Medical Journal of Babylon
Vol. 12- No. 4:1015 - 1019 , 2015
http://www.medicaljb.com
ISSN 2312-6760©2015 University of Babylon

Original Research Article

Role of Superoxide Dismutase Enzyme in Patients with Epilepsy

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Accepted 29 September, 2015

Abstract

Epilepsy is a disease of the brain in which at least two unprovoked (or reflex) seizures occurring within more than 24 hours apart Epilepsy is classified to idiopathic, cryptogenic, and mixed classification.Superoxide dismutase (SOD); an enzyme that catalyzes the dismutation of superoxide radicals have been purified by a simple procedure from bovine erythrocytes. This enzyme contains 2 equivalent of copper per mole of enzyme.The aim of the study to know the significance of antioxidant superoxide dismutase (SOD) enzyme in epileptic patients.

This is a prospective study conducted in Al-Hussein Teaching Hospital in Holy Kerbalaa for the period from January to October 2014 .One hundred ten (110) patients were included in this study.Superoxide dismutase activityis determined by using a simple and rapid method. Superoxide dismutase enzyme level in epileptic patients was significantly lower ($p<0.01$) than its level in healthy control. It was significantly lower in all subgroups of patients when compared to control subgroups ($p<0.01$).It was significantly lower in patients group in both male and female when compared to control group ($p<0.01$).

Epilepsy is linked with the defect in the antioxidant defense mechanism.

Key words: epilepsy, Superoxide dismutase enzyme.

الخلاصة

الصرع هو مرض يصيب الدماغ حيث يحدث اثنان من النوبات الاختلاجية على الأقل خلال أكثر من أربع. ويصنف الصرع إلى مجهول السبب وعضوي السبب والمختلط من كلا النوعين. سوبر اوكسايد دسميوتيس هو أنزيم مساعد لتحويل العناصر الحرة المتكونة خلال العمليات الايضية البسيطة في كريات الدم الحمراء الخاصة بالمواشي.

أجريت هذه الدراسة في مستشفى الحسين التعليمي في كربلاء المقدسة للفترة من كانون الثاني حتى تشرين الأول لسنة ٢٠١٤.تم شمول ١١٠ مريض بهذه الدراسة و فعالية سوبر اوكسايد دسميوتيس تم تحديدها باستخدام الطريقة البسيطة و السريعة. مستوى هذا الأنزيممرضى الصرع كان أقل بصورة ملحوظة عن مستواه في الأشخاص الطبيعيين الأصحاء و قد كان قليل بصورة ملحوظة في كل مجموعة صغيرة من المجاميع المريضة بعد مقارنتها بنفس المجاميع للأشخاص الطبيعيين الأصحاء . و أيضا كان مستوى هذا الأنزيم قليل في المرضى سواء كانوا إناث أو ذكور على حد سواء عند مقارنتها مع الأشخاصالأصحاء.من ذلك نستنتج ان الصرع مرض مرتبط بخلل في الآلية الدفاعية للمواد المقاومة للأكسدة.

الكلمات المفتاحية:الصرع، سوبر اوكسايد دسميوتيس .

Introduction

Normally, the biomolecules in the body are composed of firm bonds formed by paired electrons[1]. Weakened, disrupted bonds lead to creation of free radical (an atom, molecule, or ion that has unpaired electrons in outer orbital which unstable and highly reactive species)[2]. Usually, there is balance between formation and elimination of free radicals. Nevertheless, this balance may be shifted towards more generation of free radicals or less availability of antioxidants. This condition is known as oxidative stress and can result in serious cell damage if this stress is massive and prolonged[3].

Antioxidants are molecules that secure biological biomolecules against oxidative stress[4]. They protect cells by preventing creation of ROS or by eliminating them, quickly before they attack the biomolecules. Also they play a major role in the repair of the damage, reconstructing the lost functions and clearance the waste products[5].

Superoxide dismutase (SOD); an enzyme that catalyzes the dismutation of superoxide radicals have been purified by a simple procedure from bovine erythrocytes. This enzyme contains 2 equivalent of copper per mole of enzyme. The copper may be reversibly removed, and it is required for activity. An assay of several tissues indicates that superoxide dismutase is widely distributed within mammalian organisms[6]. Superoxide dismutase is a major antioxidant enzyme in the brain tissue and play a key role in preventing the neuronal damage which caused by epilepsy[3]. There are 3 major forms of superoxide dismutase. MnSOD present in mitochondria; Cu-Zn SOD in cytoplasm, and extracellular SOD (ECSOD) secreted by vascular cells into the extracellular matrix. They catalyze the reaction of superoxide dismutation to hydrogen peroxide (which is the first step in the chain of free radical scavenger systems), then undergoes further reactions and exerts numerous biological functions. This process leads not only to the removal of superoxide from the brain, but also generates an important regulatory molecule-hydrogen peroxide. Therefore, the expression and activity of superoxide dismutase may be critical for cellular defense from injury and seizure[7].

