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Review Article

estimated glomerular filtration rate monitoring in the Management of acute kidney injury among pediatric nephrology patients

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Abstract

Background: Acute kidney injury (AKI) affects more than a quarter of critically ill children and is now recognized as a driver of long-term chronic kidney disease (CKD) rather than a self-limited event [2,27].

Objective: This review synthesizes the evidence on estimated glomerular filtration rate (eGFR) monitoring, its estimating equations, complementary injury biomarkers, and its role in AKI staging and long-term surveillance among pediatric nephrology patients.

Methods: We synthesized epidemiologic cohort studies, equation-validation studies, biomarker studies, and systematic reviews addressing pediatric AKI definitions, GFR-estimating equations, tubular injury biomarkers, and post-AKI renal outcomes.

Findings: Creatinine-based eGFR, most commonly the revised Schwartz equation, remains the practical monitoring backbone but lags true GFR change during acute injury [7]; cystatin C-based measures and biomarkers such as NGAL detect injury 24-48 hours earlier in specific settings [18,21], and structured post-discharge eGFR follow-up detects substantial persistent CKD risk after apparent recovery [25,26].

Conclusions: Pediatric nephrology services are best served by combining consistent KDIGO-based staging, an appropriately chosen and, where feasible, cystatin C-augmented eGFR equation, targeted biomarker use in defined high-risk exposures, and scheduled long-term eGFR follow-up.

Keywords: glomerular filtration rate; acute kidney injury; pediatric nephrology; chronic kidney disease; biomarkers

1. Introduction

Acute kidney injury (AKI) is common among hospitalized children and is no longer viewed as a self-limited, reversible event but as an exposure that measurably raises the long-term risk of chronic kidney disease (CKD) and death. Multinational cohort data from the Assessment of Worldwide Acute Kidney Injury, Renal Angina and Epidemiology (AWARE) study, whose design was described by Basu and colleagues, found that AKI, defined by Kidney Disease: Improving Global Outcomes (KDIGO) creatinine and urine-output criteria [1], occurred in more than a quarter of critically ill children, with severe AKI independently associated with higher mortality [2,3]. Sutherland and colleagues, analyzing a large national cohort of hospitalized non-ICU and ICU children, showed this burden is not confined to intensive care [4]. Neonatal AWAKEN cohort data extended the observation to the youngest patients, linking early-life AKI to longer stay and higher mortality [5], and Lameire and colleagues, summarizing the global epidemiology of AKI, argued that under-recognition at the bedside, not true rarity, is the principal barrier to earlier intervention [6].

Estimated glomerular filtration rate (eGFR), derived from serum creatinine using a pediatric-specific equation, is what nephrology services use to translate a creatinine value into an interpretable index of kidney function, and serial eGFR trend tracks that function through an AKI episode and beyond. The bedside Schwartz equation and its 2009 update, validated in the Chronic Kidney Disease in Children (CKiD) cohort, remain the most widely used pediatric formulas because they require only height and creatinine [7,8]. Yet the same creatinine dependence that makes eGFR practical also makes it a lagging indicator during acute injury, since creatinine accumulates over one to two half-lives before a rise is apparent, a delay that has driven biomarker research aimed at detecting tubular injury earlier [9]. This review synthesizes the evidence on eGFR monitoring, its estimating equations, its integration with biomarkers, and its role in AKI staging, fluid

management, and long-term surveillance among pediatric nephrology patients.

2. Methods

We synthesized peer-reviewed literature on GFR estimation, AKI definitions and staging, AKI biomarkers, and post-AKI renal outcomes in children, drawing on epidemiologic cohorts, equation-validation studies, quality-improvement intervention studies, and systematic reviews from the major nephrology, critical care, and pediatrics literature. Search terms combined "acute kidney injury," "estimated glomerular filtration rate," "pediatric," "creatinine," "cystatin C," "neutrophil gelatinase-associated lipocalin," and "chronic kidney disease" with study names (AWARE, AWAKEN, CKiD) that anchor this literature. Priority was given to studies reporting quantitative AKI incidence, biomarker lead time, or post-AKI outcome rates, since these figures are what a nephrology service needs to calibrate an eGFR-monitoring protocol. No formal risk-of-bias scoring or meta-analytic pooling was performed; findings are presented narratively and, where a study reported concrete rates, summarized in tables and figures.

