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DRUG UTILIZATION PATTERN OF ANTIDIABETIC MEDICATIONS IN TYPE 2 DIABETES MELLITUS PATIENTS WITH CHRONIC KIDNEY DISEASE: A COMPREHENSIVE REVIEW OF PRESCRIBING TRENDS AND CLINICAL IMPLICATIONS

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ABSTRACT

Diabetes mellitus is the leading cause of chronic kidney disease (CKD) worldwide. It significantly contributes to illness, deaths, and the burden on healthcare systems. When type 2 diabetes mellitus and CKD occur together, they create major challenges for treatment. Changes in how drugs work in the body increase the risk of side effects and make blood sugar management more difficult. This review examines how antidiabetic medications are used in patients with diabetes and CKD. A thorough search of the literature was conducted using PubMed, Scopus, Google Scholar, and Science Direct, focusing on studies published between 2014 and 2025. We included relevant observational and cohort studies that looked at prescribing trends and drug use. The evidence shows that the use of antidiabetic drugs follows a pattern based on the stages of CKD as kidney function declines. Metformin is the first choice for early CKD, but its use decreases in later stages due to safety concerns, especially the risk of lactic acidosis. Insulin therapy becomes

more common in people with moderate to severe CKD. It works well and can be adjusted in dosage; however, careful management is needed to prevent low blood sugar. Newer drugs, like sodium-glucose co-transporter-2 inhibitors and dipeptidyl peptidase-4 inhibitors, are being used more often because they have better safety records and help protect the kidneys. Despite these improvements, issues like multiple medications, economic barriers, and poor prescribing practices remain, especially in areas with fewer resources. To optimize antidiabetic treatment in CKD, it is essential to prescribe drugs rationally, follow clinical guidelines, and work as a team to improve patient outcomes and reduce drug-related problems.

KEYWORDS: Diabetes Mellitus, Chronic Kidney Disease, Drug Utilization, Antidiabetic Agents.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a common, chronic, progressive, multi-factorial metabolic disease. It is characterized by defects in insulin secretion and action combined with other dysregulated metabolic processes. Over the last few decades, T2DM has become one of the greatest public health problems in the world and the prevalence rates are increasing rapidly, mainly due to urbanization, sedentary lifestyle, changing lifestyle and diet patterns, as well as growing prevalence of obesity.[11] According to current estimations on global population, hundreds of millions of people have diabetes and the number will likely be dramatically increased during the coming decades in particular in developing countries.

The most significant and clinically important complication of T2DM is chronic kidney disease (CKD). It causes the vast majority of all moribundities, morbidities, mortalities, health-care expenditures and health impacts worldwide. Kidney damage is classified as chronic kidney disease when functional or structural abnormality of the kidney is present for 3 or more months, which adversely affects health and is usually indicated by decline in estimated glomerular filtration rate (eGFR) and/or increased markers such as albuminuria.[12] Amongst the many causes of CKD, diabetic nephropathy represents one of the highest cause-specific burdens, accounting for up to 30-50% of all the CKD cases indicating the very strong link between diabetes and CKD.[1]

Diabetic nephropathy is considered to be developed secondary to long-standing hyperglycaemia and consequent abnormalities in metabolism, leading to structural and

functional damages to the renal microcirculation. The course of diabetic nephropathy begins with hyperfiltration of glomeruli, followed by deterioration of renal function and escalation of albuminuria, and then progressive loss of renal function resulting in end-stage renal disease (ESRD) if inadequately treated. It is worth mentioning that CKD in patients with diabetes is highly comorbid with a range of complications, most important one being cardiovascular disease (CVD) that results in a large number of death in these patients.[13]

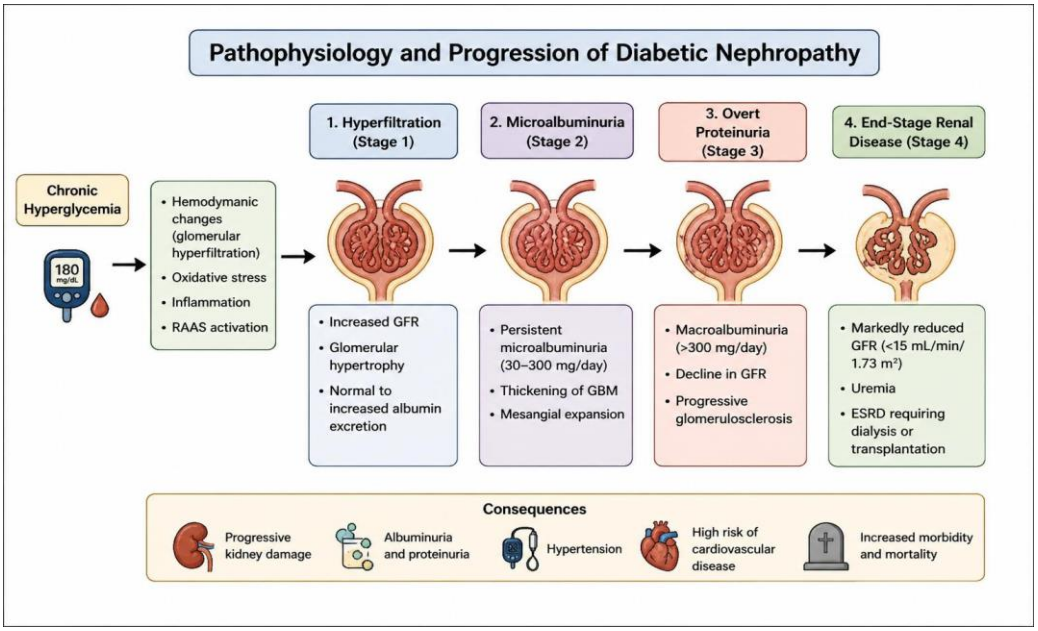


Figure highlights the pathophysiology and progression of diabetic nephropathy, which is a complication of CKD due to Type 2 Diabetes Mellitus.

