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Antioxidants and Some Biochemical Parameters in Alcoholism in Mosul City

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ABSTRACT

The study was conducted in Mosul city to show the effects of alcoholism on antioxidants and some biochemical parameters which include: Vitamin E, Vitamin A, β-carotene, Vitamin C, Folic acid, Ceruloplasmin (Cp), Total protein, Albumin, Calcium, Total bilirubin, Direct bilirubin, Uric acid, Creatinine, Total iron binding capacity (TIBC), Iron, Glutathione(GSH), Malondialdehyde (MDA), Cholesterol, Glucose, Selenium and Transferrin saturation (%). The study included (103) persons represented in two groups, the first group as a control included outside city center living, the second group represented for alcoholism was divided into two sub groups, smokers and non smokers.

The results showed a significant decreased in: calcium, transferrin saturation (%), but non decreased significant in: vitamin A, total bilirubin, direct bilirubin, creatinine, iron, MDA and glucose. While a significant increased in TIBC and selenium, but non significant increased in: vitamin E, β-carotene, vitamin C, folic acid, Cp., total protein, albumin, uric acid, GSH and cholesterol.

The results also showed a significant decrease of alcoholism smokers in: vitamin A (P=0.004), vitamin C, transferrin saturation (%) and folic acid, and non significant in: total iron binding capacity, iron, vitamin E, β-carotene, total protein, albumin and creatinine. While non significant increase in: Cp., calcium, total bilirubin, direct bilirubin, uric acid, malondialdehyde, glutathion, cholesterol, glucose and selenium was detected.

مضادات الأكسدة وبعض متغيرات الكيموحيوية لمدمني الخمور في مدينة الموصل

الملخص

شملت الدراسة تأثير تناول الكحول على مضادات الأكسدة وبعض المتغيرات الكيموحيوية في مدينة الموصل، إذ تضمنت تلك المتغيرات: فيتامين E، فيتامين A، بيتا كاروتين، فيتامين C، حامض الفوليك، سليروبلازمين(Cp.)، البروتين الكلي، ألبومين، كالسيوم، البليروبين الكلي، البليروبين المترابط، حامض اليوريك، كرياتينين، سعة الارتباط الكلي للحديد (TIBC)، الحديد، كلوتاثايون (GSH)، المالوندايألديهايد (MDA)، كوليستيرول، كلوكوز، سلينيوم وإشباع الترانسيفيرين(%). وتضمنت الدراسة (103) شخصا

مثّلوا بمجموعتين، المجموعة الاولى اعتبرت مجموعة سيطرة اختيرت من خارج مركز المدينة والمجموعة الثانية مثلت بمدمني الخمور التي قسمت إلى مجموعتين ثانويتين، مدخنين وغير مدخنين.

اظهرت النَتائِج لمدمني الخمور غير المدخنين انخفاض معنويةَ في، كالسيوم وإشباع الترانسيفيرين (%)، لكن انخفاض غير معنويةَ لـ فيتامين A، البليروبين الكلي، البليروبين المترابط، كرياتينين، حديد، MDA والكلوكوز. علاوة على ذلك، لوحظ بأن هناك زيادة معنوية في: TIBC، سلينيوم وغير معنوية لمتغيرات فيتامين E، بيتا كاروتين، فيتامين C، حامض الفوليك، .Cp، بروتين كلي، ألبومين، حامض اليوريك، GSH وكوليستيرول.

فضلا عن ذلك، لوحظ انخفاض معنوي لدى مدمني الخمور المدخنين في: فيتامين C، فيتامين A، إشباع الترانسيفيرين (%) وحامض الفوليك، وغير معنوي في: فيتامين E، بيتا كاروتين، حديد،TIBC ، بروتين كلي، ألبومين وكرياتينين. وزيادة غير معنوية لمتغيرات: .Cp، كالسيوم، البليروبين الكلي، البليروبين المترابط، MDA، كوليستيرول، الكلوكوز وسلينيوم.

INTRODUCTION

Alcohol is a term applied to members of a group of chemical compounds and, in popular usage, to the specific compound ethyl alcohol, or ethanol. Ethanol (C_2H_5OH), is a clear, colorless liquid, with a burning taste and characteristic, agreeable odor. Ethanol is the alcohol in such beverages as beer, wine and brandy (Microsoft® Encarta® Encyclopedia, 2005).

