Received: 9 March 2024 | Revised: 15 July 2024 | Accepted: 10 August 2024

DOI: 10.1002/ccr3.9367

CASE REPORT

Clinical Case Reports Open Access WILEY

Severe hypokalemia mimicking classical electrocardiographic pattern of left Main coronary artery disease: A case report and a focused review of the literature

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Key Clinical Message

Several electrocardiographic alterations due to hypokalemia have been described, but the electrocardiographic presentation meeting criteria for occlusion of the left main coronary artery is very rare. We describe a case of hypokalemia simulating it.

KEYWORDS

case reports, coronary artery disease, electrocardiography, hypokalemia, syncope

1 | INTRODUCTION

An obstructive lesion of the left main coronary artery (LMCA) has typically been described on the electrocardiogram (ECG) as a generalized ST segment depression (STD) (maximum in V4–V6) associated with inverted T waves in the same leads, and ST segment elevation (STE) in aVR.[1] Likewise, elevated SST in aVR is associated with multivessel coronary artery disease.[1] The presence of these findings, together with an adequate clinical correlation, should alert medical personnel to promptly rule out these conditions and prevent possible adverse outcomes.

However, these electrocardiographic changes are not 100% specific for an obstructive lesion of the LMCA, since elevated STE in AVR could be observed in multivessel coronary disease (three or more vessels), occlusion of the proximal segment of the anterior descending artery, and diffuse subendocardial ischemia.[2]

Besides, the pathophysiology of hypokalemia at the level of the cardiomyocytes includes generation of increased resting membrane potential which in turn increase the duration of the action potential and refractory period. Changes that are potentially arrhythmogenic, such as STD, T wave flattening, and prominent U waves, have been described as a "hallmark" of hypokalemia.[3]

However, an elevation of the ST segment in AVR simulating an LMCA lesion is a rare finding within the electrocardiographic alterations described in hypokalemia, which include: premature atrial and ventricular complexes, sinus bradycardia, prolonged QTc, junctional tachycardia, AV block, ventricular tachyarrhythmias, as well as STD, with a decrease in the amplitude and inversion of the T wave and an increase in the amplitude of

the U wave, usually from V4 to V6.[4] Below, we describe a clinical case of severe hypokalemia simulating on an ECG an obstructive lesion pattern of the LMCA.

2 | CASE HISTORY/ EXAMINATION

An 84-year-old woman with a history of high blood pressure and cholecystitis on targeted antibiotic therapy in the previous 5 days was admitted to the emergency department of a hospital specializing in cardiology and cardiovascular surgery because she had an episode of syncope while sitting in another hospital without referring other symptoms to the questioning.

Upon admission, her vital signs were within the normal range, with abdominal pain on palpation in the right upper quadrant of the abdomen, as well as a positive Murphy's sign and arrhythmic heart sounds discordant with the pulse, with no other findings on the examination.

The initial ECG showed a typical atrial flutter with rapid ventricular response, with a 0.1 mV STE in aVR, as well as a STD and inversion of the T wave in mean lead l, inferior leads, and from V2 to V5 (Figure 1), suggesting a possible obstructive lesion of the LMCA. In addition, acute myocardial injury was documented with high-sensitivity troponin T (136 pg/mL; normal value range: 0–14 pg/mL); however, chest pain was not documented, so it was considered secondary to the ongoing infectious process. Likewise, strikingly, severe hypokalemia was documented (1.9 meq/L), which, when corrected (3.7 meq/L), showed the resolution of the initially mentioned electrocardiographic findings suggestive of a LMCA lesion, also returning to a sinus rhythm in the ECG (Figure 2).

For its part, the echocardiogram demonstrated preserved biventricular function, with a left ventricular ejection fraction (LVEF) of 61%, without contractility disorders, valvular heart disease, or other relevant findings (Figure 3). Therefore, it was considered that the electrocardiographic findings were secondary to hypokalemia, thus completing the broad-spectrum antibiotic therapy for her cholecystitis and presenting a satisfactory clinical and paraclinical evolution during follow-up.

3 | DIFFERENTIAL DIAGNOSIS, INVESTIGATIONS, AND TREATMENT

In relation to the differential diagnosis of electrocardiographic alterations caused by severe hypokalemia, certain pathologies with similar electrocardiographic findings have been described.[5] These include:

1. Myocardial ischemia: T-wave inversion and STD, which are characteristic of hypokalemia, may also be indicative of myocardial ischemia.
2. Hypocalcemia: QT interval prolongation, which may occur in hypokalemia, may also be a sign of hypocalcemia.
3. Hypomagnesemia: Like hypokalemia, hypomagnesemia can cause arrhythmias and QT interval prolongation.

