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ANALYSIS OF THE RELEVANCE OF MEASURES TO IMPROVE PHARMACOTHERAPY FOR KIDNEY DISEASES

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Abstract. One of the main objectives of treating chronic kidney disease (CKD) is to slow the illness's progression and lower the chance of death. Until recently, new medication classes to supplement renin-angiotensin-aldosterone system (RAAS) inhibitors as the standard of care hardly achieved their main goals. In order to determine what new information adds to the therapy landscape, this systematic literature analysis examined therapies assessed in CKD patients since 1990. The coexistence of type 2 diabetes mellitus (T2DM) and chronic kidney disease (CKD) significantly increases cardiovascular morbidity and mortality by two to three times compared to patients without CKD. CKD is a major public health concern that is sweeping the country. The care of chronic kidney disease (CKD) includes both pharmaceutical and non-pharmacological strategies, such as dietary salt reduction and lifestyle changes to lower blood pressure. Renin-angiotensin system inhibitors (RASi), sodium-glucose cotransporter-2 inhibitors (SGLT2i), glucagon-like peptide-1 receptor agonists (GLP-1RA), and mineralocorticoid receptor antagonists (MRAs) are the four main pillars of current pharmacological management, all of which have demonstrated cardiovascular and renoprotective benefits. One of the things causing kidney damage to worsen is an incomplete block of aldosterone activity, which is still a problem. Vicadrostat and other aldosterone synthase inhibitors (ASIs) may open up new possibilities for selectively blocking aldosterone synthesis while maintaining cortisol production. Reductions in albuminuria and the possibility of renal protection have been demonstrated in early-phase trials. Is it possible for ASIs to become a fifth pillar in the management of CKD and slow its progression?

Keywords. Sglt2 inhibitors, aldosterone, mineralocorticoid receptor antagonists, chronic renal disease, ace inhibitors, renal protection.

Introduction. Over 850 million people worldwide suffer from chronic kidney disease (CKD), which is still a significant public health issue because of its progressive nature, high morbidity and mortality rates, and high medical expenses. CKD significantly reduces quality of life, frequently coexists with other chronic illnesses, and frequently results in end-stage renal disease that necessitates dialysis or kidney transplantation. The most common cause of chronic kidney disease (CKD) worldwide is type 2 diabetic mellitus (T2DM). One in three T2DM patients also have CKD, according to the Centers for Disease Control. The risk of all-cause mortality, cardiovascular mortality, cardiovascular morbidity (including myocardial infarction, strokes, peripheral artery disease, and heart failure hospitalizations), and the progression of kidney disease to failure are all significantly increased when T2D and CKD coexist. In the United States, 34.5% of persons over the age of 18 fit the criteria for prediabetes, while 13%

