active liver disease and continuing virus replication, and glomerulonephritis occurred only when both hepatitis B e antigen and antibody were simultaneously detectable in serum, thus suggesting that this antigen-antibody complex, with a molecular ratio near to equilibrium, might by of pathogenetic importance. In fact the complex has a molecular size thought to be capable of inducing glomerular disease and recently Takekoshi et al. and Ito et al. found hepatitis B e antigen without surface antigen, in the glomerular deposits of some children with membranous glomerulonephritis. Moreover an association was found between the hepatitis B e antigen/antibody status and outcome of glomerulonephritis. Resolution of the disease was often associated with hepatitis B e antibody seroconversion. Even in our patient this seroconversion was followed by a rapid return to normal of the urine analysis but only when it was associated with the end of hepatitis B virus replication and subsequent remission of liver disease. The favourable outcome of both hepatic and renal disease after the end of virus replication provides further support to the concept that both diseases, although occurring at different times and with different pathogenetic mechanisms, are due to the same aetiological agent. In our experience, more that 90% of children with chronic hepatitis type B remain chronic surface antigen carriers after hepatitis B e antibody seroconversion (unpublished observations). Our patient was one of the few in whom hepatitis B surface antigen was no longer found in serum some months after seroconversion. Why he finally developed an adequate immune response to hepatitis B virus remains unclear.

Since the end of hepatitis B virus replication seems to be a crucial event in the remission of both hepatic and extrahepatic disorders associated with the chronic infection, immunosuppressive treatment that potentiates virus replication would not find a rationale in the treatment of hepatitis B virus associated membranous glomerulonephritis. In our patient, however, short term immunosuppressive treatment, given at the onset of the nephrotic syndrome, did not seem to interfere with the favourable outcome of the infection and related disorders.

References

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Recurrent croup

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SUMMARY Thirty one of 486 children followed from birth had recurrent croup in the first four years of life. Twenty one were boys, and 10 girls. Recurrent croup occurred significantly more often in families with a positive history of allergy but was not significantly associated with the initial feeding method.

Recurrent croup was an unexpectedly common clinical occurrence identified in 6-4% of the infant population of the Isle of Wight born during a 12 month period and followed for the first four years of life. Detailed results of the first two years of the Isle of Wight infant feeding survey have been published.3-4

We wish to draw particular attention to recurrent
croup as a possible manifestation of allergic disease. We believe this form of acute stridor is at least as common as the frequently diagnosed ‘acute viral laryngotracheitis’.

Methods

Parents of children born between April 1, 1977 and March 31, 1978 were sent a questionnaire when the child had reached 5 years of age. Information was sought on the respiratory and other manifestations of disease that might have an allergic basis. This report is concerned with the occurrence and frequency of acute laryngeal stridor (croup). Croup was distinguished from asthma and wheezing in the questionnaire and we doubt that these signs have been misrecorded in this study, although we acknowledge that confusion does occur occasionally in clinical practice.

Results

Information was received from 486 families. Table 1 shows the number of episodes of croup recorded. Recurrent croup is more common in boys. Table 2 shows that recurrent croup occurs significantly more often in families where one or both parents have a history of atopic disease—eczema, asthma, or hay fever. Recurrent croup was recorded more often in the exclusively breast fed infants (8.8%) than in those given cows’ milk or mixed breast and formulae feeding (5.6%). This tendency does not achieve significance statistically and may be explained by the preference of mothers with a family history of atopy to breast feed their infants.

Discussion

Acute episodes of laryngeal stridor in infants and young children are alarming to patients and their parents and to the medical practitioners caring for them. Most frequently acute croup episodes of stridor are attributed to viral laryngotracheitis, although bacterial infections, such as acute epiglottitis, usually due to Haemophilus influenzae type B, staphylococcal tracheobronchitis (pseudomembranous croup), and diphtheria may need to be excluded.

In about 50% of cases of acute laryngotracheitis no pathogen, bacterial or viral, can be identified. This raises the possibility of a non-infective trigger for the symptoms of a substantial number of children. Allergy has been suggested as a cause of recurrent croup but there has been some reluctance to acknowledge this as an aetiologic factor. Zach and his colleagues in Melbourne recently reported their studies on 110 children nine years after each had been admitted to hospital with croup. Fifty seven were reported as having recurrent croup (in his series more than two episodes) and 33 were not considered allergic. A highly significant relation was reported between allergy and recurrent croup.

The present study confirms the findings of Zach et al of a male preponderance and a strong predisposition for croup to recur in allergic families. The prevalence of recurrent croup is very similar to that of asthma in children on the Isle of Wight. It may well be that the role of infection has been over emphasised in recurrent croup just as it has in asthma.

Table 1 Episodes and sex distribution of croup in first four years of life

<table>
<thead>
<tr>
<th>No of episodes</th>
<th>No of children</th>
<th>Boy : girl</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>426</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>29</td>
<td>16 : 13</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>2 : 3</td>
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<tr>
<td>3</td>
<td>14</td>
<td>12 : 2</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>0 : 1</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>0 : 2</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>2 : 0</td>
</tr>
<tr>
<td>&gt;6</td>
<td>7</td>
<td>5 : 2</td>
</tr>
</tbody>
</table>

Table 2 Relation between recurrent croup and parental allergy

<table>
<thead>
<tr>
<th>No (%) with croup</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive history of allergy</td>
</tr>
<tr>
<td>Negative family history of allergy</td>
</tr>
</tbody>
</table>

References


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