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CASE REPORT

Mobitz type II atrioventricular block associated with tizanidine use in a patient with left ventricular hypertrophy: a case report

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Keywords:
atrioventricular block; adverse drug reaction; left ventricular hypertrophy; electrophysiology; sinus arrhythmia (source: MeSH-NLM).

ABSTRACT

This case report describes a 52-year-old woman whose electrocardiogram revealed left ventricular hypertrophy, and whose 24-hour Holter monitoring identified supraventricular extrasystoles along with a second-degree atrioventricular (AV) block, Mobitz type II. This finding is uncommon in an asymptomatic patient with structural heart disease. The patient was receiving tizanidine for muscle pain, raising suspicion of a potential adverse drug reaction as the cause of the AV conduction abnormality. Following discontinuation of tizanidine, repeat Holter monitoring showed normalization of the conduction pattern. The patient remained clinically stable with no recurrence of symptoms. This case underscores the importance of recognizing adverse drug effects, particularly those affecting cardiac conduction, which may progress to more serious conditions if not promptly identified.

Bloqueo auriculoventricular Mobitz II asociado al uso de tizanidina en paciente con hipertrofia ventricular izquierda: reporte de caso

Palabras clave:
bloqueo auriculoventricular; evento adverso; hipertrofia ventricular izquierda; electrofisiología; arritmia sinusal (fuente: DeCs-BIREME).

RESUMEN

Se presenta el caso de una mujer de 52 años, a quien un electrocardiograma le evidenció hipertrofia ventricular izquierda y un monitoreo Holter de 24 horas mostró extrasístoles supraventriculares y un bloqueo auriculoventricular (AV) de segundo grado tipo Mobitz II. Este hallazgo resulta inusual en una paciente asintomática y con evidencia de cardiopatía estructural. La paciente se encontraba en tratamiento con tizanidina por dolor muscular, motivo por el cual se sospechó una posible reacción adversa al medicamento como causa del trastorno de la conducción AV. Tras la suspensión de la tizanidina, un nuevo Holter demostró la normalización del ritmo de conducción. La paciente se mantuvo estable y sin recurrencia de síntomas. Este caso resalta la importancia de considerar los efectos adversos de los fármacos, particularmente aquellos con impacto sobre la conducción cardíaca, los cuales pueden evolucionar a formas más severas si no se identifican sus causas de manera oportuna.

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INTRODUCTION

Second-degree atrioventricular (AV) block, Mobitz type II is a cardiac conduction disorder characterized by intermittent interruption of electrical transmission between the atria and ventricles, following a predictable pattern of PR-interval prolongation [1]. This type of block is clinically relevant because it can progress to complete AV block, increasing the risk of syncope and serious cardiovascular events. Cases of Mobitz II in young individuals—especially asymptomatic patients—are uncommon and are often detected incidentally [2].

Tizanidine is an alpha-2 adrenergic agonist widely used to manage spasticity. Reported cardiovascular adverse effects include hypotension and bradycardia. Although these effects may influence cardiac conduction, the literature has not documented a direct association between tizanidine use and the occurrence of Mobitz II AV block. Tizanidine-induced bradycardia could, in theory, contribute to conduction abnormalities; however, to date, no specific causal link with Mobitz II AV block has been established [3-6].

In an asymptomatic adult patient, the presence of Mobitz II AV block could be transient and potentially influenced by factors such as the use of medications that affect heart rate and blood pressure. Nevertheless, a thorough clinical evaluation is essential to rule out other underlying causes and determine the clinical relevance of the finding. Therefore, this report aims to explore the possible relationship between tizanidine use and Mobitz II AV block in an asymptomatic patient.

CASE PRESENTATION

A 52-year-old woman without significant personal medical history, but with a family history of heart disease in her mother, presented for a routine check-up. A baseline electrocardiogram (see Figure 1) showed a pattern compatible with left ventricular hypertrophy (LVH). Based on this finding, a 24-hour Holter monitor was ordered (see Figure 2), which revealed isolated supraventricular extrasystoles—escape beats—and daytime episodes of second-degree AV block, Mobitz type II. The patient was taking tizanidine, a muscle relaxant for musculoskeletal pain, and reported no cardiovascular symptoms at the time of evaluation.

Given the ECG finding of LVH, a transthoracic echocardiogram was requested. It showed mild concentric left ventricular hypertrophy, with an indexed left ventricular mass of 102 g/m^2, preserved ejection fraction (62%), diastolic dysfunction with an

