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SYSTEMATIC REVIEW AND META-ANALYSIS

Acute Kidney Injury and Rapidly Progressive Glomerulonephritis in Acute Post-Streptococcal and Post-Infectious Glomerulonephritis: Pooled Prevalence, Age-Related Risk and Prognostic Determinants — A Systematic Review and Meta-Analysis

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ABSTRACT

Background: Acute post-streptococcal glomerulonephritis (APSGN) commonly causes acute nephritic syndrome in children, yet how often it causes acute kidney injury (AKI) or a rapidly progressive course is unquantified.

Objective: To pool the prevalence of AKI, kidney replacement therapy (KRT) and rapidly progressive or crescentic glomerulonephritis (RPGN), and to explain the variation between cohorts.

Methods: PubMed and Europe PMC were searched. Observational studies reporting AKI, KRT or RPGN in post-streptococcal or post-infectious glomerulonephritis were eligible. Prevalence was pooled by random-intercept logistic regression, determinants as odds ratios. Risk of bias used ROBINS-E, certainty GRADE.

Result: Thirty studies were included, 26 pooled. How a study defined AKI dominated its reported risk: 62.3% (95% CI 44.7-77.2) under a bare creatinine or eGFR threshold, 35.0% (21.3-51.7) under consensus criteria and 16.0% (8.6-27.8) where none was stated (p = 0.0001). Definition class and income setting explained 66.7% of variance. Across 21 cohorts AKI complicated 34.7% (23.1-48.5) of presentations (n = 2,454; I2 = 95.2%; prediction interval 3.2-89.4%), rising to 49.6% (35.5-63.7) in the 13 cohorts stating a definition. KRT was required in 3.3% (1.4-7.8) and RPGN in 6.8% (3.4-13.2). Among children each year of age raised the odds of an adverse outcome by 17% (OR 1.17, 1.09-1.24); heavy proteinuria gave OR 2.66 (1.50-4.73). Certainty was very low.

Conclusion: Reported AKI risk depends more on how a centre defines AKI than on the population served. Between a third and a half of children develop AKI; severe outcomes are uncommon. A minimum reporting standard is proposed.

1. Introduction

Acute post-streptococcal glomerulonephritis (APSGN) is the immune-complex nephritis that follows infection of the throat or skin with nephritogenic strains of group A β-haemolytic *Streptococcus*. It remains the commonest cause of acute nephritic syndrome in children worldwide. The absolute burden is not trivial and is starkly unequal: population-based surveillance in Auckland recorded 52 cases of severe acute group A streptococcal disease per 100,000 population per year, of which the immune-mediated sequelae form part; childhood APSGN incidence in Far North Queensland was 27 per 100,000 person-years overall but 104 per 100,000 among Aboriginal and Torres Strait Islander children; and a nationwide Montenegrin series estimated approximately four cases per 100,000 children per year[1-3]. Crowding, scabies and pyoderma sustain transmission in low- and middle-income settings, and streptococcal serology reference intervals differ enough between populations that the same titre carries different meaning in different places[4]. Although the disease has largely receded from high-income populations it has not disappeared: outbreaks continue to be declared in Indigenous Australian communities and in remote First Nations populations of North America, a marked post-pandemic resurgence has been documented in central Europe, and the reverse pattern — a fall in incidence after the coronavirus disease 2019 pandemic — has been reported in New Zealand[5-8].

Cohorts describing this single disease apply mutually incompatible criteria for its commonest complication. Some use Kidney Disease: Improving Global Outcomes (KDIGO), Acute Kidney Injury Network (AKIN) or paediatric RIFLE definitions; some use a single fixed creatinine threshold applied irrespective of the child's age or size; and some report acute kidney injury as a complication without stating any criterion at all[9-12]. A standardised case definition for epidemiological surveillance of APSGN has been proposed, but it is not yet widely applied and it does not prescribe how kidney injury within a case should be graded[13]. The consequence is that two units reporting different rates of acute kidney injury may be describing the same disease with different instruments, and neither can tell.

That measurement problem sits on top of a clinical one. The teaching that has accompanied APSGN for half a century is that the acute episode is dramatic but self-limiting and that children recover completely. That teaching sits uneasily beside what individual cohorts report. Acute kidney injury is described in as few as 2% and as many as 94% of admitted children depending on the series consulted[14,15]. A minority progress to a rapidly progressive or crescentic course requiring immunosuppression, and a smaller minority require dialysis or reach kidney failure[16-18]. Twenty-year follow-up of an epidemic cohort in Brazil found appreciable chronic kidney disease and hypertension long after the acute illness resolved, and paediatric cohorts report persistent proteinuria and impaired kidney function at three months[19,20]. Understanding of the underlying immunology has meanwhile advanced considerably, with complement-pathway variants, anti-factor B and anti-C3b autoantibodies and overlap with C3 glomerulopathy now recognised in atypical and persistent disease[21-24]. None of that progress has been matched by an agreed way of counting the commonest complication.

The evidence base has never been synthesised quantitatively. Systematic reviews of APSGN exist, but they address the coexistence of acute rheumatic fever or the epidemiology of specific complications, and none pools a clinical outcome[25]. No published meta-analysis states how often the disease is complicated by acute kidney injury, by dialysis, or by a rapidly progressive course; nor has any synthesis tested whether the variation between cohorts is explained by patient factors, by geography, or by measurement. Clinicians counselling a family at admission, and services planning paediatric dialysis capacity, are working from single-centre figures whose comparability is unknown.

The novelty of this study lies in being the first quantitative synthesis of clinical outcomes in acute post-streptococcal and post-infectious glomerulonephritis, and in showing that the single largest determinant of the reported risk of acute kidney injury is not the population studied but the definition the investigators applied — a measurement effect that is quantified here, separated from geography by multivariable meta-regression, shown to outweigh every patient-level moderator tested, and translated into a concrete minimum reporting standard for future cohorts. **The aim of this study was to** estimate the pooled prevalence of acute kidney injury, kidney replacement therapy and rapidly progressive or crescentic glomerulonephritis in this disease, to identify the study-level factors that explain the heterogeneity between reports, and to pool the prognostic determinants of adverse kidney outcome that are reported on a common scale.

2. Methods

Design and reporting

This systematic review and meta-analysis was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement[26]. The review was not prospectively registered. A protocol defining the population, outcomes, moderators and planned sensitivity analyses was fixed before pooling.

Eligibility criteria

Studies were eligible if they enrolled patients of any age with a clinical or biopsy-based diagnosis of acute post-streptococcal glomerulonephritis or acute post-infectious glomerulonephritis, and reported an extractable numerator and denominator for at least one of three outcomes: acute kidney injury, kidney replacement therapy (KRT), or rapidly progressive or crescentic glomerulonephritis (RPGN). Observational designs of any type were admitted, because no randomised evidence exists in this field. Narrative reviews, treatment updates, letters, editorials, modelling studies, case reports and series of fewer than ten patients were excluded, as were studies of glomerulonephritis of non-streptococcal or unspecified non-infectious aetiology and studies reporting an outcome only as a population incidence rate.

The minimum series size of ten was set a priori on the grounds that the width of an exact binomial interval below that size makes a study's contribution to a pooled proportion negligible while its leverage on the between-study variance is disproportionate. The threshold is nevertheless low, and two contributing cohorts have denominators of 17 and 11; the pre-specified sensitivity analysis excluding cohorts with fewer than 50 participants covers both, and the influence diagnostics reported below test their leverage directly.

Information sources and search strategy

PubMed and Europe PMC were searched using two complementary strategies in each database: a broad-concept strategy combining controlled and free-text terms for post-streptococcal, post-infectious and infection-related glomerulonephritis with terms for acute kidney injury, dialysis, kidney replacement therapy, rapidly progressive glomerulonephritis, cohort and outcome; and a title-anchored strategy restricting the disease terms to the title field in order to surface cohort reports whose abstracts do not index the outcome. Complete identifier lists were downloaded for all four searches and de-duplicated programmatically, so that the counts in the flow diagram are exact rather than estimated. Reference lists of retrieved cohorts and of the existing narrative systematic reviews were hand-searched. No language restriction was applied; one included report is in French[10]. Authors of reports with missing numerators were not contacted; this is a limitation of the review process and is stated as such below.

Study selection and data extraction

Records were screened on title and abstract and then in full text. For each included study the following were extracted: country, income setting, endemic or sporadic APSGN burden in the source population, study design, recruitment period, case ascertainment route, age stratum, the aetiological label the authors themselves used, analysed denominator, event counts for each outcome, and the verbatim definition of acute kidney injury applied. Where a full text was available in PubMed Central or Europe PMC it was used in preference to the abstract. Values that could not be located were coded as not reported and the study was excluded from the affected pool rather than imputed.

Outcomes and definitions

The primary outcome was the prevalence of acute kidney injury during the acute episode. Secondary outcomes were the prevalence of kidney replacement therapy (dialysis) and the prevalence of rapidly progressive or crescentic glomerulonephritis. Kidney replacement therapy is used as the outcome label throughout; the word dialysis is retained only where a source study defined its population by that requirement or where the clinical sense requires it. The last of these combines a clinical syndrome with a histological finding, and the two are not equivalent: crescentic glomerulonephritis is verified on biopsy, whereas rapidly progressive glomerulonephritis is a clinical diagnosis made without biopsy in most of the contributing cohorts. Because biopsy is performed selectively, this composite is under-ascertained by an unknown margin, and it is rated down for indirectness accordingly.

Because acute kidney injury is defined inconsistently across this literature, each study was additionally classified into one of three definition classes — consensus criteria (KDIGO, AKIN or paediatric RIFLE), a bare creatinine or estimated glomerular filtration rate threshold, or unspecified — and this classification was carried forward as the principal moderator rather than treated as a nuisance. The unspecified class is not a measurement strategy but a missing-data category, and is interpreted as such throughout.

Risk of bias assessment

Risk of bias was assessed with the ROBINS-E tool across its seven domains: confounding, measurement of the exposure, selection of participants, post-exposure interventions, missing data, measurement of the outcome, and selection of the reported result[27]. ROBINS-E was chosen in preference to the Cochrane RoB 2.0 tool because every included study is observational and RoB 2.0 applies only to randomised trials.

Statistical analysis

Prevalence was pooled as a proportion using a random-intercept logistic regression model — a binomial-normal generalised linear mixed model with a logit link and maximum-likelihood estimation of τ^2 — and back-transformed for presentation. This estimator was chosen in preference to the Freeman–Tukey double-arcsine transformation because study sizes span 11 to 400 participants and several observed proportions lie close to 0 or 1, where the double-arcsine transformation is unstable and its back-transformation is ill-defined; the Freeman–Tukey result is reported alongside every primary estimate as a comparator. Exact Clopper–Pearson intervals were computed for individual studies. A standardised mean difference was not computed: all three endpoints are binary and none of the included single-arm cohorts has a

comparator group, so Hedges' g is mathematically undefined for these data.

