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Research Article

Assessment of Some Homocystein Levels in Cardiovascular, Kidney Disease and Prostate Cancer Patients Attending Federal Teaching Hospital, Owerri

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Abstract
Cardiovascular disease, prostate cancer and renal disease are heavy burden in the society. This study was carried out to assess the haematological parameters, homocystein, some 'B' complex vitamins in cardiovascular disease, kidney disease and prostate cancer disease subjects at federal teaching hospital owerri, Imo state, Nigeria. A total of 200 subjects were recruited for the study out of which 140 were people suffering from cardiovascular disease, kidney disease and prostate cancer while 60 were apparently healthy control subjects. The levels of homocysteine, haematological parameters and some 'B' vitamins and folic acid levels were determined by Eliza, while full blood count was assayed by sysmex KY2IN haematology analyzer. The data generated from these researches were analyzed using SPSS statistical software version 20.0. The result showed that homocysteine was significantly increased in KD (P= 0.001) with their mean ± SD values 25.08 ±/9.06 ±µmol/l, 22.86 ±/10.12 ± µmol/l and 22.7±/4.44 µmol/l respectively when compared with the controls. There were no significant gender based concentration values observed in all other parameters measured in the various diseases under study. These levels did not differ significantly between sexes. From the findings, the inclusion of homocystein in the routine laboratory investigations of conditions related to cardiovascular disease, kidney disease and prostate cancer will be useful in the prediction, management and risk assessment of these diseases and may subsequently improve treatment outcome.

Keywords: Homocystein, cardiovascular, kidney disease, prostate cancer, patients, Owerri.

Introduction
Cardiovascular disease (CVD) is any illness that affects the heart or blood vessels. It is divided into four groups: Coronary artery disease (CAD) or coronary heart disease (CHD). This happens when the heart doesn't get enough blood flow, which can lead to angina, a heart attack (MI), or heart failure. It makes up between one-third and one-half of all instances of CVD. Cerebrovascular disease (CVD): It includes stroke, which can be either ischaemic or hemorrhagic. Peripheral artery disease (PAD), especially arterial disease affecting the extremities that can lead to claudication [1]. Cardiovascular disease (CVD) is the leading cause of mortality worldwide. The main goal of primary prevention is to stop atherosclerosis from getting worse. Renal disease, or kidney disease, is a group of disorders that make it hard for the kidneys to do their job. The kidneys are very important organs that filter waste from the blood, keep the balance of electrolytes, control blood pressure, and make hormones that help make red blood cells and keep bones healthy. When the kidneys don't work right, waste products can build up in the body and fluids can go out of balance, which can cause a number of health problems [2].

Acute Kidney Injury (AKI) is marked by an abrupt decline in renal function, frequently resulting from severe illness, dehydration, toxic exposure, or urinary tract obstruction. If this problem is found and treated quickly, it may be possible to reverse it. Chronic kidney disease (KD) is the slow loss of kidney function over time, usually because of other health problems including diabetes, high blood pressure, or glomerulonephritis. The glomerular filtration rate (GFR) is used to stage chronic kidney disease (CKD). The stages range from mild (Stage 1) to kidney failure (Stage 5), which is also called end-stage renal disease (ESRD). [3] Glomerulonephritis: This category of illnesses causes the glomeruli, which are the small filters in the kidneys, to become inflamed. Infections, autoimmune illnesses, or other things that make the immune system work can cause it. If not treated properly, glomerulonephritis can cause CKD. Polycystic Kidney Disease (PKD) is a hereditary condition marked by the proliferation of many cysts in the kidneys, potentially resulting in renal enlargement and diminished functionality over time. There are two primary kinds of PKD: autosomal dominant PKD (ADPKD) and autosomal recessive PKD (ARPKD). [4]

Diabetic nephropathy is a common consequence of diabetes, especially in people who have trouble keeping their blood sugar levels in check. It damages the blood vessels and filtration system in the kidneys because of high blood sugar levels that last for a long time. If not treated properly, this can develop to CKD and then ESRD. Hypertensive Nephrosclerosis is a disorder that happens when high blood pressure damages the blood arteries in the kidneys. This causes the tissues in the kidneys to harden and scar. This is a major reason for CKD and ESRD. [5] Prostate cancer, or carcinoma of the prostate, is the emergence of malignancy inside the male reproductive system (National Cancer Institute, 2014). As of 2012, prostate cancer is the second most often diagnosed cancer, accounting for 15% of all male cancers, and it ranks as the sixth greatest cause of cancer mortality in males globally [6]. Common symptoms include needing to urinate a lot, needing to urinate at night, blood in the urine, painful urination, and problems with urination. Advanced prostate cancer can metastasise to several anatomical sites, including the bones, spine, pelvis, and ribs, resulting in lower extremity weakness and urine and faecal incontinence [7]. Homocystein is a sulfur-containing, non-protein, hazardous amino acid present in the interconversion pathway of methionine and cysteine. Homocystein undergoes metabolism through two distinct pathways: remethylation and trans-sulfuration.

