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A CASE OF AMEBIC DYSENTERY.

BY LUDVIG HEKTOEN, M.D.,
CHICAGO.

THE following case of amebic dysentery does not present any special clinical or anatomical peculiarities. It shows that this form of dysentery must be borne in mind in the diagnosis of diarrheal diseases, especially in patients that come here from the South, although it is not considered at all unlikely that the disease may originate in any part of the United States; yet, as far as I know, amebic dysentery has not been recognized in Chicago either clinically or anatomically up to the present time, but then it is only very recently that the disease has been accurately described and studied with sufficient thoroughness both in its clinical aspects as well as in regard to the etiology and the local and quite frequent secondary changes, to establish its identity as a separate and distinct form of dysentery, due without any doubt to the invasion of the wall of the large intestine by the ameba coli or dysenteric, a protozoic parasite belonging to the group of rhizopoda. It is a nucleated, granular, or homogeneous and hyaline vacuolated organism, capable of active ameboid movements, varying in diameter from 8-35 μ. when resting, and round in outline.

While a few investigators were inclined to believe the ameba to be a practically harmless parasite because it was found in various intestinal disorders and even in the feces of healthy individuals, yet of late nearly, if not all, regard it as the primary causative agent of the peculiar form of dysentery with which its name has become associated. Baumgarten expressed the view in 1890 that pyogenic organisms play an important part, together with the ameba, in producing tropical dysentery. Councilman and Lafleur believe so firmly in the etiological relation of the ameba that they gave the dysentery the name amebic; they showed the ameba to be present in the stools, in the characteristic local intestinal lesions, in secondary foci in the liver, and in the sputum after rupture of the hepatic abscess into the lung. Their conclusion is certainly warranted in view of the successful inoculation experiments of Kartulis and Hlava upon cats and dogs with pure amebic cultures in straw infusion and with dysenteric

evacuations. Wesener in a recent resumé of the whole question of dysentery says that the amebæ are the primary factor in producing the disease, the changes being, probably, to some extent, caused by bacteria which may enter the tissue alone and independently or may be carried in by the amebæ.

The excellent clinical and anatomical studies of amebic dysentery by Councilman and Lafleur demonstrate the amebæ in the stools of the disease which is one of intermissions and exacerbations tending always to chronicity and to the early production of anemia; the ulcers are quite characteristic; they result from submucous infiltration with necrosis of the mucous membrane, giving the ulcer the undermined form; in the quite frequent secondary liver abscesses, purulent inflammation does not occur, the abscess being due to colliquative necrosis caused by the ameba which in the liver were not in their cases found associated with any other organisms.

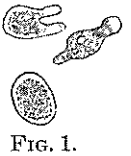

FIG. 1.

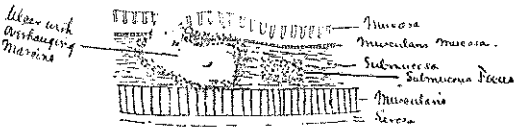

FIG. 2.

Fig. 1.—Amebæ Dysenteriæ Coli. (Jaksch).

Fig. 2.—Diagrammatic representation of excavated Ulcer and Independent Submucous Focus of Infiltration modeled after section made from Colon Ulcer.

It is much regretted that the clinical history of this case should be so imperfect.

M. M., male, age 20, professional bicycle rider, came to Chicago, which was his home, from Galveston, Tex., in November, 1891. When he reached home he was quite sick and the physician in charge made a diagnosis of typhoid fever. He had fever, diarrhea with frequent and copious hemorrhages from the bowels; he became greatly emaciated and anemic. Shortly before death, the diagnosis was changed to yellow fever and this was certified to as the cause of the patient's death after an illness of altogether six weeks.

