Case report

Ventricular tachycardia as a manifestation of acute myocardial infarction

DOI: 10.5377/alerta.v8i1.19837

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Abstract

Case presentation. We present the case of a 47-year-old male patient with a medical history of chronic arterial hypertension and type 2 diabetes *mellitus*, who presented with a history of palpitations of progressive frequency and duration, accompanied by dyspnea unrelated to exertion, for which, he went to the hospital, where paroxysmal ventricular tachycardia was evident, as a manifestation of an acute myocardial infarction without ST segment elevation.**Treatment.** Hospital management was based fundamentally on cabinet and laboratory findings. Pharmacological treatment was provided with amiodarone and dobutamine due to signs of low cardiac output, besides statins, and acetylsalicylic acid as secondary prevention, as well as percutaneous coronary intervention, with stent placement in the anterior descending artery. **Outcome.** After coronary intervention and with pharmacological management, the patient presented a notable clinical improvement, without palpitations or dyspnea.

Keywords
Tachycardia, Ventricular; Non-ST Elevated Myocardial Infarction, Cardiac Catheterization.

Resumen

Presentación del caso. Se presenta el caso de un hombre de 47 años, con antecedentes médicos de hipertensión arterial crónica y diabetes mellitus tipo 2, quien acudió por un cuadro de palpitaciones progresivas en frecuencia y duración, acompañadas de disnea no relacionada con los esfuerzos. En el hospital se diagnosticó taquicardia ventricular paroxística como manifestación de un infarto agudo de miocardio sin elevación del segmento ST. **Intervención terapéutica.** El manejo hospitalario se basó fundamentalmente en los hallazgos de gabinete y laboratorio. Se inició tratamiento farmacológico con amiodarona y dobutamina debido a la presencia de signos de bajo gasto cardíaco, además de estatinas y ácido acetil salicílico como prevención secundaria. Asimismo, se realizó una intervención coronaria percutánea, con colocación de un stent en la arteria descendente anterior. **Evolución clínica.** Posterior a la intervención coronaria y al manejo farmacológico, el paciente presentó una notable mejoría clínica, sin palpitaciones y sin disnea.

Palabras clave
Taquicardia Ventricular, Infarto del Miocardio sin Elevación del ST, Cateterismo Cardíaco.

Introduction

The European Society of Cardiology (ESC) defines myocardial infarction (MI) as acute myocardial damage, as evidenced by elevated troponins (TnI) in the setting of acute myocardial ischemia.[i] This definition has evolved to include new diagnostic methods and cardiac catheterization findings. According to ESC,[i] MI type 2 is characterized by increased troponin levels associated with an imbalance between myocardial oxygen demand and supply, without coronary thrombosis, and at least one condition with symptoms of

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Taquicardia ventricular como manifestación de un infarto agudo de miocardio. Informe de caso.

Suggested citation:
Linares Rosales CS, Arévalo Mojica IC, Luna Morales JA, Barrera Rodríguez AJ. Ventricular tachycardia as a manifestation of acute myocardial infarction. Alerta. 2025;8(1): 41-46. DOI: 10.5377/alerta.v8i1.19837

Editor:
Nadia Rodríguez.

Received:
November 27, 2023.

Accepted:
January 7. 2025.

Published:
January 22. 2025.

Author contribution:
Study conception, manuscript design, and data collection: CSLR. Literature search: JALM. Data or software management: AJBR and JALM. Data analysis: CSLR and AJBR. Writing, revising, and editing: CSLR and ICAM.

Conflicts of interest:
No conflicts of interest.

ischemia, ischemic changes on the electrocardiogram (EKG), pathological Q waves or loss of viable myocardium.

Cardiac arrhythmias are frequent complications of MI[2], with high mortality and cardiogenic shock in 30 % of cases.[1-2] Ventricular tachycardia (VT) is one of the most important arrhythmias since it can be a trigger or a consequence of MI, occurring in the acute, healing, or cicatricial phase. This presents a diagnostic challenge, especially in emergencies, to determine whether VT is the cause or effect of MI. The article describes a clinical case that highlights the diagnostic and therapeutic complexity of VT associated with MI.

