Coexistent presentation of Graves’ disease and a Riedel’s thyroiditis - Diagnostic dilemma

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Abstract

Background: The coexistence of Riedel's thyroiditis and Graves' disease is quite rare, presents a diagnostic dilemma and requires a particular clinical approach. 

Case report: We describe a 69-year-old man who had two autoimmune processes - Riedel's thyroiditis and Graves' disease, which develop at the same time in different lobes of the thyroid with contradictory manifestations. The diagnosis was confirmed by histological examination. 

Conclusions: Present case is unique by development and management, especially, it should be noted that iodine deficient thyroid diseases are endemic to the Caucasus region (Georgia), although the present case is the only one in our observation period (2016-2019). (TCM-GMJ April 2020; 5(1):P27-P30)

Keywords: Riedel's thyroiditis; Graves' disease; Autoimmune thyroiditis

Introduction

Riedel's thyroiditis (RT) is a rare, chronic inflammatory disease of the thyroid gland characterized by a dense fibrosis that replaces thyroid parenchyma (1-3). The fibrotic process invades adjacent structures of the neck and extends beyond the thyroid capsule. This feature differentiates RT from other inflammatory or fibrotic disorders of the thyroid. Extension beyond the thyroid also differentiates this from the fibrosing variant of Hashimoto thyroiditis (HT) (3-5).

In contrast, unit cases of coexistence of Riedel's thyroiditis and Graves' disease are described (6, 7). It should be noted, that the case described relates to a patient who, after surgical treatment of hyperthyroidism, developed Riedel's thyroiditis in the remaining tissue of the thyroid gland. (6). According to Bryan McIver et al. case report (2010), Graves' disease developed of the background of unilateral Riedel's thyroiditis (7). All named authors have recommended surgical treatment of Riedel's thyroiditis, which the differential diagnosis from anaplastic carcinoma was not possible, based on extensive radiological findings (6-8).

We present a case of thyroid pathology, where simultaneously developed Riedel's thyroiditis in right lobe and Graves disease in the left lobe - two different clinical-morphological forms of autoimmune thyroiditis that have not been compared in the literature as coexisting process.

Case presentation

Patient: Male, 69 years old.

Chief complaint: Increase in thyroid gland size, compression on the trachea, and shortness of breath 2 months before hospitalization.

Present medical condition: A 69-year-old man referred to the clinic for a dense formation in the anterior neck, anxiety, difficulty breathing, swallowing and discomfort in the neck area, which had been observed for about two months. Previously, the patient had not undergone any examination or treatment.

Family medical history: No medical problems.

Physical examination: The patient was medically stable with a blood pressure of 140/90 mm Hg, pulse of 92 beats per minute, respiration rate of 18 breaths per minute, and a temperature of 36.3ºC. Hard globe protrusion in the anterior neck region and middle exophthalmia were observed; scleral icterus and conjunctival anemia not present. On auscultation: systolic murmur, breath sounds in norm.

Electrocardiography: 92 beats per minute at rest, normal sinus rhythm.

Laboratory results: Complete blood and urine test are done in table (tab.1). This condition was interpreted by consilium as subclinical hyperthyroidism. 

Thyroid ultrasound: In the thyroid left lobe were observed a well-defined irregular and hypoechoic nodules measuring at 1- 6X3X0.5 cm in size, 2 - 6X4X0.7 cm in...
size, 3 - 4,5X3X5X1,5 cm in diameter, along with large scattered dense calcifications. Right lobe – dense mass, homogeneously hypoechoic with the poor demarcation of the gland borders. Enlarged cervical lymph nodes and invasion into the parathyroid glands were not present.

Thyroid fine needle aspiration (FNA) cytology: Cytopathological results (Bethesda) have shown glandular and stromal cells and few liquid substances.

Computed tomography (CT) scan of the neck: In the thyroid left lobe were: 1 - 6X3,5X0,5 cm in size, 2 - 6X4X0.7 cm in size, 3 - 4,5X3X5X1,5 cm nodular lesion occupying most of the left lobe of the thyroid. A peripheral calcified lesion was also identified. Right lobe demonstrates compression of local structures by an enlarged thyroid with low attenuation change within areas of the involved thyroid gland.

Surgery: Total thyroidectomy was taken. Complex mass with hard consistency and a thick capsule that compressed and deformed the trachea were seen.

Macroscopy: The test material contains two lobes of the thyroid gland: Left lobe is divided into 3 parts: 1 - 6X3,5X0,5 cm in size, 2 - 6X4X0.7 cm in size, 3 - 4,5X3X5X1,5 cm in size.

Adjacent to the left lobe of the gland - 3,5X1,7X0,5 cm.

Right lobe - 5X4X1cm in size, separately presented as well 1 - 4X1,5X0.6 cm in size, 2 - 3,5X0,7X0,7 cm in size.

H&E microscopy: Left lobe - micro- macrofollicular goiter with follicular cells hyperplastic buds and tall epithelial cells. Cystic degeneration and necrotic areas also are evident. Chronic lymphocytic thyroiditis with large fused germinial centers was seen (Fig. 1. A, B).

Adjacent to the left lobe of the thyroid, composed by fibrotic cords with hemorrhage foci.

Right lobe – the multifocal fibrosclerosis with thyroid architectural distortion due to the marked proliferation of fibrous tissue, severe atrophy of the follicles and evident infiltration by mononuclear inflammatory cells confirm the diagnosis of Riedel's thyroiditis (Fig. 1. C, D).

