A rare presentation of hyperthyroidism: T3 thyrotoxicosis related to bilateral pitting edema of the leg

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Abstract
Thyrotoxicosis is a clinical state of inappropriately high levels of circulating thyroid hormones (T3 and/or T4) in the body from any cause. In the literature, bilateral pretibial pitting edema in hyperthyroidism cases has been reported in a few case reports. We report a case of T3 Thyrotoxicosis who had leg swelling for three weeks and whose finding regressed rapidly with treatment. Diffuse, bilateral pitting pretibial edema, which may occur due to many etiologies, should also be considered in the differential diagnosis of hyperthyroidism.

Keywords: Thyrotoxicosis; Hyperthyroidism; Pitting edema.

Introduction
Pitting edema occurs when excess fluid builds up in the body, causing swelling. When pressure is applied to the swollen area, a “pit”, or indentation, will remain. Pitting edema usually occur in legs, feet and ankles. The risk factors of pitting edema are sitting or standing in one position for too long, low protein levels, obesity and pregnancy. While systemic edema can be seen in cases of hypothyroidism and Cushing’s syndrome, it is not common in cases of hyperthyroidism [3,5]. However, if the patient develops cardiac failure or pulmonary hypertension, peripheral edema may occur [6]. Pitting leg edema not accompanied by cardiac failure has been reported in only a few case reports, and is a rare manifestation of hyperthyroidism [7,11,13]. Here, we presented a 35 year old female patient who was presented to our medical center with the complaining swelling in both legs ongoing for three weeks and was then diagnosed with T3 Thyrotoxicosis.

Case presentation
A 35 year old female with no chronic disease presented with swelling of the legs for the last three weeks. The patient did not complain of exertional dyspnea. She did not lose her body weight. On physical examination, the temperature was 36.5 C, pulse was 126 beats/min, and blood pressure was 120/60 mmHg. On examination, heart beat was regular without murmur. Breathing sound was clear without rale and crackle. The liver and spleen were not palpable. Diffuse pitting edema was observed on the legs in pretibial areas, with no accompanying color change, induration, or tenderness (Figure 1).

In the biochemical examinations, we could find elevated Free T3 level and suppressed TSH. But FT4 was normal. On liver function test, there was slightly elevated SGOT and SGPT level. Urine test is normal. On Electrocardiogram, there was sinus tachycardia. Echocardiography revealed left ventricular ejection fraction as 65% and normal echocardiogram. We diagnosed T3 thyrotoxicosis related pitting edema and elevated liver enzyme. Carbimazole 15 mg three times a day and propranolol 40 mg two times a day treatment was started. The swelling in the leg began to improve within 1 week and completely regressed about six weeks after the onset of treatment and did not recur. After six weeks of treatment, the free T3 levels normalized and heart rate became normalized to 76 beats/min.

Figure 1: On thyroid ultrasound scan showed mild coarse texture on both lobes and size was normal.

Table 1: Patient report.

<table>
<thead>
<tr>
<th>Lab test</th>
<th>On diagnosis</th>
<th>After 6 weeks' treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin</td>
<td>3.83 g/dl (3.8-5.1)</td>
<td>3.56 g/dl</td>
</tr>
<tr>
<td>Total protein</td>
<td>5.98 g/dl (6.0-8.2)</td>
<td>6.16 g/dl</td>
</tr>
<tr>
<td>GOT</td>
<td>98 U/L (0-37)</td>
<td>15 U/L</td>
</tr>
<tr>
<td>GPT</td>
<td>131 U/L (0-42)</td>
<td>15 U/L</td>
</tr>
<tr>
<td>FT3</td>
<td>3.58 pmol/L (3.10-6.80)</td>
<td>4.13 pmol/L</td>
</tr>
<tr>
<td>FT4</td>
<td>116.06 nmol/L (57.9-150.6)</td>
<td>73.29 nmol/L</td>
</tr>
<tr>
<td>TSH</td>
<td>0.20 uIU/mL (0.4-4.2)</td>
<td>0.15 uIU/mL</td>
</tr>
</tbody>
</table>

Discussion

This case presentation demonstrated a rare presentation of hyperthyroidism, in a patient with swelling in both legs. In this case, where cardiac, renal, hepatic etiologies were excluded and hypoproteinemia was not detected. Edema began to resolve within one week of starting the appropriate treatment.

In hyperthyroidism cases, peripheral edema may develop due to congestive heart failure, pulmonary hypertension and right heart failure. While left heart failure may develop in the early stages of hyperthyroidism, congestive heart failure and pulmonary hypertension may develop in the later stages [6]. But, no evidence of left heart failure was found in the current case.

A few cases of hyperthyroidism with bilateral pitting edema have been previously reported in the literature. In a patient with bilateral leg edema and diagnosed as thyrotoxicosis due to silent thyroiditis, the symptoms regressed with angiotensin receptor blocker therapy [9]. Volke et al. reported that a patient with unilateral leg edema had been previously diagnosed with Graves’ disease [10]. In another case, bilateral leg edema was reported in a pharmacologically-induced case of hyperthyroidism [11].

A study by Kukolnikova et al. [12] showed that hyperthyroidism is one of the most common etiologies in secondary lymphedema patients. This study shows that edema in hyperthyroidism patients may be caused by impaired peripheral lymph drainage. In addition, increased thyroid hormone levels due to thyrotoxicosis activate various vascular mechanisms, which appear to have a role in the pathophysiology, however, the exact pathophysiological mechanism of this manifestation remains unclear.

Conclusion

Bilateral pitting pretibial edema is not expected in the early stages of hyperthyroidism unless there is a cardiac or pulmonary cause. We reported a rare case of T3 thyrotoxicosis presented with pitting edema. Although pitting pretibial edema is rare, it might be the first symptom in patients with hyperthyroidism. Physicians should keep hyperthyroidism in mind in the differential diagnosis of peripheral edema.

References


