The Essentials of Pouch Care Nursing

EDITED BY

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Dedication

To my family

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Contents

Foreword

The introduction of the ileal reservoir by Kock in the late 1960s to improve quality of life for the ileostomist was the start of a long development in intestinal surgery. Soon afterwards, restorative proctocolectomy with ileoanal reservoir was designed by Parks in order to avoid ileostomy altogether. Subsequently, the use of the colonic reservoir by Lazorthes aimed to improve bowel function in patients with low anterior resection. Thus, pouch surgery has been applied to inflammatory bowel disease, rectal cancer and familial adenomatous polyposis, becoming an important part of specialist colorectal surgery.

Pouches have much to do with quality of life, and this to a degree depends on the subjective perception of the patient. The discussion of indications, including counselling, is thus a vital element of achieving an optimum result. Following surgery, the patient requires help in dealing with normal recovery – even more so in the event of complications. Long-term follow-up and continued support after leaving hospital are therefore necessary. It is with these aspects of care that specialist nurses can help in a way that doctors cannot easily do. They have a different perspective on the patient's dilemma of choice of procedure and on the consideration of possible disadvantages. In addition, they can, by their accessibility, more readily offer continuity of care and advice.

Decision-making, preparation for surgery, the intermediate postoperative course and longer-term outcomes all come within the ambit of this excellent book. The vast amount of information available on pouches has been summarised in various chapters to enable a rapid and easy access to important issues, both surgical and

nursing. *The Essentials of Pouch Nursing* is the first book of its kind. It fills an educational gap and will be essential reading for any health-care professional with an interest in colorectal reconstructive surgery. It deals with all the diseases amenable to pouch formation, as well as with various techniques and aftercare, in a highly practical manner.

Professor R. John Nicholls
St Mark's Hospital, Harrow
July 2001

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Finally, sincere thanks go to my family and friends for their continued support and encouragement.

Dansac Limited are proud to have been invited to support Julia Williams with this very important publication.

Dansac Limited believes that as a leading manufacturer within the stoma care market, we have a responsibility to health-care professionals to help provide continuing education and support wherever appropriate.

We are pleased to have provided financial support for this book, and to have donated a copy to each Stoma Care Department within the UK.

CHAPTER 1
Medical aspects of ulcerative colitis

ALASTAIR FORBES

Ulcerative colitis is one of the two principal forms of inflammatory bowel disease and is somewhat more common than Crohn's disease. Although some aspects of this discussion apply equally to Crohn's disease, it should be assumed that ulcerative colitis is under consideration unless this is otherwise specified.

Epidemiology

Ulcerative colitis affects men and women at a similar rate and with a similar severity. There is a steady annual incidence of around 7 per 100,000 population, the highest figures being seen in regions most distant from the equator. The peak times for first presentation and for relapses are in the early spring and autumn/winter, with a relative lack of clinical events in the late spring and summer months: December is almost twice as problematic as May.

Aetiology and pathogenesis

The aetiology and pathogenesis of ulcerative colitis have a limited bearing on clinical practice, but it is hoped that the following will aid an understanding of the disease process and of the mechanisms by which therapeutic agents act. It should also help to provide answers to some of the questions asked by patients about their disease.

In ulcerative colitis, there are almost certainly both inherited factors and environmental influences acting together.

Genetics of ulcerative colitis

The risk of a sufferer's sibling, parent or child being affected lies at between 1 in 15 and 1 in 10, which is about 50 times the risk in the general population. However, in only around 1 out of 10 pairs of even identical twins do both members develop ulcerative colitis. Within families, colitis seems to occur at a younger age in successive generations.

Links with the HLA tissue-typing antigens have been sought. Certain HLA DR2 antigens are over-represented (being seen in around 40% of colitics compared with about 20% of control populations) and are also associated with a higher frequency of total colitis and need for surgery. In addition to the HLA sites on chromosome 6, there are putative genes coding for ulcerative colitis on chromosomes 2, 3, 7 and 12.

A circulating antibody to a neutrophil cytoplasmic antigen is rarely seen other than in colitis, its presence being partly genetically determined. Antibody levels do not reflect disease activity but do tend to fall slowly after colectomy.

The striking demarcation of the upper limit of ulcerative colitis (see below) may also be genetically determined via a variance in the anatomy of the marginal artery, a normally small branch of the inferior mesenteric artery. This hypothesis, however, remains controversial.

