Cytomegalic Inclusion Disease in the Gastro-intestinal Tract of Adults *

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CYTOMEGALIC inclusion disease, an infection with human salivary gland virus, most commonly affects infants under the age of two years. Distinctive cytomegaly, with the characteristic large intranuclear inclusion, has been found in 12 to 32 per cent of necropsies in children of that age.[3, 17] Cytomegaly is most often an incidental finding in these children, but in one to six per cent generalized cytomegalic inclusion disease is present, and is usually fatal.[12]

The disease is rare in adults and older children, but has been reported with increasing frequency in the past ten years. In 1950, Wyatt [27] could find only nine cases in adults in the world literature, seven localized to various areas of the gastro-intestinal tract, and two disseminated and fatal. In 1960, Symmers [23] was able to collect 34 cases, 19 localized and 15 disseminated. Cytomegalic inclusion disease of adults rarely is primary; rather, it is usually associated with some predisposing condition. Included are diseases of the hematopoietic, or reticuloendothelial systems (neoplastic and non-neoplastic), severe malnutrition, chronic infection, debility, steroid therapy, and treatment with cytotoxic drugs, or ionizing radiation. In the adult, the disseminated form has been known to involve lungs, adrenals, liver, digestive tract, pancreas, spleen and kidney and appears to be uniformly fatal. A *liver-adrenal-necrotizing syndrome* has been described.[13] The localized form usually involves the lungs or the gastro-intestinal tract. The gastro-intestinal form is less frequently associated with neoplasia. For an excellent discussion of all the aspects of this disease, the reader is referred to the review by Nelson and Wyatt.[13]

In the past three years, we have had experience with nonfatal disease in four adult patients, each with cytomegalic inclusion disease (verified histologically) involving the intestinal tract. One patient (Case 2) had proven liver involvement as well as an ileal lesion. In the other three, the disease presumably was localized to the intestinal tract.

In three patients, the intestinal lesion was removed surgically. One of these patients (No. 3, J. B.) has been reported in detail elsewhere [16] but will be briefly reviewed here. The fourth patient (No. 4, R. E.) has a resectable lesion, but is doing well under medical management at present. In this patient, the diagnosis was established by proctoscopic biopsy.

Case Reports

Case 1. (P. S. 77-37-44) This 27-year-old white married woman was admitted to the University of Chicago Clinics on 1-6-61 for evaluation

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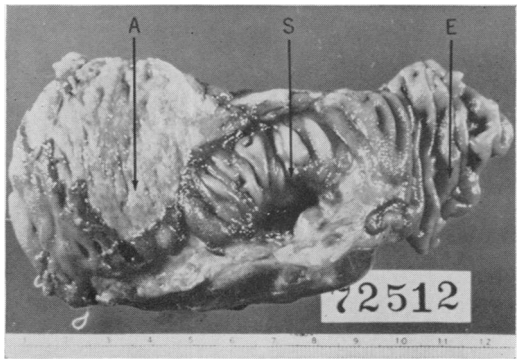

FIG. 1. Surgical specimen from Case 1, with dilated afferent limb and ulcer (A), stoma (S), and efferent limb of normal caliber (E).

of severe abdominal pain, and weight loss of ten-month duration. Beginning seven years prior to admission, she was treated elsewhere for a duodenal ulcer which proved refractory to medical management. A perforation of the ulcer was closed surgically in 1956. In 1959, after repeated hemorrhages, a subtotal gastrectomy was done. Continuity was re-established by means of an antecolic isoperistaltic, Hofmeister type of Billroth II reconstruction. A search for ulcerogenic tumors of the pancreas was made and none found. She then did well, except for progressive weight loss. After several months, however, postprandial epigastric and low back pain began, increased in severity, and was associated later with nausea and vomiting. She denied hematemesis and/or melena. Investigation in the hospital on two occasions failed to reveal the cause of her distress. Her condition deteriorated and she was transferred to the University of Chicago Clinics.

Physical examination on admission revealed an emaciated white female appearing acutely and chronically ill. She weighed 35.5 kilograms. BP was 120/90 mm. Hg; pulse 84; temperature 37.5° C. rectally; respirations 20. Apart from evidence of severe malnutrition, the significant findings were limited to the abdomen. A loop of distended bowel could be seen coursing vertically down the left side of the abdomen. Visibly active peristalsis was frequent and coincided with sensations of cramping abdominal pain.

Laboratory Data: Hematocrit 30%; ESR 32 mm.; WBC 15,600; differential: 78 polymorphs, 15 lymphocytes, 6 monocytes, 0 eosinophils and 1 basophil. Initial urinalysis showed a trace of albumin, 20 RBC/hpf. Subsequent urinalysis was normal. Fasting blood glucose, blood urea nitrogen, serum cholesterol and esters, serum bilirubin, cephalin and thymol flocculations, and thymol turbidity were normal. Serum Na 137 mEq./L., K 5.0 mEq./L., Cl 99 mM./L., Ca 9.9 mg.%, P 3.6 mg.%. Total plasma protein 5.2 Gm.%, albumin 2.7 Gm.%, globulin 2.5 Gm.%. Twelve-hour night secretion and Histalog test failed to reveal free acid. Urinary pepsinogen, expressed as milligrams of tyrosine/24 hours, ranged between 39.53 and 66.24. Postoperatively a Diagnex blue test was positive. Serum carotene was 47 μg.%, and 8.5 μg.% on two determinations. Chest x-ray and barium enema were normal. Plain film of the abdomen suggested the presence of a mass in the left mid-abdomen, an impression sustained by an otherwise normal small bowel examination. Upper GI examination demonstrated a small gastric pouch with a normal functioning gastroenterostomy. Stomal ulcer was strongly suspected, but no crater was seen.

