CASE REPORT

Torsades de Pointes as a Cause of Sudden Death in a Patient with Aortic Stenosis and Atrial Fibrillation

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The occurrence of sudden cardiac death during Holter monitoring in patients with aortic stenosis has been reported previously. In the majority of the reported cases, the cause of death was a malignant ventricular tachyarrhythmia. The presence of a strong association between frequency and complexity of ventricular arrhythmias and sudden death in patients with aortic stenosis has been proposed. We report the case of a 77-year-old woman with aortic stenosis and atrial fibrillation who had an episode of torsades de pointes that degenerated into ventricular fibrillation during Holter monitoring. A short-long-short sequence, but not increased ventricular ectopics, precipitated torsades de pointes and sudden death in this case which is strongly indicative of triggered activity as the underlying mechanism of the lethal arrhythmia.

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torsades de pointes; sudden death; aortic stenosis; atrial fibrillation

The occurrence of sudden cardiac death in patients with aortic stenosis has been reported previously.^{1,2} Monomorphic or polymorphic ventricular tachycardia was the cause of sudden death in most of the reported cases.^{1–3} Increased ectopic ventricular activity with complex forms and a significant acceleration in heart rate were observed within the last hours before the tachyarrhythmic death of patients with aortic stenosis who died suddenly during Holter monitoring.² It was hypothesized that a strong link exists between frequency and complexity of ventricular arrhythmias and sudden death in patients with aortic stenosis.²

CASE REPORT

We report a case of sudden cardiac death in a patient with aortic stenosis and atrial fibrillation who had an episode of torsades de pointes that degenerated into ventricular fibrillation. A 77-year-old woman came to the outpatient clinic to undergo a scheduled 24-hour Holter recording to investigate the adequacy of heart rate control during atrial fib-

rillation. She had a history of aortic stenosis for the last 4 years with a maximum systolic gradient of 63 mmHg as assessed by echocardiography. The aortic valve area was 1.2 cm², while significant left ventricular hypertrophy (interventricular septum and left ventricular posterior wall thickness was 1.3 cm), was encountered. The echocardiographically determined left ventricular ejection fraction was 50%. She also had a history of persistent atrial fibrillation and received panwarfin and rate control therapy with digitalis and diltiazem 270 mg daily for the last 3 months. She reported no syncope or symptoms of heart failure or angina in the past. She only reported palpitations and mild exertional dyspnea in the past. During the day of evaluation she was receiving the previously mentioned medications plus a transdermal nitrate patch and an angiotensin-converting-enzyme inhibitor. Before the start of the recording she underwent complete physical examination, 12-lead electrocardiogram and blood tests. Heart rate was 89 beats/min, blood pressure was 120/70 mmHg, and body temperature was 36.7°C. A 12-lead

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Figure 1. (A) Full disclosure strip from the Holter recording showing the last 4 minutes before and the first 4 minutes after the occurrence of torsades de pointes. (B) Enlarged ECG strips showing the onset of torsades de pointes.

electrocardiogram revealed atrial fibrillation with maximum rate-corrected (Bazett's formula) QT interval of 446 ms. Routine laboratory tests were normal. After adequate preparation, electrodes were attached and a three-channel Holter recording was initiated at 13:16:00 hours. The patient died during sleep, 11 hours later. She reported no discomfort at any time during the afternoon hours or before sudden death.

Holter analysis revealed that the cause of sudden death was an episode of torsades de pointes that was sustained and degenerated into ventricular fibrillation (Fig. 1). A short-long-short sequence precipitated the episode of torsades de pointes. No complex or numerous ventricular ectopics were recorded during the last hour before sudden death occurred (Fig. 1). The mean heart rate during the last hour before sudden death was 96 beats/min and did not differ compared to the previous hours. No electrocardiographic signs of myocardial ischemia were detected before the occurrence of sudden death.

DISCUSSION

The occurrence of sudden cardiac death during Holter monitoring in patients with aortic stenosis has been reported previously.¹⁻³ In the majority of the reported cases, the cause of death was a malignant ventricular tachyarrhythmia. The presence of a strong association between frequency and complexity of ventricular arrhythmias and sudden death in patients with aortic stenosis has been proposed, which was not found in our case. Furthermore, it was hypothesized that ventricular arrhythmias in patients with aortic stenosis appear to be related with the presence of severe heart failure and impaired left ventricular systolic function.² On the contrary, our patient displayed no symptoms of heart failure or seriously affected left

ventricular function. Although coronary arteriography was not performed, the presence of significant coronary artery disease was not apparent from the patient's history, physical examination or non-invasive diagnostic evaluation. Nevertheless, progressive prolongation of the action potential duration and increased heterogeneity of ventricular repolarization are commonly found in the hypertrophic myocardium of patients with aortic stenosis and may provide the substrate for development of arrhythmias arising from triggered activity.³⁻⁵ The association between left ventricular hypertrophy and increase in the dispersion of repolarization leading to enhanced susceptibility for torsades de pointes, has been reported previously.⁶ Thus, triggered activity was probably responsible for the occurrence of torsades de pointes and sudden death in our patient who suffered from aortic stenosis and atrial fibrillation. The presence of a short-long-short sequence which precipitated torsades de pointes and sudden death in this case is strongly indicative of triggered activity as the underlying mechanism of the lethal arrhythmia.

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