Termination and Suppression of Idiopathic Left Ventricular Tachycardia by Diltiazem

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Summary: Idiopathic left ventricular tachycardia is known to be responsive to verapamil in many cases. However, the role of other calcium-channel blockers, such as diltiazem, in treating this specific type of ventricular tachycardia is unknown. We report a case of idiopathic left ventricular tachycardia in a patient with a structurally normal heart, which was terminated and suppressed in the electrophysiology laboratory by a single dose of diltiazem intravenously, and was subsequently suppressed long-term with sustained-release diltiazem. Our finding suggests that idiopathic left ventricular tachycardia may be managed effectively with diltiazem in both the acute and chronic settings.

Key words: calcium-channel blockers, diltiazem, ventricular tachycardia

Introduction

Idiopathic left ventricular tachycardia is characterized by a right bundle-branch block morphology with left axis deviation, a site of origin in the inferoapical portion of the left ventricle, and initiation by programmed stimulation and termination with intravenous verapamil administration.¹ Although other calcium-channel blockers, such as diltiazem, have been

used commonly as atrioventricular nodal blocking agents, their role in verapamil-sensitive ventricular tachycardia is unknown. We report a case of idiopathic left ventricular tachycardia that was terminated and suppressed by diltiazem in a patient with a structurally normal heart.

Case Report

A 21-year-old healthy woman presented to a local emergency room after developing palpitations, lightheadedness, and dizziness while playing softball. She denied syncope or chest pain. She had no history of heart disease or previous episodes of palpitations and was not taking any medications. On presentation, she was alert with a blood pressure of 96/58. Physical examination was unremarkable except for tachycardia. Telemetry tracings and a 12-lead electrocardiogram (ECG) (Fig. 1A) showed a wide complex tachycardia at a rate of 219, with extreme left axis deviation and right bundle-branch block morphology. After administration of a single 20 mg dose of diltiazem intravenously, the wide complex tachycardia converted to atrial fibrillation with a controlled ventricular rate. The arrhythmia then terminated to normal sinus rhythm with resolution of her clinical symptoms. Her ECG after conversion (Fig. 1B) showed normal sinus rhythm, normal PR and QT intervals, and incomplete right bundle-branch block. The patient remained stable without further episodes of arrhythmia. Laboratory data were all within normal ranges, and there was a negative toxicological drug test. Chest x-ray was normal. An echocardiogram revealed normal left ventricular systolic function, normal wall motion, and no evidence of structural abnormality. The patient was transferred to our institution for further evaluation.

A comprehensive electrophysiology study was performed. The patient had normal sinus node recovery times and normal AH and HV intervals. Atrioventricular Wenckebach occurred at 350 ms. Sustained monomorphic ventricular tachycardia with right bundle-branch block morphology and left axis deviation was reproducibly inducible by pacing from the right atrium and right ventricular apex at cycle lengths from 240 to

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FIG. 1 (A) 12-lead electrocardiogram shows ventricular tachycardia, rate 219, with a right bundle-branch block morphology and extreme left axis deviation. (B) After administration of 20 mg diltiazem intravenously, the arrhythmia converted to normal sinus rhythm at a rate of 86 with incomplete right bundle-branch block.

280 ms (Fig. 2A). The patient remained hemodynamically stable with a blood pressure of 110/60. The ventricular tachycardia was easily terminated by burst pacing at cycle lengths of 230–240 ms. A single intravenous of 20 mg bolus dose diltiazem led to termination of ventricular tachycardia and suppressed further induction of the arrhythmia (Fig. 2B). The patient was treated with sustained-release diltiazem at a dose of 180 mg/day. She has been doing well and has resumed all activity without recurrence of arrhythmia during 5 months of follow-up.

