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Research Article

ALTERATION OF ANTIOXIDANT STATUS AND INCREASED LIPID-PEROXIDATION LEVELS IN RHEUMATOID ARTHRITIS AND OSTEOARTHRITIS PATIENTS COMPARED WITH NORMAL INDIVIDUALS

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A R T I C L E I N F O

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A B S T R A C T

Back Ground: Rheumatoid Arthritis is a chronic multisystem disease of unknown etiology. The characteristic feature of Rheumatoid arthritis is persistent inflammatory synovitis, usually involving peripheral joints in a symmetric distribution. Osteoarthritis is one of the most prevalent and disabiling chronic diseases affecting elderly. The objective of this study to evaluate the non-enzymatic antioxidant and status of lipid-peroxidation status in patients suffering RA and OA compare with normal individuals. In this study lipid-peroxidation is a marker of oxidative stress measured by MDA.

Materials and Methods: in the present study we take total 300 patients in which 100 Rheumatoid arthritis patients (RA), 100 Osteoarthritis Patients (OA) and 100 control means these people have not any disease. In this study we evaluate serum lipid-peroxidation by Utleys methods and non enzymatic antioxidant (Vitamin E & Vitamin C) measured by Emer Engel and Carl A Burtis Method with the help of colourimeter.

Result: In this study we found increased lipid-peroxidation and decreased vitamin E and Vitamin C levels. These results are highly significant in both the group as compared to control.

Conclusion: In the present study altered non enzymatic antioxidant and increased MDA levels shows that generation of free radical is a causative factor of rheumatoid arthritis and osteoarthritis.

INTRODUCTION

Rheumatoid Arthritis is a chronic multisystem disease of unknown etiology. The characteristic feature of Rheumatoid arthritis is persistent inflammatory synovitis, usually involving peripheral joints in a symmetric distribution. This synovial inflammation causes cartilage destruction and bone erosion and subsequent changes in joint integrity. The rheumatoid synoviam is witness to a complex interplay between a wide variety of cellular chemical, enzymatic, nutritional and genetic elements and is characterized by the presence of a number of secreted products of activated lymphocytes, macrophages and fibroblasts. The local production of these cytokines and chemokines may account for many of the pathological and clinical manifestation of rheumatoid arthritis.

The current concept holds that osteoarthritis involves the entire joint organ, including the subchondral bone, menisci, ligaments, periarticular muscle, capsule and synovium[1,2]. The etiology of knee OA is multifactorial, excessive musculoskeletal loading, high body mass index, previous knee injury, female gender and muscle weakness are well known factor [3] .

The imbalance between pro-oxidant and antioxidants gives rise to cellular oxidative stress, which plays an important role in the progression of OA and RA [4]. The process of free radical formation plays an major part in the development of chronic and degenerative llness such as cancer, autoimmune disorders, aging, arthritis and neurogenerative disease [5]

Lipid-peroxidation mediated by free radical is considered to be the major mechanism of cell membrane destruction and cell damage. Free radical are formed in both physiological condition in mammalian tissues [6] the uncontrolled production of free radical is considered as an important factor in the tissue damage induced by several pathophysiology. Alterations in the oxidant- antioxidant profile known to occur in rheumatic diseases.

In the present study we evaluate the non-enzymatic antioxidant (Vitamin E and Vitamin C) and levels of lipid-peroxidation in both the study group.

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MATERIALS AND METHODS

The study was undertaken in the Department of Biochemistry, S.R.N. Hospital, M.L.N. Medical College, Allahabad. In which 300 individuals of different age (20-70 years). In which 100 normal individuals and 100 rheumatoid arthritis and 100 osteoarthritis patients. All Patients were clinically evaluated.

The subjects were categorized into three groups:

1. ***Control Group:*** Normal individuals they are free from any disease and infection. The age of this group is 30-70 years.
2. ***Study Group I (RA):*** in this group patient suffering from RA the age group of patents is 30-70 years.
3. ***Study Group II (OA):*** in this group patient suffering from OA the age group of patients is 30-70 years.

