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RHYTHM DISORDERS AND ELECTROPHYSIOLOGY

CLINICAL CASE

Incessant Nonsustained Ventricular Tachycardia Unmasking Pulmonary Embolism

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ABSTRACT

BACKGROUND Ventricular tachycardia (VT) is a malignant ventricular arrhythmia that requires emergent evaluation and treatment. Ischemic heart disease is the most common cause, but its etiology can involve many other possibilities.

CASE SUMMARY A 63-year-old man without structural heart disease presented with VT originating from the right ventricular outflow tract (RVOT), which turned into electrical storm despite antiarrhythmics. A computed tomography scan revealed bilateral acute pulmonary embolism (PE), and he showed favorable progress after its treatment, without further arrhythmias.

DISCUSSION This case highlights the importance of considering PE in the differential diagnosis of VT. Early detection and specific treatment are crucial for improving prognosis of PE, in addition to achieving effective control of the tachycardia by treating its trigger. (JACC Case Rep. 2025;30:103530) © 2025 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 63-year-old man presented to the emergency department with ongoing palpitations and chest pain. He denied loss of consciousness or presyncope. On admission, his vital signs showed normotension (blood pressure 100/60 mm Hg) despite heart rate fluctuations between 150 and 220 beats/min, and his oxygen saturation was 98% without supplementation. On cardiovascular examination, auscultation revealed an irregular rhythm with no evidence of

TAKE-HOME MESSAGES

- Pulmonary embolism should be considered in the differential diagnosis of ventricular tachycardia originating from the right ventricle.
- Ventricular tachycardia triggered by a reversible cause, such as pulmonary embolism, is less likely to require long-term antiarrhythmic therapies after specific treatment of the underlying cause.

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ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

CT = computed tomography

ECG = electrocardiogram

ICD = implantable cardioverter-defibrillator

PE = pulmonary embolism

PVC = premature ventricle complex

RV = right ventricle

RVOT = right ventricular outflow tract

VT = ventricular tachycardia

poor peripheral perfusion or signs of congestive heart failure. The electrocardiogram (ECG) revealed atrial fibrillation (AF) without repolarization abnormalities but showed frequent premature ventricular complexes (PVCs) and recurrent nonsustained monomorphic episodes of wide complex tachycardia.

PAST MEDICAL HISTORY

The patient's medical history included obesity, with a body mass index of 33, hypertension, and stage III colon cancer treated with surgery and adjuvant chemotherapy in complete remission since 2013. No cardiovascular history was reported.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of wide complex tachycardia encompasses ventricular tachycardia (VT) as the most common cause, as well as supraventricular tachycardia conducted with bundle branch block aberrancy or through an accessory pathway.

After a thorough evaluation of the ECGs, it was noted that both the wide complex tachycardia runs and the PVCs shared the same morphology. Additionally, the baseline rhythm was identified as irregular AF, and the wide complex tachycardia was regular. These findings strongly suggested a diagnosis of VT.

Most patients presenting with monomorphic VT have underlying structural heart disease, primarily due to scar-related re-entry. Occasionally, re-entry mechanisms involving conduction disorders or focal sources may also be responsible. Specific VT morphologies, such as the right ventricular outflow tract (RVOT) pattern, in the absence of a family history of cardiomyopathies and with no evidence of structural heart disease, are suggestive of idiopathic VT.

INVESTIGATIONS

In our case, the morphology of the tachycardia showed a left bundle branch block pattern, inferior axis, and V4 transition, consistent with VT originating from the RVOT (Figures 1 and 2). An urgent transthoracic echocardiogram, limited by a poor acoustic window, showed preserved right and left ventricular function and normal chamber dimensions. Blood tests revealed normal electrolyte levels, a nonelevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) of 128 pg/mL (normal range: <300 ng/mL), and an initial troponin level of 11 ng/L (normal range: <53 ng/L).

