Brief Report
Volume 12:1, 2023

Journal of Integrative Oncology

ISSN: 2329-6771 Open Access

Metformin and Systemic Host Metabolism in Cancer Treatment: Effects on Mitochondrial Function and One-Carbon Metabolism for Enhanced Therapeutic Index

Vallabh Willer*

Department of Medicines, University of Barcelona, Barcelona, Spain

Introduction

Neuroendocrine neoplasms (NENs) or neuroendocrine tumors (NETs) are a rare type of cancer that arises from neuroendocrine cells, which are specialized cells that produce hormones and neurotransmitters. These tumors can originate in various organs, including the pancreas, lungs, gastrointestinal tract, and breast. NENs are divided into two subgroups: well-differentiated (low to intermediate grade) tumors, also known as carcinoid tumors, and poorly differentiated (high grade) neuroendocrine carcinomas. The diagnosis of NENs requires the expression of certain neuroendocrine markers, such as chromogranin-A, synaptophysin, or neuron-specific enolase, in at least 50% of malignant tumor cells. Neuroendocrine breast cancer (NEBC) is a rare form of breast cancer that accounts for less than 1% of all breast cancers. NEBC is a type of NEN that arises from neuroendocrine cells in the breast. NEBC is often misdiagnosed due to its rarity and the lack of awareness among healthcare professionals. The symptoms of NEBC are similar to those of other types of breast cancer, including a lump or mass in the breast, changes in the breast's appearance, and nipple discharge. NEBC can be diagnosed using imaging tests, such as mammography, ultrasound, or MRI, and confirmed by a biopsy [1].

Description

Treatment options for NEBC depend on the stage and extent of the cancer. Surgery is the primary treatment for localized NEBC, and radiation therapy and chemotherapy may be used in addition to surgery. For advanced or metastatic NEBC, systemic therapy, including chemotherapy and targeted therapy, may be used. In conclusion, NENs and NEBC are rare types of cancer that can be challenging to diagnose and treat. Increased awareness and research on these diseases are necessary to improve their diagnosis and management. Based on the provided text, it seems that the benefits of metformin in clinical trials have been modest and subject to potential biases due to the limitations of observational and review study designs. Additionally, the interpretation of preclinical models is also limited by their inability to fully replicate the complex tumor microenvironment. To address these issues and improve the design of future clinical trials investigating the effectiveness of metformin, further research is needed to identify the key factors that influence patient and tumor responsiveness to the drug. This may include factors such as tumor heterogeneity, the presence of cancer stem cells, and the immune and micro environmental context of the tumor. Overall, while the potential benefits of metformin in cancer treatment are promising, more research is needed to fully understand its mechanisms of action and how best to utilize it in clinical practice [2].

It appears that adding metformin to a treatment regimen for HER2-positive breast cancer patients may lead to changes in systemic host metabolism. Specifically, the addition of metformin was associated with an increase in the levels of the ketone body -hydroxybutyrate and the TCA intermediate -ketoglutarate, which are markers of mitochondrial function. Furthermore, the study found that patients in the metformin-containing group were more likely to achieve a pathological full response (pCR) if their homocysteine levels were significantly elevated. This suggests that metformin may enhance the therapeutic index of cancer treatments by modifying systemic host metabolism in a fasting-like manner, possibly through its effects on mitochondrial function and one-carbon metabolism. Overall, these findings support the idea of using a "drug plus diet" approach to enhance the effectiveness of cancer treatments, and suggest that metformin may be a promising candidate for pharmacologically reproducing the metabolic features of specific dietary modifications. However, further research is needed to confirm these results and to determine the optimal dosing and timing of metformin in combination with other cancer therapies.

