CASES AND TRACES

Tachycardia-Induced Cardiomyopathy in a 43-Year-Old Man

ECG CHALLENGE

A 43-year-old man with a 5-year history of palpitation and dyspnea was referred to our hospital for further evaluation and management of persistent tachycardia. Before his referral, pharmacological therapy with metoprolol and digoxin failed to alleviate his symptoms. He reported no other significant medical history. On presentation, he had an irregular heart rate of 109 beats/min. A12-lead ECG and 24-hour Holter ECG were obtained and are shown in Figures 1 and 2. The N-terminal pro-brain natriuretic peptide level was 1875 pg/mL, and a chest x-ray showed cardiac enlargement. Transthoracic echocardiography revealed a left ventricular end-diastolic diameter of 67 mm, a left ventricular ejection fraction of 23% with global hypokinesis, confirming the diagnosis of dilated cardiomyopathy.

Figure 1. The baseline ECG demonstrates a regularly irregular supraventricular tachycardia (130 beats/min).

A clearly visible P wave with morphology consistent with sinus rhythm is followed by 2 QRS complexes. Note that the PP intervals are regular.
Figure 2. Twenty-four–hour Holter tracing reveals
2 different PR intervals, with a short PR preceding the first QRS of the grouped beats, but a long PR when only 1 QRS is present, as seen after the first P wave visible on the page preceding the third QRS complex.

What is the diagnosis?
Please turn the page to read the diagnosis.
RESPONSE TO ECG CHALLENGE
The baseline ECG in this case (Figure 1) showed a pattern of grouped beating, with normal sinus rhythm followed by 2 QRS complexes in each group. We considered possible mechanisms for these findings. The first possibility was premature atrial complexes in atrial bigeminy,

![Ladder diagram of the mechanism proposed.](image)

Figure 3. Ladder diagram of the mechanism proposed.
Top, the same Holter tracing seen in Figure 2; Bottom, lead II of the ECG in Figure 1. Pink stars indicate atrial impulses originating in the sinus node. Arrows indicate the direction of conduction. Solid lines (in the AV portion of the diagram) indicate conduction via the fast pathway, whereas dashed arrows represent slow-pathway conduction. In the top ladder diagram analysis, the first and sixth P waves conduct via the slow pathway only because of refractory fast pathways, leading to PR prolongation, whereas the rest of the P waves conduct via both the fast and slow pathways. In the bottom ladder diagram analysis, note the regular PP intervals, with 1 P wave followed by 2 QRS complexes because of consecutive anterograde conduction via fast and slow pathways of the AV node. AV indicates atrioventricular.
but there was no evidence of atrial activity before the second beat in each group, and the P waves preceding the 2 QRS complexes in each group were marching through with a cycle length of \( \approx 840 \) ms. The most likely explanation became evident with the finding from the Holter monitor recording (Figure 2), which revealed 2 different PR intervals in addition to the grouped beating, indicating the presence of dual AV nodal physiology, suggesting that atrial impulses were being conducted to the ventricle via both the fast and slow pathways. As such, the most likely explanation for the ECG herein is sinus beats conducting simultaneously via a fast and slow pathway, giving the 1:2 atrioventricular (AV) relationship noted. The postulated mechanism is explained in the ladder diagram (Figure 3).

In the top ladder diagram, which reflects the same rhythm as in Figure 2, the first and sixth sinus beats show long PR intervals, indicating that the atrial impulse is conducted only via the slow pathway because of the refractoriness of the fast pathway. The rest of the beats conduct both via the fast and slow pathway, giving the 1:2 atrioventricular (AV) relationship noted. The observed variation in QRS-complex morphology, 1 narrow and 1 wide, is likely caused by an intermittent functional conduction block (aberrancy). This uncommon conduction pattern of 2 QRS complexes after a single sinus beat has been termed dual AV nodal nonreentrant tachycardia.

Dual AV nodal nonreentrant tachycardia is a rare arrhythmia that results from simultaneous antegrade conduction over the fast and slow pathways of AV node and may lead to tachycardia-induced cardiomyopathy. Because each sinus beat leads to 2 QRS complexes, the patient is effectively in incessant tachycardia, which may lead to cardiomyopathy. Rare cases of tachycardia-induced cardiomyopathy attributable to this arrhythmia have resolved by slow-pathway ablation.\(^1\,^2\) Thus, it seems that the early detection of this arrhythmia and definitive treatment, especially in cases of tachycardia-induced cardiomyopathy, is important to prevent or minimize the effects of persistent tachycardia on cardiac function. Dual AV nodal nonreentrant tachycardia may be intermittent, and the diagnosis can be easily missed because it can mimic atrial premature beats, atrial fibrillation, and ventricular tachycardia.\(^2\)

Because of his significant symptoms and left ventricular systolic dysfunction, an electrophysiological study was performed. A diagnosis of AV nodal nonreentrant tachycardia was confirmed (Figure 4). Radiofrequency catheter ablation of the slow conduction pathway was performed at the usual anatomic location. This resulted in the elimination of the tachycardia with restoration of sinus rhythm and persistent 1:1 AV conduction. After ablation, the patient reported resolution of his previous symptoms. Repeat transthoracic echocardiography 1 month after the procedure showed marked improvement of left ventricular ejection fraction to 52%, and improvement in the left ventricular end-diastolic diameter from 67 mm at presentation to 56 mm.

DISCLOSURES

None.

REFERENCES

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Yun-Tao Zhao, Lei Wang and Zhong Yi

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