Chronic Ulcerative Colitis: An Analysis of 88 Cases*

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FROM 1922 until the end of 1930 there were admitted to the Beth-El Hospital 23 cases of chronic ulcerative colitis. Since 1930 we have treated 65 patients for this illness. This marked increase in incidence of chronic ulcerative colitis on our hospital service during the present decade has prompted us to review the recent literature relative to this disease and to present an analysis of the 88 cases which have been admitted to our institution.

DEFINITION

Chronic ulcerative colitis is an inflammatory condition of the colon, usually extending upwards from the anus, involving the colon either partially or along its entire course from the anus to the cecum. It is a specific entity, not uncommon in incidence, running a prolonged and intermittent course, with serious complications and frequently fatal in its outcome.

ETIOLOGY

The signs and symptoms in the patient who presents himself for examination and treatment unquestionably point to the infectious nature of the disease. Much difference of opinion has existed relative to the etiology of this condition. Is infection the primary etiological factor? Is infection secondarily imposed upon a colon which, for other reasons, has lost its normal resistance to bacteria which ordinarily inhabit it? Is the infection similar or akin to other well known forms of colon inflammation? The confusion resulting from the answers to these questions has complicated the approach to the disease and made more difficult the method of treating it.

Evans (1) suggests that the terms pylorospasm, gastritis or duodenitis, and peptic ulcer represent steps of one disease process in the stomach or duodenum and that the disease is manifest in the colon through spastic colitis, mucous colitis, catarrhal and ulcerative colitis. Drueck (2), Kuttner (3), and many other recent writers hold that ulcerative colitis indicates a late stage of the spastic colon. On the other hand Bargen (4) states that the number of cases in which chronic ulcerative colitis follows the irritable colon is very small, and would not justify the belief that the latter is a forerunner of chronic ulcerative colitis.

Because of the appearance of symptoms of avitaminosis in some patients certain authors have felt that vitamin deficiency in some form may be the initial cause for the onset of the disease. This concept does not appear to be borne out clinically. All of our cases occurred in individuals whose dietetic habits did not deviate to any marked degree from the normal. Three of our cases showed evidence of vitamin deficiency. Two patients presented the skin manifestations of pellagra and in one the beriberi type of heart was discovered at autopsy. In view of the fact that these symptoms appeared either in the late or terminal stages of the disease, it is assumed that the vitamin deficiency is rather a result of improper absorption and utilization in, than a cause of, chronic ulcerative colitis. Dukes (5) and Bargen (4), who have carried out studies of the life habits of many series of patients found no evidence of vitamin or other deficiency playing a part in the causation of the disease.

Some authors have ascribed a disturbed metabolism as an etiological factor. Portis (6) suggests a thyrotoxic cause for the disease. Basal metabolic readings in six of our patients showed the rates to be normal in four, slightly above normal in one and slightly below normal in another.

Recently the psychogenic origin of the disease has received considerable attention. Sullivan (7, 8) found psychological difficulties in 18 of 25 consecutive cases of ulcerative colitis, in 15 of which the emotional disturbances appeared to be of definite etiological significance. He found a well marked time relationship between emotional crises and the onset of the disease or its recurrences. Murray's (9, 10) reports are confirmatory of this idea. Rechad (30) and Sullivan (8) found that psychotherapy was a successful method of treatment in chronic ulcerative colitis. 72% of our cases occurred in young adults between the ages of 16 and 40, in many of whom the problems of sex, marriage or finances might have been responsible for the creation of a psychological conflict. Five of our patients gave histories of having had "nervous breakdowns" previous to the onset of the colitis. We have called attention to the marked increase in the number of cases at our hospital since 1930. It is of interest to note that this is coincident with the period of economic depression which set in at that time. Economic and financial worries may possibly have been the exciting factors in a number of these patients.

Turner (11), Hurst (5), Hern (12) and Penner (13) feel that the disease is a form of bacillary dysentery. Winkelstein (14) reports that 20% of his cases are due to this condition. These authors base their reasoning on the response of many patients to specific treatment with anti-dysenteric serum. However, as Dukes (5) and Bargen (4) point out, the negative bacteriological and epidemiological evidence, the absence of agglutination and the uncertain effect of antidysentery serum seem to discount this point of view.

