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Unveiling Asymptomatic Atrioventricular Reciprocating Tachycardia in a Patient With Wolff-Parkinson-White Syndrome and Multiple Sclerosis

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Patient: Female, 54-year-old
Final Diagnosis: Wolff-Parkinson-White syndrome (WPW)
Symptoms: Palpitation
Clinical Procedure: Ablation
Specialty: Cardiology • Neurology
Objective: Rare coexistence of disease or pathology
Background: Intermittent accessory pathway conduction can obscure the electrocardiographic diagnosis of Wolff-Parkinson-White (WPW) syndrome on resting electrocardiograms (ECGs). Furthermore, sensory disruptions characteristic of multiple sclerosis (MS) can blunt symptom perception, masking potentially life-threatening hemodynamic instability.

Case Report: A 54-year-old woman with secondary-progressive MS (SPMS) was admitted for a complicated urinary tract infection. The admitting ECG demonstrated sinus tachycardia without preexcitation. Telemetry subsequently revealed narrow-complex tachycardia at 220 beats per minute (BPM) with concurrent hypotension. Despite hemodynamic instability, the patient explicitly denied palpitations, chest pain, or presyncope. She had discontinued fingolimod several years earlier because of palpitations, a detail retrospectively suggestive of an accessory pathway. Review of records identified historical ECGs with intermittent delta waves and short PR intervals, confirming the presence of an accessory pathway with intermittent antegrade conduction. A 12-lead ECG during the acute event confirmed orthodromic atrioventricular reciprocating tachycardia (AVRT). The patient underwent successful synchronized cardioversion followed by radiofrequency ablation of a left-sided antero-lateral accessory pathway via electrophysiological study (EPS).

Conclusions: This case illustrates the “ECG dilemma” of intermittent preexcitation, in which the absence of delta waves on a resting tracing does not exclude accessory pathway-mediated tachycardia. It further highlights that patients with neuro-modulating diseases, such as MS, may lack classical “alarm” symptoms for arrhythmia due to progressive autonomic and sensory dysfunction. Clinicians must maintain a high index of suspicion for concealed arrhythmias in this population, as neurological impairment can dissociate hemodynamic instability from subjective symptom perception. Objective monitoring with telemetry is essential to prevent adverse outcomes in this vulnerable population.

Keywords: Arrhythmias, Cardiac • Autonomic Nervous System • Demyelinating Diseases • Electrocardiography • Wolff-Parkinson-White Syndrome

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Introduction

Wolff-Parkinson-White (WPW) syndrome is characterized by antegrade conduction through an accessory atrioventricular pathway, producing the classic triad of a short PR interval, delta wave, and widened QRS complex on the resting ECG [1]. However, intermittent preexcitation, where the accessory pathway conducts only inconsistently, can render individual ECG tracings indistinguishable from normal sinus rhythm, creating a diagnostic trap for clinicians [2]. Furthermore, the symptomatic profile of WPW-associated tachycardia depends substantially on intact autonomic and sensory signaling, which can be profoundly impaired in progressive neurological disease [3].

Multiple sclerosis (MS) is an immune-mediated demyelinating disease that in its advanced progressive forms frequently disrupts both somatic and autonomic neural pathways. Dysautonomia has been reported in up to 84% of patients with progressive MS, and impaired visceral interoception may prevent the perception of otherwise severe hemodynamic events [3]. Although direct studies in MS are scarce, this cardiovascular autonomic dysfunction provides a plausible mechanism for blunted awareness of tachyarrhythmia and ischemic chest pain. This parallels the silent myocardial ischemia of diabetic autonomic neuropathy [4]. This intersection of electrophysiological concealment and neurological desensitization has not been previously reported in the literature to our knowledge.

We report a case of asymptomatic AVRT requiring synchronized cardioversion in a patient with SPMS and unrecognized WPW syndrome, presenting with hemodynamic instability in the absence of any cardiac symptoms.

Case Report

A 54-year-old woman with a history of secondary-progressive multiple sclerosis (SPMS), paroxysmal atrial fibrillation, osteoporosis, a thyroid adenoma, and depression was admitted to a tertiary care hospital with fever, nausea, and malaise consistent with a complicated urinary tract infection. Presenting vital signs included a temperature of 38.7 °C, blood pressure of 128/80 mm Hg, heart rate of 125 BPM, respiratory rate of 20 breaths per minute, and oxygen saturation of 93% on room air. The combination of fever, tachycardia, tachypnea, and mild hypoxemia met systemic inflammatory response criteria. This indicated a complicated, likely upper-tract urinary infection with early urosepsis rather than simple cystitis. Chest radiography on admission demonstrated no acute cardiopulmonary process. The mild hypoxemia (oxygen saturation 93% on room air) was attributed to the acute febrile and septic illness and resolved with fluid resuscitation, antimicrobial therapy, and serial monitoring. She was resuscitated with 30 mL/kg of

0.9% NaCl and started empirically on intravenous ceftriaxone 2 g daily. Urine culture on hospital day 2 confirmed pan-sensitive *Escherichia coli*. Due to persistent intermittent tachycardia, continuous telemetry monitoring was initiated.

