

Isolated posterior acute myocardial infarction: the responsible for subtle changes in the electrocardiogram

Infarto agudo de miocardio posterior aislado: el responsable de cambios sutiles en el electrocardiograma

Mario R. García-Arias^{1*}, Cesar Y. Salinas-Ulloa¹, Luis R. Maravilla-Jiménez¹, and Raúl Pinales-Salas²

¹Departamento de Cardiología Clínica, Instituto Nacional de Cardiología "Ignacio Chávez," Ciudad de México; ²Departamento de Medicina Interna, Hospital General de Zacatecas "Luz Gonzalez Cosío," Zacatecas. México

Clinical case

A 47-year-old female from Mexico City, with a background of heavy smoking, systemic arterial hypertension, Type 2 diabetes mellitus, and breast cancer stage IIIC on chemotherapy, attended the emergency department of a cancer medical institute with a sudden onset episode of oppressive chest pain, irradiated to both arms, intensity 5/10, accompanied by nausea and diaphoresis. Her vital signs and physical evaluation were unremarkable. The initial 12-lead electrocardiogram (EKG) showed symmetrical negative T wave inversion, a down-sloping ST segment depression, and a high R wave in leads V1-V2-V3 (Fig. 1). Two high-sensitivity troponin T measurements were performed: initial was 7 pg/mL with a second measurement 2 h after her first medical contact which reported 432 pg/mL (reference 3-14 pg/mL). The diagnosis of non-ST elevation myocardial infarction (NSTEMI) was considered, and 300 mg of aspirin, 75 mg of clopidogrel, and 80 mg of atorvastatin were administered orally. The patient was transferred to the emergency department of our institution, where a comprehensive evaluation of the case was carried out, documenting ST-segment elevation of 0.5 mm in leads V7-V9 (Fig. 2). Coronary angiography was performed and a total thrombotic occlusion at

proximal segment of circumflex artery was found with a thrombus thrombolysis in myocardial infarction (TIMI) Grade 5, deciding to place a drug-eluting stent with a final distal flow TIMI 3 (Fig. 3).

Discussion

The prevalence of isolated posterior acute perioperative myocardial infarction (PMI) is largely unknown due to the lack of records in the literature. Orai et al. mention a global prevalence of approximately 3.3%¹.

The risk factors, clinical presentation, and differential diagnosis do not vary with respect to other acute coronary syndromes (ACS)². Bayés de Luna, proposed through cardiac magnetic resonance imaging (MRI) that the posterior wall is a continuation of the inferior wall in the basal segment pointing in the same direction³; refuting Perloff's statement of a strict posterior infarct characterized by a RS morphology in V1 through V2⁴.

Enhancing the theories of many authors that suggested the anatomopathological correlation that the R on V1 was more easily explained by a lateral infarct; theories that had not echo for decades⁵.

Bayés de Luna proposed to study the ECG-MRI correlation to demonstrate in a sagittal scan of the heart

*Correspondence:

Mario R. García-Arias
E-mail: mario.aris7@gmail.com

Date of reception: 20-12-2022

Date of acceptance: 08-08-2023

DOI: 10.24875/ACM.22000287

Available online: 1-3-2024

Arch Cardiol Mex. 2024;94(1):115-119

www.archivoscardiologia.com

1405-9940 / © 2023 Instituto Nacional de Cardiología Ignacio Chávez. Published by Permanyer. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

