ASSOCIATION OF BLOOD LEAD WITH KIDNEY DISEASE IN A GROUP OF EGYPTIAN PATIENTS WITH UNEXPLAINED CHRONIC KIDNEY DISEASE.

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INTRODUCTION

- Although the most common causes of CKD in Egypt are diabetes and hypertension, it is important to also investigate for the presence of other causes, especially those with potential reversibility. For many patients, the cause of their underlying kidney disease is multifactorial or unrecognized[1].
- The association between body lead burden and kidney disease remains controversial[1]. There is good anecdotal evidence that lead nephropathy may develop after high and prolonged exposure[1,2]. However, data indicating that lead causes chronic kidney disease (CKD) and the development of end-stage renal disease (ESRD) are scarce[3]. Likewise, the possible effect from lead exposure on the progression of kidney disease is unclear[4].
- The **goal** of the current study was to determine the impact of environmental lead exposure on the development of chronic kidney disease (CKD) and end-stage renal disease (ESRD).

METHODS

- This cross-sectional study was performed in 70 egyptian patients with **unexplained** renal disease divided into 2 groups; 35 ESRD patients on regular hemodialysis, and 35 CKD patients as well as 40 age- and sex-matched subjects without known renal disease served as a control group.
- Patients with a previous history of lead poisoning were excluded. Other specific renal diagnoses were excluded based on the patient history, laboratory results, renal imaging and renal pathology.
- Besides **sociodemographic** (residence, smoking, occupation and education), **clinical** (BP, BMI) and **laboratory** (Hb, Cr, eGFR, PTH, Ca, and Pi) data, blood lead (PbB) was measured via atomic absorption spectrophotometry with a detection limit of 3 µg/dL.

RESULTS

Table (1): Clinical and laboratory characteristics of study population

	ESRD	CKD	Control	P –value*
Age (y) (Mean ± SD)	45.6 ± 16	44.5 ± 14	45.7 ± 16	> 0.5
Sex (% males)	51.4	57.1	47.5	> 0.5
High school education	11(31.4%)	13(37.1%)	15(37.5%)	>0.05
Current smoker	17 (48.6%)	15 (42.9%)	13 (32.5%)	> 0.5
High risk occupation	6(17.1%)	3(8.6%)	0 (0 %)	<0.001
Industrial/traffic residence	17 (48.6 %)	10 (28.6 %)	8 (20 %)	<0.001
BMI (kg/m²)	26.64 ± 3.8	29.1 ± 5.1	26.26 ± 4	> 0.5
Hb (g/dl)	9.1 ± 1.8	10.2 ± 1.8	11.8 ± 1.9	<0.001
Creatinine (mg/dl)	8.6 ± 1.8	2.1 ± 0.9	0.7 ± 0.2	<0.001
eGFR (ml/min)	< 15	37.2 ± 3	108.9 ± 20	<0.001
PTH (pg/mL)	417 ± 225	251 ± 99	34.4 ± 15	<0.001
Ferritin (ng/mL)	369 ± 166	234 ± 87	131 ± 56	<0.001
Blood lead (µg/dL)	14.5 ± 6.6	11.2 ± 5	5.8 ± 2.8	<0.001

**p-value is significant if < 0.05*

Mean blood lead levels were significantly higher among ESRD (14.5 ± 6.6 µg/dl) and CKD (11.2 ± 5 µg/dl) groups compared to control group (5.8 ± 2.8 µg/dl; P < 0.001). Fig. (2) shows the distribution of blood lead into pre-defined levels for ESRD and CKD cases in comparison to age- and sex-matched controls. In CKD group, PbB showed significant positive **correlation** with blood pressure (BP), sCr and parathormone (PTH); and negative correlation with eGFR. The multiple **regression** analyses revealed male gender, industrial residence, smoking, PTH and ferritin as significant independent predictors for increased blood lead. After multivariate adjustment, compared to their counterparts with blood lead less than 10 µg/dL, the odds ratio of chronic kidney disease for persons with a blood lead level ≥ 10.0 µg/dL was 16.2 (95 % confidence interval: 5.2, 50.8).

