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BALANTIDIASIS

A REVIEW AND REPORT OF CASES *

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Balantidium coli is probably the rarest pathogenic organism causing dysentery. A review of the literature shows that about 600 cases have been recorded in which this protozoon was the sole or main parasite causing colitis. It was in 2 cases of dysentery that Malmsten, in 1857, discovered the parasite in the stools in human cases and named it *Paramecium coli*. In 1863, Stein renamed it *B. coli*.

GENERAL CONSIDERATIONS

Geographic Distribution

The organism has been found as far north as Finland, Sweden, and Norway, but occurs most frequently in subtropical and tropical zones. In the Western Hemisphere, it has been identified in Canada, the United States, Mexico, Honduras, Costa Rica, Panama, Jamaica, Cuba, Puerto Rico, Venezuela, Colombia, Brazil, Uruguay, Argentina, and Chile. It is, however, world-wide in distribution. Large numbers of cases have been reported from Brazil, Mexico, the Philippines, Persia, and the United States; 61 cases had been observed in the United States up to 1950.

Incidence

The reported incidence of parasitization varies enormously, most probably depending on the methods of investigation. It is generally accepted that the parasite is found in about 0.07 per cent of all stools examined, but there are good reasons to believe that this figure falls short of the real one. Stshenovitsh, working in Khalnar, Azerbaijan (Persia), found an incidence of 5.1 per cent among 2,000 fresh stool specimens, in contrast to a study conducted in the same area a few

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years earlier, which yielded only 0.02 per cent. The discrepancy is attributed by Stshenovitsh to his predecessor's failure to examine freshly collected specimens. Stewart, in Southern Persia, discovered 17 cases among 1,430 cases of dysentery. Two of these were complicated by the Flexner type of *Shigella* and the other 2 by the Sonne type. Kipschidse observed 22 cases over a 4-year period in Tiflis, and McCarey recorded 87 cases over a 9-month period, also in Southern Persia. Burrows and Jahnes noted an incidence of 1.3 per cent among 544 patients in the Insular Penitentiary of Puerto Rico; of these cases, 332 were inmates, among whom the incidence of parasitization was 1.8 per cent. Most other authors give lower figures, as shown in Table I.

The lower values correspond to series in which no data were given as to how soon after evacuation the stools were examined. In regard

TABLE I
Reported Incidence of Balantidial Infection

Author	Country	No. of cases	Positive	Contact with pigs
Stshenovitsh*	Persia (Azerbaijan)	2,000	5.1	0
Stewart	South Persia	1,430	0.01	0
Burrows and Jahnes*†	Puerto Rico	544	1.3	+
Atilés	Puerto Rico	5,000	0.08	+
Serra	Puerto Rico	5,000	0.08	+
Maldonado	Puerto Rico	8,000	0.03	+
Cort	Siam (Chiengmai)	8,000	0.17	+
Sommerville	Argentina	5,000	0.02	+
Bowman (1909)	Philippine Islands	4,000	0.07	+
Pawel	Brazil (São Paulo)	12,500	0.02	+
Maia	Portugal	4,000	0.1	+
Potenza and Martínez	Venezuela (Caracas)	5,689	0.05	+
Young (1939)*†	United States (South Carolina)	142	4.9	0
Ferri*†	Russia (Tiflis)	68	29.0	+

* Fresh stools.

† Institutional cases.

to cases from Puerto Rico, it can be safely stated that most of the stool samples were examined many hours after passage. In Maldonado's series the specimens were received in the Department of Parasitology of the School of Tropical Medicine 2 or 3 days after evacuation, well beyond the period considered optimal for identification of the trophozoites. It seems to us that the discrepancy in incidence results mainly from rapid disintegration of the parasite in the

stools after evacuation. Maia and others have observed that 6 hours after the passage of the stools few or no trophozoites can be identified. Furthermore, it is characteristic of this condition that the balantidia are passed intermittently, so that they may be abundant one day and absent 2 or 3 days later. This must be kept in mind when conclusions are drawn as to the effectiveness of therapeutic agents.

Morphology

The parasites are readily identified, for they are the largest protozoa infecting man. In the trophozoite (Fig. 1) stage they are ovoid and vary from 30 to 150 μ in length and from 40 to 55 μ in width, although specimens as long as 300 μ have been seen. The parasite is ovoid and in its anterior portion, slightly lateral to the midline, there is a funnel-like depression, the peristome, communicating with the cytostome and the cytopharynx. The body is enveloped in a thin pellicle covered by numerous cilia that extend over the primitive buccal slit; and it is by their action that the organism is capable of very active locomotion and that food is directed into the digestive passages. At the posterior end there is a small, hardly visible, triangular opening, the cytopyge, with an excretory function. The cytoplasm contains two contractile vacuoles, and often red blood cells, starch granules, oil droplets, and inert materials. The characteristic macronucleus, which is shaped like a kidney or bean, is slightly eccentric; in its concavity a tiny micronucleus is seen occasionally. The macronucleus appears as a homogeneous mass of chromatin, staining evenly and deeply with basic dyes.

The cystic (Fig. 2) stage is characterized by a fairly large, round or ovoid structure, 40 to 65 μ in diameter. In fresh, non-stained preparations it has a dirty, opaque grayish color and presents bar-like bundles of inclusion material which is colorless and disappears on staining. It must be kept in mind that in fresh preparations the macronucleus is not visible. By using Delafield's or Heidenhain's hematoxylin the characteristic macronucleus, the granular cytoplasm, and the absence of stomata and cilia can be brought out. When the parasite is in the process of encystment, cilia may be observed under the capsule, and the organism is seen rotating actively within this capsule.

Nutrition

For their nutrition balantidia depend mostly on starches and red blood cells. Being very susceptible to the action of acids, they are quickly destroyed by the gastric juice of a normal person. Many methods of culture of the organism have been devised. Barret and

Yarbrough used 1 part of inactivated human serum and 16 parts of 0.5 per cent sodium chloride, giving a faintly alkaline reaction to litmus paper. The medium is placed in test tubes 10 by 150 mm. in size, and incubated at 37° C. for 24 hours. Dobell and Laidlaw used inspissated horse serum and Ringer's fluid-egg white with solid rice starch at a pH of 5 to 8, keeping the cultures at 37° C.; subcultures were made every 3 to 5 days. Although conjugation was observed periodically under these conditions, encystment did not occur. Schumaker (1931) used potato, corn, wheat, and arrowroot starch with equally good results, and noted that temperatures below 34° C. were harmful to the organism.

The parasite is a facultative anaerobe but it grows well in both aerobic and anaerobic media. Nelson used intestinal contents freed of coarse particles by filtration and proved that balantidia could obtain from such contents all the essentials for growth and multiplication; serum need not be added, but the normal bacterial flora of the intestinal contents was indispensable for their growth. A pH below 5 is incompatible with the life of the balantidium. Despite the high sensitivity of the ciliate to a low pH, Hegner (1926) was able to recover live trophozoites from the colons of guinea-pigs after ingestion. A diet rich in protein will decrease their growth in the colon. Schumaker (1930) found that rats maintained on a casein-rich diet (70 per cent), were prevented from developing the infection; when the diet was changed to 93.5 per cent carbohydrates, multiplication of balantidia was greatly enhanced, and the proportion of infected rats was greatly increased. Van der Reis (1923) and Schumaker (1931), independently, studied the intestinal flora of heavily parasitized rats and noted the predominance of *Lactobacillus acidophilus*, while lactose fermenters were very much diminished. The opposite was true when the balantidia were scant or absent. This compares with the studies of Sommerville in cases of balantidiasis, in which he noted a neutral or faintly alkaline reaction of the stools.

Metabolism

Schumaker (1931) observed that the parasite was unaffected by oxygen pressure of 32 lbs. per square inch for 72 hours. Daniel determined that the respiratory quotient of the parasite is 0.84, indicating that under aerobic conditions carbohydrates are not the chief source of energy for balantidium. Agosin and von Brand recently have studied its respiratory metabolism and have concluded that the aerobic gaseous exchange is characterized by a fairly high rate of oxygen

consumption, with respiratory quotients of slightly above 1.0. Anaerobically, relatively large amounts of CO₂ are given off. Aerobic and anaerobic gaseous exchanges were relatively insensitive to DL-glyceraldehyde but sensitive to glycolysis inhibitors, anaerobiosis being more sensitive to lower concentrations of a given inhibitor than the aerobic phase. Inhibition by malonate and fluoro-acetate suggested an aerobic sequence showing some characteristics of the Krebs cycle. Glaessner isolated a glycolytic enzyme and a hemolysin from the parasite, but was unable to demonstrate any proteolytic enzyme.

