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# A Study on Assessment of Systemic Inflammation, Oxidative Stress and Apolipoprotein B/A1 Ratio in Active Psoriasis Patients

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**ABSTRACT**

According to conjecture, oxidative stress and dyslipidemia are linked to psoriasis inflammation. This complex interplay is thought to be the mechanism behind the development of future CVD risk. In order to assess systemic inflammation, oxidative stress, and the apolipoprotein B/A1 ratio in patients with active psoriasis. Venoscent punctures were used to obtain roughly 10 milliliters of blood from patients who had fasted overnight and placed in sterile EDTA vials. Total cholesterol, triglycerides, and HDL cholesterol were the three components of the plasma lipid profile that were analyzed enzymatically using a kit purchased from (Randox Laboratories Limited, Crumlin, UK). Using the immunoturbidity method (Randox kit), plasma apolipoproteins B and A1 were measured, and the ratio of ApoB to A1 was determined. In comparison to healthy controls, psoriasis patients show negligible variance ($p_s<0.1$) in their age and blood pressure. Table 2 displays markers of the state of oxidative stress and systemic inflammation. In comparison to the healthy control group, the patient group's plasma CRP and erythrocyte MDA levels were found to be significantly higher ($p<0.001$, 41.02% and $p<0.05$, 29.3% high), indicating the importance of inflammation and oxidative stress in the disease process. Based on current research, it is suggested that in addition to lipid profile, apolipoproteins and indicators of oxidative stress and inflammation should be routinely assessed in order to anticipate CVD complications.

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**Key Words**
lipid profile, apolipoproteins, oxidative stress, CVD complication

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## INTRODUCTION

One of the inflammatory skin conditions that is persistent and recurrent, psoriasis has been linked to aberrant plasma lipid metabolism and oxidative stress[1]. WBC infiltration is seen in the clinically active psoriasis lesions, and multiple investigations show elevated levels of WBC activation products in the peripheral blood of these patients[2,3]. According to certain theories, C-reactive protein, an acute phase reactant produced in the liver and elevated several times after acute inflammation, is a sign of systemic inflammation in a number of diseases, such as cancer, rheumatoid arthritis, psoriasis, and myocardial infarction[4].

Large amounts of reactive oxygen species are produced in cells, which can circumvent or outweigh the antioxidant defense mechanisms of cells and tissues. This results in a variety of major, interconnected impairments to cell metabolism, such as breaks in DNA strands, increases in intracellular free calcium, damage to membrane ion transporters, and other specific proteins that ultimately cause cell death. The polyunsaturated fatty acids in the membrane lipids are prime targets for assault by free radicals, leading to lipid peroxidation, which has been identified as a key factor in the development of numerous disorders.[5,6] Of the reactive aldehydes that result from lipid peroxidation, malonaldialdehyde, lipid peroxide, is the most prevalent. It has been proposed that these aldehyde's binding to membrane proteins may change the protein's stiffness, permeability, tonicity and integrity and thus, may cause the implicated effect.[7] Furthermore, the likelihood of developing CVD is significantly increased by the presence of inflammation.[8]

It is plausible that oxidative stress, dyslipidemia, and systemic inflammation are closely related in raising the frequency of CVD risk in psoriasis patients. Even though our understanding of psoriasis from a pathologic perspective has improved, the intricate mechanisms that make psoriasis patients more likely to develop CVD in the future are still poorly understood and involve systemic inflammation, oxidative stress, and dyslipidemia. Furthermore, to the best of our knowledge, systemic inflammation, oxidative stress, and dyslipidemia have not been studied together in psoriasis patients in the past[9-11].

Patients with psoriasis may be at risk for dyslipidemia, as past meta-analyses have shown that the condition is linked to increased levels of triglycerides, low-density lipoprotein cholesterol, and total cholesterol[12]. Lipoprotein structural protein components known as apolipoproteins are essential for the movement and metabolism of lipids[13]. Thirteen Apolipoprotein A1 and B, two of the six apolipoprotein kinds, have drawn a lot of interest when discussing psoriasis. On the relationship between psoriasis and apolipoprotein A1 and B levels, however, there was no agreement[14,15]. Interestingly, obesity is a risk factor for dyslipidemia and psoriasis on its own[16]. Therefore, obesity may mask the relationship between psoriasis and apolipoprotein A1 and B levels.

## MATERIALS AND METHODS

Age and gender corresponded As controls, 35 healthy, normal volunteers without any skin conditions were gathered (Group I). Only after receiving their informed consent and the college ethics committee's approval of the protocol were these participants recruited. Each subject completed a pre-experimental questionnaire with general information about themselves, their family history and a brief physical examination that included a body area assessment based on the rule of nine.

