SERUM LIPID PROFILE AND LEPTIN LEVELS IN ASTHMATIC LIBYAN CHILDREN

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Abstract

Aim:

To study lipid profile and leptin levels in non-obese Libyan children with asthma.

Materials and Methods:

70 Libyan children from Pediatrics Department, Faculty of Medicine, Benghazi University, Benghazi, Libya were included for the present study (from 2013 to 2014). Group I formed the normal control group (N: 30 Children) and Group II is 40 Libyan children with asthma. Their age group varied from 4 to 12 years old. Serum lipid profile and leptin were measured by routine standard methods.

Results and discussion:

The Libyan children with asthma showed an increase in serum total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol and triglycerides. Serum Leptin levels did not show marked changes between the two groups. The presence of hypercholesterolemia could be a triggering factor for generation of proinflammatory response in these children. Though leptin levels did not show marked response further studies may be necessary to study the role of leptin in obese asthmatic Libyan children.

Key words:

Asthma, lipid profile, leptin, proinflammatory response, Libyan children
Introduction:
Hypercholesterolemia is shown to be responsible for the development and precipitation of atherosclerosis and coronary artery disease (CAD).(1) But its association with active immunity and asthma have been studied with conflicting results.(2-4) Very few studies are available regarding lipid profile in asthmatic children.(5) The presence of childhood obesity and asthma in Benghazi in Libyan children prompted us to carry out this studies to evaluate lipid profile in normal and non-obese asthmatic children.

Materials and Methods:
70 Libyan children attending Pediatrics Department, Faculty of Medicine, Benghazi University, Benghazi, Libya were included for the present study (from 2013 to 2014). 30 Children in Group I (normal control) and 40 children group(asthma) II formed the study groups.
The inclusion criteria used are: children below the age group 12 years (4 to 12 years old), with no associated disorders including acute infection, free from other medications, excepting anti-asthmatics. Informed consent was taken from the cases taken for the study and approval for the study was obtained from Institute’s Ethics Review Board.

Body Mass index (BMI), serum total cholesterol, HDL cholesterol, triglycerides, VLDL cholesterol were measured using standard methods. LDL cholesterol was calculated using Friedwald formula.

Statistical analysis
Data were analyzed using statistical package (SPSS Version 17). The results were presented as (mean ± SD). Data comparisons were performed using two tailed unpaired Student t-test, and correlation coefficient was used for correlations. A value of p<0.05 was considered to be significant.

Results
Comparisons of normal children and non-obese asthmatic children regarding biochemical analyses

Serum Total cholesterol concentration (TC) (mg/dL)
The serum concentration of cholesterol level in the non-obese asthmatic children. was higher than normal children respectively (P<0.001; 125.89±28.084, 152.26±30.09, respectively) see Table.1 and Fig.1 and 2.
Asignificant positive correlation was found between serum TC concentration and LDL cholesterol (P<0.01) and triglyceride (TG) (P<0.05). No significant correlation between serum cholesterol and HDL, VLDL and Leptin was observed (P>0.05) (Table 2 and 3).

**HDL-Cholesterol (mg/dL)**

The HDL-Cholesterol for normal children was 39.09±9.09 and non-obese asthmatic children was 48.74±11.20 respectively. The HDL cholesterol was higher in non-obese asthmatic children compared to the control subjects (P<0.001) see Table (1) No significant correlation was observed between serum HDL-C and TC, VLDL, TG, and leptin. (Table 3 and 4) A significant positive correlation was found between serum HDL-C concentration and LDL (P<0.05).

**LDL-Cholesterol (mg/dL)**

Serum LDL-Cholesterol for normal children was 64.53±29.54 and non-obese asthmatic children 80.97±32.79, respectively. There was a significant increase in LDL-cholesterol in asthmatic children compared to normal controls (Table 1 and 2).

A significant positive correlation between serum LDL-C and serum cholesterol (P<0.01) was observed. There was no significant correlation between serum LDL-C HDL, VLDL, TG and leptin levels (Table 3).

**VLDL-Cholesterol (mg/dL)**

The serum VLDL-cholesterol for normal children was 20.88±6.61 and in non-obese asthmatic children 25.64±21.00 respectively. A significantly positive correlation between serum VLDL-cholesterol and serum Triglycerides was observed (P<0.01).