The patient denied any history of syncope, chest discomfort, dyspnea, or recurrent palpitations. Notably, she had previously reported palpitations during a prior course of fingolimod for MS, which resolved upon its discontinuation. Her current medications included methimazole 5.0 mg every other day, baclofen 10 mg 3 times daily, naproxen 250 mg daily, venlafaxine 150 mg daily, calcium citrate 630 mg daily, vitamin D 400 IU twice daily, and magnesium oxide 400 mg daily. The admitting ECG (**Figure 1**) demonstrated sinus tachycardia at 109 BPM without evidence of preexcitation.

On the morning of hospital day 3, telemetry revealed the onset of a narrow-complex supraventricular tachycardia at 220 BPM. Bedside assessment confirmed hypotension with a blood pressure of 90/50 mm Hg. Remarkably, the patient remained comfortable in bed and explicitly denied chest pain, palpitations, presyncope, or dyspnea throughout the episode. A 12-lead ECG (**Figure 2**) confirmed narrow-complex tachycardia at 211 BPM with retrograde P waves and a long RP interval greater than 70 milliseconds, consistent with orthodromic AVRT. Morning laboratory values demonstrated hypokalemia (3.1 mmol/L), hypomagnesemia (1.6 mg/dL), and subclinical hyperthyroidism (thyroid-stimulating hormone 0.15 μ U/mL; free T4 0.84 ng/dL), consistent with her known thyroid adenoma on methimazole therapy.

A review of the electronic medical record revealed historical ECGs from years prior that intermittently demonstrated short PR intervals (82 ms), delta waves, widened QRS complexes (120 ms), and right bundle branch morphology (**Figure 3**), consistent with the WPW pattern. The patient was transferred to the intensive care unit and underwent synchronized direct current cardioversion at 250 J, successfully restoring normal sinus rhythm. Potassium (80 mEq) and magnesium (4 g) were administered following cardioversion. On hospital day 4, electrophysiological study (EPS) was performed with electroanatomic mapping, AVRT induction, and radiofrequency ablation of a left-sided anterolateral accessory pathway demonstrating both retrograde and antegrade conduction. The patient remained hemodynamically stable after the procedure and was discharged on hospital day 5 following completion of her antibiotic course. At outpatient follow-up approximately 6 months after discharge (at 55 years of age), she remained free of chest pain, palpitations, or dyspnea, with no recurrent tachyarrhythmia. Over this interval her SPMS continued to progress, ultimately requiring home health care for contractures and a largely bedbound status.

