CASE STUDY

Serial Hematologic-Inflammatory and Electrolyte Course in an 18-Month-Old Boy With Febrile Urinary Tract Infection and Kikiros Grade 5 Phimosis: A Single-Patient Case Study

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ABSTRACT

Background: Febrile urinary tract infection (UTI) in an uncircumcised toddler can coexist with severe preputial stenosis, which raises resistance to voiding outflow.

Objective: To describe the clinical, hematologic-inflammatory and electrolyte course of an 18-month-old boy with febrile UTI and Kikiros Grade 5 phimosis over four hospital days.

Methods: This single-patient case study, reported to the CARE guideline, covers an uncircumcised boy treated at K.R.M.T. Wongsonegoro Regional Hospital, Semarang, Indonesia, from January 31 to February 3, 2026. He presented after 3 days of antipyretic-refractory fever (38.6 °C) with vomiting, sunken eyes, obstructive voiding distress and Kikiros Grade 5 phimosis. Vital signs and blood counts were tabulated, with one chemistry panel. No inferential statistics were used.

Results: Urinalysis showed leukocyturia, bacteriuria and proteinuria. Leukocytes at admission were 10.4 × 10^3/µL, hemoglobin 11.4 g/dL, hematocrit 33.10% and platelets 312 × 10^3/µL. On Day 3, C-reactive protein was 16.80 mg/L, globulin 4.0 g/dL, potassium 5.50 mmol/L, sodium 136 mmol/L and ionized calcium 1.19 mmol/L. He received eight agents, including ceftriaxone, dexamethasone and furosemide. Temperature fell to 36.8 °C and heart rate from 136 to 104 beats/min. Leukocytes fell to 5.1 and then settled at 8.3 × 10^3/µL, while platelets rose from 191 to 323 × 10^3/µL. Voiding symptoms had resolved by Day 4.

Conclusion: In this child, symptoms and inflammatory markers improved within four days of combined treatment, and no single agent can be credited. Genital examination in febrile boys, renal ultrasonography after discharge and topical corticosteroid therapy for phimosis are advisable.

1. Introduction

Urinary tract infection (UTI) is among the most common bacterial infections of infants and young children. Worldwide it accounts for many emergency visits, acute admissions and empirical parenteral antibiotic courses.[1,2] Its incidence and clinical picture vary with sex, age, circumcision status and preputial retractability in boys, and with any structural or functional anomaly of the genitourinary tract.[3,4] Uncircumcised boys are the most exposed. In their first two years of life they are more susceptible to ascending infection and febrile UTI than circumcised peers, because uropathogenic Enterobacterales colonize the periurethral area and the non-retractile preputial sac.[5,6] Toddlers aged 12 to 24 months often present with non-specific signs of systemic illness, such as high fever, vomiting, anorexia, lethargy and isotonic or hyperosmolar dehydration, instead of urinary symptoms. Diagnosis is easily delayed. In acute pediatric care this remains a real hazard.[7,8]

Severe phimosis is defined anatomically as a stenotic distal preputial ring that prevents retraction of the foreskin over the glans penis. It changes both the microbial ecology and the fluid mechanics of voiding in young boys.[9,10] In asymptomatic infants, non-retractability is physiological, since congenital balanopreputial adhesions keratinize and separate as the child grows. Symptomatic or pathologic phimosis is different. Classified as Kikiros retractability Grade 4 to 5 or Kocaoglu Grade III, it forms a warm, moist and stagnant mucosal reservoir that fimbriated uropathogens colonize heavily.[11,12] Once the preputial opening narrows to a pinpoint, resistance to outflow rises with the inverse fourth power of the orifice radius, as the Poiseuille relation predicts. The toddler strains against high preputial back-pressure. Urine can reflux in turbulent jets from the urethra into the preputial space, and bacteria then ascend more readily across the bladder trigone and the ureterovesical junction.[13,14]

The usual pathogens are Escherichia coli, Klebsiella pneumoniae and Proteus mirabilis, whose strains carry virulence adhesins and sometimes determinants of extended-spectrum β-lactamase resistance.[15–19] Once they cross the superficial umbrella-cell layer of the urothelium, lipopolysaccharide engages Toll-like receptor 4 (TLR4) and activates nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) signaling.[20] The innate response draws neutrophils across the epithelium. That is leukocyturia. The mucosal barrier also loosens, which appears as proteinuria. High mobility group box-1 (HMGB1), interleukin-1β (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) enter the circulation, stimulate hepatic synthesis of C-reactive protein (CRP) and polyclonal immunoglobulins, and reset the hypothalamic thermoregulatory set-point through prostaglandin E2 (PGE2).[21,22] Tubulointerstitial inflammation can also blunt the response of the cortical collecting duct to aldosterone in febrile infants and toddlers. Reported consequences include transient distal potassium retention (hyperkalemia), sodium wasting (hyponatremia and transient secondary pseudohypoaldosteronism), and dehydration-related shifts in ionized calcium and microvascular platelet margination.[23–25]

A toddler with febrile UTI who vomits repeatedly and is micronutrient-depleted needs oral tolerance and mucosal

barrier integrity restored. Care combines 5-hydroxytryptamine type 3 (5-HT3) receptor blockade with ondansetron, isotonic crystalloid rehydration and targeted zinc supplementation, alongside parenteral bactericidal therapy.[26–28] Randomized trials and systematic reviews also suggest that a short course of a systemic corticosteroid such as dexamethasone, given with parenteral antibiotics, may reduce permanent renal cortical scarring, although trial results are mixed.[29–31] The 2026 European Association of Urology / European Society for Paediatric Urology (EAU/ESPU) guidelines and the 2026 American Academy of Pediatrics (AAP) clinical practice guidelines have both been updated. Detailed longitudinal descriptions may nevertheless be scarce. Such a description would quantify, in one child, serial hematological clearance, the partition of CRP-to-albumin and albumin-to-globulin, distal tubular electrolyte derangements and the kinetics of several drugs, in a toddler with febrile UTI and Kikiros Grade 5 phimosis.[1–5] This single-patient case study therefore had three aims. The first was to characterize the four-day clinical, hematologic-inflammatory, protein-globulin and electrolyte trajectory of a 1-year-6-month-old uncircumcised boy hospitalized with acute febrile UTI and Kikiros Grade 5 phimosis. The second was to describe the sequence of coordinated parenteral rehydration and bactericidal cephalosporin, antiemetic, micronutrient, kaliuretic loop diuretic and corticosteroid therapy, and the response to it. The third was to outline a guideline-based plan for secondary prevention, built on post-UTI renal ultrasonography and topical corticosteroid therapy for the prepuce.

2. Methods

Study Design and Setting

This single-patient case study follows the CARE guideline for case reports, and the completed checklist is provided with the submission. The patient was an uncircumcised boy aged 1 year 6 months (18.0 months). He was admitted through the Emergency Department and managed in the Pediatric Inpatient Ward of K.R.M.T. Wongsonegoro Regional General Hospital, Semarang, Central Java, Indonesia, under the academic clinical supervision of the Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia, during January and February 2026.

The timeline was assembled from the hospital record. The observation window covered a three-day febrile prodrome before admission (January 28 to January 30, 2026) and four inpatient epochs. Epoch T0 (Hospital Day 1) was emergency admission on January 31, 2026. Epoch T1 (Hospital Day 2) was ward evaluation and diagnostic urinalysis on February 1, 2026. Epoch T2 (Hospital Day 3) was extended inflammatory, protein-globulin and electrolyte profiling on February 2, 2026. Epoch T3 (Hospital Day 4) was hematological and clinical verification of resolution before discharge on February 3, 2026. The timeline itself is presented in the Results.

Participants and Eligibility Criteria

The diagnosis rested on four findings: an uncircumcised boy younger than 24 months; acute fever that did not respond to antipyretics (axillary temperature >= 38.0 °C for >= 48 hours); signs of lower urinary tract obstruction or mucosal irritation (straining during micturition, crying on voiding, frequent voids of small volume, and turbid, malodorous urine); and severe non-retractile phimosis confirmed on systematic genital inspection. Urinalysis showing leukocyturia, bacteriuria and proteinuria together confirmed the infection.[2,3,6] Two alternative diagnoses were considered from the history, physical examination and serial laboratory results. Primary acute viral or bacterial gastroenteritis was unlikely because dysuria and straining were prominent, the urine was turbid and malodorous, urinalysis showed leukocyturia and bacteriuria, and there was no watery diarrhea. Complicated acute lobar pyelonephritis or uroseptic shock was unlikely because there was no costovertebral angle tenderness or peritoneal sign, the Glasgow Coma Scale score was 15 and peripheral perfusion was normotensive. Preputial morphology was graded with the standardized six-tier Kikiros retractability scale (Grade 0: full retraction; Grade 5: no retraction at all, with a pinpoint orifice and an invisible glans) and with the Kocaoglu morphological classification.[9,10]

Variables and Outcome Measures

Treatment was prescribed by the treating pediatric team as standard care, and no research-specific intervention was given. The agents, routes and weight-based doses (9.0 kg) are listed in the Results. Follow-up was by daily bedside assessment until discharge on February 3, 2026, with vital signs, symptoms and blood counts as the outcome measures. Adverse events were looked for clinically and in the laboratory results.

Data Collection and Measurement

Clinical, hemodynamic and anthropometric parameters were recorded across Epochs T0 to T3. Baseline recumbent length (80.0 cm) and body weight (9.0 kg) were plotted on the World Health Organization (WHO) Child Growth Standards for boys aged 0 to 24 months. From these, the weight-for-age Z-score (WAZ), length/height-for-age Z-score (LAZ/HAZ) and weight-for-length Z-score (WLZ/WHZ) were derived, together with body mass index (BMI = weight [kg] / height [m]^2) and Mosteller body surface area (BSA = [(height [cm] × weight [kg]) / 3,600]$^{0.5}$). Axillary temperature (°C), heart rate (beats/min), respiratory rate (breaths/min) and peripheral capillary oxygen saturation (SpO2, %) on room air (FiO2 = 0.21) were tracked daily, and the SpO2/FiO2 ratio was derived. Emesis frequency, orbital hydration turgor and micturition mechanics were recorded at the same visits. Venous blood was sampled on January 31, 2026 (T0), February 2, 2026 (T2) and February 3, 2026 (T3). The Central Laboratory of RSUD K.R.M.T. Wongsonegoro Semarang analyzed hemoglobin (Hb, g/dL), hematocrit (Hct, %), erythrocyte count (RBC, × 10^6/µL), total leukocyte count (WBC, × 10^3/µL) and platelet count (PLT, × 10^3/µL) on automated hematology analyzers. On February 1, 2026 (T1), diagnostic urinalysis assessed macroscopic turbidity, odor, leukocyturia, bacteriuria, proteinuria and hematuria. On February 2, 2026 (T2), an extended biochemical panel measured quantitative C-reactive protein (CRP, mg/L; reference < 5.0 mg/L), serum albumin (g/dL; reference 3.4–4.8 g/dL), total protein (g/dL; reference 6.5–8.4 g/dL), serum globulin (g/dL; reference 1.8–3.2 g/dL), ionized calcium (Ca^{2+}, mmol/L; reference 1.00–1.15 mmol/L), serum potassium (K^+, mmol/L; reference 3.50–5.00 mmol/L) and serum sodium (Na^+, mmol/L; reference 135–147 mmol/L). The reference intervals are those printed on the hospital laboratory report.