The passage of a Kantor tube was followed by dramatic symptomatic relief. Each time oral feedings were reinstated, however, pain and vomiting recurred.

On 1-21-61, the 16th hospital day, an exploratory laparotomy was performed. A kink was found at the stoma with resultant obstruction of the afferent jejunal limb. This portion of intestine was markedly thickened, dilated and was filled with fluid. At the level of the stoma on the afferent side, there was inflammation and edema. The stoma was resected, including a small cuff of stomach and 6 to 8 cm. of jejunum on each side. A postcolic Billroth II reconstruction was done, and the patient made an uneventful recovery.

The surgical specimen contained a broad flat ulcer in the afferent limb of jejunum, next to the stoma (Fig. 1, 2). In the granulation tissue within the bed of the ulcer, there were numerous cytomegalic inclusion bodies, many within capillary endothelial cells (Fig. 3). The typical inclusion-bearing cells measured up to 40 microns in diameter. The nuclei were enlarged also, measuring 10 μ, or more, and contained a single round, or oval basophilic, or acidophilic inclusion, surrounded by a clear halo. The nuclear membrane was sharply etched. The cytoplasm was usually amphophilic and often contained clusters of the typical cytoplasmic inclusion bodies.

Case 2. (M. M. 63-31-62) This 54-year-old white women was first admitted to the University of Chicago Clinics on 2-12-58 for evaluation of severe diarrhea of 2½-month duration. During that time her stools were watery, foul-smelling, generally small in volume, and varied in number from two to 12 per day. They frequently contained particles of undigested food. Although abdominal

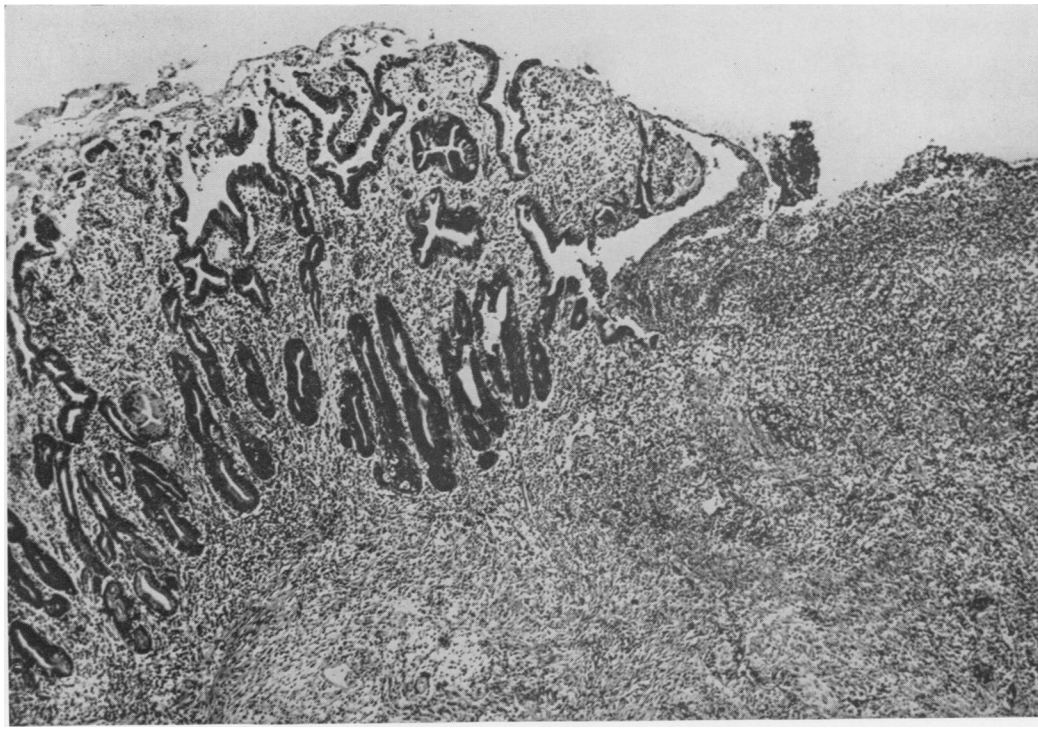

FIG. 2. Chronic ulcer in afferent limb of jejunum, Case 1 (× 60).

pain and anorexia were not significantly present, she had lost 20 pounds since the onset of her illness. Fourteen years before admission, gallstones had been demonstrated. Chest roentgenogram taken in the out-patient department six weeks before admission demonstrated a large anterior mediastinal mass intimately associated with the right side of the cardiac silhouette, and presumed to be a pericardial cyst.

Physical examination on admission revealed a well-developed, well-nourished woman in no acute distress. BP was 170/90 mm. Hg; pulse 98; respirations 20; temperature 36.5° C. There were no significant abnormal findings.

Laboratory Data: white blood cell counts fluctuated between 10,600, and 23,600; red blood cell counts, and differentials were all normal; urinalysis, serum electrolytes, and blood glucose were normal. Urea clearance was 23 by the maximum formula with a blood urea nitrogen of 15 mg.%. Total protein 4.6%, albumin 3.3%, globulin 1.3%. Serum bilirubin, cephalin and thymol flocculations, thymol turbidity, cholesterol, cholesterol esters, and prothrombin time were normal; BSP retention was 36% in 45 minutes, alkaline phosphatase was 9.6 units%. Determinations of fecal fat, xylose tolerance, and serum carotene substantiated the suspicion of malabsorption. Schilling test showed only 2.9% excretion of Co^{60} labelled Vitamin B_{12} which failed to reach normal levels with intrinsic factor. There was achlorhydria after Histalog stimulation; urinary 5-HIAA determination was normal. BMR was + 15. Blood and stool cultures, and examination of stools for ova and parasites were negative. Tuberculin, coccidioidin and histoplasmin skin tests were negative.