The supposed occurrence of a case of yellow fever stirred the Health Department up, and I was requested to make a post-mortem examination. This was done 48 hours after death. The undertaker had made what is technically known as cavity and arterial embalming both, and this interfered materially with the recognition of any possible changes in the parenchymatous organs. The body was greatly emaciated, the skin white in color. There was no peritonitis and no intestinal adhesions. For the sake of brevity it may be said that none of the organs examined, the heart, lungs, liver, kidneys, spleen,

pancreas, stomach and intestines, showed any changes of importance except the large intestine. There was, however, a marked general anemia, the solid organs containing but very little blood. The liver did not contain any abscess large enough to be recognized with the naked eye; areas of local parenchymatous necrosis could not be made out. The small intestine showed no changes in its lymphatic apparatus. The brain and spinal cord were not examined. In the large intestine the changes were found furthest advanced in the cecum and in the hepatic, splenic and sigmoid flexures of the colon. The transverse colon and the rectum showed the mucous membrane to be quite healthy in places, with here and there characteristic ulcers and excavations. The contents of the large intestine were gray or gray and yellow in color, liquid in consistence, and mixed with lumps of mucous, shreds from the intestinal mucous membrane, and occasionally a little blood could be distinguished. Selecting, for description, a section from the hepatic flexture of the colon which appears to present the changes in their greatest intensity, it will be seen that the mucous membrane is riddled with ulcerations of varying extent, shape and depth; in places may be seen small as well as extensive superficial areas of loss of substance in the mucosa only, exposing the submucosa, which may appear to be softened and gray, or quite firm, the margins of the ulcer being nearly always a little undermined; then there are also to be seen deeper ulcers with the smooth muscular coat as the floor, the cavity being either empty or else filled with a loose or slightly adherent plug or mass of softened, necrotic tissue; the margins of these deeper ulcers are overhanging, *i. e.*, undermined to such an extent that mere inspection gives one no idea at all of the extent of the ulcer; if the intestine be floated upon water, the margins of the deep ulcers will spread out and a probe may be passed to quite an extent under the margin beyond the edge, and in some instances it may emerge into another neighboring ulcer, showing the existence of submucous passages connecting adjacent cavities. Usually the edges of these overhanging margins are shreddy and apparently undergoing softening. In one place, in the hepatic flexure of the colon, is an area of softening which extends deeper than usual; it is situated on the posterior wall and, upon but very slight manipulation, a complete perforation results, the entire thickness of the wall being in a state of gelatinoid softening so that the borders of the perforation are confluent and shreddy. No retroperitoneal changes were recognized posteriorly to this extensive area of necrosis. Near by was an oblong ulcer running transversely to the long axis of the bowel and extending in a more or less terraced manner down through all the coats of the intes-

tine to the serosa. Both these very extensive foci of destruction are located in the midst or the vicinity of an area of apparently healthy mucous membrane, which covers a portion of intestine perceptibly thicker than elsewhere; on close examination, minute openings in the mucous membrane can be made out as well as more extensive superficial ulcerations; the minute openings lead down to places of submucous thickening or infiltration, and in a few places a gray, viscid, turbid, semi-solid material can be brought out, leaving a submucous cavity. In the cecum there are extensive areas of ulceration with but softened shreds of the mucosa here and there over the almost continuous ulcer, upon which are also found large brownish necrotic

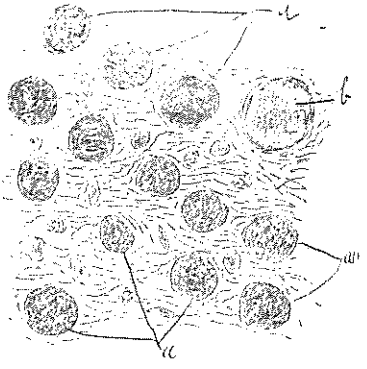

FIG. 3.

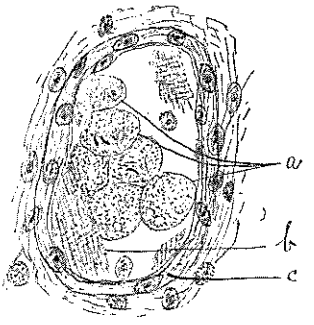

FIG. 4.

