Demonstration of Transient Entrainment in Monomorphic Sustained Ventricular Tachycardia Associated With Cardiac Sarcoidosis

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A 49-year-old man was referred for further treatment of sustained monomorphic ventricular tachycardia (VT) associated with cardiac sarcoidosis. During an electrophysiologic study (EP), dl-sotalol suppressed the spontaneous VT and prevented induction of VT. However, when prednisolone treatment was started, monomorphic VT recurred frequently. To terminate the VT, a temporal pacing lead was placed at the apex of the right ventricle, and programmed electrical stimulation was attempted from the lead. During the EP study, 2 different monomorphic VTs were repetitively induced and both types were able to be terminated by rapid ventricular pacing; in one of the VT morphologies, constant and progressive fusion was obvious during the ventricular pacing. Some monomorphic VTs associated with cardiac sarcoidosis are due to reentry with an excitable gap, but the clinical efficacy of EP-guided antiarrhythmic drug treatment seems to be less certain during steroid therapy. In the present case, a defibrillator device was implanted to prevent a possible arrhythmic event. (Jpn Circ J 2000; 64: 635–637)

Key Words: Cardiac sarcoidosis; Monomorphic ventricular tachycardia; Reentry; Transient entrainment

Ventricular tachyarrhythmias sometimes occur in patients with cardiac sarcoidosis and can be a cause of sudden cardiac death. However, the electrophysiological mechanism of the tachyarrhythmia and the role of electrophysiologic study (EP)-guided selection of antiarrhythmic drugs in such patients have not well been clarified. The present case report elucidates the electrophysiological characteristics of the VT that is associated with cardiac sarcoidosis.

Case Report

The 49-year-old man had complained of faintness and palpitation since November 1998, and his electrocardiogram (ECG) recorded ventricular tachycardia (VT) of 250 beats/min. Since April 1999, he had been taking dl-sotalol, which was effective in preventing the spontaneous VT attacks. He was referred to hospital for further treatment for sustained monomorphic ventricular tachycardia.

On admission, his hematological and serological examinations were normal except for a high serum angiotensin converting enzyme value (58.0IU/L). The 12-lead ECG showed normal sinus rhythm and complete right bundle branch block (RBBB). Chest X-ray showed mild cardiomegaly and swelling of the bilateral hilar lymph nodes. The 2-dimensional echocardiogram demonstrated diffuse hypokinesia of the left ventricle and the basal wall of the left ventricle was thin. A color Doppler study did not show any prominent valvular regurgitation and the ejection fraction of the left ventricle was 40%. His coronary angiogram was normal. A technetium scintigram revealed an abnormal accumulation in the heart and transbronchial lung biopsy confirmed noncaseating granulomatosis. From these findings, cardiac sarcoidosis was diagnosed.

After obtaining informed consent, a standard EP study was performed while the patient continued taking dl-sotalol. Programmed electrical stimulation consisted of 1–3 extrastimuli and rapid incremental ventricular pacing up to 286 ms were attempted from 2 sites of the right ventricle and one site of the left ventricle before and after using isoproterenol, but ventricular tachyarrhythmia was not induced. During sinus rhythm, endocardial mapping was performed, but the local electrograms recorded from both ventricles were normal. However, when prednisolone (40 mg/day) was started for cardiac sarcoidosis, sustained VT with a QRS morphology identical to that of the clinically documented VT occurred frequently. To suppress the VT, a temporal pacing lead was inserted in the apex of the right ventricle, and programmed stimulation was given using the lead. Sustained monomorphic VTs with 2 different QRS morphologies were repetitively induced by 1–2 ventricular extrastimuli, and one of the VT morphologies matched the clinical VT. During VT, rapid ventricular pacing was attempted for 5–10 s, starting from a slightly shorter cycle length of the VT. The pacing was then repeated at progressively shorter cycle lengths after a decrement in steps of 2–5 ms until the VT was interrupted. The cycle length of the clinical VT was 410 ms and was terminated at a paced cycle length of 402 ms (Fig 1). The cycle length of the other VT was 560 ms and it was terminated at a paced cycle length of 500 ms (Fig 2). During ventricular pacing, constant and progressive fusion were confirmed on surface ECG in...
Fig 1. Monomorphic ventricular tachycardia (VT) with a QRS morphology matching the spontaneous VT was induced by a single ventricular extrastimulation, and its cycle length was 410 ms (Panel A). During the VT, rapid ventricular pacing was started from 408 ms and repeated after a decrement of the pacing cycle length in steps of 2 ms. The paced QRS morphology was slightly different in each pacing cycle length (Panels B–D), and the VT was terminated at the paced cycle length of 402 ms. S1/S1, basic stimulation; S1/S2, interval of premature stimulation; VTCL, cycle length of VT; PCL, pacing cycle length.

Fig 2. Rapid ventricular pacing was attempted during non-clinical monomorphic ventricular tachycardia (VT) with a cycle length of 560 ms. The VT was entrained, and constant and progressive fusion was observed on the surface ECG (Panels A, B). The VT was terminated by 500 ms pacing (Panel C). Abbreviations as in Fig 1.
the latter VT morphology and the mechanism was confirmed to be reentry with an excitable gap. Transient entrainment was not obvious in the former VT morphology because it was easily terminated by the ventricular pacing at the cycle length that approximated the VT cycle length (Fig 1) and the rapid pacing was only applied from the apex of the right ventricle. Although RBBB on ECG and the abnormal tehnecium accumulation in the heart disappeared with prednisolone treatment, a defibrillator was implanted in August 1999.

Discussion

Monomorphic VT associated with ischemic and non-ischemic organic heart diseases are highly entrained by rapid pacing, so the most common mechanism of these VTs is considered to be reentry with an excitable gap. However, the electrophysiological characteristics of the VT that is associated with cardiac sarcoidosis has not yet been clarified, probably because EP studies have rarely been performed in patients with cardiac sarcoidosis and because spontaneous VT is considered a manifestation of disease activity or inflammation.

In the present case, no VT was induced during the first EP study, but sustained monomorphic VTs with clinical and non-clinical morphology were repetitively induced by programmed electrical stimulation during the second EP study. Both VTs were terminated by rapid ventricular pacing, and transient entrainment was obvious in one of them, findings that suggested that the electrophysiological mechanism was reentry with an excitable gap. To our knowledge, this is the first case of transient entrainment demonstrated in monomorphic VT associated with cardiac sarcoidosis.

Ventricular pacing from the left ventricle is required to demonstrate transient entrainment in the clinical VT morphology but in the present case we only attempted ventricular pacing from the temporal lead placed at the apex of the right ventricle.

Because reentrant monomorphic VT is usually reproduced by programmed electrical stimulation and antiarrhythmic drugs that can prevent induction of VT during the EP study are considered to effectively suppress the clinical recurrence of VT. However, standard EP studies cannot predict the long-term efficacy of treatment in patients with cardiac sarcoidosis and VT can recur during steroid therapy. In the present case, prednisolone may have altered the conduction property of the heart and caused the recurrence of clinical VT. Therefore, in VT associated with cardiac sarcoidosis, we have to consider early defibrillator treatment.

References


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