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Olanzapine-Induced Bradycardia: A Clinical Case Report

Olanzapin Kullanımı Sonrası Gelişen Bradikardi: Bir Olgu Sunumu

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ÖZ

Giriş: Atipik antipsikotiklerin kardiyak iletim sistemi üzerine etkileri çoğunlukla QT uzamasıyla ilişkilendirilirken, bradikardi oldukça nadir bildirilen ancak klinik olarak önem taşıyan bir yan etkidir. Bu olgu sunumunda, olanzapin tedavisi başlandıktan kısa süre sonra gelişen belirgin bradikardi vakasının klinik seyri ve olası mekanizmaları tartışılmıştır.

Olgu: Yirmi üç yaşında erkek hasta, psikotik bozukluk tanısıyla yatırılarak olanzapin 10 mg/gün tedavisine başlanmıştır. Başlangıç EKG'sinde 1. derece AV blok ve 55–60/dk istirahat nabzı saptanmıştır. Tedavinin 7. gününde nabız 35/dk'ya düşmüş, kan basıncı 95/60 mmHg ölçülmüştür. Laboratuvar değerleri normal sınırlarda olup, hipoglisemi, elektrolit bozukluğu, tiroid disfonksiyonu ve ilaç etkileşimi gibi ikincil nedenler dışlanmıştır. Olanzapin tedavisi kesilerek aripiprazol başlanmış, takipte nabız 60–65/dk düzeyine yükselmiştir.

Tartışma: Olanzapine bağlı bradikardi nadir görülse de özellikle mevcut iletim kusuru veya düşük istirahat nabzı olan bireylerde ciddi klinik sonuçlara yol açabilir. Bu olgu, QT uzaması olmaksızın da antipsikotiklerin kalp hızını etkileyebileceğini göstermektedir. Antipsikotik başlanmadan önce EKG değerlendirmesi yapılmalı, tedavi süresince vital bulgular yakından izlenmelidir. Gerektiğinde alternatif antipsikotiklere geçilmesi kardiyak güvenlik açısından önem taşır.

Anahtar Kelimeler: Olanzapin; Bradikardi; Antipsikotik; Şizofreni; Kardiyotoksisite

ABSTRACT

Introduction: Although the cardiac effects of atypical antipsychotics are often associated with QT prolongation, bradycardia is a much rarer but clinically significant adverse effect. This case report aims to describe the clinical course and possible mechanisms of marked bradycardia developing shortly after initiation of olanzapine treatment.

Case: A 23-year-old male patient was hospitalized with a diagnosis of psychotic disorder and initiated on olanzapine 10 mg/day. Baseline electrocardiogram revealed a first-degree AV block and a resting heart rate of 55–60 bpm. On the 7th day of treatment, his pulse decreased to 35 bpm, and blood pressure was 95/60 mmHg. Laboratory results were within normal limits, and secondary causes such as hypoglycemia, electrolyte imbalance, thyroid dysfunction, and drug interactions were excluded. After olanzapine discontinuation and switch to aripiprazole, the heart rate improved to 60–65 bpm.

Discussion: Although bradycardia associated with olanzapine use is rare, it may have clinically significant consequences in patients with pre-existing conduction abnormalities or low resting heart rate. This case highlights that antipsychotics may affect cardiac chronotropy even in the absence of QT prolongation. Baseline ECG and close monitoring of vital signs are essential during treatment, and switching to alternative agents should be considered when necessary.

Keywords: Olanzapine; Bradycardia; Antipsychotic; Schizophrenia; Cardiotoxicity

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INTRODUCTION

Atypical antipsychotics (second-generation antipsychotics) are widely used as first-line treatment agents in severe psychiatric disorders such as schizophrenia, bipolar disorder, and treatment-resistant major depression. However, it has long been recognized that this drug class may adversely affect cardiac conduction and repolarization. While QT/QTc prolongation and associated torsades de pointes (TdP)-type ventricular arrhythmias have been extensively discussed, bradycardia remains a much less frequently reported adverse effect. Bradycardia associated with atypical antipsychotics can occur across a wide clinical spectrum, ranging from asymptomatic heart rate reduction to severe presentations such as syncope or hypoperfusion, regardless of concomitant QT prolongation (1–3).

Recent reviews on the cardiac effects of antipsychotics suggest that, beyond hERG (human ether-à-go-go-related gene) potassium channel blockade, multiple mechanisms may be involved, including effects on sodium and calcium channels, autonomic nervous system modulation, anticholinergic and α-adrenergic actions, as well as pharmacokinetic drug–drug interactions (4,5).

In this context, the present case report aims to highlight a rare but potentially serious clinical condition by describing a patient with a psychotic disorder who developed marked bradycardia shortly after the initiation of olanzapine treatment.

CASE

A 23-year-old male university student, previously living with his mother and sister, was involuntarily hospitalized due to a two-month history of irritability, the belief that his friends in the neighborhood were aware of events occurring at school and were mocking him, the perception of being subjected to insinuations through others' glances, and suspicions that his mother and sister might harm him.

His past psychiatric history revealed a childhood diagnosis of attention-deficit/hyperactivity disorder (ADHD), for which methylphenidate had been prescribed but not administered at the family's request. At the age of 11–12, aripiprazole was recommended due to behavioral problems and had reportedly been used for approximately ten years. There was no family history of psychiatric disorders.

Mental status examination revealed that the patient was conscious, oriented, and cooperative. Speech rate and quantity were within normal limits, although his associations were occasionally loose. His thought content included persecutory and referential delusions, and he lacked insight. Laboratory test results were within normal limits. Vital signs showed a blood pressure of 122/86 mmHg and a heart rate of 58 beats per minute. Electrocardiography (ECG) demonstrated a first-degree atrioventricular (AV) block with a QTc interval of 428 ms.