Epidemiology

The protozoon has been found in the hog, wild boar, sheep, horse, bovines, guinea-pig, fowl, turtle, cockroach, *Macaca mulatta*, orang-outang, baboon, chimpanzee, and other species. However, of all animals in relatively close contact with man, the hog is the one that is more frequently and most heavily parasitized. Ostroumov, in Central Soviet Russia, found 62 per cent of the pigs heavily infected; Shegalow, in St. Petersburg, 40 per cent; Kipschidse, in Tiflis, 63.3 per cent; and Füsthy, in Szeged, 92 per cent, and in Budapest, 90 per cent. Walker, Liu, Shun-Shin, Pawel, Cruz and Domingo, and others referred to the heavy rate of infection among hogs in the countries from which they reported. Twenty-five per cent of the cases give a history of contact with hogs, and Selimkhanov and Ferri, among others, believed that there is an important relationship between the heavy infection of hogs and human cases. Our review of the literature shows that more than 50 per cent of cases give a history of contact with pigs. On the other hand, Liu, Shun-Shin, and Shegalow remarked that despite the heavy infection of hogs in their countries the number of human cases was very small. The relationship between man and hog also is rendered doubtful by McCarey's series; being Mohammedans, his patients did not eat pork nor live in close contact with pigs. In Young's series (1939) there was no history of contact with hogs, and Awakian remarked that although the number of hogs is high in England, cases of human balantidiasis are extremely rare, and that in Armenia, where hogs are scarce, balantidiasis often affects man. Tzaturian observed 24 cases in 7 years, of which only 2 patients admitted contact with pigs.

All of these controvertible observations have led some authors to believe that there is a difference between the human and porcine balantidia; the former has been called *B. coli* and the latter *B. suis*. Hegner (1934), Selimkhanov, Walker, and Pritze, among others,

despite extensive and painstaking studies, have been unable to differentiate one from the other on a morphologic basis. Differentiation on the basis of size alone is fallacious, since balantidia may vary enormously in size, probably depending on the dietetic habits of the host. Chichulin reported on a family, all members of which developed balantidiasis after eating raw hog sausage, presumably heavily parasitized. Awakian found 29 per cent of the rats in Southern Russia infected with the ciliate, and Klein discovered it in the sewer system of London. The parasite also has been observed in the diarrhetic stools of monkeys and chimpanzees, and in a capybara. Tsuchiya and Kenmore attributed an important rôle to flies in the transmission of their case.

There must be other factors to explain the irregular incidence of balantidiasis. Worthy of note is the fact that the more severe infestations have been reported from underprivileged areas where standards of hygiene and nutrition are inadequate. Furthermore, the only epidemics have been reported from mental institutions where the patients were untidy, negativistic, or even coprophagic (Young, 1939; Ferri). Elliott and Hotson, and Junqueira also mentioned coprophagia among their patients.

PATHOLOGY

Pathogenicity

Some believe that *B. coli* is completely harmless. Others think that it is pathogenic only on tissues previously damaged otherwise, as by bacteria. Still another group considers that the ciliate is pathogenic per se. The natural resistance of man to this parasite is evidenced by the persistent failure by Young (1950) and Knowles and Das Gupta to transmit the infection to human volunteers. On the other hand, Walker was able to reproduce the disease experimentally in monkeys by feeding and by rectal inoculation with trophozoites from pigs. Brumpt infected monkeys with material obtained from pigs, and vice versa. Harms was unable to induce the disease in pigs with trophozoites from a human case. Hegner (1926) recovered live balantidia from the colons of guinea-pigs that had ingested trophozoites.

Morphologic Features in Man and Experimental Animals

The number of cases studied histopathologically is small. The first large series published was that of Strong, who in 1904 collected 40 cases from the literature and added 7 of his own. After that only isolated reports appeared. The most recent series came from this laboratory in 1947, when Koppisch and Wilking described the necropsy findings in 4 cases.

Balantidiasis usually involves the colon (Fig. 4). Grossly, the lesions involve the large intestine from cecum to rectum, although the rectosigmoidal segment is the region more commonly and more severely affected. The earlier lesions appear as small, flask-shaped ulcers a few millimeters in diameter. Later these lesions expand into ulcers resembling those of amebic colitis. They may be numerous or scarce; occasionally they replace almost the entire colonic mucosa. The edges of these ulcers are frayed, ragged, swollen, and frequently undermined. They are covered with mucous material or with a necrotic, grayish white, slate black, or black membrane. The mucosa about the ulcers may be reddened and swollen, or may appear practically normal. The ulcers are for the most part superficial, but some may affect the whole thickness of the intestinal wall, so that perforation may occur. After perforation, the omentum may be plastered over the involved segment or a generalized peritonitis may result. Although the ulcers usually stop at the ileocecal valve, in some cases the process may extend to the distal portions of the ileum, as in 2 cases seen by Koppisch and Wilking.

The earlier lesions, as described by Walker in the experimental animal, appear as zones of slight hyperemia of the mucosa with or without punctiform hemorrhages. Foci of vascular dilatation and perivascular round cell infiltration associated with eosinophils are seen also. The absence of polymorphonuclear leukocytes and the abundance of round cells and eosinophils is a characteristic that distinguishes balantidiasis from the bacterial infections. The balantidia penetrate through the intact epithelium of the colon, chiefly that of the crypts. At the site of contact between the ciliate and the epithelium, the cells may become shrunken (Fig. 3), the cytoplasm turning markedly acidophilic and the nucleus pyknotic. The glandular epithelium may be thinned out. Whether entering directly through the surface epithelium or through glands, the parasite usually penetrates the basement membrane and then the muscularis mucosae, to reach the submucosa (Fig. 5). At this level it often is found in dilated lymphatics (Fig. 6). The lymph vessels and small capillary blood vessels are then much dilated. The mucosa and submucosa undergo necrosis, and the limiting portions become infiltrated chiefly with lymphocytes, although in places neutrophils may be numerous (Fig. 7). Foci of hemorrhage are frequent, and congestion is very prominent.

When the overlying mucosa is cut off from its blood supply, necrosis and ulceration follow. The ulcers are irregular and have undermined edges (Fig. 8). They are partly covered by a coagulum of fibrin, red blood cells, leukocytes, and cellular débris (Fig. 9). Occasionally an

abscess is found in the submucosa beneath an intact, overlying mucosa; serial sections will usually demonstrate that this occurs in the vicinity of an ulcer. Balantidia are difficult to identify in the necrotic portions of ulcers, but they are numerous in the periphery.

The organisms are seen also at a distance from the ulcers, in the muscular coat, in the serosa (Fig. 10), and even in regional lymph nodes (Fig. 11). Campos observed that those found in the muscle coat were surrounded by a clear space which separated them from the tissue; however, this is probably the result of fixation and shrinkage rather than of any specific change. In these locations there is very little reaction, if any, about them. Manlove claimed that this is explained by the tendency of this ciliate to migrate post mortem, and his experiments seemed to confirm that impression. In the appendix the lesions are similar to those of the colon, so that no special reference is necessary (Fig. 12). No lesions have been reported as occurring in either regional lymph nodes or in the liver.

Clinical Features

The presence of balantidium in the feces is not always an indication of disease. Swartzwelder classified the clinical picture as follows: (1) asymptomatic, the main danger of this form being the rôle these patients play as carriers, chiefly in mental institutions or penitentiaries; (2) a chronic form with intermittent episodes of diarrhea; and (3) the dysenteric form, which may be mild, severe, or fulminating.

The *chronic form* is characterized by the presence of loose bowel movements alternating with episodes of constipation. There may be epigastric distress, colicky abdominal pain, and tenesmus. The number of bowel movements varies from 3 to 20 a day and mucus often is seen, but pus and blood only rarely. Loss of weight is moderate, but may be marked when the course is protracted. Balantidia are identified in the stools only sporadically, so that repeated examinations and the study of fresh stools are mandatory.

The *acute form* appears suddenly with 3 to 15 bowel movements daily, accompanied by tenesmus; the stools contain mucus, blood, and neutrophils. The patient complains of epigastric distress, nausea, and abdominal pain, with tenderness along the colon. Loss of weight may be rapid, some patients having lost 40 kg. over a period of 3 months. Weakness is marked and is related to dehydration and undernourishment. Swartzwelder claimed that these patients have a peculiar odor to their breath, recalling a "pigpen." Proctoscopic examination reveals diphtheritic patches, 1.5 to 3 cm. in diameter, surrounded by a bright

red, swollen mucosa. Occasionally tiny ulcers also are seen, requiring differential diagnosis from bacillary or amebic dysentery. Balantidia may be extremely numerous in the scrapings of such ulcers. Pallor and hypochromic anemia may result from hemorrhage and undernourishment. Some authors have attributed a megaloblastic anemia to the parasite, but these patients either suffered from sprue or the gastric juice showed achlorhydria, so that pernicious anemia could not be ruled out. Leukocytosis and eosinophilia are not found unless there are complications such as perforation or association with other parasites, such as *Uncinaria*.

In the *fulminating type*, seen ordinarily in emaciated patients, or in the late stage of some other severe disease, diarrhea starts suddenly with from 5 to 25 bowel movements a day. Overt hemorrhages may occur, leading to death by exsanguination; otherwise there is dehydration and rapid deterioration, with death in 3 to 5 days. Although most cases are afebrile, temperatures as high as 38.1° C. have been observed in the chronic and dysenteric forms. The chronic form may last for years; McEwen has reported a case of 20 years' duration.

