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 hepatitis 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 A, B, and C antibodies to hepatitis C virus, and serum samples were sent to 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 that hepatitis B and C viruses were passively transfused 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.2 Similarly, passive transfer of antibody to hepatitis B virus has been described after infusions of IVIG.2 3 Passively transferred antibody to hepatitis B virus has been described after infusions of IVIG.2 3 Passive transfer of antibody to hepatitis B virus may also be due to the use of unconcentrated IVIG preparations.3

The increases in serum transaminases in this case were likely to have been due to Kawasaki disease. Increases in serum transaminases and bilirubin values occur frequently in Kawasaki disease and are due to microscopically to cholangitis, periductitis, vasculitis, and portal perivasculitis.6

IVIG has become an important therapeutic agent with ever increasing uses. This patient demonstrates that infusion of IVIG may induce false positive serological and immunohaematological tests, and therefore the results of such studies must be interpreted with caution.

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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 an 11 year old girl who presented with a 12 month history of four episodes of right sided parotid swelling, without redness or tenderness, but associated with low grade fever. There was a history of recurrent, unexplained fever 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 but denied recurrent ear infections from 5 years of age, for which adenotonsillectomy was performed and ventilating tubes inserted, and had a chronic non-productive cough since age 7 years.

Sialogram showed right sided sialectasis with a normal left parotid duct. Chest radiography 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 0.5–5), and serum IgM 0.13 and 0.23 g/l (normal 0.3–2). HIV antibodies were negative. T cell subsets 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 recognized 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 did not state whether or not it was recurrent.5 One of the two patients described by Fris et al had IgA deficiency, gluten enteropathy and high titres of antinuclear antibodies, all features of evolving common variable immunodeficiency.6 As long ago as 1960, Mosbech and Kristensen felt that autoimmune phenomena might play a part in the pathogenesis of recurrent parotitis.5 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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