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Renal lithiasis in a patient with primary hypothyroid-ism, case report

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Abstract

Introduction: Idiopathic hypercalciuria 35%, persistently acid urine 13.3%, hyperuricosuria 9.3%, hypocitraturia 5%, hypomagnesuria 4%, primary hyperparathyroidism 2%, hyperoxaluria 1.4% and cystinuria 1% are causes of most re-incidences. of kidney stones. A sporadic case of association between kidney stones and primary hypothyroidism not previously reported is presented.

Clinical case: This is a 16-year-old boy who debuted with kidney stones (2 stones) with recurrent episodes in the sixth month (1 single stone in the renal pelvis) that required lithotripsy and new episodes of subsequent microlithiasis. Additionally, the patient presented non-glomerular persistent isolated hematuria, alopecia, and intolerance to cold. Dietary alterations with low water intake, high sodium, and protein intake.

Diagnostic workshop: The patient was classified as a carrier of metabolically active stones. The metabolic study established the presence of hypercalciuria (calciuria > 140 and < 300 mg/day in men) dependent on diet (absorptive type II), with a calciuria/creatinuria ratio < 0.11mg/mg. Primary hypothyroidism was also diagnosed.

Evolution: With hydration and vegan dietary treatment, hydrochlorothiazide, citrate replacement, and levothyroxine, it has been possible to reduce calciuria and eliminate episodes of kidney stones to date.

Conclusion: Primary hypothyroidism should be considered as another cause of type II absorptive hypercalciuria. Hypothyroidism can be a cause of isolated hematuria.

Keywords:

MESH: Nephrolithiasis, Hypothyroidism, Adolescent, Hypercalciuria, Single-Case Studies as Topic.

Received: May 9, 2022
Acepted: September 3, 2022
Published: September 22, 2022
Editor: Dr. Franklin Mora Bravo.

Cite:
Silva S. Renal lithiasis in a patient with primary hypothyroidism, case report. REV SEN 2022;10(2):64-73. DOI: htpp://doi.org/10.56867/17

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Renal lithiasis is a disease characterized by the concretion of minerals present in the urine. Under favorable conditions, form a solid mass partially or completely obstructs the urinary tract. It constitutes a health problem, with a prevalence of 8.8% (95% CI 8.1%-9.5%) [1], with a prevalence in men of 10.6% (95% CI 9.4%-11.9%) vs 7.1% (95% CI 6.4% - 7.8%) in women. Regarding weight, there was a higher prevalence in obese individuals (11.2% [95% CI 10.0%-12.3%] vs 6.1% [95% CI 4.8-7.4], *P* <0.001). in people with normal weight. Hispanics develop a higher risk of kidney stones than other ethnicities (OR: 0.60 [95% CI 0.49-0.73]) vs non-Hispanics (OR: 0.37 [95% CI 0.28-0.49]), P < 0.001 [1].

In a cross-sectional study carried out in our country, a prevalence of renal lithiasis of 9.43% (CI 9.07%-9-79%) was determined, with an incidence of 33.33% among the age groups of 48 to 85 years, and lithiasis due to elevated calciuria was present in 78.26%, with no correlation between age and calciuria levels. (*P*=0.34) [2], a study conducted in the highlands of Ecuador. However, the incidence of renal lithiasis also seems to be conditioned by environmental factors since a study carried out on the Ecuadorian coast (Guayaquil) determined the casuistry of lithiasis in patients treated in the emergency service of a state reference center, which determined an incidence of 22.3% (CI95% 22.2%-22.47%) [3].

The incidence of urolithiasis is so common that a first episode is not considered sufficient to start a metabolic study; the reported recurrence rates are 11%, 20%, 31%, and 39% at 2, 5, 10, and 15 years, respectively [4].

In the chemical structure of stones, there is a higher incidence of calcium oxalate (67%), followed by hydroxyapatite (16%), uric acid (8%), struvite (3%), brushite (0.9%), and cysteine (0.35%) [5].