Ethanol absorbed into the body is mainly metabolized in the liver by an alcohol dehydrogenase enzyme to form the acetaldehyde (Garrett and Grisham, 2005):

$$\underset{\text{ethanol}}{CH_3CH_2OH} + NAD^+ \longrightarrow NADH + H^+ + \underset{\text{acetaldehyde}}{CH_3CHO}$$

While some studies have found that moderate use of alcohol has beneficial health effects, including protection from coronary heart disease. Red wine has been shown to inhibit in vitro oxidation of human LDL (Kaur and Kapoor, 2001). There appear to be other heart benefits of alcohol consumption as well, such as (Gaziano, 1993): Increasing high-density lipoprotein (HDL or (good)) cholesterol, preventing blood clots, raising blood levels of vitamin B6, thereby reducing the levels of homocysteine (a substance that increases the risk of heart disease).

Prolonged use of large amounts of alcohol might cause serious liver damage (George et al., 2004). In the first stage of liver disease caused by alcohol, fat accumulated in the liver and known as fatty liver.

Some people with fatty liver develop hepatitis, which inflames and kills liver cells (Paton, 1994). Hepatitis is marked by jaundice, which gives a yellowish tint to the eyes and skin. Others may develop cirrhosis, an irreversible condition in which normal liver tissue is replaced by scar tissue. The scarring prevents blood from traveling freely through the liver, building blood pressure in the veins that run from the intestine to the

liver. Consequently, the liver can no longer process toxins efficiently, causing poisons to build up in the blood. This buildup can be fatal (Paton, 1994).

Heavy drinking also damages heart muscle. Nearly half of all cases of cardiomyopathy are caused by alcohol abuse. In this heart disease, the heart muscles, particularly the right and left ventricles, enlarge and become flabby, reducing the heart's blood-pumping efficiency. This inefficiency reduces the flow of blood through the kidneys, which normally filter excess salts and water out of the blood. Eventually the blood volume rises, causing a potentially fatal backup of fluid in the lungs (Stipanuk, 2000).

Alcoholic persons tend to have high blood levels of the hormone epinephrine and deficiencies of the mineral magnesium. This combination produces severe arrhythmias, or heartbeat irregularities, a common cause of sudden death in heavy drinkers. Chronic drinkers typically develop hypertension, a leading cause of stroke (Beilin and Puddey, 1993).

Injecting of large dose of ethanol into rats has decreased the SOD activity and increased lipid peroxidation in the liver of rats by accumulation of conjugated dienes or ethan production and asignificant decreased in the GSH concentrations of liver and kidney cells of rats, but not in other tissues (Halliwell and Gutteridge, 1985).

Alcoholism is a major health problem afflicting people all over the world (George et al., 2004) and it is one of the main causes of damaging the human health (Burim et al., 2004), it is distributed throughout the body water, so most tissues heart, brain and muscles are exposed to the same concentrations as in blood (Paton, 1994).

The aim of the research is to obtain the effects of alcoholism (smokers and non smokers) on antioxidants and some biochemical parameters in Mosul city.

MATERIALS AND METHODS

The study included (103) individuals divided into two groups: The first non smokers control group (40), But the second smokers (35) and non smokers (28) alcoholism group. In alcoholism mean age of smokers group was (34.1) years (range 25-53 years) and Body Mass Index (BMI) in mean (27.11) kg/m^2 (range 25.39-30.71). But the mean age of non smokers group was (39.5) years (range 26-53 years) and Body Mass Index (BMI) in mean (28.78) kg/m^2 (range 26.34-32.12).

The following kits were used for determination of Total Protein No. (0303), Albumin No. (0801), Calcium No. (2403). Total Bilirubin and Direct Bilirubin No.(0401), Iron No. (0502), Total Iron Binding Capacity No. (0512), Glucose No. (0903), Cholesterol No. (0603), were obtained from Syrbio kits, Syria.

Vitamin A, β-carotene, vitamin E, vitamin C, Folic acid, Malondialdehyde, Glutathion, Selenium, Uric acid and Creatinine were determined using manual methods (Table 1).

Table 1:Methods used for determination of biochemical parameters.

