RESEARCH ARTICLE

The Estimation of Oxidative Stress from Alcohol Use Disorders in Iraqi Population

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ABSTRACT

Alcohol is the most widely used addictive substance in the world which linked to a variety of health, economic, and societal issues, reactive oxygen species (ROS) are extremely reactive chemicals that normally expressed in tiny level during metabolic processes in the body and can damage complex biological components like lipids, proteins, or DNA, so the current study aim to detect the oxidative stress relation with alcohol level in drunks in Iraqi population, the result showed a non-significant differences were observed in age, body mass index (BMI), ROS and thromboangiitis obliterans (TAO), also a Non-significant differences observed in study variables (BMI, ROS, TAO, Alcohol and duration) except the duration of abuse ($p = 0.001$) in the two age categories less than 30 and more than 30 years, The result of BMI showed non-significant differences about study categories, the study of mean differences of study variables belong to residence higher percent was observed in urban (77.77%) while less than in rural (22.22%) area and non-significant was observed in age, BMI, ROS, TAO, Alcohol, duration of abuse. We conclude that there were weak association between oxidative stress and alcoholic drinking, so we need further studies to understand the relationship between alcohol addiction and oxidative stress complication.

Keyword: Alcohol use disorder, Oxidative stress, Reactive oxygen species, Total antioxidants.

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INTRODUCTION

Alcohol (ethanol) is processed in the liver.[1] Because of the production of reactive oxygen metabolites throughout the process of alcohol metabolism, it is sensitive to oxidative stress, and as a result, Alcoholic Liver Disease has become an important cause of morbidity and mortality.[2,3]

The most major risk factor for the development of adrenoleukodystrophy (ALD) is the amount and kind of alcohol consumed. Increased pro-oxidant generation induced by alcohol, as well as a reduction in antioxidant defense mechanisms (enzymatic and non-enzymatic) in the liver, may contribute to the development of ALD, when pro-oxidants and antioxidants are in balance, oxidative stress occurs, which is characterized by increased The ROS are tiny, highly reactive oxygen–containing molecules produced normally in minute amounts throughout the body's metabolic processes and can react with and destroy complex biological components like lipids, proteins, or DNA. Several activities, notably in the liver, increase the production ROS and/or interfere with the body's natural defensive systems against these molecules.[5] For example, when alcohol is broken down in the liver, molecules are formed, which then undergo additional metabolism in the cell, resulting in the creation of ROS. Alcohol also increases the activity of cytochrome P450 enzymes, which contribute to the formation of ROS. Furthermore, alcohol can change the amounts of specific metals in the body, making ROS generation easier. Furthermore, alcohol lowers the amounts of antioxidant agents (i.e., antioxidants). Oxidative stress is the consequence of this process, and it leads to cell damage. The generation of ROS and oxidative stress in liver cells.[6]

The high concentration of ROS and the oxidative stress that results have been linked to a range of human illnesses; alcohol-induced oxidative stress is linked to cell injury, notably liver cell damage. Many investigations have shown that alcohol induces lipid peroxidation and protein modification; however, it's not always apparent whether these changes are the source or the result of alcohol-induced tissue damage. Nonetheless, several studies have indicated that providing antioxidants,

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agents that lower free iron levels, or agents that restore GSH levels can help to avoid or mitigate the harmful effects of alcohol.[7]

MATERIAL AND METHOD

Drunk individuals attended Al-Mhaweel hospital for forensic alcohol level detection enrolled in the present study, after ethical approval of each volunteer, blood samples and data were collected to serum isolation, ROS and total antioxidants (TAO) were detected. The alcohol was detected by forensic center in Ministry of Interior Affairs. Data represented by mean ± stander error, ANOVA one-way, independent t-test were used for significant evaluation at $p < 5$.

RESULT

The current work conducted to detect the oxidative stress relation with alcohol levels in drunks in the Iraqi population, study characteristics baseline was clarified in the Table 1, non-significant differences were observed in Age, BMI, ROS and TAO.

