A Case of Recurrent Silent Thyroiditis

Tekrarlayan Sessiz Tiroidit Olgusu

Suleyman Ipekci, Mehtap Cakir
Selcuk University, Meram School of Medicine, Endocrinology and Metabolism, Konya, Turkey

Abstract
Silent thyroiditis constitutes approximately 1% all of thyroiditis. Rarely, silent thyroiditis can present with recurrent attacks. Here, we present the case of a patient who had four episodes of painless (silent) thyroiditis. Turk Jem 2011; 15: 47-9

Key words: Thyroiditis, silent thyroiditis, recurrent silent thyroiditis

Özet

Anahtar kelimeler: Tiroidit, sessiz tiroidit, tekrarlayan sessiz tiroidit

Introduction
The term thyroiditis encompasses several variants including Hashimoto's, subacute, postpartum, drug-induced, suppurative, Riedel’s and silent thyroiditis (1). Silent thyroiditis constituting approximately 1% all of thyroiditis cases (1) is distinguished from postpartum thyroiditis by the absence of postpartum period (2), and from subacute thyroiditis by the absence of a viral prodromal period and pain or tenderness in physical examination (3). It is also differentiated from Hashimoto’s thyroiditis by both mild elevation of thyroid autoantibodies (1,4) as well as less extensive fibrosis in pathology specimens and lack of germinal centres (5). Silent thyroiditis is characterized by a temporary thyrotoxicosis. While 10-20% of patients develop permanent hypothyroidism, 80-90% of patients fully recover within three months (1,3). Rarely, silent thyroiditis can present with recurrent attacks (3,6-9). Here, we present the case of a patient who had four episodes of painless (silent) thyroiditis.

Case Report
A 26-year-old medical doctor presented to the endocrinology outpatient clinic for palpitation, anxiety and sweating. She had these complaints for ten days but in her medical history, she reported that in the last seven years she was diagnosed with thyroiditis for three times based on similar complaints. Neither in her previous attacks nor this time, there was a history of pregnancy, symptoms suggestive of upper respiratory infection, drug use or anterior neck pain. Patient’s past medical history revealed that she was a non-smoker and had no known chronic disease. Family history showed that her father was on L-thyroxine treatment for hypothyroidism due to Hashimoto’s thyroiditis. On physical examination, her blood pressure was 110/70 mmHg, pulse 92 beats/minute and temperature was normal. Thyroid was nonpalpable and there was no pain in the thyroid region upon palpation. Her skin was mildly sweaty and fine tremor was noted in both hands. There were no specific signs of Graves’ disease like ophthalmopathy and dermopathy. On laboratory examination, free thyroid hormone levels were high and TSH was suppressed (free T3 (fT3): 5.16 (2-4.4) pg/ml; free T4 (fT4): 2.14 (0.93-1.7) ng/dl; TSH: 0.045 (0.27-4.2) µIU/ml). Anti-thyroperoxidase (anti-TPO) antibody was mildly positive [112 (<34) IU/ml], anti-thyroglobulin (anti-Tg) antibody was negative [30.6 (<115) IU/ml] and thyroglobulin level was normal [24.03 (1.4-78) ng/ml]. A complete blood count, erythrocyte
sedimentation rate (ESR) and C-reactive protein (CRP) levels were normal. Table I shows the laboratory data during the patient's previous attacks. Although she had three previous attacks, the last two attacks and the current laboratory values are presented, because some results have been lost on follow-up. A thyroid ultrasonography has revealed a heterogeneous parenchyma with several hypoechoic regions with ill-defined margins. On Tc99m pertechnetate thyroid scan, the tracer uptake was prominently low (Figure 1). Fine-needle aspiration biopsy showed abundance of lymphocytes and occasional oncocytic cells. The patient was just given beta-blocker therapy for symptomatic relief. Her thyroid hormone levels were compatible with subclinical hyperthyroidism at the end of two months and normal at the end of four months. As in her previous episodes, she did not become hypothyroid during follow-up.

**Discussion**

Silent thyroiditis occasionally can recur and usually requires only symptomatic treatment. In a patient series by Nishimaki and colleagues, 16 of 48 patients with silent thyroiditis had recurrent attacks (8). In that study, high fT4 levels, early age of onset, male gender and positive autoantibodies were defined as predisposing factors for recurrent episodes of silent thyroiditis. From this point of view, early age of onset, higher peak fT4 and antibody positivity were the predisposing factors in our case.

