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University of Minnesota

Chronic Ulcerative Colitis

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I. FIRST MONTH

During October the first 4 meetings of the 5th series of Staff Meetings were held in the Recreation Room of the Nurses' Home. The program was as follows: Spontaneous Hypoglycemia (diagnostic problem), Aplastic Anemia (therapeutic problem), Diagnostic Radiological Reports (departmental report), Hand Infections (common condition with review of end-results). The attendance has been satisfactory as exactly 500 staff members and guests came to the four meetings. The discussions have been unusually good and very much to the point, 32 members participating. In addition to the 500 copies of the bulletin printed for the meetings, 260 were sent to our regular mailing list, 240 copies to all teaching hospitals in the United States of 300 beds and over, and 60 given out to various persons after the meetings.

With the exception of one meeting, we have kept within our time limit fairly well. If those who discuss our problems will limit their remarks to a reasonable limit it will be possible for everyone to be heard and for the members to get back to their regular duties at the appointed time (1:15).

Today we have as our guest the Association of American Medical Colleges. We appreciate very much the opportunity of having so many medical leaders with us and we hope they will join in the discussion. We are starting on another round of therapeutic problem, diagnostic problem, departmental report and common condition for November. The subject is best classed as a therapeutic problem -- Chronic Ulcerative Colitis.

II. CASE REPORT

CHRONIC ULCERATIVE COLITIS

Case is white male, 59 years old, admitted to Minnesota General Hospital 2-23-33, expired 7-9-33 (136 days).

Onset: Attacks of Diarrhea

5- -32 - Previously well except for occasional attacks of diarrhea for about 3 years (not severe, recurring about 2 or 3 times a year). Now began having persistent diarrhea with 6 to 8 bowel movements a day. Stools thin, watery, pale yellow color with considerable mucus.

Remission

8- -32 - Several gastro-intestinal studies (private physician). Diagnosis of colitis made. Treatment given (nature not known). Diarrhea disappeared.

Recurrence

10- -32 - Diarrhea recurred as severe as before. No blood in stools. Unable to work because of diarrhea. Lost weight, very weak and has vague abdominal pain.

2-23-33 - Condition same. Persistence of diarrhea. 25 lbs. weight loss. Occasional difficulty in starting urine and nocturia 2 to 3 times a night.

Admitted

Physical examination: Cooperative and intelligent. Mucous membranes pale. Area of dermatitis on upper part of right leg. Heart - blood pressure 112/74, appears enlarged to left, loud systolic murmur at left border of sternum (not transmitted). Lungs - negative. Abdomen - colon visible and palpable in upper part of abdomen, no induration or masses, slight tenderness over colon, inguinal hernia on each side. Extremities - pitting edema of ankles, watch crystal nails of upper and lower extremities. Rectal - large external hemorrhoids, normal prostate.

Laboratory

Urine - negative. P.S.P. - 2 examinations - 45% and 50% excretion. Blood - Hb. 84%, wbc's 7, 150, Pmn's 68%. N.P.N. - 34.3 mgs. Stool - benzidene 4+, urobilin +, numerous wbc's, occasional rbc's, no parasites. Agglutination test - typhoid, paratyphoid A. and B., tularemia; melitensis - negative; repeated and result checked.

Electrocardiogram

Split P2 and P3. X-ray - R.U.B.,

colon - Barium enema went through colon without any trouble, it poured right on to cecum into ileum so that cecum was not distended very well. No evidence of disease. Kidney shadows fairly well seen. No evidence of disease.

Gastrointestinal Study

Negative stomach and duodenum. X-ray of chest - negative. X-ray of sinuses - both maxillary sinuses (almost completely blocked) show marked degree of chronic sinusitis. 6 Ft. Heart and esophagogram - negative. Repeated X-ray of colon - On barium examination, there was spasticity of sigmoid portion of colon and roughening along upper part of transverse colon which might suggest an early ulcerative colitis. X-ray of gall-bladder-negative.

Special consultations:

Dentistry - marked caries and pyorrhea.

Dermatology - dermatitis on anterior surface of leg (senile pruritis).

Surgical - both internal and external hemorrhoids present; surgical treatment not advised until colonic condition clears up.

Rhinology - both maxillary antra and walls examined - no pus obtained.

Eye - refraction - right 20/15 - 1, left 20/20 -3. Fundi - media clear, O.D.- disc slightly blurred, number of hylaine exudates in deep portion of retina just nasal to disc and along inferior nasal vessels about 2 disc diameters from disc; larger arteries are irregular in caliber and slightly more tortuous than normal. O.S. - disc also somewhat blurred, medium sized cotton-wool exudate along inferior nasal vein near disc;larger arteries irregular in caliber and more tortuous than normal. Impression - arteriosclerotic fundi.