Heterogeneity was quantified by τ^2, by I^2 with its 95% confidence interval, and by Cochran's Q. A 95% prediction interval derived from the t-distribution accompanies every pooled estimate, because with heterogeneity of the magnitude seen here the confidence interval around the mean does not describe the range a clinician should expect in the next setting. Moderators were examined by subgroup analysis and by mixed-effects meta-regression on the logit scale, with candidate models compared by the corrected Akaike information criterion and by incremental likelihood-ratio tests. Definition class, income setting, case ascertainment and age stratum were pre-specified moderators; endemic-versus-sporadic burden was a pre-specified alternative to income setting; publication year and study size were exploratory. No adjustment was made for multiplicity across the moderator tests, and the p values should be read accordingly.

Prognostic determinants reported by at least three studies on a common scale were pooled as odds ratios by generic inverse-variance weighting on the log scale with restricted maximum-likelihood estimation. Robustness was examined by leave-one-out analysis across every contributing cohort, by two alternative pooling methods, and by a pre-specified set of restricted analyses. Influence was assessed by studentised residuals with a Bonferroni-corrected two-sided outlier test, Cook's distance, DFFITS, hat values and Baujat contributions. Small-study effects were examined by Peters' regression on the inverse of the sample size, which is the appropriate test for proportion and binary outcomes, with Egger's regression, Begg's rank correlation and trim-and-fill reported alongside. Analyses were performed in R version 4 using the *meta* and *metafor* packages.

Certainty of the evidence

Certainty was rated using the GRADE framework adapted for prevalence and prognostic-factor evidence. All bodies of evidence started at low certainty because every included study is observational, and were then rated down for risk of bias, inconsistency, indirectness, imprecision and publication bias as applicable.

3. Results

Study selection

The four searches retrieved 1,435 records. After removal of 112 within-source and 377 cross-source duplicates, 946 unique records were screened on title and abstract and 866 were excluded. Eighty reports were assessed in full text — 77 from the searches and three identified through citation chasing — and 50 were excluded with reasons, most commonly because they were narrative reviews or treatment updates (n = 17) or because no outcome numerator could be extracted (n = 12). Thirty studies met the inclusion criteria, of which 26 contributed to at least one pooled estimate. Adding Europe PMC to PubMed was not redundant: 294 of the 946 unique records came from Europe PMC alone, and five of the 21 cohorts contributing to the primary outcome were identified only through that source. The selection process is shown in Figure 1.

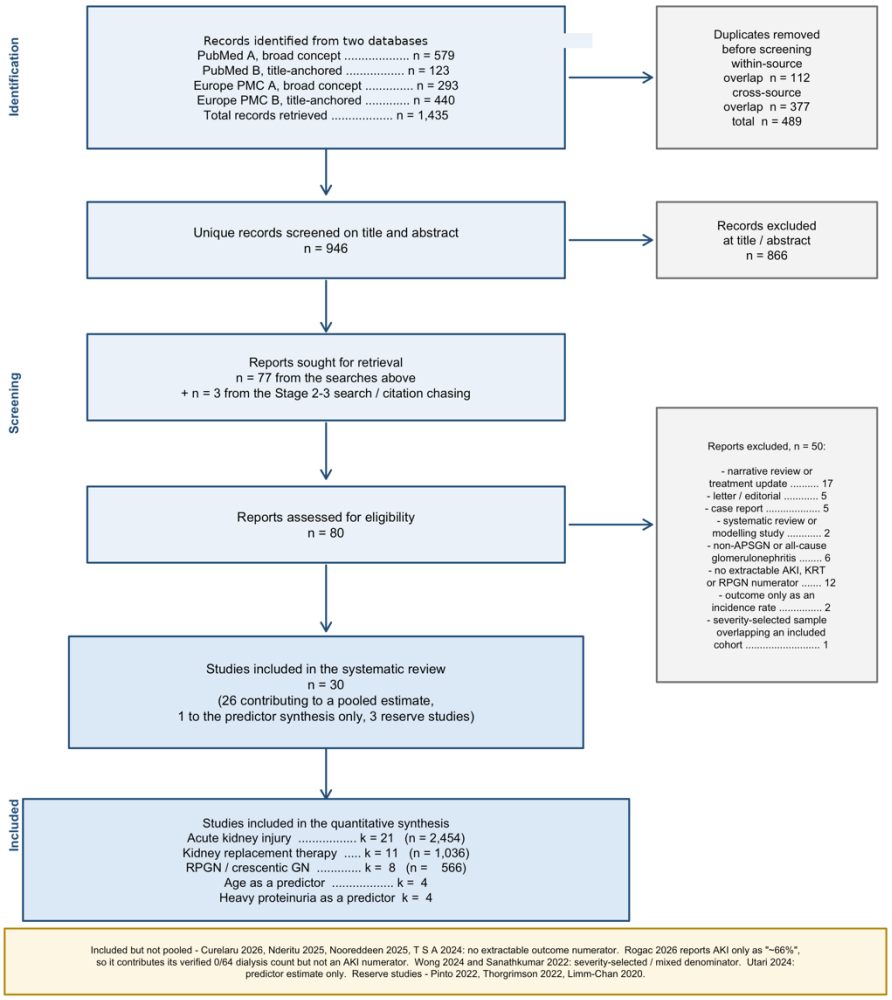

Figure 1. PRISMA 2020 flow diagram for the identification, screening and inclusion of studies reporting acute kidney injury, kidney replacement therapy or rapidly progressive glomerulonephritis.

Characteristics of the included studies

The 26 pooled cohorts comprised patients from 20 countries across six regions and were published between 2014 and 2026. Twenty-five enrolled children and one enrolled adults. Twenty-two were retrospective or cross-sectional and four were prospective. Analysed denominators ranged from 11 to 400 participants. Four cohorts were referral-selected — recruited through a nephrology referral or biopsy pathway rather than through general admission.

Seventeen of the 26 cohorts explicitly labelled their population as post-streptococcal; six used the broader post-infectious label, including the sole adult cohort, which is a biopsy-proven post-infectious series in which the streptococcal fraction is not stated; and three described their population only as acute glomerulonephritis without an aetiological qualifier. This label is recorded for every study in Table 1 and is the principal source of indirectness in this synthesis, discussed further below.

Table 1. Characteristics of the 26 studies contributing to a pooled estimate, with the aetiological label used by each source and the acute kidney injury definition each investigator team applied.

Study	Country	Aetiology	Design	Period	Age	N	AKI n (%)	KRT n (%)	RPGN n (%)	AKI definition applied
Ali el-TM 2014[28]	Sudan	APSGN	Retrospective and prospective cohort	[NR]	Child	69	—	20 (29.0)	—	Severe acute disease requiring dialysis
Gunasekaran K 2015[29]	India	PIGN	Prospective observational	[NR]	Child	72	15 (20.8)	1 (1.4)	—	AKIN criteria
McGil Ugwu GI 2015[30]	Nigeria	AGN	Retrospective records review	[NR]	Child	20	8 (40.0)	—	—	Recorded as a complication; criteria not stated
Ayoob RM 2016[14]	United States	APSGN	Single-centre one-year case series	[NR]	Child	17	16 (94.1)	3 (17.6)	3 (17.6)	eGFR <80 mL/min/1.73 m²
Dagan R 2016[31]	Israel	PIGN	Single-centre retrospective cohort	Two decades	Child	125	87 (69.6)	—	—	Azotaemia (elevated urea or creatinine)
Demircioglu Kilic B 2018[32]	Türkiye	APSGN	Single-centre retrospective cohort	[NR]	Child	75	22 (29.3)	—	—	GFR <90 mL/min/1.73 m²
Maalej B 2018[10]	Tunisia	APSGN	Single-centre retrospective review	12 years	Child	178	66 (37.1)	—	—	Creatinine ≥70 µmol/L, fixed threshold
Bhalla K 2019[33]	India	AGN	Prospective observational	[NR]	Child	48	38 (79.2)	2 (4.2)	—	Serum creatinine >1 mg/dL
Chong HC 2023[12]	Australia	APSGN	Single-centre retrospective cohort	2012–2017	Child	96	42 (43.8)	—	—	Reported as AKI; criteria not specified
Dhakal AK 2023[20]	Nepal	APSGN	Cross-sectional records and follow-up	2015–2022	Child	77	—	1 (1.3)	—	Not tabulated
Karakaya D 2023[16]	Türkiye	APSGN	Single-centre retrospective cohort	2010–2022	Child	153	—	—	19 (12.4)	Not tabulated
Matsell DG 2023[34]	Canada	PIGN	Retrospective referral cohort	[NR]	Child	119	79 (66.4)	—	—	AKI at initial presentation (KDIGO-based)
Abugrain K 2024[17]	South Africa	APSGN	Single-centre retrospective cohort	2015–2020	Child	100	59 (59.0)	4 (4.0)	7 (7.0)	Age-banded serum creatinine reference ranges
Bajracharya P 2024[11]	Philippines	APSGN	Single-centre retrospective descriptive	5 years	Child	77	49 (63.6)	—	—	Creatinine above age-specific normal
Bhat IA 2024[35]	India	APSGN	Prospective observational	[NR]	Child	50	12 (24.0)	—	—	Stated AKI criteria not specified
Leventoglu E 2024[36]	Türkiye	APSGN	Single-centre retrospective cohort	[NR]	Child	11	—	1 (9.1)	1 (9.1)	Not tabulated
Mengstie LA 2024[37]	Ethiopia	APSGN	Multicentre retrospective cross-sectional	[NR]	Child	322	59 (18.3)	—	—	Recorded as a complication; criteria not stated
Shrestha M 2024[15]	Nepal	PIGN	Retrospective cross-sectional	2020–2023	Child	63	1 (1.6)	—	1 (1.6)	AKI recorded as a clinical complication
Konopasek P 2025[7]	Czech Republic	APSGN	Multicentre (15 centres) retrospective	2016–2023	Child	95	56 (59.0)	3 (3.2)	2 (2.1)	KDIGO creatinine and/or oliguria
Ranawaka R 2025[38]	Sri Lanka	APSGN	Retrospective records cohort	[NR]	Child	39	8 (20.5)	—	1 (2.3)	pRIFLE estimated creatinine clearance <50%
Tizki S 2025[18]	Morocco	APSGN	Single-centre retrospective cohort	2019–2023	Child	83	13 (15.7)	5 (6.0)	17 (20.5)	Stated AKI criteria not specified
Wan Yusof WA 2025[9]	Malaysia	APSGN	Single-centre retrospective cohort	2008–2018	Child	400	86 (21.5)	2 (0.5)	—	KDIGO, modified to an age-based baseline
Moges TA 2026[39]	Ethiopia	APSGN	Multicentre retrospective cohort	2018–2024	Child	332	27 (8.1)	—	—	Not stated
Rogac Z 2026[3]	Montenegro	PIGN	Nationwide retrospective cohort	2006–2022	Child	64	—	0 (0.0)	—	Not stated; AKI given only as “approximately 66%”
Samal AN 2026[40]	India	AGN	Prospective observational	[NR]	Child	70	21 (30.0)	—	—	KDIGO
Thammathiwat T 2026[41]	Thailand	PIGN	Single-centre biopsy-proven cohort	1996–2023	Adult	73	7 (9.6)	—	—	Not stated

AGN, acute glomerulonephritis (no aetiological qualifier used by the source); AKI, acute kidney injury; AKIN, Acute Kidney Injury Network; APSGN, acute post-streptococcal glomerulonephritis; eGFR, estimated glomerular filtration rate; KDIGO, Kidney Disease: Improving Global Outcomes; KRT, kidney replacement therapy; [NR], not reported; PIGN, post-infectious glomerulonephritis; pRIFLE, paediatric Risk, Injury, Failure, Loss, End-stage; RPGN, rapidly progressive glomerulonephritis. An em dash indicates that the outcome was not reported by that study, so it contributed no numerator to that pool. The Sri Lankan cohort reports its acute kidney injury outcome on a denominator of 39 and its crescentic glomerulonephritis outcome on a separately reported group of 44; the two rows enter different pools and are never double-counted, but the discordance is as published and could not be reconciled from the report. The Sudanese cohort defined its population by the requirement for dialysis, not its outcome; it therefore contributes only a kidney replacement therapy numerator and is handled separately in the sensitivity analysis.

