

Ventricular Tachycardia of Right Bundle-Branch Block—Left Axis Deviation Morphology and Organic Heart Disease

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Summary: Most series of patients with ventricular tachycardia (VT) of right bundle-branch block (RBBB)—left axis deviation (LAD) morphology include young individuals with no overt evidence of structural heart disease. In the present report, the clinical and electrophysiologic findings in two patients with verapamil-responsive VT and organic heart disease are described.

Key words: mitral valve prolapse, ischemic heart disease, bundle-branch block, ventricular tachycardia

Introduction

The verapamil-responsive ventricular tachycardia (VT) with a QRS morphology resembling right bundle-branch block (RBBB) and left axis deviation (LAD) has received increasing attention in recent years.^{1–10} As this type of VT was widely described in young individuals without overt heart disease, there has been little emphasis on its possible appearance and prognostic implications in patients with organic heart disease as described herein.

Case One

A 16-year-old boy was admitted because of palpitations lasting for several hours. An ECG demonstrated a wide

QRS tachycardia at 150 beats/min with an RBBB–LAD pattern. The tachycardia ended spontaneously and the ECG taken during sinus rhythm was normal. Physical examination disclosed a mid-systolic click and a late systolic murmur. Echocardiography showed bileaflet mitral valve prolapse. A radionuclear scan showed normal right and left ventricular function. Past medical history was unremarkable. Family history elicited that the patient's father died suddenly at the age of 42 and one of his brothers had an undefined heart murmur. His mother and three brothers were subsequently evaluated. Clinical and echocardiographic findings were compatible with mitral valve prolapse in all the patient's brothers.

An electrophysiologic study was performed in the non-medicated state. During sinus rhythm, the AH and HV intervals were normal. Dual AV nodal pathways were demonstrated. VT, identical in morphology to the clinical arrhythmia, was induced multiple times by rapid atrial and ventricular pacing and by ventricular extrastimulus testing (Figs. 1, 2); it was terminated following the administration of intravenous verapamil (2 mg). Restoration of sinus rhythm was preceded by a gradual lengthening of the tachycardia cycle length. VT could not be reinduced following the administration of verapamil. The patient was discharged on oral verapamil (80 mg t.i.d.). One month later he was readmitted with VT which again responded to intravenous verapamil (Figs. 3, 4). The dose was increased to 120 mg t.i.d., and no clinical recurrences have been observed for a follow-up period of 24 months.

Case Two

A 66-year-old man was admitted to the Coronary Care Unit with acute inferior wall myocardial infarction. The course was complicated by complete AV block, requiring temporary pacemaker installation, and postinfarction angina. A coronary angiogram performed on the Day 8 postinfarction demonstrated significant proximal left anterior

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FIG. 1 Induction of ventricular tachycardia (VT) by atrial pacing. Note AV dissociation and lack of H potentials preceding the VT. A = atrial electrogram, H = His bundle electrogram. Paper speed is 100 mm/s.

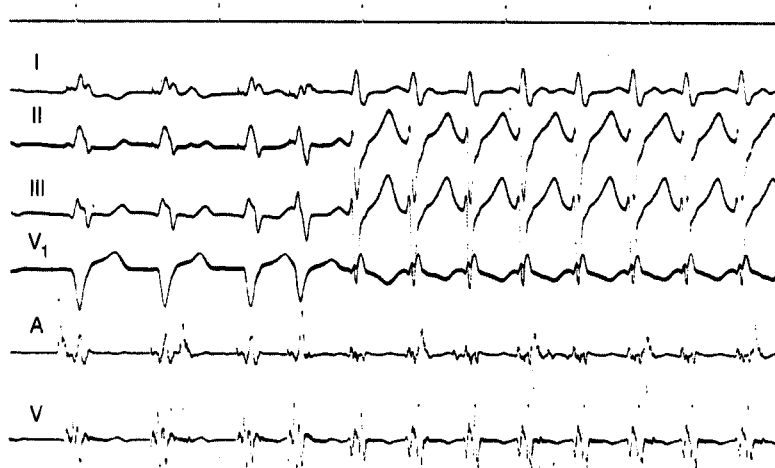


FIG. 2 Induction of VT by ventricular extrastimulus testing. Paper speed is 50 mm/s. V = ventricular electrogram.

