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Hypokalemia paralyzing revealing a rare association of autoimmune diseases: type 1 diabetes, thyroiditis and tubulopathy about a case.

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Abstract: *Hypokalemia is a Paralyzing uncommon pathology, primitive often genetic autosomal dominant. Type 1 diabetes is an autoimmune disease of the pancreas responsible for a total deficit of insulinsecretion and hyperglycaemia. Someclinical manifestations extraglandular, canbe a discovery mode of the disease. We report the case of a patient with hypokalemiamoroccan Paralyzingrevealingthis pathology Whose combination with a renaltubulopathy by distal tubular acidosis (DTA) type 1 and autoimmune diseases of specific organ is rare.*
The DTA type 1 is related to the Inability of the distal tubule to secrete sufficient amount of hydrogen ions, making it possible to acidification of the urine below pH 5.5. This DTA is responsible for a severe rarely hypokalemia, neuromuscular complications, or revealing of a type 1 diabetes as is the case of our patient. A case of hypokalemiaparalyzingpuring a Sjögren syndrome (GSS) is associated with a thyroiditis was reported.
Hypokalemia paralyzing revealing combination of autoimmune diseases annually (type 1 diabetes, thyroiditis and tubulopathy) isuncommon and difficulties to diagnose. Thus, faced with an unexplained hypokalemia neuromuscular paralysis in young patients, we must think although rare to a tubulopathy in the context of autoimmune diseases.

Keywords:*HypokalemiaParalyzing, Tubulopathy, Distal renaltubular acidosis (DTA) type 1, type 1 diabetes, Thyroiditis.*

Summary:
Hypokalemia is a rare paralyzing condition, often primitive genetic autosomal dominant. The type 1 diabetes is an autoimmune disease of the pancreas responsible for a total deficit of insulinsecretion and hyperglycaemia. Glandular extra clinical manifestations can be a way of discovering the disease. We report the case of a Moroccan patient with hypokalemia paralyzing revealing this pathology whose association withtubulopathy by distal renal tubular acidosis (ATD) type 1 and autoimmune diseases specific organ is rare.
The type 1 ATD is related to the inability of the distal tubule to secrete sufficient amount of hydrogen ions, making it impossible to acidification of the urine below pH 5.5. This ATD is very rarely cause severe hypokalemia or revealing neuromuscular complications of type 1 diabetes as is the case of our patient. A case of paralytic hypokalemiaduring a Sjögren's syndrome (SS) associated with thyroiditis was reported. The type of association 1 ATD-specific autoimmune diseases is exceptional organ.
Hypokalemia paralyzing revealing an association of autoimmune diseases (type 1 diabetes, thyroiditis and tubulopathy) is rare and difficult to diagnose. Thus, faced with an unexplained hypokalemia neuromuscular paralysis in young patients, we must think although rare, a tubulopathy in the context of autoimmune diseases.

Keywords: Hypokalemiaparalyzing, Tubulopathy, Tubularacidosistype 1 (ATD), Type 1 diabetes, Thyroiditis.

I. Introduction

The hypokalemic paralysis is a rare condition, usually primitive genetic autosomal dominant. The type 1 diabetes is an autoimmune condition characterized by pancreaticinsulinopenia and hyperglycemia, officials of a cardinal inaugurating syndrome mostoftendiabeticketo-acidosis. Some extra glandular manifestations may apply inaugurate the clinical picture. We report the case of a Moroccan patient with hypokalemia paralyzing revealing this pathology whose association withtubulopathy by distal renal tubular acidosis (ATD) type 1 and autoimmune diseases specific organ is rare.

II. Observation

This is Mrs. NA, a Moroccan aged 28, who like history, miscarriage 05 months ago, diabetic heredity, no notion of taking medication or exposure to toxic; hospital through the emergency to tetraparesis.

One week before admission, the patient describe episodes of muscle weakness of the lower limbs and difficult climbing with higher and maintain the static position. These events were preceded by a polyuropolydipsic syndrome withpolyphagia and significant weight loss. On the eighth day appeared a significant functional impairment of all four limbs more marked in the lower limbs, but no isolated disorders of consciousness or respiratory disorders or swallowing, no digestive problems. Furthermore, the patient does not report similar symptomatology before, or similar cases in the family.

On admission, there was dehydration, polypnéique, afebrile, blood pressure (BP) to 110 / 70mmHg without orthostatic hypotension, flaccidtetraparesiswithmuscularhypotonia members and an abolition of tendon reflexes without damage of sensory functions.