Fradkin (15), Winkelstein (14) and others feel that the ameba may be a cause of chronic ulcerative colitis. It is undoubtedly true that a number of cases of amebic colitis are mistakenly diagnosed as chronic ulcerative colitis. But where the diagnosis is not supported by laboratory evidence or by effects of anti-

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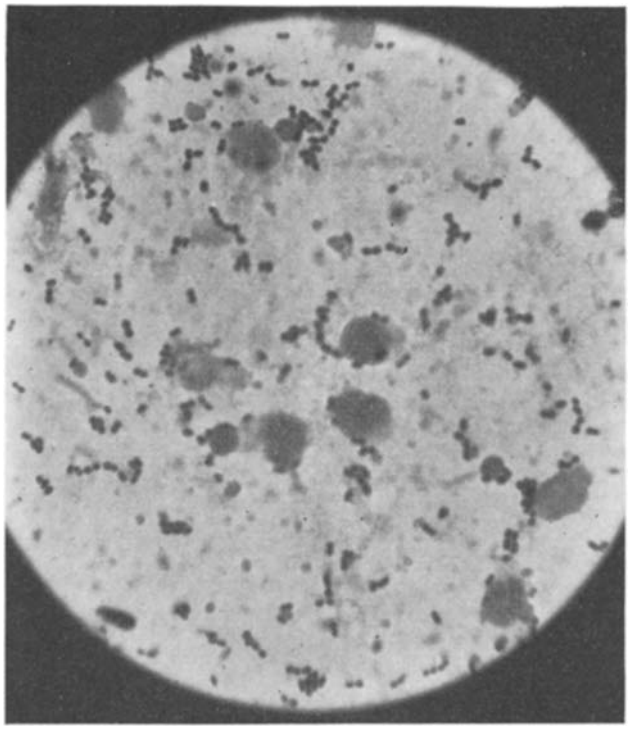

Fig. 1

amebic therapy, one can hardly see any reason for attributing to this organism the responsibility for this disease.

Bassler (16) found that in young subjects Clostridium Welchii, enterococcus and green and non hemolytic streptococcus is the infective trilogy and that in adults the hemolytic streptococcus is most often met with.

Bargen (4) and other workers have established a diplostreptococcus of characteristic morphologic and biologic properties as the main causative organism of chronic ulcerative colitis. The diplococci, when isolated according to the method of Bargen, are fused in pairs, almost merging into chains of four cocci, are lancet shaped and gram positive. The diplococcus has been isolated in pure culture from the rectal lesions of 80% of his cases at the Mayo Clinic. The lesions of ulcerative colitis have been reproduced in rabbits and dogs by the intravenous injections of many strains of this organism. It has been recovered from the blood in acute fulminating cases. It has been found in foci of infection such as peridental or peritonsillar abscesses and cultures of these reproduced the disease in animals. Cook (17) produced periapical dental infections in dogs and two to 16 months later the proctoscopic picture of chronic ulcerative colitis was demonstrated. People in normal health have been found to be carriers of this organism. It has been isolated in the stools of patients with vague abdominal complaints who later developed the typical picture of chronic ulcerative colitis.

Many authors have cast doubt upon the contention of Bargen that the diplostreptococcus is the chief offending organism. Portis (6) states that no positive blood cultures are found. However, one can rarely obtain positive blood cultures in most diseases which have been initiated by infection from a distant focus. Bassler (17) recovered the diplococcus in 44% of his cases of ulcerative colitis and in 37% of 500 cases of non-ulcerative colitis, yet feels that it is not the etiological factor in the disease. Rafsky (18) states that the diplococcus is a strain of the enterococcic group. Bargen has definitely shown significant differences between the two; namely that on mannite agar the appearance of the colony of the diplococcus is fine and translucent; the enterococcus grows as a large, white opalescent colony on this medium. The diplococcus does not grow on plain agar; it does not grow on gelatin; it usually does not coagulate milk. The enterococcus does all of these readily. Dukes (5) feels that the diplococcus and other types of fecal streptococci are secondary invaders and that the bloody diarrhea produced by intravenous injection in rabbits does not follow the course of human chronic ulcerative colitis and that the same result is produced by other bacteria.

In our series of cases the diplostreptococcus was isolated from 16 patients or 18% of the total. Rarely did we make repeated examinations after failure to isolate the organism on the first attempt. It is probable that, if the technical details and suggestions of Bargen were carefully followed out, many more cases would reveal the diplococcus as the offending organism. The enterococcus was isolated in six cases or 7% of the total. A green streptococcus was isolated in two patients and a hemolytic staphylococcus in one. No specific organism was isolated in 63 patients or 72% of the entire group.