The patient was diagnosed with a psychotic disorder according to DSM-5 criteria, and olanzapine 10 mg/day was initiated. Initially, the heart rate ranged between 55–60 bpm without symptoms. On the seventh day of treatment, approximately five hours after taking the medication, his pulse dropped to 35 bpm while lying down and increased to 65–70 bpm after 15 minutes of sitting upright. His blood pressure was recorded as 95/60 mmHg, and ECG showed a QTc of 385 ms. The patient remained asymptomatic with no dizziness, syncope, or respiratory distress, and laboratory results were unremarkable. The cardiology department was consulted. It was considered that the bradycardia might be attributable to olanzapine, and a change in the antipsychotic medication was recommended.

Table 1. Electrocardiogram parametres

	1. day	**7. day**	**20. day**
QTc	428	385	358
PR	222ms	224ms	220ms
Pulse	54	35	65

Due to the decrease in heart rate from 60 bpm to 35 bpm following olanzapine administration, the medication was gradually discontinued. Aripiprazole 5 mg/day was initiated and titrated to 10 mg/day. On day 20 of follow-up, his pulse had increased to 60–65 bpm. After titration to 20 mg/day of aripiprazole, the patient's paranoid ideations decreased in frequency and intensity, and he was discharged in partial remission. Written informed consent was obtained from the patient.

DISCUSSION

Although cases of olanzapine-induced bradycardia were previously considered exceedingly rare, an increasing number of reports have emerged in recent years (6,7). In most of these cases, a marked decrease in pulse rate was observed shortly after the initiation of treatment or following a dose increase, with normalization of heart rate upon discontinuation of the medication (7). Particularly in cases without accompanying QT prolongation, bradycardia is thought not to result solely from repolarization abnormalities. Instead, alternative mechanisms such as ion channel blockade at the sinoatrial or atrioventricular (AV) node level, or increased vagal tone, have been proposed (5). Experimental studies have also demonstrated a dose-dependent bradycardic effect of antipsychotics, emphasizing that significant alterations in heart rate can occur even in the absence of QT prolongation (8).

In the present case, bradycardia developed on the seventh day of olanzapine treatment. Laboratory analyses and secondary causes (such as hypoglycemia, electrolyte imbalance, hypothyroidism, or β-blocker use) were systematically excluded. The presence of a baseline first-degree AV block and a low resting heart rate (55–60 bpm) may have rendered the patient more susceptible to developing bradycardia. The prompt normalization of pulse rate after discontinuation of olanzapine, along with the stable heart rate observed during aripiprazole treatment, may suggest a possible causal association, although other contributing factors cannot be completely excluded.

The cardiac effects of olanzapine can be explained through several mechanisms, including α-adrenergic antagonism, anticholinergic sedation, increased vagal tone, and direct sodium/potassium channel blockade at the sinoatrial node. In addition, case reports have described bradycardia associated with olanzapine-induced hypothermia or hypoglycemia (9). Taken together, these findings indicate that antipsychotics may influence not only cardiac repolarization but also chronotropy and conduction pathways.

In patients initiated on antipsychotic treatment—particularly those with preexisting conduction abnormalities, low resting heart rate, coexisting cardiac disease, or renal/hepatic impairment—baseline electrocardiogram (ECG) and pulse monitoring are recommended. The British Heart Rhythm Society (BHRS) advises performing ECGs at admission, prior to discharge, and at least annually in patients receiving antipsychotics (10). Similarly, guidelines from the American College of Cardiology (ACC), the American Heart Association (AHA), and the Heart Rhythm Society (HRS) regard resting ECG evaluation as the standard of care for assessing bradycardia and conduction abnormalities (11).

Based on this case, when selecting antipsychotic therapy for young patients with preexisting conduction disturbances and low resting heart rate, olanzapine should be prescribed cautiously—preferably at a low dose and with close vital and ECG monitoring. If bradycardia develops, secondary causes should be excluded, and appropriate measures such as dose reduction or switching to an alternative agent with a different receptor and ion-channel profile (e.g., aripiprazole) should be implemented.

Bradycardia is not a risk unique to olanzapine; similar cases have been reported with risperidone, quetiapine, clozapine, and amisulpride. For instance, in an elderly patient using amisulpride, symptomatic bradycardia was observed and resolved promptly after drug discontinuation (5). This suggests a potential class effect among antipsychotics that may predispose to bradycardia in susceptible individuals. In the literature, aripiprazole has been shown to be superior to other antipsychotics in terms of cardiac side effects. Therefore, based on the side-effect assessment, the treatment was switched from olanzapine to aripiprazole (12).

The present report is limited by its single-case design, which restricts the generalizability of findings. Furthermore, plasma drug levels and direct assessments of autonomic nervous system activity were not obtained. Future multicenter, prospective studies incorporating ECG and pulse monitoring are warranted to better elucidate the chronotropic effects of antipsychotic medications.

This case report demonstrates that antipsychotics can directly affect cardiac chronotropy and conduction pathways, even in the absence of QTc prolongation. Therefore, personalized monitoring strategies, including baseline and follow-up ECG assessment, multidisciplinary evaluation (collaboration between psychiatry and cardiology), and close monitoring of vital signs, are crucial to ensure treatment safety in patients receiving antipsychotics.

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Conflict of Interest

The authors declare no potential conflicts of interest regarding scientific or medical committee memberships, consultancies, expert witness activities, employment, shareholding, or any other relationships that could be construed as a conflict of interest related to this work.

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