Status of Serum Lipids and Oxidative Stress in Psoriatic Cases

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ABSTRACT

Aim: Psoriasis is a common chronic inflammatory skin disease with an unknown etiology. Our aim is to evaluate the status of serum lipids and oxidative stress in psoriatic cases. Materials and methods: Study group comprised 30 newly diagnosed cases of psoriasis attending OPD of dermatology department, Mamata General hospital, Khammam. 30 age and sex matched healthy subjects were recruited as controls with informed consent. Ethical committee clearance was taken. Cholesterol, Triglyceride, HDL cholesterol, LDL cholesterol, Malondialdehyde (MDA) and Total antioxidant capacity were measured in serum of psoriatic cases and controls. Results: Mean serum levels of MDA, Cholesterol, Triglycerides and LDL cholesterol were significantly increased and TAC and HDL cholesterol were significantly decreased in psoriatic cases compared to controls. Conclusion: Our results revealed that psoriasis is associated with dyslipidemia and altered oxidative stress status.

KEYWORDS: MDA, Psoriasis, TAC, dyslipidemia.

INTRODUCTION

Psoriasis is a chronic and recurrent skin disorder characterized by marked inflammatory changes in the epidermis and dermis. The worsening of psoriasis has been linked with oxidative stress [1]. Disorders in the antioxidant defense mechanisms are known to be involved in the pathogenesis of psoriasis [2, 3].

The skin is a potential target for oxidative injury, as it is continuously exposed to UV radiation and other environmental stress generating reactive oxygen species (ROS) [4]. ROS mediated oxidative damage involves a vast number of biological molecules since it causes lipid peroxidation, DNA modification, and secretion of inflammatory cytokines [5]. Plasma membranes of the skin cells in the psoriatic lesions have a significant increase in arachidonic acid, which is the natural substrate for synthesis of malondialdehyde (MDA), an end product of lipid peroxidation [6]. Antioxidants can protect the epidermis from the events that contribute to epidermal toxicity and diseases. Deficiencies in any of the antioxidant defense systems can cause a reduction in the total antioxidant capacity (TAC) of an individual [7].

However, inadequate antioxidant protection or excess ROS production creates a condition known as oxidative stress, contributing to the development of cutaneous disease and disorders [8]. In vivo antioxidant status can be assessed by measuring individual plasma or tissue levels of antioxidants. Measuring the levels of these specific antioxidant molecules can yield valuable information, and low levels of such antioxidants provide suggestive, but not definitive, evidence of oxidative stress. However, determining total antioxidant capacity provides an index of the sum of the activities of all antioxidants [7].

Psoriasis is a common chronic and recurrent inflammatory skin disorder [2] that has been associated with abnormal lipid metabolism and high frequency of cardiovascular events [1]. This prevalence seems to be related to the severity of psoriasis, as it occurs more frequently in patients presenting large areas of the body affected with psoriasis lesions. Though dyslipidemia is known to occur, less is known about its status and association with oxidative stress in patients of psoriasis. Hence, this study was carried out to determine the status of serum lipids and oxidative stress in psoriatic cases.
evaluate the oxidative stress and dyslipidemia in patients with psoriasis.

MATERIALS AND METHODS

Study group is comprised of 30 newly diagnosed cases of psoriasis attending OPD in dermatology dept of Mamata General Hospital, Khammam. Cases were diagnosed based on Auspitz sign, clinical features of psoriasis like erythema, itching, thickening and scaling of the skin. The clinical severity was determined according to the Psoriasis Area and Severity Index (PASI) score. 30 age and sex healthy subjects were recruited as controls from hospital staff and patient attendants.

Informed consent and clearance from ethical committee was taken. The subjects having Diabetes mellitus, Hypertension, Coronary heart disease, renal disease, endocrine disorders, liver dysfunction, are excluded. Smokers, chronic alcoholics and subjects on antioxidant supplements and lipid lowering drugs are excluded from the study.

Under aseptic precautions 5ml fasting blood sample was collected and serum was separated after clot retraction. The serum was analyzed for levels of serum lipids on the same day. Remaining serum was aliquoted and stored at -80 °C until analysis of Malondialdehyde and Total antioxidant capacity.

Fasting lipid profile [Total cholesterol, High density lipoprotein Cholesterol (HDL-cholesterol), Triglycerides] was done to all patients by using enzymatic kits on biochemistry autoanalyser [tulip diagnostics, India].

Cholesterol:Enzymatic-Cholesterol oxidase peroxidase method

Triglyceride:Glycerol phosphate oxidase peroxidase method

HDL cholesterol: Phospho tungstate precipitation method

LDL cholesterol values have been estimated using the Friedwald formula:

[Total cholesterol]–[HDL cholesterol+Triglycerides5] =Estimated LDL-cholesterol [9].

