Mini Review
Volume 09:01, 2024

Journal of Diabetic Complications and Medicine

ISSN: 2475-3211 Open Access

Metformin in Esophageal Carcinoma: Exploring Molecular Mechanisms and Therapeutic Insights

Daniel Benny*

Department of Medical Social Sciences, Medical University of South Carolina, South Carolina, USA

Abstract

Esophageal carcinoma is a challenging malignancy with limited treatment options and poor prognosis, necessitating the exploration of novel therapeutic strategies. Metformin, a widely used drug for type 2 diabetes, has shown promising anticancer effects in various malignancies, including esophageal carcinoma. This article provides an overview of the molecular mechanisms underlying the anticancer properties of metformin in esophageal carcinoma and discusses its therapeutic potential.

Keywords: Metformin • Esophageal carcinoma • Molecular mechanisms • Therapeutic insights

Introduction

Esophageal carcinoma ranks among the deadliest cancers worldwide, characterized by its aggressive nature, limited treatment options and poor prognosis. Despite advancements in therapy, the overall survival rates for esophageal carcinoma remain low, highlighting the urgent need for innovative treatment approaches. Metformin, an oral hypoglycemic agent, has garnered attention for its potential anticancer effects [1]. In this article, we delve into the molecular mechanisms through which metformin exerts its anticancer activity in esophageal carcinoma and explore its therapeutic implications.

Literature Review

Metformin activates AMPK, a key cellular energy sensor, leading to inhibition of mTOR signaling pathway. AMPK activation by metformin suppresses cell proliferation, induces cell cycle arrest and promotes apoptosis in esophageal carcinoma cells. Metformin disrupts cancer cell metabolism by inhibiting mitochondrial complex I, leading to decreased ATP production and altered cellular metabolism [2]. This metabolic reprogramming attenuates cancer cell growth and survival in esophageal carcinoma. Metformin inhibits the insulin/IGF signaling pathway, which is implicated in tumorigenesis and cancer progression. Suppression of IGF signaling by metformin impedes proliferation, migration and invasion of esophageal carcinoma cells [3,4].

Discussion

By inhibiting EMT, metformin impedes invasion and metastasis of esophageal carcinoma cells, enhancing treatment efficacy. Metformin holds promise as an adjuvant therapy in esophageal carcinoma, complementing standard treatment modalities such as surgery, chemotherapy and radiotherapy. Clinical trials evaluating the efficacy of metformin as an adjuvant therapy in esophageal carcinoma are underway, with preliminary results showing encouraging outcomes. Combining metformin with conventional chemotherapeutic agents or targeted therapies may enhance treatment efficacy and overcome resistance mechanisms [5]. Synergistic interactions between metformin and other anticancer agents have been observed in preclinical studies, underscoring the potential for combination therapy in esophageal carcinoma. Identification of biomarkers predictive of metformin response can facilitate personalized treatment approaches in esophageal carcinoma. Biomarker-guided selection of patients likely to benefit from metformin therapy may optimize treatment outcomes and minimize unnecessary side effects [6].

Conclusion

Metformin emerges as a promising therapeutic agent for esophageal carcinoma, exerting anticancer effects through multiple molecular mechanisms. Its ability to target various hallmarks of cancer, including dysregulated metabolism, proliferative signaling and inflammation, underscores its potential as a multifaceted anticancer agent. Further research, including well-designed clinical trials, is warranted to elucidate the therapeutic efficacy and safety profile of metformin in the management of esophageal carcinoma.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Bray, Freddie, Jacques Ferlay, Isabelle Soerjomataram and Rebecca L. Siegel, et al. “Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries.” *CA Cancer J Clin* 68 (2018): 394-424.
2. Thrumurthy, Sri G., M. Asif Chaudry, Sasha SD Thrumurthy and Muntzer Mughal. “Oesophageal cancer: Risks, prevention and diagnosis.” *Bmj* 366 (2019).
3. Kamangar, Farin, Dariush Nasrollahzadeh, Saeid Safiri and Sadaf G. Sepanlou, et al. “The global, regional and national burden of oesophageal cancer and its attributable risk factors in 195 countries and territories, 1990–2017: A systematic analysis for the global burden of disease study 2017.” *Lancet Gastroenterol Hepatol* 5 (2020): 582-597.
4. Santucci, Claudia, Silvia Mignozzi, Matteo Malvezzi and Giulia Collatuzzo, et al “Global trends in esophageal cancer mortality with predictions to 2025 and in incidence by histotype.” *Cancer Epidemiol* 87 (2023): 102486.

***Address for Correspondence:** Daniel Benny, Department of Medical Social Sciences, Medical University of South Carolina, South Carolina, USA; E-mail: bennyd333@gmail.com*

Received: 19 January, 2024, Manuscript No. jdcm-24-129729; **Editor Assigned:** 22 January, 2024, PreQC No. P-129729; **Reviewed:** 05 February, 2024, QC No. Q-129729; **Revised:** 10 February, 2024, Manuscript No. R-129729; **Published:** 17 February, 2024, DOI: 10.37421/2475-3211.2024.9.247

5. Bolger, Jarlath C., Claire L. Donohoe, Maeve Lowery and John V. Reynolds. "Advances in the curative management of oesophageal cancer." *Br J Cancer* 126 (2022): 706-717.

6. Soenen, Stijn, Chris K. Rayner, Michael Horowitz and Karen L. Jones. "Gastric emptying in the elderly." *Clin Geriatr Med* 31 (2015): 339-353.

How to cite this article: Benny, Daniel. "Metformin in Esophageal Carcinoma: Exploring Molecular Mechanisms and Therapeutic Insights." *J Diabetic Complications Med* 9 (2024): 247.