Infection as the cause of inflammatory bowel disease

Although most attention to infection as a possible cause of inflammatory bowel disease has focused on Crohn's disease, there is also evidence relevant to ulcerative colitis. The seasonal variation in clinical activity has already been referred to, and patients often link gastrointestinal infections to the onset or relapse of their colitis.

Hydrogen sulphide, a product of sulphate-reducing bacteria, is specifically implicated. Many colitis patients describe offensive flatus, and although it is for obvious reasons difficult to perform studies of flatus, data do exist. Colitis patients produce up to four times as much hydrogen sulphide as normal people. This is not only unpleasant, but also toxic, with an effect in animals similar to that of cyanide.

There may also be an immune response to normal intestinal organisms, with a significant level of circulating antibodies to a

variety of their antigens. Purified bacterial components can produce intestinal inflammation, but there is little evidence that ulcerative colitis is truly an autoimmune disease.

In animal models of inflammatory bowel disease, it is striking that bacteria are almost always involved in addition to the specific stimulus employed. Few animals spontaneously develop a disease at all like colitis, an endangered monkey, the cotton-top tamarin, being an exception. Tamarin colitis is very similar to ulcerative colitis, including the propensity for colorectal carcinoma to develop; the disease seems only to affect animals held in captivity.

Non-steroidal anti-inflammatory drugs

Non-steroidal anti-inflammatory drugs may be implicated in the causation of some cases of inflammatory bowel disease. These drugs can certainly cause colonic inflammation, watery diarrhoea and chronic blood loss. Even if not causative, they tend to exaggerate symptoms in many (if not most) patients with colitis. They may also be responsible for a high proportion of cases of acute self-limiting colitis.

Smoking and colitis

Smoking is an intriguing environmental factor in the context of inflammatory bowel disease. It definitely contributes adversely to all aspects of Crohn's disease but appears in some way protective in ulcerative colitis. Smoking is less common in ulcerative colitis patients than in healthy controls, the highest frequency of the disease being found in ex-smokers. It also influences the ileo-anal pouch, pouchitis being less common in current smokers. The reason(s) for this remain speculative, but a disruption of mucus production and of protective barrier function is probably important. Nicotine may also be helpful, and this has led to several trials of its use.

Appendicectomy

For reasons that are not clear, previous appendicectomy is protective against, or is associated with a protective factor against, ulcerative colitis. Patients with colitis are only about a twentieth as likely to have had their appendix removed as are age-matched controls.

Diet as an aetiological factor

Although diet is almost certainly important in Crohn's disease, and patients are always concerned to explore a link in ulcerative colitis, there is no clear association, other than in Japanese patients in whom the consumption of a Western-type diet is associated with the disease. Clearly, diet is not an independent risk factor here.

Clinical presentation and investigation

Ulcerative colitis is usually responsible for diarrhoea and rectal bleeding. Less often, there is weight loss, anorexia and fatigue. When there is only a short history, gastrointestinal infection will be the most probable alternative diagnosis. General examination usually contributes little; although perianal disease is more typical of Crohn's disease, some changes are seen in a minority (up to 10%) of patients with ulcerative colitis.

Oral aphthous ulcers are common in the general population but are over-represented in groups of patients with inflammatory bowel disease. They can be troublesome and may need specific treatment with topical steroids to achieve their resolution.

Investigation

At sigmoidoscopy, the rectum is almost always obviously involved in ulcerative colitis. There is a loss of the normal vascular pattern, confluent erythema and, to a greater or lesser degree, ulceration, the latter two being continuous. That is to say, the disease is present distally and extends proximally without uninvolved colon between the abnormal areas.

Investigation will typically commence with routine laboratory tests such as a full blood count and serum biochemistry. These will rarely contribute to the diagnostic process itself, except where there is uncertainty over whether the patient has atypical functional bowel disease (effectively excluded by an elevated platelet count, a low haemoglobin level or low albumin, for example). Blood tests assessing the degree of inflammation, including the C-reactive protein (CRP) level and the erythrocyte sedimentation rate, are also used. These, however, have only modest reliability.

Differential diagnosis

The differential diagnosis of ulcerative colitis includes infection, non-steroidal drug-related colitis and acute self-limiting colitis. The most likely organisms are all are fairly readily identified (or excluded) by a conventional laboratory examination of the stools. 'Pseudomembranous colitis', arising from *Clostridium difficile* infection should be sought, by culture and by examination for the cytotoxin, especially if the patient has recently been exposed to antibiotics. The patient with ulcerative colitis may also present because of a superimposed gastrointestinal infection.

