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Opinion Article

Significance in Tumor Metastasis: A Brief Note

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DESCRIPTION

Tumor metastasis is accountable for 90% of cancer-related mortality but is still one of the most poorly understood components of cancer pathogenesis. The majority of solid tumors are carcinomas, cancers that originate in the epithelial cells. Successful invasion-metastasis cascades need several steps that include Epithelial Mesenchymal Transition (EMT) of cancer cells, invasion through the Extra Cellular Matrix (ECM) and stromal cell layers, intravasation into the vasculature lumina, conveyance through the circulatory system, extravasation into parenchyma of aloof tissues and organs, seeding at the premetastatic niche, and finally existence and growth at the metastatic site. Multistep metastatic process in two stages, in the first stage, cancer cells translocate physically from the primary tumor to the site of delivery; and in the second stage, colonization happens at the secondary site. As simple as these courses may sound, the clinical impact of these changes is immense. Many of the events complicated in these stages are the result of reciprocal and developing crosstalk between the tumor microenvironment and carcinoma cells. The EMT package plays an important role in tumor metastasis by disassembling adherens and tight connections, transforming polarized epithelial cancer cells into a mesenchymal cell phenotype, and facilitating the detachment of mesenchymal cells from initial sites to let passage through dismantled Basement Membranes (BMs). Once they have touched the distant organs, these mesenchymal cells may return to an epithelial phenotype through a Mesenchymal Epithelial Transition (MET) and thereby recover the ability of cancer cell proliferation and difference in metastatic sites.

These inflammatory cells play an important role in tumor progression by promoting tumor cell proliferation, matrix remodeling, angiogenesis, suppressed adaptive immunity, and EMT. Classically, they do this by creating various cytokines such as the interleukins, interferon and other tumor-promoting factors. Recent has now established that EMT-mediated premetastatic tumor cells are also involved in the recruitment, activation, and differentiation of TAMs. The important concept of EMT inspiring cancer development and progression is well accepted, but the links between EMT and TAMs in these developments present a gap. For example, although the tumor inflammatory cell populations seem to promote growth and progression of tumors, the exact interactive pathways are not well distinct.

By defining the roles of EMT and TAMs in cancer development, therapeutic strategies that target these processes may be developed to advantage cancer patients. The purpose of this is to deliver an overview of experimental and clinical evidence that validates the crosstalk between TAMs and cancer cells undergoing EMT during metastasis and develop an understanding of the translational significance of this information, for development of new diagnostic and therapeutic strategies targeting TAMs and/or EMT.

CONCLUSION

A major complication of cancer development is the metastatic feast of cancer cells from the primary tumor. Similarities between the process of EMT in embryogenesis and wound healing and that seen in feast of epithelial-derived cancers are now becoming clear. There is cumulative evidence that the tumor metastatic cascade relies on a complicated communication between EMT-modified cancer cells and TAMs. Cytokines released by tumor cells promote the recruit of macrophages to the tumor site and distinguish them into TAMs that then become active in the tumor microenvironment. Reciprocally, as a major component of solid tumors, TAMs promote cancer cell attack and metastasis by secreting various cytokines, for example, TGF-β, NF-κB, VEGF, and CCL18. Therefore, a positive feedback circuit forms between TAMs and EMT-modified cancer cells in the tumor microenvironment. Despite some remaining gaps in knowledge concerning links between EMT, TAMs, and cancer invasiveness, it seems that targeting transcriptional factors and the signaling pathways between TAMs and EMT can break the cycle of crosstalk to combat metastasis. Though most evidence detailed here was from experimental models, some results connecting patient-derived cancers support the preclinical experimental data. With further elucidation about the mechanism of tumor metastasis, it may be soon conceivable to translate these fundamental research detections successfully into clinic practice.

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