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Venetoclax-Associated Cardiotoxicity in Acute Myeloid Leukemia: A Review of Mechanisms, Clinical Evidence, and Therapeutic Implications

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ABSTRACT

Venetoclax (VTX) is an antineoplastic agent that inhibits the BCL-2 protein and modulates mitochondrial apoptosis. It has demonstrated efficacy in the treatment of acute myeloid leukemia (AML) and chronic lymphocytic leukemia (CLL). The combination of VTX with a hypomethylating agent (HMA) has resulted in improved survival outcomes for patients with AML, exceeding those observed with monotherapy. The toxicity profile of venetoclax includes neutropenia, gastrointestinal disturbances, and diarrhea. Cardiotoxicity is a significant concern, particularly when venetoclax is administered at high doses or over extended periods. This adverse effect can result in serious complications, including myocardial infarction, new-onset atrial fibrillation, and sudden cardiac death. Studies indicate that approximately 20% of patients receiving venetoclax in combination with a hypomethylating agent (HMA) experience cardiac event, frequently necessitating therapy suspension. Venetoclax-associated cardiotoxicity has been linked to oxidative stress, inflammation, and apoptosis. Clinical studies have identified an imbalance between the production of reactive oxygen species (ROS) and the antioxidant defence system, as well as elevated levels of inflammatory markers, which contribute to the activation of apoptosis in cardiac cells. Elucidating these mechanisms are necessary for the development of targeted oncologic therapies that optimize treatment efficacy while ensuring cardiovascular safety.

Keywords: Venetoclax; Cardiotoxicity; Acute Myeloid Leukaemia; Targeted Oncologic Therapies

Abbreviations: VTX: Venetoclax; AML: Acute Myeloid Leukaemia; CLL: Chronic Lymphocytic Leukaemia; HMA: Hypomethilating Agent; ROS: Reactive Oxygen Species; MDS: Myelodysplastic syndrome; MPS: Myeloproliferative Neoplasms; FAERS: FDA's Adverse Event Reporting System; LVEF: Left Ventricular Ejection Fraction; RVR: Rapid Ventricular Response; LSCs: Leukaemia Stem Cells; TnT: Cardiac Troponin T

Introduction

Haematological malignancies (HMs) are diverse neoplasms characterised by differences in pathogenesis, prognosis, and treatment [1]. Acute myeloid leukaemia (AML) is a rapidly progressing myeloid neoplasm. It originates from the stem cell precursors of the myeloid lineage, which include red blood cells, platelets, and white blood cells [2]. AML is defined by the clonal proliferation of immature myeloid cells, called blasts, in the peripheral blood and bone marrow. This process leads to bone marrow failure and results in underproduction of red blood cells and platelets. It is associated with infection, anaemia, and bleeding [3]. The classification of AML is based on mutational profiles according to the European Leukaemia Network (ENL) 2022 recommendations. High-risk myelodysplastic syndrome (MDS) and myeloproliferative neoplasms (MPS) can both lead to AML [4]. Patients exposed to alkylating agents or radiation during chemotherapy, as well as those exposed to environmental factors such as radiation, tobacco smoke, and benzene, may develop therapy-related MDS/AML or de novo AML [5].

In the United States, the annual incidence of new AML cases exceeds 20,000. The median age of diagnosis is 68 years. The prevalence is significantly higher in non-Hispanic whites. The male-to-female ratio is 5:3 [4]. Despite advances in treatment, AML accounts for

about 1.68% of all cancer deaths in the EU in 2021. This highlights the need for targeted prevention and improved health policies to improve patient outcomes [6]. The use of targeted anticancer therapies with small-molecule inhibitors has been shown to be an important therapeutic strategy for overcoming haematological tumours. Venetoclax (VTX) is an orally bioavailable small-molecule inhibitor of B-cell lymphoma 2 (BCL-2). By modulating the mitochondrial apoptotic pathway, it shows great efficacy in the treatment of Acute myeloid leukemia (AML) and Chronic lymphocytic leukaemia (CLL) [7]. When evaluating overall survival (OS), complete remission/complete remission with incomplete blood count recovery (CR/CRi), and progression-free survival (PFS), the combination of a hypomethylating agent (HMA) with VTX therapy has shown improved response rates. These results are superior to those of VTX monotherapy in AML patients [8]. Besides haematological toxicities and upper respiratory tract infections as side effects, symptoms of cardiotoxicity can also occur with prolonged use of the drug [9].