The exacerbations of this disease following upper respiratory infections and the removal of foci of infection lend support to the belief that the disease is infectious in nature. Seven of our patients had acute exacerbations after upper respiratory infections and one after a pleurisy. Tonsillectomy caused an exacerbation in one. The extraction of a number of teeth led to an acute fulminating exacerbation in one case with death from sepsis.

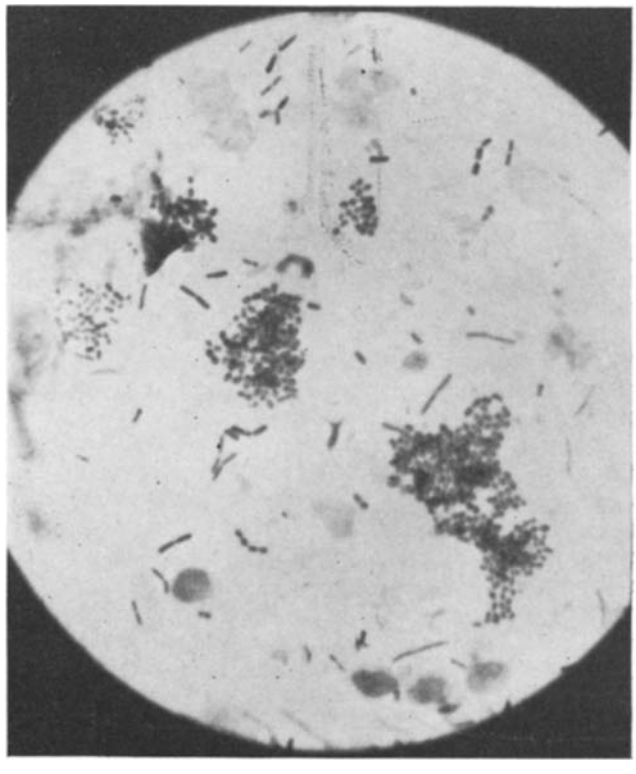

Fig. 2

The following report substantiates in every detail Bargen's claims as to the etiology of chronic ulcerative colitis and clearly illustrates the role of a focus of infection in the onset and recurrence of symptoms.

Case No. 57081, J. S., male, age 17. Admitted January 9, 1933, with a history that two months prior to admission, following a cold, the patient developed a diarrhea and shortly thereafter noticed blood in the stools. After treatment with medicines his condition improved. He then had another upper respiratory infection which brought with it a more severe recurrence of symptoms and hospitalization was advised. On admission, patient was having numerous loose stools containing blood and pus and on many occasions passed only clots of blood. There were severe abdominal cramps and marked weakness. Temperature was 103. White blood cells numbered 20,200 with 80% polymorphonuclear cells. Hemoglobin was 55% and red cell count 2,575,000. Barium enema showed a spastic colon which filled very rapidly. Sigmoidoscopy disclosed numerous ulcerations in the rectum and sigmoid, exuding a bloody and muco purulent discharge. Smears taken from the stool and stained revealed a preponderance of diplococci singly and in chains (Fig. 1). Culture of the stool showed almost pure culture of the same organism. Repeated transfusions were given to tide the patient over the acute phase of his illness. A vaccine prepared from the organisms isolated in the stools was administered. Five weeks after administration of the vaccine direct smears from the stool showed clumps of agglutinated diplostreptococci (Fig. 2). (This was probably caused by the presence of agglutinins in the serum of the blood which had exuded through the colonic mucosa and agglutinated the organisms). The patient was discharged markedly improved on February 16, 1933. Vaccine treatment was continued at home for three months. At the end of this time no diplococci were found in either the smear (Fig. 3) or culture from the stool. Patient felt well and had three to four bowel movements per day which were occasionally streaked with blood. On November 4, 1933, patient was again ill with an upper respiratory infection. Shortly thereafter there was a recurrence of the severe diarrhea. He was readmitted to the hospital November 18, 1933, for tonsillectomy. Stool examination at this time failed to show the diplococcus. The number of stools lessened under medical treatment and a few weeks after admission a tonsillectomy was performed. This was followed the next day by an increase in the number of stools which contained blood. Examination of the stool at this time showed numerous diplococci. Culture of the tonsils disclosed a pure culture of diplostreptococci, the same organisms which were noted in the stool. Another autogenous vaccine was prepared and administered for six weeks. Patient was discharged as cured on January 3, 1934. There has been no recurrence of symptoms to date.

