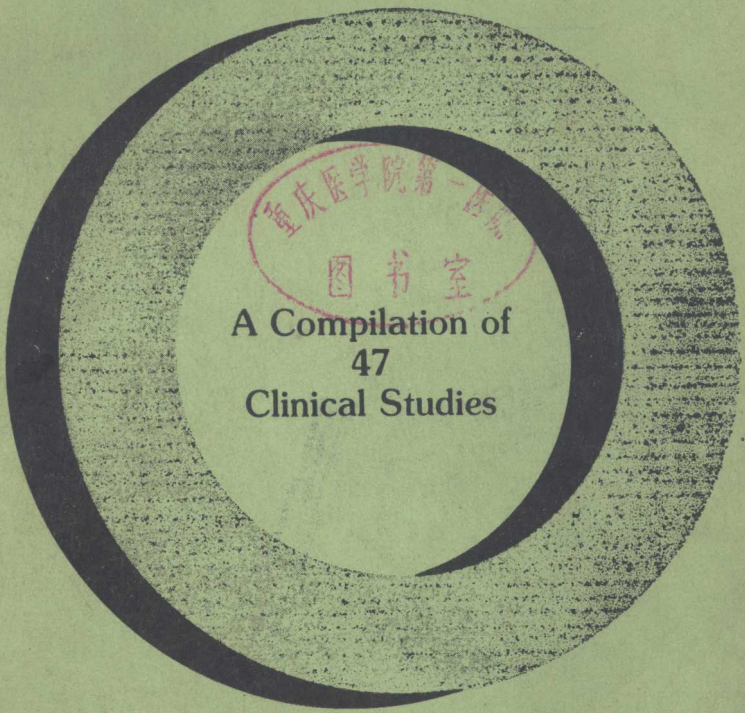
Parasitic Diseases Case Studies



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Parasitic Diseases

Case Studies

A Compilation of 47 Clinical Studies

By

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preface

Over the course of my many years of teaching Parasitic Diseases, I have found the most useful tool in illuminating the nature of these to be the case history. Herein are compiled case histories accumulated over a period of time. I have seen most of the cases described in this book. My mentor, Harold W. Brown, M.D., has shared with me a number of cases, and a few have been acquired from colleagues who have known of my interest in this area of medicine.

The purpose of this book is to review for the practitioner and inform the student of the salient features of the parasites that affect humans. Because parasites have peculiar life cycles and oftentimes a unique epidemiology, an outline of almost every parasite follows the appropriate case histories. For in-depth discussion the reader is referred to the texts listed below. The references cited with the case histories either supplement the texts or contain material that updates them, but in almost every instance they are clinically oriented.

The following four texts are recommended to the reader:

Brown, Harold W.: *Basic Clinical Parasitology,* Fourth Edition. New York: Appleton-Century-Crofts, 1975.

Faust, Ernest C., Russell, Paul F., Jung, Rodney C.: *Craig and Faust's Clinical Parasitology,* Eighth Edition. Philadelphia: Lea and Febiger, 1970.

Hunter, George W. III, Swartzwelder, J. Clyde, Clyde, David F.: *Tropical Medicine,* Fifth Edition. Philadelphia: W.B. Saunders Company, 1976.

Wilcocks, Charles, Manson-Bahr, P. E. C.: *Manson's Tropical Diseases,* Seventeenth Edition. Baltimore: Williams and Wilkins Company, 1972.

In addition, because of the peculiar geographic distribution of some of the parasites, an epidemiological table has been provided in the Appendix as a guide to the clinician both in elucidating the possible causes of a patient's illness and in giving advice to an ever-increasing number of travelers.

I trust that this book will prove stimulating. Suggestions for improvement will always be welcome. The opinions expressed herein are my own and do not necessarily reflect those of the U.S. Government.

David N. Reifsnyder, M.D.

contents

PARASITIC DISEASES

CASE 1: DIARRHEA AND WEIGHT LOSS IN AN EXECUTIVE

HISTORY

A 50-year-old perfume manufacturing executive was in Leningrad, Russia, working on an agreement for his company. Towards the end of the second week he had noted the onset of 4-6 liquid, nonfoul smelling stools daily, with occasional blood and mucus. He took Lomotil with some relief.

