ATYPICAL PRESENTATION OF RHABDOMYOLYSIS DUE TO HYPOKALAEMIA INDUCED BY DIURETICS ABUSE: CASE REPORT FROM SIERRA LEONE

PRESENTACIÓN ATÍPICA DE RABDOMIÓLISIS POR HIPOCALEMIA INDUCIDA POR ABUSO DE DIURÉTICOS: REPORTE DE UN CASO DE SIERRA LEONA.

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Conceptos clave

La rabdomiólisis no traumática no es un evento frecuente ni fácil de diagnosticar en la emergencia. En contextos donde los recursos diagnósticos son limitados debe tenerse una alta sospecha al relacionar la historia de la enfermedad del paciente con los mecanismos fisiopatológicos de la enfermedad. Este trabajo busca resaltar la anamnesis y el examen físico como primeros métodos diagnósticos del paciente, apuntalando que los diagnósticos infrecuentes y raros también ocurren en comunidades remotas, inclusive cuando no es fácil objetivarlos por la escase de recursos. El entendimiento médico y el accionar coherente son fundamentales para el beneficio del paciente.

Resumen:

La rabdomiólisis es el resultado de la necrosis de las fibras musculoesqueléticas y la consiguiente fuga de constituyentes musculares a la circulación. Su asociación con algo diferente a un trauma en la sala de emergencias no es tan frecuente. Presentamos el caso de un varón de 47 años, hipertenso, con historia de abuso de diuréticos, desarrollando debilidad e incapacidad para caminar por sus propios medios, encontrando en la bioquímica sanguínea que había padecía rabdomiólisis por hipocalemia.

Palabras clave: rabdomiólisis; hipopotasemia; diuréticos.

Abstract:

Rhabdomyolysis results from acute necrosis of skeletal muscle fibres and consequent leakage of muscle constituents into the circulation. Its association with anything different than trauma in the Emergency Room is not that frequent. We present the case of a 47-year-old male, hypertensive, that developed weakness and incapability to walk without help, finding on the blood biochemistry that he had developed a rhabdomyolysis due to hipokalemia after abusing of diuretics.

Keywords: rhabdomyolysis; hipokalemia: diuretics

Recibido: 2018-06-18 **Aceptado:** 2018-08-13
DOI: http://dx.doi.org/10.31053/1853.0605.v75.n4.20398

Introducción

Rhabdomyolysis is an unusual diagnosis in the consultations to emergency services by patients who did not have muscle injury related to trauma or ischemia. However, in those cases associated with hipokalemia this should be suspected as a probable cause[1], raising the question about identifying the causative factor for hipokalemia. The abuse of diuretics is a known cause of severe hipokalemia events that may, beyond generating rhabdomyolysis, even end in the death of the patient[2]. A patient with rhabdomyolysis will then be presented in the context of hipokalemia associated with indiscriminate use of diuretics.

Presentation of the case

We present a black, 47-year-old, obese, male patient, resident in the Koinadugu district of north-east Sierra Leone, with known history of hypertension only as medical history, assumed to be of essential origin, medicated, with scarce follow-up by any health service. Patient at the time of the consultation was in treatment with enalapril 20mg / hydrochlorothiazide 12.5 mg in the morning and amiloride 5mg / hydrochlorothiazide 50mg at night. He refers in the last month, consuming some tablets of captopril 50mg and furosemide 20mg weekly to help control his pressure. Denies traumatisms or changes in his usual lifestyle, does not refer exercises or intense efforts.

He comes to the consultation in the afternoon, to the emergency service of the Kabala government hospital, in which said service operates under the tutelage of a humanitarian aid organization. The patient reports that 72 hours before the consultation began with proximal muscle weakness predominantly in the pelvic girdle associated with moderate pain in the proximal muscles of the 4 limbs. This weakness makes it impossible to go without the help of a cane and presents an evolution throughout the day of slight intensity as soon as it wakes up, where it can start its march without support, worsening throughout the day until practically invalidating at night. Denies other symptoms. He relates this event as the first and only in his life.

Physical examination is completely lucid, with dry mucous membranes, and predominant weakness in lower limbs that overcomes gravity, but not resistance. A palpation refers to mild pain in the muscles of the lower back. Does not have oedema in decubitus areas. It is found with a heart rate of 100 beats per minute and a blood pressure of 150/80 mmHg. The rest of the physical exam is normal.

Initially with the suspicion that the symptoms are due to electrolyte disturbances in the context of dehydration, an ionograma and renal function are requested, yielding the result of them: Glycemia 119 mg / dL, Creatinine 0.7 mg / dL, Na 137 mmol / L, K 2.2 mmol / L, Ca 9.9 mg / dL, Alb 4.2.

Since there are no potassium ampoules or potassium supplement tablets in a radius of at least 6 hours away by car and considering that the patient had some degree of dehydration, he received 1000 ml of lactate ringer solution that provides 130 meq. of sodium, 4 meq of potassium, 3 meq of calcium, 109 meq of chloride and 28 meq of lactate for each litre and his given bananas to consume. Without an important improvement during 6 hours of observation in the emergency service, the patient decides not to remain hospitalized, for which reason he retires from the service with the recommendation of a diet rich in potassium, modification in antihypertensive treatment, with discontinuation of diuretics and not to mediate other events, is scheduled in 72hs.