Five patients had acute recurrences during pregnancy and two had therapeutic abortions performed for this reason. Two patients had recurrences after appendectomy and three after hemorrhoidectomy.

Patients of all ages and of both sexes are affected by this disease. The youngest in our series was seven years of age, the oldest 70. 62% of our cases occurred in females (Table I).

In only one instance were two members of one family affected; a young girl who developed symptoms shortly after the death of her sister from chronic ulcerative colitis.

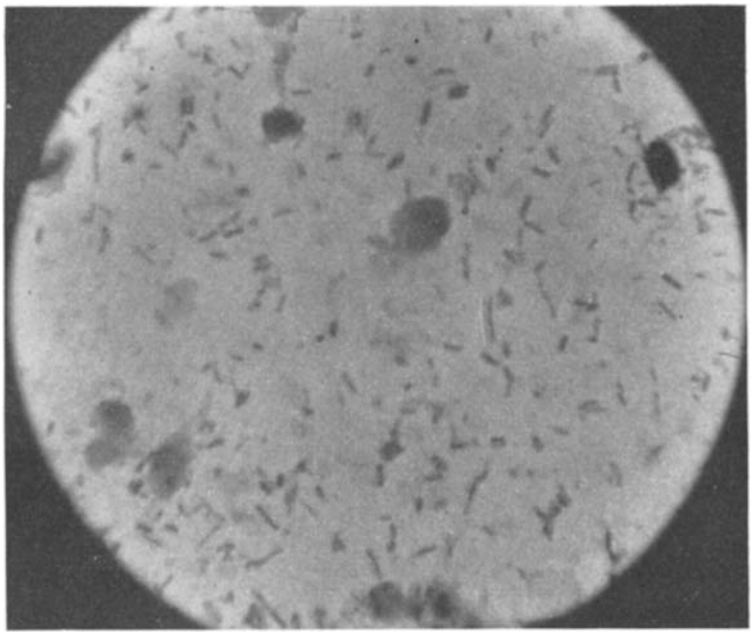

Fig. 3

SIGMOIDOSCOPY (GROSS PATHOLOGY)

In the greatest number of cases the pathologic process is initiated within the lower limits of the rectum and extends upward toward the sigmoid and descending colon. The initial stage is characterized by an acute inflammation of the mucous membrane of the colon. It appears diffusely hyperemic. This is shortly followed by an edema of the mucosa which becomes boggy and readily bleeds when slightly traumatized by the sigmoidoscope. Minute abscesses appear beneath the surface of the mucosa which slough away and leave small, pin point, bleeding ulcers. At this stage the mucous membrane appears diffusely granular and bleeding. The union of these minute ulcers may produce ulcers of larger dimensions and secondarily imposed infection will alter their appearance. During the stage of remission the pin point ulcers may heal and leave small scars. In the late stages of chronic ulcerative colitis, when great difficulty may be encountered in differentiating this disease from other forms of

colitis with ulcerations, the presence of these small, pit-like scars is of utmost importance in making the diagnosis. As a result of the diffuse scarification with formation of fibrous tissue the wall of the bowel becomes contracted and the lumen narrowed from slight to extreme degrees. Strictures may develop in cases where the pathologic process has been more severe and fibrosis more marked. Purulent exudate, dammed up by these strictures, may produce large abscesses.

TABLE I

Age incidence

Age	7	11-15	16-20	21-30	31-40	41-50	51-60	61-70	Total
Male	1	1	1	14	9	4	1	2	33
Female	0	3	12	21	6	6	5	2	55

When the ulcers heal, ragged polypoidal tags of mucous membrane remain. (These must not be confused with true adenomatous proliferations). When these infected ulcers extend into the deeper layers of the colon the danger of perforation is imminent.

The following chronologic reports exemplify the sequence of protoscopic findings in one of our cases:

Case No. 82697, F. L., female, age 16. Admitted March 4, 1937, with history of diarrhea of five weeks duration, the stools during the latter two weeks admixed with blood and pus. Sigmoidoscopy March 11th; markedly congested mucosa. Bleeds readily. Many small, whitish areas under the mucous membrane (Abscesses). Many small ulcerations. May 26th: Acutely congested mucosa. Many small and larger ulcerations. Many small, pit-like scars noted throughout the mucosa. July 7th: Mucosa much paler but thickened and scarred. Few polyps noted.