Fig. 3.—*a*. Amebæ from floor of deep ulcer. *b*. Vessel with fibrinous clot. Amebæ deeply stained with methylene blue; vacuoles very faint; no nuclei to be seen. Litz Obj. ⅛ Oil immersion; eye-piece 3; tube length; 160 mm.; camera lucida drawing with paper on level with base of microscope.

Fig. 4.—*a*, Amebæ in interior of vessel; cell and deeply stained, rod shaped bodies in interior, *b*, Fibrinous clot; *c*, Wall with cell infiltration. Hæmatoxylin staining; otherwise same as Fig. 3.

masses. In but very few places are any signs of healing visible in the shape of ulcers with smooth walls and floor, and hard, rounded and thickened margins. In shape the ulcers are, as already indicated, of great variety; some are round, others oval, others oblong, and others again irregular; the large, oblong ulcers are usually transverse to the long axis of the bowel. Where the ulcers were largest and most extensive there would not be found so much thickening in the intestinal wall as about the small openings, apparently because in the case of the large ulcers cavities had been exposed and emptied of their gelatinous contents. Microscopic examination of the intestinal contents and of the viscid masses from the cavities in the submucosa showed no moving amoeba at all, but some large, granular bodies,

usually oval, sometimes oblong, which were thought to be dead and motionless amebæ; there were also found pus and red blood corpuscles. Pieces of intestine were hardened in alcohol, imbedded in paraffin or celloidin, microtomized and stained in various ways, with carmine, hematoxylin and Löffler's solution of methyl blue. From a study of these sections it is seen that beyond a general loss of surface epithelium there does not seem to be any change in the mucosa except directly about the ulcers. The ulcers almost invariably present a flask-shaped appearance, the body of the flask being located in the deeper parts of the mucosa and in the submucosa, while the neck, as it were, passes through the glandular stratum and opens upon the surface of the mucous membrane. The bottom of these ulcers is usually formed by the muscular coat; sometimes the necrosis extends further and may, as shown above, pass clear through the intestinal wall. The smaller ulcers of this description plainly show the process to be further advanced in the submucosa than elsewhere, giving us the characteristic undermined edges; in some places the cavity contains a mass of homogeneous material through which are scattered cells and nuclei, and the submucous tissue at the sides of the ulcers gradually loses its fibrillated structure and becomes clear and homogeneous. There is much cellular infiltration into the edges of the overhanging mucosa which often appears to have become folded inward so as to line the neck of the flask-shaped ulcer with glandular follicles; in places, again, the mucosa appears to be undergoing destruction, and the large, flattened ulcerations do not show the submucous excavation so well. The tissue around the ulcers is, as a rule, infiltrated with small, round cells; perhaps this is most marked at the floor, and it is in this locality that the amebæ are found most numerously; occasionally they are also found between the glandular follicles and in the necrotic contents *in situ* in the ulcers. The amebæ found are invariably granular; a nucleus is not always demonstrable; vacuoles are barely indicated in sections stained with hematoxylin under high power; no such structural details can be made out, no matter what staining is used, as was done by Councilman and Lafleur, and others. The amebæ usually stain uniformly and rather faintly compared to the cell nuclei around them, but sometimes one part of an amebæ stains deeper than the rest; they often contain other and smaller cells; they appear about twice, or more, as large as the leucocytes and gave the impression of being smaller than those usually described in literature; they lie in the tissue in groups of a few, as a rule, and often they are found in the interior of lymphatic vessels. The muscular coat often shows the infiltration of round cells in rows

or groups. The vessels around the smaller ulcers are usually full of blood corpuseles and often partly filled with fibrinous thrombi; the wall sometimes appears very much thickened, at other times it is the seat of a round cell infiltration; some of these changes may be found in the subserous vessels. The lymph follicles appear to be involved only by continuity of tissue.

