

EFFECT OF OMEGA-3 AND MULTIVITAMINS ON ALUMINUM-INDUCED CHANGES IN SERUM AND TISSUE ENZYME ACTIVITIES IN RATS

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ABSTRACT

The present work was designed to study the effect of omega-3 and multivitamins on aluminum-induced changes in some tissue enzymes level in male albino rats. For the present work, seventy adult male rats were randomly divided into seven groups; fed on normal diet and treated as follows: Control group 1 supplied with tap water. Group 2 supplied with 0.2 ml/rat of the vehicle (oil). Group 3 supplied orally with 0.2 ml/rat aluminum solution. Group 4 supplied orally with 0.2 ml/rat aluminum solution and diluted acetic acid solution (0.5%). Group 5 supplied orally with 0.2 ml/rat aluminum solution and omega-3. Group 6 supplied orally with 0.2 ml/rat aluminum solution and multivitamins. Group 7 supplied orally with 0.2 ml/rat aluminum solution and both antioxidants, omega-3 and multivitamins. After treating the animals with the tested materials daily for 30 days, the activities of serum alkaline Phosphatase (ALP), acid Phosphatase (ACP), Alanine Transaminase (ALT) and Aspartate Transaminase (AST) were increased while their activities in liver and brain tissues were significantly decreased. The toxicity of Aluminum was enhanced in the presence of acid as indicated by further changes in the levels of above parameters as compared with those of Al treated animals. The toxic effect of Al, in the presence of antioxidants such as omega-3 and multivitamins, was comparatively reduced and the studied parameters showed tendencies to change toward the normal levels. Thus, animals treated with Al in the presence of above antioxidants showed slight decreases in the activities of tissues ALP, ACP, ALT and AST. From the results of the present study, it can be concluded that the toxic effect of Al in the presence of acid was enhanced, whereas its toxicity was greatly reduced in the presence of the antioxidants such as omega-

Keywords: Aluminum, omega-3, multivitamins, ALP, ACP, ALT, AST and multivitamins individually or in combination.

INTRODUCTION

Aluminum (Al) is the third most abundant element on the earth's crust. Humans are exposed to Al from various environmental sources and interventions, e.g., ingestion of antacids, dialysis, using Al utensils....etc. This metal can also be encountered in some foods, dust, and other sources, including drinking water (Tripathi *et al.*, 2009). In human, the consumption of aluminum contaminated drinking water has been supposed to be the major environmental factor for the development of Alzheimer disease (AD) (Yokel, 2000). Defense mechanisms include: removal of Oxygen (O_2), scavenging reactive oxygen/nitrogen species (ROS) or their precursors, inhibition of reactive oxygen species ROS formation. Vitamin C is an essential micronutrient required for normal metabolic functioning of the body. Humans and other primates have lost the ability to synthesize Vitamin C as a result of a mutation in the gene coding for L-gulonolactone oxidase, an enzyme required for the biosynthesis of Vitamin C via the glucuronic acid pathway, thus, Vitamin C must be obtained through the diet (Carr and Frei, 1999). Vitamin E is the most potent antioxidant that can break the propagation of the free radical

chain reaction in the lipid part of the biological membrane. Among the antioxidants, Vitamin E has shown some promises in the treatment of AD (Vatassery, 1992). Omega 3 fatty acid from fish and fish oil can protect against chronic heart disease (CHD). Acetic acid, or its ionic form acetate, is also part of the normal cellular metabolism (Von Oettingen., 1960). Acetate enters naturally in to the metabolism of the body; it is absorbed from the gastrointestinal tract and through the lungs and almost completely oxidized by tissues (Von Oettingen., 1960). In the light of above information about the toxicity of Al and lack of detailed studies on the toxic effect of Al on various biological and biochemical studies, the present work was undertaken to study the protective effect of omega-3, multivitamins and their combination on Al-induced changes in serum and tissue enzymes.