Objective of the Work

Venetoclax is widely used in oncological treatment, particularly for patients with chronic lymphocytic leukemia and acute myeloid leukemia, often in combination with a hypomethylating agent [10]. The toxicity profile of Venetoclax includes neutropenia, gastrointestinal issues, and diarrhea, while there is growing concern regarding its potential cardiotoxicity in patients undergoing treatment for Acute Myeloid Leukemia. High doses or prolonged use of Venetoclax may lead to serious cardiac complications, such as myocardial infarction, new-onset atrial fibrillation, and even sudden cardiac death [10]. Javaria Ahmad et al. analysed the FDA's Adverse Event Reporting System (FAERS), which is focused on the FDA's pharmacovigilance mechanism. They collected reports on VTX and other chemotherapeutic agents associated with adverse events, such as atrial fibrillation. Their analysis indicates that the percentage of adverse cardiac events attributed to AF is higher than that of 30 other antitumoral drugs [11]. Our review focuses on the current literature about the potential cardiotoxicity of venetoclax. It also explores the mechanisms by which this drug may cause cardiac damage. This highlights the need for personalised cancer therapies that address these risks while maximising efficacy.

Clinical Pharmacology

Venetoclax (VTX) is defined as part of antineoplastic agents "BH3 mimetics" which inhibit antiapoptotic proteins such as BCL-2, BCL-w and BCL-XL [12]. Overexpression of BCL-2 has been demonstrated in patients with lymphoblastic leukemia, which mediates tumour cell survival and leads to resistance to chemotherapeutics [13]. By binding selectively to BCL-2, it removes the binding of pro-apoptotic proteins (such as BIM) that contain BH3 motifs, resulting in mitochondrial outer membrane permeabilisation (MOMP) and caspase activation. ((EMA.EUROPA.EU)). VTX is able to function as an antagonist and therefore can aid in the restoration of programmed cell death [14]. This action leads to the displacement of proapoptotic proteins, such as BIM, and induces caspase-dependent apoptosis. It enhances tumour cells' sensitivity to programmed cell death [15]. It is advised to take VTX with a high-fat meal, as this can increase its exposure by 5.3 times compared to fasting. Once in the bloodstream, VTX has a strong affinity for plasma proteins, binding within a concentration range of 1- 30 μM (0.87-26 μg/ml). The apparent volume of distribution (Vdss/F) of VTX in patients is estimated to be between 256 and 321 litres. Maximum plasma concentration is typically reached approximately 5 to 8 hours after repeated oral dosing.

VTX is primarily metabolised by CYP3A4 with an estimated terminal elimination half-life of approximately 26 hours. Of note, 20.8% of the drug is eliminated from the body in an unmetabolized form [13]. The combined regimen of the BCL- 2 inhibitor venetoclax (VTX) with a hypomethylating agent, such as Azacytidine (AZA), at a daily dose of 400 mg, has shown high synergy in the therapeutic response's efficacy [10]. Indeed, patients who are ineligible for intensive chemotherapy and have received VTX + AZA, demonstrated an improvement in their response rates and overall survival rates with durable remissions [16]. This could be due to these drugs' ability to target Leukemia stem cells, which leads to the creation of interferences in the Tricarboxylic Acid cycle (TCA cycle). Furthermore, decreased levels of alpha-ketoglutarate and increased succinate levels, are followed by the inhibition of electron transport chain complex II; which leads to perturbations that suppress oxidative phosphorylation (OXPHOS) and target Leukemia stem cells (LSCs). Finally, through the disruption of the metabolic machinery driving energy metabolism, AML patients can be cured of LSCs and have a better therapeutic response [17].