**Inclusion Criteria:** Participants in the trial were those who provided informed consent, had no prior history of cardiovascular illness, were not receiving any medical treatment (such as phototherapy or local steroid medication), and had been taking antioxidant supplements for at least a month before blood work.

**Exclusion Criteria:** Individuals who had a history of chest pain, deep fungal or disseminated localized gonococcal infection, diabetes mellitus, arthritis, hypertension, renal insufficiency, hepatic disease, acute illness (fever, joint pain, abdominal complaint), cancer, were taking any medications were excluded.

Venoscent punctures were used to obtain roughly 10 milliliters of blood from patients who had fasted overnight and placed in sterile EDTA vials. Blood was centrifuged at 1000 g for 15 min at room temperature to separate the plasma, which was then kept at -80°C until needed. Commercially available ELISA kits (R&D Systems, USA) were used to measure plasma CRP levels in accordance with the manufacturer's instructions. Total cholesterol, triglycerides, and HDL cholesterol were the three components of the plasma lipid profile that were analyzed enzymatically using a kit purchased from (Randox Laboratories Limited, Crumlin, UK). Using the immunoturbidity method (Randox kit), plasma apolipoproteins B and A1 were measured and the ratio of ApoB to A1 was determined. Following the preparation of hemolysate, the amounts of erythrocyte malondialdehyde (MDA), a thiobarbituric acid reactive compound, were tested. A trimethine-colored material was produced as a result of the heat-induced reaction between MDA and thiobarbituric acid (TBA) in the acid solution, and its spectrophotometric measurement was made at 532 nm[15].

**Statistical Analysis:** The study group individual's data were input individually into a Microsoft Excel 2007 sheet and the values were reported as Mean±SD. Student's t test was used to examine the significance of mean difference between research group subjects.

## RESULTS AND DISCUSSIONS

Table 1 shows the demographic indices of the study group subjects as observed in the current investigation, including their mean age and blood pressure. In comparison to healthy controls, psoriasis patients show negligible variance ($p<0.1$) in their age and blood pressure. Table 2 displays markers of the state of oxidative stress and systemic inflammation. In comparison to the healthy control group, the patient group's plasma CRP and erythrocyte MDA levels were found to be significantly higher ($p<0.001$, 41.02% and $p<0.05$, 29.3% high), indicating the importance of inflammation and oxidative stress in the disease process.

According to conjecture, oxidative stress and dyslipidemia are linked to psoriasis inflammation. This complex interplay is thought to be the mechanism behind the development of future CVD risk. In comparison to healthy controls, psoriasis patients in the current study had significantly higher plasma CRP levels, a perturbed lipid profile, and an elevated apolipoprotein B/A1 ratio. Our results are consistent with a number of earlier investigations that were carried out independently and with patient groups that were not very large[4,9,11]. They claim that increased inflammation causes psoriasis patients' atherogenic dyslipidemic profiles by influencing lipoprotein lipase activity through antilipoprotein lipase antibodies. Moreover, an oxidative inflammatory effect increases the production of atherogenic complexes of autoantibodies to oxidized LDL, which in turn raises the risk of CVD in psoriasis patients by promoting the accumulation of LDL in the endothelium wall[17].

Impaired lipid metabolism and cholesterol buildup may result from this condition. Notably, elevated cholesterol levels may worsen the inflammatory state of psoriasis, and dyslipidemia may also be a significant factor in the pathophysiology of the condition. In the meantime, it has been documented that apolipoprotein B increases inflammation by activating the pathways that trigger the production of TNF-α and IL-6 via p38 mitogen-activated protein kinase and NF-κB. Apolipoprotein B could be a significant independent immunomodulator that connects systemic and local inflammatory responses to lipid metabolism. Thus, aberrant lipid metabolism and psoriasis may be related in both directions. Regardless of the reciprocal association between dyslipidemia and psoriasis, screening for lipid metabolism may have good clinical consequences for psoriatic patients, given the advantages of early detection and therapy of comorbidities.