Statistical Analysis

The patient's course was set against international pediatric nephrology and urology standards in three steps. First, hydrodynamic outflow resistance across the stenotic preputial orifice was illustrated with the Hagen-Poiseuille relation ($\Delta P = 8\mu LQ / [\pi r^4]$), which links pinhole narrowing to raised voiding pressure, straining (a bearing-down effort at every void), small-volume pollakiuria and retrograde periurethral bacterial seeding.[9–12] Second, derived ratios described the acute-phase protein pattern and the electrolyte balance: the Platelet-to-Leukocyte Ratio (PLRWBC = PLT / WBC), the Albumin-to-Globulin Ratio (A/G = Albumin / Globulin), the CRP-to-Albumin Ratio (CAR = CRP [mg/L] / Albumin [g/dL])

and the Serum Sodium-to-Potassium Ratio (Na^+/K^+). No mechanism was tested.[21–25] Third, the diagnostic workup, the inpatient drug regimen and the plan for post-discharge renal and bladder ultrasonography (RBUS) and preputial management were compared with the 2026 EAU/ESPU pediatric urology guidelines,[1] the 2026 AAP UTI clinical practice guidelines and companion technical reports,[2–5] the 2024 WikiGuidelines consensus statement,[6] and recent evidence on adjuvant anti-inflammatory therapy and imaging of the urinary tract after UTI.[29–35]

Because this was a single patient, no inferential tests (p values, t tests, analysis of variance or χ^2 tests) were applied; they carry no meaning for one subject. All serial clinical and laboratory measurements from T0 to T3 were instead described. Change from baseline was given as an absolute difference and, for ratio-scale variables only, as percentage change, Δ% = [(Tn − T0) / T0] × 100%. Inter-epoch step changes, Δ%(T_3 vs T_2) = [(T3 − T2) / T2] × 100%, and linear daily velocities, v = (Tn − T0) / Δt, were also calculated. Temperature was reported as an absolute change in °C because a percentage change of a Celsius value has no meaning. Erythrocyte corpuscular indices were derived at each venipuncture: Mean Corpuscular Volume (MCV [fL] = [Hct (%) × 10] / RBC [10^6/µL]), Mean Corpuscular Hemoglobin (MCH [pg] = [Hb (g/dL) × 10] / RBC [10^6/µL]) and Mean Corpuscular Hemoglobin Concentration (MCHC [g/dL] = [Hb (g/dL) / Hct (%)] × 100). Each biomarker was also expressed against the upper reference limit (URL) printed on the laboratory report as %URL = (Observed Value / URL) × 100%.

Ethical Considerations

Clinical information was taken from the hospital record and from the history given by the patient's mother, and it was de-identified for publication. After bedside counseling on February 1, 2026, the patient's mother (legal guardian) gave written informed consent. The consent explicitly covered anonymization, scientific analysis and open-access publication of the clinical history, the serial laboratory results and the photograph of the genital examination. To protect the privacy of the patient and caregiver, as the International Committee of Medical Journal Editors (ICMJE) and the Committee on Publication Ethics (COPE) require, permanent solid black bars were applied to the upper peripheral and caregiver-identifying features in the clinical photograph. The distal preputial pathology remained fully visible.

CMHC Research Center Ethics Committee, Indonesia, reviewed the protocol and granted ethical clearance (Protocol Approval Number: 2026/0429). The work complied with the clinical governance of K.R.M.T. Wongsonegoro Regional Hospital Semarang and of the Faculty of Medicine, Universitas Tarumanagara, Jakarta, Indonesia. All diagnostic, laboratory, photographic and therapeutic procedures followed the Declaration of Helsinki (2013 Fortaleza revision) and the privacy and confidentiality standards of the ICMJE.

3. Results

Participant Flow and Baseline Characteristics

An uncircumcised Indonesian boy aged 1 year 6 months (18.0 months) came to the Emergency Department of K.R.M.T. Wongsonegoro Regional Hospital Semarang on January 31, 2026 (Epoch T0, Hospital Day 1). His chief complaint was continuous high-grade fever for three days. His mother gave a structured history on February 1, 2026 (Epoch T1, Hospital Day 2). The fever had begun abruptly 72 hours before arrival, had persisted day and night, and had not responded to oral antipyretics at home. The child also vomited repeatedly, with more than two non-bilious episodes per day, refused solid and liquid intake, and became progressively less active. Of particular note, the mother described obstructive voiding. At every micturition the child cried intensely and visibly strained the abdomen and pelvis. He passed small volumes frequently (pollakiuria), and the urine was turbid and strongly malodorous, without macroscopic hematuria. He had never been circumcised and had no documented earlier UTI, congenital renal anomaly or hospitalization, as detailed in Table 1.

Table 1. Baseline Demographic, Anthropometric, and Clinical Findings at Admission (T0: January 31, 2026, to T1: February 1, 2026).

Clinical & Biometric Domain	Baseline Patient Finding (T0 / T1)	Reference Standard / Pediatric Cutoff	Pathophysiological & Clinical Interpretation
Chronological Age & Biological Sex	1 year 6 months (18.0 months); Male (XY)	Toddler / Early Childhood (< 24 months high-risk febrile UTI window)	A recognized window for non-retractile preputial colonization and ascending infection
Admission Setting & Dates	Emergency Department (T0: Jan 31, 2026); Inpatient Ward (T1: Feb 1, 2026)	RSUD K.R.M.T. Wongsonegoro Semarang (Tertiary Referral Hospital)	Acute inpatient admission necessitated by 3-day antipyretic-refractory fever, emesis, and dehydration
Chief Complaint & Febrile Duration	Continuous fever for 3 days prior to admission; T0 axillary temperature = 38.6 °C	Normal pediatric axillary temperature: 36.5–37.5 °C (Febrile UTI threshold >= 38.0 °C)	Sustained pyrexia (+1.1 °C above upper limit) refractory to outpatient oral antipyretics
Gastrointestinal & Hydration Status	Vomiting > 2 episodes/day, anorexia, hypoactivity, bilateral sunken eyes	WHO Mild-to-Moderate Dehydration Scale (sunken orbits, reduced oral intake, tachycardia)	Intolerance to oral fluids and emesis-induced volume depletion mandating parenteral crystalloid resuscitation and antiemetic therapy
Obstructive & Irritative Voiding Symptoms	Crying on micturition (dysuria), straining, pollakiuria (increased frequency, small voided volumes)	Normal toddler voiding: effortless stream without straining or vocal distress	High-resistance outflow through stenotic preputial ring coupled with acute trigonal-urethral mucosal inflammation
Macroscopic & Microscopic Urinalysis (Feb 1, 2026)	Turbid and malodorous urine; Leukocyturia (+), Bacteriuria (+), Proteinuria (+), Hematuria (−)	Clear, odorless urine; negative pyuria, negative bacteriuria, negative proteinuria	Active urinary tract infection with leukocyte exudation and protein leakage
Cardiorespiratory & Neurological Status	Moderately ill; Compos mentis, GCS 15 (E4V5M6); HR 136 bpm; RR 24 breaths/min; SpO2 98% (room air, FiO2 0.21; SpO2/FiO2 = 466.7)	Toddler awake HR: 80–130 bpm; RR: 20–30 breaths/min; SpO2 >= 95% (SpO2/FiO2 >= 452)	Preserved sensorium and oxygenation; heart rate +4.6% above the 130 bpm limit used (fever and dehydration)
Anthropometrics & WHO Growth Z-Scores	Body weight = 9.0 kg; Length/Height = 80.0 cm; BMI = 14.06 kg/m²; BSA = 0.45 m²; WAZ: −2 SD to 0 SD (−1.68 SD); LAZ/HAZ: −2 SD to 0 SD (−0.85 SD); WLZ/WHZ: −2 SD to −1 SD (−1.52 SD)	WHO Child Growth Standards (Boys 18 months): Median weight 10.9 kg, length 82.3 cm; Normal nutritional status (−2 SD <= Z <= +2 SD)	Within WHO normal limits; weight-for-length −1.52 SD, possibly from the 3-day febrile illness and dehydration
Genital & Preputial Morphology	Uncircumcised; severe distal preputial narrowing with pinpoint stenotic orifice; glans penis completely non-visualized; no external erythema, edema, or purulent discharge	Kikiros Retractability Grade 5 / Kocaoglu Grade III (completely non-retractile prepuce with pinhole meatal opening)	Symptomatic obstructive phimosis; a stagnant preputial reservoir for uropathogens and voiding back-pressure are proposed mechanisms

Abbreviations: BMI, body mass index (kg/m²); BSA, body surface area (Mosteller formula, m²); FiO_2, fraction of inspired oxygen; GCS, Glasgow Coma Scale; HAZ/LAZ, height/length-for-age Z-score; HR, heart rate; RR, respiratory rate; SpO_2, peripheral capillary oxygen saturation; UTI, urinary tract infection; WAZ, weight-for-age Z-score; WHO, World Health Organization; WHZ/WLZ, weight-for-height/length Z-score.

At emergency triage (T0) the child looked moderately ill but was fully alert: compos mentis, with a pediatric Glasgow Coma Scale (GCS) score of 15 (E4V5M6). Heart rate was 136 beats/min, which is +4.6% above the upper awake toddler reference limit of 130 beats/min. Respiratory rate was 24 breaths/min with regular mechanics. SpO2 was 98% on room air (FiO2 = 0.21), giving an SpO2/FiO2 ratio of 466.7. Axillary temperature was 38.6 °C, which is +1.1 °C above the 37.5 °C normothermic threshold. Both eyes were sunken, indicating mild-to-moderate dehydration from repeated vomiting, anorexia and febrile insensible losses. Cardiopulmonary auscultation and abdominal palpation found no hepatosplenomegaly, suprapubic mass or costovertebral angle tenderness. The penis was uncircumcised. The distal prepuce was severely narrowed and ended in a pinpoint, stenotic orifice that hid both the glans penis and the external urethral meatus. It was classified as Kikiros retractability Grade 5 and Kocaoglu Grade III phimosis. There was no external preputial erythema, balanoposthitis edema or purulent discharge (Figure 1).

