



Case report

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# Stemi and Malignant Ventricular Tachycardia Triggered by Rare Posterior Left Ventricular Artery Thrombosis: A Case Report



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## Abstract

**Background:** Acute posterior myocardial infarction (MI) caused by thrombotic occlusion of the posterior left ventricular (PLV) artery is an exceptionally rare presentation and may be complicated by malignant ventricular arrhythmias.

**Case Presentation:** We report the case of a 52-year-old Asian male who presented with acute dyspnea, angina, palpitations, and diaphoresis. ECG demonstrated ST-segment elevation in posterior leads (V7-V9) and depression in anterior leads, suggestive of acute posterior MI. Echocardiography revealed postero-inferior wall hypokinesis. Coronary angiography identified a thrombotic occlusion in the PLV artery, and successful percutaneous coronary intervention (PCI) improved TIMI flow from grade 2 to 3. Standard pharmacological therapy, including beta-blockers, aspirin, ACE inhibitors, statins, and initial thrombolysis with streptokinase, was administered.

**Clinical Course:** On day four post-MI, the patient developed hemodynamically significant monomorphic ventricular tachycardia with loss of consciousness. Emergency management included multiple DC cardioversions and intravenous amiodarone, followed by oral therapy. Subsequent echocardiography showed mild left ventricular dysfunction and regional wall motion abnormalities without significant valvular pathology.

**Conclusion:** This case highlights the clinical importance of considering PLV artery thrombosis as a potential etiology of posterior MI and the associated risk of malignant ventricular tachycardia. Prompt diagnosis, timely reperfusion, and aggressive arrhythmia management are crucial for improving outcomes. Long-term care requires strict adherence to pharmacotherapy, lifestyle modification, and cardiac rehabilitation.

**Keywords:** Posterior myocardial infarction, PLV artery, thrombotic occlusion, ventricular tachycardia, percutaneous coronary intervention.

## Introduction

The posterior left ventricular (PLV) artery typically branches from the right coronary artery (RCA), but in left-dominant circulation, it can branch from the circumflex artery, and rarely from the left anterior descending (LAD) artery. In general, it is responsible for supplying the posterior-inferior portion of the heart such as the inferior ventricle. Specifically, it rarely branches from the anterior descending left or circumflex arteries [1]. Thrombotic occlusion of the PLV artery leading to ST-segment elevation myocardial infarction (STEMI) is extremely rare, with

no documented cases reported in a recent study by Ahmed IAM et al. [2]. Generally, the main coronary arteries such as the RCA, left anterior descending artery (LAD) or the left circumflex artery (LCx) are frequently involved in culprit lesions leading to STEMI that rarely occlude branches like PLV [3].

This is a rare case of thrombotic occlusion of the PLV artery leading to STEMI and complicated by monomorphic ventricular tachycardia post-angioplasty. Therefore, it is crucial to understand and handle these uncommon coronary artery infarcts

and their complications appropriately. This report underscores the importance of recognizing and managing these uncommon coronary artery occlusions and their complications.

## Case Presentation

### Case history and examination

The patient is a 52-year-old Asian male who presented to the medicine outpatient department with a one day history of dyspnea (NYHA Grade IV), angina, perspiration, and palpitations. He did not have any history of diabetes mellitus, hypertension, alcohol consumption, or prior ischemic heart disease. However, he had a history of an addiction to chewing tobacco. He reported adhering to a nutritionally diverse and balanced diet, suggesting an overall awareness of dietary health.

Physical examination findings at the initial presentation revealed a pulse of 84 beats per minute and a blood pressure of 110/80 mm Hg. He was afebrile. There were no signs of peripheral

edema, pallor, or digital clubbing. He underwent initial testing and was referred to the cardiology department, where he was admitted for further management.

Routine laboratory tests were performed, including complete blood counts, liver and kidney function tests, and serum electrolytes, and all results were within normal limits. Electrocardiogram (ECG) findings on the initial presentation demonstrated ST-segment elevation in the posterior leads V7, V8, and V9, which were not included in Figure 1, and ST-segment depression in leads V2, V3, and V4. The posterior leads V7-V9 were specifically used due to clinical suspicion of acute posterior myocardial infarction, as they are not part of the routine 12-lead ECG. At this point the differential diagnosis of acute posterior myocardial infarction, inferobasal myocardial infarction and left circumflex artery occlusion were made. He underwent an urgent echocardiography, demonstrating hypokinesis of the postero-inferior myocardial wall. The echocardiogram also revealed trivial tricuspid and mitral regurgitation.