No.	Parameters measured	Method used	References
1	Vitamin A	Needld-Pearson method	Neeld and Pearson,1963
2	β-carotene	Needld-Pearson method	Neeld and Pearson,1963
3	Vitamin E	Emmerie-Engel reaction	Emmerie and Engel, 1938
4	Vitamin C	2,4-dinitrophenylhydrazine derivatization method	Roe and Kuther, 1943
5	Folic acid	Microbiologyical assay	AOAC, 1950
6	Glutathion	Modified procedure utlilizing Ellman`s reagent.	Sedlak and Lindsay, 1968
7	Malondialdehyde	Thiobarbituric acid method	Lunec, 1990
8	Uric acid	Phosphotungstic acid method	Varley, 1967
9	Total bilirubin	Diazo method	Toro and Ackermann, 1975
10	Direct bilirubin	Diazo method	Toro and Ackermann, 1975
11	Total protein	Biuret method	Kingsley,1942
12	Albumin	Bromocresol green mthod (dye binding method)	Doumas et al., 1971
13	Ceruloplasmin	p-Phenylenediamine oxidase method	Sunderman and Nomoto, 1970
14	Glucose	Glucose oxidase method	Trinder, 1969
15	Cholesterol	Cholesterol estrase method	Richmond, 1973
16	Creatinine	Jaffĕ method	Jaffĕ, 1886
17	Total Iron Binding Capacity	Ramsay method	William et al., 1977
18	Transferrin saturation	Transferrin saturation(%)= Serum Iron /TIBC X 100	Burtis and Ashwood, 1999
19	Iron	Bathophenanthroline method	Carter, 1971
20	Calcium	Methylthymol blue method	Rbertson and Marshall., 1979
21	Selenium	Selenium-orthophenylenediamine complex	Cummins, 1965

Body Mass Index (BMI) was determined using the formula: weight (kg)/height^2(m^2) was used (Al-Abbad and Al-Sowielem, 1998).

RESULTS AND DISCUSSION

Effects of alcohol on the biochemical parameters:

The results of biochemical parameters for non smokers alcoholism and control group were listed in Table (2).

Table 2: The biochemical parameter of control and non smokers alcoholism.

Parameters	Non smokers Alcoholism (n=28)		Control group (n=40)		t-test
	mean	SD	mean	SD	P-value
Age(year)	39.5	8.09	35.7	7.9	0.986
Weight(kg)	83.0	5.65	76.31	4.5	0.424
Height(cm)	172.5	17.6	169.0	10.57	0.795
B.M.I(k.g./m2)	28.78	2.01	26.71	2.45	0.032
Vit.E(mg/dl)	1.23	0.08	1.0	0.06	0.198
Vit.A(µg/dl)	43.5	7.91	46.42	8.4	0.133
β-carotene(µg/dl)	126.9	12.72	80.49	11.52	0.421
Vit.C(mg/dl)	0.93	0.15	0.85	0.07	0.065
Folic acid(ng/ml)	7.8	0.61	6.85	0.78	0.054
Cp.(mg/l)	152.6	22.2	151.1	19.4	0.147
T.p.(gm/dl)	5.97	0.59	5.5	0.38	0.298
Alb. .(gm/dl)	4.96	0.49	4.36	0.22	0.346
Calcium(mg/dl)	11.1	0.78	11.78	1.24	0.004
Total Bilir. (mg/dl)	0.13	0.08	0.41	0.27	0.053
Direct Bilir. (mg/dl)	0.07	0.025	0.19	0.03	0.191
Uric acid(mg/dl)	5.4	0.64	5.05	0.58	0.421
Creatinine(mg/dl)	0.66	0.03	0.82	0.05	0.041
TIBC(µg/dl)	222.3	88.55	186.8	32.16	0.021
Iron(mg/l)	0.84	0.11	1.6	0.19	0.0021
GSH(µmol/l)	16.06	2.09	13.7	2.83	0.356
MDA µmol/)	6.43	0.51	6.5	1.97	0.214
Cholesterol(mg/dl)	188.9	28.51	180.8	40.13	0.389
Glucose(mg/dl)	60.56	11.2	68.1	12.8	0.132
Selenium(µg/dl)	82.25	7.58	35.7	8.06	0.047
Tansferrin saturation%	26.06	2.25	36.65	5.75	0.0031

Different Significantly at $P \leq 0.05$.