Co-existence of T2DM and CKD creates challenging issues, especially with regard to pharmacotherapy. Renal impairment causes changes in pharmacokinetic parameters including absorption, distribution, metabolism and excretion. Reduction in clearance of drugs causes accumulation and enhances drug toxicity. In addition, alterations in plasma protein binding due to hypoalbuminemia and uremia also elevate free fraction of drugs and enhance drug efficacy. Tissue distribution and hepatic metabolism might also play significant role in changing drugs fate.

In terms of pharmacodynamics, patients with CKD may also exhibit an altered drug sensitivity; for example, slowed degradation of insulin due to renal impairment may increase insulin action and enhance risk of hypoglycemia. Same effects may occur with other insulin secretagogues such as sulfonylureas due to impaired clearance. As such, careful consideration must be given in selecting, titrating, and monitoring doses of antidiabetic drugs in patients

with diabetes and impaired renal function.

A variety of oral agents and drugs for management of T2DM in CKD patients are currently available, with different modes of actions, metabolism, routes of excretion, and adverse effects. The traditional oral antidiabetic agents, such as metformin and sulfonylureas, were widely used; but in the later stages of CKD, the administration of these agents must be limited due to risk of toxicity. Lately, various new classes of antidiabetic agents have been introduced to overcome such obstacles: namely dipeptidyl peptidase-4 (DPP-4) inhibitors, sodium-glucose co-transporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists have become increasingly popular due to their improved safety profiles and potential beneficial effects on kidneys and CVD.[14]

Despite these advancements in pharmacotherapy, effective management of diabetes in CKD patients remains complicated. Diabetic patients with CKD usually carry a number of comorbidities namely hypertension, dyslipidaemia and cardiovascular disease; requiring use of multitude of drugs which in turn may potentiate problems through polypharmacy leading to drug-drug interactions and adverse drug reactions.[5] Economic constraints in many cases might also limit the access of patients to more optimal therapies and thus create a gap between standard guideline recommendations and actual practice.[6]

The study of drug utilization helps in understanding prescription patterns, rationale behind drug use, guidelines compliance, and identifying areas that need improvement. With respect to drugs for treating CKD patients with diabetes, drug utilization research is crucial given the importance of tailoring treatments and minimizing risk of adverse drug effects.

Recent clinical guidelines, such as from Kidney Disease: Improving Global Outcomes (KDIGO) and American Diabetes Association (ADA), recommend a patient-focused approach to managing diabetes in CKD, prioritizing individualized therapy at each stage of disease progression and favoring agents with established benefits on both renal function and cardiovascular status.[13] Changes in clinical guidelines prescribing patterns have become significant influences on drug utilization.

Given the challenges posed by combination T2DM and CKD, and rapid development in diabetes medications, there is a critical need to extensively examine antidiabetic drug use in patients with diabetes and CKD. Information about current drug utilization practices in

relation to their appropriateness with respect to existing evidence-based guidelines can provide critical input for improving patient care in this complex high-risk group.

This review will thus be dedicated to critically review antidiabetic drug use patterns in patients with T2DM and CKD including aspects of pharmacological adaptations and clinical outcomes to identify limitations in clinical practice, knowledge gaps and suggest ways for improvement.

KDIGO guidelines recommend

- Use of **metformin in early CKD (eGFR ≥30 mL/min/1.73 m²)**
- Preferential use of **SGLT2 inhibitors for renoprotection**
- Careful dose adjustment of antidiabetic agents in advanced CKD

ADA guidelines emphasize

- Individualized glycemic targets
- Minimization of hypoglycemia risk
- Use of agents with cardiovascular and renal benefits

These recommendations highlight a shift toward **patient-centered and outcome-based therapy**, which is reflected in evolving drug utilization patterns observed in recent studies.