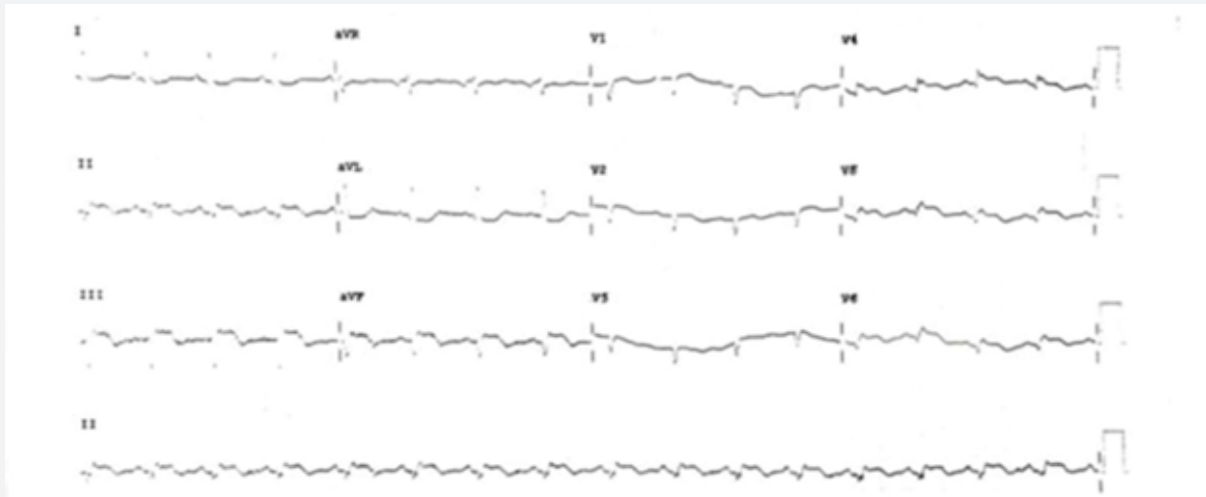


Figure 1: ST-segment elevation, at the time of initial presentation

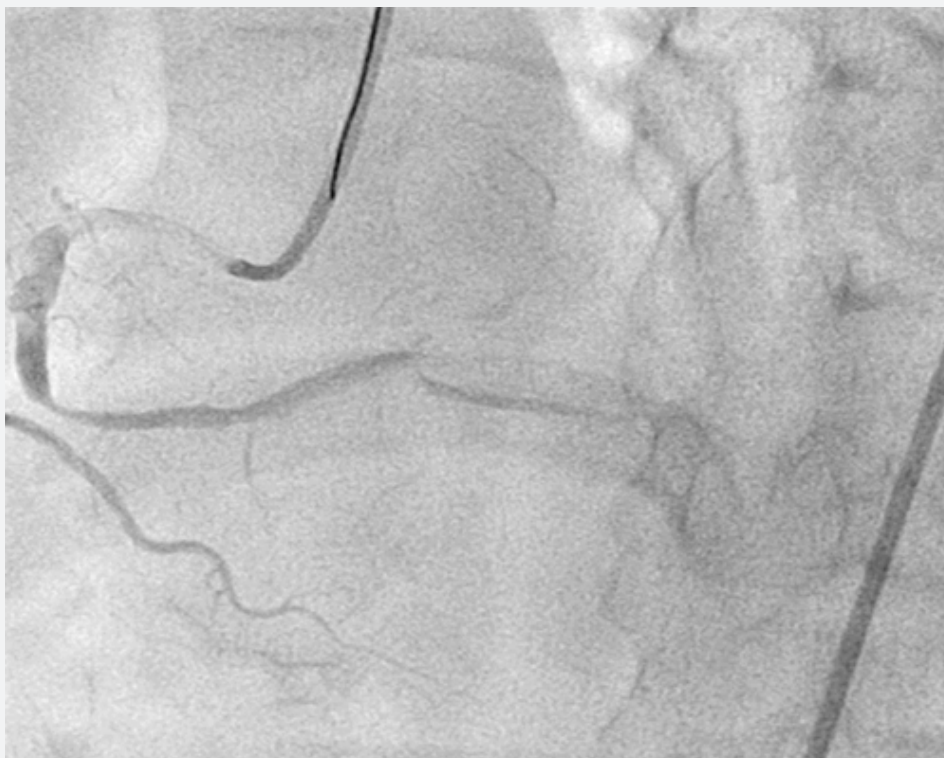
He was promptly scheduled for cardiac catheterization to diagnose the condition further, for which he provided his written informed consent. The procedure, conducted under local anesthesia, revealed a filling defect in the patient's PLV artery (figure 2). Successful angioplasty was performed, improving the pre-procedural Thrombolysis in Myocardial Infarction (TIMI) flow from 2 to a post-procedural flow of 3 (figure 3). Post-dilation was not required. Following the procedure, he was admitted to the coronary intensive care unit (ICU) for monitoring.