The simple metabolic alterations that cause the majority of recurrences are idiopathic hypercalciuria (35%), persistently acid urine (13.3%), hyperuricosuria (9.3%), hypocitraturia (5%), hypomagnesuria (4%), primary hyperparathyroidism (2%), hyperoxaluria (1.4%) and cystinuria. 1% [6]. The combined incidences of metabolic disturbances were hypercalciuria plus hyperuricosuria in 35% and hypercalciuria plus hypocitraturia in 9.4%. Hypercalciuria plus hyperuricosuria occurs in 6.8% of patients [6]; thus, hypercalciurias make up the majority of the pathophysiology. of kidney stones, the most relevant being absorptive hypercalciuria (type I and II), renal-type hypercalciuria (elevated PTH) and fasting hypercalciuria (normal to low PTH) according to the classification of Pak C. et al. [7]. Complementary to this classification is the one described by Bataille P. et al. in diet-independent hypercalciuria (idiopathic or absorptive hypercalciuria type I) and diet-dependent hypercalciuria equivalent to absorptive type II of the Pak et al. classification [8].

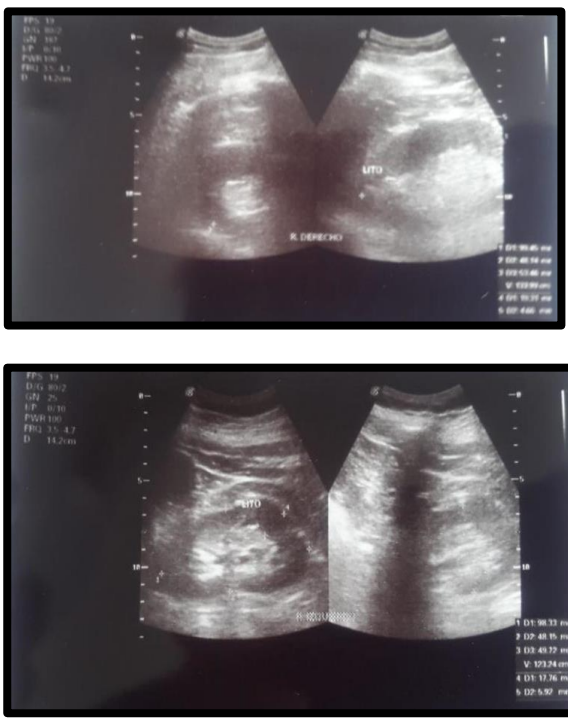

Figure 1. Ultrasound prior to lithotripsy and initiation of metabolic treatment for lithiasis, the presence of kidney stones in lower and middle calyces is observed. Top image: right kidney, bottom: left kidney..

Treatment should generally be multidisciplinary in coordination with urology, nutrition, and endocrinology and is based on calcium-sparing diuretics (hydrochlorothiazide), healthy habits, a diet low in protein and salt, and plenty of fluids and without calcium restriction in the case of hypercalciurias [9]. Other measures are added depending on the LRMS result, stone chemistry analysis, or associated comorbidities [9].

Until now, there have been no association studies between hypothyroidism and recurrent renal lithiasis; however, the association between thyroid dysfunction (hypo- and hyperthyroidism) and its effects on renal physiology (at the prerenal and renal levels) has been described [10]. A sporadic case of this previously unreported association is presented.

Clinical case

Background and clinical history

The present case is of a 17-year-old man with a history of trauma to the right lower limb with a closed tibia and fibula fracture at 13 years of age; the fracture was surgically reduced. On one occasion, he had isolated urinary infections during his childhood

with mild proteinuria (150 to 300 mg/24 hours), which was attributed to the presence of a urinary infection. In adolescence, he was diagnosed with Gilbert's syndrome. The first episode of renal lithiasis presented at the age of 16. Bilateral kidney stones were diagnosed, and a metabolic study for kidney stones (LRMS) was not performed (Figure 1). He was prescribed amiloride 2.5 mg + hydrochlorothiazide 25 mg every day.

Family history and habits

He had a history of kidney stones in his paternal grandfather and a paternal uncle. Within the eating habits, the intake of > 1 liter of milk per day, the intake of 1500 ml of water/day, and a large intake of proteins, including red meat and sausages, additionally referred to a habit of eating few fruits and vegetables.

