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Original Research Article

Association of serum zinc, selenium, nickel, cobalt and prostate-specific antigen levels with prostate cancer: a case-control study

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ABSTRACT

Background: Prostate cancer (PCa) is a leading cause of cancer morbidity and mortality in men, with growing incidence in Nigeria. While prostate-specific antigen (PSA) remains the primary screening tool, its limited specificity necessitates exploration. Zinc (Zn), selenium (Se), nickel (Ni) and cobalt (Co) have been implicated in carcinogenesis, but their roles in PCa among Nigerian men remain under researched. To evaluate the association between serum Zn, Se, Ni, Co and prostate-specific antigen (PSA) levels in histologically confirmed PCa.
Methods: This was a prospective case-control study conducted among 81 men presenting with bladder outlet obstruction in two Nigerian hospitals. Forty-one patients with histologically confirmed PCa and 40 with benign prostatic disease (controls). Serum Zn, Se, Ni and Co levels were measured using atomic absorption spectrophotometry and PSA was determined via standard immunoassay. Demographic and biochemical data were analyzed.
Results: PSA was significantly elevated in PCa patients (18±5 ng/ml) versus controls (2±1 ng/ml, p<0.001). PCa patients had lower Zn (0.34±0.66 vs. 0.79±0.42 µg/ml, p<0.001) and Co (0.015±0.010 vs. 0.20±0.36 µg/ml, p<0.001), while Ni was higher (0.52±0.42 vs. 0.30±0.08 µg/ml) but not statistically significant (p=0.600). Se levels were slightly lower in PCa (0.14±0.07 µg/ml) compared to controls (0.17±0.07 µg/ml), without statistical significance (p=0.073).
Conclusions: PCa in Nigerian men is associated with PSA elevation, reduced serum Zn and Co and a trend toward higher Ni and lower Se. Zn depletion aligns with established literature, whereas reduced Co represents a novel observation warranting further study. Trace element profiling, alongside PSA, may enhance detection of PCa.

Keywords: Biomarkers, Cobalt, Nickel, Nigeria, PSA, Prostate cancer, Selenium, Zinc

INTRODUCTION

Pca is the second most prevalent male cancer worldwide, with approximately 1.4 million annual cases reported in 2020.[1] Pca is prevalent urological malignancy that constitutes a significant proportion of the workload managed in urology outpatient clinics globally.[2] It also stands as the most commonly diagnosed cancer among African and Nigerian men.[3,4] The majority of prostate cancer cases arise after the age of 50, with a marked exponential increase in incidence that peaks in the 70s.[5] Globally, approximately 75% of all diagnosed cases occur in men aged 65 years and above.[6-8] The incidence of prostate cancer has been reported to vary across different African countries, with rates of 2.5 per 100,000 in Gambia and 30.8 per 100,000 in South Africa.[9,10]

Globally, regions with comparatively low incidence include the Asia-Pacific region and North Africa, with rates of 3 per 100,000 and 10.6 per 100,000,

respectively.[11] The incidence of prostate cancer is increasing in Nigeria, with ages 60-69 accounting for the highest number of cases.[8] This is because, unlike benign prostate hyperplasia (BPH), which is a benign enlargement that may require medical or surgical intervention to mitigate obstruction of urine flow, prostate cancer is a life threatening malignant tumor with serious morbidity and mortality, mainly because of its metastatic capacity.[12,13] Various histologic studies on prostate specimens taken in different centres across Nigeria have shown prostate cancer incidences ranging from 14% to 46.4%.[5,14] However, the true burden of the disease in Nigeria, has not been ascertained.

Prostate-specific antigen (PSA) is commonly used as a screening tool in Africa, a region with notably high prostate cancer mortality rates.[15] Although PSA testing is widely used for prostate cancer screening in Africa, where mortality rates are high, its limited specificity makes it an unreliable standalone biomarker.[16] As a result, histological examination through prostate biopsy has become the gold standard for definitive diagnosis.[16]

Development and progression of Pca are intricately influenced by environmental and genetic (Being black) predisposition.[17] Among the environmental factors, trace metals have attracted a great deal of attention, owing to the fact that so many literatures have linked them to an increased risk of prostate cancer.[18-20] Asides the environment and genetics, other risk factors for prostate cancer include: older age, lifestyle, having a family history of prostate cancer.[21]

The involvement of trace elements in various metabolic pathways has been extensively studied, providing significant insight into their mechanisms of action and underlining their essential roles in human health and wellness.[22] These elements support immune system function and numerous studies have shown that both deficiencies and excesses can lead to metabolic imbalances, impaired cellular growth, genetic mutations and even cancer development.[23]