Balantidial Appendicitis

Jaffé and Kann (1943), from Venezuela, were the first to describe balantidial appendicitis. They reported 6 cases, in 5 of which the complaints were of chronic nature and in the sixth there was a sudden onset of abdominal pain, shown at operation to be due to acute appendicitis. Jaffé and Kann were unable to identify balantidia in the appendiceal walls of the 5 chronic cases, but they were abundant in the lumina. In the other case balantidia were seen throughout the wall, and the ciliate was considered the cause of the appendicitis. In 1947, Potenza and Martínez, also from Venezuela, reported the second case of acute appendicitis due to *B. coli*. The patient was a child who developed abdominal pain in the right lower quadrant, with positive McBurney and Rovsing signs, as well as neutrophilic leukocytosis. To the best of our knowledge, these are the only cases of acute appendicitis ascribed to the *B. coli* that have been recorded. Worthy of note is the fact that repeated examination of the stools for a period of 6 months after the operation failed to reveal any cysts or trophozoites.

DIAGNOSIS AND THERAPY

The diagnosis is based on the identification of the parasite in the stools or in scrapings obtained at proctoscopy. Any dysenteric process ought to include also a careful search for amebae and dysentery bacilli;

combinations of these diseases sometimes occur. Overlooking another parasite by the identification of only one species may lead to grave consequences for the patient. The material obtained must be examined immediately, keeping in mind the rapid deterioration of the trophozoite. Cysts are seldom found; Young (1939), among 142 examinations, observed trophozoites in 87.5 per cent and cysts in only 8.9 per cent. Ferri found no cysts in his cases. The material may be examined by making a saline suspension or after staining with Delafield's or Heidenhain's hematoxylin. As the ciliate is large, the use of a low-power lens usually is adequate; however, considering the enormous variations in size, a more careful search should be made at a higher magnification if low-power examination proves negative. The parasite has been identified in the urine, although only once, by Maliwa and Haus who later demonstrated the organism in sections of ureter and bladder in the same patient. Stokvis discovered the parasite in the sputum of a patient supposed to have a liver abscess perforating through the diaphragm into the lung; however, this supposition was never proved. Finally Hinkelmann claimed to have encountered *B. coli* in the blood of a patient who also showed it in the urine, but this case is open to question.

The prognosis of balantidiasis, especially for the acute and fulminating types, was very poor until 1950, with a mortality of up to 30 per cent. The introduction of antibiotic drugs has improved the outlook. The number and varieties of drugs and treatments previously used speak for the poor results and inefficacy of most. Greene and Scully, in 1923, used a starch-free diet consisting of 2½ quarts of milk a day, to which soft eggs were added. Silva also obtained good results in the cases thus treated, but such measures have failed in the hands of others. Cort, after the unsuccessful use of emetine, neoarsphenamine, and acetarsone, gave his patients 15 ml. of oil of chenopodium in 150 ml. of olive oil per rectum, obtaining complete cures in all cases. Serra treated a patient of his following the same technique, and the patient died of oil of chenopodium poisoning within a few hours. Kipschidse considered emetine the best drug, but Cruz and Domingo, and DeLanney and Beahm got no results with it. Iodine compounds, either alone or in combination with other drugs, have been used successfully by some authors (DeLanney and Beahm, Swartzwelder, Young and Walker, Pramanik) while others had complete failures (McCarey, Cort). Of all the chemotherapeutic agents, the arsenicals, such as stovarsol, carbarsone, acetarsol, tryparsamide, neoarsphenamine, and spirocid, gave consistently good results. Carbarsone, alone

or in combination with iodine derivatives, was considered the best. Sulfaguanidine and other sulfa derivatives also have been prescribed with contradictory and irregular benefits. As complementary therapy, enemas of methylene blue, yatren, protargol, and quinine sulfate also were employed.

In 1950, Castellanos *et al.* reported on the excellent results obtained with bacitracin in two children with balantidial dysentery. In the same year Agosín *et al.* published their observations on the action, both *in vivo* and *in vitro*, of aureomycin and terramycin against balantidia; they attributed this action not to any change in the pH or in the intestinal flora but to a direct effect of the drug on the parasite. Beneficial results also were reported by Neghmé *et al.* in 1951, who obtained complete disappearance of the parasite from the feces in 2 to 4 days. Later on, Weinstein *et al.*, Jarpa and Allende, and Hoekenga used terramycin, while Santos, Burrows and Jahnes, and Neghmé *et al.* used aureomycin with very good results. The dose of terramycin has been 500 mg. 4 times daily for 10 days and for aureomycin, 2 gm. daily for a total of 28 gm.

REPORT OF CASES

In a previous communication (Koppisch and Wilking) one of us gave examples of two modalities of balantidiasis. Case 1 (necropsy no. 1196) represented a very acute case of balantidial dysentery with involvement of the colon and terminal ileum in a girl 19 years old. Death was due ultimately to two perforations of the cecum and one of the sigmoid colon, with diffuse peritonitis. Case 2 (necropsy no. 1384), a 4-year-old mulatto girl, was an example of the chronic form, with six ulcers distributed along the colon, and with extensive pulmonary and glandular tuberculosis as the main disease.

Case 3

A 7-year-old boy was admitted to the hospital because of severe diarrhea, vomiting, and headache for 3 days. At onset, the patient vomited seven worms (*Ascaris*), after which he was unable to retain food or liquids. Temperature was 99° F. The patient was poorly developed, poorly nourished, markedly dehydrated, and slightly cyanotic. The pupils were dilated, did not react to light, or in accommodation. He was given 5 per cent glucose in saline solution intravenously, and shortly thereafter became restless, slightly cyanotic, and dyspneic. Râles were heard and in a few minutes apnea supervened. He was placed in a Drinker respirator. The liver was palpable 3 fingerbreadths below the costal margin. The extremities were cold and flaccid and reflexes could not be elicited. The diarrheal stools were greenish and bloody. The urine showed traces of albumin and a few casts. The condition of the patient deteriorated rapidly and he expired 19 hours after admission.

Gross Examination

The body (necropsy no. 2128) was that of a poorly developed, poorly nourished and dehydrated boy. The abdominal cavity contained 200 ml. of turbid yellow liquid with multiple flecks of fibrin. The peritoneum was smooth and glistening. The liver was enlarged and the bladder rose 7 cm. above the pubis. Approximately 75 ml. of yellow fluid was found in each pleural space and there were several fibrous pleural adhesions over the posterior aspect of the lungs. In the right frontal region there was a hematoma, 1.5 cm. in diameter, and another in the occipital region, measuring 1.6 cm. across. The bones of the skull, the meninges, and the surface of the brain were normal. There was an early thrombus in the confluence of the sinuses. The heart was normal. The lungs were markedly congested and edematous. The small intestine showed numerous large, discrete areas of mucosal congestion and several adult *Ascaris*. The mucosa of the colon showed congestion and tiny ulcers measuring about 1 mm. across. Whipworms were abundant in the rectum, sigmoid, and appendix. The other organs were normal.

Microscopic Description

Heart. There was moderate serous atrophy of the subepicardial fat. A slight diffuse infiltration of the myocardium with lymphocytes and eosinophils was present, together with slight interstitial edema.

Lungs. Marked congestion of the interlobular septa was present. Coagulated protein filled the alveolar spaces. A few macrophages and neutrophils were seen in air sacs and occasional hemorrhages were present also.

Spleen. Congestion and hemorrhages into the splenic pulp were evident.

Liver. Most hepatic cells contained fat droplets. The portal spaces were broadened by fibrosis. The central zones revealed slight congestion.

Adrenal Glands. The adrenal cortex was almost completely depleted of lipids. The zona fasciculata showed focal atrophy. There was an extracortical adenoma.

Ileum. In the ileum edema of the submucosa was accompanied by infiltration with eosinophils.

Colon. Numerous balantidia were found in the colon between the detached mucosa and the basement membrane. Some had migrated into the submucosa and there they had induced a marked infiltration with eosinophils, lymphocytes, and plasma cells. This was diffuse but

in some areas it became more dense. In such regions there were minute foci of necrosis and hemorrhage. Balantidia were found in the margins of these abscesses. The overlying mucosa was thinned but not ulcerated. The blood vessels were filled with eosinophils and lymphocytes and the capillary network of the mucosa also was diffusely dilated and filled with eosinophils. The lymphatic vessels were dilated; in some the endothelium was slightly swollen. About a few lymphatics there were groups of lymphocytes and eosinophils forming nodules that bulged into the lymphatic lumen and narrowed it. Sections from an early ulcer showed numerous balantidia in the mucosa and upper portion of the submucosa, surrounded by lymphocytes and a few eosinophils. A few balantidia were seen also in the mesenteric lymph nodes where they had induced a moderate hemorrhage; one was found in an afferent lymph vessel in the hilum of the node without any reaction about it.

Case 4

An 11-year-old Negro boy (S.P. no. 75066) was admitted to the hospital because of abdominal pain of a few hours' duration. He had felt well until March 31, 1955, when, on awakening, he complained of mild pain in the right lower abdominal quadrant. He vomited shortly thereafter, and the pain, nausea, and vomiting increased in severity. There had been no bowel movement since the onset. He had had no similar attacks in the past. Physical examination revealed the temperature to be 103° F.; pulse, 116 per minute; blood pressure, 115/70 mm. of Hg. The abdomen was soft; there were positive McBurney and Rovsing signs. There was also tenderness and pain over the right lower quadrant. The white blood cells were 13,200, with a differential of 80 segmented leukocytes and 20 lymphocytes. Red blood cells were 4,600,000 and the hemoglobin 12.7 gm. per 100 ml. Appendectomy was performed the same day.