Psychiatric consultation - impression that patient is very depressed, although he had no desire to die. Could be a potential suicide and should be considered so. (Day this opinion was rendered, attempted suicide. He slashed left side of throat with razor. Taken to operating room where bleeding was controlled and wound sutured).

Cystoscopic examination under local anesthesia - catheterized specimen from bladder showed no pus but numerous rbc's. Median intravesical and lateral intraurethral prostatic hypertrophy, grade I. Trabeculation II of bladder. Ureteral orifices normal. Indigo-carmine appears within normal limits. Diagnosis - early benign hypertrophy of prostate with beginning obstruction.

Stool cultures negative

4-21-33 - During 2 months interval, condition same except for progressive weakness. Hemoglobin dropped from 84% to 73%. Numerous cultures of stools gave no information.

No Improvement

5-10-33 - Condition same except for more weakness. Apprehensive. Difficulty in starting urine. Vague pain in abdomen. Temperature chart shows daily rises of slight degree, 100 or slightly higher. Various forms of treatment given, all without any improvement.

7-5-33 - Condition same.

Sudden change

7-7-33 - Pains in right lower chest. Very irritable and moaning.

Cardio-respiratory Failure

7-8-33 - Difficulty in breathing. Chilly. Skin clammy. Great deal of pain in right side of chest and abdomen. Blood pressure 66 systolic. 450 cc. citrated blood given. Very weak. Rales in right base.

7-9-33 - Temperature 102. Complains of weakness and pain. Cyanosis present. Some dyspnea. Later in day, abdomen distended, rigidity and tenderness present. Weak pulse. Dyspnea and cyanosis increased. Unconscious. 10:40 P.M. - Expired.

Autopsy

Poorly nourished

Body is well-developed but poorly

nourished, white male, 59 years of age, measuring 164 cm. in length and weighing 110 lbs. Rigor present. Hypostasis purplish and posterior. No jaundice. Slight edema about ankles. Some cyanosis of base of neck and upper part of chest. Pupils equal. Ragged wound in left side of neck below jaw (attempted suicide) which is well-healed.

Slight excess of clear fluid in Peritoneal Cavity. No evidence of peritonitis. Two small herniae present in inguinal region, readily admit finger and are about 3 inches in length. Appendix not inflamed, hangs free.

Bloody fluid

Pleural Cavities free of adhesions. Slight excess fluid present on left side; right contains about 100 cc. slightly blood-tinged fluid. Pericardial Sac contains small amount of clear fluid.

Brown Atrophy

Heart weighs 300 grams. Very marked brown atrophy present. Musculature of dusky brown color. Large epicardial plaque over anterior surface of left ventricle. Musculature of heart somewhat soft. No areas of fibrosis or thrombosis. Mural endocardium smooth. Valves well-formed. No recent or old endocarditis. Root of Aorta shows no change. Coronaries are soft. Few atheromatous plaques in large branches.

Embolus, Infarction

Right Lung weighs 900 grams, Left 650. Lower lobe of right lung completely infarcted, being nodular, very dark, on cross section intensely infiltrated with blood. Dissection of artery to this lobe shows complete obstruction with old antemortem clot immediately beyond bifurcation of right pulmonary artery. Upper lobe on this side is free of emboli and well expanded. Moderate degree of collapse present in lower left lobe, with minimal bronchopneumonia. Considerable edema and engorgement.

Spleen Small

Spleen weighs 150 grams, not very large or particularly soft. Markings well retained. Moderate amount of pulp can be scraped away.

Fatty change. No abscesses

Liver weighs 1800 grams, soft yellowish and markings somewhat faint. No abscesses. Periportal spaces appear normal.

Gall-Bladder has thin wall. No stones. Ducts not dilated.

Colitis, Superficial Ulceration. Inflammatory Polyps.

Gastro-Intestinal Tract: Esophagus, stomach and duodenum show no ulceration, polyp, or tumor. Small bowel somewhat thickened and edematous. Several coils of small bowel show slight redness of mucosa (patchy). No ulceration. Lymphoid follicles not hyperplastic. Beginning in cecum and extending through colon to mucocutaneous junction of rectum, a well-developed colitis is present. Bowel is edematous. Mucosa is red in a patchy, irregular manner. Sigmoid portion of colon somewhat more involved than upper large bowel. Several dark, circumscribed polyps present. One of these has a small pedicle. Several dark areas of ulceration in upper colon as well as in sigmoid. Ulcerations measure about 1 cm. in diameter up to 1.5 cm. in length. They are superficial and appear grossly like a circumscribed atrophy of the mucosa rather than a true ulcer. No undermining or submucous tracts present. No abscesses.

Pancreas shows no fibrosis, tumors or cysts.

Adrenals well-developed and show no adenomas.