Laboratory tests showed: diabetic ketoacidosis (DAC) withhyperchloraemicmetabolicacidosis (blood glucose 4 g / L, acetonuria +++, bicarbonatémie: 15mmol / L, HbA1c 7.5%, serum chloride: 105mmol / L), severe hypokalemia (K +: 1,6mmol / L), elevation in urinary potassium (98mmol / 24 hours), with good renalfunction, normocalcemia, rhabdomyolysis (CPK: 4100U / L); hypothyroidism autoimmunethyroiditis (TSHus to 19,97µUI / ml antibody (Ab) thyroperoxidase (ATPO) positive 938,54UI / ml). The neck ultrasonography was in favor of thyroiditis. The acts of the negative celiac disease.

The ECG showed T wave flattened. Electromyogram for a pure motor axonal neuropathy mechanism; lumbar puncture came back normal. Antinuclear antibodies (ANA), anti-Ac DNA and rheumatoid factor are negative. The dosage of complement fractions is normal. The hepatitis B and C serology and VIH1-2 are negative. The rest of the balance sheet shows a cortisol to 12µg / dl, normal ESR, CRP to 18mg / L, leukocytosis, and positive urine culture C.albucans, a normochome normocytic anemia 10g / dl.

The patient received sodium bicarbonate administration 1-2 mg / Kg / day of potassium supplementation, a DAC protocol (rehydration, insulin), an antifungal treatment with Fluconazole, a basal-bolus insulin therapy scheme with therapeutic education and a replacement therapy with L-Thyroxine.

The evolution was marked by the gradual remission of motor deficit, muscle pain and normalization of laboratory parameters (bicarbonatémie, potassium, CRP, ESR, CPK). The euthyroidclinical and biological standardization of HbA1c and complete remission of motor deficit are obtained after 03 months.

III. Discussion

The hypokalemic paralysis is a rare condition, usually primitive genetic autosomal dominant due to mutations in the gene SCN4A (encoding muscle sodium channel) or CACLN1A3 gene (encoding the muscle calcium channel). This is a rare muscle channelopathymanifested by the occurrence of recurrent paralysis access for a few hours to days, ranging from a para- or tetraparesis to a complete paralysis of 4 members with risk of respiratory disorders and swallowing. In addition to the familial forms (Westphall of disease), thyrotoxic periodic paralysis and barium poisoning are common etiologies. These conditions are caused by an abnormality of the transfer membrane K + [1]. In our observation, potassium loss is of renal origin by increases in urinary potassium. Normal blood pressure and metabolicacidosishyperchloraemiceliminatehypokalemia by aldosteronism but rather in favor of the distal renal tubular acidosis (ATD) type1 [2]. The latter is most often secondary to an autoimmune disease [3] but rarely associated with diabetes type 1 or Hashimoto's thyroiditis, as in our case. The age of our patient, the absence of antibodies systemic diseases and family history, eliminate a primitive renal etiology. There is no guide element to an iatrogenic or toxic cause. However, hyperglycemia in diabetes is likely to cause renal potassium depletion by polyuria. The pathophysiologic mechanism of tubular damage in diabetes is most likely related to the complications of the latter. Now the microangiopathic complications in type 1 diabetes are later, but can be pre-existing diverse etiologies.

The type 1 ATD is related to the inability of the distal tubule to secrete sufficient amount of hydrogen ions, making it impossible to acidification of the urine below pH 5.5. Biologically, the ATD is characterized by metabolicacidosis, hypokalaemiahyperchloraemic and an alkaline urinary pH; moderate renal impairment is sometimes observed. This ATD is very rarely cause severe hypokalemia neuromuscular complications [4] or indicative of a type 1 diabetes as is the case of our patient. This is a quick installation table leading to a flaccidtetraparesisusuallypredominant on the proximal muscles but up to the respiratory muscles or cranial nerves; cardiac arrhythmias may also occur [5]. Hypothyroidism by Hashimoto's thyroiditis can be an aggravating factor neuromuscular event related to hypokalemia and rhabdomyolysis. Moreover hypothyroidism in general is responsible for the occurrence of tubular acidosis deficit by pumps H + / ATPase at the collector tubes [6].

Sporadic cases of ATD develop most often in adults and can be primary (almost always in women) or secondary to variousdiseases [Sjögren's syndrome (SS) or renal acidosis, renal transplantation, nephrocalcinosis, medullary néphrospongiose or chronic kidney obstruction] or drug reactions [7]. A case of paralytic hypokalemia during a SGS associated thyroiditis has been reported [8].
The type of association 1 ATD-specific autoimmune diseases is exceptional organ [9].

IV. Conclusion

Hypokalemia paralyzing revealing an association of autoimmune diseases (type 1 diabetes, thyroiditis and tubulopathy) is rare, especially in our difficult diagnostic context without medical history. Thus, faced with an unexplained hypokalemia neuromuscular paralysis in young patients with no notable medical history, although rare, look tubulopathy (type 1 ATD) in the context of autoimmune diseases (type 1 diabetes, thyroiditis of Hashimoto).

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