Gastroduodenal and colon x-rays were normal. Cholecystogram confirmed the presence of gallstones. Small bowel examination demonstrated changes compatible with malabsorption, but no evidence for regional enteritis. Proctoscopy, and gastric and duodenal cytology were normal.

Therapy for *idiopathic steatorrhea* was instituted. This included Prednisone 10 mg. q.i.d., anticholinergic drugs, and a low-residue, nonlaxative diet. On this program she improved and was discharged on the 53rd hospital day.

Seventeen days later she was re-admitted for evaluation of severe abdominal pain of six-hour duration.

Physical examination showed the patient to be apprehensive and acutely ill. Blood pressure was 136/90 mm. Hg; pulse 130; respirations 18;

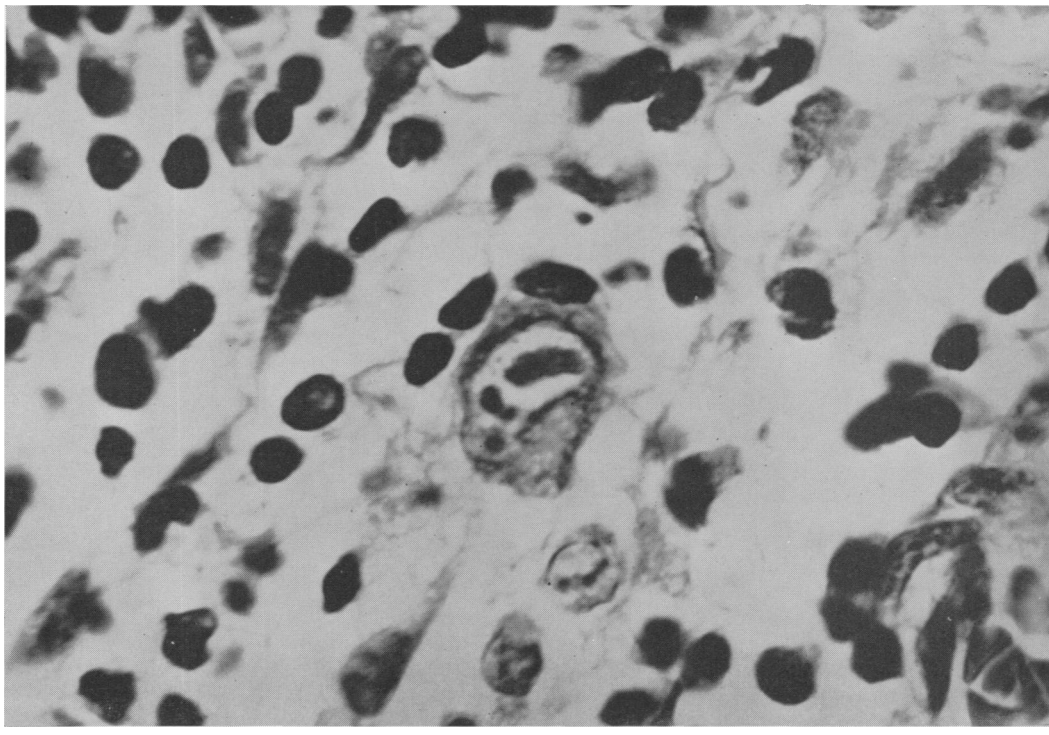

FIG. 3. Cytomegalic cell with inclusion and typical halo, in granulation tissue of ulcer, Case 1 (× 1,650).

and temperature 37.6° C. The abdomen was moderately distended, and direct tenderness was present in both lower quadrants. Rebound tenderness was noted in the right lower quadrant.

White blood cell count was 12,150 with a *normal* differential. Serum carotene level was increased as compared to previous levels and alkaline phosphatase was 5.1 units%. There were no other changes in laboratory data.

She improved with conservative therapy, including the continuation of steroids, but low-grade fever persisted. On the 21st hospital day, an exploratory laparotomy was done. The terminal four feet of ileum were found to be thickened, inflamed and firm. There were enlarged regional lymph nodes. A liver biopsy specimen was removed and approximately 12 inches of ileum representing the transition from normal to abnormal bowel was resected. The diseased distal stump of ileum was closed, and continuity was re-established with a bypassing end-to-side ileo-ascending colostomy. During the first 14 postoperative days, steroids were gradually discontinued. She was discharged improved and afebrile on the 31st postoperative day. She has been lost to follow up.

The surgical specimen was a segment of ileum containing both isolated and confluent ulcers (Fig. 4, 5). In the portion with most severe involvement, the intestinal wall was thickened. Dense infiltration of inflammatory cells was present in the base of the ulcers, and here there were numerous cytomegalic inclusion bodies in the granulation tissue (Fig. 6), many in capillary endothelial cells. Fibrin thrombi occluded some vessels, and angiitis was apparent. Next to the ulcers, the glands were disrupted, with loss of villi and distention of crypts by leukocytes and fibrin. In the surgical biopsy of the liver there were severe reactive changes and fatty metamorphosis. A cell containing a cytomegalic inclusion was present in one portal area.

