Opinion
Volume 20:01, 2024

Journal of Surgery [Jurnalul de chirurgie]

ISSN: 1584-9341 Open Access

Triple-negative Breast Cancer: Challenges, Advances and Future Directions

Robert Clan*

Department of Internal Medicine, University of Münster, Münster, Germany

Introduction

Triple-Negative Breast Cancer (TNBC) represents a distinct subtype of breast cancer characterized by the absence of Estrogen Receptor (ER), Progesterone Receptor (PR), and Human Epidermal Growth Factor Receptor 2 (HER2) expression. TNBC accounts for approximately 15-20% of all breast cancer cases and is associated with aggressive clinical behavior, higher rates of recurrence, and poorer prognosis compared to other breast cancer subtypes. Despite advances in breast cancer treatment, TNBC remains a significant clinical challenge due to its heterogeneity, lack of targeted therapies, and limited treatment options. This manuscript aims to provide a comprehensive overview of TNBC, exploring its epidemiology, molecular features, clinical presentation, current treatment strategies, challenges, recent advances, and future directions [1].

TNBC is more commonly diagnosed in younger women, African American women, and those with a family history of breast cancer. Molecular profiling studies have revealed that TNBC is a heterogeneous disease comprised of multiple molecular subtypes with distinct biological characteristics and clinical outcomes. These subtypes include basal-like, mesenchymal-like, immune-rich, and Luminal Androgen Receptor (LAR) subtypes, each characterized by specific gene expression patterns and signaling pathways. Basal-like TNBC, in particular, is associated with a higher risk of metastasis and poorer survival outcomes [2].

Description

TNBC often presents as a palpable breast lump or mass, with symptoms such as breast pain, nipple discharge, or skin changes. Imaging studies, including mammography, ultrasound and Magnetic Resonance Imaging (MRI), are used to evaluate the extent of disease and guide treatment decisions. Histopathological examination of tumor tissue is essential for confirming the diagnosis of TNBC, with characteristic features including high histological grade, high mitotic index, and lymphocytic infiltration. Immunohistochemical staining for ER, PR, and HER2 expression is used to classify tumors as TNBC. The management of TNBC involves a multimodal approach, including surgery, chemotherapy, radiation therapy, and, in some cases, targeted therapies. Neoadjuvant chemotherapy is commonly administered to downstage locally advanced tumors and increase the likelihood of breast-conserving surgery. Anthracycline and taxane-based regimens are the standard of care for TNBC, although platinum-based chemotherapy may be considered for patients with BRCA1/2 mutations. Despite initial responses to chemotherapy, TNBC is associated with a higher risk of recurrence and distant metastasis [3].

TNBC poses several challenges in clinical management, including the lack of targeted therapies and the development of chemotherapy resistance. The absence of ER, PR, and HER2 expression precludes the use of endocrine therapies and HER2-targeted agents, limiting treatment options for TNBC patients. Additionally, TNBC exhibits significant molecular heterogeneity, making it difficult to predict treatment responses and identify actionable targets. Furthermore, disparities in access to healthcare and clinical trials disproportionately impact underserved populations, exacerbating the burden of TNBC in certain demographic groups [4].

Despite these challenges, recent advances in TNBC research have led to the identification of novel therapeutic targets and the development of targeted therapies. Immune checkpoint inhibitors, such as pembrolizumab and atezolizumab, have shown promising results in clinical trials, particularly in patients with PD-L1-positive tumors. Poly (ADP-ribose) polymerase (PARP) inhibitors [5], such as olaparib and talazoparib, have demonstrated efficacy in patients with BRCA1/2 mutations, providing a targeted treatment option for a subset of TNBC patients. Additionally, ongoing research efforts are exploring combination therapies, biomarker-driven approaches, and immunotherapy-based strategies to further improve outcomes in TNBC.

Conclusion

TNBC represents a challenging subtype of breast cancer characterized by aggressive clinical behavior, limited treatment options, and poorer prognosis compared to other breast cancer subtypes. Despite these challenges, recent advances in TNBC research have provided insights into the molecular underpinnings of the disease and paved the way for the development of novel therapeutic strategies. By embracing innovation, fostering collaboration, and advocating for equitable access to care, the healthcare community can work towards improving outcomes and quality of life for individuals affected by TNBC.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Jiang, Shuai, Hong-Wei Zhang, Ming-Hua Lu and Xiao-Hong He, et al. "MicroRNA-155 functions as an OncomiR in breast cancer by targeting the suppressor of cytokine signaling 1 gene." *Cancer Res* 70 (2010): 3119-3127.
2. Zhang, Chun-Mei, Jing Zhao and Hua-Yu Deng. "MiR-155 promotes proliferation of human breast cancer MCF-7 cells through targeting tumor protein 53-induced nuclear protein 1." *J Biomed Sci* 20 (2013): 1-10.
3. Kong, William, Lili He, Marc Coppola and Jianping Guo, et al. "MicroRNA-155 regulates cell survival, growth, and chemosensitivity by targeting FOXO3a in breast cancer." *J Biol Chem* 285 (2010): 17869-17879.
4. Kyriakis, John M., Harald App, Xian-feng Zhang and Papia Banerjee, et al. "Raf-1 activates MAP kinase-kinase." *Nature* 358 (1992): 417-421.

***Address for Correspondence:** Robert Clan, Department of Internal Medicine, University of Münster, Münster, Germany; E-mail: clan.robert@yahoo.edu*

Received: 01 January, 2024, Manuscript No. JOS-24-129953; **Editor Assigned:** 02 January, 2024, PreQC No. P-129953; **Reviewed:** 17 January, 2024, QC No. Q-129953; **Revised:** 23 January, 2024, Manuscript No. R-129953; **Published:** 31 January, 2024, DOI: 10.37421/1584-9341.2024.20.135

5. Moodie, Shonna A., Berthe M. Willumsen, Michael J. Weber and Alan Wolfman. "Complexes of Ras- GTP with Raf-1 and mitogen-activated protein kinase kinase." *Sci* 260 (1993): 1658-1661.

How to cite this article: Clan, Robert. "Triple-negative Breast Cancer: Challenges, Advances and Future Directions." *J Surg* 20 (2024): 135.