CASE REPORT

It Is Not Always Digitalis: Bidirectional Ventricular Tachycardia in Left Ventricular Hypertrophy

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electrophysiology; ventricular tachycardia & clinical, noninvasive techniques; Holter/event recorders & clinical

A 68-year-old man with a history of coronary heart disease presented to the emergency department with hypertensive crisis (systolic blood pressure 240/90 mmHg), angina pectoris, and cold sweat. The patient denied having either palpitations or syncope. Cardiac markers, blood count, and serum chemistry were normal. There were no ST segment alterations in the 12-lead ECG on admission. Echocardiography showed normal left ventricular function with significant left ventricular hypertrophy, as well as normal right ventricular function and dimensions. After recompensation and normalization of blood pressure the patient was referred to the catheterization laboratory. Coronary angiography revealed insignificant coronary heart disease.

On the cardiologic ward the asymptomatic patient underwent routine 24-hour Holter ECG recording exhibiting multiple episodes of sustained bidirectional ventricular tachycardias (BVTs) at 120 bpm lasting up to 1 minute (Figs. 1 and 2). There were no significant alterations of serum electrolytes during the time of the Holter recording—the medication was aspirin, amlodipine, and ramipril. The patient has never received digitalis glycosides, which is frequently associated with BVTs.¹ Significant ischemia, reduced left ventricular function,² aconite poisoning,³ and familial history of sudden cardiac death⁴ could all be ruled out. After a clinical and electrocardiographic negative exercise testing (cycle ergometry) up to a work rate of 100 W without significant ventricular ar-

rhythmias, the patient was referred to the electrophysiology (EP) laboratory for further investigation of the BVTs. But also the EP-study showed normal times and there were no inducible sustained ventricular arrhythmias in programmed electrical stimulation.

Because of the lack of pathophysiological findings, the BVTs in the present case were most likely due to massive left ventricular hypertrophy—a type of structural heart disease, which has so far not been reported to be the underlying cause of BVTs. The coexistence of BVTs in patients with congenital catecholaminergic polymorphic VT (CPVT) due to an underlying genetic disorder that leads to intramyocardial calcium overload and triggered activity has also been described in patients with left ventricular hypertrophy.⁵ Another genetic cause of left ventricular hypertrophy with associated CPVT was observed in mice due to calsequestrin mutations leading to chronic intracellular calcium overload.⁶ These genotypic causes of BVTs might also explain the occurrence of this uncommon arrhythmia in association with left ventricular hypertrophy. The patient was treated conservatively with optimal antihypertensive medication and amiodarone (treatment with beta-blockers was not considered for long-term therapy due to a history of beta-blocker intolerance). Under medical therapy there were no recurrences of BVTs in control-Holter ECG recordings within 5 years of follow-up.

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Figure 1. Bidirectional ventricular tachycardia with alternating QRS axes and left bundle branch block type morphology. Note intermittent fusion (†) and capture beats (*).

Figure 2. A Holter sample tracing showing sinus rhythm with intermittent premature ventricular complexes (PVCs) (blue) as well as multiple episodes of bidirectional ventricular tachycardia (red).

The present case is unique for the demonstration of BVTs in a patient without digitalis therapy and stresses the importance of the arrhythmogenic properties of left ventricular hypertrophy.

REFERENCES

1. Kummer JL, Nair R, Krishnan SC. Bidirectional ventricular tachycardia caused by digitalis toxicity. *Circulation* 2006;113:e156-e157.

2. Al-Khafaji A, Corwin HL, Adhar GC, et al. Bidirectional tachycardia: Two cases and a review. *Anesth Analg* 2002;95:310-315.
3. Tai YT, Lau CP, But PP, et al. Bidirectional tachycardia induced by herbal aconite poisoning. *Pacing Clin Electrophysiol* 1992;15:831-839.
4. Cohen TJ, Bing Liem LB, Hancock EW. Association of bidirectional ventricular tachycardia with familial sudden cardiac death syndrome. *Am J Cardiol* 1989;64:1078-1079.
5. Rosso R, Kalman JM, Rogowski O, et al. Calcium channel blockers and beta-blockers versus beta-blockers alone for preventing exercise-induced arrhythmias in catecholaminergic polymorphic ventricular tachycardia. *Heart Rhythm* 2007;4:1149-1154.
6. Song L, Alcalai R, Arad M, et al. Calsequestrin 2 (CASQ2) mutations increase expression of calreticulin and ryanodine receptors, causing catecholaminergic polymorphic ventricular tachycardia. *J Clin Invest* 2007;117:1814-1823.