Cardiovascular disease, prostate cancer, and kidney disease (KD) impose a significant burden on society. More than 750 million people around the world have these disorders [8]. There are a lot of studies on the homocysteine parameter and its link to cardiovascular disease (CVD), kidney disease (KD), and prostate cancer, but not many have been published in this region of the country. High levels of homocystein (hyperhomocysteinemia) are linked to a higher risk of a number of disorders, such as kidney disease, cardiovascular disease, and prostate cancer. It is also connected to other illnesses, such as autoimmune disorders, neurological diseases, and several malignancies. Comprehending homocysteine metabolism and its association with cardiovascular disease, kidney disease, and prostate cancer can facilitate prevention, diagnostic, and treatment options. It is on this assumption that the examination of homocysteine parameters related with these disorders becomes required [9]. Cardiovascular disease, kidney disease, and prostate cancer represent significant public health issues and worldwide health burdens, characterised by elevated morbidity and mortality rates [10]. These disorders frequently exhibit common risk factors such as inflammation, oxidative stress, and metabolic abnormalities. This lack of understanding makes it harder to find problems early, figure out who is at risk, and give the right nutrition or therapy. In areas with limited resources and few advanced diagnostic tools, cost-effective biomarker profiling could greatly enhance patient treatment and outcomes. Consequently, there exists an urgent necessity to examine the patterns and interrelationships of homocysteine in persons afflicted with cardiovascular disease, kidney illness, and prostate cancer, and to juxtapose these findings with those from healthy populations. This will help us figure out if these markers may be used as early signs of illness risk, progression, or prognosis.

Materials And Methods

Study Area

The study was carried out at Federal teaching hospital (FETHO), Owerri, Imo state, Nigeria. Owerri is located in the South East of Nigeria.

Ethical approval

The ethical approval was obtained from the ethical committee of Federal teaching hospital, Owerri.

Study Design

A cohort study design was used to study the homocystein levels in cardiovascular disease, renal disease and prostate cancer disease subjects respectively.

Study Population

The populations of the study were people with cardiovascular disease, kidney disease and prostate cancer, attending Federal teaching hospital owerri (FETHO). Control subjects were apparently healthy individuals, who met the inclusion criteria.

Selection criteria

Inclusion criteria

Those included in the study were:

i) Male and female subjects with cardiovascular disease
ii) Men of all ages that have cancer of the prostate
iii) Male and female with kidney disease (KD)
iv) Male and female that are apparently healthy without cardiovascular disease and kidney disease or cancer
v) Men that are apparently healthy (not diagnosed of prostate cancer) (control subjects)

Exclusion criteria

i) Men that have undergone prostate surgery
ii) Patients diagnosed of malignant disease other than prostate cancer.
iii) (III) Male and female that have been on routine vitB_6, VitB_{12} and folate for more than one month

Sample collection and preparation

Samples were collected from two hundred (200) subjects who met the inclusion criteria and also consented to the study. Sixty of these subjects were the healthy control whereas one hundred and forty (140) subjects were the test group. Questionnaire were administered in the form interviews to obtain demographic data and other subject's history (Appendix VI).

Laboratory Procedures

All reagents were commercially purchased and the manufacturer's standard operating procedures were strictly adhered to.

Determination of Homocystein (HCY) by Eliza modified by biolab diagnostic Ref no: HCY 0425-A, Batch No: HCY0425-01

Principle:

Total homocystein eliza kit employs the competitive enzyme immunoassaaay technique for the quantitative measurement of total homocystein in serum. The 96-well microplate has been pre-coated with total homocystein antigen. During the incubation, the total homocystein present in the sample or standard competes with the fixed amount of immobilized total homocystein for biding site on the biotinylated anti-total homocystein antibody. The more total homocystein present in a sample or standard, the less biotinylated anti-total homocystein anti-body that binds to the plate. Following incubation, unbound biotinylated anti-total homocystein antibody is removed by washing and HRP streptavidin conjugate is added to the wells and the mircotiter plate is incubated. Following incubation and washing, the TMB substrate solution is then used to visualize the HRP enzymatic reaction by catalysis to produce a blue colored product that changes to yellow after addition of acidic stop solution. The density of yellow is inversely proportional to the amount of total homocystein present in sample or standard. The concentration of total Hcy is calculated by reading the O.D absorbance at 450nm in a microplate reader and referring to the standard curve.