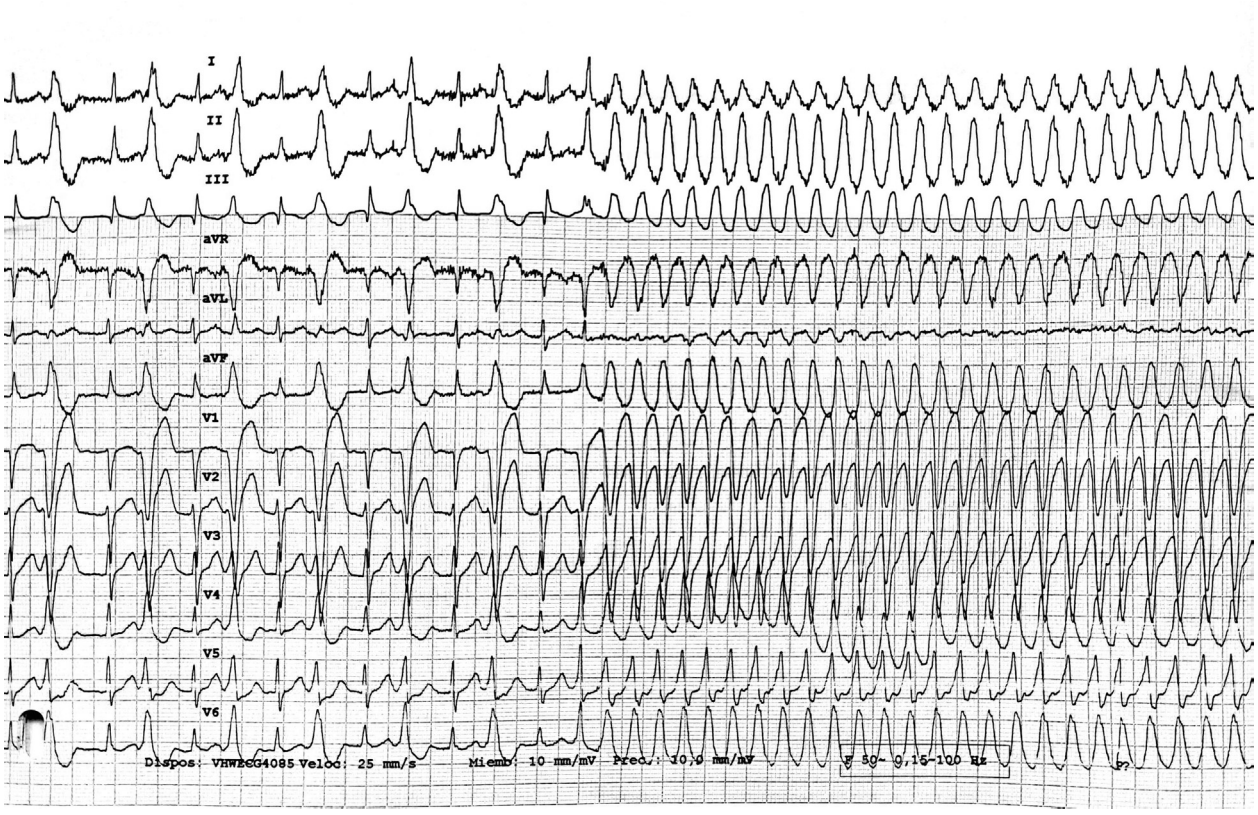
Despite procainamide and esmolol infusion, the frequency of the VT episodes increased, including episodes of sustained and hemodynamically instable monomorphic VT. At this point, rapid sequence intubation was required. After continuous deep sedation with midazolam, propofol, and fentanyl, the patient's arrhythmic storm was controlled, and he did not present any more episodes of VT. Spontaneous conversion to sinus rhythm was also achieved. In addition, anticoagulation with low-molecular-weight heparin at 1 mg/kg every 12 hours was started for AF.

After 48 hours of hemodynamic stability, while the patient was still under sedative agents but with tapering antiarrhythmic agents, a computed tomography (CT) scan excluded coronary artery disease but revealed a bilateral pulmonary embolism (PE) (Figure 3). A limb Doppler ultrasound ruled out deep vein thrombosis. A cardiac magnetic resonance scan showed no structural heart disease or late gadolinium enhancement suggestive of fibrosis (Figure 4). On the

VISUAL SUMMARY **Brief Summary of Clinical Events**

Timeline	Events
Date of Submission	A 63-year-old man with palpitations was admitted to the hospital for ventricular tachycardia. He required deep sedation and intubation due to recurrent episodes despite antiarrhythmic agents. Anticoagulation was initiated for paroxysmal AF.
Day 2	A transthoracic echocardiogram excluded structural heart disease, and a CT angiogram revealed bilateral PE with no evidence of coronary artery disease.
Day 3	No fibrosis was detected on cardiac magnetic resonance. A limb Doppler ultrasound ruled out deep vein thrombosis.
Day 5	After tapering antiarrhythmics with no recurrence of ventricular arrhythmias, the patient was successfully extubated.
Day 7	After heart team discussion, PE was identified as the trigger for VT. He was transferred from the coronary care unit to the cardiology ward under telemetry.
Day 10	The patient was discharged from the hospital with an insertable cardiac monitor and was prescribed indefinite anticoagulation therapy and low dose of β-blockers.

FIGURE 1 **Sustained Ventricular Tachycardia**



Electrocardiogram demonstrating ventricular tachycardia (VT) with probable origin in the right ventricular outflow tract (RVOT): QRS with inferior axis, left bundle branch block morphology, and precordial transition in lead V_4. Both premature ventricle complexes and runs of VT were consistent with RVOT morphology.

fifth day of anticoagulant therapy, successful weaning from sedation and extubation were achieved. Finally, the antiarrhythmic agents were discontinued, and in-hospital telemetry monitoring revealed no further arrhythmias.

