Brugada Syndrome

Since Brugada et al. published their first paper in 1992 on the syndrome which became known as Brugada syndrome, many papers have been published concerning the role of gene SCN5A as its cause, along with studies on characteristic electrocardiographic findings, mechanism of arrhythmia, aggrivating factors and prognosis. Such studies gradually clarified the pathogenesis and clinical characteristics of this syndrome. However, SCN5A variation is found only in 18–30% of Brugada syndrome patients.

Although the coved type pattern is thought to be important for correct diagnosis, it is widely accepted that fluctuation of the electrocardiographic patterns is one of the features characteristic of Brugada syndrome. Sakabe et al. (Eur Heart J 2003; 24:1488-1493) examined electrocardiographic findings in medical records representing 3,339 cases over one to ten years, and they observed waveform changes between a saddle-back and coved type in 56.5% among patients exhibiting Brugada-type electrocardiograms.

Implantation of an implantable cardioverter and defibrillator (ICD) is the first choice treatment for patients with a history of cardiac events. But there is no agreement on therapeutic policy for asymptomatic cases. Brugada et al. (Circulation 2003; 108:3092-3096) have reported that the incidence of cardiac events observed during a period of 27±29 months in 190 asymptomatic patients with Brugada syndrome was 8.4%. In the Sakabe’s study there was only one patients who suffered a cardiac event and, moreover, all the electrocardiograms recorded at annual medial examinations over ten year period for this patient were found to be the saddle-back type. However, a typical coved-type pattern was recorded for the same patient immediately after an episode of ventricular fibrillation following the implantation of the ICD.

Few asymptomatic patients with Brugada type electrocardiograms who have no history of cardiac events or family history of sudden cardiac death agree to undergo electrophysiological examinations. It may be possible to prevent cardiac events in these patients if their electrocardiograms normalize by oral administration of certain drugs. Recently, drugs such as quinidine sulfate and phosphodiesterase III inhibitors have been reported to be effective in preventing cardiac events in Brugada syndrome. But the efficacy of these drugs for this indication has not yet been verified.

I would expect that the following problems such as the participation of gene variation other than SCN5A in the origin of Brugada syndrome, the prognosis of the cases which show saddle-back type Brugada electrocardiograms, and prevention of the cardiac events by means of the oral administration of the drugs which can convert the Brugada type electrocardiogram to the normal pattern, will be revealed in the near future.