3. Results

Epidemiology and staging of AKI in pediatric nephrology practice

Consistent staging is the precondition for any eGFR-monitoring protocol, because a creatinine-based eGFR trend is only actionable once mapped onto a shared severity framework. The pediatric modification of the RIFLE criteria (pRIFLE), introduced by Akcan-Arikan and colleagues, adapted the adult staging system to pediatric creatinine clearance and urine output and remains foundational to subsequent KDIGO pediatric staging [10]. Zappitelli and colleagues showed that reported AKI incidence in a pediatric ICU cohort varied substantially depending on which creatinine-based definition and baseline reference were applied, a finding directly relevant to benchmarking a service's own eGFR-based surveillance

data [11]. Sutherland and colleagues compared pRIFLE, AKIN, and KDIGO definitions directly in a large hospitalized pediatric cohort and found meaningful discordance in classified stage for the same patient [12]. In the AWARE cohort, severe AKI (KDIGO stage 2-3) occurred in roughly one in nine critically ill children and carried an independent mortality signal, giving nephrology teams a concrete benchmark [2]. Hui-Stickle and colleagues, describing an earlier single-center pediatric ARF cohort, showed that the case mix of pediatric AKI had already shifted from primary renal disease toward AKI as a complication of systemic illness and multiorgan support [13]. Renal angina scoring, combining early risk factors with small changes in fluid balance and creatinine, was derived and validated by Basu and colleagues to flag, ahead of a diagnostic creatinine rise, children who warrant intensified eGFR and biomarker surveillance [14].

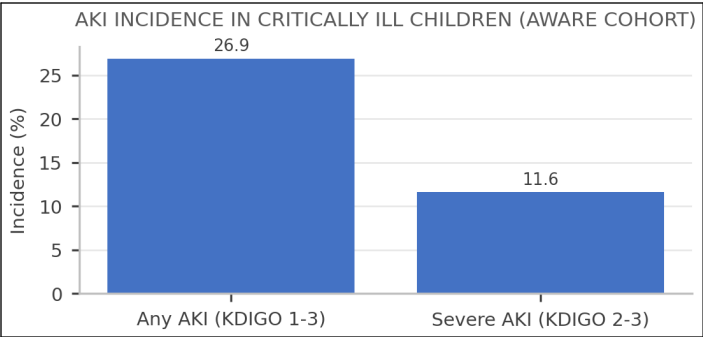


Figure 1: Incidence of any acute kidney injury and of severe (KDIGO stage 2-3) acute kidney injury among critically ill children and young adults in the multinational AWARE cohort [2].

eGFR estimating equations in children

Because pediatric AKI monitoring is built on serial GFR estimates, the equation used matters as much as testing frequency. The revised Schwartz bedside equation, calibrated against enzymatic creatinine assays and validated in the CKiD cohort, corrected the overestimation produced by the original 1976 formula and remains embedded in most pediatric electronic health record eGFR calculators [7]. Schwartz and Work's review of GFR measurement and estimation in children noted that creatinine-based equations perform less reliably at

extremes of muscle mass, during critical illness, and immediately after an AKI episode, before creatinine has re-equilibrated with true GFR [15]. Cystatin C, a low-molecular-weight protein with less dependence on muscle mass than creatinine, was reviewed by Filler and colleagues as a complementary marker, and combined creatinine-cystatin C equations have improved estimation precision across the pediatric CKD spectrum [16]. Pottel and colleagues derived a single eGFR equation spanning infancy to adulthood using height-independent creatinine and cystatin C terms, addressing the discontinuous transition between pediatric and adult formulas as adolescents move into adult care [17]. In the acute setting, Herget-Rosenthal and colleagues showed serum cystatin C rose one to two days before creatinine in patients developing AKI, and Bagshaw and Bellomo's review concluded this earlier signal, and cystatin C's smaller dependence on volume status, gives it a meaningful advantage over creatinine-based eGFR alone for early detection [18,19].

Table 1: Pediatric GFR-estimating equations in common clinical use, their required inputs, and their principal limitation for AKI monitoring [7,15,16,17].