Case 3. (J. B. 72-84-35) This 18-year-old white boy was admitted to the University of Chicago Clinics in March, 1959, for evaluation of bloody diarrhea of two months' duration. At age six years, the patient had had an episode of recurrent emesis lasting two weeks. He was allergic to sulfonamides. There was nothing else remarkable in the past history. Colon roentgenograms, performed elsewhere six weeks before admission,

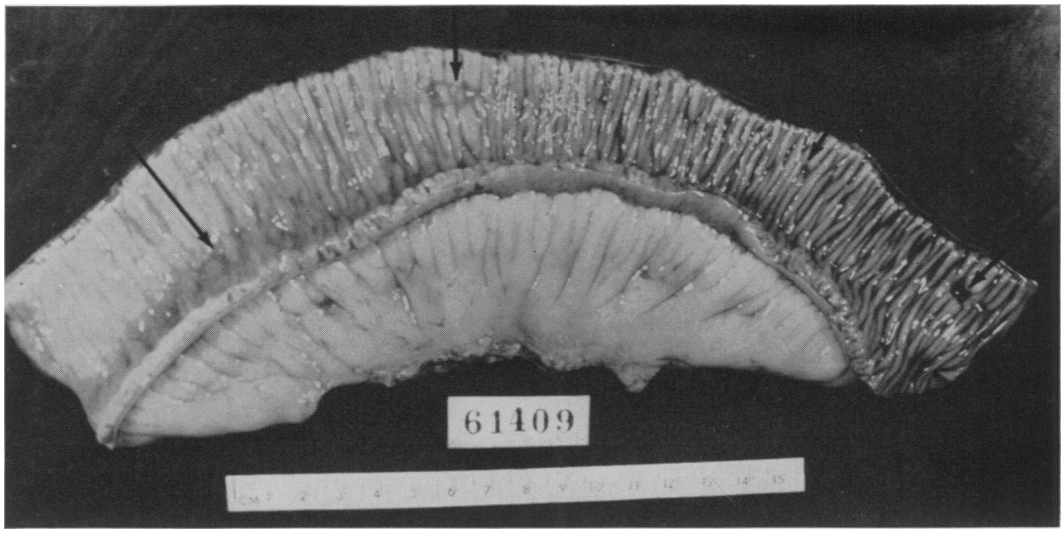

FIG. 4. Ileum from Case 2 with isolated ulcers (right) and confluent ulcers (left).

were reported to be normal. On admission, examination revealed emaciation, pallor, fever, and tachycardia. There was tenderness to palpation with rebound tenderness in the lower abdomen. Rectal examination caused pain. Other physical findings were normal. Laboratory studies included: Hemoglobin 12 Gm.; hematocrit 35%; white blood cell count 14,700; urinalysis normal;

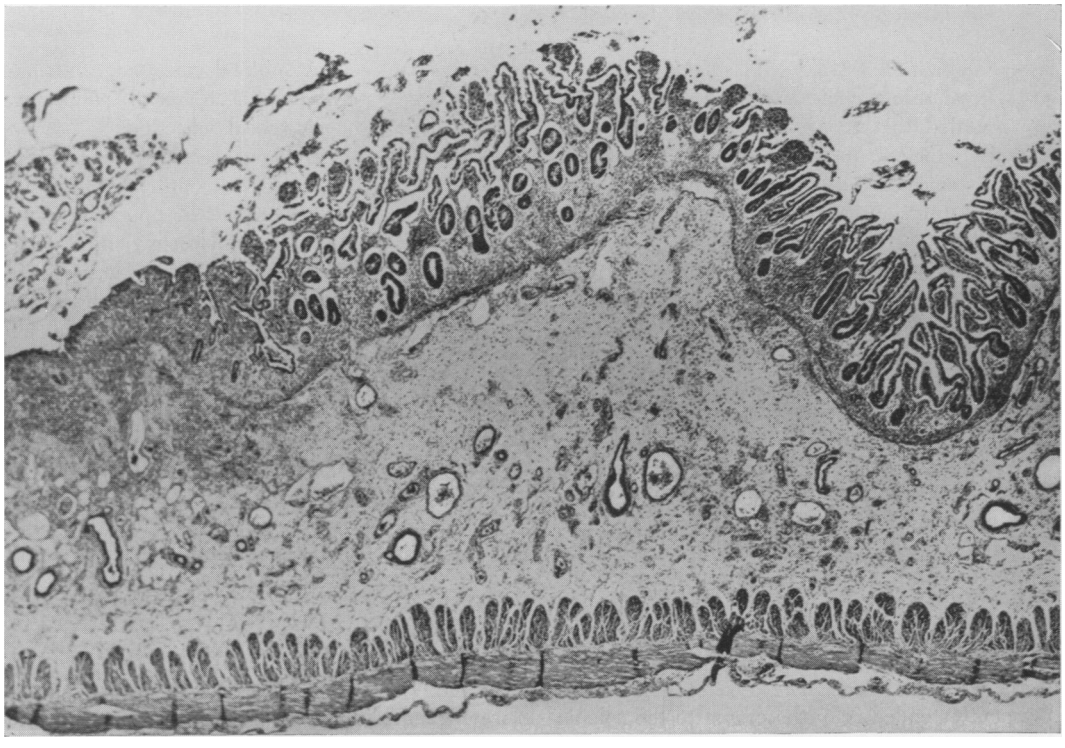

FIG. 5. Ileum from Case 2 with margin of ulcer (left) and inflammation and edema (× 21).

