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From Molecular Background to Therapy Approach: Bronchial Carcinoids

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Introduction

The group of tumours that make up neuroendocrine tumours (NETs) of the lung is heterogeneous, ranging from well-differentiated bronchial carcinoids (BCs) to incredibly aggressive small cell lung cancers (SCLCs) and large cellphone neuroendocrine carcinoma (LCNEC). On the one hand, neuroendocrine carcinomas (NECs), such as SCLC and LCNEC, are clinicopathologically distinct entities compared to BCs since they manifest more frequently in patients who have a history of smoking and develop extremely suddenly. On the other hand, BCs are rare, frequently slowly progressing neuroendocrine epithelial tumours that frequently affect people who have never smoked and account for less than 2% of all lung cancer cases [1].

But over the past 30 years, the prevalence of BCs has increased at a rate of about 6% annually. Normal carcinoids (TCs) and atypical carcinoids (ACs), the latter of which are extremely rare (approximately 0.2%), are two categories into which BCs can be separated. While ACs are more aggressive tumours and are more likely to metastasize to other tissues, TCs are slow-growing tumours that seldom spread outside of the lungs. Up to 5% of patients with multiple endocrine neoplasia type 1 (MEN1) cases—the majority of which are sporadic—have BCs, typically TCs. Additionally, they could develop in the vicinity of a rare form of hereditary disease called a familial lung carcinoid tumour [2].

Description

It is acknowledged that NECs and BCs are distinct molecular entities since information from gene expression profiling and comparative genomic hybridization shows that BCs and NECs are clustered separately. Chromosomal abnormalities are more common in NECs than BCs in this regard, with the exception of the deletion of 11q, which affects the full range of lung NETs. Additionally, various molecular and genetic alterations in BCs have been linked to survival rates and likely the response to treatment. In general, understanding the molecular past of BC is important for understanding the prognosis and for guiding medicinal decisions in metastatic contexts. Most BCs can be cured surgically when it comes to treatment [3].

The surgical plan is determined by the tissue type, size, and placement. Conservative resection, such as sleeve resection, segmentectomy, or wedge resection, is the preferred treatment for TCs with central localization, whereas anatomic resection is frequently needed for ACs, particularly those with peripheral localization. Bronchoscopic excision may also be a treatment option in cases of intraluminal carcinoids that are centrally located. However, systemic treatment approaches must be taken into account in superior BCs. Somatostatin analogues (SSAs) are frequently used as the first-line treatment for advanced BCs and to manage the symptoms of carcinoid disease. Peptide receptor radionuclide therapy (PRRT) is a treatment option for selected patients with advanced and/or metastatic BCs that are SSTR-great at imaging and exhibit disease progression while receiving SSAs [4].

Given their modest proliferative potential, standard chemotherapy regimens frequently have little success in BCs. According to a section III scientific trial in non-functioning extra-pancreatic NETs, targeted therapy and everolimus are the only medications approved in BC. Although promising, the effectiveness of several treatments such as immunotherapy and antiangiogenic drugs must be considered experimental. In this paper, we provide a thorough analysis of the molecular and genetic background of TCs and ACs, concentrating on the differences between them and NEC, as well as practical prognostic and predictive biomarkers. In addition, a summary of the treatment of local and metastatic disease is included, as well as information on the key paraneoplastic syndromes associated with BCs, such as the carcinoid syndrome, the ectopic Cushing syndrome (ECS), and the syndrome of inappropriate antidiuretic hormone (SIADH) [5].

Conclusion

A better understanding of the genetic and molecular background of BCs would enable a better assessment of the risk of disease progression and the personalization of treatment in cases of advanced disease. Reduced gene expression and MEN1 gene mutations and deletions have been linked to a poor prognosis in BCs. The PI3K/AKT/mTOR pathway represents a mutation that can be targeted with kinase inhibitors, and the ATRX mutation has additionally been associated to a shorter disease-specific survival. Localized BC usually heals after surgical resection. There are specific systemic therapeutic options for advanced BCs, including SSAs, PRRT, chemotherapy, radiation, everolimus, antiangiogenic drugs, and immunotherapy. In slowly developing excellent SSTR lung NETs, SSAs are frequently the first-line treatment because they can trigger stabilisation in 30–70% of patients. For patients with progressing, disseminated illness, including SSTR tumours for PRRT treatment or following SSAs, Everolimus and PRRT are effective solutions. Systemic treatment with cytotoxic chemotherapy must be evaluated as refractory to other high-quality medicines such SSAs, everolimus, or PRRT, or as a startling modern illness. Due to the lack of viable clinical trials to assess novel techniques, the treatment sequence is no longer fully stable. A crucial component of this setting is the patient decision, which enables patients to receive personalised care.

Conflict of Interest

None.

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