Leading article

Diet in the management of Crohn’s disease

There is an ebb and flow in the tides of medical opinion concerning the treatment of diseases of unknown aetiology, especially those with a prolonged or variable course. The role of diet in the management of Crohn’s disease illustrates this well. Burrill B Crohn in his notable description of the disease that commonly bears his name in this country (but not in the United States), stated in 1932 that ‘medical treatment is purely palliative and supportive. In general, the proper approach to complete care is by surgical resection of the diseased segment’. Sir Arthur Hurst said in 1937: ‘the only satisfactory treatment is operative’.2

In the following decades, however, greater emphasis was placed on dietary management. Almy in Cecil and Loeb (1959) recommends ‘a high calorie and protein diet, low in seasoning and cold fluids. Roughage should be reduced’.3 Avery Jones suggests that ‘the inflammation of Crohn’s disease . . . responds well to a period of rest and good food . . . A low residue diet is important if the bowel is narrowed’.4 Donaldson puts it more pithily: ‘although many patients and a few physicians pay compulsive attention to diet there is no evidence that what the patient eats in any way affects the symptoms, or course of Crohn’s disease with one notable exception – those patients with narrowing of the gut lumen . . . should avoid all foods that contain cellulose’.5 Paradoxically the new 1500 page British textbook of gastroenterology does not mention dietary treatment of Crohn’s disease.6

In recent years the climate has changed. Appreciation of the high recurrence rate after surgery (20–50% in 10 years), the realisation that the disease may involve much more extensive areas of the gastrointestinal tract than is apparent radiologically and the failure of extensive immunological studies to identify a specific process or pathophysiological mechanism, has lead to further enquiry into the role of diet in the aetiology and management of the disease. Interest at present centres on elemental diets, elimination diets with identification of specific food intolerance and high fibre low refined sugar diets. Epidemiological studies have stimulated enquiries into the possible role of cornflakes,7 sugar, smoking, oral contraceptives, or an infective agent in the initiation or perpetuation of the disease process. None of the studies appear to stand up to further critical evaluation.8–10

The very high incidence of nutritional deficiencies in patients with Crohn’s disease which can be global, with weight loss, wasting and anaemia and specific, with deficiencies of iron, folic acid, B12 and protein, has lead to enhanced interest in correcting these problems. Their aetiology is clearly multifactorial: decreased intake because of anorexia and pain, malabsorption due to surgical resection, thickened diseased bowel and bacterial
overgrowth, exudation of blood and protein due to inflammation, hypercatabolism from infection and fever, all are important. New technical developments allow patients to be fed enterally, or parenterally – at high cost and with substantial side effects, especially in inexperienced hands. Enteral diets especially provide a challenging model for understanding and studying the pathophysiological processes perpetuating the disease. Initial uncontrolled studies\(^{11-15}\) suggesting the value of elemental, or liquid diets in the treatment of acute Crohn’s disease with fistulae, growth retardation and gross malnutrition has been substantiated by the first small, but controlled study of an elemental diet against prednisolone in the treatment of acute Crohn’s disease in patients sufficiently ill to need admission to hospital.\(^{16}\) This study showed that both treatments were equally effective. The interest of these observations is whether the improvement in the activity of the disease process was entirely due to improved nutritional state, or whether the improvement was due to removal of some perpetuating agent from the intestinal environment. Was the improvement due simply to bowel rest, to removal of an allergenic component, to decreased gastrointestinal protein loss, to a change of the bowel flora or to a ‘medical bypass’?\(^{17}\) These results await further confirmation, but should provide an important model for further study of the pathophysiological mechanisms in the absence of extraneous factors, such as steroids. The interest in nutritional factors has lead to a number of observations. Heaton et al\(^{18}\) studied the effect of a diet with increased fibre and low in refined sugar on the natural history of Crohn’s disease and found that 32 patients treated with the diet for 52 months had significantly fewer and shorter admissions to hospital than 32 matched control patients. Five of the controls and one diet-treated patient needed an operation. Inpatient stay was 111 in the treated, compared with 533 days in the control patients. Unfortunately historical controls were used in this study, which was not randomised; this seriously confounds interpretation of the results, though it has received wide attention. The preliminary results of the St Mark’s prospective multicentre double blind trial though not yet published, are said to show no striking difference between patients treated with the Heaton diet compared with controls. (Personal communication).

The study reported in this issue on page 989 by Levenstein et al is therefore interesting. It appears that in Italy a low residue diet is normally more often prescribed than would be the custom in this country. The authors prospectively compared the effect of a low residue diet with a normal Italian diet in 70 patients with non-stenosing Crohn’s disease over 29 months. They found no difference in the outcome and recommend that patients should eat more balanced, appetising food and not an unnecessarily restricted menu, which may diminish the intake of vitamins. It is noteworthy that a substantial number of patients from both groups eliminated one, or more foods, because of presumed exacerbation of symptoms. We are not given details whether any particular foods commonly provoked symptoms. It is hoped the authors will continue their studies.

Steatorrhoea after surgical resection, bile salt deficiency, diseased bowel and bacterial overgrowth may all contribute to the diarrhoea of Crohn’s disease. Andersson and coworkers showed that a moderate decrease of fat intake from 100–40 g daily may provide improvements in the symptoms
and often also in the nitrogen and electrolyte balance. Care needs to be taken, however, that the lower dietary fat does not lead to an unacceptable low calorie intake.

Hunter and coworkers have stimulated considerable interest in their preliminary findings that some patients with non-specific diarrhoea and Crohn's disease may symptomatically improve by exclusion of specific foods from their diet. The offending foods are most frequently wheat, corn, dairy products, citrus fruits, tea, and coffee. Aided by the media and women's magazines, these studies have stimulated enormous public interest, so that now gastroenterologists are constantly asked by their patients whether they should go onto an elimination diet and whether a specific food intolerance is the cause of their disease. The difficulties of initiating exclusion diets and assessing intolerance to specific foods should not be underestimated by the patient, or by the clinician. Ultimately double blind challenges as well as controlled trials, are required. At present the place of exclusion diets in the management of Crohn's disease is not known. Lactose intolerance may occur in about 10% of normal Europeans, but considerably more often in other populations. Kirschner et al found that 34% of children, or adolescents with Crohn's disease had hypolactasia. Minor histological abnormalities of the jejunal mucosa are frequent in Crohn's disease and this may lead to hypolactasia. Such observations should stimulate the assessment of a low lactose diet. The hypolactasia should be specifically diagnosed, however, because prolonged low lactose diet can lead to inadequate intake of calcium which is particularly important in children and adolescents.

Patients with gastrointestinal conditions often feel that their disease is caused by or needs, a specific diet. It is very difficult to be sure which of the present ideas will ultimately stand the test of time. Further clinical investigation of the different diets should be valuable in the greatest unsolved challenge of gastroenterology - the aetiology and treatment of Crohn's disease.

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References