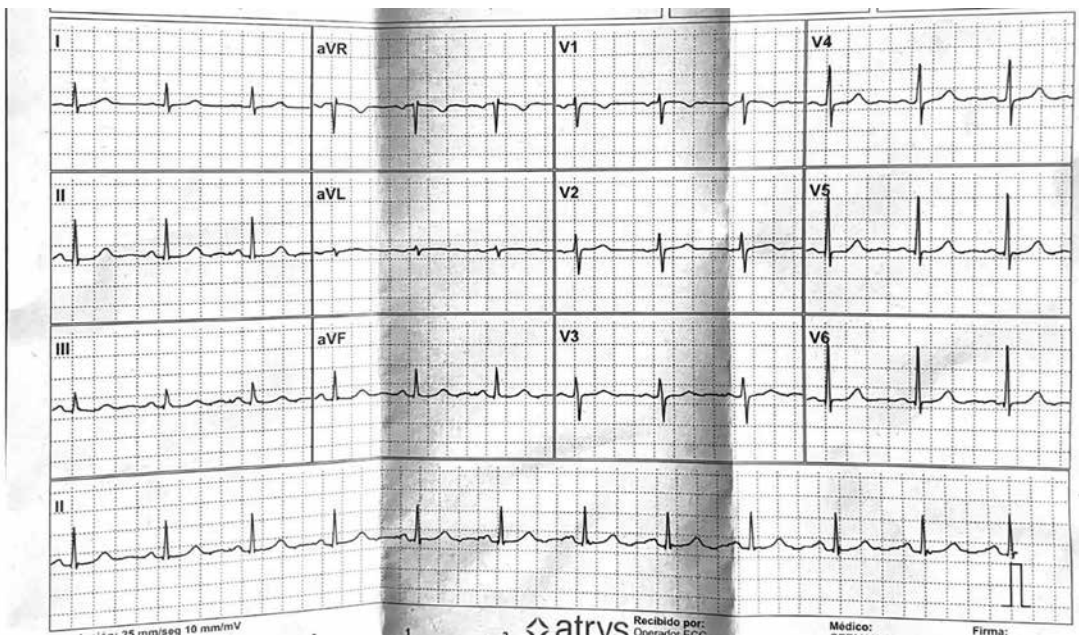

Figure 1. Twelve-lead electrocardiogram showing sinus rhythm and left ventricular hypertrophy

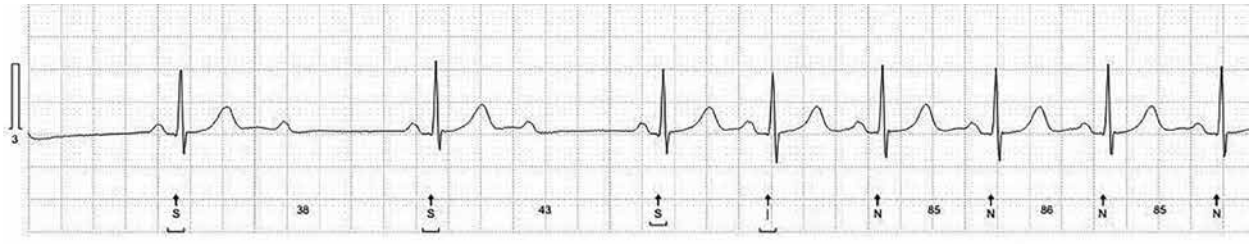

Figure 2. Isolated supraventricular extrasystoles (escape beats) and isolated episodes of second-degree AV block, Mobitz type II

abnormal relaxation pattern, a mildly enlarged left atrium, and no significant valvular abnormalities. There was no dilation of right-sided chambers and no pericardial effusion (see Figure 4). Cardiac magnetic resonance imaging was not deemed necessary, as the echocardiogram adequately characterized myocardial structure.

Laboratory tests showed fasting glucose 96 mg/dL, serum creatinine 0.82 mg/dL, sodium 139 mEq/L, potassium 4.1 mEq/L, magnesium 2.0 mg/dL, and a lipid profile within normal limits. There was no evidence of thyroid dysfunction. Ambulatory monitoring did not document arterial hypotension or bradycardia.

One week later, the patient was evaluated by the Electrophysiology Service. An exercise stress echocardiogram was negative for ischemia, and a second Holter monitor showed resolution of the previous findings (see Figure 3), with no evidence of AV block or extrasystoles. The patient remained hemodynamically stable, with no symptoms attributable to the conduction abnormality.

Given suspicion of a possible association between tizanidine use and the transient AV block, the medication was discontinued and outpatient follow-up was arranged. On subsequent evaluations, cardiac parameters remained within normal ranges and the patient experienced no new events.

DISCUSSION

The second-degree AV block, Mobitz type II represents a significant alteration in cardiac electrical conduction due to its potential to progress to complete AV block and trigger adverse cardiovascular events such as syncope or, in extreme cases, sudden death [1,2,7]. The incidental identification of this type of block in asymptomatic patients is uncommon, particularly in the absence of structural heart disease or other relevant risk factors [8-13].

In this case, a Mobitz II AV block was observed during a 24-hour Holter monitoring in a patient with no known structural heart disease who was under tizanidine treatment. This finding raised the possibility of an adverse drug reaction, as tizanidine's sympatholytic effects include bradycardia and hypotension [3-6], although a direct association with advanced AV blocks has not been previously established.

