Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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Post KT Management Int the CKM Syndrome Era

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Kidney transplantation markedly improves survival and quality of life in patients with kidney failure, but it does not normalize cardiovascular–kidney–metabolic (CKM) risk. Kidney transplant recipients remain a unique high-risk population in whom residual chronic kidney disease, single allograft physiology, donor-related factors, rejection history, calcineurin inhibitor nephrotoxicity, and lifelong immunosuppression interact with traditional metabolic risk factors. Therefore, post-transplant care in the CKM syndrome era should move beyond graft-centered monitoring toward integrated cardiorenal-metabolic risk assessment. This framework is particularly relevant after kidney transplantation because metabolic risk is dynamically reshaped after transplantation. Some uremia-related abnormalities may improve, but new or persistent metabolic derangements frequently emerge, including weight gain, increased fat mass, relative sarcopenia, post-transplant diabetes mellitus, dyslipidemia, hypertension, and hyperuricemia. Recent studies suggest that newly developed metabolic syndrome after transplantation is associated with graft loss, whereas persistent metabolic syndrome is associated with cardiovascular events and mortality. These findings indicate that post-transplant metabolic changes are clinically meaningful phenotypes linked to both patient and allograft outcomes. Immunosuppressive therapy is a major modifier of CKM risk. Corticosteroids promote weight gain, insulin resistance, hyperglycemia, and dyslipidemia; tacrolimus is associated with β-cell dysfunction and post-transplant diabetes; cyclosporine is linked to hypertension, hyperuricemia, and dyslipidemia; and mTOR inhibitors may aggravate lipid abnormalities. Thus, CKM management cannot be separated from immunosuppressive strategy, although metabolic optimization must be balanced against alloimmune protection. In native CKD and diabetic kidney disease, treatment is rapidly evolving toward combination therapy with renin–angiotensin system blockade, SGLT2 inhibitors, GLP-1 receptor agonists, and

nonsteroidal mineralocorticoid receptor antagonists. However, kidney transplant recipients were largely excluded from pivotal trials, and transplant-specific guidance remains limited. Emerging data support the cautious use of SGLT2 inhibitors and GLP-1 receptor agonists in selected stable recipients, whereas finerenone remains investigational. In the CKM syndrome era, post-transplant management should be phenotype-based, carefully monitored, and tailored to transplant-specific risks.

Keywords: Kidney transplantation, Cardiovascular-kidney-metabolic syndrome, Post-transplant diabetes mellitus, SGLT2 inhibitors , GLP-1 receptor agonists