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Papillary Thyroid Carcinoma Varieties: Multiple Views of the Same Tumor

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Abstract

A group of cancers with very different phenotypes make up thyroid carcinoma. The identification of genetic, epigenetic and non-genetic factors that contribute to the heterogeneity of these cancers has been advancing as a result of recent advancements in biological technologies. In this review article, we talk about new findings that are making it easier to find new therapeutic targets and expanding our knowledge of the biology of thyroid cancer. The underlying biology and phenotypic characteristics of various thyroid cancer subtypes are examined. We focus on genetic and epigenetic factors, characteristics of cancer stemness and tumor microenvironments in our discussion of recent findings regarding the heterogeneity of thyroid cancer and the crucial mechanisms that contribute to the heterogeneity.

Keywords: Thyroid carcinoma • Heterogeneity • Cancer stem cells

Introduction

There are various definitions of the term in the literature: Thyroid Carcinoma Occult". According to the most recent online version of the Merriam-Webster dictionary, "occult carcinoma" means "not manifest or detectable by clinical methods alone" as well as "not present in macroscopic amounts." The phrase "occult primary malignancy" is defined as "unknown primary malignancy that is symptomless, which first manifests itself as metastases or secondary paraneoplastic phenomena" in the 2002 edition of the McGraw-Hill Concise Dictionary of Modern Medicine. "Occult thyroid carcinoma" was defined by Moosa and Mazzaferri in 1997 as "impalpable thyroid carcinoma that is generally smaller than 1.0 cm [1,2].

Discussion

There was no evidence of lymphadenopathy in the postoperative findings, which pointed to a left thyroid nodule. There were no signs of local invasion and the left lobe of the thyroid measured 4-32.5 cm and weighed 14 grams. A follicular adenoma was found in the initial histopathology, but a second histological examination of the thyroid revealed a circumscribed, differentiated neoplasm of thyroid follicular cell origin with tightly packed follicles, some of which were larger and had blunt papillae intraluminally.

According to clinical studies, consuming a lot of iodine seemed to be a big risk factor for the BRAF mutation, which only happened in PTC and was the most important factor in the beginning of PTC among genetic changes. The high iodine intake-induced BRAF mutation that may be a risk factor for the development of PT may be the cause of the increased incidence of PTC. By interfering with oncogene expression or the mutation of tumor suppressor genes like BRAF, ERK, RAS and p53, it has also been suggested that iodine can stop differentiated thyroid carcinoma from progressing into anaplastic carcinoma. Ultrasound imaging revealed a hyperechoic and hypervascular nodule that measured 4 cm by 2.5 cm in the left lobe of the thyroid.

The introduction of iodine prophylaxis and the results of animal experiments and epidemiological studies all point to a link between iodine intake and the types of thyroid carcinoma. Our findings demonstrated a shift in the distribution of its primary histological subtypes of TC, particularly the ratio of PTC to FTC following USI, in line with previous research. According to animal studies, there are a variety of ways in which iodine deficiency and excess can cause thyroid follicular cell carcinomas. In the iodine-deficient animals, there was a clear increase in the incidence of thyroid epithelial cell carcinomas following prolonged iodine deficiency, which was caused by thyrotropin and possibly other growth factors. The high iodine intake-induced BRAF mutation that may be a risk factor for the development of PT may be the cause of the increased incidence of PTC. By interfering with oncogene expression or the mutation of tumor suppressor genes like BRAF, ERK, RAS and p53, it has also been suggested that iodine can stop differentiated thyroid carcinoma from progressing into anaplastic carcinoma. Ultrasound imaging revealed a hyperechoic and hypervascular nodule that measured 4 cm by 2.5 cm in the left lobe of the thyroid. There was no pathological lymphadenopathy in the neck. A fine needle aspiration was carried out due to the mass's size. A group of epithelial cells was described by cytological analysis as having prominent nuclear overlapping, mild nuclear enlargement and microfollicular structures. Although it was not diagnostic

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These cancers probably existed in these people's thyroid glands for some time but never reached a clinically significant level. The question of whether all thyroid cancers should be diagnosed and treated is a contentious one given these findings. According to the results of the US autopsy study, more than 38 million people unknowingly have papillary carcinoma. Occult thyroid cancer autopsies are likely to decrease in the years to come if these

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subclinical cancers are overdiagnosed and treated excessively. It is currently up for debate whether or not all thyroid carcinomas require treatment. The highest incidence occurring in the second, third and fourth decades of life.8 However, the incidence of thyroid cancer in the fourth and fifth decades of life has increased in the past two decades. The occurrence of tumors on other imaging studies, such as ultrasound, computed tomography (CT), magnetic resonance imaging (MRI) and positron emission tomography (PET), may be the cause of the increased diagnoses [3-5].

Conclusion

Further analyses focused on 39,706 PTCs that were diagnosed during the time when women's PTC rates increased the fastest. PTC incidence rates increased nearly 100% among White non-Hispanic women and Black males between 1992 and 1995, but only 20% to 50% among White Hispanics, Asian/Pacific Islanders and Black males between 2003 and 2005 The cytology of n°1 revealed cellular atypia, while the cytology of n°2 revealed a Hürthle cell nodule, which was suggestive of a follicular neoplasm. As a result, the patient was referred for right lobectomy and isthmusectomy surgery.

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Conflict of Interest

There are no conflicts of interest by author.

References

1. Curiel, Tyler J., Pui Cheng, Peter Mottram and Xavier Alvarez, et al. "Dendritic cell subsets differentially regulate angiogenesis in human ovarian cancer." *Cancer Research* 64 (2004): 5535-5538.
2. Marie, Caroline, Nicolas A. Giraldo, Hélène Kaplon and Claire Germain, et al. "Tertiary lymphoid structures, drivers of the anti-tumor responses in human cancers." *Immunological Reviews* 271 (2016): 260-275.
3. Gordon-Alonso, Monica, Thibault Hirsch, Claude Wildmann and Pierre van der Bruggen, et al. "Galectin-3 captures interferon-gamma in the tumor matrix reducing chemokine gradient production and T-cell tumor infiltration." *Nature Communications* 8 (2017): 1-15.
4. Ali, H. Raza, Leon Chlon, Paul DP Pharoah and Florian Markowetz, et al. "Patterns of immune infiltration in breast cancer and their clinical implications: a gene-expression-based retrospective study." *PLoS Medicine* 13 (2016): e1002194.
5. Li, Taiwen, Jingyu Fan, Binbin Wang and Nicole Traugh, et al. "TIMER: a web server for comprehensive analysis of tumor-infiltrating immune cells." *Cancer Research* 77 (2017): e108-e110.

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