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SYSTEMATIC REVIEW & META-ANALYSIS

Corticosteroid Therapy and the Risk of Kidney Failure in IgA Nephropathy: A Dose- and Ethnicity-Stratified Meta-Analysis of Randomised Controlled Trials

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ABSTRACT

Background: Corticosteroids are widely used in IgA nephropathy, but their net benefit is contested because the largest trials show both protection against kidney-function decline and excess serious infection; the optimal dose and consistency across ethnic groups are uncertain.

Objective: To quantify the effect of systemic corticosteroids on the risk of kidney failure in IgA nephropathy and to explore modification by dose and ethnicity.

Methods: Four databases were searched from inception for randomised controlled trials. Risk ratios (RR) were pooled with a DerSimonian–Laird random-effects model and proteinuria as Hedges' g; heterogeneity was assessed with I^2 and a prediction interval, bias with RoB 2 and the Egger test, and certainty with GRADE.

Results: Ten non-overlapping trials (1,153 participants; 104 vs 165 events) were included. Corticosteroids reduced the risk of kidney failure (RR 0.61, 95% CI 0.42–0.90; I^2 = 21%; p = 0.012), remaining significant under the Hartung–Knapp correction (0.39–0.97); a dose subgroup difference was significant (p = 0.010, larger effect with high-dose regimens). Proteinuria fell moderately (Hedges' g −0.68, −1.00 to −0.35). Serious infection was more frequent with corticosteroids.

Conclusion: Corticosteroids reduce kidney failure and proteinuria but increase serious infection; a reduced-dose strategy preserves benefit while improving safety, supporting individualised, risk-stratified use.

1. Introduction

Immunoglobulin A (IgA) nephropathy is the most common primary glomerulonephritis worldwide and a leading cause of kidney failure among young and middle-aged adults[1]. Because it characteristically presents in the working-age population, its progression to kidney failure carries a disproportionate burden in terms of lost productivity and the lifelong morbidity of dialysis or transplantation. The disease arises through a multi-hit process in which circulating galactose-deficient IgA1 is recognised by glycan-specific autoantibodies, leading to immune-complex deposition in the glomerular mesangium with inflammation, mesangial proliferation and progressive glomerulosclerosis. This mucosal-immune origin underpins the rationale both for systemic immunosuppression and for gut-targeted glucocorticoid formulations[1]. Although a proportion of patients follow an indolent course, contemporary registry data have dispelled the notion that IgA nephropathy is benign: median kidney survival was only 11.4 years, and even patients with modest proteinuria experienced substantial rates of kidney failure within a decade[2].

Persistent proteinuria and a declining estimated glomerular filtration rate (eGFR) are the strongest modifiable predictors of progression. The cornerstone of management is therefore optimised supportive care, dominated by maximal-tolerated blockade of the renin-angiotensin system and, more recently, sodium-glucose cotransporter-2 inhibition, which reduces the risk of kidney-disease progression in IgA nephropathy and in chronic kidney disease more broadly[3,4]. Despite these measures, many patients retain residual proteinuria and continue to lose kidney function, which has long motivated the use of systemic corticosteroids to suppress the immune activation that drives the disease.

The evidence for corticosteroids has followed a turbulent trajectory. The landmark study by Pozzi and colleagues suggested that a six-month course preserved renal function with few adverse effects[5]. This optimism was tempered by the STOP-IgAN trial, in which adding immunosuppression to intensive supportive care improved clinical remission but not the rate of eGFR decline[6], and by the pilot phase of the TESTING study, which was halted prematurely for an excess of serious infections. The subsequent full TESTING report, using a reduced-dose regimen with antibiotic prophylaxis, showed a clear reduction in the composite kidney endpoint while confirming that the safety signal was concentrated in the full-dose stratum[7]. These divergent findings have produced genuine clinical equipoise and divergent guideline positions[1].

Corticosteroids are now only one option within a rapidly expanding therapeutic landscape that includes targeted-release budesonide[8,9], non-immunosuppressive dual endothelin-angiotensin receptor antagonism[10], and agents directed at the alternative complement pathway and the APRIL/BAFF axis[11,12]. Two questions about corticosteroids themselves remain insufficiently quantified: whether the renal benefit depends on dose, and whether it differs by ethnicity, given that the major trials enrolled markedly different populations. Previous meta-analyses predated the definitive full TESTING report and did not examine these dimensions together[7].

