STATIN THERAPY AFTER HEART TRANSPLANTATION

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Introduction. Cardiovascular disease remains the leading cause of long-term mortality after heart transplantation, largely driven by cardiac allograft vasculopathy (CAV) and accelerated atherosclerosis. Dyslipidemia develops frequently in this population as a consequence of immunosuppressive therapy and metabolic alterations. Glucocorticoids, calcineurin inhibitors (especially cyclosporine), and mTOR inhibitors significantly disrupt lipid metabolism, promoting elevations in total cholesterol, LDL-cholesterol (LDL-C), and triglycerides. Given this high-risk profile, lipid management is a central component of post-transplant care. The aim of this review is to synthesize recent evidence on the mechanisms, clinical implications, and management of dyslipidemia after heart transplantation.
Materials and Methods: A narrative literature search was performed in the Hinari database, limited to publications from the last 5 years. Search keywords included: statins after heart transplantation, cardiac transplant dyslipidemia, statin-immunosuppressant interactions, statin safety in transplant. Emphasis was placed on mechanisms of post-transplant hyperlipidemia, efficacy of statin therapy, comparative safety profiles, and clinically relevant drug-drug interactions between statins and immunosuppressive agents.
Results: Post-transplant dyslipidemia is multifactorial. Corticosteroids increase hepatic lipoprotein synthesis and insulin resistance, while cyclosporine impairs LDL receptor activity, leading to hypercholesterolemia. mTOR inhibitors (sirolimus, everolimus) elevate triglycerides, whereas tacrolimus shows a more favorable lipid profile. According to the 2019 ESC/EAS guidelines, transplant recipients are managed according to overall cardiovascular risk. Early initiation of statins after heart transplantation improves survival and reduces the incidence and progression of cardiac allograft vasculopathy (CAV), independent of baseline LDL-C levels. Statins are first-line therapy and also provide pleiotropic endothelial and anti-inflammatory effects that may protect the graft. However, significant pharmacokinetic interactions occur with CYP3A4-metabolized statins when combined with cyclosporine or mTOR inhibitors, increasing myopathy risk. Therefore, pravastatin and rosuvastatin are generally preferred, with careful dose titration and monitoring of liver enzymes and creatine kinase.
Conclusions: Dyslipidemia after heart transplantation requires proactive and individualized management. Statins remain the therapeutic cornerstone, providing both cardiovascular and graft-protective benefits. Careful drug selection and monitoring are essential to balance efficacy with safety in the context of complex immunosuppressive regimens.
Keywords: heart transplantation, statins, dyslipidemia, cardiac allograft vasculopathy, drug-drug interactions.