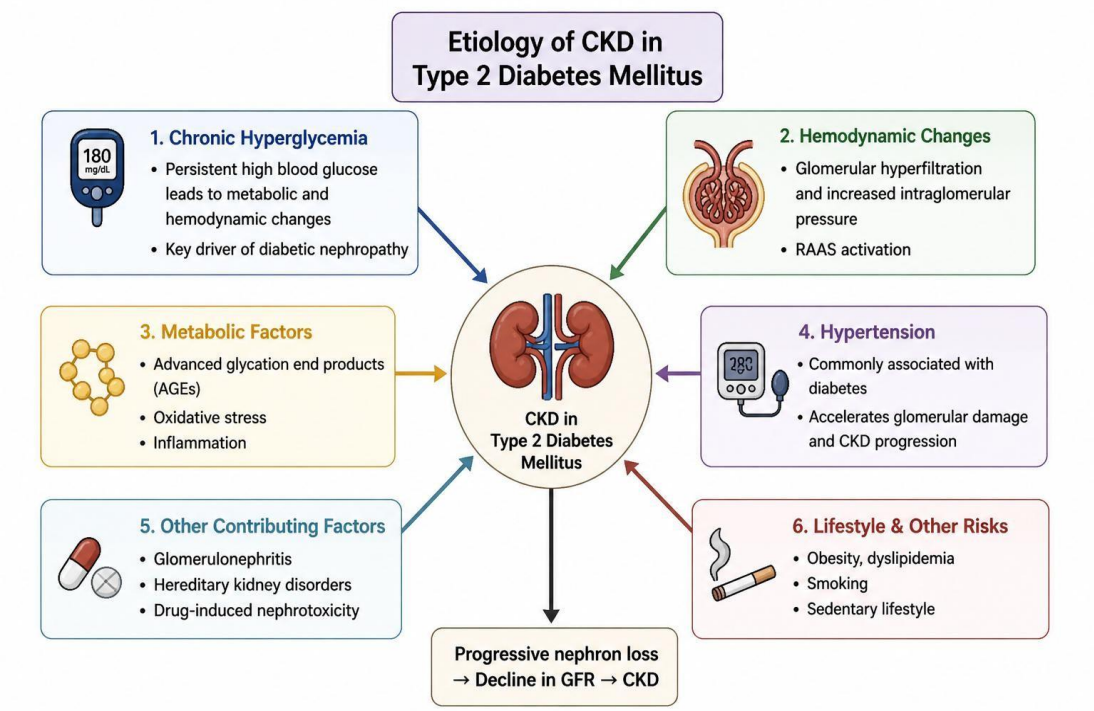
ETIOLOGY

CKD is an extremely multifactorial process, but by far the leading cause of CKDworldwide is Type 2 Diabetes Mellitus, which accounts for around 30-50% of cases.[1] Diabetic nephropathy is essentially a renal complication of diabetes caused by long-standing hyperglycaemia, and the corresponding metabolic and hemodynamic changes.

Chronic hyperglycaemia stimulates AGEs, oxidation and endothelial dysfunction, all leading to structural damage to the glomerular capillaries.[2] Stimulation of the RAAS further adds to insult through increasing intraglomerular pressure and glomerular hyperfiltration.

Diabetes is often coupled with hypertension and thus this should be regarded as a powerful promoter and enhancer of diabetic glomerular injury. While causes such as glomerulonephritis, genetic abnormalities or drug-induced nephrotoxicity can cause CKD, in diabetics they are generally only co-factors influencing rate of disease rather than original cause.

In essence, the cause of CKD in diabetes consists of both metabolic and hemodynamic influences upon a nephron population, eventually culminating in nephron loss. The impact of the foregoing on the prescribing of drugs cannot be overestimated.



ETIOLOGY OF CKD in Patients with T2DM

EPIDEMIOLOGY

CKD impacts between 10% and 14% of the worldwide population and is significantly more prevalent among Type 2 DM patients.[11] From the epidemiological literature, between 20% to 40% of patients with T2DM are found to be positive for diabetic nephropathy by some time point in their disease course.[12]

CKD is now a worldwide epidemic and the number of individuals with CKD in low- and middle-income countries is disproportionately high due to increased incidence of diabetes, diagnosis of patients late in their disease, and access to health care that results in accelerated CKD progression and complications.[6] Within a given population and locale such as South Asia, diabetic CKD constitutes a major public health problem due to rising rates of metabolic disorders.

Diabetic CKD is associated with a significantly higher rate of cardiovascular morbidity and mortality. Even at very early stages of renal disease, there is increased risk of cardiovascular

events such as MI and stroke.[13] Importantly, more patients with CKD are found to succumb to cardiovascular events than to progressive ESRD.

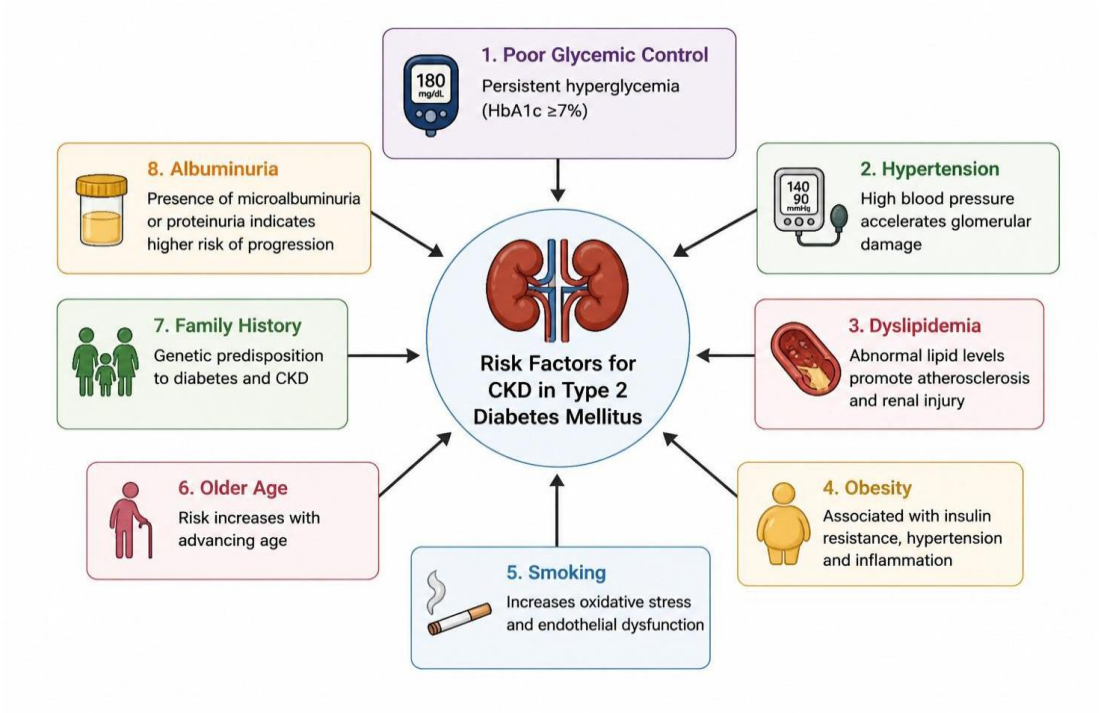
As early CKD is mostly asymptotic and leads to late diagnoses, it underscores the importance of routine screening in high-risk groups. Clinically, increasing T2DM prevalence increases the prescription of renal- function adjusted antidiabetic agents, thereby affecting drug utilization.

