Immunosuppression and rejection

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The rate of acute cellular rejection for intestinal grafts is higher than for any other abdominal organ. This is reflected in the magnitude of immunosuppression required. As with other solid organ transplants acute cellular rejection is most common in the first few months after transplantation, although it can occur at any time. Michael Anthony Epstein , b. 1921, Professor of Pathology , University of Bristol, Bristol, UK. Yvonne Barr , 1931–2016, virologist who emigrated to Australia. Epstein and Barr discovered this virus in 1964. regimens commonly utilise an induction agent, usually a lymphocyte-depleting antibody such as antithymocyte glob - ulin (ATG) or alemtuzumab. Paediatric regimens commonly use non-depleting antibody induction. Maintenance imm uno - suppression comprises a calcineurin inhibitor (most commonly tacrolimus), an antimetabolite (mycophenolate or azathioprine) and prednisolone. In addition, some programmes supplement this with early addition of an mTOR (mammalian target of rapamycin) inhibitor (sirolim us or everolimus). Acute cellular rejection is usually initially manifested in the terminal ileum. Rarely other organs (liver/pancreas) may be a ff ected without intestinal rejection. Rejection most commonly presents with increased stoma output or diarrhoea, sometimes with an associated fever. Sev ere (exfoliative) acute cellular rejection with loss of intestinal mucosa results in rapid bacterial translocation and sepsis . In any intestinal transplant recipient presenting with sepsis or septic shock, a diagnosis of rejection needs to be considered. Endoscopic assessment and biopsy of the graft are required for the diagnosis and assessment of the severity of acute cel - lular rejection. Graft surveillance in a protocolised manner is performed in some programmes; others respond to symptoms suggestive of rejection (i.e. an incr ease in stoma output). At endoscopy , rejection can range from mild, with some erythema of the mucosa, to moderate, with some ulceration, to severe, with confluent loss of mucosa. At biopsy , salient fea - - tures of rejection are an inflammatory infiltrate in the lamina propria, crypt loss and ulceration and an increase in apoptotic bodies in the base of the crypts. Rejection is treated primarily with high-dose intravenous pulsed steroids. If the rejection is steroid resistant then fur - ther lymphocyte depletion may be needed with either ATG or alemtuzumab. - -

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