Physical exam

There were no relevant data on physical examination. The patient's blood pressure was normal for his age: 110/70 mmHg, pulse 65 per minute. She had normal body development for her age, adequate height and weight percentiles, 56 kg, and a size of 1.78 m. She had a body mass index of 19.8 kg/m^2.

Evolution in the first month of diagnosis

One month after the first control, he did not present renal colic or urinary infection. The simple MRLS diagnostic workshop diagnosed borderline hypercalciuria, hypernatriuria, and hypovitaminosis D (Tables 1 and 2). It was decided to continue with amiloride 2.5 mg + hydrochlorothiazide 25 mg, and oral vitamin D, a low sodium diet, and adequate hydration with > 3 liters per day were added.

Evolution in the sixth month

Six months after the diagnosis, he was referred from the pediatric nephrology service to the clinical nephrology hospitalization service for coming of age, with an episode of hematuria and new renal colic.

Diagnostic workshop

General urinalysis confirmed the presence of isolated hematuria with the absence of proteinuria. A complete EMLR was performed (Tables 1 and 2 and Figure 3), the result of which was borderline absorptive hypercalciuria type II, hypomagnesuria, persistent acid urine, and hypocitraturia with the persistence of microlithiasis in more significant number and size than that reported in an ultrasound. (the most oversized measured 3 mm), However, the stone was only located in the right renal pelvis on this occasion. He was prescribed the necessary water intake to maintain diuresis of 2.5 liters per day, potassium citrate 4 mEq/day (300 mg sachet, with 8 mEq per sachet, ½ sachet each day), magnesium citrate 1 g/day and hydrochlorothiazide plus amiloride 50 mg daily, vitamin D in the form of calcidiol 10,000 IU daily for three months. He referred to nutritional control to correct the intake of sodium, water, and protein in an acceptable way. Extracorporeal shock wave lithotripsy (ESWL) was planned in an outpatient urology clinic, which was able to remove stones located in the renal pelvis. The post lithotripsy control ultrasound showed the absence of stones.

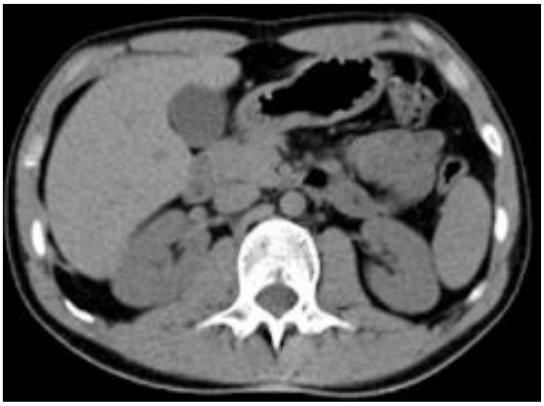

Figure 2. Abdominal and renal computerized axial tomography without bilateral stones (2/10/2021).

Subsequent controls

In a new six-month check-up, the patient reported the absence of renal colic; however, microscopic hematuria persisted in the urine study. It was verified that the patient had better adherence to the diet with weight loss and better hydration, and there was less consumption of sodium and protein in the diet. The 24-hour urine analysis reported normal urinary pH and the presence of borderline uricosuria with normal calciuria. It was planned to continue with potassium citrate at a dose of 16 mEq/day (2 Sachets of 300 mg per day), magnesium citrate 1 tablet per day of 1000 mg, and hydrochlorothiazide 50 mg per day.

Recurrence of the third frame of renal lithiasis

In the teleconsultation control during the COVID-19 pandemic, eight months after the last consultation, the patient presented a loss of adherence with less control in the diet: the patient increased the consumption of sodium, proteins, and dairy products with

