Journal of the Postgraduate Institute of Medicine 2025; 12(2): E281 1-6

https://doi.org/10.4038/jpgim.8567

Case Report

Posterior fascicular ventricular tachycardia complicated by reversible tachycardia induced cardiomyopathy: A diagnostic and therapeutic challenge

N M M Risly[1,2], I K Jayasinghe[2], N Athauda[2], P D Fonseka[1,2], J F Sahana[3], M H A Muneer

[1]Postgraduate Institute of Medicine, University of Colombo,[2]National Hospital, Kandy, [3]Teaching Hospital, Peradeniya

Key words: Salbutamol, Posterior Fascicular Ventricular Tachycardia (LPF-VT), Ischemic Cardiomyopathy, Tachycardia-Induced Cardiomyopathy, Arrhythmia Management, Implantable Cardioverter-Defibrillator (ICD)

Corresponding Author: N M M Risly, E-mail< Rislycool123@gmail.com >

https://orcid.org/0009-0000-4743-5034

Received: 04 Mar 2025, Accepted revised version: 04 Sep 2025, Published: 07 Jan 2026

Competing Interests: Authors have declared that no competing interests exist

Introduction

Left posterior fascicular ventricular tachycardia (LPF-VT) is a rare arrhythmia that can occur in age group, from newborns to adults [1]. It is often misdiagnosed as supraventricular tachycardia (SVT) with right bundle branch block (RBBB) and left anterior hemiblock aberrancy in emergency situations [2,3]. In this patient, posterior fascicular VT presented a diagnostic challenge, initially mimicking supraventricular tachycardia with bundle branch block. In our case, the LPF-VT was triggered or unmasked either due to withdrawal of beta blocker therapy in case of symptomatic bradycardia or due to salbutamol therapy. Our case is unique because LPF-VT is rarely reported, is usually highly verapamil sensitive which was not seen in our case and rarely causes reversible tachycardia induced cardiomyopathy.

Case Presentation

A 69-year-old male with a history of hypertension, heart-failure with preserved ejection fraction, ischemic heart disease with triple vessel disease with ectatic coronary arteries and right coronary artery total occlusion (treated with stenting) presented to the medical ward with multiple syncopal episodes. He had no accompanying chest pain or neurological symptoms and arrived without any previous medical records except for a drug prescription.

https://doi.org/10.4038/jpgim.8567

The patient was on oral anticoagulants (rivaroxaban for ectatic coronaries), dual antiplatelet therapy (DAPT), statins, beta-blockers and ACE inhibitors under regular cardiology follow-up. On initial assessment, his airway and breathing were stable, but his blood pressure was 80/50 mmHg, and his heart rate was 30-35 bpm. An ECG showed sinus bradycardia, 1st-degree AV block and infrequent ventricular ectopics, suggestive of beta-blocker-induced bradycardia. He was treated with IV atropine 0.5 mg stat (three doses) which improved both heart rate and blood pressure [ECG 1].