Risk of bias

Across the 26 pooled cohorts, 20 were judged at high risk of bias overall, one at very high risk and five at some concerns; among the 21 cohorts contributing to the primary outcome the corresponding figures were 16 high, one very high and four some concerns — that is, 17 of 21 at high or very high risk. Domain 6, measurement of the outcome, was the recurring failure and drove the overall judgement in most studies. Missing data was the second most frequent problem, reflecting substantial and non-random loss to follow-up. Only three studies were at low risk across every substantive domain, and all three applied consensus criteria either prospectively or by explicit protocol[7,29,40]. Domain-level judgements are given in Table 2 and displayed in Figure 2.

Figure 2. ROBINS-E traffic-light plot of domain-level and overall risk-of-bias judgements for the 26 pooled cohorts. Domain 6, measurement of the outcome, is the dominant weakness and is the same problem that the definition subgroup analysis quantifies statistically.

Table 2. ROBINS-E domain-level risk-of-bias judgements.

Study	D1	D2	D3	D4	D5	D6	D7	Overall
Ayoob RM 2016	High	Some	High	Some	Some	High	Some	High
Matsell DG 2023	Some	Low	High	Some	Some	Low	Low	High
Bajracharya P 2024	High	Some	Some	Some	High	High	Some	High
Abugrain K 2024	Some	Some	Some	Some	High	High	Low	High
Konopasek P 2025	Low	Low	Low	Some	Low	Low	Low	Some concerns
Chong HC 2023	Some	Some	Some	Some	High	High	Some	High
Maalej B 2018	High	Some	Some	Some	Some	High	Some	High
Demircioglu Kilic B 2018	Some	Some	Some	Some	Some	High	Some	High
Bhat IA 2024	Some	Low	Some	Some	Low	High	Some	High
Wan Yusof WA 2025	Some	Low	Some	Some	Some	Some	Low	Some concerns
Gunasekaran K 2015	Low	Low	Low	Some	Low	Low	Low	Some concerns
Ranawaka R 2025	Some	Some	High	Some	High	Some	Some	High
Tizki S 2025	Some	Some	Some	Some	Some	High	Some	High
Thammathiwat T 2026	Some	Some	High	Some	Some	High	Some	High
Moges TA 2026	Some	Some	Some	Some	Some	High	Some	High
Shrestha M 2024	High	Some	Some	Some	Some	Very high	Some	Very high
Rogac Z 2026	Some	Some	Low	Some	Some	High	High	High

Table 2. ROBINS-E domain-level risk-of-bias judgements.

Study	D1	D2	D3	D4	D5	D6	D7	Overall
Dhakal AK 2023	Some	Some	Some	Some	Some	High	Some	High
Ali el-TM 2014	High	Some	Some	Some	High	Some	Some	High
Karakaya D 2023	Some	Some	Some	Some	Some	Some	Some	Some concerns
Leventoglu E 2024	High	Some	High	Some	Some	High	High	High
Mengstie LA 2024	Some	Some	Some	Some	Some	High	Some	High
Samal AN 2026	Some	Low	Some	Some	Low	Low	Some	Some concerns
Dagan R 2016	Some	Some	Some	Some	Some	High	Some	High
Bhalla K 2019	High	Some	High	Some	Some	High	Some	High
McGil Ugwu GI 2015	High	Some	Some	Some	High	High	High	High

D1, confounding; D2, measurement of the exposure; D3, selection of participants; D4, post-exposure interventions; D5, missing data; D6, measurement of the outcome; D7, selection of the reported result. "Some" denotes some concerns.

The definition of acute kidney injury dominates the reported risk

The principal finding of this review is presented first because it conditions everything that follows. The definition of acute kidney injury applied by each study explained more of the variation between cohorts than any patient-level or geographical factor examined. Studies applying a bare creatinine or estimated glomerular filtration rate threshold reported a pooled prevalence of 62.3% (44.7 to 77.2); studies applying consensus criteria reported 35.0% (21.3 to 51.7); and studies that never stated a criterion reported 16.0% (8.6 to 27.8), with a test for subgroup differences of Q = 17.85 on two degrees of freedom (p = 0.0001). This is a fourfold spread in reported risk across three ways of counting the same event, and it runs in the direction that measurement theory predicts. The gradient is shown in Figure 3, where the separation between the three subgroup diamonds, and not the overall diamond, is the finding.

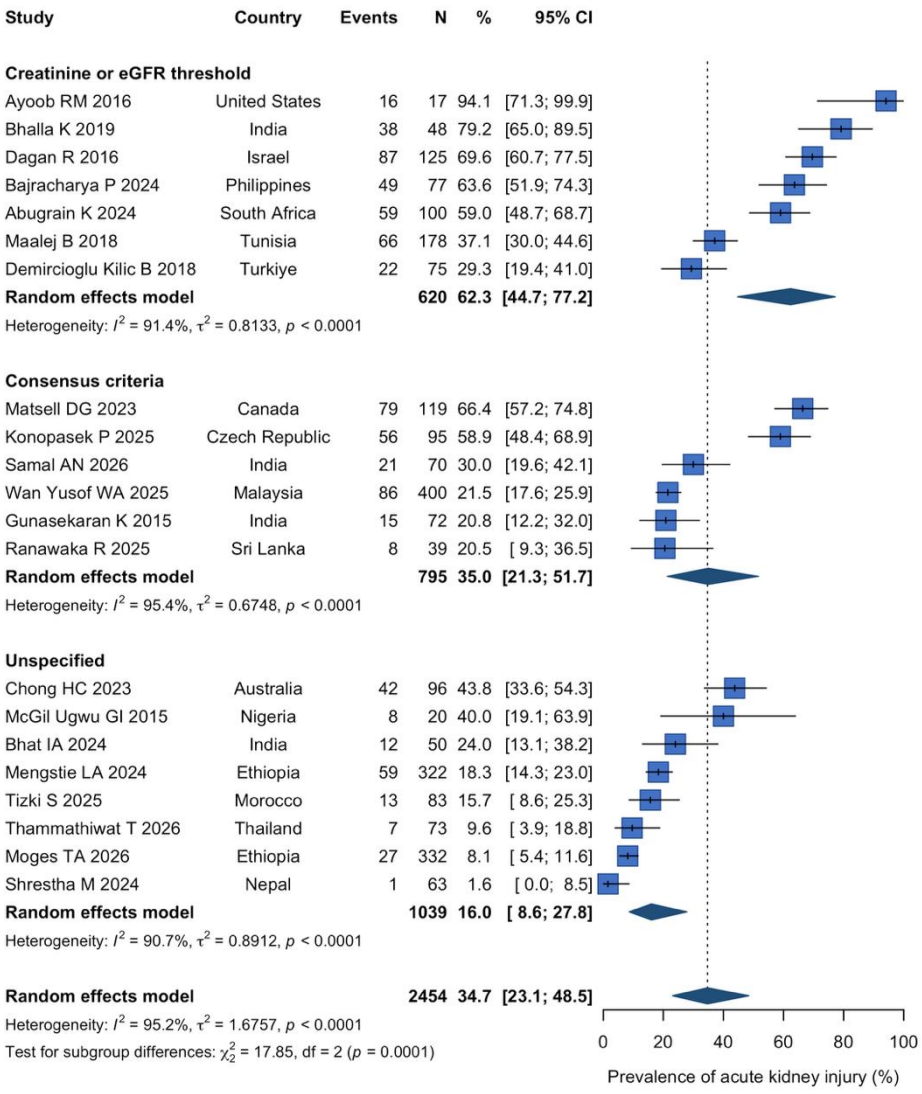

Figure 3. Prevalence of acute kidney injury grouped by the definition each study applied. The gradient across the three subgroups, not the overall diamond, is the substantive result of this review.

The unspecified group requires care in interpretation. It is not a measurement strategy but a missing-data category, and it is heterogeneous by construction: it contains studies that applied a rigorous definition and failed to report it as well as studies that counted whatever appeared in the discharge record. Its pooled estimate of 16.0% should therefore not be read as the risk associated with any coherent approach to measurement.

Pooled prevalence of acute kidney injury

Across all 21 cohorts and 2,454 patients, with 771 events, acute kidney injury complicated 34.7% of presentations (95% CI 23.1 to 48.5). Heterogeneity was extreme (I^2 = 95.2%, 95% CI 93.8 to 96.3; τ^2 = 1.676; Q = 418.8 on 20 degrees of freedom, p < 0.001) and the 95% prediction interval spanned 3.2% to 89.4%. Individual study estimates ranged from 1.6% in a Nepali cross-sectional series to 94.1% in a small United States referral case series[14,15].

Because eight of these 21 cohorts never stated a definition, a co-primary analysis restricted to the 13 cohorts that did state one is reported alongside the all-studies figure. That analysis gives a pooled prevalence of **49.6% (35.5 to 63.7)**, with I^2 = 94.6% and τ^2 = 1.045. The difference of nearly fifteen percentage points is larger than the effect of any moderator examined and larger than the leave-one-out range, and both figures are therefore reported throughout, including in the abstract. The all-studies estimate describes what this literature reports; the definition-stated estimate describes what it reports when the outcome can be verified to have been measured. The pooled estimate and the contributing cohorts are shown in Figure 4, and all three outcomes with their prediction intervals and Freeman–Tukey comparators are summarised in Table 3.

