Invasive fibrous thyroiditis (Riedel’s disease) with signs of giant cell arteritis

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Abstract

OBJECTIVES: The authors described a case of Riedel’s thyroiditis, a relatively rare disease affecting the thyroid gland.
RESULTS: A 38-year-old female was diagnosed with Riedel’s thyroiditis by a frozen section examination during attempted surgery for the suspicion of malignancy. The histopathologic examination confirmed the diagnosis of Riedel’s thyroiditis combined with signs of giant cell arteritis. After hemithyroidectomy, the patient had paresis of the recurrent laryngeal nerve for five days and signs of hypothyroidism and hypoparathyroidism for three months after surgery. The level of thyroid peroxidase antibodies titer was higher. The patient had no compression symptoms, no signs of systemic immunopathology, and no signs of extracervical fibrosis. She was put on prednison 5 mg daily.
CONCLUSIONS: Riedel’s thyroiditis can be looked upon as a cervical fibrosis of unknown etiology affecting the thyroid. In our case it was combined with signs of giant cell arteritis.
Introduction

Invasive fibrous thyroiditis is a rare chronic inflammatory disease of the thyroid gland. It is characterized by a fibrosis destroying the gland and extending into surrounding structures. The disease has some clinical and histologic similarity to retroperitoneal, mediastinal and retroorbital fibrosis as well to sclerosing cholangitis. It can be looked upon as a cervical fibrosclerotic process of unknown etiology that involves the thyroid.

Case report

A 38-year-old female was diagnosed elsewhere with thyroiditis and hypothyroidism requiring replacement therapy with thyroid hormones. After she had been put on steroids for one month, she improved slightly, however 6 months later, the patient was referred to our department for thyroid surgery. She complained of pain at the anterior part of the neck, she had slight cervical discomfort, but no problems with breathing. The goiter was stone hard, relatively small. Both vocal cords moved normally. At ultrasonography the right lobe was 20 ml, the left lobe was 18 ml, and no cervical lymph nodes were seen. The patient refused aspiration biopsy. A CT scan showed a diffuse goiter with hypodensities after contrast, and no cervical lymph nodes were seen. The thyroid functions. The results showed the following: levels of total calcium 1.85 mmol/l (normal 2.24 to 2.64), ionized calcium 1.04 mmol/l (normal 1.13 to 1.32), phosphorus 1.62 mmol/l (normal 0.65 to 1.61), free triiodothyronine 2.09 pmol/l (normal 2.5 to 5.6), free thyroxin 12.23 pmol/l (normal 10.0 to 25.0), and thryotropin 15.28 mIU/l (normal 0.3 to 4.0). Antithyroglobulin antibodies were negative and thyroid peroxidase antibodies titer was higher (260 kU/l). Level of parathormon intact was 1.94 pmol/l (normal 1.3 to 7.6). This examination confirmed hypothyroidism, hypocalcemia and the presence of thyroid antibodies. A detailed immunological examination showed only a higher level of immunoglobulin G 17.10 g/l (normal 9.00 to 16.0). The examination of blood cells showed slight changes, leukocytosis 11.8 x 10^9/l (normal 4.0 to 10.0), lymphopenia 0.203 (normal 0.250 to 0.330) and neutrophilia 0.709 (normal 0.570 to 0.680). FW was normal and C reactive protein was normal as well. The examination did not reveal any signs of systemic immunopathology. Extracervical fibrosis was excluded by CT of thorax and abdomen. Three months after surgery the patient felt better, however she still had slight cervical discomfort and intermittent hoarseness. She had resumed all her activities but she felt tired. The daily medication was prednisolone (Prednison) 5 mg, levothyroxinum (Letrox) 150 microgram and dihydrotachysterolum 0.1% (Tachystin) 20 drops.
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Fig. 1. Dense hyalinized collagen tissue with mild chronic inflammatory infiltrate spreading among atrophic muscles.

Fig. 2. Granulomatous arteritis with giant cells along the internal elastic membrane, obliteration by intimal fibrosis, hyalinized tissue around the artery.
Discussion

It is nearly impossible to make a diagnosis of Riedel’s thyroiditis based on the results of needle biopsy, because it contains only fibrous tissue [1]. A thyroidectomy is recommended because of the suspicion of other diagnoses, above all suspicion of malignancy, as it was in our case. The diagnosis of Riedel’s thyroiditis is made during surgery by the frozen section examination [1] and with a definitive histopathological finding.

Riedel’s thyroiditis can coexist with Hashimoto’s thyroiditis [2–4], though it is not considered the fibrosing variant of Hashimoto’s thyroiditis [5]. We proved peroxidase antithyroid antibodies in our patient, but it has proven positive in 19% of miscellaneous nonautoimmune thyroid diseases [6]. In our case the histological finding revealed changes consistent with diagnosis of Riedel’s thyroiditis, however the presence of giant cell arteritis was an unusual finding hitherto not described in cases of typical Riedel’s thyroiditis. Since the cause of Riedel’s thyroiditis is unclear we suggest the possible role of this arteritis in the pathogenesis of the thyroid lesion in our patient.

No consensus on standard therapy exists. The surgical treatment of Riedel’s thyroiditis is recommended only to relieve pressure symptoms, which comprises mostly wedge resection of the isthmus and mobilization of the lateral lobes [1]. We did not complete the total thyroidectomy after the frozen section showed no malignancy, and the diagnosis of Riedel’s thyroiditis was highly probable. The surgical problem we encountered in our case was not the recurrent laryngeal nerve but parathyroid glands. Even after hemithyroidectomy the calcium level dropped significantly after surgery and there was a need for therapy with dihydrotachysterol. During three months of follow-up, the calcium level did not come to normal level. Hypoparathyroidism caused by Riedel’s thyroiditis was reported by other authors [7]. The therapy of Riedel’s thyroiditis can be conservative. Good results were reported with steroids [8, 9] and very good results were reported with Tamoxifen [10]. We started our patient’s therapy with small doses of steroids, because she had no compression symptoms, no signs of systemic immunopathology, and no signs of extracervical fibrosis.

REFERENCES