Equation	Marker(s)	Inputs	Principal limitation in AKI
Bedside Schwartz (2009)	Creatinine	Height, serum creatinine	Lags true GFR change during acute injury/recovery
Combined creatinine-cystatin C	Creatinine + cystatin C	Height, creatinine, cystatin C	Requires cystatin C assay, not universally available
Full age spectrum (FAS)	Creatinine and/or cystatin C	Age, creatinine and/or cystatin C	Continuity gain across pediatric-adult transition, not injury-specific
Cystatin C alone	Cystatin C	Serum cystatin C	Earlier signal than creatinine but less standardized across assays

Biomarkers and early detection of AKI

The core limitation of a creatinine-based eGFR trend during acute injury is temporal lag, and much of the past

two decades of pediatric AKI research has focused on tubular injury biomarkers that rise before creatinine does. Devarajan's review established neutrophil gelatinase-associated lipocalin (NGAL) as one of the earliest, most extensively validated markers of tubular injury in children [9], and Zappitelli and colleagues confirmed in a critically ill cohort that urine NGAL rose well before the diagnostic creatinine threshold was met [20]. In cardiac surgery, where the timing of the nephrotoxic insult is precisely known, Krawczeski and colleagues showed NGAL measured hours after bypass predicted subsequent AKI well before creatinine rose [21], and Parikh and colleagues found a panel combining NGAL with other tubular markers improved prediction of AKI and downstream outcomes beyond postoperative creatinine trend alone [22]. Basu and colleagues showed combining a functional marker (eGFR change) with a tubular damage biomarker improved diagnostic precision for AKI after pediatric cardiac surgery beyond either marker alone, supporting a combined-marker strategy over eGFR trend in isolation [23]. Bhatt and Das's systematic review concluded that NGAL, alongside markers such as kidney injury molecule-1 and interleukin-18, consistently shortens the interval to detection relative to creatinine-based eGFR, though none has yet fully replaced serial eGFR monitoring in routine practice, largely for reasons of assay standardization and cost [24].

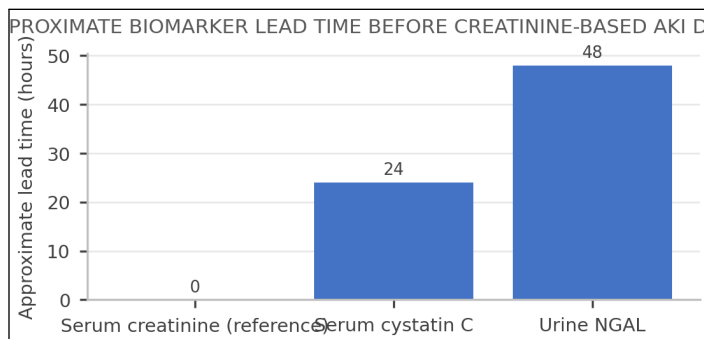


Figure 2: Approximate lead time by which a rise in urine NGAL or serum cystatin C precedes the diagnostic rise in serum creatinine/eGFR after a defined renal insult, relative to creatinine as the zero-hour reference [20,21,18].

Long-term renal outcomes after AKI

Because AKI is now understood as a risk factor for CKD rather than a fully reversible event, eGFR monitoring extends well past discharge. Askenazi and colleagues followed children three to five years after acute renal failure and found a substantial minority with persistently reduced eGFR, proteinuria, or hypertension, establishing the basis for structured post-AKI follow-up [25]. Mammen and colleagues, in a prospective ICU cohort followed one to three years, found a majority of survivors had at least one marker of kidney damage or reduced eGFR, and a clinically significant minority met formal CKD criteria [26]. Greenberg, Coca, and Parikh's systematic review pooling pediatric AKI follow-up cohorts confirmed a consistently elevated pooled risk of CKD and mortality after AKI, despite heterogeneity in follow-up duration and CKD definition [27]. Goldstein and Devarajan's review of AKI-to-CKD progression argued that this heterogeneity is not reassuring, since even the most conservative studies still detected excess risk, and proposed structured eGFR surveillance as standard pediatric nephrology aftercare [28]. Menon and colleagues sharpened this picture by linking high nephrotoxic medication exposure specifically to AKI that was, in turn, associated with CKD at six months, tying eGFR trend to a defined, modifiable exposure rather than illness severity alone [29].

