LETTERS TO
THE EDITOR

Prevalence of antidelta in Turkish children with chronic hepatitis B infection

Delta hepatitis is caused by a dual infection with the hepatitis delta virus (HDV) and hepatitis B virus (HBV). HDV depends on the helper function of hepatitis B surface antigen (HBsAg) for its replication. Therefore, epidemiology of delta hepatitis usually follows that of HBV infection and it is particularly prevalent in endemic areas for HBV infection. The incidence of anti-HDV positivity appears to increase with age, especially among anti-HBe positive carriers. Infection with HDV occurs either as coinfection with the HBV or as superinfection in a chronic HBV carrier. Although it is often associated with HBV and progressive liver disease, the natural course may vary.

To establish the prevalence of delta infection in children, we investigated total anti-delta using an ELISA system (Organon Technica) in 206 children who were chronically infected with HBV (121 asymptomatic carriers, 59 with chronic persistent hepatitis, 13 with chronic active hepatitis, and 13 with cirrhosis) aged between 8 months and 17 years (mean SD) 7.76 (3.70) years. We detected antidelta in only six patients (2.95%): in three with cirrhosis, two with chronic active hepatitis, and one with chronic persistent hepatitis. Their ages ranged between 8 and 13 years. Four of them were positive for serum HBeAg and two were positive for anti-HBe. None of the asymptomatic carriers had antidelta. When we take into consideration the prevalence of antidelta in children with chronic liver disease it was 7.1% (6 of 85 children with chronic hepatitis or cirrhosis).

During four to seven years of follow up clinical and laboratory findings of our patients remained relatively stable.

Turkey has an intermediate endemicity for HBV infection and the prevalence of HBsAg carriers varies from 4% to 10%, and antidelta in adult patients with chronic hepatitis B has been found up to 36% in prevalence studies. Farci et al. in Italy, found a prevalence of 12.5% of antidelta in chronic hepatitis B infected children. However, in their study all children had chronic liver disease. The prevalence of antidelta in Turkish children is lower than that in Italian children, even if we consider only the patients with chronic hepatitis or cirrhosis (7.1% - 12.5%, respectively). There was no difference in the mean age of the patients and in the follow up duration between two studies. In Egypt a low prevalence of antidelta in children was reported (4.2%), whereas Ruiz Moreno et al. found a high prevalence in Spain (13%). The prevalence of antidelta in adults in Italy is similar to our country. The route of transmission of HDV infection in children might be different in various countries. The percentage of delta infection parallels the severity of the disease; in our study antidelta positivity was also high in patients with chronic active hepatitis (2/13), while none of asymptomatic carriers had antidelta. As previously shown in children and adults, a correlation between chronic delta infection and presence of anti-HBe was not observed in our patients. Although it was believed that delta infection usually worsened the course of the disease, clinical and laboratory findings of our patients were stable and follow up was similar to that found in the study of Bortolotti et al.2

We conclude that in our country the prevalence of HDV infection is not high during childhood, and prevalence increases with age suggesting that HDV infection is usually acquired as a superinfection rather than coinfection and vertical transmission is uncommon. The course of the disease is usually stable. The epidemiology of delta infection is different in various countries. To explain the differences in geographical distribution of HDV infection further studies are needed.

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Misdiagnosis of pancreatitis during valproate treatment in gastro-oesophageal reflux

Pancreatitis should always be suspected in patients with severe gastrointestinal symptoms during sodium valproate treatment. We report a child, receiving sodium valproate, in whom we misdiagnosed pancreatitis because of high serum amylases, which were later shown to be mainly of salivary origin (90%). We believe that a combination of gastro-oesophageal reflux plus the use of crushable sodium valproate (rather than enteric coated) led to ulcerative oesophagitis and absorption of luminal irritants.

A 16 year old, severely retarded child was admitted to the hospital because his epilepsy worsened. Ten days later the boy was reported to be having ‘fits’ of a new type: electroshock-like jerks, sweating, and hyperventilation provoked by turning the body, washing, and feeding. Feeding became difficult because of frequent attacks and associated cervical dystonia.