RISK FACTORS

The development and progression of Chronic Kidney Disease in patients with Type 2 Diabetes Mellitus are influenced by multiple risk factors, which can be classified into the following types:

Diabetes-Specific Risk Factors

- **T2DM Duration:** Longer duration is strongly associated with increased risk of nephropathy.
- **Poor glycemic control:** Persistent hyperglycemia accelerates microvascular damage.
- **Insulin resistance:** Contributes to metabolic dysfunction and renal injury.
- **Genetic predisposition:** Certain genetic factors increase susceptibility to diabetic nephropathy.[1]



Risk factors associated with CKD progression

CKD Progression Risk Factors

- **Hypertension:** Increases intraglomerular pressure, leading to structural damage.
- **Proteinuria:** Strong predictor of CKD progression and poor prognosis.
- **Obesity:** Associated with increased renal workload and inflammation.
- **Smoking:** Promotes oxidative stress and endothelial dysfunction.
- **Dyslipidemia:** Contributes to atherosclerosis and renal vascular damage.

These risk factors not only influence disease progression but also significantly affect drug response, safety, and therapeutic outcomes in CKD patients.

PATHOPHYSIOLOGY

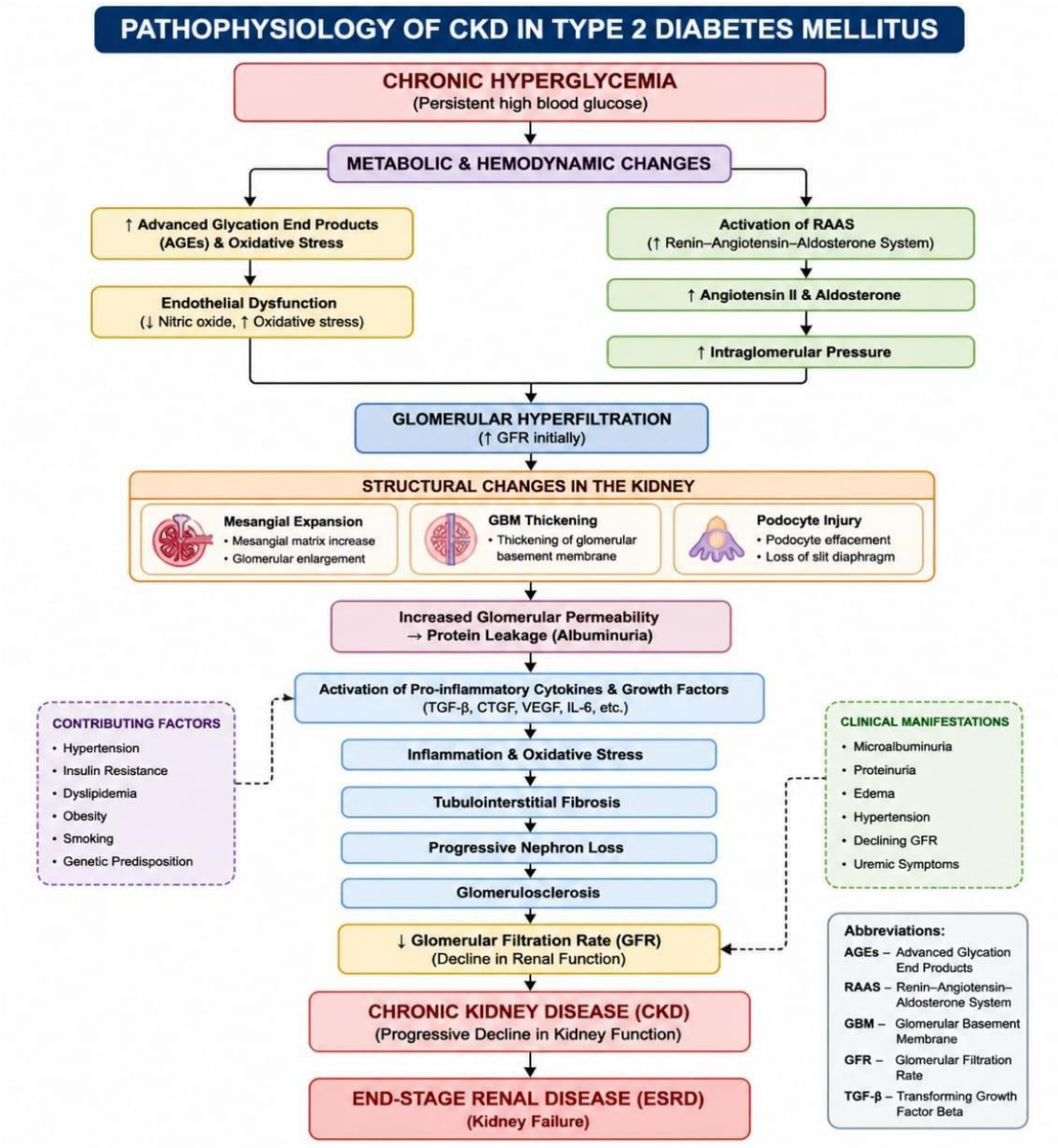
The pathophysiology of Chronic kidney disease in diabetic patients involves metabolic and hemodynamic mechanisms initiated by chronic hyperglycemia.

