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Determination of blood indices of albino rats treated with aluminum chloride and investigation of antioxidant effects of vitamin E and C

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Abstract

The current study aims to investigate hematological and biochemical blood indices of albino rats administrated aluminum chloride ($AlCl_3$) for eight weeks, and to study the therapeutic effects of vitamin E and C. $AlCl_3$ decreased the total red blood cell count (by 18%), hemoglobin (7%) and hematocrit (20%), and increased white blood cell count (67%), lymphocytes (29%), mean corpuscular volume (14%), mean corpuscular hemaglobin (6%) and platelets (33%). Administration of vitamin E with or without vitamin C failed to restore levels of red blood cell counts, hematocrit, mean corpuscular volume, mean corpuscular hemaglobin or platelets, but vitamin E on its own restored levels of white blood cells, hemaglobin and lymphocytes.

$AlCl_3$ decreased serum glucose levels by 30%, and increased triglyceride (28%) and cholesterol (20%) levels; neither vitamin treatments restored the levels of these components. $AlCl_3$ increased levels of urea (12%), uric acid (77%) and creatinine (25%) compared to the controls, and vitamin E separately or together with vitamin C restored the levels of these nitrogen compounds.

The activities of alanine aminotransferase, alkaline phosphatase, and aspartate aminotransferase were also increased by the $AlCl_3$ treatment; the first two but not aspartate aminotransferase were restored by vitamin E separately or together with vitamin C.

We conclude that vitamin E separately or together with vitamin C suppressed cytogenetic injury and damage to some biochemical pathways of rat organs induced by $AlCl_3$.

Keywords: albino rats, aluminum chloride, blood indices, rat organs, vitamin E, vitamin C.

Introduction

Aluminum is a well-known toxic agent and represents a severe problem in a variety of medical (Nicolini *et al.* 1992) and environmental situations (Meranger 1989). The evidence implicating aluminium as a neurotoxin has been continuously mounting. Research on both animals and humans has linked it with neurocognitive dysfunction and in some cases death (Rifat *et al.* 1990). The major sources of aluminum include air, food and water (Michel 1990), and the gastrointestinal tract constitutes the main route of entry into the body. However, the absorption rate is low in normal human subjects (Brown *et al.* 1986).

Aluminum hydroxide, administered therapeutically in large quantities as an antacid and phosphate binder has been suggested to contribute to aluminum accumulation and toxicity (Lione 1985). Chronic exposition can cause alterations in skeletal, nervous, hematopoietic and respiratory systems (Chen *et al.* 2002; Cambell 2002). Blood urea is the principal end product of protein catabolism and a good indicator of kidney function. Uric acid is the end product of catabolism of purine bases; increased concentrations in the blood over the normal range might be due to extra degradation of purines in the liver, or an inability to excrete uric acid by the kidneys (Varely 1987). Creatinine appears in the serum in amounts proportional to the body's muscle mass and is more readily excreted by the kidneys than urea or uric acid (Pevicharova *et al.* 1997).

Blood enzymes are normally found in small amounts in circulation because of normal tissue turnover. Alanine aminotransferase as a liver enzyme significantly elevates in hepatobiliary disease, but also in connection with damage to the heart or skeletal muscle as well as liver parenchyma. Alkaline phosphatase is present on the cell surfaces in most human

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tissues, and belongs to a group of enzymes that catalyze the hydrolysis of phosphomonoesters at alkaline pH. High activity is found in the intestine, liver, bone, spleen and kidneys (Stryer 1995). Aluminum ions alter the properties and structure of cellular membranes, inhibiting many enzymes (Platt *et al.* 2001; Abreo & Glass 1993), and can act as antagonists for other elements such as calcium, magnesium, iron, silicon, phosphorus, copper and zinc (Ward *et al.* 2001).

Vitamin C is essential for the formation of collagen and intracellular material, bone, teeth and for the healing of wounds. It helps maintain elasticity of the skin aids the absorption of iron and improves resistance to infection. Vitamin E is the primary liposoluble antioxidant, perhaps important in scavenging free oxygen radicals and in stabilizing cell membranes, maintaining permeability (Packer 1993). Antioxidants such as vitamins E and C, coenzyme Q, glutathione and selenium ions can act synergistically, preventing lipid peroxidation and cell destruction (Escott-Stump & Mahan 2000).