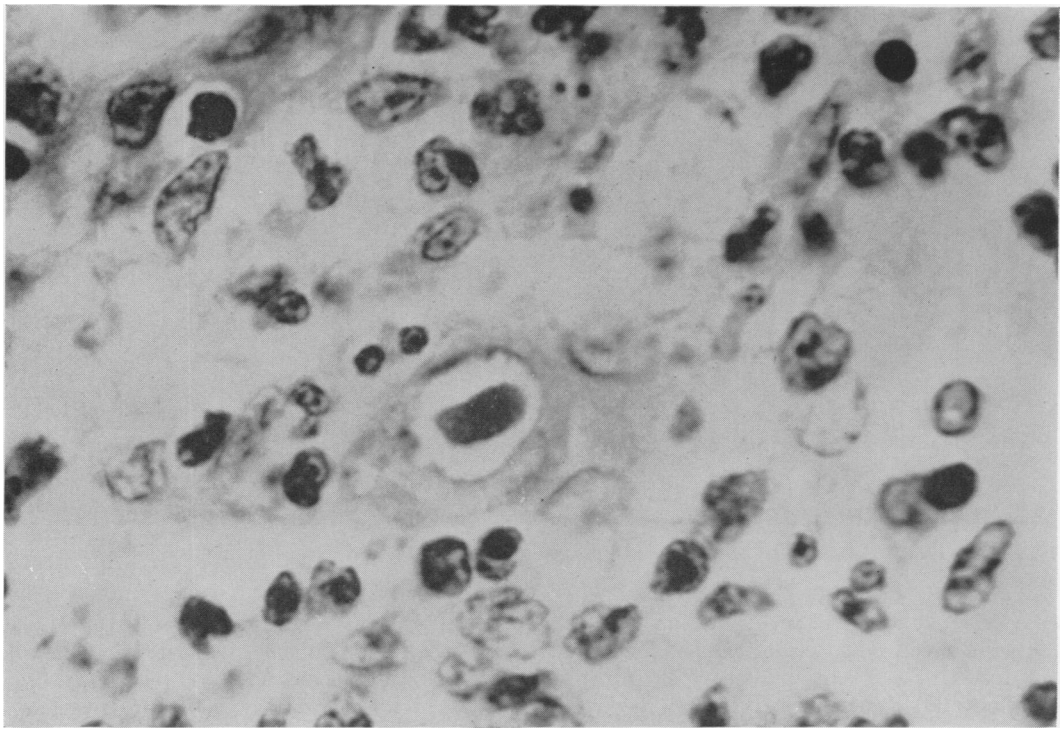

FIG. 6. Typical inclusion-bearing cell in base of ulcer of ileum, Case 2 (× 1,650).

plasma albumin 2.9 Gm.%, plasma globulin 2.4 Gm.%, total serum cholesterol 80 mg.%. Proctoscopy revealed that the rectal mucosa was *beefy red* and friable. Stool examinations were negative for pathogenic bacteria, ova and parasites. The clinical impression was nonspecific ulcerative colitis.

The patient received intravenous glucose, electrolytes, vitamins, antibiotics, and hydrocortisone 150 mg. daily, but diarrhea persisted. Following the administration of intravenous ACTH, 30 units daily, diarrhea and abdominal pain gradually improved. In his seventh hospital week, fever, abdominal pain, and bloody diarrhea recurred. Despite maximal measures, including intravenous antibiotics, hydrocortisone, 250 mg. daily, whole blood, and plasma, his status deteriorated. There was massive blood loss per rectum. Surgery was necessary during the patient's ninth week in the hospital.

At operation, the entire colon was thickened and edematous with a clear serosal exudate. The haustra were preserved. The area of greatest involvement extended from the cecum to the splenic flexure; the sigmoid and rectosigmoid appeared least involved. No perforation or abscess formation was encountered. A subtotal colectomy and ileostomy were performed. The rectal stump was closed below the peritoneal reflection.

During the period of recovery, attempts to culture salivary gland virus, utilizing specimens of colon frozen at the time of operation, specimens obtained during proctoscopy nine weeks after the operation, and urine were unsuccessful. Adrenal cortical steroid medication was gradually reduced in dosage, and then discontinued nine weeks following surgery. The patient's appetite gradually improved, his weight increased, and increasing ambulation was well tolerated.

Improvement was sustained and additional weight gain occurred. In the fall of 1959, the patient was able to return to school. The patient's most recent examination in February, 1961 (21 months following surgery) revealed that the rectal tissue was thickened; bloody discharge was not evident. The patient is now working.

The resected colon contained more than a dozen ulcers, and there was bullous edema of the remaining mucosa. Typical cytomegalic inclusions were abundant in granulation tissue of the ulcers and in vascular endothelium, with associated angiitis and necrosis. Acute, subacute and healed

ulcers were present. The appendix was ulcerated also, but the terminal ileum was not, and it had neither ulcers nor scars.

Case 4. (R. E. 76-73-92) This 60-year-old white married man developed acute cramping abdominal pains and bloody diarrhea on 9-11-60 while on a fishing trip. No other member of the group had similar symptoms. The diarrhea did not respond to the use of opiates. Other than a mild anorexia there were no systemic symptoms. A past history of pneumonia with empyema surgically drained at age 17, poliomyelitis with minimal residual at age 36, and a family history of tuberculosis proving fatal to the patient's mother were elicited. The patient's physical examination was within normal limits. Temperature was 37.5° C. On admission, 10-14-60, laboratory studies revealed white blood cell count 5,900 ranging from 2,700 to 10,000 during the hospitalization, with 70% lymphocytes, 8 monocytes and 5 eosinophils, and 12.7 Gm. hemoglobin with a hematocrit of 39%, but falling and requiring 2,000 cc. of whole blood during the first four weeks of hospitalization. Urinalysis was normal; total plasma proteins were 5.3 Gm.% with albumin 2.9 Gm.%, globulin 2.4 Gm.%. Examinations of the stools were negative for pathogenic organisms, ova and parasites. The diagnosis of ulcerative colitis was confirmed by the presence of gross blood in the stool, the proctoscopic appearance of an extremely friable and edematous mucosa, and the roentgen appearance of the colon demonstrating sigmoidal serrations and loss of the haustral pattern.