Pyelitis, Prostatic Hypertrophy

Each Kidney weighs 160 grams. Capsules trip easily. They are quite pale. No abscesses. Cortices and pyramids well differentiated. Ureter and pelvis not inflamed or dilated on right side. Left ureter and pelvis show definite pyelitis, evidenced by purulent urine and thickening and injection of mucosa of pelvis. Bladder shows very slight trabeculation. Wall is thin and shows no cystitis. Small projection into bladder from prostatic area and very slight enlargement of prostatic

lobes (lateral).

Aorta shows minimal atheromatous change, particularly in lumbar region.

Lymph Nodes show very little hyperplasia. Search for lymph nodes to culture but none large enough found.

Organs of Head and Neck - not examined.

Source of Emboli

Veins: Femoral and pelvic veins milked upward, several thrombi found in prostatic plexus and several fragments of old clot in right femoral vein.

Microscopic

Colon: 4 sections show uniform process. Ulceration is of patchy type. Over most of mucosa, bottoms of glands are retained. Foci of complete loss also present. Entire mucosa infiltrated with mononuclears and few neutrophiles. Occasional hyperplastic lymphoid follicles present. There is marked engorgement of vessels without any thrombosis. Submucosa markedly fibrotic and thickened, muscularis and serosa less so. Numerous large collections of mucus present in submucosa forming what might be called mucous cysts. No amebae seen.

Liver: extensive fatty replacement.

Lungs: advanced bronchopneumonia.

Spleen and lymph nodes: hyperplasia. No infiltration with large mononuclear cells.

Heart, pancreas, adrenal, kidney, prostate - no significant change other than noted above.

Cultures: (B.Olson) (Ulcers, spleen, liver, bile) Rosenow's technique for isolation of Bargen diplococcus carried but none recovered.

Diagnosis:

1. Chronic non-specific ulcerative colitis.
2. Inflammatory polyps of colon.
3. Thrombosis of prostatic plexus and right femoral vein.
4. Pulmonary embolism and infarction.
5. Brown atrophy of heart.
6. Acute bronchopneumonia.
7. Fatty change of liver.
8. Cloudy swelling of kidneys.
9. Pyelitis, left.
10. Prostatic hypertrophy.
11. Depressive psychosis (clinical).
12. Attempted suicide.
13. Inguinal herniae.

III. ABSTRACT

CHRONIC ULCERATIVE COLITIS

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Introduction

The various publications on chronic ulcerative colitis present an odd contrast. Some groups consider the question of etiology and treatment solved while others believe that they are still unsolved problems. We hear of the "conquering hero" who has not yet appeared and the field saturated with the blood of best human efforts.

Regardless of conflicting views, the Mayo Clinic group commands most attention because of the large series of cases studied.

History

1829-1842, Cruveilhier's text illustrates a case of chronic ulceration of colon.

1875, Wils and Moxan described disease in lectures.

1885, Allchin, first demonstration of autopsy specimen.

1888, White, first comprehensive study (clinical).

1909, Allchin, review of pathology.

1919, Logan and Bargen revive interest with bacteriological studies.

Etiology

The term chronic ulcerative colitis has apparently come to stay in spite of its objectionable features. The term by common consent carries the implication that the chronic ulceration of known conditions, such as tuberculosis, amebiasis, dysentery, tumors, etc., is excluded. The general run of cases can be divided into two groups: (1) true chronic ulcerative colitis and (2) cases of specific disease masquerading as the former. (The latter group is not insignificant). Chronic bacillary dystentery and amebiasis are finally diagnosed in many cases often after long study. It appears from the reports that the isolation of the causative organisms may be so difficult that often it is only a happy accident. It is the consensus of opinion that in any series of cases a small percentage will be of this type. The view that dysentery and amebiasis are rare in the northern states can no longer be upheld. This is borne out by the amebic cases seen at the Mayo Clinic. In 1101 cases between 1921 and 1931, the percentage per individuals examined was: Florida .19, Alabama .15, Mississippi .27, Louisiana .16, Texas .12, Arizona .18, California .18, Minnesota .04.

Illinois .19, Iowa .12, Wisconsin .06, Indiana .16, Michigan .16, N. Dakota .10, Canada .12. Mackie feels that bacillary dysentery in its chronic stages is frequently called chronic ulcerative colitis. In 935 convalescent cases of dysentery, he found 52 Flexner and 13 Shiga carriers. 20% of the former and all the latter became permanent carriers. Negative cultures mean little. 85% of cultures in the chronic forms were negative in the Flexner group. Agglutination tests were negative in 20%. Flexner types are frequently sporadic. This author feels that these cases are much more frequent than is generally appreciated.