Common Side Effects of Venetoclax Uses

The most common adverse event (AE) associated with venetoclax is neutropenia. This occurs with both monotherapy and combination regimens [18]. Chemotherapy-induced neutropenia (CIN) is the most common haematological toxicity associated with cancer chemotherapy. The National Cancer Institute Common Toxicity Criteria (NCI-CTC) categorizes neutropenia's severity levels into four grades: grade 1 when the absolute neutrophil count [ANC] is < 2000/mmc, grade 2 when the ANC is < 1500/mmc, grade 3 when the ANC is < 1000/mmc, and grade 4 when the ANC is < 500/mmc [19]. A review of conducted clinical studies revealed that the predominant adverse hematological effects observed in the treatment of chronic and acute myeloid leukemia with venetoclax were grade III/IV neutropenia and febrile neutropenia. Phase II study on venetoclax monotherapy conducted by Stilgenbauer, et al. [20] showed that haematological toxicity was a prevalent adverse effect. The analysis reported that 40% of patients had grade III/IV neutropenia, 15% had thrombocytopenia, and 25% had anaemia.

The infection rate reached 81%, including pneumonia in 10% of patients. Consequently, 17% of patients required a reduction in venetoclax dosage, while 40% were obliged to discontinue treatment [20].

According to this, monoclonal antibody treatment has been identified as an factor contributing to the development of neutropenia. Therefore, combination therapies should be considered as an aggravating factor increasing the incidence of neutropenia [21]. Matthew Waggoner et al. (2021), showed that another class of AEs, due to the use of venetoclax, is represented by gastrointestinal (GI) disorders. Nausea instead, is defined as low to minimal risk as side effect. However, antiemetics, such as metoclopramide and 5-hydroxytryptamine 3 antagonists, are suggested to be used prior to the therapy. Finally, an additional common side effect is diarrhea. The concurrent use of Venetoclax and Azacytidine in patients with AML significantly increases the probability of experiencing diarrhea as a side effect [22].

Cardiotoxicity

One of the most significant health concerns following anticancer therapy has emerged to be cardiovascular damage [9]. This leads to an important issue in cancer treatment, influencing the mortality of patients with cancer and causing a delay or a discontinuation of the therapy. Patients following AML therapy with HMA have reported cardiac events, clinically manifested with new onset heart failure HF, acute myocardial infarction, new onset atrial fibrillation AF and sudden cardiac death [23]. Isla M. Johnson et al., through a monocentric retrospective study examined all patients with AML treated with venetoclax plus HMA in the initial stage or in relapsed/refractory setting at the Mayo Clinic between November 2018 and November 2020. 170 patients were studied, of which 109 received venetoclax plus HMA as initial therapy and the remaining 61 patients with relapsing/refractory AML. Forty-eight cardiac events were documented in 34 (20%) patients; cardiac event rates were similar despite treatment context or HMA used: 23/109 (21%) in patients treated in initial therapy compared to 11/61 (18%) in relapse context. Of these last 10 had received previous anthracycline therapy and 3 of them had anthracycline-related cardiomyopathy. The most frequent events included a reduction in left ventricular ejection fraction (LVEF) on echocardiography, atrial fibrillation with rapid ventricular response (RVR), followed by elevation of troponin without electrocardiogram alterations.

