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Targeting Tumor Microenvironment and Metabolic Aberration Against TNBC

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Abstract

Triple Negative Breast Cancer (TNBC) is a highly heterogeneous and aggressive subtype of breast cancer. Tumor microenvironment has been identified as a major regulator of carcinogenesis and imparting drug resistance to the proliferating cancer cells. Microenvironment conditions has been identified to promote altered metabolism which is one of the major factors in contributing to drug resistance of TNBC. Thus, identification of specific metabolic inhibitor and use of combinational drug therapy may be proven as a mainstay of treatment for triple negative breast cancer. Oxidative stress caused by ROS a byproduct of metabolic processes is associated with tumorigenesis and metastasis of breast cancer. Generally high levels of ROS have been identified in TME which contributes in cancer progression and aggressiveness. Thus, early detection of ROS level may be clinically advantageous to prevent further progression and aggressiveness of TNBC.

Keywords: TNBC; Tumor microenvironment; Oncometabolites

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Introduction

Triple negative breast cancer and tumor microenvironment

Triple Negative Breast Cancer (TNBC) is a highly heterogeneous, aggressive and fatal subtype of breast cancer which is negative for estrogen receptors (ER-), progesterone receptors (PR-) and human epidermal growth factor receptor 2 (HER2-). The present communication highlights on the importance of drug synergism which implies targeted drug therapy as different molecular regulators [1]. Special emphasis has been given to oncometabolites and ROS as they are important part of tumor microenvironment which undergoes which greatly influence and promote cancer metastasis. Tumor microenvironment (TME) has been identified as a major regulator of carcinogenesis, thus exploring this complex mechanism may bring an insight on understanding tumorigenesis. Exploring TME and its specific biomarkers related to metabolic aberration can be used to predict the clinical outcome may be proven as a promising approach in treatment of TNBCs [2]. In addition to neoplastic cells, the breast microenvironment is rich in extracellular matrix (ECM), stromal cell, endothelial cells, immune cells, fibroblasts, and adipocytes. TMC requires soluble factors like PDGF, TGF-β, EGF for communication and interactions which increases the aggressiveness of the disease. Cancer-associated fibroblasts (CAFs) are predominated in cancer stroma and affect the tumor microenvironment such that they promote cancer initiation, angiogenesis, epithelial-mesenchymal transition (EMT) invasion and metastasis. In breast cancer, CAFs not only promote tumor progression, but also induce therapeutic drug resistances and best example can be given as paclitaxel. Collagen type I secreted by CAFs contributes to decreasing chemotherapeutic drug uptake in tumors and plays a significant role in regulating tumor sensitivity to a variety of chemotherapies [3]. Chemotherapy and radiation therapy induce DNA damage in fibroblasts which ultimately, promote secretion of WNT16B and consequently result in aggressiveness of the disease and induce mitoxantrone (MIT) resistance by NF-κβ pathway activation [4].

Stemness in tumor microenvironment of TNBC

TNBC stem cells exhibit unique abilities including self-renewal, differentiation potential, and drug resistance to chemo- and/or radiotherapy, which contributes to the development of aggressiveness and metastatic lesions [5]. Indeed, exposure various treatments promote “stemness” in non-stem cancer cells, which explain failure of clinical improvement. Acquisition of stemness involves epithelial-mesenchymal transition (EMT), in which epithelial cells are transformed into a mesenchymal phenotype characterized by increased capacities for migration, invasiveness, and resistance to apoptosis. Many approaches have