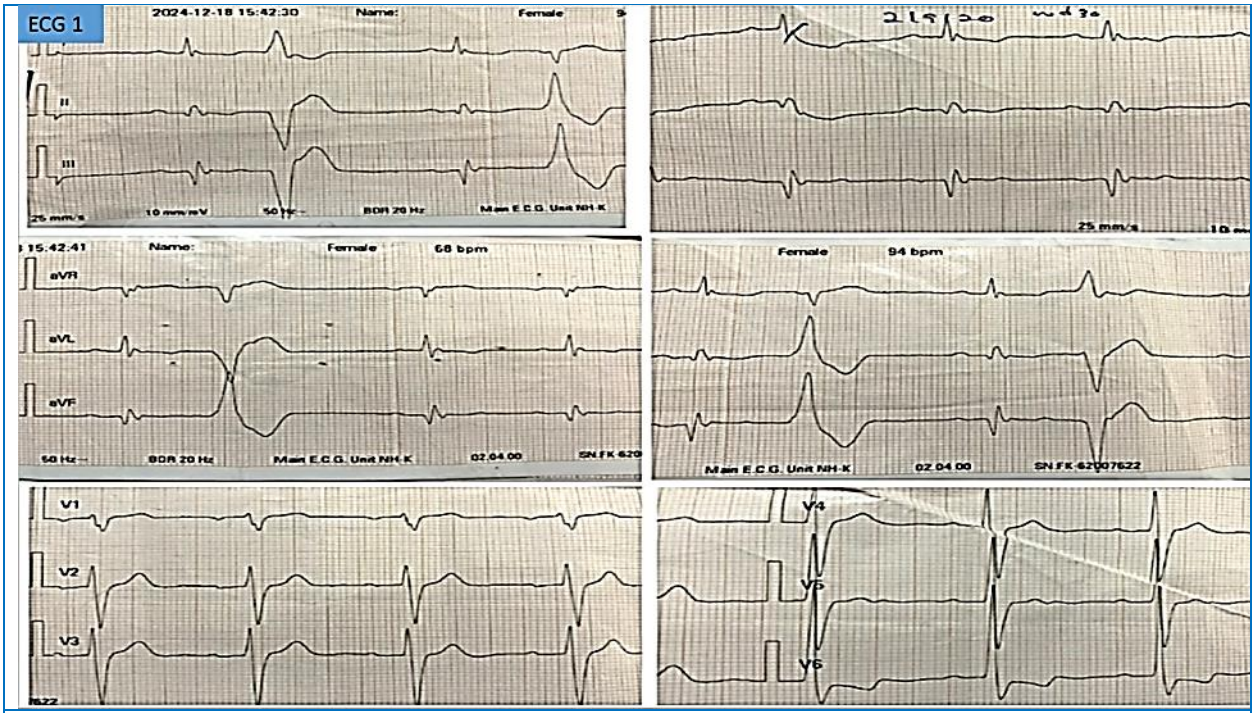

Figure 1: ECG 1

The electrophysiology (EP) team was consulted and a Holter study was arranged with initiation of salbutamol (2mg TDS dosing regime) to address rebound bradycardia with a diagnosis of betablocker associated symptomatic bradycardia. Further evaluation excluded acute coronary syndrome (ACS), hypothyroidism and common causes of bradycardia. He had normal electrolyte levels. An initial echocardiogram revealed moderately impaired LV function with an ejection fraction of 40-45%, global hypokinesia, and preserved LV mass. The Holter study reported frequent onset polymorphic ventricular ectopics with alternating bigeminy and trigeminy patterns, prompting the discontinuation of salbutamol due to its pro-arrhythmic potential. Despite the discontinuation, the patient developed broad complex tachycardia with an RBBB pattern [ECG 2] during continuous cardiac monitoring, initially suspected to be SVT with aberrancy, and was treated with adenosine (6mg → 12mg → 18mg) without effect.

