Journal of Clinical and Nursing Research, 2026, Volume 10, Issue 4
http://ojs.bbwpublisher.com/index.php/JCNR
Online ISSN: 2208-3693
Print ISSN: 2208-3685

Dyslipidemia in Kidney Transplant Recipients: Implications for Graft Prognosis and Lipid Management

Jingru Chen, Jianghua Chen*

The First Affiliated Hospital, Zhejiang University School of Medicine, Hangzhou 310000, Zhejiang, China

****Author to whom correspondence should be addressed.***

Abstract: Dyslipidemia is a common metabolic complication after kidney transplantation, affecting 75-95% of recipients. Increasing evidence links post-transplant lipid abnormalities to graft dysfunction and eventual graft loss. However, studies on conventional lipid markers have reported inconsistent associations with transplant outcomes. Emerging lipid indicators may better reflect the burden of atherogenic lipoproteins and provide additional value for risk stratification after transplantation. This review summarizes current evidence on the epidemiology, mechanisms, graft outcome associations, and management of dyslipidemia in kidney transplant recipients, with a focus on postoperative monitoring and prognostic assessment.

Keywords: Kidney transplantation; Dyslipidemia; Graft outcomes; Lipid management

Online publication: Apr 30, 2026

1. Introduction

Kidney transplantation is the preferred treatment for end-stage renal disease, offering better survival and quality of life than dialysis [1]. Although advances in surgical techniques and immunosuppressive therapy have improved outcomes [2], long-term graft survival remains a major clinical challenge. Identifying modifiable risk factors for graft loss, particularly those linked to metabolic injury, is a clinical priority.

Dyslipidemia is a common post-transplant complication [3], largely driven by immunosuppressive therapy [4]. Dyslipidemia has been associated with graft dysfunction and late graft loss [5,6], but conventional lipid indices show inconsistent associations with graft outcomes [7]. This inconsistency has prompted interest in emerging markers that better reflect atherogenic burden, including remnant cholesterol (RC), non-high-density lipoprotein cholesterol (non-HDL-C), and apolipoproteins [8]. This review examines the epidemiology, mechanisms, graft outcome associations, and management of dyslipidemia in kidney transplant recipients.

2. Dyslipidemia and graft injury

Dyslipidemia affects 75–95% of kidney transplant recipients after transplantation [3]. The typical pattern includes increased total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), with reduced high-density lipoprotein cholesterol (HDL-C) [4]. Qualitative abnormalities (e.g., small dense LDL, dysfunctional HDL) are also described but not captured by routine testing.

In addition to diet, obesity, and genetics, immunosuppressive therapy plays a major role: glucocorticoids induce TG and reduce HDL-C by inhibiting lipoprotein lipase activity; calcineurin inhibitors increase lipid levels by interfering with cholesterol metabolism and lipoprotein clearance pathways; mammalian target of rapamycin inhibitors elevate TC and TG possibly through inhibition of lipoprotein lipase activity and promotion of lipolysis [8,9]. Post-transplant weight gain, insulin resistance, and new-onset diabetes after transplantation may further aggravate post-transplant dyslipidemia [5,9].

Dyslipidemia affects the allograft via interconnected pathways: oxidative stress/inflammation, metabolic toxicity, and vascular injury. A cholesterol-rich environment promotes renal inflammation via macrophage cytokine release, and oxidized LDL further triggers inflammation, impairs vascular repair, and accelerates microvascular rarefaction and graft dysfunction [10]. Dyslipidemia may also disrupt insulin signaling via excess free fatty acids and lipotoxicity, worsening glomerular hyperfiltration and proteinuria [11]. Additionally, hypercholesterolemia contributes to intimal thickening and graft vascular sclerosis [10].

3. Conventional lipids and graft prognosis

Post-transplant hypercholesterolemia often coexists with impaired kidney function, reflected by lower estimated glomerular filtration rate (eGFR) [12]. Recipients with higher TC levels had lower 5-year and 10-year graft survival rates [13]. TC > 5.2 mmol/L was associated with more severe interstitial fibrosis in the graft [14].

