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Molecular Characterization of Bronchial Cancer and its Therapy

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Introduction

Bronchial cancer remains one of the leading causes of cancer-related mortality worldwide. It is a heterogeneous disease with various histopathological and molecular subtypes, each having distinct clinical and biological characteristics. Understanding these subtypes is crucial for accurate diagnosis, effective treatment and the development of targeted therapies. This article provides a comprehensive overview of the histopathological and molecular profiling of bronchial cancer subtypes, focusing on their diagnostic features, molecular alterations and clinical implications.

Description

Bronchial cancer is broadly classified into two major categories: Non-Small Cell Bronchial Cancer (NSCBC) and Small Cell Bronchial Cancer (SCBC).

Non-Small Cell Bronchial cancer (NSCBC)

NSCBC accounts for approximately 85% of all bronchial cancer cases and is further divided into three main subtypes:

Adenocarcinoma, this is the most common subtype, representing about 40% of bronchial cancers. It arises from the glandular cells of the lung and is often found in the outer regions of the lung. Histologically, adenocarcinomas can be identified by glandular formation and cumin production. Subtypes include cigar, papillary, micro papillary and solid adenocarcinomas. Squamous Cell Carcinoma (SCC), accounting for about 25-30% of bronchial cancers, SCC arises from the squamous cells lining the airways. It typically occurs in the central part of the lungs. Histopathological features include keratinization and intercellular bridges. SCC is associated with smoking and tends to present with necrosis and cavitation. Large Cell Carcinoma, this subtype constitutes about 10-15% of bronchial cancers and is characterized by large, undifferentiated cells. It can occur in any part of the lung and lacks the glandular or squamous differentiation seen in adenocarcinoma and SCC.

SCLC accounts for approximately 15% of bronchial cancers and is strongly associated with smoking. It is an aggressive form of bronchial cancer, often presenting with rapid growth and early metastasis. Histologically, SCLC is characterized by small, round to oval cells with scant cytoplasm, finely granular chromatin and a high mitotic rate. It often exhibits neuroendocrine features and expresses markers such as synaptophysin, chromogranin and CD56 [1-3]. Molecular profiling has revolutionized the understanding and treatment of bronchial cancer by identifying specific genetic alterations that drive tumour genesis. These alterations serve as targets for personalized therapies and include mutations, gene rearrangements and amplifications.

Molecular alterations in NSCLC

Epidermal Growth Factor Receptor (EGFR) mutations are prevalent in approximately 10-15% of NSCLC cases, particularly in adenocarcinomas and non-smokers. Common mutations occur in exons 18-21, leading to constitutive activation of the EGFR pathway. Targeted therapies, such as Tyrosine Kinase Inhibitors (TKIs), have shown significant efficacy in patients with EGFR-mutant NSCLC. KRAS mutations are found in about 25-30% of NSCLC cases, primarily in adenocarcinomas and smokers. These mutations lead to the activation of the RAS/MAPK pathway. Unlike EGFR, targeted therapies for KRAS-mutant NSCLC have been challenging, but recent developments, such as KRAS G12C inhibitors, offer new treatment options. Anaplastic Lymphoma Kinase (ALK) rearrangements occur in approximately 3-5% of NSCLC cases. The EML4-ALK fusion gene results in constitutive kinase activity. ALK inhibitors, such as crizotinib and alectinib, have demonstrated substantial clinical benefits in ALK-positive NSCLC patients. ROS1 gene rearrangements are present in about 1-2% of NSCLC cases. Similar to ALK, ROS1 fusions lead to constitutive kinase activation. ROS1 inhibitors, such as crizotinib, are effective in treating ROS1-positive NSCLC. BRAF mutations, particularly V600E, are found in about 1-2% of NSCLC cases. These mutations activate the MAPK pathway. BRAF inhibitors, either alone or in combination with MEK inhibitors, provide therapeutic options for BRAF-mutant NSCLC. MET exon 14 skipping mutations occur in approximately 3-4% of NSCLC cases. These mutations lead to the activation of the MET pathway. MET inhibitors, such as capmatinib and tepotinib, are effective in patients with MET exon 14 alterations.

Molecular alterations in SCLC

SCLC is characterized by frequent alterations in tumour suppressor genes, such as TP53 and RB1. TP53 mutations are present in nearly all SCLC cases, leading to the loss of p53 tumour suppressor function. This contributes to the high genomic instability and aggressive nature of SCLC.

1. **Rb1 mutations:** RB1 mutations occur in almost all SCLC cases, resulting in the loss of retinoblastoma protein function. This promotes uncontrolled cell cycle progression and tumour growth.
2. **MYC amplifications:** MYC gene amplifications are found in a subset of SCLC cases and are associated with aggressive tumour behaviour and poor prognosis.

Clinical implications

The integration of histopathological and molecular profiling into clinical practice has significant implications for the management of bronchial cancer: The identification of specific molecular alterations has enabled the development of targeted therapies, improving treatment outcomes and reducing toxicity. Examples include EGFR TKIs, ALK inhibitors and ROS1 inhibitors. Molecular profiling allows for the customization of treatment plans based on the individual genetic makeup of the tumour. This approach maximizes therapeutic efficacy and minimizes adverse effects. Certain molecular alterations serve as prognostic biomarkers, providing information on disease progression and survival outcomes. For instance, EGFR mutations are associated with better response rates to TKIs. Molecular alterations can predict the likelihood of response to specific therapies. For example, ALK rearrangements predict responsiveness to ALK inhibitors. Molecular profiling can aid in the early detection and screening of bronchial cancer, particularly in high-risk populations. Liquid biopsies, which analyse circulating tumour DNA, offer a non-invasive method for monitoring molecular changes. The integration of histopathological and molecular profiling is essential for accurate diagnosis and personalized treatment. Histopathological examination provides initial insights into the subtype of bronchial cancer, guiding subsequent molecular

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testing. Molecular profiling then identifies specific genetic alterations, enabling tailored therapeutic approaches. This combined approach enhances the precision of bronchial cancer management, leading to better prognostic outcomes and improved survival rates [4,5].

Conclusion

Histopathological and molecular profiling has transformed the landscape of bronchial cancer diagnosis and treatment. Understanding the distinct histopathological features and molecular alterations of bronchial cancer subtypes is essential for accurate diagnosis, effective treatment and the development of targeted therapies. Continued research and advancements in molecular profiling will further enhance the precision and personalization of bronchial cancer management, ultimately improving patient outcomes.

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Conflict of Interest

None.

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