Current Practice

CLINICAL PRACTICE

Colonic Diverticula

Pathology and Natural History

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British Medical Journal, 1970, **3**, 565-567

Diverticula of the colon are the commonest pathological process in the large bowel; yet until relatively recently little general interest was shown in their frequency, aetiology, pathology, and treatment.

No mention of this easily recognized entity appeared before the nineteenth century, but in 1849 Cruveilhier[1] described colonic diverticula and in 1857 Habershon[2] of Guy's Hospital gave a surprisingly accurate account of "pouches of the colon which sometimes becomes a considerable size . . . the orifices of these small sacs are bounded by the hypertrophied circular and longitudinal fibres and their contents remain almost shut off. These pouches are the result of constipation, muscular fibres become hypertrophied, but their effort to propel onward their contents lead to these minute hernial protrusions." W. H. Cripps described vesicocolic fistula due to inflammatory changes in the colon in 1888,[3] and in 1903 R. Morison carried out a resection of the sigmoid colon for what he thought to be a carcinomatous obstruction but which proved to be what he preferred to call "sacculitis."[4] By 1910, however, A. Keith could collect only seven specimens of diverticula of the colon in the museum of the Royal College of Surgeons of England and the London medical schools compared with 149 other examples of alimentary diverticula.[5] Aetiologically he regarded the colonic diverticula as being due to high pressure within the bowel lumen. Seven years later W. H. M. Telling and O. C. Gruner were able to collect 324 cases of colonic diverticula and gave a detailed analysis of the complications of this condition.[6]

An enormous impetus to the study of diverticular disease was provided by the introduction of the barium enema examination. In 1926 E. I. Spriggs and O. A. Marxer noted colonic diverticula in 100 of 1,000 barium enema examinations and described a "prediverticular" phase of contraction of the bowel with a "saw-tooth" irregularity.[7] Later Spriggs reported that he had noted the subsequent development of diverticula in this zone.[8] He believed that some toxic or bacterial damage from contained faeces produced local irritation and that resultant contraction of the bowel was responsible for the formation of these dilatations.

Incidence—Age and Sex

There is evidence that the incidence of diverticula of the colon is increasing in civilized communities, that its sex distribution is changing, and that it is being seen increasingly in younger age groups. Nevertheless, until recently our knowledge of the distribution of this condition depended on radiological investigations of patients having at least some sort of alimentary disturbance, or postmortem studies, often retrospective, on subjects dying in hospital; both methods obviously involved population selection.

In 2,179 consecutive barium enema examinations in Sheffield, J. L. A. Grout reported an 8% incidence of diverticula.[9] J. D. J. Pemberton and his co-workers found an 8·5% incidence in 47,000 examinations at the Mayo Clinic,[10] H. K. Ransom a 12% incidence in 14,695 examinations at the University of Michigan,[11] and C. E. Welch and his colleagues a 20% incidence in patients over the age of 60 in Boston.[12] These studies showed that the condition was rare in young adults and increased in frequency with age.

Early retrospective reviews of postmortem findings yielded a low incidence of colonic diverticula, which was about 2% at the London Hospital,[13] at the Cook County Hospital, Chicago,[14] and in Adelaide.[15] In all cases the incidence rose with increasing age. Nevertheless, unless looked for specifically, diverticula of the colon can easily be overlooked. T. G. Parks, studying 130 colons obtained post mortem found that 45 bore diverticula,[16] and recently L. E. Hughes has reported the results of painstaking dissection of the large bowel in 200 cases, mostly in people in their 70's.[17] No fewer than 85 of these showed colonic diverticula, and another five bore a solitary diverticulum of the caecum.

The only study of a perfectly healthy population reported is that of O. N. Manousos and his colleagues who investigated 109 subjects in Oxford without gastrointestinal symptoms.[18] This series comprised medical staff, visitors to hospital, and a small group of geriatric patients. A follow-through radiological method was used after drinking radio-opaque material. Diverticula were found in one in every three persons over the age of 60; below that age the condition was present in 7·6% of the population studied. These same workers analysed previous reports over the last 60 years and have shown an interesting change in sex incidence; earlier papers showed a male preponderance of up to 2:1, which has swung over since the early 1950's to a female preponderance varying in different reports from 1·2:1 to 2:1 proportion of females to males.

