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TO ASSESS THE EVALUATION OF ANTIHYPERLIPIDEMIC DRUG PRESCRIBING PATTERNS IN CHRONIC KIDNEY DISEASE PATIENTS

Anila N.*, Soumya R. V.[2], Dr. Nithin Manohor[3], B. Aswinipriya[1], Atmaj[1], Roshin Robert[1], Dr. Prasobh[4]

[1]Student Fifth Year Doctor of Pharmacy Student, Sree Krishna College of Pharmacy and Research Centre, Parassala, Thiruvananthapuram, Kerala, India.
[2]Associate Professor, Department of Pharmacy Practice, Sree Krishna College of Pharmacy and Research Centre, Parassala, Thiruvananthapuram, Kerala, India.
[3]Professor and HOD, Department of Pharmacy Practice, Sree Krishna College of Pharmacy and Research Centre, Parassala, Thiruvananthapuram, Kerala, India.
[4]Principal, Sree Krishna College of Pharmacy and Research Centre, Parassala, Thiruvananthapuram, Kerala, India.

***Corresponding Author: Anila N.**

Student Fifth Year Doctor of Pharmacy Student, Sree Krishna College of Pharmacy and Research Centre, Parassala, Thiruvananthapuram, Kerala, India.

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ABSTRACT

Chronic kidney disease (CKD) is associated with significant alterations in lipid metabolism, leading to an increased risk of cardiovascular morbidity and mortality. Dyslipidemia in CKD patients is characterized by elevated triglycerides, reduced high-density lipoprotein (HDL), and variable low-density lipoprotein (LDL) levels. Appropriate use of antihyperlipidemic drugs plays a critical role in reducing cardiovascular risk in this population. This study aims to evaluate the prescribing patterns of antihyperlipidemic drugs in CKD patients, focusing on drug selection, dosage adjustments, and adherence to clinical guidelines. Commonly prescribed agents include statins, fibrates, ezetimibe, and combination therapies. The study also assesses the pharmacological mechanisms, safety profiles, and potential adverse effects of these drugs in CKD patients. Findings suggest that statins remain the cornerstone of therapy, though dose adjustments and careful monitoring are essential due to altered pharmacokinetics in CKD. Rational prescribing practices can significantly improve patient outcomes while minimizing drug related complications.[2]

KEYWORDS: Chronic kidney disease, dyslipidaemia, lipid abnormalities, Rational drug therapy, Anti-Hyperlipidemic drugs.

INTRODUCTION

Chronic kidney disease is a progressive condition characterized by a gradual loss of renal function over time. It is a major public health concern worldwide due to its association with high cardiovascular risk. Cardiovascular disease (CVD) is the leading cause of death in CKD patients, and dyslipidaemia is one of the major contributing factors.

Lipid abnormalities in CKD differ from those in the general population and require specific therapeutic approaches. Management of dyslipidaemia using antihyperlipidemic drugs is essential to reduce atherosclerotic complications. However, CKD alters drug metabolism and excretion, making prescribing decisions more complex.

Evaluating prescribing patterns helps ensure rational drug use, adherence to guidelines, andimproved clinical outcomes.[3]

