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QUANTITATIVE ANALYSIS OF ANTIOXIDANT ENZYME SUPEROXIDE DISMUTASE IN PARKINSON'S DISEASE

Medical Science

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ABSTRACT

Introduction: Parkinson's disease affects the nerve cells in the brain that produce dopamine. Oxidative stress may contribute to the neurodegenerative process in Parkinson's disease (PD) and the presence of an altered antioxidant defense system in upper motor neurons, suggesting that oxidative stress may be a widespread phenomenon in PD .This might result from increased exposure to free radicals coupled with deficit of antioxidants. Antioxidant enzyme, erythrocytic superoxide dismutase (ESOD), has demonstrated its efficacy in exploring the mechanism of PD. The present study was undertaken to find the levels of ESOD in PD.

Materials and Methods: 39 Parkinson's disease patients and 61 age matched controls were studied for their red cell superoxide dismutase levels. Analysis was performed by using ANOVA test to compare the values between Parkinson's disease patients and controls. Age and gender wise statistical analysis was performed of the ESOD in the PD and controls.

Results: Mean of ESOD in controls was 335.62units/ml of whole blood with S.D. of ± 30.36. Mean of ESOD in PD was208.00 with S.D. of ± 47.72 units/ml of whole blood, when compared statistically with control the 'mean difference was108.58 and was highly significant i.e. 'p' value is <0.001. In case of ESOD mean value for males it was 338.69 and females it was 330.18. When compared statistically it was found to be nonsignificant with t value 1.052.

Conclusion: There was significant decrease in ESOD levels among PD patients which might provide supportive tool in understanding prognosis of the disease.

KEYWORDS

Erythrocytic superoxide dismutase, Parkinson's disease., neurodegeneration

Introduction

Parkinson's disease affects the nerve cells in the brain that produce dopamine. Parkinson's disease symptoms include muscle rigidity, tremors, and changes in speech and gait. Degeneration of basal ganglia results in movement disorder, which manifests as either too little or too much movement depending on structures involved. Parkinson's disease was named after the British doctor James Parkinson, who in 1817 first described the disorder in detail as "shaking palsy."[1] Examples are Parkinson's disease, Huntington's disease and cerebellar degeneration [2,3] (usually causes ataxia).

In cases of PD, various indices of reactive oxygen species (ROS) damage have been reported within the specific brain region that undergoes selective neurodegeneration The enzyme SOD converts these superoxide free radicals to the less reactive hydrogen peroxide. Significance of SOD in various types of pathogenic response, especially toxic chemical injury to cells, makes the study of functional status of SOD isoenzymes an important aspect of research in biomedical studies[4]. There is increasing evidence that oxidative stress is a casual or at least an ancillary factor in neuropathology of several adult neurodegenerative disorders as PD[5].

Oxidative stress may contribute to the neurodegenerative process in Parkinson's disease (PD). It is planned to measure erythrocytic superoxide dismutase (SOD), antioxidant enzyme involved in primary defense against free radical damage, in PD and normal control subjects. Although there are some evidence to support this, the data are unlimited and needs additional more research work on these antioxidant enzymes.

MATERIALS AND METHODS

Present work was carried out in the department of biochemistry, Krishna institute of medical sciences, Karad. Subjects were taken from department of medicine of Krishna hospital, Karad and Kripamayee hospital, Sangli and some other private hospitals in Karad. The total number of subjects included were one hundred. Out of 100 cases, 61 were healthy controls of matching age and sex as that of disease group, 39 were suffering from Parkinson's disease. Diagnosis was done by Neuropsychiatrists and physicians from department of medicine.

Sample Collection: After obtaining patients written consent blood specimens were collected from anticubital vein, by taking all aseptic precautions. 5 ml blood samples from all subjects were collected in EDTA bulbs. Hemolysates were prepared from the blood samples in EDTA bulbs on the same day of collection and analyzed immediately using standard protocols [6].

Name of method: Erythrocytic superoxide dismutase by RANSOD Kit from Randox Lab. ESOD units/ml of whole blood = ESOD units/ml [7,8].

Statistical analysis was done by students “t test and POST HOC”, by use of SPSS10.0(Statistical Program for Social Service) software.

Results & Discussion

Table 1: Age wise distribution of ESOD in study population

Age groups	Statistics	E. SOD U/ml whole blood PD	ESOD U/ml whole blood in controls
51 – 60 Years	Range	208 to 330	312 to 380
	Mean ± SD	246.25 ±37.94	348.36 ±18.20
61 – 70 Years	Range	160 to300	208 to360
	Mean ± SD	189.47 ±34.66	314.81 ± 8.914
> 70 years	Range	150 to342	300 to398
	Mean ± SD	195.50±54.31	345.57±33.786

Table 2: comparison of ESOD levels in controls and PD.