In the patient presenting without acute dysenteric symptoms, the differential diagnosis includes colorectal carcinoma, ischaemic colitis and radiation enteritis.

Up to a third of patients with predominantly distal colitis may, despite a history of diarrhoea, prove to be constipated on abdominal palpation. A distinction from functional bowel disorders such as irritable bowel syndrome is usually obvious because of the presence of bleeding in colitis.

Barium enema and other imaging

The double-contrast enema using both air and barium has almost completely superseded the single-contrast examination, but the unprepared ('instant') enema is still helpful in the evaluation of acute colitis (Figure 1.1). In early or mild colitis, the only abnormality may be a granularity of the mucosa, but the changes will be continuous from the rectum upwards. The so-called 'hose-pipe' colon is now rarely seen but when present strongly supports the diagnosis of chronic ulcerative colitis.

Computed tomography scanning with computer reconstruction to generate 'virtual colonoscopy' is very impressive as a technological feat, but is not yet as sensitive as colonoscopy. In radio labelled white cell scanning, the patients' own leukocytes are labelled and returned to the circulation. These migrate to areas of inflammation and hence provide a relatively non-invasive definition of the extent of disease in colitis.

Colonoscopy

While there is certainly a place for the barium enema and the other options, none is yet as sensitive as colonoscopy, and none of the

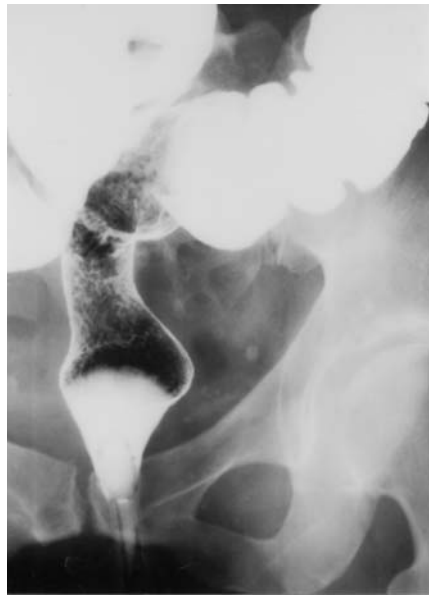

Figure 1.1 Barium enema showing superficial ulceration in the rectum but normal appearances more proximally in a patient with ulcerative colitis.

others permits biopsy samples to be obtained. At colonoscopy, the features are essentially those already described for sigmoidoscopy. The proximal limit of ulcerative colitis is often very clearly demarcated, and the small bowel is never involved (Figures 1.2 and 1.3).

There may be (post-inflammatory) polyps at sites of past inflammation, which can develop into long, interlinked lesions with mucosal bridging. Pseudopolyps, which represent islands of normal or regenerative mucosa, occur only when there is marked active inflammation.

Histology

A firm diagnosis of ulcerative colitis should not be made without histological support. The histological changes are confined to the mucosa and submucosa. There will typically be an acute inflammatory reaction with neutrophils, crypt abscesses and goblet cells depleted of mucus. With time, architectural changes develop. Irregular, short and excessively branched crypts develop. These features are more conclusive in the distinction from acute infective colitis or self-limiting colitis (see Figure 1.2).

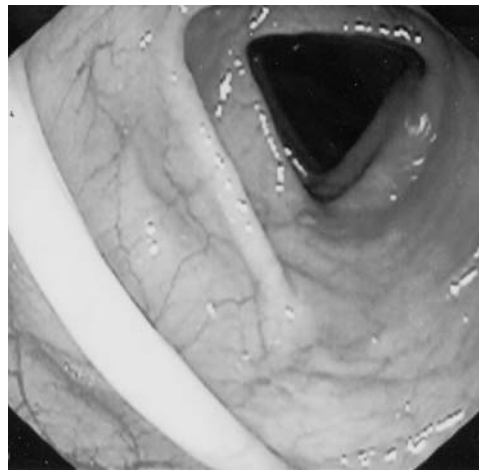

Figure 1.2 Colonoscopic appearance of normal transverse colon in a patient with left-sided colitis (see Plate 1).

