The Relationship between Serum Aluminum Level and some neurological diseases in Human

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Abstract

This study was performed to explorealuminium exposure in healthy individuals, andoccupationally exposed populations compared with neurological diseases patients. The results indicate that a significant differences were found between aluminium concentration in sera of healthy individuals compared with occupationally exposed populations ($p<0.01$). No significant difference($p>0.05$) was observed in occupationally exposed populations compared with Parkinson's disease victims. Serum aluminium levels were much higher in Alzheimer's disease victims compared with aluminiumplants workers ($p<0.01$). The results indicate that aluminium is not a risk factor to healthy non-ccupationally exposed populations, while the adverse effects were observed in occupationally exposed populations, and neurological disease victims.

((العلاقة بين مستوى الألمنيوم في مصل دم الإنسان مع بعض الأمراض العصبية))

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مفتاح البحث: الألمنيوم و الأمراض العصبية

الخلاصة

تم انجاز هذه الدراسة لتوضيح علاقة التعرض للألمنيوم بين الأفراد الأصحاء غير المعرضين والأفراد المعرضين للألمنيوم مقارنةً مع مرضى الأمراض العصبية. النتائج أشارت إلى وجود فروقات معنوية ملحوظة بين تركيز الألمنيوم في مصل الدم للأفراد الأصحاء عند مقارنته بمصل دم العاملين في مصانع الألمنيوم ($P<0.01$) بينما لم تكن هناك أي فروقات معنوية ($P>0.05$) بين الأفراد العاملين في مصانع الألمنيوم عند مقارنتها بمرضى الشلل الرعاش. أوضحت النتائج بأن مستويات الألمنيوم في مصل دم مرضى الخرف المبكر أعلى من العاملين بمصانع الألمنيوم($P<0.01$)، كما أشارت النتائج بأن عنصر الألمنيوم لا يشكل عامل خطر على الأفراد الأصحاءغير المعرضين له بينما لوحظت التأثيرات عكسية بين العاملين في المصانع ومرضى الجهاز العصبي.

Introduction

Aluminium is a non-essential trace element of ubiquitous distribution. It may cause adverse effect on the nervous system [1,2] and can also cause anemia, osteomalacia and cardiac arrest. [3,4]Aluminium may be found in manyproces- sed food, cosmetics, tooth paste, antiperspirants and adjuvants in various parenteral preparations [5.6]. Foods naturally high in aluminium include baked potato, barley, corn, okra, cucumbers, eggplant [7,8]. Moreover, spinach dried spices thyme and particularly tea leaves [1,3,9]. The main source of aluminium that is able to enter the blood is drinking waters.

Aluminiumsulphate is often added to remove the brownish colour from water [9,10,11].Alumin- ium levels in blood and urine have been used to determine exposure levels. Current reference intervals for healthy individuals are estimated to be (1-5 µg /100ml) for serum or plasma and (3 – 8 µg/L) for urine while above this amount was found in dialysis, encephalopathy and este-omalacia patients[9,12,13]. It has been implicated as an etiological factor in some pathologies inc-luding Alzheimer's disease (AD), Parkinsonism dementia (PD), seizure amyotrophic lateral scl-erosis, encephalopathy and osteomalacia[10,14,15]Aluminium may accumulate in the brains of AD victims because the disease causes a defect in the normally protective blood brain barrier which makes the brain a willing host for alum- inium deposition [16,17].

The aim of this study is to determine the serum aluminium levels in healthy individuals, and occupationally exposed populations, comp-ared with patients suffering from a number of neurological disorders .

Patients and Methods

A total of 160 individuals were chosen from three Iraqi governorate(Baghdad, Salahddin and Thi-Qar). These were divided into three groups: 1- Control group: included 52 healthy individu-als from several regions in Baghdad, Tikrit and Al- Nassiriyah. Their ages ranged from 18-73 year.

2- Aluminium manufactories workers:-

included 58 Workers , 33 of them werechosen from Al-Nassiriyahaluminium manufactory and the other 25 were chosen from aluminium manufactories in Jamela quarter in Baghdad . Their age s ranged from 20-65 year.

3-Patientsgroups:

3-1 Seizure patients: included 25 patients from Salahddin governorate. Their age s ranged from 17-63 year.

