Linear Growth in Infants and Children with Atopic Dermatitis
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Introduction
Skin barrier defects play central role in the pathogenesis of atopic dermatitis (AD) affecting local immunity and skin hydration. Severe AD is seen in 1-15% of cases and its effects on growth and nutrition are not know

OBJECTIVES
1) To measure the effect of AD on linear growth in 162 infants and children
2) to study the effect of hypoalbuminemia and hypo-proteinemia on the growth of these children

Methods
We studied linear growth and BMI all children with severe AD (<14 years) (n = 162) seen at Pediatric Allergy-Immunology clinics of Hamad General Hospital during June 2014-2015 with severe AD. SCORAD and anthropometric data were collected Serum total protein, albumin, 25OHD, and IgE concentrations were measured

Results
Children with severe AD had height SDS (HtSDS) = -0.75 +/- 0.8.
22/162 (13.60%) of children had HtSDS < -2, 57/162 (35%) had HtSDS < -1. 
BMI of the patients = 15 +/- 6.4. BMISDS was < -2 in 14% of patients. 16% of patients had hypoalbuminemia.
AD severity scores (SCORAD) was 61.3 ±22.3.
Twenty five patients with hypoalbuminemia had low BMI 11.2±2 % compared to 26 normo-albuminemic patients who had BMI 19.1±38.1%.
SCORAD was higher in hypo-albuminemic-low-BMI patients compared to normo-albuminemic-normal-BMI patients (67.9±22.1 vs 58.3±22.5).
Vitamin D deficiency was found in 58% of the patients. HtSDS and BMI did not correlate significantly with the severity of the disease (SCORAD).

Discussion
Children with severe AD had high prevalence of hypoalbuminemia due to loss of albumin through the diseased skin. Albumin loss may lead to malnutrition and low BMI in these patients. HtSDS of 35% of children was < -1. It is important to closely monitor growth, nutrition and biochemical makers in the management of severe AD.

SCORADS number %
mild 8/162 5%
moderate 35/162 22%
severe 118/162 73%

HtSDS in children with AD

<table>
<thead>
<tr>
<th>BMI Status</th>
<th>HtSDS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; -2</td>
<td>13-60%</td>
<td>HTSDS &lt; -2</td>
</tr>
<tr>
<td>&lt; -1</td>
<td>35%</td>
<td>HTSDS &lt; -1</td>
</tr>
</tbody>
</table>

Biochemical status

<table>
<thead>
<tr>
<th>Biochemical</th>
<th>BMI (%)</th>
<th>SCORAD (index)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-Albumin</td>
<td>11.2±2*</td>
<td>67.9±22.1*</td>
</tr>
<tr>
<td>Normal-Albumin</td>
<td>19.1±38.1</td>
<td>58.3±22.5</td>
</tr>
<tr>
<td>Low-Protein</td>
<td>11.2±1.2*</td>
<td>73±21.1*</td>
</tr>
<tr>
<td>Normal-Protein</td>
<td>22.5±11.8</td>
<td>59.9±20.5</td>
</tr>
</tbody>
</table>

Severe AD may lead to hypoproteinemia, low BMI and delayed linear growth. Hypoalbuminemia is associated with low BMI in 42% of patients. These findings confirm a harmful effect of severe AD on albumin loss and growth. It is important to closely monitor growth, nutrition and its biochemical makers (albumin, IGF-I) in the management of severe AD.