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## MEDICINE

(INTERNAL DISEASES, PEDIATRICS, NEUROLOGY, DIAGNOSTIC METHODS, ETC.)

### ABSCESS OF THE LIVER\*

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I wish to report 116 cases of abscess of the liver, in which the diagnosis was confirmed by either aspiration, operation, or post-mortem findings. Of the 116 cases, 91 are from the Charity Hospital and 25 from Touro Infirmary, both in New Orleans, and occurred during the years 1910-1914, inclusive.

Forty-seven (47) of these cases gave a definite history of dysentery, some dating back many years, while others had just recovered from acute attacks. Twenty-eight (28) gave definite histories of *chills and fever*. All but a very small percentage gave most definite histories of pain in the region of the liver as the most prominent symptom. Of these 116 cases, 73 or 68.9% recovered, 40 or 34.5% died, and 3 deserted. Of those dying, six were practically moribund on admission. Six (6) cases were not operated upon; of these, one died on operating table, one died with diagnosis of pneumonia; two died with diagnosis of tuberculosis; two ruptured into lung and were cured by the use of emetin without further operative interference.

In 49 of these cases, the average leucocyte count was 18,000; the average neutrophile count in 59 cases was 79%. In 16 cases showing a positive infection with the amoeba histolytica, the average neutrophile count was 77.2%.

Amoebic infection was found positively in only 25 cases. A definite history of trauma was given in only four cases. In 10 cases, the left lobe, only, was involved. Seven (7) of the 116 cases ruptured into lung. Of the seven positive cases of multiple abscess, all died. No attempt has been made to separate the single from the multiple abscess cases. In those recovering after the drainage of a single abscess, it is presumed that no other focus of infection was present; in those dying after operation, only a post-mortem could decide the number of foci present. The average length of time the case was acutely sick before entrance was 11½ weeks in the Charity Hospital, and 7½ weeks in the Touro series.

I wish to discuss briefly several points arising in the analysis of these cases, and the very first is the question of etiology. I commenced this investigation under the firm belief that the amoeba histolytica was responsible for at least 80% of all the cases of hepatic abscess, occurring in this sub-tropical climate, and yet, from the above records, it would seem that it is the primal cause in only 21.5%.

\*The Jerome Cochran Lecture, delivered before the Medical Association of the State of Alabama, Birmingham, April 21, 1915.

A further analysis shows, however, that the causative factor was not looked for in over 30% of the cases, and even in those where inquiry was made, the surgeon was often satisfied to send only the aspirated pus to the pathologist for examination. This method is not satisfactory, for it is well known that the pus from an amoebic abscess is sterile, and rarely ever shows the amoeba, which must be obtained by scraping the walls of the abscess cavity rather roughly, not merely swabbing out the cavity with a sponge. Moreover, the specimen must be kept warm and examined at once. It is imperative that this investigation be made at the time of operation, for often in 48 hours after operation in a positive amoebic case, scrapings prove negative, due to the fact that the serum which has poured out seems to dissolve and liquify this most delicate organism.

It is well known, and often proven in my wards, that the amoeba histolytica is most difficult to find in the bowel even after the use of a rectal tube, and we now use the proctoscope in all suspicious cases, scraping the ulcer with a curette in order to obtain satisfactory specimens.

Dr. S. K. Simon has recently reported (personal communication) a case of abscess of the liver coming to operation, in which no amoeba or other organism was found at the time of operation, and yet one month afterwards, he was able to demonstrate amoeba in the stools. In the work of Wherry, Rogers, Vedder and others in 1912-1913, we have a therapeutic test of the greatest value in emetin hydrochloride. Given a case of diarrhoea of unknown origin; if this clears up in three or four days under the use of emetin, we can be fairly positive that it was of amoebic origin; in the same way, given an abscess of the liver of unknown etiology; if this does not improve after drainage, but does after emetin, we can state almost positively that here, too, the same organism has caused the original insult.

A case occurred two months ago in the charity ward of Touro Infirmary, under the

care of Dr. Weis, confirming this. The patient was emaciated to the last degree, septic, exhausted with constant coughing, spitting up enormous amounts of foul pus, which was at once recognized as coming from the liver; no amoeba were found, but the case cleared up promptly, one might almost say magically, under the use of emetin in large doses.