Table 1. Follow-up of laboratory results

	06/13/2013	08/24/2018	05/11/2019	02/22/2020	03/12/2020	10/13/2020	05/03/2021	*06/09/2021	*01/25/2022
Hct (%)	33	45.2		47		43		46	44
Hbg /dL	10.9	fifteen		17		15.4		fifteen	14
Leukocytes u/uL	11,600	6,400		5,300		6,890		7,520	6,080
Urea mg/dL		25	26	33					
Creat mg/dL		1.2	0.9	0.8		0.8	0.75	0.76	0.7
pH			5.9		7.3				
HCO 3mmol /L					28				
CO 2 µg/m3					29.7				
Glucose mg/dL		95			80	87	97		
Uric acid mg/dL		5.5	6.7		6.2		5		4.3
OGT U/L		23		33	26	18	13		
TGP U/L		18		27	25	14	12		
F alkaline U/L				225	197	211	190		
Bill. Total mg/dL		1.1		2.5	1.1	0.65	1.5		
Bill. Add mg/dL		0.5		0.75	0.48	0.3	0.3		
Albumin g/dL							4.3		
NamEq /L		140		134	143	136	13 5	138	139
KmEq /L		4	4.3	3.6	3.6	4.3	3.5	3.8	3.6
Clmmol /L		104		100	102	100	100	100	100.6
Calcium mg/dL		9.8	10	8.4	9.5	9	10	8.7	
Phosphorus mg/dL			3.8		3.4		4.8		
Mgmmol /L					1.8		two		
PTH pg/mL		44					48		
Col. T mg/dL						152	102		116
HDL mg/dL						23			
LDLmg /dL						128			
trimg /dL						54	83		60
DV ng/mL		twenty-one			21.7		13		
TSH mIU/L							7.7		2.7
T4 mIU/L							6.6		10.9
C3 mg/dL								124	
C4 mg /dL								37	
CRP mg/L									
Hep B Ags IU/L								negative	
HCV IU/L								negative	
HIV IU/L								negative	

* under treatment with levothyroxine (T4) 100 mcg daily. Hct: hematocrit; Hb: hemoglobin; creat: creatinine, ph: blood pH; HCO3: Bicarbonate, Co2: hydrogen dioxide, TGO: glutamic oxalacetic transaminase, TGP: glutamic pyruvic transaminase, Fal: alkaline phosphatase, bile.T: total bilirubin, bile.d: direct bilirubin, Album: albumin, Na: sodium, K: potassium, Cl: chlorine, Mg: magnesium, Col. T: total cholesterol, Hdl: high-density lipoprotein, LDL: low-density lipoprotein, Tg: triglycerides, Vit D: vitamin d 25OH. TSH: thyroid stimulating hormone, T4: thyroxine. C3-C4: complement fractions C3-C4. CRP: c-reactive protein; HCV: hepatitis C; HBV Ags: hepatitis B surface antigen; HIV: HIV, PTH: parathyroid hormone.

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Increase weight by 3 kg (59 kg). Renal ultrasound revealed unilateral renal microlithiasis in the right pelvis. A simple EMLR study was performed in which borderline calciuria persisted. It was planned to resume the change in eating habits and continue with magnesium citrate and the same medication prescribed in the previous control.

Alopecia and cold intolerance

Between January and June 2021, the patient reported the presence of abdominal colic accompanied by diarrhea and fever, for which he went to the emergency room of the institution. Possible bacterial and viral infectious causes were ruled out, including SARS-CoV-2. The colic was attributed to the gastrointestinal system; however, isolated hematuria was demonstrated on two occasions. Despite complying with the suggested diet, the patient continued with borderline hypercalciuria. Due to a secondary effect of dyspepsia related to the use of potassium citrate and magnesium citrate, this medication was discontinued. The prescription of hydrochlorothiazide 75 mg plus amiloride 5 mg/day and a strict diet were maintained.

Isolated hematuria was studied separately as a possible glomerular disease, requesting the identification of acanthocytosis, serology for hepatitis C, hepatitis B, HIV, antinuclear antibodies, complement, and inflammatory markers such as CRP. During this period, the patient developed intolerance to cold and diffuse alopecia, for which thyroid function tests were requested.

After a month of control, the diagnosis of hypothyroidism was established with a TSH of 7.7 mIU/L. Levothyroxine 1.6 mcg/kg/day (100 mcg/day) was prescribed, and she was referred for further follow-up with endocrinology. Additionally, there was an episode of nontolerance to hydrochlorothiazide with the presence of cramps and hypotension, so the dose was lowered to 50 mg per day.