Using electronmicroscopic investigation method (osmium tetraoxide fixation, epon-araldyte embedding, Tesla BS500 microscope) the following results were obtained: electron micrographs of thyroid gland samples from left lobe were shown multilayered follicles with large euchromatic nuclei, heterogenous colloid in lumen, underlying basal lamina and blood capillary with erythroblastosis (Fig. 2. A, B, C, D).

Outcome: Not since of complication after surgery and during postsurgical replacement therapy with levothyroxine.

Discussion

It is known, that Riedel's thyroiditis is a thyroid gland rare, chronic inflammatory disease (1-3), but etiology not well known. Recent concept defines RT as autoimmune disease with systemic extrathyroidal fibrosis mainly associated with IgG4-related systemic disease (IgG4-RSD) (9-12). However, IgG4-RSD may unify two well known pathogenesis postulate: One theory of pathogenesis postulates that RT results from an autoimmune process. A second theory holds RT to be a primary fibrotic disorder (13).

The presented study demonstrate unusual case, because two different functionally opposite autoimmune diseases progress within one organ, into right and left thyroid lobe. Based on laboratory and FNA data, we cannot determine which are the lieder, primary developed - Riedel's thyroditis or Graves' disease, as the characteristic changes of both processes are pronounced.

Histologic and ultrastructural images of a surgically removed thyroid gland confirm in left lobe the process characteristic of Graves’ disease: follicular cell hyperplasia, extensive lymphoplasmocytic infiltration with the development of fuse active germinal centers, and secondary degenerative changes. In the right side there was hard avascular tissue with atrophy of the glandular parenchyma so-called stone-like, dense fibrosis, which confirm characteristic of Riedel's thyroiditis.

But, elevated TSH level, abnormal cardiac signs and middle exophthalmia contribute to trend of parenchyma hyperplasia and subclinical hyperthyroidism.

Due to the low incidence of Riedel's thyroiditis, there are no guidelines or large clinical studies that refer to the optimal management of the condition as extensive comprehensive reviews present (6-8, 14), moreover, with Graves’ disease combination. This creates a diagnostic difficulty as laboratory data show subclinical hyperthyroidism, on one hand, and high levels of thyroid peroxidase (TPO) and thyrotropin receptor antibodies (TRAb), on the other hand, which is strongly conflicting.

Marine-Lenhart-Syndrom was excluded, because our patient has two types of autoimmune thyroditis by different pathophyslogic mechanisms, moreover, RT and Graves’s disease are postulated from CT and FNA results. We can share with Wu, Leung et al. (15) that mechanisms underlying Marine-Lenhart-Syndrom include somatic constitutively active mutations of the TSH receptor, but the absence of TSH receptor autoantibodies and over expression of Thyrotropin receptor antibodies (TRAb), Thyroid peroxidase (TPO) antibodies and Thyroglobulin antibody confirm that reported case is unusual and baseline symptoms of hyperthyroidism coexist with other type of autoimmunity, such as RT.

Overall, we believe that the case to be of special interest as a diagnostic dilemma, which may have a similar pattern of Riedel's thyroiditis and Graves’ disease. It is emphasized that iodine deficient thyroid diseases are endemic to the Caucasus region (Georgia), although the present case is the only one in the period from 2016 to 2019 (our observation period).

Conclusion

The reported case is to focused on two different histopathologically and pathophysiologically opposite autoimmune processes that present clinical risk and required different management.
Acknowledgement

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Conflict of interest disclosure

Authors declare no potential conflicting interests related to this paper.

References


Table 1. Blood and urine test

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<th>Test</th>
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<tr>
<td>Blood Urea Nitrogen (BUN)/Creatinine</td>
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<td>7-20/ 0.6-1.2 mg/dL</td>
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<td>Total protein/albumin</td>
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<td>6.8-7/ 3.5-5.5 g/dL</td>
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<td>AST/ALT</td>
<td>21/10 IU/L</td>
<td>8-20/5-40 IU/L</td>
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<td>Na</td>
<td>140 mEq/L</td>
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<tr>
<td>K</td>
<td>5.0 mEq/L</td>
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<tr>
<td>Cl</td>
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<td>96-106 mEq/L</td>
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<tr>
<td>Ca</td>
<td>4.8 mEq/L</td>
<td>4.5-5.2 mEq/L</td>
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<td>Erythrocyte sedimentation rate (ESR)</td>
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<td>0-22 mm/hr</td>
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<td>C-reactive protein</td>
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<td>TSH</td>
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<tr>
<td>Thyroglobulin antibody</td>
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Figure 1. H&E. A. Graves disease, proliferative thyroid epithelial cells and cystic degeneration (1), X100; B. Graves disease, large fused lymphoid nodule with germinal center (arrow), X200. C. Riedel thyroiditis, fibrous tissue replaced thyroid follicles, follicles atrophy and inflammatory mononuclear cells infiltration, X200; D. Riedel thyroiditis, extensive fibrosis replaced thyroid parenchyma, X200.

Figure 2. Electron micrographs. A. B. Graves disease, X2000. Multilayered follicles with large euchromatic nuclei (1), underling basal lamina and blood capillary with erythrotrastis, heterogeneous colloid in lumen (2). C. Riedel’s thyroiditis, fibrosclerosis area with active fibroblast (arrows) and dense collagen fibers replaced follicles, X4000; D. Riedel’s thyroiditis, extensive fibrosis and atrophy of follicles, pyknosis of nuclei (arrows), X2000.