Persistent hyperglycemia leads to **glomerular hyperfiltration**, increased intraglomerular pressure, and activation of the renin-angiotensin-aldosterone system (RAAS). These changes result in structural alterations, including **mesangial expansion**, **glomerular basement membrane thickening**, and **podocyte injury**. Activation of pro-inflammatory cytokines and growth factors accelerates tissue damage and promotes **tubulointerstitial fibrosis**.[2]

As the disease progresses, there is

- Progressive nephron loss
- Glomerulosclerosis
- Decline in glomerular filtration rate (GFR)

These pathological changes ultimately lead to Chronic Kidney Disease and, in severe cases, end-stage renal disease.



Pathophysiology of CKD in patients with T2DM

Staging of CKD

CKD is classified based on estimated glomerular filtration rate (eGFR) and albuminuria levels, both of which are critical in diabetic nephropathy.

Stage	eGFR (mL/min/1.73 m²)	Description
G1	≥90	Normal function with kidney damage
G2	60–89	Mild decrease
G3a	45–59	Mild to moderate decrease
G3b	30–44	Moderate to severe decrease
G4	15–29	Severe decrease
G5	<15	Kidney failure (ESRD)

MANAGEMENT OF TYPE 2 DIABETES MELLITUS IN CKD

The management of T2DM in patients with Chronic kidney disease requires to be individualized and stage specific, by assessing their renal function, the risk of hypoglycemia, comorbidities and drug safety profiles.[12]

STAGE-WISE MANAGEMENT OF TYPE 2 DIABETES MELLITUS IN CKD

CKD STAGE	eGFR (mL/min/1.73 m²)	STAGE DESCRIPTION	RECOMMENDED ANTIDIABETIC DRUGS	DRUGS TO USE CAUTIOUSLY	DRUGS TO AVOID / CONTRAINDICATED
G1–G2 (Early CKD)	≥60	Normal to mild decrease in kidney function	• Metformin (first-line) • SGLT2 inhibitors (for renoprotection) • DPP-4 inhibitors • GLP-1 receptor agonists • Lifestyle modification	• None specific at this stage	• Not usually required to avoid
G3a (Mild to Moderate CKD)	45–59	Mild to moderate decrease in kidney function	• Metformin (continue with dose reduction if eGFR ≥30) • SGLT2 inhibitors (if tolerated) • DPP-4 inhibitors (dose-adjusted) • GLP-1 receptor agonists • Insulin (if required)	• Sulfonylureas (risk of hypoglycemia) • Pioglitazone (risk of fluid retention)	• Glyburide • High-dose metformin (if eGFR <45)
G3b (Moderate to Severe CKD)	30–44	Moderate to severe decrease in kidney function	• Metformin (use only if eGFR ≥30, in reduced dose) • DPP-4 inhibitors (dose-adjusted) • GLP-1 receptor agonists • Insulin (preferred if glycemic control not achieved)	• Sulfonylureas (risk of severe hypoglycemia) • SGLT2 inhibitors (limited efficacy)	• Glyburide • High-dose metformin • Acarbose
G4–G5 (Severe CKD / ESRD)	<30	Severe decrease in kidney function or kidney failure	• Insulin (mainstay of treatment) • DPP-4 inhibitors (linagliptin preferred) • GLP-1 receptor agonists (if not contraindicated)	• Most oral antidiabetic drugs	• Metformin • Most sulfonylureas • SGLT2 inhibitors (ineffective at very low eGFR)

GENERAL PRINCIPLES

- Individualize glycemic targets
- Avoid hypoglycemia
- Adjust drug dose based on eGFR
- Monitor renal function regularly
- Manage comorbidities (HTN, Dyslipidemia)
- Lifestyle modification and patient education

Stage wise maagement of T2DM in CKD patients

Dose adjustment and careful selection of drug becomes necessary as renal impairment can significantly alter drug pharmacokinetics and pharmacodynamics.

GENERAL PRINCIPLES OF MANAGEMENT

- Individualized glycemic targets (avoid strict control in advanced CKD)
- Prevention of hypoglycemia
- Use of renally safe drugs
- Dose adjustment based on eGFR
- Management of comorbidities (hypertension, dyslipidemia)

STAGE-WISE MANAGEMENT

Stages G1–G2 (eGFR ≥60 mL/min/1.73 m²)

Mild Renal Impairment

Most anti diabetic drugs can be utilized safely at this stage.

Preferred drugs

- **Metformin** (first-line therapy)
- **SGLT2 inhibitors** (early use for renoprotection)
- DPP-4 inhibitors
- **GLP-1 receptor agonists**

Rationale

Metformin is used as the first line agent as it can improve insulin sensitivity and has a favourable safety profile in patients with mild renal impairment. SGLT2 Inhibitors are also recommended as they can reduce intraglomerular pressure and slow the progression of chronic kidney disease.[4]

Stage G3 (eGFR 30–59 mL/min/1.73 m²)

Moderate renal impairment

As kidney f unction worsens, the drug selection process becomes more cautious.

