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RESEARCH ARTICLE

LIPID PEROXIDE LEVEL AND ANTIOXIDANT STATUS IN PSORIATIC PATIENTS

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ABSTRACT

Psoriasis is a chronic inflammatory, proliferative skin disease characterized by pathological skin lesions due to various exogenous and endogenous factors. It is associated with a number of biochemical and immunological disturbances. Recently, it has been suggested that increased reactive oxygen species (ROS) production and compromised function of antioxidant system may be involved in the pathogenesis of this disease. This study was an attempt to detect the levels of antioxidants as well as to assess the possible role of lipid peroxidation in patients with psoriasis. It was a case control study of seventy five (clinically proved and histopathological confirmed) psoriatic patients and an equal number of age and sex matched normal healthy individuals served as controls. All the subjects were interviewed as per the proforma designed. The study compared lipid peroxidation product in the form of Malondialdehyde (MDA) levels and antioxidant status in the form of Superoxide dismutase (SOD) in psoriatic patients and healthy controls. The results were tabulated and the data was analyzed statistically to draw the relevant inferences.

INTRODUCTION

Psoriasis is a common, chronic, disfiguring, inflammatory and proliferative condition of the skin, in which both genetic and environmental influences have critical role. It presents chronic sharply demarcated, dull red, scaly plaques, particularly on extensor prominences and the scalp.[1, 2] Normally, skin cells mature and shed after about a month. However in psoriasis, the normal cycle of replacing old skin cells with new one becomes unbalanced.[3]

Psoriasisis characterized by red, scaly patches, papules and plaques, which usually itch. The skin lesions seen in psoriasis may vary in severity from minor localized patches to patches that covers the complete body. [4] The disease affects 2–4% of the general population. [5]

The cause of psoriasis is not fully understood, but a number of theories exist. Around one-third of people with psoriasis report a family history of the disease, and researchers have identified genetic loci associated with the condition. Identical twin studies suggest a 70% chance of a twin developing psoriasis if the other twin has the disorder. The risk is around 20% for non-identical twins. These findings suggest both a genetic susceptibility and an environmental response in developing psoriasis.[6]

Oxidative stress is currently receiving attention as an important mediator of psoriasis. It is a condition that occur when the steady state balance of pro-oxidants and anti-oxidants is shifted in the direction of the former leading to alteration of cell's 'redox state' to 'oxidized state' creating the potential for oxidative damage leading to cell death and even necrosis.

The skin is a potential target for oxidative injury, as it is continuously exposed to UV radiation and other environmental stresses generating reactive oxygen species (ROS).[7] ROS mediated oxidative damage involves a vast number of biological molecules since it causes lipid peroxidation, DNA modification, and secretion of inflammatory cytokines.[8]

Plasma membranes of the skin cells in the psoriatic lesion have a significant increase in arachidonic acid, which is the natural substrate for synthesis of malondialdehyde (MDA), an end product of lipid peroxidation. [9]

One important line of defense is a system of enzymes which include superoxide dismutase (SOD) and other antioxidants which decrease the concentration of the most harmful

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oxidants.[10]A major contribution to the total antioxidants comes from antioxidant molecules in plasma. Antioxidants can protect the epidermis from the events that contribute to epidermal toxicity and diseases. Deficiencies in any of the antioxidant defense system can cause a reduction in the total antioxidant status (TAS) of an individual.[3] However, inadequate antioxidant protection or excess ROS production creates a condition known as oxidative stress, contributing to the development of cutaneous disease and disorders.[11]

Until recently, it was difficult to treat psoriasis, due to incomplete understanding of the factors behind pathogenesis of psoriasis which points out that there may be some lacuna in our understanding of etiopathogenesis of psoriasis. Therefore, the present study was planned to investigate the possible involvement of lipid peroxidation-antioxidant status in psoriatic patients.

MATERIAL AND METHODS

The present study was undertaken in the Department of Biochemistry; Government Medical College, in Collaboration with Department of Dermatology, Guru Nanak Dev Hospital affiliated to Govt. Medical College Amritsar.

