Nutrition, eating and gastrointestinal conditions in adolescence

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Adolescence is the period in development when puberty occurs and peak growth velocity is achieved (mean: girls 12 years, boys 14 years). Optimal nutrition is therefore particularly important at this time. Certain gastrointestinal (GI) diseases, some affecting nutrition and growth (eg Crohn’s disease), may present during this period.

Attitudes to eating

As teenagers develop greater freedom and independence, they are increasingly likely to take more meals away from home and decide for themselves what they want to eat. School exams, peer pressures, relationship or sexuality issues become part of their life. It is at this stage that meals may start to be missed, especially breakfast. Several studies have shown that breakfast improves the intellectual and physical performance of schoolchildren. As young people begin to choose their own food, their diet may gradually deteriorate in nutritional quality and consist of more ‘junk food’, high in saturated fat and sugar. Snack and convenience foods are particularly low in iron, calcium, folate and fibre.

Nutritional education is important and is part of health education and the national curriculum. The ‘principles of good eating’ are vital (see Table 1) and have a role in preventing adult disease.

Reduction of obesity is now one of the key targets of The health of the nation. Currently, 13% of men and 16% of women in the UK are obese, the percentages having doubled since 1980. Children, too, are becoming heavier for their height, and the nation as a whole, including children, takes less physical exercise than a decade ago.

On the other hand, there are teenagers who feel they need to lose weight. Sometimes a degree of weight reduction can be recommended and is appropriate, but some individuals diet unnecessarily. Some dieting regimens can be both restrictive and nutritionally deficient. The degree to which the media and the fashion industry portray the ‘ideal body’ and their influence on young people is debatable. In extreme cases, the psychiatric conditions anorexia nervosa and bulimia nervosa can develop. Perfectionism and negative self-evaluation are particularly important risk factors for both eating disorders. Parental obesity, early menarche and parental psychiatric disorder are more associated with bulimia nervosa than anorexia nervosa.

Inflammatory bowel disease

The incidence of Crohn’s disease is increasing. Cosgrove et al found that the incidence of Crohn’s disease in South Glamorgan more than doubled over a 10-year period, although the incidence of ulcerative colitis (UC) remained the same. Recent prospective data from the British Paediatric Surveillance Unit

Table 1. Principles of good eating.

- Enjoy your food
- Eat a variety of different foods
- Eat the right amount to be a healthy weight
- Eat plenty of foods rich in starch and fibre
- Don’t eat too much fat
- Don’t eat sugary foods too often
- Look after the vitamins and minerals in your food
Table 2. Presenting symptoms for inflammatory bowel disease.

| Presenting symptom       | %   |
|--------------------------|-----|
| Weight loss              | 48* |
| Lethargy                 | 23* |
| Anorexia                 | 17* |
| Pain                     | 71  |
| Diarrhoea                | 64  |
| Bleeding:                |     |
| ulcerative colitis       | 85  |
| Crohn's disease          | 24  |
| Joint problems           | 8   |
| Growth failure           | 3   |
| Primary amenorrhoea      | 1   |
| Liver involvement        | 1   |
| Erythema nodosum         | 1   |
| Acute appendicitis       | 1   |

* significantly more cases in Crohn's disease.

Crohn's disease, can be vague (poor growth velocity, general ill health), sometimes without GI symptoms, and this can make diagnosis difficult. Presenting symptoms are shown in Table 2. Increased erythrocyte sedimentation rate, C-reactive protein, platelet count, stool alpha-1-antitrypsin, and low haemoglobin and albumin may suggest ongoing inflammation and are useful screening tests. Perinuclear antineutrophil cytoplasmic antibodies are associated more with UC than with Crohn's disease.

Patients with suspected IBD require endoscopy and biopsy to confirm the diagnosis and extent and severity of disease. Barium meal is needed to visualise the small bowel in Crohn's disease. The technetium-labelled white-cell scan, which is non-invasive, was previously thought to be sensitive (90%) and specific, and was recommended as a first-line investigation. However, more recent evidence suggests that it can give false negative and false positive results, and is therefore not a reliable screening test (S Murphy; personal communication).

Treatment depends on the type and extent of the disease. Distal colitis can be treated with mesalazine and predofoam enema. Severe UC requires oral steroids. Small bowel Crohn's disease responds well to elemental feed (Fig 1); if that fails, steroid-sparing drugs may be needed, for example, azathioprine or newer treatments such as anti-tumour necrosis factor alpha. Treatments used for IBD are shown in Table 3.

Coeliac disease

Coeliac disease (CD) is a lifelong intestinal intolerance to dietary gliadin, causing a 'gluten-sensitive enteropathy' which results in malabsorption. Previous studies have quoted a UK incidence of one per 1,000, but it is now thought that the incidence is increasing. This is probably due to an increase in the number of cases being diagnosed later in life, in the teenage years and beyond. Catassi et al. screened 3,351 students aged 11–15 years and found one subclinical case per 300 students. The number of cases discovered was described as 'the tip of the iceberg'. Clinicians should be aware of the wide variation in presenting symptoms. Children presenting under two years of age are more likely to have classical GI symptoms, but they may also be constipated and have delayed psychomotor development. Later features range from anorexia, abdominal pain and anaemia to delayed puberty, infertility, epilepsy (intracerebral calcification), depression, arthritis, dental enamel hypoplasia and recurrent mouth ulcers. Diagnosis has been aided by the use of serological tests (Table 4).

