



Research article

STUDY OF MALONDIALDEHYDE, VITAMIN E AND VITAMIN C LEVELS IN DEPRESSIVE PSYCHAIITRIC ILLNESS PATIENTS

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ABSTRACT

Background: Incidence of psychomotor and psychiatric illness has promoted an awareness regarding the mental and emotional well-being as an important criterion of health. Research on depression is one of the important researches in psychiatric medicine. Negative reactions to life's situations become repetitively intense and frequent leading to symptoms of depression. **Aim:** A study on changes between antioxidant levels in cases of depression. **Method:** It was a Case Control Observational Study. The patients newly diagnosed by psychiatrist & having depression were assessed with Hamilton's depression scale are included in the study. The controls were free from depression. The blood sample was collected from each case and control at fasting. Serum Malondialdehyde (MDA), Serum Tocopherol (Vitamin-E) Serum Ascorbic acid (Vitamin-C) levels were estimated. **Result:** Category I: Group I, II, III MDA levels were 2.02 ± 0.16 , 2.02 ± 0.16 , 2.02 ± 0.15 respectively. In Category II: Group I, II, III MDA levels were 6.28 ± 0.77 , 5.31 ± 1.33 , 5.74 ± 1.13 respectively. Category I: Group I, II, III Vit E levels were 11.27 ± 1.21 , 10.99 ± 1.03 , 11.44 ± 1.20 respectively. In Category II: Group I, II, III Vit E levels were 9.92 ± 1.14 , 10.44 ± 1.35 , 10.21 ± 1.30 respectively. Category I: Group I, II, III Vit C levels were 0.87 ± 0.06 , 0.89 ± 0.14 , 0.89 ± 0.06 respectively. In Category II: Group I, II, III Vit C levels were 0.82 ± 0.01 , 0.82 ± 0.01 , 0.82 ± 0.01 respectively. **Conclusion:** There is a significant difference between mean values of height & weight in Category I and Category II. Thus, the decreased antioxidant levels in depression cases show marked "oxidant - antioxidant imbalance" with evidence of increased oxidative stress.

KEYWORDS: Psychaitric illness, Serum Malondialdehyde, Serum Tocopherol, Serum ascorbic acid.

INTRODUCTION

Incidence of psychomotor and psychiatric illness has promoted an awareness regarding the mental and emotional well-being as an important criterion of health. Research on depression is one of the important researches in psychiatric medicine. Negative reactions to life's situations become repetitively intense and frequent leading to symptoms of depression: Depression is very common i.e. seventeen per cent of all people experience depression [1].

Age groups, in almost all walks of life. Children or adults, develop depression symptoms. Intense sadness, loss of interest or pleasure in normal activities, sleep disturbances or oversleeping, change in appetite and decreased energy level; feelings of helplessness and thoughts of suicide are sequels to stress induced depression.

Exact mechanism of depression is not known. Depression may be due to Genetic Factors [2], Environmental Factors [2,3],

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Neurobiological Factors [4], Depressed person as a parent [3]. In addition, there are many environmental factors, such as loss of a loved one, unemployment, an unexpected medical illness, that appear to increase the likelihood of depression [5]. The present study was designed to find the association between antioxidant enzymes malondialdehyde, Vitamin C and Vitamin E in depressive subjects & control.

Aim: A study on changes between antioxidant levels in cases of depression.

Objectives:

1. To estimate and compare serum Malondialdehyde, ascorbic acid (Vitamin C) & tocopherol (Vitamin E) levels in patients with mental depressive group and controls
2. To find out changes between severity of depression & antioxidants levels
3. To estimate severity of depression & levels of antioxidants

MATERIAL & METHOD

Study Design: It was a CaseControl Observational Study.

Ethics committee: The study protocol was approved by the institutional ethics committee.

Sample size: The depressed group includes 83 patients whereas the 100 normal individuals without any psychiatric disorder were taken as controls.

Sampling method: Non Probability Purposive sampling method was adopted for selection of subjects.

The patients newly diagnosed by psychiatrist & having depression were assessed with Hamilton's depression scale are included in the study.

Matching of case and control: Controls were matched with depression patients with regard to age.

Group I consist of – 20-30 years, Group II consist of – 31-40 years and Group III consist of – 41-55 years.

Selection of Control group:

Inclusion criteria: The controls were free from depression, age varies from 20 to 55 years, free from clinical morbidities other than depression and willing to participate in the study

Exclusion criteria: History of consumption of psychotropic substance and suffering from hypertension

Selection of depression cases:

Inclusion criteria: The depression cases were diagnosed by psychiatrist & rated on the basis of Hamilton's depression scale []. Age group varies from 20-55 years, the cases were free from clinical morbidities, willing to participate in the study and able to cooperate in conduction of autonomic function test

Exclusion criteria: History of substance use disorder, suffering from hypertension

Groups: Collection of blood sample: First the written consent was obtained from all subjects. The blood sample was collected from each case and control at fasting (in the

morning to avoid diurnal variation of oxidative stress) from cubital vein with all aseptic precautions. Eight ml blood was collected (4ml in plain bulb and 4ml in chemistry bulb) and serum was separated by centrifugation at 3000 rpm for 10 minutes.