Table 2. Simple and complete metabolic study of kidney stones (EMLR)

	08/24 2018	*02/14/2019	*02/15/2019	**02/20/2019	03/12/ 2020	10/13/ 2020	12/22/ 2020	05/03/ 2021	&*06/09/ 2021	&01/25/ 2022
Diuresis 24 h (ml)	2300	1345	2218	2800	2100	2100	2650	2000	1800	2050
Creatininuria (mg/ml)	1100	990	1100	1230	1000	890			871	807
Clearance creat. (ml/min)	41	106	106		83.6				79.6	78
Sodium U. (mEq/24h)	287	100	204	230	97	123	98	129	209	60
Uric acid U. (mg/24h)	526	473	432		449	416	465	316	282	243
Calcium U. (mg/24h)	250	187	201	100	133	85	269	180	2.7	90
Calcium index./create	0.22	0.18	0.18	0.08	0.13	0.09			0.003	0.11
Magnesium U. (mg/24h)	30	39	30	30	48	65	72	78	89	
Proteins U. (g/24h)	184	0.05	0.05		0.04	0.08				0.02
Oxalate U. (mg/24h)		27	26.6							0.02
Cystine U. (mg/24h)		0	0							
Citraturia U. (mg/24h)		90	110						600	
Ph U.	5	5.6	5.5		6	6	6	6.5	6	
Urinary red blood cells		7	6			8	7	7	9 – NA	
Density U		1015	1020			1015	1025	1025	1015	
Appearance		yellow	yellow			yellow	yellow	yellow	yellow	
Sediment protection	2+	black	black			traces	traces	traces	traces	

* Full EMLR. **Simple EMRL after a restricted diet for five days, calculated with a patient's weight of 59 kg on a 5-day diet without dairy intake, with 100 meq of sodium and <1 g of animal protein. & EMRL in treatment with levothyroxine (T4) and suspended his treatment for hypercalciuria, pH: urinary pH, U: urinary. ND: Nondysmorphic

