

Secondary Hypokalemia of Acute Renal Tubular Acidosis: Case Report

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Abstract

Acute Renal Tubular Acidosis is a rare cause of severe hypokalemia. The research for this etiology and the initiation of its treatment is very important because it is a vital diagnosis and therapeutic emergency.

Introduction

Hypokalemia is a common reason for hospital admissions (more than 20% of patients in hospital) [1].

It is often asymptomatic in the healthy patient, but can threaten the life of the patient with heart problems.

In this context, it is important to have a good grasp of the evaluation and management of this frequent disorder and to systematically seek its cause [2].

Among the etiologies of hypokalemia we encountered acute tubular acidosis for the first time in the medical emergencies of CHU Oran, which etiology we report in this case [3].

CASE

Mrs D. Fatima, 42 years old, without children admitted in Intensive Care Unit for asthenia and shock.

Patient with personal history:

Medical: irritable bowel syndrome treated symptomatically

Surgical: cholecystectomy on the May 7th, 2018 in Ain Türk hospital

Family: normal

The beginnings of disorders date back to 9 months by the sudden onset of abdominal colic pain, nausea and vomiting, constipation, anorexia, weight loss and debility, which made her go to see a gastrologist who treated these disorders as an irritable bowel with laxative prescription.

After 6 months of unfavorable evolution, the patient visited

a general practitioner, an ultrasound has been made showing gallstones treated surgically, with simple postoperative instructions but the debility, anorexia and weight loss have increased, motivating the patient to see the tri medical area on the 21st may 2011.

In the tri medical area

The patient had clouding of consciousness, Blood Pressure: high, Heart rate: 130 BPM, dehydrated (skin fold). The action to be taken: oxygen therapy, 2 peripheral vein, volume replacement, standard checkup. The blood electrolytes checkup revealed: normal natremia ($Na^+=135$), severe hypokalemia ($K^+=1,29$). An electrocardiogram (EKG) revealed severe hypokalemia signs: flattening of ST segment, the U wave appearance. The Abdominal echography and the thoracic radiography were normal.

The correction of the potassium was started with 1g/h.

The 22nd of May 2018 at 23h the patient has degraded her neurological score with state of shock motivating a filling and intubation with artificial ventilation. The correction of the potassium has been continued.

The 23rd of May 2018 at 9h: the patient was conscious, cooperating, good cough and swallowing reflexes BP: 11/06 HT: 90 BPM, the patient is extubated and transferred to intensive care.

In intensive care unit (ICU)

A blood electrolyte checkup was realized: normal natremia $Na^+=142$ m mole /l, hypokalemia $k^+=2,1$ m mole /l, hypocalcemia $Ca^{++}=72,4$ mg /l, hypermagnesemia $Mg= 16,6$ mg /dl, possibly iatrogenic.

A urinary electrolyte checkup was also realized: $NAu =255$

meq /24h, Ku=127meq / 24h knowing that the potassium is in process of correction.

A thyroidal test has been made in order to detect a hyperthyroidism but it was normal.

We asked for a blood count: White blood cell=25200, hemoglobin=10g/dl, platelets=213000. We searched an infectious cause: cyanobacterial check-up of urines was negative; the lumbar puncture was negative too. The patient was under cefizox 1g/6h.

The patient was conscious, cooperating, afebrile, but always asthenic with ongoing constipation, bloating and abdominal pain with the persistence of hypokalemia despite the daily correction of the potassium.

An abdominal scanner has been asked for, in order to search an abdominal cause causing a digestive leak of the potassium which became without any anomaly.

We realized a digestive endoscopy, we noticed a reflux esophagitis, congestive gastritis, healthy bulb, normal duodenal light.

A blood gas checkup showed: PH= 7,37, PCO₂=18mmhg, HCO₃⁻=10,8 mole/an offset metabolic acidosis with HCO₃⁻ broke down.

Plasma cortisol was made to find hypercortisolism that came back normal.

We made another urinary electrolyte checkup: NA u = 387meq /24h, Ku =100meq /24h, the probability of a urinary leak of K⁺ is taken back.

A gastric casing was carried out 3 days in a row looking for a cause of this debility and weight loss in favor of pulmonary Tuberculosis came back negative.

After eliminating all the possible causes of hypokalemia and after the results of the urinary ionograms confirming high kaluresis

and the results of the gas blood checkup, the hypothesis of a renal leak of K⁺ is carried over, tubular acidosis is selected as a probable diagnosis, the treatment is taking bicarbonates by mouth diluted in water and distributed throughout the day.

The patient has taken bicarbonates diluted in water and distributed throughout the day and every day, without correction of the potassium in direct intravenous injection, only with increasing dietary value with oral intake of potassium, we notice the disappearance of the asthenia and refeeding.

A gas blood checkup was realized revealing: PH=7,45, PCO₂=36,4mmhg, HCO₃⁻=25,8mmole/. Blood ionogram revealed: NA+=136mmole/l, K+=3,01mmol/l, electrocardiogram realized was normal.

Discussion

Hypokalemia is a difficult clinical diagnosis. The clinician's attention should be drawn to a range of clinical signs that can range from simple constipation to respiratory distress through paralysis of the respiratory muscles. the search for the cause is essential and allows the prescription of a suitable treatment. Several etiologies are at the origin of this disorder, among which acute tubular acidosis which is rare and few cases have been reported in the literature. The treatment is simple; it consists of alkaline supplementation by oral route.

References

1. H. Corraze, J. Levraut (2007) Hypokaliémies Journal Européen des Urgences 20: 86-90.
2. Guidon-Attali, Jean-Christian Colavolpe, Georges François. Dyskaliémies de l'adulte. EMC Anesthésie-Réanimation [36-860-A-15].
3. John R. Asplin, Frédéric L (2002) Col Maladies tubulaires héréditaires. Principes de Médecine Interne 1598-1606.