

A rare manifestation of atrial fibrillation in the presence of Wolff-Parkinson-White syndrome: tachycardia-induced cardiomyopathy

Wolff-Parkinson-White sendromu zemininde gelişen atriyum fibrilasyonunun nadir bir prezentasyonu: Taşikardiye bağlı kardiyomiyopati

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Summary– We report a 68-year-old man who presented with heart failure and atrial fibrillation (AF) with rapid ventricular response and wide QRS complexes. Tachycardia-induced cardiomyopathy (TIC) due to persistent AF developing on the basis of Wolff-Parkinson-White (WPW) syndrome was considered. Signs and symptoms of heart failure improved with restoration of sinus rhythm. This case suggested that persistent AF in a patient with WPW syndrome is one of the rare causes of TIC.

Tachycardia-induced cardiomyopathy (TIC) is caused by persistent supraventricular or ventricular tachyarrhythmias. It is characterized by ventricular systolic dysfunction and clinical manifestations of heart failure, which are reversible with normalization of the heart rate. Although atrial fibrillation (AF) is one of the most important causative tachyarrhythmias that can lead to TIC, it is rare in the presence of Wolff-Parkinson-White (WPW) syndrome.

In our case, we present reversible tachycardiomyopathy due to persistent AF in a patient with WPW syndrome.

CASE REPORT

A 68-year-old male was presented to our outpatient clinic with dyspnea and leg swelling. He had been

Özet– Geniş QRS kompleksleri olan hızlı ventrikül cevaplı atriyum fibrilasyonu (AF) ve kalp yetersizliği semptomları ile başvuran 68 yaşındaki bir erkek hasta sunuldu. Hastada Wolff-Parkinson-White (WPW) sendromu zemininde meydana gelen sürekli AF ve bunun sonucunda gelişen taşikardiye bağlı kardiyomiyopati (TBK) düşünüldü. Kalp yetersizliği bulgu ve semptomları sinüs ritmi elde edildikten sonra düzeldi. Bu olgu WPW zemininde gelişen sürekli AF'nin TBK'de nadir bir sebebi olabileceğini göstermektedir.

diagnosed previously with heart failure and persistent AF. His echocardiographic and coronary angiographic examination showed left ventricular (LV) systolic dysfunction (ejection fraction: 40%) and normal coronary arteries, leading to a diagnosis of idiopathic dilated cardiomyopathy. In accordance with this diagnosis, treatment consisting of metoprolol, furosemide, digitalis, and warfarin had been prescribed. On admission, his blood pressure was 120/80 mmHg and heart rate 153 beats/min in spite of the metoprolol (100 mg) and digitalis. He had bilateral pretibial edema. The cardiac sounds were arrhythmic with a 2/6 systolic murmur on the apical area, and he had basal pulmo-

Abbreviations:

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|-----|------------------------------------|
| AF | Atrial fibrillation |
| ECG | Electrocardiography |
| EPS | Electrophysiological study |
| LV | Left ventricular |
| TIC | Tachycardia-induced cardiomyopathy |
| WPW | Wolff-Parkinson-White |

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nary rales on auscultation. The laboratory examination revealed mildly elevated blood urea nitrogen (33 mg/dl, normal range <20 mg/dl), therapeutic digoxin level (0.8 ng/ml) and an effective international normalized ratio (INR) level (2.1). Complete blood count, thyroid function and other biochemical tests were all within normal ranges. Electrocardiography (ECG) revealed AF with rapid ventricular response and wide QRS complexes (Figure 1a, top). Chest radiography showed cardiomegaly with signs of pulmonary congestion. LV systolic dysfunction (ejection fraction was 32% measured using the modified Simpson method) and mild dilatation of the LV and both atria, together with mild mitral regurgitation, were observed on transthoracic

echocardiography. The patient was hospitalized for further assessment and treatment.