Though a condition found predominantly in the elderly, there have been sporadic reports of diverticula occurring in teenagers and young adults.[19,20] It is a distinct clinical impression, however, that more and more examples of acute diverticulitis occurring in young adults are now being seen.

Aetiology

Colonic diverticula usually appear in two longitudinal rows between the mesenteric taenia and the two lateral taeniae. H. Drummond pointed out that these sites correspond to the points of penetration of the colic blood vessels through the bowel wall and postulated that diverticula occurred there because of the resultant weakening of the muscle coat.[21] R. J. Noer confirmed these findings by injecting radio-opaque material into the colonic blood vessels and then *x*-raying the bowel wall;[22] each diverticulum was shown to correspond exactly to the site of blood vessel penetration, thus accounting for the well known complication of massive haemorrhage which may occur in diverticular disease.

For a long time pathologists and clinicians came to regard diverticula as outpouchings of mucosa occurring in degenerate, fatty infiltrated, and weakened bowel.[23,24] In 1963, however, B. C. Morson redirected interest to the true state of affairs by showing that specimens of sigmoid colon resected for diverticular disease showed thickening of the taenia coli

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and of the circular muscle, the latter being arranged in concertina-like folds with the orifices of the diverticula opening between the corrugations.[25 26] The mucosa itself is thrown into folds and appears to contribute to the narrowing of the bowel lumen; the thickening he explained as being due to shortening and spasm of the muscle layers of the bowel rather than hyperplasia or hypertrophy. This muscle thickening has now been described in detail by numerous investigators.[17 27-29]

Recent pressure studies have thrown further light on the aetiology of diverticula. S. Arfwidsson found higher intrasigmoid pressure waves in patients with diverticula compared with normal subjects whether at rest, after food, or after prostigmine.[28] N. S. Painter has reported normal pressure tracings in the sigmoid colon in patients with diverticular disease at rest but an appreciable increase in pressure as a result of morphine and prostigmine.[30-32] Cineradiographic studies showed segmentation of the bowel during phases of high pressure with gross distension of the diverticula as local pressure increased in the affected segments.

Mechanisms

We thus have a concept of some predisposing factor or factors producing spasm and contraction of the muscle wall of the colon. The resulting high pressure acts on points of maximum weakness—the sites of blood vessel penetration—thus producing pulsion diverticula.

Interestingly R. R. Wilson was able to produce diverticula experimentally in the walls of normal freshly removed appendices by opening the muscle down to the mucosa and inducing synchronous contraction of the muscularis of the appendix.[33] Clinically this was confirmed many years ago by S. Barling,[34] who recorded how he explored a man of 43 with a three-year history of diarrhoea, rectal bleeding, and pain. Initially the sigmoid colon appeared normal except for its walls, which were rather thicker than usual. He then observation twice more as the colon relaxed and then and rigid, and a row of diverticula to appear; he repeated the observation twice more as the colon relaxed and then contracted again.

The exact nature and the aetiology of the muscular thickening in diverticular disease remain unknown.[35] Patients with spastic colon (or the "irritable colon syndrome") have similar physiological responses of the colon to patients with diverticular disease; both have a shorter transit time for food than normal people and both have increasing motility after eating a meal. Indeed, patients with the irritable colon syndrome may go on to develop diverticula.[36]

Geographical Variation

There is a definite geographical variation in the distribution of diverticular disease; though common in the so-called civilized Western World, it is rare in the undeveloped countries of Africa and Asia. Though almost unknown in rural African communities, examples are now being found in subjects living under urban conditions, and the Negro in the U.S.A. apparently develops diverticula with frequency equal to his white compatriot.[37] Possibly this may be associated with the emotional and psychological tensions of civilized life, since stress has been shown to increase the pressure within the sigmoid colon.[38] A more likely explanation might be that the low bulk of the Western diet, by producing a small constipated stool, may provide inadequate dilatation of the colon; this could allow progressive contraction of the circular muscle bundles of the large bowel. This contrasts with the soft bulky stools of primitive peoples partaking of high roughage diet.

Isolated diverticula develop in the rat colon after prolonged feeding on a high fat or a low residue diet,[39 40] but we have been unable to confirm this on a small group of rats maintained on a roughage-free diet for a period of between 1 and 1½ years.[41] We are at present engaged in a long-term study of a large colony of rats which is being maintained on a no-roughage diet from the time of weaning.