Remarks.—As far as the clinical history of this case goes it answers but fairly well to the clinical description of amebic dysentery; had amebæ been looked for in the stools they would undoubtedly

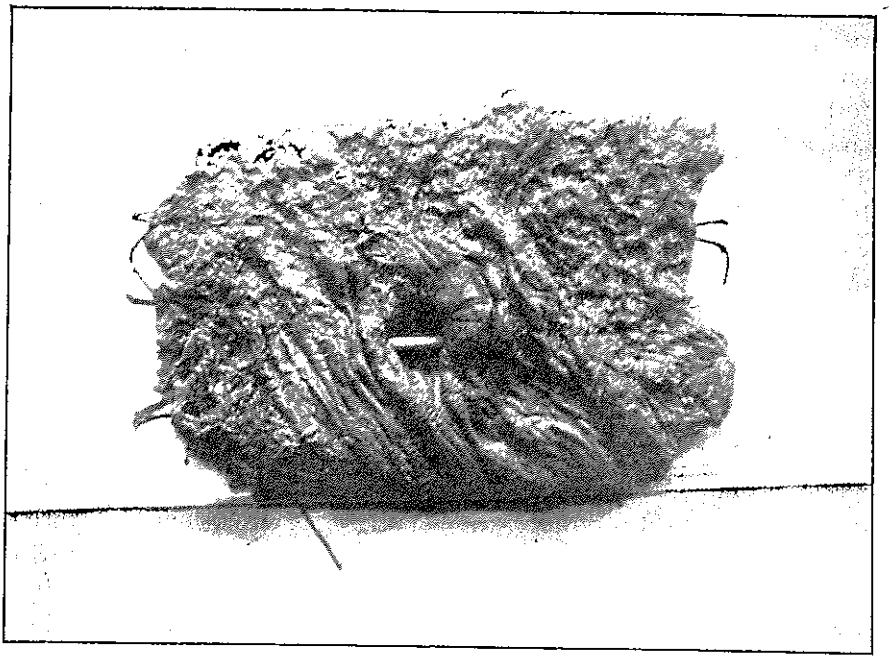

Fig. 5.—Photograph of part of the large intestine described, showing amebic ulcers and one perforation, margins of which are held apart by small stick of wood.

have been found. The thickening and the ulcers in the colon are so typical of this variety of dysentery that an anatomical diagnosis could unhesitatingly be made; microscopically the changes are identical with those described by Councilman and Lafleur, and show the origin of the ulcers to be due to a primary submucous infiltration and necrosis extending to the mucosa and resulting in the formation of a submucous cavity with undermined edges, which may be filled with a mass of softened, gelatinous necrotic tissue or, when older, it may be empty; the floor and the walls of the ulcer are infiltrated with round cells, and scattered through the tissue are found the quite characteris-

tic amebæ which it is right to regard as the primary etiological factor. The absence of distinct vacuolation, the somewhat feeble affinity for some stains and the uniform granularity of the amebæ are perhaps wholly or in part traceable to the length of time between death and the post-morten examination; at any rate they could not be satisfactorily demonstrated in the feces 48 hours after death, and this agrees with the experience of others, who all advise that they are best demonstrable in the warm discharges; as the body temperature declines, spontaneous movements cease in the amebæ, which become granular and difficult to recognize.

119 Loomis Street.

1. Path. Mykologie, Bd. II, 1890, quoted by Councilman and Lafleur.
2. The Johns Hopkins Hospital Reports, Vol. II. Nos. 7, 8, 9, 1891.
3. Centrabl. für Bacteriologie, Bd. 8, 1890; Bd. 9, 1891, and Virchow's Archiv., Bd. 105; 1885 and Bd. 118, 1889.
4. Centralbl. für Bacteriologie, Bd. 1, 1887.
5. Centrabl. für Allg. Path. and Path. Anat., Bd. III, Nos. 12 and 13, 1892.

26.- Traumatic detachment of a shred of the prepatellar bursal lining causing chronic inflammation; removal; recovery.
West. Med. Recorder 1892, 14, p. 121.

27.- Extrophy of the bladder, epispadias, rudimentary penis, pubic diastasis and inguinal retention of testicle.
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