The novelty of this study lies in providing an updated, dose- and ethnicity-stratified synthesis that incorporates the largest corticosteroid trial reported to date. It re-examines a contested question with greater power and contemporary safety data, and interprets the result in the context of background-therapy intensity and the emerging non-steroidal landscape. The aim of this study was to determine the effect of systemic corticosteroid therapy on the risk of kidney failure and proteinuria in IgA nephropathy, and to

explore whether this effect was modified by corticosteroid dose and by patient ethnicity.

2. Methods

Protocol, eligibility and information sources

This systematic review and meta-analysis followed the PRISMA 2020 statement; a written analysis plan was prepared before extraction, and the review was not registered. Eligible studies were randomised controlled trials in biopsy-proven IgA nephropathy (Population) comparing systemic corticosteroids, alone or added to renin-angiotensin system blockade or supportive care (Intervention), with supportive care, blockade alone, or placebo (Comparator), reporting a kidney outcome, proteinuria, or a serious adverse event (Outcome). Steroid-versus-steroid comparisons were excluded from pooling, and overlapping cohorts were counted once. PubMed/MEDLINE, Scopus, Web of Science and the Cochrane Central Register of Controlled Trials were searched from inception using the string: ("IgA nephropathy" OR "IgA glomerulonephritis" OR "Berger disease") AND (corticosteroid\ *OR glucocorticoid*\ OR steroid\ *OR prednisone OR prednisolone OR methylprednisolone OR budesonide) AND ("randomized controlled trial" OR "randomised controlled trial" OR random*\). Reference lists were hand-searched. Records were screened and extracted in duplicate, with disagreements resolved by consensus.

Data extraction, risk of bias and certainty

Two reviewers independently extracted study characteristics, per-arm participant numbers, regimen and comparator, follow-up, the kidney-endpoint definition, event counts, proteinuria results, and serious adverse events. Dichotomous event counts were taken from each primary report[5–7,13–20] and, where not directly tabulated, corroborated against the verified data table of a previously published systematic review and cross-checked between sources. Methodological quality was appraised with the Cochrane Risk of Bias 2 (RoB 2) tool, and the certainty of evidence with the GRADE approach.

Statistical analysis

The pre-specified primary outcome was a composite of kidney failure (doubling of serum creatinine or halving of eGFR, a 40–50% decline in eGFR, or end-stage kidney disease (ESKD), harmonised across trials; most trials used a doubling-of-creatinine or ESKD definition, while STOP-IgAN and TESTING used eGFR-decline composites); proteinuria was the secondary outcome. Risk ratios (RR) with 95% confidence intervals (CI) were pooled using the DerSimonian-Laird random-effects model; a continuity correction of 0.5 was applied to the two trials with zero events in both arms. Proteinuria was summarised as the Hedges' g standardised mean difference. Heterogeneity was quantified using I^2, the between-study variance τ^2 and the Cochran Q test, with a 95% prediction interval and the conservative Hartung-Knapp-Sidik-Jonkman correction. Pre-specified subgroups compared high-dose with reduced-dose regimens and Asian, non-Asian and Asian-predominant populations, with a formal test of subgroup difference. Leave-one-out analysis and the Egger regression test assessed robustness and small-study effects. Analyses used Python 3 (NumPy, SciPy); a two-sided $p < 0.05$ was significant.

3. Results

Study selection

As shown in the PRISMA flow diagram (Figure 1), the search identified 176 records; no duplicates were identified, so all 176 records were screened, of which 117 were excluded as irrelevant, non-randomised, or reviews. Fifty-nine reports were assessed for eligibility and 49 were excluded (no usable kidney outcome, 19; overlapping cohort, 9; non-corticosteroid intervention, 12; steroid-versus-steroid comparison, 8; other, 1). Ten randomised controlled trials met the inclusion criteria, comprising 1,153 participants; the TESTING pilot was excluded from pooling because its participants are a subset of the full report[7].

