Kardiologia Polska 2013; 71, 9: 991; DOI: 10.5603/KP.2013.0242 ISSN 0022–9032

STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Ventricular fibrillation with a 2:1 conduction block over the right ventricle in a Brugada syndrome patient

Migotanie komór z blokiem przewodzenia 2:1 u pacjenta z zespołem Brugadów

Marek Jastrzębski[1], Piotr Kukla[2]

[1]1st Department of Cardiology, Interventional Electrocardiology and Hypertension, University Hospital, Krakow, Poland
[2]Department of Internal Medicine, Specialist Hospital, Gorlice, Poland

A 24-year-old male who survived an aborted sudden cardiac death was admitted for a diagnostic work-up and a defibrillator implantation. He had experienced an episode of ventricular fibrillation at 5am during his sleep. His roommate promptly administered cardiac massage, and an emergency team subsequently performed a successful defibrillation. He recovered without any neurological deficits. No cardiac abnormality was detected on echocardiogram or coronary angiogram, and his medical history was unrevealing. However, the ECG showed a spontaneous Brugada type 1 pattern in lead V_1, and after ajmaline administration also in lead V_2, albeit only when recorded in the second intercostal space. Consequently, Brugada syndrome was diagnosed and a defibrillator was implanted. Ventricular fibrillation that was induced during the implantation procedure showed a very peculiar pattern, with the ventricular rate in lead V_1 being half of that observed in other leads, while in lead V_2 alternans in QRS amplitude was present. These observations suggested a local 2:1 conduction block (Fig. 1) over the right ventricular free wall.

The mechanism underlying Brugada syndrome remains a matter of controversy (Wilde AA et al. J Mol Cell Cardiol, 2010; 4: 543–553). Data supporting a depolarisation abnormality mechanism, i.e. local conduction delay seen during the electrophysiology study (Nademanee K et al. Circulation, 2011; 12: 1270–1279), late potentials, and a right-bundle branch block QRS morphology, conflict with experimental data suggesting a repolarisation mechanism (Yan GX, Antzelevitch C, Circulation 1999; 15: 1660–1666). However, depolarisation abnormalities accentuate during a ventricular rate increase, in contrast to repolarisation abnormalities. In the current case, an increase in the rate during ventricular fibrillation/flutter resulted in an augmentation of the depolarisation problem over the right ventricle manifested by a local 2:1 block. These findings support the depolarisation theory of Brugada syndrome.

We are not aware of such an ECG pattern having been previously reported. However, Wilde AA et al. (J Mol Cell Cardiol, 2010; 4: 543–553) reported ST-T alternans in lead V_1 during an increase in the heart rate, and this also may be indicative of a similar phenomenon as in our case.

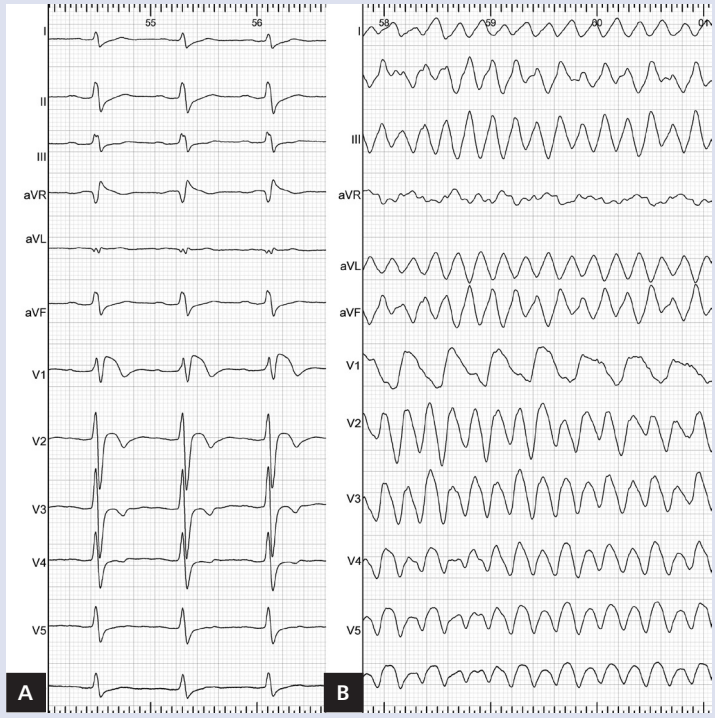

Figure 1. **A**. Electrocardiogram with Brugada type 1 pattern in lead V_1 during sinus rhythm; **B**. Electrocardiogram showing a ventricular fibrillation/flutter in a patient with Brugada syndrome. Note the 300 bpm ventricular rate in all leads with the exception of lead V_1, where the rate seems to be a regular 150 bpm. Moreover, in lead V_2 alternans in QRS amplitude is present. These patterns are suggestive of a local 2:1 conduction block over the right ventricle

Address for correspondence:
Marek Jastrzębski, MD, PhD, 1st Department of Cardiology, Interventional Electrocardiology and Hypertension, ul. Kopernika 17, 31–501 Kraków, Poland, tel: +48 12 424 73 01, +48 12 424 73 14, fax: +48 12 424 73 20, e-mail: mcjastrz@cyf-kr.edu.pl

Conflict of interest: *none declared*