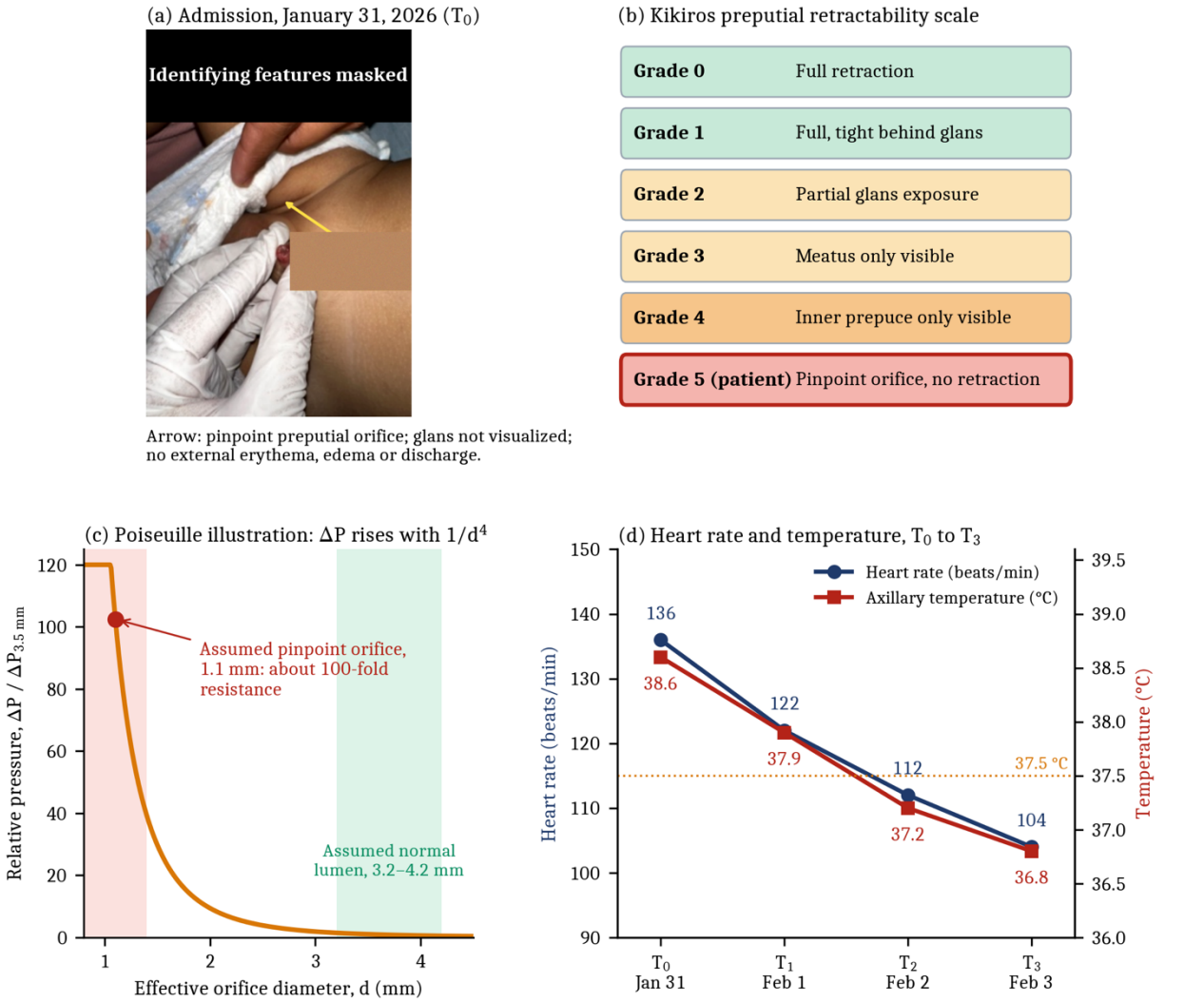


Figure 1. Kikiros Grade 5 Phimosis and Clinical Course Across Four Hospital Days. (a) Genital examination at admission (January 31, 2026): severe distal preputial narrowing with a pinpoint orifice that hides the glans, without erythema, edema or discharge; identifying features are masked with a black bar. (b) Kikiros preputial retractability scale, Grades 0–5. (c) Illustration of the Hagen-Poiseuille relation between orifice diameter and relative voiding pressure, using assumed diameters. (d) Heart rate and axillary temperature, T0 to T3.

Body weight at baseline was 9.0 kg and recumbent length was 80.0 cm, giving a body mass index (BMI) of 14.06 kg/m² and a Mosteller body surface area (BSA) of 0.45 m². The WHO Child Growth Standards for 18-month-old boys give a median weight of 10.9 kg, a median length of 82.3 cm and a median weight-for-length at 80.0 cm of 10.4 kg. Against these references, the weight-for-age Z-score (WAZ) fell in the −2 SD to 0 SD corridor (exact WAZ = −1.68 SD), and so did the length/height-for-age Z-score (LAZ/HAZ), which lay in the −2 SD to 0 SD corridor (exact LAZ/HAZ = −0.85 SD). The weight-for-length Z-score (WLZ/WHZ) lay in the −2 SD to −1 SD corridor (exact WLZ/WHZ = −1.52 SD). Overall, baseline nutritional status was within WHO normal limits, although weight-for-age and weight-for-length were close to −2 SD. The modest dip in WLZ (−1.52 SD) may reflect weight loss and fluid depletion during the three-day febrile and emetic prodrome; no earlier weight was available for comparison. The Holliday-Segar maintenance formula for a 9.0 kg toddler (100 mL/kg/day) set the daily fluid requirement at 900.0 mL/24 hours. This target guided immediate parenteral crystalloid resuscitation in the Emergency Department and a staged transition to full enteral hydration over the next 96 hours, as shown in Figure 2.

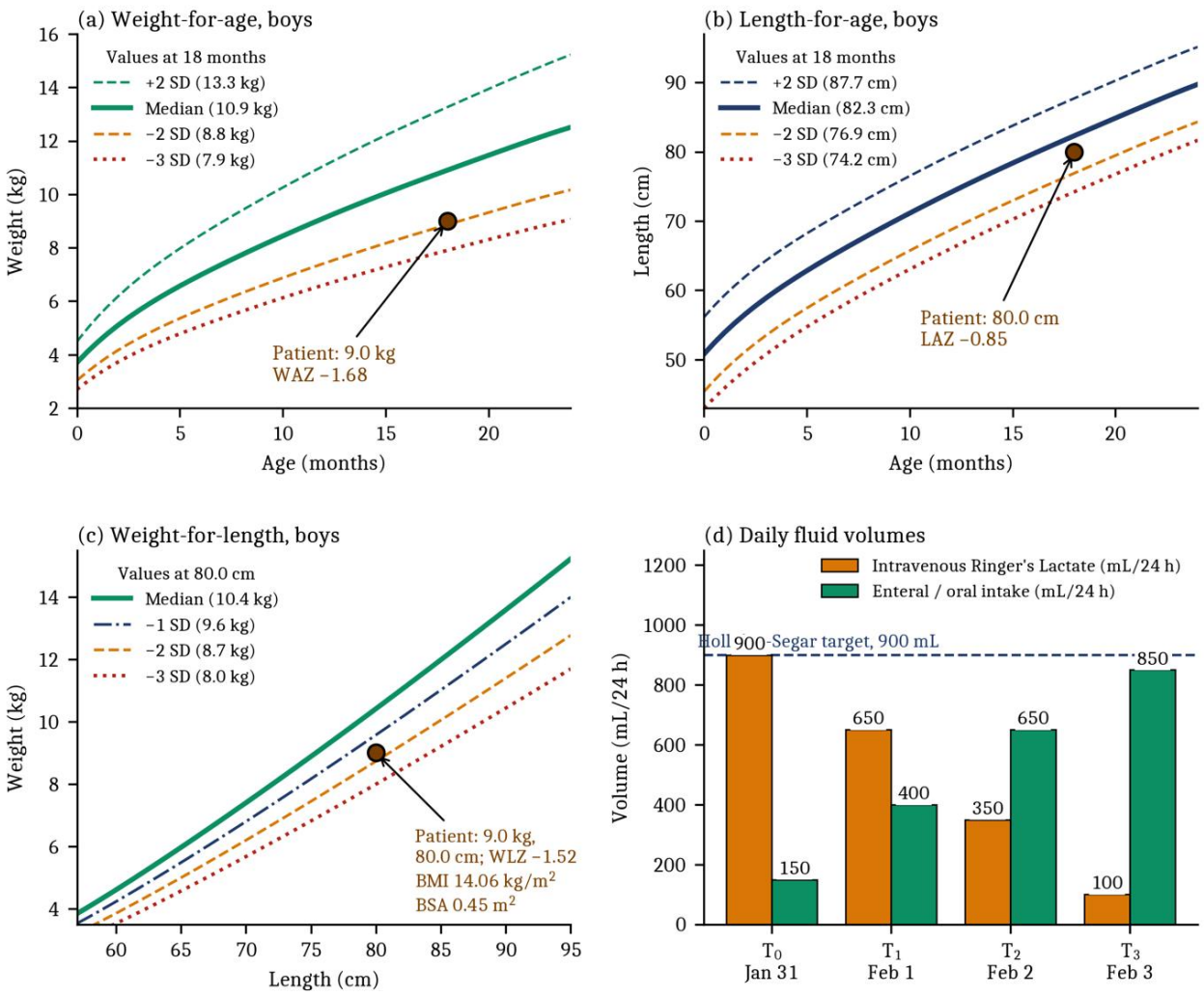


Figure 2. WHO Child Growth Standards Z-Score Plots (Boys 0–24 Months) and Daily Fluid Intake. (a) Weight-for-age, (b) length-for-age and (c) weight-for-length of an 18-month-old boy (9.0 kg, 80.0 cm) plotted on WHO reference lines (WAZ −1.68, LAZ/HAZ −0.85, WLZ/WHZ −1.52). (d) Daily intravenous and enteral fluid volumes against the Holliday-Segar maintenance target (900 mL/24 h).

Primary Outcome

Bedside surveillance across the four inpatient epochs (T0: January 31, 2026; T1: February 1, 2026; T2: February 2, 2026; T3: February 3, 2026) showed a rapid, coordinated reversal of fever, tachycardia, dehydration and obstructive dysuria after multimodal therapy began. Axillary temperature fell steadily from 38.6 °C at T0 to 37.9 °C at T1, reached 37.2 °C and afebrile normothermia at T2, and settled at 36.8 °C by T3 (net Δ = −1.8 °C; linear thermal clearance velocity = −0.60 °C/day). Heart rate slowed from 136 beats/min at T0 to 122 beats/min at T1 (Δ% = −10.29%), 112 beats/min at T2 (Δ% = −17.65%) and 104 beats/min at T3 (net Δ = −32 beats/min; net Δ% = −23.53%; deceleration velocity = −10.7 beats/min/day). Respiratory rate stayed regular (24 to 22 breaths/min; Δ% = −8.33%), and SpO2 rose from 98% to 99% on room air (SpO2/FiO2 ratio = 466.7 to 471.4; Δ% = +1.02%). After parenteral ceftriaxone, dexamethasone, furosemide and ondansetron were given, vomiting had stopped completely by T2 (Δ% = −100.0%) and the sunken eyes had resolved, with normal periorbital turgor restored. Crying and straining at micturition disappeared entirely by T3, and the urine cleared of turbidity and malodor (Table 2). The changes followed the start of treatment but cannot be attributed to a single drug.

Table 2. Clinical Timeline of Vital Signs, Hydration, Voiding, and Urinalysis From T0 to T3 (January 31 to February 3, 2026).