Regarding the cause of true ulcerative colitis, there are two general theories: (a) Non-specific, and (b) specific infection (aside from above). The majority of writers believe in the non-specific theory. The pathogenesis, predisposing factors and other conditions advanced by advocates of the specific-germ theory are accepted but the organism itself is considered to be non-specific. Any one of the normal inhabitants of the bowel or symbiotic combinations of them under suitable conditions may be the exciting factor according to this theory. The supporting evidence may be summarized as follows:

1. Failure of the "specific" serum to give uniform results.

2. Failure to recover the "specific" organism in all (or even majority of) cases.

3. Occasional success of non-specific polyvalent serum (Streicher serum, etc.).

4. Finding of other occasional causes (such as dysentery bacilli, amebae, tumors, etc.).

The Logan-Bargen organism is called by them the diploccoccus of ulcerative colitis. This theory is based on a large series of cases. The work began in 1919. Between Jan. 1930 and Aug. 1931, 472 cases were treated with the "specific serum". Extensive experimental and clinical studies have been done. The organism is a diplococcus closely fused in pairs almost merging into short chains. It is lancet shaped, slightly larger than the pneumococcus, gram-positive and not bile-soluble. The cultural reactions are characteristic. On blood agar, it forms a green halo with a faint rim of hemolysis. On mannite agar, the colonies are fine and translucent in contrast to the large opalescent colonies of the related (?) enterococcus. It does not grow on plain agar; it contains a specific polysaccharide. It ferments dextrose, lactose, saccharose, maltose, raffinose and salicin. It does not ferment inulin and mannite. The enterococcus is heat resistant while the specific diplococcus is not. The primary culture must be taken from the base of an ulcer and not from the feces. It must be made in a medium with diminished oxygen tension. The Rosenow tall dextrose brain broth is ideal for this purpose. On the basis of these characteristics, the organism can be readily identified. Other workers than Bargen's group state that they have no difficulty in identification when the organism is present.

The immunological reactions are being studied. Agglutinins and precipitins are formed in experimental animals. In 32 human cases, 15 showed agglutination in dilutions varying from 1 : 40 to 1 :320. The presence or absence of agglutination in normals is not stated. Various skin reactions are present. These also are positive for S. fecalis and the enterococcus but the results of similar tests on normals is not stated. In 57 cases, 50 normals "exhibited local skin reactions of varying intensity to the diplostreptococcus of chronic ulcerative colitis, or to S. faecalis, or the enterococcus or to all." The skin reactions are "not constantly commensurate with the gravity of the disease. In this group of 57 cases, 12 gave positive tests before treatment and negative ones after treatment." It is claimed that "this study furnishes additional data supporting (the) contention that the disease is of specific etiology."

Specific localization is claimed

for the organism. Of 459 rabbits injected intravenously, 268 (58%) "gave evidence of disease of the large intestine" (marked diarrhea, hemorrhage, ulceration). Diplococci, "in some essentials like those found in cases of chronic ulcerative colitis" isolated from normals (16 times in 98 persons, 16%) produced similar changes in only 2% of animals. In 21 of 25 dogs (84%), more striking results were obtained with the "specific" organism.

Incidence of Organism

In approximately 80% of the patients examined over a period of several years, organisms of this type have been recovered.

Conflicting data

Apparently the majority of writers of the last few years do not accept these findings. Various reasons, cited above, are given. Many of these reports are based on isolated groups of cases and details are not given. Rafaky and Manheims, however, give adequate details of their study. 314 patients suffering from a variety of conditions were studied. The distribution and incidence of the Bargen diplococcus and the enterococcus is summarized as follows:

30 Ulcerative Colitis cases
- Feces culture - none made
- Proctoscoped cases - 30
  - Enterococcus - 28
  - Bargen diplococcus - 2 (7%)

111 Spastic Colitis cases
- Feces cultures - 89
  - Enterococcus - 89
  - Bargen diplococcus - 0
- Proctoscoped cases - 22
  - Enterococcus - 20
  - Bargen diplococcus - 2 (9%)

173 Spastic colitis cases
- Feces cultures - 132
  - Enterococcus - 123
  - Bargen diplococcus - 1
  - Neither - 8
- Proctoscoped cases - 36
  - Enterococcus - 36
  - Bargen diplococcus - 0
- Stomach cultures - 4
  - Enterococcus - 0
  - Bargen diplococcus - 1

Bargen's group find the organism in areas other than the colon. These are said to constitute the foci for the colonic spread. The great difference is in the percentage of cases in which the "specific" organism is found - Bargen, 80%; authors, 7%. The fermentation reactions, according to these men are similar to those of the enterococcus. In animal experiments, the results (on small series) were radically different. 5 rabbits - 1 control, 2 intravenous Bargen diplococcus, 2 intravenous enterococcus. All remained healthy and at autopsy showed no gross or microscopic lesions. In another series 10 rabbits were used. 4 intravenous Bargen diplococcus, 4 intravenous enterococcus, 2 controls. In all ten, no diarrhea developed and none showed lesions at autopsy.

These authors conclude that "the Bargen diplococcus is a strain of the enterococcus group. It is not regarded as the specific factor in ulcerative colitis--".