At the end of the third week he returned to the United States. The liquid stools persisted and a ten-pound weight loss ensued. He felt warm at times but had no chills or frank fever. He denied anorexia, nausea, vomiting, malaise, or hematochezia. He finally consulted a physician when home remedies did not result in an abatement of the diarrhea. The physician performed sigmoidoscopy, which revealed some ulcerations. Ulcerative colitis was considered, but because of the travel to Leningrad, he was referred to a specialist for further examination. Swabs were not taken at the time of sigmoidoscopy.

EXAMINATION

The patient presented as a well-developed, well-nourished man. Gross evidence of weight loss was lacking. Except for tenderness in the RLQ and LLQ of the abdomen, the physical examination was normal.

LABORATORY

White blood cell count - 11,000 with 80% neutrophils, 19% lymphocytes, 1% monocytes. Examination of a freshly passed liquid stool revealed motile trophozoites of Entamoeba histolytica. This was confirmed with stained smears. An indirect hemagglutination test for ameba was negative.

CLINICAL COURSE

The patient was treated with a combination of diiodohydroxyquinoline (Diodoquin) and oxytetracycline (Terramycin). There was some improvement. However, at the end of the course of treatment he had the onset of liquid stools occurring 6-8 times a day, and over a period of three weeks he lost another five pounds. On re-examination of the stool, amebas were still present. Metronidazole (Flagyl) was then prescribed and over a three- to four-day period the stools returned to normal. After a three-year follow-up he had no recurrence of the symptoms.

QUESTIONS

1. Amebiasis may mimic
   A. ulcerative colitis
   B. appendicitis
   C. bacillary dysentery
   D. depressive neurosis

2. The indications for a purged stool examination are
   A. nondiarrheal stool for ameba
   B. routine ova and parasites
   C. schistosomiasis

3. The drugs of choice for acute intestinal amebiasis are
   A. diiodohydroxyquinoline and tetracycline
   B. emetine
   C. metronidazole
   D. chloroquine

4. The asymptomatic cyst passer should be treated with
   A. diiodohydroxyquinoline
   B. metronidazole
   C. paromomycin
   D. no drug

ANSWERS

1. (A, B, C, D) All are true. Any patient in whom a diagnosis of ulcerative colitis is considered should have serum tested for amebic antibodies using the indirect hemagglutination test. The treatment for ulcerative colitis can cause dissemination of ameba with disastrous consequences. In one series, two out of seventeen patients with apparently confirmed ulcerative colitis had amebic colitis as determined by serology and responded to anti-amebic

treatment. Although not common, depression may be a manifestation of amebiasis even without overt intestinal symptoms. A predilection of the ameba for the appendix can be a cause for misdiagnosis, especially in highly endemic areas.

2. (A) The only indication in parasitology for a purged stool is a search for ameba in a patient without diarrhea. There is no reason to purge a patient for any other parasitological study. We had a patient with amebiasis whose wife had no symptoms and a casual stool specimen was negative for ameba. We purged her and found amebic cysts in her stool. Most likely, she was a carrier who had transmitted the infection to her spouse. A continuing epidemic of amebiasis among Peace Corps volunteers was finally traced to an asymptomatic cook in the boarding house most often frequented by the volunteers. Purging her yielded diagnostic stools.

3. (A, C) Most authorities will try metronidazole first. Emetine is used in extremely acute amebiasis. It will help abate the dysentery, but does not eliminate the amebas. Chloroquine has no effect on intestinal amebiasis. Some authorities recommend giving chloroquine as a prophylaxis against hepatic amebiasis concomitantly when treating intestinal amebiasis. If metronidazole is used, chloroquine is not needed since metronidazole is usually effective against hepatic disease. Should metronidazole fail in acute amebiasis, diidohydroxyquinoline with tetracycline should be employed. An alternate drug available only from the Center for Disease Control is diloxanide furoate (Furamide).

4. (A, C) The asymptomatic cyst passer should be treated because he is a reservoir and serves as a means of transmission. It is the cyst stage that is infective. Metronidazole is so well absorbed that it is not usually effective for eliminating cysts in the asymptomatic individual. Paromomycin (Humatin) is an excellent amebicide and is useful in acute intestinal disease when given with diiodohydroxyquinoline, or alone as a luminal amebicide in a carrier. Although more expensive, paromomycin can be given in five days and has no side-effects. Diiodohydroxyquinoline (Diodoquin) occasionally mimics amebiasis, with bloating and colicky diarrhea.