Case presentation

A 47-year-old man with a medical history of chronic arterial hypertension, treated with enalapril (20 mg daily) and type 2 diabetes *mellitus*, treated with glibenclamide (5 mg daily), who consulted for a medical history of one week after beginning with the perception of sporadic palpitations, not associated with exercise or other stimuli. The duration and frequency of the palpitations increased progressively and were later accompanied by dyspnea unrelated to physical exertion, so he decided to consult the emergency unit of a departmental hospital in western El Salvador; his vital signs were: blood pressure of 80/40 mmHg, heart rate: 180 beats per minute, respiratory rate of 22 per minute, oxygen saturation of 97 %, temperature of 37. 1 ° C. At that time, he was transferred to the Maximum Emergency Service. The following paraclinical tests were reported: creatinine:1. 31 mg/dL, urea nitrogen: 30 mg/dL, sodium: 139 mEq/L, potassium: 4. 5 mEq/L, magnesium: 2. 10 mg/dL, phosphorus: 4. 35 mg/dL, calcium: 8. 51 mg/dL, chlorine: 104 mEq/L, TGO: 63 IU/L, TGP: 58 IU/L, total CPK: 1264 IU/L, CPK MB: 28. 0 IU/L, Leukocytes: 9450 cell/uL, neutrophils: 67, 8 %, hemoglobin: 14. 3 g/dL, hematocrit: 40. 6 % and platelets: 233 000 cells/uL.

The tachyarrhythmia was classified as VT 10 minutes after the initial consultation (Figure 1), and it was decided to perform electrical cardioversion using three shocks of 100, 150, and 200 joules, respectively. After the cardioversions, rate control and an improvement in blood pressure were achieved approximately 20 minutes after the initial consultation. However, this improvement lasted only 30 minutes, after which the patient presented with VT again, although without hypotension. Given this situation, labetalol hydrochloride 20 mg was administered intravenously in a single dose, and, despite this management, neither the rate nor the rhythm was controlled, so it was decided to refer the patient to the National Hospital of Santa Ana.