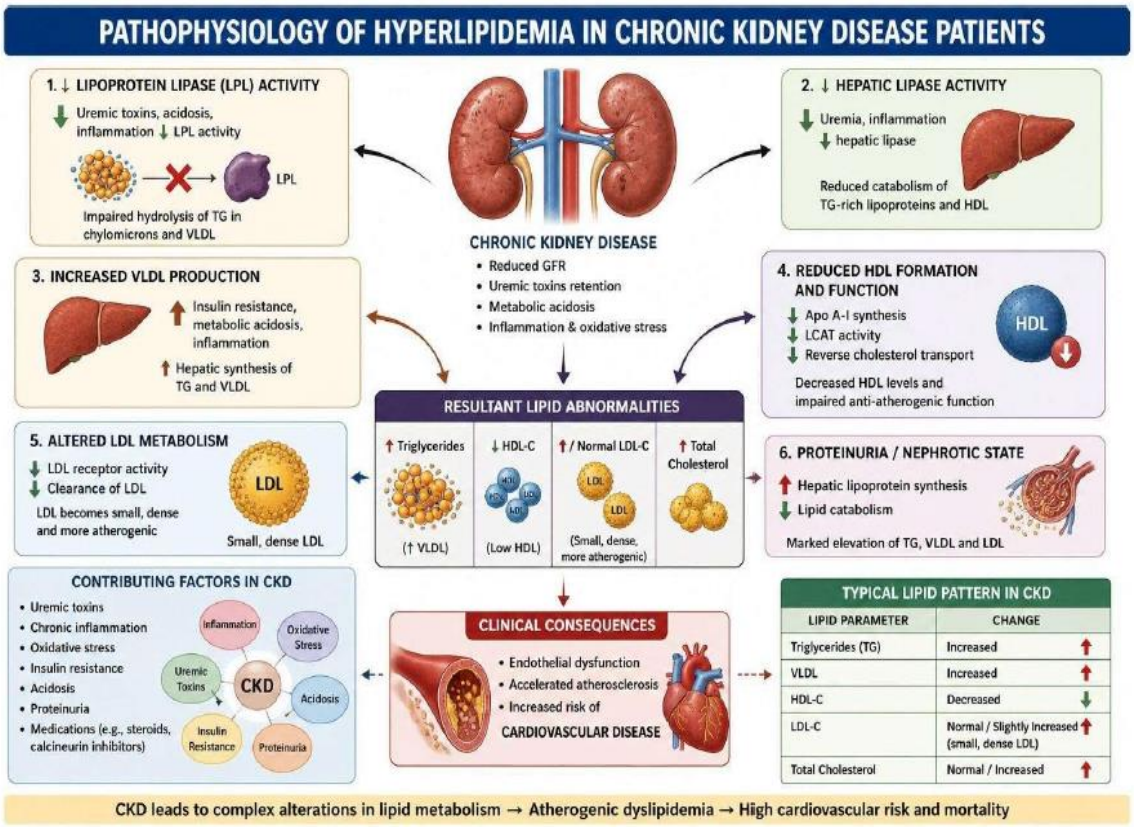
Hyperlipidaemia in Chronic Kidney Disease Patients

Hyperlipidaemia in CKD is primarily characterized by:

- Elevated triglycerides
- Reduced HDL cholesterol
- Increased very low-density lipoprotein (VLDL)
- Normal or mildly elevated LDL

Pathophysiology

- Reduced activity of lipoprotein lipase
- Impaired clearance of triglyceride-rich lipoproteins
- Increased oxidative stress and inflammation
- Proteinuria leading to altered lipid metabolism[8]



These abnormalities contribute significantly to accelerated atherosclerosis and cardiovascular comp chronic kidney disease (CKD) is a medical condition characterized by lasting structural or functional abnormalities in the kidneys, persisting for a duration of over three months and significantly impacting an individual's health. The classification of CKD is based on various factors, including the underlying cause, glomerular filtration rate (GFR), and the category of albuminuria. The presence of CKD in patients with established CVD is associated with a substantially higher mortality rate compared to those with CVD but normal kidney function. In addition to other cardiovascular risk factors, the decline in GFR itself is strongly associated with an increased risk of cardiovascular disease (CVD) among adults. Both vascular disease and structural heart disease are more prevalent in individuals with chronic kidney disease. It is crucial to recognize the heightened risk of cardiovascular complications in individuals with CKD. By understanding this association, healthcare professionals can implement appropriate preventive measures and treatment strategies to mitigate the impact of CVD in patients with CKD, Consequently, patients with CKD, particularly those in stage 3 CKD, or at a more advanced stage (stage 4-5 CKD) requiring dialysis, are considered to be at a high or very-high risk of developing CVD. The evidence of use of statin in these patients has been explored in many trials.

1. Lipid abnormalities in chronic kidney disease

In patients with chronic kidney disease, it is commonly observed that triglyceride levels are elevated, while levels of high-density lipoprotein cholesterol are decreased, additionally there is an increase in the concentration of small dense low-density lipoprotein particles. Furthermore, kidney dysfunction leads increased levels of LP(a) due to impaired catabolism of lipoprotein(a) (resulting in elevated (a) levels. These lipid abnormalities along with other factors are the important reason of increased cardio-vascular morbidity and mortality. However, these acquired lipid abnormalities can be reversed through kidney transplantation or the remission of nephrosis.

