symptoms and remained positive for 10 months. IgM fell to low concentrations but was still positive up to 10 months after disease onset. The initial haemoglobin was 10·6 g/dL, white cell count $4·2 \times 10^9$/L, erythrocyte sedimentation rate 56 mm per h, and antinuclear antibody titre was 40 with a speckled pattern. Double-stranded DNA binding, rheumatoid factor, extractible nuclear antigen, complement profiles, and antibody to chlamydia, yersinia, salmonella, borrelia, rubella, campylobacter, Epstein-Barr virus, and Coxsackie virus showed no recent contact.

An initial diagnosis of parvovirus arthritis was made but because the symptoms were uncontrolled on flurbiprofen 50 mg three times a day, hydroxychloroquine 400 mg and prednisone 10 mg daily were added. The hand and feet radiographs, although initially normal, at 8 months showed definite cortical erosions of several small joints (figure). Therefore, sodium aurothiomalate 20 mg per week was started in place of hydroxychloroquine with gradual clinical improvement.

Our case provides a well-documented description of a definite parvovirus infection followed by an erosive polyarthritis studied early (less than 3 months) into the disease. It highlights the need for accurate diagnosis of recent parvovirus B19 infection which may give falsely low positive IgM with some tests. The association between parvovirus B19 and some forms of rheumatoid arthritis deserves further study, especially early in the process.

A Tyndall, W Jelk, H H Hirsch

Department of Rheumatology, University of Basel, 4055 Basel, Switzerland; and Institute for Medical Microbiology, University of Basel

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Non-Hodgkin lymphoma in heart/lung transplant recipients

SIR—Opelz and Henderson (Dec 18/25 p 1514) emphasise the importance of immunosuppression in the development of non-Hodgkin lymphoma (NHL) by comparing renal and heart transplant recipients in North America and Europe. Although immunosuppression is important, its role in the aetiology of NHL after transplantation might have been over-emphasised. Variations in HLA matching of donor and recipient might contribute to the difference in incidence of NHL in the populations studied. Prospective HLA matching is done in renal transplantation in Europe, but it is not standard in North American renal transplant centres[1] and is not done in heart transplantation. The absence of prospective HLA matching could increase immunosuppression requirements, thereby contributing to incidence of NHL in North American renal transplant recipients and heart transplant recipients world wide.

Additional factors might be important, as suggested by studies of heart/lung recipients who also do not undergo prospective HLA matching. At our centre, 3 of 166 (1·8%) heart transplant recipients and 2 of 26 (7·6%) heart/lung transplant recipients have developed post-transplant lymphoproliferative disease (PTLD). 4 of the 5 subjects had high-grade B-cell NHL. This finding is similar to experience in North America, where in one large series 3·4% of heart transplant recipients and 7·9% of heart/lung transplant recipients have developed PTLD.[2] In our centre, both heart and heart/lung transplant recipients receive rabbit antithymocyte globulin (RATG) 2 mg/kg on three consecutive days after transplant, but heart transplant recipients additionally receive further RATG for persistent grade 3a rejection. Thus heart/lung transplant recipients seem more liable to develop PTLD despite equivalent or less immunosuppression than in heart transplant recipients.

In both our heart/lung transplant patients the initial clinical sign of PTLD was an ulcerative bronchitis,[3] implicating bronchus-associated lymphoid tissue (BALT) in the transplanted lung as the important initial site for uncontrolled B-cell proliferation. It is recognised that BALT is important in the histogenesis of pulmonary lymphomas,[4] and that the presence of BALT itself is dependent on antigenic stimulation.[5] The higher incidence of PTLD in heart/lung than in heart transplant recipients might result from transplanting substantial aggregates of BALT in which antigenic stimulation promoted by HLA mismatching results in uncontrolled B-cell proliferation.

Another important co-factor for the development of NHL is latent Epstein-Barr virus (EBV) infection. We have studied the expression of Epstein-Barr nuclear antigen 2 (EBNA 2) and latent membrane protein (LMP), both of which are associated with B-cell transformation. With anti-EBNA2 monoclonal antibody PE2 and anti-LMP monoclonal antibody pool CS1-4 (Dakopatts, Copenhagen)[6] both EBNA 2 and LMP were shown in nests of transformed B cells in the paraffin blocks of four of five patients.

Opelz and Henderson assert that the difference in immunosuppression underlies the different incidence of NHL seen in North America and Europe. In heart-lung transplant recipients we believe that chronic antigenic stimulation resulting from the absence of prospective HLA matching and latent EBV infection in transplanted BALT are critical factors. We suggest that these factors may be as important as baseline immunosuppression in accounting for the disparity between incidence of NHL in heart and renal transplant recipients and between North American and European renal transplant recipients.

Jim Egan, James Stewart, Nizar Yonan, John Arrand, Ashley Woodcock

North West Lung Centre, and Department of Cardiothoracic Surgery, Wythenshawe Hospital, Manchester M23 9LT, UK; and Department of Molecular Biology, Paterson Institute for Cancer Research, Christie CRC Cancer Centre, Manchester

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SIR—Opelz and Henderson report a high incidence of NHL in kidney and heart transplant recipients. For this complication, they found positive correlations to aggressiveness of immunosuppression and age of the recipient.

These results accord with data from our group,[1] showing that 13 of 28 heart and 5 of 28 liver transplant patients had transient, changing, or stable monoclonal immunoglobulins