Six hours after the initial evaluation, the vital signs were blood pressure of

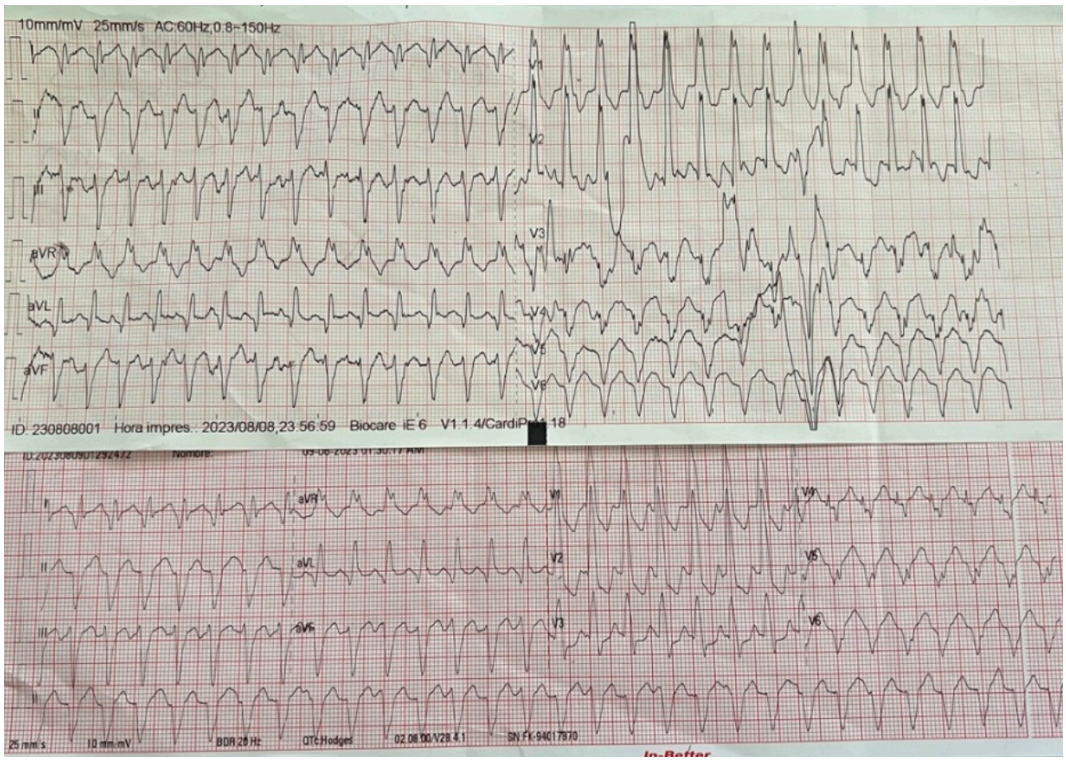

Figure 1. EKG, with evidence of the Marriott sign, absence of SR in precordial leads, AV dissociation, and initial R and Rr' in aVR.

100/60 mmHg, heart rate of 160 beats per minute, respiratory rate of 23 per minute, oximetry of 95 %, temperature of 37. 1 °C, and glycemia of 101 mg/dL. A new electrocardiogram was performed, which showed VT, and approximately 30 minutes after the electrocardiogram was taken, and without any therapeutic intervention, the heart rate and rhythm returned to sinus rhythm.

The patient remained in sinus rhythm and hemodynamically stable for approximately five hours; hospital admission was indicated with a diagnosis of paroxysmal VT. However, eight hours later, he presented a new episode of VT that, suddenly and without medical intervention, returned to sinus rhythm. On this occasion, ST-segment depression and symmetrical T-wave inversion in the diaphragmatic face were observed, as well as Q waves in leads DI, aVL, V5, and V6 (Figure 2).

Treatment

Eight hours after the initial intervention, amiodarone impregnation was started: 300 mg intravenously (EV) in ten minutes, followed by 900 mg at 1 mg/ min for six hours and then 0. 5 mg/min for 18 hours, completing 24 hours. For acute coronary syndrome, enoxaparin 30 mg bolus EV, acetylsalicylic acid 300 mg, clopidogrel 300 mg, atorvastatin 80 mg, and carvedilol 6. 25 mg every 12 hours were administered.

The echocardiogram reported ischemic heart disease in the dilated phase, with an ejection fraction of 20 %, akinesia of the inferior, lateral, and apical wall, mild mitral insufficiency, mild diastolic dysfunction, and normal pulmonary pressures. Holter showed a sinus-based rhythm with T-wave depression in leads DII, DIII, aVF, and V3 to V6, without tachyarrhythmias. The patient was admitted to the Intensive Care Unit with a diagnosis of non-ST-elevation infarct-type acute coronary syndrome and paroxysmal VT. Four days later, percutaneous coronary intervention (PCI) was performed with stenting of the left anterior descending coronary artery and TIMI 3 flow. Four days later, percutaneous coronary intervention (PCI) was performed with stenting of the left anterior descending coronary artery and TIMI 3 flow.

Outcome

The patient remained hospitalized for ten days, receiving double platelet antiplatelet therapy, anticoagulants, statins, a selective beta-blocker, angiotensin II-converting enzyme inhibitors (ACE inhibitors), and oral nitrates. Multiple control electrocardiograms, an echocardiogram, Holter monitoring, and coronary angiography with cardiac catheterization were performed. He was discharged in stable clinical condition, with no new episodes of tachyarrhythmias

