Alzate et al. Int J Clin Cardiol 2015, 2:6
ISSN: 2378-2951

International Journal of Clinical Cardiology

Case Report: Open Access

Electrocardiographic Changes in Primary Hyperparathyroidism

Alexander Alzate[1], Marcela Muñoz Urbano[2]* and Kenny Buitrago-Toro[2]

[1]Faculty of Health Sciences, Internal Medicine department, Universidad Tecnológica de Pereira, Pereira, Risaralda, Colombia

[2]Faculty of Health Sciences, Medicine program, Universidad Tecnológica de Pereira, Pereira, Risaralda, Colombia

***Corresponding author:** *Marcela Muñoz Urbano, Faculty of Health Sciences, Medicine program, Universidad Tecnológica de Pereira, 660003 27th avenue #10-02 Alamos neighborhood, Pereira, Risaralda, Colombia, E-mail: marcemu_02@hotmail.com*

Abstract

Electrolytic variability modifies normal structures of segments and intervals on the electrocardiogram (ECG). We present a case that was referred to the Internal Medicine department by the Neurosurgery division for several pathological fractures.The electrocardiogram led us to a diagnostic approach and finally we show how electrocardiographic tracing changed after a treatment implementation. Finally, in this study we highlight the importance of an electrocardiogram on a patient´s approach.

Keywords

Hypercalcemia, Electrocardiogram, Hyperparathyroidism

A 43-year old African-Colombian man with personal clinical history of arterial hypertension and urolithiasis was referred by the Neuro surgery and Spine Surgery service to the Internal Medicine and Hematology Department with a 6-year medical history of joint pain in knees, elbows, hips, cervical and thoracolumbar spine. There was no history of synovitis. The patient showed weakness at the lower limbs with evidence of chest, thoracolumbar spine and leg deformities that caused gait limitations and made a wheelchair for mobilization necessary. Three years ago, he had a right femoral neck fracture due to a fall from his own height which required surgical fixation. He has been treated by Neuro-surgery and Spine Surgery because of multiple vertebral fractures and a diffuse alteration in the intensity of bone marrow. The patient was never subjected to studies of his symptomatology, partly because he lived far from any major city where he could undertake related treatment. In figure 1, an admission ECG is shown.

It shows sinus rhythm at 110 beats per minute, normal axis, left ventricular hypertrophy, normal PR and QRS length, QT 230 ms, Short QTc 320 ms, Q-waves in V3 and an ST elevation in V2-V4.

Numerous studies were performed. A bone scintigraphy revealed multiple abnormal accumulations of radiotracer affecting the thoracic and lumbar vertebral bodies and diffuse widespread uptake. Initial ionized calcium level was 2.02 mmol/L (*reference value: 1.1-1.4 mmol/liter*), serum calcium in 13.2 mg/dL (*reference value: 9.0-10.5 mg/dl),* serum creatinine1.1 mg/dL (*reference value: <1.5 mg/dl),* urea nitrogen 17 mg/dl (*reference value:10-20 mg/dl),* parathyroid hormone (PTH) in 1900 pg/mL (*reference value: 10-60 pg/ml*) and phosphorus levels 2,7 mg/dL (*reference value: 3-4.5 mg/dl*). The patient underwent thyroid ultrasound that showed a hypoechoic heterogeneous lesion and well demarcated measuring 25 × 23 × 18 mm. Later, parathyroid scintigraphy was performed, which revealed an abnormal accumulation of radiotracer in the left thyroid lobe. The patient received a clinical diagnosis of primary hyperparathyroidism (PHPT) secondary to hyperfunctioning parathyroid adenoma. Therapy with zoledronic acid was started and he was referred to the Head and Neck Surgery and Oncology department, just for surgical removal of a solitary parathyroid tumor or subtotal resection of all pathologic parathyroid tissue. The report of pathology in which the diagnosis would be confirmed has not yet been provided. The last ionized calcium measurement was 1.8 mmol/L. Control ECG is shown in figure 2.

