Another case of HBV associated membranous glomerulonephritis resolving on lamivudine
Connor and colleagues (see page 446) report rapid resolution of a hepatitis B associated membranous glomerulopathy and nephrotic syndrome after two months of oral lamivudine. We would like to add our experience in a similar case.

A 5 year old female of Vietnamese origin presented with a two week history of periorbital swelling and weight gain. She had 3 plus protein, 20-200 dysmorphic red blood cells, and red blood cell casts in her urine. Serum albumin was low at 19 g/l, cholesterol was 9.8 mmol/l, and serum complement C3 and C4 were reduced (0.61 g/l and <0.1 g/l, respectively). She was hepatitis B surface antigen positive and hepatitis B surface antibody negative. The physician caring for her at that time placed her on prednisone 60 mg/m\(^2\) per 24 hours, and there was microscopic haematuria with lamivudine in a similar case. She continued to be hepatitis e antigen positive without detectable e antibodies. The average duration of proteinuria is 30 months. We believe that treatment with lamivudine in this case likely suppressed the virus and resulted in early remission of clinical nephrotic syndrome; however, the subsequent rebound in viral load and renal biopsy results probably indicates loss of viral suppression, leading to the subclinical relapse. It is unknown at this time if the strain of hepatitis B has developed resistance to lamivudine. Effective viricial agents may be needed to prevent relapses of hepatitis B induced membranous glomerulonephritis. Further, finally work is needed to investigate the efficacy of this treatment in a larger cohort and to establish guidelines about the duration of such therapy.

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References


PCD or not PCD
In response to the leading article on primary ciliary dyskinesia (PCD) and the commentary by Dr Andrew Boon, we write as clinicians with an interest in PCD who work in general paediatrics and neonatology. We agree with Dr Boon that the identification of an uncommon medical disorder from the large number of children presenting with common symptoms and signs is a major challenge for the general paediatrician. We also share the view that it is undesirable and certainly impractical to refer every child with recurrent episodes of cough, rhinitis, and scrotal oedema for further investigation. However, we believe that the aim of the editorial by Professor C O’Callaghan and Dr A Bush was to provide information on subtle differences in the clinical presentation of PCD to help us differentiate these patients from those with common non-specific childhood respiratory problems. For example, it is uncommon for a term infant to be admitted to a neonatal unit with significant respiratory concerns following a vaginal delivery but common in infants with PCD. We performed an as yet unpublished questionnaire survey of individuals belonging to the PCD support group which identified that 47% had been admitted to a neonatal unit with unexplained respiratory problems following a normal vaginal delivery. Rhinitis is also very rarely seen in normal neonates but is extremely common in patients with PCD. Other subtle clues increasing the likelihood of PCD are the characteristic cough and middle ear problems especially the development of persistent otitis media after tympanostomy tube insertion.

There is of course no doubt that a cheap reliable screening test would significantly help promote early diagnosis of PCD but it is not yet on the horizon. A detailed history especially of the neonatal period will help those working in neonatology or general paediatrics to highlight the patient that should be referred for further investigations including cilia studies.

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PCD or not PCD

Hazards in the epidemiological study of sudden infant death syndrome

The study of Platt and Pharaoh, confirms the increased risk of SIDS in twins compared with singletons. They point out that a major component of that higher accrued risk is that twins tend to be of low birthweight. Their finding that like-sex twins are at no greater risk than unlike-sex twins adds to the substantial evidence concerning the very limited role of genetic susceptibility for SIDS, and the rarity of recurrence in siblings of victims.

The authors illustrate the gratifying fall in the number of SIDS during the six years of their 1990s study. As the number of infants categorised each year as SIDS in England and Wales comes nearer to that of 200, so it becomes more important for those involved in epidemiological studies to be sure that the categorisation (i.e. the diagnosis) is correct. I refer to infants who, a few years after they have been categorised as SIDS, have been re-assessed, usually because of a subsequent child being abused or killed, and, in the course of court proceedings, findings are made that...
the previous infant(s) were killed by the par-
ent, rather than dying of natural causes. Cur-
rently, there does not seem to be a mechanism for cor-
correcting the national childhood mor-
tality statistics when later, correct diagnoses are made. For instance, in the 1990s, I am aware that 20 infants who were initially categorised as SIDS, but who in later years, after extensive child protection investigations, were deemed to have been killed, usually by smothering. Colleagues will know of other cases: the true number will be higher. It is unfortunate that the official statistics do not seem to be altered retrospectively, and remain a misleading figure for any research worker. I should add that, since none of the cases of parental killing of which I am aware involves twins, the conclusions of Platt and Pharoah are more likely to have been strengthened rather than weakened by such false diagnosis. Hence, the number of SIDS continues to fall, it will become ever more difficult for research workers to compare small sub-
groups of SIDS within national mortality sta-
tistics unless the statistics are revised retro-
spectively in response to later correct diagnosis.

It is appropriate to warn of an additional hazard for research workers in this field. In the same issue of Archives there was an inter-
view with an epidemiologist in France concern-
ing the possibility of vagal over-
activity as a cause of sudden infant death. They referred to a “positive family history of SIDS”. A past epidemiologist in France concern-
ing SIDS, yet subsequent questioning of the relevant grandparent has revealed that no such infant deaths occurred. Presumably, the mother responsible for smothering or kill-
ing her child has invented the family history, the mother responsible for smothering or killing her child has invented the family history, the mother responsible for smothering or killing her child has invented the family history, the mother responsible for smothering or killing her child has invented the family history, the mother responsible for smothering or killing her child has invented the family history.

In the reported cases, the children had been administered substantially (up to 5 times) higher than the glaso SmithKline (GSK) Core Data Sheet recommended Flixotide dose of 400 mg/kg/day and use of fluticasone (FP) at such doses is certainly not endorsed by GSK. Within the recommended doses, there are a wealth of data from controlled clinical trials that Flixotide is a well tolerated and effective drug in adults and children.10 There are a number of recent studies in children which identified no cases of adrenal crisis and no effect on growth during 12 months treat-
ment with FP at licensed doses.11 There are also a number of methodological deficiencies in this survey, the most important being that the survey is not case-controlled and lacks information on true incidence against the overall FP use or exposure. In addition, it is unclear from the survey what attempts were made to closely monitor any adrenal suppression that might occur with increasing doses of FP or whether patients were down-titrated to the lowest effective FP dose, as routinely recommended. The survey data also imply that fluticasone has been implicated in the great majority of cases of adrenal failure even though it is the least frequently prescribed form of inhaled corticosteroid. Prescribing data in relation to fluticasone propionate with budesoindc and beclometasone dipropionate at half the microgram dose or less. Respir Med 1998;92:95–104.


DIN-LINK Data, Compufile Ltd, (March 2002).


Hazards in the epidemiological study of sudden infant death syndrome

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