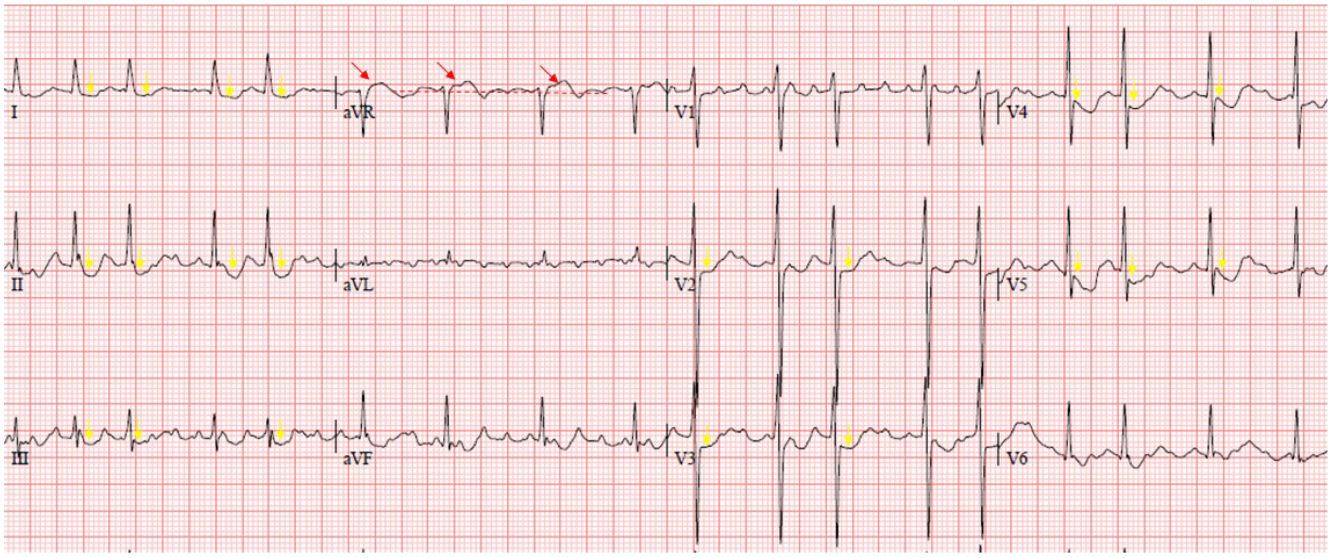


FIGURE 1 Admission electrocardiogram showing a typical atrial flutter with rapid ventricular response, associated with a 0.1 mV elevation of the ST segment in aVR (red arrows) and also depression of the ST segment in Dl, Dll, Dlll, aVF, and from V2 to V5 (yellow arrows).

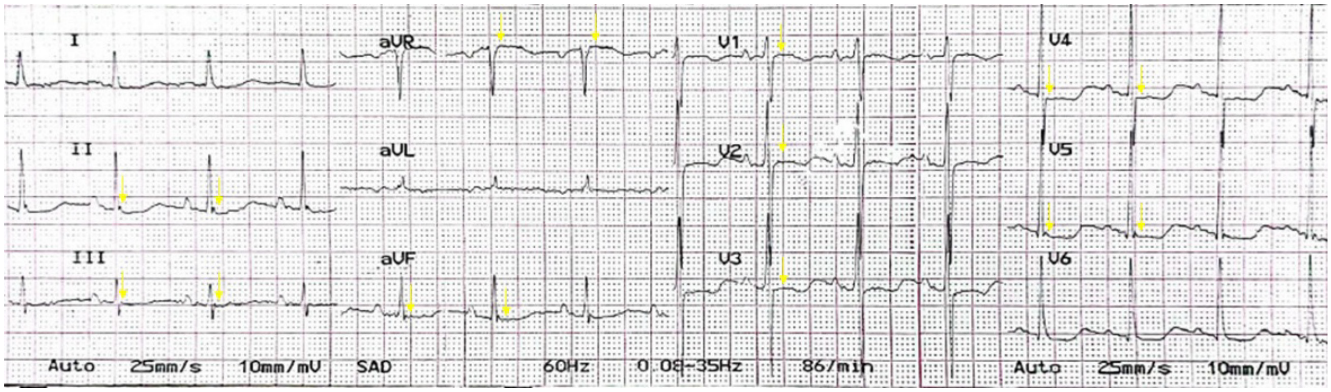
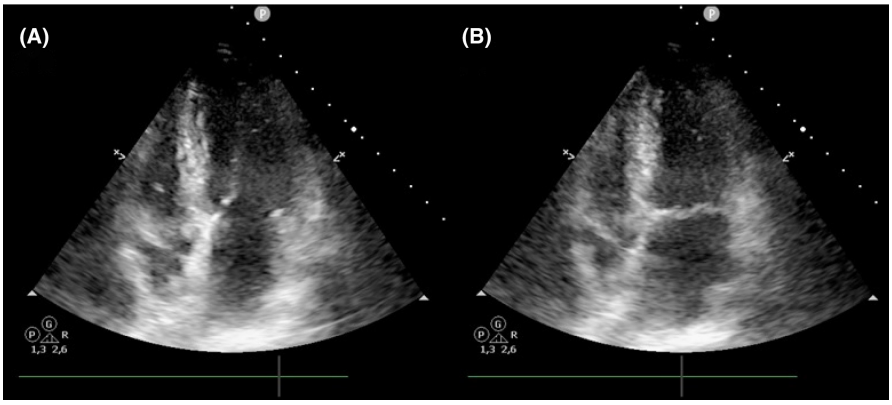


