

3-1963

## Fever, Chills, Hepatomegaly And Debility

Boy Frame

Joseph Eschbach Jr.

W. S. Haubrich

J. E. Garman

Follow this and additional works at: <https://scholarlycommons.henryford.com/hfhmedjournal>



Part of the [Life Sciences Commons](#), [Medical Specialties Commons](#), and the [Public Health Commons](#)

---

### Recommended Citation

Frame, Boy; Eschbach, Joseph Jr.; Haubrich, W. S.; and Garman, J. E. (1963) "Fever, Chills, Hepatomegaly And Debility," *Henry Ford Hospital Medical Bulletin* : Vol. 11 : No. 1 , 65-69.

Available at: <https://scholarlycommons.henryford.com/hfhmedjournal/vol11/iss1/6>

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons. For more information, please contact [acabrer4@hfhs.org](mailto:acabrer4@hfhs.org).

## FEVER, CHILLS, HEPATOMEGALY AND DEBILITY

Case Report from Medical Grand Rounds

BOY FRAME, M.D., FIFTH MEDICAL DIVISION  
JOSEPH ESCHBACH, JR., M.D., MEDICAL RESIDENT  
W. S. HAUBRICH, M.D., DIVISION OF GASTROENTEROLOGY  
J. E. GARMAN, M.D., DEPARTMENT OF RADIOLOGY

*Dr. Boy Frame:*\*\* Our case this morning represents a problem in the diagnosis of "pyrexia perplexa".

*Dr. Joseph Eschbach, Jr.:*\*\* This 47 year old man had been previously healthy except for several seemingly self-limited, "flu"-like illness marked by diarrhea over the last 6 months. Six weeks before admission to this hospital, he experienced the abrupt onset of fever, diaphoresis, and shaking chills. He was twice admitted to another hospital and given antibiotics with only temporary relief. At the second admission, he was icteric and had an enlarged liver. A diagnosis of hepatitis was made. The jaundice cleared in one week. However, he continued to have shaking chills, fever, and sweats. Diarrhea recurred. Meanwhile, the patient lost 25 pounds.

Upon his transfer here, the patient appeared cachectic. His blood pressure was 80/40, and his pulse was 120. A smooth and only slightly tender liver extended 15 cm. below the right costal margin. Because of bulging flanks and a protuberant abdomen, ascites was thought to be present. The hemoglobin was 8.3 Gm. The white blood cell count was 16,000 and later rose to 30,000. The serum bilirubin was 0.64 mg., and the alkaline phosphatase was 6.3 Bodansky units. The serum transaminase was initially 97 units and later dropped to 18 units.

Soon after his admission, the patient developed shaking chills and fever (104°) followed by signs of shock. The blood pressure was supported by Aramine and blood transfusions.

Suspecting an intra-abdominal infectious process, laparoscopy was performed. The peritoneum was clear, and there was no ascites. The liver appeared enlarged. Visible on the smooth, dark red liver surface were fairly well circumscribed white and yellow areas in both the right and left lobes. These were interpreted as areas of focal necrosis. In a liver biopsy the parenchymal cells were pleomorphic and attended by a sparse, inflammatory infiltrate suggesting a non-specific hepatitis.

W. S. Haubrich, M.D. kindly serves as Chairman of the Grand Rounds Committee.

## MEDICAL GRAND ROUNDS

Because it was felt that the patient was proceeding to a subacute liver necrosis, antibiotics were stopped and prednisone (20 mg. q.i.d.) was initiated. During the next week there were 3 distinct episodes of shaking chills, the last 2 with shock. A Gram-negative septicemia was suspected. Numerous blood cultures were negative. Streptomycin and tetracycline were given. Finally, a single culture obtained during the patient's chills yielded *Streptococcus viridans*, and large doses of penicillin were begun. The patient continued to have mild diarrhea. Proctoscopy was negative, and no parasites were seen in several fresh stool specimens.

At this point, 4 features stood out: 1) a persistent leukocytosis, 2) persistent fever despite massive antibiotic therapy, 3) an enlarging liver, and 4) the patient's steady deterioration.