Recommended approach

- **Continue metformin with dose reduction** (if eGFR ≥30)
- **Add SGLT2 inhibitors** (if tolerated)
- Use DPP-4 inhibitors (dose-adjusted)
- **Consider GLP-1 receptor agonists**

Drugs to use cautiously

- Sulfonylureas (risk of hypoglycemia)

Rationale

As renal function declines, the risk of drug accumulation and toxicity increases significantly, hence the drug selection process must be carried out with utmost caution.

Stages G4–G5 (eGFR <30 mL/min/1.73 m²)

Severe renal impairment / ESRD

At this stage, many oral antidiabetic drugs are contraindicated.

Preferred therapy

- Insulin (mainstay of treatment)
- DPP-4 inhibitors (select agents like linagliptin)

Contraindicated / avoided

- Metformin (risk of lactic acidosis)
- Most sulfonylureas
- Certain SGLT2 inhibitors (limited efficacy in low eGFR)

Rationale

Reduced renal clearance significantly increases the risk of drug toxicity. Insulin is preferred due to its predictable efficacy, although dose reduction is required due to decreased renal degradation.

CLINICAL CHALLENGES

- Increased risk of hypoglycemia
- Drug accumulation due to reduced clearance
- Poor adherence due to complex regimens
- Cost limitations in newer therapies

DRUG UTILIZATION PATTERN OF ANTIDIABETIC MEDICATIONS IN CKD

The drug utilization pattern of anti diabetic medications in patients with Type 2 Diabetes Mellitus and Chronic kidney disease demonstrates a stage dependent prescribing pattern which is influenced by safety, efficacy and clinical evidence. Several studies have consistently shown that prescribing practices are progressively shifting toward more individualized and evidence-based therapy.[8]

DRUG UTILIZATION PATTERN OF ANTIDIABETIC MEDICATIONS IN CKD

ANTIDIABETIC MEDICATION CLASS	MECHANISM OF ACTION	UTILIZATION PATTERN IN CKD	ADVANTAGES / BENEFITS	LIMITATIONS / CAUTIONS IN CKD
1. METFORMIN (Biguanide)	↓ Hepatic glucose production, ↑ insulin sensitivity, ↑ peripheral glucose uptake	• First-line drug in early CKD (G1–G2) • Use declines as CKD progresses • Avoided in advanced stages (G4–G5)	• Effective • Safe in early CKD • Low cost • Weight neutral	• Risk of lactic acidosis in advanced CKD • Accumulates with reduced eGFR • Discontinue if eGFR <30 mL/min/1.73m²
2. SGLT2 INHIBITORS (e.g., Dapagliflozin, Empagliflozin)	Inhibit glucose reabsorption in proximal tubule (SGLT2 inhibition) → ↑ glucosuria	• Increasing use across CKD stages (G1–G3) • Limited efficacy in G4–G5 but still used for renoprotection in selected cases	• Renoprotection • Cardioprotection • Low risk of hypoglycemia • Weight & BP reduction	• Reduced efficacy in low eGFR (<30) • Risk of dehydration, UTI, genital infections • Cost is a major barrier
3. DPP-4 INHIBITORS (e.g., Sitagliptin, Vildagliptin, Linagliptin)	Inhibit DPP-4 enzyme → ↑ incretin levels (GLP-1, GIP) → ↑ insulin secretion, ↓ glucagon secretion	• Utilization increasing in all CKD stages • Preferred in moderate to severe CKD due to safety • Dose adjustment required (except Linagliptin)	• Low risk of hypoglycemia • Well tolerated • Safe in CKD with dose adjustment	• Dose adjustment needed (except Linagliptin) • Cost
4. GLP-1 RECEPTOR AGONISTS (e.g., Liraglutide, Dulaglutide)	Mimic GLP-1 action → ↑ insulin secretion, ↓ glucagon secretion, ↓ appetite, slows gastric emptying	• Increasing trend in CKD • Used as add-on therapy • Preferred for patients with CVD & obesity	• Weight loss • Cardiovascular benefit • Low risk of hypoglycemia • Possible renoprotection	• GI side effects (nausea, vomiting) • Cost • Limited data in advanced CKD
5. INSULIN (Basal / Bolus)	Replaces or supplements endogenous insulin → ↑ glucose uptake in tissues	• Utilization increases as CKD progresses (G3b–G5) • Mainstay treatment in severe CKD & ESRD • Flexible dosing	• Most effective in advanced CKD • No renal excretion • Adjustable dosing	• High risk of hypoglycemia (due to ↓ renal insulin degradation) • Requires careful dose titration
OVERALL TREND: Shift from conventional agents (Metformin, Sulfonylureas) to newer, safer agents (SGLT2 inhibitors, DPP-4 inhibitors, GLP-1 RAs) and as CKD progresses, with emphasis on renal & cardiovascular protection and minimizing hypoglycemia.				

Drug Utilization pattern of Anti Diabetic medications in CKD

Metformin Utilization

Metformin is the first line therapy in the early stages of CKD due to it's efficacy, safety profile and affordability. As CKD progresses, the utilization of metformin drops due to higher risk of lactic acidosis and drug accumulation. KDIGO suggest that careful use or stopping the medication in later stages of CKD. Decrease in metformin usage shows that healthcare providers are adhering to the standard guidelines.[8]

Sulfonylureas

Sulfonylureas use is limited due to increased risk of hypoglycemia, although they are being used in some settings. Gliclazide and Glipizide are considered to be safer alternatives due to their relatively lower risk profiles.