In a case series by Mittra and colleagues, four recurrent cases of silent thyroiditis were presented who had developed nine, four, three and two episodes (3). The first three cases were treated with radioiodine therapy during the recovery phase of an episode. There was a period of 27 years between the first and last episode of the first case. The second patient had developed hypothyroidism after two thyrotoxic episodes. All cases presented with elevated total T3 and/or free T3, free T4, suppressed TSH levels and low uptake of radioiodine on thyroid scan. Anti-Tg was positive in all cases and anti-TPO was positive in three of the cases. The lacks of a viral prodrome and neck pain or tenderness were considered to be enough for differentiating from subacute thyroiditis by the authors. The first three patients became hypothyroid after radioiodine therapy and thyroxine replacement therapy was given. The symptom-free period between episodes was 1-4 years. In another case report by Cho and colleagues, a case with four episodes was described (7). This case who had highly elevated anti-TPO levels (>4000 IU/ml), became hypothyroid after radioiodine treatment. Another very interesting case experienced severe transient thyrotoxicosis every two years (9). During each episode, the patient had to take bed rest for about 1 month. He was treated on two occasions with radioiodine therapy for recurrence of thyrotoxicosis two years apart.

In the differential diagnosis of recurrent thyrotoxicosis apart from silent thyroiditis, Hashimoto’s thyroiditis, subacute thyroiditis, postpartum thyroiditis (1,4), and factitious thyrotoxicosis should be included. Due to mildly positive anti-TPO antibodies and low uptake on thyroid scan during the thyrotoxic phase and absence of development of hypothyroidism after the attacks, Hashimoto’s thyroiditis was not considered in our case. Increased tracer uptake is expected in the thyrotoxic phase of Hashimoto’s thyroiditis, but rarely, low tracer uptake may also be noted (1). In the differential diagnosis of this patient, absence of fever and normal serum inflammatory markers ruled out subacute and suppurative thyroiditis. In a case report by Choe and colleagues, a thyroiditis case initially considered as subacute thyroiditis was finally diagnosed as silent thyroiditis at the third attack and the patient was treated with radioactive 131I ablation after the fourth attack (7). Fine-needle aspiration biopsy in such a situation, as well as in our case, could be helpful for definitive diagnosis.

Recurrence of thyrotoxicosis and the profession of the patient brings to mind factitious thyrotoxicosis which will also cause low uptake on thyroid scan, but in these cases, thyroglobulin levels will either be low or undetectable (10). However, thyroglobulin autoantibodies make this measurement less reliable when the patient has an autoimmune thyroid disease.

Postpartum thyroiditis is not different from silent thyroiditis clinically, except that it occurs after pregnancy. In the course of the postpartum thyroiditis, first a period of hyperthyroidism, then hypothyroidism and finally a euthyroid state are observed, like in silent thyroiditis. However, some patients may develop permanent hypothyroidism.

**Table 1. Laboratory results of the patient during attacks. Abnormal values are marked in bold**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Previous episodes</th>
<th>Current episode</th>
</tr>
</thead>
<tbody>
<tr>
<td>fT3 (2-4.4 pg/ml)</td>
<td>6.28</td>
<td>4.92</td>
</tr>
<tr>
<td>fT4 (0.93-1.7 ng/dl)</td>
<td>1.89</td>
<td>1.51</td>
</tr>
<tr>
<td>TSH (0.27-4.2 μIU/ml)</td>
<td>0.01</td>
<td>0.007</td>
</tr>
<tr>
<td>Anti-TPO &lt; 34 IU/ml</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Anti-Tg (&lt; 115 IU/ml)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Thyroglobulin (1.4-78 ng/ml)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Erythrocyte Sedimentation Rate</td>
<td>-</td>
<td>9</td>
</tr>
<tr>
<td>CRP (0-10 mg/l)</td>
<td>-</td>
<td>10.4</td>
</tr>
<tr>
<td>Leukocyte count (4000-10000 /μl)</td>
<td>-</td>
<td>6400</td>
</tr>
</tbody>
</table>
Conclusions

As a conclusion, absence of delivery in the last six months, presence of moderately high anti-TPO, normal serum thyroglobulin levels and recurrent episodes were diagnostic of recurrent silent thyroiditis in our case. Our diagnosis was also supported by the cytological findings. In the differential diagnosis of recurrent thyrotoxicosis, silent thyroiditis should be included and supported by thyroid fine-needle aspiration biopsy, when necessary.

References