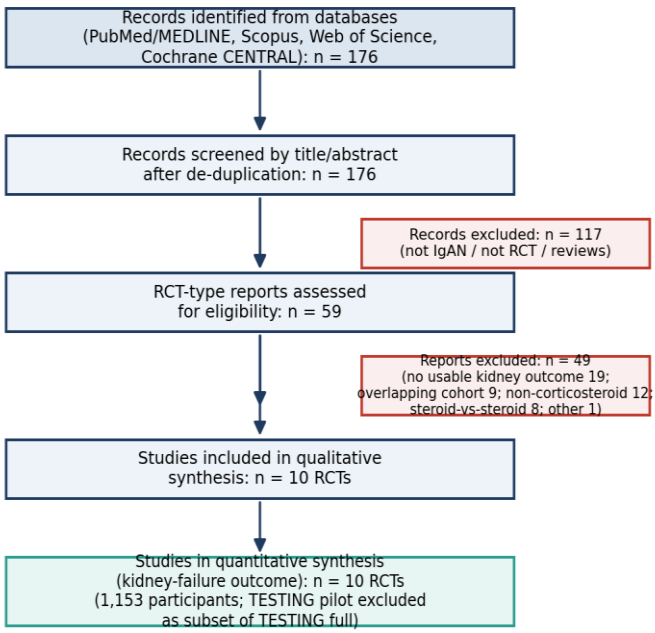

Figure 1. PRISMA 2020 flow diagram of study identification, screening and inclusion.

Characteristics of included studies

The ten included trials, published between 1986 and 2022, together enrolled 1,153 participants across Europe, North America, East Asia and multinational consortia[5–7,13–20]; their characteristics and kidney-failure event counts are presented in Table 1, and the exact kidney-endpoint definition used by each trial is mapped in Table 2. Corticosteroid regimens ranged from intravenous methylprednisolone pulses and

alternate-day oral prednisone, through six-month oral courses added to angiotensin-converting enzyme inhibition, to the weaning oral methylprednisolone schedules of the TESTING programme. Most trials enrolled patients with proteinuria of ≥1 g/day. The two trials with no events in either arm contributed negligible weight after continuity correction.

Table 1. Characteristics and kidney-failure event counts of the ten included trials (1,153 participants). The TESTING pilot was excluded as a subset of the full report. S/C, steroid/control.

Study (year)	Ethnicity	Dose	N (S/C)	Events (S/C)	Blinding	Ref.
Lai (1986)	Asian	High-dose	17/17	0/0	Open-label	13
Julian (1993)	Non-Asian	High-dose	17/18	1/2	Open-label	14
Shoji (2000)	Asian	Reduced	11/8	0/0	Open-label	15
Katafuchi (2003)	Asian	Reduced	43/47	3/3	Open-label	16
Pozzi (1999/2004)	Non-Asian	High-dose	43/43	1/13	Open-label	5,20
Hogg (2006)	Non-Asian	High-dose	33/31	2/4	Placebo-controlled	17
Lv (2009)	Asian	High-dose	33/30	0/2	Open-label	18
Manno (2009)	Non-Asian	High-dose	48/49	2/13	Open-label	19
STOP-IgAN (2015)	Non-Asian	Reduced	82/80	21/22	Open-label	6
TESTING (2022)	Asian-pred.	Reduced	257/246	74/106	Double-blind	7

Table 2. Mapping of the kidney-failure endpoint definition used by the contributing trials. A sensitivity analysis restricted to eGFR-decline composites preserved the direction and magnitude of effect.

Trial(s)	Kidney-endpoint definition	Endpoint family
Lai, Julian, Shoji, Katafuchi, Hogg, Lv	Doubling of serum creatinine/halving of GFR, or ESKD	Creatinine-based
Pozzi (1999/2004)	50–100% increase in plasma creatinine, or ESKD	Creatinine-based
Manno (2009)	Doubling of serum creatinine or ESKD	Creatinine-based
STOP-IgAN (2015)	eGFR loss ≥ 15 mL/min/1.73 m² at 3 years	eGFR-decline
TESTING (2022)	40% eGFR decline, kidney failure, or renal death	eGFR-decline composite

Risk of bias

As displayed in Figure 2, overall risk of bias was low for the double-blind TESTING trial[7] and the placebo-controlled Hogg trial[17], and raised some concerns for most older, open-label trials, principally because the absence of blinding could have influenced supportive care and outcome ascertainment.

