A study on lipid profile and apolipoprotein levels in psoriatic patients

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INTRODUCTION

Psoriasis is a common, recurrent, genetically determined, inflammatory, and proliferative disease of the skin. The characteristic lesion is a chronic sharply demarcated, dull-red, scaly plaque, situated particularly on the extensor surfaces and scalp 1. Patients with psoriasis face increased mortality and morbidity from cardiovascular events, particularly in severe and longstanding disease 2. The link between cardiovascular disease (CVD) and psoriasis has been reported in several studies 3,4. One of the factors that may explain the increased susceptibility of patients with psoriasis to CVD is an abnormal lipid profile 5. Another factor is some of the pharmacological agents that are used in the treatment of psoriasis such as retinoids and cyclosporine, which may...
induce hyperlipidemia. Apolipoprotein B is the major structural protein in lipoproteins like low-density lipoprotein (LDL), intermediate-density lipoprotein (IDL), and very-low-density lipoprotein (VLDL). Each compound carries one molecule of apolipoprotein B. Therefore, the total value of apolipoprotein B represents the total level of atherogenic lipoproteins. Studies have shown that apolipoprotein B is more accurate than LDL, especially in patients with hypertriglyceridemia, very low LDL values, or non-fasting samples.

Furthermore, accumulating evidence suggests that apolipoprotein B is superior to LDL in predicting atherosclerotic CVD. Additionally, an elevated apolipoprotein B level is more strongly associated with coronary heart disease incidence than a similar elevation in LDL cholesterol level. Consequently, apolipoprotein B has been designated as a secondary target to monitor statin therapy in several guidelines.

This study aimed to approximate the lipid profile, apolipoprotein A-I, and apolipoprotein B levels in psoriatic patients in comparison with healthy subjects.

MATERIALS AND METHODS

This was a cross-sectional, case-control study conducted in Alsader Teaching Hospital in Basrah, Southern Iraq from 1st of May 2017 till 1st of May 2018. A total of 93 patients with plaque psoriasis were enrolled in this study. Psoriasis was diagnosed clinically by a consultant dermatologist and confirmed in borderline cases by histopathological examination of a skin biopsy taken by the same dermatologist. The control group (113 subjects) included 50 males and 63 females. Regarding the studied biochemical parameters, serum levels of TC, TG, VLDL-C, LDL-C, and apolipoprotein B were significantly higher in the psoriatic group as compared with the control group. Conversely, the serum level of HDL-C was significantly lower in the psoriasis group. On the other hand, there was no significant difference between patients and controls in terms of the apolipoprotein A-I level, as shown in Table 1.

Regarding the effect of the severity of psoriasis on the lipid profiles of the patients, it was found that the values of the investigated parameters,

RESULTS

A total of 93 patients (48 females) with plaque psoriasis were enrolled in this study. Their age ranged from 15-45 years. The control group (113 healthy subjects) included 50 males and 63 females.
Lipid profile in psoriasis

except for HDL-C, were significantly higher among patients with moderate and severe psoriasis relative to those with mild psoriasis (Table 2).

The effect of psoriasis severity on apolipoprotein A-I and apolipoprotein B levels is also shown in Table 2. There was a statistically significant higher level of apolipoprotein B among patients with moderate and severe psoriasis than those with mild psoriasis. However, there was no significant difference in the level of apolipoprotein A-I among the three groups.

**DISCUSSION**

This study aimed to approximate the lipid profile values and apolipoprotein A-I and apolipoprotein B levels in psoriatic patients in comparison with healthy subjects. The overall results of this study showed that the lipid profile was clearly disturbed among patients with psoriasis. Serum levels of TC, TG, VLDL-C, LDL-C, and apolipoprotein B were higher while HDL-C was lower among psoriatic patients in comparison with the control group. It is important to clarify whether these lipid abnormalities are secondary to psoriasis or whether the reverse is true, or if both are caused by a common genetic abnormality.

There are several possible explanations for the increased prevalence of CVD mortality and morbidity in patients with psoriasis. Firstly, patients with psoriasis could be considered as a group with increased atherosclerotic risk because of increased oxidant stress, decreased antioxidant capacity, abnormal lipid profiles, and increased lipoprotein content 8. At the cellular level, even patients with mild psoriasis display disequilibrium between markers of oxidative stress and antioxidants. In addition, psoriasis may further promote oxidative stress through an association with decreased folic acid levels 9. Secondly, recent evidence strongly suggests that chronic inflammation, a characteristic of psoriasis, plays a role in the initiation and progression of atherosclerosis 8. Furthermore, elevated levels of high-sensitivity C-reactive protein (hs-CRP), a non-specific marker of inflammation, is one of the emerging risk factors for CVD 10. Thirdly, abnormalities in plasma lipids and lipoproteins are likely to play an important role in the increased risk of psoriatic patients for developing atherosclerosis and CVD 5. Besides, there is evidence that established treatments of psoriasis such as retinoids and cyclosporine may induce hyperlipidemia that can promote the development of CVD 6,7. The lipid abnormalities seen in psoriasis might facilitate and maintain the

