CASE REPORT



Primary thyroid T cell lymphoproliferative disorder with a 10-year history of neck mass: a case report and literature review

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Abstract

Thyroid lymphoproliferative disorder is known to be associated with Hashimoto thyroiditis. The major histological types of thyroid lymphoproliferative disorders are malignant lymphoma, such as diffuse large B cell lymphoma, extranodal marginal zone B cell lymphoma of mucosa-associated lymphoid tissue (MALT), and follicular lymphoma. All of these are B cell lymphomas; however, T cell lymphoproliferative disorders represent only a minority of cases. Since T cell lymphoproliferative disorder rarely develops and T cell lymphoma is a heterogeneous entity, it is difficult to concisely summarize this complex topic. Here, we present a case of primary thyroid T cell lymphoproliferative disorder reminiscent of indolent T cell lymphoproliferative disorder of the gastrointestinal tract: the pathological findings from this case also were similar to those of MALT lymphoma such as small lymphoid cells, lymphoepithelial lesions, and a low Ki-67 labeling index. T cell lymphoproliferative disorders with these pathological features have been reported in the thyroid gland, lymph nodes, palate, spleen, and gastrointestinal tract. Furthermore, we reviewed and summarized the studies on thyroid T cell lymphoproliferative disorder to cell lymphoproliferative disorder so the cases and their clinicopathological characteristics.

Keywords Thyroid gland \cdot T lymphocytes \cdot Lymphoproliferative disorders \cdot Hashimoto disease \cdot Tertiary lymphoid structures

Introduction

Primary thyroid T cell lymphoproliferative disorder is extremely rare; therefore, it is difficult to consider it a differential diagnosis. In particular, if the cytology is bland and the morphology is similar to that of extranodal marginal zone B cell lymphoma of mucosa-associated lymphoma tissue (MALT), achieving a precise diagnosis is more difficult. Here, we present a case of peripheral T cell lymphoproliferative disorder of the thyroid, which is composed of small lymphoid cells with a low Ki-67 labeling index. Moreover, we thoroughly reviewed the literature to summarize the clinicopathological features of primary thyroid T cell

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Bomi Kim domabem96@paik.ac.kr lymphoproliferative disorder and compared them with those of our case.

Clinical history

A 64-year-old woman presented with a 10-year history of a thyroid mass, the size of which had rapidly increased recently; therefore, she was recommended for surgery. Fine-needle aspiration revealed adenomatous goiter. Laboratory data on admission were as follows: hemoglobin level, 12.5 g/dL (normal range, 11.2-14.6 g/ dL); white blood cell count, 9.97×10^9 /L (normal range, $3.15-8.63 \times 10^{9}$ /L); platelet count 258×10^{9} /L (normal range, $138-347 \times 10^{9}$ /L); lactate dehydrogenase level, 198 IU/L (normal range, < 250 IU /L); β2-microglobulin level, 2.00 mg/L (normal range, 0.81-2.19 mg/L); thyroidstimulating hormone level, 7.61 µIU/mL (normal range, 0.27-4.2 µIU/mL); T3 level, 127.0 ng/dL (normal range, 80-200 ng/dL); free T4 level, 0.58 ng/dL (normal range, 0.93-1.70 ng/dL); anti-thyroglobulin level, > 10,000.0 IU/ mL (normal range, 0-4.1 IU/mL); and anti-thyroid

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peroxidase antibody level, 26.3 IU/mL (normal range, 15-65 IU/mL). On physical examination, a solid mass was palpable on the neck. Computed tomography (CT) of the thyroid revealed a diffusely enlarged thyroid gland (Fig. 1a). Lymph node enlargement was also found in bilateral lateral neck (Fig. 1b). Total thyroidectomy with central lymph node dissection was performed. The diagnosis was peripheral T cell lymphoproliferative disorder, associated with Hashimoto thyroiditis. Post-operative neck CT revealed the decrease in the size of small lymph nodes. Chest, abdominal, and pelvic CT exhibited no remarkable abnormality. As revealed through positron emission tomography, lymph nodes in the right neck level III and left neck levels II-IV showed mild uptake of 18F-fluorodeoxyglucose, indicating that the figures were indeterminate for malignancy. Core needle biopsy was performed on the ovoid-shaped, slightly enlarged lymph nodes in the neck at level II on the left side, in which benign lymphadenopathy was radiologically suspected. Hematoxylin and eosin staining showed no evidence of T cell lymphoproliferative disorder. While the physicians were assessing cyclophosphamide, hydroxydaunorubicin, vincristine, etoposide, and prednisone chemotherapy, she refused bone marrow