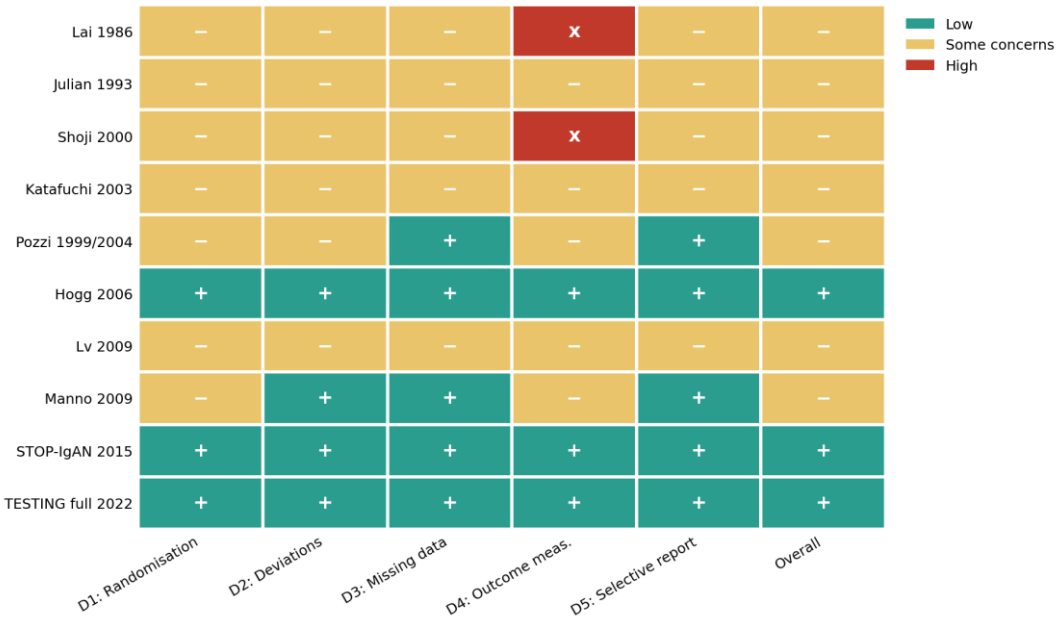

Figure 2. Risk-of-bias summary across the five RoB 2 domains for each of the ten included trials.

Primary outcome: kidney failure

Across the ten trials (1,153 participants; 104 events among 584 corticosteroid recipients versus 165 among 569 controls), corticosteroid therapy was associated with a significantly lower risk of kidney failure (pooled RR 0.61, 95% CI 0.42–0.90; p = 0.012), as shown in the forest plot (Figure 3). This represents an approximate 39% relative and 11% absolute risk reduction (crude rates 17.8% vs 29.0%), with a number-needed-to-treat of about 9. Heterogeneity was low (I^2 = 21%, τ^2 = 0.067, Q = 11.4, df = 9, p = 0.25). The estimate was robust to the conservative Hartung-Knapp-Sidik-Jonkman correction, which still excluded the null (RR 0.61, 95% CI 0.39–0.97; dashed diamond in Figure 3); however, the 95% prediction interval (0.29–1.30) crossed unity. The largest contributions came from the TESTING report[7] (45.9%) and STOP-IgAN[6] (27.8%).