4. Discussion

Three practical implications follow. First, eGFR monitoring must be interpreted against a consistent staging framework, since the incidence and severity distribution a service reports depends materially on which creatinine-based definition is applied [10,11,12]. Adopting KDIGO staging consistently gains comparability with the largest published cohorts, including AWARE and AWAKEN, letting a service benchmark its own severe-AKI rate and mortality signal [2,5]. Second, the choice of GFR-estimating equation is not neutral: the revised Schwartz equation is adequate for most bedside monitoring, but its creatinine dependence means it lags

true GFR change during acute injury and recovery, and incorporating cystatin C meaningfully shortens that lag where feasible [7,15,16,18,17]. Nephrotoxic medication surveillance illustrates how eGFR trend becomes actionable: Goldstein and colleagues showed that electronic health record identification of high-risk nephrotoxin exposure, paired with mandated creatinine/eGFR checks and a subsequent quality-improvement program, measurably reduced nephrotoxic-medication-associated AKI [30,31].

Table 2: Long-term renal outcome signals reported by pediatric AKI follow-up cohorts and the corresponding follow-up interval [25,26,27,29].			
Study	Cohort	Follow-up	Key renal outcome signal
[25]	Pediatric acute renal failure survivors	3-5 years	Substantial minority with reduced eGFR, proteinuria, or hypertension
[26]	Pediatric ICU AKI survivors	1-3 years	Majority with kidney-damage marker; clinically significant CKD minority
[27]	Pooled pediatric AKI cohorts (systematic review)	Variable	Consistently elevated pooled CKD and mortality risk vs. no-AKI
[29]	High nephrotoxic medication exposure with AKI	6 months	AKI from nephrotoxin exposure associated with incident CKD

Third, biomarker-augmented monitoring and fluid management deserve a defined but bounded role alongside eGFR trend, not a wholesale replacement. NGAL and related tubular injury markers reliably detect injury before eGFR falls, valuable in time-critical settings such as cardiopulmonary bypass, but no single biomarker yet has the availability, standardization, and outcome validation to replace serial eGFR as the monitoring backbone [20,21,22,24]. Fluid balance is a further confounder nephrology teams must track alongside the number itself: Sutherland and colleagues' analysis of the prospective pediatric continuous renal replacement therapy registry found fluid overload at initiation independently associated with mortality, and Selewski and colleagues

extended this to children on extracorporeal membrane oxygenation requiring renal replacement therapy [32,33]. Because dilution from positive fluid balance can mask a true GFR fall, and because Goldstein and colleagues' early hemofiltration outcomes showed smaller fluid overload at initiation associated with better survival, an eGFR protocol that ignores fluid balance risks underestimating injury severity in the sickest children [34]. The ADQI 16 consensus on acute kidney disease and renal recovery, and Lameire and colleagues' recognition that AKI is a growing global concern, together support treating eGFR trend, biomarker data, and fluid balance as an integrated triad through both the acute admission and the post-discharge surveillance period where CKD risk accrues [35,6,27,36].

5. Conclusion

Estimated glomerular filtration rate, calculated from serial serum creatinine using pediatric-specific equations and refined where possible with cystatin C, remains the practical backbone of AKI monitoring in pediatric nephrology because it is inexpensive, widely available, and comparable across the large epidemiologic cohorts that define current staging and outcome benchmarks [7,2]. Its principal limitation, a lag between true GFR change and the creatinine signal, is real but bounded: tubular injury biomarkers such as NGAL shorten that lag in specific high-risk settings, fluid balance tracking prevents dilutional misclassification, and structured post-discharge eGFR surveillance captures the CKD risk that persists after apparent recovery [9,32,25,26]. Services that combine consistent KDIGO-based staging, an appropriately chosen and, where possible, cystatin C-augmented eGFR equation, targeted biomarker use in defined high-risk exposures, and scheduled long-term eGFR follow-up are best positioned to translate this evidence into measurably better outcomes for children with, or at risk of, acute kidney injury.

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