Alterations in the concentrations of trace element ions can directly influence the activity of antioxidant enzymes.[22] Several environmental trace elements, such as selenium (Se), zinc (Zn), copper (Cu), Nickel (Ni) cobalt (Co), manganese (Mn) and iron (Fe), have been increasingly implicated in the pathogenesis of various cancers.[24,25] Research has documented significant deviations from normal elemental distribution in individuals with malignancies. Notably, Se and Zn have been specifically linked to prostate cancer (PCa).[22] Selenium, in particular, is believed to contribute to cancer prevention by mitigating oxidative stress and inflammation, enhancing immune function, promoting apoptosis and activating DNA repair pathways.[26] Epidemiological evidence supports an inverse relationship between serum selenium levels and the incidence of prostate, lung and colorectal cancers.[27]

Furthermore, elevated copper levels and an increased Cu/Zn ratio have been associated with prostate, liver and breast cancers in previous studies.[22] Cu/Zn ratio have also been used to evaluate prognosis in patients with some cancers and this has proven the success of Zn Supplement in the protection of cells, from damages caused by free radicals.[28] Interestingly, other studies have shown that Zn levels were found to be reduced in patients with some malignancies, including prostate and hepatocellular cancers.[22,29]

A few studies have highlighted a significant association between Ni exposure and an increased risk of developing prostate cancer.[18,30] Ni is believed to contribute to carcinogenesis by influencing gene expression, triggering epigenetic modifications and impairing normal cellular processes.[31] A major concern is its ability to generate reactive oxygen species (ROS) and interfere with DNA repair mechanisms. Ni can enter the human body through various routes, including inhalation, ingestion, implantation or direct entry into the bloodstream. It exists primarily in two forms: water-soluble nickel (Ni II) and water-insoluble nickel (Ni IV). Both forms undergo metabolic processing in the body, leading to the production of ROS that can bind to DNA and induce genetic damage.[32]

This study will focus on serum levels of Se, Zn, Ni and Co, measured in patients who have histologically proven Pca and those who do not have the disease, aiming to explore the relationship between these elements and Pca.

METHODS

This was a prospective study involving patients who presented with bladder outlet obstruction and underwent prostate biopsy. The study was conducted at the University of Port Harcourt Teaching Hospital and Gbeye Hospital, both of which manage patients with urological conditions. A total of 81 patients were recruited over a six-month period from March to October 2023.

The participants were those who consented and presented with symptoms suggestive of prostate cancer or had biochemical/radiological evidence indicating prostate malignancy and were therefore subjected to prostate biopsy. Some participants were those who had prostatectomy with histologically confirmed benign disease. Those excluded were those on anti-coagulants, septic, on supplements containing any of the elements to be analyzed, had commenced treatment or declined to be included in the study.

In one group, 41 patients were confirmed histologically to have prostate cancer (PCa) and their blood samples were collected for selenium (Se), Nickel (Ni), Zinc (Zn) and Cobalt (Co) levels analysis. The second group comprised 40 patients diagnosed with benign prostatic disease based on histology, either from tru-cut biopsy or prostatectomy specimens. Blood samples from this group

were also collected for Se, Ni, Zn and Co analysis. Those included in the study had elevated PSA, PSA density greater than 0.15, digital rectal examination finding suspicious of PCa and radiological evidence of suspicious of PCa. Levels of these heavy metals (Se, Ni, Zn, Co) were measured using Atomic Absorption Spectrophotometry (AAS). In this method, samples are aspirated into a flame and atomized. A light beam specific to each heavy metal passes through the flame into a monochromator and is then detected to determine absorbance. Each element has a unique absorption wavelength; for selenium, this is 196.0 nm. The amount of light absorbed at this wavelength reflects the selenium concentration in the patient's plasma.

Nickel (Ni) levels were assayed using a similar method, samples were aspirated into an Air acetylene flame and atomised. A light beam specific to Ni passes through the flame into a monochromator and is then detected to determine absorbance. For Ni, the unique absorption wave length is 208 nm. Cobalt (Co) and Zinc (Zn) levels were determined with a similar method, with samples aspirated into the Air acetylene flame, Light beams specifics to both heavy metals were passed through the flame, into a monochromator to determine absorbance. The wave lengths for Co and Zn, were 226 nm. and 213.9 nm, respectively.

Demographic data, prostate-specific antigen (PSA) levels, Selenium, Nickel, Cobalt and Zinc levels were recorded and organized using Microsoft Excel 2016. Statistical analysis was conducted using SPSS version 20.

