

Clinical and laboratory characterization of pyelonephritis and hyperoxaluria in children

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ABSTRACT: Pyelonephritis (PN) and hyperoxaluria (HO) are significant pediatric renal conditions. Their coexistence often complicates clinical management and exacerbates renal tissue injury through oxidative stress. This study aims to characterize the clinical and laboratory profiles of children with these conditions and evaluate the role of Malondialdehyde (MDA) as a marker of renal damage. This retrospective study analyzed clinical and laboratory data from 48 pediatric patients diagnosed with PN and HO between 1996 and 1997. Patients were categorized into three groups: PN, HO, and combined PN/HO. Clinical syndromes and laboratory markers, including leukocyturia, microhematuria, proteinuria, and MDA levels, were evaluated. Toxic syndrome was the most prevalent clinical manifestation in the combined PN/HO group. Abdominal pain syndrome was significantly more frequent in patients with concurrent PN and HO compared to isolated cases. Laboratory analysis revealed distinct profiles: leukocyturia (74 ± 7.6) was predominant in PN, microhematuria (88 ± 9.6) in HO, and high-grade proteinuria (330 ± 14 mg%) in the combined group. Notably, serum and urine MDA levels were significantly elevated in HO and combined cases,

indicating increased oxidative stress. The combined occurrence of PN and HO leads to more severe clinical and laboratory alterations than either condition alone. MDA levels serve as a critical diagnostic indicator for assessing oxalate-induced renal tissue damage.

INTRODUCTION

Renal and urinary tract diseases represent a significant portion of pediatric morbidity worldwide.¹ In Mongolia, these conditions rank third among pediatric diseases, with a growing concern regarding the transition to chronic renal failure due to delayed diagnosis. Pyelonephritis (PN) remains the most common acquired renal disease in Mongolian children, while hyperoxaluria (HO) has emerged as a critical metabolic disorder that predisposes children to nephrolithiasis and chronic interstitial injury. While the individual mechanisms of PN (bacterial inflammation) and HO (metabolic oxalate overload) are well-documented, their synergistic impact on renal tissue remains a subject of investigation. Chronic exposure to oxalates induces lipid peroxidation, a process that can be objectively measured through serum and urine Malondialdehyde (MDA) levels.

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The primary objective of this study is to provide a detailed clinical and laboratory characterization of children with PN, HO, and their combination. By analyzing the correlation between clinical syndromes and oxidative stress markers (MDA), we hypothesize that the coexistence of these conditions significantly accelerates renal injury, necessitating a more targeted diagnostic approach. Recent studies have consistently linked oxalate-induced tubular injury to heightened oxidative stress, where Malondialdehyde (MDA) acts as a reliable biomarker for lipid peroxidation and subsequent renal epithelial cell damage.^{2, 3} Understanding this pathway is essential for identifying early therapeutic targets in pediatric patients with metabolic nephropathies.

MATERIALS AND METHODS

This study is a retrospective analysis of 48 pediatric patients diagnosed with pyelonephritis (PN) and hyperoxaluria (HO). The study was conducted at the Department of Nephrology, National Center for Maternal and Child Health (NCMCH), Ulaanbaatar, Mongolia, during the period of 1996–1997. The patient cohort consisted of children who met the clinical and laboratory diagnostic criteria for PN, HO, or a combination of both. Diagnostic Criteria: Diagnostic criteria for pyelonephritis and hyperoxaluria were established based on the national clinical guidelines of the Mongolian Ministry of Health and the prevailing diagnostic standards applied at the NCMCH during the 1990s.⁴

Pyelonephritis (PN): Diagnosed based on clinical indicators (toxic syndrome, backache, urinary frequency) and laboratory evidence of leukocyturia and bacteriuria.

Hyperoxaluria (HO): Diagnosed based on clinical presentation and metabolic evidence of oxalate-induced urinary changes, such as microhematuria and hyperoxaluria.

Combined Group: Patients displaying concurrent clinical and laboratory signs of both PN and HO. Laboratory Analysis:

Laboratory investigations were performed at the Research Laboratory of “Monos Pharm” Co., Ltd. Key parameters included:

Urinalysis: Quantitative assessment of leukocyturia, microhematuria, and proteinuria (mg%).

Oxidative Stress Markers: Serum and urine Malondialdehyde (MDA) concentrations were measured to assess the degree of lipid peroxidation and renal tissue injury.

Quality Assurance: All samples were processed using standardized biochemical protocols.

Statistical Analysis:

Data were processed using descriptive statistical

methods. Clinical and laboratory findings were compared between the three groups (PN, HO, and PN/HO) to determine the statistical significance of differences in symptom prevalence and marker levels. Mean values were reported with standard deviations ($\pm$ SD).