This control ECG shows sinus rhythm at 80 beats per minute, normal axis, left ventricular hypertrophy, normal PR and QRS length, QT 320 measured at DII, QTc 350 ms, and an ST elevation in V2-V5.

Discussion

Primary hyperparathyroidism is the most common etiology for the hypercalcemia diagnosis that was made in this case. To determine the presence of primary hyperparathyroidism the clinician must prove an elevated or inappropriately normal serum level of intact PTH associated hypercalcemia [1]. Many patients with primary hyperparathyroidism are asymptomatic. Our patient presented with a variety of chronic symptoms and signs, including renal, bone, neurological and cardiovascular manifestations [1-4]. The disease is commonly manifested with hypertension, low bone mineral density and consequently an increased rate of fractures; all of which were shown in this patient [2].

It is well known that potential action in cardiac cells is generated by the movement of electrolytes across the cardiac cell membrane and abnormal ion's levels may lead to altered electrical activity. Hypercalcemia may induce electrocardiogram abnormalities such as QT interval shortening, sometimes associated with prolongation of PR segment and QRS interval, increased duration of the T-wave, decreased T-wave amplitude and prominent U-waves [3,5].

ClinMed International Library

Citation: Alzate A, Urbano MM, Buitrago-Toro K (2015) Electrocardiographic Changes in Primary Hyperparathyroidism. Int J Clin Cardiol 2:068

Received: October 14, 2015: **Accepted:** December 12, 2015: **Published:** December 15, 2015

