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Mild Clinical Manifestations in a Patient with Severe Uremia

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Abstract: Nowadays, it is rare to treat anyone patients with very severe uremia. Most of them are monitored by a doctor and long before they reach the end stage of chronic kidney failure they smoothly end up in some method of dialysis. However, there are also some cases who are not monitored, who appear with some symptoms, usually metabolic acidosis, hyperkalemia, or vomiting's, in which case uremia is usually not very severe, and the cause diagnosed by laboratory exams. A young patient was brought to our hospital by his relatives, because during his sleep he developed tonic-clonic spasms and loss of consciousness, without any other particularly subjective or objective findings. It is presented because of the very severe laboratory picture it had and its mild clinical semiology.

Keywords: Severe Uremia, Chronic Kidney Disease, Severe Acidosis, Severe Hypocalcemia, Severe Hyperuricemia, Severe Hyperphosphatemia.

Case Report

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INTRODUCTION

Over 75 individual clinical manifestations or signs have been described in cases of uremia [1]. In severe uremia, the accumulation of many toxins and in fact in large quantities will obviously accompany by heavier clinical manifestations. Moreover, these are expected to be even more pronounced when the kidney disease is unknown to the patient, who will obviously not follow any dietary restriction or some medical instructions. This means that he/she exposed to all the complications of uremia, with the result that particularly serious manifestations are expected. Of course, the anemia that usually accompanies chronic kidney disease (CKD) could be tolerated, however heavy it may be, because it sets in slowly and the body adapts. Thus, he treats some of the uremic disorders, without having significant clinical manifestations or consequences, despite his very heavy laboratory picture.

PATIENT CASE

A 38-year-old man was brought to the hospital because he had a fainting episode and generalized tonic-clonic seizures during his sleep (family members reported seizures, loss of consciousness, biting of the tongue, which was objectively diagnosed in the emergency department as bleeding and trauma of the tongue, which was edematous), which was treated with diazepam. He had a history of arterial hypertension in the last 10 years (which he reported was relatively well regulated), for which he was taking medication, without any follow up by a doctor and without laboratory monitoring. From the general examination of the blood it was found that hematocrit (Hct)=23.3%, hemoglobin (Hb)=7.9 gr/dL, white blood cells=18.4 x 10^9 μ/L (with 76% polymorphonuclears) and serum urea 403 mg/dL (143.9 mmol/L), creatinine 30 mg/dL (2,652 mmol/L), urate 22 mg/dL (1308.6 mmol/L), phosphate (Pi) 11 mg/dL (3.55 mmol/L), total calcium 6.1 mg/dL (1.52 mmol/L), magnesium (Mg) 3.9 mg/dL (1.6 mmol/L), albumin 3.8 g/dL (38 g/L), potassium 6.28 mmol/L, sodium 137 mmol/L, creatine phosphokinase (CPK) 1,009 IU/L and parathormone (PTH) 1,688 pg/ml (1,688 ng/L). The patient reported from his history easy fatigue (however he worked without problems as a waiter), sleepiness (during the morning hours) and decreased libido. On admission he had an arterial pressure of 160/90 mmHg, pulse 94/min and 28 breaths/min.

An impression was made, at the emergency department, by the very low blood pH (6.873) and the small increase of lactates (8.42 mmol/L), apparently due to the convulsions. From the blood gases, it appears that this was metabolic acidosis with an increased anion gap (pH 6.783, HCO_3^- 3 mmol/L, anion gap 35.6 mmol/L). The increased anion gap did not appear to be solely due to the increased lactates. The expected $PaCO_2$ (compensation) was equal to 40-$DHCO_3^-$x1.2=40-21x1.2=40-25.2=14.8 mmHg, which is approximately

what he had ($PaCO_2$=16.9 mmHg). The delta gap was equal to: Na^+-CI^--39=135-102-39=135-141=-6 mmol/L, that means that metabolic alkalosis may have been also present, possibly due to the diuretic he was taking (he was taking irbesartan in combination with hydrochlorothiazide).

An ultrasound scan of the kidneys revealed a kidney length of 9 cm bilaterally, with increased cortical echogenicity. Echocardiographic examination of the heart revealed an ejection fraction of 65%, with diastolic dysfunction, without other pathological findings. Fundoscopy revealed stage II hypertensive retinopathy. Electrocardiogram showed no abnormalities, despite very low ionized calcium. In the computed tomography of the brain, no pathological findings were found.

A temporary jugular catheter was placed and beginning haemodialysis without any anticoagulation (with flushes of the filter with 100 ml 0.9% NaCl/hour), with a 1.6 m^2 surface area filter (polyethersulfone), with a blood pump flow of 200 ml/min and a dialysate flow of 500 ml/min, lasting the session 2 hours. A 0.9% NaCI solution with 6 amp of calcium gluconate 10%, was given to the patient in each of the first 3 hemodialysis sessions.

After 3 days he restored normal diuresis (800-2,300 ml/24h), but without any noticeable renal clearance ([Clearence creatinine+Clearance Urea]/2 = 4 ml/min). The patient's blood gases (venous sample) after three days and 2 consecutive dialysis sessions (of 2 hours the first and of 2.5 hours the second) showed pH 7.36, HCO_3^- 21.5 mmol/L, anion gap 15 mmol/L, while total calcium was 1.78 mmol/L (corrected for serum albumin).