Discussion

A unique entity of ventricular tachycardia with a QRS morphology of right bundle-branch block and left axis deviation has been well recognized and studied.^{1–5} Idiopathic left ventricular tachycardia may be refractory to conventional treatment but may respond quite well to verapamil.^{1, 5–11} The pathogenesis of idiopathic left ventricular tachycardia is un-

known, although it has been suggested that the mechanism is reentry with an area of slow conduction.^{9, 11–13} Recent study has indicated that this entity of ventricular tachycardia encompasses a heterogeneous group of tachycardias that result from multiple cellular electrophysiologic mechanisms.¹⁴ The mechanism by which verapamil terminates and suppresses this type of ventricular tachycardia may be by prolongation of conduction within an area of slow conduction.¹² Although the effectiveness of verapamil in treating this type of ventricular tachycardia is well established, the role of alternative calcium-channel blockers such as diltiazem has not previously been investigated. As we have found, diltiazem may be effective in the acute and chronic setting of the management of this type of ventricular tachycardia. Although there are no published studies to date, dihydropyridine calcium-channel blockers, such as nifedipine, may be effective in suppressing and terminating this specific type of ventricular tachycardia due to their minimal effects on the conduction system *in vivo*.

Be that as it may, it is very important to emphasize that calcium-channel blockers, such as verapamil and diltiazem, may be effective in managing idiopathic left ventricular tachycar-

FIG. 2 (A) Induction of ventricular tachycardia by ventricular pacing at a cycle length of 280 ms. (B) Termination of ventricular tachycardia by a 20 mg bolus of intravenous diltiazem.

dia; they are, however, contraindicated for the majority of ventricular tachycardias, particularly those associated with underlying ischemia.

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Venous Thrombosis Causing Arterial Embolization to the Same Limb through a Patent Foramen Ovale

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Summary: We describe a patient who presented with acute ischemia affecting the left lower limb. Because a transthoracic echocardiogram was abnormal, a transesophageal study was arranged. This demonstrated an atrial septal aneurysm and right-to-left shunting of contrast, raising the possibility of paradoxical embolism. The diagnosis was confirmed by contrast venography, which showed extensive thrombosis in the deep veins of the left thigh, and a ventilation-perfusion scan which was consistent with multiple pulmonary emboli. Among the lessons from this case was the finding that in patients with arterial embolism the likely origin of the embolus should be considered and, in the absence of common risk factors (atrial fibrillation, rheumatic heart disease, left ventricular dilatation, widespread atheroma), occult venous thrombosis and a right-to-left shunt should be sought. In this select group of patients, transesophageal echocardiography is significantly more sensitive than transthoracic study and should be the investigation of choice. Second, in the patient described in this report the clinical signs of deep venous thrombosis (DVT) were masked

by the more prominent features of acute arterial ischemia. Without the incidental echocardiographic abnormality, it is likely that the important diagnoses of DVT, pulmonary embolism, and paradoxical embolism would not have been made.

Key words: paradoxical embolism, transesophageal echocardiography, limb ischemia, patent foramen ovale, deep venous thrombosis

Case Report

A 61-year-old man was admitted with a 12-h history of pain, numbness, weakness, and coldness in the left leg. The symptoms had fluctuated over that time but had suddenly worsened 1 h before admission. He had been short of breath for 2 weeks and had had bilateral leg cramps for a week. He was an ex-heavy smoker who had been exposed to asbestos dust for many years as a power station worker. He had been treated for left calf deep venous thrombosis (DVT) and pulmonary embolism 16 years previously. This had followed varicose vein surgery to the left leg 6 weeks prior, although there had not been a prolonged period of immobility.

Physical examination revealed sinus tachycardia, a third heart sound, and signs consistent with an acutely ischemic left leg. All pulses below and including the left femoral pulse were absent. The clinical impression was of acute left ilio-femoral thromboembolism, and heparin was commenced. An electrocardiogram showed sinus tachycardia, right bundle-branch block, and a leftward R wave axis (-32°). A chest x-ray showed apical pleural thickening and no other abnormality. Hemoglobin, platelet count, protein C, protein S, anti-thrombin III, and fibrinogen levels were within the normal

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