If I Had...

Ulcerative colitis

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Enter patient with peculiar shuffling gait. 1st medical student: "I bet he's got Parkinson's disease." 2nd medical student: "And I bet he's a case of myxoedema." Patient: "You thought I had Parkinson's and you're wrong. He thought it was myxoedema, and he's wrong. I thought I was passing wind and I was wrong." Shuffles off right.

That old joke used to go down well with the patrons of the music hall, I grimly reflected as I was once again forced to leave the outpatient clinic. There was no doubt about it, my irritable bowel seemed to be getting worse. Diarrhoea was interrupting my daily work more and more, especially in the mornings and after meals, but I was also inconvenienced at other times and was sometimes forced to get up at night. Nevertheless, the abdominal colic was really no more than I could expect with any bout of diarrhoea. True, I had been feeling more tired recently but I had been working particularly hard. This might have also explained the frequent crops of aphthous ulcers in my mouth. But on that particular day I was forced to admit to myself that the stool was not only watery but appeared to contain blood.

Now there was nothing for it except to have a word with my general practitioner. The rectal examination showed little apart from a trace of blood on the finger stall, but I was relieved to hear that there was no palpable carcinoma. The history extended back over some months and we both thought that a bowel infection was unlikely, but three stool specimens were sent to the laboratory to exclude infective or parasitic conditions. After all, there was a slight chance that all my troubles might be cured by a course of chemotherapy. The stool contained red and white cells but no pathogens were isolated and no ova, cysts, or parasites seen. My haemoglobin concentration was 12.0 g/dl, ESR 18 mm in the first hour, and plasma albumin concentration 35 g/l, but the white cell count and electrolytes were normal. With these findings, we could no longer ignore the possibility of organic disease of the bowel, or defer consultation with a colleague in hospital.

I asked to see a physician who has a particular interest in gastroenterology and works closely with a surgeon interested in bowel disease. Inflammatory bowel disease does not respond so well to the occasional practitioner. The crucial investigation will be the sigmoidoscopy, which I hope will be performed in the left lateral position. I think that the knee-elbow position is both undignified and unnecessary. Air insufflation will be the

most uncomfortable part and should be kept to a minimum if active inflammation is present, but I would expect the operator to introduce the scope as far as possible to see if he could get above the inflamed area and into a segment showing normal mucosa.

A rectal biopsy specimen will have to be taken and I should prefer it to be done by suction. If the suction pressure is not excessive there will be only a slight risk of haemorrhage and the specimen obtained will be fit for the histologist. I would refuse biopsy with conventional forceps, because the mucosa in ulcerative colitis is often smooth with no folds to grip and there is a risk of stripping it away longitudinally with consequent haemorrhage. After all this, the specimen is frequently too distorted for accurate diagnosis. Although there are no pathognomonic signs of ulcerative colitis on biopsy, other signs may point towards Crohn's disease and, if this diagnosis is being entertained, I should like to have all available evidence before me. I regard the differentiation of ulcerative colitis from Crohn's disease as vital. It can often be achieved by the various investigations outlined above together with barium meal and follow-through. Unfortunately, in some patients it may not be possible to separate the two conditions. The prognosis and management of the two diseases are different; I would certainly prefer to have ulcerative colitis if I had the choice.

A barium enema would be the next step but not until a few days after the rectal biopsy because of the risk of perforation. This examination is essential to determine the extent of the disease and hence the prognosis. A straight radiograph of the abdomen would be unnecessary if I was in good general condition. At this stage I would refuse to undergo colonoscopy. This unpleasant and time-consuming investigation requires equally unpleasant preparation (although the latter might be modified in the presence of active colitis). I accept that the extent of the disease can be most accurately estimated by this method and that introduction of the instrument may be helped by the shortening of the colon that occurs in advanced colitis. Nevertheless, the main reason for determining extent is to gauge prognosis and this can be done satisfactorily by conventional radiography. In fact, the natural history of ulcerative colitis has yet to be worked out using endoscopy as the criterion. I would not, however, refuse colonoscopy later if polypi or strictures needed close inspection, biopsy, or treatment.

Two types of colitis?