Clinical Parameter	T0: Day 1 (Jan 31, 2026) Emergency Dept	T1: Day 2 (Feb 1, 2026) Inpatient Ward	T2: Day 3 (Feb 2, 2026) Inpatient Ward	T3: Day 4 (Feb 3, 2026) Pre-Discharge	Change T0 → T3 and clinical resolution
Axillary Temperature (°C)	38.6 °C (3-day continuous fever)	37.9 °C (early defervescence)	37.2 °C (afebrile)	36.8 °C (sustained normothermia)	Δ = −1.8 °C; cooling rate = −0.60 °C/day; afebrile by T2
Heart Rate (beats/min)	136 bpm (febrile tachycardia)	122 bpm (improving volume status)	112 bpm (normocardic)	104 bpm (baseline resting rhythm)	Δ = −32 bpm (Δ% = −23.53%); deceleration rate = −10.7 bpm/day, alongside rehydration and fever clearance
Respiratory Rate (breaths/min) & SpO2 (%)	24 breaths/min; SpO2 98% (FiO2 0.21; S/F = 466.7)	24 breaths/min; SpO2 98% (FiO2 0.21; S/F = 466.7)	22 breaths/min; SpO2 99% (FiO2 0.21; S/F = 471.4)	22 breaths/min; SpO2 99% (FiO2 0.21; S/F = 471.4)	RR Δ% = −8.33%; SpO2 Δ% = +1.02%; regular breathing throughout hospitalization
Emesis Frequency & Oral Intake Tolerance	> 2 episodes/day; severe anorexia & reduced activity	1 episode/day; partial oral liquid tolerance	0 episodes/day; restored appetite & active play	0 episodes/day; 100% oral nutritional tolerance	Emesis Δ% = −100.0% by T2, after IV ondansetron, Ringer's Lactate, ranitidine and zinc were started

Clinical Parameter	T0: Day 1 (Jan 31, 2026) Emergency Dept	T1: Day 2 (Feb 1, 2026) Inpatient Ward	T2: Day 3 (Feb 2, 2026) Inpatient Ward	T3: Day 4 (Feb 3, 2026) Pre-Discharge	Change T0 → T3 and clinical resolution
Orbital Hydration & Systemic Perfusion	Bilateral sunken eyes; mild-moderate dehydration	Decreasing periorbital hollowness; capillary refill < 2 s	Normal orbital turgor; euvolemic mucosa	Fully restored orbital & skin turgor; euvolemic (9.0 kg)	100% reversal of clinical dehydration signs after isotonic crystalloid and enteral rehydration
Dysuria, Straining & Voiding Frequency	Severe crying and straining at every void; frequent small-volume voids	Moderate dysuria; reduced suprapubic straining effort	Minimal discomfort; consolidated voiding volumes	Zero crying or straining; normal age-appropriate stream	100% resolution of dysuria and straining by T3, after IV ceftriaxone, dexamethasone and furosemide were started
Urine Macroscopic & Dipstick/Sediment Profile	Cloudy (turbid), malodorous; Hematuria (−)	Urinalysis (Feb 1): Leukocyturia (+), Bacteriuria (+), Proteinuria (+), Hematuria (−)	Clearing amber urine; resolution of malodor	Clear, pale-yellow, non-malodorous urine	Macroscopic clearance of turbidity and malodor by T3; repeat urinalysis not reported
Preputial & Glans Physical Examination	Distal preputial stenosis, pinpoint orifice, glans non-visualized (Kikiros Grade 5)	Distal preputial narrowing; zero balanoposthitis erythema/edema/discharge	Unchanged stenotic preputial ring; zero local tissue inflammation	Persisting Kikiros Grade 5 anatomic phimosis; scheduled for post-discharge topical steroid / urology follow-up	Acute periurethral mucosal irritation resolved; structural phimosis remains for definitive management

Abbreviations: S/F, SpO_2/FiO_2 ratio. Percentage change is (later value − T0) / T0 × 100 and is not given for temperature.

By T3 the child was afebrile with a heart rate of 104 beats/min, had no vomiting, passed clear urine without crying or straining, and was discharged on February 3, 2026. The Kikiros Grade 5 phimosis persisted at discharge (Table 2). The treating team judged the outcome favorable for survival, renal function and cure. Discharge planning included outpatient renal and bladder ultrasonography (RBUS) and first-line topical corticosteroid therapy for the phimosis.

Secondary and Additional Analyses

Urinalysis on February 1, 2026 (T1) was positive for leukocyturia, bacteriuria and proteinuria, with no microscopic hematuria. This supported the working diagnosis of acute febrile UTI together with Kikiros Grade 5 phimosis.

Serial hematology at T0 (January 31, 2026), T2 (February 2, 2026) and T3 (February 3, 2026) described the course of the myeloid, megakaryocytic and erythroid lineages. At admission (T0) the total leukocyte count (WBC) was 10.4 × 10^3/µL, within the laboratory interval of 3.8–10.6 × 10^3/µL (98.1% of the upper limit printed on the report). At T2, after parenteral ceftriaxone and dexamethasone had been started on T1, WBC had fallen by −50.96% to 5.1 × 10^3/µL. This may reflect treatment response, hemodilution or margination; a differential count was not reported. It then settled at a mid-range 8.3 × 10^3/µL on T3 (Δ% T2→T3 = +62.75%; net Δ% T0→T3 = −20.19%). The platelet count (PLT) followed a biphasic acute-phase course within the normal reference range (150–400 × 10^3/µL). It decreased from 312 × 10^3/µL at T0 to a transient nadir of 191 × 10^3/µL at T2 (Δ% = −38.78%), possibly from microvascular margination, and then rebounded by +69.11% to 323 × 10^3/µL on T3 (net Δ% T0→T3 = +3.53%). The derived Platelet-to-Leukocyte Ratio (PLRWBC = PLT / WBC) therefore rose at every venipuncture, from 30.00 at T0 to 37.45 at T2 (Δ% = +24.83%) and 38.92 at T3 (net Δ% = +29.73%). In the erythroid compartment, hemoglobin stayed within the 11.0–15.0 g/dL reference range (11.4 g/dL at T0, 11.6 g/dL at T2 and 11.6 g/dL at T3; net Δ% = +1.75%). Hematocrit changed from 33.10% at T0 to 32.40% at T2 (Δ% = −2.11%), which probably reflects plasma volume expansion after Ringer's Lactate rehydration, and recovered to 33.80% on T3 (Δ% T2→T3 = +4.32%; net Δ% T0→T3 = +2.11%). Erythrocytes moved from 4.96 to 4.87 and 5.01 × 10^6/µL (net Δ% = +1.01%). The derived corpuscular indices were stable (MCV: 66.73 → 66.53 → 67.47 fL; MCH: 22.98 → 23.82 → 23.15 pg; MCHC: 34.44 → 35.80 → 34.32 g/dL), as shown in Figure 3. MCV was below, and MCH at the lower edge of, the intervals printed in Table 3 (MCV 70–86 fL; MCH 23–30 pg).

(a) Hospital laboratory record

Nama pemeriksaan	31/01/2026	02/02/2026	03/02/2026	Nilai rujukan
Hemoglobin	11,4	11,6	11,6	11-15 g/dL
Hematokrit	33,10	32,40	33,80	40-52%
Eritrosit	4,96	4,87	5,01	4,7-6,1 /µL
Leukosit	10,4	5,1	8,3	3,8-10,6 /µL
Trombosit	312	191	323	150-400 /µL
CRP	-	16,80	-	<5,0 mg/L
Albumin	-	4,0	-	3,4 - 4,8 g/dL
Protein total	-	8,0	-	6,5 - 8,4 g/dL
Globulin	-	4,0	-	1,8 - 3,2 g/dL
Kalsium	-	1,19	-	1,00 – 1,15 mmol/L
Kalium	-	5,50	-	3,50 – 5,00 mmol/L
Natrium	-	136	-	135 – 147 mmol/L

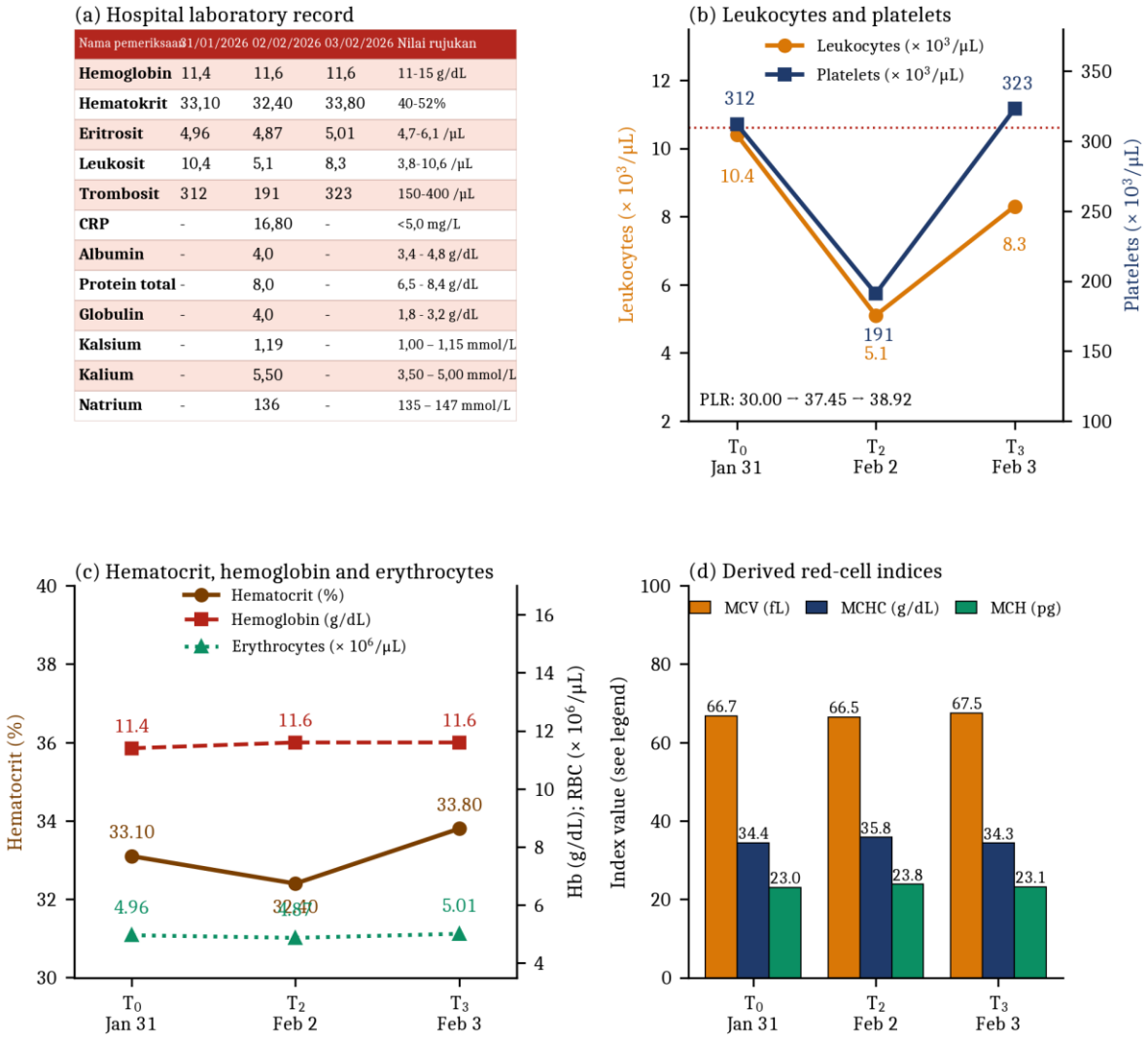


Figure 3. Serial Laboratory Results and Derived Red-Cell Indices on January 31 (T0), February 2 (T2), and February 3, 2026 (T3). (a) Hospital laboratory record, retyped with its Indonesian headings, decimal commas and units as printed. (b) Leukocytes and platelets; the dotted line is the printed upper limit for leukocytes. (c) Hematocrit, hemoglobin and erythrocytes. (d) Derived red-cell indices.

Extended biochemistry on February 2, 2026 (Epoch T2, Hospital Day 3) showed systemic inflammatory activation and a borderline potassium value (Table 3; Figure 4a). Quantitative C-reactive protein (CRP) was clearly elevated at 16.80 mg/L against a reference of < 5.0 mg/L. That is 336.0% of the upper reference limit (+236.0% above the cutoff), which is compatible with an acute-phase response to the infection. CRP was not measured at admission or at discharge. Total protein was 8.0 g/dL (reference: 6.5–8.4 g/dL; 95.2% URL), and albumin was well preserved at 4.0 g/dL (reference: 3.4–4.8 g/dL; 83.3% URL), so the CRP-to-Albumin Ratio (CAR) was 4.20 (mg/L per g/dL), equivalent to 0.42 mg/g. Globulin was raised at 4.0 g/dL against a reference of 1.8–3.2 g/dL (125.0% URL; +25.0% above the upper limit). This lowered the Albumin-to-Globulin (A/G) ratio to 1.00. Globulin is the difference between total protein and albumin, and protein electrophoresis was not done, so the class of globulin cannot be stated; the child was on the sixth day of illness. Electrolytes showed a borderline-high potassium, with serum potassium (K^+) of 5.50 mmol/L (reference: 3.50–5.00 mmol/L; 110.0% URL; +10.0% above the upper limit), and low-normal serum sodium (Na^+) of 136 mmol/L (reference: 135–147 mmol/L; Na^+/K^+ molar ratio = 24.73). The cohort cited for comparison defines hyperkalemia as potassium above 5.5 mmol/L,[23] so this value lies at the threshold rather than above it. The pattern is compatible with, but does not prove, transient distal nephron aldosterone resistance; renin, aldosterone, urine electrolytes and a repeat potassium were not measured. Ionized calcium (Ca^{2+}) was 1.19 mmol/L, which is 3.48% above the upper limit printed on the report (1.00–1.15 mmol/L; 103.5% URL). Acid-base status was not measured.