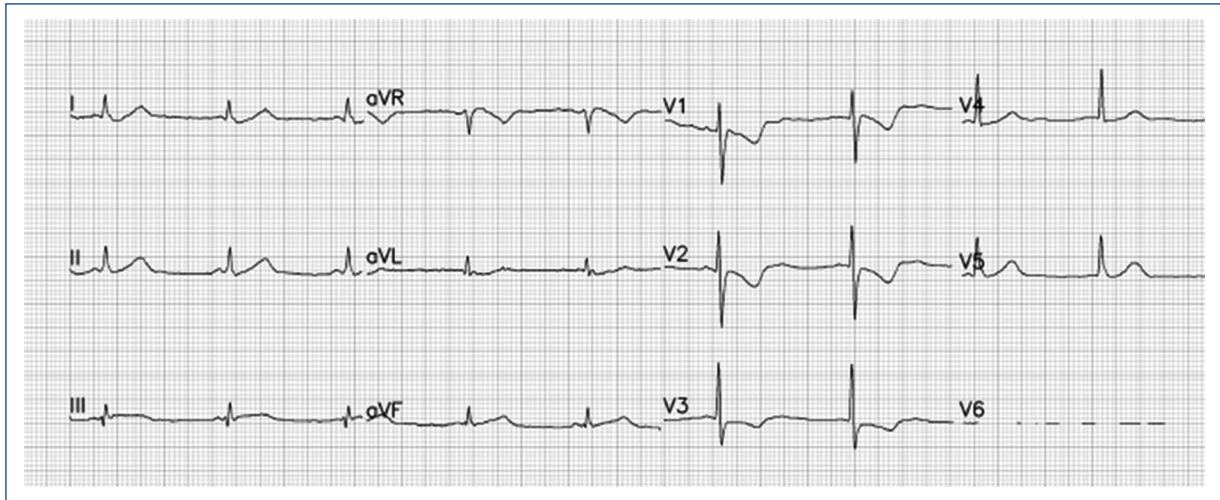


Figure 1. Initial 12-lead electrocardiogram of the patient where a down-sloping ST segment depression and a high R wave from V1-V3 is observed, which is normally interpreted as subendocardial ischemia.

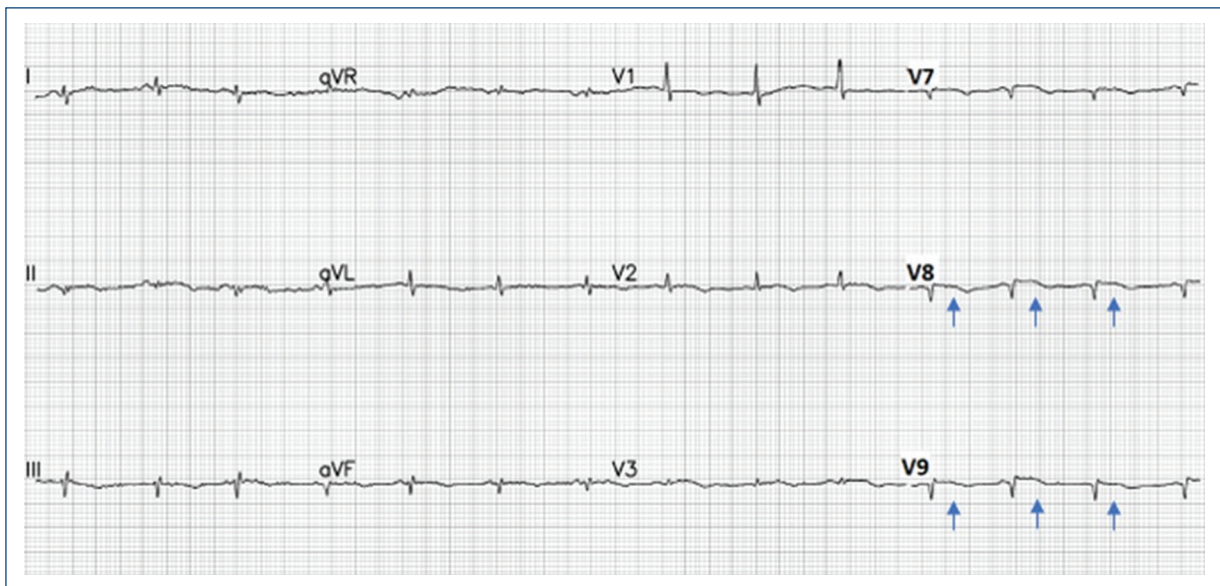


Figure 2. The electrocardiographic thoracic circle was performed in which an elevation of 1 mm can be observed in leads V8 and V9 (blue arrows), representing subepicardial ischemia of the posterior wall of the heart. Performing this electrocardiographic complementation in patients who are evaluated for chest pain allows to offer early reperfusion strategies and improves prognosis.

that in more than two-thirds of the cases, the posterior wall did not exist, because the basal part of the inferior wall was simply a continuation of said wall in the same direction; arguing that contrary to what was expected, there was an RS morphology instead of RS⁶.

The images in MRI, in the horizontal scans, allowed to prove that the heart was not situated in an exclusive posteroanterior sense. Therefore, in the case of an

infarct of the posterior wall, now inferobasal segment of the inferior wall, the necrosis vector headed towards V3, showing no increase in the R wave in V1 because it was being masked in the RS morphology because it normally exists in V3^{6,7}.

