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# Cardiovascular Diseases Correlation with Antioxidants in Hilla City, Iraq

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### Abstract

Cardiovascular disease (CVD) covers any disease of the circulatory system, namely the heart (cardio) or blood vessels (vascular), includes heart attack, angina and peripheral vascular heart disease, however antioxidants are vital substances that possess the ability to protect the body from damage caused by free radicals induce oxidative stress. This study aimed to find out the possible mechanisms of correlation between CVD and antioxidants. The results showed that total protein (TP),total antioxidant status (TAS), albumin levels, catalase (CAT), superoxide dismutase (SOD) have significance decreased in patients compared to control the group.

Keywords: Cardiovascular diseases, Antioxidants, Catalase, Total protein, serum albumine

## Introduction

Free radicals are very reactive chemical species with an unpaired electron. Because of their reactivity lipids, proteins and DNA can be damaged by the radical's action. In consequence, they are responsible for many diseases, such as cancer, cardiovascular disorder, atherosclerosis, asthma, arthritis, neurodegenerative disorders: Alzheimer's, Parkinson's diseases and dementia[1]. As a first level of defense, the AOS includes enzymes, mostly intracellular located, such as several forms of superoxide dismutase (SOD), catalase (CAT) and a group of macromolecules including albumin [2]. The chemistry of antioxidants involves the mechanism of action of antioxidant that two mechanisms of action have been proposed for antioxidants. The first is a chain-breaking mechanism by which the primary antioxidants donate electrons to the free radicals present in the system, example lipid radicals. The second mechanism involves removal of ROS (reactive oxygen species) and RNS (reactive nitrogen species) initiator by quenching chain initiator catalyst [3] On this basis, there are five known antioxidant mechanisms which describe antioxidant reactions[4, 5].

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# **Materials and Methods**

1. Patients with AMI (n= 60), Control (n= 40) 2.All chemicals used in this study with highly purified and no further purification done.

### Collection of specimens:

Venous blood samples of 10 milliliters were drawn from patients of AMI within 12 hours of the heart attacks by using disposable syringes (10mL) in sitting position (Ethical committee certificate No.5/2014), 1.8 mL of fresh blood drawn in a plane tube contains (0.2mL) trisodium citrate solution 3.8% as anticoagulant for measuring fibrinogen, the remaining of the blood sample pushed slowly into plain disposable tubes. The blood was allowed to clot at 37° C for 15 minutes, and then centrifuged at 3000 rpm for 10 minutes then the sera were obtained and stored at  $-20^{\circ}$  C until analysis of TP, TAS, SOD, CAT, Albumin level.

### Determination of Total Protein

Serum TP was measured using kit supplied by Spinreact (Spain) [6]

#### Determination of TAS

Serum TAS was measured using kit supplied by Bio cat. (Germany) [7]

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#### Determination of SOD

(SOD) activity measured using the method as described previously [7]

### Determination of CAT

(CAT) activity measured using the method as described by Cohen et al., **[8]** 

### Determination of Serum albumin :

Serum albumin was measured by colorimetric assay using kit supplied by Manufacturer Biolabo (France) [9]

## **Results and Discussion**

The results of this study (Tables 1-5) showed a significant decrease in total protein levels in Patients group compared with the control group (P<0.001). A decrease in total protein

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concentration is the result of the fall in albumin, that A: G ratio change due to either reduction of albumin or elevation of globulins, however, decreased levels (hypoproteinemia) are found in Renal diseases, malnutrition, albuminuria, and terminal liver failure. Serum albumin is a negative acute-phase reactant that is synthesized by the liver, in inflammatory states, the activity of macrophages and other immune system cells is enhanced, leading to the production of cytokines, which switch protein synthesis in the liver from serum albumin to other acute-phase proteins. The albumin-MI association was modified by hypertension, in those hypertensive subjects showed a steeper relation of albumin to MI risk. There was evidence for an increased risk of all-cause mortality with lower albumin levels among women (R1). Albumin at concentrations less than physiological can inhibit copper-stimulated peroxidation and hemolysis. It also inhibits the production of free hydroxyl radicals from systems containing copper ions and H<sub>2</sub>O<sub>2</sub> and can scavenge peroxyl radicals, and its inhibits copper

Table	1: Serum	levels of S	Serum Tota	l Protein	(gm/dl)	of patients	and control
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Samples	Number	Total Protein (gm/dl) Mean ±SD	P value
Control	40	7.7938 ± 0.33092	P<0.001
Patients	60	5.6752 ± 0.49884	

### Table 2: Serum levels of Serum Albumin (gm/dl) with AMI patients and controls

Samples	Number	Serum Albumin (gm/dl) Mean ±SD	P value
Control	40	4.5420 ± 0.1283	P<0.001
Patients	60	3.4065 ± 0.18431	

### Table 3: Serum levels of SOD (U/L) with AMI patients and controls

Samples	Number	SOD (U/mL) Mean ±SD	P value
Control	40	23.5925 ± 2.12885	P<0.001
Patients	60	16.8517 ± 0.78167	

### Table 4: Serum levels of TAC (mmol/L) with AMI patients and controls

Samples	Number	TAC Mean ±SD	P value
Control	40	3.5450 ± 0.75037	P<0.001
Patients	60	1.3520 ± 0.33081	

### Table 5: Serum levels of CAT (U/L) with AMI patients and controls

Samples	Number	TAC Mean ±SD	P value
Control	40	3.5450 ± 0.75037	P<0.001
Patients	60	1.3520 ± 0.33081	



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dependent lipid peroxidation systems. LDL oxidation is one of the early steps in the atherosclerotic process that serum albumin may inhibit endothelial apoptosis [10]. The result is in agreement with results of [11,12] (Table 1, 2).

In this study, there was a significant decrease in total antioxidant levels in Patients group compared with a control group (P<0.001). Low dietary intakes of antioxidant vitamins or reduced synthesis of non-dietary antioxidants such as albumin, bilirubin, glutathione peroxidase cholesterol and uric acid are likely to result in an oxidant-antioxidant imbalance that exacerbates inflammation and tissue damage [13]. This result is in agreement with results of [14]. There was a significant decrease in superoxide dismutase levels in Patients group compared with the control group (P<0.001). Superoxide dismutase decreases levels of oxidative stress and improves endothelial dysfunction and diminishes the development of atherosclerosis [15]. The results of this study show that reduce in copper/zinc SOD (Cu/ZnSOD) could associate with increased vascular oxidative stress, which agree with the results of [17]. There was a significant decrease in Catalase levels in Patients group compared with the control group (P<0.001). To balance basal ROS production, the cellular redox milieu is maintained by a number of key antioxidant enzymes that limit intracellular and extracellular accumulation of deleterious reactive oxygen metabolites. These antioxidant defense enzymes include the superoxide dismutases, catalase, which reduces hydrogen peroxide to water and others, and the result is in agreement with results of [17] (Table 3-5).

## Conclusion

Antioxidants like SOD and CAT have shown promise as possible therapy for prevention and treatment of Ischemic heart diseases.

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