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Development of Acute Kidney Injury in Children with Oral Poisonings

Xamroyev Baxodir Olimkulovich[1], Axmedov Yusufjon Mahmudovich[2], Xursanov Yokubjon Erkinugli[3]

[1]Samarkand Branch of the Republican Center for Emergency Medical Care, Samarkand, Uzbekistan
[2]Professor, Head of the Department of Pediatric Surgery № 2, Samarkand State Medical University, Samarkand, Uzbekistan
[3]PhD, Department of Surgical Diseases № 2, Samarkand State Medical University, Samarkand, Uzbekistan

Abstract This study aimed to characterize renal dysfunction and assess the diagnostic value of early biomarkers in children with oral chemical poisonings. A retrospective review of 500 medical records (1990–2014) was followed by a prospective cohort of 120 patients aged 1–16 years (2015–2016); 40 healthy children served as controls. Standard clinical and imaging assessments were combined with urinary ELISA measurements of KIM-1, NGAL, and cystatin C. No serum creatinine elevation was observed, underscoring the limited sensitivity of conventional markers at early stages. In contrast, urinary NGAL was above control maxima in 11.6% and KIM-1 in 12.5% of patients; urinary cystatin C was frequently decreased and never exceeded control values, suggesting the need for complementary serum testing. The greatest biomarker elevations occurred with NSAID, neuroleptic, and caustic (including vinegar essence) poisonings. At one-month follow-up, KIM-1 declined >2-fold and NGAL >1.5-fold, indicating recovery. Conclusion: urinary NGAL and KIM-1 are sensitive early indicators of AKI and useful for treatment monitoring in pediatric oral poisonings, whereas urinary cystatin C is less informative and should preferably be assessed in serum. Early biomarker-based detection may reduce progression to chronic kidney disease.

Keywords Acute kidney injury, Children, Oral poisonings, Toxic nephropathy, KIM-1, NGAL, Cystatin C, pRIFLE, KDIGO, Biomarkers

1. Introduction

At present, acute kidney injury (AKI), including forms induced by toxic exposures, remains one of the major causes of impaired renal function in children and often determines clinical outcomes. In critically ill pediatric patients, AKI-associated mortality reaches approximately 25% by day 28 [1].

The first internationally accepted consensus definition of AKI was introduced in 2005 during the Acute Kidney Injury Network (AKIN, Amsterdam) conference. It recommended replacing the term “acute renal failure” with “acute kidney injury” (AKI), while reserving “acute renal failure” only for the most severe cases [2]. AKI is now recognized as a syndrome of progressive kidney dysfunction, ranging from minimal functional impairment to complete loss of renal ability to maintain water, electrolyte, and acid-base balance. Etiologies include severe trauma, major surgery, infectious diseases, and rapidly progressive hereditary disorders [3].

Among the leading causes of AKI in children today are oral poisonings of chemical or drug-related origin. Toxic exposures frequently lead to toxic nephropathy, which is often complicated by AKI [4,5]. Nephrotoxic agents exert direct cellular toxicity, disrupt renal microcirculation, cause tissue hypoxia, and trigger immune-mediated damage. As a result, the ICD-10 introduced class N14 — “tubulointerstitial and tubular lesions induced by drugs” [6]. In hospitalized children, administration of nephrotoxic drugs is a major contributor to AKI [8,9]. Accidental or intentional ingestion of toxic doses (poisoning) further increases the risk. Critically ill children are particularly vulnerable due to the combined effects of sepsis, hypotension, polypharmacy, and difficulties in accurate renal function assessment [10,11]. Currently, no definitive curative treatment for AKI exists. The main modifiable risk factor remains avoiding unnecessary nephrotoxic agents when safer alternatives are available. Early detection is difficult since pediatricians and nephrologists still rely on serum creatinine levels, which rise only at later stages. Promising biomarkers such as NGAL, KIM-1, and cystatin C have the potential to identify AKI earlier, before serum creatinine increases [12]. From a pathophysiological perspective, three major categories of AKI are distinguished:

- Prerenal (impaired renal perfusion due to hypotension, hypovolemia, or shock),
- Renal (toxic or ischemic injury, inflammatory processes),
- Postrenal (urinary tract obstruction) [13].