have diabetes. Due to its connections to metabolic problems and early renal alterations, prediabetes is becoming recognized as an early warning indicator for chronic kidney disease (CKD) [1-5]. Because of this, early intervention is crucial to slowing or stopping the development of full-blown CKD. In addition to difficulties resulting from electrolyte imbalances, such as hyperkalemia, metabolic acidosis, and hypocalcemia, advanced stages of chronic kidney disease (CKD) are linked to a variety of uremic symptoms, including fatigue and pruritus. Reduced estimated glomerular filtration rate (eGFR), severe albuminuria, and eventually renal failure result from tubular and glomerular hypertrophy, sclerosis, and fibrosis as kidney function deteriorates. In addition to the danger of kidney death, individuals with chronic kidney disease (CKD) are more likely to experience cardiovascular problems prior to end-stage kidney disease. Even those with stage 2 CKD or an eGFR of less than 90 mL/min/1.73 m^2 showed an increase in cardiovascular-related mortality, according to a meta-analysis involving 1.4 million people. The National Kidney Foundation, the Kidney Diseases Outcomes Quality Initiative (KDOQI), and the international guideline group are working to better manage CKD and enhance patient care [6-11]. A grading system for CKD based on albuminuria and eGFR was created by Kidney Disease Improving Global Outcomes (KDIGO). The KDIGO guidelines state that CKD is characterized by six eGFR stages. Impaired renal function is indicated by an estimated GFR of less than 60 mL/min over a three-month period; the severity increases as eGFR declines. An eGFR of ≥90 and 60-89 mL/min/1.73 m^2, respectively, are indicative of kidney impairment (e.g., albuminuria or structural abnormalities) in CKD stages 1 and 2. Stages 3a (eGFR: 45-59 mL/min/1.73 m^2) and 3b (eGFR: 30-44 mL/min/1.73 m^2) of moderate chronic kidney disease (CKD) indicate a gradual decline in kidney function. Stage 4 (eGFR: 15-29 mL/min/1.73 m^2) and Stage 5 (eGFR: <15 mL/min/1.73 m^2 or end-stage renal disease) of advanced chronic kidney disease (CKD) indicate severe impairment or failure of the kidneys. The albumin-to-creatinine ratio (ACR) is used to categorize albuminuria into three groups. Higher levels of albuminuria are associated with worse renal and cardiovascular outcomes. The three categories of albuminuria are A1 (normal to mildly increased, <30 mg/g), A2 (moderately increased, 30-300 mg/g, formerly known as microalbuminuria), and A3 (severely increased, >300 mg/g, formerly known as macroalbuminuria). Adverse outcomes are independently predicted by both albuminuria and eGFR, and their combination increases this risk even more [12-18]. Albuminuria, which reflects persistent glomerular damage and systemic endothelial dysfunction, is a crucial part of CKD staging and a significant independent predictor of cardiovascular events and renal progression. In people with T2D, managing CKD becomes increasingly challenging. Lifestyle changes, such as giving up smoking, maintaining blood sugar levels, lowering blood pressure below 130/80, and using statins to decrease cholesterol, are the mainstay of managing cardiovascular and kidney disease in people with type 2 diabetes. Additionally advised are reducing sarcopenia, increasing stepping, limiting sitting, improving the quality and duration of sleep, aiming for at least 150 minutes of sweating per week, strengthening with resistance training. Renin-angiotensin system inhibitors (RASi) are a well-established medication for chronic kidney disease (CKD). When paired with RAS inhibition, sodium-glucose cotransporter 2 inhibitors (SGLT2i) have recently been shown to be beneficial in preventing the development of renal failure in a variety of patient populations. The "aldosterone breakthrough" phenomena that many patients receiving long-term RAS inhibition experience is noteworthy. The phenomenon is the continuation or recurrence of high aldosterone levels in spite of ongoing treatment with angiotensin receptor blockers or angiotensin-converting enzyme inhibitors. Aldosterone breakthrough reduces the long-term reno-protective advantages of RAS inhibitors by contributing to persistent sodium retention, inflammation, fibrosis, and cardiovascular remodeling [19-24].

The main purpose of the presented manuscript is to analyze the relevance of measures to improve the pharmacotherapy of kidney diseases based on the results of authoritative scientific works.

Angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) are examples of RAS inhibitors that help lower blood pressure, lessen proteinuria, and delay kidney injury. Regardless of diabetes status, new research indicates that SGLT2 inhibitors not only reduce the risk of renal failure and cardiovascular disease, but they also slow the evolution of chronic kidney disease. The main pharmacological toolkit for treating chronic kidney disease (CKD) and its related problems consists of diuretics, which help control fluid retention and hypertension, and statins, which target dyslipidemia. Because they can lower renal and cardiovascular problems by specifically blocking the RAS pathway, RAS inhibitors have become common treatments for chronic kidney disease (CKD). Angiotensin II, a strong vasoconstrictor and aldosterone release stimulant, is efficiently suppressed by ACE inhibitors and ARBs. RAS inhibitors assist maintain renal function and slow the course of CKD by lowering intraglomerular pressure, decreasing proteinuria, and minimizing renal fibrosis. RAS inhibitors lessen the mechanical stress on the glomerular capillaries by dilating the efferent arterioles, which lowers intraglomerular pressure [3-10]. This reduces proteinuria and prolonged glomerular damage. Additionally, they improve cardiovascular health by lowering blood pressure and decreasing unfavorable cardiovascular events in individuals with chronic kidney disease. Oparil et al. gave thorough explanations on how to treat hypertension, emphasizing the function of RAS inhibitors. To put it briefly, angiotensin II is a powerful vasoconstrictor that ACEs and ARBs can prevent from being produced or acting. RAS inhibitors lower systemic blood pressure by reducing peripheral resistance through the blockage of angiotensin II. It has been demonstrated that ACEs and ARBs prevent end-organ damage by stabilizing and lowering blood pressure. To counteract hyperkalemia, RAS inhibitors can require additional medications. The safety profile of RAS inhibitors has been much improved by more recent potassium binders such patiromer and sodium zirconium cyclosilicate (SZC). By attaching to potassium, which is then eliminated in stool and not further absorbed by the body, patiromer and SZC act on the gastrointestinal system. Serum potassium falls when less potassium is absorbed. These medications lessen the risk of hyperkalemia that might result from RAS inhibition by lowering serum potassium levels, particularly in patients with impaired renal function. As a result, they enable healthcare professionals to securely administer RAS inhibitors at therapeutic dosages [13-23].