The status of a specific causative organism in chronic ulcerative colitis may be summarized in the statement that the Bargen group with over 10 years experience with a large group of cases and with extensive experimental investigation believe their organism is the specific cause whereas nearly all other investigators usually working with smaller numbers of cases have not been able to confirm Bargen's findings.

Predisposing Factors

Larson personally interviewed 233 cases. His results are tabulated as follows:

Sex	Male	100
	Female	133

Age:	Decade	Number
	1	5
	2	22
	3	58
	4	69
	5	46
	6	23
	7	7
	8	3

Range: 20 Months to 73 Years

Maximum: 73% between 20 to 50 yrs.

Nationality, residence: No significance.

Definite preceding (predisposing) Conditions present in 124 (53%). These conditions may be divided into 3 groups:

A.	Infectious group	52
	Definite infection	31
	Influenza	11
	Head colds	2
	Sore throat	2
	Appendicitis	1
	Otitis media	1
	Sinusitis	1
	Whooping cough	1
	Pleurisy	1
	Cholecystitis	1
	Epidemic jaundice	1
	Removal of focal infections	3
		107
B.	Low resistance of bowel	
	Constipation, cathartics, etc.	18
	Acute diarrhea (after bad food or water)	13
	Amebic Dysentery	8
	Acute diarrhea (undetermined origin)	6
	Perineal abscess	3
	Hemorrhoids	2
	Anal fistulae	2
		62
C.	Miscellaneous	
	Psychic	6
	Pregnancy	4
	Change in climate	3
	Urethral dilation	1
	Injestion of bottleful of (Rid-O-Fat"	1
		15

Acute flare-ups were attributed to definite cause in 60%. Infection was the most frequent cause. Some (23) cases gave as causative factors such conditions as menstruation, chilling, hay fever, migraine, etc.

Impression: The proof of an actual relation between the colitis and those various factors is extremely difficult but it appears that infections of various sorts may act as predisposing factors.

Pathology

The pathology is of additional interest because the changes can be observed through the proctoscope and these observations are a factor in diagnosis.

The process begins in the rectum and progresses toward the cecum. Usually the progression is by continuity but occasional cases show patchy or even isolated areas of involvement. In about 700 cases at the Mayo Clinic, 23 showed this localized type of process without involvement of the distal colon.

The earliest change consists of edema and congestion of the mucosa. This bleeds freely on manipulation. Localized areas of reddening progressing on to tiny abscess formation next appear. Histologically, these areas are preceded by a hyaline plugging of the vessels beneath the point of reddening. The nature of this plugging is not known. It is seen in other types of colitis. The abscess is a true suppurative process. The abscesses rupture and produce ulcers. There is a marked tendency to confluence. Irregular ulceration with undermined edges and tunnels are produced.

The discharge is purulent with small amounts of mucus. The mucosa between the ulcers is characteristic: edematous, congested and bleeding freely on the slightest trauma. The bowel wall becomes thick due to edema and fibrous reaction. Later, contraction begins. The lumen becomes narrow at the most involved areas, usually first at the

rectum and frequently as one or more separate strictures higher up. Loss of haustrations and the obliteration of the bulging ampulla of the rectum is characteristic. In late stages, the colon is transformed into a thin straight tube making right angle turns at the spleen and liver, with a thick fibrous ulcerated wall.

Complications are local and distant. The ulcerated tags of mucosa often become transformed into inflammatory polyps. Strictures have been mentioned. Between these, huge pockets of pus may collect. Perforation can occur. Perirectal or pericolic abscess, mesenteric thrombosis are other complications. Malignant degeneration of the polyps may occur.

Secondary changes

both local and distant are as follows:

693 cases

Polyposis	69
Strictures	59
Arthritis	30
Perirectal abscess	26
Neurologic changes	25
Perforation	23
Cutaneous lesions	17
Malignancy	15
Splenomegaly	10
Nephritis	8
Endocarditis	7
Ocular disease (inflammatory)	5
Hemorrhage (fatal)	3
Mesenteric thrombosis	3
Renal calculi	2
Tetany	1
	278

Mental changes "many cases"

Differential Diagnosis

On gross appearance of mucosa between chronic ulcerative colitis, chronic dysentery, amebiasis and tuberculosis is often difficult. When late, markedly involved cases are observed, gross differentiation may be impossible. The characteristic features are limited to the very early lesions. Those of chronic ulcerative colitis consist of reddened spots with a central miliary abscess or ulcers. The early lesions of dysentery are found on the tips of the mucosal folds and are closely set ulcerations (not abscesses). The confluent ulcers, therefore, are transverse to the bowel axis and on the tips of the mucosal folds. In amebiasis, the earliest lesions are small necrotic plaques (membrane) which can be pulled away from the ulcer. Tuberculosis is characterized by primary involvement of the submucous lymph follicles and lymph vessels with subsequent ulceration. In this disease the intervening mucosa, characteristically, appears normal and does not bleed on manipulation. The site of involvement usually is not of much value. Dysentery and chronic ulcerative colitis show maximum involvement in the lower colon; tuberculosis and amebiasis in the upper half.

