Journal of Pregnancy and Child Health
ISSN: 2376-127X

Mishra, J Preg Child Health 2023, 10:2

Editorial Open Access

The Ovarian Cancer due to Death of Non-Apoptotic Cell: Resistance, Treatment, and Prognosis

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Abstract

Due to the lack of reliable screening procedures and distinctive symptoms, ovarian cancer is typically discovered at an advanced stage. Low survival rates and a poor prognosis are caused by repeated treatment resistance and recurrence prior to the advent of PARPi as maintenance medicines. According to recent studies, apoptosis plays a significant role in ovarian cancer. With the further advancement of targeted therapy, non-apoptotic cell death, such as autophagy, ferroptosis, necroptosis, immunogenic cell death, pyroptosis, alkaliptosis, and other mechanisms of cell death, has demonstrated significant potential in tumour prevention and treatment. The involvement of non-apoptotic cell death in the initiation, progression, and prognosis of ovarian cancer has been the subject of a systematic review of the literature.

Keywords: Ovarian cancer; Autophagy; Ferroptosis; Treatment; Resistance

Introduction

A deeper theoretical foundation is provided in this review for investigating therapeutic targets and medication developing risk prediction models that aid in the clinical transition of essential medications, and resistance in refractory ovarian cancer [1]. Only breast and cervical cancer have a higher fatality rate than ovarian cancer, which is the third most deadly female malignancy [2]. In 2020, there were 313,959 new cases of OC and 207,252 deaths from OC, according to morbidity and mortality data reports of 36 forms of cancer from 185 countries [3]. In the same year, there were 55,342 new instances of OC in China, making up 17.62% of all new cases worldwide [4]. Additionally, 37,519 people died from OC in China in 2020, making up 18.10% of all deaths worldwide [5]. The majority of patients experience an insidious start due to the lack of reliable screening techniques and distinct symptoms [6]. About 70% of people receive an advanced stage diagnosis, and less than 50% of people survive for five years total [7].

Discussion

Therefore, improving the prognosis of patients with OC by identifying new therapeutic targets and overcoming the resistance to poly-ADP-ribose polymerase inhibition and platinum-based chemotherapy [8]. A rigorous genetic mechanism that controls programmed cell death directs an autonomous cell death process to preserve a constant internal environment [9]. The most effective form of PCD, apoptosis, is the main mechanism of action of the majority of currently available anticancer medications [10]. However, by overexpressing anti-apoptotic proteins, tumour cells become resistant to apoptosis, resulting in the development and growth of tumour. Non-apoptotic cell death has demonstrated considerable promise for tumour prevention and treatment due to the continual development of targeted therapies. In autophagy, the most typical type of cell death that isn't apoptotic. Cytoplasmic macromolecules, aggregated proteins, damaged organelles, or pathogens are carried to the lysosome when cells are malnourished or infected by inflammation. As a result, normal cell and tissue homeostasis is maintained. Lysosomal hydrolase, a mediator of cell self-digestion, maintains cell metabolism and survival during hunger and stress. The increased metabolic needs brought on by the fast expansion of cancer stem cells are addressed by autophagy. Once a tumour has developed, elevated autophagy flux promotes tumour cell survival and proliferation in hypoxic and nutrient-rich environments. Therefore, preventing autophagy is advantageous for getting rid of malignant cells. Ferroptosis, which ultimately results in cell death, is dependent on the ongoing buildup of lipid peroxides in the cell membrane. However, ferroptosis can activate immune systems associated with inflammation. Causing tumour growth in the tumor's microenvironment. Ferroptosis-targeting is a new anticancer tactic. Finally, non-apoptotic cell death opens up a new avenue for the diagnosis and therapy of tumours. To aid in clinical diagnosis and therapy, the research on non-apoptotic cell death in OC, including therapeutic targets, drug resistance, efficacy prediction, and the association of various non-apoptotic cell death, is carefully reviewed in this study.

Conclusion

The mechanism of autophagy in the onset and progression of OC would aid in the development of new clinical biomarkers to monitor autophagy, find appropriate therapeutic timing and effective therapeutic targets, and suppress and even reverse drug resistance through a combination of treatments, which is thought to be an emerging strategy for the prevention and treatment of OC. Lysosomal-driven catabolism, or autophagy, is the most prevalent form of PCD. The metabolic transformation of tumour and stromal cells, which influences the development of the tumour microenvironment and becomes a target for tumour therapy, is highly valued by autophagy. Depending on the types and stages of carcinogenesis, autophagy can play a variety of roles. Autophagy slows down the development of tumours and stops tumour growth in its early stages. once the tumour reaches an advanced stage and the internal pressure Autophagy aids in

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Received: 02-Feb-2023, Manuscript No. jpch-23-87989; **Editor assigned:** 06-Feb-2023, PreQC No. jpch-23-87989 (PQ); **Reviewed:** 20-Feb-2023, QC No. jpch-23-87989; **Revised:** 24-Feb-2023, Manuscript No. jpch-23-87989(R); **Published:** 28-Feb-2023, DOI: 10.4172/2376-127X.1000578

Citation: Mishra S (2023) The Ovarian Cancer due to Death of Non-Apoptotic Cell: Resistance, Treatment, and Prognosis. J Preg Child Health 10: 578.

the survival and growth of the initial tumour and, through encouraging metastasis, increases the invasiveness of the tumour. Inhibiting autophagy in this situation may lead to better treatment outcomes for cancer patients.

Acknowledgement

None

Conflict of Interest

None

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