CROHN'S DISEASE AND DIETARY TREATMENT: WHERE ARE WE NOW?

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Since my last article for *NHD* on dietary treatment of Crohn's Disease (CD)[1], we have had an update in the dietary guidelines from the BDA and research has moved on at a pace in the area of the gut microbiome. This article discusses current treatment of adult CD with dietary therapy.

CD is a chronic inflammatory autoimmune condition, exhibiting transmural skip lesions, affecting any area of the gastrointestinal tract. The aetiology of CD is poorly understood; there is a genetic component, an inappropriate immune response to microbiota and environmental causes including diet. Patients have significant morbidity, 70-80%[2] will have surgery and this has considerable effect on nutritional status, particularly if resections are substantial.

Access to a dietitian is vital in the treatment of patients with CD, as malnutrition is common. Along with protein energy malnutrition, there has been reported inadequate intake of micronutrients[3]. The latest inflammatory bowel disease (IBD) audit[4] highlighted that, whilst dietetic access has improved, the importance that standards escalate the level of care in areas where change is challenging was emphasised. The audit showed 23% of patients have no access to specialist dietetic input, only 67% of services have 0.5 WTE dietitian and only 61% of services reported a 90% level of nutritional risk screening during inpatient admission. No less than 100% of assessment with a validated tool (e.g. MUST) is the standard; the authors report that services should implement a business case to improve dietetic accessibility. However, BMI has been suggested to be falsely reassuring and grip strength has been proposed as a better predictor of lean mass, presence of sarcopenia and osteoporosis risk.[5]

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EXCLUSIVE ENTERAL/LIQUID DIETS

Enteral or oral liquid diets have been provided as treatment to reduce inflammation and induce remission. Theories suggest that the mechanism is avoidance of presumed dietary antigens or alteration of the bowel microbiota profile. A major paper in CD was published in 2007[6] and suggested that, whilst evidence was clear that enteral diets induce remission, corticosteroids were more effective in this regard. Evidence was unclear that steroids were more effective; some of the studies reported concomitant additional drug therapy and exclusion of these studies showed a lack of superiority of steroids over diet, but exclusion reduced the power of the research.

A recent meta-analysis suggested that enteral nutrition is more effective in inducing and maintaining remission in combination with infliximab (a medical anti TNF treatment) compared with infliximab treatment alone.[7] Liquid diets remain a secondary consideration in adults, where palatability and adherence are still sited as major drawbacks in

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treatment. Liquid diet therapy can maintain nitrogen balance and reduce the risk of development of osteoporosis in the long term in adult females when compared with steroids[8] and has also been found to maintain remission.[9] The liquid diet ideally needs to be followed for a minimum of four to six weeks to allow for mucosal healing, although benefits have been shown from 10 days onwards[10] and reintroduction needs to be commenced once inflammation has resolved.

FOOD ELIMINATION/REINTRODUCTION DIETS

LOFFLEX (or LOw Fibre, Fat Limited EXclusion diet,) researched by a team at Addenbrooks Hospital[11], is recommended by guidelines.[10] Liquid diet can be continued as nutrition support during food reintroduction when needed, particularly during the first restricted stage. The LOFFLEX diet is used for two weeks and then foods are reintroduced to tolerance. The initial phase can be increased to four weeks if needed.[10] Standards state that when compared with elimination diets, there is limited weak evidence that the LOFFLEX diet is of similar efficacy to elimination diets and patients are more compliant and prefer LOFFLEX as a reintroduction diet option.

THE SPECIFIC CARBOHYDRATE DIET

This diet eliminates disaccharides, oligosaccharides, polysaccharides and fruits and vegetables containing a higher ratio of amylopectin to amylose. Some patients are following this diet as a self-care approach. Individual case reports have been published and a case series report has recently been published of 50 IBD patients (36 with CD) choosing this diet.[12] Currently, there is no randomised control trial (RCT) level evidence for its efficacy as a treatment. The average case in this publication followed the diet for three years, the longest 13 years, but the concern is that this diet has not been investigated for adequate nutrient provision. A reintroduction protocol is provided to reintroduce some foods, how many foods people reintroduce again remains unclear. It allows fructose and, therefore, fructose malabsorption is not considered as a possibility of symptom causation in CD.

