

Developing the Therapeutic Potential of Beta-Blockers in Leukemia and Future Directions

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DESCRIPTION

Leukemia, a group of cancers that typically begin in the bone marrow and result in high numbers of abnormal white blood cells, poses significant challenges in terms of treatment and management. While chemotherapy, radiation, and bone marrow transplantation have been the mainstays of treatment, the role of cardiovascular drugs, particularly beta-blockers, in leukemia is an emerging area of interest [1]. Beta-blockers, traditionally used to manage cardiovascular conditions like hypertension and arrhythmias, have shown potential in modulating cancer progression, including leukemia. This article delves into the potential role of beta-blockers in leukemia, exploring their mechanisms, therapeutic potential, and challenges. Beta-blockers are a class of medications that inhibit the effects of adrenaline (epinephrine) and noradrenaline (norepinephrine) on beta-adrenergic receptors, thereby reducing heart rate, cardiac output, and blood pressure. These drugs are widely used to manage cardiovascular conditions, such as hypertension, heart failure, and arrhythmias, and are also employed in managing anxiety and preventing migraines [2-4].

The primary beta-adrenergic receptors targeted by beta-blockers are Beta-1 receptors are predominantly found in the heart, these receptors, when blocked, lead to a decrease in heart rate and contractility. Beta-2 receptors are Located mainly in the lungs, vascular smooth muscle, and skeletal muscle, blocking these receptors can lead to bronchoconstriction and vasodilation. Commonly used beta-blockers include propranolol, atenolol, metoprolol, and carvedilol. These drugs vary in their selectivity for beta-1 and beta-2 receptors, as well as their pharmacokinetic properties, which can influence their therapeutic and adverse effects [5-7]. The interest in beta-blockers in oncology, including leukemia, stems from their potential to interfere with the adrenergic signaling pathways that are implicated in cancer progression. Chronic stress and elevated levels of catecholamines (adrenaline and noradrenaline) have been associated with tumor progression, metastasis, and poor prognosis in various cancers. Beta-blockers, by inhibiting adrenergic signaling, could

potentially reduce these effects and improve outcomes in cancer patients [8].

Adrenergic signaling can promote the formation of new blood vessels (angiogenesis) that supply nutrients to tumors. Beta-blockers may inhibit this process, thereby starving the tumor of essential nutrients. Beta-blockers may enhance the immune system's ability to recognize and destroy cancer cells by reducing the immunosuppressive effects of chronic stress. Adrenergic signaling is implicated in the metastatic spread of cancer cells. By blocking this pathway, beta-blockers may reduce the likelihood of metastasis. Some studies suggest that beta-blockers may directly inhibit the proliferation of cancer cells through mechanisms independent of adrenergic signaling. Leukemia is characterized by the uncontrolled proliferation of abnormal white blood cells, which can lead to bone marrow failure, immune suppression, and infiltration of other organs. While the use of beta-blockers in solid tumors has been extensively studied, their role in hematological malignancies like leukemia is less well understood [9].

Leukemia cells, like other cancer cells, can be influenced by adrenergic signaling. Beta-adrenergic receptors have been identified on leukemia cells, suggesting that these cells may respond to catecholamine's. The activation of these receptors could potentially promote the proliferation and survival of leukemia cells. Beta-blockers, by inhibiting these receptors, may therefore reduce leukemia cell growth and enhance the effectiveness of existing therapies. Preclinical studies have provided some insights into the potential role of beta-blockers in leukemia. For example, studies in animal models have shown that beta-blockers can reduce the growth of leukemia cells and enhance the effects of chemotherapy. In one study, the beta-blocker propranolol was found to inhibit the growth of Chronic Lymphocytic Leukemia (CLL) cells *in vitro* and *in vivo*, suggesting that this class of drugs may have direct anti-leukemic effects. While preclinical data are promising, clinical evidence for the use of beta-blockers in leukemia is limited. Some observational studies have suggested that beta-blocker use is associated with improved survival in patients with hematological malignancies, including leukemia. However, these studies are often confounded

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by factors such as the indication for beta-blocker use (e.g., cardiovascular disease), and randomized controlled trials are needed to establish a causal relationship. While the potential role of beta-blockers in leukemia is intriguing, several challenges and considerations must be addressed.

