



Congress Abstract

Clinical Case: Diagnosis And Treatment of Distal Tubular Acidosis (Type 1)

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ABSTRACT

Introduction: Renal tubular acidosis (RTA) in adults remains an underdiagnosed condition, often masked by other diseases. Distal RTA is particularly challenging as it may present primarily with neurological symptoms for a long time, including muscle weakness, flaccid pareses, and episodes of paralysis against the background of hypokalemia. In clinical practice, such patients are often initially hospitalized and monitored in neurological departments with suspected primary neurological pathology, which leads to delays in establishing the correct diagnosis. The true cause of these symptoms is metabolic and electrolyte disturbances caused by RTA.

Late diagnosis increases the risk of complications such as nephrocalcinosis, nephrolithiasis, and progression of chronic kidney disease. Thus, reporting clinical cases of RTA with “neurological masks” is important to raise awareness among doctors of various specialties and ensure timely differential diagnosis.

Objective: To analyze a clinical case of distal RTA in an adult patient who presented with neurological symptoms at onset.

Clinical case: A 19-year-old male was admitted with severe weakness in all limbs and inability to move independently. He had been monitored by a cardiologist for mitral valve prolapse and was one of triplets. At 17, he experienced two episodes of muscle weakness progressing to severe movement restriction after physical exertion. At 18, he was hospitalized twice for periodic paralysis with hypokalemia (1.6–3.3 mmol/L). Treatment included potassium supplements and metabolic therapy.

On admission, potassium was 1.6–2.8 mmol/L and chloride 126–127 mmol/L, while sodium remained normal (140–143 mmol/L). Immunological and hormonal tests, including ANA, anti-dsDNA, ANCA, antiphospholipid antibodies, thyroid hormones, and anti-TPO, were normal. CT showed bilateral renal microcalculi and right-sided pyeloectasia. Neurological evaluation was performed, and AChR and MuSK antibodies were ordered to exclude myasthenia gravis.

Severe hypokalemia was corrected with intravenous KCl. Considering hypokalemia, hyperchloremia, and nephrolithiasis, distal renal tubular acidosis was suspected.

Conclusions: Distal RTA in adults can present initially with pronounced neurological symptoms, mimicking primary nervous system diseases.

The presence of hypokalemia combined with hyperchloremia requires exclusion of RTA, especially in cases of recurrent episodes of muscle weakness.

Detection of nephrolithiasis or kidney microcalculi is an important diagnostic marker supporting distal RTA.

The presented case highlights the need for interdisciplinary approach and increased vigilance among physicians to consider metabolic causes of neurological disorders.

Keywords: Distal Tubular Acidosis; Hypokalemia; Metabolic Acidosis