After completion of the diagnostic studies, the patient was treated with a bland diet, sedation, antispasmodics, and Azulfidine without significant improvement. His course was characterized by uncontrollable bloody diarrhea, daily temperature spikes to 39° C. with negative blood cultures, progressive anemia and weight loss. On the 11th hospital day, a dendritic ulcer of the left cornea developed. On the 16th hospital day, 120 units of intramuscular ACTH, blood, plasma, and antibiotics, including Chloromycetin and Erythromycin, were given daily without subsequent alteration of the patient's course. Proctoscopic biopsy specimen on the 19th hospital day revealed numerous inclusion bodies within the rectal mucosa, some in vascular endothelium, and changes compatible with ulcerative colitis (Fig. 7, 8). On the 32nd hospital day, the patient was given 200 mg. of hydrocortisone intramuscularly daily and the

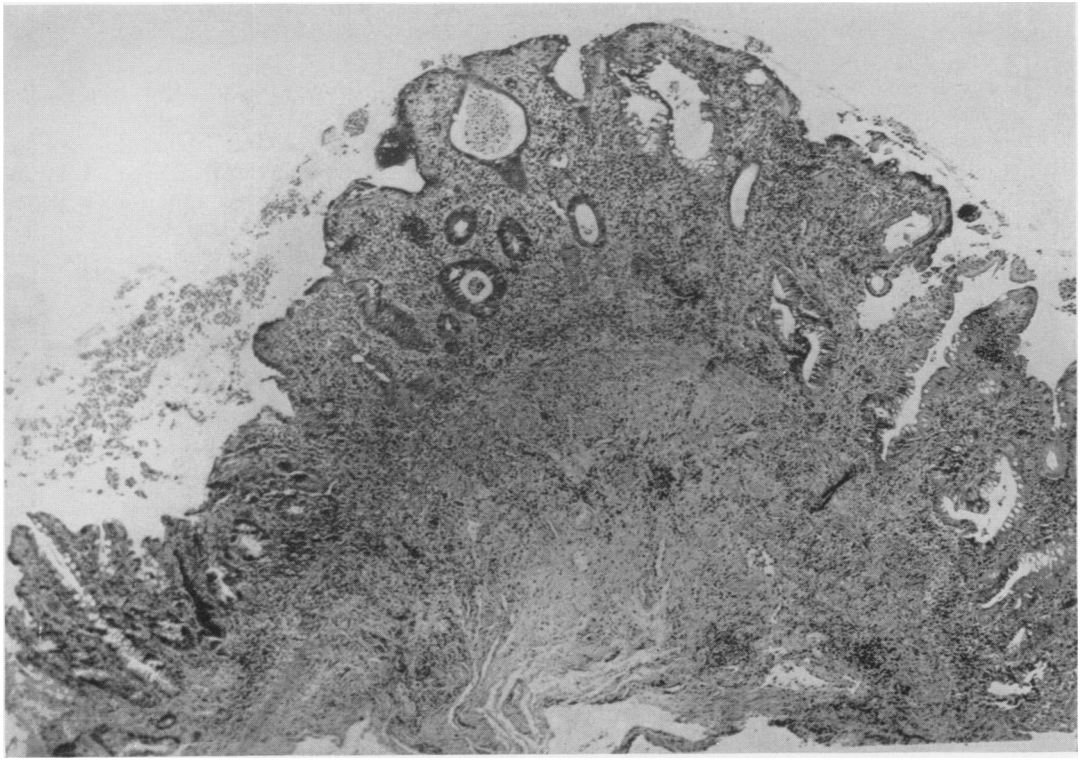

FIG. 7. Acute and chronic ulcerative proctitis in biopsy of rectum, Case 4 (× 55).

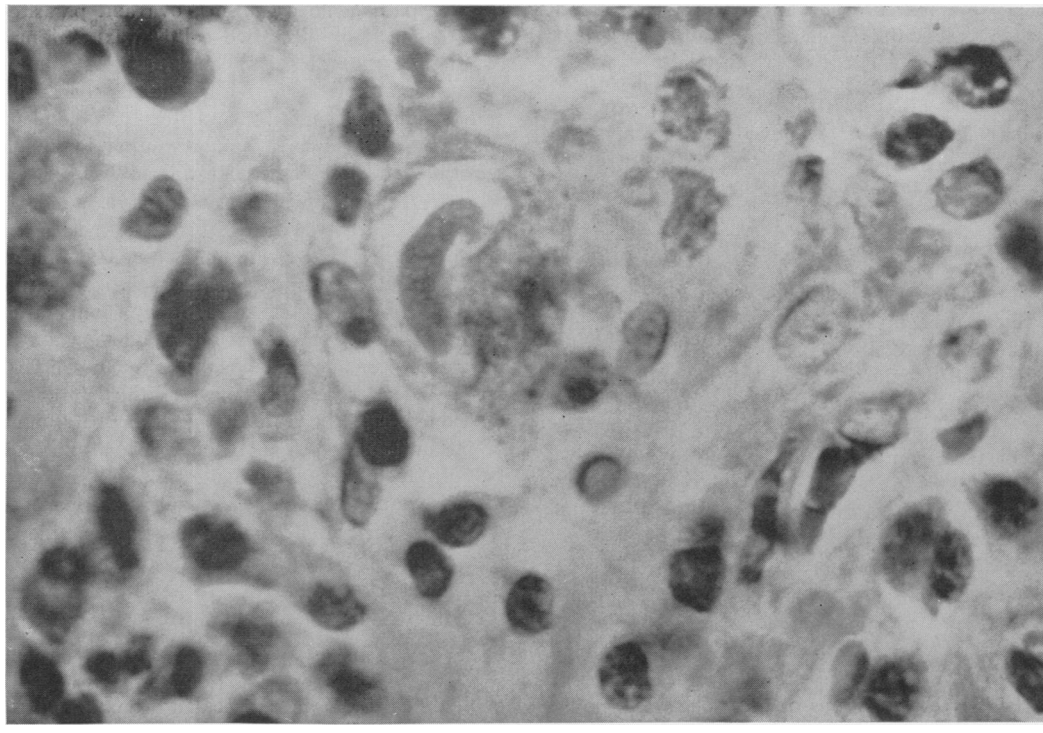

FIG. 8. Cytomegalic cell with large oval intranuclear inclusion, in biopsy of rectal mucosa, Case 4 (× 1,670).