Gross Examination

The appendix measured 4 cm. in length, and its diameter was increased to 1.2 cm. The serosa was grayish yellow and was covered with fibrinopurulent exudate. The wall was friable. The lumen was dilated and filled with pus.

Microscopic Description

Microscopically, the appendiceal mucosa was extensively ulcerated and infiltrated with polymorphonuclear cells, lymphocytes, and eosinophils. Abundant balantidia were present in the base of the ulcers and in the wall. In the latter location they tended to be arranged in groups in the lymphoid follicles, where they had elicited a necrotizing reaction. They were found also in the lumina of lymph and blood vessels. One ulcer had perforated through the wall into the meso-appendiceal fat. The advancing border of this ulcer was represented by a group of

balantidia, some of which had degenerated. In general, the muscular part of the appendix was diffusely infiltrated with polymorphonuclear leukocytes, and edema was conspicuous everywhere.

The postoperative course was uneventful; unfortunately the feces were not examined, and it has been impossible to have the patient return for that purpose.

Case 5

A 37-year-old white woman (S.P. no. 75437) was admitted because of sharp pain in the right thigh, radiating to the abdomen. She felt perfectly well until the evening prior to admission, at which time the pain developed. It became worse on motion, which she was careful to avoid. She denied having had nausea, vomiting, diarrhea or urinary symptoms, or having had similar episodes in the past. She was in acute distress and the abdomen was distended. There was tenderness over the abdomen, which was accentuated in the right lower quadrant. There was rebound tenderness. A mass was felt in the right lower quadrant. Intestinal peristalsis was active. She was a gravida, XII, para XII, and had no history of any previous serious illness. The leukocyte count was 16,750, with a differential of 76 segmented leukocytes, 2 band cells, 16 lymphocytes, and 4 eosinophils. Urinalysis was negative. At laparotomy, both adnexae were inflamed. The appendix was removed. Grossly, its serosa was smooth and glistening; the serosal blood vessels were moderately congested and the distal end was slightly dilated. It measured 10 cm. in length and 1.2 cm. in diameter.

Microscopic Description

Microscopically, the serosa of the appendix revealed congestion and infiltration with eosinophils and polymorphonuclear cells. The muscle coat also was infiltrated with eosinophils and showed moderate interstitial edema. Sections of the distal end showed abundant balantidia and moderate numbers of leukocytes. There was no ulceration of the mucosa nor were any protozoa identified in the mucosa or other parts of the wall. There was no evidence of inflammation. The postoperative course was uneventful.

DISCUSSION

The severity of the histopathologic changes found in our cases corroborates the opinion of those who believe firmly in the pathogenic nature of *B. coli*. They also illustrate several other factors which we deem advisable to discuss further. Bowman (*J. A. M. A.*, 1911) and later Jaffé (1919) stated that the living protozoon secretes a toxic substance, but in such small amounts that only mild or no inflammatory reaction is induced in the surrounding tissues. However, when they are very numerous or degenerating, a larger release of this toxin provokes an inflammatory infiltrate composed chiefly of lymphocytes and eosinophils. Harms corroborated these findings. The presence of polymorphonuclear cells is secondary to necrosis and possibly also to secondary bacterial infection.

It is obvious to us that the simple presence of balantidia is not enough to induce pathologic alterations. Other factors must act before this protozoon becomes pathogenic. In regard to dietetic habits, it has been repeatedly stressed that in most cases the patients lived on diets composed chiefly of carbohydrates with very little or no protein and scarce animal fats. Schumaker (1931) was able to correlate diet and intensity of infection in the hog as an experimental animal. However, despite the fact that poor eating habits affect large sectors of the population in many parts of the world, the number of cases of balantidiasis is indeed low.

Other circumstances must be present so that the combination of all will lead to a terrain favorable to the attack of balantidia. Masing was the first to point out the presence of achlorhydria or severe hypochlorhydria. Similar observations have been made by Brea and Nieto, Jarpa and Allende, Elliott and Hotson, Ferri, Cruz and Domingo, and Logan. This aspect of the problem has not been studied systematically. It is of interest that most cases, particularly the more severe ones, occur among elderly individuals, whose tendency to low free-acid values in the gastric contents is well known. In general, any condition that tends to depress the natural defenses of the body, such as chronic infection, undernourishment or starvation, alcoholism, and filth, seems to predispose man to infections by this organism. Furthermore, *B. coli* is not, in most patients, the only intestinal parasite present. Burrows and Jahnes found intestinal parasites of two to seven different species in their cases; likewise Bowman (*Philippine J. Sc.*, 1911), Santos, Masing, Kipschidse, Atilas, Sommerville, Koppisch and Wilking, Pramanik, and Stewart all observed concomitant parasitizations. The accompanying parasites were mostly *Ascaris lumbricoides*, *Necator americanus*, *Trichuris trichiura*, and *Strongyloides stercoralis*, but *Diphyllobothrium latum*, *Taenia saginata*, *Giardia lamblia*, *Blastocystis hominis*, and *Endamoeba histolytica* also have been reported. Atilas claimed that the degree of parasitization, state of nutrition of the host, nature of the host's diet, and quality of the intestinal flora all have an influence on the development of balantidiasis.

All of our cases came from a sector of the population in which both food habits and hygienic conditions were substandard. Undernourishment was evident in all, and two had severe chronic infectious diseases (tuberculosis and a typhoid carrier state). All cases which were necropsied showed multiple infestation by several kinds of intestinal parasites (Fig. 14). Unfortunately, no gastric analyses were done.

Our case of acute balantidial appendicitis is the third one recorded.

Campos' case was observed at necropsy as part of a diffuse inflammation of the whole colon and appendix, so that it does not count as surgical material. Our case 1 is interesting also because it was complicated by one of the less common manifestations of balantidiasis, namely, ulceration of the ileum with perforation. Only one case, that of Strong, has so far been reported in which a similar situation occurred.

The lesions of balantidiasis cannot be differentiated grossly from those of amebic dysentery and only histologic examination will reveal the true nature of the causal agent. However, because of the rapidity with which balantidia disintegrate, the organism may not be identified in cases in which the necropsy has been delayed for many hours, especially under the unfavorable circumstances of the tropics, unless a careful and painstaking search is carried out.

SUMMARY

This study was based on 5 necropsies of patients dying with *Balantidium coli* dysentery and two surgically removed appendices, also infected by this protozoon. The lesions are similar, grossly, to those seen in amebic dysentery. The histopathologic differentiation is made by the identification of the ciliate, which usually is seen at the invading edge of the ulcers or at the periphery of submucosal abscesses. The lesions are seen also deep in the intestinal wall, commonly within lymphatic vessels. Occasionally also they are identified in the regional lymph nodes, where a mild reaction may occur. Only one case has been reported previously in which this parasite affected the terminal ileum; our case is the second one. Similarly, only two cases of acute balantidial appendicitis had been recorded, ours being the third. The literature in regard to geographic distribution of the parasite, incidence, epidemiology, biology of the ciliate, pathogenetic aspects, clinical picture, and recent therapeutic trends is reviewed.

BIBLIOGRAPHY

- Agosín, M.; Christen, R., and Rubio, M. Acción de la aureomicina sobre el *Balantidium coli*. I. Estudio "in vitro." *Bol. Inform. Parasit. Chile*, 1950, 5, 32-33. Acción de la terramicina sobre el *Balantidium coli*. I. Estudio "in vitro." *Ibid.*, 1950, 5, 42-43.
- Agosín, M., and von Brand, T. Studies on the respiratory metabolism of *Balantidium coli*. *J. Infect. Dis.*, 1953, 93, 101-106.
- Atiles, A. D. A study of balantidiasis coli. Report of two cases in children successfully treated with stovarsol. *Puerto Rico J. Pub. Health & Trop. Med.*, 1942-43, 18, 287-299.

