Urinary tract stones in immobilized children[1]

Paul W. Musselman, M.D.
Robert Kay, M.D.

Urinary tract stones in children are uncommon in the absence of structural abnormalities or metabolic disorders. Prolonged immobilization is a recognized precursor of hypercalcemia and other disturbances in mineral metabolism. The authors describe 3 children (7, 9, and 11 years of age) who were found to have stones in the upper urinary tract following prolonged immobilization for treatment of orthopedic abnormalities. Hypercalcemia was documented in 2 patients and hypercalciuria in 1. The mechanism of immobilization-induced hypercalcemia and hypercalciuria is discussed.

Index terms: Immobilization · Urinary calculi

Cleve Clin Q 52:11–13, Spring 1985

Pediatric urolithiasis is uncommon, accounting for only one in 3,000–8,000 pediatric hospital admissions.[1,2] The etiology of pediatric urolithiasis is usually divided into four broad categories: *a*) endemic, *b*) metabolic, *c*) infectious, and *d*) idiopathic. Half of all calcium-containing stones in children represent a metabolic disorder. One such disorder resulting in hypercalcemia involves prolonged immobilization, usually imposed for treatment of musculoskeletal or neurologic abnormalities. First described by Albright in 1953,[3] prolonged immobilization affecting bone metabolism and leading to hypercalcemia and hypercalciuria has been well documented.[4–10] The resulting calcium-containing stones represent between 2% and 18% of all pediatric urolithiasis.[2,11] We present three additional cases of calcium-containing stones related to prolonged immobiliza-

[1] Section of Pediatric Urology, Department of Urology, The Cleveland Clinic Foundation. Submitted for publication Oct 1984; accepted Dec 1984.

0009-8787/85/01/0011/03/$1.75/0

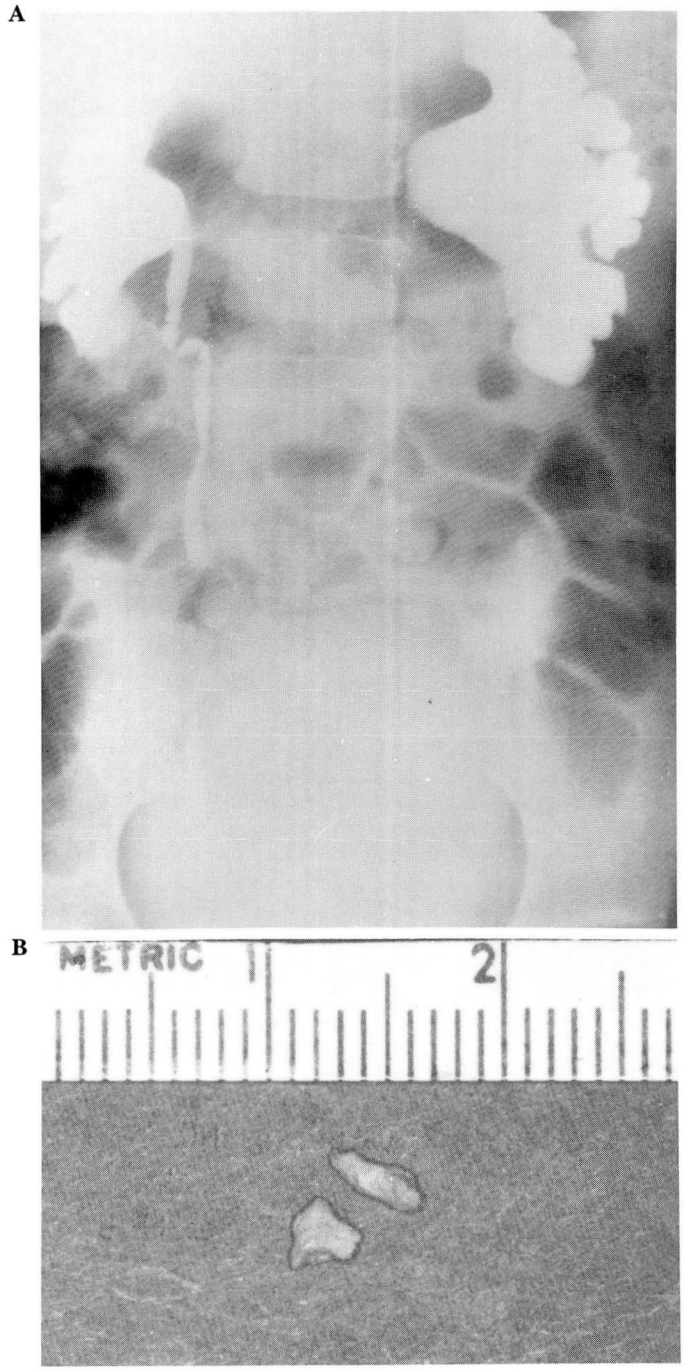

Fig. 1. Case 1.
A. Intravenous pyelogram demonstrates bilateral hydronephrosis immediately prior to passage of two calculi.
B. The calcium oxalate and calcium phosphate calculi passed spontaneously.

tion in an effort to understand the pathophysiology of this disease and to suggest further management of the immobilized child to avoid urolithiasis.