The aetiology of ulcerative colitis is unknown and so the disease continues to be subdivided according to its extent as judged by sigmoidoscopy and barium enema. Conventional teaching regards disease of differing extent as part of the same process and additionally relates prognosis according to the severity of an individual attack and the development of complications. Nevertheless, most patients referred to an outpatient

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clinic nowadays are suffering from distal colitis, and its management is different from that of more extensive disease.

Ulcerative proctitis is present when an upper limit to the inflammation can be seen on sigmoidoscopy. It is now generally accepted that it has a good prognosis without complications and rarely spreads. The patients are not systemically ill or toxic but a severe flare-up usually indicates spread. A recent survey¹ has calculated the following five-year probabilities: spread as far as splenic flexure (5%), spread up to hepatic flexure (3%), and need for operation (2%). These patients nearly all have an irritable bowel which causes symptoms in its own right. They often complain of bleeding and their complaints seem out of proportion to the amount of blood lost. This is probably because their frequent bowel actions produce only fresh blood and mucus, which may easily be seen in the lavatory and adds to their anxiety. Treatment must be directed towards the irritable bowel as well as the mucosal inflammation. Most patients do not require psychotropic drugs and respond to reassurance with an explanation of the underlying pathophysiology.

There is a variant of ulcerative proctitis in which it is impossible to get above the inflammation with the sigmoidoscope, although the barium enema appearances are normal. This carries a slightly worse prognosis than proctitis, with double the chance of operation at five years and more tendency to extend. This group does not show uniform appearances because in some patients the inflammation may cease at about 15 cm from the anus but the limit will not be seen sigmoidoscopically because of a sharp rectosigmoid angle, whereas in others the sigmoidoscope may be introduced up to 30 cm and the colitis is still present. Routine use of the fiberoptic sigmoidoscope, which is supposed to allow direct inspection up to the lower descending colon, may help to define the natural history. Once barium enema demonstrates disease in the sigmoid (distal colitis) the chance of further spread and operation increases.

Management

The probabilities are that I would have one of the forms of distal colitis which could easily be managed on an outpatient basis. I would attack my high-roughage diet with determination and initially add a bulking agent to the regimen. There is evidence that bouts of constipation initiate relapses and also that, in the presence of a constipated irritable bowel, sulphasalazine is not delivered in its active form to the site of inflammation. If I were anaemic and the red cell indices suggested iron deficiency, I would take iron in spite of the rumours that it might precipitate a relapse: what little hard evidence there is suggests that oral iron preparations are harmless. Similarly I would not restrict my milk intake, although I might reconsider this and even undergo a lactose tolerance test if the symptoms became intractable.

I would, however, avoid antibiotics for fear of aggravating the colitis. My first line of specific treatment would be sulphasalazine, which I would take at half dose before increasing to maximum in the hope of reducing side effects. Should I be one of the 30% who experience adverse effects with sulphasalazine, I would probably switch to cromoglycate by mouth even though its therapeutic role has not been fully assessed. At the same time I would start steroid retention enemata at night. I hope that a seven-day course would be sufficient, but I would extend it should bleeding continue. If I were one of those unfortunates with distal colitis who continue to bleed more or less continuously at a low level, I would take a weekly course of enemata every one month or so. I would not take oral corticosteroids except for an acute relapse severe enough to stop me from working. On this regimen I would expect the diarrhoea to settle, and would hope not to need regular long-term anti-diarrhoeal agents in addition. For prophylaxis, I would attempt to live up to the copywriter's dream of sulphasalazine ad infinitum, but I wonder if I, like so many of my patients, would fall by the wayside once restored to better health.

Should I develop an acute severe attack of colitis against a background of more extensive disease I would expect to be admitted to hospital. The sheet-anchor of treatment would then be corticosteroids and I would prefer to start with corticotrophin in high dosage before switching to oral prednisolone as the symptoms subsided. I would expect to come off the drug completely after 2-3 months of reducing dosage. I would not wish to take azathioprine or any other cytotoxic agent, preferring to undergo surgery if symptoms were still not controlled. In my view immunosuppressive treatment achieves less reduction of symptoms than corticosteroid treatment, and carries more chance of side effects. I would also resist the routine administration of intravenous antibiotics unless there was a proved infection to be treated.

