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Nephrogenic diabetes insipidus and chronic tubulointerstitial nephritis caused by lithium toxicity

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Case Study: Background: Only few cases of tubulointerstitial nephritis due to lithium side effects have been reported. Herein, we report the case and our findings. Case: A 54-year-old female with manic depressive illness who had been receiving treatment at the neuropsychiatry department visited the emergency department 2 hours before the hospital visit, with altered mentality as the chief complaint. Her vital signs were stable and peripheral blood test results were as follows: hemoglobin level, 11.0 g/dL; white blood cell count, 9300/mm^3; and platelet count, 247,000/mm^3. The BUN and Cr increased to 20.7 and 2.9 mg/dL. Serum lithium level, 3.76 mMol/L (reference value: 0.6–1.2 mMol/L). Renal ultrasonography revealed no specific finding. Hemodialysis was performed under the diagnosis of lithium poisoning. On day 4, the serum lithium level decreased to 0.16 mMol/L, and the hemodialysis was stopped. On day 5, acute renal failure improved with a BUN 6.2 mg/dL and Cr 1.2 mg/dL. However, her daily urine volume increased to 6995 mL and serum sodium level to 159 mMol/L. Her urine osmolality was 211 mOsm/kg, indicating nephrogenic DI. Under the diagnosis of nephrogenic DI due to lithium poisoning, treatment with amiloride was started, but her daily urine volume further increased to 7830 mL and urine osmolality decreased to 135 mOsm/kg, showing no treatment response. Thus, indomethacin was added to increase amiloride dose. Renal biopsy was performed on day 33 to determine the renal damage caused by the lithium and prognosis. On biopsy findings, she was diagnosed as having acute tubular necrosis and chronic tubulointerstitial nephritis. On day 46, the daily urine volume was <3000 mL and the urine osmolality was reduced to 268 mOsm/kg, showing improvement. Conclusion: The findings of this study show that with long-term use of lithium, acute lithium poisoning, nephrogenic DI, and chronic tubulointerstitial nephritis may occur simultaneously.

Figure 1. Inflammtory cell infiltration in the interstitium with tubular invasion are seen (PAS stain).