6. Pastor MA, Izquierdo MJ, Vargas-Machuca I, Carrasco L, Fariña MC, Martín L, et al. Enfermedad de Grover en un paciente con insuficiencia renal crónica en hemodiálisis. Actas Dermosifiliogr. 2003;94:169–72.
7. Boutli F, Voyatzi M, Lefaki I, Chaidemenos G, Kanitakis J. Transient acantholytic dermatosis (Grover's disease) in a renal transplant patient. J Dermatol. 2006;33:178–81.
8. González-Sixto B, Rosón E, de la Torre C, García-Doval I, Cruces M. Grover's disease in a patient undergoing peritoneal dialysis with resolution after renal transplant. Acta Derm Venereol. 2007;87:561–2.
9. Bassi E, Roujeau JC, Grimbert P, Ortonne N, Bagot M. Grover's disease in a renal transplant patient, after hemodialysis renewal. G Ital Dermatol Venereol. 2012;147:222–3.
10. Jatem E, Agraz I, Semidei ME, Ferrer B, Ramos R, Fort J. Grover's disease in a peritoneal dialysis patient. Nefrologia. 2013;33:608–9.

Laura Rodríguez-Pazos[a,*], Alejandro Vilas-Sueiro[a], Daniel González-Vilas[a], Cristina Durana[b]

[a] *Servicio de Dermatología, Complejo Hospitalario Universitario de Ferrol, Ferrol, A Coruña, Spain*
[b] *Servicio de Anatomía Patológica, Complejo Hospitalario Universitario de Ferrol, Ferrol, A Coruña, Spain*

[*] *Corresponding author.*
E-mail address: ladrizos@hotmail.com (L. Rodríguez-Pazos).

Severe arrhythmia due to hypokalemia. Influence from diuretic substances☆

Arritmia cardiaca grave por hipopotasemia. Influencia de las sustancias diuréticas

Dear Editor,

This was a case of a 25-year-old woman with no known allergies or relevant medical history and no toxic habits. She is an attorney and drinks 500–750 ml of beverages containing taurine and 1 l of caffeinated soda per day due to stress. The following details were observed: height 170 cm, weight 58 kg and BMI 20. She was admitted to the hospital for headache and tachycardia during the last two days after she did some sports and coinciding with an increase in the consumption of a beverages containing taurine. She denied chest pain or dyspnoea. Had no vomiting or diarrhoea and had no change in diuresis. She did not consume herbal products, drugs, teas, diuretics, liquorice or alcohol. *Physical examine*: Conscious, oriented, blood pressure 108/86, heart rate 110 beats per minute. Afebrile. Anodyne cardiopulmonary auscultation. Rest of the examination was normal. *Blood test*: normal red cell count, no elevation of cardiac or hepatic enzymes and coagulation test without alterations; creatinine 1.04 mg/dL, urea 31 mg/dL, potassium 1.73 mEq/L, sodium 134 mEq/L, magnesium 2.2 mg/dL, chloride 85 mEq/L, Albumin 4 g/dL. Arterial blood gas: Ph 7.580, PCO_2 46 mmHg, PO_2 86 mmHg, bicarbonate 43.1 mmol/L. Plasma anion gap (AG): 5.9 mEq/L. Urine: chloride 22.2 mEq/L, potassium 68.28 mEq/L, sodium 210 mmol/L, urea 920 mg/dL, creatinine 192.72 mg/dL, glucose 15 mg/dL. Urine anion gap: 256 mEq/L. Plasma osmolality: 278.2 mOsm/L. Urine osmolality: 573.3 mOsm/l. Transtubular potassium gradient: 15. Cortisol at 8 am and aldosterone in supine position were within the normal range. No alterations in urinary sediment. ECG: sinus rhythm, markedly enlarged QT (580 ms; corrected 700 ms); with frequent polymorphic ventricular tachycardia (Fig. 1). An infusion of CLK was initiated via central line: 80 mEq within two hours and maintained with an infusion of 120 mEq/day. After 18 h, urine test was: sodium 25.3 mEq/L, potassium 6.2 mEq/L; in serum: sodium 142 mEq/L, potassium 2.8 mEq/L transtubular potassium gradient: 4. Venous blood gas: Ph 7.380, PCO_2 52 mmHg, HCO_3 30.8. Blood test at discharge: sodium 143 mEq/L, potassium 4.84 mEq/L, chloride 105 m Eq/L, pH 7.380, pCO_2 49 mmHg, bicarbonate 29 mmol/L. Urine: potassium 11.59 mmol/L, sodium 89 mmol/L, creatinine 266.27 mg/dL, urea 642 mg/dL. The ECG was normal. The evolution of ions in the urine suggested the presence of a diuretic substance that was suspended at admission. *Diagnoses*: hypokalaemia due to diuretic substances: taurine and caffeine, but not being able to rule out the presence of other diuretics, aggravated by the increase of insensitive losses and alkalemic state. A Bartter vs Gitelman-type tubulopathy was ruled out given the evolution of the ions in urine and the hormonal axis normality. Alteration in heart conduction due to hypokalaemia. Mixed alkalaemia: Chloride-resistant metabolic alkalosis due to diuretic substances and reactive respiratory alkalosis.