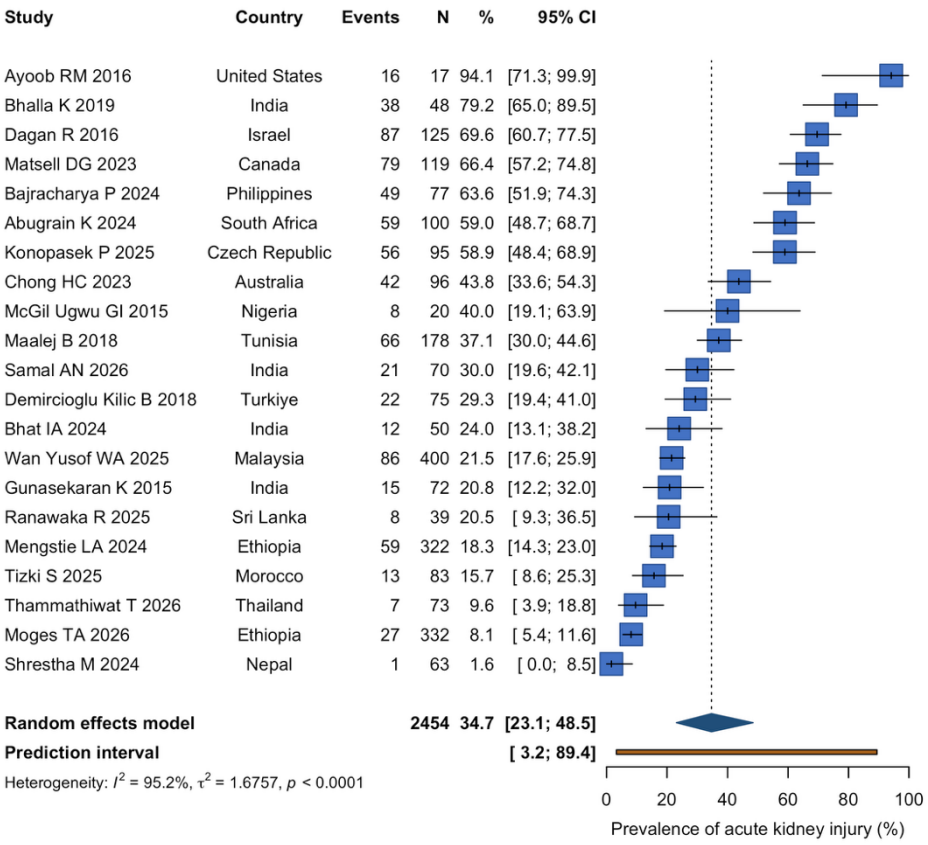

Figure 4. Forest plot of the pooled prevalence of acute kidney injury (random-intercept logistic regression, k = 21, n = 2,454). Squares are individual study proportions with exact confidence intervals; the diamond is the pooled estimate.

Table 3. Pooled prevalence of each outcome, with prediction intervals and the Freeman–Tukey comparator.

Outcome	k	Patients	Events	Pooled prevalence, % (95% CI)	95% prediction interval, %	I^2 (95% CI), %	τ^2	Cochran Q (df), p
Acute kidney injury	21	2,454	771	34.7 (23.1–48.5)	3.2–89.4	95.2 (93.8–96.3)	1.676	418.8 (20), <0.001
Kidney replacement therapy	11	1,036	42	3.3 (1.4–7.8)	0.2–41.7	84.4 (73.6–90.7)	1.647	64.0 (10), <0.001
RPGN or crescentic glomerulonephritis	8	566	51	6.8 (3.4–13.2)	0.8–39.1	69.0 (35.2–85.2)	0.702	22.6 (7), 0.002
Acute kidney injury — Freeman–Tukey comparator	21	2,454	771	36.7 (25.3–48.8)	0–90.3	96.6 (95.6–97.3)	0.077	580.7 (20), <0.001
Kidney replacement therapy — Freeman–Tukey comparator	11	1,036	42	4.3 (0.9–9.2)	0–28.0	86.3 (77.4–91.8)	0.020	73.2 (10), <0.001
RPGN — Freeman–Tukey comparator	8	566	51	7.2 (2.8–13.1)	0–29.5	76.7 (53.7–88.3)	0.013	30.1 (7), <0.001

CI, confidence interval; RPGN, rapidly progressive glomerulonephritis. The primary estimator is a random-intercept logistic regression on the logit scale; the Freeman–Tukey double-arcsine result is shown for comparison only. The co-primary analysis restricted to the cohorts that stated a definition (49.6%, 35.5 to 63.7) is reported in the text of the preceding subsection and in the sensitivity analyses below.

Kidney replacement therapy and rapidly progressive glomerulonephritis

Kidney replacement therapy was required in 3.3% of patients (1.4 to 7.8) across 11 cohorts and 1,036 patients, with 42 events, substantial heterogeneity (I^2 = 84.4%) and a prediction interval of 0.2% to 41.7%. The upper limit of that prediction interval is not clinically credible and its origin is identifiable. One contributing cohort defined its *population* by the requirement for dialysis and consequently reports a dialysis prevalence of 29.0%, roughly an order of magnitude above every other cohort in the pool[28]. A study that selects patients on the outcome cannot contribute an unbiased estimate of that outcome's prevalence. Excluding it moves the estimate to **2.5% (1.2 to 5.1)** and halves the heterogeneity, from an I^2 of 84.4% to 54.4%. That analysis is reported here as the preferred estimate of dialysis requirement, with the all-cohorts figure retained for completeness.

Rapidly progressive or crescentic glomerulonephritis occurred in 6.8% (3.4 to 13.2) across eight cohorts and 566 patients, with 51 events, and a prediction interval of 0.8% to

39.1%. This was the least heterogeneous of the three outcomes (I^2 = 69.0%, 95% CI 35.2 to 85.2), which is expected given that crescentic disease is verified histologically where it is reported — though, as noted in the Methods, that same selectivity means the composite is under-ascertained. The corresponding forest plots are given in Figures 5 and 6.

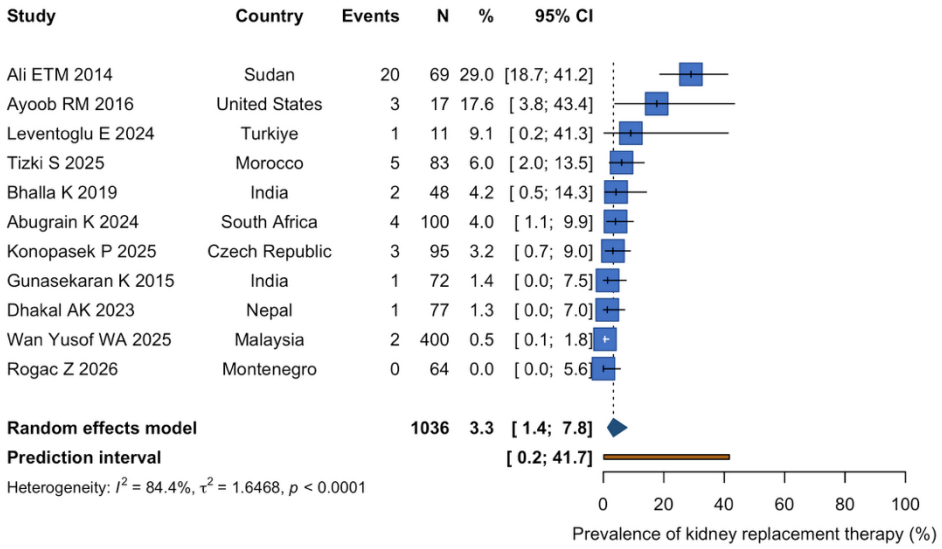

Figure 5. Pooled prevalence of kidney replacement therapy (k = 11, n = 1,036).

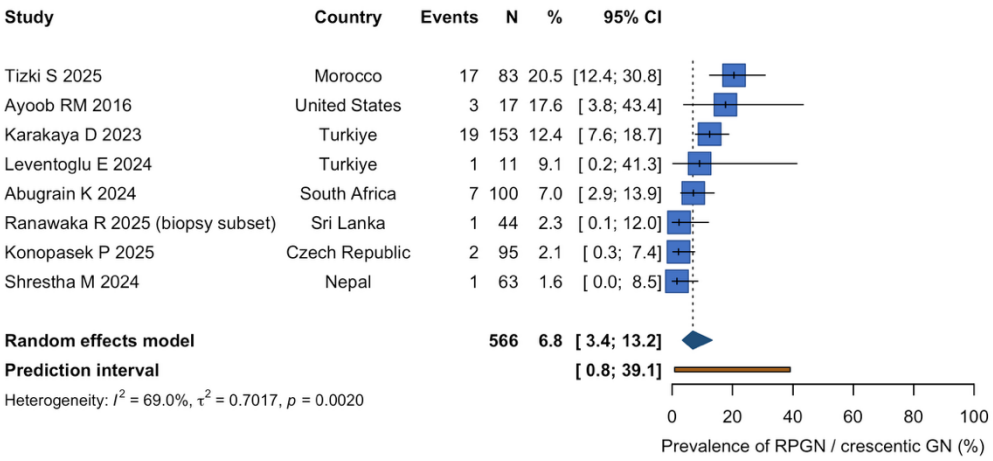

Figure 6. Pooled prevalence of rapidly progressive or crescentic glomerulonephritis (k = 8, n = 566).

Other sources of heterogeneity

Income setting was also strongly associated with reported prevalence (60.5% in high-income versus 25.4% in low- and middle-income settings, p = 0.0018). Case ascertainment did not reach significance (referral-selected 64.3% versus general admission 29.4%, p = 0.131). All subgroup analyses are set out in Table 4. The age-stratum row of that table compares 20 paediatric cohorts against a single adult cohort; a subgroup containing one study has no estimable between-study variance, so that comparison is descriptive only and no inferential claim is made from it.

Table 4. Subgroup analyses for all three outcomes.

Outcome	Moderator	Level	k	Patients	Prevalence, % (95% CI)	I^2, %	$Q_{between}$ (df)	p
AKI	AKI definition applied	Creatinine or eGFR threshold	7	620	62.3 (44.7–77.2)	91.4	17.85 (2)	0.0001
AKI		Consensus criteria (KDIGO, AKIN, pRIFLE)	6	795	35.0 (21.3–51.7)	95.4		
AKI		Unspecified	8	1,039	16.0 (8.6–27.8)	90.7		
AKI	Income setting	High-income	6	527	60.5 (42.7–75.9)	89.1	9.73 (1)	0.0018
AKI		Low- and middle-income	15	1,927	25.4 (15.7–38.5)	94.1		
AKI	Case ascertainment	Referral-selected	4	257	64.3 (22.5–91.8)	94.9	2.28 (1)	0.131
AKI		General admission	17	2,197	29.4 (20.0–41.0)	94.8		
AKI	Age stratum	Child	20	2,381	36.6 (24.5–50.6)	95.3	11.74 (1)	0.0006
AKI		Adult	1	73	9.6 (4.6–18.8)	—		
AKI	APSGN burden in the source population	Sporadic	6	504	54.4 (27.1–79.3)	93.7	2.79 (1)	0.095

Table 4. Subgroup analyses for all three outcomes.

