Metabolic Reprogramming in Triple-Negative Breast Cancer: Covid 19

Paola M Brown*

Department of Pathology, Dalhousie University, Halifax, Canada

Corresponding Author*

Paola M Brown

Department of Pathology,

Dalhousie University,

Halifax, Canada

E-mail: brownM@gmail.com

Copyright: © 2021 Brown PM. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Received date: April 07, 2021**; Accepted date:** April 21, 2021**; Published date:** April 28, 2021

Description

The COVID-19 pandemic has necessitated the development of breast cancer treatment prioritisation policies, with patients with advanced disease, such as triple-negative breast cancer (TNBC), receiving top priority for clinical intervention. We summarise how COVID-19 has affected TNBC management so far and highlight areas where more data is required to fine-tune changing guidelines. TNBC management during the pandemic poses problems beyond the limits of overburdened healthcare systems, due to the immunocompromised state of most TNBC patients seeking treatment. During the COVID-19 outbreak, we performed a literature review of clinical recommendations for both primary and targeted TNBC therapeutic methods, noting improvements in treatment timing and drugs of choice [1]. Cancer cells are increasingly exhibiting distinct metabolic phenotypes to drive their proliferation and progression, a phenomenon known as metabolic reprogramming. New methods for overcoming malignant cancer, like triple-negative breast cancer, are now possible thanks to major advances in metabolic reprogramming. TNBC is linked to a high histologic score, violent phenotype, and a poor prognosis. Despite the fact that triple-negative breast cancer patients benefit from conventional chemotherapy, they also have a high risk of recurrence and are more likely to develop drug resistance. As a result, there is a pressing need to investigate the limitations of triple-negative breast cancer and create new therapeutic drugs to increase triple-negative breast cancer patients' clinical outcomes. Metabolic reprogramming may be a potential therapeutic target for triple-negative breast cancer treatment. We mainly address how triple-negative breast cancer cells and stromal cells in the microenvironment reprogram their metabolic phenotype to function in nutrient-poor conditions in this paper [2]. Given the fact that metastasis is a possibility, we also focus on the role of metabolic adaption in mediating metastasis and chemo resistance of triple-negative breast cancer tumors. And chemo resistance are the key contributors to mortality of triple-negative breast cancer patients, we also focus on the role of metabolic adaption in mediating metastasis and chemo resistance of triple-negative breast cancer tumors. TNBC is a heterogeneous group of breast cancers that lack expression of estrogen receptors, progesterone receptors, and amplification of the human epidermal growth factor receptor 2 gene, rendering it resistant to endocrine therapy and HER2-targeted care. TNBC makes up almost 15% of all invasive breast cancers, and it has the highest risk of metastatic spread and the worst overall survival of all breast tumor subtypes. TNBC makes up almost 15% of all invasive breast cancers, and it has the highest risk of metastatic spread and the worst overall survival of all breast tumor subtypes [3]. While chemotherapy improves TNBC patients' clinical outcomes, recurrence rates are still high, and TNBC tumors often develop resistance to chemotherapeutic agents. Given the restricted treatment options and aggressive phenotypes of TNBC, it's critical to enhance our understanding of the disease's characteristics and identify new therapeutic targets to help in the creation of successful treatments. The ability of cancer cells to receive nutrients from a nutrient-depleted environment and use these nutrients to maintain their transformed state, create biomass, and increase cell proliferation is a common feature. Studies started to concentrate on cancer-related metabolic reprogramming within critical metabolic pathways, such as altered glucose, lipid, and amino acid metabolism, to investigate possible metabolic vulnerabilities during cancer progression. The main molecular features of TNBC related to metabolic reprogramming are first introduced in this study. In addition, we summarize the possible metabolic targets and corresponding agents for TNBC treatment in the preclinical and clinical stages, as well as provide an overview of the main metabolically adapted pathways in TNBC tumors, mainly glucose, fatty acid, and amino acid metabolism, and then investigate potential therapeutic targets for metabolic vulnerabilities to direct TNBC therapy. We also discuss how metabolic adaptation in TNBC tumors affects the metastatic process and chemo resistance, as well as the metabolic relationship between TNBC tumors and their microenvironment. Over the last few decades, we've learned a lot more about how cancer cells change their metabolism. TNBC tumors' malignancy is unquestionably aided by an improved glycolytic phenotype. Nonetheless, unlike the standard Warburg effect, which shows decreased OXPHOS activity, TNBC tumors have been shown to have dual OXPHOS alterations, indicating the need to classify the oncogenes responsible for the increased OXPHOS activity. Even though fatty acid synthesis and FAO are thought to be opposites in the metabolic reprogramming of cancer cells, they can work in tandem to help TNBC cells advance. TNBC tumors have a glutamine-addicted phenotype, making glutamine-related enzymes possible therapeutic targets. Despite the fact that serine and glycine share the same synthetic pathway, serine metabolism is more important for TNBC cell growth. Since TNBC tumor cells must survive and proliferate in a new microenvironment, metabolic adaptation of TNBC tumor cells often entails interaction with adjacent stromal cells [4]. Despite the fact that serine and glycine share the same synthetic pathway, serine metabolism is more essential for TNBC cell growth than glycine metabolism. TNBC tumor cells must thrive and proliferate in a new microenvironment, so metabolic adaptation also necessitates contact with nearby stromal cells.

References

1. Papautsky, E., et al. “Patient-Reported Treatment Delays in Breast Cancer Care during the COVID-19 Pandemic.” Breast Cancer Res. 184.1(2020): 249-254.
2. Yin, K., et al. “Breast Imaging, Breast Surgery, and Cancer Genetics in the Age of COVID-19.” Cancer. 126.20(2020): 4466-4472.
3. Carey, L. A., et al. “Race, Breast Cancer Subtypes, and Survival in the Carolina Breast Cancer Study.” JAMA. 295(2006): 2492.
4. Elghazawy, H., et al., “Implementation of Breast Cancer Continuum of Care in Low- and Middle-Income Countries during the COVID-19 Pandemic.” Oncol. 10(2020): 0574.

Cite this article: Brown PM. Metabolic Reprogramming in Triple-Negative Breast Cancer: Covid 19. Eur J Clin Oncol, 2021, 3(S1), 001.