DPP-4 Inhibitors

Across multiple studies, an increase utllization of dipeptidyl peptidase-4 (DPP-4) inhibitors was observed. These agents are favoured possibly due to their low risk of hypoglycemia and minimal adverse effects. Dose adjustment is still required for these agents in CKD, with a sole exception of linagliptin, as it is primarily excreted via liver.[8]

SGLT2 Inhibitors

Soidum-glucose co transporter-2 inhibitors have gained importance as they contribute to glycemic control as well as renal protection.[14] They promote renal protection by reducing intraglomerular pressure and slower CKD progression, while also preventing cardiovascular events.[2]

Impact of Economic Constraints

Cost related barriers especially in low and middle income families, limit the utilization of the new anti diabetic agents, resulting in reduced accessibility and suboptimal adherence, highlighting a gap between guideline based recommendations and real word practice.[6]

Polypharmacy in CKD Patients

With an average of 5-8 drugs per prescription, polypharmacy is a significant finding in CKD patients.[7] This is largely due to the presence of multiple comorbidities such as hypertension, dyslipidemia and cardiovascular diseases. As the number of drugs increase, so does the risk of interactions, ADRs, and poor medication adherence, which are worsened by altered pharmacokinetics in CKD.[4]

Irrational Prescribing Practices

Irrational prescribing practices such as inappropriate drug selection, lack of dose adjustment, etc. contribute to a major persisting problem.[10]

Drug-Related Problems

Problems such as therapeutic duplication, inappropriate dosing and drug interactions are frequently reported in patients with CKD.[7] Such drug related problems highlight the importance of prescription review, monitoring and pharmacist intervention in optimizing therapy.

Overall Pattern

The current drug utilization pattern reflects a transition from conventional anti diabetic agents to newer, safer alternatives, as the treatment procedures are adhering to the standard guidelines and becoming more individualized which results in better therapeutic outcome. However, economic burden, polypharmacy and irrational prescribing acts as the limiting opposition to optimal therapeutic outcomes.

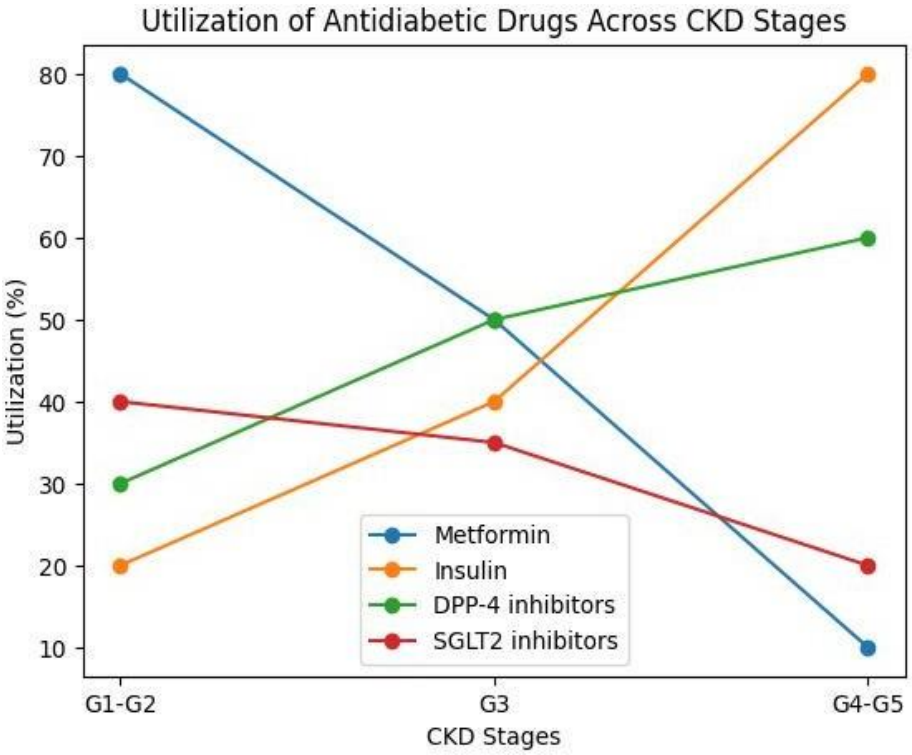
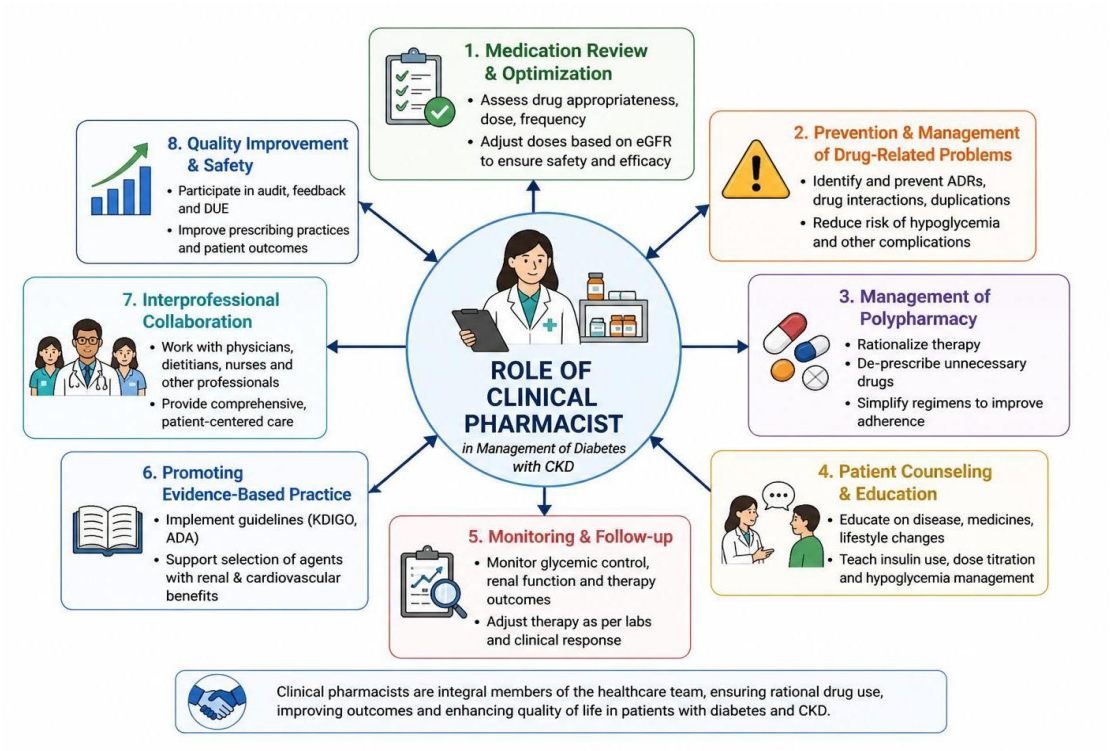