Elevated LDL-C predicts adverse outcomes. A machine learning model identified pre-transplant LDL-C as a predictor of renal dysfunction [15]. Maintaining LDL-C < 2.6 mmol/L is an protective factor against early graft failure (graft loss within 5 years) [16], and early statin therapy is associated with lower graft loss risk [17].

The role of HDL-C is less clear, as studies show inconsistent associations with graft prognosis; this suggests that HDL particle functionality, rather than absolute concentration, may be more clinically relevant [15].

TG elevation has been linked to graft dysfunction and lower graft survival [18]. Metabolic abnormalities (≥ 2 of: hypertension, hyperglycemia, high TG, low HDL-C) increase graft dysfunction risk within 2 years [18]. TG levels have also been reported to correlate inversely with eGFR.

Overall, conventional parameters capture only part of the risk, and their mixed findings suggest they do not fully reflect lipid-related injury [7,19].

4. Emerging markers and graft prognosis

RC is derived mainly from triglyceride-rich lipoprotein remnants. In the FAVORIT trial, higher RC levels were associated with all-cause mortality, with a trend toward higher graft failure risk [20]. RC has also been associated with the urinary albumin-to-creatinine ratio, suggesting renal injury [21].

Non-HDL-C, a clinically useful marker of atherogenic burden, has shown prognostic value for post-transplant renal dysfunction [15]. In stable graft function recipients, non-HDL-C ≥ 4.2 mmol/L was a sig-

nificant risk factor for reduced eGFR [22].

Apolipoprotein B (ApoB) has been associated with late graft failure [6]. Apolipoprotein A1 (ApoA1) is considered anti-atherogenic [19]. Increased ApoB, decreased ApoA1, and a higher ApoB/ApoA1 ratio are associated with chronic kidney disease progression and post-transplant complications [19].

The triglyceride-glucose (TyG) index, a surrogate marker of insulin resistance, is associated with graft outcomes. The highest TyG quartile was associated with a 2.13-fold higher risk of death-censored graft loss and 1.46- and 1.78-fold higher risks of eGFR decline (≥ 30% and ≥ 50%, respectively) [23].

These markers better reflect atherogenic burden and insulin resistance, offering enhanced risk stratification, especially when conventional profiles are only mildly abnormal.

5. Lipid management

Lipid management should be part of long-term metabolic care, not an isolated effort to normalize values. The high prevalence of dyslipidemia and its implications for graft outcomes support regular monitoring [24]. Diet, exercise, and weight loss remain foundational: healthy dietary patterns lower TC and LDL-C; structured physical activity increases HDL-C and improves creatinine clearance; weight loss improves lipid-related abnormalities [25,26].

Statins are first-line. Meta-analyses show that statin therapy reduces all-cause mortality in kidney transplant recipients [17]. KDIGO guideline recommends statin therapy for recipients aged ≥ 30 years or those with cardiovascular risk factors [24]. For statin intolerance or inadequate response, proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors are an alternative, reducing LDL-C without significant immunosuppressive interactions [27]. Adding ezetimibe to statin therapy may further reduce TC, whereas fibrates remain most effective for TG lowering [24].

Management needs to be individualized [24]. In patients with persistent dyslipidemia, emerging markers may help refine risk assessment but are not yet used as routine treatment targets. If lipid control remains suboptimal, modification of the immunosuppressive regimen may be considered, while carefully balancing the risk of rejection [5]. However, prospective studies are needed before emerging markers can guide routine treatment.

6. Conclusion

Dyslipidemia is highly prevalent after kidney transplantation and is associated with graft injury. Conventional lipid parameters remain clinically useful but have important limitations. Emerging markers may provide a more detailed view of risk related to graft outcomes. Current gaps include non-standardized measurement timing, heterogeneous cutoff values, and a lack of intervention trials targeting these emerging markers. Addressing these requires large multicenter prospective studies.

Disclosure statement

The authors declare no conflict of interest.

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