**Serum Triglyceride (mg/dL)**

The serum triglyceride in normal children was (109.91±35.94) and non-obese asthmatic children was (112.54±36.81) respectively. A significant positive correlation between serum TG and serum VLDL (P<0.01) was seen in normal children only and not in asthmatic children.
Table 1. Serum Lipid profile and Leptin in non-obese asthmatic Libyan children

<table>
<thead>
<tr>
<th></th>
<th>Normal Children (N:30)</th>
<th>Non-obese asthmatic children (N:40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Total Cholesterol (mg/dL)</td>
<td>125.89</td>
<td>28.08</td>
<td>152.26</td>
</tr>
<tr>
<td>LDL-cholesterol (mg/dL)</td>
<td>64.53</td>
<td>29.57</td>
<td>80.97</td>
</tr>
<tr>
<td>VLDL-cholesterol (mg/dL)</td>
<td>20.88</td>
<td>06.62</td>
<td>25.60</td>
</tr>
<tr>
<td>HDL-cholesterol (mg/dL)</td>
<td>39.10</td>
<td>09.10</td>
<td>48.70</td>
</tr>
<tr>
<td>Leptin (ng/ml)</td>
<td>02.44</td>
<td>01.63</td>
<td>02.62</td>
</tr>
</tbody>
</table>

Number of sample N=70; P Values shown only if statistically significant. < *P0.01 highly significant < **P0.001 very highly significant < ***P HDL (high-density lipoprotein) LDL (Low-Density Lipoprotein) VLDL (Very Low-Density Lipoprotein) TG (Triglyceride) HDL, LDL, VLDL, TG, (P>0.05)

Fig.1. SERUM LIPID PROFILE AND LEPTIN IN CONTROL
Table 2. Correlations between Serum lipid and Leptin in Normal children (CONTROL)

<table>
<thead>
<tr>
<th></th>
<th>leptin</th>
<th>TG</th>
<th>VLDL</th>
<th>LDL</th>
<th>HDL</th>
<th>CHOL</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHOL</td>
<td>0.75</td>
<td>0.20</td>
<td>0.06</td>
<td><strong>0.00</strong></td>
<td>0.35</td>
<td></td>
</tr>
<tr>
<td>HDL</td>
<td>0.98</td>
<td>0.64</td>
<td>0.29</td>
<td><strong>0.01</strong></td>
<td></td>
<td>0.35</td>
</tr>
<tr>
<td>LDL</td>
<td>0.93</td>
<td>0.91</td>
<td>0.33</td>
<td></td>
<td><strong>0.01</strong></td>
<td><strong>0.00</strong></td>
</tr>
<tr>
<td>VLDL</td>
<td>0.11</td>
<td><strong>0.00</strong></td>
<td>0.33</td>
<td>0.29</td>
<td>0.06</td>
<td></td>
</tr>
<tr>
<td>TG</td>
<td>0.06</td>
<td></td>
<td>0.91</td>
<td>0.64</td>
<td>0.20</td>
<td></td>
</tr>
<tr>
<td>leptin</td>
<td>0.06</td>
<td>0.11</td>
<td>0.93</td>
<td>0.98</td>
<td>0.75</td>
<td></td>
</tr>
</tbody>
</table>

Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).
Table 3. Correlations Between Serum lipid and Leptin in non-Obese asthmatic children

<table>
<thead>
<tr>
<th></th>
<th>CHOL</th>
<th>VLDL</th>
<th>TG</th>
<th>LDL</th>
<th>HDL</th>
<th>CHOL</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHOL</td>
<td>0.41</td>
<td>0.79</td>
<td>0.12</td>
<td>0.00**</td>
<td>0.34</td>
<td></td>
</tr>
<tr>
<td>HDL</td>
<td>0.95</td>
<td>0.90</td>
<td>0.68</td>
<td>0.19</td>
<td></td>
<td>0.34</td>
</tr>
<tr>
<td>LDL</td>
<td>0.33</td>
<td>0.99</td>
<td>0.70</td>
<td></td>
<td>0.19</td>
<td>0.00**</td>
</tr>
<tr>
<td>TG</td>
<td>0.92</td>
<td>0.10</td>
<td></td>
<td>0.70</td>
<td>0.67</td>
<td>0.12</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.32</td>
<td></td>
<td>0.10</td>
<td>0.99</td>
<td>0.90</td>
<td>0.79</td>
</tr>
<tr>
<td>leptin</td>
<td>0.32</td>
<td>0.92</td>
<td>0.33</td>
<td>0.95</td>
<td></td>
<td>0.41</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 4. Correlations Between Serum lipid, and Leptin in Normal children and non-Obese asthmatic children