The Gaza strip was exposed to Israeli bombing from Dec 27 2008 to Jan 18 2009, resulting in high concentrations of heavy metals. Such environmental contaminants can be transmitted to humans, causing many health complications (Manduca *et al.* 2009). Although many studies have been carried out on the toxic effect of aluminium ions and the antioxidant effects of vitamin E and C (Hayes *et al.* 2001; Eastmond *et al.* 2001; Manduca *et al.* 2009; Al-Faisal 2010), their effects on the body at a molecular level are still controversial. The present study investigates the different effects of $AlCl_3$ on blood indices of albino rats, and the subsequent response of rat tissues to therapeutic actions of vitamins E and C.

Materials & Methods

The study design involved one control and three treatment groups. It used 24 adult male albino rats, each weighing 100-120 gm, purchased from the breeding unit of the Biology Department, IUG. They were kept in plastic cages with wire mesh covers for one week before experimentation, and then divided in groups of six into one control and three treatment groups. Group one was administered 40 mg/l $AlCl_3$ dissolved in the drinking water (Fyiad 2007); group two was given 40 mg/l $AlCl_3$ plus vitamin E (150 mg/kg) (El-Nahas 1993); and group three had 40 mg/l $AlCl_3$ plus vitamins E and C (150 mg/kg) (El-Nahas 1993). Commercial balanced diet and water were continuously and regularly supplied *ad libitum* to the animals throughout the experimental period. The duration of the experiment was 8 weeks, when blood samples were collected from the jugular vein for hematological and biochemical examination.

Routine hematological parameters and a complete blood count was carried out using an automated 18-parameter hematology analyzer (ABX Micros 60, Horiba ABX, France). Clear serum samples were separated by centrifugation at 3000 rpm. for 20 min, collected and stored in a deep freeze at (-20 ^{0}C) for biochemical analysis. Glucose, triglyceride and cholesterol were determined using classical methods described by Trinder (1979), Fossati & Prencipe (1982) and Allain *et al.* (1974), respectively. Serum urea measurement was based on cleavage of urea with urease (Fawcett & Scott 1960). Serum uric acid was determined according to Fossatti *et al.* (1980). Serum creatinine was measured without protein precipitation according to Bartels *et al.* (1972). Activities of serum aspartate aminotransferase and alanine aminotransferase were determined according to the classic method of Reitman & Frankel (1957); measurement of serum alkaline phosphatase activity was also based on the method of Bessey *et al.* (1946).

Data were analyzed using SPSS version 13 for Windows. ANOVA was used to test for differences among groups; differences were considered significant if $p < 0.05$.

Results

Table 1 summarizes the effect of $AlCl_3$ and vitamins C and E on hematological parameters. After eight week of $AlCl_3$ administration, there was significant decrease in the total red blood cells, red blood cells, hemoglobin and hematocrit compared to the control. In contrast, white blood cells, lymphocytes, corpuscular volume, corpuscular hemoglobin and platelets showed a significant increase compared to the control. There was a non-significant increase in corpuscular hemoglobin concentration. Administration of vitamin E alone, or with vitamin C, failed to counteract the effect of $AlCl_3$ on red blood cells, hematocrit, corpuscular volume, corpuscular hemoglobin, corpuscular hemoglobin concentration and platelets. Vitamin E alone counteracted the effect of the ion on white blood cells, lymphocytes and hemoglobin.

Parameter	**control**	**$AlCl_3$**	**$AlCl_3$ + vitamin E**	**$AlCl_3$ + vitamins E & C**
White blood cells (x 10^3 cell/µl)	$3.90^b \pm 0.19$	$6.5^a \pm 0.24$	$4.10^b \pm 0.36$	$5.85^a \pm 0.29$
Lymphocytes	$60.6^b \pm 2.5$	$78.2^a \pm 3.0$	$62.6^b \pm 3.2$	$75.9^a \pm 2.3$
Red blood cells (x 10^6 cell/µl)	$11.19^a \pm 0.20$	$9.14^b \pm 0.35$	$9.25^b \pm 0.30$	$9.51^b \pm 0.31$
Hemoglobin (g/dl)	$16.26^a \pm 0.70$	$15.18^b \pm 0.18$	$16.18^a \pm 0.19$	$15.45^b \pm 0.20$
Hematocrit (%)	$65.9^a \pm 1.1$	$53.0^b \pm 1.2$	$52.5^b \pm 1.3$	$51.6^b \pm 1.3$
Corpuscular volume (fi)	$14.5^c \pm 0.2$	$16.6^b \pm 0.2$	$17.4^b \pm 0.3$	$16.2^b \pm 0.2$
Corpuscular hemoglobin (pg)	$27.14^b \pm 0.20$	$28.65^a \pm 0.16$	$30.84^a \pm 0.19$	$29.97^a \pm 0.23$
Corpuscular hemoglobin concentration (g/dl)	$53.52^b \pm 0.25$	$57.95^a \pm 0.31$	$56.70^a \pm 0.36$	$54.20^a \pm 0.42$
Platelets (x 10^3/µl)	$595.1^c \pm 23.3$	$790.0^a \pm 25.2$	$731.8^b \pm 31.3$	$723.0^b \pm 33.9$

Table 1: Hematological indices of the rats administrated $AlCl_3$, vitamin E and vitamin C (all values expressed as mean ± SE). Means with different subscripts in the same row differ significantly ($p<0.05$).

Table 2 shows changes in glucose, triglycerides and cholesterol concentrations in the experimental groups. Aluminium treatment significantly decreased serum glucose levels (by 30%) and increased significantly triglycerides (28%) and cholesterol (20%). Treatments with vitamin E alone or with vitamin C did not restore these compounds to control levels.

Parameter	**control**	**$AlCl_3$**	**$AlCl_3$ + vitamin E**	**$AlCl_3$ + vitamins E & C**
Glucose (mg/dl)	$95.2^a \pm 3.1$	$66.8^b \pm 2.2$	$68.8^b \pm 2.2$	$67.5^b \pm 2.3$
Triglycerides(mg/dl)	$88.5^b \pm 2.5$	$113.3^a \pm 2.3$	$105.7^a \pm 3.4$	$103.7^a \pm 2.2$
Cholesterol (mg/dl)	$129.1^c \pm 2.3$	$155.0^a \pm 3.2$	$152.2^a \pm 3.3$	$143.6^b \pm 2.2$
Urea (mg/dl)	$25.3^b \pm 0.1$	$28.3^a \pm 1.1$	$26.5^b \pm 1.2$	$25.4^b \pm 2.1$
Uric acid (mg/dl)	$3.51^b \pm 0.20$	$6.21^a \pm 0.26$	$3.77^b \pm 0.24$	$4.11^b \pm 0.25$
Creatinine (mg/dl)	$0.60^b \pm 0.01$	$0.75^a \pm 0.02$	$0.65^b \pm 0.01$	$0.62^b \pm 0.02$
Alanine aminotransferase (IU/ml)	$25.5^b \pm 0.3$	$30.8^a \pm 0.4$	$28.1^b \pm 0.3$	$27.4^b \pm 0.3$
Aspartate amino transferase (IU/ml)	$21.9^c \pm 0.4$	$31.4^a \pm 0.4$	$28.7^b \pm 0.5$	$29.7^b \pm 0.6$
alkaline phosphatase (IU/ml)	$81.5^b \pm 0.6$	$100.1^a \pm 4.3$	$90.1^b \pm 2.2$	$88.2^b \pm 2.2$

Table 2: Chemical concentrations in albino rats administrated $AlCl_3$, vitamin E and vitamin C (all values expressed as mean ± SE). Means with different subscripts in the same row differ significantly ($p<0.05$).

Levels of non-protein nitrogenous constituents for treatment groups are also given in Table 2. The aluminium treatment significantly increased urea (by 12%), uric acid (77%) and creatinine

levels (25%) compared to the control. Vitamin E separately or together with vitamin C significantly counteracted the effects of aluminium.

Activities of serum aspartate amino transferase, alanine aminotransferase and alkaline phosphatase (Table 2) increased significantly following aluminium treatment. As for the nitrogenous compounds, these increases were counteracted by treatment with vitamin E alone or together with vitamin C. In contrast, aspartate amino transferase activity was not counteracted at all.