MATERIALS & METHODS

Animals

During the present study, seventy adult male albino rats were used. The rats were 8-10 weeks of age with an average weight ranged between 150-170gm. The rats were placed in polypropylene cages (30 × 25 × 17 cm), reared

in animal house (University of Zakho) with free access to standard diet and water. The animals were maintained under laboratory conditions at about 24 °C and exposed to a photoperiod of 12\12 (L\D). Animals were acclimated to the laboratory condition for about 10 days before the application of experimental protocols. Rats were divided into seven groups, each of ten male individuals. Animals were treated as follow. In 1st group, animals were fed on standard diet and supplied with water *ad libitum*. In 2nd group, animals were supplemented with oral intubations of 0.2 ml oil/rat. The 3rd group animals were supplied orally with aluminum chloride AlCl₃ (20 mg/kg b.w). In 4th group, animals were supplied orally with AlCl₃ (20 mg/kg .b.w) plus 0.2 ml acetic acid (0.5%) per rats. In 5th group, animals were supplied orally with AlCl₃ (20 mg/kg b. w.) and 0.2 ml of 5% of Omega-3 (O3) dissolved in oil. In 6th group, animals were supplied orally with AlCl₃ (20 mg/kg b.w) plus 0.2 ml multivitamins solution (8.5 mg/100 ml). In 7th group, animals were supplied orally with AlCl₃(20 mg/kg b. w.) plus 0.2 ml multivitamins (8.5mg/100 ml and 0.2 ml omega-3 (5 %).. The doses of Aluminum, multivitamins, omega-3 and acetic acid were calculated according to the animal's body weight before dosing. The desired doses of multivitamins, omega-3, aluminum chloride and acetic acid for each animal was intubated into oesopharyngeal region, using small syringe connected to thin silicon tube. The tested materials were given daily for 30 days.

Preparation of serum

After thirty days of treatments, the rats were deprived from food for 24 hours, but left free access to water. The animals were anesthetized with Diethyl ether and blood samples were obtained directly by heart puncture 5ml of blood was collected in non-heparinized tube, dry clean centrifuge tubes, it was allowed to clot at room temperature for 30 minutes, then centrifuged at 3000 rpm for 15 minutes to separate the serum from the blood (Dacie and Lewis, 1984). Serum samples were placed in Eppendorf tubes and used for determination of ALT, AST, ACP and ALP activities.

Determination of ALT and AST

For the determination of tissue ALT and AST activity, 10 % (w/v) tissue homogenate was prepared from liver and brain in ice-cold of Tris Buffer (pH 7.4). The homogenate was

centrifuged at 10000 g for 10 minutes. The activities of ALT and AST in the supernatant was determined according to the colorimetric method of Reitman and Frankeusing Transaminases–Kits (Biolabo, France)

Determination Acid Phosphatase

For the determination of tissue ACP activity, 10% (w/v) tissue homogenate was prepared from liver and brain in ice-cold of Citrate Buffer (PH 4.8). The homogenate was centrifuged at 10000 g for 10 minutes. The ACP activities in the supernatant was determined by the method described by Fishman, and Richterich using ACP–Kit (Biolabo, France)

Determination of Alkaline Phosphatase

For the determination of tissue ALP activity, 10% (w/v) tissue homogenate was prepared from liver and brain in ice-cold of Carbonate-Bicarbonate Buffer (PH 10.5). The homogenate was centrifuged at 10000 g for 10 minutes. The supernatant was used to determine ALP activity according to the Optimized method based on the DGKC (German Society of Clinical Chemistry, 1972) using Alkaline phosphatases–Kit, (Biolabo- France).

Statistical analysis

Analysis of data was performed by using (Graphpad prism 5). Results were expressed as mean ± S.E.M. Statistical differences were determined by Dunnetts test for multiple comparisons after ANOVA (Dunnetts test treats one group as a control and compares all other groups against it)

P values <0.05 were considered statistically significant.