The zones that corresponded to the posterior wall, now inferobasal or segment 4, are depolarized after a delay, of 40 ms, and therefore cannot originate a Q

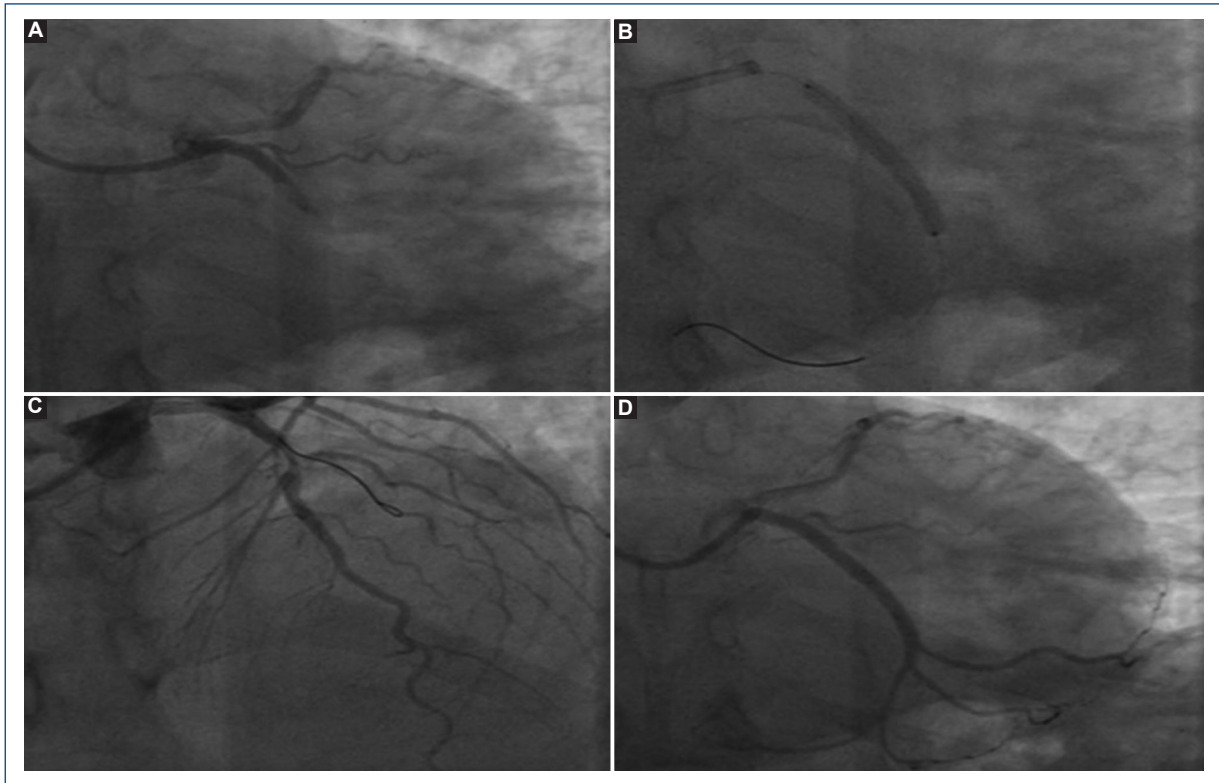


Figure 3. Percutaneous coronary angioplasty. **A:** caudal right anterior oblique view (30/30) total proximal occlusion circumflex artery thrombus thrombolysis in myocardial infarction (TIMI) IV. **B:** caudal right anterior oblique view: preballooning and placement of medicated stent. **C:** cranial right anterior oblique view flow recovery. **D:** caudal right anterior oblique view final flow TIMI III.

wave (or an R wave as a mirror image in V1 through V2) because the QRS complex has already started to register⁷.

On the other hand, since 1950 the Mexican Cardiology School has referred the fact that it is an isolated wall⁸ and this nomenclature continues to be in the current guidelines of the European Society of Cardiology in 2017 and the American College of Cardiology Committee and the Joint Cardiology Clinical Practice Guidelines/American Heart Association in 2021⁹⁻¹².

The 12-lead EKG does not directly orient towards the posterior wall, therefore, none of the leads reveals the classic signs of posterior acute myocardial infarction (AMI), such as ST-segment elevation⁸, which leads to errors or delays in diagnosis². However, it can be suspected when the opposite leads to the posterior wall (V1-V3) manifest “mirror” images characterized by a higher R wave voltage, ST-segment depression, and symmetrical, vertical, acuminated T wave in V1-V2; these changes can be predictive of PMI^{8,9}.

