Rapamycin-induced hypokalaemic nephropathy in a middle-aged hypertensive male

Sir,

Rapamycin-induced hypokalaemic nephropathy is rarely reported in renal transplant patients. Rapamycin, an mammalian target of rapamycin (mTOR) inhibitor, acts by preventing cell-cycle progression; it prolongs delayed graft function (DGF) and decreases repair of tubular cells [1]. There are reports of thrombotic microangiopathy when rapamycin is used with calcineurin inhibitors [2]. We hereby report a man who developed hypokalaemic nephropathy secondary to rapamycin.

A 31-year-old male with end-stage renal disease (ESRD) underwent a renal transplantation on 4 May 2005, the donor being his mother. He was inducted with dacluzimab 20 mg and immunosuppressed with ciclosporin 275 mgBD, prednisolone 35 mgOD and sodium salt of mycophenylate, 360 mgBD. He weighed 68 kgs. He had a biopsy-proven secondary to hypokalaemia and the drug was withheld. Two weeks later his creatinine declined to 2.1 mg/dl with serum potassium 4.1 mmol/l. His immunosuppressants were tacrolimus 3 mgBD and prednisolone 12.5 mgOD.

At the molecular level, mTOR is necessary for maintaining the integrity and regeneration of tubular epithelial cells, which account for the majority of potassium reabsorption [1]. The sustained improvement in creatinine and potassium following discontinuation of rapamycin strongly implicates this agent in the development of hypokalaemic nephropathy. Though the incidence of hypokalaemia on rapamycin is 34%, profound hypokalaemia is very rare [3]. The temporal relationship of recovery of renal function following discontinuation of rapamycin and correction of hypokalaemia suggests rapamycin as the inciting agent for acute kidney injury. This case highlights the profound hypokalaemic response to minimal maintenance dose of rapamycin in the absence of diarrhoea, vomiting or medications causing hypokalaemia.

Conflict of interest statement. None declared

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