Among the 7 patients with troponin elevation; 4 experienced events in the setting of another inciting factor such as severe anaemia, while the remaining of them showed elevation levels of troponin without another explanation. Other cardiac events included worsening heart failure with retained ejection fraction (HFpEF), non-atrial fibrillation arrhythmia and pericardial effusion or pericarditis. In particular, 2 patients had a fatal cardiopulmonary arrest. Of the 34 patients who experienced cardiac events, 11 (32%) had no known pre-existing cardiac disease. In addition, 4 patients (12%) had no known cardiovascular risk factors. These events have had a significant impact on AML therapy. About half of the patients stopped (27%) or discontinued (21%) treatment with HMA plus venetoclax due to the cardiac event. Furthermore, most (89%) events required hospital care, with a considerable proportion being treated in intensive care (27%). Cardiac management included the initiation of new cardiac medications (77%), whereas 2 patients required emergency procedural intervention. Unfortunately, the cardiac event itself was fatal in 9 patients (27%) [24]. Although the link between venetoclax and cardiac events is not well-established, it is intriguing to explore the potential for cardiotoxicity given its mechanism of action. Therefore, Venetoclax could be cardiotoxic through different mechanisms.

Oxidative Stress

Oxidative stress, defined as an imbalance between the production of free radicals (ROS) and the body's antioxidant capacity, has been identified as a pivotal mechanism underlying cardiotoxicity induced by VTX and many other anticancer drugs [9]. In a prospective in vivo experimental study conducted by Al- asmari et al., the levels of malondialdehyde (MDA), catalase (CAT), and glutathione (GSH) were measured in three groups of rats. The first group was a control group treated with NaCl. 0,9% and a group treated with VTX at low (50 mg/kg) and high (100 mg/kg) doses over a 21-day period. The results showed that both low- and high-dose VTX groups showed elevated levels of MDA, a marker of lipid peroxidation and oxidative damage, compared with the control group. Concurrently, a clear decline in endogenous antioxidants important for maintaining cell homeostasis and ROS levels in response to various toxic insults, including glutathione (GSH) and catalase (CAT), was observed compared with the control group. These data support the hypothesis that VTX treatment induces an imbalance between ROS production and antioxidant capacity, promoting oxidative damage in cardiac cells and causing cardiotoxicity [9].

Inflammation

Excessive cellular stress, alterations in intracellular redox balance, and increased oxidant species lead to myocardial tissue damage and inflammation [25]. According to Al-Asmari et al, gene and protein expression of several markers related to inflammation and oxidative stress were analysed. The results showed that cytokine gene expression levels for IFN-γ and TGF-β were increased in both the high- and low-dose VTX treatment groups relative to the control group, with a dose-dependent relationship. High-dose VTX treatment also increased the inflammatory protein NF-κB p65, the pro-inflammatory cytokine TNF-α and the inflammatory cytokine IL-6 [9]. This suggests that high levels of Venetoclax therefore lead to the production of inflammatory proteins in the heart, causing cardiac damage

Apoptosis

Numerous studies have linked myocardial dysfunction and toxicity to apoptosis. BH3-only proteins can induce apoptosis in response to significant stress, such as genetic damage [26]. As mentioned above, venetoclax is a BH3-mimetic protein that regulates apoptosis, inhibiting BCL-2, an anti-apoptotic protein that prevents cell death, promoting apoptosis of cancer cells in AML patients. In particular,

elevated levels of BCL-2 can neutralise BH3-only proteins, allowing cells to escape apoptosis [27]. In the clinical setting, venetoclax has been associated with cardiomyopathy and arrhythmias [28]. Investigators, including AlAsmari et al, have suggested that VTX treatment may cause cardiac toxicity, possibly due to Bcl-2 inhibition, which can induce apoptosis in multiple organs, including the heart. Subsequently, Al-Asmari et al. measured gene and protein expression of apoptotic markers to investigate apoptosis induction in cardiac tissue. After 21 days of VTX treatment, gene expression of Bax (an apoptosis regulator also known as "bcl2 like protein 4") along with a significant reduction in Bcl-2 gene and protein expression were found in the VTX-treated group [25]. In particular, high-dose VTX treatment led to a significant increase in cleaved caspase-3 protein levels, which are known to be responsible for biochemical changes in apoptosis. Consequently, the induction of apoptotic markers can lead to cardiotoxicity [29].