Table 3. Serial Laboratory Results and Derived Indices at T0, T2, and T3 (January 31, February 2, and February 3, 2026).

Laboratory Parameter & Unit	T0: Jan 31, 2026 (Admission Day 1)	T2: Feb 2, 2026 (Ward Day 3)	T3: Feb 3, 2026 (Ward Day 4)	Reference Interval (as printed on the hospital report)	Percentage change (T0→T2 | T2→T3 | net T0→T3) and interpretation
Hemoglobin (Hb, g/dL)	11.4	11.6	11.6	11.0–15.0 g/dL	Δ% (T0→T2) = +1.75% | Δ% (T2→T3) = 0.00% | Net Δ% = +1.75%; stable within the reference range
Hematocrit (Hct, %)	33.10%	32.40%	33.80%	40.0–52.0%	Δ% (T0→T2) = −2.11% | Δ% (T2→T3) = +4.32% | Net Δ% = +2.11%; possible hemodilution after IV Ringer's Lactate; the interval printed is an adult interval

Laboratory Parameter & Unit	T0: Jan 31, 2026 (Admission Day 1)	T2: Feb 2, 2026 (Ward Day 3)	T3: Feb 3, 2026 (Ward Day 4)	Reference Interval (as printed on the hospital report)	Percentage change (T0→T2 | T2→T3 | net T0→T3) and interpretation
Erythrocytes (RBC, × 10^6/µL)	4.96	4.87	5.01	4.70–6.10 × 10^6/µL	Δ% (T0→T2) = −1.81% | Δ% (T2→T3) = +2.87% | Net Δ% = +1.01%; stable
Derived Red Cell Indices (MCV [fL] / MCH [pg] / MCHC [g/dL])	66.73 fL / 22.98 pg / 34.44 g/dL	66.53 fL / 23.82 pg / 35.80 g/dL	67.47 fL / 23.15 pg / 34.32 g/dL	Toddler MCV: 70–86 fL; MCH: 23–30 pg; MCHC: 32–36 g/dL	MCV net Δ% = +1.11%; MCV below, and MCH at the lower edge of, the printed intervals; cause not established
Total Leukocytes (WBC, × 10^3/µL)	10.4	5.1	8.3	3.8–10.6 × 10^3/µL	Δ% (T0→T2) = −50.96% | Δ% (T2→T3) = +62.75% | Net Δ% = −20.19%; fall by T2, after IV ceftriaxone and dexamethasone were started on T1, then partial recovery
Platelet Count (PLT, × 10^3/µL)	312	191	323	150–400 × 10^3/µL	Δ% (T0→T2) = −38.78% | Δ% (T2→T3) = +69.11% | Net Δ% = +3.53%; nadir on Day 3 with rebound on Day 4
Platelet-to-Leukocyte Ratio (PLRWBC = PLT / WBC)	30.00	37.45	38.92	Derived index	Δ% (T0→T2) = +24.83% | Δ% (T2→T3) = +3.93% | Net Δ% = +29.73%; rising across the three draws
C-Reactive Protein (CRP, mg/L)	Not assayed at T0	16.80	Not measured	< 5.0 mg/L	3.36 × upper limit on Day 3 (+236.0% above the 5.0 mg/L cutoff), compatible with an acute-phase response to infection; no admission or discharge value
Serum Total Protein (g/dL) & Albumin (g/dL)	Not assayed at T0	Total Protein: 8.0 Albumin: 4.0	Not measured	Total Protein: 6.5–8.4 g/dL Albumin: 3.4–4.8 g/dL	Albumin preserved (4.0 g/dL) alongside proteinuria (+)
Serum Globulin (g/dL), A/G Ratio & CRP-to-Albumin Ratio (CAR)	Not assayed at T0	Globulin: 4.0 / A/G Ratio: 1.00 / CAR: 4.20 (mg/L per g/dL)	Not measured	Globulin: 1.8–3.2 g/dL Normal A/G: 1.1–2.2	Globulin +25.0% above the 3.2 g/dL limit; calculated as total protein minus albumin; class not determined
Serum Potassium (K^+, mmol/L) & Sodium (Na^+, mmol/L)	Not assayed at T0	K^+: 5.50 Na^+: 136 Na^+/K^+ Ratio: 24.73	Not measured	K^+: 3.50–5.00 mmol/L Na^+: 135–147 mmol/L	Potassium +10.0% above the 5.00 mmol/L limit printed on the report (at, not above, 5.5 mmol/L); sodium low-normal; mechanism not tested
Ionized Calcium (Ca^{2+}, mmol/L)	Not assayed at T0	1.19	Not measured	1.00–1.15 mmol/L	3.48% above the 1.15 mmol/L limit printed on the report

Abbreviations: A/G, albumin-to-globulin ratio; CAR, C-reactive protein-to-albumin ratio ((mg/L)/(g/dL)); CRP, C-reactive protein; Hb, hemoglobin; Hct, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PLRWBC, platelet-to-leukocyte ratio; PLT, platelets; RBC, red blood cells; WBC, white blood cells. Day 4 CRP, potassium, sodium, ionized calcium and proteins were not measured.

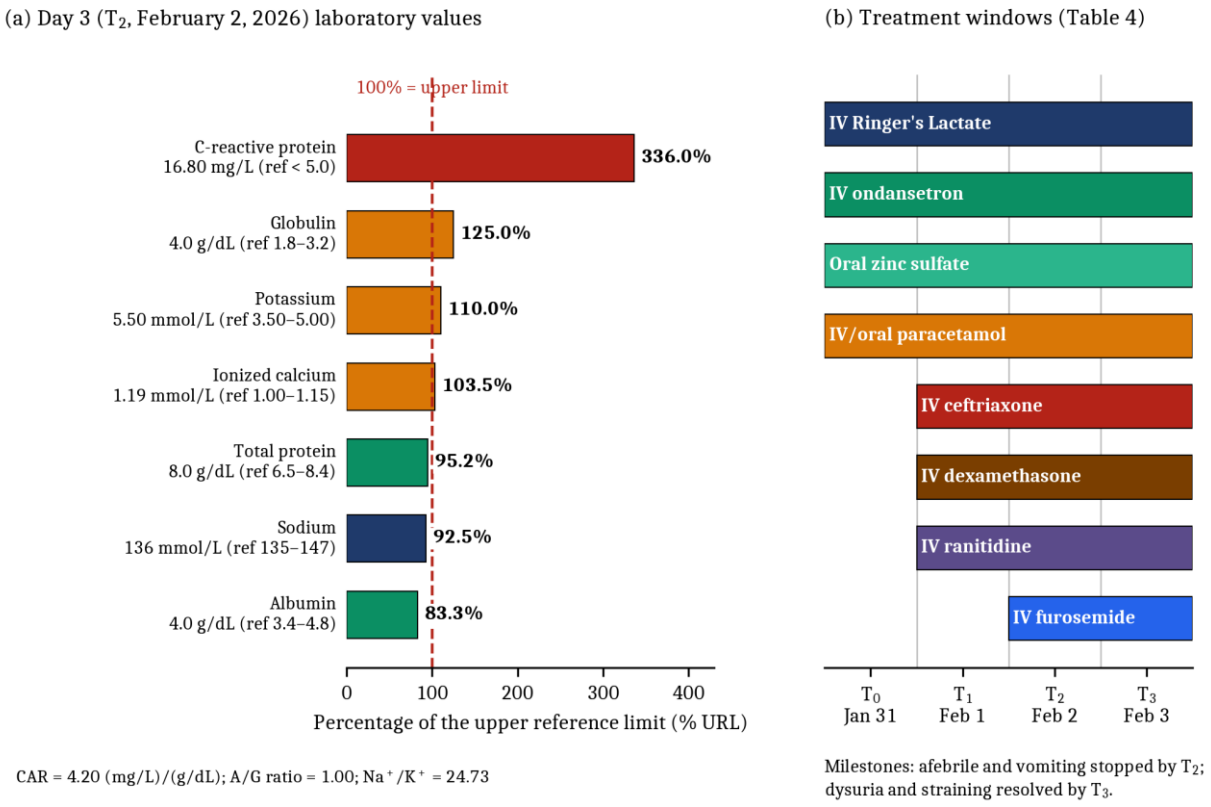


Figure 4. Day 3 Laboratory Deviation Profile and Treatment Windows. (a) Values on February 2, 2026 (T2) as a percentage of the upper reference limit printed on the report. (b) Exposure windows of the eight agents (Table 4) and clinical milestones.

Treatment aimed to eradicate the uropathogen, reverse intravascular dehydration, suppress tubulointerstitial and periurethral inflammation, and lower the borderline-high potassium and ionized calcium. It used a coordinated eight-agent regimen across the Emergency Department and the Pediatric Inpatient Ward. In the Emergency Department (T0) the child received isotonic Ringer's Lactate for immediate volume resuscitation. He also received intravenous ondansetron (0.15 mg/kg/dose; a selective 5-HT3 antagonist) to stop emesis, intravenous paracetamol (10–15 mg/kg/dose) for central antipyresis and analgesia, and oral zinc sulfate (Zn^{2+}, 20 mg/day) to support urothelial and enteric mucosal barrier integrity. After transfer to the ward (T1 to T3), empirical parenteral antibiotic therapy with intravenous ceftriaxone (50–75 mg/kg/day) began. It was paired with short-course intravenous dexamethasone (0.15–0.30 mg/kg/day) to limit NF-κB-mediated tubulointerstitial and preputial-meatal inflammatory edema, and with intravenous ranitidine (1–2 mg/kg/dose q12h) as stress-ulcer prophylaxis during corticosteroid use. On T2 a low dose of intravenous

furosemide (Lasix, 0.5–1.0 mg/kg/dose) was added to inhibit the Na^+-K^+-$2Cl^-$ cotransporter (NKCC2) of the thick ascending limb. The intent was to speed urinary kaliuresis (lowering K^+ from 5.50 mmol/L) and calciuresis (lowering Ca^{2+} from 1.19 mmol/L), and to produce a diuretic washout across the stenotic preputial-urethral tract (Table 4; Figure 4b). Neither potassium nor calcium was measured again, and no urine output is reported.

Table 4. Agents Given, Weight-Based Doses (9.0 kg), Targets, and Documented Adverse Events.

