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Amiodarone, Extreme AV Block and Pseudo-Pacemaker Syndrome: A Malady Caused By A Cure

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ABSTRACT

Amiodarone is a potent antiarrhythmic used to treat supraventricular tachycardia (SVT). However, its substantial impact on *atrioventricular* (AV) nodal conduction can obscure or exacerbate underlying delays, resulting in rare conditions such as pseudo-pacemaker syndrome (PPMS). This syndrome is the result of an extreme prolongation of the PR interval, which causes AV dyssynchrony and hemodynamic compromise in the absence of an implanted device. A 25-year-old male presented with SVT, further complicated by hypokalemia. The case revealed a pre-existing AV nodal conduction delay. Therefore, we present this case. The patient transitioned to sinus rhythm after receiving amiodarone; however, he experienced a persistent and severe PR interval prolongation that exceeded 400 ms. The symptoms of vertigo and palpitations, consistent with PPMS, were induced by this severe conduction block and persisted for more than ten days—reflecting the long half-life of amiodarone. This case demonstrates that amiodarone can dangerously exacerbate latent AV nodal disease in young adults, underscoring the necessity of recognizing PPMS as a clinical consequence of iatrogenic PR prolongation and emphasizing the need for cautious use and monitoring of antiarrhythmic drugs in patients with any evidence of conduction abnormalities.

Keywords: *Amiodarone; Pseudo-Pacemaker Syndrome; Atrioventricular Conduction Delay*

INTRODUCTION

Supraventricular tachycardia (SVT) refers to a range of arrhythmias originating at or above the atrioventricular (AV) node, often characterized by heart rates between 150 to 220 beats per minute.(Patti et al., 2025) The AV node is crucial for modulating electrical impulses to the ventricles, and its dysfunction can lead to conduction abnormalities like first-degree AV block, where the PR interval exceeds 200 milliseconds.(Greene et al., 1978) While traditionally considered benign in young individuals, emerging evidence suggests first-degree AV block can be associated with risks like atrial fibrillation and pacemaker needs, especially if the PR interval is markedly prolonged (>300 ms), potentially causing atrioventricular dyssynchrony and symptoms.(Han B et al., 2019; Oldroyd et al., 2025)

Amiodarone, a potent Class III antiarrhythmic, also exhibits Class I, II, and IV properties, effectively prolonging cardiac action potential and refractory periods.(Florek et al., 2025) Its calcium channel and antisympathetic actions slow AV nodal conduction, prolonging the PR interval, which is beneficial for SVT but can also cause or worsen AV block.(Florek et al., 2025; Singh et al., 2015) Pseudo-pacemaker syndrome (PPMS) is a rare condition resulting from extreme PR prolongation (often >300-350 ms), where atrial systole occurs close to the preceding ventricular systole, leading to hemodynamic instability without an implanted

pacemaker.(Carroz et al., 2010) This atrioventricular dyssynchrony disrupts efficient cardiac hemodynamics.(Catrina et al., 2024)

In this case report, we aim to describe a young adult male who presented with SVT, revealing an underlying AV nodal conduction delay. Amiodarone administration exacerbated this delay, leading to an extremely prolonged PR interval (>400 ms) mimicking a pseudo-pacemaker syndrome. The primary objective of this study is to present a case of a young adult with SVT and an underlying AV nodal conduction delay, which was severely exacerbated by amiodarone, resulting in PPMS. By analyzing this case, we aim to elucidate the electrophysiological mechanisms behind extreme PR prolongation induced by amiodarone in patients with latent conduction abnormalities. Additionally, this research underscores the importance of thorough AV conduction assessment before administering AV nodal-blocking agents, particularly in young populations where subtle conduction defects may go unrecognized. The findings of this study hold significant clinical value, as they enhance awareness among physicians about the risks of amiodarone in patients with pre-existing conduction delays and emphasize the need for vigilant monitoring post-administration. Furthermore, this report provides diagnostic insights for identifying PPMS in patients with markedly prolonged PR intervals and advocates for personalized therapeutic approaches, including alternative antiarrhythmic strategies or early intervention in high-risk cases. Through this case, we highlight the delicate balance between the therapeutic benefits and potential adverse effects of antiarrhythmic drugs, contributing to improved patient management and outcomes in clinical practice.