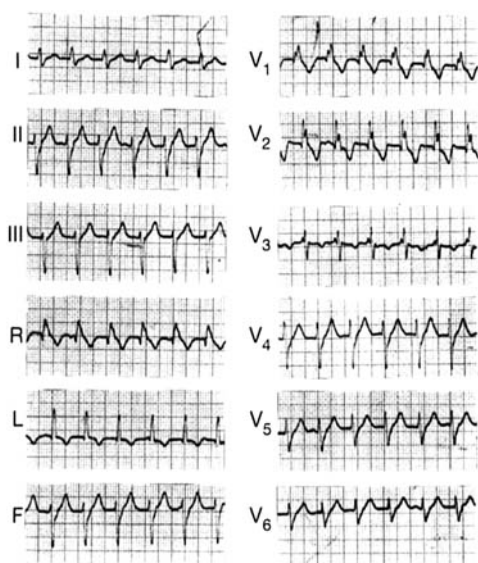


FIG. 3 Case No. 1: 12 lead ECG showing VT with a RBBB-LAD pattern.

descending and mid right coronary artery stenotic lesions. The patient underwent an aortocoronary bypass grafting, and he was discharged following one week period of uneventful recovery. He was placed on oral therapy of aspirin, persantin, amiodarone (200 mg b.i.d.) and diltiazem (60 mg t.i.d.). Six months later he started experiencing a new onset of effort angina accompanied with frequent premature ventricular contractions that were resistant to conventional therapy. Nuclear study showed global left ventricular dysfunction with an ejection fraction of 30%. At this stage, aspirin, nitrates, amiodarone, and diltiazem were taken regularly until he was readmitted for chest pain that did not respond to nitroglycerin sublingually. Upon admission, all oral medications with the exception of aspirin and amiodarone were withdrawn. Physical examination revealed a fourth heart sound and pulmonary rales. An ECG showed Q waves in the corresponding inferior leads and frequent multifocal premature ventricular beats. Intravenous nitrates and heparin were given, resulting in complete relief of pain and no increase in enzyme levels. After 36 h following withdrawal of diltiazem, the patient complained

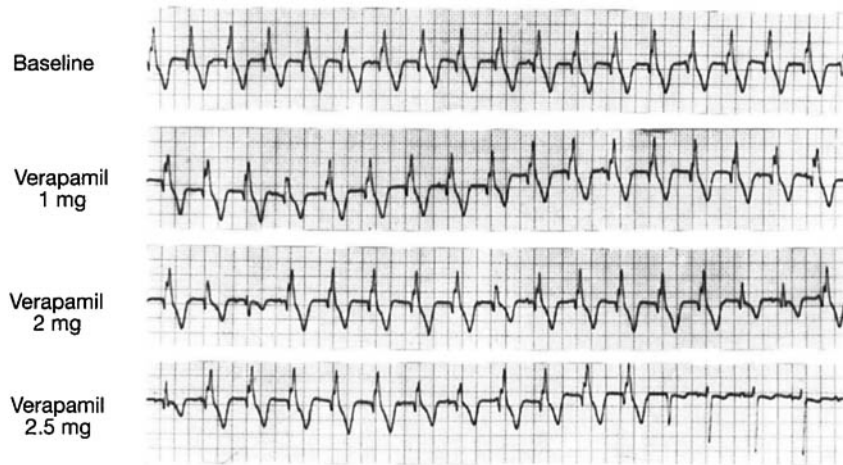


FIG. 4 Termination of VT with the administration of intravenous verapamil.

of chest discomfort, palpitations, and shortness of breath. An ECG disclosed VT with a rate of 105 beats/min and QRS morphology of RBBB–LAD pattern (Fig. 5) interrupted by frequent premature ventricular contractions. Neither VT nor premature contractions were responsive to lidocaine. At this point, oral diltiazem (60 mg) was given, resulting in gradual deceleration of VT and restoration of sinus rhythm. One further self-terminating bout of VT occurred four h later in conjunction with recurrency of chest pain. An off-diltiazem electrophysiologic study was later performed demonstrating normal AH and HV intervals during sinus rhythm. Symptomatic VT of RBBB–LAD pattern, induced by atrial extrastimulus and rapid atrial pacing, was promptly terminated by intravenous administration of verapamil (2 mg). During tachycardia, His potential activation was evident 10 ms before the QRS complex. The patient was discharged on diltiazem (60 mg t.i.d.) and remained asymptomatic for a follow-up period of one year.