RESULTS

The mean values of the effects of treatment on the serum AST activity are shown in Table (1). Treatment of animals with Al and Al+ acid significantly (P<0.001) increased AST activity from 55.56 to 65.8 ± 1.3 and 66.8 ± 0.79, respectively. Furthermore, the treatment of animals with Al+Omega-3 and Al+ multivitamins reduced the toxicity as indicated by lower AST activity. However, in the treated rats, the activity of serum AST, was still higher but at lower significant levels (P<0.05), whereas, the activity of AST in animals treated with Al+omega-3+multivitamin was very close to its activity in control rat. In rats treated with Al and

Al + acid, the activity of ALT was significantly increased as compared with the control group. However, animals treated with omega-3,

multivitamins and their combinations reduced Al toxicity since the ALP activity was more or less similar to that of the control animals (Table 1).

Table (1) Shows the effect of aluminum on some serum enzyme activities

| Treatment Enzyme activity(IU) | Control | Oil | AlCl ₃ | AlCl ₃ +Acetic acid | AlCl ₃ +O ₃ | AlCl ₃ +Mv | AlCl ₃ +O ₃ +Mv |
|----------------------------------|-------------|--------------|-------------------|--------------------------------|-----------------------------------|-----------------------|---------------------------------------|
| AST | 55.56 ± 0.7 | 56.33 ± 1.17 | 65.8*** ± 1.3 | 66.8*** ± 0.7 | 60.2* ± 1.25 | 61.1** ± 0.76 | 56.38 ± 0.924 |
| ALT | 55.38 ± 0.5 | 51.8 ± 0.66 | 74.6*** ± 0.6 | 69.4*** ± 0.6 | 55.2 ± 0.59 | 55.00 ± 0.86 | 55.88 ± 0.833 |
| ALP | 56.5 ± 0.89 | 56.3 ± 1.017 | 76*** ± 0.9 | 67.6*** ± 0.98 | 60* ± 0.72 | 63** ± 0.48 | 58.43 ± 0.841 |
| ACP | 10.1 ± 0.2 | 10.15 ± 0.14 | 14.6*** ± 0.13 | 13.8*** ± 0.07 | 12.09** ± 0.3 | 11.66** ± 0.25 | 11.38** ± 0.159 |

* (P< 0.05) , ** (P< 0.01), *** (P< 0.001)

Serum ALP activity was significantly increased (P<0.001) in rats treated with Al and Al + acid to 76± 0.9 and 67.6 ± 0.98 was reduced, respectively. Furthermore, in animals treated with omega-3 and multivitamins along with Al, reduced its toxicity as indicated by a lower ALP activity as compared with Al treated groups. However, the ALP was still significantly higher than the control. On the other hand, in rats treated with a combination of omega-3 and multivitamins along with Al, ALP activity was more or less similar to that of the control rats. Treatment of rats with Al, Al + acid and with the addition of antioxidants significantly (P<0.01 - 0.001) increased the activity of ACP (Table 1). However, the activity of ACP was comparatively higher as compared with its activity in animals treated with antioxidants and their mean values were 12.09± 0.3, 11.66 ± 0.258 and 11.38 ± 0.159, respectively.

Table (2) shows the effect of aluminum without or with acid and antioxidants on liver and brain AST activity. In animals treated with Al without

or with acid, AST activity in the liver and brain was greatly reduced as indicated by the presence of highly significant difference (P<0.01-0.001) in AST activity compared with that of the control. On the other hand, the toxic effect of aluminum was greatly reduced as indicated by AST activity similar to that of the control. Statistical analysis the results revealed that there are no significant differences between the control and rats treated with Aluminum and antioxidants (P>0.05). Aluminum with or without acid produced a significant (P< 0.05-0.01) reduction in liver and brain ALT activity as compared with its activity in the control group (Table 3). However, the effect of Al in the presence of acid on liver and brain GPT activity was much more pronounced (P< 0.001) as compared with rats treated with Al alone. On the other hand, the effect of Al in the presence of omega-3, multivitamins and their combination reduced the toxic effect of liver and brain as indicated by ALT activity which was more or less similar to that of the control.

