EDITORIAL

Amebic Abscess of the Liver

A n increased incidence of amebic abscess of the liver was noted in the United States as a result of World War II, and a similar rise can be expected from the war in Vietnam. Recent articles in The American Journal of Digestive Diseases by Wenger and Coberly,1 and Knauer2 (this issue), and by Cain et al.3 and Johnson et al.4 in 1968 reflect this increased incidence and a renewal of interest in the disease. The articles in this issue invite comment.

The clinical picture presented by Knauer's 15 patients is most unusual because of the high incidence of secondary bacterial infection (8 cases). The reason for this high infection rate is not clear, but it should alter the usual clinical findings in amebic abscess of the liver, causing more prominent fever, leukocytosis, pleural effusion, and abnormal results in liver function tests. Although Knauer emphasizes the frequency of abnormal results in liver function tests in his patients, most still fit the usual pattern of minimal or no jaundice, elevated levels of sulfobromophthalein and serum alkaline phosphatase, and little, if any, elevation of serum glutamic transaminase levels. He correctly indicates that this pattern of abnormality is not specific for amebic abscess, but also occurs with other focal lesions of the liver, such as hepatoma, metastases, and echinococcal cysts. Although for many years Entamoeba histolytica could not be cultured in sterile medium, recently the organism has been cultured successfully on a sterile medium by Diamond at the National Institutes of Health.

Authors of both articles correctly emphasize the value of radioisotope scanning of the liver and serologic tests for rapid diagnosis of amebic abscess of the liver. I particularly recommend gold-198 scanning, and often use it as an emergency procedure to identify and locate a suspected abscess. I also prefer to attempt aspiration of suspected lesions prior to antiamebic therapy to prove the presence of an abscess and to obtain material for bacterial culture and examination for amebae. Demonstration of pus becomes more important when the patient is in the cancer-suspect age range or when symptoms suggestive of an infectious or inflammatory process are lacking, since amebic abscess occasionally mimics the clinical findings of cancer of the liver.

The indirect hemagglutination (IHA) test for amebiasis, as performed by Lewis and Kessel5 or Milgram et al.,6 also is extremely useful in diagnosis of invasive amebic disease, although this test is not readily available at the present time. Care must be exercised in applying this test clinically. While most patients with amebic abscess have a positive IHA test, we have encountered a
few with a negative test. The IHA test also is positive in the majority of patients with invasive amebiasis of the colon. Titers can remain elevated for months after successful treatment of invasive amebiasis, as is nicely illustrated by titers in the patient described by Wenger and Coberly. Thus, a positive IHA test for amebiasis simply indicates recent infection, but does not "prove" the presence of either an amebic abscess or invasive amebic colitis; it does increase the odds for such a diagnosis, however.

Aspiration of bacteriologically sterile fluid from a liver abscess (in which case the fluid usually lacks a foul odor) is sufficient for a presumptive diagnosis of amebic abscess. Although highly suggestive and considered classic, brown or anchovy-sauce-colored fluid often is not obtained from amebic abscesses of the liver on the initial aspiration—the fluid is just as likely to be whitish or greenish in color. Demonstration of trophozites of *E. histolytica* is the only proof of the diagnosis. However, an adequate clinical response to treatment with antiamebic drugs generally is considered as confirmation of the presumptive diagnosis of amebic abscess (when there is evidence that an abscess is present) since amebae usually are found in the abscess fluid in only 50% of cases. The presence of *E. histolytica* in the stool also does not "confirm" the presence of amebic abscess of the liver, but does increase the odds somewhat for such a diagnosis when the proper clinical findings are evident.

The antiamebic therapy used by Wenger and Coberly, based on the article by Sepulveda *et al.*, is not in the form recommended by most authorities since the dose of emetine hydrochloride is half and the dose of chloroquine phosphate twice that usually used. Diiodohydroxyquin also is generally given for 3 weeks, rather than for 1 week. The most reliable clinical information about amebiasis comes from the Amoebiasis Research Unit, Durban, South Africa; their experience is summarized in Wilmot’s book *Clinical Amebiasis*. They have experienced few instances of serious difficulty when emetine hydrochloride is used in proper dosage. They recommend administration of both emetine hydrochloride and chloroquine phosphate since either drug alone is associated with a small but significant incidence of treatment failure. The dose of emetine hydrochloride is 1 mg./kg. of body weight (maximum dose 65 mg.) daily in two divided doses intramuscularly for 7–10 days. Chloroquine phosphate is given orally in two divided doses daily as follows: a loading dose of 1 gm. on each of the first 2 days, then 0.5 gm. daily for 3–4 weeks. Emetine hydrochloride and chloroquine phosphate can be given concurrently, or emetine hydrochloride first, followed by chloroquine phosphate. I have used half the above dose of emetine hydrochloride for a few patients who were critically ill and appeared to be unusually poor risks for this drug, but this is rarely necessary.

Two new drugs not yet approved for treatment of amebiasis in the United States eventually may replace the above agents: Dehydroemetine is as effective clinically as emetine hydrochloride, but the former is less toxic because of a