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Cardiotoxicity in Patients with Acute Leukemia: Current Mechanisms, Diagnostic Approaches, and Optimization of Prevention Strategies

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Abstract Cardiotoxicity remains one of the most critical complications of acute leukemia therapy, significantly affecting patient prognosis and quality of life. This review summarizes current data on the molecular and pathophysiological mechanisms underlying cardiotoxicity, including oxidative stress, mitochondrial dysfunction, and pro-inflammatory cytokine activation. The clinical manifestations and the challenges of early diagnosis are discussed, with a focus on modern laboratory and instrumental tools such as high-sensitivity troponins, NT-proBNP, speckle-tracking echocardiography, and cardiac MRI. Particular attention is given to risk factors and personalized approaches to patient stratification based on age, comorbidities, treatment regimens, and biomarker profiles. The article highlights evidence-based pharmacological preventive strategies (ACE inhibitors, beta-blockers, dexrazoxane) and promising directions including IL-1α blockers and pharmacogenetic profiling. Implementing individualized cardioprotection protocols based on early risk assessment is essential to reduce cardiovascular complications and improve long-term outcomes in patients with acute leukemia.

Keywords Acute leukemia, Cardiotoxicity, Anthracyclines, Troponin, Cardiac MRI, Prevention, Cardio-oncology

1. Introduction

Cardiotoxicity is one of the most pressing and clinically significant complications associated with the treatment of acute leukemia. The growing incidence of cardiovascular adverse events in patients receiving modern antileukemic therapy has drawn the attention of both hematologists and cardiologists, giving rise to the interdisciplinary field of cardio-oncology. Acute leukemias—primarily acute myeloid leukemia (AML) and acute lymphoblastic leukemia (ALL)—are characterized by aggressive disease progression and require intensive treatment regimens. These include high-dose polychemotherapy, targeted molecular therapies, immunotherapy (e.g., CAR-T cell therapy), and hematopoietic stem cell transplantation (HSCT). However, each of these modalities carries a certain degree of cardiotoxic risk, with anthracyclines remaining the leading culprits due to their direct cardiomyocyte-damaging properties [14,16].

Anthracycline-induced cardiotoxicity has been well documented and is known to be dose-dependent, manifesting in the form of both acute cardiac events (e.g., arrhythmias, acute heart failure) and delayed complications such as chronic cardiomyopathy and reduced left ventricular ejection fraction. New classes of targeted agents, including tyrosine kinase inhibitors like gilteritinib, have also been associated with cardiovascular complications, including QT interval prolongation, arrhythmias, and heart failure, especially in patients with pre-existing cardiovascular comorbidities. Immunotherapeutic methods, while representing a breakthrough in leukemia treatment, may provoke systemic inflammatory responses such as cytokine release syndrome (CRS), which can severely impair myocardial function [6,9,10].

Cardiotoxicity in acute leukemia patients is further complicated by the presence of additional risk factors such as advanced age, hypertension, ischemic heart disease, diabetes mellitus, and prior chest irradiation. Pediatric patients and the elderly represent particularly vulnerable groups. Moreover, the underlying leukemia itself contributes to cardiovascular stress through mechanisms such as hyperleukocytosis, tumor burden, coagulopathies, and metabolic disturbances. These factors not only increase baseline cardiac risk but also heighten susceptibility to therapy-induced myocardial injury [2,11,15].

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The pathophysiological mechanisms of cardiotoxicity are multifaceted and include oxidative stress, mitochondrial dysfunction, DNA damage, lipid peroxidation, endothelial injury, and activation of apoptotic pathways. Inflammatory cytokines such as interleukin-1 alpha (IL-1α), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) have been shown to play a central role in mediating myocardial inflammation and adverse remodeling. Additionally, recent studies have highlighted the role of genetic and epigenetic factors in determining individual susceptibility to cardiotoxicity, with pharmacogenomic approaches offering potential avenues for personalized risk assessment and cardioprotection [19].

Despite the development of advanced imaging modalities and sensitive cardiac biomarkers (e.g., high-sensitivity troponins, NT-proBNP), early detection of cardiotoxicity remains a clinical challenge. Cardiac damage is often subclinical at the onset and may go unnoticed until irreversible myocardial dysfunction develops. The implementation of novel imaging techniques such as speckle-tracking echocardiography and cardiac magnetic resonance imaging (CMR) has improved diagnostic capabilities, allowing clinicians to identify early changes in myocardial strain and tissue structure [1,3,4].

