Original Article

Chromium Toxicity

Comparative Study of Chromium Toxicity with Hypertension and End Stage Renal Failure Cases

1. Nadeem ul Haq 2. Muhammad Sajid Khan 3. Adnan Anwar
t. Assoc. Prof. of Biochemistry, 2. Asstt. Prof. of Physiology, 3. Lecturer of Physiology,
Al-Tibri Medical College, Karachi

ABSTRACT

Objective: To assess the association between hypertension and chronic renal failure with lethal effects of chromium on the beginning of the Chronic Kidney disease.
Study Design: A prospective cross sectional study**.**
Place and Duration of Study: Jinnah post graduate medical centre and Kidney centre Karachi during December 2012 to December 2013.
Materials and Methods: The present study was conducted on a total of 150 patients (age > 40 years) divided into three groups. Patients in Group I included 50 patients with hypertension whereas Group II included 50 patients with hypertension associated with chronic renal failure (CRF) and Group III consisted of 50 healthy controls.
Results: Results showed that levels of fasting blood sugar and HbA1c in both group I and group II were significantly higher as compared to controls. The levels of serum urea (**132.0 ± 28.2**) and creatinine (**7.8 ± 1.38**) in group II patients were significantly high as compared to group I and group III patients. The Creatinine clearance (**55.1 ± 9.61**) in group II patients were significantly less as compared to group I and group III patients. Serum chromium levels were significantly high in group II patients(**59.6 ± 6.73)** as compared to group I and controls.
Conclusion: Serum chromium level has significant correlation with glycemic index in both group I and group II patients compared to controls, while correlation with renal failure was significant only in group II patients.
Key Words: Hypertension, Serum chromium, Chronic renal failure, Creatinine

Citation of article: Haq N, Khan MS, Anwar A. Comparative Study of Chromium Toxicity with Hypertension and End Stage Renal Failure Cases. Med. Forum 2015;26(12):23-25.

INTRODUCTION

Kidney damage are common problems. The causes of nephropathies are different and some time life threatening (Sabolic 2006)[1].
Researchers found that derangements in serum creatinin level are some time fatal.(Chawla et al 2011) [2]. Chronic renal disease is a community health crisis. National Kidney Foundation on the source of clinical practice recommendations, found that twenty million subjects in the USA have ESKD, with eight million of these classified as having moderate or severe renal disease (Weiner DE et al 2004)[3].
Chronic kidney diseases may leads to several other diseases CVS disease and renal failure, also known as CKD, need treatment with dialysis or a kidney transplant for continued existence. (Jennette et al 2010)[4].
The main functions of the kidneys are regulation of water and electrolyte balance, excretion of hormones and many foreign substances, specifically drugs and regulation of arterial blood pressure and to eliminate waste products and excess water from the blood,. Disturbance of kidney functions leads to various health harms, such as bone disease , hypertension, anemia and hypercholesterolemia (Guttmann et al 2008)[5]. Some time mild failures in kidney function may leads to death, prolonged span of stay, and increased costs. The sign and symptoms of ESKD are mostly, GIT, CVS, blood and CNS symptoms. Dyspnia is a result of hypervolaemia, metabolic acidosis and anaemia (Hsu et al 2008)[6]. Numerous well-known as well as minor known relations exist between ESKD and both environmental causes and conditions, such as diabetes mellitus, hypertension, heavy metals, industrial chemicals, elevated ambient temperatures, and infections(Das and Singh 2011)[7].
Systemic hypertension and increased glomerular filtration lead to progressive nephron damage. Effective blood-pressure control delays the advancement of renal disease in adults with chronic kidney disease(Iyer et al 2010)[8].
High blood pressure is frequent in older patients is a risk factor for event of cardiac failure and CKD (Wohl et al 2009)[9].
Chromium is [Cr (VI)] a strong oxidizing agent, is carcinogenic, mutagenic, potent inducers of tumors in experimental animals; neurotoxic, immunotoxic, genotoxic reproductive toxic, and can cause DNA

Correspondence: Dr Nadeem-ul-haq,
Associate Professor of Biochemistry
Al-Tibri Medical College, Karachi
Cell No, 03222577272
E-mail: nadeemulhaq252@gmail.com

damage, sister chromatid exchanges gene mutations, and chromosomal aberrations (Soderlalnd et al 2010)[10]. Cr(VI) and Cr(III) are the predominant stable oxidation states. Many Cr(VI) compounds are highly soluble. Cr(VI) can readily cross the skin, and is transported into cells via an anion carrier (cr3). Most of Cr(III) compounds are generally insoluble and do not easily cross cell membranes Toxic effects of Cr(VI) result from its cellular activation via one- or two-electron reduction processes. (Singh SK et al 2012)[11].