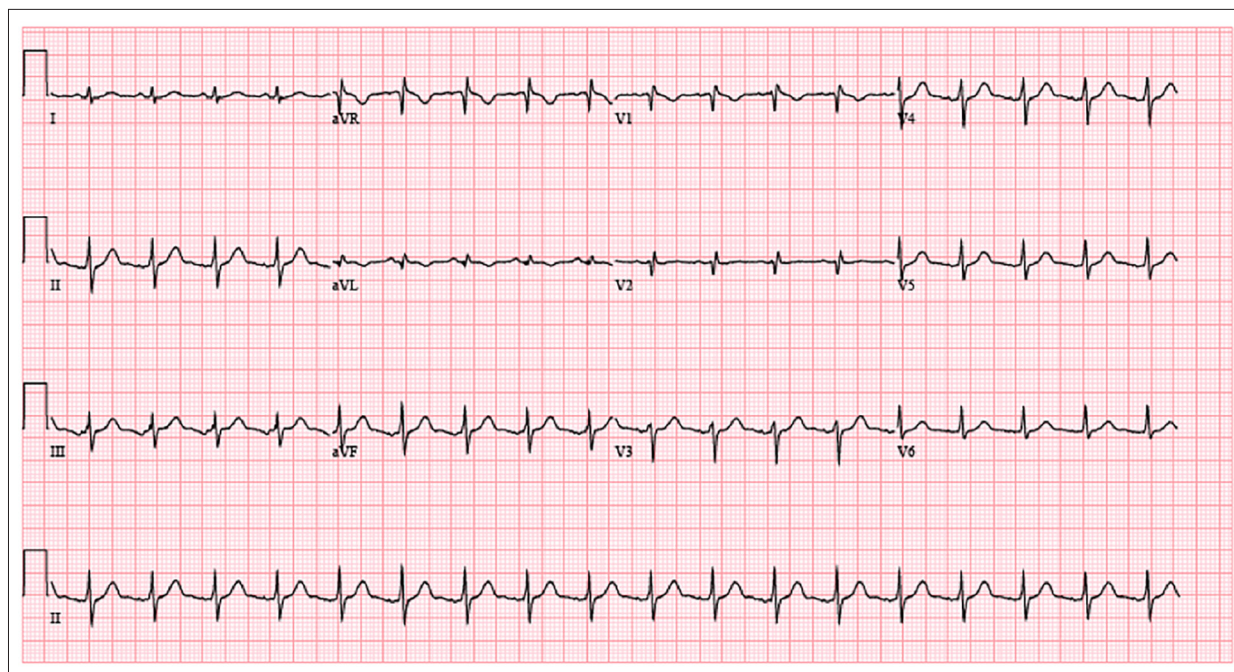


Figure 1. Presenting 12-lead ECG consistent with sinus tachycardia. ECG demonstrates normal sinus tachycardia at 109 BPM with a normal PR interval (112 ms) and without evidence of preexcitation. ECG, electrocardiogram; BPM, beats per minute.

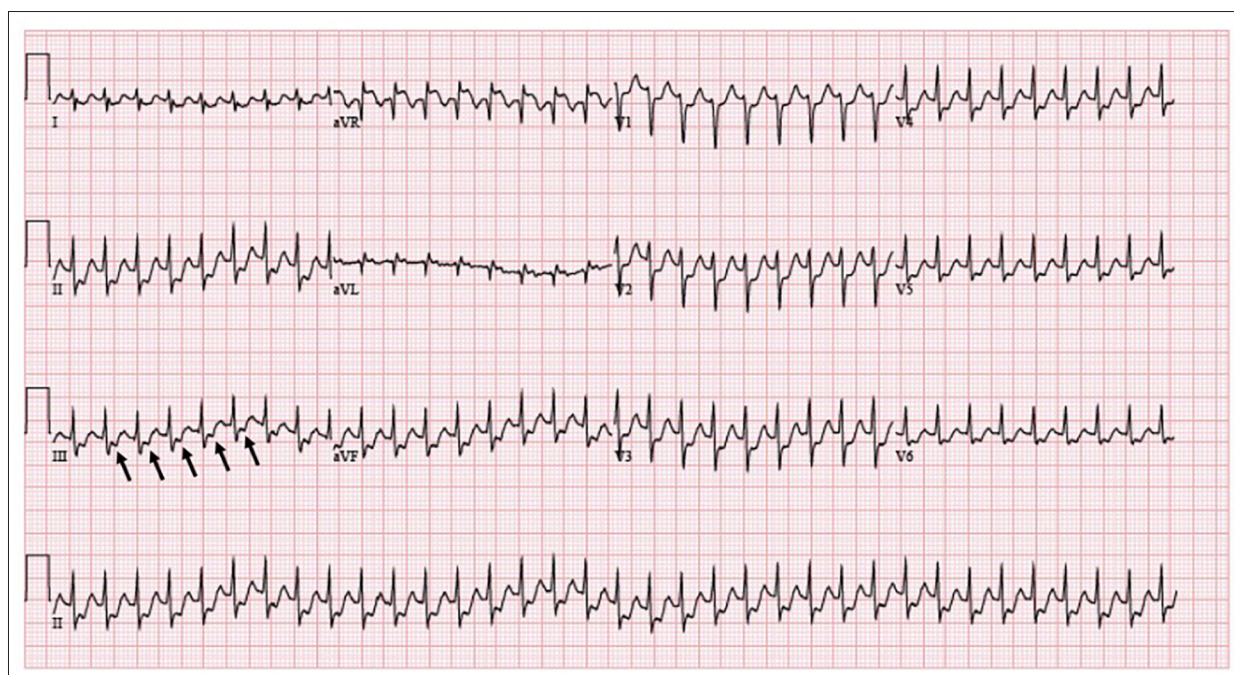


Figure 2. 12-lead ECG demonstrating narrow-complex orthodromic atrioventricular reciprocating tachycardia. ECG demonstrates narrow-complex tachycardia at 211 BPM with retrograde P waves (black arrow) and a long RP interval (> 70 ms), consistent with orthodromic AVRT. ECG, electrocardiogram; AVRT, atrioventricular reciprocating tachycardia; BPM, beats per minute.