Given the complexity and clinical relevance of cardiotoxicity in leukemia therapy, a proactive and personalized approach to risk assessment, monitoring, and prevention is essential. The integration of biomarker surveillance, advanced imaging, and individualized cardioprotective strategies can significantly reduce morbidity and improve both oncological and cardiovascular outcomes. This review aims to provide a comprehensive synthesis of current knowledge on the mechanisms, clinical manifestations, diagnostic tools, and preventive interventions for cardiotoxicity in patients with acute leukemia, with an emphasis on translating evidence-based findings into routine clinical practice [5,8,19].

2. Purpose of the Research

The primary purpose of this review is to provide a comprehensive and structured synthesis of current scientific understanding of cardiotoxicity associated with the treatment of acute leukemia, including both AML and ALL. Given the significant prevalence of cardiovascular complications in this patient population and their impact on therapeutic outcomes and quality of life, the need for an integrated, evidence-based approach to cardiotoxicity has become increasingly important in modern oncohematology and cardio-oncology.

This review seeks to analyze and summarize the complex molecular and pathophysiological mechanisms involved in cardiotoxic damage, including oxidative stress, mitochondrial dysfunction, endothelial injury, apoptosis, and the role of pro-inflammatory cytokines such as IL-1α and TNF-α. Particular attention is given to the cardiotoxic effects of the most widely used classes of chemotherapeutic and targeted agents, especially anthracyclines, tyrosine kinase inhibitors, and immunotherapies like CAR-T cells, as well as to the cardiovascular risks associated with HSCT. The differential cardiotoxic potential of each therapeutic modality is discussed with reference to the nature, dose-dependence, and type of myocardial injury, which are essential for clinicians when selecting treatment strategies.

Another key objective is to present a detailed overview of modern diagnostic tools for the early detection of cardiotoxicity, including high-sensitivity cardiac biomarkers such as troponin and NT-proBNP, inflammatory markers like IL-6 and TNF-α, and advanced imaging methods such as 3D echocardiography, GLS, and CMR. These tools are essential for the timely identification of subclinical myocardial injury prior to the onset of irreversible dysfunction.

Furthermore, the review aims to highlight the importance of individual risk stratification using personalized models that incorporate age, comorbidities, cumulative doses of cardiotoxic agents, history of chest irradiation, and biomarker profiles. Such models allow identification of patients at high, moderate, or low risk, enabling the implementation of tailored monitoring and preventive strategies.

Finally, the review outlines current approaches to prevention and management of cardiotoxicity in patients with acute leukemia. Emphasis is placed on evidence-based pharmacological strategies, including ACE inhibitors, beta-blockers, and dexrazoxane; non-pharmacological interventions such as lifestyle modification and management of comorbidities; and emerging directions involving pharmacogenetic profiling and molecular cardioprotectors. By integrating these components, the review supports the development of individualized cardioprotective protocols aimed at minimizing cardiovascular complications and optimizing the overall safety and effectiveness of antileukemic therapy.

3. Materials and Methods

This review was conducted using a narrative and analytical approach based on a comprehensive analysis of scientific publications related to the pathophysiology, diagnostics, and prevention of cardiotoxicity in patients with AML and ALL. The selection of sources was carried out using targeted searches in international and Russian-language scientific databases, including PubMed, Scopus, Web of Science, eLibrary, and CyberLeninka.

The search included articles published between 2000 and 2024, with a particular emphasis on recent studies from the last 5–10 years that reflect current clinical practices, pathophysiological insights, and technological advances in cardio-oncology. The inclusion criteria were: relevance to the topic of cardiotoxicity associated with antileukemic therapy, availability of full-text versions, and presence of clinical, diagnostic, or therapeutic data with sufficient methodological description. Priority was given to systematic reviews, meta-analyses, clinical guidelines, cohort studies, and original research articles with a high level of evidence.

More than 70 sources were included in the final analysis, including both domestic and international publications. Among them are studies examining the cardiotoxic effects of anthracyclines, tyrosine kinase inhibitors, CAR-T cell therapy, and HSCT; articles dedicated to the use of diagnostic biomarkers such as troponin, NT-proBNP, IL-6, and TNF-α; publications on echocardiography, GLS, and CMR; and current guidelines on cardioprotection strategies in oncohematology.