The parameters mainly observed were oxidative stress markers lipid-peroxidation, cerulloplasmin, and non-enzymatic (Vitamin E and Vitamin C) antioxidants were evaluated. Differences within and between groups from baseline to end using T-test.

RESULT

In the present study we found increased serum lipid-peroxidation, superoxide-dismutase and also elevated levels of serum cerulloplasmin levels and decreased levels of vitamin E and vitamin C in both the study groups. These results are statistically significant in both the group. The result shows in the table. In this study we got serum vitamin C levels in the osteoarthritis patients and rheumatoid arthritis patients was 0.53±0.07 and 0.64±0.06mg/dl) which was increased in OA and RA as compared to control.

Serum Vitamin E levels in OA and RA was 0.45±0.03mg/dl) which was increased in both the study group as compared to control. These are statistically significant.

Serum lipid-per oxidation levels in Osteoarthritis and Rheumatoid arthritis patients was 2.19±1.21 and 4.64±0.229 increased that of controls. Serum SOD levels in OA and RA was 2377±38.5 which was increased in study group as compared control. Cerulloplasmin levels also increased in study group as compared normal individuals the result are OA and RA 43.65±16.05 and 56.75 ±19.5.

Observation Table

Levels Non-enzymatic antioxidant status and Lipid-peroxidation, Superoxide-dismutase, Cerulloplasmin levels in both the study groups compared to control groups

S.N.	Particulars	Control (n=100)	OA (n=100)	RA Patients (n=100)
1	Vitamin C	40.87±0.25	0.53±0.07	0.64±0.06
2	Vitamin E	0.907±0.25	0.45±0.03	0.33±0.02
3	Lipid-peroxidation	1.68±0.99	2.19±1.21	43.65±16.05
4	Superoxide-dismutase	2166±145	2377±38.5	2450±40.10
5	Cerulloplasmin	40.81±11.35	0.64±0.06	56.75±19.5

DISCUSSION

Rheumatoid arthritis is one of the most common inflammatory diseases worldwide. In the present study elevated levels of MDA and multidirectional antioxidant were native in both arthritic patients as compared to control.Rheumatoid arthritis is a major cause of morbidity as it affects the joints, causing stiffness and loss of mobility. The cause of rheumatoid arthritis is mainly joint inflammation initiated by oxidative stress. Involvement of oxygen free radicals (OFR) in the pathophysiology of inflammation in a number of organs and tissues has been reported in literature [13], Free radicals are enormously produced at the site of inflammation and tissue injuries. Lipid peroxide that are generated at the site of inflammation of tissue injury into blood and can be estimated in serum or plasma, which inturn reflect the severity of the tissue damage [14]. Significantly lower levels of Vitamin E and Vitamin C were found in RA patients as compared to control group. Vitamin E helps to trap free radicals and interrupt the chain reaction that damages the cells. Regeneration of Vitamin E depends on Vitamin C. As there is an increased oxidative stress in RA there may be increased consumption of Vitamin C and Vitamin E. This reduction in ascorbate levels suggests its role in combating oxidative stress.

In the present study serum MDA was found significantly higher in OA and RA patient as compared to control. Our findings for lipid-peroxidation in both the study groups are similar to the study of Akyol *et al* [15]. They reported elevated MDA levels in their study. In contrast of our study, Kajanachumpol *et al* [16] reported no significant changes in MDA levels in rheumatoid arthritis patients as compared to control. The elevated MDA level in our OA patients coincide with result of Surapaneni *et al* [17], Maneesh *et al*.[18] & Rubyk *et al*.[19] who reported significantly increased serum MDA levels in OA patients compared to control. Thus, these findings are in keeping with possible evidence of free radical production & damage in OA.