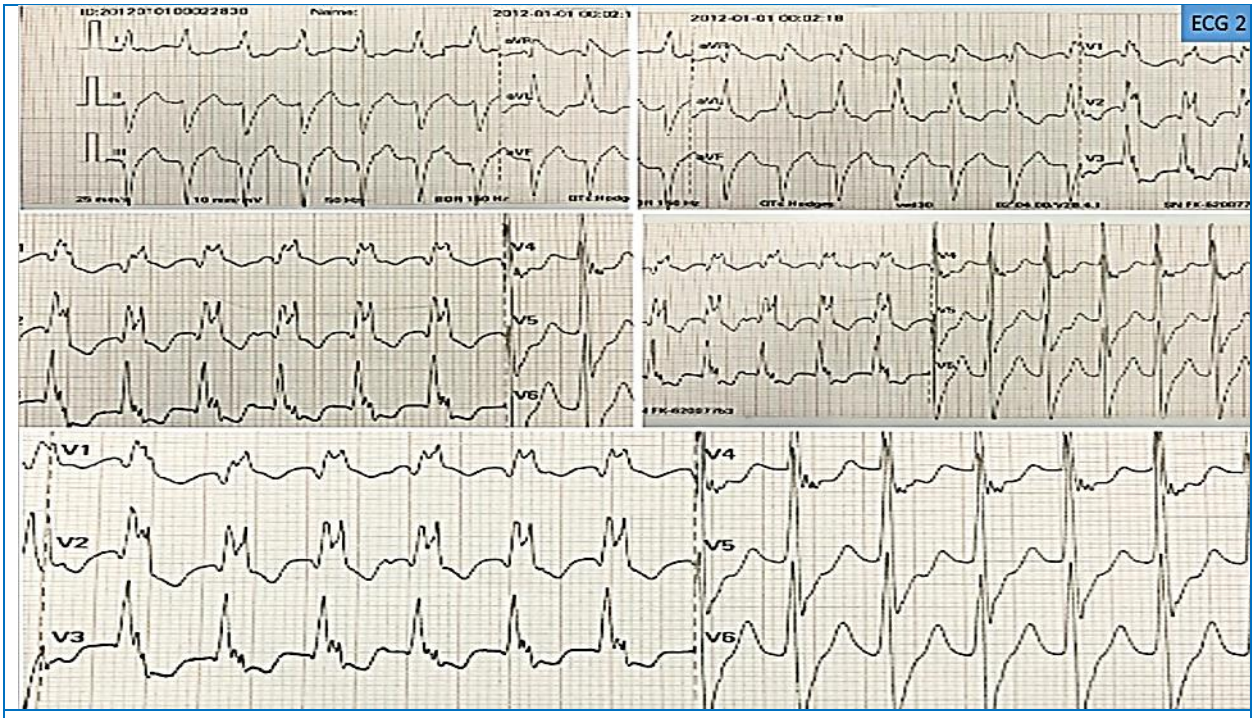

Figure 2: ECG 2

As his condition deteriorated, synchronized DC cardioversion (DCCV, 70J) was performed, successfully restoring sinus rhythm. Following an EP team assessment, the rhythm was identified as Left Ventricular Posterior Fascicular VT, which is typically verapamil sensitive. Despite administration of IV verapamil the rhythm reverted to VT within 30 minutes, requiring escalating DCCV (70J → 120J → 170J) to achieve sinus rhythm. However, recurrent VT episodes necessitated further DCCV (up to 220J) and initiation of IV amiodarone (150mg loading dose followed by another 150mg) alongside inotropic support with IV noradrenaline. The patient was subsequently transferred to the Coronary Care Unit (CCU) for continuous cardiac monitoring. Repeat Holter monitoring showed multiple runs of non-sustained VT and a follow-up 2D echocardiogram demonstrated findings of dilated cardiomyopathy with a reduced ejection fraction of 20-30%, likely due to tachycardia-induced cardiomyopathy. After stabilization, the patient underwent Implantable Cardioverter-Defibrillator (ICD) implantation with pacing [ECG 3] and was eventually discharged on optimized medical therapy including oral anticoagulants, lower dose beta-blockers, ACE inhibitors, and statins. On later follow-up echo in 2 months, patient had gained increased ejection fraction closer to his initial baseline values.