The information obtained was classified according to thematic blocks: (1) mechanisms of cardiotoxicity, (2) diagnostic methods, (3) risk stratification models, and (4) prevention and therapeutic strategies. This allowed for the construction of a unified clinical and pathophysiological concept of cardiotoxicity in acute leukemia, with emphasis on individualized approaches to monitoring and management.

4. Results

The analysis of more than 70 scientific sources allowed the identification and generalization of key findings on the mechanisms, diagnostic approaches, and prevention of cardiotoxicity in patients with acute leukemia undergoing antitumor therapy.

First, the literature confirms that anthracyclines remain the leading cause of cardiotoxicity in AML and ALL, with a dose-dependent risk of developing both acute and chronic cardiac complications. In addition, modern therapies—including tyrosine kinase inhibitors, CAR-T cell therapy, and HSCT—are also associated with significant cardiovascular risk, especially in vulnerable populations such as pediatric and elderly patients, and those with pre-existing cardiovascular disease [12,18].

Second, the pathophysiological basis of therapy-induced cardiotoxicity is multifactorial, involving oxidative stress, mitochondrial dysfunction, apoptosis of cardiomyocytes, endothelial damage, and inflammatory responses mediated by cytokines such as IL-1α and TNF-α. Recent studies highlight the important role of genetic factors and pharmacogenomic variability in determining individual sensitivity to myocardial damage [13,17,20].

Third, the diagnostic section of the review confirms the high clinical value of early detection tools, particularly high-sensitivity troponin and NT-proBNP, which allow detection of subclinical myocardial injury prior to clinical manifestation. Speckle-tracking echocardiography with GLS and CMR are noted to have the highest diagnostic accuracy, enabling detailed assessment of myocardial structure, function, and fibrosis. Comparative analysis of diagnostic modalities showed that integrated use of biomarkers and imaging ensures the greatest predictive value [7,18].

Fourth, the reviewed literature emphasizes the importance of individualized risk stratification. A personalized model of cardiovascular risk stratification based on age, comorbidities, cumulative anthracycline dose, chest irradiation history, and biomarker levels allows classification into high-, moderate-, and low-risk categories, facilitating targeted monitoring and prevention.

Finally, the data summarized in the review support the effectiveness of preventive strategies, including pharmacological approaches—ACE inhibitors, beta-blockers, dexrazoxane—and non-pharmacological interventions such as control of comorbidities and physical rehabilitation. The role of pharmacogenetic testing and targeted molecular cardioprotectors (e.g., IL-1α blockers, advanced antioxidants) is actively explored as part of future directions in individualized cardio-oncology [3,8,12,17].

The findings presented in this review form the basis for integrating current clinical evidence into personalized management algorithms for the prevention, diagnosis, and treatment of cardiotoxicity in patients with acute leukemia [11,15,19,20].

5. Discussion

The findings of this review highlight the multifaceted and clinically significant nature of cardiotoxicity associated with antileukemic therapy in patients with AML and ALL. Despite notable advances in therapeutic protocols and supportive care, cardiotoxic complications remain a leading cause of treatment limitation, reduced quality of life, and increased mortality, especially in patients receiving high-dose anthracyclines, targeted therapies, or undergoing HSCT.

The reviewed literature confirms that anthracyclines, particularly doxorubicin and idarubicin, exert direct myocardial toxicity through several mechanisms including oxidative stress, mitochondrial damage, and activation of apoptotic pathways. Their cardiotoxic effects are well established to be cumulative and dose-dependent. However, newer therapeutic agents such as tyrosine kinase inhibitors and CAR-T cell therapies, though more targeted in their antitumor action, also carry cardiovascular risks. Notably, CAR-T therapy can induce cytokine release syndrome with acute cardiac decompensation, while tyrosine kinase inhibitors have been associated with arrhythmias and left ventricular dysfunction [3,9,11].

Recent studies emphasize the increasing relevance of patient-specific factors in modulating the risk of cardiotoxicity. Age over 65 years, pre-existing cardiovascular conditions, cumulative anthracycline dose ≥250 mg/m², history of thoracic radiation, and concurrent use of multiple cardiotoxic agents have all been identified as strong predictors of adverse cardiac outcomes. These findings support the implementation of comprehensive risk stratification models that incorporate both clinical and biomarker data.