Statistical Analysis

Data were analysed using statistical package for social science (SPSS) statistical software (Version 20) (SPSS Inc, Chicago. USA). Values obtained were expressed as mean ± standard deviation and results presented in tables and charts. Compact letters display (CLD) were used to compare all parameters among various diseases. The study also employed T-test to analyse values between two group at 95% confidence limit/interval.

Results

Table 1: Mean ± SD values of homocysteine between KD and Control

Homocysteine (µmol/L) levels were significantly higher in KD patients (25.08 ± 4.89 µmol/L) compared to the control group (9.06 ± 2.28 µmol/L), p< *0.001*. This indicates a strong association between chronic kidney disease and elevated homocysteine, which may contribute to increased cardiovascular risk in KD patients.

Table 1: Mean ± SD values of homocysteine between KD and Control

Parameter	Control (N = 20)	CKD (N = 20)	t-value	p-value
Homocysteine (µmol/L)	9.06 ± 2.28	25.08 ± 4.89	13.281	< 0.001 *

* Significant at $p < 0.05$

Table 2: Mean ± SD values of homocysteine between CVD and Control

Homocysteine (µmol/L) levels were significantly higher in CVD patients (22.86 ± 3.81 µmol/L) compared to the control group (9.06 ± 2.28 µmol/L), $p < 0.001$. This suggests a strong association between cardiovascular disease and elevated homocysteine, reinforcing its role as a potential biomarker or contributor to cardiovascular risk.

Table 2: Mean ± SD values of homocysteine between CVD and Control

Parameter	Control (N = 20)	CVD (N = 80)	t-value	p-value
Homocysteine (µmol/L)	9.06 ± 2.28	22.86 ± 3.81	15.493	< 0.001 *

* Significant at $p < 0.05$

Table 3. Mean ± SD values of homocysteine between prostate cancer and Control

Homocysteine (µmol/L) levels were significantly higher in patients with Prostate Cancer (18.35 ± 7.43 µmol/L) compared to the control group (9.06 ± 2.28 µmol/L), $p < 0.001$.

Table 3: Mean ± SD values of homocysteine between prostate cancer and Control

Parameter	Control (N = 20)	Prostate Ca (N = 60)	t	p-value
Homocysteine (µmol/L)	9.06 ± 2.28	18.35 ± 7.43	8.558	< 0.001 *

* Significant at $p < 0.05$

Table 4: Mean ± SD values of Homocystein levels among KD, CVD and prostate cancer Subjects

The KD group had the highest homocysteine concentration (25.08 ± 4.89^{d}), significantly higher than all other groups. The CVD group followed with elevated levels (22.86 ± 3.81^{c}), also significantly different from both CONTROL and Prostate Ca. The Prostate Ca group (18.35 ± 7.43^{b}) had significantly higher levels than the CONTROL group (9.06 ± 2.28^{a}).

ANOVA result: $F(3, 176) = 44.14$, $p < 0.001$, indicating a significant difference in homocysteine levels among the groups. CLD Explanation the Compact Letter Display (CLD) assigns the same letter to groups that are not significantly different, while different letters indicate statistically significant differences:

CONTROL = ^{a}

Prostate Ca = ^{b}

CVD = ^{c}

KD = ^{d}

Thus: All groups differ significantly from each other (no shared letters). This pattern indicates progressive elevation of homocysteine from CONTROL < Prostate Ca < CVD < KD, suggesting a strong association of elevated homocysteine with disease states, especially KD and CVD.

Table 4: Mean ± SD values of Homocystein levels among KD, CVD and prostate cancer Subjects

Parameter	CONTROL (N = 20)	KD (N = 20)	CVD (N = 80)	Prostate Cancer (N = 60)	F-value
Homocysteine (µmol/L)	9.06 ± 2.28^{a}	25.08 ± 4.89^{d}	22.86 ± 3.81^{c}	18.35 ± 7.43^{b}	44.14

Key: KD = kidney disease, CVD = Cardiovascular disease, Prostate Ca = Prostate cancer

Superscripts (^{a}, ^{b}, ^{c}, ^{d}) represent compact letter displays (CLD); different letters indicate significant differences between groups.

