IMMUNOSUPPRESSION FOLLOWING LIVER TRANSPLANTATION

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Liver is considered to be an ‘immunoregulatory’ solid organ with specialised venous endothelial turnover, a high number of extramedullary haematopoietic stem cells and the ability to produce numerous immunoregulatory substances. The privileged state of liver in transplantation is highlighted by the relatively lower need for human leukocyte antigen (HLA) or blood-group matching. Compared with other organs, such as kidneys and lungs, the liver allograft has the advantage of demonstrating lower rates of acute and chronic rejection, a resistance to antibody-mediated rejection and a higher /uni25CF /uni25CF /uni25CF /uni25CF Harvey Williams Cushing , 1869–1939, Professor of Surgery , Harvard University Medical School, Boston, MA, USA. agents such as antithymocyte globulin (ATG), CD25 mono - clonal antibodies (basiliximab and daclizumab) or cluster of di ff erentiation (CD)52 monoclonal antibodies (alemtuzumab; Campath-1H), w hich routinely form part of kidney , pancreas and other organ transplants, are rarely used in L T . They are only considered in selected patients with a high immunolog - ical risk or renal compromise, in the latter case to delay the introduction of calcineurin inhibitors (CNIs). CNIs (tacrolimus and ciclosporin) are the mainstay of LT maintenance immuno - suppression, with mycophenolate mofetil (MMF), azathioprine and corticosteroids consider ed as the essential adjuncts to CNIs (Table 89.3). Mammalian target of rapamycin inhibitors (mTORi) such as sirolimus and everolim us have established roles in patients with worsening renal function and in those with LT for HCCs and incidental cancers on explant. The side e ff ects of prolonged immunosuppression are one of the limitations of long-term survival among LT recipients, espe - cially those due to immunosuppression-induced metabolic syndrome, cardiovascular disease, renal impairment and malignancy . Several studies have shown tha t 20% of LT patients can achieve operational tolerance, whereby there is long-term survival of the allograft in the absence of immunosuppression. However, there is a need for more research to understand this better and to identify the group of patients who will benefit from withdrawal of immunosuppression.

TABLE 89.3 Common immunosuppression medications after liver transplant. Drug Mechanism of action Calcineurin inhibitors Inhibit T-cell signalling, prevent (tacrolimus, ciclosporin) lymphocyte activation and block cytokine transcription Mycophenolate mofetil Inhibits T-cell and B-cell proliferation Azathioprine Purine analogue, impedes DNA and RNA synthesis Corticosteroids Decrease cytokine production Decrease lymphocyte activation and proliferation Decrease antibody production Decrease phagocytosis and release of proteolytic enzymes mTORi (sirolimus/everolimus) Mammalian target of rapamycin inhibitor

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