

The role of faecal *Candida albicans* in the pathogenesis of food-intolerant irritable bowel syndrome

S.J. Middleton, A. Coley and J.O. Hunter

Department of Gastroenterology, Addenbrooke's Hospital, Cambridge CB2 2QQ, UK

Summary: *Candida albicans* was sought in stool samples from 38 patients with irritable bowel syndrome and 20 healthy controls. In only three patients with irritable bowel syndrome was *C. albicans* discovered and these patients had either recently received antibiotics or the stool sample had been delayed more than 24 hours in transit. *C. albicans* was isolated from none of the control stool samples. We conclude that *C. albicans* is not involved in the aetiology of the irritable bowel syndrome.

Introduction

The irritable bowel syndrome (IBS) is the most common gastroenterological disorder in clinical practice in Western society.^{1,2} It is now known that many patients with IBS have specific food intolerances.^{3,4} Diets have enabled many patients to control their symptoms but limited diets are expensive and socially inconvenient. *C. albicans* has been implicated in antibiotic-associated diarrhoea in elderly patients⁵ and it has been suggested that food intolerance may be a consequence of the overgrowth of *C. albicans* in the gut.⁶ This theory has received much publicity in the popular press but little scientific analysis. In this study we report the results of a search for *C. albicans* in the stools of patients with IBS proven to be caused by food intolerance and in those of normal healthy controls.

Materials and methods

Patients

The diagnosis of IBS was based upon the standard Manning criteria following exclusion of organic disease by a combination of investigations including normal stool culture, full blood count, ESR, acute phase proteins, plasma, glucose, liver function tests, urea, creatinine, electrolytes and amylase, together, where clinically indicated, with lactose tolerance test, barium enema, abdominal ultrasound, intestinal permeability and radio-labelled granulocyte scans. Food intolerance was

detected by the relief of symptoms following two weeks on a standard exclusion diet.⁷ Patients subsequently reintroduced foods to see which if any precipitated their symptoms and the intolerances thus discovered were confirmed by double blind challenge. Normal volunteers were recruited from hospital staff and patients relatives who were in the same age range as the patients studied.

Microbiological investigations

Stool samples were collected by patients using a wooden spatula and 20 ml metal container with screw top and delivered to the laboratory within 24 hours. Validation of *C. albicans* viability during transport was performed by seeding 1 g samples of *C. albicans*-negative stool with a known concentration of the organism and incubating these for 24 hours at various temperatures. The number of colony-forming units present on Sabouraud's dextrose agar was determined for each sample. A 10-fold increase in *C. albicans* counts occurred at 22°C and 37°C, but not at 4°C, which remain unchanged. On the receipt of faecal samples, 1 g of homogenized faeces was cultured on Sabouraud's medium. A colony count was performed after 48 hours of aerobic incubation. Colonies of *Candida*-like organisms were incubated in horse serum for 3 hours at 37°C, and a wet film was then examined for filamentous overgrowth or 'germ tubes', a finding specific for *C. albicans*.⁸

Results

There were no *C. albicans* colonies detected after incubating the faeces of the control group. Moderate numbers of *C. albicans* (approximately 10⁴/g

wet faeces) were grown from faecal samples taken from three IBS patients. Two of the three had received antibiotic therapy near the time of sampling and the third faecal specimen had been exposed to an extended period of transit following collection prior to incubation. On resampling these individuals there were no *C. albicans* colonies seen after 48 hours incubation.

Discussion

IBS is not a homogeneous entity but a collection of poorly understood conditions presenting with similar symptoms, namely abdominal pain and an abnormal bowel habit. It is now clear that in the United Kingdom approximately half the patients diagnosed as IBS may be benefitted by dietary manipulation, at least temporarily. The pathogenesis of such food intolerance is still unknown.⁹ However, it has been suggested that changes in the bacterial flora of the gut may be a factor involved. Other workers have suggested that overgrowth of *C. albicans* in the colonic flora is a common feature of patients suffering food intolerance.^{6,10} They have advocated treatment with so-called 'yeast-free diets' and antifungal drugs, such as nystatin and ketoconazole. As these drugs have caused harmful

side effects,^{11,12} it is important to establish whether or not *C. albicans* in the gut is a significant factor producing symptoms in these patients.

We were concerned that the delay in culturing the stools of our patients caused by transportation to the laboratory might lead to reduced *C. albicans* counts. However, the studies we have performed in which stools were spiked with the organism and then cultured after incubation for 24 hours after collection at 4°C, 22°C and 37°C showed that this did not occur.

We found *C. albicans* to be no more prolific in the faeces of people with IBS than those of our healthy controls. Our results are similar to those found on review of 168 patients who claimed that their symptoms were related to *Candida* in the gut. As in our study, no evidence was found to substantiate an association between symptoms and *C. albicans* infection.¹³ Anecdotal reports of improvement after treatment for *C. albicans* may be a consequence of simultaneous dietary measures (the 'yeast-free diet') in patients who are food intolerant.^{3,4}

Overgrowth of *C. albicans* is not the cause of food intolerance in patients with IBS and anticandidal drugs should not be used in the treatment of this condition.

References

1. Ferguson, A., Sircus, W. & Eastwood, M. Frequency of 'functional' gastrointestinal disorders. *Lancet* 1977, **ii**: 613.
2. Painter, N.S. The high fibre diet in the treatment of diverticular disease of the colon. *Postgrad Med J* 1974, **50**: 629–635.
3. Alun Jones, V., McLaughlan, P., Shorthouse, M., Workman, E. & Hunter, J.O. Food intolerance: a major factor in the pathogenesis of irritable bowel syndrome. *Lancet* 1982, **ii**: 1115–1117.
4. Nanda, R., James, R., Smith, H., Dudley, C.R.K. & Jewell, D.P. Food intolerance and the irritable bowel syndrome. *Gut* 1989, **30**: 1099–1104.
5. Danna, P.L., Urban, C., Bellin, E. & Rahal, J.J. Role of candida in pathogenesis of antibiotic-associated diarrhoea in elderly patients. *Lancet* 1991, **i**: 511–514.
6. Crook, W.G. *The Yeast Connection*, 2nd ed. Professional Books, Jackson, Tennessee, 1984.
7. Workman, E., Alun Jones, V. & Hunter, J.O. *The Allergy Diet*. Dunitz, London, 1984.
8. Douglas Sleight, J. & Timbury, M.C. *Candida*. In: *Notes on Medical Bacteriology*. Churchill Livingstone Medical Text, Edinburgh, 1981, p. 133.
9. Hunter, J.O. Food allergy – or enterometabolic disorder? *Lancet* 1991, **ii**: 495–496.
10. Galland, L. Nutrition and candidiasis. *J Orthomol Psychiat* 1985, **14**: 50–60.
11. Lewis, J.H., Zimmerman, H.J., Benson, G.D. *et al.* Hepatic injury associated with ketoconazole therapy. *Gastroenterology* 1984, **86**: 503.
12. Quinn, J.P. & Venezia, F.R. Ketoconazole and the yeast connection. *JAMA* 1986, **255**: 3250.
13. Renford, L. *et al.* Yeast connection among 100 patients with chronic fatigue. *Am J Med* 1989, **86**: 165–168.