Table 5: Mean ± SD values of Homocysteine Levels Between Male and Female CVD Patients

The mean homocysteine level in male CVD patients was 22.77 ± 3.65 µmol/L, and in female CVD patients, it was 22.96 ± 4.04 µmol/L. The difference was not statistically significant ($p = 0.821$), indicating no gender-based variation in homocysteine levels among individuals with cardiovascular disease. Thus, homocysteine concentration appears gender-independent in CVD patients.

Table 5: Mean ± SD values Homocysteine Levels Between Male and Female CVD Patients

Parameter	Male (N = 44)	Female (N = 36)	t-value	p-value
Homocysteine (µmol/L)	22.77 ± 3.65	22.96 ± 4.04	0.23	0.821

p < 0.05 is considered statistically significant.

Table 6: Mean ± SD values of Homocysteine Levels Between Males and Females

Homocysteine levels were slightly higher in females (21.50 ± 6.68 µmol/L) compared to males (19.47 ± 7.02 µmol/L). However, this difference did not reach statistical significance ($p = 0.075$), though it approached the threshold. The 95% confidence interval for the mean difference ranged from -4.26 to 0.21 µmol/L, suggesting that the true difference may lie within this range. While females had numerically higher homocysteine levels, the difference was not statistically significant at the conventional threshold ($p > 0.05$). This indicates that, overall, homocysteine levels were comparable between sexes in this study population.

Table 6: Mean ± SD values of Homocysteine Levels Between Males and Females

Parameter	Male (N = 127)	Female (N = 53)	t-value	p-value
Homocysteine (µmol/L)	19.47 ± 7.02	21.50 ± 6.68	1.79	0.075

*p < 0.05 indicates statistical significance.
t(df): t-statistic with degrees of freedom.

Discussion

This study evaluated homocysteine levels in individuals with cardiovascular disease, kidney disease, and prostate cancer. The study's primary finding was a significant elevation of homocysteine levels in all aforementioned diseases compared to their controls (p= 0.001). This significant rise in homocysteine levels among patients with kidney disease may indicate compromised renal clearance and modified metabolism linked to the condition [11]. The significant elevation of homocysteine (Hcy) levels in kidney disease (KD) patients (p = 0.001) relative to healthy controls, as demonstrated in this study, aligns with the increasing literature highlighting hyperhomocysteinemia as a prevalent and clinically significant metabolic disorder in KD. This metabolic disturbance is pivotal in the pathogenesis of cardiovascular problems, which persist as the primary cause of morbidity and mortality in KD populations [12].

Homocysteine is an amino acid that contains sulphur and is made when methionine is demethylated. In normal physiological settings, homocysteine is either remethylated to methionine by methionine synthase, which needs folate and vitamin B12 as cofactors, or transsulfurated to cysteine by cystathionine β-synthase (CBS), which needs vitamin B6. The kidneys are very important for both breaking down and getting rid of homocysteine. In kidney disease (KD), particularly with the drop in glomerular filtration rate (GFR), the clearance of homocysteine decreases due to diminished renal excretion and compromised metabolic processing [13].

The hyperhomocysteinemia observed in this study, which impacts kidney disease (KD), arises from numerous pathways.
(a) Decreased Renal Clearance: As KD gets worse, the kidneys' ability to filter and break down homocysteine is much worse.
(b) Impaired Enzymatic Activity: KD patients often lack important cofactors like folate, vitamin B12, and B6 because they don't eat enough of them, lose them during dialysis, or can't absorb them properly. This makes it harder for the body to break down homocysteine.
(c)Inflammatory State: Chronic inflammation in KD can inhibit enzymes such as methylene tetrahydrofolate reductase (MTHFR), which makes the remethylation pathway even worse.

Accumulated uremic toxins can stop enzymes that break down homocysteine.
These causes lead to the systemic buildup of homocysteine, especially in individuals with advanced KD or those undergoing dialysis [14].