Cardiac Damage

Cardiac enzymes, such as creatine kinase-MB (CK-MB) and cardiac troponin I (cTn-I), in combination with histopathological examination of cardiomyocytes, serve as pivotal markers for identifying cardiotoxic effects associated with anticancer drugs [30]. Changes in the levels of these enzymes indicate an early response to cardiac damage caused by toxic substances [31]. In the prospective experimental study conducted by Al-Asmari et al., in addition to assessing oxidative damage in cardiac cells, serum levels of CK-MB and cTn-I were analysed. Rats were divided into two groups and subjected to 21 days of VTX treatment at different drug doses to assess potential cardiac damage. The results showed increased levels of both enzymes compared to the control group, which had received no treatment. Histopathological observations on the Venetoclax-treated group revealed a nuclear enlargement of cardiac cells. In addition, the production of a chronic inflammatory response and a dose-dependent increase in cardiac myocyte size confirmed the structural and functional damage to the heart in VTX-treated patients.

A retrospective analysis in a clinical study by [32] investigated venetoclax's cardiotoxic effects, focusing on serum cardiac toxicity markers in 55 adult patients undergoing treatment. The research specifically examined cardiac troponin T (TnT) levels, a marker of heart muscle damage, in relation to venetoclax dosage and patient outcomes. The researchers measured TnT levels before, during, and after therapy, along with venetoclax serum levels (quantified via mass spectrometry-high- performance liquid chromatography). Statistical analyses, including Wilcoxon signed-rank tests and mixed-effects regression models, were employed to evaluate changes in TnT, its correlation with venetoclax dose, and the impact of creatinine levels. The study revealed a significant increase in TnT during venetoclax treatment, indicating a potential risk of cardiac injury associated with the drug [32].

Discussion

This review was conducted via an accurate retrospective analysis of literature from the PubMed scientific database. The time range defined in the bibliographical sources was set from 2020 to 2025. The inclusion criteria used to select the sources were as follow:

1. *In vitro* studies.
2. Clinical studies.
3. Articles referring to the mechanism of action of Venetoclax, to explore its potential cardiotoxicity profile in patients with AML.
4. Studies reporting cardiac events associated with Venetoclax in combination with HMA in the treatment of AML.
5. Studies that were not case reports.

Studies that met the inclusion criteria were submitted for data extraction. Numerous studies have reported the potentially cardiotoxic effect induced by VTX. Major adverse cardiac events (MACE) have been reported, including new-onset heart failure (HF), acute myocardial infarction and new-onset atrial fibrillation (AF). An in vivo study, conducted by Brock et al. from 2025, found a significant increase in cardiac troponin during Venetoclax treatment, indicating a potential risk of cardiac damage associated with the drug. Moreover, Isla M. Johnson et al through a retrospective analysis on patients with AML found that forty-eight cardiac events were documented in 34 patients. These events had a significant impact on therapy for AML, resulting in therapy discontinuation and the need of hospitalization. Furthermore, intensive care admissions and cardiac management treatment were included [24].

Conclusion

To conclude, venetoclax is emerging as a promising novel agent with significant efficacy in the treatment of acute myeloid leukaemia. However, through mechanisms such as apoptosis, oxidative stress and inflammation, it may induce cardiotoxic effects such as acute myocardial infarction and new-onset atrial fibrillation. The potential cardiotoxicity associated with high doses or prolonged use of Venetoclax represents a significant challenge in cancer therapy. This issue is critical in cancer therapy as it may lead to delays or discontinuation of chemotherapy, ultimately affecting patient mortality. It is therefore crucial to evaluate the cardiotoxicity profile of compounds such as Venetoclax in order to develop effective, tailored oncological regimens that also manage side effects and protect cardiac function.

Conflict of Interest

The authors declare no conflicts of interest.