The purpose of this study was to look at the relationship between cancer development and metformin therapy. On the basis of sample cohort data from the National Health Insurance Service, a population-based cohort study of adult diabetic patients was carried out in 2010. Those who had received recurrent oral metformin medication over a 90-day period were considered metformin users. Even in the high daily dosage metformin groups (>1 g/day), we failed to detect a correlation between metformin therapy and the risk of cancer among diabetes patients. To corroborate these results, additional prospective, sizable population-based cohort studies are required because there may still be residual confounders or bias. As a clinical pharmacodynamics biomarker connecting metformin's antifolate-like effect and biological tumour response, circulating homo cysteine may be investigated. There is debate in the literature right now on whether metformin can increase patients' chances of surviving colon cancer. In order to determine the relationship between metformin and the survival rate of type II diabetic individuals with colorectal cancer (CRC), we performed a meta-analysis. Although metformin is known to have an antitumor impact, its role in cancer prevention is still debatable [3-5].

Conclusion

There is still no solid evidence linking metformin use to an increased risk of prostate cancer. To assess a possible correlation between metformin use and prostate cancer risk, we conducted a systematic review and meta-analysis of all relevant cohort studies. The second-leading cause of cancer-related mortality in men is prostate cancer, which is the most prevalent malignant cancer in males worldwide after lung cancer. The aim of this study was to examine the association between male metformin use and prostate cancer. In developed nations, breast cancer is one of the main causes of cancer mortality. We conducted a meta-analysis of randomised clinical trials to examine the association between dose and response as well as the impact of metformin on biomarkers linked to outcomes in breast cancer.

Acknowledgement

We thank the anonymous reviewers for their constructive criticisms of the manuscript. The support from ROMA (Research Optimization and recovery in the Manufacturing industry), of the Research Council of Norway is highly appreciated by the authors.

Conflict of Interest

None.

***Address for Correspondence:** Vallabh Willer, Department of Medicines, University of Barcelona, Barcelona, Spain, E-mail: barbato@yahoo.com*

Received: 02 January, 2022, Manuscript No. jio-23-94558; **Editor assigned**: 03 January, 2022, Pre QC No. P-94558; **Reviewed:** 16 January, 2023, QC No. Q-94558; **Revised:** 21 January, 2023, Manuscript No. R-94558; **Published:** 28 January, 2023, DOI: 10.37421/2329-6771.2023.12.412

References

1. Nanni, O., D. Amadori, A. De Censi and A. Rocca. "Metformin plus chemotherapy versus chemotherapy alone in the first-line treatment of HER2-negative metastatic breast cancer. The MYME randomized, phase 2 clinical trial." *Breast Cancer Res Treat* 174 (2019): 433-442.
2. Fenn, Kathleen, Matthew Maurer, Shing M. Lee and Katherine D. Crew. "Phase 1 study of erlotinib and metformin in metastatic triple-negative breast cancer." *Clin Breast Cancer* 20 (2020): 80-86.
3. Zell, Jason A., Christine E. McLaren, Timothy R. Morgan and Michael J. Lawson. "A phase iia trial of metformin for colorectal cancer risk reduction among individuals with history of colorectal adenomas and elevated body mass index clinical trial of metformin in obese patients with adenoma." *Cancer Prev Res* 13 (2020): 203-212.
4. Molenaar, Remco J., Jons W. van Hattum, Iris S. Brummelhuis and Jorg R. Oddens. "Study protocol of a phase II clinical trial of oral metformin for the intravesical treatment of non-muscle invasive bladder cancer." *BMC cancer* 19 (2019): 1-9.
5. Bilusic, Marijo, Nicole J. Toney, Renee N. Donahue and Susan Wroblewski. "A randomized phase 2 study of bicalutamide with or without metformin for biochemical recurrence in overweight or obese prostate cancer patients (BIMET-1)." *Prostate Cancer Prostatic Dis* (2022): 1-6.

How to cite this article: Willer, Vallabh. "Metformin and Systemic Host Metabolism in Cancer Treatment: Effects on Mitochondrial Function and One-Carbon Metabolism for Enhanced Therapeutic Index." *J Integr Oncol* 12 (2023): 412.