3-2 Neurodegenerative patients: included 25 patients. They were evaluated and chosen as victims of Alzheimer's and Parkinson's diseases according to their past medical history. Their ages ranged from 25-70 year. 5 ml of venous blood sample was drawn. This blood was trans-ferred to disposable polypropylenetubes and allowed to clot. After centrifugation at 3000 rpm for15 minutes, the serum was removed and placed in collection tubes. Sera samples were stored frozen at – 20^{o}C till the time ofanalysis. Aluminium was determined by using calibrat-ion curve method. A shimadzu model AA-680G,atomic absorption spectrometer equipped with a shimadzu GFA-4B graphite furnace ato-mizer was used in this method. 5,10,15 and 20 µg/100ml of aluminium standard solutions were prepared daily. Atomization of the above solut-ions were carried out according to the optimum instrumental parameters recommended. Calibr-ation curve was established by plotting alumini-um absorbance against its concentration. Stati-stical analysis was used to find out the signify-cance of the results according to the p value.

Results:

Table 1 shows that aluminium concentration expressed as µg /100ml of aluminiummanuf-actories workers are significantly higher than healthy individuals.The means of aluminium concentration in sera of healthy individuals and aluminium manufactories workers were 3.55 and 23.81 µg/100mlrespectively.

A statistical significant elevation ofalumini-um concentration in sera of seizure disease vict-ims (15.52 µg/100ml, compared with healthy individuals 3.55 µg/100ml), (table 2). Also, a statistical significant elevation of aluminium concentration was observed in sera of neurode-generative diseases victims (39µg/100ml) com-pared with healthy individuals (3.55 µg/100ml), (tables 3). No relationship was found between serum aluminium, gender and ages.

Discussion

The results suggest that aluminium is not a risk factor for healthy non-occupationally exposed humans. An increasing serum aluminium levels in µg/100ml, were observed in sera of occupan-tionally exposed population (23.8 ± 6.08) com-pared with seizure disease victims (15.52 ± 6.02), ($p<0.01$) and this suggests that workers having long-term exposure to aluminium may be at increased risk of adverse health effects. These results are in agreement with the litera-ture[18], It is likely that high aluminiumconce-ntration in sera of neurodegenerative diseases victims might be implicated in the pathogenesis of Alzheimer and Parkinson's diseases. The res-ults are in agreement with the results of Kilbu-rn[13], who reported that workers in aluminium plants exhibited neurobehavioral impairment and symptoms of pulmonary toxicity. These results are supported by Walton study which states that increased blood aluminium is the only route by which ingested aluminium can bioaccumulate in the human central nervous system, contributing to development of Alzhei-mer's diseases in some people [19]. The positive relationship between elevated serum aluminium levels and seizure disorders, can not be totally dismissed.

Conclusions:

Aluminium has not been shown to pose a heal-th risk to healthy, non occupationally exposed humans. However workers having long term occupational exposure to aluminium may be at increased risk of adverse health effects. It is lik-ely that highaluminium concentration in sera of neurodegenerative diseases victims, might be implicated in the pathogenesis of Alzheimer and Parkinson's diseases. The positive relation-ship between elevated serum aluminium levels and seizure disorders, can not be totally dismi-ssed.

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(Table 1)
Aluminium concentration in sera of healthy individuals and aluminium manufactories workers (µg / 100 ml).

Statistical parameters	Healthy individuals	Aluminium manufactories workers
Sample size	$n_1 = 52$	$n_2 = 58$
Means	$X_1 = 3.55$	$X_2 = 23.81$
Standard deviation	$S_1 = 4.47$	$S_2 = 6.08$
t-test	14.04	
Probability	($p<0.01$) Highly significant	

(Table 2)
Aluminium concentration in sera of healthy individuals and seizure disease victims (µg / 100 ml)

Statistical parameters	Healthy individuals	Seizure patients
Sample size	$n_1 = 52$	$n_2 = 25$
Means	$X_1 = 3.55$	$X_2 = 15.52$
Standard deviation	$S_1 = 4.47$	$S_2 = 6.02$
t-test	5.22	
Probability	($p<0.01$) Highly significant	

(Table 3)
Aluminium concentration in sera of healthy individuals and neurodegenerative diseases victims (µg / 100 ml).

Statistical parameters	Healthy individuals	Neurodegenerative patients
Sample size	$n_1 = 52$	$n_2 = 25$
Means	$X_1 = 3.55$	$X_2 = 39.3$
Standard deviation	$S_1 = 19.1$	$S_2 = 6.02$
t-test	8.45	
Probability	($p<0.01$) Highly significant	