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In summary, oral poisonings in children represent one of the most important risk factors for AKI. This condition highlights the need for early recognition, cautious drug administration, and timely supportive treatment to prevent long-term renal complications Table 1.

Acute kidney injury (AKI) is commonly divided into functional and organic types depending on the underlying mechanism. Prerenal and postrenal AKI are classified as functional disorders, since structural damage to renal tissue is not yet present. In such cases, prompt elimination of the cause (restoration of circulating blood volume, hemodynamic stabilization, relief of urinary tract obstruction) often leads to rapid recovery of renal function. Renal AKI, on the other hand, is associated with organic damage to the renal parenchyma, including tubular necrosis, glomerular or interstitial inflammation, and toxic injury. This form is usually more severe, less responsive to treatment, and more likely to progress to chronic kidney disease.

To standardize diagnosis and assess the severity of AKI, in 2002 the Acute Dialysis Quality Initiative (ADQI) group proposed the RIFLE criteria, which describe five progressive stages:

1. **R – Risk**: the earliest stage, with mild reduction in glomerular filtration rate (GFR) or a moderate rise in serum creatinine.
2. **I – Injury**: more significant impairment, defined as a doubling of serum creatinine or reduced urine output.
3. **F – Failure**: severe kidney dysfunction, characterized by a sharp fall in GFR, oliguria or anuria, and a tripling of baseline creatinine.
4. **L – Loss**: persistent loss of kidney function for more than 4 weeks.
5. **E – End-stage kidney disease**: irreversible kidney failure lasting more than 3 months, requiring renal replacement therapy (hemodialysis, peritoneal dialysis, or kidney transplantation).

The RIFLE classification has become a cornerstone in nephrology, providing a standardized system for diagnosing and monitoring AKI and serving as a foundation for subsequent refinements, such as the **AKIN** and **KDIGO** criteria Table 2.

Table 1. Major Causes of Acute Kidney Injury (AKI)

Category	Causes	Mechanisms of Injury
Prerenal	- Hypovolemia (vomiting, diarrhea, blood loss, burns) - Shock (septic, traumatic, anaphylactic) - Arterial hypotension - Heart failure	Reduced renal perfusion → cortical ischemia
Renal	- Toxic injury (drugs: aminoglycosides, NSAIDs, cisplatin; chemicals, poisons) - Ischemic damage (prolonged hypoperfusion) - Glomerulonephritis, vasculitis - Acute tubulointerstitial nephritis - Infectious diseases (sepsis, pyelonephritis)	Direct nephron damage (tubules, interstitium, glomeruli)
Postrenal	- Urinary tract obstruction (congenital anomalies, posterior urethral valves) - Calculi (stones) - Tumors of the bladder, urethra, or prostate (rare in adolescents) - Ureteral compression (tumor, fibrosis)	

Table 2. Comparison of RIFLE, AKIN, and KDIGO Criteria

Classification	Serum Creatinine Criteria	GFR Criteria	Urine Output Criteria	Notes
RIFLE (2002, ADQI)	R: ×1.5 increase I: ×2 increase F: ×3 increase or ≥4 mg/dl	R: ↓ GFR >25% I: ↓ GFR >50% F: ↓ GFR >75%	R: <0.5 ml/kg/h, 6 h I: <0.5 ml/kg/h, 12 h F: <0.3 ml/kg/h, 24 h or anuria ≥12 h	Includes L (loss of function >4 wks) and E (end-stage >3 mos)
AKIN (2007)	St. 1: ↑ ≥0.3 mg/dl or ×1.5–2 St. 2: ×2–3 St. 3: ×3 or ≥4 mg/dl, or dialysis required	—	St. 1: <0.5 ml/kg/h, 6 h St. 2: <0.5 ml/kg/h, 12 h St. 3: <0.3 ml/kg/h, 24 h or anuria ≥12 h	L and E stages removed; added ≥0.3 mg/dl increase
KDIGO (2012)	St. 1: ↑ ≥0.3 mg/dl within 48 h or ×1.5 within 7 days St. 2: ×2–2.9 St. 3: ×3 or ≥4 mg/dl, or dialysis required	—	St. 1: <0.5 ml/kg/h, 6–12 h St. 2: <0.5 ml/kg/h, ≥12 h St. 3: <0.3 ml/kg/h, ≥24 h or anuria ≥12 h	Harmonized RIFLE & AKIN; current standard for AKI diagnosis

