Open Journal of
Clinical & Medical
Case Reports

Volume 8 (2022)
Issue 02

ISSN: 2379-1039

Idiopathic hypocalcemia with hypercalciuria: A case report

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Abstract

Idiopathic renal hypercalciuria can cause a deficit in serum calcium and thus lead to osteoporosis. Earlier diagnosis and management are necessary to prevent recurrence of stone formation and reduce morbidity associated with the condition. We present to you a case of a 61-year-old male with a past medical history of osteopenia, kidney stones, gout, osteopenia, and chronic kidney disease stage 3. We diagnosed him with idiopathic hypercalciuria, and he was put on hydrochlorothiazide and a low-sodium diet. His hypercalciuria was controlled and serum biochemistry normalized. An earlier diagnosis of Idiopathic hypercalciuria (IH) may lead to earlier treatment intervention and decreased loss of bone density. However, increased awareness regarding this condition is necessary and will lead to prompt diagnosis and earlier screening of the relatives and sooner access to genotyping.

Keywords

Hypercalciuria; Hypocalcemia; Polyendocrinopathy; Hypoparathyroidism; Nephrolithiasis.

Introduction

Hypocalcemia is usually a tell tale sign of hypoparathyroidism. Hypoparathyroidism can occur solely or as a component of autoimmune Polyendocrinopathy. The autosomal dominant disorder occurs when there is a mutation in the calcium-sensing receptor (CaSR) leading to increased urinary calcium and decreased serum calcium [1,2]. Hypocalcemia can cause muscle contractions and limb numbness [3]. Hypercalciuria on the other hand can lead to nephrolithiasis and nephrocalcinosis. Calcium oxalate stones are the most common type of kidney stone and are mainly composed of calcium oxalate. In contrast, some calcium phosphate stones are either made of brushite or hydroxyapatite. Pharmacological and nutritional management is necessary to prevent new stone formation. Restricting the salt intake and increasing the fluid intake will decrease the risk of stone formation. Thiazide diuretics decrease urinary calcium and hence dietary management includes increasing fluid intake, salt restriction, animal protein restriction, and

maintaining a normal calcium intake. Thiazide diuretics have proven effective in preventing calcium stone formation by reducing the urinary excretion of calcium.

We describe a patient with idiopathic hypocalcemia with hypercalciuria associated with polyuria, polydipsia, and nephrocalcinosis. A detailed metabolic evaluation of this patient suggested idiopathic hypocalcemia with hypercalciuria.

Case Presentation

A 61 year old male presented to the clinic for follow-up of his renal function evaluation. He had a past medical history of osteopenia, kidney stones, gout, hypogonadism, left renal cyst, osteopenia, and chronic kidney disease stage 3. The patient was taking Allopurinol 300 MG Tablet 1 tablet Orally Once a day, AndroGel 25 MG/2.5 GM (1%) Gel 2 packets Transdermal Once a day, Vitamin D (Cholecalciferol) 1000 UNIT Capsule 1 capsule Orally Once a day, Calcium 500 MG Tablet 1 tablet with meals Orally once a day, Vitamin C 500 MG Tablet Chewable 2 tab Orally Once a day, Vitamin B12 TR 1000 MCG Tablet Extended Release 1 tablet Orally Once a day. He was a nonsmoker and did not drink alcohol. The patient denied any new urinary complaints. His physical examination was unremarkable. A basic metabolic profile is shown in Table 1.

Table 1: Basic Metabolic Profile.

Blood urea and nitrogen (BUN)	15	7-25 (mg/dl)
Creatinine	0.97	0.70-1.33(mg/dl)
eGFR	85	> OR = 60 (mL/min/1.73m2)
Sodium	142	135-146 (mmol/L)
Potassium	3.7	3.5-5.3 (mmol/L)
Chloride	104	98-110 (mmol/L)
Carbon dioxide	30	20-32 (mmol/L)
Calcium	9.4	8.6-10.3 (mg/dL)

His vitamin D levels were 34 ng/mL, parathyroid hormone levels were 36 pg/mL. His urinalysis is shown in Table 2.

