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OPINION ARTICLE

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The Pathology of Extrapulmonary Small Cell Carcinoma

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Description

Extrapulmonary Small Cell Carcinomas (EPSCCs) are a rare malignant tumour. They have been discovered in almost every organ system since their initial description in 1930. The same multipotent stem cell is assumed to have given rise to EPSCCs as well as their more prevalent pulmonary cousins. Small cell elements, however, may develop as a late-stage phenomena in the genetic evolution of more organ-typical carcinomas, according to current molecular findings. Similar morphologic, immunohistochemical, and ultrastructural characteristics to Pulmonary Small Cell Carcinomas have been reported (PSCCs). PSCC, other neuroendocrine tumours, tiny round blue cell tumours, metastatic melanoma, lymphoma, and poorly differentiated non-small cell carcinomas are included in the differential diagnosis of EPSCC. Abnormalities documented in PSCC and modifications found in carcinomas more frequently found in the organ from where they originate are among the molecular changes known to occur in EPSCCs. In this article, we explore theories of histogenesis, sites of occurrence, diagnostic characteristics, differential diagnoses, molecular abnormalities, and clinical behaviour in order to discuss the pathogenesis of EPSCC.

Histogenesis

Similar to how the Kultchisky cell was formerly thought to be the source of Pulmonary Small Cell Carcinomas (PSCCs), 5 Amine-Precursor Uptake and Decarboxylase (APUD) cells identified by Pearse in 1969 were thought to be the source of EPSCCs. It was claimed that these cells were part of a widespread neuroendocrine system and shared ultrastructural characteristics with numerous small cell carcinomas. As a result, the APUD system offered EPSCC a tempting putative cell of origin. But this theory doesn't really give anything.

Diagnosis

EPSCC has the same shape as its pulmonary equivalent. Although irregular organioid clusters, trabeculae, and rosette-like patterns may exist, the architecture is typically solid. The cells have sparse cytoplasm, inconspicuous nucleoli, and finely distributed chromatin, and are typically two to three times the diameter of mature lymphocytes. Often, nuclei will align themselves around the nuclei of neighbouring cells (nuclear molding). Necrosis and mitotic activity are typically noticeable.

Multiple diagnoses

There are many possible diagnoses for EPSCC. It comprises poorly differentiated carcinoma, metastatic PSCC, other neuroendocrine tumours, numerous tiny round blue cell tumours, metastatic melanoma, and lymphoma. A normal chest CT scan, normal plain radiograph, normal sputum cytology, or a negative bronchoscopy are required to rule out metastatic PSCC, which has morphology identical to that of EPSCC.

The majority of knowledge about the molecular changes in EPSCC comes from individual case reports and short series. Despite the paucity of available data, EPSCCs often seem to share some molecular abnormalities with both PSCC and carcinomas more frequently found in the affected organ. The loss of genetic material from the short arm of a chromosome is among the most often observed chromosomal abnormalities in PSCC. With an incidence of 0.1% to 0.4%, or roughly 1,000 new cases diagnosed each year in the United States, EPSCC is a rare condition. Patients must, by definition, have a small cell carcinoma on histology, normal chest plain radiographs and computed tomography scans, normal sputum cytology, or negative bronchoscopy results. Patients of middle age or older are most affected by EPSCCs, with more than 70% of patients being older than 50.

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A rare form of cancer known as an EPSCC is hypothesised to develop from either a multipotent stem cell or as a late-stage phenotype in the genetic course of a more organ-typical carcinoma. They are aggressive tumours that frequently spread at presentation. In terms of morphology, immunohistochemistry, and ultrastructure, they are identical to their more prevalent pulmonary counterparts.