According to Mezes *et al* [20] and Ciemen MY *et al* [21], SOD is the important antioxidant enzyme having an antitoxic effect against superoxide anion. The over expression of SOD might be an adaptive response and it result in increased in dismutation of superoxide to hydrogen peroxide. Ostalowska *et al* [22] have reported increased activities of superoxide dismutase in synovial fluid of patients with primary and secondary osteoarthritis of the knee joint.Recklies *et al* [23] mentioned that SOD is the first line of defense against ROS; it catalytes thedismutation of the superoxide anion into hydrogen peroxide. Ceruloplasmin, Cu containing protein, has been found to be increased in RA patients as compared to control. Increased levels of Ceruloplasmin may be related to its scavenging action of superoxide radicals that are generated during the inflammatory process of RA [24]. Cerulloplasmin (Cp) is an acute phase protein that is primarily synthesized in the liver and secreted into the blood. It is a prominent antioxidant that can scavenge ROS [25]. We observed a significant increase in plasma Cp in OA patients was significantly higher than in control. In agreement with our findings, many authors [26] observed increased plasma Cp level in RA. On the other hand, Ashour *et al*. [27] stated that the raised levels of Cp are significantly increased in RA group but not in OA group. This outstanding agreement about Cp level in RA was emphasized by Nagler *et al*. [28] as Cp is considered the principal plasma and synovial antioxidant in RA, being responsible for up to 70% of the protective capacity against superoxide free radicals. Nevertheless, Louro *et al*. [29] stated that although the increase in the concentration of Cp might offer an additional safeguard against oxidative stress.

Our findings inconcordance with the findings of Surapaneni KM *et al*.[17]. They observed significantly increased levels of

erythrocyte SOD and significantly decreased Vitamin E and ascorbic acid levels in osteoarthritis patient as compared to control. The result suggest higher oxygen free radical production, evidence by increased SOD, increased MDA and decreased Vitamin E and Vitamin C activity, support to the oxidative stress in osteoarthritis. The increased activities of antioxidant enzymes may be a compensatory regulation in response to increased oxidative stress.

CONCLUSION

In the present study we observed elevated levels of lipid-peroxidation, superoxide-dismutase and extracellular antioxidant Cerulloplasmin in patient with osteoarthritis and rheumatoid arthritis as compared to control. Oxidative stress may be involved in rheumatoid arthritis and osteoarthritis.

