Diabetes Following Mumps in Sibs

Diabetes mellitus is a well-documented but rare complication of mumps. Hinden (1962) found that less than 20 cases had been described. Since then occasional further cases have been reported. To our knowledge the condition was noted in sibs only once (King, 1962). We here present 2 more cases of this rare and serious complication of mumps in sibs.

Case Reports

Case 1. Female, 6 years old, was admitted to hospital in January 1970, with a history of gradual loss of weight, polydipsia, and enuresis of four months' duration. Two months before the onset of symptoms she had developed bilateral parotid swellings with fever, diagnosed as mumps.

On examination she was found to be moderately dehydrated, with a dry skin. Respiration was rapid and deep, and acetone was obvious in the breath. The diagnosis of diabetes mellitus was established by the high levels of blood sugar and the presence of sugar and ketones in the urine. The main laboratory findings are shown in the Table.

The patient was treated with parenteral fluids and insulin and was soon stabilized on 15 units of lente insulin.

Case 2. Male, 9 years old, was admitted to hospital in January 1970. He had poor appetite, polydipsia, polyuria, and enuresis for 4 months. He had fallen ill with fever and bilateral parotid swellings a fortnight before his sister. Because of his continuing symptoms and the diagnosis of diabetes in his sister, his blood and urine were examined, and a blood sugar of 250 mg/100 ml and a urine loaded with sugar and ketones were found. The laboratory findings in the ward are shown in the Table.

He was discharged from the hospital stabilized on 25 units of lente insulin.

Further investigations for establishing the diagnosis of mumps and the exclusion of prediabetic state in parents were performed. Complement-fixation tests as shown in the Table gave titres for both S and V mumps antigens, indicating past infection. Blood glucose tolerance test was normal in both parents. There was no family history of diabetes in three generations.

Discussion

Clinical and epidemiological evidence shows that there is a close relation between apparent or in-apparent mumps infection and diabetes mellitus (Gundersen, 1927; Melin and Ursing, 1958; Hinden, 1962). Inapparent infections with the virus of mumps, on the basis of serological evidence, represent no more than one-third of all infections (Maris et al., 1946), which means that diabetes mellitus will still be a rare complication of mumps even if serological tests are performed during or after epidemics. The pathological basis of the condition seems to be the involvement of the pancreas by the mumps virus. Clinical pancreatitis as a complication of mumps is not common in children, but to what extent the condition passes unnoticed is not known (Blumenstock, Mitheofcr, and Santulli, 1957; Hinden, 1962; McCree, 1963). This complication is rarely followed by diabetes, thus indicating that apart from the involvement of the pancreas other factors may play a role in the pathogenesis.

A latent state of diabetes mellitus in children can be activated during the course of a viral or bacterial infection. In our patients the negative family history for diabetes in three generations, and the normal glucose tolerance test in the parents are against, though not excluding, the existence of a prediabetic state.

The almost simultaneous occurrence of diabetes mellitus in both sibs is in favour of a common aetiological agent.

Whether diabetes in the reported cases has been developed by a diabetogenic strain of mumps virus or by mere chance is not easy to conclude.

Summary

Symptoms of diabetes mellitus began 2 months after mumps in a 9-year-old boy and his 6-year-old sister. A causal relation between the mumps and the diabetes is suggested.

TABLE

<table>
<thead>
<tr>
<th>Laboratory Findings</th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood sugar (mg/100 ml)</td>
<td>264</td>
<td>260</td>
</tr>
<tr>
<td>Serum sodium (mEq/L)</td>
<td>123</td>
<td>130</td>
</tr>
<tr>
<td>Serum potassium (mEq/L)</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Serum chloride (mEq/L)</td>
<td>106</td>
<td>99</td>
</tr>
<tr>
<td>Alkaline reserve (mEq/L)</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>Sugar in the urine (g/100 ml)</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Ketones</td>
<td>+ + + +</td>
<td>+ + + +</td>
</tr>
<tr>
<td>Complement-fixation test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mumps S</td>
<td>1 : 32</td>
<td>1 : 32</td>
</tr>
<tr>
<td>Mumps V</td>
<td>1 : 64</td>
<td>1 : 64</td>
</tr>
</tbody>
</table>

References


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Diphenoxylate Hydrochloride (‘Lomotil’) Poisoning in Children

Three cases of diphenoxylate hydrochloride (Lomotil) poisoning in children have been reported (Harries and Rossiter, 1969; Henderson and Psaila, 1969; Riley, 1969). We describe a fourth case, to emphasize the great importance of early diagnosis and prompt therapy.

