Total Antioxidant Capacity and Some Risk Factors in Iraqi Smokers with Acute Myocardial Infarction

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Abstract

Background: Acute myocardial infarction (AMI) is one of the major causes of mortality and morbidity in the world. Reactive oxygen species (ROS) may play important role in the pathogenesis in myocardial infarction. Cigarette smoke is a major exogenous source of free radicals which is a heterogeneous aerosol consisting of more than 4000 compounds including high concentrations of free radicals, and reactive oxygen and nitrogen species. Total antioxidant capacity (TAC) was studied in most cases in serum, and may be used as a marker of the body antioxidant status. The recent applications of the TAC status in medical and nutritional studies as well as future possible uses of TAC level as a biomarker for diagnosis, prognosis and prevention of respiratory, diabetes, heart and vascular system, chronic renal injury, neurological, cancer diseases.

Aim: Study the correlation between the total antioxidant capacity level with lipid profile changing

Materials and Methods: This study was conducted during the period from Nov. 2014 to Sep. , 2015. Fifty eight patients presented with typical chest pain to the cardiac care unit in Al-Hussein Teaching Hospital, Al-Hussein Medical City/ Kerbela Health Directorate and Department of Biochemistry–College of Medicine / University of Kerbala. The diagnosis was based on the clinical history, presentation confirmed by ECG and various investigations of cardiac biomarker. Thirty five persons age – matched healthy volunteers were selected as a control group. The age, weight, height, lipid profile, TAC were measured in sera of all subjects

Results: The results revealed that serum total cholesterol, triglyceride, VLDL-C and LDL-C were significantly increased ($P < 0.01$) but serum HDL-C level was significant decreased ($P < 0.01$) in non-smoker AMI patients as compared with that found in non-smoker controls, while serum TAC , BMI and age was non-significantly different between non-smoker patients and non-smoker healthy control groups ($p > 0.05$). The results of smoking AMI show non-significant differences in levels of total serum cholesterol, triglyceride, VLDL-C, LDL-C and a significantly decreased in serum HDL-C as compared with smoker control. On the other hand BMI was non-significantly different between smoker patients and smoker healthy control groups ($p > 0.05$), while the results obtained show that a significant decrease in TAC in smoker AMI patient group in comparison with smoker normal control group ($p < 0.05$), and significant different age in smoker AMI patient group in comparison with smoker normal control group ($p < 0.05$).

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الكلمات المفتاحية : احتشاء العضلة القلبية الحاد, إجمالي القدرة المضادة للأكسدة ,مستوى الدهون, أنواع الأوكسجين التفاعلية

الخلاصــة

الخلفية: احتشاء العضلة القلبية الحاد (AMI) هي واحدة من الأسباب الرئيسية للأمراض والوفيات في العالم . أنواع الأوكسجين التفاعلية (ROS) قد تلعب دورا هاما في التسبب في احتشاء عضلة القلب. ويعتبر دخان السجائر هو مصدر خارجي كبير للجذور الحرة الذي هو عبارة عن دخان غير متجانس يتكون من أكثر من 4000 مركب بما في ذلك تركيز عالية من الجذور الحرة، وأنواع الأوكسجين والنيتروجين التفاعلي. ويمكن دراسة إجمالي القدرة المضادة للأكسدة (TAC) في معظم الحالات في مصل الدم، ويمكن استخدامها كعلامة لوضع الجسم من مضادات الأكسدة. ان التطبيقات الأخيرة لاختبار (TAC) في الدراسات الطبية والغذائية وكذلك الاستخدامات المستقبلية المحتملة لـ TAC كعلامات بيولوجية لأغراض التشخيص والتشخيص والوقاية من أمراض الجهاز التنفسي والسكري والقلب والأوعية الدموية، والإصابة الكلوية المزمنة والعصبية، وأمراض السرطان.