Initially, he recovered well, with stable vitals and no immediate complications. However, on the fourth day after the myocardial infarction (MI), he developed hemodynamically significant monomorphic ventricular tachycardia, presenting with profuse sweating and subsequent loss of consciousness.

His  $spO_2$ , measured via pulse oximetry, was 94%, and his blood pressure was 98/72 mm Hg, with a heart rate of 182 beats per minute. A bedside electrocardiogram confirmed the development of monomorphic ventricular tachycardia (figure 4).

### Treatment, outcome, and follow-up

Upon presentation with symptoms suggestive of STEMI, the patient received 1.5 million international units of streptokinase as initial thrombolytic therapy to mitigate ischemic damage while preparations for percutaneous coronary intervention (PCI) were underway. This decision was influenced by a delay in access to immediate PCI facilities. Streptokinase administration was complemented by standard acute coronary syndrome therapy, including beta-blockers, acetylsalicylic acid (ASA), an angiotensin-converting enzyme inhibitor, and statins.

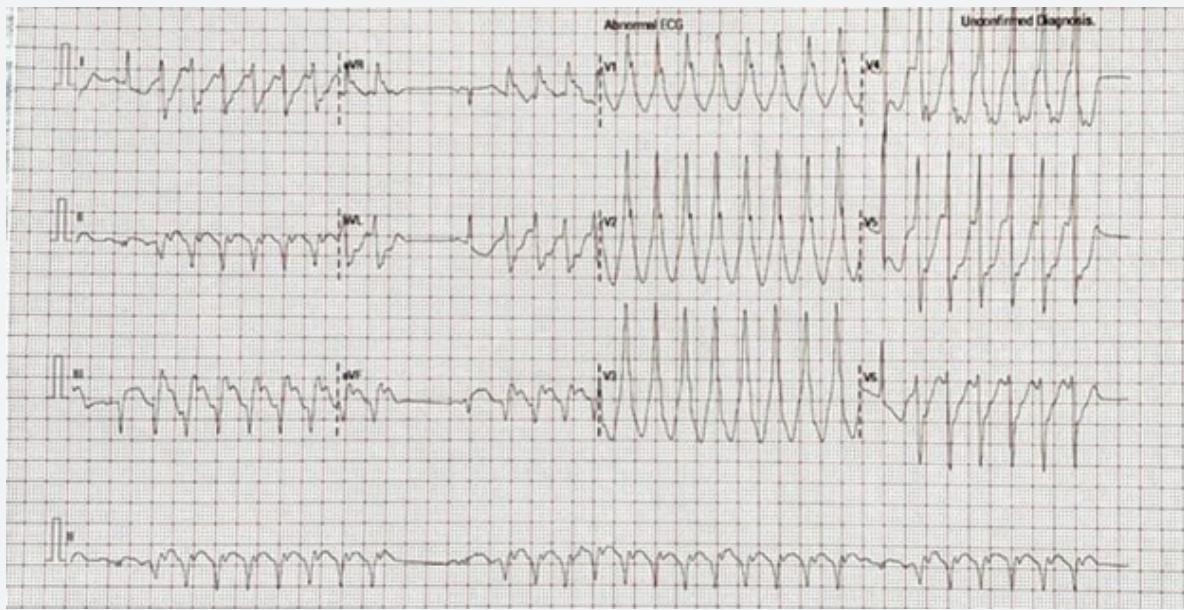


**Figure 2:** Coronary angiogram demonstrating a filling defect in the PLV artery, a branch of the distal right coronary artery



**Figure 3:** After successful angioplasty, resolution of filling defect is seen in the PLV artery





**Figure 4:** On day 4 post-angioplasty, electrocardiogram demonstrates the development of monomorphic ventricular tachycardia

Subsequently, the patient underwent urgent coronary catheterization, which revealed a posterolateral ventricular (PLV) infarct associated with two lesions in the right coronary artery (RCA). A single-vessel PCI was performed, including the successful cannulation of the RCA, lesion crossing with a run-through wire, and pre-dilation with a Trek balloon. Stent deployment utilized Xience Prime Drug-Eluting Stents, achieving proper stent positioning and distal TIMI flow improvement from grade 2 to grade 3. The procedure was conducted under local anesthesia, and post-dilation was not required. He was then admitted to the coronary intensive care unit (ICU) for post-procedural monitoring.