Outcome	Moderator	Level	k	Patients	Prevalence, % (95% CI)	I^2, %	$Q_{between}$ (df)	p
AKI		Endemic	15	1,950	28.1 (17.9–41.3)	94.1		
KRT	Income setting	High-income	4	187	3.8 (1.0–14.1)	37.3	0.05 (1)	0.830
KRT		Low- and middle-income	7	849	3.2 (1.1–9.0)	89.8		
KRT	Case ascertainment	Referral-selected	2	65	8.0 (2.8–20.9)	63.6	2.17 (1)	0.141
KRT		General admission	9	971	2.7 (1.0–7.3)	86.9		
RPGN	Income setting	High-income	4	276	8.0 (3.5–17.5)	57.7	0.24 (1)	0.628
RPGN		Low- and middle-income	4	290	5.7 (1.8–16.4)	80.4		
RPGN	Case ascertainment	Referral-selected	1	17	17.6 (5.8–42.7)	—	2.53 (1)	0.112
RPGN		General admission	7	549	6.0 (2.8–12.6)	72.7		

AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; KRT, kidney replacement therapy; RPGN, rapidly progressive glomerulonephritis. The age-stratum and referral-selected rows for RPGN contain a single study; the associated p values are reported for completeness but are not interpretable as tests and no inference is drawn from them. No adjustment was made for multiplicity across these comparisons.

Definition class and income setting are not interchangeable. In a multivariable meta-regression each remained significant after adjustment for the other (definition class added to a model already containing income setting, likelihood-ratio statistic 13.27 on two degrees of freedom, p = 0.0013; income setting added to a model already containing definition class, 6.02 on one degree of freedom, p = 0.014). Together the two moderators explained 66.7% of the between-study variance and reduced the residual I^2 to 88.6%, and this model was the best-fitting of the eleven candidates by corrected Akaike information criterion. Neither publication year (R^2 = 18.3%) nor study size (R^2 = 11.0%, p = 0.118) explained anything material. Model comparison is given in Table 5 and the coefficients of the best-fitting model in Table 6.

Table 5. Mixed-effects meta-regression model comparison for the prevalence of acute kidney injury, ranked by corrected Akaike information criterion.

Model	Coefficients	Q_M	p	τ^2	Residual I^2, %	R^2, %	AICc	ΔAICc
Definition + income setting	4	30.71	<0.001	0.459	88.6	66.7	65.61	0
Definition + ascertainment	4	24.62	<0.001	0.554	90.6	59.8	68.09	2.48
AKI definition class alone	3	18.47	<0.001	0.659	92.0	52.2	68.13	2.52
Definition + burden setting	4	20.69	<0.001	0.616	91.3	55.3	70.33	4.72
Definition + burden + ascertainment	5	25.00	<0.001	0.547	90.2	60.3	71.93	6.32
Income setting alone	2	7.61	0.006	0.982	94.8	28.8	72.29	6.68
Publication year	2	4.71	0.030	1.126	95.4	18.3	74.61	9.00
Case ascertainment alone	2	3.53	0.060	1.197	95.8	13.1	75.64	10.03
Burden setting alone	2	3.02	0.082	1.207	95.8	12.4	76.13	10.52
Null (random effects only)	1	5.34	0.021	1.378	96.3	—	76.28	10.67
log study size	2	2.45	0.118	1.226	95.7	11.0	76.67	11.06

AICc, corrected Akaike information criterion; Q_M, omnibus test of the moderators. R^2 is the proportion of between-study variance explained relative to the null random-effects model.

Table 6. Coefficients of the best-fitting meta-regression model (definition class plus income setting).

Term in the best-fitting model	logit β	SE	z	p	Odds ratio (95% CI)
Intercept (consensus criteria, high-income)	0.039	0.382	0.10	0.918	1.04 (0.49–2.20)
Creatinine or eGFR threshold (vs consensus criteria)	0.970	0.410	2.36	0.018	2.64 (1.18–5.89)
Unspecified (vs consensus criteria)	−0.732	0.409	−1.79	0.073	0.48 (0.22–1.07)
Low- and middle-income (vs high-income)	−0.985	0.373	−2.64	0.008	0.37 (0.18–0.78)

Odds ratios are on the odds of acute kidney injury relative to the reference category shown in the intercept row. eGFR, estimated glomerular filtration rate.

A pre-specified alternative explanation was tested and rejected. It is tempting to read the income-setting effect as a proxy for endemic streptococcal burden, particularly because the Australian Top End cohort serves a population with among the highest APSGN incidence in the world yet is classified as high-income[2,12]. Every study was therefore reclassified as arising from an endemic or a sporadic burden setting and the analysis repeated. The reclassified moderator was not significant (endemic 28.1% versus sporadic 54.4%, p = 0.095) and fitted worse than income setting (ΔAICc = +2.5, Table 5). The hypothesis did not survive its own test and income setting was retained.

Prognostic determinants

Two determinants were reported by enough studies on a common scale to pool. The outcomes those studies used are not identical, and the pooled estimates should be read as associations with adverse kidney outcome broadly defined rather than with a single endpoint: the four age estimates derive from acute kidney injury, a composite kidney injury at last follow-up, full nephritic syndrome, and stage 1 or higher acute kidney injury within thirty days respectively, and the four proteinuria estimates range from acute kidney injury to chronic glomerulonephritis to a composite poor treatment outcome.

Across all four contributing cohorts, the odds of an adverse kidney outcome rose by 13% per additional year of age (OR 1.13, 1.04 to 1.22, p = 0.005), with substantial heterogeneity (I^2 =

77.1%) that proved attributable to population. Restricted to the three paediatric cohorts the pooled estimate was OR 1.17 (1.09 to 1.24) with I^2 = 0% — three studies from Malaysia, Canada and Israel, using different adjustment sets and different outcome definitions, agreeing closely[9,31,34]. The single adult cohort, a Japanese real-world database of 1,002 patients, gave a much flatter slope of OR 1.04 per year[42]. Because the adult stratum contains one study, the contrast between the two slopes is hypothesis-generating rather than a formal test, and is presented as such.

Heavy or nephrotic-range proteinuria at presentation was associated with an odds ratio of 2.66 (1.50 to 4.73, I^2 = 29.6%), and the finding survived restriction to the three adjusted estimates (OR 2.24, 1.28 to 3.90)[37,40,43]. One contributing estimate used a 2+ dipstick threshold rather than a nephrotic-range definition, which will bias that study towards the null and therefore, if anything, makes the pooled association conservative. Both determinants are summarised in Table 7 and displayed in Figures 7 and 8.

Table 7. Pooled prognostic determinants of adverse kidney outcome.

Determinant	k	Pooled OR (95% CI)	95% prediction interval	p	I^2, %	τ^2	Contributing studies
Age, per additional year — all cohorts	4	1.13 (1.04–1.22)	0.87–1.46	0.005	77.1	0.005	Wan Yusof 2025; Matsell 2023; Dagan 2016; Sakoda 2026
Age, per additional year — paediatric cohorts only	3	1.17 (1.09–1.24)	1.01–1.34	<0.001	0	0	Wan Yusof 2025; Matsell 2023; Dagan 2016
Heavy proteinuria at presentation	4	2.66 (1.50–4.73)	0.63–11.24	<0.001	29.6	0.119	Matsell 2023; Utari 2024; Samal 2026; Mengstie 2024
Heavy proteinuria — adjusted estimates only	3	2.24 (1.28–3.90)	0.45–11.01	0.005	14.3	0.057	Matsell 2023; Utari 2024; Mengstie 2024

OR, odds ratio. Outcome definitions differ between contributing studies and are listed in the text; the pooled estimate should be read as an association with adverse kidney outcome broadly defined rather than with a single endpoint. One proteinuria estimate uses a 2+ dipstick threshold rather than a nephrotic-range definition.

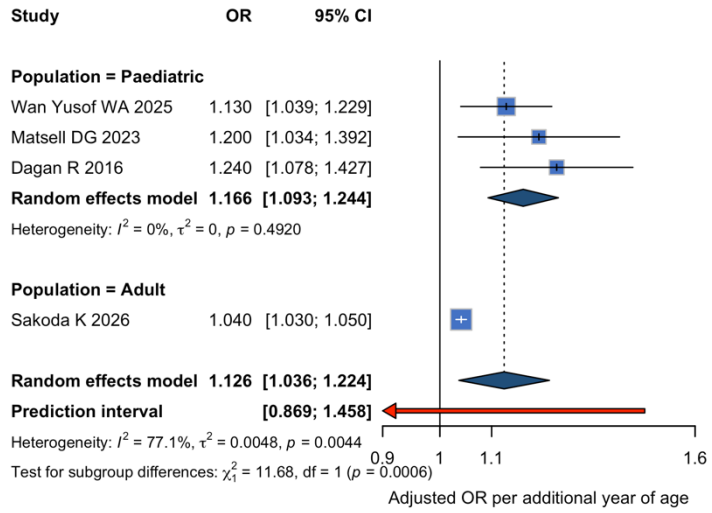

Figure 7. Age, per additional year, as a determinant of adverse kidney outcome, stratified by population. The paediatric slope is steep and homogeneous; the single adult cohort is much flatter.

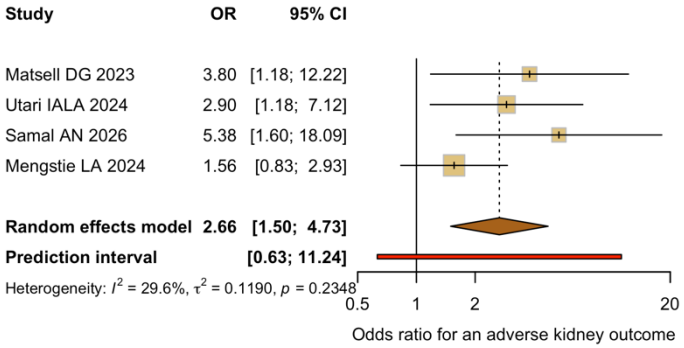

Figure 8. Heavy or nephrotic-range proteinuria at presentation as a determinant of adverse kidney outcome.

A third candidate determinant, acute kidney injury at presentation as a predictor of poor recovery, was reported by only two cohorts — a crude risk ratio of 3.07 in one Ethiopian series and an adjusted odds ratio of 1.70 in another — and was not pooled[37,39]. Other predictors reported in individual cohorts, including low complement C3, oliguria, macroscopic haematuria and blood urea nitrogen, appeared in only one or two studies each or on incompatible scales and could not be combined. Fever at presentation, examined directly in one Israeli cohort, was associated with longer admission but not with worse short- or medium-term kidney outcome[44].

Extrarenal and hypertensive complications

Hypertensive and extrarenal complications are frequently the reason a child with this disease is admitted to intensive care, but

they are reported too inconsistently across the included cohorts to pool: denominators differ, definitions of hypertensive emergency are not standardised, and neurological complications are captured by clinical impression in some cohorts and by brain imaging in others. They are therefore tabulated narratively in Table 8, with the range reported across the cohorts that described each, so that readers can see the spread without a pooled estimate being implied. No formal synthesis of these outcomes should be inferred from that table.

Table 8. Extrarenal and hypertensive complications as reported by the included cohorts. These were not pooled and the ranges are descriptive.