Tuberculosis, however, rarely involves the bowel primarily. Apparently several years ago chronic ulcerative colitis was erroneously called tuberculosis. Martin at the Chicago Municipal Hospital (tuberculosis) found that lower colonic lesions were rare except in advanced pulmonary tuberculosis. In 930 private patients with all varieties of rectal complaints, he found only 2 cases of tuberculous colitis. Among 150 tuberculous cases, in those without intestinal symptoms, there were no colonic ulcers; in those with mild symptoms, there were 1.3% ulcers and in those with advanced symptoms, there were 25% ulcers (proctoscopic examination).

Intestinal localization in tuberculosis is summarized as follows:

	Fenwick and Dodwell		Goldberg, Sweany and Brown
	Incidence %	Incidence as sole G.I. lesion	
Duodenum	3.4	0	3.8
Jejunum	28.0	1.4	21.2
Ileum	60.2	4.4	83.2
Cecum	--	--	87.0
Ascending colon	51.4	1.8	--
Transverse colon	30.6	1.0	--
Descending colon	21.0	0	--
Colon	21.0	0	71.0
Sigmoid	13.5	0	16.3
Rectum	14.1	0	16.3

Clinical Features

Individual cases of the disease present a variable picture due to differences of acuity, extent, complications and reactions of the patient. The onset is insidious with the most marked remissions during the winter. Later, the symptoms are continuous. The intractable rectal discharge of blood, pus and mucous mixed with feces is the foremost symptom. Pains, cramps, tenesmus, distention, abdominal paraesthesias, malaise, weakness, loss of weight, anemia, fever and aches in other parts of the body mingle together in the individual cases. On proctoscopic examination, the pathological changes outlined above can be seen. In late cases bizarre combinations are found.

Roentgenoscopic Studies

In the average uncomplicated case, characteristic features are present. The rectum must receive closest attention since in 20% of cases the disease is limited to this area. The ampulla is small and straight. Transverse striation (spasm) is seen. In the colon itself, the barium fills to the cecum "in an instant". All redundancy is gone, the diameter and length is decreased. Haustrations are absent. Spastic areas are frequent. The destruction of mucosa is variable in extent and depth and the contour therefore varies extensively from fringed to smooth. The regional or localized areas of ulceration, especially when unassociated with disease in the distal colon, present the most difficult problems for diagnosis. Tuberculosis or tumor is usually suspected. When complicating features of stricture, polyps, etc. appear the roentgenologic examination becomes more complicated.

Treatment

"There have been almost as many types of treatment for chronic ulcerative colitis as there have been contributors to the literature on the subject." Most of the contributors would agree with the following statement by Bargen: the rational treatment must rest on the facts: (1) that the condition is an infectious disease of the large intestine; (2) that uncomplicated cases constitute medical problems; and (3) that certain complications are definite indications for operation. The methods of medical treatment advocated by this group, however, are not entirely accepted.

Of the non-specific forms of treatment, there is no data found to indicate preference for any one type. A partial list of these forms of therapy is as follows: non-specific polyvalent serum (Streicher serum and others), anti-dysentery serum, parathormone and calcium, "ionization" of colon with copper sulphate, foreign protein "shock" therapy, antiseptics (usually dyes) by mouth and unnumerable forms of rectal irrigations. The most commonly used are dyes, silver nitrate, mercurochrome, argyrol, acetyl-tannic acid (tannigen), copper sulphate, plain water, saline solutions, bismuth, olive oil, witch-hazel, etc. Supportive measures and bland non-residue high caloric diet are also given. The German literature cites numerous cases benefitted by

transfusions. In a circular letter to various clinics, the results were encouraging in the mild cases of not too long duration. The benefit was attributed to the various factors which could be expected from frequent transfusion.

Specific Serum Treatment

Bargen and his associates (Logan and others) have developed a "specific antibody serum" (concentrated serum). It has been used in approximately 700 cases.

The serum is given intramuscularly every 9 to 12 hours beginning with .1cc. and increasing each injection by .1 cc. until the average maximal amount of 3 cc. is given. The duration of treatment and other details apparently are not yet standardized. When improvement occurs, the patient is given an autogenous (?) serum. Administration is continued for 6 to 8 weeks, followed by a monthly course each four months during the symptom free period. This is continued for 2 years.

The Statistical Summary of Results is as follows:

Jan. 1, 1930 to Aug. 1 1931 - 472 cases
"Returned to usual occupation - 352 (74.3%)
Free of symptoms (3 to 5 stools per day)- 250 (53%)
Free of proctoscopic and x-ray evidence of disease- 49 (10%)

The presence of 3 to 5 stools per day seems abnormal but when considered in light of the mental and nervous changes induced by the acute illness and in light of the usual amount of mucosal change this becomes quite satisfactory. In addition to the above improved cases, 26 more received 50% improvement, 45 had not yet sufficient treatment and the other 49 did not respond satisfactorily because of complications or inability to take serum.