Reference

1. Deborah PM. Epidemiology of rheumatoid arthritios: determinants of onset, persistence and outcome. *Clin Rheum* 2002; 111:172-7.
2. Anandacoomarasamy A., March L. (2010) Current evidence for osteoarthritis treatments.Ther Adv Musculoskel Dis 2:17-28.
3. Takeda H., Nakagawa T.,Nakamura K., Engebretsen L. (2011) Prevention and management of knee osteoarthritis and knee cartilage injury in sports. *Br J Sports Med* 45: 304-309.
4. Martin J. A., Buckwalter J A. (2002) Aging, articular cartilage chondrocyte senescence and osteoarthritis. *Biogerontology* 3: 257-264.
5. Halliwell B.(1995) Oxygen radicals, nitric oxide and human inflammatory joint disease. *Ann Rheum Dis* 15: 494-501.
6. Cox MM, Nelson DL. Lehninger Principles of Biochemistry. 5th Edition, p. 259, W.H.Freeman & Company, New York, 2008.
7. American College of Rheumatology and HDC Committee on Clinical Guidelines 1996.
8. Hasim, S.A. & Schuttranger, G 1996. *Am. J. Clin. Nutrl* August 19; 137-141**.**
9. Burtis, C.A. 1994 Tietz Text book of Clinical Chemistry. 2 WEI.
10. Utley, H.G. Bernhein, F& Hochstein, P. (1967) Arch. Biochem, Biophys,118-28
11. Marklund S, Markuland G. A simple assay for superoxide dismutase using autooxidation of pyrogallol. *European Journal Biochem*. 1974: page No, 469-72.
12. Ravins, H.A. (1961), *J. Lab.Clin.Med,* 58,161.
13. De Bandt M, Grossin M, Driss F, Pincemail J, Babinchevaye C, Pasquier C. Vitamin E uncouples joint destruction and clinical inflammation in a transgenic mouse model of rheumatoid arthritis.
14. Gotia, S., Popovici, I. and Hermeziu, B. 2001. Antioxidant enzymes levels in children with juvenile rheumatoid arthritis. *Southeast Asian J Prop Med Public Health*; 31 (2): 335-8.
15. Akyol O, Isci N, Temel I, Ozgocmen S, Uz E. The relationships between plasma and erythrocyte antioxidant enzymes and lipid peroxidation in patients with rheumatoid arthritis. *Joint Bone Spine* 2001; 68: 311-7.
16. Surapaneni KM, Venkataraman G. 2007. Status of lipid peroxidation, glutathione, ascorbic acid, vitamin E and antioxidant enzymes in patients with osteoarthritis. *Indian J Med Sci.* Jan; 61 (1): 93-14.
17. Krishna Mohan Superaneni, G. Venkataraman, 2007. Status of lipid peroxidation, Glutathione Ascorbic acid, Vitamin E and Antioxidant enzymes in patients with osteoarthritis.
18. M Maneesh, H Jayalekshmi, T Suma Chatterjee, A Chakrabarti and TA Singh, 2005. *Indian Journal of Clinical Biochemistry,* 20 (1) 129-130.
19. Rubyk BI, Fil chagin NM, Sabadyshin RA. Change in lipid peroxidation in patient with primary osteoarthosis deformans. *Ter Arkh* 1988; 60 (9): 110 -113.
20. Mezes M, Bartosiewiez G 1983. Investigation on vitamin E and lipid-peroxide status in rheumatic disease. *Clin Rheumatol*; 2 (3):259-63.
21. Cimen MY, Cimen OB, Kaemaz M, Ozturk HS, Yorgancioglu R, Durak I. 2000. Oxidant/ antioxidant status of the erythrocytes from patients with rheumatoid arthritis. *Clin Rheumatol*: 19 (4): 275-7.
22. Ostrakhovitch, E.A. and Afanas'ev, I.B. 2001. Oxidative stress in rheumatoid arthritis leukocyte: suppression by rutin and other Antioxidants and chelators. Biochem. *Pharmacol*. 62, 743-746.
23. Recklies AD, Poole AR, Banerjee S, Bogoch E, Dibattista J, Evans CH, et.al. Pathophysiologic aspects of inflammation in diarthroidal joints. In: Buckwalter JA, Einhorn TA, Simon SR, editors. Orthopaedic basic science: biology and biomechanics of the musculoskeletal system, 2nd ed. Rosemont, IL: AAOS; 2000.p.489-530.
24. RoshanAlam, Saba Khan and Khushtar Anwar Salman. Evaluation of antioxidants and lipid peroxidation status in rheumatoid arthritis patients. *NJIRM* 2013; 4(5): 1-5.
25. Gambhir JK, Lali P, Jain AK. Correlation between blood antioxidant levels and lipid peroxidation in rheumatoid arthritis. *Clin Biochem* 1997; 30:351-5. 2003;42(10):1234-41.
26. Kamanl A, Naziroglu M, Aydilek N, Hacievliyagil C. Plasma lipid peroxidation and antioxidant levels in patients with RA. *Cell Biochem Funct* 2004;22(1):53-7.
27. Ashour M, Salem S, Hassaneen H, El-Gadban H, Elwan N, Awad A, *et al*. Antioxidant status in children with juvenile rheumatoid arthritis living in Cairo, Egypt. *Int J Food Sci Nutr* 2000; 51: 85-90.
28. Nagler RM, Salameh F, Reznick AZ, Livshits V, Nahir AM. Salivary gland involvement in rheumatoid arthritis and its relationship to induced oxidative stress. *Rheumatology*
29. Louro MO, Cocho JA, Mera A, Tutor JC. Immunochemical and enzymatic study of ceruloplasmin in rheumatoid arthritis. *J Trace Elem Med Biol* 2000; 14(3):174-8.

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