الهدف: دراسة العلاقة بين مستوى إجمالي القدرة المضادة للأكسدة مع مستوى الدهون المتغيرة وبعض عوامل الخطر الأخرى (العمر،ومؤشر كتلة الجسم، التدخين)في المرضى المصابين باحتشاء عضلة القلب

المواد وطرق العمل: أجريت هذه الدراسة خلال الفترة من نوفمبر 2014 إلى سبتمبر2015 على ثمانية وخمسون مريض يعانون من آلام في الصدر حضروا إلى وحدة العناية القلبية (CCU) في مستشفى الحسين التعليمي/مديرية الصحة/مدينة الحسين الطبية / كربلاء واستند التشخيص على التاريخ الطبي، والتي أكدها التخطيط الكهربائي لعضلة القلب ECG. ، وتحقيقات مختلفة من العلامات البيولوجية القلب العرض وقد تم اختيار خمسة وثلاثين شخصا من المتطوعين الأصـــــحـــاء كمجـــمـــوعـــة تحـــــكــــم .تم قياس العمر، الوزن، الطول، والدهون الثلاثية, و إجمالي القدرة المضادة للأكسدة (TAC) في مصل جميع العينات وتمت إجراء كافة القياسات في فرع الكيمياء الحيوية /كلية -الطب /جامعة كربلاء.

النتائج: أظهرت النتائج زيادة جوهرية في مستويات الكولسترول الكلي في الدم، الدهون الثلاثية، VLDL-C، وLDL-C (P<0.01)في المرضى غير المدخنين بالمقارنة مع غير المدخنين لمجموعة التحكم .وسجلت HDL-C انخفاضات كبيرة في المرضى غير المدخنين بالمقارنة مع غير المدخنين لمجموعة . في حين كانت مستويات TAC في مصل الدم،ومؤشر كتلة الجسم والعمر لا تختلف اختلافات كبيرة بين المريض غير المدخرين وغير المدخنين الأصحاء(p>0.05). أما نتائج مجموعة المدخنين المرضى AMI لم تظهر اختلافات ملحوظة في مستويات الكولسترول الكلي في الدم ، الدهون الثلاثية VLDL -C ، LDL-C ،في حين انخفض وبشكل ملحوظ مستوى HDL-C مقارنة مع مجموعة التحكم المدخنين. ومن ناحية أخرى فان BMI لم يختلف اختلافا معنويا بين المرضى المدخنين والمدخنين لمجموعة التحكم الأصحاء (p>0.05)، في حين أن النتائج التي تم الحصول عليها تظهر انخفاضا بنسبة كبيرة في مستويات TAC في مجموعة المرضى المدخنين بالمقارنة مع المدخنين لمجموعة التحكم الأصحاء (p<0.05) والفرق في السن كبير بين المدخنين المرضى بالمقارنة مع المدخنين لمجموعة التحكم الأصحاء (p<0.05).

Introduction:

Acute myocardial infarction (AMI) is one of the major causes of mortality and morbidity in the world [1]. Myocardial infarction have a many causes and risk factors such as : previous cardiovascular disease, old age, high blood levels of certain lipids such as increased in serum of each of triglycerides (TG), low-density lipoprotein- cholesterol (LDL-C). Low level of serum high density lipoprotein-cholesterol (HDL-C) , tobacco smoking, diabetes mellitus , obesity, chronic kidney diseases, heart failure, high blood pressure, excessive alcohol consumption, and chronic high stress level [2,3]. The most common cause of AMI is atherosclerotic coronary artery disease with erosion or rupture of a plaque causing transient, partial or complete arterial occlusion [4]. Reactive oxygen species (ROS) may play important role in the pathogenesis in myocardial infarction [5]. The formation of the atherosclerotic plaque is the oxidation of the LDL particle by the oxidants. This oxidation triggers a number of events that enhance the atherosclerotic plaque development [6]. Cigarette smoke is a major exogenous source of free radicals which is a heterogeneous aerosol consisting of more than 4000 compounds including high concentrations of free radicals , and reactive oxygen and nitrogen species [7]. Cigarette smoke contains organic compounds, free radicals and many oxidants such as superoxide and nitric oxide. In addition, inhalation of cigarette smoke into the lung caused to

