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Deciphering the Omental Microenvironment: Fibroblast-Mediated Enhancement of Ovarian Cancer Aggressiveness

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Abstract

Ovarian cancer remains a formidable challenge in oncology, characterized by its aggressive nature and propensity for metastasis. The omental microenvironment, enriched with fibroblasts, emerges as a critical determinant of tumor progression in ovarian cancer. Fibroblasts, traditionally recognized for their structural role, undergo phenotypic alterations in response to tumor-derived cues, transforming into cancer-associated fibroblasts (CAFs). In this review, we delineate the multifaceted roles of CAFs in promoting ovarian cancer aggressiveness. Activated CAFs remodel the extracellular matrix (ECM), facilitating cancer cell invasion and metastasis through ECM degradation and deposition of pro-migratory proteins. Additionally, CAFs secrete a plethora of growth factors, cytokines, and chemokines, fostering a pro-tumorigenic microenvironment that sustains cancer cell proliferation, survival, and epithelial-to-mesenchymal transition (EMT). Moreover, CAFs contribute to immune evasion by recruiting immunosuppressive cells to the tumor milieu. Targeting the interactions between fibroblasts and cancer cells represents a promising therapeutic strategy in ovarian cancer. Disrupting fibroblast activation or inhibiting key signaling pathways implicated in fibroblast-mediated tumor progression holds therapeutic potential. Combination therapies that concurrently target cancer cells and stromal components may offer synergistic benefits. This review underscores the pivotal role of fibroblasts in driving ovarian cancer aggressiveness and highlights the therapeutic opportunities afforded by targeting the tumor microenvironment.

Introduction

The omentum, a fatty tissue draped over the intestines like an apron, is increasingly recognized as a pivotal player in cancer metastasis, particularly in ovarian cancer. Within this microenvironment, fibroblasts, traditionally viewed as structural support cells, have emerged as key orchestrators of tumor progression. Understanding the interplay between omental fibroblasts and ovarian cancer cells sheds light on novel therapeutic strategies to combat this aggressive disease [1].

The omental microenvironment

The omentum serves as a dynamic reservoir of various cell types, extracellular matrix (ECM) proteins, and soluble factors, forming a complex microenvironment conducive to tumor growth and dissemination. Fibroblasts, the predominant stromal cells in the omentum, undergo phenotypic changes in response to signals from neighboring cancer cells, cytokines, and growth factors, transitioning into activated cancer-associated fibroblasts (CAFs).

Fibroblast-mediated enhancement of ovarian cancer aggressiveness

Activated CAFs exert multifaceted effects on ovarian cancer cells, promoting their aggressiveness through various mechanisms. Firstly, CAFs remodel the ECM, facilitating cancer cell migration and invasion. Increased deposition of ECM proteins, such as fibronectin and collagen, creates physical paths for cancer cell movement and enhances their ability to infiltrate surrounding tissues. Additionally, CAFs secrete matrix metalloproteinases (MMPs), enzymes that degrade ECM barriers, further facilitating tumor cell dissemination.

Moreover, CAFs actively communicate with cancer cells via paracrine signaling, fostering a pro-tumorigenic environment. Secretion of growth factors such as TGF-β, FGF, and HGF by CAFs promotes cancer cell proliferation, survival, and epithelial-to-mesenchymal transition (EMT), a process associated with increased metastatic potential and therapy resistance [2-5]. Furthermore, CAF-derived cytokines and chemokines recruit immune cells to the tumor microenvironment, creating an immunosuppressive niche that shields cancer cells from immune surveillance.

Therapeutic implications

Targeting the crosstalk between omental fibroblasts and ovarian cancer cells holds promise for therapeutic intervention. Strategies aimed at disrupting fibroblast activation or inhibiting key signaling pathways implicated in fibroblast-mediated tumor progression represent attractive therapeutic avenues. In preclinical studies, inhibition of CAF-derived factors, such as TGF-β or FGF signaling, has shown efficacy in attenuating tumor growth and metastasis in ovarian cancer models. Furthermore, combination therapies that simultaneously target cancer cells and stromal components may offer synergistic benefits. For instance, combining conventional chemotherapy with agents targeting CAFs or ECM remodeling enzymes could enhance treatment response and prevent the emergence of drug-resistant phenotypes.

Roles of activated fibroblasts in ovarian cancer cell adhesion and invasion

Ovarian cancer remains one of the deadliest gynecological malignancies, largely due to its propensity for metastasis and therapeutic

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resistance. The tumor microenvironment, comprising various stromal cell types, extracellular matrix (ECM) components, and soluble factors, plays a crucial role in driving disease progression. Among these stromal cells, activated fibroblasts have emerged as key orchestrators of ovarian cancer cell adhesion and invasion, contributing to metastatic spread and treatment challenges. This review explores the intricate interplay between activated fibroblasts and ovarian cancer cells, shedding light on their roles in promoting tumor aggressiveness.

Activation of fibroblasts in ovarian cancer

In response to signals from neighboring cancer cells, cytokines, and growth factors, quiescent fibroblasts within the tumor microenvironment undergo phenotypic and functional changes, transitioning into activated cancer-associated fibroblasts (CAFs) [6]. These activated fibroblasts exhibit a spectrum of alterations, including enhanced ECM remodeling capacity, increased secretion of growth factors and cytokines, and augmented contractility, all of which contribute to tumor progression.

Role of activated fibroblasts in ovarian cancer cell adhesion

Activated fibroblasts play a pivotal role in facilitating ovarian cancer cell adhesion to the ECM and neighboring cells. Through ECM remodeling, CAFs create a permissive niche for cancer cell attachment, promoting their retention within the primary tumor and facilitating subsequent invasion. Moreover, CAF-derived factors such as fibronectin and integrins contribute to the formation of focal adhesions, adhesive structures that mediate cancer cell-ECM interactions and signaling, thereby promoting tumor cell survival and growth.

Contribution of activated fibroblasts to ovarian cancer cell invasion

In addition to promoting adhesion, activated fibroblasts profoundly influence ovarian cancer cell invasion, a critical step in metastasis. By secreting matrix metalloproteinases (MMPs) and other proteases, CAFs facilitate ECM degradation, allowing cancer cells to breach physical barriers and invade surrounding tissues. Furthermore, CAF-derived soluble factors, including growth factors and chemokines, induce epithelial-to-mesenchymal transition (EMT) in cancer cells, a process associated with increased motility, invasiveness, and resistance to apoptosis.

Therapeutic implications

Targeting the interactions between activated fibroblasts and ovarian cancer cells represents a promising therapeutic strategy to impede tumor progression and metastasis. Strategies aimed at inhibiting fibroblast activation, disrupting ECM remodeling, or interfering with paracrine signaling pathways hold potential to attenuate tumor aggressiveness and improve patient outcomes. Furthermore, combination therapies that concurrently target cancer cells and the tumor microenvironment may offer synergistic benefits [7,8]. In conclusion, understanding the roles of activated fibroblasts in ovarian cancer cell adhesion and invasion provides insights into tumor biology and unveils therapeutic opportunities for combating this deadly disease.

Conclusion

The omental microenvironment plays a pivotal role in promoting ovarian cancer aggressiveness, with fibroblasts emerging as central mediators of this process. Elucidating the molecular mechanisms underlying fibroblast-cancer cell interactions provides valuable insights into tumor biology and unveils potential therapeutic targets. Future research efforts aimed at unraveling the complexities of the omental microenvironment and developing innovative treatment strategies hold promise for improving outcomes in ovarian cancer patients.

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