

[PICTURES IN CLINICAL MEDICINE]

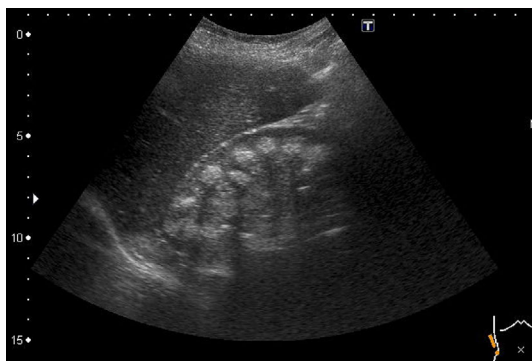
Induction of Macroscopic Nephrocalcinosis by Acetazolamide and Vitamin D

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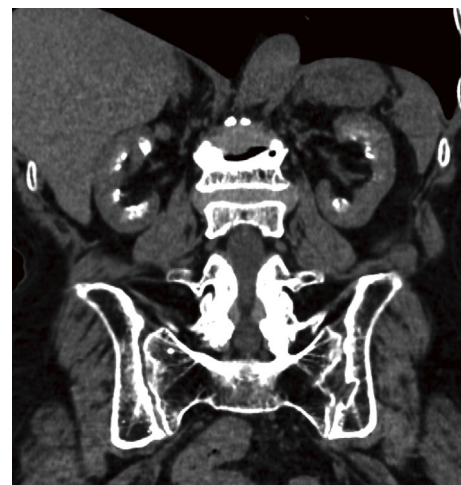
Key words: nephrocalcinosis, acetazolamide, eldecalcitol

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Picture 1.



Picture 2.

An 80-year-old woman with a history of epilepsy was admitted to the hospital with a distal radius fracture. Blood samples revealed hypercalcemia and acute kidney injury prior to surgery. Ultrasonography was performed to test for acute kidney injury, revealing aligned nephrocalcinosis (Picture 1). Computed tomography also showed findings suggestive of calcification (Picture 2). Blood gas studies revealed metabolic acidosis despite hypercalcemia. Regarding her history of oral medication, she had taken eldecalcitol to treat osteoporosis for 1.5 years and acetazolamide as an antiepileptic drug for at least 10 years. Her medical history, physical findings, and test results showed no positive findings suggesting nephrocalcinosis due to other diseases, including Sjögren's syndrome, sarcoidosis, or primary hyperparathyroidism. Acetazolamide is a carbonic anhydrase inhibitor that acts mainly in the proximal tubules and causes type 2 renal tubular acidosis (RTA). Although type 2 RTA is generally considered less likely to cause nephrocalcinosis than type 1 RTA, carbonic anhydrase inhibitors are exceptional because they decrease the urinary excretion of citric acid and inhibit stone dissolution (1). In a previous report, 15%

of patients treated with acetazolamide developed renal calcification, which should be recognized as a frequent complication (2).

The authors state that they have no Conflict of Interest (COI).

References

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