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Review Article

Internal Environment Disturbances (Electrolytes and Acid-Based), what to Correct First? From Theory to Practice, Concerning a Clinical Case

Ceballos Gabriella Elizabeth*, Añasco Daniela, Plos Carlos, Rodriguez Bell Margaret and Medrano Tatiana

Ricardo Gutierrez Children's General Hospital - Buenos Aires, Federal Capital City, Argentina

***Corresponding Author:** Ceballos Gabriella Elizabeth, Ricardo Gutierrez Children's General Hospital - Buenos Aires, Federal Capital City, Argentina.

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Abstract

Introduction: In clinical practice, hydroelectrolytic and acid-base state imbalances are very common. In the presence of several alterations at the same time, it is important to consider the patient's medical history, and his or her basic disease, if any, and prioritize what to correct first.

In patients with ureterosigmoidostomy, the main complication is hyperchloremic metabolic acidosis. Other associated alterations are hypomagnesemia, hypocalcemia, hyperammonemia and increased creatinine and blood urea.

Long-term follow-up is important for early diagnosis and treatment, thus avoiding serious life-threatening complications.

Objective: To describe a clinical case with multiple electrolyte disturbances of difficult correction and focus on the management of these disturbances.

Description: A 14-year-old female adolescent seeks medical assistance due to vomiting and diarrhea for 48 hours. A laboratory study was performed, and multiple electrolyte disorder was found.

Diagnosis: Hypernatremic dehydration, hyperchloremic metabolic acidosis with acidemia, hypokalemia without electrocardiographic repercussion, hypophosphatemia, and hypomagnesemia associated with muscle aches and spasms (that required IV fluids treatment), and exacerbation of chronic renal failure in the patient, with Rokitansky syndrome and ureterosigmoidostomy.

Conclusion: The proper correction of the internal environment stabilized the patient; this was a challenge when deciding what to correct first, in the face of multiple electrolyte disturbances.

Keywords: *Electrolytes; Acid-Based; Ureterosigmoidostomy*

Introduction

In clinical practice, hydroelectrolytic and acid-base state imbalances are very common. The management of these should be very precise and cautious to obtain a balance and reverse or avoid situations that could be serious, such as cardiac electrical alterations by potassium or pontine myelinolysis, by rapid corrections of sodium, among others. In the presence of several alterations at the same time, it is important to consider the patient's medical history, and his or her basic disease, if any, and prioritize what to correct first.

Unlike the urothelial mucosa which is relatively impermeable, the absorptive and secretory properties of the gastrointestinal mucosa, make the intestine, even being the best substitute for the urinary tract, produce a series of water exchanges and solutes between urine and blood, when the urine meets the intestinal mucosa, which triggers metabolic disorders.

The alterations are related to the degree of absorption of the intestinal wall, which is influenced by the segment of the gastrointestinal tract used, contact surface, contact time, urine solutes concentration, osmolarity and fluid pH, and renal function levels.

In patients with ureterosigmoidostomy, the main complication is hyperchloremic metabolic acidosis due to resorption of NH_4^+ and secretion of HCO_3^-, it is only symptomatic in a small percentage of cases, being generally, in those with impaired renal function, unable to compensate for the higher acid load.

In the epithelial cells of the distal colon, the epithelial sodium channel (ENaC) and potassium channel (K) are expressed and play a role in sodium reabsorption (Na) and K secretion, which is like the kidneys. These actions are regulated by the renin-angiotensin system through the mineralocorticoid receptor.

In the context of dehydration, both renin activity and aldosterone concentration are elevated, inducing K secretion and Na reabsorption in both the kidneys and the distal colon, explaining hypokalemia.

Other associated alterations have been described, hypomagnesemia, hypocalcemia, hyperammonemia, and increased creatinine and blood urea. As complications can appear in the short and long term, it is necessary to perform in these patients a prolonged follow-up for early diagnosis and treatment, with special attention on those patients with chronic kidney disease [1-5].

Objective of the Study

To describe a clinical case with multiple electrolyte disturbances of difficult correction, and the emergence of thinking about what to correct first, since an error could cause serious complications.

Description

A 14-year-old female adolescent seeks medical assistance due to vomiting and diarrhea for 48 hours.

At the time of physical examination, the patient was in a regular general state, afebrile, normotensive, and tachycardic, with semi-moist mucous membranes; then moderate dehydration was also assessed, so a rapid replenishment of physiological solution was performed and a laboratory, which showed altered renal function (U 118 Cr 1,2) with hypokalemia of 2,7. After this, the patient was admitted for clinical control and treatment.

Relevant background information:

- SGA Baby. Controlled pregnancy.
- Complete vaccines by reference.
- Schooling Interrupted 2 years ago (Complete primary, but not started secondary).
- Prenatal diagnosis of bilateral hydroureteronephrosis by bilateral ectopic ureters with ureter outlet below the bladder neck.
- Bilateral skin ureterostomy at 3 months of age.
- Nephrectomy of the left kidney at 8 years + ureterosigmoidostomy (Caffey surgery).
- Multiple hospitalizations for acute pyelonephritis, without bacteriological rescue.

- Episode of urosepsis.
- Diagnosis of Rokitansky syndrome with total hysterectomy, at 13 years, preserving both ovaries for a second time by hematometra and hematosalpinx.