Modern Concepts

Our ideas about diverticular disease have recently changed.[42 43] Nevertheless, until recently clinicians, radiologists, and pathologists talked about two entities. The first, diverticulosis, implied a colon studied with diverticula which were symptomless. The second, diverticulitis, was further subdivided into acute and chronic. The clinical features of the former, with paracolic infection or free perforation, were easy to define. Those of the latter, chronic or recurrent diverticulitis, were considered to be due to long-standing inflammatory changes and presented with pain, tenderness, constipation, or subacute obstruction—while radiologically a narrowing and distortion of the affected segment of sigmoid colon could be seen. This concept was rudely shaken by Morson, whose detailed studies showed that frequently specimens of colon resected as examples of so-called chronic diverticulitis showed no evidence at all of inflammation.[25 26] He used the term "diverticular disease" to denote this state of affairs. S.-C. Ming and F. G. Fleischner, studying 62 specimens of sigmoid colon resected for diverticulitis, found not a single example of 'chronic diverticulitis";[44] 16 of the specimens showed no inflammatory disease and others showed acute inflammation of a diverticulum with its consequences—paracolic abscess, sinus or fistula formation, or free perforation with peritonitis. Finally, Parks has shown the difficulties of differentiating between "diverticulosis" and "diverticulitis" clinically and the impossibility of doing this by barium enema examination.[45 46]

Three groups of patients can now be defined. The first and very large group comprises subjects with symptomless diverticula—that is, *colonic diverticulosis*. The second consists of those patients presenting with acute inflammation of the diverticulum with its complications—that is, *acute diverticulitis*. The third group is made up of patients developing colicky pain in the left lower abdomen, bowel disturbance, dyspeptic symptoms, and subacute or even acute intestinal obstruction, with no pathological evidence of inflammatory disease. The term *diverticular disease* is a useful description at this stage. The fascinating question, at present unsolved, is the factor which determines whether a patient whose colon bears diverticula will be completely free from symptoms, as occurs in most examples, or will develop any of the protean manifestations of this non-inflammatory condition. Possibly further investigations using manometry or cineradiology may show significant differences between these two groups.

Complications of Diverticular Inflammation

The clinical syndrome of acute diverticulitis results from extravasation of colonic contents into a diverticulum associated with intramural inflammation, with foreign body granuloma or abscess formation.[47] This may lead to perforation of the affected diverticulum with either a local paracolic abscess or free perforation with general peritonitis which may either be purulent or faecal. The inflamed diverticulum may become adherent to, or form a fistula into, an adjacent viscus—particularly the bladder but also the small bowel, the uterus, the uterine adnexae, or the vagina. Erosion of a blood vessel at the neck of the diverticulum may result in a severe haemorrhage into the lumen of the colon.

Inflammation of one or more diverticula produces the features of "left-sided appendicitis" with fever, nausea, and vomiting and with localized pain, tenderness and guarding. This condition may resolve on conservative treatment but may progress to abscess formation with a localized tender

mass and a raised leucocyte count. When this occurs surgical drainage, together with a defunctioning transverse colostomy, must be performed. When the acute episode has settled the affected sigmoid segment must be resected followed by subsequent closure of the colostomy. Rarely acute local infection may be complicated by a psoas abscess[48] or by portal pyaemia.[49-51]

Fistula Formation

Fistula formation into the bladder (vesicocolic fistula) is a fascinating condition, seen more often in men than women since in the latter the uterus obtrudes between bladder and colon. The patient presents with cystitis, pneumaturia, and the passage of faecal material in the urine—though, surprisingly enough, the patient rarely notices urine passed through the rectum. It is also surprising how often in an old and feeble patient this condition causes little systemic disturbance; if his general medical state is poor it may indeed be advisable to leave well alone. When he is relatively fit, however, closure of the fistula with resection of the affected segment of colon must be carried out with either a two-stage or three-stage operation. C. W. Mayo and C. P. Blunt encountered 46 examples of vesicocolic fistula in 200 cases of diverticulitis over a period of 10 years.[52] 39 of these occurred in men and the age variation was from 29 to 76 years: 40 complained of pneumaturia, 21 of dysuria and frequency, 21 of the passage of faeces per urethram, and 14 of either suprapubic or perineal pain. Ten patients noticed haematuria and only one complained of the passage of urine per rectum. Of 45 patients who were examined by barium enema, nine showed the fistulous track; 33 had cystoscopy performed and in 28 of these the appearances suggested the presence of a fistula.

