Commentary

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Tumor-Stroma Interactions in Head and Neck Squamous Cell Carcinoma

Chen Xen*

Department of Oncology, University of Science and Technology, Kunming, China

Corresponding author: Chen Xen, Department of Oncology, University of Science and Technology, Kunming, China, E-mail: Xen_C@gmail.com

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Description

Head and Neck Squamous Cell Carcinoma (HNSCC) manifests as a formidable cancer often accompanied by heightened Lymph Node Metastasis (LNM) upon diagnosis. Tumor budding, defined by the emergence of isolated cells or small clusters of tumor cells (clusters containing fewer than five malignant cells) at the invasive edge, along with the composition of the fibrotic cancer stroma, has emerged as a significant factor influencing the dynamics of solid tumours. Despite this recognition, the precise contribution of these elements remains undefined, necessitating the establishment of a standardized scoring system for comprehensive evaluation. The journey of understanding LNMets has unfolded across five interwoven phases of exploration: Initial clinical, radiological and pathological observations; the evolution of surgical interventions for treatment; the correlation of LNMets with patient survival rates, aiding prognostication; an ongoing investigation into the mechanisms underlying tumor dissemination to lymph nodes; and the refinement of treatment strategies for LNMets.

Lymph nodes

This trajectory of discovery has gained momentum with the collaborative efforts of scientists and clinicians, who continually contribute novel insights and advancements in both fundamental and applied research concerning lymphatics and lymph nodes. This synergy between bedside challenges and interdisciplinary collaboration exemplifies how clinical issues can be tackled through the interconnectedness of medical practitioners and researchers. While substantial progress has been made, there remains a wealth of knowledge yet to be uncovered. As our understanding of LNMets deepens, so too will our ability to optimize patient management strategies in the future. Lymphatic spread stands out as a critical clinicopathological feature of Colorectal Cancer (CRC), ultimately culminating in patient mortality. On a molecular level, the tumor microenvironment in the colon experiences a dysregulation between oxygen availability and consumption, leading to hypoxia, a significant independent prognostic factor in CRC and nearly all solid tumors.

In this outline hypoxia-sensitive molecular mechanisms that drive lymphatic metastasis in CRC, highlighting similarities between vascular and lymphatic processes, while also delineating lymphatic-specific pathways activated downstream of hypoxic signaling. Additionally, we explore potential therapeutic strategies aimed at mitigating intrametastatic hypoxic signaling to enhance the prognosis of CRC patients. Around half of childhood malignancies are attributed to solid tumors. Among the most prevalent are brain tumors, lymphoma, neuroblastoma, osteosarcoma and rhabdomyosarcoma. Given that chemotherapy and radiation, common treatments for these malignancies, can affect bone marrow function, patients may experience complications akin to those seen in acute leukemia cases. Notably, bleeding tendencies and susceptibility to infections are prominent medical concerns. Overall, dental management for patients with solid tumors closely mirrors that of those with acute leukemia.

Squamous cell carcinoma

The primary environmental factors contributing to the development of Head and Neck Squamous Cell Carcinoma (HNSCC) include alcohol and tobacco use, immunosuppression and exposure to high-risk strains of Human Papillomavirus (HPV). However, it's important to note that not all individuals who use alcohol or tobacco, or who are exposed to HPV or EBV, develop cancer. For instance, only a small percentage of smokers develop lung cancer, and an even smaller fraction are diagnosed with HNSCC. This suggests that HNSCC is influenced by multiple factors, including genetic predisposition and environmental elements, which collectively contribute to tumorigenesis. Head and Neck Squamous Cell Carcinoma (HNSCC) is a challenging malignancy known for its aggressiveness, frequent recurrence and poor prognosis. EGFR, a key player in HNSCC, has been closely associated with the disease, with cetuximab being the pioneer anti-EGFR antibody sanctioned for HNSCC treatment.

However, despite the initial success of cetuximab, many patients eventually experience relapse due to the development of acquired resistance. Additionally, the signaling pathway is commonly activated in HNSCC and has been identified as a contributor to tumorigenesis and resistance to treatment, often intertwining with EGFR signaling. In a recent study outlined, researchers observed that prolonged exposure to EGFR inhibition led to the upregulation of the transcription factor GLI1

in HNSCC cells, coinciding with the emergence of resistance to cetuximab. Conversely, when inhibited, HNSCC cells became more reliant on EGFR, which consequently hindered or delayed tumor recurrence when combined with cetuximab. Furthermore, the combination of cetuximab and the inhibitor demonstrated superior anti-tumor efficacy compared to cetuximab alone, as evidenced by experiments conducted on patient-derived xenografts of HNSCC.