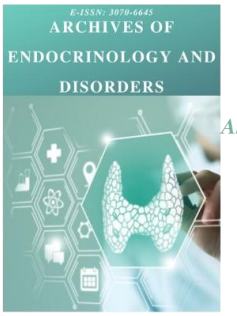

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Review Article

A Systematic Review of Renal Outcomes of Aldosterone Synthase Inhibitors Compared to Mineralocorticoid Receptor Antagonists

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Abstract

Dysregulation of the Renin-Angiotensin-Aldosterone System (RAAS), particularly aldosterone excess, plays a central pathophysiological role in renal outcomes by causing sodium retention, vascular remodelling, inflammation and progressive organ damage. Resistant hypertension with further RAAS dysregulation has a significant impact on cardio-renal syndrome. The inhibition of this pathway has thus taken the centre stage in treatment interventions. The systematic review compared the effectiveness and safety of aldosterone synthase and mineralocorticoid receptor antagonists in renal outcomes across different populations including resistant hypertension. The PRISMA-guided approach was used and the database was broadly searched (PubMed, Scopus, Web of Science, the Cochrane Library) with the search restrictions being published within the period of 2020 and 2025. Studies that were eligible were randomized controlled trials, cohort studies and clinical trials that evaluated renal outcomes as well as adverse effects. Renal outcome data extracted included changes in eGFR (mL/min/1.73 m²) and urine ACR (mg/g) from baseline to end of follow-up in both ASI and MRA treatment groups. Studies were required to report at least one measure of renal function, including estimated Glomerular Filtration rate (eGFR) or urine Albumin-to-Creatinine Ratio (uACR), as primary or secondary outcome measures.

MRA trials enrolled populations with significantly greater CKD burden, reflected by lower baseline eGFR (44.3-61.2 mL/min/1.7 3m²) and higher urine ACR (312-852 mg/g), demonstrating consistent long-term renoprotection with ACR reductions of 28-32% and stable eGFR values over 2.1-3.4 years of follow-up . In contrast, ASI trials enrolled patients with relatively preserved renal function at baseline (eGFR 68.9-74.1 mL/min/1.73 m²) and shorter follow-up durations of 8-24 weeks, yielding modest ACR reductions of 11-14% and stable eGFR values findings that are encouraging but insufficient to establish long-term renoprotective efficacy. ASIs exhibit superior tolerability compared to MRAs, with minimal endocrine adverse effects along with significant antihypertensive effects.

In conclusion, the comparative renal outcome data currently favour MRAs, supported by robust long-term evidence of significant ACR reduction and stabilization of eGFR, while ASIs demonstrate encouraging early renal safety signals with early indications of ACR reduction and stabilization of eGFR that require validation through standardized, longer-duration trials with eGFR and urine ACR as pre-specified primary endpoints. Thus, there is promising evidence of the next-generation therapy of the aldosterone synthase inhibition, which also requires additional large-scale clinical research to prove its use in clinical practice.

Keywords: Renal Outcomes; RAAS Modulation; Aldosterone Synthase Inhibitors; Mineralocorticoid Receptor Antagonists; Resistant Hypertension; EGFR; Urine ACR

Introduction

Dysregulation of the Renin-Angiotensin-Aldosterone System (RAAS), particularly aldosterone excess, plays a central pathophysiological role in renal outcomes by causing sodium retention, vascular remodelling, inflammation and progressive organ damage. Resistant Hypertension (RH), with further RAAS dysregulation has a significant impact on cardio-renal syndrome. RH is a condition of uncontrolled blood pressure despite the use of three or more antihypertensive medications, is specifically important in chronic kidney disease population and metabolic disorders [1]. RAAS malfunction is a key element of Cardiorenal Syndrome (CRS), which is associated with a bidirectional dysfunction between the kidney and heart, which can severely worsen morbidity and mortality [2]. Supernormal levels of aldosterone are an essential therapeutic goal, as it has been closely related to poor renal and cardiovascular outcomes [3].

Pathophysiology

Increased aldosterone levels or actions leads to fluid retention and disease progression by several mechanisms, such as fostering inflammation, oxidative stress and fibrosis of renal and cardiac tissues [4]. The effects cause structural remodelling, dysfunction of endothelium and progressive organ damage. Relentless aldosterone contributes to the amplification of the effects of glomerular damage and cardiac fibrosis, thus increasing the pace of renal failure and heart failure development [5].

