JKU 15(2) (2026)

Jurnal Kedokteran Unram

https://journal.unram.ac.id/index.php/jku

Suspected Acute Myocarditis in Wolff Parkinson White Syndrome Presenting with Supraventricular Tachycardia and ST Elevation: A Case Report

I Gusti Ayu Aruna Krisnadewani[1], Bayu Setia[2]

[1] General Practitioner, Intern of Cardiology and Vascular Department, RSUD Kota Mataram, Indonesia.

[2] Cardiologist, Cardiology and Vascular Department, RSUD Kota Mataram, Indonesia.

DOI: https://doi.org/10.29303/jk.v15i2.9653

Article Info

Received : **February 23, 2026**

Revised : **June 23, 2026**

Accepted : **June 24, 2026**

Abstract

Myocarditis defined as inflammation of myocardium in response to acute injury. It can cause general and unclear symptoms. On ECG, it may show ST-segment elevation, which often mimicking ACS. Meanwhile, Wolff Parkinson White syndrome is a pre-excitation cardiac condition that is characterized by an accessory conduction pathway that predisposes individuals to tachyarrhythmias, including supraventricular tachycardia. When both conditions co-exist, diagnosis and management become particularly challenging. A 20-year-old male was admitted to our hospital with palpitations and diaphoresis 8 hours prior to admission, after playing soccer a night before, accompanied with shortness of breath and crushing chest pain. History of recent viral infections were denied. His initial BP was 100/70 mmHg and HR was 200 beats per minute, with normal physical examination. Admission ECG showed AVRT orthodromic. After vagal maneuver, ECG converted to sinus rhythm with ST elevation on inferior leads and suggesting WPW syndrome, troponin level was elevated. Coronary angiography revealed normal coronary arteries except for myocardial bridging in LAD. CRP and ASTO titers were elevated. Cardiac MR and endomyocardial biopsy were not done yet. This patient then diagnosed with WPW syndrome and suspected myocarditis. In WPW patients, the conduction of electrical pulses is no longer limited to the AV pathway due to the presence of accessory pathway, Bundle of Kent, that has considerable potential to cause supraventricular arrhythmias, which the important route is the AVRT route. In this case, myocarditis is thought to be the cause. Myocarditis can trigger arrhythmias due to various factors, including alteration of cardiac structural, vascular involvement, and changes in membrane potential triggered by inflammation. This case report highlights that myocarditis can act both as a substrate and a trigger for SVT in patients with WPW syndrome. When SVT occurs in WPW patients, we should consider myocarditis as the underlying cause. Early recognition could guide to appropriate management and prevent potential complications associated with both arrhythmia and myocardial inflammation.

Keywords: myocarditis, Wolff-Parkinson-White syndrome, supraventricular tachycardia, ST-elevation

Citation: Krisnadewani, I.G.A.A., & Setia, B. (2026). Suspected Acute Myocarditis in Wolff Parkinson White Syndrome Presenting with Supraventricular Tachycardia and ST Elevation. *Jurnal Kedokteran Unram, 15(2)*, 76-79. DOI: https://doi.org/10.29303/jk.v15i2.9653

Introduction

Myocarditis defined as inflammation of myocardium in response to acute injury. It can cause

Email: arunakrisnadewani@gmail.com

general and unclear symptoms. On an electrocardiogram (ECG), it may show ST-segment elevation, which often mimicking acute coronary syndromes (ACS) (Fernando et al., 2020; Drazner, et al.,2025). Meanwhile, Wolff Parkinson White (WPW) syndrome is a pre-excitation cardiac condition that is characterized by an accessory conduction pathway, known as Bundle of Kent. This accessory pathway predisposes individuals to tachyarrhythmias, including supraventricular tachycardia (SVT) (Vijay, et al., 2022). When both conditions co-exist, diagnosis and management become particularly challenging.