Should I develop a toxic megacolon, I would hope for close collaboration between physician and surgeon leading to early operation. If I improved dramatically within a few hours on intensive medical treatment, I might be happy for the operation to be postponed until I was in better shape. Nevertheless, I would not pretend to myself that I was out of the wood, because in my experience the colon always seems to be irreversibly damaged leading to persistent diarrhoea at the least. There would therefore be no point of postponing elective surgery once the toxic episode had settled enough to allow panproctocolectomy with the minimum of risk.

Choice of operation

This is an emotive subject and has been well discussed in the *BMJ* recently.² My own preference would be to have a panproctocolectomy with ileostomy, but I might allow myself to be persuaded by my colleagues if their preference was for ileorectal anastomosis. As I see it, total excision of the disease means cure. Complications of ileostomy are unusual provided that the surgeon is good at his job and fashions and sites the ileostomy correctly. Ileostomy aftercare is now good and the availability of a stoma therapist would be an additional inducement for surgery. I hope that I would not continue with failing medical treatment too long so that I would need an emergency rather than an elective operation. In that case I would expect the rectum to be left in situ for link-up or removal at a later date, when there would be less chance of sexual problems resulting.

If I were twenty years younger I would ask for an ileorectal anastomosis, particularly if I were guaranteed less than four bowel actions per day. Nevertheless I doubt if any surgeon would give me such a guarantee, and I would find a greater frequency of defaecation an enormous nuisance, especially if my sphincter were to become less efficient with advancing years. I would also tolerate poorly the repeated check-ups with sigmoidoscopy and biopsy, which would probably induce a degree of cancerophobia, even though the risk is small. I would not be persuaded to have any form of reservoir ileostomy unless the operation were to be performed by a surgeon with considerable experience—and there are few of them around at the moment. The concept is tempting, but I suspect that the result would be less than ideal.

The risk of carcinoma

Although some papers in recent years have suggested that the cancer risk is somewhat less than had been suggested, the danger remains. It is negligible in distal colitis, but, once the disease has spread to involve most of the colon, the risk increases year by year. It will not become significant until 10 years have elapsed and so colectomy need only be recommended at this stage. Luckily few patients are in this category, and many will still be suffering from symptoms so that the risk of cancer may be the final inducement to submit to surgery.

The real problem occurs with the occasional patient whose disease is quiescent. If I were in this position, I would still

prefer to have a panproctocolectomy and get it over with. I would not want an ileorectal anastomosis because the rectal stump would still be a hazard. If I lived in an area where there was a pathologist who could confidently and consistently diagnose premalignant changes in the rectal biopsy specimen, then I might procrastinate. Even so, I am sure that I would find six-monthly biopsies irksome and I have listened to too many arguments about the incidence and relevance of biopsy findings to place my faith in pathology.

Conclusion

Should I be unlucky enough to develop ulcerative colitis, I would try to regard it as more of a nuisance than a disability.

Are antihistamines of value in treating "catarrhal deafness"?

Antihistamines have some value in treating catarrhal deafness as they decrease secretions that exacerbate the condition. An antihistamine combined with a decongestant such as xylometazoline hydrochloride (Otrivine-Antistin) nasal spray would probably be of greater value than antihistamines given by mouth and cause less sedation. Such a preparation should not be used for prolonged periods, however, as rebound congestion can occur. One of the more sedative antihistamines such as promethazine hydrochloride (Phenergan) might help at bedtime for the relief of nasal congestion interfering with sleep.

Can nicotine be found in the blood of non-smokers after using the cigarette-making machine to make, say, 40 cigarettes, as this would include firmly pressing down two ounces (56.7 g) of moist tobacco with the pulp of the fingers?

In 1933 Faulkner¹ reported an interesting case of nicotine poisoning by absorption through the skin. A florist, using a nicotine insecticide spray, accidentally soaked the seat of his trousers with spray, and within 15 minutes became critically ill with nicotine poisoning. He was rushed to hospital, admitted for four days, and recovered; but his clothes had been tied up in a bag and not dried. When he dressed to leave his trousers were still damp, and one hour later he was readmitted with exactly the same symptoms. Depending on pH, absorption of nicotine through the skin can be extremely rapid and efficient or it can be negligible. This was dramatically shown by Travell.² She applied nicotine solutions to the skin of cats. An amount of nicotine that had no pharmacological effect in acid solution was rapidly fatal in alkaline solution. "Green tobacco sickness," an occupational hazard of tobacco croppers in the USA, is another manifestation of cutaneous absorption of nicotine.³ It occurs when freshly cropped tobacco comes in contact with the damp sweaty clothes of the tobacco cropper. To answer the question, it is doubtful whether more than negligible amounts of nicotine would be absorbed by non-smokers who rolled cigarettes. Apart from those working in tobacco factories, it is difficult to see why non-smokers would wish to roll cigarettes. Besides, non-smokers are known to absorb nicotine by breathing air polluted by tobacco smoke,⁴ and the amounts taken in by this "passive smoking" are far greater than is likely to occur by rolling cigarettes.