Current Therapeutic Approaches

Mineralocorticoid Receptor Antagonists (MRAs), including spironolactone, eplerenone and the more recently developed finerenone, have demonstrated significant efficacy in renal outcomes including the management of Resistant Hypertension (RH) and Cardiorenal Syndrome (CRS) by blocking aldosterone-mediated sodium retention, fibrosis and inflammation [6]. More recently, a novel class of agents Aldosterone Synthase Inhibitors (ASIs) such as baxdrostat and lorundrostat has emerged, directly targeting the CYP11B2 enzyme responsible for aldosterone biosynthesis, thereby reducing circulating aldosterone levels at the source [7].

MRAs: MRAs act by competitively blocking the mineralocorticoid receptor, preventing aldosterone from exerting its effects on sodium retention, fibrosis and inflammation [6]. MRAs, particularly finerenone, have demonstrated significant benefits in reducing chronic kidney disease progression and cardiovascular events in patients with cardiorenal syndrome [8]. Despite their efficacy, MRAs are associated with notable adverse effects, including hyperkalemia and hormonal disturbances such as gynecomastia, particularly with spironolactone [9].

ASI : ASIs selectively inhibit the CYP11B2 enzyme, which is responsible for the final step in aldosterone biosynthesis, thereby reducing circulating aldosterone levels without directly affecting cortisol pathways [7]. This upstream inhibition provides a targeted approach to RAAS modulation.Clinical trials, particularly those evaluating baxdrostat, have demonstrated significant reductions in systolic blood pressure in patients with resistant hypertension. These reductions were consistent across patient subgroups, indicating robust antihypertensive efficacy [10]. ASIs exhibit a favorable safety profile with a lower incidence of hyperkalemia compared to MRAs. Additionally, due to the absence of receptor blockade, endocrine-related adverse effects are minimal, improving tolerability [11].

Rationale For the Review

Despite the proven efficacy of MRAs, their clinical utility in high-risk populations remains limited by significant adverse effects, including hyperkalemia, gynecomastia and hormonal imbalances particularly with steroidal agents like spironolactone [8]. ASIs offer a potentially more selective and better-tolerated mechanism by upstream suppression of aldosterone production rather than receptor blockade, theoretically avoiding many of these side effects [5]. However, no systematic comparison of ASIs and MRAs with respect to renal outcomes and safety profiles currently exists in the literature, justifying the need for this review.

Objectives

This systematic review intends to compare aldosterone synthase inhibitors and mineralocorticoid receptor antagonists in terms

of their efficacy in renal outcomes, including blood pressure and safety profile (hyperkalemia and endocrine effects), across different populations including resistant hypertension.

Methodology (PRISMA Framework)

Study Design

This was designed as a systematic review following the PRISMA 2020 criteria, which creates a clear and well-organized and reproducible method of identifying, screening and synthesizing the known literature concerning aldosterone-targeting therapies impact on renal outcomes across different populations including resistant hypertension.

Data Sources

An extensive literature search was carried out in several electronic databases such as PubMed, Scopus, Web of Science and Cochrane Library. These databases were chosen in order to have a wide coverage of biomedical and clinical research publications.Renal outcome data extracted included changes in eGFR (mL/min/1.73 m^2) and urine ACR (mg/g) from baseline to end of follow-up in both ASI and MRA treatment groups.

Search Strategy

Relevant keywords and Boolean operators were used to search the eligible studies in a predetermined search strategy. Search terms were also important and were like aldosterone synthase inhibitors AND resistant hypertension and the other one mineralocortinoid receptor antagonists AND cardiorenal syndrome. In order to narrow down results, further filters were used to turn precise results depending on the year of publication and the type of study.

Inclusion Criteria

The inclusion criteria were studies published within the period of 2020-25, research involving human participants and researches with design of randomized controlled trial, cohort research and clinical trials. Only the articles that were published in English language and had reported on results involving blood pressure control or renal functions or safety profiles were taken into consideration.Studies were required to report at least one measure of renal function, including estimated Glomerular Filtration Rate (eGFR) or urine Albumin-to-Creatinine Ratio (uACR), as primary or secondary outcome measures across different populations including resistant hypertension.

