Case Report

Simultaneous Coexistence of Thyrotropin-Prolactin-Secreting Adenoma and Papillary Thyroid Carcinoma

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Background. The thyrotropin-secreting adenomas (TSHomas) are very rare; their prevalence accounts for 0.5–3% of all pituitary tumors [1]. The sensitive laboratory assays and the accuracy of magnetic resonance imaging currently allow early and precise diagnosis. Simultaneous coexistence of TSHoma and papillary thyroid carcinoma (PTC) is still exceptional, and so far, only sixteen cases have been reported [2–14]. This association suggests the involvement of TSH in thyroid carcinogenesis and raises the difficulty of monitoring levothyroxine suppressive therapy in thyroid carcinoma after a total thyroidectomy.

1. Introduction

The thyrotropin-secreting adenomas (TSHomas) are very rare; their prevalence accounts for 0.5–3% of all pituitary tumors [1]. The sensitive laboratory assays and the accuracy of magnetic resonance imaging currently allow early and precise diagnosis. Simultaneous coexistence of TSHoma and papillary thyroid carcinoma (PTC) is still exceptional, and so far, only sixteen cases have been reported [2–14]. This association suggests the involvement of TSH in thyroid carcinogenesis and raises the difficulty of monitoring levothyroxine suppressive therapy in thyroid carcinoma after a total thyroidectomy.

2. Observation

A 50-year-old Moroccan woman, followed for type 2 diabetes, who underwent a total thyroidectomy with histology confirming papillary thyroid carcinoma (measuring 10 mm) classified T1b Nx M0 (Figure 1). She also received an adjuvant therapy with 100 mCi of radioactive iodine. Posttherapy whole-body scanning revealed fixation in the right thyroid bed compatible with thyroid remnant without metastasis. Cervical ultrasound did not objectify any residue or lymphadenopathy. We noted persistently unpressed TSH after gradual dose levothyroxine suppressive therapy up to 300 μg per day (4 μg/kg/day); her serum TSH level fluctuating from 4.5 to 50 μIU/mL and thyroglobulin (Tg) level was 6.8 ng/mL. We had quickly eliminated noncompliance, interfering medicines, and thyroid malabsorption, and we suspected a syndrome of inappropriate TSH secretion (SITSH). A pituitary MRI showed a macroadenoma in the sella turcica (10 × 12 mm), without local mass effect on adjacent structures, especially optic chiasm (Figure 2). The specific investigation was in favor of TSHoma, in particular the difficulty of monitoring thyroid carcinoma in nonremission of a T3.

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suppression test (100 μg/day divided in 3 administrations), the serum alpha-subunit levels ($\alpha$-GSU) was 4.9 ng/mL (normal postmenopausal value 0.6 to 1.5 ng/mL), and $\alpha$-GSU/TSH molar ratio was 1.4 (normal <1). Other investigations to evaluate pituitary function were normal (Table 1).

Accordingly, the patient has had transsphenoidal pituitary adenomectomy with histology confirming a thyroid-tropin-prolactin-secreting adenoma (50% of $\beta$TSH immunoreactivity and 25% of prolactin immunoreactivity) (Figure 3).
Table 1: Pertinent laboratory findings.