On the fourth day post-MI, the patient developed hemodynamically significant monomorphic ventricular tachycardia, necessitating urgent intervention. Initial management included serial DC cardioversions (50J, 90J, 200J, and 250J), successfully restoring sinus rhythm. An intravenous loading dose of amiodarone was administered. However, the arrhythmia recurred later that day, presenting with profuse sweating, loss of consciousness, and ECG-confirmed monomorphic ventricular tachycardia. A 200J DC cardioversion was performed, restoring sinus rhythm. The patient was transitioned to oral amiodarone for discharge.

A pre-discharge echocardiogram revealed mild left ventricular dysfunction with regional wall motion abnormalities consistent with a posterior infarct. The mitral and tricuspid valves were normal, with no residual regurgitation.

The patient was educated on the importance of medication

adherence, dietary and lifestyle modifications, and was provided a tailored cardiac rehabilitation plan. He was discharged in stable condition with scheduled follow-up visits to monitor recovery and optimize therapy.

## Discussion

The posterior left ventricular (PLV) artery is one of the terminal branches of the coronary arterial system (1). Occlusion in the PLV artery has been rarely reported in the literature. Patient's clinical presentation and investigations were consistent with a diagnosis of ST-segment elevation myocardial infarction (STEMI), confirmed by electrocardiographic findings. Subsequent catheterization revealed a significant PLV occlusion. In a study conducted by John C Wang et al. on 208 patients presenting with STEMI, they found and reported that none of the patients had a thrombotic occlusion of the PLV artery, highlighting the rare nature of a PLV artery infarct [4]. The patient presented with no previous co-morbidities or similar past medical history. However, chewing tobacco is a risk factor for the development of MI [5]. A similar case was reported in Korea, where a 51-year-old male presented with cardiac arrest, and catheterization showed occlusion of the posterolateral branch of RCA [2]. Therefore, PLV occlusion is vital due to its potential for MI, resulting in sudden cardiac arrest with ventricular arrhythmias. The patient exhibited posterior wall hypokinesia on the echocardiogram. In a study conducted on patients with ST elevation in posterior chest leads, it was found that 41% of the patients had lateral wall dyskinesia, and 61% had a dyskinetic or an akinetic posterior wall, indicating a severely abnormal condition [6].

Treatment for STEMI is directed to restore coronary flow either via thrombolytic agents or angioplasty. The Streptokinase was administered to lyse intracoronary thrombotic occlusions, improve ventricular function, and reduce mortality. Early angioplasty was done as a reperfusion therapy for the patient, which helped clear the occlusion [7]. On the fourth day after angioplasty, the patient developed monomorphic ventricular tachycardia.

Ventricular Tachycardia is a broad complex that originates from ventricles. The most common ventricular tachycardia is monomorphic, originating from a single focus point. The patient's ECG showed regular broad complex arrhythmia and uniform QRS complex in each lead [8]. Monomorphic ventricular tachycardia has multiple causes, which include ischemic heart disease, dilated cardiomyopathy, hypertrophic cardiomyopathy, and Chagas disease. It can lower cardiac output, leading to hypotension, collapse, and acute cardiac failure if hemodynamically significant [9], out of which the patient presents mainly with low blood pressure. Moreover, a decreased cardiac output results in decreased myocardial perfusion that can degenerate into ventricular fibrillation [10]. Ventricular tachycardia can occur via various mechanisms, but in certain cardiac tissues, such as the atrioventricular (AV) node, there can be dual conduction pathways—one fast and one slow. Under normal conditions, the impulse travels down the fast pathway, preventing re-entry. However, if there is conduction block or tissue damage (e.g., scar tissue), the impulse may travel slowly through an alternative pathway and then re-enter the previously excited tissue, forming a re-entry circuit. This aberrant electrical activity can lead to arrhythmias [11-12]. Ventricular tachycardia is associated with increased mortality irrespective of the type of infarct and the presence or absence of bundle block. In a study conducted on 16842 patients, it was found that late sustained ventricular tachycardia was retained as a strong, independent predictor of 6-week mortality after myocardial infarction [13].