Therapeutic Agent & Route	Clinical Phase & Weight-Normalized Regimen (BW = 9.0 kg)	Primary Pharmacological & Pathophysiological Target	Observed Longitudinal Clinical & Biomarker Response	Adverse events documented
Ringer's Lactate (Isotonic Crystalloid, IV)	Emergency Dept (T0) & Ward Maintenance (~900 mL/24 h Holliday-Segar = 100 mL/kg/day)	Intravascular volume expansion; correction of emesis-induced dehydration (sunken orbits) and renal perfusion restoration	Reversal of sunken eyes, HR reduction from 136 to 104 bpm (Δ% = −23.53%), Hct 33.10% → 32.40% → 33.80%	None documented
Ceftriaxone (Third-Generation Cephalosporin, IV)	Inpatient Ward (T1–T3): Parenteral empirical regimen (50–75 mg/kg/day = 450–675 mg/day IV)	Bactericidal inhibition of penicillin-binding proteins (PBPs) in ascending uropathogenic Enterobacterales within urinary tract	Defervescence (38.6 → 36.8 °C by T3), WBC 10.4 → 5.1 × 10^3/μL at T2 (Δ% = −50.96%); macroscopic clearing of urine; repeat urinalysis not reported	None documented; platelet dip to 191 × 10^3/μL on Day 3 (within the reference interval) with rebound to 323 × 10^3/μL on Day 4
Ondansetron (Selective 5-HT3 Antagonist, IV)	Emergency Dept (T0) & Ward (T1–T3): 0.15 mg/kg/dose (~1.35–1.5 mg IV q8–12h)	Blockade of vagal afferent and area postrema 5-HT3 receptors	100% cessation of vomiting (> 2/day → 0/day by Day 3), enabling transition from parenteral hydration to full oral nutrition	None documented; no ECG reported
Paracetamol (Acetaminophen, IV / Oral)	Emergency Dept (T0) & Ward PRN/Scheduled: 10–15 mg/kg/dose (90–135 mg q6–8h)	Central inhibition of prostaglandin E2 synthesis; analgesia for dysuria	Temperature fell by 1.8 °C; crying at micturition resolved by T3	None documented
Zinc Sulfate (Oral Micronutrient Cofactor, Zn^{2+})	Emergency Dept (T0) & Ward: 20 mg/day elemental Zn^{2+} oral supplementation	Support of urothelial and intestinal barrier function and innate immunity	Appetite restored by T2; an effect on mucosal recovery cannot be shown	None documented
Furosemide (Lasix, Loop Diuretic, IV)	Inpatient Ward (T2–T3): Low-dose parenteral loop diuretic (0.5–1.0 mg/kg/dose = 4.5–9.0 mg IV)	Inhibition of the thick ascending limb Na^+-K^+-$2Cl^-$ cotransporter (NKCC2) with the aim of increasing kaliuresis (K^+ = 5.50 mmol/L) and diuresis	Potassium and ionized calcium not remeasured; urine cleared by T3	None documented; urine output, creatinine and repeat electrolytes not reported
Dexamethasone (Systemic Corticosteroid, IV)	Inpatient Ward (T1–T3): Short-course parenteral anti-inflammatory adjunct (0.15–0.30 mg/kg/day IV)	Anti-inflammatory action through NF-κB and AP-1 transrepression, aimed at renal tubulointerstitial and preputial-meatal edema	Straining and dysuria resolved by T3 (several agents given at once)	None documented
Ranitidine (H2-Receptor Antagonist, IV)	Inpatient Ward (T1–T3): 1–2 mg/kg/dose (9–18 mg IV q12h)	H2-receptor blockade to prevent emesis- and dexamethasone-associated gastroduodenal mucosal erosion	No hematemesis, melena or epigastric distress during corticosteroid and antibiotic therapy	None documented

Abbreviations: 5-HT_3, 5-hydroxytryptamine type 3 receptor; AP-1, activator protein-1; BW, body weight; COX, cyclooxygenase; IV, intravenous; NF-κB, nuclear factor kappa-light-chain-enhancer of activated B cells; NKCC2, sodium-potassium-chloride cotransporter 2; PBP, penicillin-binding protein; PRN, pro re nata.

Adverse Events

No adverse drug reaction was documented during the four days. Platelets fell to 191 × 10^3/μL on Day 3, a value within the 150–400 × 10^3/μL reference interval, and rebounded by +69.11% to 323 × 10^3/μL on Day 4 while ceftriaxone continued. This pattern is more in keeping with an acute-phase change than with drug-induced thrombocytopenia, although it cannot prove that. Table 4 lists the agents and doses. Blood pressure, urine output, creatinine and an electrocardiogram were not reported, so cardiac, renal and electrolyte safety of ondansetron and furosemide could not be assessed from the record.

4. Discussion

Principal Findings

This case study describes a four-day course of acute febrile urinary tract infection together with severe Kikiros Grade 5 distal preputial stenosis in an uncircumcised boy aged 1 year 6 months. In early childhood, recognizing UTI takes a high index of suspicion. Systemic features such as persistent high-grade fever (38.6 °C), repeated vomiting (> 2 episodes/day), anorexia, lethargy and sunken eyes often overshadow local urinary signs and mimic acute viral gastroenteritis.[1–3,15,16] Here, the history and urogenital examination revealed a clear triad of obstructive and irritative features: crying and abdominal straining during micturition, pollakiuria with small voided volumes, and turbid, malodorous urine. Dipstick and microscopic urinalysis corroborated this by showing leukocyturia, bacteriuria and proteinuria together.[3,6,8] Combining these findings with the serial hematologic-inflammatory and biochemical data let us outline a five-tier pathophysiological cascade. It runs from distal preputial stenosis to systemic acute-phase inflammation and a borderline electrolyte disturbance (Figure 5). Only the clinical and laboratory steps were observed; the mechanisms linking them are hypotheses drawn from the literature.

At the anatomical and hydrodynamic level (Tier I of Figure 5), the line between benign physiological preputial adhesions and symptomatic pathologic phimosis is fundamental to pediatric urological decisions.[9–12] Inspection of this 18-month-old showed a severely narrowed distal preputial ring. Its orifice was a pinpoint. The glans penis and external urethral meatus were invisible (Kikiros Grade 5 / Kocaoglu Grade III), although there was no external balanoposthitis erythema, edema or purulent discharge. A prepuce that cannot retract prevents natural desquamative clearance of epithelium and keeps a warm, moist, low-oxygen sub-preputial environment. Such an environment may favor dense colonization by uropathogens. Uropathogenic Escherichia coli expresses mannose-sensitive Type 1 fimbriae (FimH) and mannose-resistant P-fimbriae (PapG).[17] The Hagen-Poiseuille equation ($\Delta P = 8\mu LQ / [\pi r^4]$) shows the hydrodynamic consequence. Narrowing the effective orifice diameter from a normal toddler lumen (~3.5 mm) to a pinpoint (~1.1 mm) raises outflow resistance about 100-fold in the model ($(3.5/1.1)^4 \approx 10^2$). During micturition, detrusor contraction pushes urine into the distensible sub-preputial space faster than it can leave through the stenotic ring. The high back-pressure explains the intense straining, the crying and the fractionated small-volume voids. It also produces turbulent retrograde jets that carry periurethral bacteria into the anterior urethra and bladder.

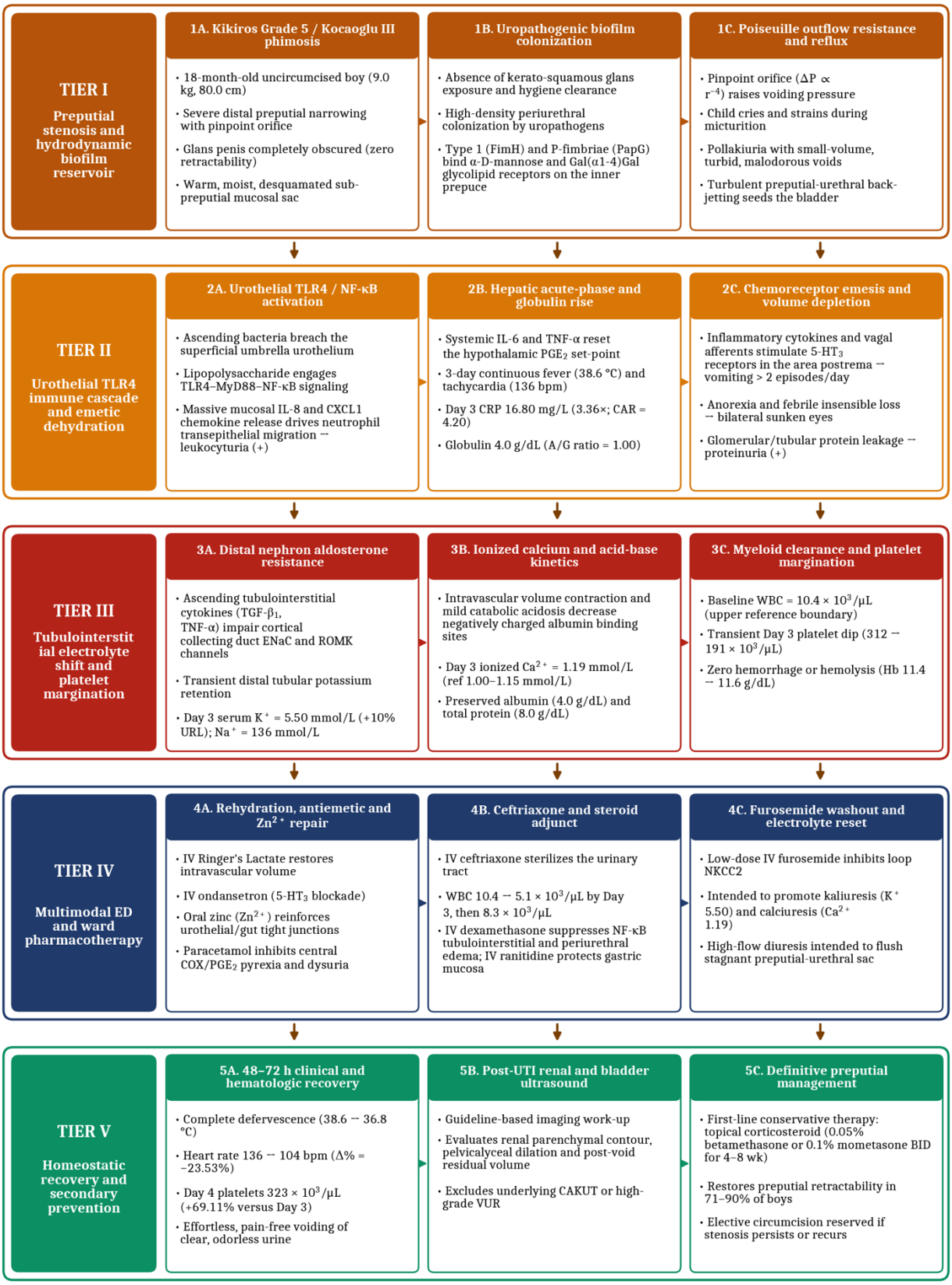


Figure 5. Proposed Five-Tier Pathophysiological and Treatment Cascade in Febrile Urinary Tract Infection With Kikiros Grade 5 Phimosis.

Once uropathogenic bacteria ascend into the bladder and upper urinary tract (Tier II of Figure 5), bacterial lipopolysaccharide and fimbrial adhesins engage urothelial Toll-like receptor 4 (TLR4) and activate the myeloid differentiation primary response 88 (MyD88) and NF-κB transcriptional cascade.[21,22] This innate signal attracts neutrophils across the epithelium into the urinary lumen, seen as leukocyturia and turbid, malodorous urine. It disrupts the tight junctions that govern protein reabsorption in the glomerulus and tubule, seen as proteinuria. It also releases IL-1β, IL-6 and TNF-α into the bloodstream. The systemic footprint of the three-day pre-hospital illness was seen on three biomarker axes. First, the admission leukocyte count was upper-borderline at 10.4 × 10³/μL at T0. It fell by −50.96%

to 5.1 × 10^3/μL by T2, after intravenous ceftriaxone had been started on T1, and then stabilized at 8.3 × 10^3/μL on Day 4. Second, Day 3 C-reactive protein was 16.80 mg/L (3.36 × the 5.0 mg/L upper reference limit; CAR = 4.20 (mg/L per g/dL)). Third, there was polyclonal hyperglobulinemia of 4.0 g/dL (+25.0% above the 3.2 g/dL upper reference limit; A/G ratio = 1.00), with preserved hepatic albumin (4.0 g/dL) and total protein (8.0 g/dL). The transient Day 3 platelet dip from 312 to 191 × 10^3/μL (Δ% = −38.78%) was followed by a +69.11% rebound to 323 × 10^3/μL on Day 4. It may reflect acute-phase platelet margination and later recovery of platelet production; neither was measured.