Chart highlighting the utilization of Anti Diabetic Drugs across CKD stages

ROLE OF CLINICAL PHARMACIST IN MANAGEMENT OF DIABETES WITH CKD

Clinical pharmacists play a crucial role in optimizing pharmacotherapy, as the management of patients with T2Dm and CKD is complicated and requires a multidisciplinary approach.

Clinical pharmacists can improve patient outcomes and minimize drug related problems. In CKD, due to altered pharmacokinetics and pharmacodynamics, the role of clinical pharmacists become more essential to ensure the safety and efficiacy of anti diabetic medications.



Role of Clinical Pharmacist in Management of T2Dm in CKD patients

Medication Review and Optimization

Reviewing medication profile of patients to ensure that the selected drug is given at an appropriate dose is one of the main roles of a clinical pharmacist. In CKD patients, dose adjustment is particularly important, as dose calculation is based on eGFR, which is essential to prevent drug accumulation and toxicity. Review of medication chart help to identify inappropriate medications as well as optimize the therapy based on the individual, leading to rational use and desired therapeutic outcome.[15]

Prevention and Management of Drug-Related Problems

Patients with Chornic Kidney disease are at a higher risk of drug related problems such as ADRs, interactions and dosing errors. Clinical pharmacists play a crucial role in identifying such problems, and are able to prevent such problems by continuous monitoring and intervention. Hence the incidence of hypoglycemia and other complications are significantly reduced.[15]

Management of Polypharmacy

By conducting medication chart review, a clinical pharmacist is able to identify therapeutic duplication as well as unnecessary medications, hence rationalizing therapy, simplifying regimens and ensuring compatibility between drugs. This significantly reduces polypharmacy

as well as increasing patient adherence towards medications and reduces the risk of ADRs.[15]

Patient Counseling and Education

One of the main roles of a clinical pharmcist is providing patient counseling so as to improve the patient's knowledge regarding disease management, medicaiton adherence and lifestyle modifications. Counseling on proper insulin usage, and hypoglycemia management is important in patients with CKD. This improves overall therapeutic outcomes.[15]

Monitoring and Follow-Up

Clnical pharmacists play a crucial role by monitoring blood glucose, renal function and drug therapeutic outcomes in CKD patients, as they can interpret laboratory data and adjust the therapy accordingly. They also play a role in ensuring adherence to standard treatment guidelines.[15]

Promoting Evidence-Based Practice

Clinical pharmacists play a crucial role in the implementation of clinical guidelines such as KDIGO recommendations, by supporting healthcare providers in selecting anti diabetic agents most suited for each patient, promoting newer and safer drugs with renal benefits, while considering the economic burden and patient specific factors.[15]

Role in Healthcare System Improvement

At a broader level, clinical pharmacists improve prescribing pattern through feedback and participation in drug utilization studies. Their involvement assists in identifying gaps in therapy, rationalizing therapy, reducing irrational prescribing and contribute to an increase in overall quality of care and therapy provided.

CONCLUSION

The reivew hightlights the patterns in the utilization of anti diabetic medications in patients witth Type 2 Diabetes Mellitus and Chronic Kidney Disease. The findings indicate that the prescribing practices are influenced by CKD stages. In early stages of CKD, metformin is the commonly prescribed drug which is gradually transitioned in to insulin and other safer alternatives in later stages of CKD.

The use of newer agents such as DPP-4 inhibitors and SGLT 2 inhibitors highlights the shift towards evidence based and outcome based therapy. However, economic burden acts as a barrier leading to limited accessibility of these newer agents, commonly seen in developing

countries.

The high prevalence of polypharmacy, and irrational prescribing practices highlght the need for improved drug selection, dose adjustment, decision making and adherence to standard guidelines.

Overall, optimizing antidiabetic therapy in CKD requires a patient-centered approach that considers renal function, comorbidities, drug safety, and economic factors. Regular prescription review, dose individualization, and the involvement of clinical pharmacists are essential to minimize adverse drug reactions and improve therapeutic outcomes. Future research should focus on large-scale, multicentric studies to generate more robust evidence and support the development of standardized prescribing protocols for this population.

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