A case of TSH secreting pituitary adenoma with Evans’ syndrome

Toshio Seki, Atsushi Yamada, Natsumi Kikuno, Masayuki Oka, Masami Seki, Atsushi Takagi, Masahiro Fukuogi
Division of Neuroendocrinology, Endocrinology and Metabolism, Division of General Internal Medicine, Department of Internal Medicine, Tokai University School of Medicine, Shizuoka, Japan

Introduction
TSH-secreting pituitary adenoma (TSHoma) is rare. Tumors may lead to the development of hyperthyrotropism through a mechanism called “disconnection” of hypothalamic control of TSH (pituitary). In contrast, Evans’ syndrome is a rare complication of autoimmune hypophysitis (IAHA) and idiopathic hypophysitis (thought to have a common immunological mechanism). In recent years, various cases of Evans’ syndrome associated with hyperthyrotropism have been reported. Here, we report a case of TSHoma with Evans’ syndrome.

Physical examination
- Height: 163.0 cm, Weight: 49.0 kg
- Vital signs: BP 127/70 mmHg, HR 148 min, RR 24 min
- Neck: diffusely enlarged thyroid gland
- Chest: Clear, no edema, no heart murmur
- Abdomen: Soft, no tenderness, normal bowel sounds, no hepatomegaly
- Ext: scapular ecchymosis, edema

The patient presented with hyperthyrotropism, ecchymosis, and edema.

Sero logical findings
- Anti-TSHR: +
- Anti-TG: +
- Anti-TPO: +
- Anti-thyroid antibodies: +
- Anti-thyroid peroxidase antibodies: +

The serological test results showed positive antibodies for thyroid antigens.

Routine laboratory data
- WBC: 4900 µl, Hb: 14 g/dl
- BUN: 18 mg/dl
- Cr: 0.9 mg/dl
- TSH: 49.7 μIU/ml
- FT3: 1.9 pg/ml
- FT4: 1.9 pg/ml
- TSHR antibodies: Positive
- Thyroid antibodies: Positive

Routine laboratory data showed positive thyroid antibodies and increased TSH levels.

Endocrine laboratory data
- Glucose (mg/dl): 110
- TSH (mIU/ml): 49.7
- FT3 (pg/ml): 1.9
- FT4 (pg/ml): 1.9

Endocrine laboratory data showed high TSH levels with normal glucose levels.

MRI findings on admission
- Pituitary macroadenoma
- Hypothalamic involvement
- Edema in the surrounding area

MRI revealed a large pituitary adenoma with hypothalamic involvement.

Discussion

Discussion 1
- In the face of our knowledge, there is no case report of Evans’ syndrome associated with TSHoma. It would be interesting to determine whether these two disorders exist concomitantly. Several studies suggest that both diseases have the same immunological mediation, includingAITLA suppression of cell proliferation and cytokine production.

Discussion 2
- The diagnosis of TSHoma is challenging due to the lack of specific symptoms. No clear guidelines for the treatment of TSHoma have been established.

Discussion 3
- Another possible relation in patients with TSHoma and Evans’ syndrome is the presence of a genetic polymorphism related to the pituitary tumor-transforming gene (PTTG). TSHoma and Evans’ syndrome are both autosomal dominant disorders.

References

Conclusion
- In summary, we describe here a case of TSHoma associated with Evans’ syndrome. To the best of our knowledge, there is no case report of Evans’ syndrome associated with TSHoma.

Clinical course of the patient
- Before admission, the patient had no symptoms suggestive of thyroid disease, and no family history of thyroid disorders was noted.

Laboratory results revealed low hemoglobin, hematocrit, red blood cell, and platelet levels. A hematologic disease was suspected, and the patient was referred to our hospital. She had a history of malnutrition and thyroid gland enlargement for 4 years prior to admission.

75-g OGTT and octreotide suppression test
- The 75-g OGTT revealed abnormal glucose intolerance (BMI 25.0 kg/m²). A concomitant TSH suppression test had a positive result (BMI, 90 kg/m²).

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75-g OGTT after surgery
- Before admission, the patient had no symptoms suggestive of thyroid disease, and no family history of thyroid disorders was noted.

Laboratory results revealed low hemoglobin, hematocrit, red blood cell, and platelet levels. A hematologic disease was suspected, and the patient was referred to our hospital. She had a history of malnutrition and thyroid gland enlargement for 4 years prior to admission.