MANAGEMENT

After discussion by the heart team and the exclusion of potential cardiac causes, including coronary artery disease and structural heart disease, PE was identified as the trigger of VT from the RVOT. Subsequently, after antiarrhythmic treatment had been withdrawn, an isoprenaline test was performed, but neither ventricular arrythmias nor any PVCs were induced.

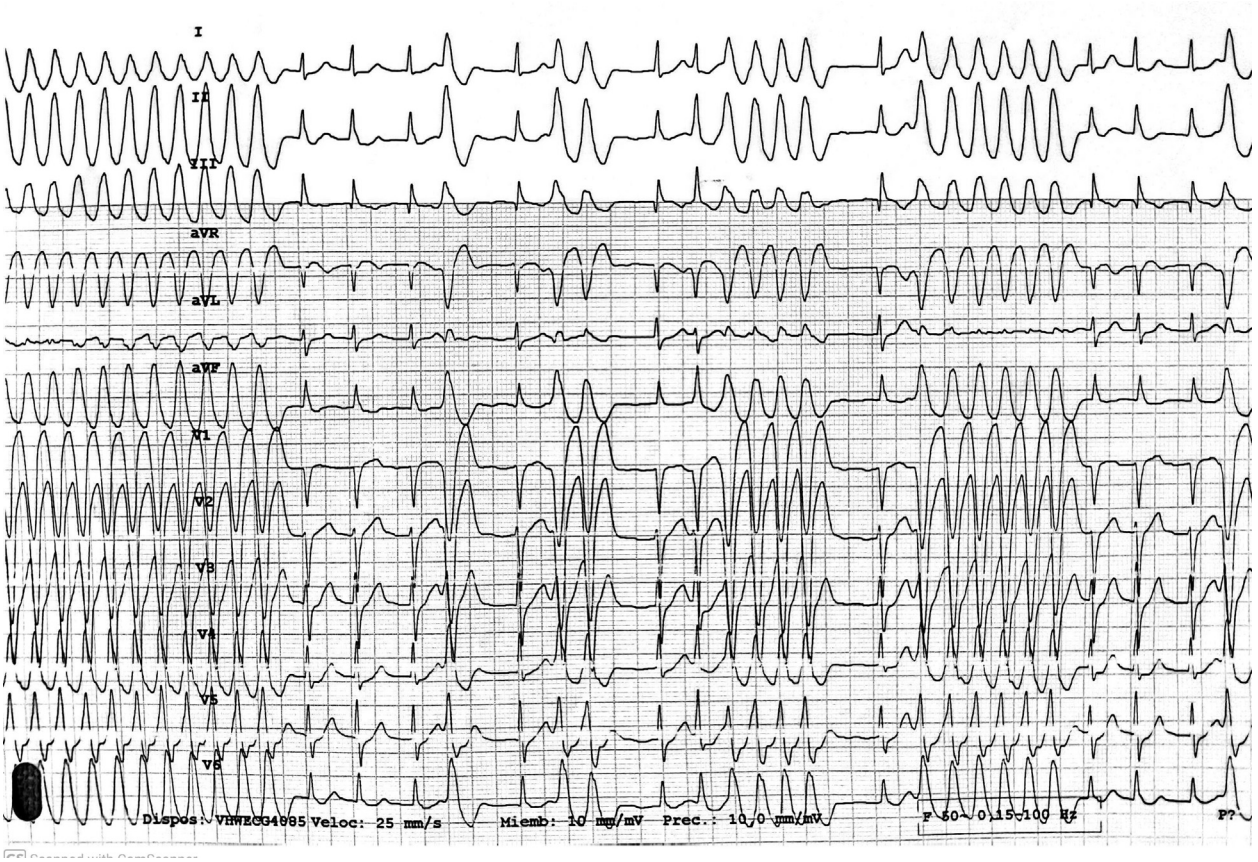
The consistency between the PVCs and the tachycardia morphology was highly suggestive of VT, even without its confirmation from an electrophysiology study. Taking into consideration potential complications during the procedure, such as thrombus migration, and the absence of new arrhythmic episodes after anticoagulation therapy, it was assessed that the risks outweighed the potential benefits of an electrophysiology study. Consequently, it was considered unnecessary to implant a cardioverter-defibrillator (ICD).

In the end, the patient was discharged with an insertable cardiac monitor, indefinite anticoagulation therapy, and low-dose β-blockers.