CASE PRESENTATION

A 25-year-old male arrived at the emergency department with a primary complaint of palpitations. The patient indicated that the palpitations had persisted for the last three months. These episodes were linked to dyspnea and chest pain that radiated to the left arm and back. The symptoms manifested suddenly and without identifiable causative circumstances. During the preliminary evaluation, the patient was aware and alert. Vital signs showed a blood pressure of 146/83 mmHg and a heart rate of 130 beats per minute. His respiratory rate was 20 breaths per minute, with an oxygen saturation of 98-100% in room air. The physical examination yielded no significant findings.

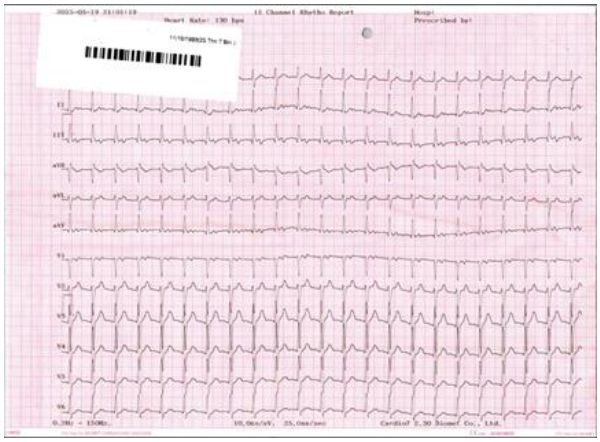

Figure 1. The initial ECG showed SVT with possible aberrancy, AVNRT, or AVRT, with a prolonged PR interval suggestive of underlying AV nodal delay.

An initial electrocardiogram (ECG) suggested supraventricular tachycardia (SVT) accompanied by a prolonged PR interval (Figure 1), implying a potential underlying atrioventricular (AV) nodal conduction delay. Laboratory studies revealed substantial hypokalaemia (3.18 mmol/L), which was swiftly rectified (3.82 mmol/L) with intravenous potassium chloride (KCL). Amiodarone 150 mg was subsequently provided to treat the supraventricular tachycardia. A follow-up ECG post-treatment indicated a return to a normal heart rhythm; however, it exhibited an excessively extended PR interval exceeding 400 ms, suggesting a pseudo pacemaker syndrome (Figure 2). The patient was admitted for a three-day hospitalisation for clinical observation and commenced on Ramipril for hypertension treatment.

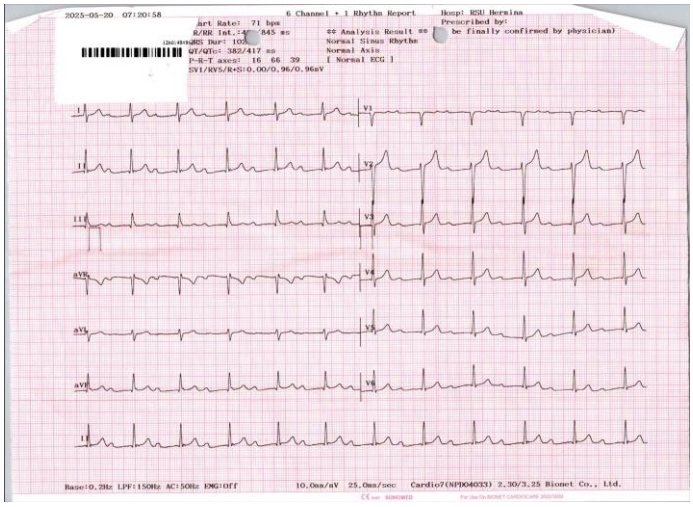

Figure 2. The ECG following amiodarone administration indicated a markedly prolonged PR interval above 400 ms, accompanied by normal heart rates resembling pseudo pacemaker syndrome.

Thyroid function tests were conducted to exclude an underlying thyroid condition as a potential cause of the arrhythmia, and the results were normal. Before discharge, a final ECG was performed, confirming the continuation of the markedly prolonged PR interval (>400 ms) with a normal heart rate (Figure 3). Upon release, the patient indicated the absence of major symptoms and was booked for a follow-up appointment one week subsequently.