Surgical Treatment

Operative interference becomes necessary only in the presence of complicating features. Fulminating course, marked suppuration in the bowel wall, abscesses, strictures, polyposis and malignancies call for interference. Three procedures are commonly done: cecostomy (or appendicostomy), ileostomy and colectomy. Cecostomy or appendicostomy for irrigation is generally discredited. No physiological rest to the colon is obtained and the irrigations which can be done through the opening can be done just as effectively through the rectum. Ileostomy with complete deviation of the fecal stream is an accepted procedure in the presence of the conditions stated above. One warning is frequently stated: the colon is not to be handled in any way. Its wall is heavily infected and the manipulation frequently induces peritonitis. At the Mayo Clinic with serum treatment, the number of ileostomies has decreased. Colectomy is a very radical operation. Not many have been done. In these, the mortality has been low. The indications are strictures with severe infection, polyposis and the presence of metastatic infections which are being fed by the heavily infected colon.

Impressions:

1. Chronic ulcerative colitis by common consent is that form in which a specific factor is not demonstrated, e.g., tubercle bacilli, amoeba or dysentery bacilli.

2. In a certain number, a prolonged search may reveal amoeba or dysentery organisms, seldom tubercle bacilli. The attitude toward chronic ulcerative colitis varies from extreme pessimism to optimism. Interest in the disease has been developed during the last few years, chiefly through the work of the Mayo group.

3. There are 2 theories as to its cause (non-specific organisms and a specific organism). Proponents of the first theory accept the Mayo group studies of pathogenesis, predisposing factors, etc. but disagree with the statement that the diplococcus described by Logan and Bargen is the specific pathogen.

4. Bargen's opponents point to (a) the failure of the "specific" serum to given uniform results, (b) failure to recover the "specific" organism in even the majority of cases (practically all workers except Bargen), (c) occasional success of non-specific polyvalent serum, (d) delayed finding of specific factor in some cases (see 2).

5. Bargen has described the cultural characteristics of his organism which other workers have verified with ease. He has found it in 80% of the cases, they in only about 10% of ulcerative and spastic colitis.

6. Most of the other series are very small in comparison with Bargen's.

7. Only 1 fairly large negative study of 314 cases is found in the literature (Rafaky and Manheims). These authors conclude the Bargen bacillus is a strain of the enterococcus group and not a specific factor in ulcerative colitis. Animal inoculations were negative in their experience.

8. Larson, in 233 cases (personal interviews), found little sex difference (a few more females), wide range of age (20 months to 73 years) with most falling between 20 and 50 years (73%). The majority give a story of infections, lowered bowel resistance and miscellaneous factors as predisposing circumstances. Flare-ups are also thought to be on an infectious basis.

9. The disease begins in the rectum and progresses toward the cecum. The process is usually continuous. Edema, congestion and bleeding are early signs. Abscesses turn into ulcers, fibrous thickening develops and a tube-like structure is produced.

10. Some of the complications are polyps, malignancy, strictures, perforation, mental changes, etc.

11. The differential diagnosis in the late cases is often difficult.

12. The clinical symptoms vary. Remissions are usually present in earlier stages.

13. The x-ray findings are fairly characteristic but may be confusing in some.

14. Treatment in uncomplicated cases is medical. Sera, calcium, parathormone, foreign protein, dyes, etc. have been tried. A bland non-residue high caloric diet is helpful. In some, transfusions have been used with success. Bargen claims excellent results with his specific anti-serum: Sure 10%, symptomatic-free 53%, returned to work 75%.

15. When surgical treatment is necessary, complete ileostomy rather than cecostomy is recommended. The danger is peritonitis. Colectomy is said to carry a relative low mortality and is advocated in the presence of polyposis or heavily infected bowel. All operative procedures should be carried out with minimum handling of the bowel.

Rudolph Koucky.

IV. MEETINGS

1. STAFF MEETING

Date: Oct. 19, 1933.

Place: Recreation Room, Nurses' Home.