Perforation

Free perforation with general peritonitis is naturally an extremely serious complication. The prognosis is particularly poor in elderly patients who are admitted with shock, and especially if the peritonitis is faecal rather than purulent.[53 54] In the past a rather conservative surgical approach has been advised with drainage of the peritoneum and transverse colostomy or exteriorization of the perforation.[55 56] More recently, however, excellent results have been reported following resection of the perforated segment with either immediate anastomosis or exteriorization resection with later closure.[57-61] In the hands of an experienced abdominal surgeon, this would appear the sounder procedure.

Haemorrhage

Recently increasing emphasis has been placed on haemorrhage as a complication, both as a cause of minor blood loss and as a source of major intestinal haemorrhage.[62-65] Fortunately in most cases the haemorrhage ceases spontaneously and emergency surgery is not required, but for this very reason not a great deal is known about the exact origin of the bleeding. Possibly a vessel lying near the diverticulum becomes eroded by localized inflammatory disease. W. W. Slack studied 36 surgical specimens removed for diverticular disease.[66] Twelve of the patients had complained of intermittent rectal bleeding, but in only four of the specimens was ulceration of a diverticulum found. In these cases granulation tissue was seen with open-ended blood vessels in the walls of the diverticula. J. M. Young and M. B. Howorth reported that in one specimen of colon resected for continued rectal bleeding several diverticula had inverted into the lumen of the bowel, and one of these had undergone ulceration.[67]

Continued haemorrhage from the bowel presents a serious, but fortunately rare, indication for surgical intervention. If the diverticular disease is localized at laparotomy then this segment should be resected and the specimen opened to confirm that the source of haemorrhage has been removed. Where the disease is widespread exploration through multiple colotomies —with or without insertion of a sterilized sigmoidoscope—has been advocated.[68] However, both J. C. Goligher and W. R. Olsen advised total or near total colectomy with ileorectal or right colorectal anastomosis in these circumstances.[69 70]

Differential Diagnosis

Obviously the symptoms and signs of both diverticular disease and of carcinoma of the left colon closely resemble each other, and no feature in either the history or the examination may clearly define one from the other.[71] A barium enema examination may give classical features of one or other condition but every experienced clinician will recall many cases where the radiologist was in reasonable doubt about the diagnosis. A carcinoma may be found by sigmoidoscopy, and a positive biopsy will clinch the diagnosis, but in most cases the tumour is out of reach of the instrument. Even at laparotomy the surgeon may have considerable difficulty in telling whether a large mass in the sigmoid colon, perhaps adherent to adjacent structures, is due to one or other disease.

Differentiation is important since, believing mistakenly that he is dealing with cancer, the surgeon may perform a radical operation—on occasions even an abdominoperineal excision of the rectum—for what proves to be benign disease. Under such circumstances we have practised a very simple manoeuvre; a small incision is made into the lumen of the bowel above the diseased area and a finger passed distally into the affected segment. If the mass is due to diverticular disease, there will be no ulceration of the mucosa and this, though oedematous, will feel completely smooth, since the condition is produced, as we have already stressed, by pericolic disease. A carcinoma, in contrast, will always give the typical feel of a malignant ulcer, with heaped up edges.

Perhaps one should not strive too hard to effect this differential diagnosis. I have already stressed how common diverticular disease of the colon is in our elderly population; carcinoma of the large bowel is now the second commonest killing cancer in our community, so obviously we will encounter many cases in which these two conditions co-exist. I have personally operated on several patients in whom one or other lesion has been diagnosed only to find *both* present in the same resected specimen. Moreover, other colonic diseases apart from cancer may co-exist with diverticulosis of the large bowel; polyps, for example, are far from unusual and may be responsible for some cases of haemorrhage from the rectum attributed to the diverticula. G. T. Schmidt and his colleagues have recently reviewed 26 examples of Crohn's disease in segments of diverticular disease of the colon.[72]

This century has witnessed diverticula of the colon progress from pathological curiosities to a common entity. Much remains to be discovered about their aetiology, especially about the coexisting (and probably pre-existing) abnormality of the muscle wall of the sigmoid colon. Only then can we plan rational prophylaxis and treatment of diverticular disease.