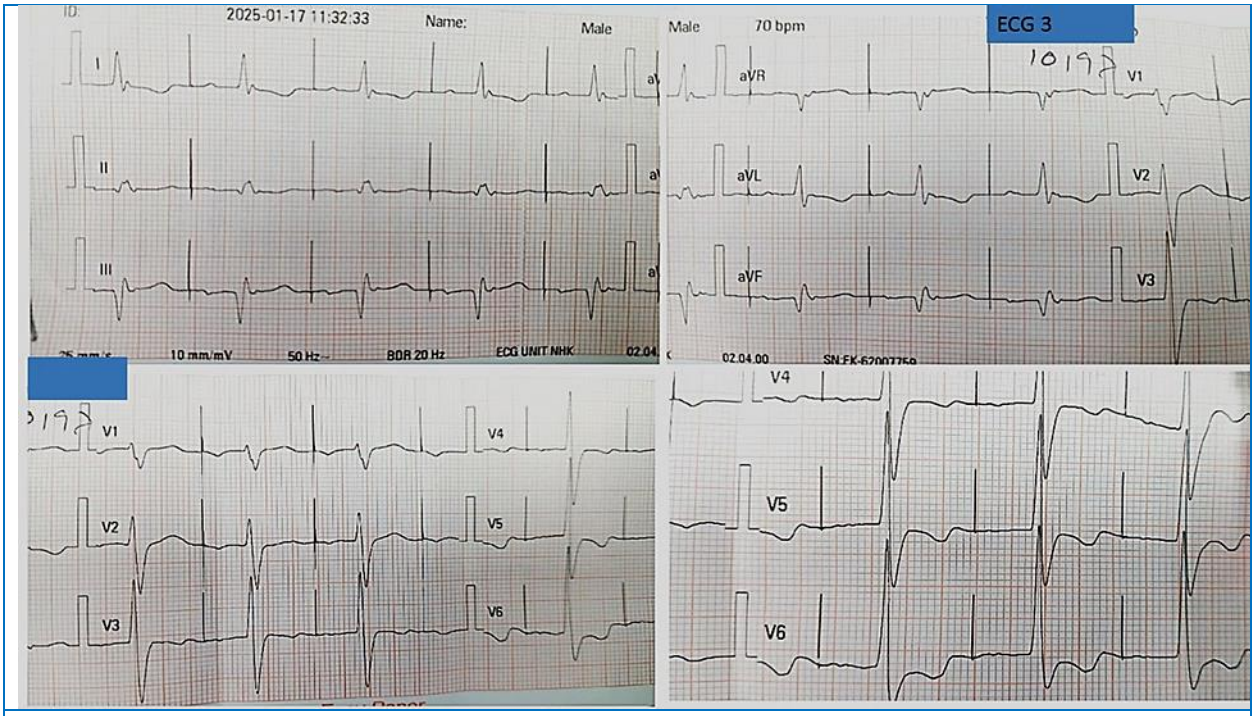

Figure 3: ECG3

Discussion

Fascicular VT is often seen in structurally normal hearts but can coexist with ischemic heart disease as in our case and can complicate the management [3]. These arrhythmias are classically sensitive and terminated with intravenous verapamil which directly targets the re-entry circuit involved in fascicular VT [2]. However, in this case, the arrhythmia was refractory to verapamil, necessitating repeated synchronized direct current cardioversion (DCCV) and initiation of IV amiodarone, which remains a cornerstone in VT management.

Our case is unique because the LPF-VT was thought to be triggered either due to unmasking of pre-existent LPF-VT by beta blocker withdrawal or initiation of therapeutic doses of proarrhythmic salbutamol, which has been reported very rarely in the literature. The reports of VT due to salbutamol are confined mainly to salbutamol overdose or abuse [4,5,6]. Cases of ventricular arrhythmias, including ventricular fibrillation (VF) and tachycardia, have been attributed to electrolyte disturbances, particularly hypokalemia, and increased myocardial excitability [5. In severe cases, fatal dysrhythmia has been observed, emphasising the importance of cautious administration, especially in high-risk cardiac patients [3]. In day-to-day clinical practice, certain elderly individuals are hypersensitive even to therapeutic doses which may have contributed to our case.

When the pathophysiology of LPF-VT is considered, it involves a macro-reentrant circuit that comprises of ventricular myocardium, part of the left posterior fascicle, a slow conduction zone and occasionally a specially conducting fiber [7]. Diagnosis can be confirmed through electrophysiological studies (EPS) and genetic evaluation which may reveal associated channelopathies such as Brugada syndrome [1]. ECG characteristics distinguishing LPF-VT include atypical RBBB-like V1 morphology, positive QRS in aVr, V6 R/S ratio ≤1 and QRS ≤140 ms [5]. Treatment typically involves radiofrequency ablation targeting specific potentials, which has shown success in eliminating the arrhythmia [3]. Electrophysiological studies (EPS) and continuous cardiac monitoring via Holter ECG are vital in exactly mapping the arrhythmic focus and guiding treatment [2].

In our case, due to recurrent runs of VT and continuously higher ventricular rate, the patient developed features of worsening heart failure which was considered as new onset tachycardia induced cardiomyopathy (TIC) backed by clinical and echocardiogram evidence. Studies have emphasized the risk of TIC, where prolonged ventricular arrhythmias lead to a reversible decline in left ventricular function [8]. Early recognition and management of TIC are crucial as prolonged arrhythmic episodes can result in irreversible myocardial damage [8]. TIC is a reversible form of heart failure caused by persistent tachyarrhythmias when the sinus rhythm restored [9]. It occurs in approximately 10% of focal atrial tachycardia cases and is strongly associated with incessant or frequent paroxysmal tachycardia [10]. Though TIC can develop gradually, recurrent tachycardia can lead to a rapid decline in left ventricular function as in our case [11]. After successful treatment, such as radiofrequency ablation, left ventricular function usually improves or normalizes within months [10,11]. However, long-term outcomes may include recurrent heart failure exacerbations with uncontrolled tachyarrhythmia and sudden death, suggesting its persistent nature. Therefore, careful long-term follow-up is necessary for patients with TIC, even after initial recovery [8]. The final decision to implant an implantable cardioverter-defibrillator (ICD) with pacing aligns with guidelines for secondary prevention in patients with recurrent hemodynamically unstable VT and severely reduced ejection fraction [2,11]. This approach significantly reduces the risk of sudden cardiac death and improves long-term outcomes in such high-risk individuals.