MATERIALS AND METHODS

The present study is a cross sectional prospective included 150 patients. The patients are divided into three groups.

Group I included 50 patients (age > 40 years) having hypertension Group two comprises of fifty subjects (age more than forty years) having hypertension with end stage kidney diseases and Group three comprises of fifty normal subjects (age more than forty years). Experimental groups which treated to heavy water contamination by the poisonous metals were particular for this study.

Written approval was taken from all subjects and total examination and history was taken. The exclusion criteria are patients having liver disease, endocrine disease, pregnancy and females using oral contraceptive pills.

Patient's blood was collected for examination the serum level of urea, Creatinine, Creatinine clearance and chromium level (Owiredu et al, 2013)[12]. The data was analyzed on SPSS. Less than 0.05 p-value considered to be significant.

RESULTS

One hundred and fifty samples of blood were examined, who were divided into three groups having different age and gender were examined.

Results showed that group two subjects having high blood pressure and end stage renal diseases almost analogous with group one subjects with high blood pressure . Thus no obvious changes were noted in group one subjects when compared with group one subjects. FBS and HbA1c in both Group one and Group two were not significant as compared with normal subjects shown in Table 1.

Table No.1: Hypertension with end stage kidney diseases and controls

Glycemic index	(Group I) Hypertension (n=50)	(Group II) Hypertension with CRF (n=50)	(Group III) Controls (n=50)	p-Value
Fasting Blood Sugar (mg/dl)	100.5 ± 14.48*	101.3 ± 14.65	100.8 ± 15.47	0.001
HbA1c (%)	5.5 ± 0.88	5.6 ± 0.90	5.5 ± 0.94	0.001

* As compared to controls p<0.01

Table No.2: Relationship of kidney function in subjects with Hypertension , Hypertension with end stage kidney diseases and normal subjects

Renal function and serum chromium levels	(Group I) Hypertension (n=50)	(Group II) Hypertension with CRF (n=50)	(Group III) Controls (n=50)	p-Value
Urea (mg%)	24.2 ± 8.73	132.0 ± 28.2	22.3 ± 8.49	0.001
Creatinine (mg%)	1.15 ± 0.22	7.8 ± 1.38	1.12 ± 0.23	0.001
Creatinine Clearance (ml/min)	115.3 ± 13.91	56 ± 9.51**	108.8 ± 14.36	0.001
Chromium (ug/dl)	1.95 ± 0.64	59.6 ± 6.73	1.5 ± 1.07	0.001

* As compared to controls p<0.01. **As compared to Type II diabetic p<0.01

Table No.3:- Relationship between glycemic index, Chromiu and kidney parameters in groups.

		FBS	HbA1c	Urea	Creatinine	Creatinine clearance
Chromium (Pearson Correlation)	Control &HTN	.075	.051	.001	.073	.085
	Control & HTN with CRF	.126	.087	.340**	.358**	-.388**

** Relationship is significant at the 0.01 level (2-tailed)
* Relationship is significant at the 0.05 level (2-tailed)

The results showed that levels of serum urea (132.0 ± 28.2) and creatinine (7.8 ± 1.38) in group II patients (Diabetes with CRF) were significantly elevated (p<0.01) as compared to group I (Hypertension) and group III (Controls) patients. However Creatinine clearance (55.1 ± 9.61**) in group II patients (Diabetes

with CRF) were significantly less ($p<0.01$) as compared to group I (hypertension) and group III (Controls) patients. Serum chromium levels were considerably high ($p<0.01$) (59.6 ± 6.73) in group II patients (hpertension with CRF) as compared to group one (hypertension) and controls (group III) as shown in Table 2.

