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.

AYSEL YÜCE
NURTEN KOÇAK
HASAN ÖZEN
FIGEN GÜRAKAN
Division of Paediatric Gastroenterology, Department of Paediatrics, Hacettepe University, Faculty of Medicine, Ankara, Turkey
(Correspondence to: Dr Aysel Yüce, Hacettepe Üniversitesi, Çocuk Gastroenteroloji Ünitesi, 06100 Ankara, Turkey)

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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 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]

R RIIKONEN
Children's Hospital, University of Helsinki, Stenbäckink 11, Helsinki 00290, Finland

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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-

line levels of antimyeloperoxidase antibodies were detectable by enzyme linked immunosorbent assay (ELISA). Antinuclear (titre 1:640 by indirect immunofluorescence) and antihistone autoantibodies (strongly positive by ELISA) were also detectable. Antibodies to native DNA were moderately positive by ELISA but negative by indirect immunofluorescence 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.[2-4]

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

R LAUX-END
D INAEBNIT
H A GERBER
M G BIANCHETTI
University of Berne, Inselspital, CH-3010 Bern, Switzerland

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AUTUMN BOOKS

Physical Signs of Child Abuse. A Colour Atlas. By Christopher J Hobbs and Jane M Wynne. (Pp 245; £60 hardback.) W B Saunders, 1996. ISBN 0-7020-1778-7.

When the history of the eighties comes to be written there may be more than a passing reference to the epidemic of child abuse uncovered during this decade. Together with the collapse of communism and the march of materialism, there was enormous social change. The rapid transformation throughout the developed democracies from smoke stack to service economies had severe consequences for the large communities of labouring populations no longer required in the manufacturing conurbations. This social involution destroyed longstanding support networks, some of which had concealed or contained abusive behaviour towards children. Coupled with changes in family formation, this resulted in increasing pressures on parents: some of whom found that declaring abuse uncovered resources.

Beliefs influence decisions and diagnoses—even if we are unaware of them. There is a human tendency to conform with firmly expressed opinions rather as religious sects fall into doctrinal rigidity. One belief comes to dominate and any opposition is damned as heretical. Paediatricians in Britain will be aware of the problems that arose after overzealous investigations in communities as disparate as Rochdale and Orkney; American readers would be similarly aware of Merivale and Wenatchee.

Two of the pioneering workers in the field of child abuse in the 1980s were doctors Hobbs and Wynne who worked together in Leeds. They have published prolifically but, like many pioneers, not without opposition or criticism. The time may now be opportune to reflect objectively on this body of work. The atlas may assist in the process suggested by Vandeven and Emans whereby photographs of lesions should be reviewed by panels of experts to establish an audit of clinical criteria for a diagnosis of child abuse (*Arch Dis Child* 1995; 73: 469-71). Hobbs and Wynne's atlas provides a firm pictorial record of the material on which they based their opinions.

This atlas is beautifully presented with many colour illustrations, radiographs, and growth charts that summarise the sad catalogue of effects of violence towards the vulnerable. The introduction suggests that it is important as a resource for practically everybody involved in child protection (from judges to nursery nurses and police to paediatricians). The text however is limited, banal in places ('don't forget the battery in the flash') and in many cases too technical for a non-medical readership. A simple description of both the normal and the injuries would have been helpful. An indication of the range of possible causes for a given illustration would be expected; ambiguous illustrations are not helped by the text 'this is a worrying sign'. Differential diagnoses, including accidents and rare conditions, are required. An index would also be useful.

The book would benefit from strict editing with a layout in a more logical form to facilitate access and cross referencing. More rigid selection of photographs is needed to indicate the relative importance of different conditions.

It is always refreshing to review colleagues' views. There is much to learn in this atlas for many professionals. Teachers may be interested to know that 'spanking may have sexual overtones', dentists that 'untreated dental caries are part of the picture of neglect', dietitians that 'failure to thrive and obesity may be part of the same attachment difficulty which amounts to abuse', and gynaecologists to learn that 'children who insert foreign bodies have almost always been sexually abused'. The inclusion of accidental burns and evident deprivation as abusive acts serves only to confuse, weakens efforts to help the underprivileged, and may indicate a lack of objectivity in the authors.

International referees cited by the editor of *Child Abuse and Neglect* questioned the very high level of positive findings among Leeds children, together with the low level of allegations by these children (*Child Abuse Negl* 1989; 13: 165). Such a fraught area requires careful reflection, repeated reassessments of objectivity together with full assessment of all aspects of the child's history. Overstatement may lead to scepticism with consequent neglect of those in need of help. The time is right for the establishment of clear criteria and guidelines for the diagnosis of child abuse. Unfortunately this book does not serve this purpose.

There are several excellent alternative teaching aids I prefer: *ABC of Child Abuse* (edited by S R Meadow); London: BMA Publications, 1989 (this includes the work of the authors of this atlas). *Atlas of Child Sexual Abuse* (edited by D Chadwick *et al*); Chicago: Yearbook Publications, 1989 (a masterly monograph). *The Battered Child Syndrome* (by S M Smith); London: Butterworth, 1975 (which in 27 illustrations shows most aspects of physical abuse and has a useful historical introduction). *Child Abuse. A Handbook for Health Care Practitioners* (by I Blumenthal); London: Edward Arnold, 1994 (uses line drawings rather than photographs in a balanced comprehensive text). *Clinical Forensic Medicine* (edited by W S McLay); London; Pinter Publications, 1990 (the chapters on child abuse and child sexual abuse have clear uncontentious text with line illustrations).

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.

R SUNDERLAND
Consultant paediatrician

The Child with a Disability. 2nd Ed. By David M B Hall and Peter D Hill. (Pp 386; £65 hardback.) Blackwell Science, 1995. ISBN 0-86542-850-6.

David Hall and Peter Hill offer this book to hospital paediatricians and general practitioners, professionals allied to 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 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 passed on 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 development are 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 seems somewhat arbitrary with half a page on the genetic variants of