- Awakian, A. Studies on the intestinal protozoa of rats. II. Rats as carriers of *Balantidium*. *Tr. Roy. Soc. Trop. Med. & Hyg.*, 1937-38, 31, 93-98.
- Barret, H. P., and Yarbrough, N. A method for the cultivation of *Balantidium coli*. *Am. J. Trop. Med.*, 1921, 1, 161-164.
- Bowman, F. B. Two cases of *Balantidium coli* infection, with autopsy. *Philippine J. Sc.*, 1909, 4, 417-422.
- Bowman, F. B. A case of dysentery caused by *Balantidium coli* with coincident filarial infarction of the spleen. *Philippine J. Sc.*, 1911, 6, 147-153.
- Bowman, F. B. The pathogenesis of the *Balantidium coli*. *J. A. M. A.*, 1911, 57, 1814-1817.
- Brea, R. J., and Nieto, C. A. Balantidiasis humana en el Uruguay. Contribución a su estudio. *Arch. urug. de med., cir. y especialid.*, 1937, 11, 720-733.
- Brumpt, E. Démonstration du rôle pathogène du *Balantidium coli*. Enkystement et conjugaison de cet infusoire. *Compt. rend. Soc. de biol.*, 1909, 67, 103-105.
- Burrows, R. B., and Jahnes, W. G., Jr. The effect of aureomycin on balantidiasis. *Am. J. Trop. Med.*, 1952, 1, 626-630.
- Campos, E. de S. Sur un cas de balantidiose suivie d'autopsie: colite, appendicite et lésions des ganglions lymphatiques. *Compt. rend. Soc. de biol.*, 1924, 90, 1341-1343.
- Castellanos, A.; Prado, E.; García, O., and Montero, R. Uso de bacitracin en el tratamiento de las enterocolitis producidas por *Balantidium coli*. *Rev. cubana Pediat.*, 1950, 22, 542-549.
- Chichulin, G. N. [Rôle of *Balantidium coli* in intestinal disorders.] *Voyenno-Med. J., S.-Peterb.*, 1900, 78, med. spec. pt., p. 2059. (Cited by Young and Walker.)
- Cort, E. C. Infection with *Balantidium coli*. Twelve cases treated with oil of chenopodium. *J. A. M. A.*, 1928, 90, 1430-1431.
- Cruz, A. F., and Domingo, C. M. Sobre un caso de balantidiosis. *Med. colon., Madrid*, 1945, 5, 121-131.
- Daniel, G. E. The respiratory quotient of *Balantidium coli*. *Am. J. Hyg.*, 1931, 14, 411-420.
- DeLanney, L. A., and Beahm, E. H. *Balantidium coli*: report of case with proctoscopic study. *J. A. M. A.*, 1943, 123, 549-550.
- Dobell, C., and Laidlaw, P. P. On the cultivation of *Entamoeba histolytica* and some other entozoic amoebae. *Parasitology*, 1926, 18, 283-318.
- Elliott, G. B., and Hotson, R. Balantidial dysentery. *Canad. M. A. J.*, 1953, 69, 317-319.
- Ferri, L. V. [Contribution to the epidemiology of balantidiosis.] *Med. Parasit. & Parasitic Dis., Moscow*, 1942, 11, 108-112. Abstract in: *Trop. Dis. Bull.*, 1943, 40, 459-460.
- Füsthly, Ö. Untersuchungen über das Vorkommen der Balantidiosis in Ungarn. *Zentralbl. f. Bakt.*, 1938, 142, 133-137.
- Glaessner, K. Ueber Balantidienenteritis. *Zentralbl. f. Bakt., Orig.*, 1908, 47, 351-362.
- Greene, J. L., and Scully, F. J. Diet in the treatment of *Balantidium coli* infection. *J. A. M. A.*, 1923, 81, 291-293.

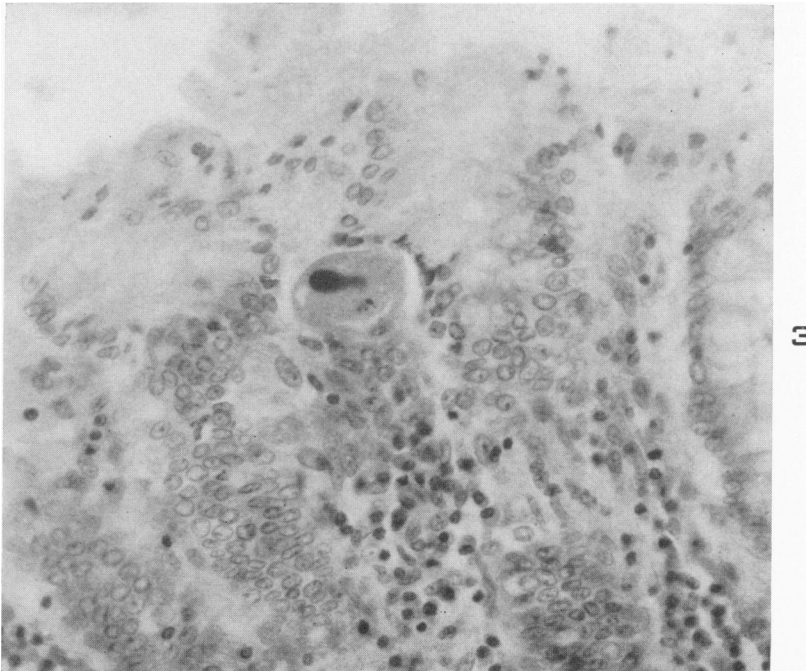
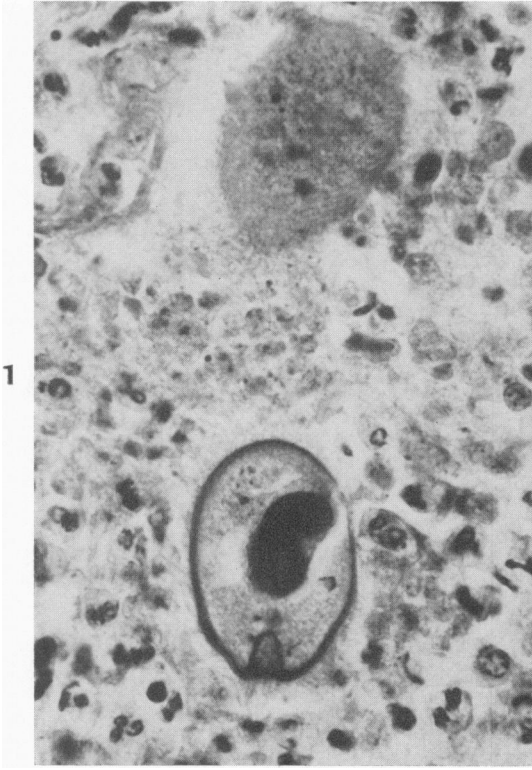
- Harms, H. Beitrag zur Histopathologie der Balantidiocolitis. *Virchows Arch. f. path. Anat.*, 1932, **284**, 422-437.
- Hegner, R. W. Animal infections with the trophozoites of intestinal protozoa and their bearing on the function of cysts. *Am. J. Hyg.*, 1926, **6**, 593-601.
- Hegner, R. Specificity in the genus *Balantidium* based on size and shape of body and macronucleus, with descriptions of six new species. *Am. J. Hyg.*, 1934, **19**, 38-67.
- Hinkelman, A. J. Pathogenicity, cultivation, and reproduction of protozoan parasites. *New York M. J.*, 1919, **109**, 235-240. (Cited by Wenyon.)
- Hoekenga, M. T. Terramycin treatment of balantidiasis in Honduras. *Am. J. Trop. Med.*, 1953, **2**, 271-272.
- Jaffé, R. Zur Pathologie der Balantidien-Colitis. *Zentralbl. f. allg. Path. u. path. Anat.*, 1919-20, **30**, 145-152.
- Jaffé, R., and Kann, C. Sobre el *Balantidium coli* en el apéndice y la apendicitis balantidiosa. *Rev. sudam. morfol.*, 1943, **1**, 74-82.
- Jarpa, A., and Allende, J. Un caso de balantidiasis humana. *Bol. Inform. Parasit. Chile*, 1953, **8**, 14.
- Junqueira, M. A. Balantidiose. *Hospital, Rio de Janeiro*, 1940, **18**, 811-818.
- Kipschidse, N. Zur Frage der pathologischen Bedeutung des *Balantidium coli*. *Arch. f. Schiffs- u. Tropen-Hyg.*, 1928, **32**, 253-255.
- Klein, E. *Paramaecium coli* and trichomonas in sewage. *Brit. M. J.*, 1896, **2**, 1852. (Cited by McEwen.)
- Knowles, R., and Das Gupta, B. M. Some observations on *Balantidium coli* and *Entamoeba histolytica* of macaques. *Indian M. Gaz.*, 1934, **69**, 390-392.
- Koppisch, E., and Wilking, V. N. Balantidial dysentery: report of 4 cases with post-mortem study. *Puerto Rico J. Pub. Health & Trop. Med.*, 1947-48, **23**, 185-224.
- Liu, H. L. Balantidium infection in man: report of a case from Chefoo. *Chinese M. J.*, 1941, **59**, 476-479.
- Logan, A. H. *Balantidium coli* and pernicious anemia: report of four cases. *Am. J. M. Sc.*, 1921, **162**, 668-674.
- Maia, C. da C. Aspectos clínicos y epidemiológicos da balantidiase humana. *An. Inst. med. trop.*, 1952, **9**, 1453-1465.
- Maldonado, R. Q. Personal communication.
- Maliwa, E., and Haus, V. v. Über Balantidieninfektion der Harnwege. *Ztschr. f. Urol.*, 1920, **14**, 495-501. (Cited by Wenyon.)
- Malmsten, P. H. Infusorien als Intestinal-Thiere beim Menschen. *Virchows Arch. f. path. Anat.*, 1857, **12**, 302-309.
- Manlove, C. H. Two cases of balantidial colitis. *Philippine J. Sc.*, 1917, s. B, **12**, 149-163.
- Masing, E. Über die Bedeutung des Magens für die Infektion mit *Balantidium coli*. *Klin. Wchnschr.*, 1929, **8**, 2380-2382.
- McCarey, A. G. Balantidiasis in South Persia. *Brit. M. J.*, 1952, **1**, 629-631.
- McEwen, F. J. Balantidium colitis. *M. Clin. North America*, 1924, **7**, 1289-1294.
- Neghmé, A.; Miranda, M.; Agosín, M., and Sanz, R. Contribución a la quimioterapia del *Balantidium coli*. II. Estudio clínico. *Bol. Inform. Parasit. Chile*, 1951, **6**, 7-8.