FIGURE 2 Control electrocardiogram showing sinus rhythm along with resolution of ST segment elevation in aVR and in addition to ST segment depression in Dll, Dlll, aVF, and in V2–V5.

FIGURE 3 A four-chamber apical axis echocardiogram in the end-diastolic (A) and end-systolic (B) phases shows adequate contraction of the left ventricle and the absence of global and segmental contractility disorders.



4. Use of certain medications: Some medications, such as antiarrhythmics, tricyclic antidepressants, and antipsychotics, can cause electrocardiographic alterations similar to those of hypokalemia.

After performing an exhaustive interrogation and assessing serum levels of calcium by colorimetric assay (9.5 mg/dL) and serum magnesium (1.9 mg/dL), these conditions were ruled out. Consequently, it is crucial to keep in mind that, in addition to the ECG, the differential diagnosis should always be supported by a thorough clinical evaluation that includes the patient's medical history, physical examination, and laboratory results.

4 | OUTCOME AND FOLLOW UP

During the hospitalization, her electrocardiographic findings resolved after replenishing her potassium levels to normal values, and her clinical and paraclinical condition also improved after completing antibiotic therapy for her cholecystitis. During the 3-month follow-up, she denied having syncope or other symptoms.

5 | DISCUSSION

After performing a review of the available medical literature in the major clinical databases (PubMed, Google Scholar, and Scielo), we found two case reports of hypokalemia resembling a pattern of obstruction of the LMCA.[3,6]

In the first case, described by Burgos et al., a 42-year-old female patient with a medical history of gestational hypertension and acute gastroenteritis, consulted the emergency department due to palpitations. Her initial electrocardiogram presented sinus tachycardia, with elevation of the ST segment of 0.2 mV in aVR and V1 and diffuse depression of the ST segment in more than seven leads. These findings are similar to those found in our case, however differing equally, given that in our case a typical atrial flutter with rapid ventricular response was documented.

Furthermore, as in our case, hypokalemia was also documented, but moderate (2.8 meq/L), and an echocardiogram was within normal parameters, which ruled out left ventricular dysfunction, segmental contractility disorders, or valvular heart disease, among others. However, unlike our case, the case described by Burgos et al. did not present

myocardial injury, which allowed them to further doubt a possible occlusion of the trunk of the left coronary artery.

The second case describes a 39-year-old female with a medical history of previous laxative abuse and major depressive disorder who denied any recent use of laxatives or binging behavior and consulted due to gastroenteritis. Her initial electrocardiogram showed widespread deep ST segment depressions and T wave inversions, with STE in aVR. Also, the corrected QT interval was prolonged at 534 milliseconds. Her serum potassium was 2.3 mmol/L on presentation, and her troponin levels were within the normal range. And as in our case, these electrocardiographic findings resolved with intravenous replacement and correction of potassium (3.1 meq/L).[6]

Finally, it should be noted that despite the differences in age, clinical presentation, the presence of acute myocardial injury, and the additional finding of typical atrial flutter in our case, in all three cases the pattern resembling the occlusion of the trunk of the left coronary artery was resolved by correcting the hypokalemia. Table 1 of the article presents and contrasts the main characteristics of the two cases mentioned and our own case.