Materials and Methods

This is a prospective study conducted in Al-Hussein Teaching Hospital in Holy Kerbalaa for the period from January to October 2014 in order to evaluate the role of superoxide dismutase in epilepsy. One hundred ten (110) patients were included in this study with age range from (1-<18) years. These patients were subdivided into four groups as follows: the first group (1-<3 years); the second group (3-<5 years); the third group (5-<10 years); and the fourth group (10- <18 years) [8,9]. All patients are newly diagnosed with idiopathic epilepsy, none of them took any medication that may cause epilepsy or on antiepileptic drugs (AEDs).

Superoxide dismutase activityis determined by using a simple and rapid method, based on the ability of the enzyme to inhibit the autioxidation of pyrogallol. The Cu-Zn SOD activities are expressed as units/milliliter (U/ml). One unit of Cu-Zn SOD activity being defined as amount of enzyme required to cause 50% inhibition of pyrogallol autioxidation[10].

Results

Superoxide dismutase (SOD):

Superoxide dismutase enzyme level in epileptic patients was significantly lower ($p<0.01$) than its level in healthy control as shown in table (1).

Table 1 : Superoxide dismutase(SOD) enzyme level in patients and healthy control groups

SOD	Patients	Healthy control	P value
Mean ±SD U/ml	16.09±0.52	23.23±3.71	p< 0.01*

*p value < 0.01 is highly significant.

Superoxide dismutase enzyme and age:
Superoxide dismutase enzyme (SOD) was significantly lower in all subgroups of patients when compared to control subgroups (p<0.01). At the same time there is no significant difference of (SOD) between age groups of patients or healthy control (p>0.05) as shown in table (2).

Table 2 : Superoxide dismutase enzyme level in patients and control group in different age groups

Age group (year)	SOD Mean ±SD U/ml		P value
	Patients	Healthy control	
1-< 3	15.82±0.21	22.98±0.20	p< 0.01*
3-< 5	15.72±0.86	23.01±0.6	p< 0.01*
5-< 10	16.29±0.16	23.73±0.16	p< 0.01*
10-< 18	16.54±0.43	23.14±0.38	p< 0.01*
P value	p>0.05	p>0.05	

*p value < 0.01 is highly significant.

Superoxide dismutase enzyme and gender:
Superoxide dismutase enzyme was significantly lower in patients group in both male and female when compared to control group (p<0.01), on the other hand, there was no significant difference of SOD level between males and females in the same study group (p>0.05) as shown in table (3).

Table 3 : Superoxide dismutase enzyme level in patients and control group in both gender groups

Gender	SOD Mean ±SD U/ml		P value
	Patients	Healthy control	
Male	16.27±0.48	23.32±0.41	p< 0.01*
Female	15.91±0.56	22.96±0.06	p< 0.01*
P value	p>0.05	p>0.05	

*p value < 0.01 is highly significant.

Discussion

In the present study, superoxide dismutase enzyme (SOD) level in epileptic patients was significantly reduced in comparison with healthy controls (Table 1,2), this finding was consistent with many previous studies [6,7,11].

In the current study, the study groups were subdivided according to sex (Table 3), there was no significant difference in SOD level between males and females whether they are patients or healthy control which is similar to other studies that stated that the sex did not affect SOD level [3].

References

1. Halliwell B. Free radicals and antioxidants: updating a personal view. Nutrition Reviews. (2012) ;70(5) 257-65.
2. Sharma K.; Reynolds N. ; Rakhit M. and Agarwal A. Oxidative Stress in Applied Basic Research and Clinical Practice (2013). 33-60.
3. Poltorak E.P.; Zagrodzki P.; Nicol F.; Kryczyk J. and Barton H. Antioxidant agents and physiological responses in adult epileptic patients treated with lamotrigine (2013). Pharmagological Reports; 65, 99-106.
4. Agrawal, L.; Louboutin, J. P.; Marusich, E.; Reyes, B. A.; Van Bockstaele, E. J. and Strayer, D. S. Dopaminergic neurotoxicity of HIV-1 gp120: reactive oxygen species as signaling intermediates (2010). Brain Res. 1306C, 116–130.
5. Niki E. Assessment of antioxidant capacity in vitro and in vivo. (2010). Free Radical Biology &Medicine;. 49(4): 503-515.
6. Surekha T.N. and Melinkeri R.R. Oxidative and Antioxidative Status in Epilepsy. (2010).*Pravara Med Rev*; 2(4).
7. Ogunro P.S.; Mustapha A.F. and Salau A.A. (2013). Lipid peroxidation and antioxidant status with primary generalized epilepsy. Scholer Research Library; 5 (1):68-74.
8. Gill D. and Obrien N. Pediatric clinical examination. (1988). 1st ed.,Churchill living stone comp. , p 5-7.
9. Behrman R. E; Kleigman R. M. and Jenson H.B. Growth and development. (2004). Nelson textbook of pediatrics .Saunders .USA,p 25.
10. Magnani L.; Gaydou M. and Hnbaud J.C. Spectrophotometric measurement of

antioxidant properties of flavones and flavonols against superoxide anion.
(2000). Anal. Chim. Acta 411,1-2, 1: 209-216.
11. Chen D.; Lu Y.; Yu W.; Luo J.; Xiao Z.; Xiao F. and Wang X. Clinical value of decreased superoxide dismutase 1 in patients with epilepsy. (2012). Seizure;21(7):508-11. doi: Epub.