Table 2: Urinalysis.

	Value	Reference Range
Color	Yellow	Yellow
Appearance	Cloudy	Clear
Bilirubin	Negative	Negative
Ketones	Negative	Negative
Specific Gravity	1.014	1.001-1.035
Occult Blood	Negative	Negative
PH	7.5	5.0-8.0
Protein	Negative	Negative
Nitrite	Negative	Negative
Leukocyte Esterase	Negative	Negative
Glucose	Negative	Negative

His stone risk diagnostic profile showed hypercalciuric nephrolithiasis, hyperoxaluric, hyperuricosuria nephrolithiasis is shown in Table 3.

Table 3: Stone risk assessment 24 hour urine.

Total urine	1.99 Liter	>2.00 Liter a day
Ph urine	6.5	5.5-7.0
Calcium,24-hour urine	408	<250.0(mg/day)
Oxalate,24-hour urine	83	<45(mg/day)
Uric acid,24-hour urine	706	<700(mg/day)
Calcium oxalate	5.80	<2.00
Brushite	4.86	<2.00
Sodium Urate	1.87	<2.00
Struvite	4.01	<75.00
Uric acid	0.54	<2.00

There was no evidence for renal tubular acidosis. We diagnosed him with idiopathic hypercalciuria, and he was put on hydrochlorothiazide 12.5 mg once a day and a low-sodium diet. His hypercalciuria was controlled and serum biochemistry normalized. He is being followed up regularly at the clinic with repeat renal function tests, bone mineral density and education to adhere to a normal level of calcium intake, low protein, low salt, and high fiber diet.

Discussion

Hypocalcemia and hypercalciuria are usually attributed to hypoparathyroidism and are usually associated with parathyroid levels in the low to the normal range [4,5]. Our patient had normal parathyroid levels. Idiopathic hypercalciuria can cause urolithiasis. Our patient had a history of nephrolithiasis, and he passed the stones spontaneously. Hypercalciuria and urolithiasis can cause a decrease in bone density [6,7]. Hence our patient was diagnosed with osteopenia as well. Dietary factories can also influence calcium excretion in the urine [8]. Increased protein, calcium, and sodium in the diet can increase urinary calcium [8]. However, our patient was not taking any dietary calcium supplements and had a moderate sodium intake. Urinary calcium greater than 4 mg per kilogram per 24 hours (0.1 mmol/kg body weight) is an indication of idiopathic hypercalciuria [9,10]. Our patient had urinary calcium of 408 mg/day. The metabolic disturbances associated with increased susceptibility to stone formation calcium oxalate stone are hypercalciuria, hyperoxaluria, hypocitraturia [11]. Our patient had hypercalciuria and hyperoxaluria.

Conclusion

Idiopathic renal hypercalciuria can cause a deficit in serum calcium and lead to osteoporosis; earlier diagnosis and management are necessary to prevent recurrence of stone formation. Adolescents with unexplained urinary symptoms should always be evaluated for IH. Thiazide diuretics are valuable and cost-effective. Further studies are required to determine the association between hypercalciuria and fracture association. An earlier diagnosis of IH may lead to earlier treatment intervention and decreased loss of bone density. IH is a complex trait and people having IH may have first degree relatives with the same condition. However, increased awareness regarding this condition is necessary and will lead to prompt diagnosis and earlier screening of the relatives and sooner access to genotyping.

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Manuscript Information: Received: November 29, 2021; Accepted: February 23, 2022; Published: February 28, 2022

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Citation: Patel K, Waleed MS, Pathalapati R, Gondara V. Idiopathic hypocalcemia with hypercalciuria: A case report. Open J Clin Med Case Rep. 2022; 1832.