evaluation and discharged. She had been well at follow-up after discharge.

Materials and methods

Immunohistochemistry and Epstein Barr virus-encoded ribonucleic acid (EBER) in situ hybridization (ISH)

Immunohistochemical staining was performed using the Vantaa BenchMark ULTRA automatic immunostainer and Leica BOND MAX automated system according to the manufacturers' instructions. The following primary antibodies were used: anti-bcl-2 (Bcl-2/100/D5), anti-bcl-6 (NCL-L-Bcl-6–564), anti-CD3 (LN10), anti-CD4 (SP35), anti-CD8 (SP57), anti-CD10 (56C6), anti-CD20 (L26), anti-CD30 (Ber-h2), anti-CD56 (1B6), anti-CXCL-13 (AF801), anti-Ki-67 (MIB-1), anti-pan-cytokeratin (AE1/AE3), anti-PD-L1 (SP263), anti-terminal deoxynucleotidyl transferase (TdT) (SEN28), anti-T cell receptor (TCR) beta F1 (8A3), anti-TCR gamma (H-41), and anti-T cell intracellular antigen-1 (TIA-1) (2G9A10F5). EBER ISH was performed using the Ventana

Fig. 1 Thyroid computed tomography and gross examination. a Thyroid CT disclosed diffuse enlargement of bilateral thyroid glands with one enhancing nodule in the left thyroid lobe. The white arrow indicates one of the enlarged lymph nodes. b Multiple lymph node enlargement with perinodal infiltration in both sides of the neck was also noted. c The thyroid was diffusely enlarged and the cut surface was homogenous and yellow, and soft without hemorrhage, necrosis, or any nodular lesion





Fig. 2 Microscopic examinations. **a** (Hematoxylin and eosin, $\times 1$) Normal parenchyma of the thyroid is obliterated almost totally by dense lymphocytic cells. **b** (Hematoxylin and eosin, $\times 400$) Lymphoid cells in interfollicular areas show minimal cytological atypia. **c** (Hematoxylin and eosin, $\times 400$) Atypical small lymphocytes invade follicular epithelial cells, creating lymphoepithelial lesions. **d** (Hematoxylin and eosin, $\times 100$) Reactive lymphoid follicles are distributed throughout the thyroid. **e** (Hematoxylin and eosin, $\times 12.5$) Normal structure of neck lymph node is effaced and extra-lymph nodal exten-

Benchmark ULTRA automatic immunostainer according to the manufacturer's instructions (EBER1 DNP Probe).

Immunoglobulin heavy chain (IgH) gene and T cell receptor (TCR) gene rearrangement study

DNA was extracted from formalin-fixed paraffin-embedded tissue, and IgH gene and TCRγ gene rearrangement polymerase chain reactions (PCRs) were performed using IdentiCloneTM IGH Gene Clonality Assay and IdentiCloneTM TCRG Gene Clonality Assay. To cover IgH gene, primers that targeted conserved framework (FR1-3) and diversity sion is noted. **f** (Hematoxylin and eosin, ×400) Small lymphoid cells, which are morphologically identical to those of the thyroid, infiltrated the lymph node, and filled the sinuses of the lymph node. **g** (CD3 immunohistochemistry, ×100) Atypical lymphoid cells which proliferated in interfollicular areas. Lymphoepithelial lesions are positive for CD3. **h** (CD4 immunohistochemistry, ×200) CD4 immunohistochemistry × 200) CD4 immunohistochemistry × 200) CD8-positive lymphoid cells are scattered

(DH1-7) regions and the downstream consensus JH gene segments were used. The probes for the gene for TCR γ targeted V γ lf, V γ 9, V γ 10, V γ 11, J γ 1.3/2.3, and J γ 1.1/2.1.

