NHD
THE DIETITIANS' MAGAZINE

eArticle with CPD

SPONSORED BY

NUTRICIA Advanced Medical Nutrition

NHDmag.com Volume 4.07- April 10th 2014

CROHN'S AND COLITIS: CLINICAL PRESENTATION AND MEDICAL MANAGEMENT

Arash Assadsangabi
Specialist Registrar in Gastroenterology
Royal Hallamshire Hospital
Sheffield Teaching Hospitals NHS Foundation Trust

Dr Mark McAlindon
Consultant Gastroenterologist
Royal Hallamshire Hospital
Sheffield Teaching Hospitals NHS Foundation Trust

For authors' details please see next page.

Inflammatory bowel disease (IBD) is an idiopathic chronic condition which encompasses two distinct disease categories, namely ulcerative colitis and Crohn's disease (CD). In ulcerative colitis (UC) the mucosal inflammation starts at the rectum and could spread proximally but always in a continuous fashion. It only affects the colon but could sometimes cause a backwash ileitis. On the other hand, CD can involve any part of the gastrointestinal lumen from mouth to anus. It generally follows a discontinuous pattern.

Northern Europe, the UK and North America have the highest incidence and prevalence of IBD. Low incidence areas include southern Europe, Asia and most developing countries, although the rate of the disease is on the rise on these regions (1).

Current widely accepted pathogenesis of the disease postulates that IBD results from an inappropriate response to an innocuous antigen in a defective mucosal immune system. Once inflammation begins, the primary difference between patients with IBD and unaffected persons lies in an impaired ability to down-regulate mucosal inflammation.

IBD is a multi-factorial disease with various aetiologic risk factors being linked to its development. A positive family history is the largest independent risk factor for the disease (2). There is strong evidence of genetic factors attributed to the high concordance rate of IBD in studies on identical twins (3).

A higher accumulation of IBD in urban areas compared with rural communities has been shown in several large epidemiological studies that may reflect the effect of improving hygiene as a risk factor (2). The traditional low incidence of IBD in developing countries, which is now on the rise, also suggests the possible socioeconomic changes such as sanitation, industrialisation and diet as risk factors (4). Excessive sanitation is thought to interfere with the normal functional maturation of the mucosal immune system that requires exposure to various environmental antigens for its normal development and induction of immune tolerance in early stages of intestinal maturity. This in turn results in inappropriate immune responses when exposed to these antigens later in life.

Cigarette smoking aggravates the course of CD; on the other hand it is associated with less frequent flares of UC (5). An association between certain diets such as high polyunsaturated fats and high carbohydrate diet and the increased risk of IBD has been shown in several studies (6,7). Breastfeeding helps with intestinal immune system maturity and probably confers immunity to IBD (8).

ULCERATIVE COLITIS

UC is a chronic inflammatory process of the colonic mucosa. It is a clinical diagnosis, confirmed by other ancillary findings from endoscopic and histological examinations. Acute UC typically presents with gradual onset of bloody diarrhoea, pus or mucus passing, urgency and abdominal cramps during bowel movements. The severity of symptoms correlates with the extent of disease. When the disease extends beyond the rectum, blood is usually mixed with stool. It is worth mentioning that non-IBD causes of colitis and enteritis including bacterial, parasitic, viral, inflammatory, toxic, vasculitic and malignant should be excluded prior to confirmation of diagnosis.

CD treatment is usually follows a sequential 'step-up' approach, in which less aggressive and less toxic treatments are first initiated, followed by more potent medications or surgery if the initial medical therapy fails.

Several criteria have been described for better objective assessment of the UC severity. Truelove-Witts criteria is one of the most widely used methods dividing the disease to mild, moderate or severe.

A variety of drug therapies is available for induction of treatment during acute flare of UC. Current approved treatment options include 5-aminosalicylic acid (5-ASA), steroids, ciclosporin A, tacrolimus, infliximab and surgery. On the other hand, certain medications such as 5-ASA, E coli Nissle 1917/ VSL#3, azathioprine/mercaptopurine, infliximab (not currently approved by the NICE) are used to maintain UC remission.

