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Cyclophosphamide in Transplant Immunology: Mechanistic Precision, Immune Reprogramming, and Toxicity

Miss. Sakshi Vijay Khandare, Dr. Avinash S. Jiddewar, Asst. Pranit G. Kubare
Miss. Vaishnavi D. Dhokane, Miss. Shivam B. Waghamare
NSPM College of Pharmacy, Darwha, Yavatmal

Abstract: *Cyclophosphamide is an alkylating prodrug widely used as an immunosuppressant in oncology, autoimmunity and transplantation. Its immunomodulatory efficacy stems from hepatic bioactivation to DNA-alkylating metabolites that preferentially damage proliferating lymphoid populations, modulate T-cell subsets, and alter regulatory networks involved in allograft rejection. Recent work highlights dose- and timing-dependent effects: high-dose, peri-transplant cyclophosphamide depletes alloreactive T cells and prevents graft-versus-host disease (GVHD) in hematopoietic stem cell transplantation, while low-dose regimens exert selective effects on regulatory T cells and myeloid-derived suppressor cells with potential to reshape tolerance induction. Cyclophosphamide's therapeutic window is constrained by predictable toxicities — myelosuppression, hemorrhagic cystitis, gonadal injury, cardiotoxicity and infection risk — whose incidence depends on cumulative dose, metabolite exposure, and host pharmacogenetics (CYPs, POR). Balancing efficacy and toxicity requires regimen optimization (dose, schedule, mesna hydration), pharmacogenetic awareness, and combination strategies with targeted agents (calcineurin inhibitors, MMF, PTCy schedules). This review synthesizes mechanistic, pharmacologic, clinical-trial, and toxicity data to inform rational use of cyclophosphamide in organ transplantation*

Keywords: Cyclophosphamide, Immunosuppression, Post-transplant cyclophosphamide (PTCy), Organ transplantation, T-cell modulation, Toxicity, CYP2B6, Clinical trials, Tolerance induction

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