Results

Macroscopic and microscopic features

The thyroid glands weighed 121 g. The lobes were wellencapsulated and diffusely enlarged. The cut surface was homogeneous and yellow without evidence of hemorrhage or



Fig. 3 T cell receptor rearrangement (TCR) polymerase chain reactions. Capillary gel electrophoresis shows clonal TCR β (**a**–**d**) and TCR γ (**e** and **f**) rearrangement products in the thyroid gland (**a**, **c**, and **e**) and the lymph node samples (**b**, **d**, and **f**). Each pair of thyroid gland and lymph node has similar clonal TCR β and TCR γ gene rearrangement patterns

necrosis (Fig. 1c). Small, mildly cleaved lymphoid cells with marked proliferation infiltrated the thyroid follicles (Fig. 2a and b), thus forming lymphoepithelial lesions similar to those in MALT lymphoma (Fig. 3c). Reactive lymphoid follicles were distributed throughout the thyroid (Fig. 2d). Plasma cells and eosinophils were rarely observed. Prominent arborizing high endothelial venules were not observed. The thyroid follicular cells showed oncocytic change that are occasionally found in Hashimoto thyroiditis. Four small (<1 cm in size) perithyroidal lymph nodes excised from the central part of the neck showed effacement of nodal architecture. The sinuses were filled with small lymphoid cells, which were morphologically identical to tumor cells of the thyroid (Fig. 2e and f).

Immunohistochemical staining and EBER ISH

The tumor cells were positive for CD3 (Fig. 2g) and CD4 (Fig. 2h), suggesting that the tumor originated from a helper T cell. Sparsely scattered small lymphoid cells were positive for CD8 (Fig. 2i). The CD4:CD8 ratio was 9:1. CD20 did not react with tumor cells and was only expressed in lymphoid follicles. Cytokeratin staining highlighted the lymphoepithe-lial lesions. The Ki-67 labeling index was 10% in the interfollicular area. EBER ISH showed negative results. CD10, bcl-2, bcl-6, CD30, CD56, CXCL-13, TdT, and TIA-1 were all negative. Tumor cells were positive for TCR beta F1 and CD5, and negative for TCR γ .

IgH and TCRy gene rearrangement

PCR analysis of the TCR β (Fig. 3a–d) and TCR γ (Fig. 3e and f) genes revealed clonal gene rearrangement (dominant predominant peaks) in the thyroid gland (Fig. 3a, c, and e) and lymph node samples (Fig. 3b, d, and f). Each pair had similar clonal TCR β and TCR γ rearrangement patterns. In contrast, IgH had a polyclonal background.