Generally speaking, the choice of treatment depends on several factors including location, severity, comorbidities and degree of responsiveness to initial medical therapies as well as the patient's choice.

Arash is a senior registrar in gastroenterology currently based at the Royal Hallamshire Hospital. His main area of research interest is proteomics in IBD. He is currently undertaking a joint laboratory/ clinical research on the subject attached with the University of Sheffield.

Since being appointed as a consultant in Sheffield in 1998, Mark has led endoscopy and nutrition services and developed capsule endoscopy. He is active in research in these areas, has published original research papers and reviews and lectures nationally and internally on capsule endoscopy.

CROHN'S DISEASE

CD is a transmural inflammatory disease of the gastrointestinal mucosa that can affect the entire gastrointestinal tract, but most frequently involves the distal small intestine and proximal colon. At diagnosis, the ileocecal region is involved in about 47 percent of cases, followed by the colon in about 20 percent and the small intestine alone in about 30 percent. The stomach and mouth are rarely affected. The oesophagus is also very rarely involved (9). It can cause complications such as strictures, abscesses, sinus tracts, fistulas or adhesions. These features may also contribute to bowel obstruction. The inflammatory process usually evolves toward one of two pattern of disease: a fibrostenotic-obstructing pattern or a penetrating-fistulatous pattern. The behaviour and anatomical location of the disease can change over time (10).

The clinical presentation is largely dependent on disease location and can include prolonged diarrhoea with abdominal pain, low-grade fever, weight loss, generalised fatigability, clinical signs of bowel obstruction, as well as passage of blood, pus and/or mucus.

The diagnosis is made on the basis of history and physical examination, supplemented with objective findings from endoscopic, radiological, laboratory and histological studies. Multiple scoring systems incorporating the patient's history, physical examination findings, and laboratory data have been developed to objectively assess disease activity in adults with CD. The Crohn's Disease Activity Index (CDAI) is one such scoring system that is widely used in research. Another commonly used criterion is the Harvey-Bradshaw Index (HBI) which has more applicability in the clinical ground due to its easy scoring system.

CD treatment is usually follows a sequential 'step-up' approach, in which less aggressive and less toxic treatments are first initiated, followed by more potent medications or surgery if the initial medical therapy fails. Induction of treatment usually includes 5-ASA, azathioprine/ mercaptopurine, steroids, infliximab/adalimumab or surgery depending on the location, severity, disease behaviour (fistulating versus obstructing) and previous drug responsiveness whilst taking in to account the patient's preference. Azathioprine/mercaptopurine, infliximab/adalimumab or methotrexate on the other hand can be used to help keeping the disease in remission. There is no good evidence to support any role of probiotics in the maintenance of Crohn's disease.

ROLE OF NUTRITIONAL THERAPY IN IBD

Malnutrition is a common occurrence in IBD; hence validated tools such as Malnutrition Universal Screening Tool (MUST) should be used in clinical practice to guide objective assessment of these patients (13,14). Nutritional deficits in calcium, vitamin D, other fat soluble vitamins, zinc, iron and vitamin B12 status (Post terminal ileal resection) is relatively common especially during the disease activity (14).

In certain situations, such as short bowel syndrome or peri-operative patients with low BMI or significant weight loss, macronutrients support in the form of total parenteral nutrition (TPN) may be indicated (15). There is no good evidence to support the use of TPN and bowel rest as the main or adjunct induction therapy in IBD (16, 17).

Therapeutic liquid feeding is not indicated in the treatment of UC (14, 18). On the other hand, exclusive enteral nutrition (EEN) can be used as an alternative therapy to corticosteroids for

For **fast, effective** management of Crohn's disease that's **ready** when they are...

...Elemental 028 Extra Liquid

Elemental 028 Extra Liquid is the quick, effective[1,2] and long lasting[3] solution to Crohn's disease episodes and flare ups.