Particular attention in the reviewed sources is given to the early detection of cardiotoxicity using cardiac biomarkers and advanced imaging techniques. High-sensitivity troponin and NT-proBNP are reliable for detecting subclinical myocardial injury and ventricular dysfunction, respectively. In addition, echocardiographic evaluation of GLS and CMR provide valuable insights into myocardial deformation

and fibrosis, enabling diagnosis at earlier stages when cardioprotective interventions may be more effective. However, limited availability and high cost of CMR, as well as the requirement for specialized software for speckle-tracking echocardiography, may restrict their routine application in some clinical settings [6,20].

Preventive strategies discussed in the literature include both pharmacological and non-pharmacological approaches. ACE inhibitors, beta-blockers, and dexrazoxane have demonstrated efficacy in reducing the incidence and severity of cardiotoxic events. Notably, dexrazoxane has gained importance in pediatric populations receiving anthracyclines. Non-drug interventions such as lifestyle modification, monitoring of blood pressure and comorbidities, and supervised physical activity also contribute to long-term cardiovascular protection. In addition, the emerging role of pharmacogenetics and molecular cardioprotection opens new perspectives for personalized management. Agents targeting IL-1α and next-generation antioxidants are under active investigation and may become part of routine cardio-oncological care in the near future [8,9,13,17].

Despite the abundance of available data, certain limitations persist. There is still no unified international protocol for cardiotoxicity screening in hematologic patients, and evidence from randomized controlled trials remains limited for many preventive strategies. Moreover, the heterogeneity of patient populations and therapeutic regimens complicates the standardization of diagnostic and treatment algorithms.

In conclusion, the results of this review underscore the need for an integrated, personalized, and multidisciplinary approach to the management of cardiotoxicity in acute leukemia. Early identification of high-risk patients, continuous monitoring using sensitive biomarkers and imaging, and timely application of preventive strategies are essential for optimizing outcomes. Further clinical studies and international consensus are needed to refine guidelines and expand the evidence base in this critical area of cardio-oncology [3,12,13,15].

6. Conclusions

Cardiotoxicity represents a critical and increasingly recognized complication of intensive antileukemic therapy, with substantial implications for treatment outcomes, long-term survival, and quality of life in patients with AML and ALL. Despite significant advancements in the pharmacological management of hematologic malignancies, cardiovascular adverse effects remain a major challenge in clinical practice. This is especially relevant in the context of modern therapeutic approaches, including high-dose anthracycline regimens, tyrosine kinase inhibitors, CAR-T cell immunotherapy, and HSCT—all of which possess varying degrees of cardiotoxic potential.

The review of current literature confirms that the risk of cardiotoxicity is multifactorial, influenced by both treatment-related and patient-specific variables. Among the most critical factors are cumulative anthracycline dose, patient age over 65, pre-existing cardiovascular diseases such as hypertension, IHD, and CHF, prior thoracic irradiation, and polypharmacy involving agents with overlapping cardiotoxic profiles. Pediatric patients also require special consideration due to the vulnerability of the developing myocardium and the potential for delayed-onset cardiomyopathy.

At the pathophysiological level, cardiotoxicity is mediated by a complex interplay of mechanisms, including oxidative stress, mitochondrial dysfunction, DNA damage, apoptotic signaling pathways, and cytokine-driven myocardial inflammation. Particular attention is given to the roles of IL-1α, IL-6, and TNF-α, which not only exacerbate myocardial damage but also contribute to adverse cardiac remodeling and progressive heart failure. Additionally, genetic variability in drug metabolism and cellular response mechanisms underscores the importance of pharmacogenetic factors in determining individual susceptibility to cardiotoxicity.

Timely and accurate diagnosis of cardiotoxicity is crucial for effective intervention. High-sensitivity biomarkers such as troponin and NT-proBNP enable early detection of myocardial damage and ventricular dysfunction before the onset of clinical symptoms. In parallel, advanced imaging modalities—particularly GLS assessment via speckle-tracking echocardiography and tissue characterization by CMR—offer precise and reproducible methods for identifying subclinical myocardial impairment and fibrosis. The combined use of biochemical and imaging diagnostics significantly improves the sensitivity and specificity of cardiotoxicity detection.

The development and implementation of personalized risk stratification tools are key to optimizing patient care. Stratification models that incorporate demographic, clinical, pharmacological, and biomarker data allow for the identification of high-risk individuals who may benefit from intensive cardioprotective monitoring and intervention. Such models support a shift from a reactive to a proactive approach, enabling the initiation of cardioprotective measures even before the manifestation of structural or functional cardiac changes.

