referred from GP's and general paediatricians requesting us to investigate the suspected condition of coeliac disease. Coeliac disease has a broad clinical spectrum, and, as Bell and Mackovitch state, antigliadin, antireticulin, and antienterodial antibody tests are not perfect, although they are used as screening tests in epidemiological surveys. Therefore, on an individual patient basis, we choose to perform a small intestinal biopsy as the most efficient means of investigation. Clearly there is a difference between children who have a permanent disorder and those with a temporary condition such as cows' milk sensitive enteropathy. In centres where small intestinal biopsy is not available a therapeutic trial of milk elimination may be in order, but this will lead to the over diagnosis of the condition and overuse of milk substitutes.

It was certainly our aim to add to the body of knowledge on chronic diarrhoeal disease in childhood, and trust that other centres, both national and international, would view our experience in the light of the context of their own work situation.

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Positive hepatitis serologies with treatment for Kawasaki syndrome

Sir,—Intravenous immune globulin (IVIG) is currently the treatment of choice for Kawasaki syndrome in that it shortens the duration of symptoms and lowers the incidence of coronary artery aneurysms associated with this disorder. We describe a 3 year old boy who had hepatomegaly and raised hepatic transaminases associated with Kawasaki syndrome and transiently developed false positive serologies to hepatitis B and C after treatment with IVIG.

Case report

Seven days before hospitalisation, this 3 year old boy developed fever, abdominal pain, and vomiting. During the next five days he developed a generalised maculopapular skin eruption sparing the palms and soles, diffuse tender cervical adenopathy, oedema of the hands and feet, non-purulent conjunctivitis, right upper quadrant abdominal tenderness with mild hepatomegaly, and a systolic ejection murmur. Serum alanine aminotransferase was 133 IU/l (normal 6–65), serum aspartate aminotransferase was 191 IU/l (normal 7–40), and serum lactate dehydrogenase was 775 IU/l (normal 232–619). Prothrombin time, alkaline phosphatase, and bilirubin values were normal, as was an abdominal sonogram. Past and family history were negative; he had received no transfusions nor been exposed to infectious hepatitis. A diagnosis of Kawasaki syndrome was made and IVIG administered. The child's symptoms resolved within 48 hours but serum transaminases remained raised. On the third hospital day, alanine aminotransferase was 164 IU/l and aspartate aminotransferase was 191 IU/l. Alkaline phosphatase and bilirubin values remained normal. Serum was obtained for measurement of hepatitis B and C virus antibodies and for hepatitis C virus RNA detection (HCV Ab ELISA, Ortho Diagnostics), as were antibodies to core and surface antigens of hepatitis B virus. Clinically the child did well and was discharged from the hospital at the conclusion of IVIG treatment. In the year since hospital discharge he has remained asymptomatic and his serum transaminases have remained normal. Three months after discharge, antibodies to hepatitis C virus and core and surface antigens of hepatitis B virus were detectable. However, at six, nine, and 12 months after discharge, all antibody studies were negative.

The history and serological findings in this case suggest hepatitis B and C viruses were passively transferred by infusion of IVIG. The disappearance of detectable antibody to both viruses six months after the infusion is consistent with the 30 day half life of IVIG.3 Passive transfer of antibody to hepatitis B virus has been described after infusions of IVIG.4 Similarly, passive transfer of antibody to human immunodeficiency virus has been described after infusions of hepatitis B virus and core and surface antigens of hepatitis B virus were detectable. However, at six, nine, and 12 months after discharge, all antibody studies were negative.

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Recurrent parotitis

Sir,—Cohen et al report 11 children with recurrent parotitis.1 They state that immunological factors may be involved in the pathogenesis of this condition, although in general the immunological abnormalities described have been fairly subtle.2,3 We report a recent case which supports the theory that autoimmune mechanisms are important.

We have recently seen a 11 year old girl who presented with a 12 month history of four episodes of right sided parotid swelling, without红ness or tenderness, but associated with low grade fever. There was a history of recurrent, unexplained fevers from infancy until 7 years of age. She also gave a history of dry, sore eyes since infancy, recently diagnosed by an ophthalmologist as being allergic conjunctivitis. She had a chronic non-productive cough for four years. She had recurrent ear infections from 5 years of age, for which adenotonsillectomy was performed and ventilating tubes inserted, and had a chronic non-productive sialaictis since age 7 years.

Sialogram showed right sided sialaictis with a normal left parotid duct. Chest radiograph was normal. Full blood count was normal, with no eosinophilia. Serum Immunoglobulin concentrations that were performed on two occasions showed serum IgG 3:2 and 2:4 g/l (normal 6:5–15:0), serum IgA 0:07 and 0:09 g/l (normal 0:5–3:0), and serum IgM 0:13 and 0:23 g/l (normal 0:9–3:0). HIV antibodies and anti-HTLV-1, anti-HIV, and C3d were negative. T cell counts were normal but B cells were raised (CD19 27%, normal 1–15%), a diagnosis of common variable immunodeficiency (late onset hypogammaglobulinaemia) was made, and the patient was started on intravenous immunoglobulin replacement treatment.

It is well recognised that autoimmune phenomena may occur in common variable immunodeficiency, including recurrent parotitis. Conley et al described parotitis in two of eight children with common variable immunodeficiency, although it did not state whether or not it was recurrent.4 One of the two patients described by Fries et al had IgA deficiency, gluten enteropathy and high titres of antinuclear antibodies, all features of evolving common variable immunodeficiency.5 As long ago as 1960, Mosbech and Kristensen felt that autoimmunity might play a part in the pathogenesis of recurrent parotitis.6 We believe the case we report here supports that theory. Our case shows the importance of measuring serum immunoglobulin concentrations on children with recurrent parotitis.

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1 10.1136/adc.68.1.151-a on 1 January 1993. Downloaded from http://adc.bmj.com/ on September 14, 2023 by guest.