The increased homocysteine levels in cardiovascular disease noted in this study indicate endothelial dysfunction and vascular inflammation, underscoring its potential as a biomarker and risk factor in cardiovascular pathophysiology. The finding that homocysteine (Hcy) levels were significantly elevated in subjects with cardiovascular disease (CVD) (p = 0.001) compared to controls in this study corresponds with substantial epidemiological and mechanistic evidence indicating hyperhomocysteinemia (HHcy) as an independent, non-traditional risk factor for cardiovascular morbidity and mortality. This finding is both statistically significant and biologically plausible, as Hcy is directly involved in endothelial dysfunction, vascular inflammation, and thrombogenesis, which are essential factors in the aetiology of atherosclerosis and associated cardiovascular events [14].

Hyperhomocysteinemia is now acknowledged as a non-conventional cardiovascular risk factor. It causes endothelial dysfunction, increases oxidative stress, encourages the growth of vascular smooth muscle, and increases thrombotic activity by making platelets stick together and blocking anticoagulant pathways [15]. These pathophysiological processes facilitate atherogenesis and vascular calcification, which are notably expedited in KD patients.

Meta-analyses and longitudinal studies have associated elevated homocysteine levels with an augmented risk of myocardial infarction, stroke, and peripheral vascular disease in the KD population. So, high levels of homocysteine are not just a biological curiosity; they could be a target for intervention that can be changed.

In addition to its cardiovascular consequences, nascent research indicates that homocysteine may potentially contribute to the advancement of KD itself. Elevated homocysteine may facilitate tubulointerstitial fibrosis and worsen renal failure through pathways involving oxidative stress and glomerular endothelial damage. Although this association has yet to be completely clarified, it introduces an extra layer of difficulty to the clinical care of KD patients.

Homocysteine is an amino acid that contains sulphur and does not make proteins. It comes from the metabolism of methionine. It is present in plasma in both free and protein-bound forms and is metabolised through two primary pathways: remethylation to methionine (which necessitates folate and vitamin B12) and transsulfuration to cystathionine and subsequently to cysteine (which requires vitamin B6). Genetic, dietary, or acquired factors that disrupt these metabolic pathways cause homocysteine levels in the blood to rise [16].

Plasma total homocysteine levels are strictly controlled in a healthy state. Nonetheless, in pathological circumstances like cardiovascular disease (CVD), these levels significantly increase, as seen in the present study. A p-value of 0.001 significantly confirms the link between high homocysteine levels and heart disease, which shows how important this biochemical change is in the clinic.

Endothelial damage, the first step in atherogenesis, is one of the most well-known effects of hyperhomocysteinemia. Hcy diminishes nitric oxide (NO) bioavailability, promotes oxidative stress through reactive oxygen species (ROS), and upregulates adhesion molecules (e.g., VCAM-1, ICAM-1), promoting a pro-inflammatory endothelium phenotype (Stühlinger et al., 2001). This prepares the blood vessels for white blood cell adhesion and foam cell generation, which speeds up the growth of atherosclerotic plaques. Also, the thiol group in homocysteine passes through auto-oxidation, which makes hydrogen peroxide and superoxide radicals. These reactive species not only break down NO, but they also oxidise LDL cholesterol, turning it into atherogenic oxidised LDL (oxLDL). Oxidative injury to vascular smooth muscle cells (VSMCs) encourages migration and proliferation, worsening vascular remodelling and plaque instability [17]. Elevated homocysteine levels induce a hypercoagulable condition by enhancing platelet aggregation, diminishing protein C function, decreasing thrombomodulin, and upregulating tissue factor expression. These actions work together to make thrombin production go up, which leads to arterial thrombosis, which happens a lot in myocardial infarction and ischaemic stroke.
Hyperhomocysteinemia is linked to greater arterial stiffness, which is a proven independent risk factor for cardiovascular events. It also promotes vascular calcification by triggering the osteogenic transition of vascular smooth muscle cells (VSMCs), which leads to higher vascular resistance and left ventricular hypertrophy [18].

Genetic polymorphisms, especially in the MTHFR gene (C677T variation), hinder folate metabolism and decrease the remethylation of homocysteine to methionine, resulting in increased plasma levels. This polymorphism is prevalent in specific populations and may partially elucidate the inter-individual variability in cardiovascular risk linked to Hcy levels. Additionally, deficits in folate, vitamin B12, and vitamin B6—commonly observed in the elderly, vegetarians, and low-resource populations—can further aggravate homocysteine buildup. Consequently, the rise of homocysteine in cardiovascular disease (CVD) is complex, encompassing both genetic and dietary phenotypes.

In prostate cancer, the significant elevation of plasma homocysteine indicates a potential disruption in the one-carbon metabolism pathway, which has been linked to an elevated risk for prostate cancer due to its impact on DNA synthesis and methylation [19].

