CLINICAL VIGNETTE

Speckle tracking and global longitudinal strain in endomyocardial infiltration during FIP1L1-PDGFRA chronic eosinophilic leukaemia

Andrzej Tomasik, Edyta Nowak-Radzik, Katarzyna Pigoń, Grzegorz Banasik, Ewa Nowalany-Kozielska

2nd Department of Cardiology, Medical Faculty with Dentistry Division in Zabrze, Medical University of Silesia, Katowice, Poland

A 29-year-old male patient was admitted to hospital with severe decompensated congestive heart failure (HF) with pleural effusion, ascites, peripheral oedema, and worsening dyspnoea in New York Heart Association class IV. He had already been diagnosed with hypereosinophilic syndrome (HES) with endomyocardial fibrosis, irreversible pulmonary hypertension, and chronic atrial fibrillation (AF). Previous diagnostic work-up confirmed chronic eosinophilic leukaemia with fusion gene FIP1L1-PDGFRA as the underlying cause of HES, and the patient was successfully treated with imatinib, although HF rapidly worsened. Endomyocardial fibrosis was diagnosed using magnetic resonance imaging with late gadolinium enhancement in left ventricular (LV) apical and mid-posterior regions. Electrocardiographic examinations revealed AF and right ventricular hypertrophy (Fig. 1A). Echocardiography showed cardiomegaly (**Suppl. Video 1 — see journal website**) with normal LV systolic function biplane, LV ejection fraction 59%, significant mitral regurgitation (Fig. 1B, **Suppl. Video 2 — see journal website**), and significant tricuspid regurgitation (**Suppl. Video 3 — see journal website**) with a pressure gradient of 73 mmHg (Fig. 1C). LV diastolic function was restrictive with monophasic transmitral flow with E wave of 112 cm/s, medial e' wave of 5 cm/s, and E/e' med ratio equal to 22.4. Two-dimensional speckle tracking analysis revealed a severe segmental decline of longitudinal strain within anterior, lateral, and posterior walls with global longitudinal strain reduced to –14.2% (Fig. 1D). After intravenous diuretics and inotropic drugs, the patient's condition has improved and he lost 8 kg of weight. He was discharged home on oral treatment, and a readmission for right heart catheterisation and cardiac transplant evaluation was scheduled. However, before this could happen the patient unexpectedly died without witness. We have to consider imatinib cardiotoxicity as one of the presumed underlying causes of rapid HF progression [1, 2]. Myocardial cells may be extremely susceptible to cardiotoxic properties of tyrosine kinase inhibitors in patients with pulmonary hypertension [3]. This is the second report on the use of speckle tracking echocardiography in chronic eosinophilic leukaemia with cardiac involvement [4]. Similarities in distribution of disturbed myocardial strain and reduced global longitudinal strain are evident. Such casuistic reports may lead to better understanding and care of these unique patients.

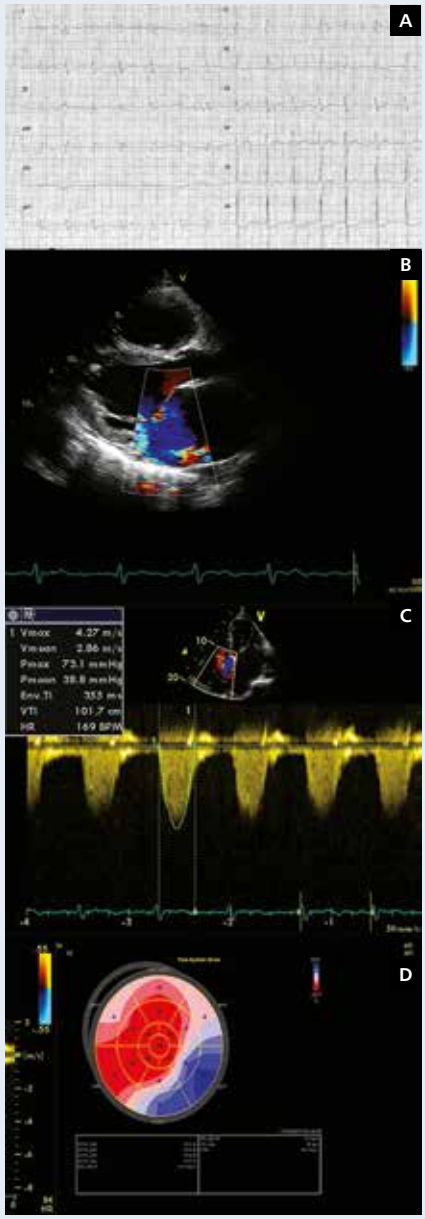

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Figure 1. Echocardiographic findings in eosinophilic leukaemia with endomyocardial infiltration: electrocardiography presenting atrial fibrillation and right ventricular hypertrophy (**A**), significant mitral regurgitation in parasternal long-axis view (**B**), severe pulmonary hypertension assessed by tricuspid regurgitation pressure gradient (**C**), disturbed myocardial strain within lateral segments of the left ventricle and reduced global longitudinal strain (**D**)

Address for correspondence:
Andrzej Tomasik, MD, PhD, 2nd Department of Cardiology, Medical Faculty with Dentistry Division in Zabrze, Medical University of Silesia in Katowice, ul. Skłodowskiej 10, 41–800 Zabrze, Poland, tel: +48 32 373 23 72, fax: +48 32 372 10 10, e-mail: atomasik@sum.edu.pl

Conflict of interest: *none declared*