The following case exemplifies the ascending nature of chronic ulcerative colitis and the danger of pronouncing a patient cured without thorough and long follow up:

Case No. 32941, E. K., female, age 20. Admitted March 9, 1929, with a history of three days of bloody diarrhea. March 11th—proctoscopy reveals an ulcerative proctitis extending only two inches above the anus. Patient was apparently cured with rectal instillations of bismuth subnitrate in olive oil and discharged April 29th. Patient was readmitted on November 18, 1929. There was a recurrence of bloody diarrhea after an upper respiratory infection. Sigmoidoscopy revealed pin point abscesses with small and button sized ulcers throughout sigmoid and rectum, penetrating through the mucosa and submucosa.

The following gross and microscopic pathologic description indicates the marked destructive process in a case of longer standing:

Case No. 81796, M. J., female, age 29. Admitted January 13, 1937, with a history of diarrhea of four years duration. Patient died February 1, 1937, of a generalized peritonitis due to perforation of the colon. At about the hepatic flexure, small mucosal ulcerations are first noted. These grow very extensive along the transverse colon partly encircling the bowel. They possess ragged, undermined edges with a base made up of submucosa, muscularis, and along the attachment of the lesser omentum only peritoneum. The peritoneum presents longitudinal rents encircling the bowel, affording communication between the colon and peritoneal cavity. The descending colon, sigmoid and rectum present a more advanced degree of the lesions. The bowel is thickened to about 0.5 cm. No normal mucosa is seen. Only ragged strips and bridges of mucosa course irregularly across the inner aspect. Between these lie exposed submucosa, muscularis or serosa.

MICROSCOPIC EXAMINATION

The mucosa is for the most part absent. In one place most of the thickness of the submucosa is also gone. The cells along the surface of the mucosa show degeneration. In the mucosa there is an infiltration of lymphocytes and plasma cells and some edema. A few polymorphonuclear cells are also noted. Where the mucosa is absent the surface is covered with cellular debris and polymorphonuclear cells. There is an increase in fibrous tissue and some edema of the submucosa. It is infiltrated with lymphocytes, plasma cells, histiocytes and polymorphonuclear cells. A lymph node in the serosa shows a widening of its sinuses and these hold histiocytes.

SYMPTOMS AND DIAGNOSIS

The disease may start insidiously. The patient may complain of vague abdominal pains, increased frequency of stools with an excessive amount of mucous. The stools are loose in consistency. After a variable period of time the patient will notice blood admixed with the stool and later the presence of pus. There may be slight or even no elevation of temperature. The patient has frequent calls to stool and may pass only hemorrhagic or purulent rectal discharge without fecal matter. In the fulminating case there is a rather abrupt onset with high and occasionally septic type of temperature. The patient appears acutely ill. There are numerous loose purulent and bloody stools. The patient shows all evidence of a profound toxemia.

Later the patient presents a picture of inanition and dehydration. There is marked loss of weight, pallor and anxiety. 46% of our cases had a marked secondary anemia. 8% had achylia gastrica. The leucocyte count and sedimentation rate were increased especially in cases with hyperpyrexia.

The diagnosis rests upon the history and sigmoidoscopic findings as aforementioned, the physical examination and the appearance of the colon on the X-ray film after examination with the barium enema.

Physical examination of the abdomen will disclose a firm, tube-like descending colon and sigmoid which may be tender to palpation. Digital examination of the rectum discloses a spastic sphincter. The lumen is narrowed. The mucosa is granular and, if ulcerations are deep, they may be noted by the examining finger.

The appearance of the colon with the barium enema depends upon the stage of the disease, the extent of the ulceration and the presence of sequellae. The colon fills rapidly. Where the ulcerations are small and superficial the outline of the colon is smooth and shows a notable lack of haustration. Where the ulcerations are deeper the outlines of the colon are feathery, a

characteristic picture of the disease. Where ulceration is deep, and irregular fibrosis has occurred, the outline is rough and irregular. Because of the fibrosis of its wall the colon becomes narrowed and shortened. Examination by the contrast enema, where air is injected into the colon following the expulsion of the barium, will more easily outline ulcers and polyps.

COMPLICATIONS

Stricture of the bowel is commonly seen. It is the result of the fibrosis which takes place during the stage of healing. One is struck by this condition when one finds marked difficulty in passing the sigmoidoscope because of the narrowed lumen. It is wiser to stop the examination than to attempt to forcibly push one's way past the stricture. Perforation of the bowel resulted from this procedure in one of our early cases. 13 of our cases were complicated by stricture.