COMMENTS

The onset of disease in amebiasis may be as early as ten days after exposure or may be delayed for several weeks or months depending on the intensity of initial exposure. As the amebas do indeed multiply in the intestine, a critical number may have to be reached before symptoms are induced. A happy balance

between amebas and normal intestinal flora can be upset by antimicrobial therapy, change in diet, physical or emotional stress, and other factors that are as yet unknown. Garlic is somewhat amebicidal, and an individual whose diet is usually high in garlic may develop overt amebiasis when circumstances, such as travel to another country, result in a marked decrease in garlic intake. For example, I have seen a number of Colombians who had never had symptomatic amebiasis develop it when they came to the United States.

Intensity of exposure has been mentioned. Although amebiasis is cosmopolitan, certain areas seem to be richer reservoirs of Entamoeba histolytica. Notable among these is Leningrad (where amebas in man were first described by Losch in 1875), Mexico, South America, and South Africa. It is usually more prevalent in the tropics and subtropics but has been found in Alaska and Finland.

If stools are negative for ameba but Charcot-Leyden crystals are reported in a nonasthmatic individual, amebiasis should be strongly suspected. Although eosinophilia is not characteristic of amebiasis, or for that matter of any protozoal disease, Charcot-Leyden crystals, which are a coalescence of eosinophilic granules, are often seen in patients with intestinal amebiasis. Even though eosinophils are not seen in the exudate of amebic colitis, these crystals may be present. Charcot-Leyden crystals may be seen with hookworm or trichuriasis; however, when so seen, the eggs of these parasites are readily apparent.

Amebic granuloma, which may resemble carcinoma of the colon on x-ray, is an inflammatory lesion loaded with eosinophils. Several years ago a 72-year-old man with diarrhea of one year's duration was seen in our department. He also had had an intermittent fever, and a barium enema revealed a lesion consistent with carcinoma of the ascending colon. Due to his cardiac condition, surgery was not possible and medical management was considered. However, the patient was questioned more closely and it was revealed that he was one of the people who had had amebiasis in the Chicago epidemic in 1933. Because of this, a serological test for amebiasis was done and a titer of 1:32,000 was obtained. Metronidazole was given, and in 48 hours the diarrhea had stopped and the patient was afebrile. Barium enema done at a later date was negative.

Serology is useful in both the diagnosis of and in the determination of the extent of amebiasis. Asymptomatic cyst passers tend to have low or negative titers. Patients with symptomatic invasive intestinal amebiasis will have positive titers in 60% or so

of such individuals. Those with hepatic amebiasis will have a positive test in more than 95% of instances. Amebic granuloma can be differentiated from a carcinoma by the very high titer universally obtained.

Dientamoeba fragilis is the only other intestinal ameba that is pathogenic to humans. It causes a diarrheal syndrome. However, it is not invasive like E. histolytica and never involves the liver. It has two distinct nuclei and does not exhibit a cyst stage, and thus can be differentiated from other ameba, as well as E. histolytica. It responds to diiodohydroxyquinoline or tetracycline. Its distribution is worldwide and should be considered in the differential of diarrheal syndromes.

CASE 2: FEVER AND RUQ TENDERNESS

HISTORY

A 31-year-old importer presented with a chief complaint of fever of one week's duration. This man is in the importing business and periodically travels to Europe, India, and Pakistan. Three months prior to his admission he had returned from a trip to Pakistan. During his stay there, and for about two weeks after his return to the United States he experienced loose, watery stools without gross blood. He noted mild fever and a five-pound weight loss. The diarrhea and fever disappeared spontaneously. One week prior to admission he began experiencing fever and malaise; this persisted, and on the day of admission his temperature was 104°F. He denied pain, nausea, vomiting, or diarrhea.