The gold standard for diagnosis still remains small bowel biopsy (Fig 2). It should be performed if anti-endomysial antibodies (AEA) are positive or there are strong clinical grounds for suspecting CD, and is best done by endoscopy. Diagnostic histological changes are villous atrophy, crypt hyperplasia, thickened mucosa and increased numbers of inflammatory cells in the lamina propria.

For an adolescent or any child over the age of two diagnosed with CD the revised European Society of Paediatric

survey of the incidence of inflammatory bowel disease (IBD) found 3.9–4.8 cases per 100,000 per year amongst children below the age of 16, with Crohn's disease being twice as common as UC.

IBD particularly affects adolescents, the mean age at diagnosis being 11.7 years, with a peak at 13 years. Symptoms, particularly in the case of
Gastroenterology and Nutrition (ESP-CAN) 1990 guidelines state that no challenge is necessary if there is complete clinical remission after one typical biopsy and antibodies return to normal. Regular follow-up for this lifelong disease is important. At the appropriate time, patients should be made aware of the long-term increased risk of intestinal lymphoma (twice that of the general population) and possible other malignancy, and that risks are reduced with a strict gluten-free diet. This may be difficult to follow for individuals with few symptoms, so yearly review—possibly with measurement of antibody levels and discussion with a dietician on new gluten-free products available—will re-emphasise the importance of diet. Good dietary compliance also decreases the risk of osteoporosis later in life.

Table 3. Treatment options for inflammatory bowel disease.

Elemental diet:
- induces remission rates equivalent to steroid treatment, no side effects
- taken orally, by nasogastric tube or via gastrostomy
- six weeks initial treatment—possibly exclusive dietary intake
- mechanism of action—bowel resting, decreased antigenic load, nutritional effect, trophic amino acids, modification of gut flora, intestinal permeability, faecal pH
- for relapses, may be used in combination with diet

Steroids:
- hydrocortisone or oral prednisolone, starting with 60 mg/m² and weaned to alternate-day dose depending on severity
- predfoam enema for disease distal to splenic flexure

Aminosalicylates:
- used to maintain remission
- sulphasalazine is a combination of 5-aminosalicylic acid and sulphapyridine
- sulphapyridine acts only as a carrier to the colonic site of action
- newer mesalazine and olsalazine avoid the sulphonamide related side effects
- monitor for blood dyscrasia

Immunosuppression:
- azathioprine
- cyclosporin
- infliximab—monoclonal antibody against tumour necrosis factor-alpha

Others:
- metronidazole, may be followed by lactobacillus, as a method of altering bowel flora
- oral budesonide

Psychological support:
- counselling if necessary, support groups, liaising with schools, home tuition in extreme cases

There is a genetic association with HLA phenotypes DR3-X and DR5-7. When a new diagnosis is made, first-degree relatives should be screened using serological tests, as 10% of them will have CD. A clear association has also been shown between diabetes mellitus and CD: 2–4% of diabetics will have CD, and testing of antigliadin antibody and AEA should form part of initial assessment soon after diagnosis. In most cases, CD develops soon after the diagnosis of diabetes. Children with Down’s syndrome also have a higher incidence of CD.

Peptic ulcer disease

Although relatively unusual (one per 2,500), peptic ulcer disease may occur for the first time during adolescence. In most cases, this will be a primary diagnosis and not secondary to drugs or other conditions. Thirty per cent of cases present with haematemesis or night pain, but some have persistent epigastric or periumbilical pain and melaena. There is a positive family history in up to 50% of cases; its presence is therefore a strong indicator for investigation.

Most cases are associated with Helicobacter pylori infection. H. pylori is a Gram-negative rod containing a urease enzyme which catalyses the conversion of urea to ammonia and bicarbonate, creating an alkaline environment. This promotes the secretion of gastrin, which stimulates acid production in the gastric mucosa.

Table 4. Serological tests as aids to the diagnosis of coeliac disease

Antigliadin antibody:
- IgA: more specific, less sensitive
- IgG: less specific, more sensitive

Anti-endomysial antibody:
- IgA: highly specific and sensitive (90–100%)

Note: Ideally IgA levels should be tested as patients with coeliac disease have a higher incidence of IgA deficiency than the general population (1 in 50 vs 1 in 500), and this may give a false negative result.

Ig = immunoglobulin
Suspected ulcer disease is an indication for upper GI endoscopy and biopsy. Active ulcers are treated with an H$_2$-antagonist or proton pump blocker for at least 6–8 weeks. If histology is positive for H. pylori, patients will need, in addition, triple therapy with omeprazole, metronidazole and clarithromycin for one week. A recent study of 16 children with duodenal ulcers showed ulcers healed in all of them after eradication of H. pylori. All the children remained asymptomatic for a median of 37 months, with none requiring subsequent admission to hospital.

Positive serology only indicates previous contact with H. pylori rather than active disease. Histology is diagnostic. Labelled carbon urea breath test, if available, is ideally suited for monitoring eradication following a course of treatment. Studies have shown a link between H. pylori and poor socio-economic factors, especially overcrowding.

H. pylori infection remains an area of controversy. Many normal children show serological evidence of H. pylori, and no studies have shown convincing evidence of its association with recurrent abdominal pain, weight loss or failure to thrive in infancy.

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