Non-significant differences observed in study variables except the duration of abuse (p 0.001) in the two age categories less than 30 and more than 30 years (Table 2).

The result of BMI showed non-significant differences about study categories (Table 3).

Table 4 shows mean differences of study variables belong to residence higher percent was observed in urban (77.77%) while less than in rural (22.22%) area and nonsignificant difference was observed in age, BMI, ROS, TAO, Alcohol, duration of abuse.

Table 5 shows mean differences of study variables belong to employment, higher percent was observed in the employment (61.1%) while less than in non-employee (27.8%) and lesser than in free work (11.1%) and there is a significant difference was observed in both Age and duration of disease (p 0.002,0.001), respectively.

Table 6 shows mean differences of study variables according to duration of abuse higher percent was observed in <5 years categories (61.1%) while less than in 5–10 years (33.3%) and lesser than in >10 years (5.6%) and there is a significant difference was observed in only Age (0.000).

DISCUSSION

The unbalanced in Oxidative stress plays a key role in the pathogenesis of alcoholic liver damage. Molecules produced through ethanol metabolism, such as ROS and reactive nitrogen species (RNS), structurally and functionally alter organic molecules. As a result, physiological processes are disrupted, and hepatocytes become more sensitive to cytokines like tumor necrosis factor-, as well as endotoxins, activating signaling pathways for instance nuclear factor kappa B, and extracellular signal regulated kinases.[8]

Chronic inflammation is the most significant mechanism associated with oxidative stress and the production of ROS. When cytokines are released, they activate oxidant production enzymes like NADPH oxidase and the NFB, which then activate LOX, Cox-2, and iNOS, culminating in the creation of ROS. Furthermore, ROS can be produced by the enzyme CYP2E1, which is increased by chronic alcohol intake, as well as in NASH, where free fatty acids and acetone (primarily in diabetics) induce CYP2E1. ROS causes lipid peroxidation, which results in the formatted of lipid peroxidation products including 4-HNE and MDA. Both substances can bind to DNA and create extremely harmful ethno-DNA adducts.[9]

Our study showed that the duration of abuse has significant differences about other variant and this result agree with a

Table 1: The study characteristics baseline of drunks and control group with alcohol level

Variables	*Age (year)*	*BMI kg/m²*	*ROS µmol/L*	*TAO µmol/L*	*Alcohol mg/cm³*	*Duration of abuse*
Drunks	28.44 ± 2.22	26.87 ± 0.69	22.91 ± 0.91	884.16 ± 12.01	65.88 ± 6.14	3.72 ± 0.74
Control	26.66 ± 0.85	24.81 ± 0.73	23.56 ± 0.63	887.07 ± 12.21	0	-
Sig	0.400	0.058	0.552	0.871	-	

Independent t test, $p < 0.05$

Table 2: The mean differences of study variables according to age categories

Age categories	*Percentages*	*BMI kg/m2*	*ROS µmol/L*	*TAO µmol/L*	*Alcohol mg/cm³*	*Duration of abuse*
<30	66.7%	27.07 ± 0.912	22.06 ± 0.49	877.67 ± 15.42	67.83 ± 7.54	2.16 ± 0.36
>30	33.3%	26.47 ± 1.06	24.63 ± 2.58	897.13 ± 19.33	62.00 ± 11.395	6.83 ± 1.470
Sig		0.696	0.195	0.462	0.668	0.001

Independent t test, $p < 0.05$

Table 3: The mean differences of study variables belong to BMI categories

BMI categories	*Percentages*	*Age (year)*	*ROS µmol/L*	*TAO µmol/L*	*Alcohol mg/cm³*	*Duration of abuse*
Normal	27.8%	26.60 ± 2.06	24.57 ± 3.24	866.70 ± 23.28	74.80 ± 15.23	3.40 ± 0.81
Overweight	55.6%	26.80 ± 2.159	22.37 ± 0.534	900.08 ± 13.75	54.30 ± 2.41	3.00 ± 0.63
Obese	16.7%	37.00 ± 11.13	21.97 ± 0.68	860.16 ± 41.77	89.66 ± 22.16	6.66 ± 3.84
Sig		0.238	0.559	0.349	0.071	0.211