Discussion

This study aimed to determine the toxic effects of the aluminium ion, and the therapeutic effects of vitamin E and vitamin C, on rats. We found highly significant decreases in hemoglobin, red blood cells and hematocrit among aluminium-treated rats, as have others (Karmaker *et al.* 2000). The reduction in hemoglobin content might be due to increased rate of destruction or reduction in the rate of formation of red blood cells. This intepretation was supported by the low levels of red blood cells in the treated groups. Reductions in hematocrit, red blood cells and hemoglobin might be attributed to hyperactivity of bone marrow, leading to production of red blood cells with impaired integrity that are easily destroyed in the circulation (Karmaker *et al.* 2000). On the other hand, these decreases could alternatively reflect a lower oxygen supply to different tissues, resulting in low energy production in the rats. The decrease in hemoglobin could be not only due to decrease in red blood cells count but also to impaired biosynthesis of heme in the bone marrow.

The significant increase in white blood cell levels of aluminium-treated rats might indicate activation of the immune system, a normal cell-mediated immune response (El-Demerdash 2004). The increase in lymphocytes could be due to the toxic action of the aluminium ion that stimulates the hemopoietic system to release more of these cells, causing an increase in their number in the blood stream.

The increase in corpuscular volume, corpuscular hemoglobin, corpuscular hemoglobin concentration and platelets were consistent with changes in red blood cell counts and hemoglobin levels. These changes may be correlated with some pathological changes developed in blood-forming organs, or with the destruction of red blood cells, or with both factors. In this regard, from similar results Naylor (1971) concluded that anemia resulted from hemodilation, extra vascular hemolysis and toxic dyshemopiosis.

Our findings show that vitamin E on its own counteracted the effects of the aluminium ion on white blood cells, lymphocytes and hemoglobin. Vitamin E and vitamin C separately increase the activities of antioxidant enzymes in various tissues of rats, especially liver tissues and also vitamin E on bone marrow, where the different blood cells are formed (Shireen *et al.* 2008).

Our findings also revealed a decrease in serum glucose levels in response to aluminium. Indirectly, aluminium is known to play a specific role in carbohydrate metabolism (Thirunavukkarasu & Sakthisekaran 2003). Concerning lipid metabolism, our results demonstrated that triglycerides and total cholesterol levels increased in response to aluminium, consistent with increasing lipogenesis in the liver (Thirunavukkarasu & Sakthisekaran, 2003). Vitamin E separately or together with vitamin C could not counteract the effects of the aluminium ion on glucose, cholesterol and triglycerides. This indicates that vitamin E is only indirectly involved in metabolism of these compounds via defending the integrity of cells against oxidating agents. Vitamin E mediates mitochondrial superoxide generation, suggesting a possible mode of action at tissue level, and it also modulates the expression and/or activation of redox-sensitive biological response modifiers that regulate important cellular events (Chow 2004).

Enhanced protein catabolism and accelerated amino acid deamination in response to low glucose levels caused by aluminium ion administration is the best interpretation for the elevated levels of urea. The presence of toxic compounds can increase blood urea and decrease plasma protein (Berne & Levy 1998). The observed increase in uric acid concentration might be due to extra degradation of purines in the liver, or an inability to excrete uric acid by the kidneys (Varely 1987). An increase in creatinine has been seen, interpreted as caused by a decrease in muscle mass (Pevicharova *et al.* 1997) or abnormal glomerular function of the kidneys induced by $AlCl_3$ administration (Berne & Levy 1998). The observation that vitamin E separately or together with vitamin C counteracted the toxic effects of the aluminium ion in forming these nitrogenous compounds indicates that the vitamins reverse and thus inhibit interactions with metabolic enzymes involved in their synthesis in the liver and muscles.

Serum transaminases (aspartate amino transferase and alanine aminotransferase) and alkaline phosphatase exhibited a significant increase in treated rats, perhaps indicating persistent cellular injury (Bansal *et al.* 2005). Elevated activities of serum transaminases could be a sign of impaired liver function. Alkaline phosphatase has a specific location within both sinusoidal and bile canalicular membranes, accounting for its more predominant elevation in certain disorders (Bansal *et al.* 2005): acute cell necrosis liberates alkaline phosphatase into the blood circulation and its level is elevated. As with biosynthetic enzymes of nitrogen compounds, vitamin E separately or together with vitamin C reversed the toxic effects of aluminium ions on the activities of alkaline phosphatase and alanine aminotransferase, but not aspartate amino transferase. Their effect on aspartate amino transferase requires further investigation.