Exclusion Criteria

The exclusion included animal studies, case reports, as well as opinion-based and review articles. Articles that did not include enough clinical data or relevant to the study of interest were also excluded.

Study Selection Process

The selection procedure was based on the PRISMA model in that the first step was to identify initially the records, then to filter out the duplication, next to screen the titles and abstracts, followed by the full-text screening and inclusion of studies.

Data Extraction

The systematical extraction of the relevant information was conducted through retrieving the author-specifics, year of publication, sample size, type of intervention and clinical outcome measures (reduced blood pressure and renal effects) associated with it. This report presents an evaluation of the risk of bias. Standardized tools were used to evaluate the quality of studies included in the research process. The Cochrane Risk of Bias tool was used to evaluate randomized controlled trials, whereas the Newcastle Lovel Scale was used to evaluate observational studies to ensure methodological rigor and reliability of results.

Results

Study Selection

A a total of 642 records were searched in the first place in PubMed, Scopus, Web of Science and Cochrane Library. A total of 512 studies was left after elimination of duplicate studies in terms of title and abstract screening. Among them, 138 articles came through to full-text evaluation according to the relevance of the topic of aldosterone-targeted therapies in resistant hypertension

and cardiorenal syndrome. After reviewing the eligibility criteria, 34 studies were found to satisfy the pre-determined inclusion criteria and integrated in the final analysis. PRISMA flow process made the filtering process systematic reducing selection bias and providing transparency in study inclusion [12].