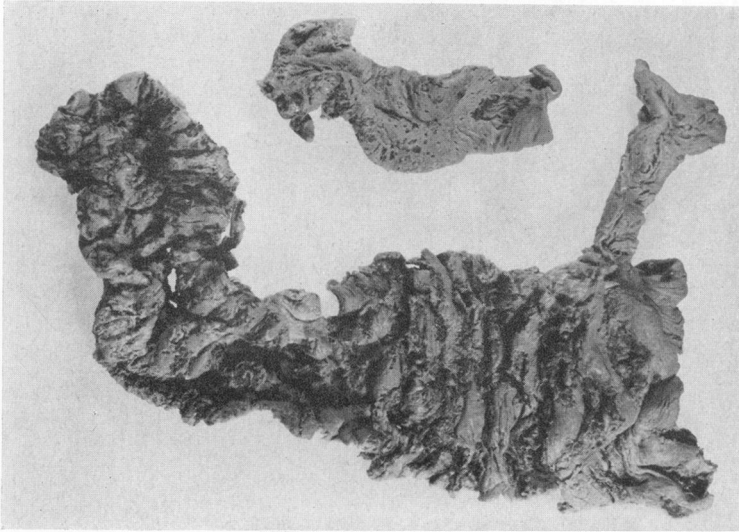
- Nelson, E. C. An intestinal content cultivation medium. I. Methods of preparation and use and data obtained in the cultivation of *Balantidium coli* from the pig. *Am. J. Trop. Med.*, 1940, 20, 731-745.
- Ostroumov, V. G. [Materials on the problem of the identity of *Balantidium suis* and *B. coli*.] *Med. Parasit. & Parasitic Dis., Moscow*, 1946, 15, 43-44. Abstract in: *Trop. Dis. Bull.*, 1947, 44, 904-905.
- Pawel, M. Um caso de balantidiose humana. *Arq. de biol.*, 1943, 27, 93-95.
- Potenza, L., and Martínez, A. Apendicitis balantídiana en un niño. *Rev. sudam. morfol.*, 1947, 5, 142-149.
- Pramanik, S. Balantidiasis. *Brit. M. J.*, 1947, 2, 794.
- Pritze, F. Beiträge zur Kenntnis des *Balantidium coli*. *Ztschr. f. Parasitenk.*, 1928, 1, 345-415.
- Santos, J. A. Aureomicina en el tratamiento de balantidiasis coli. *Bol. Asoc. méd. de Puerto Rico*, 1951, 43, 482-484.
- Schumaker, E. *Balantidium coli*: host specificity and relation to the diet of an experimental host. *Am. J. Hyg.*, 1930, 12, 341-365.
- Schumaker, E. Relation of *Balantidium coli* infection to the diet and intestinal flora of the domestic pig. *Am. J. Hyg.*, 1931, 13, 576-584.
- Selimkhanov, K. A. [Balantidial colitis in Azerbaidjan.] *Med. Parasit. & Parasitic Dis., Moscow*, 1945, 14, 70-73. Abstract in: *Trop. Dis. Bull.*, 1947, 44, 91.
- Serra, A. Balantidial dysentery in a child. Death following rectal administration of oil of chenopodium. *Puerto Rico J. Pub. Health & Trop. Med.*, 1930-31, 6, 443-444.
- Shegalow, J. P. Ein Fall von *Balantidium coli* bei einem 5-jährigen Mädchen. *Jahrb. f. Kinderh.*, 1899, 49, 425-441.
- Shun-Shin, M. Balantidial dysentery in Rodríguez and its treatment with mercury biniodide. *Brit. M. J.*, 1947, 2, 417-418.
- Silva, G. S. de P. Balantidiase humana: aspectos clínicos e therapeuticos. *Brasil-med.*, 1938, 52, 1005-1015.
- Sommerville, E. T. W. Sobre un caso de *Balantidium coli*. *Rev. Inst. Bact., Malbrán*, 1945-48, 13, 58-61.
- Stein, F. (1863) Ueber *Paramoecium(?) coli*. Malmst. Amtl. Ber. 37. Vers. Deutsch. Naturf. u. Artze, 165. (Cited by Wenyon.)
- Stewart, I. S. Dysentery in South Persia. *Brit. M. J.*, 1949, 1, 662-663.
- Stokvis, B. J. Paramecium in sputa. *Nederl. tijdschr. v. geneesk.*, 1884, 20, 4-5. (Cited by Wenyon.)
- Strong, R. P. The clinical and pathological significance of *Balantidium coli*. Bureau Publ. Print., Manila, 1904, 77 pp.
- Stshenovitsh, V. [On the occurrence of *Balantidium coli* and other intestinal protozoa in man.] *Med. Parasit. & Parasitic Dis., Moscow*, 1941, 10, 252-260. Abstract in: *Trop. Dis. Bull.*, 1943, 40, 313.
- Swartzwelder, J. C. Balantidiasis. *Am. J. Digest. Dis.*, 1950, 17, 173-179.
- Tsuchiya, H., and Kenamore, B. Report on a case of balantidiasis. *Am. J. Trop. Med.*, 1945, 25, 513-514.
- Tzaturian, A. Transactions of the Tropical Institute of Armenia (Russian), 1935. (Cited by Awakian.)

- van der Reis. Ueber die Bakterienflora des Darms. (*Balantidium coli* und pathologische Dünndarmbesiedlung.) *München. med. Wchnschr.*, 1923, 70, 835-836.
- Walker, E. L. Experimental balantidiasis. *Philippine J. Sc.*, 1913, s. B, 8, 333-349.
- Weinstein, P. P.; Garfinkel, B. T., and Miller, M. M. Treatment of a case of balantidial dysentery with terramycin. *Am. J. Trop. Med.*, 1952, 1, 980-981.
- Wenyon, C. M. Protozoology. Baillièrre, Tindall & Cox, London, 1926, 2, 1201-1210.
- Young, M. D. Balantidiasis. *J. A. M. A.*, 1939, 113, 580-584.
- Young, M. D. Attempts to transmit human *Balantidium coli*. *Am. J. Trop. Med.*, 1950, 30, 71-72.
- Young, A. D., and Walker, O. J. *Balantidium coli* infection in Oklahoma. *J. A. M. A.*, 1918, 70, 507-508.
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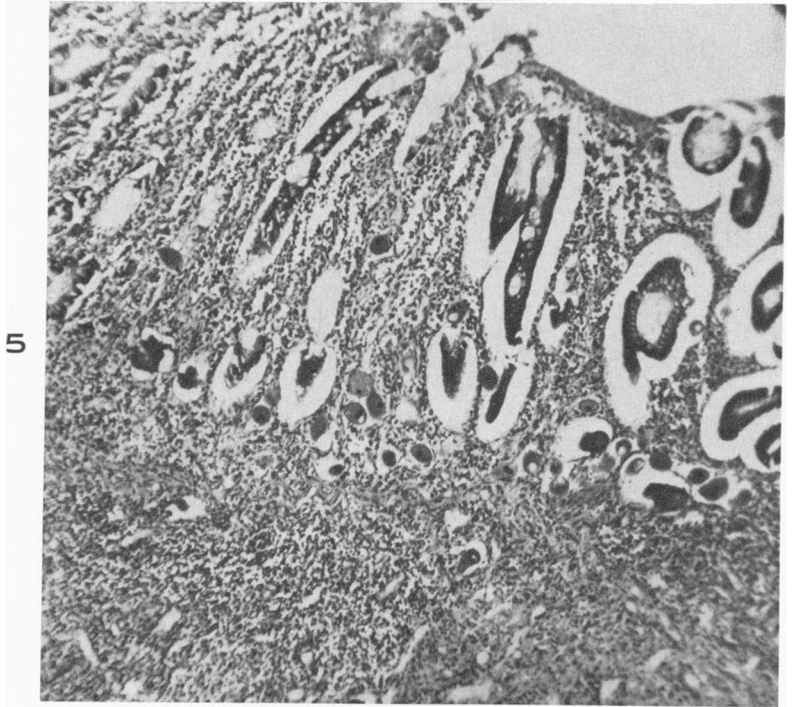
LEGENDS FOR FIGURES

- FIG. 1. Trophozoite stage of *Balantidium coli*, degenerated above, well preserved below. Of note are the kidney-shaped nucleus, peristome, and cilia. Phosphotungstic acid-hematoxylin (PTAH) stain. $\times 800$.
- FIG. 2. Cystic stage, in feces. Delafield's hematoxylin stain. $\times 800$.
- FIG. 3. Immediately about the penetrating organism the cells may be shrunken, with acidophilic cytoplasm and pyknotic nuclei. Hematoxylin and eosin stain. $\times 360$.