Case reports

Case 1. A 7-year-old boy sustained a fracture of his right femur following a bicycle accident. He was placed in traction for nine weeks. Total gross painless hematuria developed after the eighth week. Left-sided flank pain ensued, and an intravenous pyelogram showed bilateral hydronephrosis with delayed function on the left (*Fig. 1A*). He was subsequently transferred to the Cleveland Clinic where he was noted to have a serum calcium of 9.2 mg/dL. Shortly after the urogram was obtained at our institution, left-sided flank pain developed again and was associated with nausea and vomiting. He passed a 5-mm calculus. At this time, serum calcium was elevated at 11.0 mg/dL. Urine calcium excretion was 119 mg/24 hrs. The expected upper limit of normal urinary calcium for this patient was 74 mg/24 hrs calculated by a daily excretion rate of 4 mg/kg/24 hrs.[9] He subsequently passed two additional small calculi (*Fig. 1B*). Analysis revealed 95% calcium phosphate and 5% calcium oxylate. The patient has remained asymptomatic and free of stone disease for three years.

Case 2. An 11-year-old girl was struck by a car while riding her bicycle. The patient suffered multiple pelvic fractures, a fractured left tibia and left fibula, and a bladder laceration. Initially, a Hoffman apparatus was placed to align her multiply fractured pelvis, and subsequently, a Steinmann pin was inserted and traction instituted. She was immobilized for three months. During this time, her serum calcium was noted to be 10.4 mg/dL. One week later, gross painless hematuria developed. Abdominal radiographs revealed bilateral renal pelvic stones (*Fig. 2*). She subsequently underwent surgical removal of all upper tract stones. Stone analysis revealed calcium oxylate and calcium phosphate. No further stones have developed after three-and-a-half years.

Case 3. A 9-year-old boy was struck by a car and suffered a severe head injury and multiple fractures involving the pelvis, right femur, and right humerus. The patient was in a prolonged coma. He was placed in traction for five weeks and hospitalized for three months. Two months after discharge, hematuria developed. An intravenous pyelogram revealed osteopenia, left-lower-pole calculus, and a left ureteral calculus. He underwent a left ureteral lithotomy. Since reambulation, the patient has remained free of urolithiasis for four years.

Discussion

Bone is a complex metabolic organ that is in a dynamic equilibrium between calcium deposition and calcium reabsorption. According to Donaldson et al,[12] reabsorption is controlled by the parathyroid hormone, calcitonin, and mechanical factors. The mechanical factors are unknown, but are thought to be changes in electrical forces within the bone caused by pressure transferred to it or tension applied to it by surrounding muscular tissues. Immobilization changes the intrinsic mechanical forces on the bone and thereby upsets this balance, resulting in increased bone reabsorption.

An increase in the calcium reabsorption from bone may result in elevated serum calcium which suppresses parathyroid hormone levels which in turn affect urinary excretion, intestinal absorption of calcium, and synthesis of vitamin D. The natural history of hypercalcemia and hypercalciuria during immobilization has been demonstrated.[12] In this study, healthy volunteers were put to bed for 30 to 36 weeks. Urinary calcium was elevated from baseline throughout bed rest, peaking in the seventh week. Although this decreased later, it did remain elevated for at least three weeks after ambulation. In an additional study by Stewart et al,[10] the increase in urinary calcium persisted for 12 to 18 months following prolonged immobilization. During immobilization, normal healthy volunteers lost 1.5 g of calcium per week (approximately 0.5% of their total body calcium per month). Skeletal radiodensity was reduced as much as 30% after three to six months of immobilization.

