Elemental diet in acute Crohn's disease

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SUMMARY  Fifteen patients with acute Crohn's disease aged between 6 and 20 years were given an elemental diet. One patient received it by nasogastric tube and another would not tolerate it. All 14 patients have been reviewed for periods of between 1 and 3 years. An elemental diet appears to be a safe and effective method of inducing a remission in acute Crohn's disease.

Failure to gain weight and short stature are difficult to correct in children with Crohn's disease. Management of acute Crohn's disease may include corticosteroid therapy,1 sulphasalazine,1-3 or surgery.4-6 Corticosteroids may cause stunting of growth or vertebral collapse. Although surgery often results in a growth spurt, recurrence of active disease is common.7 Nutritional treatment in the form of total parenteral nutrition8-10 or special diets,11-12 or a combination of both,13 14 has been effective in a few children. We have studied 14 young patients with Crohn's disease treated with an elemental diet which was given initially to induce a remission and later as a nutritional supplement.

Patients and investigations

The 15 patients were aged between 6 and 20 years and 10 of them, including the oldest, were prepubertal (Table). Six of 11 patients younger than 18 years were below the 3rd centile for weight and 2 were below the 3rd centile for height. The diagnosis was suspected clinically and confirmed radiologically in each. The disease was confined to the small bowel in 10 of the patients and in 5 both the small and large bowel were affected. Patients with small bowel disease had no abnormality detected by barium enema studies. In 5 the diagnosis was confirmed by biopsy, 3 by rectal and 2 by colonoscopy which showed granuloma formation, and in a further 2 by

