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# Changes in the Cardiovascular System in Patients with Acute Leukemia after the Use of Polychemotherapy

Amerova Dilafruz Abdikhalimovna[1], Isametdinova Zebo Salokhetdinovna[2]
[1]*Assistant teacher of the department of Hematology, Samarkand State Medical University*
[2]*Therapist of Republican Scientific Center for Emergency Medical Services*

***Abstract:*** ***This article examines the seasonal changes in the vascular system and the problems that arise in this process in a patient with leukemic flow after polychemotherapy. Also, in the article, the results of the analysis of the cardiotoxic effects of polychemotherapy in patients with acute leukemia are cited and discussed.***
***keywords: cardiotoxic effect, cardiovascular system, clinical manifestations, drugs, leukemia, organotoxic complications, polychemotherapy.***

## I. INTRODUCTION

In the structure of critical conditions associated with polychemotherapy (PCT), an important place is given to: cardiovascular failure, coagulopathies, acute respiratory failure, central nervous system disorders, acute renal failure, liver failure. Organotoxic complications of PCT were diagnosed in 82.2% of patients with acute lymphoblastic leukemia; toxicity grades I and II predominated. In the first place, damage to the nervous system was noted (45.6%), in second place was damage to the heart (19.7%), and in third place was damage to the liver (14.8%).

## II. LITERARY REVIEW AND METHODOLOGY

The cardiotoxic effect of PCT occurs mainly with the use of anthracyclines (adriamycin, rubomycin) and less often with the use of other drugs (cyclophosphamide, 5-fluorouracil, etoposide, etc.) [6-10].

The toxic effect on the myocardium of anthracycline antibiotics used in the chemotherapy of malignant tumors (adriamycin, doxorubicin, etc.) in most cases manifests itself as chronic cardiomyopathy. Both in its morphological substrate (widespread irreversible degeneration of cardiomyocytes with replacement sclerosis) and in its clinical manifestations, it resembles idiopathic dilated cardiomyopathy. Such myocardial damage usually develops 3–6 (up to 12) months from the start of treatment after receiving a total dose of chemotherapy of at least 500 mg/m2 and is refractory to the therapy. Less commonly, anthracycline antibiotics cause acute myocarditis, in some cases accompanied by pericarditis [11].

Morphological examination of the myocardium reveals lymphocytic infiltration and foci of cardiomyocyte necrosis. Such myocarditis usually occurs soon after the administration of chemotherapy and, unlike chronic cardiomyopathy, undergoes complete reversal after their withdrawal, usually without recurrence when cytostatic treatment is resumed [11].

The cardiotoxicity of anthracyclines is dose-dependent and is caused by excess Ca2+ in the sarcoplasmic reticulum. With a total dose of adriamycin less than 400 mg/m2, the incidence of cardiomyopathy is 0.14%, with a dose of 550 mg/m2 – 7%, and if the dose exceeds 700 mg/m2 – the probability increases to 18%. Synergistic cardiotoxic effect

with rubromycin vincristine exhibits. Cardiotoxicity of chemotherapy is more often observed in old age. Signs of myocarditis are: rapid heartbeat, shortness of breath, enlarged heart, poor circulation. Clinical manifestations of cardiotoxicity (cardialgia, tachycardia, rhythm disturbance) are more often observed in patients with acute and chronic anthracycline-induced inhibition of cardiac contractile function (50% and 63%, respectively) compared with patients without changes in left ventricular contractility in response to doxorubicin administration ( 20%) [9].

The criterion for early and late chronic cumulative cardiotoxicity of cytostatic chemotherapy in patients with malignant neoplasms is a decrease in the left ventricular ejection fraction by 10% or more, established using radionuclide ventriculography and/or ECG-synchronized perfusion tomoscintigraphy [6,13-15].

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Acute cardiotoxicity with rapid intravenous administration of the drug is manifested by vasodilation, hypotension, and cardiac arrhythmias [10]. Subacute complications are characterized by myocarditis and pericarditis.

Chronic cardiotoxicity in the form of dilated cardiomyopathy is reported in 4.5–7.0% of patients, usually at the end of the course of treatment or shortly after its completion. In recent years, the possibility of later development of this complication has been reported [3].

A severe course of doxirubicin-induced cardiomyopathy was described, which developed 15 years after children were cured of cancer, while there were no signs of heart damage in the immediate period after treatment. Patients suddenly, with virtually no warning, experience severe left ventricular heart failure. Morphological signs of late cardiotoxic effects.

– cardiac dilatation, intramural thrombosis, reduction in the number and lysis of muscle fibers, interstitial edema and fibrosis.