The presence of possible accessory pathways was considered for two reasons: the possible presence of a delta wave in a complex QRS morphology and intractable rapid ventricular response in spite of optimal medication. Intravenous furosemide for improving congestive symptoms and intravenous amiodarone for decreasing accessory pathway conduction were started, and digitalis was discontinued for improving AV node conduction. Following this, the patient's heart rate was observed to decrease to normal ranges and his symptoms diminished (Figure 1a, bottom).

Elective electrical cardioversion was being considered; however, on the tenth day of this new treatment, spontaneous sinus rhythm with the pattern of WPW syndrome developed (Figure 1b, top). Electrophysiological study (EPS) and ablation were planned for the WPW syndrome, but the patient refused this treatment and was discharged with amiodarone 200 mg, metoprolol 100 mg, furosemide 40 mg, and warfarin therapy. During the follow-up examination five weeks later, his previously reported symptoms were no longer present and his ECG showed a sinus rhythm without prominent delta wave appearance (Figure 1b, bottom). The follow-up echocardiographic examination revealed both normal LV systolic function and dimensions. By the third month following treatment, he was observed to be asymptomatic without diuretic therapy and had a normal ECG.

DISCUSSION

Tachycardia-induced cardiomyopathy (TIC) occurs in association with supraventricular tachyarrhythmias, such as AF, atrial flutter, automatic atrial tachycardia, atrioventricular nodal re-entry tachycardia, automatic atrioventricular junctional tachycardia, and accessory pathway tachycardia, as well as with ventricular tachycardia,^[1] but it is rare

