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A case of hypokalemic paralysis secondary to distal renal tubular acidosis as a presenting symptom in Sjogrens syndrome

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Sjogrens syndrome is a chronic inflammatory disease characterized by lymphocytic infiltration of the exocrine glands, particularly the lacrimal and salivary glands. Secondary Sjogrens is found in patients with existing autoimmune diseases such as rheumatoid arthritis or systemic lupus erythematosus. Extraglandular manifestations of Sjogrens such as distal renal tubular acidosis are well reported in the literature, however hypokalemic paralysis associated with distal RTA is a much rarer complication. Here, we present a case of a 41 year old female with a history of SLE and RA who presented to our facility with bilateral lower extremity paralysis of one day duration. Patient was evaluated by neurology with an initial differential diagnosis of Guillian Barre, cervical lesion in her spinal cord or Myasthenic crisis. Initial lab work revealed a severe non-anion gap metabolic acidosis with a positive urine anion gap and severely depleted potassium ($K=1.4$). Patient was immediately started on potassium supplementation and IVIG, however her weakness progressed to her upper extremities and patient subsequently developed hypercapnic respiratory failure requiring intubation and mechanical ventilation. Patient's acidosis was corrected with a sodium bicarbonate infusion and her hypokalemia eventually improved with aggressive potassium supplementation. She was weaned from the ventilator and downgraded from the ICU. Further workup revealed positive anti-SSA antibodies indicating secondary Sjogrens syndrome although patient did not report xerostomia or sicca like symptoms. This case is an uncommon presentation of Sjogrens associated distal RTA presenting as hypokalemic paralysis.

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The diagnosis and management of acute kidney injury: A complete audit cycle

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Introduction: In 2009 NCEPOD reported only 50% of patients who had died of acute kidney injury received good care. In 2013 NICE produced guidelines on how to prevent, detect and manage those in AKI.

Aim: This study aims to know does the Royal Surrey County Hospital comply with NICE Guidelines (CG 139) for the identification and management of acute kidney injury.

Method: Patients in AKI were identified by their renal function blood results on the program 'WinPath'. Data was collected on a total of 20 medical and 20 surgical patients. By retrospectively reviewing their case notes we identified whether the correct diagnosis was made, the associated risk factors and if the appropriate investigations and management strategies were implemented. Interventions included teaching to junior doctors, distributing AKI alert cards and displaying posters on each ward.

Results: The initial audit showed that only 38% of patients with AKI were correctly diagnosed. After the intervention this increased to 63% (70% for medicine, 55% for surgery). 95% of the patients had three or more risk factors. The most common risk factors included being over the age of 65, taking nephrotoxic drugs and having a diagnosis of hypertension. The Royal Surrey implemented 2 and a half times more management strategies only when AKI was correctly diagnosed in comparison to no diagnosis.

Conclusion: The intervention has proven to be a success with the Royal Surrey increasing the rate of AKI diagnosis from 38% to 63%. This was achieved using relatively inexpensive, practical to deliver and straight forward techniques. Once AKI diagnosis is made, this allows for a better management plan. Nevertheless there is still room for improvement. Future considerations include a computerized warning system, focused drug chart reviews, an accessible guide to AKI management and a proforma to identify those at risk with the aim of prevention.

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