Table (2) Shows the effect of aluminum on AST activity on selected body organ

| Treatment | Control | Oil | AlCl ₃ | AlCl ₃ +Acetic acid | AlCl ₃ + O3 | AlCl ₃ + Mv | AlCl ₃ + O3+Mv |
|----------------|-----------------|------------------|--------------------|--------------------------------|------------------------|------------------------|---------------------------|
| AST (IU/100mg) | | | | | | | |
| Liver | 146.0±1.6 15 | 139.1±1.0 6 | 133.4* ±6.7 | 112.9***±1 .64 | 150.3±1.6 3 | 151.1±2. 17 | 148.1±2. 123 |
| Brain | 92.00±1.3 02 | 91.22 ± 3.601 | 75.00***±0 .601 | 71.22***±0 .43 | 93.3±1.11 8 | 92.78±1. 14 | 92.22±0. 4006 |

* (P< 0.05) , ** (P< 0.01), *** (P< 0.001)

Table (3) Shows the effect of aluminum on ALT activity on selected body organ

| Treatments | Control | Oil | AlCl ₃ | AlCl ₃ +Acetic acid | AlCl ₃ + O3 | AlCl ₃ + Mv | AlCl ₃ + O3+Mv |
|------------|-----------------|-----------------|-------------------|--------------------------------|------------------------|------------------------|---------------------------|
| ALT | | | | | | | |
| Liver | 155.3 ± 1.23 | 152.0 ± 0.77 | 150.1*± 0.76 | 149.9** ± 0.94 | 153.6 ± 1.15 | 156.1± 1.08 | 155.0 ± 1.50 |
| Brain | 153.4 ± 0.57 | 149.6 ± 0.42 | 123.7*** ± 0.9 | 126.7***± 0.64 | 151.7 ± 0.94 | 151.7 ± 0.94 | 152.3 ± 0.644 |

* (P< 0.05) , ** (P< 0.01), *** (P< 0.001)

Treatment with aluminum significantly (P<0.001) reduced liver and brain ALP activity (Table 4). The toxic effect of Al in the presence of acid was further enhanced the activity of liver and brain ALP. On the other hand, the toxic effect of Al in the presence of omega-3, multivitamins and their combination diminished the toxic effect as indicated by ALP activity which was more or less similar to that of the control rats. Liver and brain ACP activity showed significant (P< 0.01-0.001)

Table (4) Shows the effect of aluminum on ALP activity on selected body organ

| Treatment | Control | Oil | AlCl ₃ | AlCl ₃ +Acetic acid | AlCl ₃ + O3 | AlCl ₃ +Mv | AlCl ₃ + O3+Mv |
|----------------|------------------|------------------|-------------------|--------------------------------|------------------------|-----------------------|---------------------------|
| ALP (IU/100mg) | | | | | | | |
| Liver | 45.07 ± 0.614 | 43.42 ± 0.689 | 23.3***± 0.81 | 15.5***± 0.84 | 43.12 ± 0.271 | 43.30 ± 0.521 | 43.97± 0.437 |
| Brain | 39.38 ± 1.034 | 37.38 ± 0.565 | 30.25***± 0.83 | 23.7***± 0.81 | 40.25 ± 1.15 | 37.38± 0.88 | 39.13 ± 0.53 |

* (P< 0.05), ** (P< 0.01), *** (P< 0.001)

Reduction in ACP activity in rats treated with Al in the presence or absence of acid as compared with that of the control (Table 5). However, the toxic effect of Al was enhanced in the presence of acid. Al in the presence of omega-3 also caused a significant reduction in the liver ACP activity (P<0.01). On the other hand, multivitamins in the presence or absence of omega-3 diminished the toxic effect of Al and liver and brain and ACP activity was more or less similar to that of the control. Also the vehicle produced a mild, but statistically non-significant reduction in liver ACP activity (P>0.05).