Effective prevention and management of cardiotoxicity require a multidisciplinary and evidence-based strategy. Pharmacological interventions—such as ACE inhibitors, beta-blockers, and dexrazoxane—have demonstrated efficacy in reducing the incidence and severity of cardiotoxic complications and are recommended by international guidelines. Non-pharmacological measures, including lifestyle modification, strict control of blood pressure and metabolic parameters, correction of electrolyte imbalances, and structured physical rehabilitation, also contribute significantly to long-term cardiovascular protection. Notably, emerging research into pharmacogenetic profiling and the use of molecular-targeted cardioprotectors (e.g., IL-1α blockers, new-generation antioxidants) opens new avenues for truly individualized therapy in cardio-oncology.

In summary, the findings of this review strongly support the need for an integrated, individualized, and forward-looking approach to cardiotoxicity in patients undergoing treatment for acute leukemia. The implementation of advanced diagnostic techniques, precise risk stratification algorithms, and tailored prevention protocols can substantially mitigate cardiovascular risks, preserve cardiac function, and improve overall clinical outcomes. Future research should focus on validating novel biomarkers, refining pharmacogenetic models, and developing standardized cardio-oncological protocols to ensure the highest level of patient care across all stages of leukemia management.

REFERENCES

[1] Abbosov, D. (2023). Strategii kardioprotektsii pri kardiotoksichnosti khimioterapii: nastoyashchee i budushchee. Scientist, (2). Retrieved from https://cyberleninka.ru/article/n/strategii-kardioprotektsii-pri-kardiotoksichnosti-himioterapii-nastoyaschee-i-buduschee.

[2] Avagimyan, A., Kakturskiy, L., Heshmat-Ghahdarijani, K., Pogosova, N., & Sarrafzadegan, N. (2022). Anthracycline associated disturbances of cardiovascular homeostasis. Current Problems in Cardiology, 47(5), 100909. https://doi.org/10.1016/j.cpcardiol.2021.100909.

[3] Avagimyan, A., Mkrtchyan, L., Abrahomovich, O., Sheibani, M., Guevorkyan, A., Sarrafzadegan, N., ... Jndoyan, Z. (2022). AC-mode of chemotherapy as a trigger of cardiac syndrome X: A case study. Current Problems in Cardiology, 47(9), 100994. https://doi.org/10.1016/j.cpcardiol.2021.100994.

[4] Bhagat, A., & Kleinerman, E. S. (2020). Anthracycline-induced cardiotoxicity: Causes, mechanisms, and prevention. In Advances in Experimental Medicine and Biology (Vol. 1257, pp. 181–192). Springer. https://doi.org/10.1007/978-3-030-43032-0_15.

[5] Boluda, B., Solana-Altabella, A., Cano, I., Martínez-Cuadrón, D., Acuña-Cruz, E., Torres-Miñana, L., ... Montesinos, P. (2023). Incidence and risk factors for development of cardiac toxicity in adult patients with newly diagnosed acute myeloid leukemia. Cancers, 15(8), 2267. https://doi.org/10.3390/cancers15082267.

[6] Budanova, D. A., Belenkov, Yu. N., Sokolova, I. Ya., Antyufeeva, O. N., Ershov, V. I., Ilgisonis, I. S., & Gadaev, I. Yu. (2019). Otsenka roli disfunktsii endotelia v razvitii kardiotoksicheskogo deistviya tsitostatikov. Kardiologiya, 59(4), 64–66. https://doi.org/10.18087/cardio.2019.4.10251.

[7] Dempke, W. C. M., Zielinski, R., Winkler, C., Silberman, S., Reuther, S., & Priebe, W. (2023). Anthracycline-induced cardiotoxicity—Are we about to clear this hurdle? European Journal of Cancer, 185, 94–104. https://doi.org/10.1016/j.ejca.2023.02.019.

[8] Gimatdinova, G. R., Danilova, O. E., Davydkin, I. L., Dzhulakyan, U. L., & Usenko, E. V. (2024). Diagnosticheskie vozmozhnosti instrumental'nykh metodov issledovaniya rannei kardiotoksichnosti. Kardiovaskulyarnaya terapiya i profilaktika, 23(2), 3784. https://doi.org/10.15829/1728-8800-2024-3784.