Discussion

Considering the histologic features, the main pathologic differential diagnosis in this case was Hashimoto thyroiditis although TCR gene rearrangement PCR showed monoclonality. Degeneration of follicular cells in Hashimoto thyroiditis occurs via CD8 + T cell-mediated cell death, cytokinemediated cell death through the activation of CD4 + Th1 cells, and antibody-dependent cell-mediated cytotoxicity. Therefore, dense infiltration of CD4 + and CD8 + lymphocytes is expected in Hashimoto thyroiditis. In the present case, lymphoepithelial lesions and interfollicular areas were diffusely positive for CD4. However, CD8-positive cells were rarely found throughout the thyroid gland. Moreover, TCR monoclonality is detected in both the thyroid gland and lymph node, which shows almost identical clonal rearrangement patterns. Considering these features, the diagnosis in the present case was T cell lymphoproliferative disorder. The World Health Organization (WHO)'s 2017 edition of hematopoietic and lymphoid tissues added this entity: indolent T cell lymphoproliferative disorder of the gastrointestinal tract [1]. This entity consists of monotonous mature-appearing small round lymphoid cells with an indolent clinical course [1]. Table 1 presents a summary of the primary thyroid T cell lymphoproliferative disorders in the literature. There were five cases with pathological findings similar to those in our case [2–4]. Interestingly, Hayashi et al. suggested a new subgroup of T cell lymphoma, "indolent peripheral T cell lymphoma," characterized by small monotonous lymphoid cells with mild nuclear atypia, low Ki-67 labeling index, and good prognosis compared to conventional peripheral T cell lymphoma, NOS [3]. If we consider that the reported cases often have missed Ki-67 labeling indices or mitosis value, there are several reported cases similar to ours [5-10]. In summary, thyroid T cell lymphoproliferative disorder mimicking indolent T cell lymphoproliferative disorder of the gastrointestinal tract occurs in the age range of 32-81. They were found at an almost equal frequency in both males and females. Eighteen of 19 patients were alive. We were not able to confirm if the patient who died could be classified as an indolent T cell lymphoproliferative disorder because the proliferative index or mitotic count has not been mentioned. Considering these points, it is reasonable that this disease can be classified as a T cell lymphoproliferative disorder at extranodal sites. However, these pathological features do not always indicate good prognosis. Of the patients with nodal "small-cell peripheral T-cell lymphoma" with a low Ki-67 labeling index, all three patients died excluding two who could not be followed up [11]. Despite the "indolent" pathological findings, most of these patients showed aggressive clinical behavior such as systemic lymph node enlargement or bone marrow involvement [11]. Additionally, this case had lymphoepithelial lesions, similar to the pathological features of MALT lymphoma. Uherova et al. analyzed eight patients in whom peripheral T cell lymphomas resembling MALT lymphoma: 4 patients with T cell lymphomas of the lymph nodes, 1 with T cell lymphoma of the lymph node and salivary gland, 1 with T cell lymphoma of the lymph node and tonsil, 1 with T cell lymphoma of the hard palate, and 1 with T cell lymphoma of the tongue [12].

Table 1 St	ummary of clinicopathologic	characteristics of re-	ported pr	imary thyroid	1 T cell lymphomas resemb	ling indolent T cell lyr	mphoproli	ferative disorder		
Case no	Reference	Age (years)/sex	HT	HTLV-1	FNA	Cell size	LEL	Proliferation (Ki-67 LI or mitotic count)	Diagnosis	Follow-up period (mo.)/ status
1	Colovic (2007) [13]	34/M	1		NA	Small		NA	PTCL, NOS	13/dead
2	Hayashi (2013) [3]	60/F	NA		NA	Small	+	0.89	PTCL, NOS	62/alive
3	Hayashi (2013) [3]	67/F	NA	ı	NA	Small	+	0.48	PTCL, NOS	36/alive
4	Hayashi (2013) [3]	59/M	NA		NA	Small	+	1.07	PTCL, NOS	18/alive
5	Kim (2010) [4]	48/F	+		Not diagnostic	Small	+	1	PTCL NOS	NA
9	Koida (2007) [5], Yoshida (2013) [10]	61/M	+	ı	NA	Small to medium	+	NA	PTCL, NOS	28/alive
L	Mizukami (1990) [14]	W/6L	ı	NA	NA	Small	NA	NA	TCL	48/alive
8	Motoi (2005) [7]	71/F	+	ı	NA	Small to medium	+	10/10HPF	PTCL, NOS	27/alive
6	Okamoto (2005) [15]	86/F	·		Not diagnostic	Small	NA	NA	PTCL, NOS	48/alive
10	Raftopoulos (2001) [8]	72/M	+	NA	Atypical lymphocytes	Small to medium	+	NA	TCL	12/alive
11	Reeders (2017) [2]	59/M	+1	NA	NA	Small to medium	+	10 - 15%	PTCL, NOS	NA
12	Suzuki (1994) [16]	57/F	+	NA	Atypical lymphocytes	Small	NA	NA	TCL	9/alive
13	Yang (2008) [17]	32/M	ı	ı	Malignant lymphoma	Small to medium	+	NA	PTCL, NOS	12/alive
14	Yokoyama (2012) [18]	70/F	+		Chronic thyroiditis	Small to medium		0.6	PTCL, NOS	22/alive
15	Yoshida (2013) [10]	63/F	ı	NA	NA	Small to medium	+	NA	PTCL, NOS	15/alive
16	Yoshida (2013) [10]	51/M	+	NA	NA	Small to medium	+	NA	PTCL, NOS	97/alive
17	Yoshida (2013) [10]	67/F	+	NA	NA	Small to medium	+	NA	PTCL, NOS	70/alive
18	Yoshida (2013) [10]	83/M	+	NA	NA	Small to medium	ı	NA	PTCL, NOS	13/alive
19	This case	64/F	+1	NA	Adenomatous goiter	Small	+	10%	PTCL, NOS	NA
HT, Hashi PTCL, NO	moto's thyroiditis; <i>HTLV-1</i> , <i>S</i> , peripheral T cell lymphor	human T cell lymp na, not otherwise spe	hotrophic scified; To	virus; <i>FNA</i> <i>CL</i> , T cell lyr	, fine-needle aspiration; <i>LE</i> nphoma	EL, lymphoepithelial 1	esion; LI,	labeling index; n	<i>••.</i> , month(s); <i>NA</i>	, not applicable;