| Investigations          | Before admission | At admission | After surgery | After lanreotide | After cabergoline | Reference range |
|-------------------------|------------------|--------------|---------------|------------------|------------------|-----------------|
| TSH (μIU/mL)            | 4.5–50           | 35           | 20            | 6                | 0.5              | 0.35–4.95       |
| FT4 (pmol/L)            | —                | 23           | 24            | 22               | 19               | 12–22           |
| FT3 (pmol/L)            | —                | 5.3          | 6.7           | 6.6              | 5.9              | 2.8–7.1         |
| Thyroglobulin (ng/mL)   | —                | 6.8          | 22.7          | 8                | 0.4              | <1              |
| α subunit (ng/mL)       | —                | 4.9          | —             | —                | —                | 0.6–1.5         |
| α-GSU/TSH molar ratio   | —                | 1.4          | —             | —                | —                | <1              |
| LH (IU/L)               | —                | 36.8         | —             | —                | —                | 2.4–12.6        |
| FSH (IU/L)              | —                | 84.1         | —             | —                | —                | 3.5–12.5        |
| Estradiol (pmol/L)      | —                | 51           | —             | —                | —                | 46–607          |
| Prolactin (mIU/L)       | —                | 250          | 230           | —                | 90               | 72–511          |
| Cortisol (nmol/L)       | —                | 525          | —             | —                | —                | 171–536         |
| ACTH (pg/mL)            | —                | 40           | —             | —                | —                | 7–63            |
| IGF1 (μg/L)             | —                | 102          | —             | —                | —                | 93–245          |

(a)

Figure 3: Continued.
Three months after surgery, no residual tumor was apparent on pituitary MRI, but we noted a persistence of inappropriate secretion of TSH under 300 μg/d of levothyroxine. TSH level was 20 μIU/mL, FT4 level was 22.4 pmol/l, FT3 level was 5.6 pmol/l, and Tg level was 22.7 ng/mL (Table 1). Given the absence of biological remission after surgery, the patient underwent lanreotide therapy at 90 mg per month for three months, but the TSH level was still increased which provokes the stop of lanreotide. The TSH level was lowered for the first time after switching to cabergoline therapy and it allowed a complete response under 3 mg per week (Table 1). The follow-up is eight years after cabergoline therapy with a good cardiac tolerance: the patient has managed to achieve complete remission of thyroid cancer without any biochemical or structural recurrence (low nonstimulated thyroglobulin levels, normal cervical ultrasound, and negative 131I-whole-body scan). Automatically levothyroxine dosage was gradually reduced to 125 μg/day.

3. Discussion

We present here a case of thyroid cancer in coexistence with thyrotropin-prolactin-secreting adenoma. The sixteen published cases are reviewed and compared with our patient at the level of many parameters: age, sex, size of thyroid cancer, size of TSHoma, immunohistochemical study results, therapies used, and observed evolution (Table 2) [2–14].

TSH-suppressive hormonal therapy is a cornerstone of thyroid carcinoma therapy. Unrepressed TSH after levothyroxine suppressive therapy first evokes noncompliance, interfering medicines, thyroid malabsorption, and biological interference, before conducting further investigations for

Figure 3: Histopathological and immunohistochemical study showing (a) histopathological features of pituitary adenoma (x200), (b) 50% of βTSH immunoreactivity (x400), and (c) 25% of prolactin immunoreactivity (x400).
These diagnoses can be easily excluded by questioning, monitoring of drug intake, testing malabsorption, and repeating serum TSH measurement with several laboratory methods to exclude transitory changes and biological interference [15, 16]. Once these diagnoses are excluded, we are therefore faced with SITSH and the second step is to differentiate between TSHoma and syndromes of thyroid hormone resistance (RTH) [16, 17]. The differential diagnosis is complicated, so clinical presentations, laboratory assessment, and imaging advances may help and guide diagnosis (Table 3). T3 suppression test is the most specific and sensitive test, especially in patients with thyroid ablation. α-GSU may also be used; an elevated α-GSU concentration or a high α-GSU/TSH molar ratio favors the diagnosis of TSHoma in particular, in macroadenoma; these parameters are within the normal range in the case of a