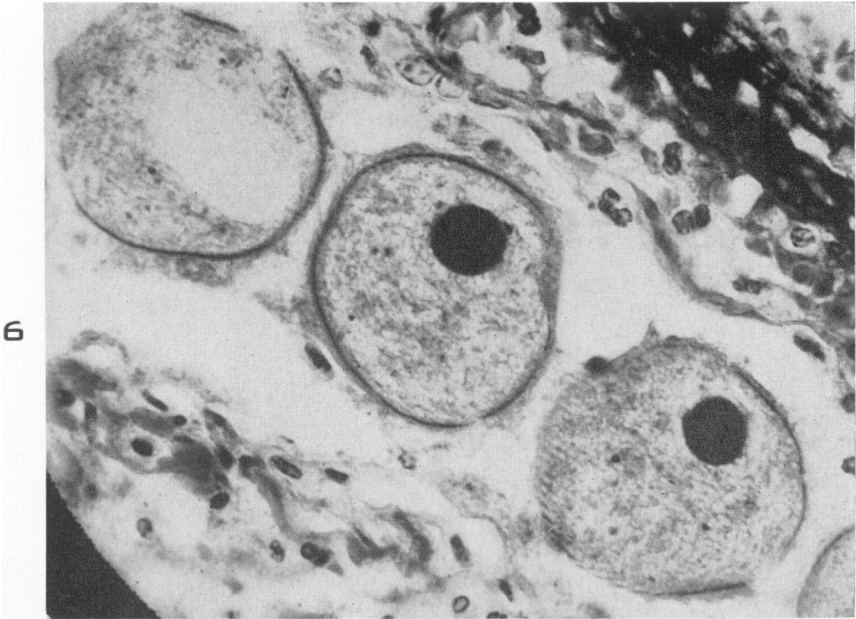
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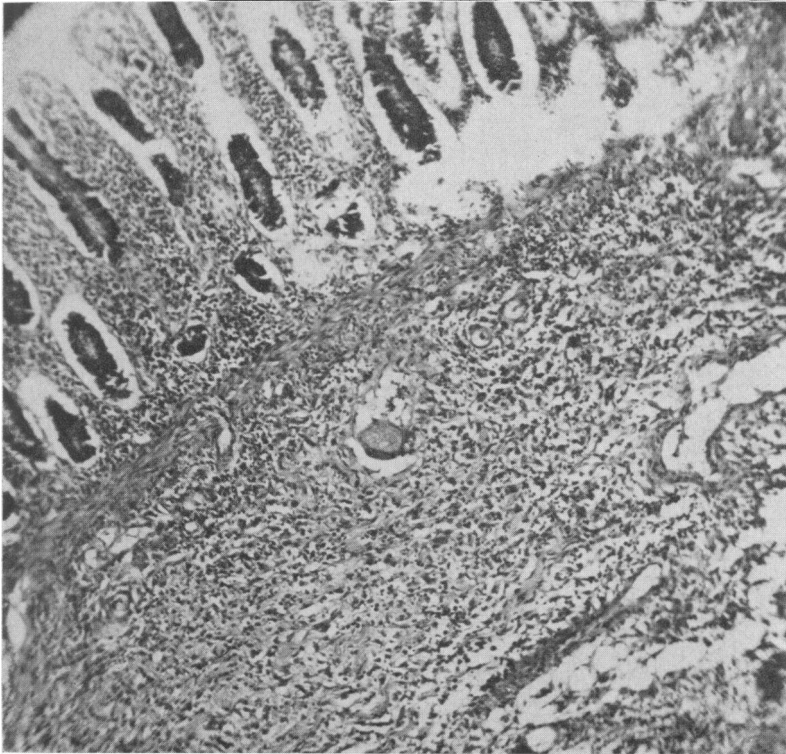
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FIG. 4. Fatal case of balantidial dysentery. The ragged appearance of the mucosa is evident. There were multiple ulcers from the rectum to cecum. Perforation of the colon had occurred.

FIG. 5. Balantidial colitis: numerous organisms in tissues and in dilated lymphatics; marked lymphocytic and monocytic infiltration without ulceration. Hematoxylin and eosin stain. $\times 80$.



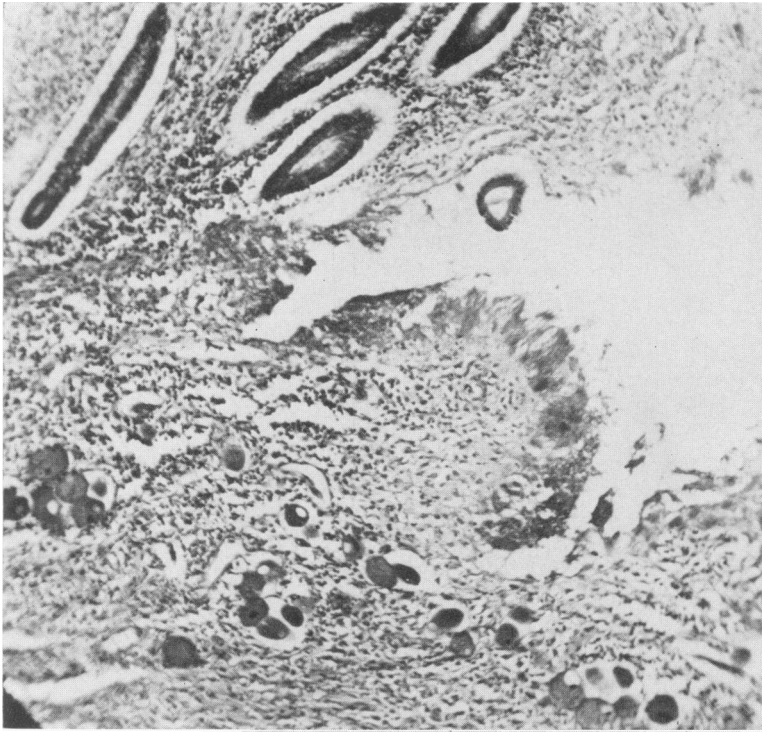
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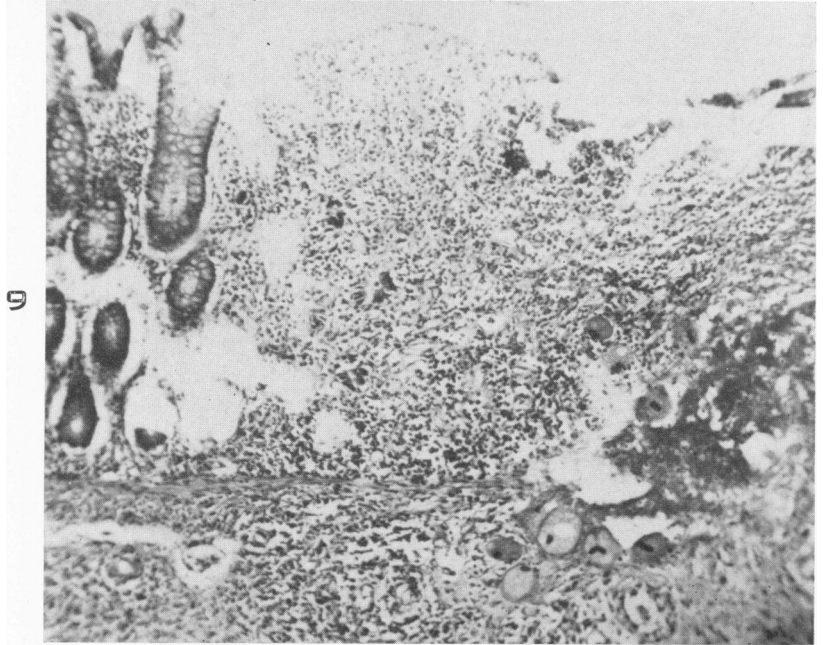
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FIG. 6. Balantidia in dilated lymphatic vessels of submucosa. PTAH stain. $\times 800$.

FIG. 7. Zone of necrosis in submucosa surrounded by numerous round cells; in the center a degenerated balantidium may be noted. Hematoxylin and eosin stain. $\times 80$.