Polyposis (improperly termed because the polypoidal tags of mucous membrane which remain after healing are not true polyps) is frequently noticed on proctoscopic examination. If present higher up in the colon they are well visualized on the X-ray by means of the contrast enema. 11 of our cases were complicated by polyposis. When large these false polyps may aggravate the symptomatology.

Perforation of the colon with a resulting peritonitis occurs when ulceration has penetrated the serosa of the colon. This was observed in three and the cause of death in two of our cases. Penetration of ulcers in the perirectal and perianal regions may form perirectal abscesses which then may rupture through the skin, vagina or bladder and produce fistulae. Nine of our cases showed these complications.

Vitamin B deficiency was found in a case which revealed a beriberi type of heart at autopsy. There was myocardial atrophy, edema and vacuolation of the heart muscle. Two cases of vitamin G deficiency were noted — a roughness and dark discoloration of the backs of the hands and forearms as is seen in pellagra.

There was an inversion of the serum albumin and globulin ratio in two cases. There was hypoproteinemia in three cases, two of which revealed evidence of a nutritional edema.

Arthritis complicated two of our cases. Hepato and splenomegaly was found in one case.

Bargen (4) and Hurst (19) in addition note such complications as renal insufficiency, occular lesions, peripheral neuritis, progressive arterial occlusion, multiple abscess of the liver, tetany and carcinoma. Carcinoma is not as common a complication as is found in true polyposis or multiple adenomata of the intestine. This is probably due to the fact that the polypoidal masses are not true adenomatous proliferations.

PROGNOSIS

Primary recovery within a few weeks has been reported. We have never seen it. Exacerbations and remissions are common. One must not fall into the error of pronouncing a case cured merely on freedom of symptoms or even with apparently healed lesions on examination of the colon, unless the patient has been followed up for a reasonably long period of time.

Hurst (19) feels the mortality should not exceed 5 to 10% if the patients receive adequate treatment for sufficient periods. He states that the large majority should recover eventually so completely that they are able to lead a life of normal activity. Bargen (20) states that X-rays show a tendency of the bowel to return to normal in 40% of the cases. Crohn and Rosenak (21) in a series of 75 cases report 33 cured, 23 improved, 8 unimproved and 11 died. Kiefer (22) divides his cases into the non toxic—non sclerotic of which he reports 78% cured, the non toxic—sclerotic of which 50% recovered and the toxic where 38% showed satisfactory improvement and where the mortality was 25%.

In our series of cases only two or 2.3% have been pronounced cured, one after a period of four years and another after two years. 21 cases or 23.8% were unimproved at the time of discharge. 10 patients or 11.4% died; two from perforation with peritonitis, three from severe toxemia, one from sepsis resulting after extraction of teeth, one from broncho pneumonia, one after appendectomy, one after appendicostomy and one after ileostomy. 55 cases or 62.5% were discharged as improved, many of these to return later with exacerbations. The best that we could say for the latter group of cases was that the patients were improved because the degree of involvement of the colon on sigmoidoscopic examination was less on discharge than on admission.

TREATMENT

Many varieties of treatment have been used in the attempt to cure chronic ulcerative colitis. Hare (23) feels that no treatment has been found that acts as a specific cure in an attack of this disease or that will prevent a relapse.

Rest in bed is essential when the patient is acutely ill, i.e. when there is hyperpyrexia, leucocytosis, increased sedimentation rate and very frequent bowel movements. Bargen (4) feels that when the acute infection has subsided the patient should be given some form of occupational therapy to distract his attention from the need of remaining near a toilet room.

Where the psychiatric background has been studied and psychological difficulties or emotional disturbances appear to be of etiological significance, Murray (9, 10), Rechad (30) and Sullivan (8) have found psychotherapy to be of distinct value.

DIET

Hurst (24), Bargen (4), Kiefer (22) and others specify various diets used in treatment. Suffice it to say that the ideal menu consists of a well balanced, high caloric, high vitamin diet, with foods digested mostly in the stomach and small intestine, and leaving as little residue as possible for the colon. Because of the rapid peristalsis there is little absorption in the colon and undigested food in the bowel is a source of irritation. Where hypoproteinemia is present, the protein content of the food should be increased. Synthetic preparations of all the vitamins are used orally, and parenterally when the patient is unable to retain them by mouth.