activates some endogenous mechanisms, such as macrophages and accumulation of neutrophils, that caused of further increasing the oxidant injury [8]. Total antioxidant capacity (TAC) was studied in most cases in serum, and may be used as a marker of the body antioxidant status [9]. Measuring of TAC can provide information about the general situation of antioxidant status of the individual which may include those antioxidants do not recognize yet or not easily to measured [10]. The applications of the TAC test in medical and nutritional studies as well as future possible uses of TAC as a biomarker for diagnosis, prognosis and prevention of respiratory, diabetes, heart and vascular system, chronic renal injury, neurological, cancer diseases [11].

The aim of the presented study is to correlate between the total antioxidant capacity with lipid profile and some other risk factors (age, BMI, smoking) in AMI patient .

Materials and Methods:

The study was conducted during the period from Nov. 2014 to Sep. , 2015. Fifty eight patients presented with typical chest pain to the cardiac care unit (CCU) in Al-Hussein Teaching Hospital, Al-Hussein Medical City/ Kerbala Health Directorate. The diagnosis was based on the clinical history, presentation confirmed by ECG and various investigations of cardiac biomarker. Thirty five persons age – matched healthy volunteers were selected as a control group. Both groups divided to smokers and nonsmokers (39 AMI smokers patient and 17 smoker control) and (19 nonsmokers AMI patient and 18 nonsmoker control).

Lipid profile parameters include total serum cholesterol (TC), serum triglyceride (TG), and serum HDL-C which were investigated by enzymatic methods [12].

The level of VLDL-C and LDL-C were evaluated by use Friedewald formula listed below [13].

Total cholesterol (TC) = (VLDL-C) + (LDL-C) + (HDL-C)
[LDL-C] = [Total Cholesterol] - [HDL-C] - [Triglyceride/5]

Serum total antioxidant capacity was done by colorimetric method. Body mass index (BMI) was calculated from weight and height of patients and controls in kilogram/meter^2 according to the following equation.

BMI = Weight (kg) / Square Height (m^2)
Statistical analysis:

Lipid profile results indicated that serum total cholesterol, triglyceride, VLDL-C and LDL-C were a significant increase ($P < 0.05$) in non-smoker AMI patients in comparison with non-smoker controls, while serum HDL-C determined was significantly decreased in non-smoker AMI patients in comparison with non-smoker controls ($P < 0.05$) as shown in Table 1.

Table (1) The measured parameters of lipid profile in sera of non-smoker (AMI

and control groups).

Parameters	AMI patients (N = 19) Mean ± SD	Control Group (N = 18) Mean ± SD	P value
TC (mg/dl)	201.16 ± 26.63	168.67 ± 28.92	P < 0.05
TG (mg/dl)	205.25 ± 44.39	155.95 ± 67.35	P < 0.05

HDL-C (mg/dl)	30.37 ± 6.72	39.73 ± 10.26	P < 0.05
VLDL-C (mg/dl)	41.05 ± 9.74	31.19 ± 13.35	P < 0.05
LDL-C (mg/dl)	129.74 ± 29.80	97.75 ± 32.71	P < 0.05

No significant increased in levels of TC, TG, VLDL-C, LDL-C and a significantly decreased in HDL-C levels were obtained in smoker AMI patients as compared with smoker controls group ($p>0.05$) as shown in Table-2.

Table (2) The measured parameters of lipid profile in sera of smoker (AMI and control group).

Parameters	AMI patients (N= 39) Mean ± SD	Control group (N=17) Mean ± SD	*P value*
TC (mg/dl)	193.36 ± 31.11	180.76 ± 37.11	p>0.05
TG (mg/dl)	198.36 ± 54.00	170.88 ± 62.64	p>0.05
HDL-C (mg/dl)	32.46 ± 6.67	41.71 ± 15.94	P <0.05
VLDL-C (mg/dl)	39.67 ± 11.68	34.17 ± 10.02	p>0.05
LDL-C (mg/dl)	121.23 ± 33.20	104.88 ± 34.23	p>0.05

Total antioxidant capacity (TAC): the result show no significant different between non-smoker AMI patients and non-smoker healthy control groups ($p>0.05$) and significant decreases TAC in smoker AMI patient group in comparison with smoker normal control group ($p<0.05$) as shown in table (3).