2. Management of lipid abnormalities in CKD patients

The management of lipid abnormalities in CKD patients involves a multifaceted approach aimed at reducing the risk of cardiovascular morbidity and mortality. Lifestyle modifications by adopting a healthy diet which is low in saturated and Trans fats. This is also to be complemented by engaging in regular physical activity, and smoking cessation. Pharmacological interventions are often necessary to achieve lipid control along with the life style changes in CKD patients.

The use of statins, which inhibit cholesterol synthesis and promote LDL. Receptor expression is the cornerstone of lipid-lowering therapy. The use of statins in managing the dyslipidaemia in CKD patients has been extensively studied and investigated in trials. It is important to consider the specific statin dosage adjustments which may be required in patients with CKD due to altered drug metabolism and potential drug-drug interactions. Other lipidlowering agents, such as fibrates or ezetimibe, may also be considered as adjunctive therapy in certain cases, although their use should be individualized based on patient characteristics and potential side effect.

Evidence from trials suggests despite the high CV risk among haemodialysis patients the statin therapy may not offer significant benefits. Several early trials, including the Die Deutsche Diabetes Dialyse Studies (the 4D study) and the An Assessment of Survival and Cardiovascular Events (AURORA) trial found no benefit in haemodialysis patients. The studies done later such as the Study of Heart and Renal Protection (SHARP) study, simvastatin and ezetimibe combination therapy reduced the risk of major atherosclerotic events in people with CKD stage 3A-5,

Statin therapy consistently provided beneficial effects in a retro-spective cohort study of adult patients with end stage renal disease (ESRD) who were on maintenance haemodialysis, these beneficial effects were more pronounced in patients who received statin on a continuous basis throughout the study period and also patients who received statin/ezetimibe combination therapy. After adjusting for age, gender, and Charlson Comorbidity Index (CCI), the benefits of statin therapy remained significant. Sensitivity analysis revealed that statin therapy provided consistent benefits across various subgroups, including older (aged 75 years) and younger (aged 40 years) patients.

Statin therapy is clearly effective in mild-to-moderate CKD, but a major question that remained after the publication of the 4D, AURORA, and SHARP studies was whether statin therapy is effective in more. Advanced CKD, particularly in dialysis patients.

The Cholesterol Treatment Trial lists (CPT) Collaboration investigators by combining data from the three CKD trials with other trials in the existing database, discovered a trend toward smaller relative reductions in major atherosclerotic events per mmol/l redaction in LDL-C as eGFR declines (with little evidence of benefit among dialysis patients). This decrease in relative risk reduction as GFR decreases implies that more intensive LDLlowering regimens are required to achieve the same benefit, at least in non-dialysis patients.

3. Initial lipid assessment and follow-up measurements

For all adults recently diagnosed with chronic kidney disease (CKD), it is advisable to conduct initial evaluation of lipid profile. This should encompass measurements of total cholesterol, LDL-C, HDL-C, and tri glycerides. The primary goal of this assessment is not only to assist in determining when to start statin treatment for CKD patients under the age of 50 but also to identify potential underlying causes of abnormal lipid levels, such as nephrotic syndrome, and to detect hyper-triglyceridemia, a common lipid issue in CKD patients. In patients have fasting triglyceride levels exceeding 1000 mg/dl. or LDL-C levels passing 190 mg/dl, it is recommended to involve specialists.

4. Safety of lipid lowering drugs in patients with chronic kidney disease

Safety issues and dose adjustments are important in advanced stages of CKD (stages 35), as adverse events are commonly dose-related and due to increased accumulation in blood.

Pharmacological Treatment of Antihyperlipidemic Drugs in CKD Patients

Treatment strategies include:

1. Statins (HMG-CoA Reductase Inhibitors)

- First-line therapy
- Reduce LDL cholesterol and cardiovascular risk
- Examples: Atorvastatin, Rosuvastatin, Simvastatin

2. Fibrates

- Primarily reduce triglycerides
- Used cautiously in CKD due to renal excretion

Examples: Fenofibrate, Gemfibrozil

3. Ezetimibe

- Inhibits intestinal cholesterol absorption
- Often used in combination with statins

4. PCSK9 Inhibitors

• Used in high-risk patients
• Reduce LDL significantly

5. Omega-3 Fatty Acids

 Lower triglyceride levels[3]

Dose adjustment is crucial in CKD to avoid toxicity.