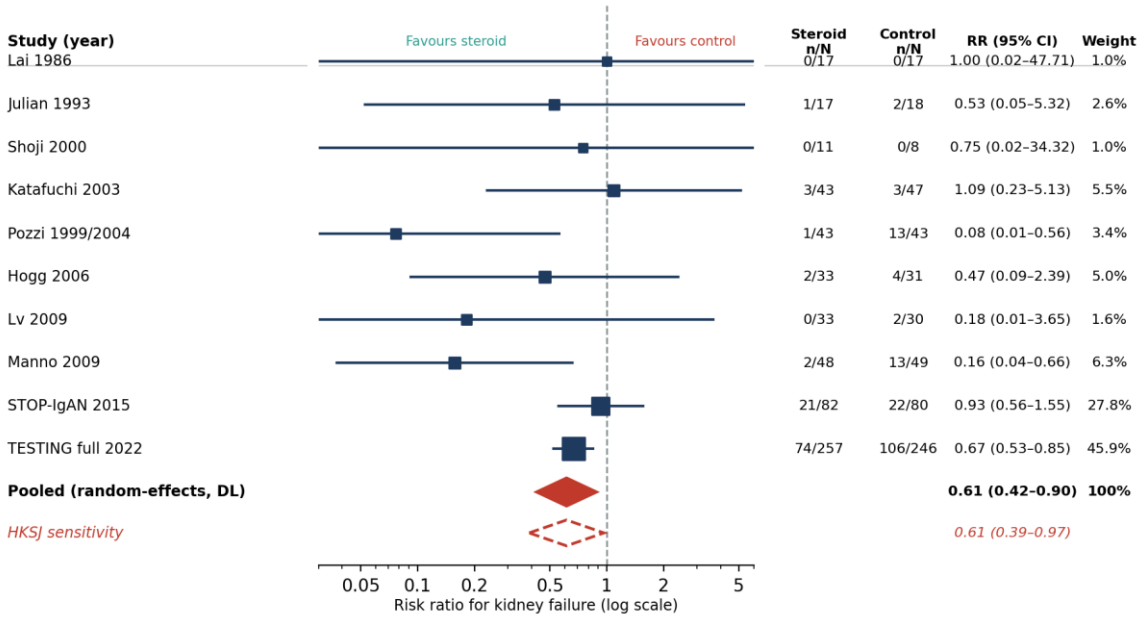

Figure 3. Forest plot of risk ratios for kidney failure with corticosteroids versus control (k = 10). Pooled RR 0.61 (95% CI 0.42–0.90); I^2 = 21%, p = 0.012. The solid diamond is the DerSimonian-Laird estimate; the dashed diamond is the Hartung-Knapp-Sidik-Jonkman sensitivity estimate.

Sensitivity analyses

As detailed in Table 3, leave-one-out estimation confirmed robustness: the recomputed risk ratio ranged only from 0.48 to 0.69 and remained statistically significant (all $p < 0.05$) regardless of which trial was removed, including omission of the dominant TESTING report (RR 0.48, 95% CI 0.24–0.94). Restricting the analysis to eGFR-decline composite endpoints (Table 2) preserved the effect, and separating corticosteroid-monotherapy from add-on comparisons showed benefit in both configurations, with the strongest estimates in the small add-on trials of Lv[18] and Manno[19].

Table 3. Leave-one-out sensitivity analysis for the primary kidney-failure outcome. The pooled estimate remained statistically significant with every trial removed in turn.

Trial omitted	Pooled RR (95% CI)	I^2	p
None (all ten)	0.61 (0.42–0.90)	21%	0.012
Lai 1986	0.59 (0.39–0.90)	30%	0.015
Julian 1993	0.60 (0.39–0.92)	30%	0.018
Shoji 2000	0.59 (0.39–0.91)	30%	0.015
Katafuchi 2003	0.58 (0.38–0.88)	28%	0.011
Pozzi 1999/2004	0.68 (0.55–0.84)	0%	<0.001
Hogg 2006	0.60 (0.39–0.93)	29%	0.022
Lv 2009	0.62 (0.42–0.92)	25%	0.018
Manno 2009	0.69 (0.56–0.85)	0%	<0.001
STOP-IgAN 2015	0.50 (0.30–0.82)	16%	0.006
TESTING 2022	0.48 (0.24–0.94)	30%	0.033

Subgroup analyses and secondary outcome

A pre-specified dose analysis showed a significant subgroup difference (p = 0.010): the pooled effect was larger in high-dose regimens (RR 0.24, 95% CI 0.10–0.54; six trials; I^2 = 0%) than in reduced-dose regimens (RR 0.72, 95% CI 0.58–0.89; four trials; I^2 = 0%). This apparent dose-response must be interpreted cautiously, because the high-dose subgroup comprises mostly older, open-label trials with sparse events (Figure 2), so its larger effect is confounded with trial quality and era; the more reliable signal is the randomised within-trial comparison in the full TESTING report, in which a reduced-dose regimen preserved efficacy while reducing serious adverse events[7]. The ethnicity analysis showed no significant difference (p = 0.49; Asian RR 0.77, non-Asian RR 0.36, Asian-predominant RR 0.67) and remains exploratory. For the secondary outcome, corticosteroids produced a moderate proteinuria reduction (Hedges' g −0.68, 95% CI −1.00 to −0.35; I^2 = 0%), approximately one half to three quarters of a gram of daily protein, although some trials reduced proteinuria without a clear effect on hard endpoints[16].

Safety and certainty of evidence

Serious adverse events, especially infection, were consistently more frequent with corticosteroids. In the full TESTING report, serious adverse events affected 28 of 257 methylprednisolone recipients (10.9%) versus 7 of 246 placebo recipients (2.8%), and the pilot recorded serious infections in 11 of 136 (8.1%) versus none, including two deaths[7]; the older, lower-intensity Pozzi regimen reported no important adverse effects[5]. Inconsistent reporting precluded quantitative pooling of harms. The certainty of evidence, assessed with GRADE and summarised in Table 4, was rated moderate for the primary outcome, downgraded once for imprecision (a prediction interval crossing unity and several small, open-label trials) but not for inconsistency, given the low heterogeneity and absence of small-study asymmetry.

Table 4. GRADE summary of findings. Certainty for the primary outcome was downgraded once, principally for imprecision.