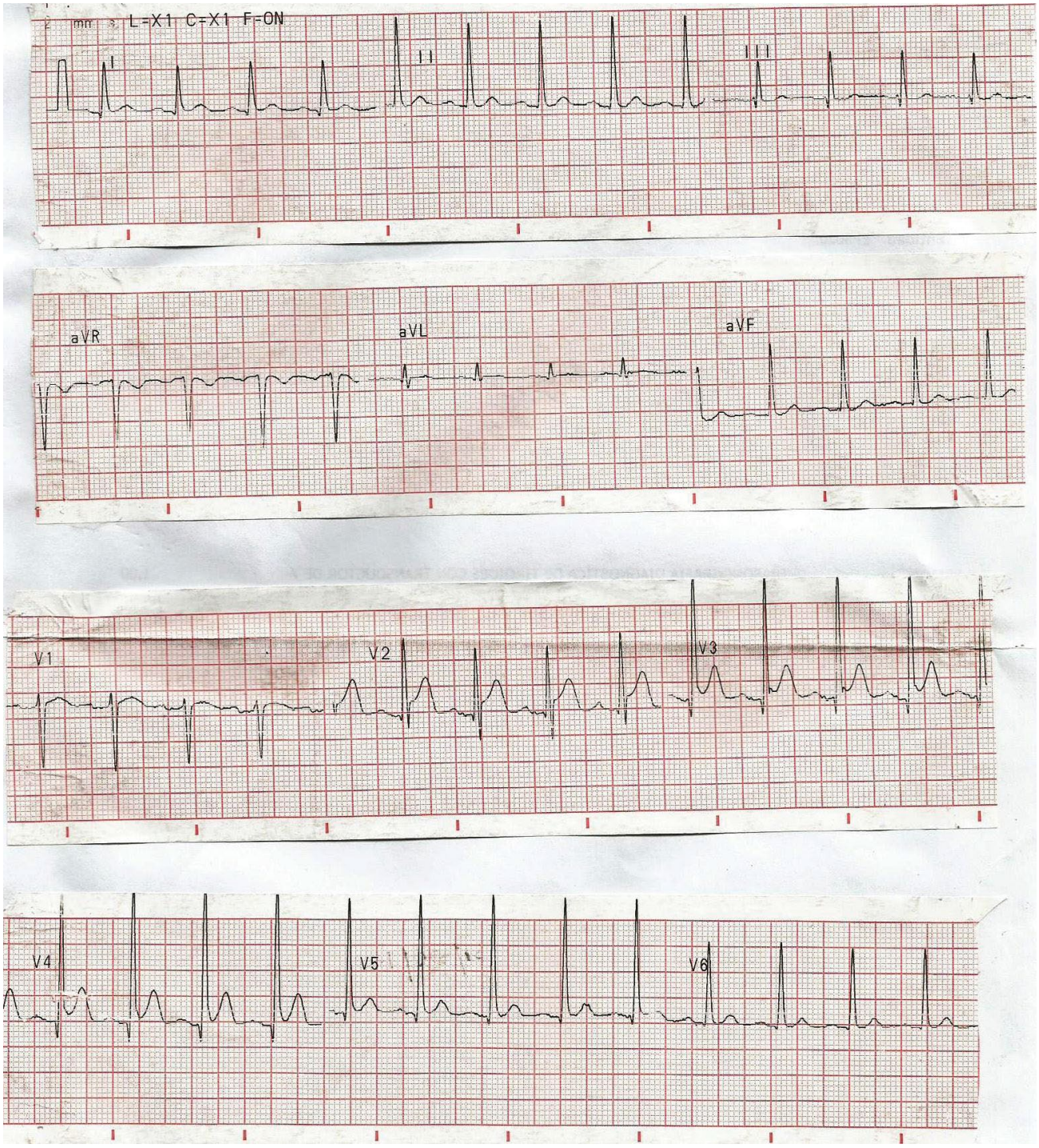

Figure 1: Admission ECG. Which electrolyte abnormality is shown in this tracing?

In our case, the PR segment was normal as well as QRS complex. However, it has been described that the amplitude of the QRS decreased as the ionized calcium level decreased, and the presence of short QTc has also reverted once the hypercalcemia has been corrected. The abnormal short QTc that was evidenced in the first ECG was not shown in the second tracing, with a QTc of 355 ms as is shown in figure 2 [4,6]. It has been reported in previous cases that patients who underwent a surgical remove of adenoma as the cause of primary hyperparathyroidism, recover normal ionized calcium levels as well of ECG regular features. Our patient's parathyroid adenoma has not been resected but his calcium ionized levels decreased considerably and this was reflected on the control ECG tracing [7]. On the ECG, the lower limit for the duration of QTc is not well established but it is reasonable to consider a normal QTc interval between 360 ms and 450 ms in males and 370 ms to 470 ms in females [4,8].

The patient never reported thoracic pain and cardiac enzymes taken initially were negative. It is important to mention this since the ECG showed ST elevation in V2-V4 which could correspond to myocardial ischemia, a finding that has been reported in some similar cases with hypercalcemia [4].

In this case, the presence of J waves which occur in certain conditions like hypothermia, early repolarization and, the Brugada syndrome could be observed. These findings are most prominent in precordial leads V2 to V5. In the patient, they are clearer in the second tracing when the calcium level was lower but still high, so it is difficult to distinguish between early repolarization and real Osborn waves since those waves are commonly correlated with severe hypercalcemia [6].

On the other hand, the ECG evidenced signs of left ventricular hypertrophy (according to Sokolow criteria). The patient did not have an echocardiogram for demonstrating any left ventricular