OUTCOME AND FOLLOW-UP

Three months after hospital discharge, the patient attended a follow-up appointment. He was asymptomatic, NYHA functional class I, and reported no

FIGURE 2 **Atrial Fibrillation and Ventricular Tachycardia**



Electrocardiogram shows alternance between an irregular narrow complex tachycardia indicative of atrial fibrillation and periods of a regular wide complex tachycardia consistent with nonsustained ventricular tachycardia.

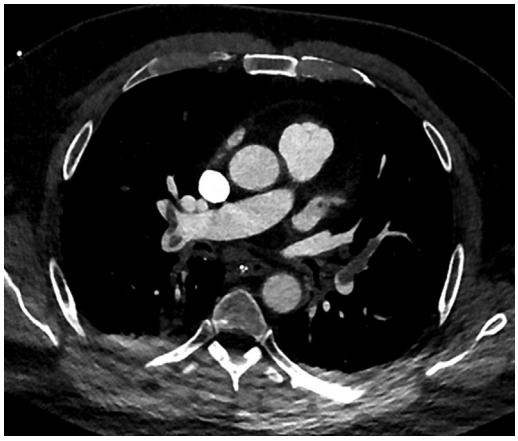
recurrence of palpitations. A follow-up CT angiogram showed complete clot resolution. Thrombophilia screening was negative, and positron emission tomography ruled out recurrence of the previous colon cancer. The insertable cardiac monitor recorded no arrhythmic events. Anticoagulation was switched to direct oral anticoagulants.

DISCUSSION

VT is a rapid heart rhythm originating from the ventricles that requires prompt and accurate diagnosis for effective management. The most common causes are related to cardiac conditions, including ischemic heart disease, cardiomyopathies, congenital heart diseases, and channelopathies.[1] However, noncardiac causes such as toxic substances or metabolic abnormalities can trigger this arrhythmia in structurally normal hearts. The remaining cases, where none of the disorders detailed here have been identified, are classified as idiopathic VTs, accounting for 10% of the presentations.[2]

Outflow tract VTs, typically originating from the RVOT, are the most common form of idiopathic VT.

FIGURE 3 Bilateral Pulmonary Embolism



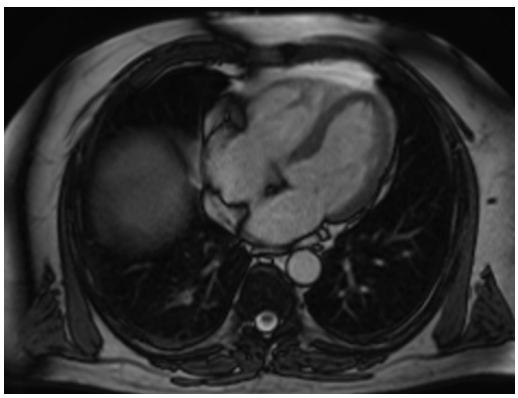
Computed tomography scan revealing perfusion defects in the right main pulmonary artery extending segmentally and in the left lower lobe divisions, besides mild pleural effusion, in accordance with bilateral acute pulmonary embolism.

They are often linked to adrenergic responses in young people due to exercise or emotional stress.[2,3]

This case describes an extremely rare presentation of PE as a RVOT VT. PE is a known cause of cardiac arrest, typically presenting as pulseless electrical activity or asystole. PE guidelines mention atrial arrhythmias and various ECG patterns such as S1Q3T3 or T-wave inversions in leads V_1-V_4 as potential presentations.[4] However, there is limited evidence of VT as a presentation of PE, with only a few cases reported in the literature.[5-8] In our case, the thrombus could have caused the VT due to a mechanical effect when passing through the RVOT or due to right ventricle (RV) overload.

This case emphasizes the importance of considering PE in the differential diagnosis of VT, particularly when the VT originates from the RV, such as RVOT[8] or RV apex morphologies.[6,7] Early diagnosis and specific treatment of PE are crucial for improving the prognosis and reducing in-hospital mortality.[9] Moreover, when VT is triggered by a reversible cause like PE and the acute episode is successfully managed with no evidence of underlying heart disease, recurrence is unlikely. This could potentially eliminate the need for additional interventions such as ablation, chronic antiarrhythmic medications, or ICDs.

FIGURE 4 Cardiac Magnetic Resonance



Cardiac magnetic resonance showing normal chamber dimensions without detection of late gadolinium enhancement.

CONCLUSIONS

VT is a very rare presentation of pulmonary embolism, as reported in a few cases. Considering PE as a potential cause of idiopathic VT is important to avoid diagnostic delays and initiate early appropriate treatment. This approach differs from typical VT management, and these patients are less likely to require long-term antiarrhythmic therapies, ablation, or ICD implantation.

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KEY WORDS **anticoagulation, thrombosis, ventricular tachycardia, vascular disease**