

## Invited Editorial / Davetli Editöryal Yorum

### Coexistence of Wolff-Parkinson-White and Brugada ECG

#### Wolff-Parkinson-White ile Brugada EKG birlikteliği

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The co-existence of Wolff-Parkinson-White (WPW) syndrome and Brugada syndrome (BS) or a Brugada electrocardiogram (ECG) pattern (BECG) is a well-known condition, and has been reported in numerous case presentations.<sup>[1-3]</sup> However, due to the lack of a prospective, controlled study, management of these patients unfortunately relies on speculative assumptions, rather than established scientific data. Management and treatment protocols are primarily based on symptoms and individual characteristics, as well as proposals from published case reports.

In this issue of the journal, Aksan et al.<sup>[4]</sup> published an interesting case report regarding WPW and BECG and discussed how to manage and treat a patient according to current knowledge and suggestions. Briefly, in their presentation, they were faced with a difficult case: a patient with a history of palpitations and recurrent syncope along with an ECG diagnostic for WPW and BECG. They first ablated the accessory pathway and then performed an ajmaline provocation test, which revealed type 1 BECG. After induction of ventricular fibrillation during an electrophysiological study, they decided to implant an intracardiac defibrillator.

Their approach to this patient was thoughtful and analytical; however, I would like to add some comments and emphasize a few useful points that should always be kept in mind. First of all, there is a lack of

established data regarding how to appropriately manage symptomatic patients with WPW and BECG. Therefore, as the

authors suggested, one should proceed on an individual basis, according to the patient's clinical features and risk factors. The presented patient reportedly experienced recurrent syncopal attacks, which may have been simple vasovagal faints, commonly encountered in patients with BS, or arrhythmogenic, due to supraventricular or ventricular tachycardia. Unfortunately, the authors did not provide any other knowledge used in the differential diagnosis of syncope, such as associated prodromal symptoms, family history, etc. Generally, if syncope is recurrent and present for a long time, it is suggestive of a benign etiology. However, in this particular case, one must accept that the exclusion of any arrhythmogenic etiology as the cause of syncope is impossible based only on history and initial clinical findings. Although no arrhythmia was induced, the authors proceeded with ablation of the accessory pathway, which I would also suggest. However, ablating asymptomatic accessory pathways is still debatable and not recommended by many electrophysiologists. The second point that I would raise is whether performing an ajmaline test and/or an electrophysiological study was necessary. In Figure 1, lead V2 unequivocally shows an apparent

#### Abbreviation:

BECG	Brugada electrocardiogram
BS	Brugada syndrome
ECG	Electrocardiogram
WPW	Wolff-Parkinson-White

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J point elevation and the typical coved pattern of type 1 BECG. Based on this ECG and new definition criteria, I would strongly suggest that this patient could be diagnosed as type 1 BS, obviating the need for a provocative test. The ECG performed after ablation showed type 2 BECG in lead V2 for an unknown reason. It may have been due to limited cardiac memory and an ajmaline test was performed to better exaggerate the J point elevation, which was quite reasonable. Based on a recent consensus statement, an electrophysiological study is not recommended and is regarded as non-diagnostic and useless, especially when one performs the standard protocol with 3 extra stimuli, similar to the protocol used for this patient. In addition, the use of 3 extra stimuli and very short coupling intervals just above the ventricular effective refractory period inducing ventricular fibrillation is generally considered nonspecific. Hence, in this particular case, I would not recommend an ajmaline test or an electrophysiological study because of their poor specificity and lack of additional diagnostic value. Finally, the decision to implant an intracardiac defibrillator is a challenging matter of debate. Is an intracardiac defibrillator implantation wise, given its well known pros and cons? Is this patient at high risk of sudden cardiac death? To answer those questions appropriately, as I mentioned above, one needs first to determine the underlying cause of the syncope, which was very difficult in the present case, and second, one must estimate the risk of sudden cardiac death. Syncopal attacks in a male patient, especially within the previous 6 months, along with evidence of spontaneous type 1 BECG should be considered sufficient to justify that the patient is at high risk for sudden car-

diac death when compared with other potential risk factors, such as family history and a positive genetic test. Therefore, I also agree with the authors' decision to implant an intracardiac defibrillator due to the presence of high-risk factors related to BS. However, a subcutaneous rather than an intracardiac defibrillator implantation would perhaps be more rational, given the long-term high rate risk of complications, such as lead-related problems, in the latter technique.

In conclusion, current practice suggests that patients with WPW and BECG should be cautiously managed according to their symptoms, individual characteristics, and risk factors until further data from prospectively designed controlled studies are available.

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