Table (5) Shows the effect of aluminum on ACP activity on selected body organ

| Treatments | Control | Oil | AlCl ₃ | AlCl ₃ +Acetic acid | AlCl ₃ +O ₃ | AlCl ₃ +Mv | AlCl ₃ +O ₃ +Mv |
|---------------|-------------|-----------------|-------------------|--------------------------------|-----------------------------------|-----------------------|---------------------------------------|
| ACP(IU/100mg) | | | | | | | |
| Liver | 20.9±0.74 | 18.48 ± 0.92 | 15.1*** ± 0.61 | 12.33*** ± 0.89 | 16.6** ± 0.54 | 18.12 ± 1.20 | 19.85 ± 0.435 |
| Brain | 15.9 ± 0.79 | 15.44 ± 0.49 | 12.57** ± 1.34 | 11.20** ± 0.66 | 15.4 ± 0.54 | 15.22 ± 0.75 | 18.3 ± 0.65 |

* (P< 0.05) , ** (P< 0.01), *** (P< 0.001)

DISCUSSION

In animals treated with Al and Al with acid, significant elevation in serum ALT and AST activities were resulted from the disturbances produced in the plasma membrane integrity and a subsequent leak in the cellular enzymes. This in turn followed by significant reduction in the levels and activities in liver and brain tissue enzymes. These results are in agreement with those reported by Hassoun and Stohs (1995); Chinoy and Memon (2001) and El-Demerdash, (2004). They found that exposure to AlCl₃ caused liver necrosis and subsequent escape of AST from the liver to the plasma. Also elevation in ALT level indicates the presence of liver disease and as a result of cellular destruction increases the level of these enzymes in the blood (Harper *et al.*, 1979). Céline *et al.*, (2006) observed in animals treated with Al and acetic acid showed a drastic elevation in the levels of serum ALT and AST and decrease in the level of this enzyme in body organs (such as liver and brain).

Treatment with Al along with omega-3 and multivitamins reduced the toxic effect of Al on body organs and there was a tendency to return serum ALT and AST levels toward normal values. Furthermore, in rats treated with Al and a combination of above antioxidants caused great improvements in the body organs as indicated by the return of enzyme activities in the serum, and the body organs (liver and brain) to their normal values. This may be due to the lipids protection from peroxidation by antioxidants via the donation of their own electrons to free radicals and the subsequent break down of the chain reaction of oxidation. Once the free radicals gained electrons, it will produce no more harm or damage to the cellular components. This agree to some extent with the explanations given by Dekkers (1996). Furthermore, a similar reduction in the toxic effect of heavy metals in

the presence of antioxidants was reported by Abdel-Tawwab *et al.* (2007a).

Attia and Nasr (2009) observed in rats treated with antioxidants (omega-3 and selenium) along with paracetamol greatly reduced the toxic effect of the metal as indicated by healing of hepatic parenchyma and regeneration of hepatocytes.

Serum acid and alkaline Phosphatase activities in rats treated with Al without or with acid were significantly increased as compared with control. A similar variation in ACP and ALP activities in Al treated rats are in accordance with the finding of El-Demerdash (2004); Szilagy *et al.*, (1994); El-Sebae *et al.*, (1997) and Ochmanski and Barabasz, (2000). Szilagy *et al.*, (1994) referred to high levels of plasma ALP in Al-treated chicken to increased osteoblastic activity, provoked by disturbance of bone formation. Ochmanski and Barabasz, (2000) reported that Al may bind to RNA, DNA and inhibits the activities of both acid and alkaline Phosphatase. Furthermore, it has been suggested that the decreased acid and alkaline phosphatase activities in different tissues might be due to increased permeability of plasma membrane or cellular necrosis.

This increase in the phosphatase activity might be due to necrosis of the liver, kidney and lung (Yousef, 2004). The presence of Al along with omega-3 or/and multivitamins, reduced the toxic effect of Al on liver, kidneys and brain phosphatase activities, which was more or less near the normal values in rats treated with both antioxidants. Due to the availability of very limited information on the subject, it is difficult to compare the results. However, Yousef (2004) and Yousef, *et al.* (2003) reported that the presence of antioxidants reduced the harmful effect on the studied parameters includes ACP and ALP in rabbits as indicated by the changes in their activities toward normal.

