

# OXIDATIVE STRESS AND ANTIOXIDANT LEVELS IN CHRONIC BACK PAIN

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**Abstract**— Increased oxidative stress in patients of chronic back pain in comparison with healthy control subjects was investigated by measuring the Malondialdehyde (Thiobarbituric Acid reactive substances), Ascorbic acid, Superoxide Dismutase (SOD), Catalase and Glutathione Peroxides (GPX) in erythrocytes. Chronic back pain patients are more prone to oxidative damage than control cases, evident from an increase in Malondialdehyde and decreased Ascorbic acid, Catalase and GPX in erythrocytes, SOD activity-increase was found and probably as an adaptive response to the stress. The role of antioxidants in chronic back pain is clear; these are beneficial to treat this condition.

**Index Terms**— Antioxidant, Chronic back pain, Oxidative stress, Adaptive response.

## I. INTRODUCTION

Chronic back pain may be caused by muscle or ligament strain (Lumbar Muscle Strain), bulging or ruptured disks (Discogenic Back Pain), Osteoarthritis, skeletal irregularities, or Osteoporosis etc. The pain may originate from the muscles, nerves, bones, joints or other structures in the spine. In the USA, acute low back pain is the fifth most common reason for physician visits. About nine out of ten adults experience back pain at some point in their life, and five out of ten working adults have back pain every year(1). Low back pain causes 40% of missed days of work in the USA (2). It is the single leading cause of disability worldwide (3). Acute pain lasts up to 12 weeks, sub acute pain refers to the second half of the acute period (6 to 12 weeks), and chronic pain is pain which persists beyond 12 weeks (4). Nonspecific back pain is believed to be due from the soft tissues such as muscles, fascia, and ligaments (5). Lumbar disc herniation and degenerative disc disease, may not be more prevalent among those in pain than among the general population, and that the mechanisms by which these conditions might cause pain are not known (6) (7). Other studies suggest that for as many as 85% of cases, no physiological cause can be shown (8) (9).

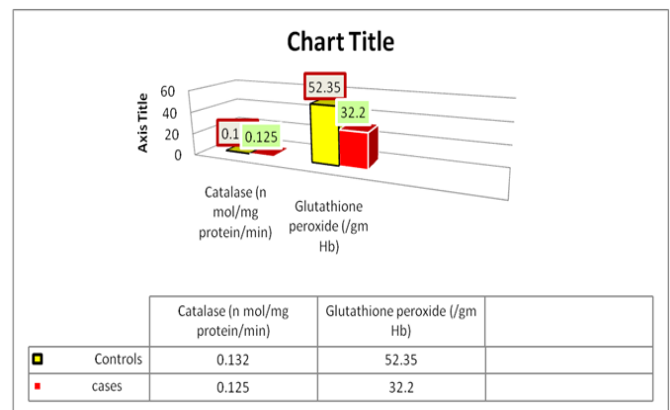
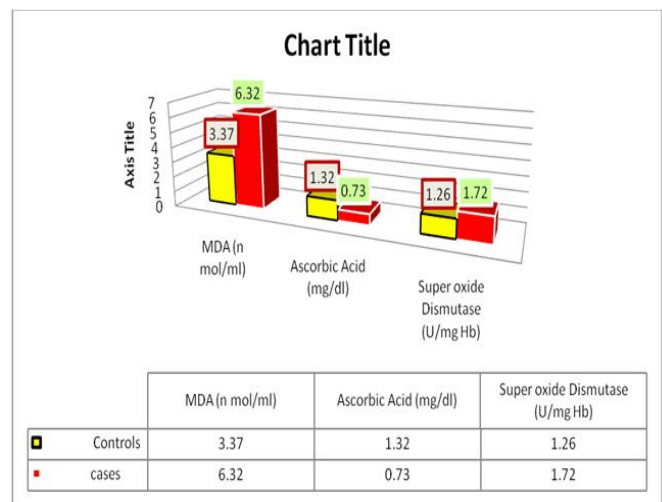
In chronic back pain, presence of free radicals mediates lipid peroxidation resulting in the soft tissue damage of these joints. Antioxidants are the substances that scavenge, suppress the formation of free radicals and even oppose their activities. The study indicates the relationship between lipid peroxidation and antioxidants in chronic back pain patients.