We conclude that aluminum ions significantly decrease red blood cell counts, hemoglobin, hematocrit and glucose, and significantly increase white blood cell counts, lymphocytes, corpuscular volume, corpuscular hemoglobin, corpuscular hemoglobin concentration, platelets, triglycerides, cholesterol, urea, uric acid, creatinine and the activity levels of alanine aminotransferase, aspartate amino transferase and alkaline phosphatase. Vitamin E on its own counteracts the effect of $AlCl_3$ on white blood cell counts, hemoglobin and lymphocytes. Vitamin E alone or together with vitamin C counteracts the effects of aluminium ions on urea, uric acid, creatinine and on the activities of alanine aminotransferase and alkaline phosphatase. Consequently, vitamin E separately or together with vitamin C suppresses cytogenetic injuries and damage to some biochemical organ pathways (e.g. liver, kidney, bone marrow) induced by $AlCl_3$ administration.

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الملخص العربى

تحديد المؤشرات الخاصة بدم الفئران البيضاء المعالجة بكلوريد الألومنيوم للتحقق من الآثار المضادة للأكسدة لفيتامينى (ج) و (هـ)

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الملخص العربي

تهدف الدراسة الحالية إلى التحقق من المؤشرات الدموية والكيميائية الحيوية لدم الفئران البيضاء المعاالجة بكلوريد الألومنيوم (AlCl3) لمدة ثمانية أسابيع لدراسة الآثار العلاجية لفيتاميني (ج) و (هـ). يؤدي كلوريد الألومنيوم إلى انخفاض عدد خلايا الدم الحمراء (بنسبة 18 ٪) والهيموجلوبين (بنسبة 7 ٪) والهيماتوكريت (بنسبة 20 ٪) ، كما يؤدي لزيادة عدد خلايا الدم البيضاء (بنسبة 67 ٪) والخلايا الليمفاوية (بنسبة 29 ٪) ومتوسط حجم خلايا الدم (بنسبة 14 ٪) ومتوسط المحتوى الهيموجلوبيني لخلايا الدم (بنسبة 6 ٪) أو الصفائح الدموية (بنسبة 33 ٪) . فشل فيتامين (هـ) عند إعطائه للفئران وحده أو مع فيتامين (ج) في استعادة المستويات الطبيعية لأعداد خلايا الدم الحمراء والهيماتوكريت ومتوسط حجم الخلايا وكذا متوسط المحتوى الهيموجلوبيني لخلايا الدم أو الصفائح الدموية، بينما نجح في استعادة المستويات الطبيعية لخلايا الدم البيضاء والهيموجلوبين والخلايا الليمفاوية.

عمل كلوريد الألمونيوم على خفض مستويات السكر في الدم بنسبة 30 ٪ وزيادة مستويات الدهون الثلاثية (بنسبة 28 ٪) والكولسترول (بنسبة 20 ٪) ولم يسهم العلاج بأياً من الفيتامينين في استعادة مستويات تلك المكونات، كما أسهم في زيادة مستويات اليوريا (بنسبة 12 ٪) وحمض اليوريك (بنسبة 77 ٪) والكرياتينين (بنسبة 25 ٪) للفئران المعالجة به مقارنةً بالفئران غير المعالجة، إلا أن فيتامين (هـ) على حدة أو بالاشتراك مع فيتامين (ج) قام باستعادة المستويات الطبيعية لهذه المركبات النيتروجينية.

من الملحوظ أن نشاط إنزيمات الألانين أمينوترانسفيريز والألكالاين فوسفاتيز والأسبارتيت أمينوترانسفيريز قد زاد نتيجة للمعالجة بكلوريد الألومنيوم، إلا أن أول إنزيمين (ما عدا الأسبارتيت أمينوترانسفيريز) قد تأثرا بالعلاج بفيتامين (هـ) وحده أو مع فيتامين (ج) مما أدى إلى إستعادة النشاط الطبيعي.

مما سبق فإننا نخلص إلى أن فيتامين (هـ) يمكنه بشكل منفصل أو مع فيتامين (ج) وقف الإصابات الخلوية والأضرار التي تلحق ببعض المسارات الحيوية لأعضاء الفئران والتي تتسبب فيها مادة كلوريد الألمونيوم.