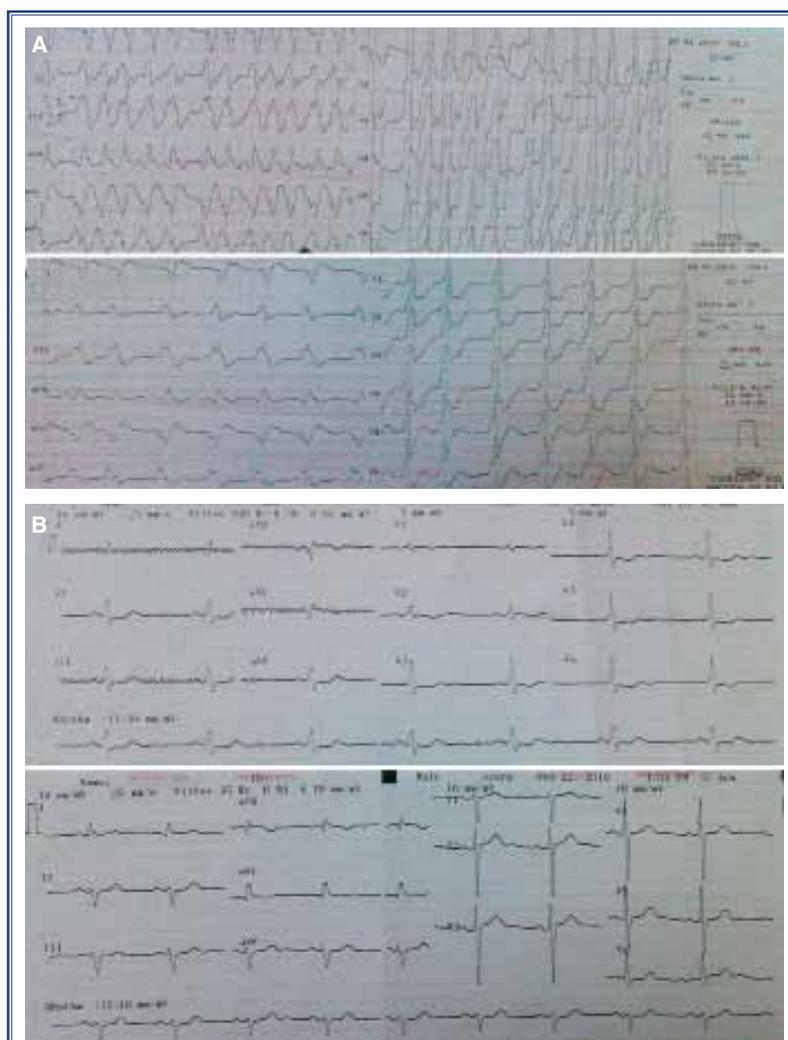


Figure 1. (A) Atrial fibrillation with rapid ventricular response and wide QRS complexes resembling delta wave (top); after heart rate control was obtained (bottom). (B) Sinus rhythm with the pattern of Wolff-Parkinson-White syndrome (top); disappearance of the prominent delta waves after five weeks (bottom).

with persistent AF in the presence of WPW syndrome. When AF appears in patients with WPW syndrome, it may be life-threatening if an extremely rapid ventricular response develops, degenerating into ventricular fibrillation,^[2] so the follow-up of such patients for an extended period is difficult.^[3]

To our knowledge, this is the first reported case in the literature in which the signs and symptoms of a patient with TIC triggered by WPW syndrome and persistent AF completely resolved with restoration of sinus rhythm. The combination of WPW syndrome and long-lasting AF has been reported in the literature only rarely.^[4,5] As in our case, they reported that the combination of WPW syndrome and long-lasting AF may lead to medically refractory tachycardia and LV dysfunction. It has been demonstrated that significant LV dysfunction and dilatation recover with treatment of the tachycardia.^[6] As patients may be unaware of their tachycardia and report only the symptoms of cardiomyopathy, it is important to consider the diagnosis of TIC in patients with cardiomyopathy with unclear etiology. In our case on admission, despite taking medication for AF and heart failure, the patient had a heart rate that was unexpectedly high, and the electrocardiogram revealed wide QRS morphology simulating conduction over an accessory pathway. Thus, the presence of possible accessory pathways was considered. Administration of amiodarone suppressed the antegrade accessory pathway conduction, but despite this, conduction over an AV node with narrow QRS morphology was not observed. A possible explanation for this situation was the extreme suppression of an AV node because of the multiple medications (metoprolol and digitalis). Although surface ECG showed the presence of a prominent delta wave after restoration of sinus rhythm, it had disappeared one month later, and only small “q” wave was observed in DI and aVL, which may indicate minimal preexcitation. Minimal preexcitation is generally associated with a left lateral accessory pathway,^[7] and the change on the ECG in these patients may be subtle after ablation therapy.^[8] Probable explanations for disappearance of the prominent delta wave in this patient are the improved AV node conduction after discontinuation of digitalis, decreased accessory pathway conduction after starting amiodarone, and the presence of a left lateral accessory pathway.

It is hypothesized that premature ventricular acti-

vation over an accessory pathway induces abnormal ventricular septal movement, LV dyssynchrony, LV remodeling, and subsequently cardiomyopathy, but it is generally related with right-sided accessory pathways.^[9] However, the surface ECG of our patient was consistent with a left lateral accessory pathway, and the initial echocardiography revealed global LV dysfunction without dyssynchrony.

Finally, although we were unable to perform EPS, the relatively low heart rate (153) at presentation for the patient who had an accompanying accessory pathway and the fact that he survived without developing ventricular fibrillation and sudden death might indicate that a presumably relatively longer antegrade refractory period is responsible for his toleration of persistent AF in the long period. However, it must be kept in mind that combination therapy of beta-blocker and digitalis may lead to fatal consequences in the presence of concurrent AF and WPW syndrome.

In conclusion, we suggest persistent AF in the presence of WPW syndrome can be a cause of TIC, which can be successfully treated by establishing sinus rhythm.

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Key words: Atrial fibrillation/complications/therapy; cardiomyopathy; heart failure; Wolff-Parkinson-White syndrome/therapy.

Anahtar sözcükler: Atriyum fibrilasyonu/komplikasyonlar/terapi; kardiyomiyopati; kalp yetersizliği; Wolf-Parkinson-White sendromu/terapi.