The earliest manifestations of the cardiotoxic effect of PCT include: hypotension, tachycardia, cardialgia, rhythm disturbance. Later symptoms of cardiotoxicity arise due to damage to the heart muscle and rhythm disturbances. Sometimes myocardial infarction may occur [7-9].

In patients suffering from systemic blood diseases, myocarditis is often encountered in practice. The toxic effect of drugs on the myocardium is realized as eosinophilic myocarditis (aminophyllini, chloramphenicol) [6].

Lymphocytic myocarditis has also been described in isolated cases of therapy with 5-fluorouracil and cyclophosphamide. These drugs, however, more often cause coronary necrosis of the myocardium due to coronary spasm, damage to the microvasculature and hypercoagulation. Clinically, various types of arrhythmias are observed, which can cause sudden death, heart failure and cardiogenic shock [2].

In children with ALL at different stages of polychemotherapy, various clinical symptoms (arterial dystonia, cardialgia, functional systolic murmur, etc.) and electrocardiographic signs of cardiovascular disorders were identified, characterized at the onset of the disease by nomotopic disorders of automaticity, recorded in 21.0% of cases by the predominance of process disorders repolarization in the LV myocardium during therapy and after its completion in 63.0% and 48.0% of cases, respectively [3].

## III. DISCUSSION AND RESULTS

Over the past five years, it has been shown that myocardial damage can occur after exposure to almost all infectious agents. However, the frequency of detection of individual pathogens varies. In the European population and among residents of the USA and Canada, the most common etiological causes of myocarditis are adenoviruses and enteroviruses, including coxsackie viruses. The most common viral genomes identified in myocardial biopsies were parvovirus, B-19, and human herpes virus-6.

The incidence of myocarditis in patients affected by the immunodeficiency virus in the era before the introduction of highly effective antiviral therapy was 50% [6]. The role of infectious agents in the development of myocarditis was verified in 61.2% of cases, collagenosis as a cause of myocarditis was noted in 14.3% of cases, a “systemic” allergic reaction developed in 5.6% of patients, in 3.7% of cases myocarditis developed in patients with burn disease. It has been established that bacterial myocarditis develops much less frequently than viral myocarditis. The most important are intracellular microorganisms – Chlamydia. Infection with this pathogen reaches 50% [5]. Among other microorganisms, corynebacterium diphtheria, haemophilusin fluenzae, legionella pneumophila, mycobacterium tuberculosis, streptococcus A, etc. have a significant effect on the incidence of myocarditis [4].

The use of trimetazidine at a dose of 35 mg 2 times a day in patients with lymphoproliferative diseases during treatment with the anthracycline antibiotic doxorubicin helps prevent the manifestation of signs of HF, worsening LV diastolic dysfunction and impaired renal filtration function [7].

Clinically, retinoid syndrome is manifested by respiratory disorders in 84% of patients, pulmonary edema – in 54%, fever – in 81%, pleural and/or pericardial effusion – in 36%, hypotension – in 18%, bone pain – in 14% and headaches – in 14%, heart failure – in 11%, acute renal failure – in 11%, specific infiltrates in the skin, muscles, and fundus [5]. Patients suffering from acute leukemia in combination with myocarditis are characterized by symptoms of a viral infection, accompanied by fever, myalgia and respiratory and gastrointestinal symptoms. The ECG revealed characteristic indicators: ST segment elevation in 2 consecutive leads - in 54% of cases, negative T wave - in 27%, ST segment depression - in 18% of cases, pathological Q wave - in 27% [6].

Modern diagnosis of myocarditis includes a number of laboratory and instrumental research methods. In a clinical blood test, ESR values may appear above normal, leukocytosis and eosinophilia. During the study of myocarditis, it was revealed that the levels of interleukin-10, interleukin-12, tumor necrosis factor α, interferon γ increase significantly. However, the prognostic role of this increase was unclear. It is currently accepted that in cases of acute myocarditis, interleukin-10 and tumor necrosis factor α reach values statically higher than in patients with acute myocardial infarction (AMI); In addition, the level of interleukin-10 has a prognostic value: the higher the level, the more likely an unfavorable prognosis is.

On the ECG, a frequent change is the formation of a negative T wave, changes in the ST segment, which puts the differential diagnosis with acute myocardial infarction in the first place [6].