## LEVELS OF MDA (TBARS), ASCORBIC ACID, AND ACTIVITIES OF SUPEROXIDE DISMUTASE,

## CATALASE AND GLUTATHIONE PEROXIDASE IN ERYTHROCYTES IN CHRONIC BACK PAIN PATIENTS;

PARAMETER	CONTROLS(n=40)	PATIENTS(n=40)	t - value
MDA(n mol/ml)	3.37 ± 0.7	6.32 ± 0.14	<0.05; S
Ascorbic ACID (mg/dl)	1.32 ± 0.07	0.73 ± 0.08	<0.05; S
Super oxide Dismutase (U/mg Hb)	1.26 ± 0.04	1.72 ± 0.03	<0.05; S
Catalase (n mol/mg protein/min)	0.132 ± 0.002	0.125 ± 0.004	<0.05; S
Glutathione peroxide U/gm Hb	52.35 ± 1.41	32.20 ± 1.14	<0.05; S

Note: S - Significance



Data presented as mean ISD p<0.05 Vs the control group.

## II. MATERIALS & METHODS

Chemicals were from sigma chemical co., USA Merck Ltd India, India.

Sis co research laboratory, India.

40 chronic back pain patients (n=40) with average age 45yr (30-60yr) were selected for the study. 40 normal healthy controls of same age group were compared with those chronic backpain patients. Control subjects are healthy, with no smoking or alcoholic habit, and without any other systemic diseases like diabetes mellitus or hypertension. Venous blood was taken, heparinised blood sample were used for the estimating lipid peroxidation and the status of other antioxidants.

**Satoh method (MDA) :-**  
Malondialdehyde levels were (n mol/L) estimated by thiobarbituric acid reaction using 40% trichloroacetic acid. Proteins were precipitated from 0.5ml serum, precipitated proteins were incubated with TBA reagent in a boiling water bath for about 1 Hr, a colored complex was obtained, at room temperature at 533 nm, 1,1,3,3- tetraethoxy propane (1u ml/l) was used as the standard for the MDA estimation. The concentration of MDA is expressed in n mol/ml.

### A. Ascorbic Acid: Determined by titration method (mg/dl)

(Varley et al) Titration with 2, 6 dichlorophenol indophenol in acid solution with ascorbic acid solution, this compound is reduced to colorless leucobase. Ascorbic acid oxidized to dehydroascorbic acid. End point used blue to red to colorless. Units are Vitamin C in mg/dl of plasma.

### B. SOD(Superoxide Dismutase)

Units are U/ mg Hb, Winter bourn method was used. The ability of tetrazolium (NBT) by superoxide which is generated by the action of photo reduced riboflavin and oxygen. The blue color formed was read at 560 nm. One unit of enzyme activity was measured as the amount of SOD which causes half of the maximum inhibition of reduction of nitro-blue tetrazolium.

### C. Catalase:

Catalase activities of all the experimental samples were analyzed by following the decomposition of Hydrogen peroxide at 240 nm as described by Bonaventura et al. The disappearance of peroxide depending on the catalase activity present in the system, was observed at 240 nm for 15 mins. 1 unit of enzyme activity is that which reduces 1  $\mu$ mole of hydrogen peroxide( $H_2O_2$ ) per minute.

### D. Glutathione Peroxide (GPX): Paglia Method, U/gm/Hb.

The oxidized Glutathione produced during GPX enzyme reaction was immediately reduced by NADPH and Glutathione reductase. NADPH consumption was monitored as a measure of formation of oxidized Glutathione. Results are expressed as U/gm of Hb.

Values have been expressed as mean  $\pm$  SEM. The results were analyzed by using students "t" test for unpaired data.