The current evidence supporting the use of beta-blockers in leukemia is largely based on preclinical studies and observational data. Randomized controlled trials are needed to establish the efficacy and safety of beta-blockers in this context. Until such data are available, the use of beta-blockers in leukemia should be considered experimental. Not all leukemia patients may benefit from beta-blockers. Factors such as the presence of cardiovascular disease, the specific subtype of leukemia, and the expression of beta-adrenergic receptors on leukemia cells should be considered when selecting patients for beta-blocker therapy. Beta-blockers are generally well-tolerated, but they can cause adverse effects such as bradycardia, hypotension, fatigue, and bronchoconstriction. In leukemia patients, who may already be frail and immunocompromised, these side effects could be more pronounced. Careful monitoring and dose adjustments may be necessary to minimize the risk of adverse effects. Leukemia patients often receive multiple medications, including chemotherapy, targeted therapies, and supportive care drugs. The potential for drug interactions between beta-blockers and other medications must be carefully considered. For example, beta-blockers may interact with certain chemotherapy drugs, leading to enhanced cardiotoxicity or reduced therapeutic efficacy. The use of beta-blockers in leukemia patients may have psychosocial implications, particularly in terms of their impact on stress and anxiety. While beta-blockers can reduce the physiological effects of stress, their impact on psychological well-being is less clear. Some patients may experience fatigue or depression as a side effect of beta-blockers, which could affect their overall quality of life [10].

CONCLUSION

The role of beta-blockers in leukemia represents an exciting and emerging area of research. While the current evidence is

promising, further studies are needed to establish the efficacy and safety of these drugs in leukemia patients. If successful, beta-blockers could offer a novel and relatively low-cost addition to the armamentarium against leukemia, potentially improving outcomes for patients with this challenging disease. As we move forward, a combination of clinical trials, mechanistic studies, and personalized approaches will be key to unlocking the full potential of beta-blockers in leukemia therapy.

REFERENCES

1. Carpenter KA, Thurlow KE, Craig SE, Grainger S. Wnt regulation of hematopoietic stem cell development and disease. *Current topics in developmental biology*. 2023; 153(1):255-79.
2. Precilla SD, Biswas I, Kuduvali SS, Anitha TS. Crosstalk between PI3K/AKT/mTOR and WNT/ β -Catenin signaling in GBM-Could combination therapy checkmate the collusion?. *Cellular Signalling*. 2022;95:110350.
3. Hao X, Zhang Y, Lu Y, Han G. STK39 enhances the progression of Cholangiocarcinoma *via* PI3K/AKT pathway. *Iscience*. 2021; 24(11):1-10.
4. Kurzer JH, Weinberg OK. Updates in molecular genetics of acute myeloid leukemia. In *Seminars in Diagnostic Pathology* 2023; 40(3): 140-151.
5. Ma Q, Yu J. Wnt/ β -catenin signaling pathway-a versatile player in apoptosis and autophagy. *Biochimie*. 2023; 211(1):57-67.
6. Sun R, He L, Lee H, Glinka A. RSPO2 inhibits BMP signaling to promote self-renewal in acute myeloid leukemia. *Cell reports*. 2021;36(7):56-96.
7. Zhang Y, Viennois E. Knockout of Ste20-like Proline/Alanine-rich Kinase (SPAK) attenuates intestinal inflammation in mice. *The American journal of pathology*. 2013;182(5):1617-1628.
8. Chen J, Zhou L. Knockdown of STK39 suppressed cell proliferation, migration, and invasion in hepatocellular carcinoma by repressing the phosphorylation of mitogen-activated protein kinase p38. *Bioengineered*. 2021;12(1):6529-6537.
9. Chiu MH, Liu HS. SPAK mediates KCC 3-enhanced cervical cancer tumorigenesis. *The FEBS journal*. 2014; 281(10): 2353-2365.
10. Delpire E, Gagnon KB. SPAK and OSR1, key kinases involved in the regulation of chloride transport. *Acta physiologica*. 2006; 187(2):103-113.