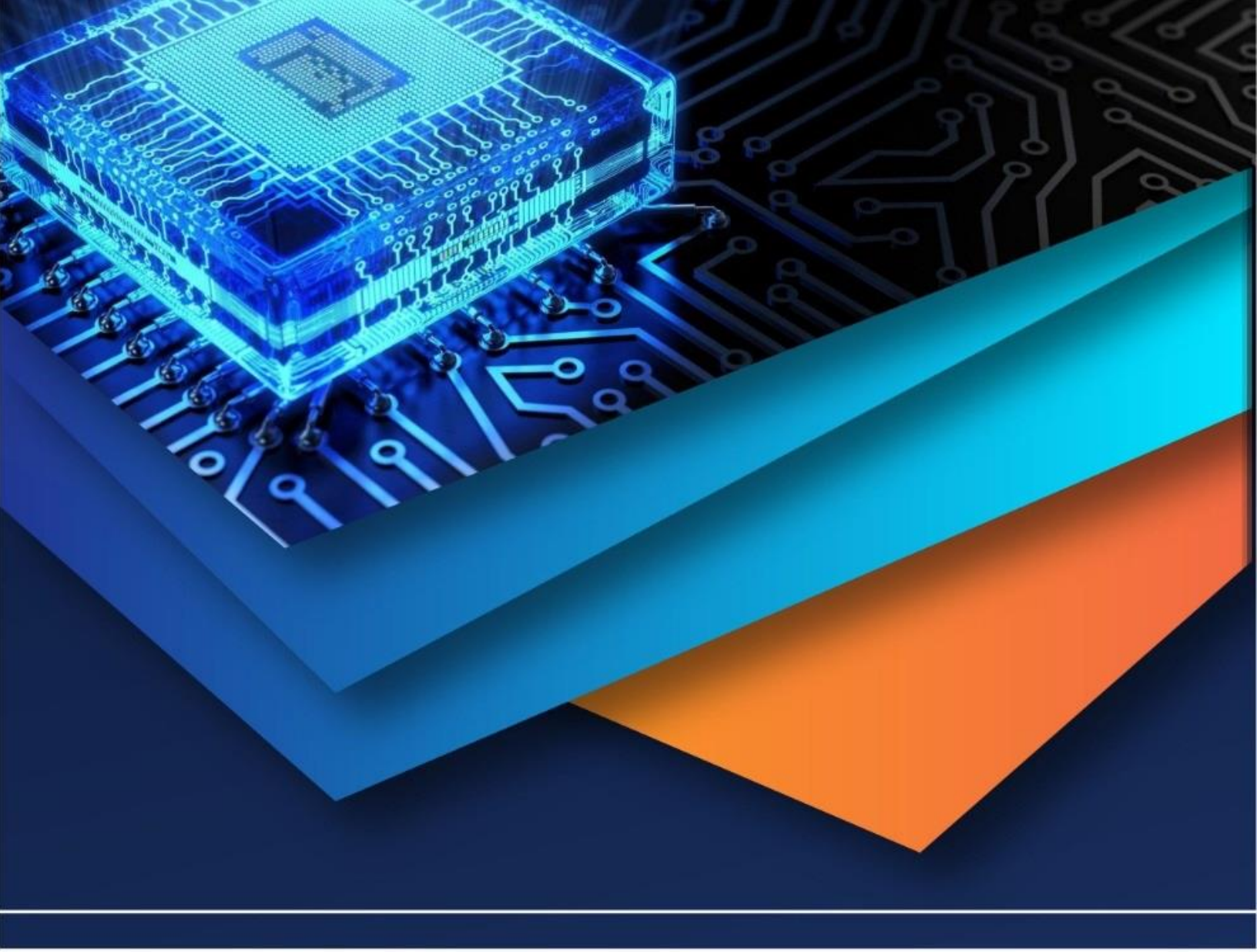
A chest x-ray shows an increase in heart size. Cardiac echocardiography is a fairly informative diagnostic method for myocarditis. This method allows you to see a violation of the contractility of the heart, an increase in the cavities of the heart and an increase in intracardiac pressure. A violation of the contraction of a portion of the myocardium – hypokinesis – is often detected. Registration of diastolic dysfunction during Doppler measurements after the first administration of anthracycline antibiotics should be considered as an early manifestation of the cardiotoxic effect of cytostatics [2].

Manifestations of cardiotoxicity in the form of rhythm disturbances of grades III and IV (multifocal ventricular extrasystole, ventricular tachycardia) require Holter monitoring and antiarrhythmic therapy. When cardiac dysfunction of degrees II and III appears during chemotherapy, in addition to the traditional prescription of cardiac glycosides, diuretics, angiotensin-converting enzyme antagonists, and potassium preparations, we consider it advisable to use mildronate, taking into account its antioxidant and cardioprotective properties [9].

In most cases, practicing doctors do not pay due attention when recognizing the cardiotoxicity of chemotherapy drugs or when formulating a diagnosis - they are not considered as a nosological unit. Thus, at various times when polychemotherapy is prescribed and completed in patients with leukemia, it is necessary to promptly identify signs of cardiotoxic myocardial damage and carry out accompanying therapy, as well as joint observation by a hematologist and cardiologist.

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