Complication, as reported by the source	Cohorts reporting it	Reported range
Hypertension at presentation	Several cohorts, variously defined	31% (Tunisia) to 87% (Nepal)
Hypertensive emergency or crisis requiring urgent treatment	Northern Territory, Australia	37.4%
Hypertensive encephalopathy	South Africa; Philippines; Montenegro	2.5% to 14%
Hypertensive seizures or convulsions	South Africa; Montenegro	8% to 10%
Posterior reversible encephalopathy on brain imaging	South Africa	8%
Pulmonary oedema	Nepal	23.4%
Nephrotic-range proteinuria at presentation	Northern Territory, Australia; South Africa; Morocco	33.7% to 57.7%
Serosal involvement (ascites, pleural effusion)	Montenegro; Philippines	3.8% to 21%

Ranges span only those cohorts that reported the complication concerned; the denominator and the definition differ between cohorts, and absence from this table does not mean the complication did not occur.

Sensitivity and influence analyses

The pooled prevalence of acute kidney injury was robust to every analysis except the removal of the undefined-outcome cohorts discussed above. Leave-one-out analysis across all 21 cohorts moved the estimate only between 31.9% and 37.8%. No study met the Bonferroni-corrected outlier criterion, no Cook's distance approached the conventional threshold of 4/k = 0.19 (the maximum was 0.147), and no study was flagged as influential; the diagnostics are shown in Supplementary Figure 9. Two specific concerns were tested and cleared. The two Ethiopian cohorts are similar in size and overlap in calendar time, raising the possibility of double counting, but they are drawn from different administrative zones and different hospitals, and dropping either or both moved the estimate only to 36.9%, 35.7% and 38.1%[37,39]. Admitting the Montenegrin cohort at its author-reported approximation of 42 of 64 gave 36.1%, so its exclusion costs nothing[3]. The full set of sensitivity analyses across all three outcomes is given in Table 9.

Table 9. Sensitivity analyses for all three outcomes. The two analyses shown in bold are reported alongside the corresponding primary estimates in the text.

Outcome	Sensitivity analysis	k	Prevalence, % (95% CI)	I^2, %	τ^2
AKI	Primary analysis — GLMM logit random effects	21	34.7 (23.1–48.5)	95.2	1.676
AKI	**Restricted to cohorts stating a definition (co-primary)**	13	**49.6 (35.5–63.7)**	94.6	1.045
AKI	Freeman–Tukey double-arcsine transformation	21	36.7 (25.3–48.8)	96.6	0.077
AKI	Inverse-variance logit (DerSimonian–Laird estimator)	21	35.0 (25.3–46.2)	95.2	1.049
AKI	Restricted to consensus AKI criteria	6	35.0 (21.3–51.7)	95.4	0.675
AKI	Restricted to creatinine or eGFR threshold definitions	7	62.3 (44.7–77.2)	91.4	0.813
AKI	Restricted to unspecified AKI criteria	8	16.0 (8.6–27.8)	90.7	0.891
AKI	Excluding referral-selected cohorts	17	29.4 (20.0–41.0)	94.8	1.069
AKI	Excluding the single adult cohort	20	36.6 (24.5–50.6)	95.3	1.615
AKI	Excluding cohorts with n < 50	17	29.7 (19.4–42.6)	95.7	1.316
AKI	Restricted to cohorts with n ≥ 70	15	33.7 (23.1–46.3)	96.1	1.032
AKI	Restricted to studies published 2020 or later	14	26.6 (16.1–40.8)	95.7	1.405
AKI	Restricted to low- and middle-income settings	15	25.4 (15.7–38.5)	94.1	1.336
AKI	Restricted to high-income settings	6	60.5 (42.7–75.9)	89.1	0.700
AKI	Endemic-burden settings only	15	28.1 (17.9–41.3)	94.1	1.248
AKI	Sporadic-burden settings only	6	54.4 (27.1–79.3)	93.7	1.979
AKI	Excluding the Northwest Amhara cohort	20	36.9 (24.9–50.7)	94.3	1.564
AKI	Excluding the East Amhara cohort	20	35.7 (23.5–50.2)	95.0	1.734
AKI	Excluding both Ethiopian cohorts	19	38.1 (25.5–52.6)	93.7	1.606
AKI	Adding the Montenegrin cohort as 42/64 (author approximation)	22	36.1 (24.4–49.6)	95.3	1.667
AKI	Excluding statistical outliers or influential studies	21	34.7 (23.1–48.5)	95.2	1.676
AKI	Leave-one-out, full range across all 21 cohorts	20	31.9 to 37.8	94.3–95.5	1.310–1.785
KRT	Primary analysis — GLMM logit random effects	11	3.3 (1.4–7.8)	84.4	1.647
KRT	**Excluding the dialysis-defined Sudanese cohort**	10	**2.5 (1.2–5.1)**	54.4	0.762
KRT	Freeman–Tukey double-arcsine transformation	11	4.3 (0.9–9.2)	86.3	0.020
KRT	Excluding referral-selected cohorts	9	2.7 (1.0–7.3)	86.9	1.826
RPGN	Primary analysis — GLMM logit random effects	8	6.8 (3.4–13.2)	69.0	0.702
RPGN	Freeman–Tukey double-arcsine transformation	8	7.2 (2.8–13.1)	76.7	0.013
RPGN	Excluding referral-selected cohorts	7	6.0 (2.8–12.6)	72.7	0.757

AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; GLMM, generalised linear mixed model; KRT, kidney replacement therapy; RPGN, rapidly progressive glomerulonephritis. The leave-one-out row reports the range across all 21 single-study exclusions rather than a single analysis.

Adding studies in year order, the cumulative estimate settled early and drifted little: it stood at 50.6% by 2018 with five cohorts and ended at 35.0% with 21. There is no secular trend towards higher or lower reported acute kidney injury, consistent with the meta-regression finding that publication year explains little. The post-pandemic increase in severity reported within a single Czech cohort is therefore not visible as a shift in the global literature, although a multicentre Saudi series likewise reported milder pandemic-era disease and an altered seasonality pattern[7,45]. The cumulative analysis is shown in Supplementary Figure 10.

Small-study effects

Peters' regression, the appropriate test for proportion outcomes, showed no evidence of small-study effects for either pool reaching ten studies (AKI p = 0.182; KRT p = 0.327), and Begg's rank correlation agreed (p = 0.629 and p = 0.392). For the AKI pool, Egger's regression was also non-significant (p = 0.729) and trim-and-fill imputed no studies, leaving the adjusted estimate at 35.0% (24.1 to 47.7). For the KRT pool, Egger's test was significant (p = 0.006) and trim-and-fill responded by imputing six studies. That result is an artefact rather than a finding: on the logit scale the standard error of a proportion is a deterministic function of the proportion itself, so cohorts reporting a low prevalence necessarily have large standard errors and manufacture funnel asymmetry whether or not any bias exists. The trim-and-fill adjusted value is therefore not reported as a result. All tests are tabulated in Table 10 and both funnel plots are provided as Supplementary Figures 11 and 12.

Table 10. Tests for small-study effects.

Outcome	Test	Statistic	p	Interpretation
AKI	Peters regression on 1/n (primary)	1.333	0.182	No evidence of small-study effects
AKI	Egger linear regression	0.352	0.729	No asymmetry; test is in any case biased for proportions
AKI	Begg rank correlation	−0.483	0.629	No evidence of small-study effects
AKI	Trim-and-fill (L^0 estimator)	0 studies imputed	—	Adjusted estimate 35.0% (24.1–47.7), essentially unchanged
KRT	Peters regression on 1/n (primary)	0.981	0.327	No evidence of small-study effects
KRT	Egger linear regression	−3.598	0.006	Significant, but an artefact of the logit standard error — see text
KRT	Begg rank correlation	−0.856	0.392	No evidence of small-study effects
KRT	Trim-and-fill (L^0 estimator)	6 studies imputed	—	Adjusted value not reported; it is a product of the same artefact

AKI, acute kidney injury; KRT, kidney replacement therapy. Peters' test is the primary test because it regresses on the inverse of the sample size and is not vulnerable to the artefact described in the text.

Certainty of the evidence

Every body of evidence was rated very low certainty, as set out in Table 11. All started at low certainty because every included study is observational. All three prevalence outcomes were rated down for indirectness, because nine of the 26 pooled cohorts did not label their population as post-streptococcal and the pooled population is therefore broader than the disease named in the research question. The prevalence of acute kidney injury was additionally rated down for risk of bias and inconsistency; the prevalence of kidney replacement therapy for risk of bias, inconsistency and imprecision; and the prevalence of rapidly progressive glomerulonephritis for inconsistency, since the confidence interval around its I^2 of 69% reaches 85% and is not compatible with homogeneity, and for imprecision. Both prognostic determinants were rated down for risk of bias and for indirectness arising from non-identical outcome definitions.

Supplementary figures

The four figures below support the robustness analyses reported in the Results and are provided as supplementary material rather than as primary findings.

Table 11. GRADE certainty assessment.

Outcome	k	Start	RoB	Incons.	Indirect.	Impreci.	Pub. bias	Certainty
Prevalence of AKI	21	Low	−1	−1	−1	0	0	Very low
Prevalence of KRT	11	Low	−1	−1	−1	−1	n/a	Very low
Prevalence of RPGN or crescentic GN	8	Low	0	−1	−1	−1	n/a	Very low
Age as a determinant (per year)	4	Low	−1	−1	−1	0	n/a	Very low
Heavy proteinuria as a determinant	4	Low	−1	0	−1	−1	n/a	Very low

Start, starting certainty (all bodies of evidence start at low certainty because every included study is observational); RoB, risk of bias; Incons., inconsistency; Indirect., indirectness; Impreci., imprecision; Pub. bias, publication bias; n/a, not assessable because fewer than ten studies contributed. AKI, acute kidney injury; GN, glomerulonephritis; KRT, kidney replacement therapy; RPGN, rapidly progressive glomerulonephritis. A value of −1 denotes rating down by one level. Final certainty is very low for every outcome, and was so both before and after the revisions to the indirectness and inconsistency domains made in response to peer review.

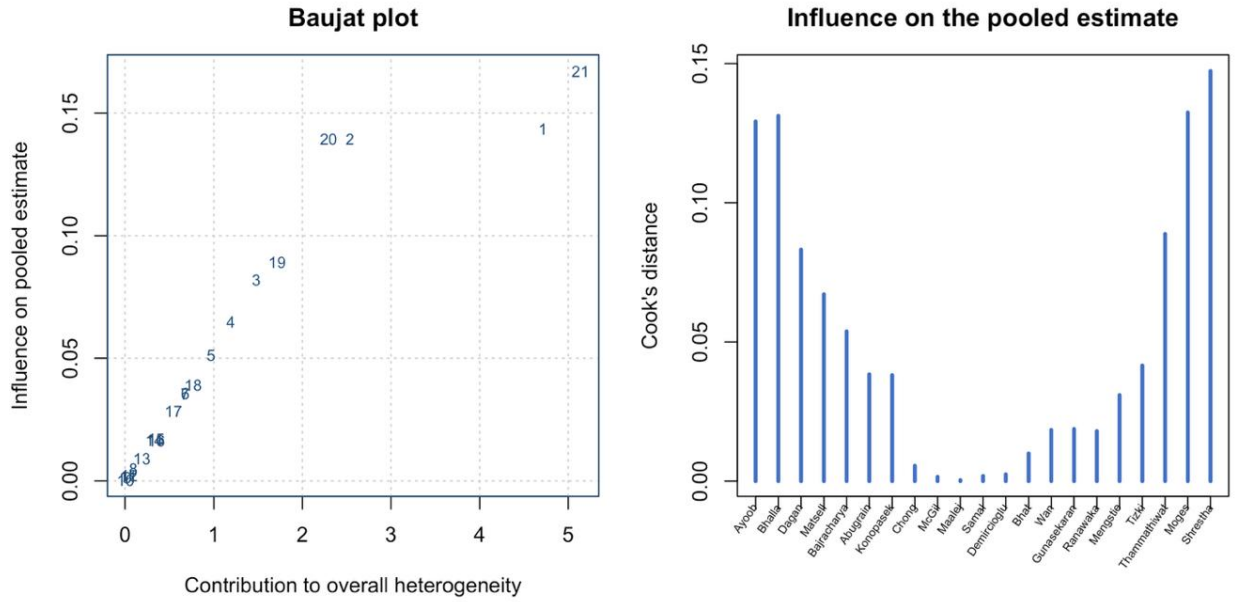

Figure 9. Supplementary. Influence diagnostics for the acute kidney injury pool: Baujat plot of heterogeneity contribution against influence, and Cook's distances by study.