Table  Clinical details of patients treated with an elemental diet

<table>
<thead>
<tr>
<th>Case</th>
<th>Gender</th>
<th>Age (years)</th>
<th>Extent</th>
<th>Principal clinical symptoms</th>
<th>ESR (mm/1st hour)</th>
<th>Hb (g/dl)</th>
<th>Albumin (g/l)</th>
<th>Prepubertal treatment</th>
<th>Other treatment</th>
<th>Follow-up</th>
<th>Time to relapse or length of follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 M</td>
<td>F</td>
<td>6-8</td>
<td>SB and LB</td>
<td>Abdominal pain and diarrhoea</td>
<td>55</td>
<td>10-9 29</td>
<td>+</td>
<td>None</td>
<td></td>
<td>Asymptomatic</td>
<td>9 months</td>
</tr>
<tr>
<td>2 M</td>
<td>F</td>
<td>7-8</td>
<td>SB</td>
<td>Abdominal pain and diarrhoea</td>
<td>38</td>
<td>9-4 25</td>
<td>+</td>
<td>None</td>
<td></td>
<td>Asymptomatic</td>
<td>3 years</td>
</tr>
<tr>
<td>3 M</td>
<td>F</td>
<td>8-9</td>
<td>SB</td>
<td>Low height and weight</td>
<td>51</td>
<td>11-4 26</td>
<td>+</td>
<td>None</td>
<td></td>
<td>Asymptomatic</td>
<td>6 months</td>
</tr>
<tr>
<td>4 M</td>
<td>F</td>
<td>10-2</td>
<td>SB</td>
<td>Abdominal pain and diarrhoea</td>
<td>45</td>
<td>12-1 33</td>
<td>+</td>
<td>Corticosteroids</td>
<td></td>
<td>Lost to follow-up</td>
<td></td>
</tr>
<tr>
<td>5 F</td>
<td>F</td>
<td>12-8</td>
<td>SB and LB</td>
<td>Abdominal pain and diarrhoea</td>
<td>26</td>
<td>11-0 38</td>
<td>+</td>
<td>Corticosteroids</td>
<td></td>
<td>Asymptomatic</td>
<td>1 year</td>
</tr>
<tr>
<td>6 F</td>
<td>F</td>
<td>13-2</td>
<td>SB</td>
<td>Subacute obstruction</td>
<td>54</td>
<td>10-0 28</td>
<td>-</td>
<td>None</td>
<td>Sulphasalazine</td>
<td>Resection</td>
<td>15 months</td>
</tr>
<tr>
<td>7 F</td>
<td>F</td>
<td>13-3</td>
<td>SB and LB</td>
<td>Abdominal pain and diarrhoea</td>
<td>35</td>
<td>10-7 40</td>
<td>+</td>
<td>None</td>
<td>Corticosteroids</td>
<td>Asymptomatic</td>
<td>1 year</td>
</tr>
<tr>
<td>8 M</td>
<td>F</td>
<td>14-4</td>
<td>SB</td>
<td>Abdominal pain and diarrhoea</td>
<td>25</td>
<td>10-5 43</td>
<td>-</td>
<td>None</td>
<td>Corticosteroids</td>
<td>Asymptomatic</td>
<td>14 months</td>
</tr>
<tr>
<td>9 F</td>
<td>F</td>
<td>15-2</td>
<td>SB</td>
<td>Abdominal pain and diarrhoea</td>
<td>63</td>
<td>10-9 35</td>
<td>+</td>
<td>None</td>
<td>Corticosteroids</td>
<td>Asymptomatic</td>
<td>1 year</td>
</tr>
<tr>
<td>10 M</td>
<td>M</td>
<td>16-1</td>
<td>SB and LB</td>
<td>Diarrhoea</td>
<td>41</td>
<td>9-3 31</td>
<td>+</td>
<td>Corticosteroids</td>
<td>Azathioprine</td>
<td>Asymptomatic</td>
<td>18 months</td>
</tr>
<tr>
<td>11 M</td>
<td>M</td>
<td>16-4</td>
<td>SB</td>
<td>Abdominal pain and diarrhoea</td>
<td>51</td>
<td>10-9 41</td>
<td>-</td>
<td>None</td>
<td>Corticosteroids</td>
<td>Asymptomatic</td>
<td>15 months</td>
</tr>
<tr>
<td>12 M</td>
<td>M</td>
<td>17-0</td>
<td>SB and LB</td>
<td>Abdominal pain and diarrhoea</td>
<td>45</td>
<td>11-3 32</td>
<td>-</td>
<td>None</td>
<td>Corticosteroids</td>
<td>Asymptomatic</td>
<td>9 months</td>
</tr>
<tr>
<td>13 M</td>
<td>M</td>
<td>18-0</td>
<td>SB</td>
<td>Subacute obstruction</td>
<td>15</td>
<td>12-7 27</td>
<td>-</td>
<td>Levamisole</td>
<td></td>
<td>Resection</td>
<td>9 months</td>
</tr>
<tr>
<td>14 M</td>
<td>M</td>
<td>18-4</td>
<td>SB</td>
<td>Abdominal pain and diarrhoea</td>
<td>52</td>
<td>11-3 45</td>
<td>-</td>
<td>Unable to tolerate diet</td>
<td></td>
<td>Resection</td>
<td>6 months</td>
</tr>
<tr>
<td>15 M</td>
<td>M</td>
<td>20-7</td>
<td>SB</td>
<td>Delayed puberty</td>
<td>20</td>
<td>11-5 27</td>
<td>+</td>
<td>None</td>
<td></td>
<td>Asymptomatic</td>
<td>3 years</td>
</tr>
</tbody>
</table>

SB = small bowel, LB = large bowel, ESR = erythrocyte sedimentation rate, Hb = haemoglobin.
histology obtained at laparotomy. The red cell sedimentation rate was greater than 25 mm in the 1st hour in 14 of the patients, 11 had serum albumin concentrations less than 35 g/l, and 12 had haemoglobin levels less than 11·5 g/dl.

At least once a week patients were reviewed and blood taken for the estimation of red cell sedimentation rate, haemoglobin, and serum protein levels. Height and weight were recorded. Patients have been followed up for periods varying from 1 to 3 years except for one patient who failed to attend after 3 months. Three have subsequently needed intestinal resection and the remainder have had remissions lasting between 6 and 36 weeks.

**Treatment**

All food was stopped for 4 weeks and replaced by an elemental diet (Vivonex, Eaton Laboratories). The strength was increased from one-third of the recommended full strength during a period of 3 days. This introduction period was prolonged if the patient complained of bloating. The child received between 40 and 50 kcal/kg with a total 8–12 g of nitrogen a day. Specific food groups were reintroduced at weekly intervals to test for an allergen at the end of the treatment period. Fruit and vegetables were given for the first week and finally eggs and milk in the sixth week. During this period the elemental diet was the main source of nutrition. Some of the patients remained on the diet as a nutritional supplement for a longer period and received mineral supplements.

