Magnetic resonance imaging in a patient with chronic lithium nephropathy

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Nephrotoxicity in patients with affective disorders and on long-term treatment with lithium salts is a well-known pharmacological side effect [1]. About 20% of the patients on long-term lithium therapy may develop renal insufficiency with ‘creeping creatinine’ and increased risk for progression to end-stage renal disease [2].

We report here the case of a 51-year-old woman treated with lithium for over 30 years due to an affective disorder. Serum lithium levels had been consistently documented within normal limits (0.55–0.95 mval/l). In 2002, her serum creatinine level was 1.19 mg/dl and further increased to 1.33 mg/dl in June 2004, which then led to withdrawal of lithium therapy. Although the patient was switched to another mood-stabilizing medication, renal insufficiency (serum creatinine 1.52 mg/dl, creatinine clearance 50 ml/min) further progressed, which led to the patient’s presentation at our university hospital in January 2005.

On admission, the patient reported polydypsia and polyuria (>5 l/day) as well as nocturia once every night. We measured reduced urine osmolality (179 mmol/kg) and urine density (1.001 kg/l) suggesting lithium-induced nephrogenic diabetes insipidus [1]. At that time, the patient was on 100 μg L-thyroxine once daily, due to lithium-induced goitre with hypothyroidism as previously reported in the literature [1]. A family history of cystic kidney disease was denied and there was no hypertension.

Physical examination demonstrated overweight (BMI 27.0 kg/m²) and slight peripheral non-pitting oedema. Urine analysis did not show any haematuria, proteinuria or glucosuria. Abdominal ultrasound revealed normal-sized kidneys with condensed, scarred renal parenchyma and a blurred demarcation from the surrounding tissue. Several small hypeechoic parapelvic cysts and hyper-echoic reflexes were displayed. A magnetic resonance (MR) tomography was subsequently performed, which displayed renal fibrosis and very abundant, uniformly and symmetrically distributed small renal cysts (1–2 mm) in the renal cortex and medulla. In the T2-weighted MR images, these cysts appeared as hyperintense (white) spots in the intermediate (gray) signal of the normal parenchyma (Figure 1). This pattern of cystic changes is

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considered to be a characteristic feature of lithium nephropathy [2]. In addition, we performed a renal biopsy, showing focal chronic tubulointerstitial damage with tubular atrophy, interstitial fibrosis and mild lymphocytic inflammation, affecting 20–25% of the cortical tubulo-interstitium. In the biopsy core, three nephrons with microcystic ectasia as well as one globally scarred glomerulum were present. The patient was discharged with the diagnosis of lithium nephropathy and recommended to avoid further lithium intake.

The leading clinical findings suggestive of lithium nephropathy were polydypsia/polyuria and abundant/symmetrically distributed renal microcysts in normal-sized kidneys. These microcysts are present in 33–63% of the patients with lithium intake and are localized in both renal cortex and medulla [2]. Differential diagnosis of renal microcysts includes simple renal cysts, autosomal-dominant polycystic kidney disease as well as glomerulocystic and medullary cystic kidney disease which can be distinguished according to number, size, location of cysts and clinical symptoms [1]. A recent study proposed that microcysts secondary to long-term lithium therapy can be detected with MR imaging [3]. Its specificity makes MR imaging sensitive for detecting renal cysts <2 mm. It enables the evaluation of the entire renal parenchyma and thus yields global information on the number and distribution of cysts in both renal cortex and medulla [3].

MR imaging was able to reveal the presence of characteristic parenchymal microcysts secondary to lithium therapy and allowed the confirmation of the clinical diagnosis of chronic lithium nephropathy. In our patient, no other cause of progressive renal insufficiency was detected in renal biopsy beyond lithium nephropathy, which was already diagnosed in the MR imaging. However, further prospective studies are required to confirm that MR imaging of parenchymal microcysts is sensitive and specific for the diagnosis of lithium nephropathy and that this novel technical approach can spare renal biopsy.

Conflict of interest statement. None declared.

References


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