Prevalence of antidualta 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 and progresses to cirrhosis, 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 luminal valproate.

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 beholding. He underwent a complete evaluation.

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. Parental fluids were given for four weeks and as a complication of this subclinical catheter he acquired bacterial sepsis and candida infection. Oesophagoscopy showed haemorrhagic ulceration, oesophagitis 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 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 agglutinins. Three cutaneous vasculitides have been reported in adults on levamisole.2 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 for 2 low nuclear antineutrophil cytoplasmic autoantibodies (titre 1:2560) were detectable by indirect immunofluorescence on ethanol fixed neutrophils and a characteristic cytoplasmic reaction was obtained on formalin fixed neutrophils. However, at most only border-
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