It was a case control study. 75 clinically diagnosed patients of psoriasis were selected for study from OPD and Ward of Department of Skin, Guru Nanak Dev Hospital, Amritsar. The criteria to diagnose psoriasis was based on physical examination, clinical examination and histopathological examination (whenever required). Patients were divided into three groups according to severity of disease.

Group 1- Mild Psoriasis (area covered is <3%)
Group 2- Moderate Psoriasis (area covered is 3-10%)
Group 3- Severe Psoriasis (area covered is >10%)

Equal number of age and sex matched healthy volunteers from the same population but without any previous and present illness were included in the study to serve as controls.
Inclusion Criteria:

The following patients were included in the study

1. The patients having clinical features of psoriasis and histopathological examination (wherever required).
2. Psoriatic patients of age group 20 to 60 years.
3. Patients who had not taken any systemic drug therapy for the disease from last two months.

Exclusion Criteria

The subjects with past or present history of any disease like:

1. Atherosclerosis
2. Coronary Artery Disease (CAD)
3. Diabetes Mellitus
4. Smoking etc.

which may affect oxidative stress were excluded from the study.

Informed consent was taken and verified.

Collection and processing of blood sample

4 ml of venous blood was taken from psoriatic patients and healthy controls in dry disposable syringe under aseptic conditions by vein puncture in anticubital vein, in a dry sterile and acid washed vial for biochemical analysis. This blood was allowed to stand for half an hour, after the clot formation, the blood sample was centrifuged at 3000 rpm for 10 minutes and serum was taken for investigations.

All the samples were assessed for serum levels of MDA and SOD.

Biochemical investigations

1. Estimation of serum MDA
2. Estimation of serum SOD

RESULTS AND DISCUSSION

Demographic Data Profile

Age Group / Sex	20-30 yrs	30-40 yrs	40-50 yrs	50-60 yrs
Male	9	9	7	12
Female	11	10	11	6

Comparison of Serum Sod Levels in Different Groups of Psoriatic Patients under Study

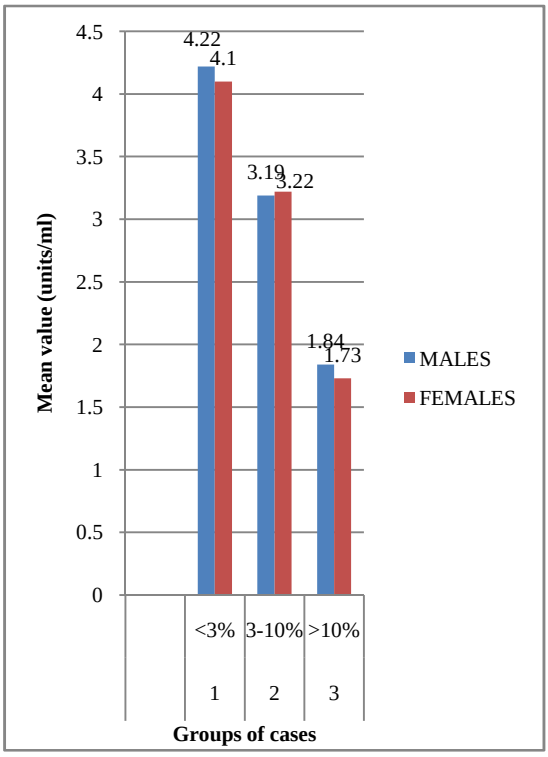

Table I

Table I shows the comparison of serum SOD levels in different groups of psoriatic patients under study. The patients were divided into three groups according to the involvement of body surface area (BSA). These groups were compared with each other. The comparison was also done between males and females of same group. The mean±SD of SOD in group I males was 4.22±0.37 and females was 4.10±0.33. The mean±SD of SOD in group II males was 3.20±0.28 and females was 3.22±0.26. And similarly mean±SD of SOD in group III males was 1.84±0.26 and females was 1.73±0.26. When males and females of same group were compared with each other, the difference in SOD levels was proved to be statistically insignificant. When group I was compared with group II and III and group II was compared with group III, then the difference in SOD levels was proved to be statistically highly significant.

