V Edition of the Clinical Cases Contest on

non-surgical clinical management of Kidney Stones

Title: Idiopathic Nephrocalcinosis – Case Report

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1. Abstract:

Objective: To present a case of nephrocalcinosis secondary to hypercalciuria, in the absence of hypercalcemia, that was successfully treated using conservative measures, such has the administration of *Lit-control Up*® and a thiazide.
Method: Data and imaging related and relevant for this case was withdrawn from the *SClinic* platform.
Results: the patient began conservative treatment with the use of thiazide together with the administration of *Lit-control Up*® and oral water reinforcement. An imaging re-evaluation 6 months after showed a very favorable response, with almost complete resolution of nephrocalcinosis and no lithiasis. The 24-hour control urine analysis showed all parameters normalized.
Conclusions: This case shows the importance of a robust study of this type of patient and how through a conservative treatment which combines dietary care and the use of correct medication, it is possible to significantly reduce the lithiasic load, preventing progression to chronic kidney disease.

2. Introduction

Nephrocalcinosis refers to the generalized deposition of calcium in the renal medulla or renal cortex, less frequently in the form of both calcium oxalate and calcium phosphate, although it has been suggested in recent studies that calcium oxalate lithiasis should be assessed independently. It is caused by diseases that cause hypercalciuria, with or without hypercalcemia, or also hyperoxaluria, hyperphosphatemia or hyperphosphaturia, in the presence of an inappropriately low pH urine. This condition develops through different mechanisms, which interfere with the increased concentration of these salts in the urine, either by increasing their serum values or by stimulating their urinary excretion. At a microscopic level, nephrocalcinosis develops through the deposition of intracellular, tubular and interstitial calcium phosphate, accompanied by a predominant interstitial infiltrate of lymphocytes and interstitial fibrosis. Macroscopically, it can be identified by imaging. Nephrocalcinosis

is commonly associated with primary hyperparathyroidism, but also with other pathologies such as distal renal tubular acidosis, medullary spongy kidney disease, sarcoidosis, tumor lysis syndrome, acquired tubulopathies, as well as iatrogenesis, such as vitamin D supplementation or excessive use of diuretics that act on the loop of Henle.
The impact of nephrocalcinosis is that it can develop into chronic kidney disease if not treated in time. The main aim of treatment is to reduce the concentration of intra-renal calcium, depending on the underlying mechanism, with a lower rate of development of lithiasis. The case presented tells of a case of nephrocalcinosis secondary to hypercalciuria, in the absence of hypercalcemia.

3. Clinical Case description

a. Patient information / Medical records

We present the case of a female patient who was seen for an isolated episode of macroscopic hematuria, with no other associated complaints. The only antecedents reported were seronegative rheumatoid arthritis and being an ex-smoker. Of her usual medication, only methotrexate stands out.

b. Diagnostic support studies and results

A summary urine analysis was carried out, which showed a low pH and hematuria (3 erythrocytes/field). Urinary tract infection was ruled out. During the study of the hematuria, which included urethrocystoscopy and computed tomography of the urinary tract (uroCT), the presence of left renal nephrocalcinosis was incidentally detected (figure 1), associated with low-volume renal lithiasis. Urethrocystoscopy was innocent. In view of the findings, the study was aimed at identifying the cause of the nephrocalcinosis.

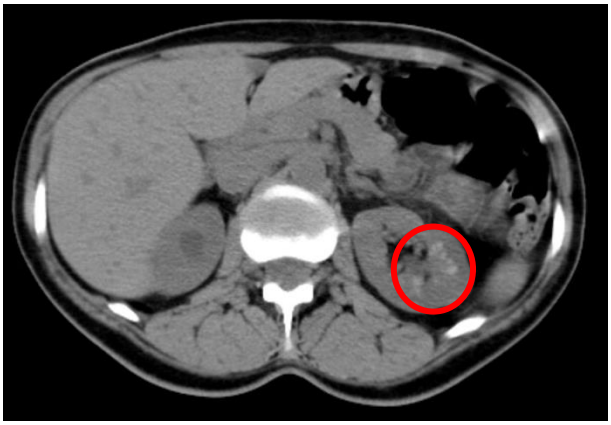

Figure 1 - Red circle shows deposition of calcium in the renal cortex, consistent with nephrocalcinosis