ACTH was discontinued. There was no response. On the 35th hospital day, the patient was given 20 cc. of immune gamma globulin intramuscularly. He became afebrile within 24 hours. The remainder of his hospitalization was characterized by gradual improvement. Ten cc. of gamma globulin were given intramuscularly weekly. At the time of discharge on the 66th day of hospitalization, the patient was taking 90 mg. of hydrocortisone orally, sulfaguanidine 6.0 Gm., and supplemental vitamins and potassium chloride.

Electrophoretic studies of the patient's plasma proteins on the 6th day of hospitalization revealed a normal percentage of gamma globulin and a slight diminution of albumin with a substantial increase in the alpha 2 globulin. A repeat pattern on the 34th day of hospitalization before administration of gamma globulin revealed an increase in the previous height of the alpha 2 peak, and elevated alpha 1 peak, and a further diminution in the percentage of the albumin. A needle biopsy of the liver on the 48th day of hospitalization revealed reactive and slight fatty changes. Rectal biopsies on the 19th and 28th hospital days revealed the presence of inclusion bodies, but none were found in the biopsies done on the 43rd hospital day, or on 1-24-61, five weeks after discharge.

The patient remained asymptomatic after discharge on 12-18-60, and the dosage of hydrocortisone was tapered over a four-week period to 60 mg. daily. Repeat proctoscopic examination after discharge revealed a hyperemic, but nonedematous, non-friable mucosa.

Rehospitalization was necessary on 2-5-61 because of jaundice and malaise for a period of approximately one week. Studies revealed a 1,400 white blood cell count with 2 neutrophils, 94 lymphocytes, 4 monocytes, hemoglobin 13.9 Gm. and hematocrit 40%. Laboratory studies were compatible with a diagnosis of homologous serum jaundice, and a liver biopsy showed nonspecific changes. The sulfaguanidine was discontinued and Gantrisin was begun. Two days after discontinuation of the sulfaguanidine, the white blood cell count was 6,800 with 74% polymorphs. Gantrisin therapy did not alter this count.

The patient's recovery was uneventful. Ten months after the onset of the illness, the patient is asymptomatic and on no therapy. The proctoscopic examination revealed an essentially normal mucosa.

TABLE 1. *Complement Fixation Titers in Three Adults with Localized Salivary Gland Virus Disease in the Gastro-intestinal Tract*

Patient	Age	Clinical Diagnosis	Days Following Histologic Dx.	Receiving ACTH or Steroid Therapy	Complement-fixing Antibody Titer to Human Salivary Gland Virus
1, P. S.	27	Stomal ulcer;	+10	No	1:64
		afferent loop	+61	No	1:64
		obstruction	+90	No	1:64
3, J. B.	18	Ulcerative	+29	Yes	0
		colitis	+360	No	1:64
4, R. E.	60	Ulcerative	−13	Yes	1:8
		colitis	+13	Yes	1:32
			+97	Yes	1:32
			+127	Yes	1:8

Serologic Data

The viral etiology of cytomegalic inclusion disease was promulgated by Van Glahn and Pappenheimer,[24] in 1925, on morphologic grounds. In recent years, human salivary gland virus has been isolated from infants with cytomegalic inclusion body disease,[20] and cytopathogenicity for human fibroblasts in tissue culture has been documented.[19, 20] The prevalence of exposure to the virus in the general population has been indicated by Rowe *et al.*[19] who obtained significant complement-fixing antibody titers to the virus in 81 per cent of apparently healthy adults over the age of 35. Three patients in this report (Cases 1, 3, 4) had determinations of complement-fixing antibodies, and in each a significant, or changing titer was obtained. These data are presented in Table 1. A serologic determination before the diagnosis became apparent from study of the surgical, or biopsy specimen was made in only one patient (Case 4).

Comments

Four cases of nonfatal cytomegalic inclusion disease in adults have been presented. In each patient the inclusions were found in an area of the intestinal tract apparently involved by another disease. Liver involvement was present in one patient. The significance of these findings is obscure, but several questions are immediately evident. Are these examples of primary cytomegalic inclusion disease, or do they represent secondary infection with salivary gland virus (SGV) in a susceptible organ? If the virus is a secondary invader, has it significantly altered the course of the primary disease?

These questions have been considered by Nelson and Wyatt, and a few salient points will be emphasized here. Clinical evidence indicates that most adult SGV infections are superimposed on a primary disease, particularly primary neoplastic, and non-neoplastic disease of the hematopoietic, and reticuloendothelial systems. The pathogenesis of secondary infection may require latent SGV infection, suggested by the high incidence of complement-fixing antibodies in adults, which becomes clinically manifest following alteration of the tissues responsible for antibody production. These tissues may be further insulted by treatment with steroids, cytotoxic agents, or large doses of radiation. Such alterations in immune response with resultant adverse change in an otherwise stable host-parasite relationship have been

postulated by others to account for most cases of adult cytomegalic inclusion disease.[4, 13, 15, 23] The suggestion that infection occurs only in patients *without* pre-existing exposure and antibody formation has received little support, except from the serologic data in Case 3.