Discussion

It has become a rule to make a distinction between VT in the presence or in the absence of organic heart disease. In this context, VT with a RBBB–LAD pattern has recently emerged as a distinct entity of VT characterized by its typical QRS configuration and its responsiveness to verapamil.^{1–10} This arrhythmia has been encountered mostly among patients with undetected structural heart disease and usually carries a good prognosis. Debate still centers on the underlying electrophysiologic mechanism involved, as findings supporting triggered automaticity as well as reentrant mechanism have been encountered in these patients. Verapamil efficacy in abolishing this arrhythmia suggests that the tachycardia is dependent on slow calcium channel activity. Our two patients fulfilled all the typical characteristics of this tachycardia. However, in contradiction with previous reports, this was encountered in the setting of organic heart disease. Among 10 patients reported by Ger-

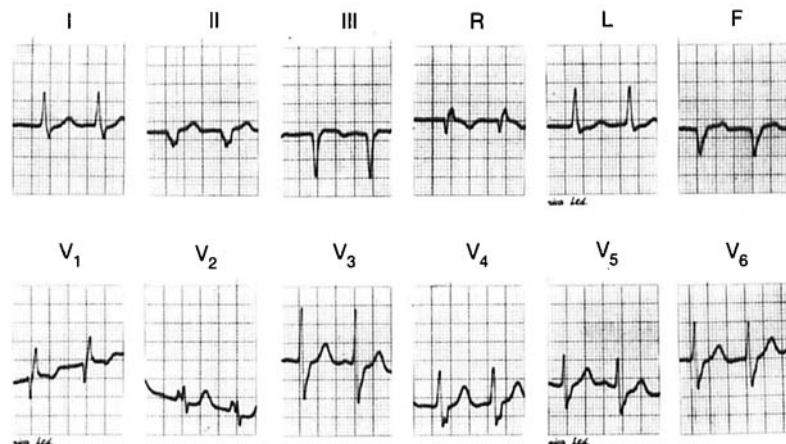


FIG. 5 Case No. 2: 12-lead ECG during VT showing RBBB–LAD morphologic characteristics.

man *et al.*¹ with this type of VT, 2 were found to show similar features of mitral valve prolapse as our patient. One of them died suddenly at the age of nineteen—16 months after an unsuccessful attempt of cryoablation of the VT focus. Two additional patients with mitral valve prolapse were described by Zipes *et al.*⁴ Generally, VT in the young population without apparent organic heart disease carried a favorable prognosis.^{11,12} However, a family history of mitral valve prolapse and sudden cardiac death, such as encountered in the present case, cannot be easily dismissed even when this benign type of VT is considered.

In the older patient described in Case 2, there was more than an anecdotal evidence suggesting that active myocardial ischemia served as a trigger for the induction of this type of VT or vice versa. The relatively slower rate of VT was attributed to prolonged amiodarone therapy. Treatment of VT, as in this case, is particularly warranted in patients with preexisting heart failure, avoiding further acceleration of the tachycardia and hemodynamic deterioration secondary to long standing VT.¹⁰ It should also be emphasized that the majority of VTs with RBBB–LAD morphology seen in patients with ischemic (postinfarction) heart disease are most probably due to reentry mechanism and they will respond to lidocaine rather than to verapamil. However, caution must be exercised when verapamil-responsive VT of RBBB–LAD morphology is seen in combination with organic heart disease as we do not know whether this type of VT may represent a harbinger of more severe ventricular arrhythmias. This peculiar VT may represent one aspect of myocardial response to ischemia, and a different prognostic implication in this subset of patients has not yet been excluded.

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