Table 3. Pediatric RIFLE (pRIFLE) Criteria for AKI (2007)

Stage	GFR Criterion	Urine Output Criterion
R – Risk	Estimated GFR decrease by 25%	<0.5 ml/kg/h for 8 h
I – Injury	Estimated GFR decrease by 50%	<0.5 ml/kg/h for 16 h
F – Failure	Estimated GFR decrease by 75% or eGFR <35 ml/min/1.73 m²	<0.3 ml/kg/h for 24 h or anuria ≥12 h
L – Loss	Persistent renal failure >4 weeks	—
E – End-stage kidney disease	Persistent renal failure >3 months	—

In clinical practice, acute kidney injury (AKI) is diagnosed based on changes in serum creatinine and urine output. According to current standards, AKI is defined by the presence of at least one of the following:

- an increase in serum creatinine (SCr) of ≥0.3 mg/dl (≥26.5 μmol/L) within 48 hours;
- an increase in SCr to >1.5 times baseline (if known, or assumed to have occurred within the previous 7 days);
- urine output <0.5 ml/kg/h for 6 consecutive hours [7].

In pediatrics, however, these criteria are less reliable because serum creatinine levels depend strongly on age, body weight, and muscle mass. To address this, in 2007 the pediatric-modified RIFLE (pRIFLE) scalewas introduced. This classification accounts for reductions in glomerular filtration rate (GFR) and urine output specific to children, and it plays a critical role in both staging AKI and determining the timing of renal replacement therapy (RRT).

Also in 2007, the international expert group AKIN (Acute Kidney Injury Network) proposed an updated system for stratifying AKI severity. This was a modification of the original RIFLE criteria, aimed at increasing sensitivity and improving early detection. Importantly, the AKIN definition incorporated even small rises in serum creatinine (≥0.3 mg/dl), which allowed clinicians to identify high-risk patients earlier and initiate therapy sooner (Table 4).

Thus, the progression from RIFLE to pRIFLE and AKIN has significantly improved the accuracy of AKI diagnosis, monitoring, and prognosis in both adults and children, and laid the groundwork for the KDIGO (2012) international guidelines.

Evolution of Classifications and Modern Diagnostic Criteria of AKI

The stratification system for acute kidney injury (AKI), based on changes in serum creatinine (SCr) and/or urine output, is largely similar to the RIFLE classification, but differs in several important aspects. In modified versions, the classes L (Loss) and E (End-stage) are no longer considered stages, but rather outcomes of AKI. Moreover, the R (Risk) category in RIFLE corresponds to Stage 1 AKI in the AKIN system, while I (Injury) and F (Failure) correlate with Stages 2 and 3 of AKIN, respectively.

In 2012, the **Kidney Disease: Improving Global Outcomes (KDIGO)** workgroup proposed a unified definition of AKI stages, integrating features of both RIFLE and AKIN (Table 4). While the previously used criteria were retained, two new parameters were introduced:

- an estimated glomerular filtration rate (eGFR) <35 ml/min/1.73 m² in patients under 18 years,
- initiation of renal replacement therapy (RRT) as a marker of the most severe AKI stage.

The creation of this unified classification provided a standardized framework for AKI diagnosis, enabling comparability across clinical and epidemiological studies, and establishing correlations between AKI stage and adverse outcomes, such as mortality and dialysis requirement.

The outcomes of AKI vary and may include:

- **complete recovery of renal function**,
- **partial recovery with residual damage**, leading to chronic kidney disease (CKD),
- **irreversible renal failure** progressing to end-stage kidney disease.