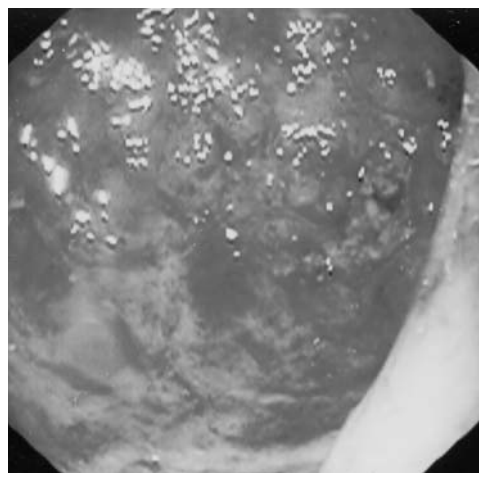

Figure 1.3 Active ulcerative colitis in sigmoid colon (same patient as above) (see Plate 2).

Clinical course and natural history of ulcerative colitis

The clinical course of colitis is not easily predicted, but the extent of colonic involvement is a guide to the future risk of colorectal carcinoma. At any one time, about 50% of patients with colitis are in full

remission, but most remain prone to intermittent relapses. Activity in any given year predicts a 70–80% risk of activity in the following year. The cumulative risk of colectomy varies from one centre to another, being partly dependent on the proportion of patients with extensive colitis seen, but it is typically around 1 in 5 at 10 years and nearer 1 in 3 on a lifetime basis.

Most patients with colitis can pursue a normal working life. The risk of relapse probably gradually diminishes with increasing age.

Frequency and significance of proximal extension of distal colitis

Ulcerative colitis is a distal disease with variable proximal extension. Because the proportion of the colon involved has a bearing on the risk of colonic carcinoma, it is used to modify the intensity of hospital follow-up. The colitis may, however, advance proximally with time. In about a quarter of those with initially limited disease, there is a progression to extensive disease by 10 years. Occasional re-evaluation is therefore appropriate.

Psychological and other clinical aspects of ulcerative colitis

It is not unusual for patients with well-established colitis to present with symptoms that sound more functional, investigations such as CRP level being normal. Perhaps as many as a fifth of all patients have irritable bowel syndrome superimposed on the colitis. This can present quite awkward management decisions.

The patient with ulcerative colitis has a chronic and often debilitating disease that can only be cured by radical surgery, which itself leaves variable long-term sequelae. The symptoms of the disease are unpleasant and are not considered ones for 'polite conversation'. It is inevitable therefore that psychological morbidity runs alongside the organic physical disease. No one now would seriously maintain that colitis is caused by psychological disease, but there can be little doubt that psychological factors can be very important, presumably contributing to the functional symptoms described above. The unpredictability of colitis is itself a major cause of anxiety and stress, especially when faecal incontinence is or has ever been a problem.

Given time, patients will often be remarkably candid about the influence of stress and emotional issues on their intestinal symptoms, appearing to find such a discussion therapeutically valuable. They may also be greatly helped by the use of low doses of constipating agents such as loperamide to help them through times of predictable stress. There does not seem to be any risk from this as long as the patient knows to discontinue the drug if true constipation develops or if abdominal discomfort occurs.

Quality of life in colitis

It is obvious that colitis has an adverse impact on the patient's quality of life, the creation and success of patient support groups, such as the National Association for Colitis and Crohn's Disease in Britain, having been partly a response to this. Many useful information leaflets, and practical aids such as the 'Can't wait' card (aiding urgent toilet access) (Figure 1.4), have been produced, in addition to counselling services and informal support. Steps have also been taken to offset discrimination in the workplace and with respect to life insurance.

Figure 1.4 'Can't wait' card.

Causes of death in ulcerative colitis

Even allowing for colorectal carcinoma, colitis has little overall effect on mortality. There is a reduced life expectancy, but at 20 years it is

still at least 90–95% that of controls, even in those with extensive colitis. The standardized mortality ratio of around 1.35 reflects an overall threefold increase in the number of colorectal carcinoma deaths and excess deaths from sclerosing cholangitis.

Medical therapy

Corticosteroids and the 5-aminosalicylate (5-ASA) drugs (sulphasalazine and its successors) still constitute the mainstay of drug therapy, azathioprine being established for resistant disease. The remitting and relapsing course of colitis, and the substantial rate of spontaneous improvement whatever therapeutic endeavours are employed, makes it crucial that any new measures should be compared with established treatments in randomized, blind, controlled trials. Previous placebo-controlled trials indicate that spontaneous improvement and an apparently full remission may reasonably be expected in around 30% and 10% of exacerbations respectively.

Steroids

Corticosteroids (usually prednisolone or hydrocortisone) provide a rapid and effective relief of symptoms in acute exacerbations, albeit not always accompanied by a full remission. There is little difference between hydrocortisone and prednisolone when equivalent doses are compared (4 mg methylprednisolone: 5 mg prednisolone: 25 mg hydrocortisone). Intravenous therapy is probably more potent than oral treatment in resistant cases.