The 24-hour urine analysis showed hypercalciuria, hypomagnesemia and a pH of 5.5 (which rules out the possibility of distal renal tubular acidosis). Analytically, the other parameters of the study, particularly serum calcium and parathyroid hormone, were within normal parameters, ruling out primary hyperparathyroidism. Citrate was also within parameters.

c. Diagnosis

After excluding the main causes and the absence of previous pathologies, particularly genetic diseases, it was assumed to be nephrocalcinosis secondary to idiopathic hypercalciuria.

d. Treatment

It was therefore decided to begin conservative treatment with the use of thiazide together with the administration of *Lit-control Up*® and oral water reinforcement, with early imaging reassessment. This alkalinizing agent was chosen because its formulation includes not only protective agents for the

development of lithiasis found in other products available on the market, but also molecules such as Theobromine, an alkaloid, potassium citrate, which is important since thiazides can cause hypokalaemia, and magnesium citrate, which makes it possible to supplement this patient's vitamin deficit at the same time.

e. Evolution and progress

An imaging re-evaluation 6 months after starting the therapy showed a clear reduction, and almost no lithiasis (figure 2). The 24-hour control urine analysis showed all parameters normalized, with pH within the target rage (pH 6.2-6.8), and no magnesium deficit.

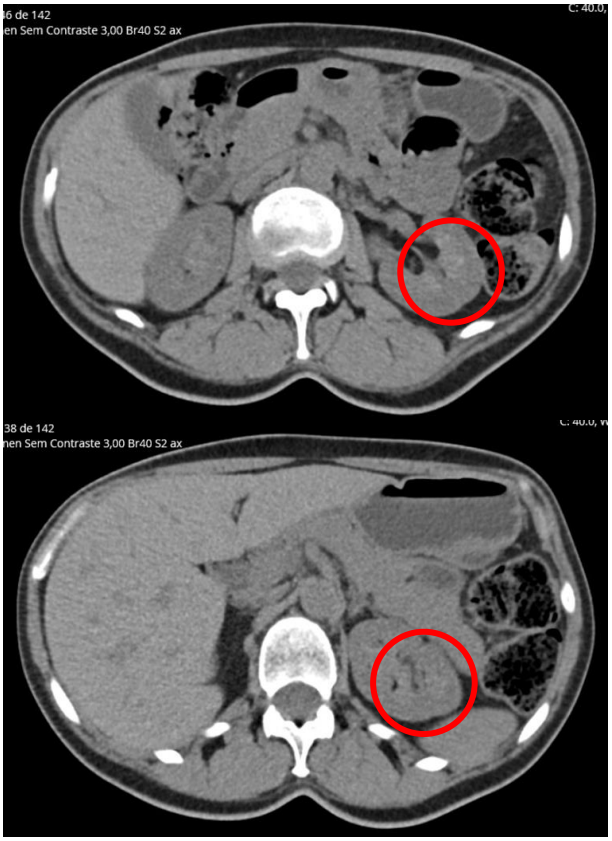

Figure 2 - imaging reassessment after 6 months of treatment, showing practically complete response with residual deposits of calcium

f. Clinical results

In view of the very favourable response, the decision was made to discontinue the thiazide and continue taking *Lit-control Up*® with urinary pH monitoring carried out by the patient herself.

4. Discussion

Nephrocalcinosis is characterized by the generalized deposition of calcium in the kidney. It is caused by diseases that interfere with the absorption and excretion of salts such as calcium (the most frequent mechanism), oxalate, citrate and phosphate. Roughly speaking, we can assign patients to 3 types of phenotypes: patients with hypercalciuria and hypercalcemia, hypercalciuria and normal calcium, and no elevation of calcium or citrate parameters. In this patient's case, hypercalciuria without hypercalcemia associated with a normal pH was identified, which ruled out two of the most frequent causes, HPT and ATR, respectively.

If not treated effectively, nephrocalcinosis can ultimately lead to chronic kidney disease. Ideally, treatment should be two-pronged: one aimed at the underlying pathology, with a view to reducing the volume of calciuria, and the other at preventing the formation of new lithiasic *foci*, by bridging molecular deficits and maintaining a normal pH. In this case, for which *Lit-control Up*® was administered, it was possible to correct the hypomagnesemia and normalize the pH value, with good results after 6 months of treatment.

5. Conclusions and recommendations

This case shows the importance of a robust study of this type of patient and how through a conservative treatment which combines dietary care and the use of correct medication, it is possible

to correct not only the ionic deficits but also significantly reduce the lithiasic load, preventing progression to chronic kidney disease.

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