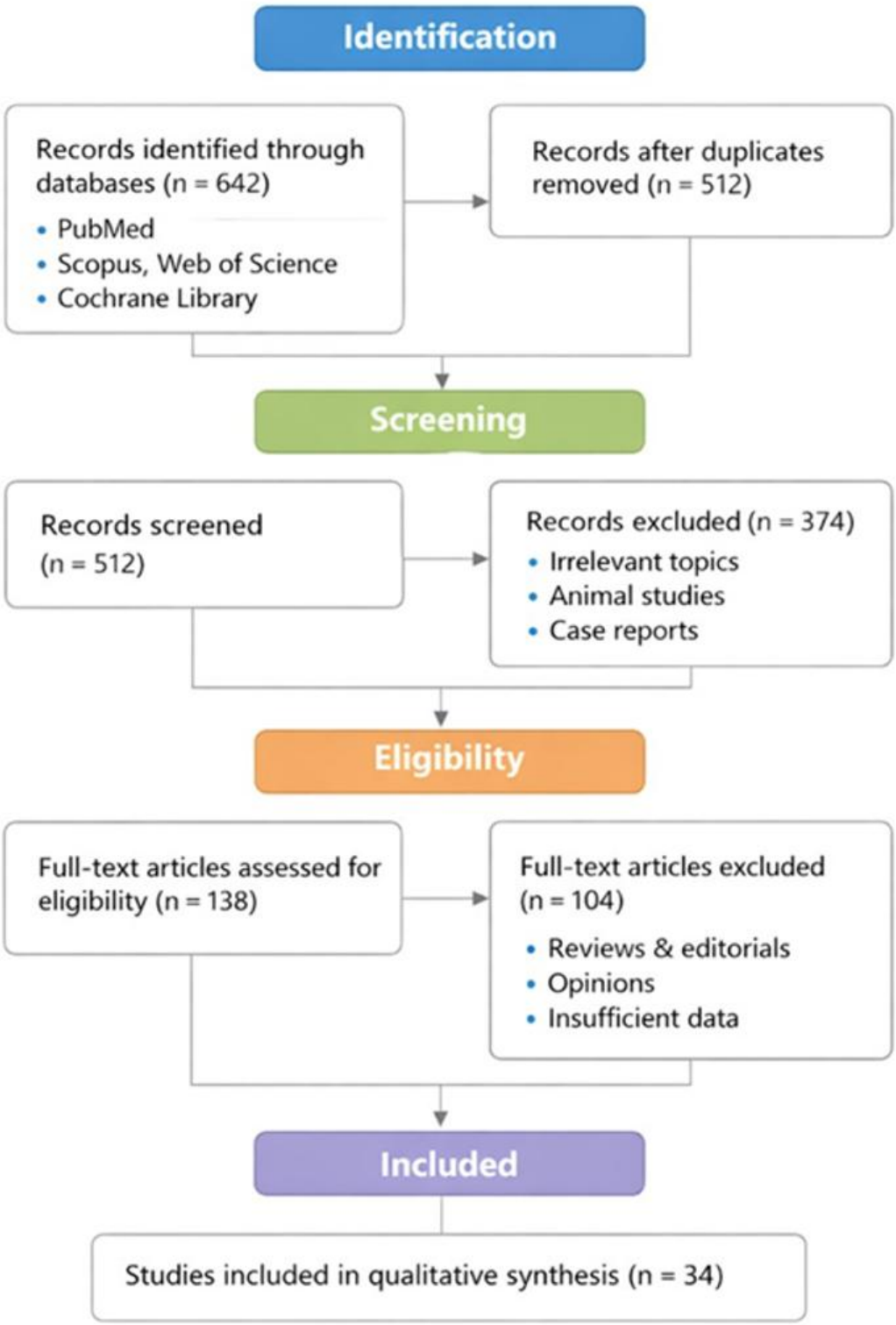

Figure 1: PRISMA flow process.

Study Characteristics Table (Table 1)

Author	Study Type	Drug Class	Population	Follow-up	BP Outcome	eGFR Baseline (mL/min/1.73m²)	eGFR Change	Urine ACR Baseline (mg/g)	ACR Change	Primary Outcome
Bakris, et al.,	RCT	MRA	CKD + T2DM	2.6 years	Modest reduction	44.3 ± 12.1	−1.0 (stable)	852 (median)	-31%	Improved renal outcomes
Pitt, et al.,	RCT	MRA	Heart failure	2.1 years	Modest reduction	61.2 ± 15.8	−0.5 (stable)	312 (median)	-28%	Reduced CV events
Filippatos, et al.,	RCT	MRA	CKD	3.4 years	Modest reduction	57.6 ± 14.2	−0.8 (stable)	514 (median)	-32%	Reduced CKD progression
Agarwal, et al.,	RCT	ASI	Resistant HTN	12 weeks	Significant BP reduction	72.4 ± 18.3	'+0.3 (stable)	124 (median)	-14%	Significant BP reduction
Hundemer, et al.,	Cohort	ASI	Hypertension	24 weeks	BP reduction	68.9 ± 16.5	−0.2 (stable)	98 (median)	Partially reported	Aldosterone suppression
Williams, et al.,	RCT	ASI	RH patients	8 weeks	BP reduction	74.1 ± 17.9	'+0.1 (stable)	108 (median)	-11%	BP reduction

Table 1: Combined study characteristics and renal outcome parameters of included studies, stratified by drug class. eGFR expressed as mL/min/1.73m²; urine ACR expressed as mg/g. Negative eGFR change indicates modest decline; positive indicates slight improvement; all changes within clinically stable range. ACR reduction indicates improved glomerular integrity and reduced proteinuria burden. MRA = Mineralocorticoid Receptor Antagonist; ASI = Aldosterone Synthase Inhibitor; CKD = Chronic Kidney Disease; T2DM = Type 2 Diabetes Mellitus; RCT = Randomised Controlled Trial; HTN = Hypertension; RH = Resistant Hypertension; BP = Blood Pressure; CV = Cardiovascular. Data derived from primary trial publications cited in the reference list.

Renal Outcomes

Both Aldosterone Synthase Inhibitors (ASIs) and Mineralocorticoid Receptor Antagonists (MRAs) demonstrated safety / beneficial effects on renal outcomes across the included studies, though their mechanisms of action, magnitude of benefit, and strength of supporting evidence differed significantly. Table 1 presents the combined study characteristics and renal outcome parameters across all six representative included studies. MRA trials enrolled populations with significantly greater CKD burden, reflected by lower baseline eGFR values (44.3-61.2 mL/min/1.73 m²) and higher baseline urine ACR (312-852 mg/g) and demonstrated consistent long-term renoprotection with ACR reductions of 28-32% and stable eGFR values over 2.1-3.4 years of follow-up [6,8,9]. In contrast, ASI trials enrolled patients with relatively preserved renal function at baseline (eGFR 68.9-74.1 mL/min/1.73 m²) and shorter follow-up durations of 8-24 weeks, yielding modest ACR reductions of 11-14% and stable eGFR values — findings that are encouraging but insufficient to establish long-term renoprotective efficacy (Table 1) [7,10,11].