EXAMINATION

Revealed a well-developed man who did not appear acutely ill. BP 110/70, P 120, R 20, T 103.6°F. Skin was warm and moist. There was no scleral icterus. Lungs were clear. Heart was normal except for tachycardia. Examination of the abdomen elicited tenderness in the right upper quadrant, and a tender liver edge was felt at the costal margin on deep inspiration. The spleen was not palpable. The remainder of the examination was normal.

LABORATORY

White blood cell count - 18,200, P 88%, L 10%, M 1%, E 1%.
Erythrocyte sedimentation rate - 68 mm/hr.
Urinalysis - normal.
SGOT - 40, bilirubin - 1.0, alkaline phosphatase - 160 I.U.
Stool for occult blood - negative.
Chest x-ray revealed the right diaphragm elevated.

CLINICAL COURSE

A liver scan was performed because of the tender liver with normal SGOT, and the history of travel and previous diarrhea (Fig. 2.1). Purged stool was positive for Entamoeba histolytica. An indirect hemagglutination test for ameba was positive with a titer of 1:1052. Treatment was initiated with metronidazole at a dosage of 750 mg by mouth three times a day. Within 24 hours he was afebrile, feeling well, and asking to be discharged from the

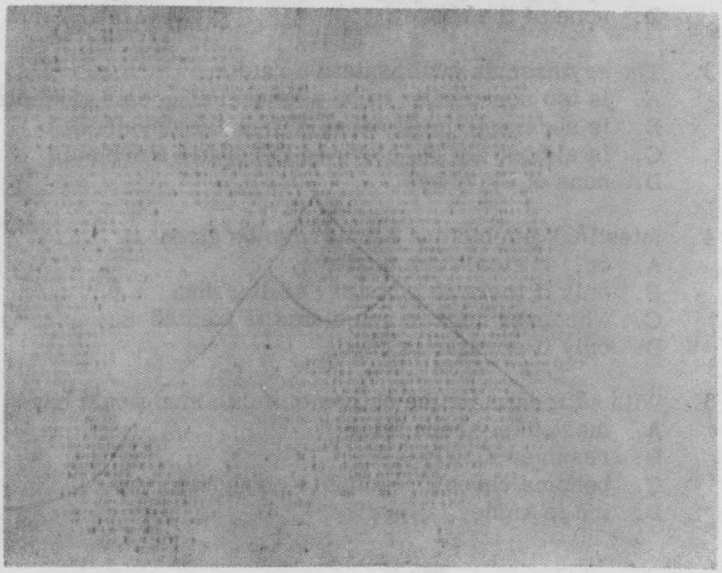

FIG. 2.1: The liver scan has a filling defect in the right lobe. The oval dotted line denotes the gallbladder.

hospital. He completed treatment at home. In addition, diidohydroxyquinoline and oxytetracycline were given for the intestinal infection, even though metronidazole might have been sufficient.

On six-month follow-up, he was asymptomatic. A liver scan revealed no abnormalities. Purged stool examination was negative for ameba.

QUESTIONS

1. Other parasitic diseases that could produce the abnormal liver scan seen in Fig. 2.1 are
   A. schistosomiasis
   B. echinococcosis
   C. cysticercosis
   D. trichinosis

2. Ameba gain access to the liver
   A. via the portal system
   B. via lymphatics

C. by migrating through the peritoneal cavity
D. none of the above

3. The erythrocyte sedimentation rate
A. is too nonspecific to be useful in diagnosis of amebiasis
B. is normal in most cases of hepatic amebiasis
C. is almost always elevated in hepatic amebiasis
D. none of the above

4. Intestinal amebicides should also be given
A. only if stools are positive
B. only if there is a history of diarrhea
C. whenever hepatic amebiasis is treated
D. only if emetine is used

5. Without treatment the outcome in this man would have
A. most likely been fatal
B. resolved slowly
C. become chronic leading to cirrhosis
D. led to kidney failure

ANSWERS

1. (B) The lesions of schistosomiasis are small and not visible as a large filling defect. Neither of the other two encyst in the liver, even though such lesions are small. Other possibilities include a pyogenic abscess, carcinoma (primary or metastatic), polycystic disease, congenital absence of lobe of liver, venous malformation, hemangioma, and estrogen-induced lesions (contraceptive pill). A defect in the liver scan is representative of a rather small differential, and amebiasis should always be considered, especially if fever is present.

