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CLINICAL VIGNETTE

Heart block, non-compaction cardiomyopathy, or athlete's heart?

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An asymptomatic 18-year-old male patient was referred to the cardiology ward for further diagnostic evaluation of electrocardiographic abnormalities repeatedly observed in the past two years. The patient was a professional basketball player, his daily training lasting around 3 to 4 h. He has never fainted nor had a family history of cardiac disorders, including sudden cardiac death. Physical examination revealed no abnormalities. The patient's blood pressure was 116/72 mmHg, body weight was 84 kg, and he was 190 cm in height. Routine laboratory tests were normal. The electrocardiogram (ECG) showed a sinus rhythm of 60 bpm with a first-degree atrioventricular block (AVB) with long PQ interval (400 ms), QRS duration of 100 ms, QTc interval of 380 ms, a normal ST segment, and features of left ventricular (LV) hypertrophy. Periodically, sinus bradycardia was recorded with attributes of isorhythmic atrioventricular dissociation and a junctional rhythm of 45 bpm, alternating with a sinus rhythm with second-degree AVB type 1 (**Suppl. Fig. 1 — see journal website**). On 24-h ECG monitoring, the average daily heart rate was 60 bpm, with episodes of second-degree AVB during the day. Additionally, a nocturnal sinus bradycardia with second-degree AVB was observed, including pauses of up to 3.6 s. Lyme disease was excluded. During the cardiac treadmill stress test, a normal chronotropic response was recorded. During exercise-induced sinus tachycardia, a persistent first-degree AVB with the same PQ interval was observed (**Suppl. Figs. 2, 3 — see journal website**). The echocardiographic examination revealed normal-sized cardiac chambers (LV end-diastolic diameter of 52 mm, LV end-systolic diameter of 31 mm) as well as normal values of LV ejection fraction (LVEF 70%) and global longitudinal strain (–22%). Parameters of mitral inflow and other indices of diastolic function were also within reference range (E 148 cm/s, A 89 cm/s, E/A 1.6, E' 17 cm/s). However, prominent trabeculation of the lateral and posterior LV walls were noticed, suggestive of a non-compaction phenotype of the myocardium (Fig. 1A, B, **Suppl. Figs. 4–10 — see journal website**). Cardiac magnetic resonance imaging was performed, however, the image did not meet the criteria for non-compaction cardiomyopathy and structural heart disease was excluded. We decided not to perform an invasive electrophysiological study. His-Purkinje disease seemed unlikely, and a long PQ interval could be explained by some baseline conduction prolongation and/or vagotony overlapping with periodic second-degree AVB (Fig. 1C). Finally, athletic heart syndrome (AHS) was diagnosed in the context of unusual, extreme adaptation to high-performance physical exercise including LV remodelling and hypertrabeculation of the myocardium. The patient was discharged home and regular cardiological follow-up was recommended. This case demonstrates that myocardial adaptation to physical effort may have different courses. The distinction between physiological versus pathological changes in athletes is crucial because the final diagnosis may have important consequences, such as exclusion from engaging in competitive sports. Alternatively, the lack of a proper diagnosis can lead to missed opportunities for effective therapeutic intervention. AHS may be associated with many electro-morphological changes in the circulatory system, including a decrease in resting heart rate with possible coexistence of benign AVB and physiological remodelling of the myocardium. Recent data indicate that LV hypertrabeculation on echocardiography is not uncommon in highly trained athletes. Comprehensive diagnosis of AHS should take into account the symptoms, family history, changes in resting ECG (any T-wave disorders, left bundle branch block, or ventricular arrhythmia), echocardiographically determined LVEF, early diastolic velocity of the mitral annulus (E'), and strain imaging.

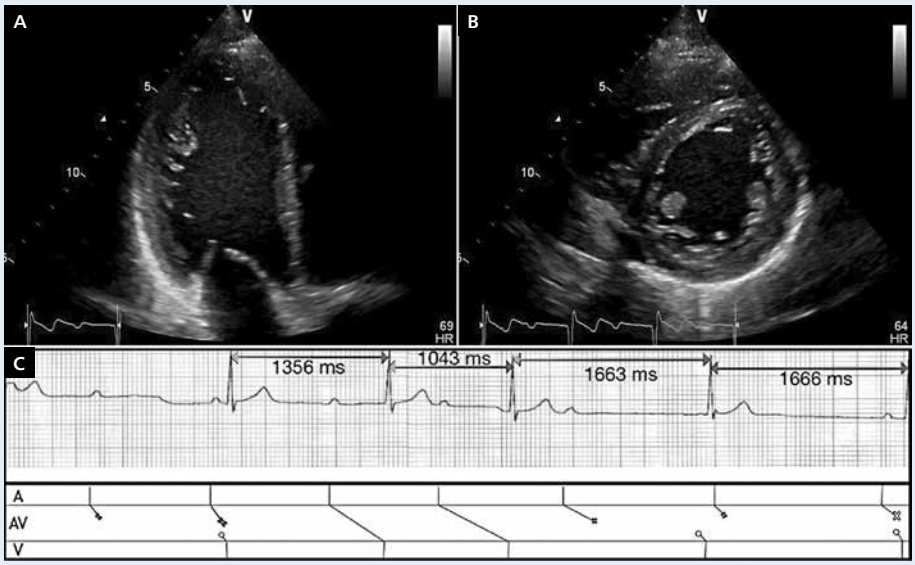

Figure 1. **A.** Echocardiography — hypertrabeculation of the posterior wall of the left ventricle (apical long axis projection, modified three-chamber view); **B.** Echocardiography — hypertrabeculation of the posterior and lateral walls of the left ventricle (parasternal, short-axis projection); **C.** A ladder diagram showing conduction through the atrioventricular (AV) node. The first, fourth, and fifth QRS complexes are junctional escape beats. Second and third QRS complexes follow conducted P waves

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