Supplied in ready-to-drink cartons, Elemental 028 Extra Liquid is shown to induce remission in 2 weeks[1,2], and may maintain remission for up to 2 years[3]. Elemental 028 Extra Liquid is a convenient way for your patients to manage their Crohn's effectively, having a minimal effect on lifestyle.

With a range of flavours and options to add variety to their elemental diet, could it be what your Crohn's patients have been waiting for?

For a sample pack of Elemental 028 Extra Liquid, just contact your Nutricia Representative or Sales Support on 01225 711969.

1. Riordan AM, et al. Treatment of active Crohn's disease by exclusion diet: East Anglian multicentre controlled trial. Lancet 1993; 342:1131-1134.
2. Teahon K, et al. The effect of elemental diet on intestinal permeability and inflammation in Crohn's disease. Gastroenterology. 1991; 101(1):84-9.
3. Verma S, et al. Oral nutritional supplementation is effective in the maintenance of remission in Crohn's disease. Dig Liver Dis 2000; 32(9):769-774.

NUTRICIA
Advanced Medical Nutrition

Nutricia Ltd. White Horse Business Park, Newmarket Avenue, Trowbridge, Wiltshire, BA14 0XQ. T: 01225 751098 www.elemental028.com

Patients with UC and CD have an increased risk of developing malignancies including colon cancer in patients with UC & CD and small bowel carcinoma in patients with Crohn's enteritis.

treating active CD although EEN has shown to be less effective than corticosteroids in the adult cohort. This could be attributed to tolerability in this group of patients (14). There is no difference in efficacy between elemental and polymeric diets as an induction treatment for active CD (19). There is also little evidence to support the use of liquid feeds as maintenance therapy for CD (20).

EXTRAINTESTINAL MANIFESTATIONS AND COMPLICATIONS

Up to about one third of patients with CD and UC will develop extraintestinal disease manifestations or complications (10). Extra-intestinal manifestations include dermatological (erythema nodusum, pyoderma gangrenosum), rheumatological (peripheral arthritis, ankylosing spondylitis), ocular (conjunctivitis, uveitis), hepatobiliary (hepatic steatosis, primary sclerosing cholangitis), urological (calcium oxalate stones), metabolic bone disorders (osteoporosis and osteonecrosis), thromboembolic disorders (venous and arterial thrombosis) and cardiopulmonary (endocarditis, interstitial lung disease).

Patients with UC and CD have an increased risk of developing malignancies including colon cancer in patients with UC & CD and small bowel carcinoma in patients with Crohn's enteritis (11). Index screening colonoscopy is currently advised for all patients about 10 years after the initial diagnosis followed by risk stratification according to disease activity, presence of complications, family history of colorectal cancer and histological findings into low, intermediate and high risk groups; follow up screening colonoscopy will then be in five, three or 10 years respectively as per current British Society of Gastroenterology (BSG) guideline (12).