Many drugs are useful in alleviating the distressing symptoms of the disease but none has as yet been proven to be specific in its cure.

Some form of opium or codeine with belladonna will slow peristalsis and relieve abdominal cramps. Bismuth and kaolin will thicken the contents of the colon and reduce the number of bowel movements. Charcoal will absorb gases and relieve colic. Dilute hydrochloric acid should be given where there is achlorhydria. Iron is prescribed for the secondary anemia.

Stimson (25) advocates the use of sodium ricinoleate by mouth in order to detoxify the upper bowel, and as an irrigation in 1% solution in order to aid the elimination of toxic and infective products from the colon. Its use has not been attended with any improvement in our cases. Castor oil has been recommended for the same reason. Its use is to be condemned. Multiple perforations of the colon with a fatal peritonitis resulted in one of our patients.

Andresen and D'Albora (26), in addition to general methods of treatment, also use mercurochrome intravenously. They begin with a dose of 15 cc. of 0.5% solution and increase the dose at four day intervals sufficient to cause a febrile reaction of 101.5 to 102.5. They cite a number of cures.

The widespread use of sulfanilamide in infection has probably prompted many investigators to use this drug in the hope that it might be the long sought for specific. We have tried it in three cases, using 30 to 40 grains daily in divided doses for a period of two weeks and then repeating the course of treatment after a period of two weeks of rest. We have found no apparent benefit from its use. In fact it may have been responsible for an increase in the diarrhea of one of our cases and for an elevation of temperature in all three where such temperature was not present before its use.

Anti-amebic drugs such as emetine, etc., should be used where there is a question, no matter how slight, of the possibility of the condition being due to amebic colitis.

LOCAL TREATMENT TO THE COLON

Solutions of saline, potassium permanganate, acriflavine, mercurochrome, etc., have been used as colonic irrigations. The rationale of this form of treatment has never been proven. At their best, irrigations will merely cleanse the surface for only a few moments. The infection situated deeper in the coats of the colon cannot be reached by the irrigating solution. Kiefer (22), Hare (23) and Bargen (4) feel that the irritation caused by this form of treatment offsets whatever possible good can be obtained from its use.

Retention enemata have been found to be of value. Hurst (24) recommends the use of tannic acid solution, ½ to 2 grains to the ounce, 1 to 1½ pints to be retained for ½ hour. Kiefer (22) uses a starch and opium retention enema to relieve tenesmus. Crohn and Rosenak (21) use neutral acriflavine (1:4000 parts of normal saline). Fradkin (27) used a mixture consisting of 20% kaolin, 10% mineral oil and 70% of a gel of aluminum hydroxide by retention enema in the convalescent patient where the stools were streaked with blood. As an adjunct to specific therapy this method aided in reducing the number of stools and healing the mucosa. Eyerly and Breuhaus (28) use a retention enema consisting of a three to five ounce mixture of kaolin and aluminum hydroxide in from three to five ounces of warm distilled water. Usually one but occasionally two retention enemata are given daily. The objection to the use of these enemata is the inability of the patients to retain them without marked discomfort for more than a few moments.

Soper (32) uses daily insufflations of calomel and bismuth subcarbonate and reports a number of cures in his early cases of chronic ulcerative colitis. We found no improvement in three of our cases from this form of therapy.

SUPPORTIVE MEASURES

The intravenous use of saline and glucose is indicated in cases of dehydration and starvation accompanying marked diarrhea. In hypoproteinemia with its resultant nutritional edema we have found the use of acacia intravenously to be of value.

TRANSFUSIONS

Bargen (4) uses repeated transfusions of about 200 cc. of blood four to seven days apart to fight the toxemia in cases of severe sepsis and for the anemia and weakness following the loss of blood. Hurst (24) recommends transfusion when the hemoglobin falls below 70%. We have used transfusions in all of our toxic or anemic patients and have found them the best supportive measure to tide the patient over the acute phase of the disease.

SERA AND VACCINES

Hurst (24, 5) and Turner (11) feel that many cases, especially the early ones, respond to specific treatment with anti-dysenteric serum. Crohn and Rosenak (21) use polyvalent antidysenteric serum or typhoid vaccine intravenously. The latter feel that the beneficial results obtained are due to the production of protein shock.