1. Statins

Mechanism of Action

Statins competitively inhibit HMG-CoA reductase, the rate-limiting enzyme in cholesterol biosynthesis. This reduces hepatic cholesterol production, leading to increased LDL receptor expression on liver cells. As a result, more LDL cholesterol is removed from the bloodstream.

Uses

* Hypercholesterolemia
* Mixed dyslipidemia

* Prevention of myocardial infarction
* Prevention of stroke
* Atherosclerotic cardiovascular disease

Dose
* Atorvastatin: 10–80 mg orally once daily
* Rosuvastatin: 5–40 mg orally once daily

Adverse Effects
* Myalgia
* Myopathy
* Rhabdomyolysis
* Elevated liver enzymes
* Gastrointestinal disturbances

2. Fibrates
Mechanism of Action
Fibrates activate Peroxisome Proliferator-Activated Receptor Alpha (PPAR-α). This increases lipoprotein lipase activity, enhancing triglyceride breakdown and reducing VLDL levels.

Uses
* Hypertriglyceridemia
* Mixed dyslipidemia
* Prevention of pancreatitis due to severe triglyceride elevation

Dose
* Fenofibrate: 48–145 mg/day
* Gemfibrozil: 600 mg twice daily

Adverse Effects
* Dyspepsia
* Gallstones
* Myopathy
* Hepatotoxicity

3. Bile Acid Sequestrants
Mechanism of Action
These drugs bind bile acids in the intestine and prevent their reabsorption. The liver utilizes more cholesterol to synthesize new bile acids, thereby lowering plasma LDL levels.

Uses
* Primary hypercholesterolemia
* Familial hypercholesterolemia
* Adjunct therapy with statins

Dose
* Cholestyramine: 4–24 g/day
* Colesevelam: 3.75 g/day

Adverse Effects
* Constipation
* Bloating
* Nausea
* Malabsorption of fat-soluble vitamins

4. Ezetimibe
Mechanism of Action
Ezetimibe inhibits the NPC1L1 transporter in the intestinal brush border, reducing absorption of dietary and biliary cholesterol.

Uses
* Hypercholesterolemia
* Familial hypercholesterolemia
* Combination therapy with statins

Dose
* 10 mg orally once daily

Adverse Effects
* Diarrhea
* Headache
* Abdominal pain
* Myalgia

5. PCSK9 Inhibitors
Mechanism of Action
PCSK9 inhibitors prevent degradation of LDL receptors in the liver. Increased LDL receptor availability enhances removal of LDL cholesterol from blood.

Uses
* Familial hypercholesterolemia
* Severe hypercholesterolemia
* Patients not responding adequately to statins

Dose
* Alirocumab: 75–150 mg subcutaneously every 2 weeks
* Evolocumab: 140 mg subcutaneously every 2 weeks

Adverse Effects
* Injection-site reactions
* Nasopharyngitis
* Influenza-like symptoms

6. Niacin (Nicotinic Acid)
Mechanism of Action
Niacin inhibits lipolysis in adipose tissue, reducing free fatty acid delivery to the liver.
This decreases VLDL synthesis and increases HDL cholesterol.

Uses
* Mixed dyslipidemia
* Hypertriglyceridemia
* Low HDL cholesterol

Dose
* 1–2 g/day orally

Adverse Effects
* Flushing
* Pruritus
* Hyperglycaemia
* Hyperuricemia

* Hepatotoxicity

7. Omega-3 Fatty Acids
Mechanism of Action
Omega-3 fatty acids reduce hepatic triglyceride synthesis and decrease VLDL production.

