Incorporated into the lamina densa with fragmentation of the latter, consistent with a membranous glomerulonephritis stage II/III. She has evidence of hyperfiltration with a glomerular filtration rate of 163 ml/min/1.73 m$^2$ as determined by $^{99}$Tc-DTPA clearance.

The ideal treatment for hepatitis B associated membranous nephropathy in children is yet to be determined. There is one retrospective analysis of six studies comprising a total of 82 children that showed 68% complete remission 12 months after the diagnosis, 7.3% renal failure, 2.4% end stage renal failure, and 30% persistent disease. Therapies should not be used as it does not appear to be beneficial, and the steroids may enhance viral replication in mononuclear cells. The average duration of proteinuria is 30 months. We believe that treatment with lamivudine in this case likely suppressed the viral load, 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 viridical 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.

**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 serous otitis media 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 of the cough and middle ear problems especially the development of persistent otitis media after tympanostomy tube placement.

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 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.

**Hazard 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, 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 parent, rather than dying of natural causes. Currently, there does not seem to be a mechanism for correcting the national childhood mortality 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 prospectively, and remain a misleading figure for any researcher. I should stress that, since most of the cases are taken at face value and become part of the national statistics unless the statistics are revised retrospectively 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 interview with epidemiologists in Paris concerning the possibility of vagal overactivity as a cause of sudden infant death. They referred to a ‘positive family history of SIDS’. A parent or a sibling, or both, of the infant in question was reported to have died of SIDS; the other sibling or sibling was not part of the relevant family history. Care has been verified in considerable detail, mistakes may be made.

In recent years I have been involved with families in which parents who have repeatedly denied involvement have provided evidence of parental involvement.

A second reason for verifying the alleged previous deaths is that the survey is not case-controlled and lacks information on true incidence against the overall PP use or exposure. In addition, it is unclear from the survey what attempts were made to closely monitor any adrenal suppression in children. The survey data also imply that fluticasone propionate 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 use in the UK (DINLINK, Doctor's Independent Network database), shows that it is in fact the most commonly prescribed inhaled corticosteroid in children with moderate and severe asthma. DINLINK is an amalgamated database of the anonymised computer records of a panel of 300 general practitioners spread across the UK selected to represent the demographic population of the UK.

In addition, the authors’ contention that adrenal effects with FP are due to its greater lipophilicity and hence accumulation over prolonged periods is misconceived and inaccurate. There are studies to show that there is no accumulation of FP at a steady state.26 It is the clearance value which determines the amount of FP in the body at steady state, and lipophilicity per se is not a relevant factor.27 I also wanted to take the opportunity to comment on the editorial by Dr Russell. The last line of the editorial recommends that if high dose inhaled corticosteroids is considered necessary, that it is advisable not to use fluticasone propionate.28 The recent publication by the CSM “Current Problems in Pharmacovigilance” states that adrenal suppression is a dose related class effect of inhaled steroids, and that all inhaled corticosteroids are associated with an increased risk of adrenal crisis when used at higher than licensed doses.

In conclusion, inhaled corticosteroids have an important place in asthma management throughout the world, and this paper by Todd et al should be reviewed in this context. Any inhaled corticosteroid used at such high doses has the potential to cause systemic effects, and paediatricians should be encouraged to treat their patients using the lowest effective dose, down-titrating as appropriate.

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