Leonard Rogers, Chauffard and others have reported similar cases.

I wish here to report a case of liver abscess which occurred in my practice, and was operated upon by Dr. Matas, showing the efficacy of emetin, but cited here as confirming the etiologic diagnosis.

Mr. A., white, male, age 46. Family history good. Past history: Periodic fever from boyhood to twenty-first year, lasting each year from 4 to 10 weeks; typho-malaria (?) fever 20 years ago. Does not use alcohol. Smokes incessantly; venereal history negative; indigestion for years.

Entered Touro Infirmary December 28, 1912. Had a chill ten days previously, which was followed by fever; these chills have recurred daily with pain in the region of the liver; no history of recent jaundice, but was jaundiced 8 months ago; normal weight 176 pounds; now 160 pounds. Examination showed: Heart normal; lungs normal; on palpation the abdomen was soft, except tenderness on pressure in right upper quadrant; a sense of resistance increased on deep pressure and some tenderness under ninth costal cartilage; liver dullness in mammary line reached two inches below costal arch; liver not enlarged upward either anteriorly or posteriorly; blood examination gave 18,800 leucocytes; neutrophils, 76 per cent. At operation an oblique incision revealed a large liver, gall bladder much distended, no calculi can be felt in it; on the inferior surface of the liver, an abscess of the spigelian lobe was found; this was evacuated and drained. The temperature at once fell to 99° and remained so for twelve days. Then followed three weeks of fever ranging from 99° to 102°. On February 3d

the liver was aspirated, but no further pus pocket found. On February 18th the sub-phrenic space was drained by a through and through drainage tube, and the temperature fell for a few days. On March 16, we aspirated the liver again through the eighth intercostal space, but no pus was found, the fever continuing to run a septic course. On March 27, Dr. Matas commenced the use of emetin by needle; in four days the temperature dropped to 99° and has remained so up to the present time.

There is another point which I think has a bearing on the etiology, namely: the fact that in so many cases where the pleura or peritoneum is opened by accident, no trouble ensues. This means that the pus must be sterile, and this sterility of the pus is an argument in favor of its being of amoebic origin. In this whole series, I have only been able to find a very small percentage of cases where the etiology was proven positively not amoebic; the appendix, gall bladder or some operation involving the rectum with subsequent infection, being the casual agent.

Another point in favor of the amoebic origin is that 73 of the 116 cases recovered after operation, in which only a single abscess was drained. Kelesch reports 500 cases of liver abscess, 85% following dysenteric attacks. Leonard Rogers "found living amoebae in 35 out of 37 consecutive cases, the two negative ones not being examined until 12 or more days after opening, when infection may have died out."

If liver abscess is due to causes other than amoeba, it should occur more often in colder climates. That it does not occur often after epidemic bacillary dysentery is a well-known fact.

You will remember that the average length of time that the patient was acutely sick before entrance to the hospital was 11½ weeks in the Charity Hospital, and 7½ weeks in the Touro series. This must make us realize the great difficulty in diagnosis, and the urgent need of further investigation into the clinical

signs and symptoms of the disease. Manson says, "Golden rules in tropical practice are, to think of hepatic abscess in all cases of progressive deterioration of health, and to suspect liver abscess in all obscure abdominal cases, associated with evening rise of temperature, and this, particularly if there be enlargement of, or pain in the liver, increased leucocyte count, and a history of dysentery."

James Cantlie says that "a man may have a pint of pus in his liver and yet there may be no pain, no increase in temperature, no cough, no great loss of appetite." In an excellent monograph, Perves and Oudard give the following rule: "Suspect an hepatic abscess if there be (1) an enlargement of liver with a sense of weight in liver region; (2) a point in that region which is distinctly painful to pressure; (3) the previous history of dysentery." Carter goes even further, declaring that in "all cases of chronic pyrexia of uncertain origin with high leucocyte count, look out for amoebic abscess."

The clinical picture of this condition is familiar to everyone; the pinched faces, hectic flush, short catching coughs, septic sweats, frequent rigors, increasing complaint of pain in right hypochondrium, and accompanying these symptoms, increased leucocytes, enlarged liver and rapid pulse.