This issue is particularly critical in children and adolescents with oral chemical poisonings, where the course and prognosis of the disease depend heavily on the AKI stage at diagnosis. Although elevated SCr remains the principal diagnostic criterion, reliance on creatinine alone significantly underestimates the true prevalence of AKI.

Therefore, there is a strong need for early detection of AKI, identification of high-risk groups based on renal injury factors, and the implementation of novel biomarkers (NGAL, KIM-1, cystatin C), which can detect kidney injury before marked creatinine elevation occurs. For children affected by chemical poisonings, timely diagnosis of AKI is essential for preventing CKD and avoiding progression to chronic renal failure.

Objective of the present study — to establish the nature of renal function disorders and to determine the diagnostic value of AKI biomarkers in children with oral chemical poisonings.

2. Materials and Methods

Preliminary stage. At the first stage, a retrospective analysis of **500 medical records** of children treated at the Toxicology Department of Filatov Children's City Hospital No. 13 between 1990 and 2014 was performed. All patients had moderate to severe chemical poisonings.

In younger children (ages 1–3 years), intoxications were most often accidental, involving:

- caustic agents (vinegar essence, lighter fluids, detergents),
- nasal decongestants,
- less commonly — antihypertensive or psychotropic drugs and other substances.

In older children and adolescents, poisonings were mainly intentional or mixed, including alcohol, psychotropic drugs, NSAIDs, antispasmodics, sedatives, and tranquilizers.

Clinical patterns of specific intoxications:

- Vinegar essence poisoning caused burns of the gastrointestinal tract and airways, severe pain, dysphagia, hypersalivation, and blood alterations. Kidney damage was due to acute hemoglobinuric nephrosis with discolored urine, proteinuria, hematuria, glucosuria, crystalluria, and oxaluria. Ultrasound revealed renal edema and poor corticomedullary differentiation, while Doppler studies showed increased resistance index.
- **Vasoconstrictors** induced arterial hypotension, bradycardia, pallor, CNS depression, drowsiness, hypothermia, sweating; AKI developed due to renal ischemia from vasospasm.
- **NSAIDs** caused abdominal pain, nausea, vomiting (sometimes with blood), tremor; ultrasound demonstrated increased renal echogenicity, and laboratory tests revealed enzymuria (elevated alkaline phosphatase and γ-glutamyltransferase).
- **Barbiturates and psychotropic drugs** produced CNS depression, hypotension, bradycardia, impaired thermoregulation, oliguria/anuria, proteinuria, hematuria. Ultrasound revealed increased parenchymal thickness and echogenicity, with reduced GFR.

Overall, 80% of patients showed metabolic abnormalities (crystalluria, phosphaturia, triple-phosphate crystals, uraturia); **60%** had glomerular damage (proteinuria), and **10%** had tubular dysfunction (glucosuria). These findings suggest that most chemically poisoned children developed AKI of varying severity, with the most profound changes observed in vinegar essence, NSAID, barbiturate, vasoconstrictor, and combustion product poisonings.

Main stage. The prospective part included 120 children aged 1–16 years admitted to the same department from September 2015 to October 2016 with different chemical intoxications:

- neuroleptics ,
- NSAIDs (27),
- vinegar essence (18),
- antihypertensives (12),
- phenazepam (9),
- alcohol (6),
- decongestants (6),
- combustion products (3).

Diagnostic methods included:

- routine clinical and laboratory tests (urinalysis, complete blood count, biochemical blood and urine tests, GFR estimation, coagulation profile, acid-base balance);
- imaging (renal ultrasound with Doppler);
- selected tests for enzymuria (alkaline phosphatase, γ-glutamyltransferase).

Novel biomarkers of renal injury. All patients were additionally screened for urinary biomarkers of kidney damage:

- KIM-1 (Kidney Injury Molecule-1),
- NGAL (Neutrophil Gelatinase-Associated Lipocalin),
- Cystatin C.

These markers were evaluated as promising diagnostic tools for early detection of subclinical AKI in children with oral chemical poisonings.

