COULD RIEDEL'S THYROIDITIS BE SUBACUTE THYROIDITIS? A CASE REPORT

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Our purpose in this paper is to make a case for the need to question whether Riedel's thyroiditis is in fact a rare variety of subacute thyroiditis. Total thyroidectomy was performed on a patient with a huge thyroid mass. The histopathological diagnosis was Riedel's thyroiditis with classical lymphoplasmacytic infiltration destroying thyroid tissue and invading perithyroidal soft tissue. However, in addition, histopathology showed epithelioid granulomas containing multinuclear giant cells. The existence of these giant cells led us to place this case somewhere between Hashimoto's thyroiditis and subacute thyroiditis (de Quervain's). Since giant cells are seen in the histopathology of subacute thyroiditis, we propose that the possibility of Riedel's thyroiditis being a form of subacute thyroiditis be observed and studied.

Key words: Riedel's thyroiditis, thyroid fibrosis.

Introduction

Riedel's thyroiditis, first described by Riedel in 1896, is defined as invasive fibrous thyroiditis [1]. It is a multifocal rare disease of unknown aetiology, observed mostly in women [2]. Clinically it resembles both anaplastic carcinoma and lymphoma. Riedel's thyroiditis is generally a massive and stony goiter characterized by extensive fibrosis attached to the adjacent organs in the neck, causing symptoms of compression [3]. The case reported here differs in its histopathology. To date, several fibroinflammatory cells have been documented in Riedel's thyroiditis with no evidence of giant cells. The few giant cells observed in this reported case may offer a new perspective on Riedel's thyroiditis as a rare stage of subacute thyroiditis.

Case report

A 46-year-old female patient was referred to our clinic in August 2009. Her primary complaints were dysphagia and otalgia going back for about 6 months. A very hard fixed mass of 5-6 cm was observed in the thyroid region mostly on the left side. The ultrasonographic examination results showed homogenous character with 2 nodules of 3 and 7 mm in the right lobe and 1 nodule of 4 mm in the left lobe. The nodules were solid and hypoechogenic in character. Fine needle aspiration cytology (FNAC) did not reveal thyrocytes, but revealed acellular squames. Neck computerised tomography (CT) showed the mass to have external attachments to the strap muscles. Laboratory analyses showed that Free T3, Free T4, TSH (third generation), TSH Receptor Antibody, Antithyroglobulin Antibody, Antimicrosomal Antibody values were normal. Carotid arteries and jugular veins were also normal as observed with Doppler ultrasonography. In early September 2009, the patient underwent total thyroidectomy.

The macroscopic findings of the specimen, on gross examination showed the left thyroid lobe measurement to be $6.8 \text{ cm} \times 4 \text{ cm} \times 3.3 \text{ cm}$. The left lobe was asymmetrically enlarged by a greyish-white, firm lesion measuring $5 \text{ cm} \times 3.2 \text{ cm} \times 2.5 \text{ cm}$. On cut-section, this lesion was obliterating the greater part of the left thyroid lobe and showing invasion beyond the thyroid capsule. Residual thyroid tissue was detected in the

neighbourhood of this lesion. Intra-operative frozen consultation biopsy was performed in order to exclude malignancy. It was assessed as benign. Sections of the right lobe did not reveal a specific lesion and was diffusely brownish.

Histopathological examination of the left thyroid lobe showed fibrosis and lymphoplasmacytic infiltration, extensive destruction of thyroid tissue and invasion of perithyroidal soft tissue, including blood vessels and striated muscle tissue (Fig. 1). The lymphocytic tissue was organized focally as aggregates. Some epithelioid granulomas containing multinuclear giant cells were detected (Fig. 2). Crystal violet stain for amyloid was negative. Microscopic examination of the right thyroid lobe showed no specific lesion except for some lymphoid aggregates and follicles. Consequently, the final diagnosis was Riedel's thyroiditis.

Discussion

Riedel's thyroiditis is a very rare form of chronic thyroiditis exhibiting an inflammatory proliferative fibrotic process, which destroys the gland and permeates throughout the capsule. In a large series of thyroidectomies, the incidence is given as 0.04% to 0.30% [1, 2].

Although pathogenesis and aetiology are obscure, there are some hypotheses for the aetiology. It is considered to be a late stage of chronic thyroiditis. A case with concurrent fibrotic Hashimoto's, Riedel's thyroiditis and acute thyroiditis have been reported [4]. An autoimmune stimulus was considered in the process of fibrosclerosis since thyroid-specific autoantibodies have been detected in some patients [1]. In this reported case, thyroid-specific autoantibody values were normal.

Ultrasound, CT and magnetic resonance imaging (MRI), FNAC do not give enough information for precise diagnosis [5, 6]. Intra-operative frozen section is definitely beneficial for the diagnosis of Riedel's thyroiditis [7]. Definitive diagnosis of Riedel's thyroiditis can be obtained by histology. Fibrotic inflammations, extensions to adjacent tissues, occlusive phlebitis, infiltration of walls of small and medium-sized veins by lymphocytes and plasma cells are observed. Lymphocytic infiltrate is composed of lymphocytes, monocytes, granulocytes, and eosinophils, with no evidence of giant cells, lymph follicles, oncocytes, or granulomas [8]. A case with giant cell arteritis has been reported [9]. Also cytologically atypical B cells with large nuclei and multiple small nuclei and of polyclonal nature, as in lymphoepithelial lesions, has been reported [10].

Histopathologically, the main diagnostic requirements for Riedel's thyroiditis are differentiating it from the fibrous form of Hashimoto's thyroiditis and from subacute thyroiditis. Generally, in subacute thyroiditis (de Quervain's), microscopic examinations show areas of marked inflammation and granulomas containing foreign body giant cells. Characteristically, these



Fig. 1. Thick fibrous bands and lymphoplasmacytic infiltration replacing thyroid tissue. HE, magnification 100×



Fig. 2. Fibrohyalinized tissue and mononuclear inflammatory infiltrate replacing thyroid tissue and a granulomatous focus (arrowhead) (HE, magnification 40×). Inset: Higher magnification of granulomatous focus: multinucleated giant cells surrounding residual colloid (HE, magnification 400×)

granulomas surround the follicles and the multinucleated giant cells engulf the colloid. Most of these giant cells are of histiocytic nature. These granulomas are not very distinct and do not have caseation necrosis. Areas of fibrosis are also seen, usually in patchy distribution [11]. There is little or no adherence to the surrounding tissues. In Hashimoto's thyroiditis, there are dense fibrous bands dividing the thyroid parenchyma into nodules, which are composed of lymphoid follicles with germinal centres, plasma cells, and oxyphilic metaplasia of follicular epithelial cells [12]. Although it is reported that Riedel's thyroiditis differs clinically and histopathologically from Hashimoto's thyroiditis and subacute thyroiditis, and thus it is considered to be an independent disease, the histopathological presence of giant cells supports our argument that Riedel's thyroiditis might be a rare variety of subacute thyroiditis.

Conclusion

Riedel's thyroiditis is a disease seen very rarely and is open to new findings in order to map its aetiology and its behaviour for designing an effective treatment. In a case like this, with a fibrotic changes extending beyond the thyroid capsule but with normal thyroid antibodies and without any clinical symptoms besides compression, the presence of giant cells is exciting. Since giant cells are seen in the histopathology of subacute thyroiditis (de Quervain's), we propose that the possibility of Riedel's thyroiditis being a form of subacute thyroiditis should be regarded and studied further.

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