References

1. Mangone L, Penna D, Marinelli F, Roncaglia F, Bisceglia I, et al. (2023) Incidence, mortality, and survival of hematological malignancies in Northern Italian patients: an update to 2020. Frontiers in Oncology, p. 13.
2. Pelcovits A, Niroula R (2020) Acute Myeloid Leukemia: A Review. Rhode Island Medical Journal 103(3): 38-40.
3. Arber D A, Orazi A, Hasserjian R P, Borowitz M J, Calvo K R, et al. (2022) International Consensus Classification of Myeloid Neoplasms and Acute Leukemias: integrating morphologic, clinical, and genomic data. Blood 140(11): 1200-1228.
4. Vakiti A, Reynolds S, Mewawalla P (2024) Acute Myeloid Leukemia. Stat Pearls Publishing.
5. Menghrajani K, Zhang Y, Famulare C, Devlin SM, Tallman MS, et al. (2020) Acute myeloid leukemia with 11q23 rearrangements: A study of therapy-related disease and therapeutic outcomes. Leukemia Research 98: 106453.
6. Patel D, Gupta M, Suppala P, Singh Hara M, Singh Daid SP, et al. (2024) AML-750 Global Burden and Trends in Acute Myeloid Leukemia in the European Union From 1990 to 2021: Insights from the Global Burden of Disease Study, 2021. Clinical Lymphoma Myeloma and Leukemia 24(S1): S337.
7. Juárez Salcedo L M, Desai V, Dalia S (2019) Venetoclax: evidence to date and clinical potential. Drugs in Context 8: 212574.
8. Griffioen M S, de Leeuw D C, Janssen J J W M, Smit L (2022) Targeting Acute Myeloid Leukemia with Venetoclax; Biomarkers for Sensitivity and Rationale for Venetoclax-Based Combination Therapies. Cancers 14(14): 3456.
9. AlAsmari A F, Alghamdi A, Ali N, Almeaikl M A, Hakami H M, et al. (2022a) Venetoclax Induces Cardiotoxicity through Modulation of Oxidative-Stress-Mediated Cardiac Inflammation and Apoptosis via NF-κB and BCL-2 Pathway. International Journal of Molecular Sciences 23(11): 6260.
10. Madarang E, Lykon J, Zhao W, Sekeres MA, Bradley T, et al. (2024) Venetoclax and hypomethylating agents in octogenarians and nonagenarians with acute myeloid leukemia. Blood Neoplasia 1(2): 100016.
11. Ahmad J, Thurlapati A, Thotamgari S, Grewal U S, Sheth A R, et al. (2022) Anti-cancer Drugs Associated Atrial Fibrillation—An Analysis of Real-World Pharmacovigilance Data. Frontiers in Cardiovascular Medicine 9: 739044.
12. Liu J, Chen Y, Yu L, Yang L (2022) Mechanisms of venetoclax resistance and solutions. Frontiers in Oncology, p. 12.
13. AbbVie Deutschland GmbH Co KG (2022, ottobre), Venclyxto (venetoclax) (2025) European Medicines Agency (n.d.). ANNEX I SUMMARY OF PRODUCT CHARACTERISTICS Venclyxto.
14. Qian S, Wei Z, Yang W, Huang J, Yang Y, et al. (2022) The role of BCL-2 family proteins in regulating apoptosis and cancer therapy. Frontiers in Oncology, p. 12.
15. Allani M, Akhilesh, Tiwari V (2024) Caspase-driven cancer therapies: Navigating the bridge between lab discoveries and clinical applications. Cell Biochemistry and Function 42(2): e3944.
16. Pollyea DA, DiNardo CD, Arellano ML, Pigneux A, Fiedler W, et al. (2022) Impact of Venetoclax and Azacitidine in Treatment-Naïve Patients with Acute Myeloid Leukemia and IDH1/2 Mutations. Clinical Cancer Research 28(13): 2753-2761.
17. Pollyea DA, Stevens BM, Jones CL, Winters A, Pei S, et al. (2018) Venetoclax with azacitidine disrupts energy metabolism and targets leukemia stem cells in patients with acute myeloid leukemia. Nature Medicine 24(12): 1859-1866.