<table>
<thead>
<tr>
<th></th>
<th>CHOL</th>
<th>LDL</th>
<th>VLDL</th>
<th>HDL</th>
<th>TG</th>
<th>leptin</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHOL</td>
<td>0.00**</td>
<td>0.25</td>
<td>0.09</td>
<td>0.04*</td>
<td>0.83</td>
<td></td>
</tr>
<tr>
<td>HDL</td>
<td>0.09</td>
<td>0.20</td>
<td>0.87</td>
<td></td>
<td>0.68</td>
<td>0.87</td>
</tr>
<tr>
<td>LDL</td>
<td>0.00**</td>
<td>0.58</td>
<td>0.20</td>
<td>0.64</td>
<td>0.50</td>
<td></td>
</tr>
<tr>
<td>VLDL</td>
<td>0.25</td>
<td>0.58</td>
<td>0.87</td>
<td></td>
<td>0.00**</td>
<td>0.63</td>
</tr>
<tr>
<td>TG</td>
<td>0.04*</td>
<td>0.64</td>
<td>0.003**</td>
<td>0.68</td>
<td></td>
<td>0.26</td>
</tr>
<tr>
<td>Leptin</td>
<td>0.83</td>
<td>0.50</td>
<td>0.63</td>
<td>0.87</td>
<td>0.26</td>
<td></td>
</tr>
</tbody>
</table>

Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
Discussion:

It was earlier reported that there was a strong correlation between serum leptin concentration, cholesterol and LDL. Similar observations were reported by Bedir et al in healthy men. (6-9). Previous studies regarding serum leptin in asthmatic patients have shown no significant correlation between serum leptin levels and asthma in children. Rather serum leptin levels show stronger correlation with BMI and therefore obesity. In the present study the children taken for the study were not obese or overweight. This was done with a view to find whether serum lipid profile and leptin levels play a role in precipitating or aggravating asthmatic condition in Libyan children. But the associations between serum leptin levels, serum lipid and obesity with asthma are not well understood. In some studies it was shown that serum cholesterol, triglyceride, LDL and VLDL levels decreased in asthmatic children compared to the control group, while HDL-C was higher than the healthy control group (10). In the present study the levels of serum total cholesterol, LDL cholesterol, triglycerides, VLDL-cholesterol and HDL cholesterol were increased in the non-obese asthmatic children. This finding is quite contrary to another study which had reported a decrease in total cholesterol, LDL cholesterol, triglycerides with an increase in HDL cholesterol in children with asthma (10). Therefore one has to take into various environmental factors along with biochemical parameters to understand what triggers proinflammatory response in asthma. One of the proinflammatory factor that has gained importance is cholesterol. A hypercholesterolemic state may induce and enhance proinflammatory mechanisms resulting in the release of higher levels of proinflammatory cytokines (11), cellular adhesion molecules (12) and inflammation sensitive plasma...
proteins. (13).

Fig. 1. Possible role of Serum Cholesterol in inflammatory response and expression of Asthma

There were scientific reports that suggested structural and functional homology between leptin and interlukin 6 (IL-6). Such structural homology may explain that leptin may influence cytokine production from T-lymphocytes eliciting greater TH1 response. (14).

Th2 cells are known to be responsible for allergic immune responses possibly through the preferential production of cytokines IL-4, (known to promote IgE production and inhibit Th1 response), IL-5 a growth and differentiation factor for Eosinophils, and IL-13 which is reported to be involved in pro-allergenic processes leading to re-modulation of the bronchial tissue that may occur in asthma.

JBMAS 2016;1:1-10
Therefore leptin may stimulate the release of proinflammatory cytokines IL-6, Interferon-L, and tumor necrosis factor (TNF-\(\alpha\)) from the adipose tissue and may promote Th1 responses (15) possibly in obese children.

On the other hand, Th1 cells produce interferon \(\gamma\) and IL-2, which inhibit Th2 lymphocytes in experimental models such as in vitro cell cultures(15,16) Such studies suggest that leptin may not be involved directly with allergic pathway like our present study though obesity is shown to be directly involved. Some other studies support the assumption that leptin may play a key role in allergic processes and therefore in asthma.(7,13,12)

References:


