

Irritable Bowel Syndrome (IBS) and Diet

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SUMMARY

The role of diet in patients with Irritable Bowel Syndrome still remains empirical, as the underlying pathophysiological disturbances of this disorder are not fully elucidated and randomised controlled trials are difficult to perform. The mainstay for dietary manipulation still remains the exclusion diets. The new, in this field is that the “forbidden” foods are fewer than in the past and the dietary advice given to the patients has a more scientific basis, although not evidence-based but based on the best evidence we have.

Fat, milk, fiber, carbohydrates and certain other substances such as coffee, alcohol, hot spices may play a greater or lesser role, in aggravating symptoms. The insistence of doctors in their personal communication with patients as well as new, well designed, trials will help towards a more rational approach to this problem.

Key words: Irritable bowel syndrome, food intolerance constipation predominant IBS

INTRODUCTION

Irritable Bowel Syndrome (IBS) is a functional disorder of the lower gastrointestinal tract. It is also accompanied by other non-colonic gastrointestinal symptoms¹⁻⁵ or extraintestinal manifestations.⁶⁻⁸ There exist now hard criteria that clearly separate this syndrome from other, mainly functional, but also organic disorders with a good discriminatory value.⁹⁻¹²

The main problem of this functional disorder is that

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the pathogenesis is multifactorial and not entirely elucidated. This fact poses difficulties in the therapeutic approach, one aspect of which is dietary management.

1. Dietary manipulation

1.1 Where do we stand?

Although reassurance and psychological support are key factors for the emotional catharsis of patients, many patients believe their symptoms to be caused, or at least aggravated, by certain foods and consequently restrict their diets unnecessarily. To date only a small number of randomised controlled trials exist in the literature most published studies being observational. No firm conclusions can therefore be extracted.¹³

Some experts propose the stratification of IBS patients based on symptoms, paying attention to dietary manipulation for those with mild or moderate disease, who are in the majority, keeping different therapeutic measures for those with severe or intractable disease.^{14,15}

1.1.1. The role of fat

It is generally accepted that fat in the diet seriously contributes to the generation of symptoms.¹⁶ It delays transit, induces bile secretion and is the stimulus for the release of several hormones, the most important of which is CCK.¹⁷ An increased responsiveness of the gut to this hormone has been observed.¹⁸ This pathophysiological basis makes the advice of avoidance of fatty foods reasonable, as no other, better evidence exists.

1.1.2 The role of fiber

Diet rich in fiber are frequently recommended, especially for those IBS patients with constipation predominant form. The most common advice is the consumption of fiber – mainly as bran – in an increasing stepwise fashion, in divided doses (12-16 gr/day upper limit, 3-4 times/day). Unfortunately, 15-25% of patients complain of aggravation of their symptoms, particularly bloating. Psyl-

lium, an hydrophilic colloid substance, or carbophilic substances can be used as an alternative to bran products, as they produce less gas by preserving stool liquidity, these compounds can, theoretically, be helpful in both constipation-predominant and diarrhoea-predominant patients. Again, however, there are arguments against this policy, as constipated IBS patients have been found to consume similar quantities of fiber to already healthy controls.^{18,19} This is one more lesson on the divergence between pathophysiology and clinical practice. Indeed, fiber reduces colonic or oroanal transit time,²⁰ without altering the rectosigmoid motility.²¹

In another clinical trial, bran is reported to be no better than placebo in relieving symptoms.²² In two other cross over randomised controlled trials using fiber comparison,^{21,22} the symptoms of the patients neither improved nor worsened.²³ It is now questionable which patients really benefit from a high-fiber diet: those with IBS-constipation predominant syndrome or those with “simple” constipation?

1.1.3 The role of lactose

It is crucial to discriminate between lactase deficient patients and lactose intolerant patients.²⁴ Although this distinction can unlabel a patient as suffering from IBS, and a mono-elimination diet be sufficient, i.e. lactose avoidance, this distinction is not always easy to make.²⁵

It has been observed that some IBS patients with confirmed lactase sufficiency feel much better whenever this substance is eliminated from their diet. Their main symptoms, such as flatulence, passing of excessive gas, pain and loose stools, if eradicated, get very much better. These patients are characterised as lactose intolerant patients.^{14,26} Food intolerance is analysed later in this text.

1.1.3 Other dietary substances

It has been shown that certain substances may cause or aggravate symptoms mainly in the diarrhoea-prone IBS patients. These include caffeine, excess alcohol, sorbitol, hot spices, and food rich in carbohydrates.^{14,16} Beans, onions, celery, bananas, Brussel sprouts and prunes are considered extremely flatulogenic and should be avoided, especially if a patient's history includes aggravation of his/her symptoms whenever these foods are consumed.²⁷

2. The perspectives

2.1 What must we hope?

As mentioned above, the pathophysiology of IBS is

multifactorial. As was believed in past, the motility disturbances are no longer believed to be the sole factor in IBS pathogenesis. Sensory abnormalities, either peripheral or central, and the recently introduced conception of food intolerance, indicate a greater complexity to the mechanisms.²⁸ Research in these fields will add knowledge that will lead to therapeutic manipulation for this disorder. The multiplicity of pathophysiology mechanisms may be reflected by the different clinical forms of IBS in discrete groups of patients. The predominant disturbance will probably have a different approach in the future.

2.2 Food intolerance

Food intolerance is a broad term of a yet unknown allergic gut reaction to certain stimuli. However, various studies have shown that several food components may aggravate symptoms through the above mechanism.¹⁵ Unfortunately, these studies have major limitations, which reduces the power of their findings.²⁹

In a meta-analysis of seven studies, the range of the positive response to an elimination diet fluctuated between 15 and 58 per cent.²⁶ Milk, wheat and eggs were responsible for the majority of symptom exacerbation. Also, in the same study, foods rich in amines and salicylate were responsible for symptom aggravation. Intolerance related to substances such as fructose and sorbitol, sugars found in fruit, soft drinks and candy.³⁰ It is interesting, that fructose malabsorption or high luminal fructose concentration has been associated with decreased plasma tryptophan and consequently serotonin levels. This has been linked to some sort of depression.³⁰ Additionally, a recent study demonstrated a specific group of IBS patients, similar to the “classic” coeliac patients, and their improvement after a gluten free diet.³¹

CONCLUSION

No hard evidence exists for IBS patients concerning their diet. It seems from observational studies that **fat** (greasy food), **lactose**, **coffee**, **hot spices**, certain **carbohydrates**, and **flatulogenic food** may play a negative central role in the dietary manipulation of patients with Irritable Bowel Syndrome. Nowadays **fiber** consumption remains a controversial issue. It seems to be beneficial to the constipated patient rather than to the constipation-prone IBS patient. More epidemiologic as well as carefully planned randomised studies, are needed, at least for those societies in which the poverty is an old nightmare. The magic carpet of the under(?) -developed countries is not yet known.

REFERENCES

1. Constantini M, Sturniolo GC, Zaninotto G, et al. Altered esophageal threshold in irritable bowel syndrome. *Dig Dis Sci* 1993; 38:206-209.
2. Ayres RCS, Robertson DAF, Naylor K, et al. Stress and oesophageal motility in normal subjects and patients with irritable bowel syndrome. *Gut* 1989; 30:1540-1542.
3. Whorwell PJ, McCallum M, Creed FH, Roberts CT. Non-colonic features of irritable bowel syndrome. *Gut* 1986; 27:37-42.
4. Kamath PS, Gaisano HY, Philips SF, et al. Abnormal gallbladder motility in irritable bowel syndrome: Evidence for target-organ defect. *Am J Physiol* 1991; 260:G815.
5. Evans PR, Bennett EJ, Young-Tae B, et al. Jejunal sensorimotor dysfunction in irritable bowel syndrome: clinical and psychosocial features. *Gastroenterology* 1996; 110:393-396.
6. Prior A, Wilson K, Whorwell PJ, Faragher EB. Irritable bowel syndrome in gynecological clinic. *Dig Dis Sci* 1989; 34:1820-1822.
7. Teruzzi V, Maggatti F, Quadri G, et al. Bladder dysfunction and irritable bowel syndrome [Letter]. *JAMA* 1992; 87:1231.
8. Vealea D, Kavanagh G, Fielding JF, Fitzgerald O. Primary fibromyalgia and the irritable bowel syndrome: Different expressions of a common pathogenetic process. *Br J Rheumatol* 1991; 30:220-222.
9. Drossmann DA, Richter JE, Talley NJ, et al. The functional gastrointestinal disorders: diagnosis, pathophysiology, and treatment. Mc Lean, VA: Degnon Associates: 1994; 1-370
10. Manning AP, Thompson WG, Heaton KW, Morris AF. Towards a positive diagnosis of the irritable bowel syndrome. *Br Med J* 1978; 2:653-654.
11. Thomson WG, Hungin AP, Neri M, et al. The management of irritable bowel syndrome: a European, primary and secondary care collaboration. *Eur J Gastroenterol Hepatol* 2001; 13(8):933-939.
12. Vanner s, Glenn D, Paterson W, et al. Diagnosing irritable bowel syndrome: Predictive value of Rome Criteria. *Gastroenterology* 1997; 112 [abstract].
13. Burden S. Dietary treatment of irritable bowel syndrome: current evidence and guidelines for future practice. *J Hum Nutr Diet* 2001; 14(3):231-241.
14. Drossman DA. A biophysiological approach to irritable bowel syndrome: Improving the Physician-Patient relationship, 1997 Solvay-Pharmaceuticals, Hannover, Germany: 1-53.
15. Drossman DA. Review article: an integrated approach to the irritable bowel syndrome. *Aliment Pharmacol Therapeutics* 1999; 13(Suppl 2): 3-14.
16. Simren M, Mansson A, Langkilde AM, et al. Food related symptoms in the irritable bowel syndrome. *Digestion* 2001; 63:108-115.
17. Sjolund K, Ekman R, Lindgren S, et al. Disturbed motilin and cholecystokinin release in the irritable bowel syndrome. *Scand J Gastroenterol* 1996; 31:1110-1114.
18. Jarrett M, Heitkemper MM, Bond EF, Georges J. Comparison of food composition in women with or without functional disorder. *Gastroenterol Nurs* 1994; 16:253.
19. Effect of dietary fiber on symptoms and rectosigmoid motility in patients with irritable bowel syndrome. *Gastroenterology* 1990; 98:66-72.
20. Cann PA, Read NW, Holdsworth CD. What is the benefit of coarse wheat bran in patients with irritable bowel syndrome? *Gut* 1984; 25:168-173.
21. Cook IJ, Irvine EJ, Campbell D, et al. Effect of dietary fiber on symptoms and rectosigmoid motility in patients with irritable bowel syndrome. A controlled, cross over study. *Gastroenterology* 1990; 98:66-72.
22. Lucey MR, Clark ML, Lowndes J, et al. Is bran efficacious in irritable bowel syndrome? A double blind placebo controlled crossover study. *Gut* 1987; 28:221-225.
23. Francis CY, Whorwell PJ. Bran and irritable bowel syndrome: time for reappraisal. *Lancet* 1994; 344:39-40.
24. Bohmer CJ, Tuynman HA. The effect of a lactose-restricted diet in patients with a positive lactase tolerance test, earlier diagnosed as irritable bowel syndrome: a 5-year follow-up study. *Eur J Gastroenterol Hepatol* 2001; 13:941-944.
25. Parker TJ, Woolner JT, Prevost AT, et al. Irritable bowel syndrome: is the search of lactose intolerance justified? *Eur J Gastroenterol Hepatol* 2001; 13:219-225.
26. Niec AM, Francum B, Talley NJ. Are adverse food reactions linked to irritable bowel syndrome? *Am J Gastroenterol* 1998; 93:2184-2190.
27. Van Ness MM, Cattau EL. Flatulence: pathophysiology and treatment. *Am Fam Physician* 1985; 31:198-202.
28. Hasler WL, Owyang C. Irritable bowel syndrome, In *Textbook of Gastroenterology* Yamada T, Alpers D, Owyang C, Powell DW, Silverstein FE (eds), JB Lippincott Company, Philadelphia, 2nd ed, 1995; Ch 81, p.1832-1855.
29. Collins S, Ismail M. Irritable bowel syndrome, In *Evidence Based Gastroenterology*, Irvine EJ, Hunt RH (eds), BC Decker Inc, Hamilton, London, 2001; Ch 16:194-206.
30. Ledochowski M, Winder B, Bair H, et al. Fructose- and sorbitol- reduced diet improves mood and gastrointestinal disturbances in fructose malabsorbers. *Scand J Gastroenterol* 2000; 35:1048-1052.
31. Wahnschaffe U, Ullrich R, Riecken EO, et al. Celiac disease-like abnormalities in a subgroup of patients with irritable bowel syndrome. *Gastroenterology* 2001; 121:1329-1338.