Taken this into account, it is suggested to perform a complete cardiac circle ECG representation which

includes posterior and right leads (V7-V9, V3R, and V4R), confirming the diagnosis with an ST-segment elevation ≥ 0.5 mm V7-V9¹².

All patients presenting with acute chest pain should have a 12-lead EKG recorded and interpreted within the first 10 min after arrival at the emergency room, to early exclude ST-elevation myocardial infarction (STEMI)^{11,12}.

One of the limitations of this initial electrocardiographic approach is that it may be inconclusive, as it does not represent all the anatomical areas of the heart, which, added to an incorrect interpretation of the findings that may preliminarily point to subendocardial ischemia. Even though its incidence among patients with lateral or inferior infarction is 20%¹³, and in isolation, it has an incidence of 3.3%¹.

Pathophysiological cardiac ischemia causes dysfunction of the sodium/potassium ATPase pump, generating a charge imbalance along the myocardial cell membrane and consequently causes the loss of vector and neutralization at the end of depolarization and the beginning of repolarization, with the final alteration of the isoelectric configuration of the J point,

electrocardiographically represented with the deviation of the ST segment and in the reciprocal changes that are observed in the infarct zone. Transmural ischemia leads to ST-segment elevation in affected leads and ST-segment depression in reciprocal leads, while sub-endocardial ischemia can manifest as T-wave inversion or ST-segment depression. In transmural posterior infarction, reciprocal changes will be observed in the leads of the anteroseptal section of the heart, which correspond to the loss of electrical force in a dorsal direction¹³. In addition to the horizontal depression of the ST segment in V1, V2, and V3 on the 12-lead EKG, there are other changes that should alert the clinician to the presence of posterior infarction, including the prominent R wave that constitutes the electrical representation of ventrally deflected electrical forces due to posterior necrosis¹³, prominent T wave, the combination of ST segment depression with a prominent positive T wave, coexistence of inferior or lateral infarction, and R/S wave ratio > 1, these latter criteria only being observed in V2^{9,14}. The presence of any of these findings should be complemented by obtaining the electrocardiographic record of posterior leads V7, V8, V9, where ST-segment elevation ≥ 0.5 mm in two of these leads would be conclusive with the diagnosis of posterior infarction¹³.

The artery responsible for this type of infarction follows the pattern of coronary dominance: if it is right, the occlusion would be in the right coronary artery, and in the left dominance, in the circumflex artery or one of its main branches¹⁴. As in the case of our patient, who presented a total thrombotic occlusion in the proximal segment of circumflex artery, requiring primary angioplasty and placement of a drug-eluting stent with favorable evolution^{13,14}.

An important fact is that up to 20% of patients who have been diagnosed with NSTEMI, which represents the most frequent phenotype of ACS, will have a posterior transmural infarction¹²⁻¹⁴. Situation that highlights the importance of an objective approach in all patients with cardiovascular risk factors who visit emergency room with typical or atypical signs of AMI, in which it is important considering complementation of posterior leads (V7, V8, and V9) on a routine basis. This profitable strategy will make it possible to identify patients who benefit from early coronary revascularization, reducing the probability of catastrophic complications derived from STEMI¹⁴.

The presence of an isolated posterior infarction is a rare clinical scenario, in which the circumflex artery is usually the culprit artery. A prompt identification and

vector understanding represented in the EKG have a high importance in the survival of patients by integrating an adequate diagnosis¹⁴.

Finally, it is important to mention the relevance of modifiable and non-modifiable risk factors for cardiovascular disease, such as in this case diabetes mellitus and high blood pressure which impact in a greater proportion to women¹⁵.

Otherwise, the hormonal response has an important role, since estrogen at the vascular level translates into a positive reaction with nitric oxide in the inhibition of vascular atherosclerotic process¹⁵. Hence, in menopause changes in lipid profile characterized by high-density lipoprotein cholesterol decrease and increase in low-density lipoprotein play a key role to the formation of atherosclerotic plaques¹⁵.

Atypical manifestations present more frequently in women such as dyspnea, palpitations, fatigue, tiredness, jaw, neck, cough, and digestive disorders such as nausea and vomiting; these last one's presented in our clinical case¹⁶.