One biochemical finding on Day 3 (Epoch T2) stood out. It was a borderline-high potassium (serum K^+ = 5.50 mmol/L; +10.0% above the 5.00 mmol/L upper limit printed on the report). It came with a low-normal serum sodium of 136 mmol/L (Na^+/K^+ ratio = 24.73) and an ionized calcium of 1.19 mmol/L (+3.48% above the 1.15 mmol/L limit printed on the report), as shown in Tier III of Figure 5. Marzuillo et al.[23] studied children hospitalized with febrile UTI. Their multicenter cohort came from the Italian Society of Pediatric Nephrology. Isolated hyperkalemia occurred in 6.4%, hyponatremia in 23.0% and full transient secondary pseudohypoaldosteronism (TPHA) in 2.9%, and younger age was the main independent predictor of hyperkalemia. A possible mechanism is ascending renal tubulointerstitial inflammation with local intrarenal synthesis of transforming growth factor-β1 (TGF-β1), IL-1β and TNF-α. These mediators transiently lower mineralocorticoid receptor sensitivity, epithelial sodium channel (ENaC) expression and renal outer medullary potassium (ROMK) channel secretion in the cortical collecting duct. Distal potassium excretion is impaired even before overt hyponatremia develops.[23–25] In addition, emesis-related contraction of intravascular volume (shown by the sunken eyes and by an initial hematocrit of 33.10% that fell to 32.40% after Ringer's Lactate resuscitation) and a mild organic acidemia could alter albumin-calcium binding. Acid-base status was not measured, so this explanation of the ionized calcium of 1.19 mmol/L remains speculative.

Comparison With Previous Studies

To place the clinical features, laboratory course and treatment outcome of this 18-month-old in the wider evidence base, the case was compared with pediatric urology and nephrology guidelines and studies published between 2021 and 2026 (Table 5). The comparison drew on the 2026 EAU/ESPU guidelines by Gnech et al.,[1] the 2026 AAP Clinical Practice Guideline and companion technical reports by Alverson et al.[2] and Forster et al.,[3] the systematic review of phimosis treatment in infant males by Heisinger-Heidler et al.,[9] the 1,550-patient severe phimosis cohort by Zhou et al.,[10] the systemic immune-inflammation marker cohort by Elgormus et al.,[21] the Italian Society of Pediatric Nephrology electrolyte imbalance cohort by Marzuillo et al.,[23] and the adjuvant corticosteroid trial by Da Dalt et al.[29] and the meta-analysis by Gkiourtzis et al.[31] The presentation and the 48- to 72-hour course of this patient were in line with these sources; mechanisms were not tested, and a single case cannot confirm agreement with cohort data.

Table 5. Comparison of the Present Case With Selected Pediatric Febrile UTI, Phimosis, Electrolyte, and Corticosteroid Sources, 2021–2026.

Study & Publication Year	Study Design, Population & Geographic Setting	Preputial / Urological Phenotype & Biomarker Profile	Diagnostic & Therapeutic Interventions	Key Quantitative Outcomes and Comparison With Present Case
Gnech et al.[1] (2026) J Pediatr Urol	EAU/ESPU Pediatric Urology Guideline Update; international pediatric UTI cohorts	Uncircumcised male infants/toddlers with non-retractile prepuce carry markedly elevated febrile UTI risk; pyuria + bacteriuria required	Parenteral cephalosporins for febrile/vomiting children unable to tolerate oral therapy; topical steroids for symptomatic phimosis	In line with our 18-month uncircumcised boy presenting with emesis, fever (38.6 °C), leukocyturia/bacteriuria and Kikiros Grade 5 phimosis treated with IV ceftriaxone
Alverson et al.[2] (2026) & Forster et al.[3] (2026) Pediatrics	2026 AAP Clinical Practice Guideline & Technical Report (children aged 8 days to 5 years)	Emphasizes combining acute urinary symptoms with urinalysis (leukocyturia, bacteriuria, proteinuria) and stewardship-guided antimicrobials	Prompt empirical parenteral therapy in vomiting toddlers with transition to targeted regimens and selective post-UTI ultrasonography	Consistent with the diagnostic findings in our toddler: dysuria, straining, turbid/malodorous urine and positive leukocyturia/bacteriuria/proteinuria
Heisinger-Heidler et al.[9] (2026) / Urology	Systematic review (3,283 abstracts, 4 studies included; male infants with prior UTI and phimosis)	Uncircumcised boys with non-retractile prepuce had 30% (8/27) recurrent UTI rate vs. 7% (2/28) when prepuce became retractile	Topical corticosteroids resolved phimosis in 90% (35/39) vs. 21% (8/39) with Vaseline and reduced recurrent UTI to 0% (0/27) vs. 16% (27/165) in untreated infants	Relevant to our plan: topical corticosteroid therapy or circumcision to prevent recurrence; the authors judge the evidence limited and insufficient for meta-analysis
Zhou et al.[10] (2021) J Paediatr Child Health	Prospective single-center cohort (n = 1,550 boys with Kikiros Grade 4–5 severe phimosis, China)	Severe non-retractile phimosis (Kikiros Grade 4–5) presenting with ballooning, voiding difficulty, or UTI	0.1% mometasone furoate topical ointment twice daily for 4 weeks with gentle preputial retraction hygiene	71.1% response at week 4 and 66.0% long-term success at 26.9 months; Grade 5 did worse than Grade 4
Elgormus et al.[21] (2023) Front Pediatr	Observational diagnostic cohort of pediatric UTI patients, Istanbul, Türkiye	Elevated WBC, CRP, NLR, PLR, and SII significantly predicted bacterial urine culture growth and systemic inflammatory burden	Empirical antimicrobial therapy guided by systemic inflammatory biomarkers and urinalysis	Parallels our patient's baseline WBC (10.4 × 10^3/μL), Day 3 CRP (16.80 mg/L; CAR = 4.20 (mg/L per g/dL)) and globulin (4.0 g/dL)
Marzuillo et al.[23] (2024) Eur J Pediatr	Multicenter Italian Society of Pediatric Nephrology cohort (children hospitalized for febrile UTI)	Evaluated Na^+/K^+ electrolyte imbalances in hospitalized febrile UTI: hyponatremia (23.0%), isolated hyperkalemia (6.4%), concurrent TPHA (2.9%)	Isotonic parenteral fluid resuscitation and parenteral antibiotic eradication of uropathogenic inflammation	Our patient's potassium of 5.50 mmol/L (at the study's 5.5 mmol/L threshold) with sodium 136 mmol/L lies in the range of electrolyte changes described
Da Dalt et al.[29] (2021) & Gkiourtzis et al.[31] (2023) / Pediatr Nephrol	Bayesian analysis of a randomized trial; systematic review and meta-analysis of randomized trials of adjuvant corticosteroids in pediatric febrile UTI	Children aged 2–24 months with febrile UTI at risk for acute tubulointerstitial inflammation and renal cortical scarring	Short-course adjuvant parenteral/oral dexamethasone alongside standard parenteral antibiotics	> 50% Bayesian probability of reducing scarring by up to 20% (Da Dalt) and a significant reduction in scarring in the meta-analysis (Gkiourtzis); faster defervescence is not reported there
Present case (Wibowo et al., 2026) / RSUD K.R.M.T. Wongsonegoro / Univ. Tarumanagara	Case report (n = 1; 18-month-old uncircumcised boy, Semarang/Jakarta, Indonesia)	3-day fever (38.6 °C), emesis, bilateral sunken eyes, Kikiros Grade 5 phimosis, leukocyturia/bacteriuria/proteinuria, CRP 16.80 mg/L, Globulin 4.0 g/dL, K^+ 5.50 mmol/L, Ca^{2+} 1.19 mmol/L	IV Ringer's Lactate, IV ceftriaxone, IV ondansetron, oral Zn^{2+}, IV paracetamol, IV furosemide, IV dexamethasone, IV ranitidine	Complete defervescence (36.8 °C), WBC reduction (−50.96% to 5.1 × 10^3/μL), platelet rebound (191 → 323 × 10^3/μL; +69.11%), and 100% voiding symptom resolution by Day 4

Abbreviations: AAP, American Academy of Pediatrics; CAR, C-reactive protein-to-albumin ratio; CRP, C-reactive protein; EAU/ESPU, European Association of Urology / European Society for Paediatric Urology; IV, intravenous; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; SII, systemic immune-inflammation index; TPHA, transient secondary pseudohypoaldosteronism; UTI, urinary tract infection; WBC, white blood cells.

Recovery took 48 to 72 hours. It shows how a targeted inpatient regimen can be combined to address infection and host homeostasis (Tier IV of Figure 5), although the contribution of each agent cannot be separated. The 2026 EAU/ESPU guidelines,[1] the 2026 AAP Clinical Practice Guideline[2,5] and the Indian Society of Pediatric Nephrology

guidelines[7] agree on parenteral antimicrobial therapy for febrile toddlers with UTI who vomit repeatedly (> 2 episodes/day), are dehydrated (sunken eyes) and cannot retain oral medication. Intravenous ceftriaxone (50–75 mg/kg/day) reaches high bactericidal concentrations in urine and renal parenchyma against community-acquired uropathogenic Enterobacterales. It was followed by a −50.96% drop in peripheral leukocytes (from 10.4 to 5.1 × 10^3/μL) and complete defervescence (from 38.6 to 36.8 °C) within 48 hours.[18,19] Intravenous ondansetron is a selective 5-HT3 receptor antagonist. It acts on vagal and area postrema emetic signaling, and vomiting had stopped completely by Day 3. A smooth change followed from intravenous Ringer's Lactate to oral nutrition. Oral zinc sulfate (Zn^{2+}, 20 mg/day) supplied a catalytic cofactor for repair of urothelial and intestinal tight junctions and for innate cellular immunity.[26–28]

Short-course intravenous dexamethasone, intravenous ranitidine and low-dose intravenous furosemide (Lasix) went beyond standard antimicrobial and rehydration care. They were meant to address two problems: acute tubulointerstitial and periurethral inflammatory edema, and distal tubular potassium retention. On the first, the Bayesian analysis of a randomized trial by Da Dalt et al.[29] reported fewer kidney scars with dexamethasone in children aged 2 to 24 months with febrile UTI (0/7 versus 2/11), with a > 50% Bayesian probability of reducing scarring by up to 20%. The randomized clinical trial by Rius-Gordillo et al.[30] found scarring in 22% of dexamethasone-treated and 21% of placebo-treated children, and the meta-analysis by Gkiourtzis et al.[31] found a significant reduction in kidney scarring. The evidence is therefore mixed. It has also been proposed that short-course adjuvant dexamethasone suppresses NF-κB-mediated urinary IL-6 and IL-8 secretion and shortens fever (mean fever duration reduced from 1.7 to 0.9 days). Ranitidine, given alongside, is intended to protect against corticosteroid- and stress-induced gastroduodenal mucosal injury. In our patient crying and straining at micturition stopped within 48 hours of the start of treatment, but several agents were given at once and no effect can be assigned to dexamethasone alone. On the second problem, low-dose intravenous furosemide on Day 3 inhibits the Na^+-K^+-$2Cl^-$ cotransporter (NKCC2) of the thick ascending limb. It increases sodium and water delivery to the collecting duct, which promotes urinary kaliuresis and calciuresis, and it produces a high-flow washout. Potassium and calcium were not measured again in this child, so the effect of furosemide on them is unknown.