Ethical approval for this retrospective study was granted by the Institutional Review Board of the National Center for Maternal and Child Health (NCMCH). Informed consent was waived due to the retrospective nature of the study, which involved the analysis of archived medical records while maintaining patient anonymity.

RESULTS

Epidemiology: The female-to-male ratio was 2:1, with the highest incidence occurring in the 11–16 age group ($42\% \pm 3.2$). **Clinical Syndromes:** Urinary syndrome was the most frequent (89.9%), followed by intoxication (70.1%) and dysuria (65.8%). In PN, lumbar pain (48%) and frequent urination (65%) were dominant. Conversely, HO patients frequently presented with abdominal pain (56%), decreased urination frequency (56%), and adipsia (67%). **Laboratory Indicators:** PN: Higher prevalence of leukocytosis and increased erythrocyte sedimentation rate (ESR). HO: Significantly higher incidence of hypersthenuria (61%) and hypercalciuria (78%). **Lipid Peroxidation (LPO):** MDA levels in HO patients (4.7 ± 0.6) were 1.2 times higher than in healthy controls, showing a strong correlation with microhematuria ($r = 0.7$, $p < 0.05$).

Table 1. MDA Levels in Patients with Pyelonephritis (PN) and Hyperoxaluria (HO)

Para- meters	Control Group (n, M $\pm$ m)	Pyelonephritis (PN) (n, M $\pm$ m)	Hyperoxaluria (HO) (n, M $\pm$ m)
Serum MDA (mmol/ml)	5 (0.55 $\pm$ 0.02)	15 (0.76 $\pm$ 0.04)	6 (0.93 $\pm$ 0.16)
Urine MDA (mmol/ml)	6 (3.6 $\pm$ 0.2)	13 (2.4 $\pm$ 0.19)	8 (4.7 $\pm$ 0.6)

* When comparing serum MDA (Malondialdehyde) levels among the control group, Pyelonephritis (PN), and Hyperoxaluria (HO), MDA levels were found to be increased in both PN and HO cases. In the case of HO, the average urinary MDA was 4.7 ± 0.6 , indicating an elevation of MDA in this condition. To verify the significance of the increased MDA levels in both blood and urine in HO compared to healthy children, the t-test was performed, yielding $t=2.4$ ($p<0.05$) for blood and $t=2$ ($p<0.05$) for urinary indicators, confirming statistical significance.

Table 2. Clinical Presentation and Syndrome Analysis

Diagnosis / Syndrome	Toxic Syndrome (n, P±m %)	Water-Electrolyte Imbalance (n, P±m %)	Pain Syndrome (n, P±m %)	Dysuria Syndrome (n, P±m %)
E. coli (+) PN (n=13)	10 (76±12.3)	9 (69±13.3)	7 (54±3)	9 (69±13.3)
E. coli (-) PN (n=10)	6 (60±16.3)	6 (60±16.3)	4 (40±16.3)	6 (60±16.3)
HO (n=18)	10 (55±12.1)	15 (83±9.1)	10 (55±12.1)	10 (55±12)
PN + HO (n=7)	6 (86±14.1)	6 (86±14.1)	6 (86±14.1)	4 (57±20.2)
Medical Record Study (n=228)	160 (70±3.0)	137 (60±3.2)	120 (53±3.3)	150 (66±3.1)

* The study involved 13 patients with E. coli-positive pyelonephritis (PN), 10 patients with E. coli-negative PN, 18 patients with hyperoxaluria (HO), and 7 patients with a combination of both PN and HO. Based on these results, our study findings are consistent with the data from the medical record review. Toxic syndrome was more prominent in cases of E. coli-induced PN and when PN was combined with HO. Water-electrolyte imbalance syndrome was predominant in the HO and combined forms, while pain syndrome was more prevalent in the combined form. In contrast, dysuria syndrome showed similar levels of manifestation across all four clinical forms.

