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Case Report

# Episodic Hematuria in a Young Boy - Do Not Miss Familial Idiopathic Hypercalciuria

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## ABSTRACT

Familial hypercalcemia is an important cause of episodic hematuria in children and can lead to nephrolithiasis if untreated. Elucidation of proper family history and screening of family members can help in clinching the diagnosis. An 8-year-old boy presented with episodic painless hematuria for 3 years, with a history of small renal stones in parents, which was managed conservatively. Ultrasound showed concretions in the renal pelvis, and urinalysis revealed hypercalciuria in the absence of renal tubular acidosis in index case as well as parents. He was started on thiazides, oral citrate supplementation, and adequate hydration, following which he became asymptomatic.

***Keywords***: *Hematuria; Hypercalciuria; Nephrolithiasis.*

## INTRODUCTION

Familial idiopathic hypercalciuria is a common cause of painless hematuria and nephrolithiasis but is often missed. Diagnosis can be made by taking a proper family history and appropriate screening of family members, and unnecessary investigations can be avoided.

## CASE REPORT

An 8-year-old boy presented with episodic painless hematuria for the past 3 years, which were aggravated by stress and dehydration. There was no history of bleeding diathesis, trauma, and transfusion of blood component, the passage of stones in urine or past surgery. Systemic examination was unremarkable.

Hemogram (Hb – 11.6 g/dl, TLC – 9800/cu mm, Platelet – 382000/cu mm), coagulogram (PT – 13.6", INR – 1.03, aPTT – 28.4"), electrolytes (Na – 144, K – 4.5, Cl – 110) and renal function (Urea – 12 mg/dl Creatinine – 0.53 mg/dl) were within normal limits. Serum calcium (9.2 mg/dl), phosphorous (4.3 mg/dl), magnesium (2.45 mg/dl), 25-hydroxycholecalciferol (32.2 ng/ml) and parathormone (37.02 pg/ml) were normal. There was no evidence of metabolic acidosis (pH – 7.418 HCO3 – 22.6 meq/l). Urinalysis revealed pH – 6.5 with calcium oxalate crystals, and no evidence of hematuria, proteinuria, pyuria or glycosuria. Urine culture was sterile. Furosemide – fludrocortisone challenge test did not reveal any evidence of distal tubular dysfunction. Calcium creatinine ratio (0.25) and 24-hour urinary calcium (4.12 mg/kg/day) were elevated. Ultrasound showed normal-sized kidneys with multiple subcentrimetric concretions in bilateral kidneys. 24-hour urinary oxalate (14 mg/24 hr) and citrate (2.44 mmol/24 hr) were normal. Evaluation of parents showed hypercalciuria (24-hour urinary calcium 4.32 mg/kg/day and 4.21 mg/kg/day) with sociological evidence of multiple subcentrimetric renal concretions.

Oral hydrochlorothiazide was started in both index child and his parents and was advised to drink adequate water (more than 2 litres) daily, along with dietary salt restriction. Child has been asymptomatic ever since, and there has been no recurrence of hematuria. 24-hour urinary calcium on follow-up was less than 4 mg/kg/day.

## DISCUSSION

Idiopathic hypercalciuria is a common cause of formation of calcium stones in the kidney and is defined by urinary calcium excretion of more than 4 mg/kg/day along with normal serum calcium levels.[1] It is usually inherited in an autosomal dominant pattern and is the commonest cause of nephrolithiasis in children. Secondary causes like renal tubular acidosis, sarcoidosis, vitamin D excess, hyperparathyroidism, Paget's disease, glucocorticoid excess, and malignant bone tumors should be ruled out.[2,3]

If untreated, 15% of patients may end up having nephrolithiasis, and it has been considered responsible for 40% of cases of renal calculi.[4] In the absence of renal stones, recurrent gross or microscopic hematuria, dysuria or lower abdominal pain can be the presenting features.[5]

Diagnosis is established by documenting 24-hour urinary calcium more than 4 mg/kg; urinary calcium: creatinine ratio >2 can serve as a useful screening test.

Management includes adequate oral hydration (at least 1500-2000 ml/1.73 m2 body surface area per day).

Oral citrate supplementation can help reduce hypercalciuria by more than 30%. The daily dose is usually 0.3-0.6 mmol/kg body weight, in sodium or potassium-based preparation. Monitoring of urine pH is essential, as alkaline urine can lead to precipitation of calcium phosphate.[6]

Severe hypercalciuria is treated with thiazide diuretics, which increase calcium reuptake in the distal tubule, as well as improve reabsorption in the proximal tubule. It is usually given as a single morning dose of 1-2mg/kg/day, titrated upwards until urinary calcium is less than 4 mg/kg/day.[7]

In conclusion, hypercalciuria is an important differential of painless hematuria in children, and detailed family history is often a soft clue. Treatment should always be initiated in the form of hydration and oral medication to prevent recurrence as well as subsequent lithiasis

## CONFLICTS OF INTEREST

We have no conflicts of interest with nobody and have nothing to declare.

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