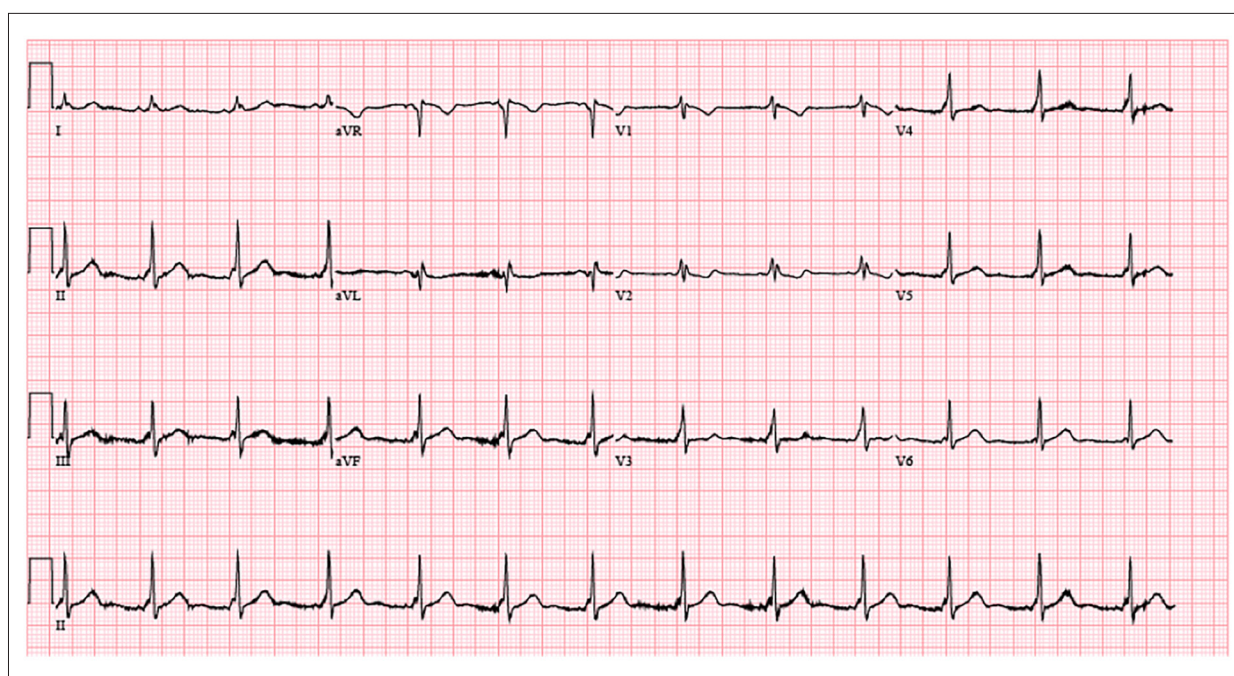


Figure 3. Historical 12-lead ECG consistent with the Wolff-Parkinson-White pattern. ECG demonstrates normal sinus rhythm at 76 BPM with a short PR interval (82 ms), delta waves, widened QRS complexes (120 ms), and right bundle branch morphology, consistent with the Wolff-Parkinson-White pattern. ECG, electrocardiogram; BPM, beats per minute.

Discussion

This case presents a unique intersection of 2 independent diagnostic challenges: the electrophysiological concealment of intermittent preexcitation and the clinical silence of hemodynamically significant tachycardia in progressive MS. To our knowledge, this is the first reported case of asymptomatic AVRT in the setting of MS.

The primary diagnostic challenge was the absence of preexcitation on the admitting ECG. Classically, the WPW pattern is identified by a resting ECG demonstrating a delta wave, signifying antegrade accessory pathway conduction [1]. However, intermittent preexcitation reflects a relatively long antegrade refractory period in the accessory pathway, which can lead to rate-dependent or autonomic tone-dependent block and an entirely normal-appearing resting tracing [2]. In this case, the pathway failed to manifest antegrade conduction on admission, providing false reassurance about the patient's arrhythmic risk. Critically, as established by both the 2015 ACC/AHA/HRS and 2019 ESC guidelines, the failure of an accessory pathway to conduct antegrade on a resting ECG does not preclude its ability to sustain orthodromic AVRT through retrograde conduction [5,6]. This case reinforces that a negative resting ECG cannot exclude accessory pathway pathology, and that historical ECG review is essential in patients with supraventricular tachycardia of unclear etiology. The heightened sympathetic tone accompanying the patient's complicated urinary

tract infection may also have compounded this concealment. Increased adrenergic drive accelerates atrioventricular nodal conduction. The normal conduction system can then depolarize the ventricles before the accessory pathway contributes, diminishing or abolishing the delta wave on the admitting tracing [2].