Case Presentation

A 20-year-old male was admitted to our hospital with palpitations and diaphoresis 8 hours prior, after playing soccer a night before. The palpitations then followed with blurry vision and shortness of breath. He also complained chest pain with similar onset. The pain was described like crushing in the substernal area, no radiation. He denied any history of recent viral infections (upper respiratory tract infection or gastrointestinal infection). He smoked 4 years ago but had stopped in the same year. His initial blood pressure in emergency room was 100/70 mmHg and heart rate was 200 beats per minute. From physical examination no cardiac murmur was found nor jugular vein engorgement, breathing sound was normal and no oedema on lower extremities. Admission ECG showed SVT AVRT orthodromic pattern (Figure 1). Vagal maneuver was done for the initial management, the ECG then change into sinus rhythm with ST-elevation on lead II, III, aVF along with short PR interval and delta waves on lead V2-V5 (Figure 2). From the laboratory findings, the troponin level was elevated (4.991 ng/dL). This patient immediately went under percutaneous coronary intervention (PCI). The coronary angiography showed normal left main coronary artery (LM), right coronary artery (RCA), and left circumflex artery (LCx), with myocardial bridging in left anterior descending artery (LAD) (Figure 3).

The day later, the patient had C-Reactive Protein (CRP) and Anti Streptolysin O (ASTO) checked with positive result, 8.1 mg/L (normal <5 mg/L) and 400 IU/mL (normal <200 IU/mL) consecutively. Transthoracic echocardiography (TTE) revealed left ventricular hypertrophy (LVH), no regional wall motion abnormalities, mild mitral regurgitation (MR), mild tricuspid regurgitation (TR), mild pulmonal regurgitation (PR) with ejection fraction (EF) by TEICH 58%. Cardiac Magnetic Resonance (CMR) and endomyocardial biopsy (EMB) were not done yet in this patient. This patient then diagnosed with WPW syndrome with suspected myocarditis. Management including benzathine penicillin, bisoprolol, ramipril, and suggested to do EP study and ablation.

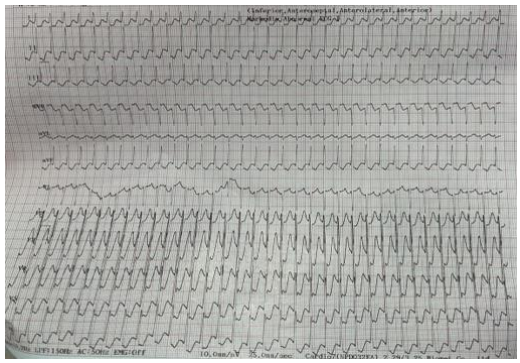

Figure 1. Admission ECG showing AVRT orthodromic

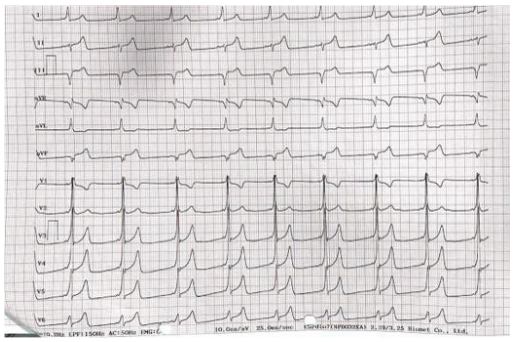

Figure 2. Post vagal maneuver ECG, showing inferior ST elevation, short PR interval and delta waves on lead V2-V5

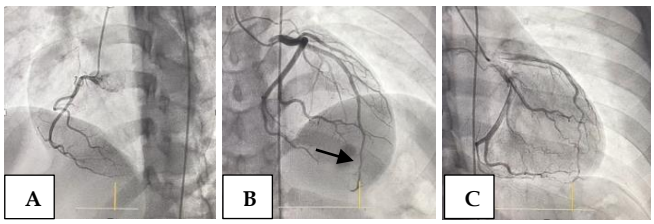

Figure 3. Patient's coronary angiography showing A: normal right coronary artery (RCA), B: myocardial bridging in left anterior descending artery (LAD) (arrow), C: normal left circumflex artery (LCx)