Groups	ESOD U/ml of whole blood Mean ± S. D.	Mean difference`	P value
PD	208.00 ± 47.72	127.13	< 0.001
Controls	335.62 ± 30.36		

Table 3: Age group wise statistics of PD Patients

Age groups	Statistics	E. SOD U/ml whole blood
51 – 60 Years	Range	208 to 330
	Mean SD	246.25 ±37.94

61 – 70	Range	160 to300
Years	Mean SD	189.47 ±34.66
> 70 years	Range	150 to342
	Mean SD	195.50±54.31

Table 4: Gender wise statistics in PD

	Sex	N	Mean	Std. Deviation	t value	Significance
Sod	M	21	218.9524	54.30882	1.503	.141
	F	18	196.2778	36.44949		

Discussion

In PD, E-SOD levels mean value was 208.00unit/ml in whole blood with S.D. of 47.72, when compared with control, the fall in E-SOD levels was found highly significant as shown in table 1 & 2 . PD patients showed a statistically significant decrease of SOD activity in whole blood and in red blood cells. Erythrocytic SOD levels in healthy control males is found to be decreased as age advanced. In age group 51-60 yr. mean is 348.36units while the same in age group in group 61-70 years314.81 units and in group > 70 years it is 345.57units as per table 3. E SOD mean value for males 218.95 and females it is 196.27 as given in table 4. When compared statistically it is found to be not significant with t value 1.503 A negative correlation between SOD activity and duration of the disease was observed, while there was no relationship between L-Dopa treatment and SOD activity. Our results indicate that a defect in SOD activity develops over time in PD. In PD, metabolism of dopamine by monoamine oxidase accelerates excessive formation of hydrogen peroxide and subsequent reaction of H2O2 with iron by Fenton reaction which generates hydroxyl radical. Hydroxyl radical is damaging radical with a half life of 10-5 seconds, and is produced from H2O2 and O2 by Haber –Weiss reaction . When E-SOD levels were decreased, the quenching of O`2 (superoxide) decreased, which further forms other toxic free radicals leads to neuronal damage in substantia nigra in PD[9] .

Whether this is a later manifestation of antioxidant mechanism deterioration or simply an epiphenomenon remains unclear. There are number of degenerative diseases affecting the basal ganglia, which present with differing combinations of slowness of movement (Bradykinesia), increased tone (rigidity), tremor and loss of postural reflexes. The most common cause of these Parkinsonian syndromes is idiopathic Parkinson's disease. Idiopathic Parkinson's disease has an annual incidence of about 0.2/1000 and prevalence of 1.5/1000 in UK. Prevalence rates are similar throughout the world, though lower rates have been reported for china and West Africa. Sex incidence is about equal. The cause is unknown and no strong genetic factors have been identified, though recent work on twins has suggested that the genetic influence may be greater than previously thought. Methyl phenyl tetrahydropyzidine (MPTP) caused server Parkinsonism in young drug users suggests that the idiopathic disease might be due to an environmental toxin. Many candidate toxins have been studied, but there is no strong evidence in favor of any of them . Present study was an honest attempt to correlate a pathogenic impact of oxidative stress on neuroinflammation in order to develop newer therapeutic strategies to reduce this process for better understanding of disease process and its cure.

Conclusion

Thus our findings suggest that elevated oxidative stress might play an important role in progression of pathogenesis of neurodegeneration. Determination of ESOD, will be a supportive additional biochemical tool for the prognosis as well as proper treatment of the neurodegenerative diseases.[10,11] Here the term prognosis is very important since the number of neurodegenerative diseases are reversible, only if these are treated properly . The prognosis is very difficult because the symptoms appear only after severe damage to the cells; In case of PD symptoms appear after 80% cells are destroyed. Usually the diagnosis of neurodegenerative diseases is missed because of the advanced age and ignorance of the patients[12].

References:

1. Abraham S. et al Erythrocyte antioxidant enzymes in PD; IJMR; Feb 2005 p111-115.
2. Merril Hayden et al ,Classification of Parkinson disease Healthwise;APRIL 29, 2005.
3. Jamie Grutzendler J Tsai, Duff K, Gan WB. Fibrillar amyloid deposition leads to local synaptic abnormalities and breakage of neuronal branches. Nat Neurosci (2004) 7:1181–3.
4. Zelko I, Mariani T, Folz R (2002). "Superoxide dismutase multigene family: a comparison of the CuZn-SOD (SOD1), Mn-SOD (SOD2), and EC-SOD (SOD3) gene structures, evolution, and expression". Free Radic Biol Med 33 (3): 337–49..
5. Kussumi et al Epidemiology of PD and MID,1996 Neuroepidmiology,;15;201-207.
6. Aminoff, M. J. Pharmacologic Management of Parkinsonism and Other Movement Disorderslogy, in Basic and Clinical Pharmacology, (Katzung, B. G., ed) Appleton-Lange, 1998, pp 450-463
7. Anthony, W. Lane et al. Mitochondrial Mutations and ageing. The Lancet March 25, 1989.; 1(8639):642-5.
8. Aoyama K, Matsubara K, Fujikawa Y, Nagahiro Y, Shimizu K, Umegae N, Hayase N, Shiono H, Kobayashi S. Nitration of manganese superoxide dismutase in cerebrospinal fluids is a marker for peroxynitrite-mediated oxidative stress in neurodegenerative diseases. Ann Neurol. 2000;47:524–527. [PubMed]
9. Araki N. et al ,effect of SOD an intracellular Ca in stroke. J cerebro. Blood flow Metab. 1992 Jan 12 (1) 43-52.
10. Asha Krishnakumar et al Understanding Dementias ,Frontline, vol.20 issue.6,March 2003,15-28.
11. A Cagnin, A Zambon, G Zarantonello, D Vianello, M Marchiori, D Mercurio, F Micciché, M Ermani, A Leon, L Battistin Serum lipoprotein profile and APOE genotype in Alzheimer's disease. J Neural Transm Suppl. 2007 ;(72):175-9.
12. Farin FM, Hitosis Y, Hallagan SE, Kushleika J, Woods JS, Janssen PS, Smith-Weller T, Franklin GM, Swanson PD, Checkoway H. Genetic polymorphisms of superoxide dismutase in Parkinson's disease. Mov Disord. 2001 Jul;16(4):705-7.