STEMI results from a total occlusion of a coronary artery. A study suggested that an infarct-related total coronary artery occlusion significantly has higher proportions of other relevant arrhythmic events at follow-up, such as electrical storm, repeated ablation, and hospital admission for ventricular tachycardia. [14] Since total coronary occlusion is associated with a higher risk of ventricular arrhythmias, it is essential to identify the occluded artery.

However, what sets this case apart is the development of monomorphic ventricular tachycardia as a complication during the post-angioplasty period due to thrombotic occlusion of the PLV artery. The rarity of this case underscores the importance of documenting such occurrences to expand the understanding of cardiac pathophysiology and its related complication [15]. The patient's management highlighted the importance of recognizing the clinical presentation and diagnostic approach for posterior left ventricular (PLV) artery occlusion, a condition often overlooked

due to its limited documentation in medical literature. Early intervention with Streptokinase and coronary catheterization allowed for timely diagnosis and revascularization, preventing extensive myocardial damage. The angioplasty procedure restored blood flow, improving the patient's prognosis [13-15]. However, despite successful PCI, the patient developed late-onset monomorphic ventricular tachycardia (VT), likely due to electrical instability in the infarcted myocardial tissue. This complication underscores the risk of arrhythmias even after revascularization, with scar tissue or slow conduction areas predisposing patients to arrhythmias [16-17].

To address this, the use of an implantable cardioverter-defibrillator (ICD) may be beneficial in managing such electrical instability, significantly reducing the risk of sudden cardiac death and secondary arrhythmic events [18]. The patient's post-PCI clinical stability, absence of significant Q waves on ECG, and lack of elevated cardiac biomarkers ruled out type 4 myocardial infarction (MI). Instead, the late VT episodes were attributed to electrical instability rather than a new ischemic event [16,17,19]. VT in this context may result from mechanisms such as reperfusion injury, myocardial stunning, or microvascular dysfunction following thrombotic occlusion and subsequent reperfusion during angioplasty [19]. Although the patient initially received streptokinase for thrombolysis rather than immediate primary PCI, angioplasty was later performed. Studies suggest that early angioplasty can prevent the development of ventricular premature complexes and other arrhythmias by improving myocardial perfusion and reducing ischemic burden [20]. In this case, despite the absence of immediate primary PCI, other contributing factors were considered in evaluating the etiology of VT.

Studies have shown that medications targeting the Renin-Angiotensin System (RAS), such as ACE inhibitors and ARBs, can have beneficial effects in reducing mortality in arrhythmic conditions like atrial fibrillation. Although our case deals with ventricular arrhythmias, these agents may offer potential therapeutic benefits in preventing complications post-infarction, particularly in patients with concurrent arrhythmias [21]. The management of rare coronary artery anomalies, such as a dual left anterior descending artery, has been extensively studied to understand the challenges in diagnosis and intervention [22]. While this case focuses on occlusion of the posterior left ventricular artery, similar diagnostic and interventional principles can be applied to rare coronary anomalies, highlighting the need for advanced imaging and catheterization techniques to accurately assess and treat such conditions. The role of inflammatory mediators, such as neutrophil extracellular traps (NETs), in the progression of cardiovascular diseases, including myocardial infarction. Although not directly implicated in this case, understanding such inflammatory mechanisms could potentially expand our understanding of arrhythmic complications, especially in cases involving rare occlusion sites like the PLV artery [23].

Inflammatory cytokines such as interleukin-6 (IL-6) have been shown to play a role in various ischemic events, including after myocardial infarction and subarachnoid hemorrhage. The modulation of IL-6 pathways may provide therapeutic potential in mitigating reperfusion injury and subsequent arrhythmias, as has been suggested in other vascular events [24]. However, further research in the future shall warrant better management of such cases.

## Conclusion

This case highlights the rare occurrence of monomorphic ventricular tachycardia following thrombotic occlusion of the posterior left ventricular artery (PLV) and subsequent PCI. Despite an initial thrombolysis-first approach with streptokinase, the patient later underwent angioplasty. However, recurrent VT episodes persisted, necessitating aggressive management, including amiodarone therapy and multiple DC cardioversions. This underscores the complexity of managing malignant ventricular arrhythmias post-PCI, emphasizing the need for continuous monitoring, timely interventions, and comprehensive post-procedural care. While angiographic diagnosis and angioplasty played a crucial role in stabilizing the patient, the persistence of arrhythmias despite revascularization highlights the necessity of individualized follow-up strategies to prevent future occurrences.

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