Case Report

Co-occurrence of Auto-Immune Thyroid Disease and Acromegaly

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Abstract

Objective: The aim of the paper is to discuss the co-occurrence of thyroid disease especially the autoimmune ones and acromegaly in conjunct of two acromegalic patient who developed autoimmune thyroid disease.

Materials and Methods: Case report.

Results: Although thyroid disease is frequently develop in acromegalic patients, the co-occurrence of autoimmune thyroid disease is rare. Because of the frequent co-occurrence, an associated thyroid disease should be on mind in the management of a complaint of an acromegalic patient. Although it is a rare co-occurrence of an autoimmune thyroid disease, this possibility also should be considered in management of acromegalic patients.

Keywords: Acromegaly, Autoimmune thyroid disease, Grave’s disease, Hashimoto thyroiditis

INTRODUCTION

Acromegalic patients frequently have thyroid disorders, even the exact prevalence is controversial (1). About 15-78% of acromegalic patients develop goitres (2,3). The definite mechanism of development of goitre remains unclear, but it is assumed that IGF-I is the main promoter in growth of thyroid gland in acromegaly (2,4), reduction in thyroid size in successfully treated acromegalics support this theory (3). The exact prevalence of auto-immune thyroid disorder in acromegalic patients is also not clear. In an Italian multi-center analysis the 0.4% of acromegalic patients had toxic diffuse goitre while 4.6% had Hashimoto thyroiditis, prevelance of positive antibody against thyroid did not differ from iodine deficient arreas (2). Grave’s disease associated with acromegaly is also a rare co-occurrence, fourteen cases have been reported up to 2005 (2,3,4,5).

We present two rare cases of acromegaly associated with autoimmune thyroiditis, one Grave’s disease and the other one Hashimoto thyroiditis. We discussed the association between acromegaly and
thyroid disease, especially the autoimmune based ones.

**CASE PRESENTATION**

**Case 1**

A fifty-four years old female patient, referred to our medical center with complaints of headache, back pain and growth of the hands and feet, hyperhydrosis for two years. She did not have loss of vision.

On her physical examination she had typical acromegalic face features with prognatism, widened teeth space and enlarged nose. Her thyroid was palpable with a 10x15 mm nodule on the left side. The other systemic examinations including the visual field examination were normal.

Her blood biochemistry and complete blood count were normal except an impaired fasting glucose. GH did not suppress, even increased after a 75 gr OGTT route. Her insulin-like Growth Factor-1 (IGF-1) was 720 ng/Ml (120-500) and IGF-1 Binding Protein (IGF-1BP) was 9.8 mg/mL (3.2-7). Her gonadotrophin levels was consistent with post-menopausal state and TSH was elevated while thyroid hormone levels remaining normal. Her anti-thyroglobulin antibody level was 423 IU/mL and anti-thyroid peroxidase was 330 IU/mL.

Ultrasonography of the thyroid revealed multinodularity and a paracrical heterogeneity. The dominant nodules were 11x9 mm on the right, 26x16 mm on the left and 20x14 mm on the isthmus. Magnetic Resonance Imaging (MRI) revealed a 10x8x8 mm mass lesion circumscribing the cavernous segment of the internal carotid artery.

A fine needle aspiration biopsy was performed for each of the nodules. Pathologic assessment was consistent with hashimoto thyroiditis without a malignant figure. We started L-thyroxin until we achieved an euthyroid state. Then a trans-sphenoidal trans-nasal approach was performed following a long acting octreotide medication because of the residual tumor and failed control of disease activity assessed by non-suppressed GH levels after an oral glucose tolerance route. After the operation hypocotisolism was diagnosed with an insulin tolerance test. She is now taking 30 mg Octreotic LAR one time a month and L-thyroxin 100 mg a day and prednisolone 7.5 mg a day. She is now symptom-free and her GH and IGF-1 levels are normal.