The results in Table (2) showed that there were significantly decreased parameters of alcoholism when compared with control group in: calcium (P = 0.004), transferrin saturation (%) (P=0.003) and creatinine (P=0.041). While non significantly decreased in: vitamin A, total bilirubin, direct bilirubin, iron, malondialdehyde and glucose which were similar in to other reported results (Chan-Yeung et al., 1981; Savage and Lindenbaum, 1986; Bjorneboe et al., 1988; Chapman et al., 1993; Gaw et al., 2005).

Iron deficiency may occur from increased gastrointestinal blood loss due to gastritis or cirrhosis (Savage and Lindenbaum, 1986).

Alcohol interferes with the balance of calcium, an essential nutrient for healthy bones. It also increases parathyroid hormone (PTH) levels, which in turn reduces the body's calcium reserves. Calcium balance is further disrupted by alcohol's ability to interfere with the production of vitamin D, a vitamin essential for calcium absorption (http://www.niams.nih.gov/bone).

Ethanol is metabolized to acetate in the liver by alcohol dehydrogenase and aldehyde dehydrogenase (Garrett and Grisham, 2005):

$$CH_3CH_2OH + NAD^+ \rightleftharpoons CH_3CHO + NADH + H^+$$

$$CH_3CHO + NAD^+ \rightleftharpoons CH_3COO^- + NADH + H^+$$

The excess NADH produced inhibits NAD_+ requiring reactions, such as gluconeogenesis, beside of alcohol inhibits gluconeogenesis, therefore showed a decrease level of glucose (Table 2) (Garrett and Grisham, 2005; Gaw et al., 2005).

The effect of alcohol might be explained by an increased clearance of substances such as creatinine caused by alcohol-induced diuresis (Goodman and Gilman, 1975), therefore may be decreased in creatinine level.

The results also showed significantly increase biochemical parameters in: total iron binding capacity (P=0.0021) and selenium (P=0.047). While non significantly increase in: vitamin E, β-carotene, vitamin C, folic acid, albumin, ceruloplasmin, total protein, glutathion, uric acid, cholesterol. Similar results were published by other investigators (Paton, 1994; Caccetta et al., 2000; Burnham et al., 2003; Parmahamsa et al., 2004; Conen et al., 2004; Gaw et al., 2005).

The role of phenolic compounds (present in wine and beer) are thought to prevent oxidative stress-linked diseases which is mainly due to their antioxidant capability while for ethanol compound hasn't this capacity as antioxidant (Denke, 2000).

A number of flavonoids have been found to possess antioxidant and free radical-scavenging properties (Hanasaky et al., 1994; Morel et al., 1994) and they able to inhibit LDL oxidation (Negré-Salvayre and Salvayre, 1992; Mangiapane et al., 1992). They can also inhibit several key enzymes in cellular systems involved in the generation of reactive oxygen species, including prostaglandin cyclooxygenase and lipoxygenase (Takahama et al., 1985; Laughton et al., 1991; Percival, 1998).

Excessive drinking of alcohol may caused many effects such as: stroke, ischaemic heart disease, other cardiac diseases, hypertensive disease, diabetes mellitus, liver cancer, cancer of mouth and pharynx, breast cancer, esophagus cancer, liver cirrhosis, epilepsy, falls, motor accidents, drownings, homicide, other intentional injuries, self-inflicted injuries, poisonings (Waluga and Hartleb, 2003). In addition, alcohol is responsible for the rise of oxygen consumption, excessive production of free radicals and increased metabolism of ethanol (Waluga and Hartleb, 2003).

Effects of smoking on the biochemical parameters in alcoholism group:

Cigarette smoking is common among persons with alcohol dependence or abuse with as many as 80 % of persons who are alcohol dependent also being smokers. Not only smoking is common with persons who have heavy alcohol consumption, but also nicotine dependence appears more severe in smokers with a history of alcohol dependence. This combined exposure to both tobacco smoke and alcohol results in major health consequences including additive risks for some diseases such as head and neck cancers. Although modest alcohol consumption has some positive health benefits, smoking typically negates these benefits (Romberger and Grant, 2004)

The results shown in Table (3) clearly predicted that the measured biochemical parameters were affected by cigarette smoking. Similar results were obtained where heavy alcohol consumption and cigarette smoking adversely affect laboratory tests (Ryback et al., 1980; Ryback et al., 1985).