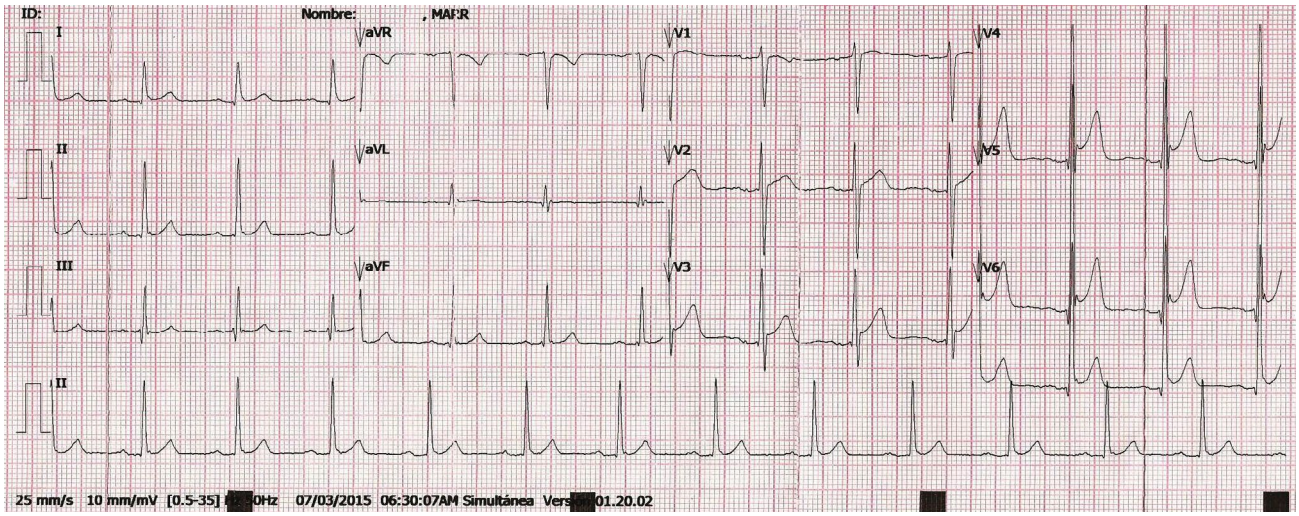

Figure 2: Control ECG.

hypertrophy but this could be related to the presence of chronic hypertension which is one of many cardiovascular manifestations of hyperparathyroidism [1,8].

As we mentioned before, the patient was referred to the Head and Neck Surgery and Oncology department for operative management which is currently the only curative therapy and is clearly indicated for all patients with classic symptoms or complications of PHPT, as in this case. The patient met criteria for surgical management such as < 50 years of age, inability to participate in an appropriate follow up, and complications of PHPT that unfortunately were associated to a chronic unattended illness [9].

References

1. Ahmad S, Kuraganti G, Steenkamp D (2015) Hypercalcemic crisis: a clinical review. Am J Med 128: 239-245.
2. Claudio Marcocci, Filomena Cetani (2011) Primary Hyperparathyroidism. N Engl J Med 365: 2389-2397.
3. Pepe J, Curione M, Morelli S, Colotto M, Varrenti M, et al. (2013) Arrhythmias in primary hyperparathyroidism evaluated by exercise test. Eur J Clin Invest 43: 208-214.
4. Robert C Schutt, Mina Elnemr, Amy L Lehnert, David Putney, Anusha S Thomas, et al. (2014) Case Report: Severe Hypercalcemia Mimicking St-Segment Elevation Myocardial Infarction. Methodist Debakey Cardiovasc J 10: 193-197.
5. Iskandar SB, Jordan RM, Peiris AN (2008) Electrocardiographic Abnormalities and Endocrine Diseases – Part D: Adrenal Gland, Diabetes, and Other Endocrine Disorders. Tenn Med 101: 35-41.
6. Chhabra L, Spodick DH (2013) Milk Alkali syndrome: an electrocardiographic masquerader for non-hypothermic Osborn phenomenon. Heart 99: 1302-1303.
7. Gardner JD, Calkins JB Jr, Garrison GE (2014) ECG diagnosis: The effect of ionized serum calcium levels on electrocardiogram. Perm J 18: e119-120.
8. Rhee SS, Pearce EN (2011) Update: Systemic Diseases and the Cardiovascular System (II). The endocrine system and the heart: a review. Rev Esp Cardiol 64: 220-231.
9. Orlo H Clark, Stephen F Hodgson, George L Irvin III, Michael Kleerekoper, Janice L Pasieka, et al. (2005) The American Association of Clinical Endocrinologists and the American Association of Endocrine Surgeons position statement on the diagnosis and management of primary hyperparathyroidism. Endocr pract 11: 49-54.