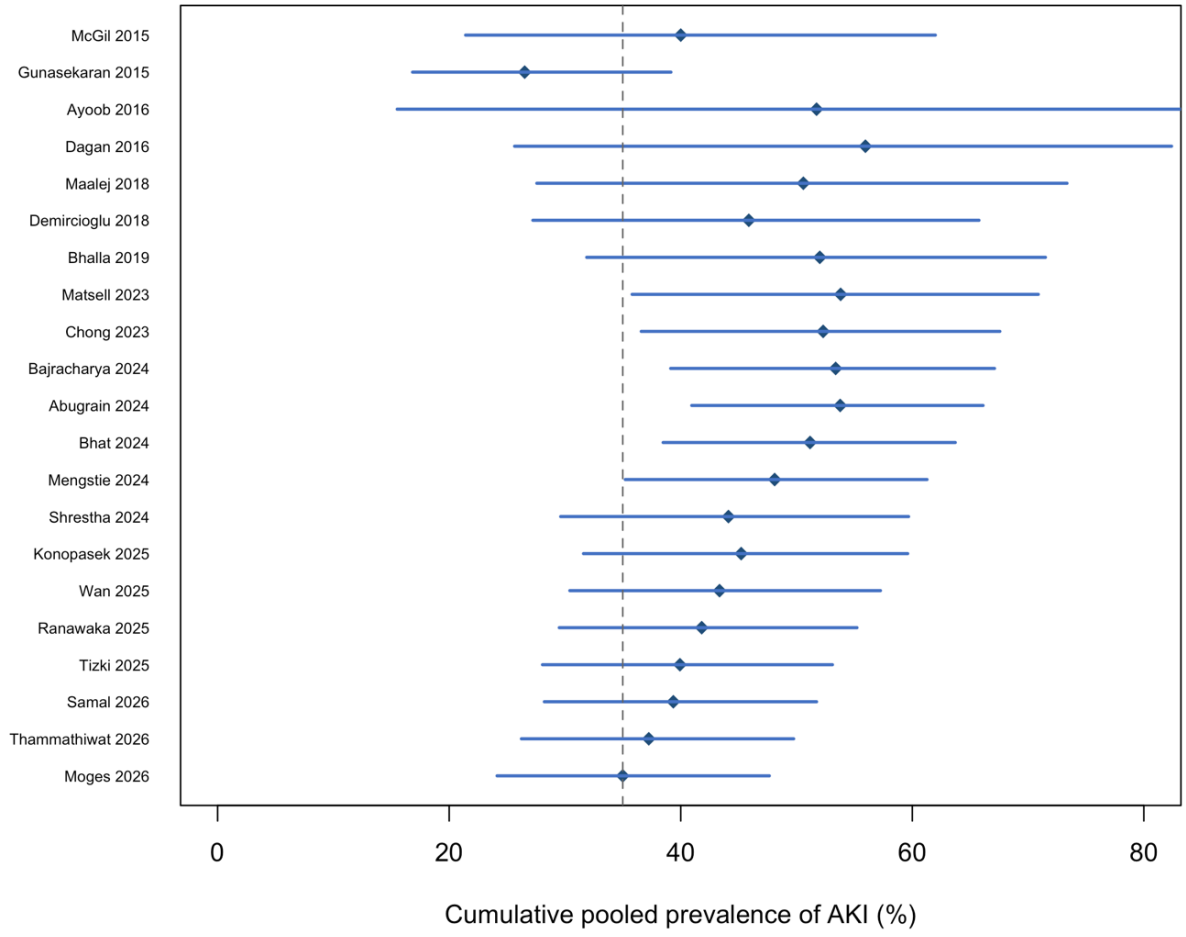

Figure 10. Supplementary. Cumulative meta-analysis of the prevalence of acute kidney injury, with studies added in year order.

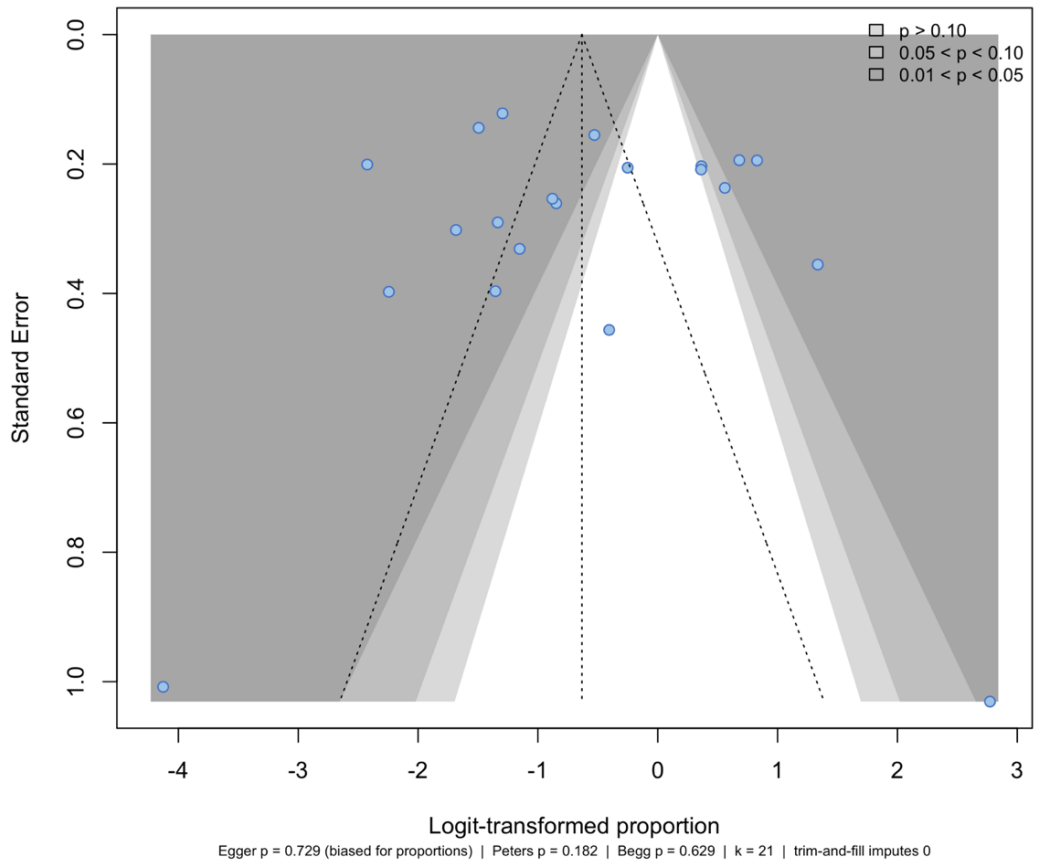

Figure 11. Supplementary. Contour-enhanced funnel plot for the acute kidney injury pool.

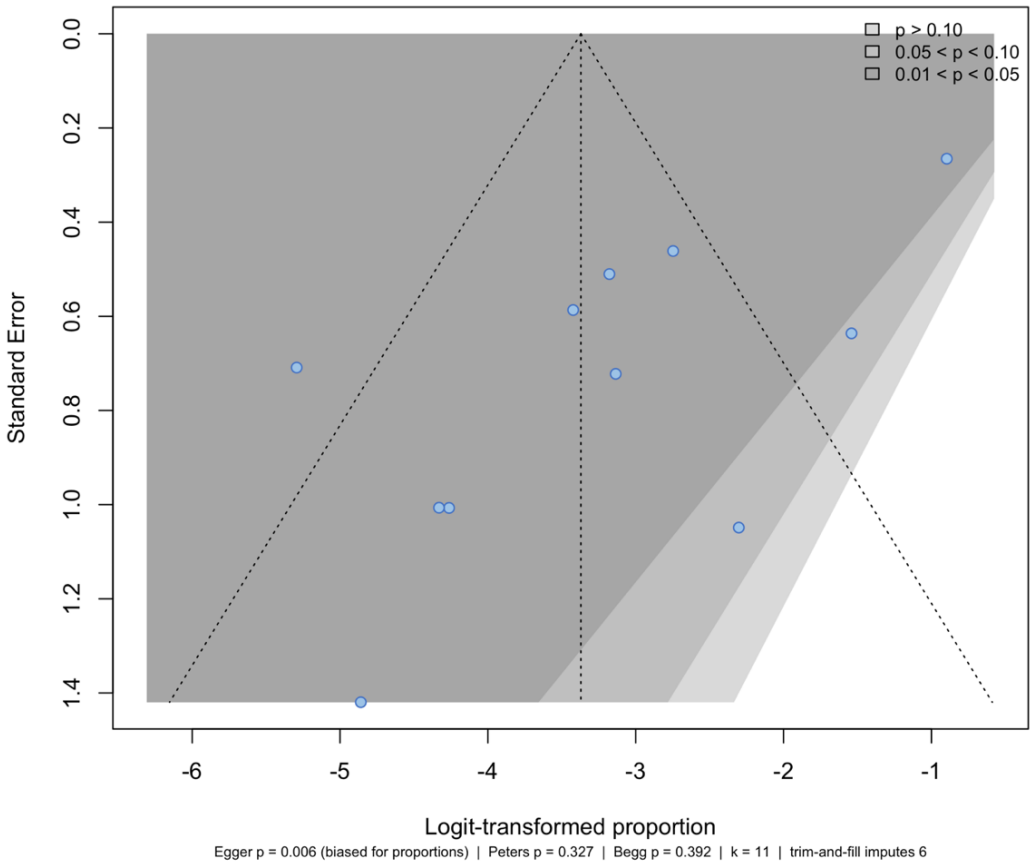

Figure 12. Supplementary. Contour-enhanced funnel plot for the kidney replacement therapy pool. The apparent asymmetry is the standard-error artefact described in the Results and is not evidence of publication bias.