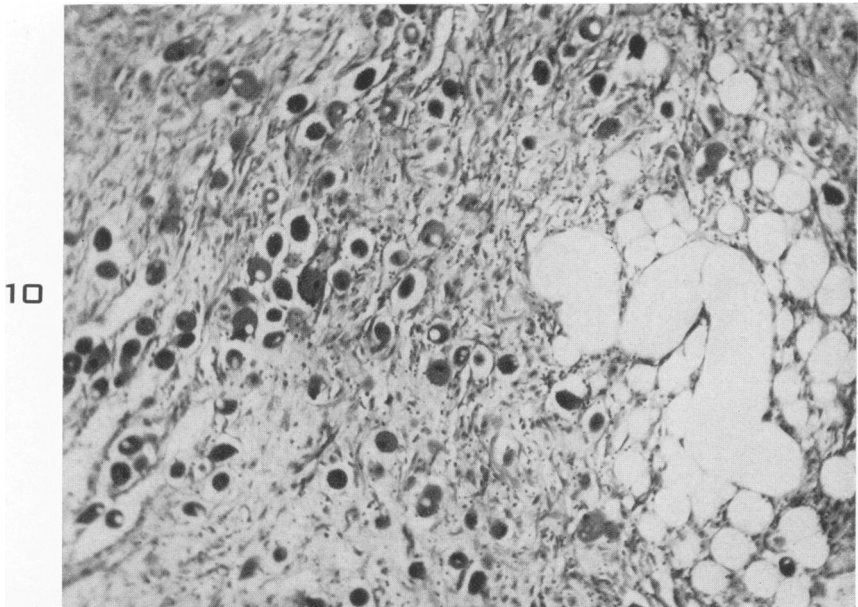
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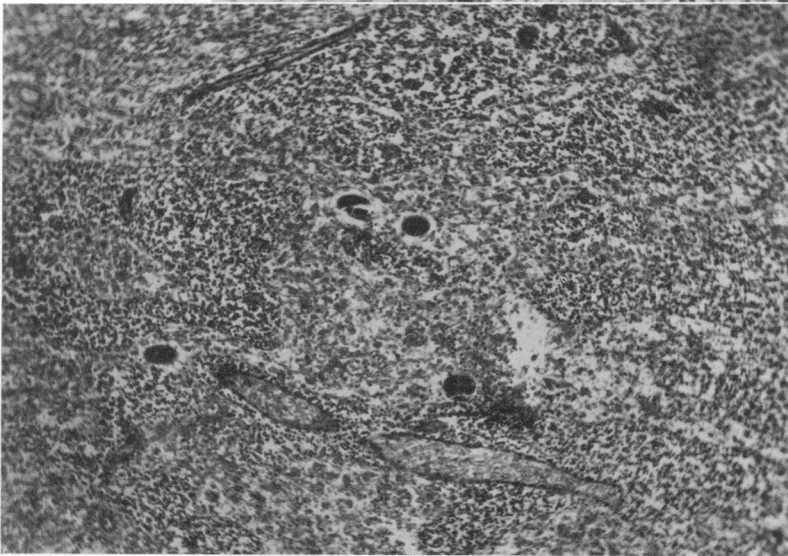
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FIG. 8. Colitis: undermined margin of ulcer with necrotic base, below which there are numerous organisms. Hematoxylin and eosin stain. $\times 80$.

FIG. 9. Necrotic material filling crater of ulcer; numerous balantidia, some perforating through the muscularis mucosae. Hematoxylin and eosin stain. $\times 80$.



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FIG. 10. Colitis: numerous balantidia invading pericolic fatty tissue, possibly by agonal or post-mortem wandering. Hematoxylin and eosin stain. $\times 80$.

FIG. 11. Lymph node from mesocolon with several balantidia; hemorrhagic and inflammatory reaction caused by the ciliate. Hematoxylin and eosin stain. $\times 80$.

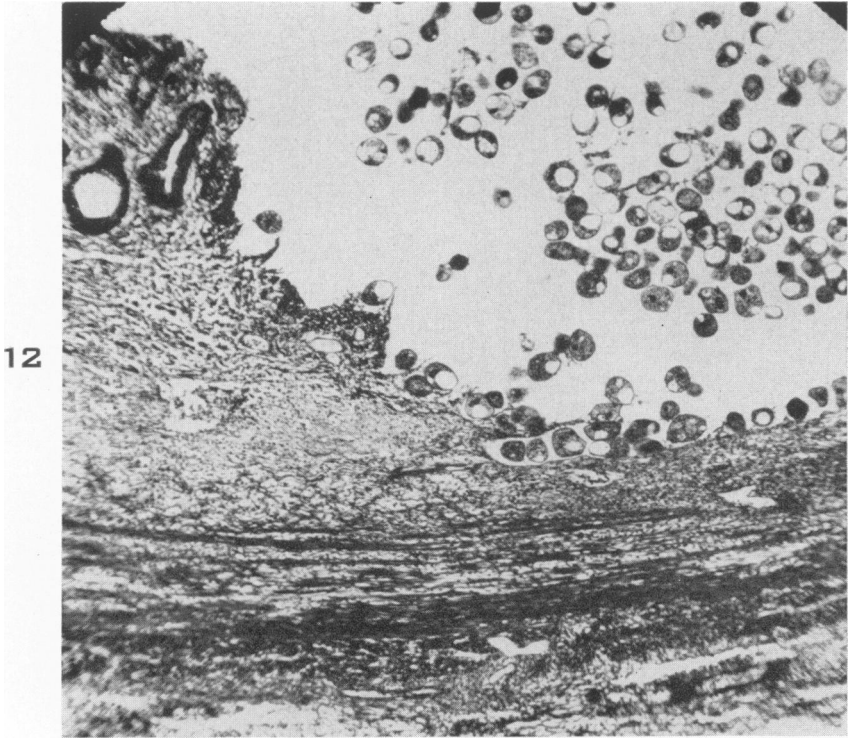
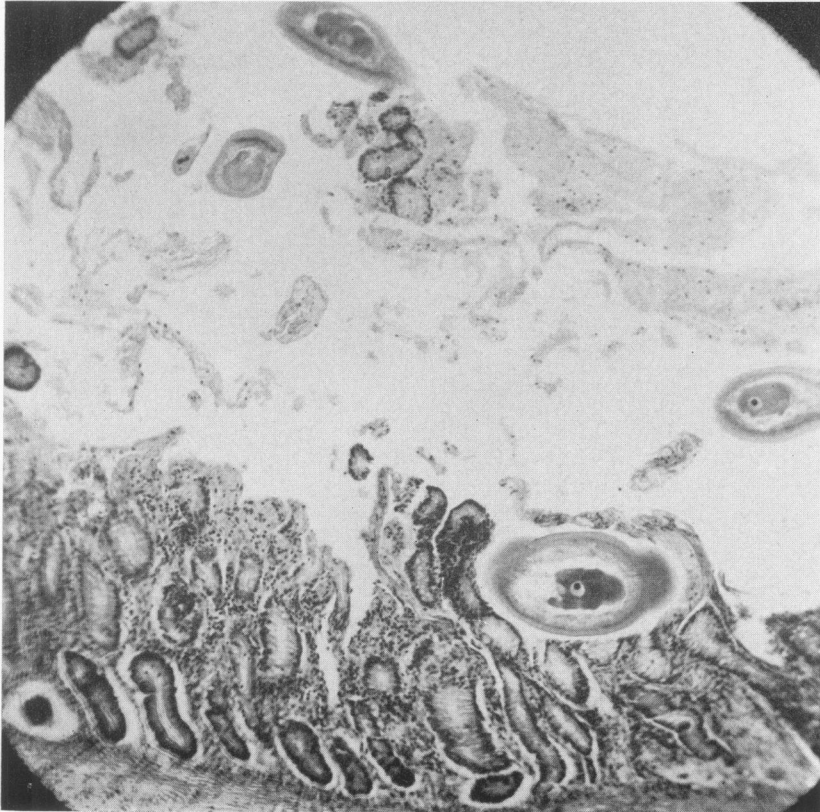
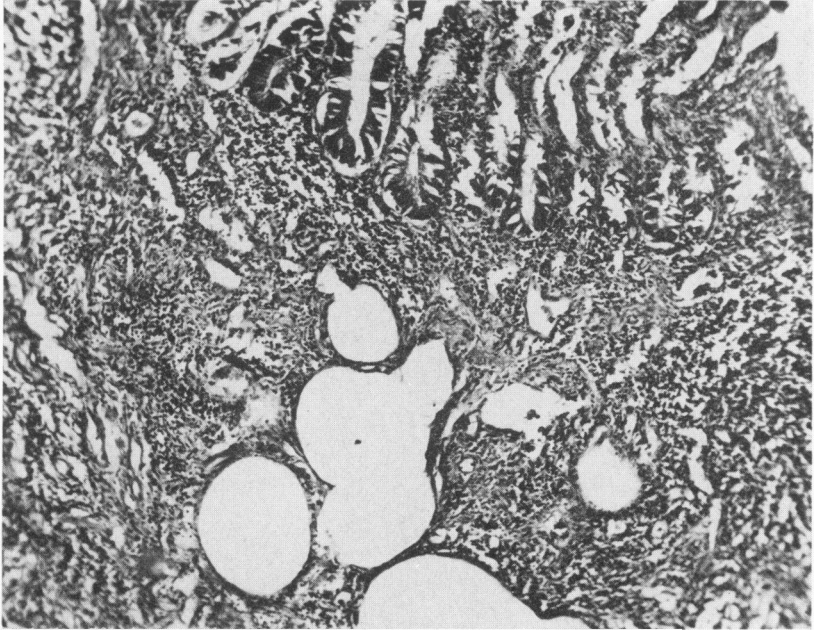


FIG. 12. Necrotizing balantidial appendicitis. Hematoxylin and eosin stain. $\times 80$.

FIG. 13. Marked dilatation of mucosal lymphatics in a case of balantidial dysentery. Hematoxylin and eosin stain. $\times 80$.

FIG. 14. Section from rectum showing implanted trichuris and two balantidia in lumen. Hematoxylin and eosin stain. $\times 80$.

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