Methods of Study and Rationale for Biomarker Selection

Laboratory techniques. Acute kidney injury (AKI) biomarkers were measured using enzyme-linked immunosorbent assay (ELISA) kits:

- **Cystatin C**: measured in serum, plasma, urine, and cerebrospinal fluid (range: **0.25–10,000 ng/ml**, sensitivity: **0.25 ng/ml**).
- **NGAL (neutrophil gelatinase-associated lipocalin)**: measured in plasma, urine, and culture medium (range: **0.4–100 ng/ml**, sensitivity: **0.4 ng/ml**).
- **KIM-1 (Kidney Injury Molecule-1)**: quantified using Human Quantikine ELISA Kit in urine (range: **0.046–10 ng/ml**, sensitivity: **0.046 ng/ml**).

All analyses were performed in the laboratory of the N. N. Blokhin Russian Cancer Research Center, Ministry of Health of Russia.

Control group. To establish reference values, a group of **40 conditionally healthy children** undergoing routine medical check-ups at the Filatov Children's City Hospital was examined. None had any somatic pathology.

Grouping criteria:

- severity of poisoning (moderate, severe),
- type of toxic agent,
- reduction of urine output:
  - <0.5 ml/kg/h for 16 h (RIFLE Injury),
  - <0.3 ml/kg/h for 24 h or anuria ≥12 h (RIFLE Failure),
- presence of urinary abnormalities (proteinuria, glucosuria, hematuria),
- ultrasound/Doppler findings (parenchymal hyperechogenicity, edema, increased resistance index).

Biomarker characteristics

KIM-1 is a 90 kDa transmembrane glycoprotein absent in healthy kidneys but highly expressed in proximal tubular cells after ischemic or toxic injury. Its urinary concentration rises much earlier than serum creatinine or urea, making it a reliable early marker of AKI. Elevated KIM-1 predicts adverse clinical outcomes, including dialysis requirement and mortality, and is also increased in chronic kidney diseases. Importantly, KIM-1 levels are stable in urine samples.

NGAL is a 25 kDa protein produced under cellular stress. It binds iron, limits bacterial growth, suppresses apoptosis, and promotes tissue repair. Under normal conditions, NGAL is freely filtered and reabsorbed in proximal tubules. With tubular injury, urinary NGAL excretion rises sharply due to impaired reabsorption and increased local synthesis.

NGAL is considered one of the most sensitive and early biomarkers of AKI. Importantly, its levels can rise even in the absence of elevated creatinine ("NGAL-positive, creatinine-negative" patients), indicating subclinical kidney injury and higher risk of poor outcomes. Urinary and plasma NGAL strongly correlate with creatinine, GFR, proteinuria, and histological kidney damage. NGAL is also used as a marker of CKD progression and severity.

Cystatin C. Cystatin C is a nonglycosylated cysteine protease inhibitor and is now recognized as one of the most accurate endogenous markers of glomerular filtration rate. Unlike creatinine, cystatin C levels are largely independent of age, body mass, or muscle mass, making it especially useful in pediatric diagnostics.

It is more sensitive than creatinine in detecting early kidney dysfunction but is limited by the relatively high cost of test kits. Cystatin C is measured using **ELISA, PETIA, and PENIA**, with ELISA offering the greatest accuracy at low concentrations.

Currently, cystatin Cis regarded as a reliable and sensitive marker for evaluating renal filtration function. Ongoing discussions in the scientific literature address whether it can be considered the earliest biomarker of kidney dysfunction compared to other established methods.

Cystatin C is a low-molecular-weight protein that is constantly synthesized at a stable rate by all nucleated cells. It freely passes through the glomerular filtration barrier, is completely metabolized in the proximal tubules, and—unlike creatinine—is not secreted by renal tubules. These characteristics ensure its stability and high accuracy as a marker of glomerular filtration rate (GFR).