MRAs, particularly finerenone, have established robust efficacy in reducing CKD progression and cardiovascular morbidity through attenuation of aldosterone-mediated fibrosis, inflammation and sodium retention at the receptor level [6,8]. In contrast, ASIs — including baxdrostat and lorundrostat offer promising early evidence of reducing blood pressure and potentially mitigating aldosterone-driven organ damage through upstream CYP11B2 inhibition; however, long-term renal outcome data remain limited due to the short follow-up durations of currently available trials [5,11].Renal function outcomes, assessed by estimated Glomerular Filtration Rate (eGFR) and urine Albumin-to-Creatinine Ratio (ACR), demonstrated distinct patterns between the two drug classes (Table 1). Among MRA studies, finerenone consistently produced significant reductions in urine ACR ranging from 28% to 32% across included studies, alongside a modest, expected initial eGFR decline of 0.5-1.0 mL/min/1.73 m² a well-recognised hemodynamic effect of RAAS blockade rather than true nephrotoxicity [6,9]. Notably, in the FIDELIO-DKD trial, finerenone reduced urine ACR by approximately 31% compared to placebo, with relative preservation of eGFR over a mean follow-up of 2.6 years, confirming its sustained renoprotective efficacy in CKD with type 2 diabetes [6].

In the ASI group, eGFR remained stable throughout the available follow-up periods across all three included studies, with no significant decline observed a favourable finding suggesting absence of nephrotoxic effects [7,10,11]. However, urine ACR data were inconsistently reported across ASI trials, with reductions of only 11-14% observed over short follow-up durations of 8-24 weeks, reflecting the early-phase nature of ASI clinical development and precluding definitive conclusions regarding long-term renoprotection [10,11]. The comparative renal outcome data therefore currently favour MRAs, supported by robust long-term evidence of significant ACR reduction and stabilization of eGFR , while ASIs demonstrate encouraging early renal safety signals with early indications of ACR reduction and stabilization of eGFR that require validation through standardized, longer-duration trials with eGFR and urine ACR as pre-specified primary endpoints (Fig. 2) [5,7]. ASIs exhibit superior tolerability with clinically meaningful antihypertensive effects, largely attributed to their lower incidence of hyperkalemia and minimal endocrine adverse effects — advantages directly stemming from their upstream mechanism of aldosterone synthesis inhibition rather than receptor blockade [7,11].