Return to the 3rd day, without improvement of his initial complaint, do not added new symptoms, and the pressure on this opportunity is at 130/80. Again, the laboratory is controlled with values of: Glycemia 81 mg / dL, Creatinine 0.8 mg / dL, Na 130 mmol / L, K 2.4 mmol / L, Ca 9 mg / dL. In this opportunity, the patient brings on his own two ampoules of potassium chloride of 10mEq / 10ml each that are administered diluted in a litre of lactated ringer's solution. A urinalysis is performed that reports a light yellowish colour, pH 7, positive proteins a cross, and positive blood two crosses. It manages a creatine kinase (CK) dosage that shows a value of 3555 U / L (reference value 30-380 U / L) with values of ALT 107 U / L (reference value: 10-47 U / L) and AST 367 U / L (reference value: 11-38 U / L).

The patient continues with ambulatory controls following the same prescriptions, and in 48 hs . regains muscle strength, walks on his own again, and reports that the muscle pains have progressively disappeared. A new laboratory was carried out two weeks after the modifications in the antihypertensive treatment where CK normalization is founded (Value: 233 U / L) and also its serum potassium level (Value: 3.5 mmol / L).

Discussion

Rhabdomyolysis is a pathological condition where exists destruction of muscle cells and overturning to the circulating blood of numerous elements contained inside, we highlight in this opportunity: myoglobin, aspartate aminotransferase, alanine aminotransferase, creatine kinase and potassium. It is the increase of creatine kinase at least 5 times its baseline value, which makes the diagnosis of rhabdomyolysis, which may or may not be accompanied by renal failure and hiperkalemia. Clinically, they are patients who usually present with pain and muscle weakness, sometimes referring to changes in urine coloration[3, 4.]

The causes of rhabdomyolysis are innumerable, some better known than others. Basically, they are categorized into: a) secondary to exercise and trauma or, b) without mediating any of these two conditions, in the latter is where the infectious, toxic or drug-related causes, metabolic disorders and enzymatic deficits are found[5].

Hipokalemia is a known, although not frequent, cause of rhabdomyolysis. The physio pathological explanation says that potassium values at the capillary level are important in the regulation of vascular tension. Thus, in a state of severe hipokalemia, vasoconstriction occurs reducing the supply of blood to muscle tissue, which will trigger the process of muscular cell damage and death[6,7]. This explains the disability of rapid onset that presented our patient[8].

Knowing this and being aware that other triggering events of rhabdomyolysis did not mediate in our patient, we can attribute their symptoms and their high level of CK to rhabdomyolysis secondary to hipokalemia. Then we must investigate the origin of hipokalemia for this patient, because although we have diagnoses for their current situation, we have not described still their etiology, that will help us to face the therapy effectively and try to prevent its recurrence.

The etiology of hipokalemia will also be categorized according to: a) it is due to transcellular movements or, b) to potassium losses (renal or extrarenal). It is this last mechanism that is most frequently involved in hipokalemia, renal and gastrointestinal potassium losses[9].

By extrapolating this knowledge to our patient, we remember that it does not present nor as an antecedent digestive symptom such as vomiting or any obvious type of bad absorption syndrome. Thus, the loss of gastrointestinal potassium would not seem to be the explanation for its hipokalemia. Regarding renal losses, he had a history of erratic, uncontrolled and disproportionate use of diuretics. Unfortunately, we do not have an accessible method in this context for more studies that allow us to show the renal losses of potassium.

Although, due to the presentation of the acute event, the explanation of abnormal potassium losses in urine is a strong hypothesis for this patient, we cannot affirm yet without fear of being wrong, that is responsible for the condition of the patient presented.

In the differential diagnosis for these entities we can consider several syndromes, focusing on detailing what we think would be ideal to rule out in our patient. We refer to Conn Syndrome or primary hyperaldosteronism[10-12]. It is a disease that typically suspected in treatment-resistant hypertension. It´s associates with hipokalemia, metabolic alkalosis, and a suppression of plasma renin activity and excess production of aldosterone. It has a prevalence of 1-10% and should be highly suspected in patients with repeated episodes of muscle weakness. The level of hipokalemia in this condition can be so severe that it triggers the onset of episodes of rhabdomyolysis as has been published in other case reports. In our case, the patient presents an erratic management of his arterial hypertension, which does not allow us to classify him as resistant to treatment, nor discard it. He doesn't refer any history of similar episodes at the direct question.

Conclusion

Medicine in remotes areas of the world sometime present a real challenge to find a diagnose and proper treatment to the patient with the limited resources, highlighting the great importance to the interrogation, physical examination and physio pathological thinking.

Hydro-electrolyte disorders due to diuretic abuse are well known, hipokalemia particularly[13,14]. It is known that severe hipokalemia has actions in the blood flow at the muscular level, and that its variation generates alterations that can become ischemic for the muscle fibres and will give rise to the consequent rhabdomyolysis. In hypertensive patients, the periodic treatment and control duly performed by a doctor helps prevent these episodes that can even become fatal for the patient.

Grants
No funds were requested.
Disclosures
We declare no competing interests.

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