Injury to the left main coronary artery disease or severe hypokalemia? Possible duality in the same electrocardiogram: A case report and a focused review of the literature

Sergio Vásquez Lozano[1], Cristian Orlando Porras Bueno[2], Gabriel Ruiz Hernández[1], Mabel Olarte Jurado[3], and Maria Blanco Rueda[3]

[1]Fundacion Cardiovascular de Colombia
[2]Pontifical Xavierian University
[3]Autonomous University of Bucaramanga

April 26, 2024

Introduction

An obstructive lesion of the left main coronary artery (LMCA) has typically been described on the electrocardiogram (ECG) as a generalised ST segment depression (SST) (maximum in V4-V6) associated with inverted T waves in the same leads and elevation. of the SST 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 SST 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].

Regarding the pathophysiology of hypokalemia, at the level of the cardiomyocytes, an increase in the resting membrane potential is generated, and the duration of the action potential and the refractory period increase. Changes that are potentially arrhythmogenic, such as ST segment depression, T wave flattening, and prominent U waves, which have been described as a "hallmark" of hypokalemia [3].

However, an elevation of the SST 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 SST depression, 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.

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 specialising 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, his 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 rise in SST in

aVR, as well as a decrease in SST and inversion of the T wave in Dl, inferior derivatives, 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; 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, 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 hypocalcemia, thus completing the broad-spectrum antibiotic therapy for his cholecystitis and presenting a satisfactory clinical and paraclinical evolution during follow-up.

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:

- Myocardial ischemia: T-wave inversion and ST-segment depression, which are characteristic of hypokalemia, may also be indicative of myocardial ischemia.
- Hypocalcemia: QT interval prolongation, which may occur in hypokalemia, may also be a sign of hypocalcemia.
- Hypomagnesemia: Like hypokalemia, hypomagnesemia can cause arrhythmias and QT interval prolongation.
- 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 interrogatory and a complete laboratory test, these conditions were ruled out in our case. 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.

Outcome and follow up

During the hospitalisation, 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, he denied having syncope or other symptoms.

Discussion

After performing a review of the available medical literature in the major clinical databases (PubMed, Google Scholar, and Scielo), we found just one case report of hypokalemia simulating a pattern of obstruction of the left main coronary artery [3].

In this 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 7 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.

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 both cases the pattern of 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.

Regarding the pathophysiology of the electrocardiographic alterations seen in hypokalemia, modified potassium ion balance in the cardiac cells causes repolarization abnormalities in hypokalemia. 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[3,6]. 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 [3,6]. Furthermore, certain sodium and calcium channels may become more active in hypokalemia, which may further modify repolarization and possibly cause arrhythmias[3,6].

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 segment ST depression, 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[4,7,8]. However, ST segment elevation in aVR with generalized descending of the same in more than 7 leads simulating an occlusion of the left main coronary artery has not been specifically described.

However, the LMCA occlusion pattern does include it, although its specificity increases, in the presence of elevated SST in V1 (less than in aVR), in aVL, in Dl and aVL, or from V2 to V6, regarding the pattern of LMCA occlusion that has been described, which corresponds to an elevation of the AVR segment with a decrease in the diffuse SST in more than 7 leads [9]. The above, according to the cohort study carried out by Chun Wei Liu et al., where these findings were found [9]. Likewise, the previously described electrocardiographic findings were also absent, as in the case of Burgos et al.

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 ST-segment elevation if there is ST-segment depression 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 contribution statement

Porras Bueno Cristian Orlando: conceptualization, data curation, formal analysis, funding acquisition, investigation, methodology, project administration, resources, supervision, validation, visualisation, writing (original draft), writing review, and editing.

Vásquez Lozano Sergio Humberto: conceptualization, data curation, formal analysis, investigation, writing (original draft), review, and editing.

Ruiz Hernández Gabriel Fernando: conceptualization, data curation, formal analysis, investigation, writing (original draft), review, and editing.

Olarte Jurado Mabel Lucero: conceptualization, data curation, formal analysis, investigation, writing (original draft), review, and editing.

Maria Paula Blanco Rueda: conceptualization, data curation, formal analysis, investigation, writing (original draft), review, and editing.

Acknowledgments

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

References

1. *Prieto-Solís JA, Benito N, Martín-Durán R. Diagnóstico electrocardiográfico de la obstrucción del tronco coronario izquierdo mediante el análisis vectorial del segmento ST y el complejo QRS. Rev Espanola Cardiol. 2008;61(2):137-145. doi:10.1157/13116201*

2. *Nikus KC, Eskola MJ. Electrocardiogram patterns in acute left main coronary artery occlusion. J Electrocardiol. 2008;41(6):626-629. doi:10.1016/j.jelectrocard.2008.06.020*

3. *Burgos LM, Domínguez M, Battioni L, Costabel JP, Trivi M. Cambios electrocardiográficos asociados a hipopotasemia: Alteraciones del segmento ST imitando isquemia miocárdica de múltiples territorios. Rev Chil Cardiol. 2017;36(3):244-248.doi:10.4067/s0718-85602017000300244*

4. *Thu Kyaw M, Maung ZM. Hypokalemia-Induced Arrhythmia: A Case Series and Literature Review. Cureus. 2022. doi:10.7759/cureus.22940*

5. *Sethuraman, C., Mohd, S. F., Govindaraju, S., Tiau, W. J., Farouk, N. D. M., & Hassan, H. H. C. (2020). Severe Hypokalemia ECG Changes Mimicking Those of Acute Coronary Syndrome (ACS) in Patient with Underlying Ischaemic Heart Disease: A Case Review. Open Journal of Emergency Medicine, 08(02), 53–58. https://doi.org/10.4236/ojem.2020.82006*

6. *Duque, M., Aristizábal-Colorado, D., Weir, D., Gaviria, M. C., & Uribe, W. (2020). La repolarización auricular y sus implicaciones clínicas. Revista Colombiana de Cardiología, 27(1), 36–40. https://doi.org/10.1016/j.rccar.2019.05.006*

7. Teymouri N, Mesbah S, Navabian SMH, et al. ECG frequency changes in potassium disorders: a narrative review. Am J Cardiovasc Dis. 2022;12(3):112-124. Published 2022 Jun 15.

8. Khan U, Afzal U, Kask L, et al. "Ion-Flux": The natural course of ECG changes in severe hypokalemia. Curr Probl Cardiol. 2024;49(1 Pt C):102158. doi:10.1016/j.cpcardiol.2023.102158

9. *Liu C, Yang F, Hu Y et al. Combining electrocardiographic criteria for predicting acute total left main coronary artery occlusion. Front Cardiovasc Med. 2022;9. doi:10.3389/fcvm.2022.936687*

Hosted file

`Table 1-word.docx` available at https://authorea.com/users/774758/articles/870367-injury-to-the-left-main-coronary-artery-disease-or-severe-hypokalemia-possible-duality-in-the-same-electrocardiogram-a-case-report-and-a-focused-review-of-the-literature