Brugada syndrome: where are you?

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This editorial refers to ‘Spontaneous Brugada electrocardiogram patterns are rare in the German general population: results from the KORA study’ by M.F. Sinner et al., on page 1338

Brugada syndrome is an arrhythmogenic disease characterized by a typical electrocardiographic (ECG) pattern with ST-segment elevation in the right precordial leads and an increased risk of sudden cardiac death related to polymorphic ventricular tachyarrhythmias or ventricular fibrillation. The cornerstone for the diagnosis of the syndrome is the presence of the typical type 1 ECG pattern characterized by the presence of a coved ST-segment elevation of at least 2 mm (0.2 mV) followed by the negative T-wave in two right precordial leads (V1—V3).1

Although the first and second consensus conferences provided clarification of the diagnosis criteria that are supposed to facilitate recognition of the disease, wide variations in the ECG interpretations still persist for this disease.2,3 For example, in the French study COBRA, 20% of the ECGs referred to the expert committee with a Brugada syndrome diagnosis were not in fact diagnostic for the disease.4 Why is it so difficult to diagnose Brugada syndrome? One of the main difficulties is the fact that in contrast to what is found for the long QT syndrome, for Brugada syndrome, there is no clear cut-off value on which to base the diagnosis. The difference between a coved aspect that indicates type 1 diagnosis and a saddleback pattern is probably not so easy to determine and then there remains a subjective interpretation of the repolarization aspect. In the same way, when presented in the literature the pattern is always very clear with a canonical ST-segment elevation, but in real life, ST-segment elevation is often just around the 2 mm cut-off value, which so often makes the diagnosis questionable. For example, the mean ST-segment elevation in a European study was 2.3 mm in the symptomatic patients and 1.9 mm in the asymptomatic patients, thus demonstrating how difficult diagnosis can be.5

Given this frequent difficulty in diagnosis, which is often at least in part suggestive, it is not surprising that there are wide variations in the prevalence of the disease.

Sinner et al.6 present an interesting study using automated ECG interpretation to evaluate the prevalence of Brugada syndrome in a general population.

First, the authors evaluated a computerized analysis of the ECG traces using a J point elevation in leads V1 or V2 or V3 >150 μV and QRS duration <150 ms. Using these criteria, they were able to detect as abnormal 100% of the 11 type 1 ECG and 3 type 2 ECG tested, demonstrating the ability of this algorithm to properly detect abnormal ECGs. Next, they evaluated 4149 ECGs from the KORA S4 study representing a representative Caucasian population selected in southern Germany. In this population, the algorithm identified 250 (6%) abnormal ECGs predominantly in males (80% of the cases), but after evaluation of the ECGs by three expert cardiologists, none of these ECGs was considered specific for Brugada syndrome.

This study is the first to use automated computer-assisted analysis to detect Brugada syndrome in a general population. However, even though the sensitivity of the algorithm is excellent (100%), the specificity is very poor. This type of algorithm can therefore clearly not be of help to the electrophysiologist cardiologist who is perfectly familiar with the Brugada syndrome ECG with all these different patterns. On the other hand, it may be particularly interesting to detect potentially affected patients when the ECG is performed and interpreted by non cardiologists, such as general practitioners or in primary care centres. The question that has still to be discussed is how relevant it is to refer 6% of the general population with abnormal ECGs to detect only a very small proportion of Brugada syndrome patients (none of the patients in the present study), given that the prognosis for asymptomatic patients truly affected by Brugada is now considered as very good.

The second important information provided by this study is the very low prevalence of Brugada syndrome in a general Caucasian population. Given the rarity of the Brugada syndrome, the sample population tested (4149) probably did not allow any definitive conclusion to be drawn as to the prevalence of the disease; indeed, this study failed to identify any Brugada syndrome patients in the entire population. This result is clearly in contrast with the previous studies where the prevalence ranged from 0.22 to 6.1% in European populations.7–10 Certainly, more strict diagnostic criteria since the
publication of the consensus report can explain, at least in part, the
difference between this and the earlier studies but there is still a con-
siderable variation between the different studies. These variations
are certainly related to the presence of clusters of Brugada syndrome
due to the familial character of the disease and this explains the
prevalence variation from one population to another, even if the
genetic background of these two populations is very similar. For
this reason, it is certainly better to consider that the prevalence of
the disease has been evaluated in a specific population rather than
in a European population.

In any case, we must thank Sinner et al. for introducing a new auto-
mated method of interpreting ECGs for Brugada syndrome which
will certainly facilitate recognition of the disease by non-cardiologists
and then allow efficient screening of a large population.

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