**Case 2**

A 48 years old female patient presented with nervousness and sweating who had a diagnosis of acromegaly for ten years. She had gone to a trans-nasal trans-sphenoidal tumor extirpation following a transcranial approach which both of the procedures failed to control the disease. Because of the failure of surgical intervention, she had a cranial radiotherapy following a long acting octreotide therapy. Because of her presenting symptoms, she was thought to have an active acromegaly despite the medical therapy.

On her physical examination, her blood pressure was 120/80 mmHg with a heart rate of 84 beats per second. She had prognatism, enlarged nose, her teeth were separated and her extremities were enlarged which are typical clinical features of acromegaly. The thyroid was diffusely palpable with no bruist or nodularity. She had thiny tremor of the hands.

Her biochemial assessment and blood count were normal. TSH:0.047 mU/mL (0.3-5), fT4:2.15 ng/dL (0.8-1.8), fT3:6.14 pg/mL (1.8-4.7), GH was 9.79 ng/mL (0.06-5) and IGF-1:643 ng/Ml (120-500).TGab was 618, TPOab was 780 IU/mL. Thyroid receptor antibody was 0.1 IU/Ml (0-10). Other pituitary hormones were normal.

On her MRI a 13x13x8 mm adenoma circumscribing the cavernous segment of the internal carotid artery was shown. Thyroid ultrasonography revealed diffusely enlarged thyroid gland with increased
vascularity consistent with thyroiditis. No nodular formation was noted. Thyroid scan with 5 mci Tc-99m pertechnetate revealed an increased heterogeneous activity and the uptakes were increased.

Propylthiouracil was started for thyrotoxicosis and disease was controlled with no adverse effect during the course of treatment. Uncontrolled disease activity was assessed after she achieved an euthyroid state and we increased the dose of the octreotide LAR. She is now taking 40 mg Octreotide LAR one time a month and propylthiouracil 25 mg three times a day. She is now symptom free and her GH levels and IGF-1 are normal.

DISCUSSION

Acromegalic patients frequently develop goitres and the prevalence is 15-78% in different series representing a high prevalence (2,3,5). The development of goitre in acromegalic patients seems independent of TSH and iodine deficiency (2,3), it is assumed that GH and IGF-1 may be the main factor in promoting the enlargement of the thyroid gland since the strict disease control reduces the thyroid size (3,4).

The exact prevalence of auto-immune thyroid disease is also controversial. In their series, Wüster et al reported no Graves disease among 80 acromegalic patient (6). In another study of Gasperi et al, the prevalence of Hashimoto thyroiditis was 4.6% while the prevalence of Grave’s was 0.4%, TGab was positive in %21 and the TPOab was positive in %23 like in iodine-deficient areas among 258 acromegalic patients (2). Female patients frequently developed Hashimoto thyroiditis in this study, also our cases which developed auto-immune thyroid disease are female as expected. Our two cases represent a rare clinical association of acromegaly and auto-immune thyroid disorder. It seems that Grave’s is more rare disorder in acromegalic patients, because only fourteen cases were reported until now (3,4,5,6).

Effected acromegalic patients and patients in iodine deficient areas may share the same mechanism of autoimmunity in which there is an increased presentation of thyroid antigens to the immune system (7,8). But we think the co-incidence of acromegaly with Grave’s disease is probably accidental, because in the reported series the prevalence of thyroid disease does not differ between acromegalic patients and patients from iodine deficient areas.

In acromegalic patients symptoms associated with thyroid disease may be confused with symptoms of active acromegaly. The high co-occurrence of thyroid disease with acromegaly should be considered while evaluating the symptoms like sweating, fatigue, myalgias in an acromegalic patient. Since acromegaly is associated with increased cardiovascular death, an associated thyroid disease may influence the disease progress of an existing cardiovascular disease. In such condition medical management should be immediately started against the hyper or hypofunctioning thyroid disorder.

Because of the same presenting sympotms, clinicians should consider an associating thyroid disease while managing an acromegalic patient. This later issue is not a rare co-occurrence. Although it seems a much more rare co-occurrence of acromegaly with autoimmune thyroid disorder, same clinical presentation may be shared with acromegaly.

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