Of concern are children who are involved in severe skeletal trauma which require immobilization. They exhibit a rapid turnover of bone and therefore are prime candidates for changes in the dynamic equilibrium of the bone and the resultant hypercalcemia and hypercalciuria. Although urolithiasis in the immobilized child is uncommon, it perhaps could be avoided altogether. A high index of suspicion combined with hydration and early ambulation will help to reduce the likelihood of urolithiasis in the susceptible child. If ambulation is impossible and prolonged immobilization is inevitable, calcitonin therapy should be considered in an effort to prevent the reabsorption of bone calcium and resultant hypercalcemia and hypercalciuria.[6,13] It is through an awareness of both the pathophysiology of stones in immobilized children as well as the potential therapies that we may help to prevent this uncommon childhood disease.

Robert Kay, M.D.
Section of Pediatric Urology
Department of Urology
The Cleveland Clinic Foundation
9500 Euclid Ave.
Cleveland OH 44106

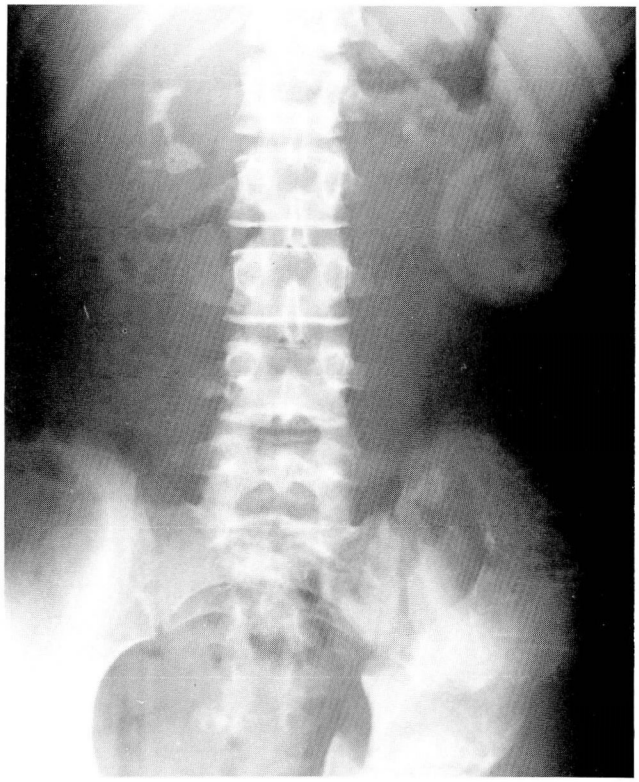

Fig. 2. Case 2. Abdominal radiograph reveals bilateral renal calculi, as well as a fractured pelvis. A colostomy collection device may be seen in the left upper quadrant.

References

1. Troup CW, Lawnicki CC, Bourne RB, et al. Renal calculus in children. J Urol 1972; **107:**306–307.
2. Bennett AH, Colodny AH. Urinary tract calculi in children. J Urol 1973; **109:**318–320.
3. Albright F, Henneman PH, Benedict PH, Forbes AP. Idiopathic hypercalciuria (abst). J Clin Endocrinol 1953; **13:**860.
4. Conley SB, Shackelford GD, Robson AM. Severe immobilization hypercalcemia, renal insufficiency and calcification. Pediatrics 1979; **63:**142–145.
5. Paulson DF. The challenge of calculi in children. Urol Clin North Am 1974; **1:**365–374.
6. Rosen JF, Wolin DA, Finberg L. Immobilization hypercalcemia after single limb fractures in children and adolescents. Am J Dis Child 1978; **132:** 560–564.
7. Wolf AW, Chuinard RG, Riggins RS, et al. Immobilization hypercalcemia: a case report and review of the literature. Clin Orthop 1976; **118:**124–129.
8. Lawrence GD, Loeffler RG, Martin LG, et al. Immobilization hypercalcemia. Some new aspects of diagnosis and treatment. J Bone Joint Surg 1973; **55A:**87–94.
9. Moore ES. Hypercalciuria in children. Contrib Nephrol 1981; **27:**20–32.
10. Stewart AF, Adler M, Byers CM, Segre GV, Broadus AE. Calcium homeostasis in immobilization: an example of resorptive hypercalciuria. N Engl J Med 1982; **306:**1136–1140.
11. Paulson DF, Glenn JF, Hughes J, et al. Pediatric urolithiasis. J Urol 1972; **108:**811–814.
12. Donaldson CL, Hulley SB, Vogel JM, et al. Affect of prolonged bed rest on bone mineral. Metabolism 1970; **19:**1071–1084.
13. Bergstrom WH. Hypercalciuria and hypercalcemia complicating immobilization. Am J Dis Child 1978; **132:**553–554.