Antagonists of the mineralocorticoid receptor (MRA). Because they target the mineralocorticoid receptor (MR) without having the negative side effects of steroidal MRAs, non-steroidal MRAs are a unique family of drugs that show promise in controlling chronic kidney disease (CKD). By specifically blocking MR activation, these drugs—like finerenone—modify the sodium and water balance, lower inflammation, and lessen fibrosis in the renal and cardiovascular system. Finerenone has a longer half-life and a stronger body of data in CKD populations than eplerenone, and it is linked to a lower risk of hyperkalemia than spironolactone as compared to earlier MRAs. Although finerenone has a high affinity for the receptor, use caution when taking drugs that can activate the cytochrome p450 enzyme 3A4 because this selective antagonism reduces the expression of profibrotic and proinflammatory mediators, ultimately maintaining renal function and cardiovascular health. Patients with diabetes who also have kidney illness can use it. Aldosterone antagonists' effectiveness is limited since they do not block non-genomic receptors. Aldosterone antagonists raise renin and aldosterone levels in response to lowering blood pressure [20-26].

The future. Because of its development and correlation with cardiovascular morbidity and mortality, chronic kidney disease (CKD) continues to be a major burden in clinical practice. Due to the increasing incidence of chronic kidney disease (CKD), particularly in individuals with type 2 diabetes, healthcare professionals must manage their patients' conditions in a way that strikes a balance between current and new treatments. Using drugs like RAS inhibitors, SGLT2is, GLP-1RA, and MRAs, the main goals of contemporary CKD care are blood pressure control, albuminuria reduction, and cardiovascular risk reduction. Despite its effectiveness, the renin-angiotensin system is still partially blocked, which exacerbates the nongenomic effects of aldosterone on the body, including kidney fibrosis and inflammation [5-12]. ASIs are a new class of medications designed to inhibit the production of aldosterone without the negative consequences of MRAs, like hyperkalemia. Vicadrostat is becoming more popular since it selectively inhibits the synthesis of aldosterone while maintaining the production of cortisol. The Tuttle study showed significant drops in the urine-to-creatinine ratio in CKD patients using standard care treatments, such as RASi and SGLT2i, indicating potential further renal protection with current treatments. In Tuttle et al.'s phase 2 trial, vicadrostat was found to reduce albuminuria by about 39.5% from baseline in the treatment group. When compared to a placebo, this decrease was statistically significant ($p<0.01$). The medical community can monitor the phase 3 trial to see whether vicadrostat effectively slows the progression of chronic kidney disease (CKD) and improves cardiovascular outcomes. It's crucial to remember that vicadrostat is still being studied and hasn't proven to be better than MRAs in terms of long-term cardiovascular or renal outcomes. It is anticipated that the ongoing phase 3 trial (NCT05536803) will be finished by 2026 and will offer more information on its safety and efficacy profile. By focusing on direct channels that were not addressed, the introduction of innovative drugs like vicadrostat into clinical practice could improve the management of chronic kidney disease (CKD). In order to control renal hemodynamics, fibrosis, and inflammation while lowering hazards like hyperkalemia, ASIs may either supplement or compete with current therapies like SGLT2 inhibitors and MRAs. It will be crucial to keep investigating these possible synergies in order to optimize customized treatment plans for CKD patients [14-24].

Discussion. Over 850 million people worldwide suffer from chronic kidney disease (CKD), which is still a major public health problem because of its progressive nature, high morbidity and mortality rates, and high healthcare expenditures. CKD significantly reduces quality of life, frequently coexists with other chronic illnesses, and frequently results in end-stage kidney disease that requires dialysis or kidney transplantation. Type 2 diabetes mellitus (T2DM) is the primary cause of chronic kidney disease (CKD) worldwide. One in three individuals with T2DM also have CKD, according to the Centers for Disease Control. A wide variety of patients with any stage of CKD (eGFR 13.9–102.8 mL/min/1.73 m2) and albuminuria (UACR 29.9–2911.0 mg/g), with (75.5%) or without (20.6%) T2D, were included in the 89 clinical trials found by this systematic literature review. While assessing RAAS inhibitors, SGLT2 inhibitors, finerenone, or other drug classes, 16 trials reported significant reductions in risks of composites comprising kidney failure (n = 12) or cardiovascular mortality without kidney failure (n = 4). Numerous trials assessed the effect of treatment on one or more composite endpoints. Comparisons were hampered by the fact that these composites were varied and evaluated in a wide range of patients. Independent outcomes that are clinically objective, including renal failure and ACM, were defined more consistently. Seven of the 32 trials that reported renal failure incidences showed significant risk decreases after treatment. These included four smaller trials of losartan, benazepril, and ramipril (n = 84–436) in patients without T2D and a minor trial of losartan (n = 751) in individuals with T2D, all of which were published prior to 2008. As a result, RAAS inhibition became the norm for treating CKD patients. However, until recently, trials of