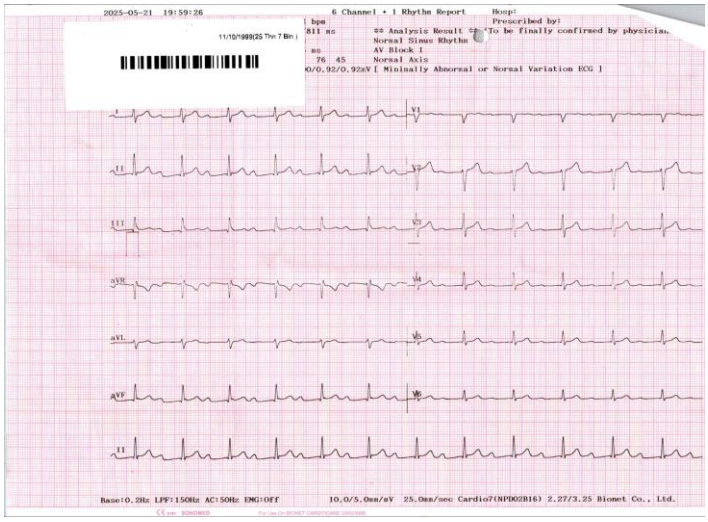

Figure 3. The ECG still suggests a markedly prolonged PR interval above 400 ms, accompanied by normal heart rates resembling pseudo pacemaker syndrome.

Seven days post-discharge, the patient returned for a follow-up appointment. He reported suffering frequent palpitations and dizziness at home, however he said that the symptoms were not more severe than those previous to his initial stay. A following ECG (Figure 4) conducted at this visit indicated that the significantly prolonged PR interval persisted, now ten days after the initial dosage of amiodarone. The patient was scheduled to be referred for a cardiac electrophysiology (EP) investigation to assess the conduction abnormalities, pending insurance authorisation.

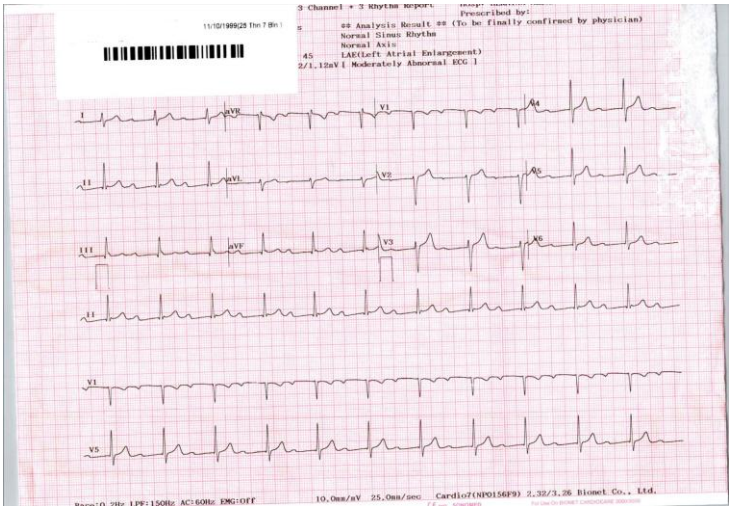

Figure 4. The ECG ten days following amiodarone administration. The ECG stil indicates a significantly extended PR interval over 400 ms.

The management and underlying electrophysiological mechanisms in this young adult presenting with SVT and subsequent profound AV nodal delay following amiodarone administration present several points of discussion. These include the evidence for pre-existing AV nodal dysfunction, the electrocardiographic characteristics of the initial arrhythmia, the role of amiodarone in exacerbating the conduction delay to an extreme degree, pharmacokinetic considerations, contributing factors, and the clinical challenges posed by limited diagnostic facilities.

Result and Discussion

A notable finding in this case is that the initial ECG captured during the SVT episode exhibited a longer PR interval prior to the administration of amiodarone (Figure 1). This finding clearly indicates the existence of a pre-existing AV nodal conduction delay. The patient likely had an undetected, asymptomatic first-degree AV block, which may have been congenital or acquired. First-degree AV block occurs in a minor proportion of young persons and is occasionally linked to increased vagal tone; nevertheless, its occurrence may also indicate a compromised or intrinsically pathological AV conduction system.(Kashou et al., 2025; Oldroyd et al., 2025) The episode of supraventricular tachycardia, characterized by a fast atrial rate(Patti et al., 2025), can exacerbate stress on the AV node and extend conduction time due to the continuous impact of impulses on the AV nodal refractory period.(Hafeez & Armstrong, 2025) However, the documentation of a prolonged PR interval during the tachycardia, as implied by the description of Figure 1, suggests a baseline conduction abnormality that extends beyond a purely rate-related phenomenon. The patient's young age does not preclude the existence of significant AV conduction disturbances or a predisposition to SVT. It has been

suggested that healthy young adults (aged 20 to 30 years) have a prevalence of 1% to 2%, which rises to 3% to 4% by the age of 60.(Nikolaidou et al., 2016) Despite of the rarity, young individuals who present with first-degree AV block, which may be transient or unmasked by other physiological or pathological conditions, have been documented in the literature.(Abdelmageed & Xiao, 2017; Ayan et al., 2011; Greene et al., 1978)

