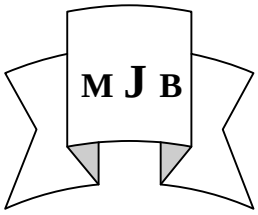
Evaluated of Serum Glutathione and Malondialdehyde for Victims of Trauma

Lamia, A.M. Al-Mashhedy

Chemistry Dept. College of Science, University of Babylon, Hilla, Babylon, Iraq.



Abstract

This paper describes a decreasing of Glutathione(GSH) concentration as antioxidant and increasing of Malondialdehyde(MDA) concentration as the end product of lipid peroxidation for victims of traumas, we studied the victims of terrorism attack due to the importance of this attack on psycho-somatic disease and on the post traumatic stress disorder. 15 patients victims of trauma in terrorism attack in jerif al-sakhar aged 12-40 years and 10 patients in animals market in Babylon city, aged 20-45 years, and 20 non terror-attack subjects aged 12-45 years, as a control group were recruited for this study.

الخلاصة

يهدف البحث الى تقييم بعض مضادات الاكسده لعدد من الأشخاص المتعرضين للصدمات النفسيه وتضمن البحث ضحايا العمليات الإرهابية التي حدثت في جرف الصخر(في سنة 2009) وسوق بيع الحيوانات في بابل (في سنة 2009) وربط المضاعفات التي تحدث لهم بعد تعرضهم للصدمه بسبب الحادث الارهابي(كونه احد الاسباب المهمه المؤديه الى العديد من الأمراض النفسجسمية) بالمتغيرات الكيميائية ومن ناحية نظرية الأكسدة.

وقد تم في هذا البحث دراسة الانخفاض في تركيز الكلوتاثيون كمضاد للأكسدة والارتفاع في تركيز المالون داي الديهايد كناتج نهائي لأكسدة الدهون في مصل 15 شخص في حادثة جرف الصخر اعمارهم تتراوح بين 12-40 سنة و10 شخص في حادثة سوق بيع الحيوانات اعمارهم بين 20-45 سنة(الحادثتين وقعتا في محافظة بابل) , (علما بأن هؤلاء الاشخاص لم يتعرضوا الى اصابات جسدية اثناء الحادث) مقارنة بمجموعة سيطرة تتكون من 20 شخص.

Introduction

Victims of terror attacks, whether or not physically injured, sometimes suffer long-term posttraumatic symptoms, although the intensity of symptoms differs among individuals, additional posttraumatic symptoms and emotional distress are evident, together with difficulty in readjusting to a normal life. [1,2] posttraumatic stress symptoms is an anxiety disorder that people get after

seeing or living though a dangerous event [3].

The use of explosives and suicide bombings has become more frequent after 2003. This terror attacks has a new markers in Iraq. We previously reported that the incidence of terrors attack has since risen. However, the rise in the incidence of victims for terror was proportionate to the rise in the incidence of terrorism trauma victims.

Oxidative stress, arising as a result of an imbalance between free radical production and antioxidant defenses, is associated with damage to a wide range of molecular species including lipids, proteins, and nucleic acids. Lipoprotein particles or membranes characteristically undergo the process of lipid peroxidation, giving rise to a variety of products including short chain aldehydes such as malondialdehyde or 4-hydroxynonenal, alkanes, and alkenes, conjugated dienes, and a variety of hydroxides and hydroperoxides. Many of these products can be measured as markers of lipid Peroxidation[4].

Glutathione(γ-glutamylcysteinylglycine, GSH) is a sulfhydryl (SH) antioxidant, antitoxin, and enzyme cofactor, glutathione is ubiquitous in animals, plant and microorganisms. GSH synthesis occurs within cells in two closely linked, enzymatically controlled reactions that utilize ATP and draw on nonessential amino acids as substrates. Cysteine is generated from the essential amino acid methionine from the degradation of dietary protein or from turnover of endogenous proteins [5, 6, 7].

Glutathione is an essential cofactor for antioxidant enzymes like GSH peroxidases and the phospholipid hydroperoxide GSH peroxidases. The GSH peroxidase serve to detoxify peroxides by reacting them with GSH, the latter enzymes use GSH to detoxify generated in the cell membranes, GSH providing electrons to help reduce oxidized biomolecules located away from the water phase.[8, 9].