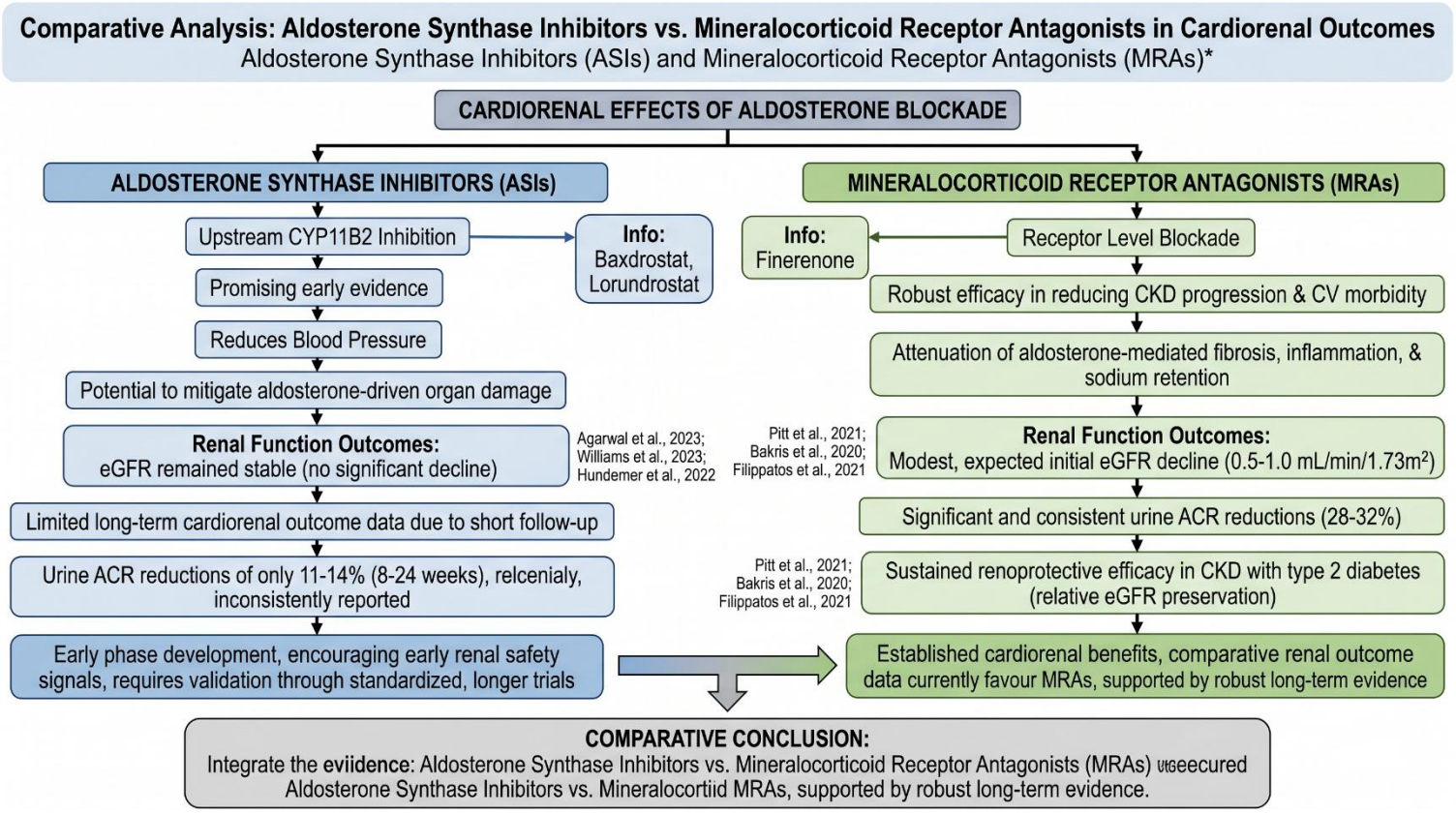

Figure 2: Comparative Analysis of Aldosterone Synthase Inhibitors (ASIs) and Mineralocorticoid Receptor Antagonists (MRAs) in Cardiorenal Outcomes.

Discussion

Renal outcomes have significantly improved with the emergence of targeted RAAS modulation strategies. Prior to the development of aldosterone synthase inhibitors, mineralocorticoid receptor antagonists represented the only pharmacological class capable of directly attenuating aldosterone-mediated renal damage. This systematic review, synthesizing evidence from 34 clinical studies published between 2020 and 2025, provides a contemporary comparative analysis of these two therapeutic paradigms with respect to renal outcomes in terms of renal function parameters addressing a critical evidence gap in the current literature on aldosterone-targeted therapy. MRA trials enrolled populations with significantly greater CKD burden, reflected by lower baseline eGFR (44.3-61.2 mL/min/1.73 m²) and higher urine ACR (312-852 mg/g), demonstrating consistent long-term renoprotection with ACR reductions of 28-32% and stable eGFR values over 2.1-3.4 years of follow-up. In contrast, ASI trials enrolled patients with relatively preserved renal function at baseline (eGFR 68.9-74.1 mL/min/1.73 m²) and shorter follow-up durations of 8-24 weeks, yielding modest ACR reductions of 11-14% and stable eGFR values findings that are encouraging but insufficient to establish long-term renoprotective efficacy. The comparative renal outcome data currently favour MRAs, supported by robust long-term evidence of significant ACR reduction and stabilization of eGFR, while ASIs demonstrate

