Kardiologia Polska 2014; 72, 7: 657; DOI: 10.5603/KP.2014.0139 ISSN 0022–9032

STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Dynamic variability of T wave polarity in 12-lead ambulatory ECG as a sign of imminent hypertrophic cardiomyopathy

Dynamiczna zmienność załamka T w 12-odprowadzeniowym badaniu EKG metodą Holtera jako wczesny objaw kardiomiopatii przerostowej bez zawężenia drogi odpływu

Krzysztof Błaszyk, Adrian Gwizdała, Ewa Straburzyńska-Migaj, Hanna Wachowiak-Baszyńska, Stefan Grajek

Department of Cardiology, Poznan University of Medical Sciences, Poznan, Poland

A 27-year-old male had suffered from eight episodes of exercise-induced syncope since he was seven years old. He had professionally trained for soccer and judo. His mother had died aged 46 of dilated cardiomyopathy. ECG revealed: sinus rhythm (SR), rSr' pattern in V_1, flat negative T waves in II, III, aVF, V_5–V_6, left ventricular hypertrophy (LVH) criteria not met. 12-lead ambulatory ECG revealed: major variability of T wave polarity, particularly in inferolateral leads; inverted T waves of various amplitude observed by day, completely normalised at night. Tilt test was negative. Echocardiography revealed: minor LVH (≤ 12 mm), normal LV function referred to as 'athletic hypertrophy'. Exercise treadmill test (ETT) revealed: achieved predicted workload, then blood pressure (BP) drop to 90/60 mm Hg at heart rate (HR) of 180 bpm, followed by progressive HR slowing to 15 bpm with ultimate pauses (≤ 6.1 s) over next 2 min (Fig. 1). Subsequently, SR at 70 bpm and BP of 130/80 mm Hg restored. Coronary angiography revealed no abnormalities. Exercise-induced neurocardiogenic syncope was diagnosed and a dual-chamber pacemaker was implanted. Further professional training was discouraged. He remained asymptomatic for the next 4.5 years. Progressive LVH (IVS 16 mm) allowed for a diagnosis of hypertrophic non-obstructive cardiomyopathy (HNOC) and the pacemaker was upgraded to implantable cardioverter-defibrillator. Two months later, a dual-chamber pacemaker was implanted to his brother (normal resting ECG, four syncopes and Mobitz type II AV block in Holter ECG). Early hypertrophic cardiomyopathy (HCM) diagnosis and prediction of life-threatening events is still challenging and the prognostic importance of ECG-based modalities remains unclear due to their relatively low sensitivity (Baggish A et al. Heart, 2009; 95: 345–347; Erice B et al. Scand J Med Sci Sports, 2009; 19: 356–363). In this report, the diagnostic investigation initially focused on reproducing the syncopal episodes. Ambulatory ECGs and tilt test failed. However, routine ETT revealed the pathomechanism of exercise-induced syncope, allowing for implantation of a pacemaker that protected against future syncope. Nevertheless it could diminish further diagnostic alertness by documenting a neurocardiogenic cause of syncope. At that stage there was no concrete rationale for further investigation for cardiomyopathy. The subtle LVH was, at that time, being linked to the physiological hypertrophy observed in athletes. The marked T wave variability observed in 12-lead ambulatory ECG is intriguing. Negative T waves in II, III, aVF, V_5–V_6 in the resting ECG were qualified nonspecific. However, T wave variability, i.e. inverted T waves by day (mostly sympathetic modulation) and then normalised during night sleep (predominating parasympathetic tone) is most probably a specific ECG picture of an initial stage of HCM. Normalisation of T waves persisting at night actually excludes new myocardial ischaemia. Also, no chest pain was reported on ETT. It seems that this day-to-night dynamic variability of T wave polarity may be a distinct diagnostic sign of an early stage of HNOC that could add to a differential diagnosis with athletic LVH.

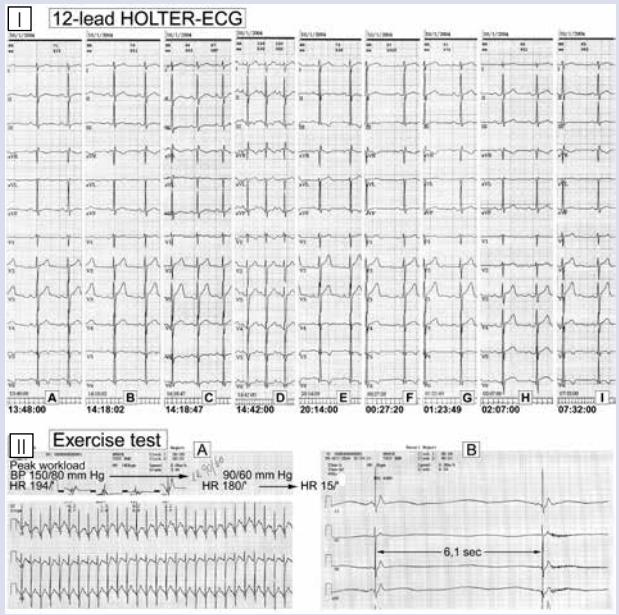

Figure 1. **Panel I.** 12-lead ambulatory ECG. Major day-to-night T wave variability (A through I). Intermittent rSr' pattern in V_1; **Panel II.** ETT: peak workload (**A**) followed by 6.1 s pause (**B**)

Address for correspondence:
Krzysztof Błaszyk, MD, PhD, Department of Cardiology, Poznan University of Medical Sciences, ul. Długa 1/2, 61–848 Poznań, Poland, e-mail: kblaszyk5@wp.pl

Conflict of interest: *none declared*