Comparison of Serum Mda Levels In Different Groups Of Psoriatic Patients Under Study

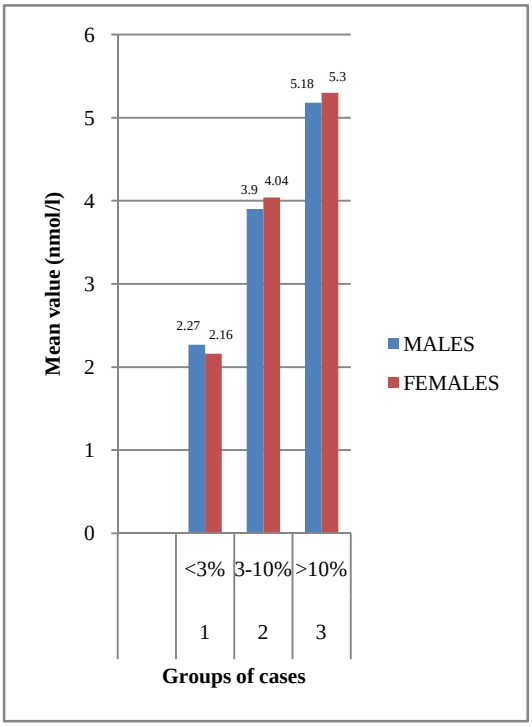

Table II

Table IIshows the comparison of serum MDA levels in different groups of psoriatic patients under study. The patients were divided into three groups according to the involvement of body surface area (BSA). These groups were compared with each other. The comparison was also done between males and females of same group. The mean±SD of MDA in group I males was 2.27±0.48 and females was 2.16±0.38. The mean±SD of MDA in group II males was 3.9±0.40 and females was 4.04±0.56. And similarly mean±SD of MDA in group III males was 5.18±0.31 and females was 5.30±0.35. When males and females of same group were compared with each other, the difference in MDA levels was proved to be statistically insignificant. When group I was compared with group II and III and group II was compared with group III, then the difference in MDA levels was proved to be statistically highly significant.
Lipid peroxidation represents tissue damage caused by hydrogen peroxide, superoxide anion and hydroxyl radicals, resulting in structural alteration of membrane with the release of cell and organelle contents, loss of essential fatty acids with the formation of cytosolic aldehyde and peroxide products.[12]

In this study, a significant increase in serum MDA levels ($p<0.001$) was observed in cases compared to controls as shown in table II. Serum MDA levels amongst cases ranged between 1.4-6.3nmol/ml with mean ±SD of 3.7±1.1 while in comparison the corresponding range amongst controls was 0.4-1.5nmol/ml with mean ±SD of 0.81±0.15.

Increase in MDA levels observed could be due to increased oxidative stress or decrease in antioxidant defense mechanism.

There was significant decrease in serum SOD levels ($p<0.001$) in cases as compared to controls (Table I) that were similar to findings of other invigilators. The serum SOD levels in psoriatic patients were ranged between 1.3–5.0 U/ml with mean ±SD 3.12 ±0.87 while in comparison the corresponding values amongst controls were 3.6 – 6.8U/ml with mean ±SD 5.13 ±0.69.

Our study indicates the possibility that, in the prediagnostic stage, serum antioxidants are low because they have been used in reducting inflammatory products. Decreased SOD activity might be related to epidermal hyper proliferation, because the ROS are thought to induce cell proliferation in various cell systems.[13,14]

Increased oxygen metabolism has been described in the psoriatic hyperproliferative epidermis, which depends on cutaneous blood flow. Increased O2•− production in the presence of decreased antioxidant activity would result in the accumulation of H_2O_2, which has an inhibitory effect on SOD activity.[13,15]

The results of our present study suggests that the oxidation is may be a very early and initiating event in overall process in sequence of events leading to psoriasis. Since, the natural antioxidant defense system SOD limits the development of inflammatory and immune processes, we believe that, the tendency to decreased SOD activity in psoriasis patients is oneof the reasons for the aggravated dermatological status.

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