P, 0.05 was considered as significant.

## III. RESULTS AND DISCUSSION

Oxidant and antioxidant parameters were measured in 40 (n=40) normal and in 40 (n=40) back pain cases. MDA levels in control and chronic back pain cases were  $3.37 \pm 0.17$  n mol/ml and  $6.32 \pm 0.14$  n mol/ml respectively. MDA levels were more in chronic back pain cases than in controls. Ascorbic acid level in control and chronic back pain cases were  $1.32 \pm 0.07$  mg% and  $0.73 \pm 0.08$  mg% respectively. Ascorbic acid level was decreased in chronic back pain cases than the controls. Super Oxide dismutase (SOD) in control and chronic back pain patients were  $1.26 \pm 0.04$  U/mg Hb and  $1.72 \pm 0.03$  U/mg Hb respectively. SOD was increased in chronic back pain cases, might be the adaptive response. Catalase in control and in chronic back pain cases were  $0.132 \pm 0.002$  n mol/mg protein/min and  $0.125 \pm 0.004$  n mol/mg protein/min respectively. Catalase was decreased in chronic back pain cases than in controls. Glutathione peroxide in control and in chronic back pain cases were  $52.35 \pm 1.14$  U/gm/Hb and  $32.20 \pm 1.14$  U/gm/Hb respectively. GPX was decreased in chronic back pain cases than the controls. Increased lipid peroxidation in chronic back pain patients as shown by the increased levels of MDA and a decrease in Ascorbic acid, Catalase activity and glutathione peroxidase seen in cases than controls. P value was  $< 0.05$  in all these parameters and were significant.

## IV. CONCLUSION:

Higher values of oxidative stress in chronic back pain were due to increase in the lipid peroxidation and to the decreased level of anti oxidants. Particularly the joint spaces damage correlates with the over production of free radicals and lack of oxygen processing enzymes and scavenging molecules. By this study we can confirm the paramount importance of antioxidant supply to the chronic backpain victims is certainly useful to reduce the symptoms of pain and favours disease prognosis along with treatment of the main etiological factor.

## REFERENCES

1. A.T. Patel, A.A. Ogle. "Diagnosis and Management of Acute Low Back Pain". American Academy of Family Physicians. Retrieved March 12, 2007.
2. Manchikanti, L; Singh, V; Datta, S; Cohen, SP; Hirsch, JA; American Society of Interventional Pain, Physicians (Jul–Aug 2009). "Comprehensive review of epidemiology, scope, and impact of spinal pain.". Pain physician 12 (4): E35–70. PMID 19668291.
3. Institute for Health Metrics and Evaluation. "2010 Global Burden of Disease Study"
4. King W (2013). Encyclopedia of pain. Springer Reference.
5. Siegfried Mense, Robert D. Gerwin, (2010). Muscle pain : diagnosis and treatment (Online-Ausg. ed.). Heidelberg: Springer. p. 4. ISBN 978-3-642-05467-9.
6. Borenstein DG, O'Mara JW, Boden SD; et al. (2001). "The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects: a seven-year

- follow-up study". *J Bone Joint Surg Am* 83–A (9): 1306–11. PMID 11568190.
7. Savage RA, Whitehouse GH, Roberts N (1997). "The relationship between the magnetic resonance imaging appearance of the lumbar spine and low back pain, age and occupation in males". *Eur Spine J* 6 (2): 106–14. doi:10.1007/BF01358742. PMC 3454595. PMID 9209878.
8. White AA, Gordon SL (1982). "Synopsis: workshop on idiopathic low-back pain". *Spine* 7(2): 141–9. doi:10.1097/00007632-198203000-00009. PMID 6211779.
9. van den Bosch MA, Hollingworth W, Kinmonth AL, Dixon AK (2004). "Evidence against the use of lumbar spine radiography for low back pain". *Clinical radiology* 59 (1): 69–76. doi:10.1016/j.crad.2003.08.012. PMID 14697378.