Time: 12:15 to 1:30

Attendance: 130

Program: Diagnostic Radiology Reports

Presented by:
L. G. Rigler
Jack Sagel

Theme: Activity of Department. (See Bulletin)

2. STAFF MEETING

Date: Oct. 26, 1933

Place: Recreation Room, Nurses' Home

Time: 12:15 to 1:35 PM

Attendance: 131

Program: Hand Infections

Discussion: M. H. Manson
Jas. Hayes
O. H. Wangensteen
H. A. Reiman
Byron Olson
R. G. Green
L. H. Fowler
E. A. Regnier
L. G. Rigler

Theme: M.H.M.: The tuberculous and gonococcal infections do not really belong with hand infections although they were confused with them at first. Earlier incision and more adequate incisions rather than improper treatment should have been the statement in regard to the pre-hospital treatment of our cases. The group, undoubtedly, represents a very selected type. If the Out-patient Department report had been included, we would probably get a different impression. (Demonstrated x-ray film made with metal strips on palms of hand to indicate fascial failure. Pasted adhesive tape on rubber glove to show flexor tendon sheaths. Discussed distribution of spaces and relationship to infection.) Among other things, the flexor sheath of middle index and ring fingers are individual and separate, but often communicate with midpalmar space. A fibrous connection is particularly strong on the proximal side but on the distal side it is less well-formed. The spaces on the back of the hands are less well-defined. It is unfortunate that we are not able to present to you the end-results in the treatment of this group.

J.H.: I hesitate to speak about this subject as I have discussed it so much in the Out-patient Department. I do not know of one instance of my treatment having been followed out in either the Out- or In-patient division. When I was here 22 years ago, I attempted to treat an infected hand by opening up every chamber. We opened many of them before they were infected and caused infection by our procedure. While out in practice, I heard of the Oschner treatment. I made a trip to Chicago and since then I have used this type of treatment in infections of the hand. To date, I have not had any bad results. The dressing is the most important part as it should be air-tight. I believe that the boric acid, phenol and alcohol are able to penetrate the tissues and destroy the bacteria. (He then recited some experimental evidence which he had carried out during the war.) It is unwise to incise before localization has taken place, as by so doing you break down the wall that nature has made. (Slides were then shown, illustrating this point.) It is important to put the gauze next to the skin and the cotton outside of this. One quart of solution should be poured on the cotton. I believe in this way I can abort the majority of infections.

O.H.W.: I agree in principle with Dr. Hayes but do not believe that his method applies in felons and infections of the tendon sheath. In felons, unless early operative interference is made, necrosis of the phalanx may take place. I do not believe that the solutions have the antibacteriocidal effect that is claimed. Acute tenosynovitis should be treated conservatively so that some functional remains. (Illustrated point by showing patient with infection of thumb.) I believe that most of the good effects of packs are from the heat and not from any other effect. Fortunately, there are very few serious infections of the hand. Many of the milder infections undoubtedly respond to conservative treatment.

H.A.R.: I wish to report 2 medical examples of infections of the hand. One, a woman of 50, gave a history of having been bitten by stray cat while

attempting to feed it. Finger became swollen and she developed lymphadenitis. She is in for another condition but has an ulcer on the back of her hand. This is characteristic of tularemia. Second case, a boy, presented a similar story after a cat bite. It is possible that some of the hemorrhages reported today may be due to the hemolytic effect of certain streptococci. Question! Did the cats have the disease or do they catch the rabbit and simply carry the infection on their teeth?

B.O.: One cat was decomposed when sent to us for treatment. We are investigating the second one now. Dr. Green knows more about the subject than I do.

R.G.G.: The cat origin of tularemia is well recognized. We have shown that the cats have the disease and may die of the infection. In studying a certain community, we found very few infections. When rabbits were taken from cats, infection was common. Apparently they are only able to catch the sick ones.

L.H.F.: We get excellent results with hand infections in the Health Service. They report promptly and are easily hospitalized. I have had no experience with Dr. Hayes treatment. With 300 to 400 students a year, during the past 8 years, I have had only one tendon sheath infection. We put on hot packs and when the condition shows attempts at localization we open wide under general anesthesia. I believe I have seen more harm result from late opening than from early. Localized induration with extreme tenderness is sufficient evidence for operative procedure. Do not use ethyl chloride.

E.A.R.: In glancing over the notes, I noticed that iodine is recommended as a prophylactic. I do not believe it is effective. I frequently teach my students that it is just as well to apply it to the door-knob. Apply hot packs immediately and keep them on for 24 hours. This keeps the wound open. I believe many cases are opened prematurely. It is a good thing to hospitalize if you can. At the Minneapolis General Hospital, we usually have 6 or 7 infections at one time in the house. Most of ours come in late. I do not believe it wise to keep a hand dressed for 48 hours without watching it. The question of diagnosis is not very difficult. Once it is established, it should be drained. We see all types of attempted drainage, including through and through stab wounds through the hand. After operation, hot packs are continued or immersion in hot water tubs. This is usually sufficient to prevent superficial sloughs. Early start should be made to rehabilitate the hand. In our experience, osteomyelitis is relatively uncommon, and I am wondering if some of the cases reported as osteomyelitis today were really true examples of this condition. I would like to ask Dr. Rigler to answer this.

L.G.R.: We see quite a few with bone changes. We believe that we are able to distinguish between atrophy and actual bone destruction. It is our impression that bone changes are fairly common.

Gertrude Gunn,
Record Librarian.

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3. N E X T W E E K

TUMORS OF THE LUNG