Discussion

Myocarditis defined as inflammation of myocardium in response to acute injury that resulting in myocytes' necrosis and degeneration. Myocarditis presentation is highly variable and may be misdiagnosed with myocardial infarction. They had overlapping clinical features including chest discomfort, palpitations, fatigue, fever, shortness of breath, leg swelling, and syncope (Fernando et al., 2020; Drazner, et al.,2025). There are three classic clinical presentations that being consistent with myocarditis: chest pain, arrhythmia, or heart failure/cardiogenic shock. Also

need to be questioned about history of antecedent viral infection, such as upper respiratory tract infection or gastrointestinal infection in a young person, a prior diagnosis of myocarditis or autoimmune disease, family history of cardiomyopathy or unexplained sudden death, and exposure to known cardiotoxin (Drazner, et al., 2025). A review of case series and research has shown that patients with myocarditis mimicking STEMI are typically younger, often report a recent infection, and present with elevated inflammatory markers. Whereas obesity (BMI >30 kg/m^2), smoking, and arterial hypertension, as risk factors, were significantly more related to myocardial infarction (Fernando et al., 2020; Drazner, et al.,2025; Wieczorkiewicz, et al., 2022). In their study, Wieczorkiewicz et al, suggested the three-parameter model: history of recent infection, CRP value on admission > 5 mg/L, and the age of <36 years could be essential in differentiating between myocarditis and myocardial infarction on admission (Wieczorkiewicz, et al, 2022).

There are several tests that can be done to evaluate a patient with clinically suspected myocarditis. Cardiac Magnetic Resonance (CMR), particularly with T_1 and T_2 parametric mapping and endomyocardial biopsy (EMB) are considered pivotal in the diagnostic workup. Due to frequent presence of chest pain, ECG abnormalities, and elevated high-sensitivity troponin levels, clinicians frequently prioritize excluding myocardial infarction through coronary angiography, especially given the urgency of managing ischemic events and the less acute nature of myocarditis in most cases. CMR offers a non-invasive method to assess myocardial tissue characteristics and plays a critical role in supporting the diagnosis of myocarditis. While distinguishing myocarditis from STEMI can be challenging in certain cases, a combination of cardiac MRI findings, a detailed clinical history, and established diagnostic criteria for suspected myocarditis can help raise clinical suspicion (Fernando et al., 2020; Drazner, et al.,2025).

Wolff Parkinson White (WPW) Syndrome is a pre-excitation cardiac condition characterized by an accessory pathway, known as Bundle of Kent, that bypasses the atrioventricular node and predisposes individuals to tachyarrhythmias (Vijay, et al., 2022). In WPW patients, the path and conduction path of electrical pulses are no longer limited to the AV pathway for the transmission from the atrium to the ventricle due to the presence of accessory pathway. In such a way that it has considerable potential to cause supraventricular arrhythmias, which the important route is the atrioventricular reciprocating tachycardia (AVRT) route. This route divided into two categories, orthodromic and antidromic. In orthodromic type, the electrical pulse enters the ventricle from the AV path and is antegrade and enters the atria through the accessory pathway as retrograde. Meanwhile, in antidromic type, electrical pulses enter the ventricle through the accessory pathway in the form of antegrade from the atrium to the ventricle and return to the atrium (Afshar, et al., 2021; Balta, et al., 2022; Aziz & Rizal, 2024). About 10% patients with WPW syndrome present with antidromic supraventricular tachycardia (SVT), where the accessory pathway functions as the anterograde conduit and the retrograde conduit might be represented by either the typical conduction mechanism or additional accessory pathway (Vijay, et al., 2022; Aziz & Rizal, 2024; Brugada, et al., 2020).

In myocarditis patients (acute or chronic), arrhythmias are commonly found and can be the only clinical symptom in the natural course of the disease. Potentially fatal tachyarrhytmias and bradyarrhythmias play a crucial role in the differential diagnosis of sudden cardiac death in patients with myocarditis. The increased risk of arrhythmias arises from alterations of myocardial structure, abnormalities in ventricular mechanics, and vascular disturbances. Inflammatory involvement of cardiac myocytes and the interstitial tissue can directly disrupt membrane potentials, predisposing to electrical instability. In addition, fibrosis, scar formation, and secondary myocyte hypertrophy or atrophy promote the development of ectopic foci, late potentials, and reentrant circuits due to heterogenous impulse conduction. Abnormal ventricular dynamics--such as increased wall stress, elevated myocardial oxygen demand, and reduced coronary reserve in the setting of impaired systolic or diastolic left ventricular function—further enhance arrhythmogenic susceptibility. Vascular factors also contributing for this process, as macrovascular and microvascular perfusion abnormalities may lead to myocardial ischemia, then amplifying electrical instability in the inflamed myocardium (Klein, et al., 2000; Peretto, et al., 2018).