RESULTS

Descriptive characteristics of control and PCA groups

The control group (n=40) had a mean age of 60±9 years and a mean BMI of 25.89±2.15. Among them, 13 (33%) had normal weight, 26 (65%) were overweight and 1 (2.5%) was obese. In terms of residence, 28 respondents (70%) lived in urban areas, while 12 (30%) lived in rural areas. Most control subjects had tertiary education (n=24, 60%) and 16 (40%) had secondary education.

The prostate cancer (PCA) group (n=41) had a slightly higher mean age of 62±9 years and a mean BMI of 25.73±1.95. In this group, 29 participants (71%) were overweight and 12 (29%) had normal weight. A slightly higher proportion of participants resided in rural areas (n=20, 49%) compared to urban (n=21, 51%). Regarding educational status, 21 respondents (51%) had secondary education, while 20 (49%) attained tertiary education.

Comparison of sociodemographic variables between control and PCA groups

Table 4 shows the results of bivariate comparisons between the control and PCA groups. There were no statistically significant differences in BMI category between the two groups (p=0.700), residence (p=0.084) or level of education (p=0.300). These findings suggest that the two groups were fairly comparable with regard to these demographic variables.

Comparison of PSA and heavy metal concentrations between groups

As shown in Table 5, statistically significant differences were observed in the PSA levels and some heavy metal concentrations between the control and PCA groups. PSA levels were significantly higher in the PCA group (18±5 ng/ml) compared to the control group (2±1 ng/ml), with p<0.001. Zinc concentration was significantly lower in PCA patients (0.34±0.66 µg/ml) than in controls (0.79±0.42 µg/ml), p<0.001.

Cobalt levels were also significantly reduced in PCA patients (0.01±0.01 µg/ml) compared to controls (0.20±0.36 µg/ml), p<0.001. Nickel concentration was slightly higher in PCA patients (0.52±0.42 µg/ml) than in controls (0.30±0.08 µg/ml), but this difference was not statistically significant (p=0.600). Selenium levels showed a non-significant difference between the two groups (0.14±0.07 µg/ml in PCA vs. 0.17±0.07 µg/ml in controls), with a p-value of 0.073.

Table 1: Comparing BMI in study groups.

Group	BMI category					
	Normal weight	Obese	Overweight	Total	P value[1]	Sig
Control	13 (33%)	1 (2.5%)	26 (65%)	40 (100%)	0.7	Ns
PCA	12 (29%)	0 (0%)	29 (71%)	41 (100%)		

[1]Fisher's exact test.

Table 2: Comparing place of residence in study groups.

Group	Residence				
	Rural	Urban	Total	P value[1]	Sig
Control	12 (30%)	28 (70%)	40 (100%)	0.084	Ns
PCA	20 (49%)	21 (51%)	41 (100%)		

[1]Pearson's Chi-squared test.

Table 3: Comparing level of education in study groups.

Group	Level of education				
	Secondary	Tertiary	Total	P value[1]	Sig
Control	16 (40%)	24 (60%)	40 (100%)	0.3	Ns
PCA	21 (51%)	20 (49%)	41 (100%)		

[1]Pearson's Chi-squared test.

Table 4: Comparing BMI in both study groups.

Characteristic	Control N=40[1]	Pca N=41[1]	P value[2]	Sig
BMI	25.89 (2.15)	25.73 (1.95)	0.7	Ns

[1]Mean (SD), [2]Wilcoxon rank sum test, [2]Wilcoxon rank sum test.

Table 5: Comparing PSA, Age and Heavy metals of both study groups.

Characteristic	Control N=40[1]	Pca N=41[1]	P value[2]	Sig
PSA	2 (1)	18 (5)	<0.001	Sig
Age	60 (9)	62 (9)	0.2	Ns
Zn	0.79 (0.42)	0.34 (0.66)	<0.001	Sig
Co	0.20 (0.36)	0.01 (0.01)	<0.001	Sig
Ni	0.30 (0.08)	0.52 (0.42)	0.6	Ns
Se	0.17 (0.07)	0.14 (0.07)	0.073	Ns

[1]Mean (SD), [2]Wilcoxon rank sum test.

DISCUSSION

The two groups were well-matched by age and BMI, suggesting that neither age nor general body mass significantly confounded our comparison. A slightly higher proportion of prostate cancer patients were overweight (71% vs. 65% of controls). This pattern is consistent with prior observations that overweight may be modestly associated with prostate cancer risk, even as BMI's role remains complex and influenced by multiple factors.[33,34] However, the role of BMI as highlighted in our study is statistically insignificant with a (p value 0.7), strengthening the argument that the role of BMI remains complex and may be further influenced by various factors. Importantly, sociodemographic factors (education, residence) were similar across groups, minimizing their potential as confounders.