| Case | Age | Sex | Size of TC (mm) | Size of PA (mm) | IHC of PA | Therapies of TSHoma | Therapies of TC | Evolution of TSHoma | Evolution of TC | References (year of publication) |
|------|-----|-----|----------------|----------------|-----------|---------------------|----------------|-------------------|----------------|-------------------------------|
| 1    | 55  | M   | 50             | 30             |           | TSH adenomectomy   | Total thyroidectomy/iratherapy | Remission       | Remission       | [2] (1991)                        |
| 2    | 37  | F   | 20             | 10             |           | TSH                | Refusing treatment               | NR              | Remission       | [3] (1998)                        |
| 3    | 27  | F   | 30             | 10             |           | TSH                | Octreotide/TS adenomectomy       | Remission       | NR              | [4] (2000)                        |
| 4    | 45  | F   | 20             | 15             |           | TSH                | Octreotide/TS adenomectomy       | Remission       | NR              | [5] (2001)                        |
| 5    | 47  | F   | 8              | 4              |           | TSH/PRL            | TS adenomectomy                  | NR              | Remission       | [6] (2006)                        |
| 6    | 50  | M   | 17             | 3              |           | TSH                | Refusing treatment               | Stability       | Remission       | [7] (2009)                        |
| 7    | 57  | F   | 8              | 26             | TSH/GH    | Octreotide         | Total thyroidectomy               | Remission       | Remission       | [8] (2010)                        |
| 8    | 38  | F   | 40             | 14             | TSH/FSH   | Octreotide/TS/放射线疗法 | Total thyroidectomy               | Stability       | Remission       | [9] (2013)                        |
| 9    | 27  | F   | 10             | 28             |           | TSH                | TS adenomectomy/lanreotide       | Partial response| Remission       | [9] (2013)                        |
| 10   | 33  | F   | 14             | 20             | TSH/GH    | TS adenomectomy    | Total thyroidectomy               | Remission       | Remission       | [10] (2014)                       |
| 11   | 47  | M   | 15             | 19             |           | TSH                | TS adenomectomy/lanreotide       | Remission       | Remission       | [11] (2015)                       |
| 12   | 46  | M   | 12             | 7              | TSH/GH    | TS adenomectomy    | Total thyroidectomy               | Remission       | Remission       | [11] (2015)                       |
| 13   | 42  | M   | 40             | 12             |           | TSH                | TS adenomectomy                  | Total thyroidectomy/iratherapy | Remission       | Remission       | [11] (2015)                       |
| 14   | 44  | F   | 30             | 16             | TSH/GH/FSH | Octreotide/TS adenomectomy | Total thyroidectomy/iratherapy | Remission       | Remission       | [12] (2017)                       |
| 15   | 27  | F   | NR             | NR             | TSH       | Surgical resection for nasopharyngeal tumor (ectopic TSHoma) | Total thyroidectomy/iratherapy | Remission       | Remission       | [13] (2017)                       |
| 16   | 57  | M   | NR             | 30             | TSH       | Octreotide/TS adenomectomy | Partial thyroidectomy       | Remission       | Remission       | [14] (2018)                       |
| Our case | 50  | F   | 10             | 12             | TSH/PRL  | TS adenomectomy/lanreotide/cabergoline | Total thyroidectomy/iratherapy | Remission       | Remission       | -(2021)                           |

NR, not reported; TC, thyroid cancer; PA, pituitary adenoma; IHC, immunohistochemistry; TSH, thyrotropin; PRL, prolactin; GH, growth hormone; FSH, follicle-stimulating hormone; TS, transsphenoidal.
and elevated biological findings (unrepressed TSH in T3 suppression test with clinical data (no family history of thyroid disease) and logical findings (pituitary macroadenoma) were associated morphologic information [24]. In our patient, the radio-
hence the interest in gathering clinical, hormonal, and association between TSHoma and RTH has been reported, and they are more frequent than TSHoma, so autonomous addition, a difficulty of imaging lies in the presence of pi-
was under a supraphysiological dose of levothyroxine. In case of TSHomas, while it is normal in RTH [20, 21] (Ta-
\[9\]). xQ his is the same mechanism described in Nelson’s syndrome after adrenalectomy in Cushing’s disease.