He was suspected of having pancreatitis caused by his sodium valproate treatment. Serum amylases rose rapidly to 3000 (normal 70-300) U/l. Valproate treatment was discontinued. Parenteral fluids were given for 5 weeks and as a complication of his subclavian catheter he acquired bacterial sepsis and candida infection. Oesophagoscopy showed haemorrhagic ulceration with active necrosis and gastro-oesophageal reflux. Surprisingly, 90% of the total serum amylase was of salivary origin and 10% of pancreatic origin. As he did not have pancreatitis, valproate was restarted, but now as enteric coated tablets. After revision of the subclavian catheter, and some days after starting antibiotics, he became afebrile and sepsis abated. His convulsions, both the original type and his new ‘fits’, became infrequent, and he was discharged.

His grandparents indicated that at home he was fed in a sitting position and the valproate was given as enteric coated tablets, whereas in hospital he was fed in a semi-recumbent position and crushable valproate tablets were used. The valproate, which is acid, was probably rapidly absorbed, and was locally irritating to the stomach and oesophagus. It may be that in these conditions, luminal amylose, which normally does not cross the gastric or intestinal mucosa, enters the blood or lymphatics.

Vasculitis associated with levamisole and circulating autoantibodies

Levamisole is used in relapsing nephrotic syndrome or as an adjuvant treatment to surgery in colon cancer. Three cutaneous vasculitides have been reported in adults on levamisole.2-4 No information is available on circulating autoantibodies in this condition. We would like to report a girl who had steroid dependent minimal change idiopathic nephrotic syndrome since the age of 6 years. The disease course was positively influenced by long term medication with levamisole and prednisone. Levamisole was stopped on two occasions but had to be resumed because of nephrotic relapses. At the age of 11.5 years, while continuing treatment with levamisole 1.2 mg/kg daily and prednisone 0.06 mg/kg every second day, the girl developed fever, arthralgia, and a non-palpable purpuric rash with a livedo pattern, chiefly on the breast, face, and arms. Physical and laboratory investigations failed to show signs consistent with systemic involvement. Circulating and pericellular antinuclear antineutrophil cytoplasmic autoantibodies (titre 1:2560) were detectable by indirect immunofluorescence on ethanol fixed granulocytes and a characteristic cytoplasmic reaction was obtained on formalin fixed granulocytes. However, at most only border-
fine levels of antimyeloperoxidase antibodies were detectable by enzyme linked immuno-sorbent assay (ELISA). Antinuclear (titre 1:640 by indirect immunofluorescence) and antithistone autoantibodies (strongly positive by ELISA) were also detectable. Antibodies to native DNA were moderately positive by ELISA but negative by indirect immunoflu- rescence using cultures of Crithidia luciliae.

The rash subsided within two weeks when levamisole was withdrawn. All autoantibodies were still detectable seven months later.

The clinical features in our case strongly resemble those described in the literature with the exception that in our patient the reaction occurred after a treatment period with levamisole of five years, by comparison, with one to three months in the reported patients.2

This observation indicates that cutaneous vasculitis induced by levamisole may be associated with circulating autoantibodies.

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Sadly, I cannot recommend this beautifully produced atlas because of its poor organisation, lack of index, ambiguous text, and lack of differential diagnoses. It serves as a useful record of the work and opinions of two pioneering paediatricians.

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David and Peter Hill offer this book to hospital paediatricians and general practitioners in training who work with children and to non-medical disciplines but curiously do not mention community paediatricians specifically as a target audience. It assumes previous knowledge of paediatrics and is clearly not an introductory text. Polnay and Hull's Community Paediatrics is therefore a better buy for undergraduates or the senior house officer entering the world outside hospital for the first time. But this book undoubtedly fills a gap in the market for specialist registrars embarking on the more specialised aspects of developmental assessment in a community setting or the child development centre. Until now such skills have had to be acquired on the basis of word of mouth backed up where possible with in-house teaching materials. The advent of a core text will be a blessing to those of us involved in specialist training.

The first seven chapters address in detail the assessment of children referred with developmental delay and the management of disclosure of developmental problems. The layout is pleasing to the eye, having two columns per page, with lots of headings. There are numerous tables giving useful hints on how to approach initial interviews and how to extract useful information by approximately phrased questions and answers (as outlined in the tables). The review of normal development is useful and hearing and vision assessment is described separately. Tests used in the assessment of intelligence, speech, and language are also generalised and outlined. The discussion is reviewed and their limitations and uses discussed. Headings will allow readers to dip into the text but the book is also eminently readable by chapters. A comprehensive index is provided.

The rest of the book is devoted to specific developmental disorders including their clinical features, investigation, and long term management. The choice of conditions described in each chapter varies, with half a page on the genetic variants of