

who received cardiotoxic treatment require special long-term attention and follow-ups (2-5). Although there is a growing base of knowledge about cardiotoxicity with classical cytostatics, targeted (biological) hormonal treatment and tumour immunotherapy, anthracycline cardiotoxicity is the most studied and understood – acute, chronic (within 1 year after treatment) and late (after 1 year or even several decades after treatment) (6-8). Despite the irreversible anthracycline-induced loss of cardiomyocytes, the heart is able to compensate this damage with no clinical manifestations for many years. As the risk factors accumulate and stressful situations increase (e.g., viral infections, excessive physical activity, increased somatic growth, pregnancy, surgeries), compensatory mechanisms cease to be effective and structural myocardial damage progresses to systolic dysfunction and clinical heart failure (1, 5, 9, 10).

Case analysis

We report a case of a 31-year-old male patient of T-cell non-Hodgkin lymphoma. He was diagnosed at the age of 4 years in 1991. He underwent diagnostic thoracotomies followed by chemotherapy (doxorubicin (cumulative dose of 230 mg/m²), cyclophosphamide, vincristine, L-asparaginase, methotrexate, mercaptopurine, cytarabine, teniposide, bleomycin (total dose of 22.5 mg/m²), prednisone) of T-cell non-Hodgkin lymphoma. He achieved long-term remission and has been monitored by his oncologist. He was also diagnosed with genetically confirmed, asymptomatic CADASIL syndrome (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) and hypothyroidism without the need for substitution therapy. In April 2019, he presented with a two months of non-productive cough, night sweats, decreased appetite, fatigue and dyspnea on exertion (after a walking a few meters). He had no chest pain and no peripheral edema. The initial physical exam demonstrated clear lungs and no evidence of edema. The laboratory evaluation showed elevated NT-proBNP (1423 ng/l). Serological examination (Chlamydia pneumoniae, Mycoplasma pneumoniae) was negative. Microbiological examination (nasal swabs/tonsils/urine/sputum) revealed no infectious agent. Chest X-ray demonstrated pulmonary venous congestion. Transthoracic echocardiogram was obtained and revealed severely decreased left ventricular systolic function. The estimate left ventricular ejection fraction was 10%. There was global left ventricular hypokinesis. Right

ventricular function was normal. The estimated right ventricular systolic pressure was elevated (50 mmHg) consistent with moderate pulmonary hypertension. (Tab. 1). PET-CT scan did not show any evidence of lymphoma recurrence. Cardiomegaly and minimal fluidopericardium were seen. We obtained these results of previous echocardiographic examinations for comparison:

- (2010): left ventricle slightly dilated, with preserved systolic and diastolic function, with diffuse hypokinesia, left ventricular ejection fraction 50-55%. Valves with good kinetics, sufficient.
- (2016): neither dilatation nor left ventricular hypertrophy, which is without significant kinetics or global systolic dysfunction, was confirmed. No hemodynamically significant valve or short-circuit disorder, left ventricular ejection fraction (LVEF) of 54%.

Based on the case history and results of the examinations, we diagnosed the condition as dilated cardiomyopathy, most likely on the basis of late anthracycline toxicity. In April 2019, at the beginning of hospitalization, we administered the beta-blocker bisoprolol for sinus tachycardia and, after the first echocardiographic examination, we also indicated the ACE inhibitor perindopril, the diuretic furosemide and the selective aldosterone blocker eplerenone. Due to symptomatic manifestations of hypotension (dizziness, collapse conditions) and worsening of renal parameters, we had to discontinue both the beta-blocker and the ACE inhibitor, titrate the dose of diuretics and indicate the selective sinoatrial node inhibitor ivabradine. The patient's condition improved during the above treatment and the patient was discharged to outpatient care. The patient then adhered to a regime of restricted of physical activity and stress and after consultation with experts of the National Institute for Cardiovascular Diseases, he was monitored by a cardiologist. The patient was taking atorvastatin (for both higher total and LDL cholesterol), eplerenone, furosemide, trimetazidine, ivabradine and 1 month after being discharged (May 2019) he underwent echocardiography, that again, showed severe diffuse hypokinesis with the above valve defects, but with an increase in left ventricular ejection fraction from 10% to 25%. In August 2019, another echocardiography showed the same LVEF of 25% and concluded: dilated cardiomyopathy with left ventricular failure. Cardiac symptoms (dyspnoea) occurred mainly under high stress levels. In early October 2019, CT coronary angiography was performed where no atherosclerotic changes in the

Tab. 1. Selected echocardiographic indicators

	2010	2016	4/2019	5/2019	10/2019	12/2020	5/2021
Diameter of the left ventricle on diastole	—	53	60	63	57	59	60
Ejection fraction	50-55	54	15	25	30	25	30
Septa thickness	—	—	10	10	10	9	9
Mitral regurgitation (degree)	0	0	2	2	1	1	1
Tricuspid regurgitation (degree)	0	0	1	1	0	0	0
Tricuspid regurgitation gradient	—	—	40	30	—	—	—
Estimation of pulmonary vein systolic pressure	—	—	50	35	—	—	—
Left atrium	—	33	43	37	37	36	36