Table 3 shows the correlation between Chromium with FBS, HBA1c and renal parameters like Urea, Creatinine and Creatinine clearance in hypertension and hypertension with end stage kidney diseases subjects. Results have shown that lead have no significant correlation with glycemic index ($P<0.01$) in each. HTN and HTN with CRF, kidney parameters are significant ($P<0.01$) only hypertension with end stage kidney diseases subjects. Correlation between lead and creatinine and HBA1c were presented respectively in figure 5 (a,b) and figure 6 (a,b). in HTN patients (fig 5a and 6a) and in HTN with CRF patients (fig 5b and 6b).

DISCUSSION

The present study showed that in metropolitan city like Karachi, particularly in industrial area , hypertension is very common and most of hypertensive patient are associated with high serum chromium level and chronic renal failure. We also found that the majority of the subjects were normal, comparable results are also noted by Chawla et al in 2010 and weiner DE at al in 2004.

In our study we also found that there is a strong correlation between heavy metal poisoning with diabetes mellitus and hypertension when compare with control and similar finding was observed by Soderland et al in 2010 and wohl et al in 2009.

In our study we do not find that accumulation of chromium is the only factor causing chronic renal failure , there are other factors involve as well, other researchers found that chronic renal failure may occur with or without exposure of chromium.

In present study the results show that in patients with hypertension and CRF increased level of FBS and HBA1c also increase the level of urea, creatinin and creatinine clearance.

The main finding of our study was that chromium exposure is one of the aggravating factor causing chronic renal failure with or without hypertension , but other factors are involve.

CONCLUSION

It is concluded that increase serum chromium level is strongly correlated with hypertension and chronic renal failure.

Conflict of Interest: The study has no conflict of interest to declare by any author.

REFERENCES

1. Soderland P, Lovekar S, Weiner DE, Brooks DR, Kaufman JS. Chronic kidney disease associated with environmental toxins and exposures. Adv Chronic Kidney Dis 2010; 17(3):254-64.
2. Chawla LS, Amdur RL, Amodeo S. The severity of acute kidney injury predicts progression to chronic kidney disease. Kidney Int 2011; 79: 1361–69.
3. Wohl E, Trivelli A, Picca S. Strict blood pressure control and progression of renal failure in children.N Engl J Med 2009; 361(17): 1639-1650.
4. Owiredu WKBA, Quaye L, Amidu N, Addai-Mensah O. Renal insufficiency in Ghanaian HIV infected patients: need for dose adjustment. African Health Sci 2013;(1):101-111.
5. Weiner DE, Tighiouart H, AMIN MG. Chronic Kidney Disease as a Risk Factor for Cardiovascular Disease and All-Cause Mortality: A Pooled Analysis of Community-Based Studies. J Am Soc Nephrol 2004;15: 1307–1315.
6. Hwang KY, Schwartz BS, Lee BK, et al. Associations of lead exposure and dose measures with erythrocyte protein kinase C activity in current Korean lead workers. Toxicol Sci 2001; 62(2):280–88.
7. Singh SK, Malik, A, and Jha V. CDKD: a clinical database of kidney diseases. BMC Nephrol 2012;13 (23):1-12.
8. Zadrazil J. Aetiology and a clinical picture of chronic renal failure. Vnitr Lek 2011;57(7-8): 607-13.
9. Jennette LE, S Vupputuri S, Hogan1 SL, Shoham DA. Community perspectives on kidney disease and health promotion from at-risk populations inrural North Carolina, USA. Rural and Remote Health 2010; 10: 1-10.27.
10. Guttmann D, Poage G, Johnston T. Reduction with glutathione is a weakly mutagenic pathway in chromium(VI) metabolism Chem Res Toxicol 2008;21(11): 2188–2194.
11. Hsu CY, Ordoñez JD, Chertow GM, Fan G. The risk of acute renal failure in patients with chronic kidney disease. Kidney Int 2008; 74(1): 101–07.
12. Sabolic I. Common mechanisms in nephropathy induced by toxic metals. Nephron Physiol 2006; 104: 107- 14.