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Lymphocyte recognition of CMV-infected endothelial cells

Rejection processes after organ transplantation are associated with upregulation of HLA class I and induction of HLA class II expression on the endothelium of the donated graft. Furthermore, it is suggested that human cytomegalovirus (CMV) infection is involved in rejection processes but the mode of interaction is still unclear. Infection of cultured human umbilical vein endothelial cells (HUVE) with CMV results in slightly enhanced expression of HLA class I antigens in vitro while no induction of HLA class II is observed. Further, in vitro studies with HUVE and cocultured peripheral blood lymphocytes (PBL) have revealed evidence for lymphocyte recognition of infected syngeneic but not allogeneic HUVE that is associated with enhanced expression of HLA class I antigens. Lymphocytes were primed against CMV antigens. One aspect of my stay in Pittsburgh for 2 months was to learn about the presentation of soluble CMV antigens to lymphocytes. CMV proteins can be synthesized and isolated utilizing the maltose-binding fusion protein system. These proteins reflect the immediate early, early, and late phases of CMV replication and are available in large amounts. In Pittsburgh, lymphocytes from bronchoalveolar lavages from lung-transplanted patients during CMV infection were cultured and challenged with soluble CMV antigens. Proliferation of these cells was accelerated as compared with adequate controls. The Pittsburgh group has recently found a higher incidence of CMV hepatitis in liver-transplanted patients when HLA-DR was matched, indicating recognition of CMV-infected cells by class II restricted T cells. It is endeavoured now to test the ability of cultured endothelial cells to present these soluble CMV antigens to syngeneic or allogeneic lymphocytes. In the allogeneic model, it is, therefore, important to determine the HLA type of the utilized cells by means of a polymerase chain reaction. Besides proliferation assays of the challenged lymphocytes, the modulation of the HLA class I and II antigen expression on cocultured endothelial cells will be quantified by means of flow cytometry. These Studies might elucidate the involvement of CMV infection in immunological mechanisms leading to graft rejection.

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Clinical monitoring of kidney transplants

I attended a training course on "clinical monitoring in kidney transplantation" at the Nephrology Department of the University of Basel (director prof. G. Thiel) between February and April 1993.

The main purpose of this course was to learn the basic criteria for working out an individual approach to immunosuppression in patients of low, average and high immunological risk, and their clinical monitoring, with special attention to renal biopsies. The training course consisted of clinical monitoring in the early posttransplant period, observation and treatment of patients after their discharge from hospital, work with the biopsy material and theoretical work. During this period, 22 kidney transplantations, including 8 from living-related donors were performed in the department. Every month, 100–150 patients attend the out-patient department. Choice of immunosuppressive protocol was based upon such parameters as recipient immunological risk, tolerance of cyclosporin A, clinical situation, condition after transplantation and development of posttransplant complications. The most interesting clinical problem was diagnosis of cyclosporin A toxicity, such as, for example, haemolytic-uraemic syndrome (HUS), which can lead to a fulminant graft loss, and the morphological differentiation between HUS and vascular rejection. Attending the Institute of Pathology (director prof, M. Mihatsch) provided an excellent opportunity to compare clinical findings with the morphological picture, and, in some cases, to analyse retrospectively the correlation between morphological and clinical data.