Bargen (4) uses a concentrated serum prepared by immunizing horses against many strains of the diplostreptococci found in the diseased tissues. This is administered intramuscularly in acute and chronic cases until improvement sets in. Following this a bacterin prepared as an autogenous vaccine from organisms found in rectal ulcers in each case is administered subcutaneously. It is given for several months in increasing doses from 0.1 to 1.5 cc. After a rest of several months it is repeated. Three or four such courses are administered or the vaccine is given until the patient is free of symptoms. After that an occasional course of vaccine is given for several years. Hare (23), Bassler (16), Hurst and Dukes (5) do not believe that Bargen's serum and vaccine are of any value. Alvarez (29) states "It is hard to understand why so many able physicians either stand cold before Bargen's achievements or else are actively hostile to his theories and statements of fact. Many men when asked if, in trying to confirm Bargen's discovery of the diplococcus, they used his technique, admitted they had not. Few made a thorough going test of his serum and vaccine. Some did not use these measures, others only half heartedly, because they felt any foreign protein would work as well."

In our series of cases the diplococcus was isolated in 16 and the vaccine prepared and given to 11 patients. One was definitely cured and 10 have shown marked improvement. Typhoid vaccine was used intravenously in three cases, two of which showed no improvement and one slight improvement. Autogenous vaccines were prepared from the stools of 11 patients where no specific organism was isolated; four were improved, seven unimproved.

Bacteriophage prepared from the organisms found in the stool has been recommended by some authorities. Both stock and autogenous preparations have been used. They may be given orally, rectally or parenterally. Three of our cases were treated with

bacteriophage, one through rectal instillation and two by means of subcutaneous injections. Two showed no improvement. In one case a stock bacteriophage was used parenterally in conjunction with an autogenous vaccine. This patient has no recurrence after two years.

SURGERY

All evident foci of infection should be eradicated. Most medical authorities feel that surgery of the colon as a cure for chronic ulcerative colitis should be used as a last resort. Hern (12) feels that "internal anastamoses and the like are too stupid in their conception to be worth more than mere mention." Kiefer (22) feels that transverse ileostomy should be done in cases of extreme toxemia. Hurst (24) feels that surgery should be used only in the complications of chronic ulcerative colitis and that colectomy is the procedure of choice. Turner (11) feels that cecostomy should be done to put the colon at rest. Flick (31) reports a case of five years standing cured by total colectomy. Soper (32) reports six of his cases cured by this operation. One of our patients died after appendicostomy, another after ileostomy. One patient showed no improvement after cecostomy.

We advise against elective surgery in the presence of chronic ulcerative colitis. Two of our patients had appendectomies and three had hemorrhoidectomies performed during the course of the disease, all of which were followed by severe exacerbations. One patient died after an appendectomy.

SUMMARY

The recent literature of chronic ulcerative colitis has been reviewed.

An analysis of 88 cases has been presented.

The youngest patient was seven years of age, the oldest 70 years of age. There were 63 cases (72%) which comprised the groups between 16 and 40 years of age. There were 55 females (62%).

No specific organism was isolated in 63 cases (72%). The diplostreptococcus was isolated in 16 cases (18%), the enterococcus in 6 (7%), a green streptococcus in two, and a hemolytic staphylococcus in one.

Five cases gave definite antecedent histories of "nervous breakdowns."

There were 20 instances of recurrences with the following conditions: Seven with upper respiratory infections, one with pleurisy, five with pregnancy, one after tonsillectomy, one after removal of the teeth, two after appendectomy, three after hemorrhoidectomy.

Many complications were encountered, the most frequent being stricture of the bowel, polyposis, perianal abscess and fistula in ano.

In addition to general methods of treatment 11 patients received autogenous vaccines prepared from the stools of whom four were improved and seven unimproved. 11 patients received an autogenous vaccine prepared from the diplostreptococci isolated from the lesions in the colon of whom one was cured and 10 were markedly improved. Three received typhoid vaccine of whom two were unimproved and one improved. Three received bacteriophage of whom two were unimproved and one apparently cured, the latter receiving at the same time an autogenous vaccine prepared from the stool. Three received two courses of sulfanilamide none of whom were improved.

Surgery of the colon was attempted in three patients; cecostomy without improvement; appendicostomy and ileostomy, subsequent to which the patients died.

All patients with toxemia, hemorrhage or secondary anemia received repeated transfusions.

Two patients (2.3%) were cured, 55 (62.5%) were improved, 21 (23.8%) were unimproved and 10 (11.4%) died.

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