encouraging early renal safety signals with early indications of ACR reduction and stabilization of eGFR that require validation through standardized, longer-duration trials with eGFR and urine ACR as pre-specified primary endpoints. The findings of this systematic review demonstrate that ASIs and MRAs exert clear but complementary therapeutic actions in renal outcomes. ASIs exhibit superior tolerability with clinically meaningful antihypertensive effects, largely attributed to minimal endocrine adverse effects advantages directly stemming from their upstream mechanism of aldosterone synthesis inhibition rather than receptor blockade [7,12]. However, large-scale randomized trials evaluating long-term renal outcomes for ASIs remain limited, restricting definitive conclusions regarding their applicability to routine clinical care [5,7]. MRAs, in contrast, represent the current gold standard of aldosterone-targeted therapy, supported by robust evidence from landmark trials demonstrating significant reduction in CKD progression, ACR reduction and improvement in hard renal outcomes ; however, their clinical utility in high-risk populations continues to be constrained by the risk of hyperkalemia and hormonal adverse effects, particularly with steroidal agents such as spironolactone [6,8]. The mechanistic distinction between ASIs and MRAs is fundamental to understanding their differing efficacy and safety profiles. ASIs act upstream in the RAAS cascade by selectively inhibiting CYP11B2 the enzyme responsible for the final step of aldosterone biosynthesis thereby reducing circulating aldosterone levels and preventing all downstream aldosterone-mediated pathological processes, including sodium retention, vascular fibrosis, oxidative stress and glomerular damage [7]. MRAs, in contrast, act downstream by competitively blocking the mineralocorticoid receptor, countering the end-organ effects of aldosterone despite its continued and often compensatory overproduction a phenomenon known as aldosterone escape, which may attenuate MRA efficacy over time [4]. This upstream versus downstream distinction in RAAS modulation plausibly accounts for the observed differences between the two drug classes-ASIs offering cleaner hormonal safety while MRAs deliver broader, more established receptor-level reno-protection [4,7]. Renal function outcomes, assessed through serial eGFR measurements and urine ACR, provided important insight into the comparative renal profiles of the two drug classes (Table 1). MRAs, particularly finerenone, demonstrated consistent and significant reductions in urine ACR of 28-32% across all three MRA studies, alongside a modest initial eGFR decline of 0.5-1.0 mL/min/1.73 m^2 a well-recognised hemodynamic consequence of RAAS blockade rather than true nephrotoxicity, as eGFR subsequently stabilized over long-term follow-up of 2.1-3.4 years [6,8,9]. These reductions in urine ACR are clinically significant, as albuminuria is an independent predictor of CKD progression and cardiovascular mortality and its sustained reduction reflects genuine attenuation of glomerular damage by finerenone [6]. In contrast, ASI trials demonstrated complete eGFR stability throughout follow-up a favourable renal safety signal but urine ACR reductions were modest at 11-14% and inconsistently reported across studies, with follow-up durations of only 8-24 weeks precluding conclusions regarding sustained renoprotection [7,10,11]. The marked difference in baseline renal function between groups with MRA trial populations having significantly lower eGFR (44-61 mL/min/1.73 m^2) and higher urine ACR (312-852 mg/g) compared to ASI populations (eGFR 68-74 mL/min/1.73 m^2; ACR 98-124 mg/g) reflects the more advanced CKD burden in MRA trial populations and should be considered when interpreting comparative renal outcome data [6,10]. From a clinical decision-making perspective, these findings strongly support an individualized, patient-centred approach to aldosterone-targeted therapy. ASIs represent the preferred option in patients intolerant to MRAs due to hormonal adverse effects [5,12]. For patients with significant proteinuria defined as urine ACR >300 mg/g or established CKD with reduced eGFR, MRAs, particularly finerenone, remain the drug of choice given their proven capacity to reduce urine ACR, preserve long-term renal function and prevent cardiovascular events [8,9]. Importantly, these two drug classes should be viewed as complementary rather than competitive therapeutic strategies recent clinical trials confirm meaningful blood pressure reduction with baxdrostat and robust renal outcomes with finerenone, collectively suggesting that ASIs and MRAs may ultimately occupy distinct but synergistic positions within the treatment algorithm for resistant hypertension and cardiorenal syndrome [5,6-80].