Conclusion

This case highlights the importance of cautious decision-making during withdrawal of beta blockers and initiation of beta-agonists in structurally abnormal hearts, early differentiation of complex arrythmias with continuous monitoring and the role of timely EP evaluation and intervention to address tachycardia-induced cardiomyopathy to optimize long-term outcomes.

References

1. Kurt E, Ak R, Bahadirli S. Posterior fascicular ventricular tachycardia: A rare cause of tachycardia. J Coll Physicians Surg Pak. 2022;32(6):804-7. https://doi.org/10.29271/jcpsp.2022.06.804

2. Francis J, Venugopal K, Khadar SA, Sudhayakumar N, Gupta AK. Idiopathic fascicular ventricular tachycardia. Indian Pacing Electrophysiol J. 2004;4(3):98-103. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC1501077/
3. Michowitz Y, Michowitz Y, Rahkovich M, Helman G, Belhassen B. Fascicular ventricular tachycardia: Electrocardiographic features and clinical implications. Circ Arrhythm Electrophysiol. 2017;10(5):e005074. https://doi.org/10.1161/CIRCEP.117.005074
4. Uysal E, Solak S, Carus M, Uzun N, Cevik E. Salbutamol abuse is associated with ventricular fibrillation. Turk J Emerg Med. 2016;15(2):87-9. https://doi.org/10.5505/1304.7361.2015.33230
5. Zheng B, Yadav K. Acute salbutamol toxicity in the emergency department: A case report. World J Emerg Med. 2021;12(1):73-5. https://doi.org/10.5847/wjem.j.1920-8642.2021.01.012
6. Boucher A, Payen C, Garayt C, et al. Salbutamol misuse or abuse with fatal outcome: A case-report. Hum Exp Toxicol. 2011;30(11):1869-71. https://doi.org/10.1177/0960327110388957
7. Liu N, Priori SG. Ventricular tachycardia involving the fascicles: Mechanisms and management. Circ Arrhythm Electrophysiol. 2016;9(4):e004272. doi: 10.1161/CIRCEP.116.004272. Available from: https://www.ahajournals.org/doi/10.1161/CIRCEP.116.004272
8. Nerheim P, Birger-Botkin S, Piracha L, Olshansky B. Tachycardia-induced cardiomyopathy: A review of animal models and clinical studies. Circulation. 2004;110(1):95-9. doi: 10.1161/01.CIR.0000135472.28234.CC. Available from: https://www.ahajournals.org/doi/10.1161/01.CIR.0000135472.28234.CC
9. Henrard V, Piérard LA. Cardiomyopathie rythmique récidivante secondaire à une tachycardie ventriculaire idiopathique [Recurrent rhythmic cardiomyopathy secondary to idiopathic ventricular tachycardia]. Arch Mal Coeur Vaiss. 2001;94(6):617-21. Available from: https://pubmed.ncbi.nlm.nih.gov/11480161/
10. Medi C, Kalman JM, Haqqani H, Vohra JK, Morton JB, Sparks PB, Kistler PM. Tachycardia-mediated cardiomyopathy secondary to focal atrial tachycardia: Long-term outcome after catheter ablation. J Am Coll Cardiol. 2009;53(19):1791-7. https://doi.org/10.1016/j.jacc.2009.02.014
11. Watanabe H, Okamura K, Chinushi M, Furushima H, Tanabe Y, Kodama M, Aizawa Y. Clinical characteristics, treatment, and outcome of tachycardia induced cardiomyopathy. Int Heart J. 2008 Jan;49(1):39-47. doi: 10.1536/ihj.49.39. PMID: 18360063. https://doi.org/10.1536/ihj.49.39