Typical regimes for moderate to severe colitis comprise oral prednisolone 0.5–1.0 mg per kilogram body weight, with a suggested minimum of 30 mg daily. There are no good data to determine how long this dose should be maintained, and most gastroenterologists commence a fairly brisk reduction once the response begins in order to avoid unnecessary toxicity. A typical regime is 7 days at the starting dose, reducing thereafter by 5 mg per week until weaning is complete.

Steroids are ineffective in maintaining remission and cause significant long-term side-effects, so they should be withdrawn once the acute episode has settled. Unfortunately, a few patients do become

steroid dependent, fostering an interest in new, less toxic, steroids. Delayed-release oral budesonide fits this description and compares reasonably well with prednisolone in efficacy, having notably less toxicity. Budesonide enemas are therapeutically equivalent to hydrocortisone and prednisolone in acute distal colitis, but they are less readily absorbed and cause less adrenal suppression. If long-term steroid use is really necessary, this drug should perhaps now be chosen.

Aminosalicylates

Sulphasalazine has been used in the treatment of ulcerative colitis since the early 1950s, proving effective in acute colitis and in the maintenance of remission. A reduction in the annual relapse rate from approximately 70% in the untreated to around 25% in those taking the drug is typical. Up to 15% of individuals are intolerant of the sulphapyridine component of sulphasalazine, but most of its therapeutic benefit results from the 5-ASA (or mesalazine) molecule. Oral 5-ASA is ineffective because it is absorbed and metabolized too proximally; alternative formulations of 5-ASA have therefore been developed. There is also no problem with oligospermia with the newer agents.

In 1999, six 5-ASA preparations were commercially available in Europe. Asacol is 5-ASA coated with a resin, which releases the drug when the pH is above 7.0, this typically occurring in the region of the caecum and ascending colon. The resin coat of Claversal and Salofalk dissolves a little more proximally. Pentasa has microgranules of 5-ASA in a semi-permeable membrane, its 5-ASA being steadily released throughout the intestine. Olsalazine has two molecules of 5-ASA linked by a bond that is broken by the same colonic bacterial enzymes that activate sulphasalazine. A potential advantage of olsalazine over other 5-ASA preparations is limited by an osmotic diarrhoea provoked by the drug, which affects up to 10% of patients but may be minimized by taking the drug with food. Balsalazide has a bond linking 5-ASA to an inert carrier and is handled like olsalazine.

The difference between all these 5-ASA agents is relatively minor, all appearing to be superior to sulphasalazine, although olsalazine and balsalazide may have the edge in more distal disease. Around

60% of patients with moderately active colitis will respond to 5-ASA therapy alone. There are strong suggestions that regular and long-term 5-ASA use not only prolongs the relapse-free interval survival, but also helps to reduce the risk of colonic neoplasia. Patients in prolonged remission nevertheless usually seek to stop maintenance therapy – or simply stop it. Aside from the issue of cancer risk, this becomes increasingly reasonable the longer the patient is from the last relapse.

The 5-ASA drugs are very occasionally responsible for renal toxicity via an interstitial nephritis, which may lead to end-stage renal failure. It is probably an idiosyncratic, dose-independent effect and occurs with all formulations including sulphasalazine, although it is recorded more often with pH delivery systems. A periodic assessment of renal function (perhaps every 6 months) is therefore wise as early changes are probably not progressive even if they are not fully reversible. 5-ASA preparations are themselves rarely the cause of an exacerbation of the colitis, the associated worsening of the underlying disease potentially leading to confusion until the correct interpretation has been reached.

Topical 5-ASA is firmly established in the treatment of active proctitis and distal colitis, typically with a response rate in excess of 70% over 3–6 weeks. The results are at least as good as, and probably better than, those of topical steroids and oral 5-ASA. In the UK, topical steroids are, however, a great deal cheaper and will probably remain the first choice in most units. Topical steroids and 5-ASA preparations may also usefully be combined. Patients almost always prefer foams to liquid enemas, but suppositories are quite often sufficient in the most distal disease, an adequate amount being delivered by a single daily 1 g dose of 5-ASA.

Immunosuppressant therapy

Immunosuppressant drugs are valuable in refractory colitis but have limited efficacy and significant toxicity. Trials of other immunomodulatory are also underway.

Azathioprine

Azathioprine and 6-mercaptopurine inhibit the synthesis of DNA and RNA. They are effective in refractory ulcerative colitis but work