Ninety percent of the potassium filtered at glomerular level is reabsorbed in the proximal tube. The distal tubule, by effect

DOI of original article:
http://dx.doi.org/10.1016/j.nefro.2015.05.008.
☆ Please cite this article as: Salanova-Villanueva L, Bernis-Carro C, Alberto-Blazquez L, Sanchez-Tomero JA. Arritmia cardiaca grave por hipopotasemia. Influencia de las sustancias diuréticas. Nefrologia. 2015;35:334–336.

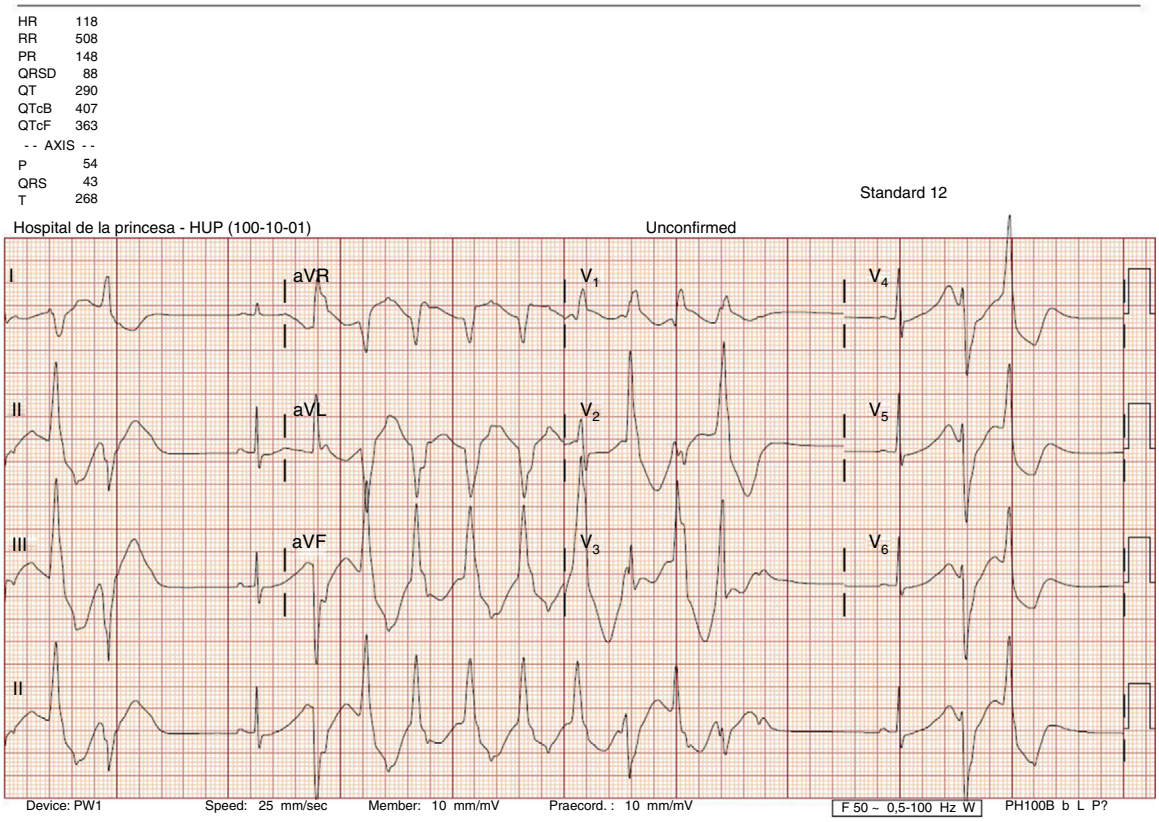

Fig. 1 – Polymorphic ventricular tachycardia. Increased QT.