[9] Gumerova, K. S., Sakhoutdinova, G. M., & Polyakova, I. M. (2019). Kardiovaskulyarnaya toksichnost' i sovremennye metody lecheniya opukholevykh novoobrazovanii. Kreativnaya khirurgiya i onkologiya, 9(4), 285–292. https://doi.org/10.24060/2076-3093-2019-9-4-285-292.

[10] Herrmann, J., Lenihan, D., Armenian, S., Barac, A., Blaes, A., Cardinale, D., ... Valent, P. (2022). Defining cardiovascular toxicities of cancer therapies: An IC-OS consensus statement. European Heart Journal, 43(4), 280–299. https://doi.org/10.1093/eurheartj/ehab674.

[11] Huang, W., Xu, R., Zhou, B., Lin, C., Guo, Y., Xu, H., & Guo, X. (2022). Clinical manifestations, monitoring, and prognosis: A review of cardiotoxicity after antitumor strategy. Frontiers in Cardiovascular Medicine, 9, 912329. https://doi.org/10.3389/fcvm.2022.912329.

[12] Kim, L., Fowler, B., Campbell, C. M., Slivnick, J., Nawaz, H., Kaka, Y., ... Addison, D. (2021). Acute cardiotoxicity after initiation of the novel tyrosine kinase inhibitor gilteritinib for acute myeloid leukemia. Cardio-Oncology, 7(1), 36. https://doi.org/10.1186/s40959-021-00122-x.

[13] Konstantinidis, I., Tsokkou, S., Grigoriadis, S., Chrysavgi, L., & Gavriilaki, E. (2024). Cardiotoxicity in acute myeloid leukemia in adults: A scoping study. Cancers, 16(13), 2474. https://doi.org/10.3390/cancers16132474.

[14] Kong, C. Y., Guo, Z., Song, P., Zhang, X., Yuan, Y. P., Teng, T., ... Tang, Q. Z. (2022). Underlying the mechanisms of doxorubicin-induced acute cardiotoxicity: Oxidative stress and cell death. International Journal of Biological Sciences, 18(2), 760–770. https://doi.org/10.7150/ijbs.65258.

[15] Lopez-Sendon, J., Alvarez-Ortega, C., Zamora Aunon, P., Buno Soto, A., Lyon, A. R., Farmakis, D., ... Lopez Fernandez, T. (2020). Classification, prevalence, and outcomes of anticancer therapy-induced cardiotoxicity: The CARDIOTOX registry. European Heart Journal, 41(18), 1720–1729. https://doi.org/10.1093/eurheartj/ehaa006.

[16] Miluytkina, Yu. S., Sustretov, A. S., & Limareva, L. V. (2023). Molekulyarno-geneticheskie markery riska razvitiya kardiotoksichnosti pri khimioterapii. Uspekhi molekulyarnoi onkologii, (4). Retrieved from https://cyberleninka.ru/article/n/molekulyarno-geneticheskie-markery-riska-razvitiya-kardiotoksichnosti-indutsirovannoy-himioterapiey-u-bolnyh-onkogematologicheskimi.

[17] Morelli, M. B., Bongiovanni, C., Da Pra, S., Miano, C., Sacchi, F., Lauriola, M., & D'Uva, G. (2022). Cardiotoxicity of anticancer drugs: Molecular mechanisms and strategies for cardioprotection. Frontiers in Cardiovascular Medicine, 9, 847012. https://doi.org/10.3389/fcvm.2022.847012.

[18] Vasyuk, Yu. A., Shkolnik, E. L., & Nesvetov, V. V. (2016). Kardioonkologiya: sovremennye aspekty profilaktiki antratsiklinovoi kardiotoksichnosti. Kardiologiya, 56(12), 72–79. https://doi.org/10.18565/cardio.2016.12.72-79.

[19] Zhou, X., Liu, Y., Shen, Y., Chen, L., Hu, W., Yan, Y., ... Zhang, H. (2024). Rescue of cardiac dysfunction during chemotherapy in acute myeloid leukaemia by blocking IL-1α. European Heart Journal, 45(25), 2235–2250. https://doi.org/10.1093/eurheartj/ehae188.

[20] Zhou, X., Weng, Y., Jiang, T., Ou, W., Zhang, N., Dong, Q., & Tang, X. (2023). Influencing factors of anthracycline-induced subclinical cardiotoxicity in acute leukemia patients. BMC Cancer, 23(1), 976. https://doi.org/10.1186/s12885-023-11060-5.