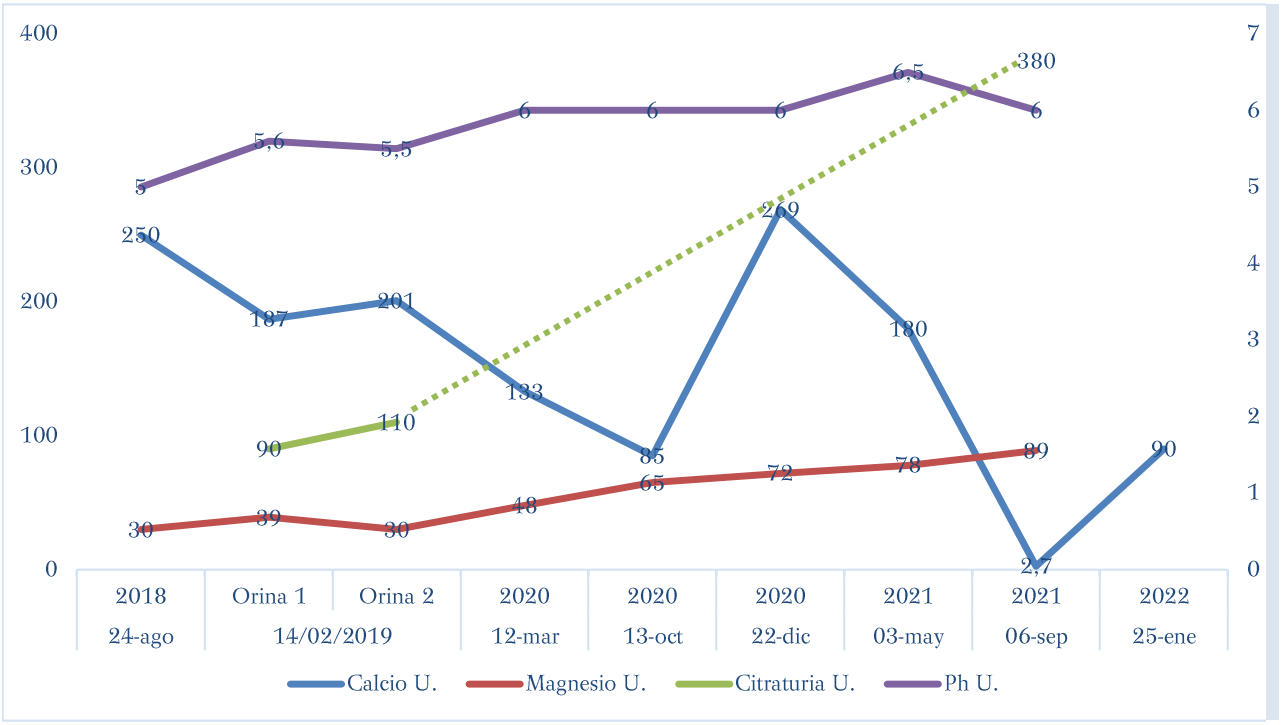

Figura 3. Cambios en el EMLR durante el tratamiento.

The control was carried out in November 2021; the patient had maintained a strict vegan-type diet and had not presented new episodes of renal colic but had an episode of macroscopic hematuria. On this occasion, glomerular hematuria (negative acanthocytosis) was ruled out, and compliment, serology, and inflammatory markers were typical. The patient also showed improvement in his energy and mood after starting levothyroxine, which was maintained at the same initial dose by endocrinology with current average TSH values. Routine thyroid ultrasound in which nodules or lesions were ruled out. The established diagnosis was primary autoimmune hypothyroidism (Hashimoto's thyroiditis). The 24-hour urine analysis reported good hydration, a salt intake of 2 g per day, and normal calciuria. Renal tomography was standard (Figure 2).

The final diagnosis was unilateral renal lithiasis of multifactorial cause due to borderline absorptive hypercalciuria type II, hypomagnesuria, and hypocitraturia with primary hypothyroidism (Hashimoto's thyroiditis).

Discussion

The main lesson of this case is the importance of EMLR monitoring in a patient with renal lithiasis with a finding of hypothyroidism, an unusual association. The patient was classified as a recurrent stone former with recurrent episodes of colic and more than one stone by imaging study as demonstrated at disease onset. The patient was not described as a carrier of stones with an aggravating or high-risk factor since this group includes patients with kidney stones in children under 12 years of age, uric acid or cystine stones, patients with only one kidney, anatomical abnormalities of the urinary tract, immunocompromised, or jobs involving increased dehydration, for example, cooks [6, 11]. From the beginning of the disease, the patient was recommended to undergo complete EMLR before the first episode of renal colic since he had two stones at debut. Other indications for performing EMLR before the first stone are the presence of systemic diseases such as chronic kidney disease, primary or secondary hyperparathyroidism, intestinal surgeries, chronic intestinal inflammatory syndromes, diabetes, gout, and obesity with or without high-risk factors [11], pathologies that were ruled out in the present case.

Within the classification of lithiasis for follow-up, the patient was a metabolically active lithiasis carrier because, despite the treatment implemented, the stones persisted in more significant numbers at the level of the right renal pelvis by ultrasound. If the goal of treatment is to convert patients with metabolically active stones (increase in size or number of stones by imaging study) to metabolically inactive, in this case, ESWL was used to fracture the single recurrent stone located in the renal pelvis in the control at the sixth month of follow-up.

In all cases, EMLR was performed on an outpatient basis, and at least one month after the stone episode, considering the recommendations, it was also ruled out that the patient had a urinary obstruction. Ideally, follow-up should be established every three months [11].

The biochemical study of the kidney stone obtained also adds much value for treatment and diagnosis since the type of metabolic disease can be determined regardless of whether EMLR is performed; therefore, patients should be encouraged to rescue the expelled stone, although this is more difficult and rare to perform, as in the present case [12].

Within the metabolic study, the presence of borderline hypercalciuria was established (calciuria > 140 and < 300 mg/day in men), dependent or absorptive diet, by a calciuria/creatininuria index < 0.11 mg/mg or calciuria < 3 mg/kg in voiding urine of the fasting and <200 mg of calcium in 24-hour urine after a 4- to 7-day diet without dairy, with 100 mEq of sodium and <1 g of animal protein [7, 8].