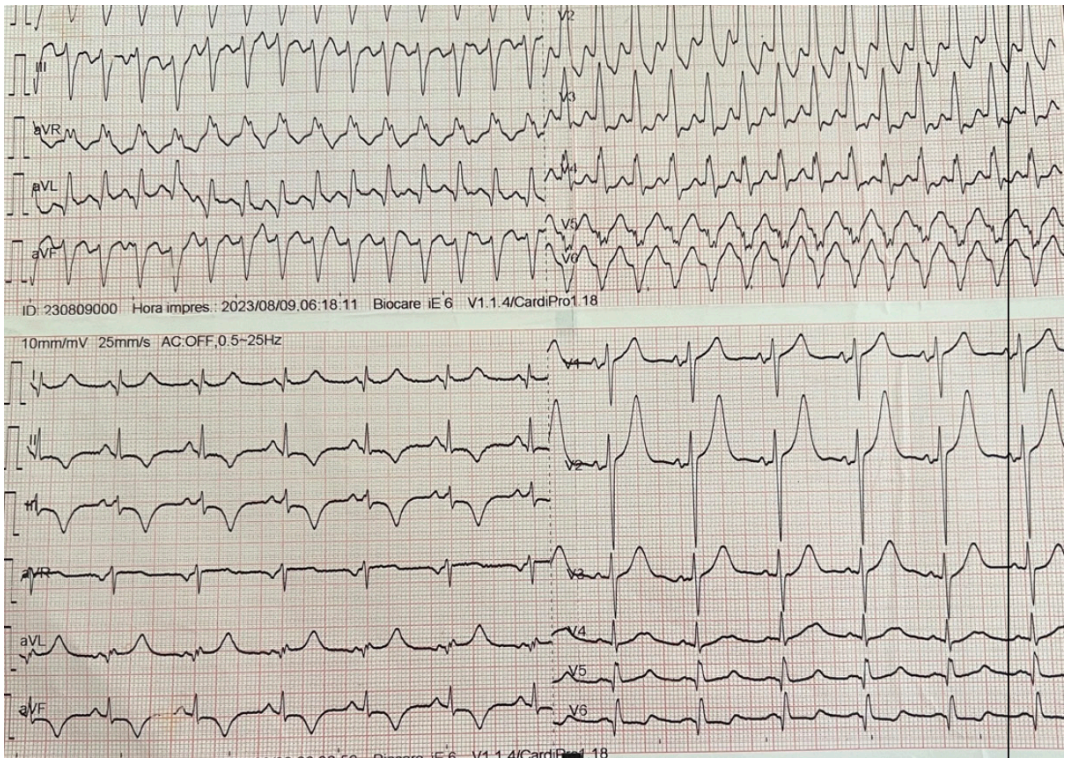

Figure 2. EKG control and ST depression are evidenced in DII, DIII, and AvF. Q waves in DI, aVL V5, and V6.

or other abnormalities, with outpatient follow-up by cardiology..

Clinical diagnosis

The diagnosis of type 2 myocardial infarction was confirmed by the finding of myocardial akinesia in the echocardiographic study. Tachycardia of ventricular origin was determined by the Brugada and Vereckei diagnostic algorithms,[iv] in addition to electrocardiographic evidence of the Marriott sign,[v] and atrioventricular (AV) dissociation allowed the diagnosis of tachyarrhythmia to be certain.

Discussion

The patient presented with a wide QRS complex tachycardia (>0. 12 seconds), so it was essential to differentiate between the causes of tachyarrhythmias with similar electrocardiographic characteristics.[vi] Arrhythmias with QRS greater than 0. 12 seconds can be classified into three main groups: ventricular, supraventricular and supraventricular with aberrant conduction.[vii.]

