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.[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 severe and progressive liver disease, the natural course may vary.[2]

To establish the prevalence of delta infection in children, we investigated total antidelta 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 (2.9%): 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% (six 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% [3 4] and antidelta positivity in adult patients with chronic hepatitis B has been found up to 36% in prevalence studies.[5] Farci *et al*, in Italy, found a prevalence of 12.5% of antidelta in chronic hepatitis B infected children.[6] 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% *v* 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%), [7] whereas Ruiz-Moreno *et al* found a high prevalence in Spain (13%).[8] The prevalence of antidelta in adults in Italy [9] is similar to our country. The route of transmission of HDV infection in children might be different in various contries. The percentage of delta infection parallels the severity of the disease[2 6]; in our study antidelta positivity was also high in patients with cirrhosis (3/13) and chronic active hepatitis (2/13,) while none of asymptomatic carriers had antidelta. As previously shown in children and adults,[5 6] 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 during follow up, 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 its 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 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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1 Rizzetto M, Durazzo M. Hepatitis delta virus (HDV) infections (epidemiological and clinical heterogeneity). *J Hepatol* 1991; **13** (suppl 4): 16-8.
2 Bortolotti P, DiMarco V, Vajro P, *et al.* Long-term evaluation of chronic delta hepatitis in children. *J Pediatr* 1993; **122:** 736-8.
3 Çakaloğlu Y, Ökten A, Yalcin S. Türkiyede hepatit B virusu seroepidemiyolojisi (tasiyicilik-seropozitiflik prevalansi). *T Klin Gastroenterohepatol Derg* 1990; **1:** 49-52.
4 Kuru Ü, Şenli S, Türel L, *et al.* Age-spesific seroprevalence of hepatitis B virus infection. *Turk J Pediatr* 1995; **37:** 331-8.
5 Özdemir S, Sonsuz A, Sentürk H, Gürakar M. The prevalence and clinical impact of delta infection in the patients with HBV-related chronic liver disease. *T Klin Gastroenterohepatol* 1991; **2:** 189-95.
6 Farci P, Barbera C, Navone C, *et al.* Infection with the delta agent in children. *Gut* 1985; **26:** 4-7.
7 Abdel-Fattah S, el-Kholy MS, Abdel-Fattah SM, *et al.* Delta virus and hepatitis B surface antigen in chronic liver diseases. *J Egypt Public Health Assoc* 1991; **66:** 427-39.
8 Ruiz-Moreno M, Camps-Rubiol T, Carren-Garcia V, *et al.* Follow-up study of delta infection in children with chronic hepatitis B. *An Esp Pediatr* 1989; **30:**255-60.
9 Smedile A, Lavarini C, Arico S, *et al.* Epidemiological patterns of infection with the hepatitis B virus associated delta agent in Italy. *Am J Epidemiol* 1983; **117:**223-9.

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 amylase.

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 belching. He appeared to be in pain.

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 required for four weeks and as a complication of his subclavian catheter he acquired bacterial sepsis and candida infection. Oesophagoscopy showed haemorrhagic ulcerative 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 semirecumbent 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.[1]

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1 Pieper-Bigelow C, Strocchi A, Levitt M. Where does serum amylase come from and where does it go? *Gastroenterol Clin North Am* 1990; **19:** 793-810.

Vasculitis associated with levamisole and circulating autoantibodies

Levamisole is used in relapsing nephrotic syndrome or as an adjuvant treatment to surgery in colon cancer.[1] 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 perinuclear 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-