Case Report

A 2-year-old girl was admitted to hospital in April 1970 at 12.30 p.m. Her parents had brought her directly to hospital because at 11 a.m. on the same day she had been found playing with a bottle of diphenoxylate (Lomotil) tablets (each containing 2·5 mg diphenoxylate hydrochloride and 0·025 mg atropine sulphate). These had been prescribed for her father’s ‘nervous diarrhoea’ and 35 to 40 tablets could not be accounted for. It was feared that this healthy 2-year-old had ingested the missing tablets between 10.30 a.m. and 11 a.m. on the day of admission.

Gastric lavage was performed at 12.30 p.m. No tablets were found in the gastric aspirate, but the child was admitted for observation. She was drowsy and the respiration rate was 12/min.

Respiration rate was recorded half-hourly and remained steady around 20/min until 8.30 p.m. when she stopped breathing completely. The heart rate and pupillary reactions were normal. Intermittent positive pressure with oxygen via an ‘Ambu’ bag was applied, and 2 mg nalorphine hydrobromide was injected intramuscularly. Two minutes later spontaneous respirations began, at first gasping in type, but within a further 10 minutes they became deep and regular at a rate of 10/min. The child remained drowsy but responsive to painful stimuli. She showed no neurological deficit. The heart rate and rhythm were normal.

After this episode, the respiration rate was continuously monitored and a second period of apnoea occurred at 3.30 a.m., more than 16 hours after the ingestion of the tablets. Oxygen was given as above for 5 minutes and a further 2 mg nalorphine hydrobromide was injected intramuscularly. This was followed in 2 minutes by the onset of spontaneous respiration.

The patient remained drowsy for 24 hours. The 2 mg injections of nalorphine were repeated every 4 hours for a full 24 hours, followed by 1 mg every 4 hours for a further 24 hours, when the drug was discontinued. The child remained fully conscious and her respiration gave no cause for further alarm. Chest x-ray was normal. She was allowed home perfectly well, 4 days after admission.

Comment

Diphenoxylate hydrochloride is a widely used drug and is often prescribed for relatively trivial and self-limiting conditions. We do not feel that the dangers of accidental poisoning with this drug are sufficiently well recognized. Diphenoxylate hydrochloride is structurally related to pethidine, is a powerful antitussive, and prolongs the transit time of the intestinal contents. The onset of action is slower and its duration longer than that of morphine, pethidine, or other opiates. The manufacturers of Lomotil* deliberately added subtherapeutic doses of atropine sulphate to diphenoxylate in order to discourage excessive self medication, but it is very difficult to legislate for the inquisitive and ubiquitous toddler. Significantly all 4 cases so far recorded were 2-year-olds: none showed signs of atropine poisoning.

The patient we describe shows clearly the slow absorption and long duration of action of diphenoxylate hydrochloride, in that the first apnoeic attack developed 10 hours after ingestion and the second one 7 hours later. Henderson and Psaila’s patient had ingested only 6 tablets, but the diagnosis was not suspected until 10 hours later. Similar lapses in time between ingestion and symptoms occurred in the cases described by Harries and Rossiter and by Riley. All 3 children were more seriously poisoned than our patient in whom the diagnosis was suspected much earlier. Though the number of tablets actually swallowed was approximate only, and the gastric aspirate contained no visible tablets, it seems likely that prompt gastric lavage played some part in our patient’s survival and recovery.

The antitussive action of diphenoxylate hydrochloride may have been partly responsible for the pneumonia which developed in Henderson and Psaila’s patient, but the cardinal sign of poisoning in all the cases described was respiratory depression with apnoea. Our patient had two apnoic attacks only, but the 3 others described were much more seriously affected, e.g. Harries and Rossiter’s patient died of irreversible CNS damage after cardiac and respiratory arrests with hypothermia, Riley’s patient was not fully conscious for 3 days.

*G. D. Searle & Co.
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