These symptoms could delay the management and timely treatments, leading to a higher rate of complications. In this way, one of the purposes of share this clinical case is to enhance a promptly identification of acute cardiovascular symptoms in women and recognized and known subtle electrocardiographic changes that could compromise the life of our patients¹⁷.

Despite the different definitions of isolated posterior AMI, presented and based on the explanations that each author or school proposes, it is important to know, describe, and above all understand the reason for each one of them; and regardless of the nomenclature, being able to identify these electrocardiographic changes and understand the vectorial changes that occur in an analytical and deductive way in favor of our patients.

Follow-up

The patient course with adequate clinical evolution and hospital discharge was decided to continue follow-up in the outpatient clinic for ischemic heart disease.

Funding

None.

Conflicts of interest

None.

Ethical disclosures

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

Confidentiality of data. The authors declare that they have followed the protocols of their work center on the publication of patient data.

Right to privacy and informed consent. The authors have obtained the written informed consent of the patients or subjects mentioned in the article. The corresponding author is in possession of this document.

Use of artificial intelligence for generating text. The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript, nor for the creation of images, graphics, tables, or their corresponding captions.

References

1. Oraili S, Maleki M, Tavakolian AA, Eftekhazadeh M, Kamangar F, Mirhaji P. Prevalence and outcome of ST-segment elevation in posterior electrocardiographic leads during acute myocardial infarction. *J Electrocardiol.* 1999;32:275-8.
2. Van Gorselen EO, Verheugt FW, Meursing BT, Oude Ophuis AJ. Posterior myocardial infarction: the dark side of the moon. *Neth Heart J.* 2007;15:16-21.
3. Bayés de Luna A. New heart wall terminology and new electrocardiographic classification of Q-wave myocardial infarction based on correlations with magnetic resonance imaging. *Rev Esp Cardiol.* 2007;60:683-9.
4. Perloff JK. The recognition of strictly posterior myocardial infarction by conventional scalar electrocardiography. *Circulation.* 1964;30:706-18.
5. Dunn WJ, Edwards JE, Pruitt RD. The electrocardiogram in infarction of the lateral wall of the left ventricle. A clinicopathological study. *Circulation.* 1956;14:540-55.
6. Cerqueira MD, Weissman NJ, Disizian V. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation.* 2002;105:539-42.
7. Durrer D, van Dam R, Freud G, Jame M, Meijler F, Arzbaecher R. Total excitation of the isolated human heart. *Circulation.* 1970;41:899-912.
8. Brady WJ, Erling B, Pollack M, Chan TC. Electrocardiographic manifestations: acute posterior wall myocardial infarction. *J Emerg Med.* 2001;20:391-401.
9. Cornejo-Guerra JA, Manzur-Sandoval D, Guadalajara-Boo JF, Briseño-de la Cruz JL. Case report: posterior myocardial infarction in presence of right bundle branch block: an old concept with new findings. *Eur Heart J Case Rep.* 2018;2:ty085.
10. Gulati M, Levy PD, Mukherjee D, Amsterdam E, Bhatt DL, Birtcher KK, et al. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR guideline for the evaluation and diagnosis of chest pain: a report of the American college of cardiology/American heart association joint committee on clinical practice guidelines. *Circulation.* 2021;144:e368-454.
11. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the task force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J.* 2018;39:119-77.
12. Hähnle L, Viljoen C, Hoefelmann J, Gill R, Chin A. Posterior infarction: a STEMI easily missed. *Cardiovasc J Afr.* 2020;31:331-4.
13. Reza M, Arash R, Akbar Shahi H. Value of posterior leads (V7-V9) in diagnosis of posterior wall ST segment elevation myocardial infarction. *Iran Heart J.* 2008;8:24-8.
14. Neill J, Owens C, Harbinson M, Adgey J. Early detection of acute posterior myocardial infarction using body surface mapping and SPECT scanning. *Coron Artery Dis.* 2010;21:420-7.
15. Murphy E. Estrogen signaling and cardiovascular disease. *Circ Res.* 2011;109:687-96.
16. Lichtman JH, Leifheit EC, Safdar B, Bao H, Krumholz HM, Lorenze NP, et al. Sex differences in the presentation and perception of symptoms among young patients with myocardial infarction. *Circulation.* 2018;137:781-90.
17. Céspedes Cuevas VM. Atypical nature of coronary artery disease in women: a proposal for measurement and classification. *Enfermería.* 2015;33:10-8.