As far as we are aware, this research represents the most extensive meta-analysis examining the connection between psoriasis and serum levels of apolipoprotein A1 and B. According to this meta-analysis, which adhered to the PRISMA statement, psoriasis patients may be more susceptible to dyslipidemia. Our study was not without limits, though. Firstly, a case-control design is used in most of the included research. As a result, it is challenging to determine the causal or temporal links between serum levels of apolipoprotein A1 and B and psoriasis. Secondly, there was evidence of heterogeneity among the included studies, which might be attributed to a variety of factors, including the clinical features and quality of the included studies. Third, because there was insufficient data available, even though we did subgroup analysis of the studies based on the kind of psoriasis and matched BMI, we were unable to match other potential factors. Likewise, insufficient information hindered our capacity to examine gender variations in the correlation between psoriasis and serum levels of apolipoprotein A1 and B. Furthermore, of the examined studies, only eight reported PASI values, and of those, the mean PASI score indicated that half had moderate psoriasis. The impact of psoriasis severity on serum levels of apolipoprotein A1 and B was not examined in our study. More research is needed to elucidate our findings in order to overcome these constraints[17,18].

Uncontrolled generation of reactive oxygen species (ROS) disrupting the systemic oxidative balance is a major factor contributing to the increased complexity of psoriasis[10]. ROS are produced by vascular smooth cells and endothelial cells, which oxidize low density lipoprotein and cause atherosclerosis. Furthermore, ROS are implicated in the destruction of cell membranes through lipid peroxidation and the products that arise from it, including lipid hydroperoxides, lipid radicals ($L^{\circ}$), lipid peroxides ($LOO^{\circ}$) and highly reactive aldehydes. These products are vital in the onset and advancement of vascular complications in psoriasis[18]. In this particular context, group II subjects exhibited a significant increase in erythrocyte MDA levels (a marker of lipid peroxidation) ($p<0.005$) when compared to healthy controls. This finding sheds light on the etio-pathogenic

**Table 1: Demographic and clinical profile of study group subjects (Mean±SD).**

| Particulars | Group I (n=35) | Group II (n=35) |
|---|---|---|
| Age (years) | 39±8 | 43±6.7 |
| Male/Female | 20/15 | 23/12 |
| Height (meter) | 2.57±0.32 | 2.61±0.30 |
| Weight (Kg) | 62.3±3.7 | 59.4±2.9 |
| BMI ($Kg/m^2$) | 24.4±1.3 | 22.8±1.2 |
| Systolic blood pressure(mmHg) | 109.6±4.1 | 113.2±4.6 |
| Diastolic blood pressure(mmHg) | 77.3±3.33 | 78.4±3.41 |

p<0.1: Non-significant, p<0.05: Significant

**Table 2: Marker of systemic inflammation, oxidative stress and plasma lipid profile study group subjects (Mean±SD).**

| Particulars | Group I (n=35) | Group II (n=35) | Increase | Decrease |
|---|---|---|---|---|
| CRP (mg/L) | 4.30±0.18 | 5.61±0.18*** | 41.02% | - |
| Malondialdehyde (µmolMDA/ml) | 3.73±0.18 | 4.42±0.25** | 29.3% | - |
| Total Cholesterol (mg/dl) | 157.61±15.4 | 208.53±20.4** | 33.2%, | - |
| Triglycerides (mg/dl) | 106.80±13.32 | 134.9±19.2** | 27.2% | - |
| HDL cholesterol (mg/dl) | 46.4±4.41 | 38.83±4.20* | - | 18.2% |
| LDL cholesterol (mg/dl) | 95.4±13.51 | 124.22±14.7** | 31.3% | - |
| Apo-B (mg/dl) | 93.51±19.0 | 115.8±27.9 ** | 23.51% | - |
| Apo-A(mg/dl) | 143.31±29.4 | 98.81±20.11** | - | 32.21% |
| Apo-B/A1 | 0.69±0.19 | 2.21±0.47*** | 83.31% | - |

*p<0.1: Non-significant, **p<0.05: Significant, **p<0.001: Highly significant

role of ROS via lipid peroxidation, which predisposes psoriasis patients to future incidence of CVD and its related complications. Our results agreed with those of an earlier study on psoriasis patients who were at risk for cardiovascular disease. They claim that lipid peroxides are harmful to the components of cells and that they are also the cause of a complex cascade that initiates the formation of atherosclerotic plaque, prostacyclin synthesis, enhancement of cytosolic free calcium, and peripheral vascular resistance, all of which contribute to the development of CVD complications in patients with psoriasis who have abnormal lipid profiles[10].

## CONCLUSION

Based on current research, it is suggested that in addition to lipid profile, apolipoproteins and indicators of oxidative stress and inflammation should be routinely assessed in order to anticipate CVD complications. Furthermore, dietary guidance can lower cardiovascular risks and aid in the management of the underlying condition. Additionally, in addition to the prescribed medication for the treatment of psoriasis, lifestyle changes, frequent exercise, and a diet high in antioxidants should be included. This will help to easily achieve two crucial CVD prevention goals: maintaining a healthy lipid profile and regulating inflammation and oxidative stress.

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