Many cases of TSHomas are completely asymptomatic which explains why the majority of TSHomas are macro-
adenomas as in our patient [20] Therefore, it is important that the clinician be alerted to persistently unrepressed TSH after levothyroxine suppressive therapy in order to be able to carry out investigations and treat TSHoma as early as possible. On the other hand, it is so difficult to control and to monitor the carcinologic side of PTC when it coexists simultaneously with TSHoma as in our case.

The unusual association of PTC with TSHomas enriches the hypothesis of a potential link between thyrotropic hypersecretion and thyroid carcinogenesis. Thus, the role of TSH in carcinogenesis and development of PTC have been suggested in several studies where we noted high incidence of PTC in the patients with TSHomas and a meta-analysis of 28 studies have objectified a significant association between TSH levels and the risk of PTC [20, 25, 26]. Conversely, thyroidectomy when performed first before resection of pituitary adenoma may affect growth rate and secretion of the TSHoma due to diminished negative feedback effect of thyroid hormones [9]. This is the same mechanism described in Nelson’s syndrome after adrenalectomy in Cushing’s disease.

| Table 3: The differential diagnosis markers between TSH-secreting adenomas (TSHomas) and resistance to thyroid hormones (RTH). |
|---------------------------------------------------------------|
| **Family history of thyroid disease**                         | TSHoma | RTH |
| Yes                                                            | Absent  | 85% |
| No                                                             | Present | Present |
| **Elevated thyroid hormone levels**                            | Present | Present |
| **Nycthemeral profile of TSH**                                | Absent  | Present |
| **High levels of SHBG (TeBG)**                                | Present | Absent |
| **Increased α-subunit**                                        | 65%     | 3%   |
| **α-GSU/TSH molar ratio**                                      | >1%     | <1%  |
| **Increase in TSH after a TRH**                                | Negative| Positive |
| **T3 suppression test**                                        | No TSH suppression | Suppression of TSH |
| **Somatostatin test**                                          | FT4 ↓ >30% | FT4 is not affected |
| **Multihormonal production**                                   | Possible | Absent |
| **MRI pituitary**                                              | 98%     | 10–20% |
| **DNA mutation analysis**                                      | –       | +    |

There are several therapeutic modalities for TSHoma, but the first line therapy is transphenoidal pituitary surgery in order to remove tumor mass and to normalize TSH, with a success rate of 80% [27, 28]. In our case, surgery allowed removing tumor mass without TSH suppression. Second-line treatments include repeat pituitary surgery, radiation therapy, and medical therapy; they should be considered after failed transphenoidal surgery and when the patient is not amenable to surgery or he declines it. Radiotherapy (conventional fractional radiotherapy or radiosurgery) may also be indicated in the treatment of TSHoma; however, the benign character of the condition, the potential side effects of radiation in particular hypopituitarism, and the long delay between radiation and efficacy will make its use less frequent, especially when medical therapies provide good control of the disease as we will see below [29]. Somatostatin analogs (SA) are the best choice of medical alternative treatment; they restore normal thyroid function in about 95% and shrink the size of TSHoma in up to 50% of patients [30–32]. Resistance to SA in our case is most likely related to nonexpression of somatostatin receptors. The presence of dopamine receptors on the thyrotoph cells justifies the therapeutic use of dopaminergic agents in TSHomas; however, efficiency in control of TSH secretion and tumor shrinkage was lower compared to SA and the best results were obtained in mixed TSH/ prolactin adenomas [30, 31, 33–36]. The immunohistochemical study had objectified an immu

50% of patients [30–32]. Resistance to SA in our case is most

4. Conclusion

The unusual association of PTC with TSHomas enriches the hypothesis of a potential link between thyrotropic hypersecretion and thyroid carcinogenesis. Our observation highlights efficiency of cabergoline in control of TSH secretion, especially in the presence of mixed TSH/
prolactin adenomas, without forgetting the difficulty encountered to control and to monitor the carcinologic side of PTC when it coexists simultaneously with TSHoma.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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