



Congress Abstract

Hyperoxaluria Associated with Hypomagnesemia in a Young Child

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ABSTRACT

Background: Urolithiasis presenting in early childhood requires evaluation for hereditary and metabolic causes, including primary hyperoxaluria. Particular attention is warranted in children with recurrent bilateral nephrolithiasis and a predominantly oxalate stone composition. We report a case of hyperoxaluria in a young child in whom evaluation for primary hyperoxaluria did not confirm an inherited disorder, while further metabolic assessment revealed severe hypomagnesemia.

Case Presentation: A 2-year-10-month-old boy with no family history of urolithiasis first developed nephrolithiasis in June 2025, at approximately 18 months of age. Computed tomography demonstrated bilateral renal calculi and pyelectasis. In July 2025, he underwent left ureteral stent placement, right-sided lumbotomy, and pyelolithotomy. Assessment of kidney function showed an estimated glomerular filtration rate (eGFR) of 75.8 mL/min/1.73 m². In April 2026, nephrolithotomy, left-sided lumbotomy, and pyelolithotomy were performed. Infrared spectroscopy of the extracted stone demonstrated 65% oxalates and 35% phosphates. Metabolic evaluation also confirmed hyperoxaluria.

Given the early age at onset, bilateral stone formation, reduced kidney filtration, and hyperoxaluria, primary hyperoxaluria was considered and molecular genetic testing was performed. Whole-exome sequencing by next-generation sequencing (NGS) detected no pathogenic variants, likely pathogenic variants, or variants of uncertain clinical significance meeting the search criteria for hereditary kidney or metabolic disorders.

In September 2026, severe hypomagnesemia was identified for the first time, with a serum magnesium concentration of 0.18 mmol/L. In view of this finding and the negative genetic evaluation, the hyperoxaluria was considered secondary and associated with magnesium deficiency. Kidney function subsequently improved, with a serum creatinine level of 34.33 μmol/L and an estimated GFR of 92.5 mL/min/1.73 m². Follow-up imaging with ultrasonography and computed tomography demonstrated reduced size of the right kidney and no calculi in the kidneys or urinary collecting system. Management included optimization of fluid intake, dietary recommendations, and magnesium supplementation.

Conclusion: Early-onset oxalate nephrolithiasis in children warrants investigation for primary hereditary causes. A negative molecular genetic result does not eliminate the need for an extended metabolic evaluation. Magnesium deficiency may alter glyoxylate metabolism in the liver and kidneys, promoting accumulation of glyoxylate, a precursor of oxalate, and may also increase intestinal oxalate absorption, thereby increasing oxalate load. This case highlights the importance of assessing not only genetic causes of pediatric hyperoxaluria but also performing a comprehensive metabolic evaluation, including assessment of magnesium status.

	Keywords: Hyperoxaluria; Hypomagnesemia; Nephrolithiasis; Primary Hyperoxaluria; Children; Next-Generation Sequencing; Whole-Exome Sequencing
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