A radioactive rose-bengal scintogram (to be shown later) and further bulging in the right flank suggested focal suppuration in the right liver lobe. It was here that the surgeons, with local anesthesia, encountered a large hepatic abscess which yielded 4 liters of thick, brownish-pink fluid. In fresh material from this drainage, sluggishly motile trophozoites were seen and identified as *Entameba histolytica*. Chloroquin therapy was immediately started, and the patient's temperature returned to normal. He has had no more diarrhea and now appears to be recovering nicely.

(The patient is introduced.)

*Dr. Eschbach:* Good morning. How are you today?

*Patient:* I'm very good this morning.

*Dr. Eschbach:* Have you travelled outside of Michigan in recent years?

*Patient:* No.

*Dr. Eschbach:* What was the most difficult part of your illness?

*Patient:* It was the chills and fever that put me in a bad way.

*Dr. Eschbach:* The drainage here, from a small polyethylene tube inserted into the right liver lobe by Dr. James Barron, is yielding less every day. Are there any questions?

*From the audience:* Did the patient have a good deal of pain?

*Patient:* No, I haven't, doctor. Once in a while, before the abscess was drained, I had a little catch in my side.

(The patient is dismissed.)

*Dr. Frame:* As you see, we had a very sick patient on our hands. Initially, because of the fever and the large liver, the possibilities of hepatoma, lymphoma, or metastatic disease were entertained. However, the liver was not particularly hard or nodular, and I think that most of us did not feel that malignancy was likely. Rather, an infectious process within the abdomen, such as a sub-diaphragmatic abscess, seemed a better explanation. The intermittent diarrhea and the enlarged liver were thought to be

## HEPATOMEGALY

related. First, ulcerative colitis or regional enteritis with hepatitis or liver abscess was considered. Tuberculosis of the cecum and terminal small bowel with extension to the peritoneum and liver were also mentioned. The extreme leukocytosis was perhaps against these latter diagnoses, but leukemoid reactions do occur in hematogenous tuberculosis. Finally, I must say that we did consider amebiasis. Ten stools, most of these warm-stage specimens, were examined for amebae, all to no avail. Proctoscopic examination with bowel scrapings were also negative for amebae.

The I<sup>131</sup> rose-bengal scintogram suggested the possibility of a large abscess in the right lobe of the liver. This was aspirated and drained by Dr. James Barron. We were fortunate in observing amebae in this pus, since usually the amebae are not abundant in the pus itself but are more apt to be found in the wall of the abscess.

*Dr. W. S. Haubrich:* First of all, this case is unusual simply because of the diagnosis. In our indexed records of the past 9 years, there have been only 2 other cases of amebic abscess of the liver. There have been 5 cases coded as amebic hepatitis, mostly instances of amebic colitis in which there were suggestions of liver involvement. Today's case illustrates several unusual features. The patient had little pain. Dr. Frame explained that the patient is a rather stoic fellow. Nevertheless, the patient thereby deprives us of one of the prime features of liver abscess, amebic or suppurative, and that is pain in the region of the liver. The patient's weight loss, chills, and fever are characteristic enough. The leukocytosis is greater than usually seen with amebic abscess, but, of course, we have to bear in mind that something else had happened in this abscess. By the time we saw this patient, there was a secondary infection. The light, yellowish-gray pus that was obtained from this abscess was not typical of amebic abscess. Pus taken from an uncomplicated amebic abscess is almost pathognomonic. It is thick and dark, often likened to anchovy paste.

At laparoscopy, I misinterpreted the focal, yellow areas in this man's liver as areas of necrosis rather than abscess. I considered that the chances were better that this man had a virus hepatitis in a subacute, necrotic phase. On the basis of probability, I might have been justified. A biopsy taken from the liver revealed, in fact, evidence of hepatitis although the changes were non-specific. Dr. Garman will demonstrate the rose-bengal scintogram.

*Dr. J. E. Garman:*\* We have both a scout film of the abdomen and an I<sup>131</sup> rose-bengal scintogram (Figure 1). In the scout film, we see a large homogeneous density occupying the right upper abdomen. This would be indistinguishable from a diffusely enlarged liver were it not for the scintogram where we see that the liver tissue emitting radiation from the rose-bengal lies, for the most part, to the left of the density. The demarcation between the area of sparse or no radiation and the area of consistent radiation is definite although somewhat serrated. This indicates that the bulk of the right liver lobe has been displaced downward and to the left by a large, space-occupying lesion.

---

\*Department of Radiology.