other medication classes hardly met their primary goals, indicating a lack of success in finding novel medicines to supplement RAAS inhibitors, slow progression, and enhance outcomes [4-12]. Patients with UACR ≥ 200 mg/g treated with SGLT2 inhibitors had a significantly lower risk of renal failure, according to two large trials (n = 2152 and 2202) published after 2019. The DAPA-CKD trial with dapagliflozin demonstrated that kidney-protective effects from SGLT2 inhibition may be extended to individuals with or without T2D, whereas the CREDENCE trial of canagliflozin exclusively included patients with T2D. The only example of a significant prolongation of survival in patients with CKD that has been documented to date is a significant reduction in ACM seen in the same dapagliflozin trial. Evidence from a recent systematic review confirms that well-designed clinical trials are necessary to optimize current treatments to meet this unmet need. Trials of comparatively long durations are necessary to enroll large patient groups because kidney failure and other clinical outcomes arise later in chronic kidney disease. In the early stages of CKD, surrogate endpoints can be used to track the course of the illness and assess treatments. Nevertheless, a wide variety of surrogate endpoints were found in this analysis, such as specific eGFR changes from baseline (33.7%), final eGFR values at follow-up (28.1%), eGFR slopes (16.9%), and percentage eGFR reductions from baseline (12.4%) [13-19]. Therefore, standardizing surrogate endpoints may be beneficial for future clinical trials assessing novel treatments for individuals in the early stages of CKD. Although it has been demonstrated previously that HRQoL declines as CKD progresses, this review emphasizes the dearth of evidence demonstrating that therapeutic improvements are accompanied by improvements in HRQoL. There were only five trials (5.6%) that measured HRQoL during treatment, and only one of those trials—a hydrochloric acid binder trial for individuals with metabolic acidosis—showed notable improvements. Recent attention has focused on challenges in recording changes in HRQoL, such as the quantity of instruments utilized and variations in their sensitivities. The omission of non-English-language publications and trials including individuals without albuminuria are just two of the review's shortcomings. 51.7% of trials did not disclose phase, and some phase 2 trials may have been included in violation of eligibility requirements. For 35% of non-double blind studies, a "higher" risk of bias was found. Lastly, patients with any stage of CKD, with or without T2D, and treated with any medication class since 1990 were included in this review due to the broad eligibility criteria. Patients with T2D and those without it had significantly different CKD etiologies, and a wide variety of comparators were found. Over time, surrogate and clinically objective measures of diminishing kidney function and treatment success have also changed, leading to the identification of 57 distinct composite outcomes. A meta-analysis was deemed unfeasible due to the volume and variety of the collected data [20-26].

Conclusions. Given the higher risk of cardiovascular disease, CKD is a serious public health concern, particularly for individuals with T2DM. As CKD advances, morbidity and death rise, highlighting the importance of early detection and efficient treatment. A framework for risk assessment and management is provided by the phases of CKD based on albuminuria and eGFR. RASi, SGLT2i, GLP-1RA, and MRA—the four pillars of CKD medication—have all shown benefits in protecting the kidneys and heart. An developing fifth pillar—a treatment that targets the progression of CKD caused by aldosterone—may be being developed with the study of ASIs like vicadrostat. Despite improvements in treatment, chronic kidney disease (CKD) is still a progressive illness that calls for a multidisciplinary approach that includes medication and lifestyle changes. It is important to understand that advancing and optimizing individual strategies may lower CKD-related morbidity and mortality, and that more study on ASIs may enhance outcomes for individuals with CKD.

Up until recently, only RAAS inhibitors have demonstrated the ability to slow the progression of chronic kidney disease (CKD) and lower the risk of kidney failure; however, this

evidence came from four smaller trials of people without T2D and one small trial of patients with T2D. According to recent findings from the CREDENCE, DAPA-CKD, and FIDELIO-DKD studies, a variety of renal and cardiovascular outcomes in patients with or without T2D can be considerably improved by adding an appropriate SGLT2 inhibitor or finerenone on top of standard of care RAAS inhibition. Furthermore, dapagliflozin combined with conventional RAAS inhibition may considerably reduce all-cause mortality in individuals with or without T2D, according to data from DAPA-CKD. These new drug classes may play a significant role in the treatment and management of chronic kidney disease (CKD) in the future due to the morbidity and mortality burden of the disease, the effects of CKD progression on HRQoL and healthcare costs, and the rising prevalence of risk factors like diabetes and hypertension in aging populations.

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