RESULTS (cont'd)

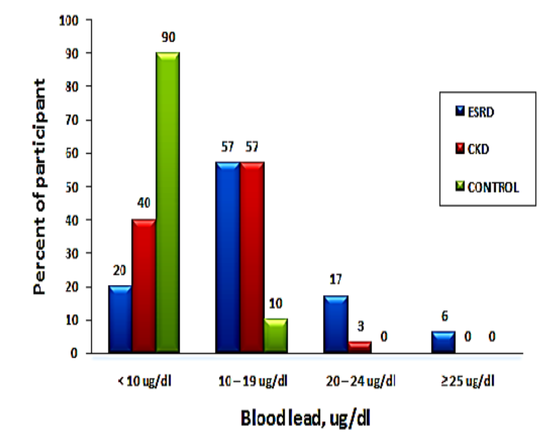

Fig.(2): Distribution of blood lead into a priori defined groupings for CKD and ESRD cases and age- and sex-matched controls

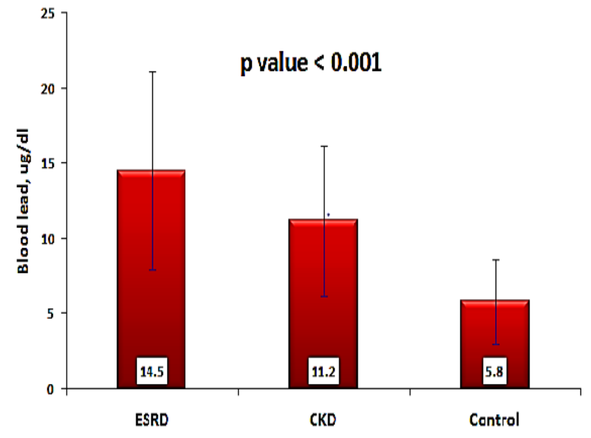

Fig. (1) : Blood lead level in different study groups

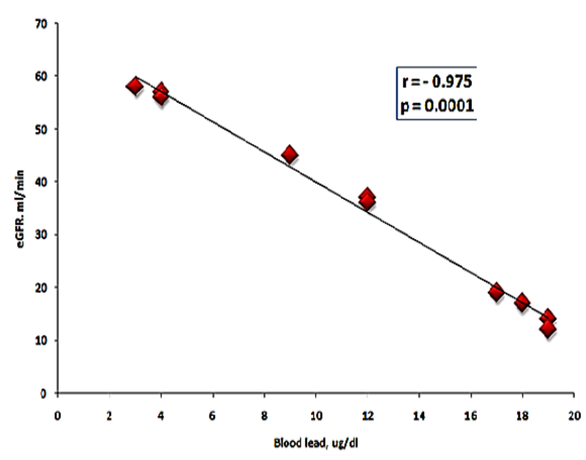

Fig. (4): Correlation of blood lead with eGFR in CKD cases

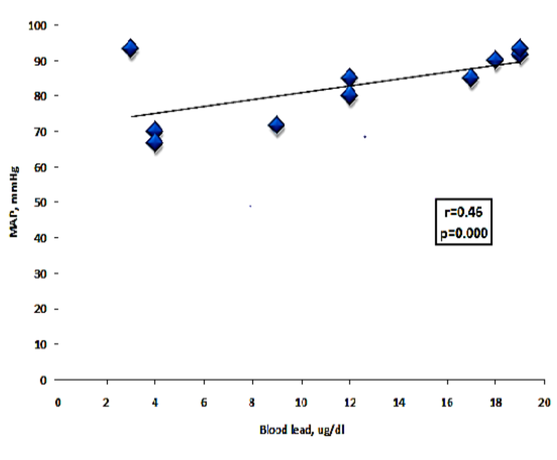

Fig. (3): Correlation of blood lead with MAP in CKD cases

DISCUSSION

- In this study, blood lead levels were strikingly higher among ESRD and CKD cases compared to their age- and sex-matched control counterparts.
- Our study showed that environmental lead exposure is a risk factor for lead nephropathy, as shown in cross sectional analysis and longitudinal data which suggest an acceleration of age-related impairment of renal function in association with long-term low-level lead exposure[4,5].
- Our study is **limited** by its cross-sectional design, so, it is difficult to make causal inference. Another limitation is small sample size. Additional limitation is the potential selection bias with the use of prevalent ESRD cases. Given the high rate of mortality associated with ESRD and an increased mortality at elevated blood lead levels, the association of elevated lead levels with ESRD incidence may be stronger than we report.

CONCLUSION

- Our cross-sectional study showed evidence for a link between PbB and different eGFR levels; and suggest that more patients with unexplained CKD might have been adversely affected by environmental exposure to lead.
- Additional well-controlled prospective longitudinal studies of patients with chronic kidney disease that include the measurement of bone lead are needed to better characterize this association. Given the potential availability of therapeutic interventions, understanding the causal impact of lead exposure on renal disease has important public health relevance.

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