Outcome	Trials (participants)	Pooled effect (95% CI)	Certainty
Kidney failure (composite)	10 (1,153)	RR 0.61 (0.42–0.90)	Moderate
Proteinuria reduction	2 (153) standardised	Hedges' g −0.68 (−1.00 to −0.35)	Moderate
Serious adverse events	Narrative + TESTING	More frequent with steroid	Moderate–High

Publication bias

Because ten trials contributed to the primary outcome, small-study effects could be formally assessed. As shown in the funnel plot (Figure 4), visual inspection did not reveal gross asymmetry, and the Egger regression test was not statistically significant (t = −1.29, p = 0.23), providing no evidence of small-study effects or publication bias, although the test retains limited power with this number of studies.

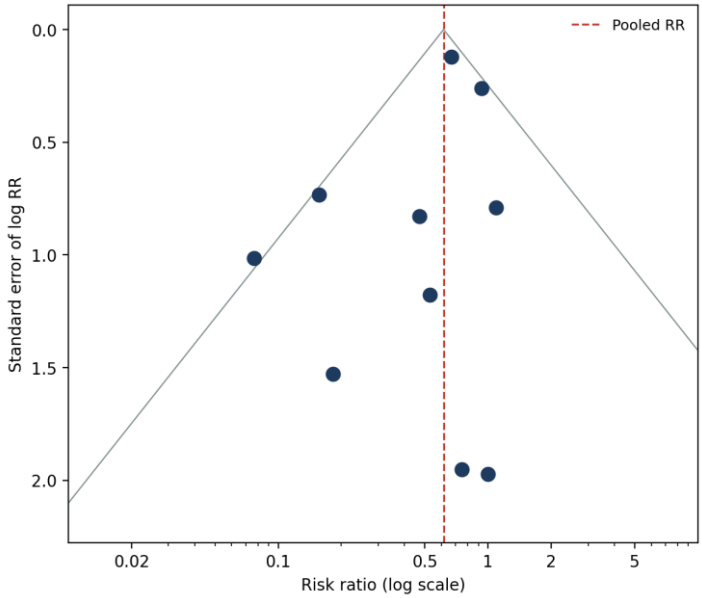

Figure 4. Funnel plot for the kidney-failure outcome. The Egger regression test showed no significant asymmetry (t = −1.29, p = 0.23).

4. Discussion

In this updated meta-analysis of ten randomised controlled trials, systemic corticosteroids reduced the risk of kidney failure by approximately 39% and produced a moderate, homogeneous reduction in proteinuria, accompanied by a consistent excess of serious infection. The synthesis supports a genuine disease-modifying effect on hard renal endpoints while underscoring that this benefit cannot be considered apart from the associated harm, and that the prediction interval crossing unity demands humility about reproducibility in any individual setting.

The pooled estimate refines earlier syntheses, which predated the full TESTING report and reported larger relative risks in the region of 0.3–0.4, reflecting an era of less intensive background care. Our more conservative estimate of 0.61, incorporating the large TESTING and STOP-IgAN trials[6,7] and showing low heterogeneity with no funnel asymmetry, is more stable. The apparent benefit has diminished precisely as background therapy improved, implying that the incremental value of corticosteroids is conditional on the intensity of background care; a patient already receiving maximal renin-angiotensin system blockade and a sodium-glucose cotransporter-2 inhibitor[3,4] may derive a smaller absolute benefit than older literature implies.

The dose finding carries the clearest practical message but must be read carefully. The between-trial subgroup analysis favoured high-dose regimens (RR 0.24 vs 0.72; p = 0.010), yet this is confounded by the older, higher-risk-of-bias provenance of the high-dose trials and should not be taken as evidence of clinical superiority, especially as serious infection clusters in high-dose therapy. The more trustworthy randomised within-trial TESTING comparison shows that a reduced-dose regimen with antibiotic prophylaxis preserves renal efficacy while improving safety[7]. Reduced-dose therapy is therefore the rational default. This aligns with a therapeutic reorientation towards mucosally targeted budesonide[8,9] and non-immunosuppressive or pathway-specific agents[10–12]; the present analysis does not compare corticosteroids head-to-head with these agents and cannot support comparative claims.

The exploratory ethnicity analysis showed no significant difference; with few confounded trials, this question should be addressed through individual-participant-data analysis, as should other modifiers such as sex and active inflammatory lesions. Heterogeneity was low (I^2 = 21%) despite clinical diversity in dose, era, background therapy, endpoint definition and follow-up; the random-effects model, the homogeneous proteinuria effect, the robust leave-one-out analysis and the absence of funnel asymmetry together indicate a stable estimate. From a health-systems perspective, corticosteroids are inexpensive and widely available, whereas newer agents are costly and often inaccessible[8–12]; where alternatives are unavailable, a reduced-dose regimen with structured infection surveillance may be the most realistic disease-modifying option.