Finer hematological and protein features add to the clinical value of this case. They matter for pediatricians who manage complicated febrile UTI in resource-variable hospitals. From T0 to T3 hemoglobin stayed normal (11.4 to 11.6 g/dL) and so did the erythrocyte count (4.96 to 5.01 × 10^6/μL). Hematocrit was 33.10% at T0, 32.40% at T2 and 33.80% at T3. It lies below the adult laboratory interval printed on the report (40.0–52.0%) but is in the range usually quoted for an 18-month-old. Derived Mean Corpuscular Volume (MCV: 66.73 to 67.47 fL) was below the lower limit printed on the report (70 fL), and Mean Corpuscular Hemoglobin (MCH: 22.98 to 23.82 pg) was at its lower edge, whereas Mean Corpuscular Hemoglobin Concentration (MCHC: 34.32 to 35.80 g/dL) was normal.[21,22] In an 18-month-old moving from liquid milk to solid family foods, an MCV of 66.5–67.5 fL with hemoglobin >= 11.4 g/dL could reflect depleted iron stores or another cause of microcytosis, and ferritin or iron studies would be needed to decide. A possible contributor during the three-day febrile prodrome is IL-6-driven hepatic hepcidin synthesis, which restricts iron export from enterocytes and macrophages. Raised urinary hepcidin and lowered serum iron have been described in infants with febrile UTI.[36] Hepcidin and iron indices were not measured in this child, so this remains a hypothesis.

The proteinuria (+) on Day 2 and the serum protein pattern on Day 3 (total protein 8.0 g/dL, albumin 4.0 g/dL, globulin 4.0 g/dL, A/G ratio = 1.00 and CAR = 4.20 (mg/L per g/dL)) fit transient febrile proteinuria better than a glomerular disease, although no quantitative urine protein was measured. In acute febrile UTI, fever, catecholamine release and proximal tubular exposure to cytokines may transiently impair megalin/cubilin-mediated reabsorption of filtered low-molecular-weight proteins, and leukocytic exudate adds Tamm-Horsfall mucoprotein and neutrophil elastase to the urinary sediment; neither was measured here. Hepatic albumin stayed at 4.0 g/dL and there was no peripheral edema, so a nephrotic mechanism is unlikely. The rise in globulin to 4.0 g/dL (A/G ratio 1.00) is compatible with an acute-phase response, but protein electrophoresis was not done. The aim of care is to interrupt the course from a first febrile UTI in infancy or toddlerhood to recurrent pyelonephritis, renal cortical scarring, secondary hypertension and chronic kidney disease. Four measures serve this aim: treating the bacterial trigger with parenteral ceftriaxone, limiting tubulointerstitial inflammation with short-course dexamethasone, restoring euvolemic renal perfusion with Ringer's Lactate and ondansetron, and arranging structured post-discharge RBUS surveillance and topical corticosteroid therapy for the prepuce.[37–41]

Strengths and limitations

Strengths of this report are the day-by-day record of a complete admission, the weight-based dosing table, the committee approval and the guardian's written consent for participation and publication, and the decision not to apply inferential tests to one patient. The limitations are considerable. This is a single patient, so no finding can be generalized. No urine culture or blood culture is documented, and the sampling method for urine is not stated, so the pathogen and its susceptibility are unknown. Only three blood counts and one chemistry panel exist; CRP, electrolytes and ionized calcium were not measured at admission or at discharge, and the potassium of 5.50 mmol/L was not repeated or checked for hemolysis. Eight agents were started within 72 hours, so recovery cannot be assigned to any one of them or separated from the natural course of the infection. Several laboratory intervals printed on the report are adult intervals, which affects how hematocrit, leukocyte count and ionized calcium are read. No renal imaging was done in hospital, and the post-discharge course is not reported.

The diagnostic limits matter most for the choice of antibiotic and for secondary prevention after discharge (Tier V of Figure 5). Acute febrile UTI was firmly established by the convergence of obstructive and irritative voiding symptoms, systemic pyrexia (38.6 °C), elevated CRP (16.80 mg/L) and urinalysis with leukocyturia, bacteriuria and proteinuria together. However, quantitative urine culture and antimicrobial susceptibility testing were not documented in the hospital record before empirical parenteral ceftriaxone was given.[3,6,8] The 2026 AAP companion technical report on UTI diagnosis by Forster et al.,[3] the WikiGuidelines consensus by Nelson et al.[6] and the Italian Delphi consensus by Autore et al.[8] all treat a catheterized or suprapubic specimen for culture before antimicrobials as the gold standard. It identifies the exact uropathogen and detects extended-spectrum β-lactamase (ESBL)-producing strains, which are increasingly common among male infants and children with urological risk factors.[16,18,19] Even so, the course is reassuring. When empirical ceftriaxone is started in an acutely vomiting toddler, defervescence within 48 hours, resolution of dysuria and a

−50.96% fall in peripheral leukocytes are compatible with a susceptible organism, although they do not identify the pathogen or its susceptibility.[7,15]

Implications

After rapid inpatient recovery, post-acute urological imaging, anatomical differentiation of upper-tract anomalies and secondary prevention of vesicoureteral reflux (VUR) and renal scarring are equally important (Tiers IV and V of Figure 5).[32–35] Beyond standard renal and bladder ultrasonography (RBUS) to screen for hydronephrosis or pelvicalyceal dilation, magnetic resonance urography and pediatric endourological studies stress the need to separate true obstructive uropathy or urolithiasis from non-obstructive calyceal variants such as megacalycosis.[42,43] Randomized trials and long-term cohorts also show that children with high-grade VUR or recurrent febrile pyelonephritis face a higher risk of permanent renal cortical scarring and adult end-stage kidney disease. Preventing ascending reinfection therefore depends on host innate immunity, careful antimicrobial or surgical stewardship, and preputial hygiene or topical corticosteroid therapy.[37–41]

Next comes imaging. Post-acute imaging of the urinary tract is an important part of longitudinal care for an 18-month-old boy with a first febrile UTI, severe Kikiros Grade 5 phimosis and a borderline-high potassium (5.50 mmol/L).[1,4,32–35] In the meta-analysis of 29 studies by Yang et al.,[32] renal and bladder ultrasonography (RBUS) found urinary tract abnormalities in 22.1% of children after a first febrile UTI. Clinically important congenital anomalies of the kidney and urinary tract (CAKUT), such as hydronephrosis, pelvicalyceal dilation, ureterocele or high-grade vesicoureteral reflux (VUR), were found in 3.1%. Marsh et al.[33] studied hospitalized children. Their cohort adds that RBUS performed before discharge, or 2 to 4 weeks after defervescence, avoids false-positive ureteral dilation from acute Escherichia coli endotoxin and also ensures that structural anomalies predisposing to renal cortical scarring are not missed.[34,35,42,43] Third, treating the Kikiros Grade 5 phimosis is recommended to reduce the risk of recurrent UTI.[1,9,13,14] In the 2026 systematic review by Heisinger-Heidler et al.,[9] uncircumcised male infants with a non-retractile prepuce had a 30% (8/27) recurrent UTI rate, against 7% (2/28) in those whose prepuce became retractile. Topical corticosteroid therapy reduced recurrence to 0% (0/27), compared with 16% (27/165) in untreated controls, and resolved phimosis in 90% (35/39) of infants. Zhou et al.[10] prospectively studied 1,550 boys with severe Kikiros Grade 4–5 phimosis. Twice-daily 0.1% mometasone furoate ointment for 4 weeks gave a 71.1% response at week 4 and a 66.0% long-term success rate over a mean follow-up of 26.9 months, and success was lower in Grade 5 than in Grade 4 phimosis. Accordingly, post-discharge care for our patient prioritizes a 4- to 8-week course of topical corticosteroid (0.05% betamethasone valerate or 0.1% mometasone furoate twice daily with gentle preputial retraction hygiene), together with outpatient RBUS screening. Elective circumcision or preputioplasty is reserved for persistent preputial stenosis or recurrent urinary tract infection.[11,12,44,45] Topical corticosteroids may increase complete resolution of phimosis after four to eight weeks of treatment,[44] and a betamethasone valerate regimen twice daily has been tested in a randomized trial.[45]

5. Conclusion

This single-patient case study describes a 1-year-6-month-old uncircumcised boy with febrile urinary tract infection and severe Kikiros Grade 5 distal preputial stenosis. His course included obstructive voiding distress (crying, straining and small-volume pollakiuria with turbid, malodorous urine), vomiting with mild dehydration, febrile tachycardia at 136 beats/min, a CRP of 16.80 mg/L (CAR 4.20 (mg/L per g/dL)) with globulin of 4.0 g/dL, and a borderline-high potassium of 5.50 mmol/L with sodium of 136 mmol/L.

Management combined isotonic Ringer's Lactate rehydration, intravenous ondansetron, oral zinc sulfate, paracetamol, intravenous ceftriaxone, short-course dexamethasone with ranitidine gastroprotection, and low-dose furosemide. Recovery followed. Defervescence was complete (38.6 to 36.8 °C), leukocytes changed from 10.4 to 5.1 × 10^3/µL and then 8.3 × 10^3/µL, platelets rebounded from 191 to 323 × 10^3/µL (Δ% = +69.11%), and obstructive dysuria resolved fully within 96 hours. No single agent can be credited with this recovery. Beyond acute treatment, three measures are advisable to reduce the risk of recurrent urinary tract infection and renal scarring. Genital examination is advisable in every febrile male toddler.[1,3] Renal and bladder ultrasonography after discharge should exclude underlying congenital anomalies.[1,4,32,33] Topical corticosteroid therapy, or elective circumcision when it fails, is first-line for symptomatic phimosis.[9,10,44,45]

Declarations

Ethics approval and consent to participate

This single-patient case study was reviewed and approved by the CMHC Research Center Ethics Committee, Indonesia (protocol approval number 2026/0429), and was conducted in accordance with institutional clinical governance at RSUD K.R.M.T. Wongsonegoro Semarang, the Faculty of Medicine, Universitas Tarumanagara, and the Declaration of Helsinki. Written informed consent for clinical participation and data collection was obtained from the patient's mother (legal guardian).

Trial registration

Not applicable.

Consent for publication

Written informed consent was obtained from the patient's mother for the publication of anonymized clinical data, serial laboratory records and the genital examination photograph in an open-access medical journal. Upper peripheral and caregiver-identifying features in the photograph were permanently masked with a solid black bar in line with ICMJE, COPE and Declaration of Helsinki privacy standards.

Data availability

All clinical, anthropometric, urinalysis and serial hematological and biochemical data generated and analyzed during this study are included in this article, its tables (Tables 1–5) and its figures (Figures 1–5).

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Conflict of interest

The authors declare no conflicts of interest.

Author contributions

H.P.W.: Conceptualization; Investigation; Data curation; Formal analysis; Visualization; Writing – original draft. Z.H.: Investigation; Resources; Writing – review & editing. H.: Supervision; Validation; Methodology; Writing – review & editing. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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Use of artificial intelligence

No generative artificial intelligence tools were used in the preparation of this manuscript.

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