¹ Faulkner, J M, *Journal of the American Medical Association*, 1933, **100**, 1664.

² Travell, J, *Annals of the New York Academy of Sciences*, 1960, **90**, 13.

³ Gehlbach, S H, *et al*, *Lancet*, 1975, **1**, 478.

⁴ Russell, M A H, and Feyerabend, C, *Lancet*, 1975, **1**, 179.

What is the treatment for atrophic vaginitis in a married woman of 68, who had a total hysterectomy 21 years ago, a mastectomy for early carcinoma eight years ago, and who suffers from angina pectoris?

Vaginal swabs should be taken at weekly intervals for about four weeks for microbiological investigation, to be quite sure there is no

The disease would probably be limited to the distal colon, so that life expectancy would be normal, and I would be able to relax even more once the first year of symptoms had passed. Severe attacks are certainly more common in the first 12 months, but they may occur at any time and I hope that I would be sensible enough to seek treatment early if I relapsed. If the inflammation spread, I would not regard life as an ileostomist as a disaster.

References

- ¹ Ritchie, J K, Powell-Tuck, J, and Lennard-Jones, J E, *Lancet*, 1978, **1**, 1140.
- ² Jones, P F, Bevan, P G, and Hawley, P R, *British Medical Journal*, 1978, **1**, 1459.

treatable infection. Then the importance of the symptoms should be weighed against the risks of treatment. Systemic treatment with oestrogens would not be sensible, but local treatment for a few weeks might be. Sometimes the area of especial discomfort is small and near the introitus. Dienoestrol cream might be applied to it without seriously adding to the oestrogen content of the blood. On the other hand, stilboestrol pessaries contain 0.5 mg of the active agent, of which an unknown quantity would be absorbed. Some alternative to oestrogens might be found in lactic acid jelly inserted with an applicator, and even the old-fashioned vinegar douches might be worth a try. And since science may be misleading sodium bicarbonate douches might be used, to see if they have any effect.

I have observed spontaneous or induced lacrimation on painful stimulation occur in patients with primary or secondary brain stem lesions. The patients generally have displayed decerebrate posturing and absence of clonic responses of the doll's eye phenomenon. When the clinical signs are correlated with post-mortem findings, the most frequent lesions have been found to be within the pons. Not all patients with lacrimation have died, but the symptom has been associated with a high mortality rate. Is this a recognised association?

This is an extremely difficult question to answer because such a case is seldom reported—probably not enough for a statistically significant answer. Any patient who has brain stem lesions for any reason is likely to die, and I would not have thought that the symptoms as described associated with lacrimation would be of more sinister significance as regards prognosis than other brain stem lesions. Nevertheless, anything of this nature that can be adequately recorded over sufficient cases would be of interest.

What is the present-day management after inserting grommets in children?

Grommets are used in the hope of providing equal air pressure in the middle ear and external auditory meatus. Although in America the grommet is sometimes referred to as a middle-ear drainage tube, there is little evidence that there is any drainage through the tube. Generally, if the grommet is going to work it should achieve maximum success within the first three months. Grommets that remain in for over six months tend either to irritate the middle fibrous layer of the tympanic membrane and produce tympanosclerosis (which is seen as calculous plaques on the drum head), or to migrate round the drum head and may give rise to pressure necrosis of the ossicular chain. If either tympanosclerosis or migration is observed the grommet should be removed immediately. There is little evidence of increased risk of bacterial otitis media while the grommet is in situ, but when bacterial otitis media does occur, the grommet should be removed if it has not been discharged at the height of the inflammatory process. Children should not be allowed to swim when grommets are in position because of the danger of a water-borne bacterial otitis media.