of the aldosterone, regulates the urinary output according to the body needs (Fig. 2). Distal excretion of Kis modified by the amount of fluid, distal contribution of sodium, mineral corticoids and excretion of non-reabsorbable anions. The most common cause of hypokalaemia due to renal losses are non-potassium-sparing and similar diuretics. Hereditary tubulopathies (Bartter and Gitelman) may not be distinguishable from the intake of diuretics. Hyperaldosteronism and hypermineralcorticoidism cause hypokalaemia due to their action on the distal nephron. Potassium is a predominant intracellular cation. The best marker to assess the renal management of potassium is TTKG in euvolemia that assesses

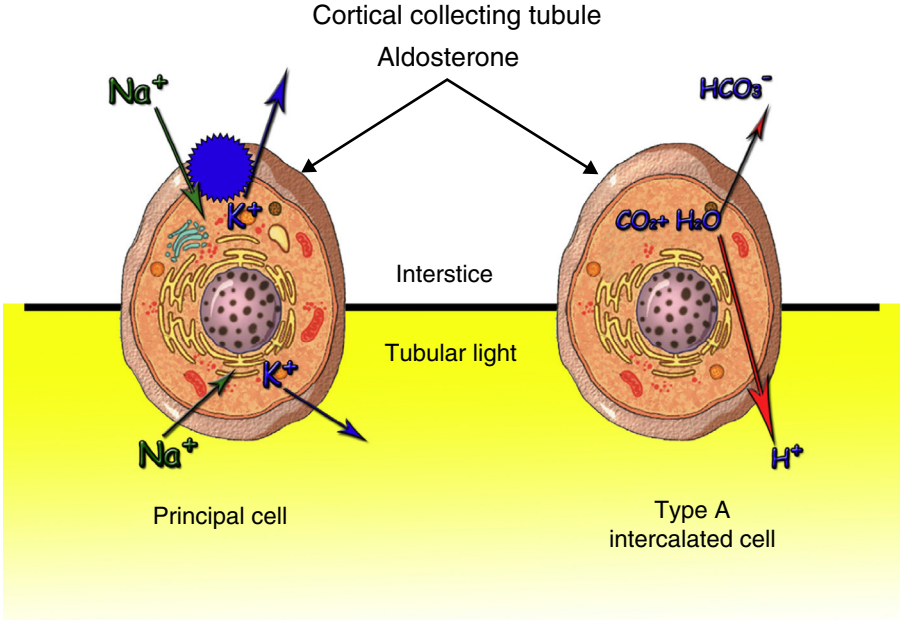

Fig. 2 – Distal tubule. Action of aldosterone on distal tubule.

the mineralocorticoid action on the distal nephron: values <4 indicates absence and >7 presence of activity. Blood pressure, extrarenal losses, acid-base state, urinary ions and urine and plasma AG have to be assessed. In the presence of metabolic alkalosis, as in our case, chloride concentration decreases in order to compensate for the elevation of bicarbonate and the AG increases in proportion to the alkalosis severity, due to the lactate and the concentration of more anionic serum proteins. In turn, the kidney tends to increase the excretion of bicarbonate at proximal and distal tube level where there is a Cl^-/HCO_3 exchange in the beta-intercalated cells of the collecting tubule. Serious chloride, potassium or extracellular space depletion inhibits this exchange. Urine AG is an indicator of urinary acidification. Positive values indicate that renal acidification is intact. Treatment must be oral. The intravenous line is reserved for serious hypokalaemia ($K < 2.5$ mEq/l), arrhythmia, acute myocardial infarction or digitalisation.[1]

Some characteristics of the energy drinks diuretic components:

Caffeine

Natural xanthine. Energy drinks have levels between 75 and 174 mg per serving, others exceed 500 mg.[2] This stimulates the central nervous system, cardiovascular system, and central respiratory system; it relaxes the smooth bronchial muscle and striated muscle, increases acid gastric secretion and renal blood fluid and has diuretic properties. Many of these effects are caused by antagonic action on adenosine receptors.[3] It is rapidly distributed through the organism and crosses the placental and blood–brain barrier. It has hepatic metabolism (cytochrome P-450). It is clinically used as a respiratory stimulant in newborns with apnoea of prematurity. The adverse effects include insomnia, agitation, headache and tachycardia at elevated doses.[4] The changes in blood pressure response are not conclusive.[5] It can produce dependence syndrome.[6]

Taurine

Conditionally essential amino acid. Its deficit is associated with cardiomyopathy, retinal degeneration and failure to thrive.[7] Metabolic actions include: bile acids conjugation, osmolar regulation, detoxification, membranes stabilisation and modification of cellular sodium and calcium levels. It has positive inotropic action and protects the cardiac membrane from the adverse effects of hyperglycaemia.[7,8] Its renalprotective effect is caused by its antioxidant action by controlling the effects generated by TGF-B1 and type I and IV collagen.[8] It increases the glomerular filtration rate, reduces sodium tubular reabsorption, reduces urine protein and inhibits antidiuretic hormone production.[8,9] Clinically, it has been used in hypercholesterolaemia, epilepsy, cardiopathy, retinal macular degeneration, Alzheimer's disease, cystic fibrosis and hepatic diseases.[10]

Both of them increase natriuresis increasing the arrival of sodium at the distal tubule: activating aldosterone and producing the entry of cellular sodium and the exit of potassium to the tubular light causing hypokalaemia.

Most of the supplements contained in the energy drinks have concentrations below the amounts associated with adverse effects.[4] The association of heart conduction alterations is not clear with studies for and against them.[11,12] The combination of these drinks with alcohol may cause arrhythmias in subjects prone to them.[13]

REFERENCES

1. Ayus JC, Caramelo C, Tejedor A. Agua, electrolitos y equilibrio ácido-base. Ed. Med. Panamericana.
2. Reissig CJ, Strain EC, Griffiths RR. Caffeinated energy drinks. A growing problem. Drug Alcohol Depend. 2009;99:1–10.
3. Haller CA, Jacob P, Benowitz NL. Enhanced stimulant and metabolic effects of ephedrine and caffeine. Clin Pharmacol Ther. 2004;75:259–73.
4. Clauson KA, Shields KM, McQueen CE, Persad N. Safety issues associated with commercially available energy drinks. J Am Pharm Assoc. 2003;48:e55–63.
5. Dalbo VJ, Roberts MD, Stout JR, Kerksick CM. Acute effects of ingesting a commercial thermogenic drink on changes in energy expenditure and markers of lipolysis. J Int Soc Sports Nutr. 2008;5:6.
6. Oberstar JV, Bernstein GA, Thuras PD. Caffeine use and dependence in adolescents: one year follow-up. J Child Adolesc Psychopharmacol. 2002;12:127–35.
7. Schaffer SW, Azuma J, Mozaffari M. Role of antioxidant activity of taurine in diabetes. Can J Physiol Pharmacol. 2009;87:91–9.
8. Mozaffari MS. Taurine modulation of renal excretory function. TJPS. 2003;16:83–90.
9. Amiry-Moghaddam M, Nagelhus E, Ottersen OP. Light- and electronmicroscopic distribution of taurine, an organic osmolyte, in rat renal tubule cells. Kidney Int. 1994;45:10–22.
10. Birdsall TC. Therapeutic applications of taurine. Altern Med Rev. 1998;3:128–36.
11. Nagajothi N, Khraisat A, Velazquez-Cecena JLE, Arora R. Energy drink-related supraventricular tachycardia. Am J Med. 2008;121:e3–4.
12. Berger AJ, Alford K. Cardiac arrest in a young man following excess consumption of caffeinated energy drinks. Med J Aust. 2009;190:41–3.
13. Wiklund U, Karlsson M, Oström M, Messner T. Influence of energy drinks and alcohol on post-exercise heart rate recovery and heart rate variability. Clin Physiol Funct Imaging. 2009;29:74–80.

Laura Salanova-Villanueva*, Carmen Bernis-Carro, Luis Alberto-Blazquez, Jose Antonio Sanchez-Tomero

Servicio de Nefrología, Hospital de La Princesa, Madrid, Spain

* *Corresponding author.*