrences adversely impact the kidney, the principal site for producing erythropoietin. Examples of such ailments are hypertension, diabetes, and HIV/AIDS (Ifeanyi & Uzoma, 2016). Erythropoiesis is the process by which the 1% of red blood cells (RBCs) lost daily are constantly replaced. Erythropoiesis is a highly controlled process that entails the growth of hematopoietic stem cells, which then develop into adult red blood cells through specific stages of commitment, proliferation, and differentiation.

Healthy adult humans generate roughly 2 million red blood cells (RBCs) each second, and these RBCs circulate throughout the body for approximately 120 days. Regulation of red blood cells (RBC) ensures tissue oxygen delivery (Palis, 2014). Haemoglobin (HGB), the main protein in red blood cells (RBCs), transports oxygen and carbon dioxide. Diagnostic tests for anemia include HGB, HCT, MCH, and MCV. These measures reveal the proportion of red blood cells, their average hemoglobin content, and their average size. Various clinical circumstances link these determinations to mortality (Brzeźniakiewicz-Janus et al., 2020). Chronic renal failure often causes erythropoietin deficiency and renal anaemia. With varying degrees of renal impairment, chronic kidney disease (CKD) often causes renal anemia. CKD risk factors include genetic or sociodemographic susceptibility and illnesses like diabetes and hypertension that can cause and propagate renal disease (Draft Background & Scope, 2020). Clinical examination and laboratory studies are essential for renal function measurement. A patient's first assessment may include a complete blood count (CBC), which measures HGB, HCT, MCH, and MCV. We will also investigate the link between these metrics and blood urea, creatinine, and uric acid.

Methods

The current study was conducted at a laboratory affiliated with a private physician's clinic in Baghdad, Iraq, from December 2022 to March 2023. The data was obtained from 60 patients who were diagnosed with hypertension using an analogue sphygmomanometer and stethoscope, with a blood pressure reading of greater than 140/90. These patients were followed up for a chronic hypertension study. The average age of these patients was 61.31 ±8.77 years. Additionally, 60 healthy volunteers acted as a control group, with an average age of 61.95±9.76 years. In order to mitigate the influence of potential confounding variables, we removed 30 individuals who had diabetes mellitus, liver disorders, and any other chronic illnesses.

Analysis of samples

Specimen collection

Blood samples were collected from patients and control individuals using a disposable syringe, with each sample being approximately 5 mL in volume. 4 milliliters of blood were transferred into gel tubes. Following centrifugation at a force of 1500 times, the acceleration due to gravity is for 10 minutes, and following centrifugation at a force of 1500 times, the acceleration due to gravity is for a duration of 10 minutes. The sera were extracted and preserved to measure blood urea, creatinine, and uric acid concentrations. The remaining blood was collected in EDTA tubes for CBC analysis.

Laboratory assessments

The COBAS c 111 (Roche/USA) analyzer was used to measure the serum concentration of blood urea, creatinine, and uric acid levels. The appropriate reagent on a COBAS INTEGRA 400 analyzer was utilized. The haematology analyzer automatically analyzed the complete blood count (CBC) and indices (Swelab et al. analyzer. Sweden).

Statistical analysis

Presenting data as mean ± SD. The statistical analysis utilized the LSD technique with a significance level of $p<0.05$. Statisticians used US-developed SPSS 13 for Windows. Compare means using the least significant difference (LSD) approach in one-way analysis of variance (ANOVA).

Results and Discussion

Table 1 presents the average disparity in renal function tests between patients with chronic hypertension and the control group. Urea, creatinine, and uric acid showed statistically significant increases ($p<0.01$). The findings demonstrated a very substantial reduction ($p<0.01$) in HGB, HCT, MCH, and MCV levels among patients with chronic hypertension, as depicted in Table 2. There is a strong and statistically significant association ($p<0.01$) between the average increase in urea and creatinine levels and the average reduction in MCH and MCV levels. Additionally, a significant correlation ($p<0.01$) exists between the uric acid level and deficiencies in HGB, HCT, MCH, and MCV. However, the levels of HGB and HCT did not exhibit a significant connection ($p>0.01$) with urea and creatinine levels, as seen in Table 3. Figure 1 shows a strong and statistically significant connection ($p< 0.01$) between renal KFTs and CBC.