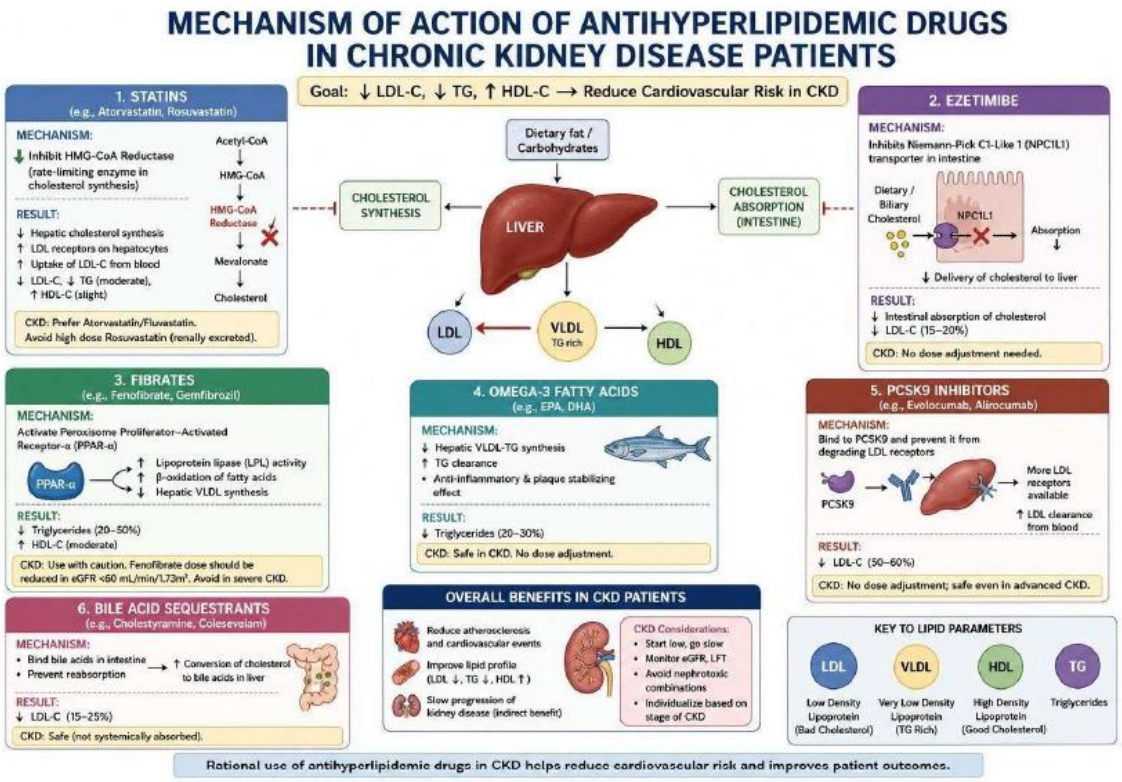
Uses
* Severe hypertriglyceridemia

Dose
* 2–4 g/day orally

Adverse Effects
* Fishy taste
* Dyspepsia
* Diarrhea
* Increased bleeding tendency at high doses

Possible Side Effects
- **Statins:** Myopathy, hepatotoxicity, rhabdomyolysis (risk ↑ in CKD)
- **Fibrates:** Renal dysfunction, gallstones, muscle toxicity
- **Ezetimibe:** Mild GI disturbances
- **PCSK9 inhibitors:** Injection site reactions
- **Omega-3:** Fishy aftertaste, mild GI upset (5)

Close monitoring is essential in CKD patients.



DISCUSSION
Prescribing patterns of antihyperlipidemic drugs in CKD patients reveal a preference for statins due to their proven cardiovascular benefits. However, inappropriate dosing and lack of monitoring may increase the risk of adverse effects.

Combination therapy (e.g., statin + ezetimibe) is often used for better lipid control. Fibrates are used cautiously due to safety concerns. Clinical guidelines recommend individualized therapy based on CKD stage, lipid profile, and comorbidities.

There is a need for improved awareness among clinicians regarding dose adjustments and guideline-based prescribing.[9]

CONCLUSION
Antihyperlipidemic therapy plays a crucial role in reducing cardiovascular risk in CKD patients. Statins are the most commonly prescribed drugs, followed by combination therapies.
Rational prescribing, appropriate dose adjustments, and regular monitoring are essential to ensure safety and efficacy.
Further studies are needed to evaluate long-term outcomes and optimize treatment strategies in CKD patients.

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