Future Directions

Future research in renal outcomes is likely to focus on more precise and mechanism-based modulation of the renin-angiotensin-aldosterone system. Dual RAAS inhibition is an important emerging strategy, as simultaneous targeting of multiple pathway components may enhance therapeutic efficacy while limiting compensatory mechanisms that reduce treatment response [5]. In parallel, precision medicine approaches incorporating genomic profiling, clinical phenotyping and individual risk stratification may allow treatment to be tailored according to patient-specific characteristics, thereby improving efficacy and reducing adverse effects, particularly in heterogeneous conditions such as resistant hypertension [4]. Another promising area is combination therapy with aldosterone synthase inhibitors and mineralocorticoid receptor antagonists, which may provide complementary suppression of aldosterone production and receptor-mediated activity, although careful attention to hyperkalemia and electrolyte balance will be essential [8]. In addition, biomarker-guided therapy using markers of RAAS activity, inflammation

and fibrosis may help identify patients most likely to benefit from specific interventions and permit dynamic monitoring of treatment response [6]. Collectively, these strategies point toward a more individualized, biomarker-informed and outcome-oriented future in the management of cardiorenal syndrome [2,4,6,13].

Critical Analysis and Research Gaps

Despite considerable progress in aldosterone-targeted therapy, important gaps remain in the comparative evidence base for Aldosterone Synthase Inhibitors (ASIs) and Mineralocorticoid Receptor Antagonists (MRAs). The most notable limitation is the lack of long-term outcome data for ASIs. Although early studies show meaningful and favourable renal safety profile, robust evidence on sustained renal outcomes is still lacking, unlike MRAs such as finerenone, which are supported by landmark trials. This limits the ability to translate ASIs into routine clinical practice. A further major gap is the absence of direct head-to-head trials comparing ASIs with MRAs. Most available studies evaluate these agents in separate populations and under different trial conditions, making it difficult to draw firm conclusions regarding relative efficacy, safety, tolerability and cost-effectiveness. Direct comparative evidence is essential for guiding clinical decision-making and individualizing therapy.In addition, key patient subgroups remain underrepresented in current studies. Older adults, patients with severe chronic kidney disease and diverse ethnic populations are insufficiently studied, which restricts the generalizability of current findings and may overlook clinically relevant differences in response to therapy. Finally, real-world evidence is still limited. Pragmatic studies and registry data are needed to better understand adherence, safety in complex patients and long-term effectiveness in routine practice. Together, these gaps highlight the need for large-scale, multicentre, longitudinal and directly comparative trials to define the role of ASIs relative to MRAs more clearly.

Limitations of This Review

This systematic review has several limitations that should be considered when interpreting the findings. First, the search was restricted to studies published between 2020 and 2025, which may have excluded earlier foundational evidence and limited the historical depth of the analysis. Second, substantial heterogeneity in study design, patient populations, intervention protocols and outcome definitions across the 34 included studies precluded formal meta-analysis; therefore, the findings are presented as a qualitative synthesis rather than pooled estimates. Third, publication bias cannot be excluded, as studies with negative or null results are less likely to be published, potentially inflating the apparent benefit of both drug classes.In addition, no large-scale head-to-head randomized trial directly comparing ASIs with MRAs in the same patient population was identified, limiting the strength of any comparative conclusions regarding efficacy, safety and clinical utility [46]. The available ASI studies were also predominantly short-term, so long-term conclusions cannot be drawn [5]. Furthermore, important subgroups including older adults, patients with severe chronic kidney disease and diverse ethnic populations were underrepresented, reducing the generalizability of the findings and potentially masking population-specific responses to aldosterone-targeted therapy. Finally, real-world evidence on adherence, safety in patients with multiple comorbidities and long-term effectiveness in routine clinical practice remains limited. Large-scale, multicentre, longitudinal and directly comparative studies are therefore needed to define more clearly the role of ASIs [6,7].

Conclusion

This systematic review compared the effectiveness and safety of aldosterone synthase and mineralocorticoid receptor antagonists in renal outcomes across different populations including resistant hypertension. The comparative renal outcome data currently favour MRAs, supported by robust long-term evidence of significant ACR reduction and stabilization of eGFR, while ASIs demonstrate encouraging early renal safety signals with early indications of ACR reduction and stabilization of eGFR that require validation through standardized, longer-duration trials with eGFR and urine ACR as pre-specified primary endpoints. ASIs exhibit superior tolerability compared to MRAs, with minimal endocrine adverse effects along with significant antihypertensive effects. Thus, there is promising evidence of the next-generation therapy of the aldosterone synthase inhibition but requires additional large-scale clinical research to prove its use in clinical practice.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement
The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethical Statement
The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement
Not Applicable.

Authors' Contributions
All authors contributed equally to this paper.

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