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.1 Infection with HDV occurs either as coinfection with the HBV or as superinfection in a chronic HBV carrier. Although it is often associated with progressive liver disease, the disease, in this case, is often transient.2-4 The epidemiology of HDV 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 lumenic amylases.

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 hyper-ventilation provoked by turning the body, washing, and feeding. Feeding became difficult because of frequent attacks and associated delirium. He also developed vomiting.

He was suspected of having pancreatitis caused by his sodium valproate treatment. Serum amylases rose rapidly to 3000 (normal 70-300) U/I. Valproate treatment was discontinued. Parenteral fluids were given for 6 weeks and as a complication of this subclavian catheter he acquired bacterial sepsis and candida infection. Oesophagoscopy showed haemorrhagic ulceration of the oesophagus 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 afibrile 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 supine 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. He may be that in these conditions, luminal amylase, 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 sur- gery in cold agglutinin disease. 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 an systemic involvement. Circulating pro nuclear antineutrophil cytoplasmic autoanti- bodies (titre 1:2560) were detectable by indi- rect immunofluorescence on ethanol fixed granulocytes and a characteristic cytoplasmic reaction was obtained on formalin fixed granulocytes. However, at most only border-

David Hall and Peter Hill offer this book to hospital paediatricians and general practitioners who are attempting to keep up to date with the evidence-based medicine 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 Hall'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 by 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 appropriately phrased questions (which are 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 and general screening are all reviewed and their limitations and uses discussed. Headings will allow readers to dip into the text but the book is also eminently readable chapter by chapter.

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 detail seem to be arbitrary with half a page on the genetic variants of
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