4. Discussion

This is the first quantitative synthesis of clinical outcomes in acute post-streptococcal and post-infectious glomerulonephritis. Its central finding is that the largest single determinant of the reported risk of acute kidney injury is not the population a cohort serves but the instrument it uses. Studies applying a bare creatinine or estimated glomerular filtration rate threshold reported almost four times the prevalence of studies that stated no criterion, with consensus criteria between the two, and this gradient runs in exactly the direction that measurement theory predicts: a fixed threshold applied without regard to age or size will capture many children whose creatinine is normal for their body habitus, whereas a complication recorded ad hoc in a discharge summary will capture only the cases that were clinically conspicuous.

That finding inverts the usual reading of heterogeneity. An I^2 of 95% would ordinarily be treated as a nuisance that undermines the pooled estimate. Here two thirds of it has a name, and the practical implication is immediate: a paediatrician comparing their unit's acute kidney injury rate against a published figure is, in most cases, comparing definitions rather than populations. Before concluding that a local rate is anomalous, the first question to ask is which criterion the comparator applied.

The consequence for the headline number is that this review reports two. Across all cohorts the prevalence is 34.7%; across the cohorts that stated a definition it is 49.6%. The gap is not a technicality. It means that the familiar figure of roughly one third, which the all-studies estimate would support, is held down by eight cohorts in which the outcome cannot be verified to have been measured at all. The honest summary is that between a third and a half of children presenting with this disease develop acute kidney injury by some defensible criterion, and that the literature is not currently able to be more precise than that.

The association with income setting is real but harder to interpret, and a negative result is reported alongside it. The obvious explanation — that high-income settings look worse because sporadic disease presents late and severely while endemic settings capture milder disease through active case finding — was pre-specified, tested by reclassifying every cohort on endemic versus sporadic burden, and rejected. A residual confound nevertheless remains: definition class and setting are correlated in this literature, since only one of the six sporadic-setting cohorts left its criterion unspecified against seven of fifteen endemic-setting cohorts, so the two coefficients cannot be cleanly separated at this sample size. Diagnostic intensity, biochemical monitoring frequency and threshold for admission plausibly differ across settings and were not measurable from the published reports.

The age finding refines rather than contradicts existing teaching. Within childhood the slope is steep and remarkably consistent: three cohorts on three continents, using different adjustment sets and different outcome definitions, agree that each additional year of age raises the odds of an adverse kidney outcome by about 17%, with no measurable heterogeneity between them. That three independent groups converge so closely on a quantity none of them set out to estimate is the most reassuring signal in this review. The much flatter adult slope suggests that the clinically important gradient lies within the paediatric age range rather than between children and adults, which is a different claim from the familiar assertion that adult-onset disease carries a worse prognosis, and one that a single adult cohort can only propose[46,47]. Heavy proteinuria at presentation behaves as a conventional marker of glomerular injury severity, consistent with single-cohort reports linking proteinuria level to renal outcome[38].

These results should be read against the recognised limitations of the underlying literature. Loss to follow-up is severe and non-random in exactly the populations at highest risk, which biases any estimate of later outcome towards benignity[12,17]. Complement-pathway abnormalities are now known to underlie a proportion of atypical and persistent cases, and none of the included cohorts screened systematically for them, so an unknown fraction of what is counted here may be C3 glomerulopathy presenting acutely[21,23,24]. Extrarenal complications, including hypertensive neurological emergencies, are reported inconsistently and could not be pooled[25].

What this review cannot answer

Three questions readers will reasonably ask cannot be answered from these data, and it is better to say so than to imply otherwise. The first is whether the children who develop acute kidney injury are the same children who later develop chronic kidney disease. The included cohorts report acute outcomes and, in a minority of cases, status at three months to one year; none links an individual's acute kidney injury status to a long-term endpoint in a form that could be pooled, and the twenty-year follow-up cohort that does report long-term outcomes arose from a non-group-A streptococcal epidemic and was held out of the primary pool for that reason[19]. The second is the true prevalence restricted to confirmed streptococcal disease: a re-analysis restricted to serologically confirmed cohorts was not computed, and no estimate for it is offered here. The third is the population incidence of the disease, which three retrieved studies report per 100,000 and which cannot be combined with prevalence data[2,6].

Strengths and limitations

The principal strengths are the breadth of the search, which paired broad-concept with title-anchored strategies across two databases and de-duplicated complete identifier lists in code; the use of an estimator appropriate to sparse and extreme proportions; the reporting of a prediction interval alongside every pooled estimate; the pre-specification and honest reporting of a moderator hypothesis that failed; and the substitution of Peters' test for Egger's, with a declared refusal to report a trim-and-fill adjusted value that the method manufactures.

The limitations are substantial and largely inherited. Every included study is observational and most are retrospective and single-centre; 17 of the 21 cohorts contributing to the primary outcome are at high or very high risk of bias, driven by outcome measurement. Certainty is very low for every outcome. Nine of the 26 pooled cohorts did not label their population as post-streptococcal, and the title of this review was broadened accordingly; a restricted re-analysis of the serologically confirmed subset was not performed. The adult stratum contains a single cohort, so every adult-versus-paediatric contrast is descriptive. Two contributing cohorts have denominators below twenty. Authors of reports with missing numerators were not contacted, so some of the twelve reports excluded for that reason might have been recoverable. Four further cohorts would have been eligible but were paywalled with no open full text and no numerator in the abstract, which is a limitation of access rather than of searching. Finally, the review was not prospectively registered.

A minimum reporting standard for kidney injury in these cohorts

The most useful thing this review can offer is not a number but a reporting requirement. If the definition a study applies accounts for a fourfold spread in its headline result, then the field cannot accumulate comparable evidence until definitions are stated. The proposed standardised surveillance case definition for APSGN establishes who counts as a case but does not prescribe how kidney injury within a case should be graded, and that gap is where this literature loses its comparability[13]. Table 12 sets out the minimum this review would ask of any future cohort. Every item in it is derived from a specific failure observed in the studies synthesised here, and none of it requires resources that a unit able to run a cohort does not already have.

Table 12. Proposed minimum reporting standard for acute kidney injury in post-streptococcal and post-infectious glomerulonephritis cohorts.

Item	What should be reported	Why it matters here
Consensus criterion and version	State whether KDIGO, AKIN or paediatric RIFLE was applied, and which version	Definition class alone accounted for a fourfold spread in reported prevalence across this literature
Baseline creatinine source	State how the reference creatinine was obtained: a true pre-morbid value, a nadir during admission, or an age-based reference range	A pre-morbid creatinine is rarely available in this population; at least one included cohort solved this explicitly by substituting an age-based baseline, and readers cannot judge comparability without knowing
Stage distribution	Report the number of patients at each AKI stage, not a binary count	A binary count conceals whether a cohort is dominated by stage 1 disease; stage distribution was available in only a minority of included cohorts
Explicit numerator and denominator	Give the count and the denominator for every outcome, including outcomes that did not occur	Twelve otherwise eligible reports were excluded from this review solely because no numerator could be recovered
Denominator for each outcome separately	State the denominator for each outcome where these differ (for example, a biopsied subset)	Discordant denominators within a single report are otherwise indistinguishable from errors
Aetiological confirmation	State how streptococcal aetiology was established, and in how many patients	Nine of the 26 cohorts pooled here did not label their population as post-streptococcal, which is the principal source of indirectness in this synthesis
Follow-up completeness	Report the proportion followed up at each time point and the characteristics of those lost	Loss to follow-up in the included cohorts reached 43% and was concentrated in the most remote and highest-risk populations

AKI, acute kidney injury; AKIN, Acute Kidney Injury Network; KDIGO, Kidney Disease: Improving Global Outcomes; RIFLE, Risk, Injury, Failure, Loss, End-stage.

Implications for clinical practice

For the clinician at the bedside, four statements are supportable. First, between a third and a half of children admitted with this disease will meet a defensible definition of acute kidney injury, most will recover the acute episode, and the probability of requiring dialysis is of the order of 2 to 3%. Second, the risk gradient across childhood is real and continuous rather than threshold-based: the pooled estimate is an increase of about 17% in the odds of an adverse kidney outcome per year of age, so an adolescent carries appreciably higher risk than a five-year-old, and no single age cut-point is supported by these data. Third, heavy or nephrotic-range proteinuria at presentation is associated with roughly a 2.7-fold increase in the odds of an adverse outcome and is the most readily available marker at admission; where it is accompanied by a rising creatinine despite diuresis, the clinical picture that the included cohorts labelled rapidly progressive glomerulonephritis, earlier consideration of biopsy is reasonable. A New Zealand biopsy series found that the pre-biopsy estimated glomerular filtration rate predicted glomerular crescents better than any other variable tested, which is the closest the literature comes to a threshold, but no included study tested a biopsy trigger prospectively and this review cannot define one[48].

Fourth, and most practically, a unit whose own acute kidney injury rate differs markedly from a published figure should compare definitions before concluding that its patients differ. The results here imply that a unit applying KDIGO will report roughly twice the rate of a unit recording acute kidney injury ad hoc, and roughly half the rate of a unit applying a fixed adult creatinine threshold to children, without any difference in the underlying population. As for monitoring, the included cohorts followed survivors at intervals ranging from a single discharge assessment to twelve weeks and beyond, and no comparative evidence exists on which schedule detects late deterioration; this review therefore cannot recommend an interval, and the absence of that evidence is itself a finding worth stating.

5. Conclusion

Acute kidney injury complicates between a third and a half of presentations of acute post-streptococcal and post-infectious glomerulonephritis — 34.7% across all cohorts and 49.6% across those that stated a definition — but the reported figure depends more on how a centre defines acute kidney injury than on the population it serves. Definition class and income setting together explain two thirds of the variation between cohorts, and definition class alone spans a fourfold range. Kidney replacement therapy is required in about 2 to 3% and rapidly progressive or crescentic glomerulonephritis occurs in about 7%. Within childhood, each additional year of age raises the odds of an adverse kidney outcome by about 17%, and heavy proteinuria at presentation roughly triples them. Certainty in all of these estimates is very low. The single change that would most improve this evidence base is the adoption of a minimum reporting standard for kidney injury, and one is proposed here.

Declarations

Ethical clearance

Ethical approval was not required. This study is a synthesis of previously published aggregate data; no new human participants were enrolled, no identifiable patient data were accessed and no additional clinical specimens or records were obtained for the purpose of this work.

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Conflict of interest

The authors declare that they have no conflict of interest.

Author contribution

Study conception and design: GPD, HH. Literature search and study selection: GPD. Data extraction and risk-of-bias assessment: GPD, HH. Statistical analysis: GPD, HH. Interpretation of results: GPD, HH. Manuscript writing: GPD. Manuscript editing and critical revision: HH. All authors read and approved the final manuscript.

Data availability

All data analysed in this review are contained within the published reports of the included studies and within the tables presented here. The extraction sheet and the analysis dataset are available from the corresponding author on reasonable request.

Generative artificial intelligence use

Generative artificial intelligence tools were not used to assist with language editing and with the preparation of tables and figures. All authors reviewed and approved the final content and take full responsibility for the accuracy, integrity and originality of the work.

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