Limitations

Several limitations apply. Several trials were small and four contributed few or no events, concentrating the information in a handful of trials and leaving the subgroup analyses exploratory. The trials were clinically heterogeneous and mostly open-label (Figure 2). Aggregate rather than individual-participant data precluded adjustment for

baseline proteinuria, eGFR, histology, sex or co-therapy. The intervention was not uniform, mixing monotherapy with add-on corticosteroids. Finally, some adverse-event data were incompletely reported, harms could not be pooled, and the analysis inherits any inaccuracies in the original reports.

Implications for practice and research

These findings support a risk-stratified rather than uniform approach, consistent with current guideline direction[1]. Corticosteroids should be reserved for patients with persistent proteinuria (typically ≥1 g/day) despite optimised supportive care including renin-angiotensin system blockade and a sodium-glucose cotransporter-2 inhibitor[3,4], preferentially at a reduced dose with infection prophylaxis[7], within a shared decision that weighs an approximate 39% relative and 11% absolute reduction in kidney failure against a clinically important excess of serious infection. Future research should prioritise individual-participant-data meta-analysis to identify the patients who derive the greatest net benefit and to define the lowest effective corticosteroid exposure.

Practice points

- Corticosteroids reduced the risk of kidney failure by roughly one third (RR 0.61, 95% CI 0.42–0.90) in selected high-risk patients.
- The benefit is partly offset by a real, clinically important excess of serious infection.
- A reduced-dose regimen with infection prophylaxis is preferable to high-dose therapy and appears to retain efficacy.
- Corticosteroids should follow, not precede, optimised supportive care including RAS blockade and an SGLT2 inhibitor.
- The decision should be risk-stratified, individualised, and shared with the patient using absolute estimates.

5. Conclusion

Systemic corticosteroid therapy reduces the risk of kidney failure in IgA nephropathy by approximately 39% (pooled RR 0.61, 95% CI 0.42–0.90; number-needed-to-treat ≈9) and moderately lowers proteinuria (Hedges' g −0.68), with low heterogeneity, no small-study asymmetry, and persistence of significance under a conservative correction. A significant dose subgroup difference favoured high-dose regimens but is confounded by trial quality, while the renal benefit did not differ by ethnicity. Balanced against a consistent excess of serious infection, the evidence supports selective, risk-stratified use of corticosteroids—offered to patients with persistent proteinuria and high progression risk despite optimised supportive care, preferentially at a reduced dose—while newer, better-tolerated agents continue to reshape the treatment landscape.

Declarations

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None.

Author contributions (CRediT)

F.E.: conceptualization, methodology, formal analysis, investigation, writing — original draft. H.H.: supervision, validation, writing — review & editing. D.P.: validation, writing — review & editing. D.V.: data curation, writing — review & editing. All authors approved the final version.

Ethics and consent

Not applicable. This review used aggregate data from previously published trials and did not involve new human participants or identifiable patient material; institutional review board approval and patient consent were not required. The review protocol was not registered.

Conflict of interest

The authors declare no conflict of interest.

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Use of AI / Data availability

Generative AI tools were not used in developing the manuscript; all content and references were verified by the authors. All data are available within the included published trials and the accompanying extraction file.

