A 56-year-old woman was hospitalised in the Department of Hypertensiology due to pericardial effusion revealed in routine echocardiographic examination. First hospitalisation: The patient reported malaise and exercise intolerance for 18 months. On physical examination regular cardiac function, 80/min with proper accentuation and blood pressure 143/91 mm Hg was seen. Comorbidities: Hypothyroidism from 8 years (during the last year TSH varied from 22.93 to 41.42 µIU/mL) and well-controlled hypertension. In ECG: Sinus rhythm, negative T wave in V1. In echocardiography: Echo-free space was distinguished behind the lateral wall of the left ventricle (LV) in the amount of 2.0–2.5 cm, and behind the posterior wall: 1.5–1.9 cm (Fig. 1). The sizes of heart cavities and systolic function (LVEF = 65%) were correct. Meagre tricuspid regurgitation was recorded (RVSP ~38 mm Hg). The inferior vena cava was undulated with correct respiratory mobility. Significant variability of flows through the atrioventricular valves during respiration was seen. Slight pressure on the right atrium and its collapse during ventricular contraction was observed. Right ventricular (RV) diastolic collapsing and paradoxical movement of the intraventricular septum were not distinguished. Despite significant pericardial effusion, symptoms of tamponade were not observed. At discharge, pericardial effusion decreased slightly (Fig. 2).

Laboratory results: Elevated levels of anti-thyroid peroxidase and antithyroglobulin antibodies; TSH = 1.7 µIU/mL within normal range. Treatment: Methylprednisolone (16 mg/day) in gradually decreasing dose, colchicine (1 mg/day), levothyroxine (175/200 µg), and perindopril. The patient was recommended to take these drugs after discharge. Control hospitalisation. After 6 weeks the patient did not report any complaints. In echocardiography: Echo-free space was distinguished behind the lateral wall of the LV in the amount of 2.1–2.6 cm (Figs. 3, 4), thus significant decrease of effusion did not occur. Pericardial thickening with echo enhancement in the area of tricuspid annulus and RV were found. LV filling profile and inflow from the pulmonary veins were correct. Laboratory results: Decreased level of TSH (0.2 µIU/mL); erythrocyte sedimentation rate, C-reactive protein, and procalcitonin were normal. Magnetic resonance imaging (MRI) of the heart revealed a substantial quantity of partially organised fluid in the pericardial sac (Fig. 5). The maximum quantity of fluid occurred behind the LV (26 mm). Over the RV and partially over the LV, pericardial visceral lamina thickening (up to 9 mm) and calcifications were distinguished. Based on MRI, constrictive pericarditis was diagnosed.

Conclusions: Our patient suffered from long-lasting hypothyroidism. This is the reason why we did not see a rapid decrease in pericardial effusion despite adequate therapy. Proper thyroid replacement therapy causes a slow process of reabsorption of pericardial effusions. According to literature, pericardial effusion incidence in hypothyroidism amounts to 3–6%. Tamponade and obstructive pericarditis occur very rarely in these patients. Echocardiographic assessment of pericardial thickening is troublesome due to the phenomenon of complete ultrasound bouncing from pericardium (between the heart muscle and lung tissue), so MRI is a powerful tool to establish constrictive pericarditis diagnosis.