Infectious complications

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Infection is the leading cause of death in multivisceral/ intestinal transplant recipients. This is as a consequence of the degree of immunosuppression and also the potential for bacterial translocation across the graft mucosa. Opportunistic infections, common to all solid organ transplant recipients, such as cytomegalovirus (CMV), Pneumocystis jirovecii (PJP), adenovirus, Epstein–Barr virus (EBV) and fungal infections, are all more common following intestinal transplantation. Appro - priate prophylaxis with antiviral (valganciclovir or aciclovir), antifungal (fluconazole) and anti-PJP (co-trimoxazole) agents is critical. Infective enteritis is common among transplant recipients and can mimic rejection, thus it is important to send stool for culture and viral polymerase chain reaction when a patient presents with a high-output stoma or diar rhoea. Rejection trig - gered by infection is possible and a high index of suspicion and early r epeat endoscopy are important if clinical improvement does not occur. PTLD is a potential complication of immunosuppression for any patient after transplant. The incidence in intestinal trans plant recipients is higher than for other solid organ transplant recipients – up to 17% in some series. This is likely to be due to both the level of immunosuppression and the amount of lymphoid tissue associated with an intestinal-containing graft. PTLD should be considered if there is a persistent positive EBV viraemia or B symptoms such as night sw eats, unexplained fevers and weight loss. If diagnosed with PTLD the first-line treatment is usually with rituximab and immunosuppression reduction. This has to be done with great caution in multi visceral/intestinal transplant recipients because of the risk of rejection and the consequences thereof. The extent of lymphoid tissue associated with the multi visceral/intestinal graft increases the risk of GVHD. Outcomes from GVHD in this patient group can be very poor. The most common pr esenting symptoms of GVHD are a rash, fever and bone marrow suppression. Optimal management of GVHD is unclear and proposed strategies include both enhancement and also reduction of immunosuppression. Management is guided by the level of peripheral T-cell chimerism. There is an increasing trend towards withdrawing immunosuppression ini tially to rebalance the equilibrium between the host and donor immune systems. Bone marrow involvement and ultimately failure is almost universally fatal. Deterioration in renal function is common to all solid org transplant groups but is most marked following multivisceral/ intestinal transplantation. The cause of this is multifactorial b ut is likely to include contributions from the physiological insult of surgery , the use of nephrotoxic medication (e.g. tac rolimus) and fluid losses associated with the stoma. It is imper ative to maintain good hydration postoperatively and home intravenous fluids may be needed initially post discharge. Immunosuppression modifications including con version from calcineurin inhibitor-based protocols to mTOR inhibitors may prevent further deterioration of renal function. These inter ventions need to be undertaken with caution as they may pre cipitate an episode of acute cellular rejection.

Revision #1
Created 2025-12-31 15:32:26 UTC by Omar Ayman
Updated 2025-12-31 15:32:26 UTC by Omar Ayman