18. Waggoner MM, Katsetos PCJ, Thomas MMPCE, Galinsky BMACI, Fox PCH (2022) Practical Management of the Venetoclax-Treated Patient in Chronic Lymphocytic Leukemia and Acute Myeloid Leukemia. Journal of the Advanced Practitioner in Oncology 13(4): 400-415.
19. Gargiulo P, Arenare L, Gridelli C, Morabito A, Ciardiello F, et al. (2021) Chemotherapy-induced neutropenia and treatment efficacy in advanced non-small-cell lung cancer: a pooled analysis of 6 randomized trials. BMC Cancer 21(1): 549.
20. Stilgenbauer S, Eichhorst B, Schetelig J, Hillmen P, Seymour JF, et al. (2018) Venetoclax for Patients with Chronic Lymphocytic Leukemia With 17p Deletion: Results from the Full Population of a Phase II Pivotal Trial. Journal of Clinical Oncology 36(19): 1973-1980.
21. Samuels C, Abbott D, Niemiec S, Tobin J, Falco A, et al. (2022) Evaluation and associated risk factors for neutropenia with venetoclax and obinutuzumab in the treatment of chronic lymphocytic leukemia. Cancer Reports 5(5): e1505.
22. DiNardo C D, Jonas B A, Pullarkat V, Thirman M J, Garcia J S, et al. (2020) Azacitidine and Venetoclax in Previously Untreated Acute Myeloid Leukemia. New England Journal of Medicine 383(7): 617-629.
23. Perino J, Mottal N, Bohbot Y, Servant V, Berroneau A, et al. (2020) Cardiac failure in patients treated with azacitidine, a pyrimidine analogue: Case reports and disproportionality analyses in Vigibase. British Journal of Clinical Pharmacology 86(5): 991-998.
24. Johnson I M, Bezerra E D, Farrukh F, McCullough K, Al Kali A, et al. (2022) Cardiac events in patients with acute myeloid leukemia treated with venetoclax combined with hypomethylating agents. Blood Advances 6(17): 5227-5231.
25. Aimo A, Castiglione V, Borrelli C, Saccaro L F, Franzini M, et al. (2020) Oxidative stress and inflammation in the evolution of heart failure: From pathophysiology to therapeutic strategies. European Journal of Preventive Cardiology 27(5): 494-510.
26. Roberts AW, Stilgenbauer S, Seymour JF, Huang DCS (2017) Venetoclax in Patients with Previously Treated Chronic Lymphocytic Leukemia. Clinical Cancer Research 23(16): 4527-4533.
27. Itchaki G, Brown J R (2016) The potential of venetoclax (ABT-199) in chronic lymphocytic leukemia. Therapeutic Advances in Hematology 7(5): 270-287.
28. Davids M S, Hallek M, Wierda W, Roberts A W, Stilgenbauer S, et al. (2018) Comprehensive Safety Analysis of Venetoclax Monotherapy for Patients with Relapsed/Refractory Chronic Lymphocytic Leukemia. Clinical Cancer Research 24(18): 4371-4379.
29. Silva F, Padín Iruegas M, Caponio V, Lorenzo Pouso A, Saavedra Nieves P, et al. (2022) Caspase 3 and Cleaved Caspase 3 Expression in Tumorogenesis and Its Correlations with Prognosis in Head and Neck Cancer: A Systematic Review and Meta-Analysis. International Journal of Molecular Sciences 23(19): 11937.
30. Tian S, Hirshfield KM, Jabbour SK, Toppmeyer D, Haffty BG, et al. (2014) Serum Biomarkers for the Detection of Cardiac Toxicity after Chemotherapy and Radiation Therapy in Breast Cancer Patients. Frontiers in Oncology, p. 4.
31. Kang YJ (2001) Molecular and cellular mechanisms of cardiotoxicity. Environmental Health Perspectives 109(S1): 27-34.
32. Brock R, Porpaczy E, Caron P, Skrabs C, Thalhammer R, et al. (2025) Changes in cardiac parameters during venetoclax treatment. Haematologica 110(10): 2503-2507.

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