

to obtain an adequate biopsy sample in a patient with severe heart failure. The procedure might be justified in borderline cases, if it could be relied upon to avoid the performance of a hopeless transplant operation with the attendant loss of a scarce donor heart.

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Commentary

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Thoracic allograft transplantation has offered the respiratory physician new challenges in diagnosis and treatment and the three case reports presented here bear witness to this.

The paper by Egan *et al* describes two patients who developed necrotic bronchitis as a presenting feature of lymphoproliferative disease following heart-lung transplantation. This condition arises via Epstein-Barr virus driven cell transformation and usually presents with malaise, fever, and nodules on the chest radiograph. An early diagnosis is imperative since the prognosis is much better when the disease is limited in extent. The incidence has increased since the widespread use of cyclosporin as the principal immunosuppressant leading to a reduced efficacy of T cell surveillance of B lymphocytes infected with Epstein-Barr virus. The disease commonly presents in the transplanted organ leading to speculation that it is clonal proliferation of donor B lymphocytes which results in lymphoproliferative disease.

The importance of this paper is that it supports the idea that donor bronchus associated lymphoid tissue provided the environment for clonal proliferation of transformed B cells. It is also of particular interest for those involved in the postoperative management of lung transplant recipients, serving to remind us that an abnormal inflamed airway following transplantation does not necessarily imply vascular injury with necrosis or primary aspergillus infection. It underlines the value of performing a biopsy on available tissue when faced with a clinical problem of unknown cause.

The paper by Sheerin *et al* describes another rare case of airway disease associated with transplantation but in this case provided the indication for single lung transplantation. Obliterative bronchiolitis is a pathological entity which is a recognised sequel of various insults and conditions including viruses, toxic gas, rheumatoid arthritis, and following bone marrow or lung transplantation. It is often diagnosed after other more common diseases of airflow obstruction have been excluded. We

have transplanted four patients with obliterative bronchiolitis, one following bone marrow transplantation, one associated with rheumatoid arthritis, and two of unknown aetiology. Histological examination of resected native lungs in both the latter cases confirmed the diagnosis but did not shed light on the aetiology. It is of interest, therefore, that in this case the proposed mechanism was identified by histological examination of the lung removed at transplantation. The diagnosis could have been made preoperatively if a lung biopsy had been carried out, and raises questions as to whether lung biopsy should have been considered in this patient at some stage during her 10 year history of progressive airflow obstruction. It is only by application of modern techniques of immuno and molecular pathology to lung tissue that the mechanisms behind obliterative bronchiolitis of unknown aetiology will be determined, hopefully leading to new approaches in therapy. It is interesting to speculate whether treatment with somatostatin or its analogue octreotide would have had any effect on tumour size or degree of obstruction in this case.

The necessity of full and careful investigation of patients undergoing any form of transplantation is borne out by the paper by Hasleton and Brooks which describes two patients who died of right heart failure after cardiac transplantation. Deaths were due to pulmonary veno-occlusive disease leading to a high pulmonary vascular resistance. It was interesting to note the morphological changes in pulmonary veins associated with the raised left atrial pressure. Many transplant groups would have been very wary of transplanting patients with a pulmonary vascular resistance of 4 or more units and transpulmonary gradients of 15 and 19 mm Hg respectively, particularly with no attempts to reduce this pharmacologically. Certainly it confirms the much greater mortality associated with heart transplantation in patients with raised pulmonary vascular resistance of whatever cause.