Among absorptive hypercalciurias is type I (urinary excretion > 200 mg of calcium in 24-h urine) and absorptive type II (normal calcium excretion < 200 mg/day) after a calcium-restricted diet [6, 7].The patient presented type II absorptive hypercalciuria.

Within the etiology of absorptive hypercalciurias are increased intestinal reabsorption of calcium secondary to increased serum calcitriol; this occurs in 30% of cases. Another cause is the increased number of calcitriol receptors in the intestine and bone (demonstrated in rats), and finally, there may also be an increase in the sensitivity of calcium receptors at the same level, all this in an idiopathic or genetic way [6, 7, 12].

Despite adequate treatment, with the initiation of a calcium-sparing diuretic (hypercalciuria), magnesium citrate (hypomagnesuria), potassium citrate (acid urine and hypercalciuria), vitamin D2 (vitamin D deficiency), diet adjustment (good adherence, he even became a vegetarian) and control with urology, the patient persisted as metabolic activity (isolated hematuria always together with episodic renal colic and persistence of microliths by echo).

Hematuria was also studied, which was isolated non-dysmorphic, which ruled out the presence of glomerulopathies or autoimmune diseases at this time, including IgA nephropathy. However, in subsequent controls in the presence of mild proteinuria, a renal biopsy would be indicated to rule out this clinical suspicion.

Simple EMLR confirmed a correction of the alterations previously described, except for borderline hypercalciuria, which led us to ask ourselves if another factor was added that contributed to the unsuccessful treatment, forcing us to more deeply study other causes, such as those described in the clinical case. A diagnosis of primary hypothyroidism was achieved due to Hashimoto's thyroiditis, which after being treated, showed a notable improvement in his symptoms with correction of his calciuria by a new simple EMLR, which raised the hypothesis of the influence of the dysfunctional thyroid gland on his lithiasis.

Currently, there are no cases describing patients with hypothyroidism and its influence on renal lithiasis; however, the relationship between an adequate function of thyroid hormones and normal renal development and growth is well established [13, 14].

Hypothyroidism produces a decrease in the glomerular filtration rate (GFR) in 55% of patients who suffer from it and less frequently in autoimmune hypothyroidism; this is secondary to a decrease in renal blood flow (RBF) by a prerenal mechanism by lowering cardiac output due to a negative inotropic and chronotropic effect, increased peripheral vascular resistance, including intrarenal (due to decreased B adrenergic receptors) and due to less vascular response to vasodilators, due to less production of the factor vascular endothelial growth factor (EVGF) and insulin-like growth factor 1 (IGF-1) [13, 14].

In addition to prerenal damage, there is also direct renal damage at the glomerular level, producing thin basement membrane nephropathy, mesangial proliferation, and increased glomerular capillary permeability, which can generate proteinuria [13]. In some cases, there may even be a reversible decrease in kidney mass [13].

Finally, there may be damage at the renal tubular level, generating a decrease in the absorption of electrolytes and water at the level of the proximal tubule, secondary to a decrease in the expression of the chloride channel (Cl. C) at the basolateral level, decreasing its absorption and increasing the amount of chloride delivered at the distal level, causing a decrease in renin secretion and therefore a decrease in RAAS activation, which contributes to a more significant decrease in GFR [13, 14].

The transport capacity of Na/K channels is decreased by a reduction in Na/K ATPase activity at the proximal level and all sectors of the nephron, generating greater excretion of sodium, potassium, and urinary calcium. Finally, there is also a decrease in the activity of the sodium/hydrogen channels (NHE), causing a decrease in their excretion, which generates defects in the acidification of the urine [13, 14].

All this generates hypotonicity in the medullary interstitium and, therefore, an inability to concentrate urine in the long term. However, in the short term, this is compensated by an increase

in the sensitivity of aquaporin receptors to antidiuretic hormone (ADH) and an increase in its secretion (secondary to the activation of the carotid baro receptors due to low cardiac output generated by hypothyroidism), promoting more significant reabsorption of water at the distal level, resulting in hypo-osmolar hyponatremia due to fluid retention at the systemic level and a trend toward higher urinary concentration at the urinary level [13, 14].