2. (A) Although only two-thirds of patients with amebic abscess of the liver give a history of antecedent diarrhea (sometimes predating the abscess by years), the route to the liver is via the portal system from the large intestine.

3. (C) Virtually all amebic abscesses of the liver cause an elevation of the erythrocyte sedimentation rate. A normal sedimentation rate should argue strongly against an amebic or pyogenic abscess.

4. (C) If only the amebas in the liver are eliminated, the intestine continues to serve as a reservoir and reinfection of the liver is possible. Since metronidazole is so well absorbed, intraluminal amebas may not have been eliminated in this man;

therefore, luminal amebicides were used. The reverse is true also; that is, when treating intestinal amebiasis, many authorities recommend giving a hepatic amebicide as well.

5. (A) If this man had been treated for other conditions, or failed to seek medical attention, his disease could well have progressed to a fatal denouement. Many cases of hepatic amebiasis do not initially appear gravely ill. Fortunately, with the advent of liver scanning, the index of suspicion has been augmented and the need for diagnostic aspiration is no longer necessary. Furthermore, many cases initially relegated to a diagnosis of viral hepatitis are now properly diagnosed early. If hepatic amebiasis is definitely suspected, treatment should be initiated pending results of patient response, serology, and even liver scanning. If surgical drainage or exploration is considered, even presuming a pyogenic abscess, preoperative antiamebic treatment should be given to prevent spillage and amebic peritonitis, which is a highly fatal complication.

Today, metronidazole is used and can be given with virtually no side-effects. However, in the doses given for amebiasis, a furred tongue and discolored urine may occur; the latter represents a metabolic product and is of no consequence. It should be remembered that metronidazole behaves like Antabuse, and alcohol is interdicted during therapy. Although intestinal and hepatic manifestations are the most common expressions of amebiasis, other extra-intestinal sites may be involved: the lung by direct extension from the liver or via hematogenous spread, the brain, kidney, and heart (also hematogenously). Cerebral abscesses have been uniformly fatal. The skin may be involved by contiguous spread from the anal area or perforation of an adherent colonic ulcer. An amebic granuloma of the colon is a rare manifestation that can be mistaken for a carcinoma. Although a pyogenic abscess may resemble an amebic abscess, the former is usually multiple, whereas the latter is usually singular. Since this rule is not infallible, serologic testing is very helpful in differentiating. Rarely, a mixed amebic and pyogenic infection may occur.

ENTAMOEBA HISTOLYTICA

MORPHOLOGY

Trophozoites usually measure 15-30 microns, but range from 10-60 microns, and may have ingested red blood cells; cysts measure 5-20 microns with a maximum of 4 nuclei. It is important to distinguish these from other ameba and white blood cells

LIFE CYCLE

Cysts ingested by man reach the colon where reversion to trophozoites occurs and multiplication ensues. Cysts are usually found in stool, whereas trophozoites are found only during diarrheal phases. Trophozoites can be carried to the liver via portal system, causing abscess → spread contiguously or hematogenously to other areas (lung and brain)

EPIDEMIOLOGY

Worldwide. Up to 50% of population in areas of poor sanitation or tropics; in the United States 1-5%, certain areas high risk for travelers, such as Leningrad, Mexico, South America

DIAGNOSIS

a) Finding cysts and/or trophozoites in stool
b) Serology

SYMPTOMATOLOGY

1. Asymptomatic
2. Diarrhea: bloody with leukocytes; may mimic ulcerative colitis; can be watery and profuse.
3. Amebic abscess of liver: right upper quadrant pain; fever; cold area on liver scan; elevated sedimentation rate; minimal to moderate leukocytosis; positive indirect hemagglutination test for ameba in 85-95% of cases
4. Ameboma - may simulate carcinoma of colon; positive indirect hemagglutination test for ameba and therapeutic trial both useful

TREATMENT

1. Acute dysentery
   a) metronidazole (Flagyl)
   b) diiodohydroxyquinoline (Diodoquin) plus
   c) tetracycline

2. Subacute or asymptomatic intestinal disease; carrier state
   a) diiodohydroxyquinoline and tetracycline
   b) paromomycin (Humatin)
   c) metronidazole, less useful

3. Liver abscess
   a) metronidazole
   b) chloroquine