Simple and noninvasive tests, including 24-hour Holter monitoring can be used in patients with myocarditis for checking ventricular late potentials and measuring heart rate variability. These tests help estimate the patient's risk but cannot predict malignant arrhythmias with high accuracy. Programmed atrial and ventricular electrophysiological stimulation can help predict ventricular tachyarrhythmias, although its accuracy is only moderate. In selected patients, immunosuppressive therapy combined with standard antiarrhythmic treatment may reduce or even eliminate spontaneous and inducible arrhythmias. However, some patients with serious arrhythmias still need additional treatment. This may include long-term

antiarrhythmic medication, ventricular tachycardia ablation, and implantation of an implantable cardioverter-defibrilator (ICD). Still, right ventricular endomyocardial biopsy remains a valuable diagnostic tool for confirming or excluding myocarditis and guiding management in patients with arrhythmias of uncertain origin (Klein, et al., 2000; Peretto, et al., 2018).

Conclusion

This case report highlights that myocarditis can act both as a substrate and a trigger for SVT in patients with WPW syndrome. When SVT occurs in WPW patients, we should consider myocarditis as the underlying cause. Early recognition could guide to appropriate management and prevent potential complications associated with both arrhythmia and myocardial inflammation.

References

Afshar, M.M., Moshaver, M.H., Khodamoradi, Z., et al. (2021). WPW Syndrome: Review of the Evidence. *Nov Appro Drug Des Des,* 6(1): 555679. doi: 10.19080/NAPDD.2021.06.555679

Aziz, I.J., Rizal, A. (2024). Recurrent Orthodromic SVT AVRT with Multiple Accessory Pathway in WPW Syndrome: Ablate All or Not?. *Heart Sci J* 2024; 5(2): 86-91

Balta, A., Ceasovschih, A., Șorodoc, V., et al. (2022). Broad Electrocardiogram Syndromes Spectrum: From Common Emergencies to Particular Electrical Heart Disorders. *J Pers Med,* 12(11). doi:10.3390/jpm12111754

Brugada, J., Katritsis, D.G., Arbelo, E., et al. (2020). 2019 ESC Guidelines for the Management of Patients with Supraventricular Tachycardia. *Eur Heart J*. 41(5):655-720. doi:10.1093/eurheartj/ehz467

Drazner, M.H., et al. (2025). 2024 ACC Expert Consensus Decision Pathway on Strategies and Criteria for the Diagnosis and Management of Myocarditis: A Report of the American College of Cardiology Solution Set Oversight Committee. *JACC,* 85:391–431

Fernando, A., Guragai, N., Vasudev, R., et al. (2020). A Rare Case of Myocarditis Mimicking ST-Elevation Myocardial Infarction. *Cureus* 12(11): e11671. doi:10.7759/cureus.11671

Klein, R.M., Vester, E.G., Brehm, M.U., et al. (2000). Inflammation of the myocardium as an arrhythmia trigger. *Z Kardiol*; 89 Suppl 3:24-35. German

Peretto, G., Sala, S., Rizzo, S., et al. (2018). Arrhythmias in Myocarditis: State of the Art. *Heart Rhythm*. doi: https://doi.org/10.1016/j.hrthm.2018.11.024

Vijay, Y., Sanjeev, T., Ratna, G.M., et al. (2022). A Wolff Parkinson-White (WPW) Electrocardiographic Pattern in Asymptomatic Patient –State-of-the-Art-Review. *J Cardiol Cardiovasc Med*, 7(2):045-052. doi:10.29328/journal.jccm.1001132

Wieczorkiewicz, P., Przybylak, K., Supel, K., Kidawa, M., Zielinska, M. (2022). Symptomatic Young Adults with ST-Segment Elevation—Acute Coronary Syndrome or Myocarditis: The Three-Factor Diagnostic Model. *J. Clin. Med*, 11, 916. https:// doi.org/10.3390/jcm11040916