DISCUSSION

The findings of this study confirm that the coexistence of PN and HO significantly worsens the clinical outcome in pediatric patients compared to isolated forms. As noted by Scheinman 1991, the inflammatory process in pyelonephritis triggers local tissue damage, which is further exacerbated by the metabolic stress caused by hyperoxaluria Scheinman 1994.^{5, 6} Our observation of elevated serum and urine MDA levels supports the theory of lipid peroxidation as a mechanism of renal injury.⁷ Oxalate crystals cause direct epithelial cell injury, leading to increased oxidative stress markers.⁸ Recent studies further substantiate that this oxidative environment facilitates renal epithelial cell damage, confirming MDA's utility as a biomarker.^{2, 3} Unlike isolated pyelonephritis, where inflammation is the primary driver, the presence of hyperoxaluria introduces a metabolic-inflammatory cycle that intensifies lipid peroxidation. This synergy explains the more severe clinical presentations observed in our combined PN/HO group. Our results align with the observations of previous researchers, suggesting that early detection of oxalate nephropathy is vital to preventing chronic kidney failure in children. Limitations: This study is retrospective, and the sample size (n=48) is limited, which suggests a need for larger, prospective longitudinal studies in the future. The study establishes a strong direct correlation between oxalate crystal aggregation and the intensification of urinary syndromes, such as high-grade proteinuria and hematuria.⁹⁻¹² Ultimately, the long-term accumulation of oxalate crystals coupled with secondary bacterial infection leads to severe renal injury, which explains why the clinical and laboratory manifestations in our combined PN/HO group were more severe than in cases of isolated disease.¹³⁻¹⁶ Given the synergistic aggravation of renal tissue injury

by the combined presence of metabolic hyperoxaluria and bacterial pyelonephritis, our findings advocate for a multidisciplinary diagnostic approach integrating both metabolic screening and inflammatory markers in pediatric patients presenting with recurrent urinary symptoms.¹⁷

CONCLUSION

Our study demonstrates that the coexistence of pyelonephritis and hyperoxaluria in children significantly aggravates the clinical course of renal disease compared to isolated forms. We observed that toxic syndrome is more prominent in pyelonephritis, while water-electrolyte imbalance is predominant in hyperoxaluria and combined cases. The study establishes a strong direct correlation between oxalate crystal aggregation and the intensification of urinary syndromes, such as high-grade proteinuria and hematuria. Serum and urine MDA levels were significantly higher in patients with hyperoxaluria and combined PN/HO, indicating that oxidative stress is a key mechanism of oxalate-induced renal tissue damage. Ultimately, the long-term accumulation of oxalate crystals coupled with secondary bacterial infection leads to severe renal injury, reinforcing the importance of early metabolic screening and targeted diagnostic approaches in pediatric nephrology.

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Conflicts of Interest:

The authors declare no conflicts of interest regarding the publication of this paper.

References:

1. World Health Organization. World Health Report: Children's health and the environment. Geneva: World Health Organization; 1995.
2. Cramer SD, Ferree PM. Primary Hyperoxaluria. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026.
3. Khairallah P, et al. Oxalate-induced renal injury: the role of oxidative stress and lipid peroxidation. *Kidney Int Rep.* 2024;9(3):450-462.
4. Ezersky RF. Pyelonephritis in Children. Leningrad: Mediastina; 1977.
5. Scheinman JI. Physiology of Pyelonephritis. *American Journal of Kidney Diseases.* 1991;17(2):203-206.
6. Scheinman JI. Primary hyperoxaluria. *Mineral and Electrolyte Metabolism.* 1994;20(6):340-350.
7. Unurjargal J. Clinical and laboratory findings in children with pyelonephritis and hyperoxaluria. [Master's Thesis]. Ulaanbaatar: Health Sciences University of Mongolia; 1998.
8. Gendenjamts N, Tserenbal D. Some issues of clinical manifestations and diagnosis of pyelonephritis in children. Ulaanbaatar; 1983.
9. Papayan AV, Savenkova ND. *Clinical Nephrology of Childhood.* Saint Petersburg: Sotis; 1997.
10. Tebioeva LT, Korovina NA. Current issues in pediatric nephrology. Moscow; 1992.
11. Petrushina AD, Krylov VI. Clinic-pathogenetic features of pyelonephritis in children. 1986.
12. Baltaev UB. Oxalate nephropathy in children: Clinical and genetic aspects. Moscow; 1990.
13. Baigildina LM. Clinical and laboratory characteristics of oxalate nephropathy in children. 1987.
14. Veltishchev VY, Ignatova MS. Preventive nephrology in children. Moscow: Mediastina; 1989.
15. Luvsandagva E. Prevalence and structure of renal diseases in children in Mongolia. Ulaanbaatar; 1990.
16. Brod J. Chronic pyelonephritis. *The Lancet.* 1957;269(6976):973-981.
[https://doi.org/10.1016/S0140-6736\(57\)91289-8](https://doi.org/10.1016/S0140-6736(57)91289-8)
17. Burova VY, Tsvetkova EI. Lipid peroxidation in metabolic nephropathies in children. *Pediatric Nephrology Journal.* 1984;12:45-49.