Unfortunately at times, many or even all of these signs fail us. I recall a case that walked into my office; had just returned from a month's sojourn on the sea coast, where he had gone for the surf bathing, which he had been told was good for one suffering from chronic malaria. He came to consult me on account of a bulging in his liver region, and declared he had no pain or fever and was not sick except for this swelling. A leucocyte count gave only 9,700 white blood cells, and temperature only 100°, and yet one pint of pus was drained from his liver the next morning.

Judging from this series, the three most constant diagnostic signs are—pain in region of liver; loss of weight and increased leucocyte count, but the first two are slow to assert



Distance from tenth intercostal space mid axillary line to left border of liver, 5 inches.

If the needle is introduced through the sixth and seventh inter-space, there is danger of wounding the lung.

These figures hold good only for a liver not diseased, and in the dead body. We must remember, however, that in hepatic abscess, the liver is much enlarged, not only by the presence of the abscess, but by the accompanying hyperaemia and hyperplasia of the remaining portion, and therefore measurements on the normal subject cannot hold. Sambuc comments on the fact that we, as a rule, aspirate too low, and declares that most abscesses are

syphilis of the liver and hepatic abscess. They both give continued fever, pain and enlargement of liver, and I wish to cite a case in point:

D. L., age 47; white, male, Italian. Entered hospital on July 7, 1914, on account of pain in liver region. Gave following history: Usual diseases of childhood; yellow fever in 1904; denies all venereal history. In November, 1913, had attack of diarrhoea; numerous actions on bowels daily, mucus and blood, and cramps with the movement. Two months ago had fever which lasted three weeks; for the next two weeks he was up, then he had



Abscess of liver not diagnosed until postmortem, although aspirated several times.

at first high up in the dome of the liver, and close to the diaphragm.

When we come to differential diagnosis, the most common mistake is that of confusing hepatic abscess with aestival-autumnal malaria and its accompanying enlargement of the liver. Osler and Manson both state that they have rarely seen cases that had not been drenched with quinine. This mistake is inexcusable, even when no plasmodia are present, for malaria gives a leucopenia, generally presents an enlarged spleen, and always answers to the therapeutic test if applied correctly.

A more difficult diagnosis is that between

a chill, with temperature one night and again next day, and followed by pain over gall bladder region. This pain has occurred at different times and often severe; never jaundiced either during or following the attack. Examination showed well-nourished man; lungs clear; heart: first sound at apex is somewhat prolonged but no murmur. Liver: tenderness over its lower border in region of gall bladder; rectus muscle little rigid and slight mass felt in gall bladder region. White blood corpuscles (7-21-14) were 13,500; neutrophiles, 67%; Widal, negative.

Fever ranges from 101° to 99° until July 24, when he was discharged, the diagnosis

being given as cholecystitis, and phosphate of soda ordered.

He returned to the hospital on September 7, 1914; says he still has pain in region of gall bladder, severe at times.

Examination shows as formerly, except liver a little larger; no tenderness or rigidity except over right upper abdomen in region of gall bladder; spleen not enlarged. Leucocyte count shows 18,500; neutrophiles, 72%; few hyaline and granular casts in urine. Temperature 99° to 101°. On September 14, this case was diagnosed as abscess of the liver and transferred at once to surgical ward. September 15, liver was aspirated in several places; no pus was found; the fluid from aspirating needle contained leucocytes, but no amoeba. Next morning rectal tube was passed, but no amoeba found in the stools. The surgeon in charge was so positive of the diagnosis that he performed a laparotomy, exposing the liver thoroughly. It was found enlarged, smooth, firm, yellowish in color, with rounded edges; under the guidance of the hand, the liver was aspirated in several directions, but still no pus was found. The blood examination on the following day showed W. B. C., 14,900; neutrophiles, 80%. On the following day, patient had a hard chill; temperature, 105°. This promptly fell to 99° in the next two days. Temperature now varied from 100° to 102° until September 30, when a Wassermann was made and found positive. He was at once given very active mercurial treatment and commenced to improve. In less than one week, the temperature was normal; the pain in region of gall bladder had subsided; the liver was slightly smaller and the patient was allowed to go home on October 16th, still taking mercury by needle and potash by mouth. He returned to the hospital on December 21, complaining of having had pain in the region of the gall bladder for the past three weeks. He had stopped entirely the use of mercury for the past month. An examination showed the heart very much displaced to the left, with a marked bulging of the whole