The changes are reversible with hormone replacement by supplying T4 hormone; that is, the physiological functions of the kidney at the prerenal, renal and tubular levels return to normal [13, 14].

In the case of the present patient with kidney stones, primary autoimmune hypothyroidism could have contributed to the formation of more concentrated urine with a higher content of sodium and calcium due to decreased tubular reabsorption, which justifies that despite the prescribed treatment and good adherence to the diet, borderline calciuria was not improved. It improved after treatment with T4 (levothyroxine).

On the other hand, the influence of hypothyroidism at the glomerular level may explain the isolated nondysmorphic hematuria experienced by the patient in the context of probable thin basement membrane nephropathy (not biopsied).

Last, no changes could be observed at the prerenal level, such as a decrease in GFR or the presence of hyponatremia, since it is a cause of autoimmune hypothyroidism, which makes it unlikely.

Conclusions

Knowing when and which patients to carry out a complete metabolic study of renal lithiasis is essential to provide a more efficient diagnosis and treatment to patients who suffer from it; for this, it is necessary to know if there are systemic risk diseases, high-risk factors for lithiasis and classifying patients into recurrent stone formers or sole formers to determine recurrence and metabolically active or metabolically inactive patients to facilitate monitoring and treatment modification. Hypercalciuria is the most common metabolic cause (single or combined) of kidney stones, so it is essential to know their classification and etiology to avoid errors in their diagnosis. Although there are no reported cases of hypothyroidism and its relationship with kidney stones, the influence of thyroid hormones on renal development, growth, and physiology must be considered. Hypothyroidism could contribute to a lithogenic state through increased urinary sodium/calcium excretion and more concentrated urine. At the glomerular level, hypothyroidism can cause isolated nondysmorphic hematuria and different degrees of proteinuria. The effects of hypothyroidism on the kidney are reversible and improve with appropriate treatment (administration of T4).

Abbreviations

EMLR: Metabolic study in kidney stones.
Hct: hematocrit.
Hb: hemoglobin
creat: creatinine
pH: blood pH;
HCO3: Bicarbonate
CO2: hydrogen dioxide,
GOT: glutamic oxalacetic transaminase,
GPT: glutamic pyruvic transaminase,
Fal: alkaline phosphatase,
bil. T: total bilirubin,
bilir.d: direct bilirubin,
Album: albumen,
Na: sodium.
K: potassium
Cl: chlorine
Mg: magnesium
Col. T: total cholesterol
HDL: high-density lipoprotein
LDL: low-density lipoprotein,
Tg: triglycerides,
Vit D: vitamin d 25OH.
TSH: thyroid stimulating hormone,
T4: thyroxine.
C3-C4: complement fractions c3-c4.
CRP: c-reactive protein;
HCV: hepatitis C.
HBV Ags: hepatitis B surface antigen.
HIV: HIV.
PTH: parathyroid hormone.

Supplementary information

Supplementary materials have not been declared.

Acknowledgments

Does not apply.

Author contributions

Santiago David Silva Tobar: conceptualization, data curation, formal analysis, fund-raising, research, methodology, project management, resources, software, writing - original draft, supervision, validation, visualization, writing: review and editing.
All authors read and approved the final version of the manuscript.

Financing

The author provided the expense of the investigation. Laboratory studies and metabolic measurements were performed as part of the regular activity of the nephrology service and did not represent an additional cost to the owners or the patient.

Availability of data or materials

The data sets generated and analyzed during the current study are not publicly available due to participant confidentiality but are available from the corresponding author upon reasonable academic request.

Statements

Ethics committee approval and consent to participate

Not needed.

Consent to publication

We have written permission from the patient's guardians to publish this case.

Conflicts of interest

The author reports having no conflicts of interest.

Author Information

Santiago Silva is a physician from the Central University of Ecuador (Quito, 2011). Medical specialist in nephrology and internal medicine from the CEMIC University Institute & Central Polyclinic of the Metallurgical Workers' Union (UOM) (Buenos Aires; 2022). Treating Nephrologist at the General Teaching Hospital of Ambato.

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