The initial ECG (Figure 1), as previously described, demonstrated SVT with discernible P waves preceding each QRS complex, as well as a prolonged PR interval. The differential diagnosis of narrow complex tachycardias is significantly influenced by this P-wave characteristic. The P waves are frequently obscured in conventional AVNRT, which is characterized by a slow anterograde pathway and a fast retrograde pathway within the AV node. This is due to the near-simultaneous activation of the atria and ventricles, which results in the P waves being buried within the QRS complex or appearing as subtle deflections at the end of the QRS, such as pseudo-R' waves in lead V1 or pseudo-S waves in the inferior leads.(Hafeez & Armstrong, 2025) In orthodromic AVRT, the retrograde P waves typically follow the QRS complex, as it employs the AV node for anterograde conduction and an accessory pathway for retrograde conduction.(Jabbour et al., 2025)

The initial SVT of this patient demonstrates the clear visibility of P waves prior to each QRS complex, which reduces the likelihood of typical AVNRT or common forms of AVRT. This ECG pattern broadens the differential to encompass atrial tachycardia with 1:1 AV conduction over a diseased AV node (hence the prolonged PR interval) or less common variants of AVNRT, such as atypical (fast-slow or slow-slow) AVNRT.(Hafeez & Armstrong, 2025) In these atypical forms, the retrograde P wave may manifest later in the cardiac cycle, occasionally presaging the subsequent QRS complex with a prolonged RP interval.(Hafeez & Armstrong, 2025) Additionally, there is the possibility of permanent junctional reciprocating tachycardia (PJRT), a rare form of supraventricular tachycardia (SVT) that is distinguished by a prolonged RP interval, frequently accompanied by inverted P waves in the inferior leads, as a result of retrograde conduction over a slowly conducting accessory pathway.(Kohli et al., 2024) The presence of dual AV nodal physiology can also result in a variety of ECG manifestations, such as varying PR intervals and conduction patterns, which can be complex at times.(M. Weiss & Ho, 2020) This atypical presentation is aptly captured in the description of "SVT with prolonged PR interval" for Figure 1. It is noteworthy that the 1:1 AV conduction was maintained during the SVT, despite the significantly extended PR interval. It implies that, despite the fact that AV nodal conduction was significantly sluggish, it had not yet reached the stage of decremental conduction, which would have resulted in Wenckebach periodicity or 2:1 block at the specific atrial rate.(Singh et al., 2015) This suggests a particular type of AV nodal pathology that leads to a uniform slowing of conduction at that rate, as opposed to the intermittent failure of conduction that is more commonly observed in more typical rate-related AV nodal fatigue.

Amiodarone, with its complex array of ion channel blocking effects, predictably slows AV nodal conduction and prolongs the PR interval.(Florek et al., 2025) However, the degree of PR interval prolongation observed in this patient, exceeding 400 ms following amiodarone administration (Figure 2), represents an extreme and uncommon electrophysiological response.(Taieb et al., 2019; Zhang et al., 2025) The administration of amiodarone appears to have profoundly exacerbated the pre-existing AV nodal conduction delay(Taieb et al., 2019),

transforming what was likely a first-degree AV block into an exceptionally long conduction time, consistent with the development of pseudo-pacemaker syndrome.(Carroz et al., 2010; Ogunlade et al., 2014) This syndrome, as previously defined, arises when the PR interval is so prolonged that atrial contraction occurs inefficiently, either against closed AV valves or into a ventricle that is already nearly full from the previous systole, leading to symptoms of hemodynamic compromise.(Barber & Kusumoto, 2020)

The persistence of the markedly prolonged PR interval (still exceeding 400 ms) even 10 days after the initial amiodarone administration (Figure 4) is entirely consistent with the drug's well-documented pharmacokinetic profile. Amiodarone has an exceptionally long half-life, and the full clinical effects may not be observed until six weeks after oral therapy. Furthermore, the pharmacological effects of amiodarone may endure for a period of 1 to 3 months following its cessation of treatment. amiodarone accumulates extensively in a variety of tissues, particularly adipose tissue, as a result of its high lipophilicity. This results in a significant volume of distribution and a very slow release back into the systemic circulation. As a result, the electrophysiological effects of the drug, whether beneficial or detrimental, may endure for weeks or even months following its discontinuation.(Florek et al., 2025; Skeoch et al., 2018) Although the pharmacokinetic characteristic of amiodarone's effects is anticipated, the sustained degree of PR prolongation (remaining >400 ms) for a minimum of 10 days following what was likely an acute loading dose regimen for SVT termination is still remarkable. It implies that the drug's effect is not easily or quickly reversible, nor is it readily compensated for by physiological mechanisms, even if plasma concentrations have begun to decline from their peak levels. This has substantial implications for the potential risk of recurrent symptoms, long-term monitoring, and perhaps even progression to more severe forms of AV block.