Tizanidine, an alpha-2 adrenergic agonist used for the management of spasticity, can indirectly affect

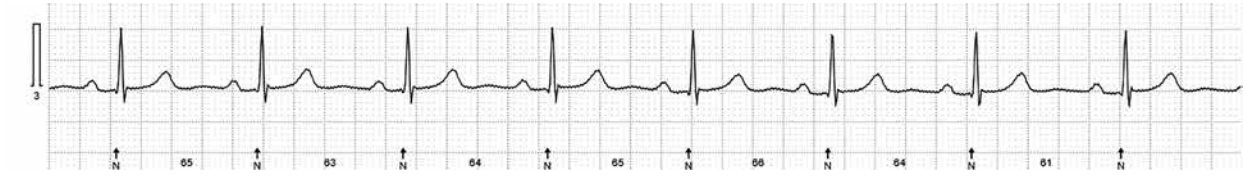

Figure 3. 24-hour Holter monitoring record

***** Regular sinus rhythm with an average heart rate between 60 and 80 beats per minute is observed. P waves precede each QRS complex, indicating normal AV conduction. No arrhythmias or significant abnormalities are evident in this tracing segment.

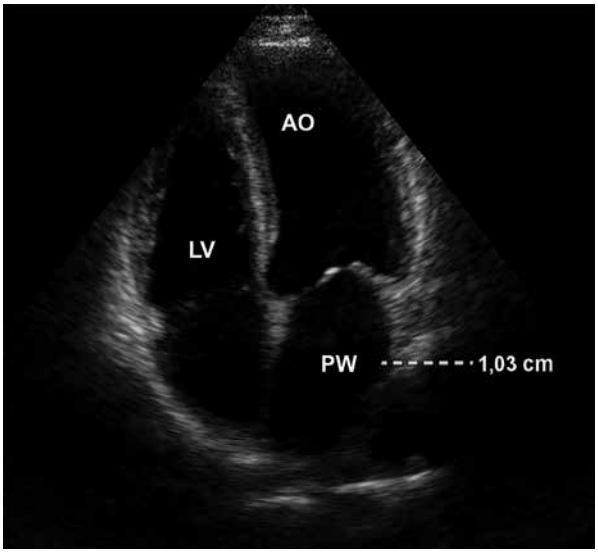

Figure 4. Transthoracic echocardiogram in parasternal long-axis view.

***** The left ventricle (LV) shows thickening of the posterior wall (PW) and interventricular septum (IVS), consistent with mild concentric LV hypertrophy. The aortic valve (AV), ascending aorta (AO), and left atrium (LA) are identified. The left ventricular ejection fraction is preserved (62%). No structural valvular abnormalities or pericardial effusion are observed.

cardiac conduction by reducing sympathetic tone [3-6]. While the literature does not document a specific relationship between its use and the occurrence of second-degree AV block, Mobitz type II, the complete resolution of the conduction disorder after discontinuation of the medication—along with a second Holter showing no abnormalities and an echocardiogram without structural heart disease or ischemia—suggests a possible causal relationship [3–6,14,15].

From a clinical standpoint, this case highlights the importance of considering potential adverse drug effects in patients presenting with unexpected electrocardiographic findings. Although tizanidine has not been classically associated with AV blocks, its effects on heart rate could, in certain contexts, contribute to the development of such abnormalities. In this regard, timely recognition of a possible pharmacologic relationship may prevent serious complications through early intervention.

This case raises important clinical considerations. First, it underscores the need for thorough evaluation of atypical electrocardiographic findings in asymptomatic patients receiving medications that can influence cardiac conduction. The use of Holter monitoring and electrophysiological evaluation was key to confirming the transient nature of the block and ruling out underlying cardiac disease.

The decision to discontinue tizanidine and pursue outpatient follow-up was fundamental, allowing observation of the block's resolution without resorting to invasive interventions. This approach suggests a conservative management alternative in situations where AV block may be drug-related and does not pose an immediate risk of progression to complete block or symptomatic manifestation.

In conclusion, this case suggests that tizanidine may act as a precipitating factor for second-degree AV block, Mobitz type II in patients without underlying heart disease. Although this is not a commonly reported adverse effect, tizanidine-induced bradycardia could interfere with AV conduction, particularly in susceptible individuals. The resolution of the block following tizanidine withdrawal indicates a possible temporal relationship that warrants consideration in clinical practice. Further studies are needed to explore the frequency and mechanisms of this association, as early identification of this side effect could prevent severe complications in patients receiving tizanidine or other drugs with similar cardiovascular effects.

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Authorship contribution

JAHN: Design and development of the research protocol, data collection, statistical analysis, discussion of results, and manuscript preparation.

JSTL: Data collection, critical literature review, discussion of results, and contribution to manuscript writing.

LADS: Research protocol design, manuscript review, and supervision of data analysis.

JAGA: Contribution to case evolution discussion, manuscript review, and treatment guidance.

JQM: Data collection, results analysis, and collaboration in manuscript writing.

VOC: Assistance with data collection, manuscript review, and contribution to results discussion.

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Conflict of interest statement

The authors declare no conflicts of interest.