Hypertension has two types: main and secondary. Ninety-five percent of hypertension cases are primary, whereas subsequent illnesses like CKD cause secondary hypertension. Most CKD patients have chronic hypertension, which contributes to its development and progression. Excellent health reduces nocturnal blood pressure by 10% to 20%. Salt excretion, the renin-angiotensin-aldosterone system, and diurnal autonomic function affect this process. Chronic kidney disease (CKD) patients with abnormal nighttime blood pressure are more likely to develop cardiovascular disease (CVD) and die from CKD. Kidney diseases affect 850 million individuals worldwide. About 10% of people worldwide have Chronic Kidney Disease (CKD), which is permanent and progresses. Due to its growing worldwide burden, predict that Chronic Kidney Disease (CKD) will be the fifth leading cause of global years lost by 2040 (Kam-Tao et al., 2020). Our study found significantly higher levels of urea, creatinine, and uric acid ($P<0.01$) in the chronic hypertension group compared to the control group.

These results match those of Isra'a Hamdani in 2010 and Marcel Tangyi Tamanji in 2017 (Isra'a, 2010; Tamanji et al., 2017). Uric acid may cause hypertension, according to Sahu et al. (2011). Uric acid increases salt sensitivity and vascular smooth muscle development, which may induce hypertension. However, multiple studies have found that uric acid increases do not indicate hypertension (Manjareeka & Nanda, 2013). Full blood count results show significant decreases ($P<0.01$) in HGB, HCT, MCH, and MCV levels in hypertensive patients compared to healthy controls. Gebrie et al. (2018) research supports this finding. Microcytic hypochromic and iron deficient anemia are characterized by low MCV and MCH. A study by Mirsad Panjeta linked chronic kidney disease patients' anemia severity to renal failure (Panjeta et al., 2015). In 2016, Divya *et al*. found a statistically significant reduction in hypertensive patient hematocrit (HCT) levels with a p-value of less than 0.01.

HCT increases whole blood viscosity during hypertension, which may explain the relationship. This may cause peripheral vasoconstriction and hypertension. HCT and HGB levels are higher in hypertensive patients than in non-hypertensives, according to Saudi Arabia, Brazil, and India studies (Divya & Ashok, 2016). A p-value of less than 0.01 indicated statistical significance. Our data matches Mcadams-Demarco, 2012, which found a significant hemoglobin reduction in chronic renal failure individuals with hyperuricemia (McAdams-DeMarco et al., 2012). However, Eun et al. (2019) observed that hyperuricemia patients without chronic renal failure had normal hemoglobin levels in 2019.

The study found a significant ($P<0.01$) positive correlation between uric acid levels and hematological parameters, including hemoglobin, hematocrit, MCH, and MCV, in hypertension patients. The connection between HGB and HCT and urea and creatinine levels was not significant ($p>0.01$). Wastiet et al. observed that renal disease patients had significantly lower RBC, Hb, and packed cell volume levels in 2013 (Wasti et al., 2013). Furthermore, the study found a substantial decrease in MCH and MCHC indices. Apart from studies that explicitly show a positive association between hematocrit (HCT) and serum uric acid, additional research, including screen-based cohorts, shows a positive correlation (Zeng et al., 2015). Uric acid is crucial to hypertension and renal dysfunction. Uric acid affects the kidney's afferent arterioles, causing elevated blood pressure independent of crystal formation or pressure. Reduced renal blood flow, ischemia, and renovascular hypertension can result from afferent arteriol (Kiuchi et al., 2020).

Conclusion

Hypertension individuals with low levels of HGB, HCT, MCH, and MCV tend to have elevated levels of urea, creatinine, and uric acid. Therefore, individuals with hypertension and renal disease may be advised to undergo anemia testing in order to identify the presence of coronary vascular disease. Managing hypertension is crucial in those with chronic renal disease as it not only slows down the course of the disease but also decreases the likelihood of detecting the risk of coronary vascular disease.

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