In the diagnostic evaluation of arrhythmias and conduction disturbances, particularly when amiodarone is involved, thyroid dysfunction must be considered. Amiodarone can cause both hypothyroidism and hyperthyroidism, especially with chronic use, and thyroid disorders themselves can independently lead to cardiac arrhythmias and conduction abnormalities.(Barrett & Bauer, 2021; Florek et al., 2025) In this case, thyroid function tests were reportedly normal, effectively ruling out a thyroid-related cause for the observed electrophysiological changes and strengthening the focus on the primary AV nodal pathology and amiodarone's direct pharmacological impact.

The patient's initial presentation included significant hypokalemia, with a serum potassium level of 3.18 mmol/L. Hypokalemia is a well-established trigger for a variety of cardiac arrhythmias, including SVT, as it alters myocardial cell membrane excitability, resting membrane potential, and action potential duration.(Thu Kyaw & Maung, 2022; J. N. Weiss et al., 2017) Furthermore, hypokalemia can itself contribute to PR interval prolongation.(Wang et al., 2020) While the hypokalemia was promptly corrected with intravenous potassium chloride , it likely played a role in the genesis of the initial SVT episode, which subsequently led to the administration of amiodarone.(Page et al., 2016) It is conceivable that the initial hypokalemia might have further compromised AV nodal conduction, synergizing with the patient's underlying AV nodal substrate to make the SVT presentation more complex. The correction of the electrolyte imbalance (3.82 mmol/L) would have removed this acute

contributing factor, but the underlying vulnerability of the AV node and its susceptibility to amiodarone's effects evidently remained.

An electrophysiology (EP) study was not performed due to limitations in facility resources. An EP study, involving the placement of intracardiac catheters to record and stimulate various parts of the heart, is the gold standard for definitively diagnosing the precise mechanism of complex SVTs (e.g., differentiating between atrial tachycardia, atypical AVNRT, or PJRT) and for localizing the exact anatomical level of AV block (e.g., within the AV node itself, or in the His-Purkinje system).(Page et al., 2016; Patti et al., 2025) The absence of such a study means that while a strong clinical and electrocardiographic diagnosis can be made, a degree of uncertainty regarding the precise electrophysiological substrate inherently remains.

In clinical settings where advanced diagnostic modalities are not readily available, the critical importance of meticulous clinical assessment, detailed and sequential ECG interpretation, and a sound understanding of arrhythmia mechanisms is underscored by this limitation. This patient's ECG series offers a compelling account of the events; however, the absence of intracardiac data has prevented the complete elucidation of the precise nature of the conduction defect and certain nuances of the SVT mechanism. This situation is not uncommon in numerous healthcare environments worldwide and underscores the practical obstacles that clinicians encounter when managing intricate cardiac electrophysiological disorders outside of highly specialized tertiary centers. It underscores the fact that clinicians frequently must make critical management decisions based on the most comprehensive diagnostic information available, despite the fact that this information is occasionally incomplete. In such circumstances, the meticulous documentation and astute interpretation of serial surface ECGs, as was demonstrated in this instance, are essential for the purpose of guiding therapy and comprehending the underlying pathophysiology. Additionally, this case reinforces the validity of referring a young patient with a significant and unusual conduction abnormality to a specialized electrophysiology center when feasible, in order to facilitate a definitive diagnosis and inform long-term management strategies.

CONCLUSION

This case underscores the critical importance of thoroughly assessing cardiac conduction in young adults before administering AV nodal blocking agents such as amiodarone, as unrecognized underlying conduction delays can be significantly worsened by the drug. Amiodarone’s potent effects may lead to extreme PR interval prolongation (>400 ms) and provoke rare but clinically significant conditions like pseudo-pacemaker syndrome, characterized by symptoms arising from AV dyssynchrony despite a normal heart rate. Given amiodarone's long half-life and persistent electrophysiologic effects, careful and prolonged clinical monitoring is essential. Future research should focus on identifying reliable predictors of latent AV nodal dysfunction and developing guidelines for safer use of AV nodal blocking antiarrhythmics in patients with subtle conduction abnormalities to minimize the risk of iatrogenic complications.

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