During hospitalization, the rest of the laboratory is received where there is evidence of multiple electrolyte disorders: hypernatremic dehydration, hyperchloremic metabolic acidosis with acidemia, hypokalemia without electrocardiographic repercussion, hypophosphatemia and hypomagnesemia associated with muscle aches and spasms (that required IV fluids treatment), and exacerbation of chronic renal failure.

After receiving 2 quick restorations of physiological solution, the patient received PHP 2000/140/40 + free water 1200 ml in 24 hours, intending to reverse hypernatremic dehydration.

Phosphorus was prioritized in corrections due to the life risk associated with its severe deficiency, and mono dipotassium phosphate IV was placed in, 30 mg/kg/day in parallel to PHP.

The K was corrected slowly as it did not present electrocardiographic alterations, magnesium sulfate IV was added, 0.8 mg/kg/ dose, every 4 hours due to symptomatology, and bicarbonate of K 3g every 6hs through the oral route, since the intravenous route is incompatible with magnesium administration.

With the IV fluids added, the patient was improving slowly until it was passed to the oral route.

In addition, due to the clinical history and the history of urosepsis without the possibility of ruling out urinary infection due to a single urinary/fecal excretory system (ureterosigmoidostomy), the patient was given Ceftriaxone. The Blood cultures x2 were negative. At 48h presented fever and table compatible with Pharmacodermia (rash + elevated inflammatory parameters + altered liver profile) so the antibiotic is rotated to Pip Tazo and was medicated with Methylprednisolone 30 mg/kg single dose, then the patient presented a slow decline with good clinical and laboratory response. The abdominal ultrasound was normal, viral serologies (EBV, CMV, HBV, HCV, HIV), and blood cultures were negative.

Laboratories	1	2 (Day 2)	3 (Day 2)	4 (Day 2)	5 (Day 3)	6 (Day 3)	7 (Day 3)	8 (Day 4)	9 (Day 5)
ABS (acid-base status)	7.22/37/15.6	7.20/34/13	7.20/26/10	7.20/26/ 10.3	7.21/31/ 12.3	7.22/31/ 12.7	7.22/28/ 12	7.22/34/ 14	7.26/38/ 17
Ionogram (Na/K/Cl/ Cai)	144/2.7/ 112/1.27	150/2.61/ 121/1.38	157/2.15/ 132/1.45	158/2.31/ 134/1.45	152/3.29/ 126/1.35	160/1.95/ 131/1.3	159/3.2/ 132/1.3	147/4.3/ 121/1.15	147/4.5/118 CAI 1.27
Ca T/P^{+}/Mg^{++}		Ca 8.5 P 1.1 Mg 2.4	Ca 8.8 P 0.9 Mg 2.3	Ca 8.9 P 0.7 Mg 2.2	Ca 7.9 P 0.6 Mg 1.9	Ca 8.5 P 0.7 Mg 1.9	Ca 8 P 2.4 Mg 1.8	CA 8 P 4.1 MG 1.5	CA8.8 P 2.2 MG 2.3
Urea/uric acid/ creatinine	U 118 AU 3.2 CR 1.21	U 98 A 2.8 CR 1.06	U 85 A 2.4 CR 0.94			U 76 A 1.9 CR 0.95	U 69 Au 1.5 Cr 0.98	U 28 Cr 1.37 AU 1.3 Glu 92	U36 Cr1 AU1.9 Glu 125

Liver Function Test	BT 0.86 BD 0.28 GPT 14 GOT 28 PT 8.5 AL 5.1	BT 0.55 GPT 10 GOT 21 PT 6.6 AL 4.1	BT 0.39 GPT 12 GOT 30 PT 7 AL 4.3			BT 0.51 GPT 10 GOT 23 PT 6.9 AL 4.4		BT 5.59 BD 4.68 GPT 304 Fal 237 ProtT 5.6 ALb 3.3	BT 0.84 BD 0.64 GPT 163 GO T23 PT6.3 ALb3.6
Hemogram	GB 12400 (64/32/4/1) HB 14 HTO 41 PL 321000	GB 9700 (65/28/7) HB 11.6 HTO 35 PL 258.000				GB 19.300 (2/83/11/4) HB 11.2 HTO 35 PL 193.000		Gb23400 (Nc3/Ns79/ L14/M1/E3) Hb11.4/ HTO 34.1 Plaq 157000	GB 19100 (MM1/NC1/ N81/L13/ M4) Hb 10.5 plaq. 229000
Others	CPK 200 LDH 324 TP 100 KPTT 38	PCR 1	CPK 785 PCR < 0.6			CPK 729 LDH 231		PCR 222.2 Proca 0.93	PCR 57 Amylase 65 Lipase 41

Conclusion

The patient presented severe electrolyte disturbances of difficult management, caused by an intercurrence that triggered dehydration and subsequently the loss of electrolytes generating greater loss than the one produced by ureterosigmoidostomy, since this, behaves like a tubulopathy, requiring hydroelectrolytic inputs exogenously and chronically.

The acute renal injury was presented as an over-imposition to chronic kidney damage and then it improved with hydration. Therefore, these alterations were a great challenge in the stabilization of the internal environment and the patient herself.

Defining what to correct first leads to a good therapeutic response.

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