Febrile convulsions and sudden infant death syndrome

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SHORT REPORT

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It has been suggested that sudden infant death syndrome (SIDS) and febrile convulsions (FC) are related aetiologically. Both conditions may be age specific reactions to fever in susceptible children, and the common mechanism may be termolabile syncope with cerebral ischaemia. The hypothesis is supported by the observations that cats’ reactions to artificially induced fever depend on age; younger kittens tend to die suddenly, older kittens tend to have convulsions, and mature cats tend to remain intact. Both SIDS and FC occur in time based clusters, and children who die of SIDS are generally younger than children with FC. Hyperthermia, which may be caused by infections, heavy wrapping, and prone sleeping, is a common finding in infants who died of SIDS.

If the shared susceptibility hypothesis is true, we would expect siblings of children with FC to have an increased risk of SIDS.

SUBJECTS AND METHODS

We performed a follow up study based on information from two nationwide registries in Denmark. The National Hospital Register contains information on almost all discharges (99.4%) from Danish hospitals since 1977. Diagnostic information is classified according to a Danish version of the International Classification of Diseases; ICD8 was used from 1984 to 1993, and ICD10 from 1994 to 1998. We included children with FC if they had ICD8 code 780.21 or ICD10 code R56.0, were between 3 and 60 months old at the time of discharge, and had no recorded history of non-febrile convulsions, cerebral palsy, severe head traumas, intracranial tumours, meningitis, or encephalitis. The Fertility Database at Statistic Denmark links several population based registries in order to obtain data on family structure, social conditions of the family, pregnancy outcome, and causes of death. We linked information from the two registries by means of the personal number assigned to all Danish citizens at birth. We then identified two cohorts of single born children who were born between 1984 and 1994. The FC+ cohort consisted of the subsequent sibling of patients who were born between 1980 and 1993 and had been hospitalised with FC between 1980 and 1998 (n = 9877). The FC− cohort consisted of the subsequent sibling of randomly selected children who were born between 1980 and 1993 and survived to the age of 5 years without being hospitalised with FC (n = 20 177). Only one sibling from each family was included. SIDS was defined as ICD8 code 795.8 and ICD10 code R95.9. The period of follow up for SIDS began at the time of birth and ended at the date of death, date of emigration, when the child reached 1 year of age, or 31 December 1995, whichever occurred first. The risk of SIDS was analysed using Cox proportional hazard regression, and the results are presented as hazard ratios (HR) with 95% confidence intervals (95% CI).

RESULTS

We followed 30 054 infants for 29 844 person years, and identified 49 infants who died of SIDS, corresponding to an overall incidence rate of 1.64 per 1000 person years in the two cohorts. We found no evidence of an increased risk of SIDS in siblings of children who had been hospitalised with FC (HR: 0.90; 95% CI: 0.49 to 1.66; table 1). We ran several Cox regression models in order to adjust for maternal age (<25, 25–34, >34 years), maternal education (7–9, 10–11, >11 years), social class of the family (low, middle, high), parity at birth of index child (primiparous, multiparous), gender (boy, girl), birth weight (<2500, 2500–4000, >4000 g), gestational age at birth (<37, 37–42, >42 weeks), interpregnancy interval (<24, 24–48, >48 months), and calendar period at birth (1984–87, 1988–91, 1992–94). None of these adjustments changed the result.

In December 1991, the health authorities in Denmark recommended a non-prone sleeping position, which was followed by a notable decrease in the incidence of SIDS. Excluding children born after 1991 had no impact on the result. Furthermore, no change in the results was seen when half siblings and children with unknown fathers were excluded.

DISCUSSION

First degree relatives of patients with FC had no overall increased risk of SIDS, and the study did not support the shared susceptibility hypothesis. Our cohort study was population based, had complete follow up, and the data did not rely on parental recall. We find it unlikely that bias or confounding have masked an association.

Abbreviations: FC, febrile convulsion; HR, hazard ratio; SIDS, sudden infant death syndrome

Table 1 Risk of SIDS in siblings of children who had had febrile convulsions (FC+) relative to the risk in siblings of children who had never been hospitalised with febrile convulsions (FC−) as measured by the Cox HR

<table>
<thead>
<tr>
<th>Cohorts</th>
<th>Person years at risk</th>
<th>SIDS No.</th>
<th>No. per 1000</th>
<th>HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FC−</td>
<td>20036.98</td>
<td>34</td>
<td>1.70</td>
<td>1.00*</td>
</tr>
<tr>
<td>FC+</td>
<td>9807.25</td>
<td>15</td>
<td>1.53</td>
<td>0.90 (0.49 to 1.66)</td>
</tr>
</tbody>
</table>

*Reference.
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