References:
1 Loftus EV Jr. Clinical epidemiology of inflammatory bowel disease: incidence, prevalence, and environmental influences. Gastroenterology 2004; 126: 1504–17
2 Daniel C Baumgart, Simon R Carding. Inflammatory bowel disease: cause and immunobiology. Lancet 2007; 369: 1627–40.
3 Tysk C, Lindberg E, Jarnerot G, Floderus-Myrhed B. Ulcerative colitis and Crohn's disease in an unselected population of monozygotic and dizygotic twins. A study of heritability and the influence of smoking. Gut 1988; 29: 990–96.
4 Desai HG, Gupte PA. Increasing incidence of Crohn's disease in India: is it related to improved sanitation? Indian J Gastroenterol 2005; 24: 23–24.
5 Cosnes J. Tobacco and IBD: relevance in the understanding of disease mechanisms and clinical practice. Best Pract Res Clin Gastroenterol 2004; 18: 481–96.
6 Riordan AM, Ruxton CH, Hunter JO. A review of associations between Crohn's disease and consumption of sugars. Eur J Clin Nutr 1998; 52: 229–38.
7 Geerling BJ, Dagnelie PC, Badart-Smook A, Russel MG, Stockbrugger RW, Brummer RJ. Diet as a risk factor for the development of ulcerative colitis. Am J Gastroenterol 2000; 95: 1008–13.
8 Schack-Nielsen L, Michaelsen KF. Breast feeding and future health. Curr Opin Clin Nutr Metab Care 2006; 9: 289–96.
9 Louis E, Collard A, Oger AF, Degroote E, Aboul Nasr El Yafi FA, Belaiche J. Behaviour of Crohn's disease according to the Vienna classification: changing pattern over the course of the disease. Gut 2001; 49: 777–82.
10 Daniel C Baumgart, William J Sandborn. Inflammatory bowel disease: clinical aspects and established and evolving therapies. Lancet 2007; 369: 1641–57.
11 Bernstein CN, Blanchard JF, Kliewer E, Wajda A. Cancer risk in patients with inflammatory bowel disease: a population-based study. Cancer 2001; 91: 854–62.
12 Stuart R Cairns, John H Scholefield, Robert J Steele, Malcolm G Dunlop, Huw J W Thomas et al. Guidelines for colorectal cancer screening and surveillance in moderate and high risk groups (update from 2002). Gut 2010; 59: 666-690.
13 Stratton RJ, Hackston A, Longmore D, et al. Malnutrition in hospital outpatients and inpatients: prevalence, concurrent validity and ease of use of the 'malnutrition universal screening tool' ('MUST') for adults. Br J Nutr 2004;92:799-808.
14 Craig Mowat, Andrew Cole, Al Windsor, Tariq Ahmad, Ian Arnott et al. Guidelines for the management of inflammatory bowel disease in adults. Gut 2011;60:571-607.
15 National Collaborating Centre for Acute Care. Nutrition Support in Adults Oral Nutrition Support, Enteral Tube Feeding and Parenteral Nutrition. London: National Collaborating Centre for Acute Care, 2006. http://www.rcseng.ac.uk.
16 Greenberg GR, Fleming CR, Jeejeebhoy KN, et al. Controlled trial of bowel rest and nutritional support in the management of Crohn's disease. Gut 1988;29:1309-15.
17 McIntyre PB, Powell-Tuck J, Wood SR, et al. Controlled trial of bowel rest in the treatment of severe acute colitis. Gut 1986;27:481-5.
18 Lochs H, Dejong C, Hammarqvist F, et al. ESPEN guidelines on enteral nutrition: gastroenterology. Clin Nutr 2006;25:260-74.
19 Zachos M, Tondeur M, Griffiths AM. Enteral nutritional therapy for induction of remission in Crohn's disease. Cochrane Database Syst Rev 2007;(1):CD000542.
20 Akobeng AK, Thomas AG. Enteral nutrition for maintenance of remission in Crohn's disease. Cochrane Database Syst Rev 2007;(3):CD005984.

THE DIETITIANS' MAGAZINE

eArticle with CPD

Volume 4.07- April 10th 2014

Questions relating to: *Crohn's and colitis: clinical presentation and medical management*
Type your answers below and then **print for your records.** Alternatively print and complete answers by hand.

Q.1	Describe the two disease categories of inflammatory bowel disease (IBD).
A	
Q.2	Outline the pathogenesis of IBD.
A	
Q.3	What are the aetiological risk factors for IBD?
A	
Q.4	What are some of the reasons for a lower incidence of IBD in developing countries?
A	
Q.5	What is ulcerative colitis (UC) and describe its symptoms.
A	
Q.6	Describe the main complications and contributing risks of Crohn's disease (CD).
A	
Q.7	What is the treatment for CD?
A	
Q.8	What nutritional therapy is used in the treatment of IBD?
A	
Q.9	Outline the follow-up treatment for patients with UC and CD.
A	

Please type additional notes here . . .