**Other treatment**

Five of the patients had received corticosteroids. Corticosteroids which failed to induce a remission in the same dosage were continued with the elemental diet in 3 patients. Another patient has a vertebral collapse as a side effect of treatment with corticosteroids before the elemental diet was started.

One patient received long-term levamisole which was used in an attempt to prolong remission; one was receiving azathioprine, and another was receiving corticosteroids, sulphasalazine, and azathioprine.

**Results**

Thirteen patients were able to tolerate the elemental diet orally, one received it by nasogastric tube and the other received corticosteroids as an alternative form of treatment. An improvement in symptoms occurred in all patients between 1 and 2 weeks after treatment started. This was accompanied by a fall in the red cell sedimentation rate, and a rise in the serum albumin and haemoglobin levels (Fig. 1).

Before the diet started all the patients showed poor growth. After 3 months of treatment 6 of the patients had crossed centiles for weight, 3 had crossed centiles for height, and the rest had not changed their weight centile (Fig. 2).

One patient with delayed puberty and short stature at age 19 years had Crohn's disease confined to the small gut. Treatment with corticosteroids and nutritional supplements had no effect. An elemental diet produced a striking increase in both height and weight (Fig. 3). Two of the other 3 patients (Cases 5 and 7), for whom corticosteroids failed to induce a remission, showed an improvement in height.

Two other patients (Cases 4 and 10) receiving the elemental diet were also given prednisolone (20 mg/day) but the diet was probably the reason that the prednisolone could be reduced quickly to 10 mg on alternate days within 6 weeks.

We were unable to identify any specific food group that induced symptoms during the reintroduction of foods.

**Discussion**

The elemental diet appeared to play a major role in inducing a clinical remission in the 14 patients with Crohn's disease. The acute symptoms were controlled, growth improved, and puberty occurred. The reason for these changes is unknown. The elemental diet may provide nutrition in patients with

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Fig. 1 Changes in various indices of disease activity of patients with Crohn's disease treated with an elemental diet.
malnutrition due to a poor intake of food as a result of anorexia and excessive protein loss from inflamed intestinal mucosa. Total parenteral nutrition either alone or in combination with oral feeding results in a ‘growth spurt’ in children with Crohn’s disease.\textsuperscript{8–11} Similarly adults with Crohn’s disease gain weight if given an elemental diet for a period up to a year.\textsuperscript{16} Four children with Crohn’s disease had a significant height and weight gain when receiving continuous elemental diet as the only form of treatment.\textsuperscript{17}

Another explanation for the effect of the elemental diet is that it contains no whole protein and therefore no allergens and may remove potential allergens from crossing the ulcerated bowel mucosa. A change in the bowel microflora may be associated. The elemental diet may also act as a medical by-pass as it reduces both gastric and pancreatic secretions\textsuperscript{18} and reduces faecal bulk as it contains no fibre. The increase in serum albumin observed in our patients may be attributed to the improvement in the nutritional status but could also be due to bowel rest. The recent findings of reduced gastrointestinal protein loss after an elemental diet lends support to this idea.\textsuperscript{19}

The unpalatability of the diet is one of its main

![Graphs showing weight and height plotted on Tanner's tables with arrows indicating the start of treatment with an elemental diet.](http://adc.bmj.com)
dissadvantages but most of our patients were able to tolerate it with encouragement of nursing and dietetic staff. The patients found the diet more acceptable if the symptoms improved. The diet was introduced gradually to avoid abdominal pain and bloating which can be caused by the hyperosmolar solution. Patients who experienced these symptoms were introduced to the diet more gradually.

Four patients were also treated with corticosteroids. The 2 receiving corticosteroids before the diet started had stunting of growth and one of them had collapsed vertebrae. One other patient still requires prednisolone 10 mg a day as maintenance treatment. The two patients who started receiving corticosteroids with the elemental diet were given prednisolone 20 mg/day which was reduced to 10 mg on alternate days by the end of the 6 weeks. The diet may reduce the corticosteroid dose and duration of treatment and avoid stunting of growth.

These preliminary results although uncontrolled suggest that an elemental diet given orally is a simple, safe method of inducing a remission of Crohn's disease resulting in an alleviation of symptoms, an improved nutritional status, and a significant gain in height and weight.

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References

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