Malondialdehyde (MDA) is determined as Thiobarbituric acid reactive substances (TBARS)[10]. Estimation of Total Antioxidant Capacity using the FRAP (Ferric Reducing Ability of Plasma) assay. [11]

Statistical Analysis: Mean±S.D values of all biochemical parameters were calculated in study and control groups and the mean difference was compared by using student ‘t’ - Test.

RESULTS

Mean serum cholesterol level in study group was 189.73 ±33.1 mg / dl & in controls was 159.63 ±17.55mg /dl .The mean difference was compared statistically & the increase was significant (P<0.0001). The mean HDL-Cholesterol in controls was 41.53 ± 3.03 mg/dl. While the mean value of HDL-Cholesterol in study group is 37.30 ± 2.98 mg/dl. There is significant (P<0.0001) difference in this HDL-Cholesterol in control group & study group. The LDL-Cholesterol mean level in normal individuals was 95.23±14.39mg/dl. In study group the LDL-Cholesterol was significantly raised (p<0.0001) being 124.43 ± 31.56 mg/dl.

Increase was observed when the mean serum triglyceride levels were compared between study (138.30±51.28 mg/dl.) and control groups (114.43 ± 38.80 mg/dl).It was statistically significant (p <0.0467) [Table1].

Table: 1 Mean Total Cholesterol, HDL, LDL, TG Levels In Control Group & Study Group

<table>
<thead>
<tr>
<th>Parameter (mg/dl)</th>
<th>Control group Mean ± SD</th>
<th>Study group Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total .Cholesterol</td>
<td>159.63±17.55</td>
<td>189.73±33.1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>HDL</td>
<td>41.53±3.03</td>
<td>37.30±2.98</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LDL</td>
<td>95.23±14.39</td>
<td>124.43±31.56</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>114.43±38.80</td>
<td>138.30±51.28</td>
<td>&lt;0.0467</td>
</tr>
</tbody>
</table>

Mean MDA level in study group was 3.067±1.168 nmol/ml and in controls was1.657 ± 0.602 nmol/ml. The mean difference was compared statistically & the increase was significant.(<0.0001). The Total antioxidant capacity in control group is 1.52± 0.338 µmol/ml, while in Psoriatic patients the mean TAC levels have significantly (p<0.0001) lowered to 0.556±0.267 µmol/ml (Table 2).

Table 2: Mean MDA & TAC Levels In Control & Study Group

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control group Mean±SD</th>
<th>Study group Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDA(nmol/ml)</td>
<td>1.657±0.602</td>
<td>3.067±1.168</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>TAC(umol/ml)</td>
<td>1.152±0.338</td>
<td>0.556±0.267</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>
DISCUSSION

In our study serum lipids and oxidative stress are assessed in psoriatic cases. We observed that mean serum levels of total cholesterol, TG, LDL-C, MDA in cases of psoriasis have increased significantly, when compared with controls. Mean serum HDL-C & TAC levels are decreased significantly when compared with controls. These observations are consistent with previously mentioned related studies Javidi Z et al [12], akhyani et al [13], and Sommer DM et al [14], Rocha Pereira P. et al. [15]and Relhan V et al. [16]. However, Yildrium et al. [17] did not find any correlation in the levels of MDA in patients of psoriasis with that of controls.

Several mechanisms including an unhealthy lifestyle, activation of type 1 helper T cells, auto antibodies recognizing oxidized LDL and some medications used to treat psoriasis such as oral retinoids and cyclosporine may induce dyslipidemia in psoriatic patients [18, 14, 19]. Also structural and functional abnormalities have been found in nearly all the segments of the gastrointestinal tract in psoriatic patients [20]. Moreover, the level of antibodies against oxidized LDL correlates with the disease severity.

Thus, psoriasis is a risk factor for hyperlipidemia and its possible subsequent sequelae such as obstructive vascular disease cannot be excluded [20]. Moreover, the level of antibodies against oxidized LDL correlates with the disease severity. ROS may be produced during the inflammatory process, in psoriasis, affecting primarily lipid metabolism of cells. Further, ROS that are produced by lipid peroxidation may activate phospholipase A2 and thus cause peroxidation of many mediators by arachidonic acid which finally metabolized to MDA [6,8,17].

The skin is constantly exposed to oxidative stress induced by ROS that are generated both from endogenous neutrophils and external pro-oxidant stimuli [5]. Evidences show that increased oxidative stress in these patients as demonstrated by the high plasma MDA levels and compromised levels of the antioxidant defense enzymes are observed at the time of diagnosis itself [21].

CONCLUSION

Dyslipidemia and increased oxidative stress are observed in patients with psoriasis. Psoriatic cases should be evaluated for status of serum lipids and oxidative stress, which can help in the management of these cases. Correction of dyslipidemias and antioxidant supplementation, can reduce the overall morbidity, enhance the prognosis of Psoriasis and minimize the future cardiovascular risk.

REFERENCES


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