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# Bacillary Dysentery and Chronic Ulcerative Colitis in World War II

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Bacillary dysentery has long been recognized as a major military disease. Next to malaria, it was the prevailing disease, during World War II, among the American forces in tropical areas and the second greatest disease threat in number of cases among overseas troops. Since 1933 a rather striking increase in incidence of the disease among civilians throughout the world has also been noted. In the United States the number of reported cases in 1944 was approximately 60 times greater than in 1933.

The subject of bacillary dysentery has been recently reviewed elsewhere (*1*). There is, however, a great paucity of data regarding the chronic manifestations of the disease. Evidence has been presented that these appear as diffuse cicatrizing, ulcerative, polypoid lesions of the small and large bowel known as regional ileitis (enteritis) and chronic ulcerative colitis. This view has met with considerable resistance in some circles, although typical chronic ileitis and ulcerative colitis have been demonstrated in many individuals followed from the acute to the chronic phase. The first extensive group follow-up was that of the atypical Flexner dysentery epidemic which occurred in Jersey City in 1934 (*2*). Of 210 patients hospitalized at the Medical Center, 122 were studied for periods varying from 9 to 12 months. Of the latter, 10.7 per cent developed chronic ulcerative colitis or ileitis. Subsequent studies elsewhere by other investigators have closely approximated this figure, so that we may reasonably assume that 1 of 10 patients with acute bacillary dysentery will probably develop the chronic form of the disease—usually ulcerative colitis. One of the major contentions of those opposing the bacillary dysentery etiology of chronic ulcerative colitis is that, if true, we should see many instances in connection with wars, since bacillary dysentery is largely a military disease.

The present communication concerns 61 cases of chronic ulcerative colitis occurring in American military personnel, including some war prisoners, of World War II. Of the total, 50 were studied from 5 to 30 months after the onset of diarrhea. In the remainder, the symptoms and signs were of slightly shorter or longer duration than the period specified. In 33 instances, the onset of the disease was definitely traced to outbreaks, 3 of them occurring aboard transports and 30 in military camps. Almost all patients incurred their initial acute bacillary dysentery in known endemic and epidemic areas, chiefly New Guinea, India, the Philippines, and North Africa. Accurate bacteriologic data during the acute phase was sparse, due to the lack of adequate laboratory facilities or the stress of combat service. In 5 of the outbreaks, *Shigella paradysenteriae* (Flexner's bacillus) was isolated. Confirmatory epidemiologic evidence was sometimes obtained in cases where initial cultural studies were not carried out. This consisted of positive cultures for *B. dysenteriae* in other military personnel who had diarrhea at the same time and place as those who were not studied bacteriologically. All cases were diagnosed as "dysentery," "GI's," "intestinal infection," "gastroenteritis," or "Delhi belly"—terms which, on the basis of previous studies, are now recognized as being practically synonymous with bacillary dysentery. All patients were treated with sulfonamides.

The general clinical picture during the acute phase in all patients included in this report was the abrupt onset of abdominal cramps, diarrhea, and fever. The bowel movements were watery, mucopurulent, or bloody. In 5–10 days these symptoms and signs subsided only to be followed by the characteristic postdiarrheal phase of constipation (healing phase). No sigmoidoscopic studies were made during the acute stage. There followed recurring episodes of bloody diarrhea, and in 8 patients the diagnosis of chronic ulcerative colitis was subsequently made in military hospitals. Fifty of the 61 patients were subjected to sigmoidoscopic study 5–30 months after their initial infection. All patients exhibited the typical hyperemic, granular mucosa or ulceration and purulent cytology of the mucosal exudate. Mural fibrosis and luminal stenosis were usually present only in cases of long duration (a year or more). Five patients in the present series also exhibited a concomitant distal ileitis, one confirmed by X-ray.

Of 12 patients receiving fecal cultures during the acute phase, 5 (41.6 per cent) were positive for *B. dysenteriae*. Of the 61 patients examined by us during the chronic phase, 6 (9.8 per cent) revealed *B. dysenteriae* by the mucosal crypt aspiration method. One patient exhibited positive cultures during both acute and chronic phases. Thus, out of a total of 61 patients with chronic ulcerative colitis, 10, or 16.4 per cent, exhibited positive cultures. This figure is significant since so few were studied initially, and the recovery of the dysentery organism in 9.8 per cent after 5–30 months appears in marked contrast to our control group where the incidence is 0.08 per cent.

The evidence presented is deemed to be of sufficient importance to lend additional support to our contention that chronic ulcerative colitis and ileitis are the result of acute bacillary dysentery. It is of particular relevancy at this time, since many of our veterans are finding it difficult to establish service-connected disability in chronic ulcerative colitis and ileitis because the initial acute phase has been forgotten or inaccurately diagnosed. It is quite probable that the present series of 61 cases forms but a small fraction of the actual number occurring in veterans of World War II.

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