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A COMPREHENSIVE STUDY ON ANTIOXIDANTS AS A BIOMARKER OF CARDIOVASCULAR RISK IN YOUNG PEOPLE

[1]Dr. Muhammad Asad Abdullah, [2]Dr. Noor-us-Syyam Sumra, [2]Dr. Khalid Nawaz
[1]Bahawal Victoria Hospital, Bahawalpur

Abstract:
Introduction: *An increasing number of studies focus on the role of reactive oxygen species (ROS) in the pathogenesis of premature ageing as well as of numerous civilization diseases, such as cardiovascular diseases. It has been suggested that higher antioxidant potential can protect the organism against undesirable ROS activity and thus prevent disease incidence.* ***Aims and objectives:*** *The basic aim of the study is to analyze the level of antioxidants as a biomarker of cardiovascular risk in young people.* ***Material and methods:*** *This cross sectional study was conducted at Bahawal Victoria hospital, Bahawalpur during 2018. There were 50 participants who selected for this study from both genders. In the course of myocardial ischemia and reperfusion the increased concentration of free radicals may also cause an increase in antioxidant enzymes activities. In order to exclude the possibility of acute ischemia-reperfusion reactions.* ***Results:*** *We collected all the demographic data of patients. Duration of disease positively correlated with HIP-1α ($r = 0.677$, P <0.001) but negatively correlated with VEGF ($r = -0.486$, P < 0.001); VEGF positively correlated with each of pyruvate ($r = 0.316$, P <0.047) and HIF-1α ($r = 0.374$, P <0.018), and, OSI positively correlated with TPX ($r = 0.969$, P <0.001), but, negatively correlated with TAC ($r = -0.469$, P <0.002).* ***Conclusion:*** *It is concluded that TAC, assayed with either the FRAS or the DPPH methods, does not differ between men with and without CVD when additional confounders are taken into consideration.*

Corresponding author:
Dr. Muhammad Asad Abdullah,
Bahawal Victoria Hospital,
Bahawalpur

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INTRODUCTION:

An increasing number of studies focus on the role of reactive oxygen species (ROS) in the pathogenesis of premature ageing as well as of numerous civilization diseases, such as cardiovascular diseases. It has been suggested that higher antioxidant potential can protect the organism against undesirable ROS activity and thus prevent disease incidence. However, the present state of knowledge on such dependence is still not complete. To highlight the central role of mineral metabolism for both, cardiovascular and skeletal integrity, the term chronic kidney disease-mineral bone disorder (CKD-MBD) was coined recently [1]. Cardiovascular disease (CVD) is the major cause of morbidity and mortality in patients with end-stage renal disease (ESRD) on haemodialysis (HD). Since ESRD frequently results from hypertension and diabetes mellitus, the increased CVD risk in these patients has been assumed to be the result of these underlying diseases [2]. Nevertheless, it has been elucidated how ESRD represents *per se* a CVD risk factor independently by both hypertension and diabetes mellitus.

Hypoxia is a state of reduced oxygen pressure below a critical threshold, which restricts the function of organs, tissues and cells [3]. A decrease in oxygen tensions of the kidney was demonstrated in a number of experimental models of CKD [4]. This led to the broad recognition that chronic cellular hypoxia of the kidney is the final common pathway in the progression of CKD leading to eventual kidney failure [5]. A group of transcription factors, designated hypoxia inducible transcription factors, are specifically induced by low tissue oxygen tension and are likely to have a role in the oxygen-sensing mechanism and reparative reaction [6].

Aims and objectives

The basic aim of the study is to analyze the level of antioxidants as a biomarker of cardiovascular risk in young people.

MATERIAL AND METHODS:

This cross sectional study was conducted at Bahawal Victoria hospital, Bahawalpur during 2018. There were 50 participants who selected for this study from both genders. In the course of myocardial ischemia and reperfusion the increased concentration of free radicals may also cause an increase in antioxidant enzymes activities. In order to exclude the possibility of acute ischemia-reperfusion reactions. We qualified the patients in whom the most recent acute coronary event, cardiac or cardio-surgery intervention had occurred at least a minimum of one month earlier.

Biochemical analysis

We measure the level of antioxidants in our selected patients groups who were suffering from CVD. Blood samples (5 mL) with and without EDTA/sodium fluoride as anticoagulant were obtained via venipuncture after the participants had fasted overnight, and serum and plasma were separated and aliquot frozen at −80°C till used. In HD patients. Baseline laboratory investigations were carried out for all patients and controls including complete blood count, serum urea and creatinine, arterial pH, arterial blood gases and infection screening, which included blood and urinary cultures by standard methods.