Parameters studied

1. Serum Malondialdehyde (MDA)

Method: Buege and Aust [6]

Normal Value: 2 to 2.5 nmoles /milliliter

Principle:- Malonyldialdehyde (MDA) is highly reactive three carbon dialdehyde, produced from lipid hydroperoxidation. It can, however, also be derived from hydrolysis of pentoses, deoxyriboses, hexoses, from some amino acids and from DNA. MDA estimation was done by thiobarbituric acid reaction. MDA is measured as an index of lipid peroxidation.

2. Serum Tocopherol (Vitamin-E)

Method: Baker & Frank [7]

Normal Value: 5- 18 micrograms/liter

Principle: Serum tocopherol is measured by its ability to cause reduction of ferric to ferrous ions which then form a red complex with a 'a' dipyrilidil. Tocopherols and carotenes are first extracted into xylene and the absorbance is read at 460 nm to measure the carotenes. A correction for the carotenes is made after adding ferric chloride and reading at 520nm.

3. Serum Ascorbic acid (Vitamin-C):

Method: Ayekyaw [8]

Normal Value: 0.5 to 1.5 milligram/deciliter

Principle:- The acid phosphotungstate is specific and sensitive for ascorbic acid determination with good reproducibility. The acid phosphotungstate used, serves not only as protein precipitant and ascorbic acid extractant but also as color reagent.

Data analysis: The data analysis was done by descriptive statistics as mean, SD, Percentage.

RESULTS

Table 1. Comparison of mean values of Anthropometric Parameters in Category I and Category II

Anthropometric Parameter	Category I (n =100)	Range	Category II (n =83) Mean ± SD	Range	'p' value
Age(Yrs)					
Group I (26)	26.35 ± 2.53	22 – 30	Group I (11) 26.45 ± 2.68	21-30	p < 0.05
Group II (38)	35.75 ± 2.80	31 – 40	Group II (39) 36.03 ± 3.10	31-40	p < 0.05
Group III (36)	46.72 ± 4.14	41 – 55	Group III (33) 46.91 ± 3.48	41-55	p < 0.05
Height (cms)					
Group I (26)	154.35 ± 4.60	146 – 163	Group I (11) 155.55 ± 8.37	132-164	p<0.05*
Group II (38)	156.71 ± 4.83	147 – 165	Group II (39) 152.54 ± 6.90	136-165	p<0.05*
Group III (36)	155.33 ± 5.30	132 – 164	Group III (33) 152.42 ± 7.33	132-164	p<0.05*
Weight (cms)					
Group I (26)	51.98 ± 8.55	40 – 75	Group I (11) 51.00 ± 7.33	42-64	p<0.05*
Group II (38)	54.11 ± 8.52	40 – 76	Group II (39) 50.62 ± 6.31	40-64	p<0.05*
Group III (36)	53.83 ± 7.47	40 - 64	Group III (33) 48.15 ± 6.10	40-64	p<0.05*

*Significant. The comparison of all variables under study was done by Students unpaired "t" test at 5% level of significance.

At the end of the study the results of the severity score, parameters and the level of antioxidants and autonomic function tests of each individual will be compared by an analysis of the variability in repeated measures.

Table 2. Comparison of mean values of Antioxidants in category I and Category II

ANTIOXIDANTS	Category I (n =100)	Range	Category II (n =83)	Range	'p' value	
	Mean ± SD					Mean ± SD
MDA nmol/ml						
Group I (26)	2.02 ± 0.16	1.84 - 2.45	Group I (11)	6.28 ± 0.77	5.01 - 7.12	p <0.05*
Group II (38)	2.01 ± 0.16	1.84 - 2.45	Group II (39)	5.31 ± 1.33	3.11 - 7.23	p <0.05*
Group III (36)	2.02 ± 0.15	4.0 - 7.23	Group III (33)	5.74 ± 1.13	4.0 - 7.23	p <0.05*
Vit E mg/l						
Group I (26)	11.27 ± 1.21	10.55 - 9.9	Group I (11)	9.92 ± 1.14	10.4 - 9.45	p <0.05*
Group II (38)	10.99 ± 1.03	10.55 - 9.9	Group II (39)	10.44 ± 1.35	10.25 - 9.45	p <0.05*
Group III (36)	11.44 ± 1.20	10.25 - 9.45	Group III (33)	10.21 ± 1.30	10.2 - 9.45	p <0.05*
Vit C mg/dl						
Group I (26)	0.87 ± 0.06	0.8 - 1.00	Group I (11)	0.82 ± 0.01	0.81 - 0.84	p <0.05*
Group II (38)	0.89 ± 0.14	0.1- 0.99	Group II (39)	0.82 ± 0.01	0.81 - 0.84	p <0.05*
Group III (36)	0.89 ± 0.06	0.81 - 0.84	Group III (33)	0.82 ± 0.01	0.81 - 0.84	p <0.05*