Respect the pathophysiology of the electrocardiographic alterations seen in hypokalemia; modified potassium ion balance in the cardiac cells causes repolarization abnormalities. Potassium channels normally open during the repolarization phase of a cardiac action potential, allowing potassium ions to leave the cell. The cell becomes hyperpolarized as a result, going back to its resting state.[3,7] This ionic balance can be disrupted by low potassium levels in the blood plasma in hypokalemia, leading to aberrant repolarization, as evidenced by alterations in the T-wave and ST-segment on the ECG.[3,7] Furthermore, certain sodium and calcium channels may become more active in hypokalemia, which may further modify repolarization and possibly cause arrhythmias.[3,7]

Respect the electrocardiographic findings described in hypokalemia; these include premature atrial and ventricular complexes, sinus bradycardia, prolonged QTc, junctional tachycardia, atrioventricular blocks, ventricular tachyarrhythmias, as well as STD, with a decrease in the amplitude and inversion of the T wave and an increase in the amplitude of the U wave, usually from V4 to V6.[4,8,9] However, STE in aVR with generalized descending of the same in more than seven leads simulating an occlusion of the LMCA has not been specifically described.

Despite the fact that the LMCA occlusion pattern does include STE in aVR with generalized descending of the same in more than seven leads, its specificity increases in the presence of STE in V1 (less than in aVR), in aVL, in mean lead l and aVL, or from V2 to V5 without V1, regarding the pattern of LMCA occlusion that has been described, which corresponds to an STE in aVR with a diffuse STD in more than seven leads.[10] The above, according to the cohort

TABLE 1 Articles describing hypokalemia with an electrocardiographic pattern of occlusion of the left main coronary artery.

Characteristic	Own clinical case	Burgos et al.[3]	CE Chua.[6]
• Age (years)	84	42	39
• Sex	Female	Female	Female
• Medical history	Cholecystitis, diarrhea and arterial hypertension	Gestational hypertension and acute gastroenteritis	Diarrhea, vomiting, laxative abuse and major depressive
• Clinical presentation	Syncope	Palpitations and autonomic symptoms	Atypical chest discomfort
• Initial electrocardiographic findings	Typical Atrial Flutter with rapid ventricular response. STE of 0.1 mV in aVR, STD and inversion of the T wave in mean lead l, lower leads, and from V2 to V5.	Sinus tachycardia STE in aVR and V1 and STD in the rest of the leads.	Widespread deep ST segment depressions and T wave inversions, with STE IN aVR.
• Myocardial injury at admission	Acute myocardial injury	Without myocardial injury	Without myocardial injury
• Resolution of electrocardiographic alterations by resolving hypokalemia	Yes	Yes	The corrected QT interval (QTc) was prolongated at 534 ms.
• Echocardiographic findings	Preserved biventricular function, with a 61% left ventricular ejection fraction (LVEF), and no valvular heart disease, contractility issues, or any pertinent findings.	Preserved biventricular function, without valvular heart disease, contractility issues, or any pertinent findings.	Without data.

Abbreviations: ms, milliseconds; STD, ST segment depression; STE, ST segment elevation.

study carried out by Chun Wei Liu et al., where these findings were found.[10] Likewise, the previously described electrocardiographic findings were also absent, as in the case of Burgos et al. and the case of CE Chen.[6]

Finally, it has been described that a mirror image of leads V5 and V6 is recorded by the lead aVR. Therefore, lead aVR will nearly always have STE if there is STD in the lateral precordial leads,[2] which could explain this finding in the Burgos et al. case and in our case. Consequently, we can conclude that in patients with an ECG suggestive of an obstructive lesion of the LMCA, severe hypokalemia should be ruled out as a differential diagnosis, mainly when there is no elevation of the ST segment in V1, nor aVL, nor concomitant chest pain among others.

AUTHOR CONTRIBUTIONS

Vásquez Lozano Sergio Humberto: Conceptualization; data curation; formal analysis; investigation. **Porras Bueno Cristian Orlando:** Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; supervision; validation; visualization; writing – original draft. **Ruiz Hernández Gabriel Fernando:** Conceptualization; data curation; formal analysis; investigation. **Olarte Jurado Mabel Lucero:** Conceptualization; data curation; formal analysis; investigation; writing – original draft. **Maria Paula Blanco Rueda:** Conceptualization; data curation; formal analysis; investigation; writing – original draft.

ACKNOWLEDGMENTS

To the Autonomous University of Bucaramanga (UNAB) and the Pontifical Xavierian University for promoting publication and research.

FUNDING INFORMATION

This article has been funded by the authors.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author.

ETHICS STATEMENT

The publication of this case has been approved by the institutional ethics committee.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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How to cite this article: Humberto VLS, Orlando PBC, Fernando RHG, Lucero OJM, Rueda MPB. Severe hypokalemia mimicking classical electrocardiographic pattern of left Main coronary artery disease: A case report and a focused review of the literature. *Clin Case Rep*. 2024;12:e9367. doi:10.1002/ccr3.9367