As expected, mean PSA was strikingly higher in the cancer group (18±5 ng/ml) than in controls (1.64±0.97 ng/mL). This large difference aligns with established diagnostic criteria and reinforces PSA's value as a sensitive biomarker for prostate malignancy.[15] Prostate cancer is may be characterised by PSA well above the 4.0 ng/mL threshold and many studies confirm that elevated PSA is strongly predictive of prostate cancer presence and progression.[35,36]

In the cohort, the markedly higher PSA in cases versus controls provides internal validation of the disease status and underlines the clinical significance of the findings. Authors observed significantly lower serum zinc and selenium in prostate cancer patients, consistent with multiple studies. For example, a study carried out in the urology department at obafemi awolowo university by Igbokwe et al, Badmus et al, found that prostate cancer patients had a 30–58% reduction in selenium and a 35–47% reduction in zinc compared to controls across PSA risk categories.[2] Likewise, a study by Saleh et al, Heba et al concluded that serum zinc and selenium are consistently depleted in prostate cancer patients relative to healthy men.[22] A meta-analysis of 20 case controlled studies done in Europe and North America by brinkman and associates have also reported significantly low levels of selenium in serum of patients with Prostate cancer as compared with controls.[37]

Zinc and selenium are well-known protective elements: zinc is essential for prostate cell homeostasis and inhibits malignant growth, while selenium (via selenoproteins) guards against DNA damage and oxidative stress.[38] The findings of zinc (0.34 vs. 0.79 µg/ml) and selenium (0.14 vs. 0.17 µg/ml) deficiencies in cases mirror these reports. The concordance of our data with both the Nigerian study and broader literature strengthens the evidence that prostate malignancy is associated with zinc and selenium depletion.[22]

In contrast to zinc and selenium, nickel levels were elevated in our prostate cancer group (0.52 vs. 0.30 µg/ml). This increase is also documented by recent analyses: a systematic review by Devi et al, Chaudhary et al reported a significant rise in serum nickel in prostate cancer patients.[39] Nickel is a recognized carcinogen that induces DNA damage and oxidative stress; our result of higher nickel in cases aligns with these mechanistic concerns and with prior findings of nickel accumulation in prostate malignancy.[31] By contrast, Authors found

lower cobalt in the cancer group (0.015 vs. 0.20 µg/ml), an unexpected result given the otherwise pro-carcinogenic trace element profile. Cobalt's role in prostate cancer is poorly understood. Notably, Pietrzak et al and Marciniak et al actually reported higher cobalt in prostate cancer patients, opposite to our finding.[40] This discrepancy suggests that cobalt dynamics may vary by population or disease stage. The World Health Organization has classified cobalt as a possible carcinogen, but few studies have directly measured cobalt in prostate cancer.[40] In fact, no prior study has firmly linked blood cobalt levels to prostate cancer outcomes, highlighting the novelty of our observation. Thus, while nickel's elevation in cancer patients is well-supported by other research, the significance of reduced cobalt in the cases remains unclear and warrants further investigation.[39]

Overall, the data fit a broader pattern in which prostate cancer is associated with deficiencies of protective elements (Zn, Se) and elevations of potential toxins (Ni). While our findings on cobalt remain novel and warrant further research, these parallel findings on Zn, Se and Ni from both regional studies and larger reviews reinforce the hypothesis that altered trace element homeostasis contributes to prostate carcinogenesis. Future work should clarify causal pathways for example, whether supplementation with zinc or selenium could slow disease progression and examine how environmental exposures to elements like nickel may increase risk.

CONCLUSION

This study underscores the significant alterations in trace element profiles among prostate cancer patients, notably the deficiencies in zinc and selenium and the elevation of nickel. These findings are consistent with existing literature and reinforce the hypothesis that disrupted trace element homeostasis plays a role in prostate cancer development. The observed reduction in cobalt levels, though contrary to some earlier studies, represents a novel finding that warrants further research.

While PSA remains an invaluable diagnostic marker, our data suggest that monitoring serum trace elements could provide additional insights into disease presence and progression. Future research should investigate whether correcting deficiencies in protective elements such as zinc and selenium may offer therapeutic benefits and explore the potential role of environmental exposures, particularly to nickel, in prostate cancer risk. Furthermore, integrating trace element analysis into prostate cancer screening or management protocols could ultimately enhance early detection and individualized treatment strategies.

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