From the current study, we can conclude that the activities of serum ALP, ACP, ALT and AST

were increased in rats treated with Al in the presence or absence of acid. The toxic effect of Al was diminished in the presence of antioxidants and the enzyme activities returned to more or less to their normal levels.

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پوخته

ئەف كاری نافرې هاته دارشتن بۆ لیکولین ل سەر کاریگه ریا باریزراو یا دژه ئوکساندنا و ئومیگا3 و فیتامینین جوراوجور ل سەر ئەله مونیومی هاندەر بۆ گورانکاریا ل هندەك پیقه رین بابوکیمیای ل جوردین نیر یین سپی. هفتی جوردین سپی . جورد هاتنه دابه شکر ب شیوهیهکی گوتره بۆ هفت کومه لا، ل ژیر خواردنا ئاسایی و هاتنه مامه له کون بقی شیوهی :

کومه لا1 (کونترول) ئافا حەنیفی هاته دان بۆ وان، کومه لا2 بۆ هەر جوردهکی 0.2% مل ژ گهینه ری هاته دان ب ریکا دهفی، کومه لا3 بۆ هەر جوردهکی 0.2% مل ژ گراوی ئەله مونیومی هاته دان ب ریکا دهفی، کومه لا4 بۆ هەر جوردهکی 0.2% مل ژ گراوی ئەله مونیومی و 0.5% مل ژ گراوی رونکری یی تفتی ئەسیتیک ب ریکا دهفی هاته دان، کومه لا5 بۆ هەر جوردهکی 0.2% مل ژ گراوی ئەله مونیومی و ئومیگا3 ب ریکا دهفی هاته دان، کومه لا6 بۆ هەر جوردهکی 0.2% مل ژ گراوی ئەله مونیومی و فیتامینین جوراوجور ب ریکا دهفی هاته دان، کومه لا7 بۆ هەر جوردهکی 0.2% مل ژ گراوی ئەله مونیومی و هەر ئیک ژ دژه ئوکساندنا و ئومیگا-3 و فیتامینین جوراوجور ب ریکا دهفی هاته دان، ئەف گیانه وه رین نافرې روژانه هاتنه سه خبیریکر ب کهرستین کاری بۆ ماوی 30 روژا.

ئەو جوردین هاتینه مامه له کون ب ئەله مونیومی زیده بونه کا کاریگه ر هاته دیتن ل هەر ئیک ژ ئاستین ل ئەنزیما تین چالاک ALP, GPT, GOT, ACP ل ناف میلاکی و میشکی، کاریگه ریا ژهراوی یا ئەله مونیومی زیده بو ب هه بونا تفتی ب ریژه بیه کا زور ب بهراوردکر دگه ل جوردین هاتینه مامه له کون ب ئەله مونیومی . کاریگه ریا ژهراوی یا ئەله مونیومی ب هه بونا دژه ئوکساندنا وهکی زهیتی میلاکا نهنگی و فیتامیناتین جوراوجور کیمبوو و نیژیکی ئاستین ئاساییت خو بوون. بقی رهنگی ئەو جوردین هاتین مامه له کون ب ئەله مونیومی ب هه بونا دژه ئوکساندنا دابه زینه کا کیم هاته دیتن ل کیشا لاشی و ههروه سا کیمبون ل ئاستین پیقه رین ئیزیماتا وهکی ALP, ACP, GOT, GPT, LDH ل ناف شانا زیده بوو و گهه شته ئاستین ئاسیین خو.

ب پی ئەنجامین فی لیکولینا نافرې دی شین بگه هینه فی ئەنجامی کو کاریگه ریا ژهراوی یا ئەله مونیومی زیده بوو ب هه بونا تفتی خەل، بهل کاریگه ریا ژهراوی کیمبوو ب هه بونا دژه ئوکساندنا (زهیتی میلاکا نهنگی و فیتامیناتین جوراوجور) ئیک ژ وان یا ههردوو دگه ل ئیک.