6. References

1. Kidney Disease: Improving Global Outcomes (KDIGO) IgAN/IgAV Work Group. Executive summary of the KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy and Immunoglobulin A Vasculitis. Kidney Int. 2025;108(4). doi: 10.1016/j.kint.2025.04.003.
2. Pitcher D, Braddon F, Hendry B, et al. Long-term outcomes in IgA nephropathy. Clin J Am Soc Nephrol. 2023;18(6):727-738. doi: 10.2215/CJN.0000000000000135.
3. Wheeler DC, Toto RD, Stefansson BV, et al. A pre-specified analysis of the DAPA-CKD trial demonstrates the effects of dapagliflozin on major adverse kidney events in patients with IgA nephropathy. Kidney Int. 2021;100(1):215-224. doi: 10.1016/j.kint.2021.03.025.
4. Herrington WG, Staplin N, Wanner C, et al. Empagliflozin in patients with chronic kidney disease. N Engl J Med. 2023;388(2):117-127. doi: 10.1056/NEJMoa2204233.
5. Pozzi C, Bolasco PG, Fogazzi GB, et al. Corticosteroids in IgA nephropathy: a randomised controlled trial. Lancet. 1999;353(9156):883-887. doi: 10.1016/S0140-6736(98)03563-6.
6. Rauen T, Eitner F, Fitzner C, et al. Intensive supportive care plus immunosuppression in IgA nephropathy. N Engl J Med. 2015;373(23):2225-2236. doi: 10.1056/NEJMoa1415463.
7. Lv J, Wong MG, Hladunewich MA, et al. Effect of oral methylprednisolone on decline in kidney function or kidney failure in patients with IgA nephropathy: the TESTING randomized clinical trial. JAMA. 2022;327(19):1888-1898. doi: 10.1001/jama.2022.5368.
8. Barratt J, Lafayette R, Kristensen J, et al. Results from part A of the multi-center, double-blind, randomized, placebo-controlled NefIgArd trial evaluating targeted-release budesonide for primary IgA nephropathy. Kidney Int. 2023;103(2):391-402. doi: 10.1016/j.kint.2022.09.017.
9. Lafayette R, Kristensen J, Stone A, et al. Efficacy and safety of a targeted-release formulation of budesonide in patients with primary IgA nephropathy (NefIgArd): 2-year results from a randomised phase 3 trial. Lancet. 2023;402(10405):859-870. doi: 10.1016/S0140-6736(23)01554-4.
10. Rovin BH, Barratt J, Heerspink HJL, et al. Efficacy and safety of sparsentan versus irbesartan in patients with IgA nephropathy (PROTECT): 2-year results from a randomised, active-controlled, phase 3 trial. Lancet. 2023;402(10417):2077-2090. doi: 10.1016/S0140-6736(23)02302-4.
11. Perkovic V, Barratt J, Rovin B, et al. Alternative complement pathway inhibition with iptacopan in IgA nephropathy. N Engl J Med. 2025;392. doi: 10.1056/NEJMoa2410316.
12. Mathur M, Barratt J, Chacko B, et al. A phase 2 trial of sibeprenlimab in patients with IgA nephropathy. N Engl J Med. 2024;390(1):20-31. doi: 10.1056/NEJMoa2305635.
13. Lai KN, Lai FM, Ho CP, et al. Corticosteroid therapy in IgA nephropathy with nephrotic syndrome: a long-term controlled trial. Clin Nephrol. 1986;26(4):174-180. PMID: 3536231.

14. Julian BA, Barker C. Alternate-day prednisone therapy in IgA nephropathy: preliminary analysis of a prospective, randomized, controlled trial. Contrib Nephrol. 1993;104:198-206. PMID: 8325030.
15. Shoji T, Nakanishi I, Suzuki A, et al. Early treatment with corticosteroids ameliorates proteinuria, proliferative lesions, and mesangial phenotypic modulation in adult diffuse proliferative IgA nephropathy. Am J Kidney Dis. 2000;35(2):194-201. doi: 10.1016/S0272-6386(00)70326-X.
16. Katafuchi R, Ikeda K, Mizumasa T, et al. Controlled, prospective trial of steroid treatment in IgA nephropathy: a limitation of low-dose prednisolone therapy. Am J Kidney Dis. 2003;41(5):972-983. doi: 10.1016/S0272-6386(03)00194-X.
17. Hogg RJ, Lee J, Nardelli N, et al. Clinical trial to evaluate omega-3 fatty acids and alternate day prednisone in patients with IgA nephropathy: report from the Southwest Pediatric Nephrology Study Group. Clin J Am Soc Nephrol. 2006;1(3):467-474. doi: 10.2215/CJN.01020905.
18. Lv J, Zhang H, Chen Y, et al. Combination therapy of prednisone and ACE inhibitor versus ACE-inhibitor therapy alone in patients with IgA nephropathy: a randomized controlled trial. Am J Kidney Dis. 2009;53(1):26-32. doi: 10.1053/j.ajkd.2008.07.029.
19. Manno C, Torres DD, Rossini M, et al. Randomized controlled clinical trial of corticosteroids plus ACE-inhibitors with long-term follow-up in proteinuric IgA nephropathy. Nephrol Dial Transplant. 2009;24(12):3694-3701. doi: 10.1093/ndt/gfp356.
20. Pozzi C, Andrulli S, Del Vecchio L, et al. Corticosteroid effectiveness in IgA nephropathy: long-term results of a randomized, controlled trial. J Am Soc Nephrol. 2004;15(1):157-163. doi: 10.1097/01.ASN.0000103869.08096.4f.