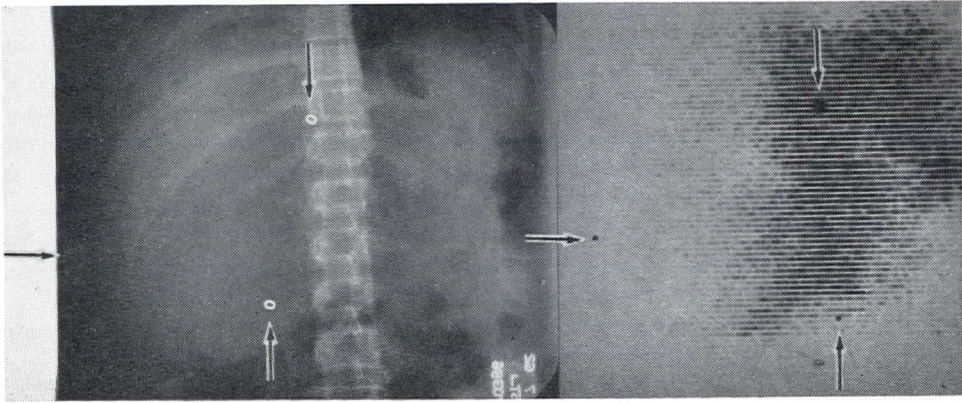


Figure 1

Scout film of the abdomen, *left*, and rose-bengal ( $I^{131}$ ) scintogram, *right*. The arrows point to the surface markers which correspond in each radiograph.

*Dr. Haubrich:* Now, a word about the pathogenesis of an amebic abscess. Amebae make their way from the intestine to the liver through the portal system. They can induce a diffuse inflammatory reaction in the liver where any number of them may gather together in small blood vessels. They produce infarcts and small areas of necrosis. Amebae possess potent cytolytic enzymes. What we call amebic "abscess" is actually an area of necrosis where dead and dying liver cells have been simply digested away by these cytolytic enzymes. As Dr. Frame mentioned, the amebae are often absent in the liquefied center of the necrosis but rather proliferate in the zone around it. Amebae are recovered from the aspirated fluid in only about one-third of cases.

*Dr. Frame:* Dr. M. J. Murray of Saginaw, who referred this patient, did a very thorough work-up and suggested the possibility of amebic abscess before the patient was transferred. In retrospect, Dr. Haubrich, do you think we might have handled this patient differently to make an earlier diagnosis?

*Dr. Haubrich:* Of course, with the 20/20 vision of hindsight, I can think of ways we might have proceeded a little differently. Nevertheless, this case evolved logically. It justifies a sound surgical axiom: pus somewhere, pus nowhere else, pus *there*.

*Dr. Frame:* Would the utilization of a complement fixation test for amebae given us an earlier clue? What is your opinion of the value of this test?

*Dr. Haubrich:* There have been several attempts to formulate a serologic test for this disease. Craig, in New Orleans, devised what, in his hands, seemed to be a potent and effective antigen. Unfortunately, other laboratories have been unable to concoct a similarly reliable substance. Moan, in Pennsylvania, claimed good results from a complement fixation test, but few have borne this out. It is my understanding that the Public Health Service laboratory in Chamblee, Georgia, is no longer accepting specimens for this test.

## HEPATOMEGALY

How do you explain this man's severe anemia?

*Dr. Frame:* This is not unusual with severe amebiasis although the exact mechanism is unknown. The extreme leukocytosis was at first disturbing, but white blood cell counts of 20,000 and 30,000 are not unusual in patients with amebic abscess of the liver, even without secondary infection.

*From the audience:* Is emetine considered too toxic in the treatment of amebiasis?

*Dr. Haubrich:* Yes, emetine has been almost abandoned. Chloroquin is the drug of choice. One must, of course, treat patients such as ours with a systemic anti-ameba agent and not merely with an agent which is going to work only within the lumen of the bowel. Should the patient not respond to chloroquin, however, this still does not vitiate the diagnosis. In such a case, one would be justified in trying a course of emetine.

(Editor's Note: The patient was given a full course of chloroquin therapy (20 grams over a 2 week period). He was given diodoquin for 3 weeks. The patient is now being observed for the results of this treatment. Presently, after 9 months, he continues to be free of symptoms, and he has regained his weight.)

