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Specific Immunohistochemical Profile of Poorly Differentiated Thyroid Carcinoma in a Diabetic Patient: One Case Report

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Abbreviations: HPFs: High-Power Fields; ATAb: Anti-Thyroglobulin Antibodies; WHO: World Health Organization; T1D: Type 1 Diabetes; PDTC: Poorly Differentiated Thyroid Carcinoma

Introduction

Several studies have demonstrated the link between the presence of diabetes disease and secondary appearance of thyroid cancer, often of follicular strain, and well differentiated form. Poorly differentiated thyroid carcinoma (PDTC) has an intermediate morphology and prognosis between differentiated and undifferentiated carcinomas, with a 50% survival rate at 10 years. Diagnosis is based on the five Turin criteria [1]. Therapeutic possibilities vary between external radiation therapy and immunotherapy with tyrosine kinase inhibitors from the outset, and -to a lesser degree- the radioactive-Iodine therapy.

The antitumor immunotherapy is not without consequences on the metabolic balance of diabetes. The coexistence of diabetes and undifferentiated cancer is not without consequence on the prognosis of both pathologies. The excess mortality attributed to the presence of diabetes was estimated at 25% [2].

Faced with diagnostic and therano-prognostic doubt related to the diabetic disease, the immunohistochemical study of anti-thyroglobulin antibodies (ATAb) on the surgical specimen or from biopsies can be carried out and makes it possible to propose other therapeutic alternatives.

Observation

We report the case of a 71-year-old patient with a long history of 25-year-type-2-diabetes, dyslipidemia and arterial hypertension who had a total thyroidectomy for multinodular goiter in April 2021. The surgery was incomplete type R2. Histological examination revealed the existence of a 6 cm right lobar PDTC, massively invading the peri thyroid soft tissues. Tumor proliferation was trabecular and solid with no papillary or anaplastic component. The postoperative course was simple. A total body CT scan (including cervical, thoracic, abdominal and pelvic regions) was performed postoperatively, and showed secondary bilateral and multifocal pulmonary release. Considering the poorly differentiated character, the incomplete surgery and the metastatic status, thus the initial therapeutic decision was to perform external radiotherapy of the neck and thyroid bed. Depending on the prognostic orientation, the irradiation dose planning to be delivered then changed according to prognostic orientation, presence of other localizations (to be irradiated during the same session). The study case was discussed in a multidisciplinary consultation committee. The radioactive iodine scan was a good mapping alternative lesion,

but the poorly differentiated tumor character was not in favor. We asked for an immunohistochemical complementary study, which objectified positive rate of ATAb. An ablative cure with 100 mCi of Radioiodine 131 was scheduled for this patient. This Radioactive iodine therapy does not have a main curative goal but rather to make a mapping of possible secondary locations avid for Iodine 131 and a prognostic classification. The post-therapeutic Wole-body scan showed multifocal hotspots at the cervical level and lung bilaterally. No bone fixation was objectified or any other new localization (Figure 1).

Figure 1: Whole body scan performed 3 days after oral administration of ablative activity of 100 mCi of Radiolodine (131I): Hot spots are objectified in cervical area and both lungs (small arrows). No bone focis.

External contamination visible on the upper part of the head related to a physiological secretion in the sweat.

As the patient was non-dyspneic and non-symptomatic, we decided to cancel he subsequent possibility of immunotherapy treatment, to postpone external radiation as second line-weapon treatment and to only continue Radio-iodine ablative cures.

Discussion

The association of diabetes with cancer takes several forms. The type 1 diabetes (T1D) is seen in endocrine cancer predisposition syndromes (hereditary paraganglioma, Multiple Endocrine Neoplasia Type 1, Multiple Endocrine Neoplasia Type 2). The type 2 diabetes (T2D) is associated with all cancers, whether they are hormone-dependent or not. Cancers are more common in diabetics than in the general population. More specifically, type 2 diabetes has been identified as a risk factor for several hormone-dependent cancers. There is a robust association between T2D and the risk of colorectal cancer or hepatocellular carcinoma. There is also a significant association between T2D and pancreas cancer, gallbladder or cholangiocarcinoma. The association with gynecological cancers is more inconsistent. The most established causative mechanisms are visceral obesity, hyperinsulinism, insulin resistance and hyperglycemia [2]. Several studies have demonstrated the link between the presence of diabetes and the secondary appearance of thyroid cancer. Follicular strain familial thyroid cancers are rare and include thyroid tumors associated with syndromes, and non-syndromic cancers [3]. In familial adenomatous polyposis, the prevalence of thyroid cancer is 2-12% (papillary carcinoma, cribriform and morular variant in 20-40% of cases). Cowden syndroma mainly comprises multiple hamartomas and 2/3 of patients develop a thyroid pathology (cancer in 35% of cases, one of the major diagnostic criteria of the syndrome). In Werner's syndroma, thyroid cancer is present in 18% of cases. For non-syndromic thyroid cancers, which represent the majority of familial cancers, they are most often papillary carcinomas [3].

The histologic features and clinical outcome of PDTC are intermediate between those of differentiated follicular-cell-derived thyroid carcinomas and anaplastic thyroid carcinomas ATC [1]. PDTC accounts for approximately 2% of thyroid malignancies in the United States [1,4]. The average patient age is 55 to 63 years, with a slight female predominance. Distant metastases are

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common, and the 5-year and 10-year survivals are approximately 70% and 50%, respectively [4-6]. The 2017 Endocrine World Health Organization (WHO) defines PDTC diagnosis on the five Turin criteria including conventional characteristics of malignancy (capsular or vascular invasion) with solid, insular, or trabecular growth pattern an absence of papillary nuclear abnormality and at least one of the following: convoluted nuclei, a mitotic count of 3 per 10 high-power fields (HPFs), or tumor necrosis [1,6]. In some cases, proliferative activity may not be uniform throughout the tumor and the diagnosis of PDTC may be missed if increased mitotic activity or focal necrosis is not appreciated. Immunohistochemical study of Anti-thyroglobulin antibodies (ATAb) on surgical specimen or from biopsies can correct the diagnosis. This is an indisputable contribution to improving the prognosis of PDTC and leading to the best treatment.

The coexistence of diabetes and a poorly or undifferentiated cancer is not without consequences on the prognosis of the two pathologies in a reciprocal way. The excess mortality attributed to the presence of diabetes was estimated at 25% [2]. Various cancer treatments can lead to a disruption of glycemic balance justifying specific multidisciplinary management to improve the prognosis. Targeted therapy with tyrosine kinase inhibitors is not without consequences on the metabolic balance of diabetes, due to its acute and unpredictable nature. Cases of fulminant diabetes linked to rapid, almost total and irreversible cell destruction have been reported in patients receiving anti-PD1 immunotherapy [2]. Radioactive-Iodine therapy has its place in the therapeutic arsenal of PCTD, depending on the degree of differentiation.

Summary

Various cancer treatments can lead to a disruption of glycemic balance in diabetic patients justifying specific multidisciplinary management to improve the prognosis. Immunohistochemistry retains an essential role in confirming the follicular nature of malignant thyroid cells and could modify the management and prognosis of this disease and make it possible to reduce clinical relapses and improve survival. Every effort should be made to prove this sensitivity by studying its immunohistochemical profile. This sensitivity to Iodine 131 is not permanent. Radioactive iodine should always be tried.

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