The following made a special impression on this patient: 1) while he had severe metabolic acidosis (pH 6.783, HCO_3^- 3 mmol/L), he had no clinical consequences (hypotension, arrhythmias), b) while he had very heavy hypocalcemia (total calcium 1.525 mmol/L), he had no clinical or ECG manifestations (despite his albumin was quite good [38 g/L]), c) while he had very severe uremia and anemia, he did not have any clinical findings before admission to the hospital, such as e.g. vomiting (he was working normally without problems) and d) while he only had arterial hypertension for 10 years and lesions of hypertensive retinopathy, he reached at the end stage of CKD, which raises serious suspicions that the cause of the hypertension maybe was some underlying glomerulopathy. The case of the patient is therefore presented for its mild clinical findings, despite the very severe laboratory picture of uremia he had.

DISCUSSION

Inadequate elimination of many potentially toxic organic metabolites from the body via the urine in CKD is associated with various clinical symptoms, which are often difficult to interpret. Almost all organs and systems of the body are affected by the uremic toxins that are not removed in uremia [1].

According to the USRDS, by 2010, less than 1.28% of patients with end-stage CKD had creatinine >20 mg/dL, with no patient reported having a creatinine value >30 mg/dL. Raj *et al*., published a case of a young patient with CKD who had very high serum creatinine (4,598 mmol/L and serum urea 154.3 mmol/L). This was an obese, African American man (1.8 m tall and 115 kg body weight) who presented to the emergency department with high blood pressure and edema of the lower extremities and face, complaining of cough, vomiting, headache, and leg cramps in the last 4 days. From the other laboratory tests, he also had hyperphosphatemia (serum phosphate 3.39 mmol/L), hypocalcemia (total serum calcium 1.575 mmol/L), anemia (Hb 5.1 gr/dL) and PTH 682 ng/L, as has our patient. Ultrasound examination revealed CKD (small kidneys, with increased echogenicity) [2]. Storm *et al*., published a patient with the highest creatinine ever published with CKD (53.9 mg/dL). This was a young man 34 years old, who presented complaining of nausea, vomiting, and diarrhea during the last 6 weeks. He was clinically hypertensive (blood pressure 184/93 mmHg), and he had ankle edema, without nocturia. Laboratory findings were serum urea 102.5 mg/dL, phosphate 5.07 mmol/L, total calcium 1.925 mmol/L (albumin 37 g/L), hemoglobin 5.6 gr/dL, and PTH 1,187.2 ng/L, without acidemia (pH=7, 33) [3].

Uremic frost is a skin manifestation of very heavy uremia, that is rarely observed nowadays, due to the early initiation of hemodialysis in patients with CKD [4]. When blood urea levels are high (urea over 71.4 mmol/L), its concentration in sweat increases significantly. Then the evaporation of sweat causes crystallization and its appearance on the skin which is like snow or salt. Our patient, despite having a very high urea value (143.9 mmo/L), did not have frost on the skin, nor morning vomiting, which is common in such severe uremia (uremic gastritis) [5]. Among 9 cases of uremic frost published in the literature, the mean blood urea level was 151 mmol/L and the mean serum creatinine was 1,547 mmol/L [6]. Relatively recently, Wlodarczyk & Zapata published a case of a 58-year-old man with abdominal pain and skin frost. Laboratory tests showed a serum creatinine of 1,768 mmol/L and a blood urea of 200 mmol/L [7].

There are two main categories of patients with seizures and CKD. Those who experience an acute symptomatic crisis in the context of existing CKD and those with epilepsy who at some point in the course of their disease develop CKD. The prevalence of epileptic attacks in patients with CKD is ~10%. These are often non-spastic and may mimic uremic encephalopathy [8]. Uremic encephalopathy is defined as an organic brain disorder that develops in patients with acute or chronic

kidney disease, usually when the eGFR is <15 ml/min. The manifestations of this syndrome vary from mild symptoms (e.g., easy fatigue), to severe, such as seizures and coma (usually at pH<6.9, as our patient had). The other laboratory disorders of our patient, such as hypocalcemia (total calcium 1.525 mmol/L) and hyperkalemia (potassium 6.28 mmol/L), may be related to the seizures, although the serum potassium level was not so high as to cause neurological symptoms.

About two-thirds of urate is excreted by the kidneys and the remaining one-third by the bile, so urate accumulates in patients with CKD. Thus, a decrease in glomerular filtration rate may lead to hyperuricemia. In CKD, in addition to the reduction in the number of nephrons [9], there is also acidosis, in which uremic organic acids accumulate, which compete with the tubular secretion of the urate, leading to hyperuricemia. This occurred in our patient, who had severe acidosis (pH 6.873) and was also receiving antihypertensive medication with a thiazide diuretic (thiazide diuretics and furosemide increase urate levels because they increase its proximal tubular reabsorption and decrease its secretion) [10, 11]. In clinical practice, elevated levels of urate in the serum of patients with CKD are also related to many other factors, such as the coexistence of obesity, dyslipidemia, alcoholism, intake of foods rich in fructose and purines, lack of exercise, etc., which our patient did not have [12, 13].

In hyperphosphatemia, hemodynamic changes are noted in patients with end-stage CKD, that are responsible for left ventricular hypertrophy [14]. It is usually asymptomatic, as was the case of our patient.

From the above it appears that our patient, despite the very heavy uremia (laboratory results and the very severe acidosis he had), his clinical picture was mild. Many of his disturbances could have been potentially fatal, but he did not appear to be severe ill and survived the whole situation, entering in a program of conventional hemodialysis. It also seems that in every case of arterial hypertension, especially in young people, is good to rule out causes that may be treatable.

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