Anova one way, $p < 0.05$

Table 4: The mean differences of study variables belong to residence

Residence	*Percentages*	*Age (year)*	*BMI kg/m²*	*ROS µmol/L*	*TAO µmol/L*	*Alcohol mg/cm³*	*Duration of abuse*
Rural	22.22%	32.00 ± 3.34	27.05 ± 1.55	22.12 ± 0.39	869.97 ± 29.36	68.75 ± 16.59	4.50 ± 1.25
Urban	77.77%	27.42 ± 2.68	26.81 ± 0.79	23.14 ± 1.18	888.21 ± 13.41	65.07 ± 6.71	3.50 ± 0.90
Sig		0.409	0.893	0.658	0.544	0.812	0.592

Independent t test, $p < 0.05$

Table 5: The mean differences of study variables according to employment ($p < 0.05$)

Employment	*Percentages*	*Age (year)*	*BMI kg/m²*	*ROS µmol/L*	*TAO µmol/L*	*Alcohol mg/cm³*	*Duration of abuse*
Employee	61.1%	23.27 ± 0.79	26.74 ± 0.89	23.55 ± 1.47	869.64 ± 17.38	56.54 ± 2.01	2.00 ± 0.35
Non-employee	27.8%	34.00 ± 1.70	26.96 ± 1.29	22.53 ± 0.34	900.76 ± 14.36	77.60 ± 19.60	5.40 ± 0.400
Free work	11.1%	43.00 ± 16.00	27.29 ± 3.44	20.40 ± 0.60	922.49 ± 7.65	88.00 ± 20.00	9.00 ± 5.00
Sig		0.002	0.971	0.585	0.295	0.145	0.001

Independent t test $p < 0.05$

Table 6: The mean differences of study variables according to duration of abuse

Duration of abuse	*Percentages*	*Age (year)*	*BMI kg/m²*	*ROS µmol/L*	*TAO µmol/L*	*Alcohol mg/cm³*
<5 years	61.1%	23.00 ± 0.58	26.69 ± 0.90	21.94 ± 0.52	878.85 ± 16.84	61.81 ± 4.99
5–10 years	33.3%	33.33 ± 1.54	26.55 ± 1.13	25.02 ± 2.50	886.22 ± 18.67	73.00 ± 16.65
>10 years	5.6%	59.00 ± 0.09	30.74 ± 0.08	21.00 ± 0.07	930.14 ± 0.08	68.00 ± 0.05
Sig		0.000	0.188	0.230	0.444	0.705

Anova one way, $p < 0.05$

research conducted by Kulkarni *et al.,*2015[10] that gives enough evidence of increased oxidative stress and compromised antioxidant defense system in patients with alcoholism, So, according to numerous studies, alcohol damage to the liver can be caused by a variety of different causes and is mediated by the harmful oxygen radicals produced by ethanol.[11] Variations in antioxidant and pro-oxidant levels have been associated with alcoholism. Alcoholism can cause a number of medical issues, such as impaired alcohol metabolism, liver cirrhosis, hormonal imbalances linked to pancreatitis, osteoporosis, immunological impairment, and decreased fertility. The metabolism of ethanol through alcohol dehydrogenase (ADH) and the resulting decreased NADH, or by the Microsomal ethanol-oxidizing system (MEOS), which involves the stimulation of cytochrome P-4502E1 and other Microsomal enzymes, can explain long-term ethanol consumption causes most toxic and metabolic diseases, acetaldehyde produced by both routes, predominantly in the liver, as well as the harmful consequences of acetaldehyde produced by both mechanisms. Ethanol use has been linked to an increase in oxidative stress, with the production of lipid peroxides and free radicals.[12]

CONCLUSIONS

We concluded that there are weak associated between oxidative stress and alcoholic drinking, and understanding the links between alcohol use, stress, and alcohol use disorders is an important field of research that is still being pursued in the Iraqi population.

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