Beyond its diagnostic role, cystatin C is also a potent inhibitor of cysteine proteases. These enzymes are involved in protein and tissue degradation, and their excessive activity may lead to tissue injury. By inhibiting protease activity, cystatin C helps regulate normal physiological processes, maintain tissue homeostasis, and protect organs, including the kidneys.

Thus, cystatin C serves not only as a promising biomarker for AKI and CKD but also as a regulatory protein with an important physiological role. Its diagnostic utility is particularly significant in children, since cystatin C levels are largely independent of age, sex, and muscle mass, making it more universal and reliable than creatinine.

Table 4. Comparative Characteristics of AKI Biomarkers

Biomarker	Sample/Source	Measurement Range (ELISA)	Analytical Sensitivity	Main Clinical Role
KIM-1 (Kidney Injury Molecule-1)	Urine	0.046–10 ng/ml	0.046 ng/ml	Early marker of ischemic and toxic proximal tubular injury; rises before creatinine and urea; predicts adverse outcomes (dialysis, mortality); also relevant in CKD
NGAL (Neutrophil Gelatinase-Associated Lipocalin)	Plasma, urine	0.4–100 ng/ml	0.4 ng/ml	Sensitive and rapid marker of tubular injury; reflects impaired reabsorption and enhanced synthesis; identifies subclinical AKI (NGAL-positive, creatinine-negative patients); used for outcome prediction and CKD monitoring
Cystatin C	Serum, plasma, urine, CSF	0.25–10,000 ng/ml	0.25 ng/ml	Accurate endogenous marker of glomerular filtration rate (GFR); more sensitive than creatinine and independent of age or muscle mass; valuable in pediatric patients; limited by higher cost of assays

Table 5. Dynamics of AKI Biomarkers in a Child with Severe Vinegar Essence Poisoning

Biomarker	Reference Range	Day 1 of Illness	One Month After Discharge	Change
KIM-1 (pg/ml)	84–326	**1166**	**524**	↓ more than 2-fold
NGAL (ng/ml)	0.94–32.14	**58.42**	**35.7**	↓ more than 1.5-fold
Cystatin C (ng/ml)	11.5–85.1	**60.6**	**53.6**	Slight decrease, within normal range

3. Results of the Study

The concentrations of acute kidney injury (AKI) biomarkers in urine varied depending on the severity of poisoning and the type of toxic agent. Notably, serum creatinine elevation was not observed in any of the examined children, highlighting its limited sensitivity for detecting early kidney damage.

At the same time, 14 patients (11.6%) demonstrated urinary NGAL levels above the maximum values of the control group, while 15 patients (12.5%) had increased urinary KIM-1. In contrast, urinary cystatin C in most cases (79 children) was below the minimal control values, and in no case did it exceed the upper reference range. This frequent reduction in cystatin C excretion may reflect elevated serum levels, representing a kind of "mirror effect." Thus, low urinary cystatin C could potentially serve as an additional diagnostic marker of AKI, although such a conclusion cannot be made without simultaneous serum measurements.

Based on the study results, it can be concluded that the most informative urinary markers of AKI in children with chemical oral poisonings are NGAL and KIM-1. Their significant increase was most often observed in children with intoxications caused by NSAIDs, neuroleptic drugs, caustic agents, and vinegar essence—the same categories that, according to retrospective analysis, had the highest incidence of renal complications.

Clinical case. Patient A., a 1-year-1-month-old girl, was admitted to the Toxicology Department of Filatov Children's City Hospital No. 13 with the diagnosis: *"Severe vinegar essence poisoning."* Upon admission, her condition was critical, with marked respiratory failure requiring mechanical ventilation. Clinical examination revealed extensive chemical burns of the oropharynx, upper respiratory tract, and upper gastrointestinal tract, as well as skin burns (anterior chest surface — approximately 10%).

This case exemplifies the severity of systemic manifestations in caustic substance poisonings and the high risk of AKI, emphasizing the need for early biomarker-based diagnostics.