Patient intake of dietary inulin type fructan has been studied.[13] Fructans are fermentable carbohydrates. Patients with active CD have lower intakes of fructans and oligo-fructans than patients with CD in remission, but again, the consequences of low fructan intake is unknown. Patients appear to be manipulating their diet to self-treat perceived symptoms from food. Research is urgently needed to investigate how effective a treatment the specific carbohydrate diet is, what nutritional limitations it provides and the long-term consequences, with a diet potentially low in prebiotics in a disease where the microbiome is implicated and higher intakes of calcium are required.

PROBIOTICS, PREBIOTICS AND SYNBIOTICS

Bacteria is associated in the pathogenicity of CD, so the theory suggests that altering gut microbiota will be beneficial. Some patients with IBD use probiotics to try to manage their disease, but less than half of users discuss this use with their healthcare provider.[14] There is still lack of evidence that these products have any effects in induction[15] or maintaining remission[16] in CD, therefore, probiotics are not supported by the new IBD standards.[10] Caution needs to be used with prebiotics and synbiotics as these products may be poorly tolerated; the patient should be informed that their use may increase risks of bloating, diarrhoea and gas, due to them containing fermentable carbohydrates.[10] Research showed that FOS inclusion in active CD did not show clinical benefit and 19% of participants in the CD arm withdrew due to worsening symptoms.[17]

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FUNCTIONAL SYMPTOM THERAPY

A major change in CD testing is use of the faecal calprotectin test, which delineates between active disease and remission. Some patients do go on to experience functional gastrointestinal symptoms during disease remission. It is important to review disease status for patients referred for dietary treatment of functional symptoms, or IBS, as self-reported symptoms during both stages may be similar.

LOW FODMAP DIET

Evidence has shown that reduction of fermentable carbohydrates (FODMAPs) may be successful in treating functional symptoms such as gas, bloating and pain during remission. A study[18] has shown that lowering the dietary fermentable carbohydrates resulted in symptomatic improvement in half of participants with IBD, this paper had a higher number of participating CD patients. In the absence of strictures/inflammation, the Low FODMAP diet could be considered as treatment.

HIGH FIBRE DIETS

High fibre diets, including wheat bran consumption, to maintain remission in CD is included as a result of searches revealing poor quality studies with small numbers of participants. One single blinded paper[19] that had very few participants reported a wide difference between the treatment and control group; this is likely to be a consequence of very low participant numbers and not strong clinical effectiveness. This is an area that needs a good understanding of the effects of foods on the digestive system at each stage of the patient's clinical condition. Inclusion of a high fibre diet to maintain remission is potentially dangerous in the presence of stricturing and wheat bran consumption may result in functional symptoms. Current guidelines[10] state that there is weak limited evidence to suggest that high fibre diets are effective in maintaining remission in CD.

CONCLUSION

Dietary self-care may be considered by patients as a consequence of poor access to dietetic services and is completely understandable when patients desire a solution to ameliorate symptoms from food. If this is the case, then the solution is to increase dietetic access to increase availability to evidenced-based care. Dietary research is challenging due to the complex nature of diet, but dietary treatments are an important adjunct in treatment. What is clear is that where randomised control trials exist, the evidence is usually limited and weak. Further research is urgently needed, to ensure that we have evidenced treatments in our dietetic care.

Stricturing disease

Reducing fibre or utilising low residue diets to reduce symptoms or prevent bowel obstructions in CD remains an area that has little evidence base, or clear demarcation as to what constitutes low fibre or low residue.[10] It is important to differentiate between strictures due to fibrotic scarring and those due to inflammation. Inflammatory strictures can resolve with liquid diets and medical treatment and may require a reduction in fibre in the short term. Symptom resolution with reducing fibre is reported anecdotally by patients; this may be due to global reduction of fermentable carbohydrates and, without research, it remains an area of controversy. It is advisable to follow guidelines or protocols with stricturing CD and UK guidelines suggest that fibre is contraindicated in stricturing disease, due to the risk of obstruction.[10]