females. The mean age of onset of psoriasis in our cohort was 29.27 yr (s.d. 14.16 yr). Overall, 98% of the samples were genotyped successfully, and all of the genotypes for the controls satisfied the Hardy–Weinberg equilibrium.

With respect to single locus associations, none of the variants examined from SLC22A4, SLC22A5, SLC9A3R1 and RUNX1 were significantly associated with PsA in the Newfoundland population either by genotype or minor allele frequencies (Table 1).

We then analysed two marker haplotypes for markers of interest in rheumatoid arthritis [SLC22A4 (rs3792876) and RUNX1 (rs2268277)] and Crohn’s disease [SLC22A4 (rs1050152) and SLC22A5 (rs2631367)] and noted no association (P = 0.342 and P = 0.81, respectively). We also examined two marker combinations for the remaining SNPs in SLC22A4 (rs3792876 and rs1050152, P = 0.48; rs3792876 and rs3763112, P = 0.580) as well as haplotypes for all four SNPs in SLC22A4 on chromosome 5 (P = 0.90). Finally we assessed the relationship of all six markers on three different chromosomes and again found no association (P = 0.74).

Thus in our study, we noted no association between the organic cation transporter genes and PsA in the Newfoundland population. This is in contrast to a recent British study that investigated 471 Caucasian PsA patients and 605 population controls for similar variants in SLC22A4 and SLC22A5 [4]. They noted two SNPs, rs3763112 mapping to SLC22A4 and rs2631367 mapping to SLC22A5, to be significantly associated with PsA (P = 0.001 and P = 0.007, respectively). Furthermore, the same haplotype as Crohn’s disease between SNPs (rs1050152 and rs2631367) was strongly associated with PsA (P = 0.0002) in their cohort. Hence, PsA in the Newfoundland population either by genotype or minor allele frequencies.

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therapy (intravenous methylprednisolone, 1 g/day for 3 days). He was transferred to our department because of his worsening clinical condition and laboratory tests (Fig. 1A). Gastrointestinal bleeding associated with disseminated intravascular coagulation (DIC) and severe jaundice were observed. T-Bil and soluble interleukin-2 receptor (sIL-2R) concentrations were extremely high at 23.5 mg/dl and 13 500 U/ml, respectively. In February 1998, the patient was treated with intravenous CyA and plasma exchange therapy, achieving a plasma CyA concentration of 100 ng/ml. Three weeks later oral CyA therapy (225 mg/day) was started instead of intravenous CyA infusion because of almost complete recovery. Before steroid therapy, a liver biopsy showed periportal steatosis with infiltration of neutrophils, lymphocytes and macrophages. Note also the lack of bile ducts (haematoxylin–eosin stain, ×100). (C) High-power photomicrographs of liver histopathological findings 40 days after commencement of CyA therapy. Note the reduction in hepatic steatosis and infiltrated lymphoid cells and histiocytic cells, and the significant increase in the number of bile ducts (arrows) in comparison with panel B (haematoxylin–eosin stain, ×400). Parts (B) and (C) of this figure may be viewed in colour as supplementary data at Rheumatology Online.
improvement, CyA and corticosteroid treatment was discontinued in 1999. He has been symptom-free and has maintained a normal life style for more than 5 yr without CyA and steroids.

WCD is a primary panniculitis, which is a histiocytic disorder described as a form of lobular panniculitis with infiltration of haemophagocytic benign histiocytes [1]. In our patient, the diagnosis of WCD was made based on the clinical features, histopathological findings of the skin and liver biopsy specimen. His WCD was steroid-resistant and he developed several complications, including DIC and severe jaundice, after steroid pulse therapy. However, CyA was remarkably effective and the clinical condition showed almost complete recovery except for mild fatty liver. Successful treatment of WCD with CyA was first described in 1987 [5] and several additional reports described a similar response to CyA [6–9]. The aetiology of WCD remains unknown. However, it has been related to an immunologically mediated reaction because of sIL-2R elevation [8, 9]. In the present case, we also observed high serum concentration of sIL-2R, which represents T-cell activation, and successful response to CyA, which acts primarily on helper T cells and interferes with the production of various cytokines. CyA therapy resulted in a dramatic decrease in the serum concentration of sIL-2R followed by rapid clinical improvement.

In addition, a striking feature of this case was the hepatic involvement. Liver biopsy before steroid pulse therapy showed perportal steatohepatitis with biliary ductopenia. Ductopenia is a rare cause of prolonged, progressive cholestatic liver disease [10]. It is mainly associated with chronic allograft rejection, graft-versus-host disease, primary biliary cirrhosis, drugs and toxins. Ductopenia associated with WCD is rare and our report is the first to describe ductopenia as one of the pathological features of WCD and to show that CyA is significantly effective in ductopenia associated with steroid-resistant WCD.

In conclusion, we report a case of WCD with biliary ductopenia successfully treated with CyA. Ductopenia should be included as a possible pathophysiological factor of hepatic lesion in WCD.

The authors have declared no conflicts of interest.

M. HINATA, T. SOMEYA, H. YOSHIZAKI, K. SEKI, K. TAKEUCHI

Department of Gastroenterology and ¹Department of Pathology, Toranomon Hospital, Tokyo, Japan
Accepted 14 January 2005

Correspondence to: M. Hinata, Department of Gastroenterology, Toranomon Hospital, 2-2-2 Toranomon, Minato-ku, Tokyo 105-0001, Japan. E-mail: mhinata@hotmail.com