



Study on chronic renal disease in a minor child

Younesi Rostami*

Department of Urology, Mazandaran University of Medical Sciences, Sari, Iran

✉ **Younesi Rostami**

Department of Urology,
Mazandaran University of Medical Sciences,
Sari 4813894393,
Iran
E-mail: roshtani34@gmail.com

Received: 03-Feb-2023, Manuscript No. PUCR-23-91892; **Editor assigned:** 06-Feb-2023, PreQC No. PUCR-23-91892 (PQ); **Reviewed:** 20-Feb-2023, QC No. PUCR-23-91892; **Revised:** 27-Feb-2023, Manuscript No. PUCR-23-91892 (R); **Published:** 06-Mar-2023, DOI: 10.14534/j-pucr.20222675602

Description

The long-term risk of Chronic kidney disease (CKD) in adult lithium users has been established by several studies [1]. Few case reports of children with lithium-induced nephropathy exist [2]. A 14-year-old patient who, after a cumulative exposure of 9 years, developed CKD and a clinical and radiological picture that suggested Chronic Tubulointerstitial Nephritis (CTIN).

In place of hyperparathyroidism, another unusual finding was hypercalcemia with suppressed Parathyroid Hormone (PTH).

Chronic tubulointerstitial or glomerular injury, or even both, may be the cause of the clinical signs and symptoms of lithium nephrotoxicity. CTIN in the form of interstitial fibrosis, dilated tubules, and microcysts is the most frequent finding on renal biopsies. Although subclinical interstitial fibrosis can be seen on biopsy as early as two years after starting treatment, clinical manifestations of lithium-induced CKD only appear after 10 to 20 years [3]. Polyuria due to impaired urinary concentrating ability (nephrogenic diabetes insipidus),

low grade proteinuria, hematuria, and elevated creatinine are possible clinical presentations. Contrarily, glomerular damage is less obvious, and people with glomerular toxicity typically have proteinuria in the nephrotic range.

There have been several proposed pathophysiologic mechanisms for lithium-induced nephropathy. Lithium transports into tubular cells and becomes trapped there because it is a poor substrate for the sodium-potassium-ATPase pump on the basolateral membrane, resulting in cytotoxic lithium concentrations. Lithium replaces sodium at Epithelial Sodium Channels (ENaC) on the apical membrane of the collecting duct. The few paediatric case reports of lithium-induced nephropathy have shown membranous nephropathy, focal segmental glomerulosclerosis, and minimal change disease as the causes of nephrotic syndrome (glomerular injury). After taking lithium for 5.5 years, one case report described a combination of glomerular and interstitial injury [4]. Our patient's initial presentation suggested that CKD was the result of CTIN, nephrogenic diabetes insipidus, and some degree of acute kidney injury. The presence of concurrent CKD was confirmed by the patient's low baseline Estimated Glomerular Filtration Rate (eGFR) both before and after the acute component had subsided. Echogenic punctate foci and other findings of microcysts with extensive involvement of the cortex and medulla were seen during a renal ultrasound. The most likely cause of this pattern is CKD brought on by lithium. The smaller microcysts represented by these punctate foci are believed to exist but are invisible to CT or MRI. The primary differential diagnosis was nephrocalcinosis,

which frequently results in echogenic and punctate foci that are primarily in the medullary region.

The most effective treatment for bipolar disorder is lithium, which may need to be continued in some patients if stopping it has negative psychiatric effects. Particularly in patients who have already developed CKD, close cooperation among Primary care-specialist (PCPs), psychiatrists, and nephrologists should aid in balancing the risk of suicide, mental health relapses, and progressive renal damage [5].

Conclusion

In these patients, hypercalcemia is much more common than hyperparathyroidism, which suggests that other mechanisms may be at play. This case study demonstrates that CKD caused by lithium can manifest in a young patient. When children are receiving lithium therapy, carers must closely monitor renal function.

References

- [1] Markowitz GS, Radhakrishnan JA, Kambham N, et al. Lithium nephrotoxicity: a progressive combined glomerular and tubulointerstitial nephropathy. *J. Am. Soc. Nephrol.* 2000; 11(8):1439-1448.
- [2] Petersen CE, Amaral S, Frosch E. Lithium-induced nephrotic syndrome in a prepubertal boy. *Jadpet.* 2008; 18(2):210-213.
- [3] Zaidan M, Stucker F, Stengel B, et al. Increased risk of solid renal tumors in lithium-treated patients. *Kidney Int.* 2014; 86(1):184-190.
- [4] McKnight RF, Adida M, Budge K, et al. Lithium toxicity profile: a systematic review and meta-analysis. *The Lancet.* 2012; 379(9817):721-728.
- [5] Werneke U, Ott M, Renberg ES, et al. A decision analysis of long-term lithium treatment and the risk of renal failure. *Acta Psychiatr Scand.* 2012; 126(3):186-97.