Analysis

Student's t-test was performed to evaluate the differences in roughness between groups. Two-way ANOVA was performed to study the contributions. A chi-square test was used to examine the difference in the distribution of the fracture modes (SPSS 19.0 for Windows, SPSS Inc., USA).

RESULTS:

We collected all the demographic data of patients. At before-dialysis session, duration of disease positively correlated with HIP-1α ($r = 0.677$, $P < 0.001$) but negatively correlated with VEGF ($r = -0.486$, $P < 0.001$); VEGF positively correlated with each of pyruvate ($r = 0.316$, $P < 0.047$) and HIF-1α ($r = 0.374$, $P < 0.018$), and, OSI positively correlated with TPX ($r = 0.969$, $P < 0.001$), but, negatively correlated with TAC ($r = -0.469$, $P < 0.002$). HIF-1α negatively correlated with each of TPX ($r = -0.529$, $P < 0.001$) and OSI ($r = -0.459$, $P < 0.003$); while, OSI positively correlated with TPX ($r = 0.944$, $P < 0.001$).

Table 01: **Correlations among the measured in CVD**

Parameters	Duration	Lactate	Pyruvate	Lactate/Pyruvate ratio	HIF-1α	VEGF	TAC	Total peroxides
TAC								
0.265 (0.098)	−0.174(0.283)	−0.147(0.366)	−0.142(0.384)	0.068(0.676)	−0.206(0.202)			
0.022 (0.894)	0.048(0.771)	−0.148(0.361)	0.105(0.517)	−0.220(0.173)	−0147(0.366)			
Total peroxides								
−0.017(0.918)	−0.032(0.843)	−0.194(0.230)	0.128(0.430)	−0.138(0.394)	0.229(0.156)	−0.247(0.124)		
0.283 (0.077)	0.036(0.825)	0.070(0.668)	−0.204(0.207)	**−0.529(0.001)**	−0.006(0.972)	0.168(0.301)		
Oxidative stress index								
−0.093(0.566)	−0.002(0.989)	0.161(0.322)	0.156(0.337)	−0.172(0.287)	0.245(0.128)	**−0.469(0.002)**		**0.969(0.001)**
0.285 (0.075)	0.015(0.929)	0.113(0.489)	−0.240(0.136)	**−0.459(0.003)**	0.062(0.705)	−0.155(0.340)		**0.944(0.001)**

Data shown are r value (*P*< value). HIF-1α= hypoxia induced factor-1α, VEGF= vascular endothelial growth factor. TAC = total antioxidant capacity.

DISCUSSION:

Previous studies provided confusing results on the relationship of the presence of cardiovascular diseases to TAC. Several studies found significantly lower blood antioxidants and TAC in patients with CVD [6,7]. Similarly, several studies found that in metabolic syndrome and HA patients exhibit decreased antioxidant protection and increased lipid peroxidation. However, no significant changes of TAC were observed during and after the incidence of MI or between hypertensive patients and normal controls [8]. Adaptive and innate immune responses are centrally involved in the chronic inflammatory process, which leads to destabilization of atherosclerotic lesions, these processes are tightly connected to metabolic factors, which are essentially influenced by life style and also the genetic/epigenetic frame. Inflammation-induced oxidative modifications contribute to all important clinical manifestations of CVD such as endothelial dysfunction and plaque disruption [9]. However, due the poor performance of antioxidant strategies in limiting atherosclerosis and cardiovascular events, it remains to be answered if oxidative modification is causal for the initiation or is an injurious response to atherogenesis. Disease underlying interactions are too complex and the understanding is too fragmentary that clear, reliable therapeutic recommendations can be given [10].

Total antioxidant capacity in renal failure group is diminished to a great extent due to antioxidant exhaustion and inhibition. Oxidative stress can also accelerate the apoptosis of leukocytes in HD patients. The activation of neutrophils and the complement pathway during HD session as the result of interactions of the blood with the dialysis membrane and endotoxin contaminated dialysate, iron overload, the presence of advanced glycation end products, high homocysteine levels, intradialytic cytokine activation, among others, could play a role [11].

CONCLUSION:

It is concluded that TAC, assayed with either the FRAS or the DPPH methods, does not differ between men with and without CVD when additional confounders are taken into consideration. Age was not a determinant affecting the antioxidative barrier, regardless of the presence of CVD.

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