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**Table 1.** Biochemical parameters of the participants.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control group</th>
<th>Case group</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD (mg/dl)</td>
<td>Mean ± SD (mg/dl)</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>163.16 ± 19.74</td>
<td>196.44 ± 45.87</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>135.15 ± 22.2</td>
<td>160.5 ± 26.77</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>VLDL-C</td>
<td>26.99 ± 6.9</td>
<td>32.22 ± 5.41</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>HDL-C</td>
<td>46.04 ± 5.49</td>
<td>41.03 ± 5.89</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>LDL-C</td>
<td>90.14 ± 19.33</td>
<td>128.79 ± 45.23</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Apolipoprotein A-I</td>
<td>144.9 ± 11.22</td>
<td>143.57 ± 10.75</td>
<td>0.83</td>
</tr>
<tr>
<td>Apolipoprotein B</td>
<td>102.04 ± 21.85</td>
<td>129.51 ± 30.07</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, SD: standard deviation, VLDL-C: very-low-density lipoprotein cholesterol.

* Significant difference (P < 0.05)

**Table 2.** Relation between psoriasis severity and lipid profile parameters as well as apolipoprotein A-I and B levels.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild (Mean ± SD (mg/dl); n=26)</th>
<th>Moderate (Mean ± SD (mg/dl); n=37)</th>
<th>Severe (Mean ± SD (mg/dl); n=30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>162.44 ± 22.5</td>
<td>185.9 ± 34.7*</td>
<td>239.1 ± 41.1ab*</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>137.2 ± 25.1</td>
<td>163.4 ± 24.3*</td>
<td>177.6 ± 14.3ab*</td>
</tr>
<tr>
<td>VLDL-C</td>
<td>27.4 ± 5</td>
<td>33 ± 4.96*</td>
<td>35.9 ± 2.9 ab*</td>
</tr>
<tr>
<td>HDL-C</td>
<td>43 ± 5.43</td>
<td>42 ± 4.55</td>
<td>37 ± 6.6 ab*</td>
</tr>
<tr>
<td>LDL-C</td>
<td>94.6 ± 31.6</td>
<td>120.6 ± 32.6*</td>
<td>168.8 ± 38.9 ab*</td>
</tr>
<tr>
<td>Apolipoprotein A-I</td>
<td>140.7 ± 11.4</td>
<td>145.9 ± 11</td>
<td>143.8 ± 9</td>
</tr>
<tr>
<td>Apolipoprotein B</td>
<td>108.69 ± 29</td>
<td>131.8 ± 26 ab*</td>
<td>145.5 ± 24.4 ab*</td>
</tr>
</tbody>
</table>

HDL-C: high-density lipoprotein cholesterol, LDL-C: low-density lipoprotein cholesterol, SD: standard deviation, VLDL-C: very-low-density lipoprotein cholesterol.

* Significant difference (P < 0.05)

a As compared with the mild group.

b As compared with the moderate group.
supported the notion that this might be genetically determined rather than acquired 20.

From what has been mentioned, it is clearly seen that the relationship between psoriasis and dyslipidemia is a matter of controversy. Based on these controversial findings, no one can conclude which one of them causes the other or whether both of them are caused by a common genetic abnormality on a solid scientific base. However, we suggest that psoriasis possibly induces secondary hyperlipidemia, which places psoriatic patients at more risk of having CVD. However, we cannot rule out the other two possibilities. Therefore, a future advanced study is advisable to clarify this issue with certainty.

The power of this study lies in the measurement of lipoprotein levels in addition to the lipid profile among the studied participants. There are limitations in this study, which include the inability to ascertain the causation in the relationship between a disturbed lipid profile and psoriasis due to its design as well as the fact that it was conducted in a single center.

**CONCLUSION**

Obtaining a lipid profile is prudent among patients with psoriasis. This practice may help clinicians to detect dyslipidemia early and to consequently treat them with lipid-lowering agents that may primarily prevent CVD. Apolipoprotein B may be used as a marker to predict those patients with psoriasis who are at risk of dyslipidemia and probably CVD.

**Conflict of Interest:** None declared.

**REFERENCES**

Lipid profile in psoriasis