A further insight concerns why the accessory pathway evaded recognition for so long. The successful ablation target (a left anterolateral pathway on the mitral annulus) itself accounts for the subtle and intermittent preexcitation [7]. Left lateral free-wall pathways lie remote from the sinoatrial node, so the sinus impulse must traverse much of the atrium before engaging the pathway. By then the atrioventricular node has frequently already begun to conduct, allowing nodal activation to predominate and producing only minimal ventricular preexcitation. Delta waves arising from such pathways are therefore often small or inapparent on the surface ECG and are easily overlooked, especially when preexcitation is also intermittent [2]. This anatomic-electrophysiologic feature plausibly explains the years-long delay in recognizing this patient's WPW pattern. It also shows the educational value of reviewing historical tracings for subtle preexcitation.

The second and equally instructive learning point is the complete absence of symptoms despite a heart rate of 220 BPM and concurrent hypotension. In the general population, such hemodynamic compromise predictably provokes palpitations, presyncope, or frank syncope. The patient's earlier history of

perceiving palpitations while on fingolimod suggests that interoception was intact at that time. Her subsequent inability to perceive the acute tachyarrhythmia likely reflects a progression of sensory neuropathy, parallel to her clinical transition from relapsing-remitting to secondary-progressive MS. Autonomic dysfunction is a well-documented complication of MS, affecting up to 84% of patients with progressive disease, and can manifest as attenuated visceral perception [3]. This phenomenon closely mirrors the loss of afferent cardiac innervation seen in diabetic autonomic neuropathy, where silent myocardial ischemia occurs in the absence of angina [4]. In patients with advanced neuro-modulating disease, the subjective review of systems may be deceptively negative, and objective monitoring becomes the only reliable safety net.

In retrospect, the discontinuation of fingolimod due to palpitations years prior may have been a missed opportunity for early diagnosis. While fingolimod is associated with bradyarrhythmias and atrioventricular block [8], it is not strictly contraindicated in WPW syndrome when the substrate has been definitively treated. Had the palpitations at that time been correctly attributed to WPW-mediated tachycardia, facilitated by recognition of the intermittently present delta waves, early catheter ablation may have been curative and could have permitted continuation of high-efficacy disease-modifying therapy. This accords with published literature supporting ablation in symptomatic patients to facilitate otherwise necessary pharmacotherapy [5]. Early intervention might have altered the trajectory of her neurological disease.

Several factors likely contributed to the precipitation of AVRT in this hospitalized patient. Hypokalemia, hypomagnesemia, and subclinical hyperthyroidism are known to increase adrenergic tone and lower the threshold for arrhythmia [5]. Importantly, the urinary infection itself was likely the proximate trigger. The adrenergic and inflammatory surge of early sepsis, fever-related catecholamine release, and the resulting electrolyte derangements together lowered the threshold for reentry. This precipitated orthodromic AVRT in a patient with a long-standing but previously quiescent accessory pathway. The presence of continuous telemetry monitoring, initiated for intermittent sinus tachycardia, was the only factor that enabled prompt detection of the hemodynamically significant, yet subjectively silent, tachyarrhythmia.

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Conclusions

This case of asymptomatic AVRT requiring synchronized cardioversion in a patient with SPMS and unrecognized WPW syndrome illustrates 2 important clinical principles. First, the absence of delta waves on a resting ECG does not exclude accessory pathway pathology, and forensic review of historical ECGs is essential when the clinical picture warrants. Second, in patients with neuro-modulating diseases such as progressive MS, neurological dysfunction can dissociate hemodynamic instability from subjective symptom perception, rendering the clinical presentation deceptively benign. Clinicians must maintain a high index of suspicion for concealed arrhythmias in this vulnerable population, and objective telemetry monitoring should be used liberally to prevent catastrophic outcomes. Finally, earlier recognition of WPW syndrome might have carried implications beyond cardiac care. Had the intermittent preexcitation been identified when the patient first reported palpitations on fingolimod, curative catheter ablation might have been feasible. This could have permitted continuation of high-efficacy disease-modifying therapy and might thereby have altered the trajectory of her multiple sclerosis.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Institution Where Work Was Done

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

Abbreviations

ECG, electrocardiogram; **WPW**, Wolff-Parkinson-White; **MS**, multiple sclerosis; **RRMS**, relapsing-remitting multiple sclerosis; **SPMS**, secondary progressive multiple sclerosis; **AVRT**, atrioventricular reciprocating tachycardia; **EPS**, electrophysiological study; **BPM**, beats per minute.

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