*Significant

DISCUSSION

Many brain chemicals ("neurochemicals") and hormones have been linked to the development of depression (e.g., norepinephrine, dopamine, thyroid hormones)^[9]. However, research studies have implicated disturbances in the serotonin (5-HT) system and the Limbic Hypothalamic-Pituitary-Adrenal (LHPA) axis as two of the neurobiological alterations most consistently associated with mood-altering illness^[10,11,12,13]. Recent works, in fact, has strongly suggested that the interaction between these two biochemical systems may play a significant role. Changes in brain chemicals occur which have also found in suicide victims with a history of depression^[11]. This suggests that a chronic or severe stress (e.g., loss of a spouse, serious illness or injury, history of abuse) may cause similar neurochemical changes in vulnerable people, therefore triggering episodes of depression^[9]. Studies of depressed patients suggest that decreased levels of serotonin in brain are associated with decreased mood, while increased levels of serotonin are associated with increased levels of mood^[14].

Antioxidants are substances that act as shields or barriers to prevent the invading free radicals from doing harm to cells^[9]. Antioxidants are as they give up their own electrons to free radicals. When free radical gains the electron from an antioxidant it no longer needs to attack the cell and the chain reaction of oxidation is broken. After donating an electron an antioxidant becomes a free radical^[9]. Antioxidants in this state are not harmful as they have the ability to accommodate the change in electrons without becoming reactive.

The human body has an elaborate antioxidant defense system. Antioxidants are manufactured within the body and can also be extracted from the food items such as fruits,

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vegetables, seeds, nuts, meats and oil. There are two lines of antioxidant defense within the cell. The first line, found in the fat soluble cellular membrane consists of vitamin E, Beta carotene and coenzyme Q. Inside the cell water soluble antioxidant scavengers are present. These include Vit C, glutathione peroxidase, superoxide dismutase and catalase^[14].

CONCLUSION

There is a significant difference between mean values of height & weight in Category I and Category II. Thus the decreased antioxidant levels in depression cases show marked "oxidant - antioxidant imbalance" with evidence of increased oxidative stress. The cause effect relationship of decrease in antioxidant capacity in subjects with depression needs further study.

REFERENCES

1. Ashutosh Bajpai, Akhilesh Kumar Verma, Mona Srivastava, and Ragini Srivastava: Oxidative stress and major depression: J CliDiagn Res. 2014 Dec; 8(12): CC04-CC07
2. Atmaca M, Koluloglu M, Tezcan E, Ustundag B. Antioxidant enzyme and melanodialdehyde levels in patients with social phobia (Journal Article): Psychiatric Res 2008 Mar 11.
3. Beere P A, Glagov S, Zarins CK : Retarding effect of lowered heart rate on coronary atherosclerosis. Science 1984; 226:180-2.
4. Carney R M, Rich MW, teVelde A, Saini J, Clark K, Freedland K E : Heart rate, heart rate variability and depression in patients with coronary artery disease. J Psychosom Res 1988; 32:159-64.
5. Carney R M, Freedland K E, Veith R C, Cryer P E, Skala JA, Lynch T, Jaffe A S : Major depression, heart rate, and plasma norepinephrine in patients with coronary heart disease. Biol res Psychiatry 1999; 45:458-63.

6. Carol Hart, Secrets of Serotonin, Chapter2, Information about serotonin
7. Chin K. Kim, MD; Susan P. Depressive Symptoms and Heart Rate Variability in Postmenopausal Women Arch Intern Med. 2005;165:1239-1244.
8. Depression: Beyond the Catecholamine Theory of Mood, A good overview of Depression
9. Dyer AR, Persky V, Stamler J, Paul O, Shekelle RB, Lepper M, Schoenberger RA, Lindberg HA. Heart rate as a prognostic factor for coronary heart disease and mortality: findings in three Chicago epidemiologic studies. Am J Epidemiol 1980;112:736-49.
10. Gold PW, Goodwin FK, Chrousos GP, 1988. Clinical and biochemical manifestations of depression. Relation to the neurobiology of stress (second of two parts). N. Eng. J. Med. 319:413-420.
11. Heidland U E, Strauer B E : Left ventricular muscle mass and elevated heart rate associated with coronary plaque disruption. Circulation 2001;104:1477-82.
12. Kannel W B, Kannel C, Paffenbarger R S, Cupples L A. Heart rate and cardiovascular mortality: the Framingham study. Am Heart J 1987;113:1489-94
13. Kathl R G, Jaeckel RS, Lopez J F, Meller W H, 1989. Pathophysiology of HPA axis abnormalities in patients with major depression: an update. Am J Psychiatry 146:311-317.
14. Kannel W B, Kannel C, Paffenbarger R S, Cupples L A. Heart rate and cardiovascular mortality: the Framingham study. Am Heart J 1987;113:1489-94.