The problem of disease apparently restricted to the gastro-intestinal tract is different. Ten other adult cases of isolated gastro-intestinal involvement have been reported.[5-9, 14, 21] Most of these patients have not had primary hematopoietic or reticuloendothelial disease. All four patients in this report suffered from debility. Only three of them (Cases 2–4) were receiving steroids at the time the diagnosis was made. Like the virus of herpes simplex, which resides *dormant* in the skin until an inciting factor such as overexposure to ultraviolet light evokes an active lesion, SGV may become locally invasive when host-immunity and/or organ-susceptibility are appropriately altered.

Fever, lack of response to therapy, and a deteriorating clinical course characterized the four cases presented. The findings, coupled with the indolent course of the disease in each case, strongly suggest that localized secondary infection with SGV may adversely affect the course of the primary disease. Furthermore, in Case 1, a stomal ulcer was present in the face of barely detectable amounts of free acid and low urinary pepsinogen levels. The pathognomonic cytomegaly and inclusions were limited to the ulcer, and we believe that this ulcer has a viral etiology. Similarly, cytomegaly in the other three cases was found primarily in areas of ulceration and frequently in proliferating vascular endothelium.

If secondary infection affects the course of the gastro-intestinal disease, then greater awareness of this condition is important among those who treat surgical disease of the gastro-intestinal tract in adults. The apparent success reported here in the treatment of this form of SGV infection with surgery in three cases, and with immune gamma globulin in another case is unique in the literature and further trials are warranted.

Certain clinical patterns should arouse suspicion of the presence of cytomegalic inclusion disease. The seriously ill patient, with a chronic, or subacute gastro-intestinal lesion, who may be severely debilitated and exhibit persistent fever without response to the most vigorous medical regimen should be suspect. Treatment which has included steroids, cytotoxins or radiation should heighten the index of suspicion. At times, typical cells may be identified in urinary sediment and gastric washings.[13] Search for these cells in the urinary sediment of Patients 1, 3 and 4 was unsuccessful. A positive complement-fixation titer against SGV is suggestive, but as demonstrated in Case 3, serial titers are needed. Involvement of the colon may be established by proctoscopic biopsy. If salivary gland virus infection is proved, involvement of other organs should be sought. Liver biopsy, and evidence of adrenal dysfunction would be helpful in determining the presence or absence of *liver-adrenal necrotizing syndrome.* It is anticipated, however, that only rarely could a localized gastro-intestinal lesion be diagnosed unless it is removed surgically, or accessible to biopsy. Further experience with the therapeutic use of gamma globulin is needed before its effectiveness can be evaluated fully, but the temporal relationship between the time of administration of gamma globulin and the improvement noted in Case 4 is suggestive.

In addition to the cases presented in the present report and ten cases of disseminated fatal infection observed at the University of Chicago Clinics, there are 40 other cases of adult cytomegalic inclusion disease.[1, 2, 10, 11, 14, 16, 18, 22, 23, 25, 26] In this group, the association with neoplastic disease of the reticuloendothelial system is prominent.

Only two cases of cytomegalic inclusion disease associated with carcinoma have been reported [7, 26] (esophagus, lung). Since advanced carcinoma might produce circumstances ideal for activation of the disease, this incidence seems unexpectedly low. The lungs and the gastro-intestinal tract are organs most frequently involved by localized salivary gland virus infection, as well as by carcinoma. It might be that this apparent disparity reflects lack of recognition of the viral disease in patients with carcinoma. In view of the interest in salivary gland virus infection by pathologists in this country and elsewhere, it seems unlikely that such cases would be overlooked or unreported. Clinical and laboratory investigation will be necessary to determine the validity of this observation. It is possible that competition for crucial metabolic pathways largely precludes mutual coexistence of the two diseases in the same host. Perhaps *both* diseases have a viral etiology, and varying degrees of cross-immunity account for the low incidence of association.

Summary

Four cases of nonfatal cytomegalic inclusion disease of the intestinal tract of adults are presented. The lesions were located in ulcerations associated with afferent loop obstruction, idiopathic steatorrhea and ulcerative colitis (2 cases). One patient also had hepatic involvement. The clinical and pathological findings and serologic data in these patients are described. All patients responded to therapy, and the disease appears to be in remission, latent or eradicated.

Current theories of pathogenesis of cytomegalic inclusion disease, and the significance of salivary gland virus infection are discussed.

To date, the incidence of carcinoma in association with cytomegalic inclusion disease is unexpectedly low. This observation requires validation by further clinical and laboratory study.

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INDICATIONS FOR OPEN-HEART SURGERY; RISKS AND RESULTS, by D. P. Morse. C. C Thomas, Springfield, 1963, $10.50.

The author describes his purpose as serving the practitioner in selection of patients operations on the heart. The book is too elementary for student, cardiologist, and surgeon; too inclusive of subject matter for critical consideration of controversy. Clinical and laboratory findings relating to diseased states of the heart and great vessels subject to treatment by closed and open procedures are summarized. The text reflects the title only briefly. Line drawings are clear but contribute no new or worthy additions to published technics. Several pages of tables summarize mortality statistics of cardiac operations culled from the literature through 1961.—ROY H. CLAUSS, M.D.

OBLITERATIVE VASCULAR DISEASE OF THE LOWER LIMB, by J. A. Gillespie and D. M. Douglas. Edinburgh: E & S Livingstone, Ltd., The Williams and Wilkins Company, Baltimore, exclusive U. S. agents, 1961, $7.00.

THIS book deals lucidly with matters of great interest to practicing and resident surgeons and medical students.

Described patterns of clinical effects of arteriosclerosis in a large personal series of patients are enhanced by observations of blood flow at rest and in response to "vasodilator" medical and surgical therapy. Lumbar sympathectomy is considered in detail. Values obtained at follow-up studies weeks and years after therapy are coupled with clinical re-

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