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The relationship between autoimmune thyroiditis and MALT lymphoma is well known. However, the number of cases with primary thyroid T cell lymphoproliferative disorder including T cell lymphoma is extremely low, making it difficult to determine whether primary T cell lymphoproliferative disorder is correlated with autoimmune thyroiditis. Based on our review, 9 of 19 cases (47.4%) with T cell lymphoproliferative disorder, including our case, had Hashimoto thyroiditis or chronic thyroiditis. In autoimmune diseases, almost all infiltrative immune cells, which are CD4 + T cells, react to T1 chemokine receptors such as CXCR3 + and CCR5 +, which are expressed in the thyroid glands [5, 19]. Koida et al. presumed that chronic stimulation of CD4 + T cells leads to the development of a neoplastic clone [5]. This theory cannot definitively explain the development of primary T cell lymphoproliferative disorders of CD8 + T cells.

It is unclear whether fine-needle aspiration cytology is useful in the diagnosis of thyroid lymphoproliferative disorders including malignant lymphoma. Although additional examinations such as flow cytometry and immunohistochemistry can help in the diagnosis of thyroid lymphoproliferative disorder [20], they are performed only when clinicians suspect malignant lymphoma as a differential diagnosis. Sangalli et al. reported that cytological studies of the thyroid showed 100% accuracy (8/8) in the diagnosis of high-grade diffuse large B cell lymphoma [21]. However, ten cases of low-grade MALT lymphoma were diagnosed with Hashimoto thyroiditis. As shown in Table 1, only three out of five cases of T cell lymphoproliferative disorder were diagnosed as malignant lymphoma or atypical lymphoid cells based on fine-needle aspiration. In only one case diagnosed as malignant lymphoma, fine-needle aspiration was performed with immunohistochemistry of CD3 and CD20 as well as TCRy gene rearrangement PCR analysis [9]. The cytological diagnosis in the present case was an adenomatous goiter. Therefore, flow cytometry or immunohistochemistry is recommended for conventional cytology if malignant lymphoma is clinically suspected.

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Author contribution BK designed and edited the study protocol. KHN reviewed clinical and pathological data. BK made pathologic diagnosis and took microscopic pictures. BK and KHN reviewed and contributed to the writing of the manuscript.

Data availability Data and images used in this study were provided by the Inje Biobank of Inje University Busan and Haeundae Paik Hospital, a member of Korea Biobank Network (IJB-20–09).

Code availability Not applicable.

Declarations

Ethics declarations Ethics approval has been obtained by the institutional review board of Inje University Haeundae Paik Hospital (IRB File no: 2020–11-006).

Consent to participate Informed consent was obtained from a participant.

Conflict of interest The authors declare no competing interests.

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