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1: [J Urol](#). 1985 Jul;134(1):11-9.

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Long-term treatment of calcium nephrolithiasis with potassium citrate.

[Pak CY](#), [Fuller C](#), [Sakhaee K](#), [Preminger GM](#), [Britton F](#).

The long-term effects of potassium citrate therapy (usually 20 mEq. 3 times daily during 1 to 4.33 years) were examined in 89 patients with hypocitraturic calcium nephrolithiasis or uric acid lithiasis, with or without calcium nephrolithiasis. Hypocitraturia caused by renal tubular acidosis or chronic diarrheal syndrome was associated with other metabolic abnormalities, such as hypercalciuria or hyperuricosuria, or occurred alone. Potassium citrate therapy caused a sustained increase in urinary pH and potassium, and restored urinary citrate to normal levels. No substantial or significant changes occurred in urinary uric acid, oxalate, sodium or phosphorus levels, or total volume. Owing to these physiological changes, uric acid solubility increased, urinary saturation of calcium oxalate decreased and the propensity for spontaneous nucleation of calcium oxalate was reduced to normal. Therefore, the physicochemical environment of urine following treatment became less conducive to the crystallization of calcium oxalate or uric acid, since it stimulated that of normal subjects without stones. Commensurate with the aforementioned physiological and physicochemical changes the treatment produced clinical improvement, since individual stone formation decreased in 97.8 per cent of the patients, remission was obtained in 79.8 per cent and the need for surgical treatment of newly formed stones was eliminated. In patients with relapse after other treatment, such as thiazide, the addition of potassium citrate induced clinical improvement. Thus, our study provides physiological, physicochemical and clinical validation for the use of potassium citrate in the treatment of hypocitraturic calcium nephrolithiasis and uric acid lithiasis with or without calcium nephrolithiasis.

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