Platelet phenolsulphotransferase activity and 'abdominal migraine'

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SUMMARY Low platelet phenolsulphotransferase activity has been reported in adult patients with dietary sensitive migraine. Platelet activity of this enzyme was therefore measured in children having 'abdominal migraine' with probable dietary trigger and in controls. No significant difference was found in activity between the two groups. There was no significant correlation between platelet phenolsulphotransferase activity and age.

'Abdominal migraine' in children is characterised by recurrent central abdominal pain, and is typically associated with pallor and with gastrointestinal symptoms such as anorexia, nausea, or vomiting.1 Despite the term being clearly self contradictory, as migraine comes from the Latin 'hemicrania' meaning '(pain in) the half of the head', it has become hallowed by usage, and reflects the suggestion that there is a relationship with common or classical migraine. Indeed, a positive family history of cranial migraine is often taken as a diagnostic criterion and, although not conclusive, evidence strongly suggests that there is a relationship between 'abdominal migraine' and common or classical migraine.1 As there is, as yet, no diagnostic laboratory test for migraine, diagnosis is purely clinical, and it remains uncertain whether common pathophysiological mechanisms underlie the two conditions.

In many cases it is thought that attacks of 'abdominal migraine' can be provoked by articles of diet, with the list of foods implicated being very similar to those suggested as being linked to dietary cranial migraine. In particular citrus fruit, cheese, and chocolate are often cited as provoking agents.2 3 Adult migraine patients who believe that dietary factors can provoke their attacks have significantly lower mean platelet phenolsulphotransferase P and phenolsulphotransferase M activity than patients with migraine not caused by diet or controls.4 5 It therefore seemed of interest to measure platelet phenolsulphotransferase activities in a group of children suffering from 'abdominal migraine' with probable dietary trigger.

Patients and methods

Platelet suspensions in sucrose were prepared from blood collected from a group of patients attending Ealing Hospital as outpatients (n=21; 10 boys, 11 girls) and from a group of controls (n=13; six boys, seven girls). The major criteria used for making a diagnosis of 'abdominal migraine' were recurrent central abdominal pain (more than three attacks in not less than three months) accompanied by pallor, nausea or vomiting, or both, and a positive family history of migraine. At the time of study patients and controls were outside an attack and drug free. The mean (SD) age of patient group was 8-8 (2-5) years and of the control group 7-9 (3-2) years with a range from 3-13 years. Platelet samples were also collected from a group of adult controls (n=16) with a mean (SD) age of 36-6 (13-2) years with a range from 22 to 60 years.

Phenolsulphotransferase activity was assayed using phenol (final concentration 10 μmol/l) and tyramine (final concentration 130 μmol/l) as substrates for phenolsulphotransferase P and phenolsulphotransferase M respectively.6 Platelet samples, prepared as described previously,4 were used as enzyme source.

Results

No significant difference was found in either platelet
Epidemiology of rheumatic heart disease

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SUMMARY We compared the incidence of rheumatic heart disease in elementary schoolchildren from low and high socioeconomic groups; children from one of the schools were rescreened 10 years later. The results showed that the incidence of rheumatic heart disease was significantly higher in low socioeconomic group but it is gradually declining.

Phenolsulphotransferase and migraine

A similar biochemical deficiency might exist in children suffering from 'abdominal migraine' with probably dietary trigger. The results of this study, however, fail to show any such common biochemical deficiency. Even so, interpretation of these negative results must be made with caution because of the relatively small numbers of patients investigated. With this reservation, we would state that, at least, no further evidence has been educed pointing to a common aetiology of so called abdominal migraine and the more firmly established 'cranial' variety.

We thank the Ministry of Agriculture, Fisheries and Food who defrayed the salary of CG. Mrs B Davies, of the department of haematology, Ealing Hospital, kindly prepared the platelet samples.

References


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Table

<table>
<thead>
<tr>
<th>Specific activity (nmol/mg protein/10 min)</th>
<th>Phenolsulphotransferase P</th>
<th>Phenolsulphotransferase M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients (n=21)</td>
<td>0.037 (0.006)</td>
<td>0.382 (0.025)</td>
</tr>
<tr>
<td>Controls (n=13)</td>
<td>0.031 (0.007)</td>
<td>0.298 (0.043)</td>
</tr>
</tbody>
</table>

phenolsulphotransferase P or phenolsulphotransferase M activity between patients and controls (table). No significant correlation was found between platelet phenolsulphotransferase activity and age (r=0.09).

Discussion

The condition to which the clinical label of 'abdominal migraine' has been applied does have certain characteristic features in common with 'cranial' migraine in terms of family history, symptomatology, and triggering factors. The fundamental difference between the two conditions is in the location of the perceived pain and, as discussed earlier, this fact renders the label etymologically unsound. The justification for its continued use would be some firm physicochemical confirmation that a relationship does exist between 'abdominal migraine' in childhood and common or classical migraine in later life. The lack, to date, however, of any diagnostic laboratory test for migraine makes this confirmation difficult.

The finding that adult patients with migraine caused by diet have lower mean platelet phenolsulphotransferase activity than those with migraine not caused by diet or controls suggested the possibility that a similar biochemical deficiency might exist in children suffering from 'abdominal migraine' with probably dietary trigger. The results of this study, however, fail to show any such common biochemical deficiency. Even so, interpretation of these negative results must be made with caution because of the relatively small numbers of patients investigated. With this reservation, we would state that, at least, no further evidence has been educed pointing to a common aetiology of so called abdominal migraine and the more firmly established 'cranial' variety.

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