In the clinical context of this case, given the hemodynamic compromise of the patient, it was essential to correct the rhythm and heart rate using electrical cardioversion, regardless of the exact origin of the arrhythmia. Once the patient was stabilized and after a retrospective analysis of the admission electrocardiographic tracing (Figure 1), the absence of RS complexes in precordial leads and atrioventricular (AV) dissociation was evidenced. This allowed the application of the diagnostic criteria of Brugada, as well as the identification of an initial R wave and Rr' morphology in aVR, compatible with the Vereckei criteria.[viii]

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In addition, the Marriott sign, a rare electrocardiographic finding consisting of an Rr' morphology in lead V1, was observed. This finding confirmed the diagnosis of monomorphic VT and made it possible to differentiate it from supraventricular tachycardia (SVT) with aberrancy, which have different electrocardiographic characteristics[ix]

In this case, the electrocardiographic changes observed, such as T-wave inversion in the diaphragmatic leads and positive T wave in aVL and DI (Figure 2), were interpreted as the result of the cardiac memory phenomenon. This term refers to persistent electrocardiographic alterations that reflect a temporal change in the electrical activation of the heart, secondary to previous episodes of anomalous depolarization, such as tachyarrhythmias.[x].

The causes of VT are divided into two large groups: those that occur in the absence of structural heart diseases, such as water and electrolyte disorders or the adverse effects of drugs that prolong the QT interval, and those that occur in the presence of structural heart diseases, such as ischemic heart disease, cardiomyopathies or valvular heart disease.[xi,xiii].

At this point, the cause of the VT in the clinical case described should be elucidated. Considering that the patient had a history of chronic arterial hypertension, type 2 diabetes *mellitus*, and a history of palpitations for a week before consultation, it is reasonable to assume that the VT could be related to scarring secondary to an old myocardial infarction, evidenced by pathological Q waves on the myocardial lateral aspect.

The history of diabetes *mellitus* suggests a high probability that the patient had suffered a previous myocardial infarction without manifesting a typical clinical picture of chest pain, possibly due to diabetic neuropathy. [xiv] The echocardiographic finding revealed a myocardium exposed to ischemia, with subsequent myocardial necrosis, which constitutes an optimal substrate for the development of VT over a myocardial scar.[iii].

However, when ST segment depression is evidenced in the recent EKG, the presence of a new myocardial infarction is confirmed due to the low output induced by the same arrhythmia. Considering that the patient had a previous structural heart disease (ischemic heart disease), which is related to the cause of VT, the complexity of controlling this condition is evident. This is because VT manifested itself on four occasions in a period of 19 hours, thus meeting the definition of an electrical storm, which is characterized by the occurrence of three or more episodes of VT (most often monomorphic) in a period of 24 hours.[xii,xv.]

In conclusion, the clinical case presented highlights the importance of an accurate diagnosis and immediate management in patients with VT associated with structural

heart disease. Although TnI elevation was observed, this finding was attributed to the ventricular arrhythmia itself. However, the electrocardiographic changes were related to cardiac memory[xvi] and also confirmed the presence of a new myocardial infarction, evidenced by ST-segment depression in the recent EKG (Figure 2). Thus, this clinical case is particularly controversial, as it combines typical elements of acute myocardial infarction with the effects of VT on existing myocardial necrosis, which poses both diagnostic and therapeutic challenges.

The initial differential diagnosis, based on specific electrocardiographic features, confirmed the ventricular nature of the arrhythmia. Furthermore, the presence of predisposing factors, such as diabetes mellitus and ischemic heart disease, underscores the critical importance of preventing and controlling comorbidities in patients at risk of thunderstorm. This highlights the need for a comprehensive approach to improve prognosis and prevent recurrences.

This clinical case presents limited information on the long-term follow-up of the patient, thereby complicating the evaluation of the sustained impact of therapeutic management on the clinical evolution and recurrence of events. In addition, there are no previous diagnostic studies available to evaluate the existence of subclinical myocardial ischemia or previous episodes of tachyarrhythmias, which could have provided a more complete context for the analysis of the case. As this is a single report, the findings are not generalizable to all patients with similar conditions, which represents a limitation inherent to the type of study in this publication.

Ethical aspects

The therapeutic decisions made were in accordance with international medical standards, thereby ensuring compliance with the ethical principles of beneficence, non-maleficence, justice, and autonomy. Informed consent was obtained from the patient for the presentation of the case.

Funding

No external funds were received for this work.

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