Table 3: The biochemical parameters of alcoholism smokers and non smokers alcoholism.

Parameters	Alcoholism smokers(n=35)		Alcoholism non smokers(n=28)		t-test
	mean	SD	mean	SD	P-value
Age(year)	34.1	9.4	39.5	8.09	0.688
Weight(kg)	92.2	5.08	83.0	5.65	0.75
Height(cm)	175.4	3.62	172.5	17.6	0.57
B.M.I(k.g./m2)	27.11	1.86	28.78	2.01	0.133
Vit.E(mg/dl)	0.95	0.05	1.23	0.08	0.273
Vit.A(µg/dl)	43.56	5.86	43.5	7.91	0.004
β-carotene(µg/dl)	86.68	14.4	126.9	12.72	0.308
Vit.C(mg/dl)	0.84	0.10	0.93	0.15	0.034
Folic acid(ng/ml)	5.63	0.48	7.8	0.61	0.045
Cp.(mg/l)	186.5	24.1	152.6	22.2	0.063
T.p.(gm/dl)	5.77	0.63	5.97	0.59	0.213
Alb. .(gm/dl)	4.36	0.19	4.96	0.49	0.45
Calcium(mg/dl)	11.8	0.61	11.1	0.78	0.39
Total Bilir. (mg/dl)	0.17	0.05	0.13	0.08	0.421
Direct Bilir. (mg/dl)	0.09	0.03	0.07	0.025	0.82
Uric acid(mg/dl)	6.8	0.81	5.4	0.64	0.051
Creatinine(mg/dl)	0.7	0.08	0.66	0.03	0.86
TIBC(µg/dl)	318.5	95.0	222.3	88.55	0.103
Iron(mg/l)	0.73	0.08	0.84	0.11	0.058
GSH(µmol/l)	17.9	1.96	16.06	2.09	0.148
MDA µmol/)	6.58	0.82	6.43	0.51	0.554
Cholesterol(mg/dl)	199.9	35.6	188.9	28.51	0.075
Glucose(mg/dl)	62.86	14.3	60.56	11.21	0.291
Selenium(µg/dl)	86.54	6.5	82.25	7.58	0.421
Tansferrin saturation%	22.91	3.21	26.06	2.25	0.045

Different Significantly at $P \leq 0.05$.

The results showed significant decreased of alcoholism smokers when compared with alcoholism non smokers in: vitamin A (P=0.004), vitamin C (P=0.034), transferrin saturation (%)(P=0.045) and folic acid (P=0.045) which were similar results with (Cano et al., 2001; Zima et al., 2001;Gueguen et al., 2003).

It was investigated that chronic ethanol drinking can lead to chromosome damage, therefore more folic acid was required for biosynthesis of biological compounds (Bergi et al., 2002; Burim et al., 2004).

The results also showed non significantly decreased in: total iron binding capacity, iron, vitamin E, β-carotene, total protein, albumin and creatinine these finding were in a good agreement with others (Chan-Yeung et al., 1981; Ryback et al., 1985; Abu-Amsha et al., 2001).

Decreased creatinine in alcoholic smokers might be related to hemodilution caused by alcohol and nicotine–induced antidiurtic hormone secretion (Goodman and Gilman, 1975), or to decreased dietary intake of protein. (Cheraskin et al., 1975). Moreover, it has been observed that there are non significant increased of alcoholism smokers in: ceruloplasmin, calcium, total bilirubin, direct bilirubin, uric acid, malondialdehyde, glutathion, cholesterol, glucose and selenium. Similar results were published by other investigators (Ryback et al., 1985; Abu-Amsha et al., 2001).

The result of this study might suggest that the Increase Cp. levels in smokers might be a part of the total antioxidant status protecting tissues from the effects of free radicals (Al-Timimi and Al-Khayat, 2001), or might merely reflect increase copper intake from the tar component of the cigarettes (Duthie et al., 1991). The increase of cholesterol might be due to increase secretion of adrenaline hormone stimulated by the nicotine in cigarettes (Cryer et al., 1976). It is known that adrenaline increase the lipolysis and cholesterol level would be increased too (Burtis and Ashwood, 1999). Glucose was reported that insulin concentration in smokers was decreased and hence glucose would be increased (Burtis and Ashwood, 1999).

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