The continual flux of single electrons to oxygen generates an endogenous oxidative stress in human tissues, superoxide, peroxide, hydroxyl radical and other free radicals derived from oxygen are highly reactive and therefore threatening to the integrity of biomolecules such as DNA and RNA, enzymes and another proteins and the phospholipids responsible for membrane integrity.[10,11]

Patients

15 victims of trauma in terrorism attack in jerif al-sakhar, aged 12-40 years and 10 victims in accident of animals market in Babylon city, aged 20-45 years, and 20 healthy subjects aged 12-45 years, as a control group were recruited for this study. Blood samples were collected from patients (after the accident directly), serum was separated by centrifugation at 3000 rpm, the analytical determinations described below were either performed immediately.

Methods

Glutathione is determined by a modified procedure utilizing Ellman's reagents, this methods principles as a reduced of 5,5-dithiobis(2- nitro benzoic acid by sulfhydryl group of GSH to yellow compound, the absorbance is measured at 412nm[12, 13].

The assessment of lipid peroxidation in serum was determined by the colorimetric thiobarbituric acid (TBA) method. Under the acidity and heat condition of the reaction the lipid

peroxides break down to form malondialdehyde(MDA) which complexes with the spectrophotometrically at 532 nm[14].

Statistical analysis.

All results are expressed as a mean ± SD(standard deviation), comparison between patients and controls were preformed by the student's t- test. Person's correlations were used to determine relationship between parameters studied. A value of $p \leq 0.05$ was considered statistically significant

Results and Discussion

The GSH levels of patients are significantly decreased, Table 1, for victims terror attack compared with non terror attack group.

Table 1 The GSH concentration (µM) for victims terror attack and non terror attack group(control group).

	N	Mean	SD	SE	P value
Control	20	19.4	5.2	2.7	------
Victims	25	12.5	12.3	5.3	0.00 Sign

MDA levels are significantly increased for victims terror attack compared with non terror attack Table 3.

Table 2 The MDA concentration(µmole/L) for victims terror attack and non terror attack group(control group).

	N	Mean	SD	SE	P value
Control	20	0.62	0.3	0.16	------
Victims	25	1.94	1.02	0.4	0.00 sign

Oxidative stress originating from outside the body is a feature of life in the world. The tens of thousands of confirmed toxic substances in our external environmental[15], and toxic substances due to terrorism attack are invariably sources of free radicals or related oxidants, Free radical production occurs continuously in all cells as part of normal cellular function. However, excess free radical production originating from endogenous or exogenous sources might play a role in many diseases. Antioxidants prevent free radical induced tissue damage by preventing the formation of radicals, scavenging them. [16]

GSH also makes major contributions to the other antioxidants that have become oxidized, such as vitamin E and vitamin C. The lists free radical quenching reactions against which GSH can be employed at below:

Hydroxyl radical(OH•) quenching:

$$GSH + HO\bullet \longrightarrow GS\bullet + H_2O$$

Reduction of Lipid peroxides:

$$2GSH + LOOH \longrightarrow GSSG + LOH + H_2O$$

Maintenance of protein-SH groups in the reduced state:

$$2GSH + PSSX \longrightarrow GSSG + P(SH)2X$$

Recycling of Ascorbic acid(ASC):

$$2GSH + 2\ ASC\bullet \longrightarrow GSSG + 2ASC$$

Another radical(R•) quenching:

$$GSH + R\bullet \longrightarrow GS\bullet + RH$$

GSH depletion lead to cell death and has been documented in many degenerative conditions, mitochondrial GSH depletion may be the ultimate factor determining vulnerability to oxidant attack[17].

The increase of MDA levels in the serum indicates the ongoing oxidative stress in patients, the serum antioxidant defense are overwhelmed and organs are no adequately protected and undergo oxidation.

There is overwhelming evidence that oxidative stress occurs in cells as a consequence of normal physiological processes and environmental interactions, and that the complex web of antioxidant defense systems plays a key role in protecting against oxidative damage. These processes appear to be disordered in many conditions, and a plausible hypothesis may be constructed implicating oxidative stress as a cause of tissue damage.

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