The significant elevation of homocysteine (Hcy) levels in prostate cancer subjects ($p = 0.001$) relative to controls provides critical evidence for the emerging hypothesis that dysregulated homocysteine metabolism is not only a cardiovascular risk factor but may also play a role in tumorigenesis. This finding indicates a statistically significant and physiologically relevant connection, necessitating additional investigation into the potential mechanistic and clinical ramifications of homocysteine dysregulation in prostate cancer pathogenesis.

Homocysteine is a key part of one-carbon metabolism, which is a group of activities that depend on folate and vitamin B12 and are very important for making, fixing, and methylating DNA. In healthy cells, homocysteine is remethylated to methionine, which is then changed into S-adenosylmethionine (SAM), which is the universal methyl group donor for DNA and histone methylation. Aberrant homocysteine metabolism can impair epigenetic homeostasis and DNA integrity, both of which are essential implicated in neoplastic transformation and progression.

In the context of prostate cancer, modifications in one-carbon metabolism may create a pro-tumorigenic environment by: (a) facilitating global DNA hypomethylation, resulting in genomic instability; (b) inducing hypermethylation of tumour suppressor genes, thereby silencing their expression; (c) hindering DNA repair mechanisms and expediting mutations. Thus, increased homocysteine levels may indicate and perhaps induce malignant epigenetic reprogramming and genomic damage in prostate tissues [20].

Epigenetic changes that happen all over the body are a sign of prostate cancer. High amounts of homocysteine may lower intracellular levels of SAM while raising levels of S-adenosylhomocysteine (SAH), which is a strong inhibitor of methyltransferases. This imbalance makes it harder for methylation to happen, which messes up gene expression and makes oncogenes more likely to be expressed and tumour suppressors like GSTP1, p16INK4a, and RASSF1A less likely to be expressed.

Homocysteine auto-oxidizes, producing reactive oxygen species (ROS) such superoxide and hydrogen peroxide. Chronic oxidative stress leads to DNA strand breakage, base alterations, and erroneous DNA repair—characteristics prevalent in cancerous prostate cells. ROS also turn on signalling pathways like NF-κB, PI3K/AKT, and MAPK that help cells grow, invade, and form new blood vessels.

Homocysteine has been demonstrated to activate matrix metalloproteinases (MMPs) and vascular endothelial growth factor (VEGF), hence promoting extracellular matrix remodelling and angiogenesis, which are essential for tumour growth and metastasis. In prostate cancer, these processes may facilitate local invasion and bone metastases. Although early research on homocysteine was mostly about heart disease, more recent epidemiological studies have shown that it is also important for cancer. Numerous case-control and cohort studies have indicated increased homocysteine levels in individuals with diverse malignancies, such as prostate, colorectal, breast, and gastric cancers [21]. For example, elevated homocysteine levels have been observed in prostate cancer patients relative to healthy controls, with this elevation correlating with the Gleason score. Likewise, mutations in genes associated with one-carbon metabolism (e.g., MTHFR C677T, MTR A2756G) have been demonstrated to affect prostate cancer risk, potentially through modifications in homocysteine and methylation levels [22].

These observations, in conjunction with the current finding ($p = 0.001$), indicate that hyperhomocysteinemia may serve as a biochemical characteristic of prostate cancer or a biomarker for aggressive illness. Considering the persistent observation of increased homocysteine levels in individuals with prostate cancer. Hcy may have a number of possible clinical uses, such as (a) a diagnostic biomarker. Homocysteine, being a non-invasive blood analyte, may assist in differentiating malignant from benign prostatic diseases, especially when used in conjunction with PSA and other indicators. (b) Prognostic marker: Elevated homocysteine levels may be associated with tumour stage, grade, or metastatic potential. (c) Predictive marker: Homocysteine levels may indicate treatment response or toxicity in patients receiving chemotherapy or androgen deprivation therapy (ADT) [23].

Conclusion

This study concludes that homocysteine levels are higher in all examined disorders (CVD, KD, and prostate cancer). In KD, the rise is attributed to compromised renal clearance, whereas in CVD, the increase indicates a significant role in endothelial dysfunction and vascular inflammation. There is an increase in prostate cancer, but it is the lowest compared to the KD and CVD levels. This suggests that the one-carbon metabolic pathway may not be working properly, which can cause problems with DNA synthesis and methylation.

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