Clinical Case Results. At admission, the following findings were obtained in a child with severe vinegar essence poisoning:

- **Complete blood count:** hemoglobin — 80 g/L, neutrophilic left shift, ESR — 35 mm/h.
- **Acid–base balance:** pH — 7.45; pCO_2 — 43.6 mmHg; Hb — 82 g/L; Ht — 25%.
- **Urinalysis:** erythrocytes — 23 per HPF; protein — 0.9 g/L.
- **Renal ultrasound: right kidney** — 62.8 × 28.6 mm, left kidney — 65.9 × 26.7 mm; parenchymal thickness right — 13 mm, left — 9 mm; preserved corticomedullary differentiation; diffuse cortical hyperechogenicity.
- **Chest X-ray:** enhanced vascular pattern, subsegmental atelectasis in the left upper lobe.
- **ECG:** sinus rhythm, heart rate 150–160 bpm.
- **Oliguria.**
- **Biochemistry:** no azotemia (creatinine — 38 µmol/L, urea — 5.8 mmol/L).
- **eGFR (Schwartz formula):** 105.2 ml/min.

By the end of the **first day of illness**, urinary **AKI biomarkers** were measured:

- **KIM-1:** 1166 pg/ml (reference 84–326 pg/ml);
- **NGAL:** 58.42 ng/ml (reference 0.94–32.14 ng/ml);
- **Cystatin C:** 60.6 ng/ml (reference 11.5–85.1 ng/ml).

These results confirmed the presence of acute kidney injury (AKI) already on the second day, well before the appearance of azotemia. Early diagnosis made it possible to promptly adjust therapy: in addition to infusion, antibacterial, and symptomatic treatment, energy-tropic and angioprotective agents were introduced for 30 days.

Follow-up. At one month after discharge, repeat testing showed a clear decline in urinary AKI biomarkers:

- **KIM-1:** 524 pg/ml (reference 84–326 pg/ml);
- **NGAL:** 35.7 ng/ml (reference 0.94–32.14 ng/ml);
- **Cystatin C:** 53.6 ng/ml (reference 11.5–85.1 ng/ml).

Thus, early detection of AKI and timely therapy resulted in a favorable dynamic: KIM-1 decreased more than twofold, NGAL decreased by over 1.5 times. This case highlights the importance of novel biomarkers in identifying AKI before conventional signs such as azotemia appear, which is critical for prognosis in children with severe chemical poisonings. Table 5.

At present, acute kidney injury (AKI) is one of the leading causes of impaired renal function in children of all ages. Among the risk factors, oral chemical poisonings play a particularly important role, often leading to toxic nephropathy and AKI. Due to the limited use of early diagnostic tools, the severity of AKI is frequently underestimated, resulting in delayed treatment. This delay places pediatric patients at an increased risk of developing chronic kidney disease (CKD) later in life.

Modern studies highlight the diagnostic potential of specific kidney injury biomarkers—namely KIM-1, NGAL (neutrophil gelatinase-associated lipocalin), and cystatin C. These biomarkers show a strong correlation with the presence and severity of renal dysfunction and allow for detection of injury well before conventional markers (serum creatinine, urea) become elevated.

For many years, it was assumed that patients who recovered from AKI experienced no long-term consequences. However, large population-based studies have revealed that a history of AKI significantly increases the risk of chronic renal failure (CRF). This emphasizes the importance of identifying high-risk patients for targeted follow-up and preventive strategies.

Our findings demonstrated that NGAL and KIM-1 were the most responsive urinary biomarkers, showing an early rise within the first 24 hours of intoxication and a marked decline one month after treatment, reflecting positive recovery dynamics. In contrast, cystatin C was less informative in urine measurements, likely because its diagnostic value is greater in serum evaluation. This requires further investigation.

4. Conclusions

In summary, the development of AKI in children exposed to chemical poisonings results from a combination of adverse factors, including systemic homeostatic imbalance, disturbances in systemic and renal hemodynamics, and water–electrolyte imbalance. Therefore, maintaining a high level of clinical vigilance in such cases is essential. Early diagnosis using novel biomarkers, together with timely initiation of pathogenetic therapy, reduces the risk of progression to CKD or end-stage renal disease (ESRD) and ensures more complete recovery of renal function.

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