Table (3) The measured parameters of TAC of Non Smoker and smoker groups

		AMI patient Mean ± SD	Control Mean ± SD	*P value*
TAC (unit/ml)	Nonsmoker	15.64 ± 8.47 (n=19)	18.96 ± 3.50 (n=18)	p>0.05
	Smoker	13.29 ± 4.58 (n=39)	15.79 ± 2.29 (n=17)	p<0.05

Body mass index and age were no significant different between non-smoker AMI patients and non-smoker healthy control groups ($p>0.05$) and significant different age in smoker AMI patient group in comparison with smoker normal control group ($p<0.05$) while BMI was no significant different between smoker AMI patients and smoker healthy control groups ($p>0.05$) as shown in table (4) and (5).

Table(4) The parameters of age and BMI of Non Smoker group .

Non Smoker	AMI patients (N=19) Mean ± SD	Control Group (N=18) Mean ± SD	P Value
BMI (kg/m^2)	30.23 ± 3.47	29.74 ± 5.47	p>0.05
Age (years)	54.47 ± 10.54	49.11 ± 11.23	p>0.05

Table(5) The parameters of age and BMI of Non Smoker group.

Smoker	AMI patients (N= 39) Mean ± SD	Control group (N=17) Mean ± SD	P Value
BMI (kg/m^2)	27.70 ± 3.83	26.74 ± 4.34	p>0.05
Age (years)	55.64 ± 12.75	48.65 ± 7.52	p<0.05

Discussion

This study showed increase significantly in serum cholesterol, triglyceride, VLDL-C and LDL-C in non-smoker patients comparison to non-smoker controls, while, HDL-C decreased significantly. This result was in agreement with studies.[14-17].

Also the results showed no significantly change in serum cholesterol, triglyceride, VLDL-C and LDL-C in smoker patients comparison to smoker controls, while, a significant decreased in HDL-C . the result agreement with studies that show the effect of smoking in lipid [18]

The major sources of oxidative stress in cardiovascular system involve: the enzymes xanthine oxidoreductase (XOR) ,NAD(P)H oxidase ,NOS as well as the mitochondrial cytochromes and hemoglobin [19,20]

ROSs including H_2O_2,$O_2^{.-}$,hydroxyl radical, and peroxynitrite have been shown to increase upon reperfusion of the heart following ischemia[21].

The results show no significant decrease in serum TAC in non-smoker patients when compared with non-smoker controls and a significant decrease in serum TAC in smoker patients comparison to smoker controls the result agreement with other studies [23-24].

The study show that the mean of age in AMI non-smoker and smoker AMI patients was 54.47 ± 10.54 years and 55.64 ± 12.75 respectively   that was higher than that of the non-smoker and smoker controls which was that the mean of age of the control is 49.11 ± 11.23 years and 48.65 ± 7.52 respectively this result was agreement with other studies[14, 25].

In this study we found that no significant different between smoker and non-smoker AMI patients and smoker and non-smoker healthy control groups in BMI this result was agreement by study[15]. But there is a study show significant different between AMI patients and control[14]. the diverging findings among such studies may be the result of BMI's inadequacy as a quantifier of true body fatness and fat distribution. BMI is an inadequate marker of overweight and obesity in patients with CAD [26].

Conclusion

1- lipid profile , significantly increase in serum cholesterol, triglyceride, VLDL-C and LDL-C with significantly decreased in serum HDL-C ware traditional risk factors in AMI and smoking

2- Serum total antioxidant capacity decreased in AMI and smokers and possible used as biomarker for diagnosis of AMI

3- age is most important clinical variable in AMI patient

4- BMI show no significant different between AMI patients and healthy control groups

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