right chest. Intercostal spaces obliterated; flatness on percussion and all signs of fluid in pleural cavity. The next day, a large amount of clear, straw-colored fluid was removed and he at once felt much better, and was placed upon mercurial treatment. On January 6, on account of bulging and tenderness and an increase in the fever, he was aspirated between the 7th and 8th rib and chocolate colored fluid obtained. This was recognized as probably liver pus, and he was transferred to surgical ward, where an operation was immediately performed and an abscess found in the dome of the right lobe of the liver. This was at once drained and the patient ordered emetin hydrochloride, half grain daily, by needle. He improved rapidly for the next three weeks, then developed a higher range of fever, which was thought to be due to a deficient drainage. This was remedied at once, and temperature again fell to normal. After one week, he developed another very high temperature, with constant sweats, and died rather suddenly, it was thought, from rupture of the abscess into abdominal cavity. This, however, could not be verified, as no post-mortem was allowed.

In the Journal of the Royal Army Medical Corps for January, 1913, a case was reported where every classical sign of hepatic abscess was present, and yet was cured by the vigorous use of mercury. The following case was one of interest to me, because I have always held that tubercular diarrhoea is a rare condition, unless there be excessive disease of the lung at the same time.

J. F., age, 39; white, male. Family history negative. Past history: Typhoid fever twenty years ago; pneumonia five years ago; at that time he was confined to bed for four months. In June, 1913, had a chill, followed by high fever, and diagnosis of pneumonia was made. The fever ranged from 99° to 102°. On July 3, his left lung was aspirated, because of dullness at the base of that lung; no fluid was found, but the X-ray showed possible tuberculosis at apex of both lungs. He commenced now to have pain in the region of liver, with

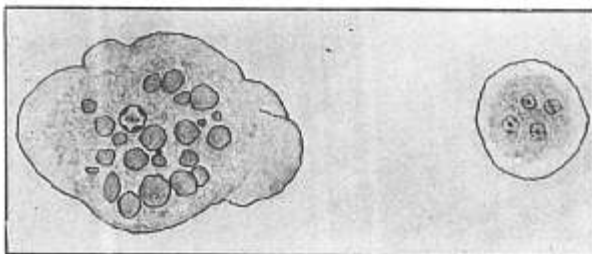
temperature daily  $100^{\circ}$  to  $101^{\circ}$  and frequent movements of the bowels, and quite a good deal of rigidity over the whole abdomen, and the case was supposed to be one of tubercular peritonitis. Blood examination was negative for Widal; showed only a slight increase in leucocytes, with a neutrophile count of 67%. The feces were negative for any parasites.

On October 15, the patient now having been in bed for three months, with loss of flesh, constant fever, poor appetite and septic temperature, a slight bulging in the region of the liver posteriorly was noticed for the first time, and a needle introduced. Chocolate colored pus was found and evacuated, and scrapings from the walls of the abscess showed amoeba.

emetin could kill the invading organism, but am loath to believe that drugs could have any effect on an abscess already formed. Carter and Cantlie are both of this opinion.

I can recall a few cases which gave histories of continued fever of rather a septic type, increased leucocyte count, enlarged liver, pain in liver and indefinite intestinal attacks in the past year; these would clear up entirely after the use of the aspirating needle, though no pus was found or ipecac given, the phlebotomy seeming to be the curative agent.

I shall not attempt to discuss the surgical aspects of hepatic abscess except to advocate an exploration in those cases where we have reason to suspect the presence of pus, and free



Vegetable stage.  
Entamoebae histolytica.

Encysted stage.  
Mann's stain.

which were also found later in the stools after a very thorough examination. The patient was discharged absolutely cured, after a very long convalescence, and the daily use of emetin.

I saw this case only one week prior to the operation, but must acknowledge that with the X-ray findings, the temperature range, and the diarrhoea, tuberculosis was the most obvious diagnosis.

In 1908, Leonard Rogers reported cases of what he termed "presuppurative stage of amoebic hepatitis" cured by the use of ipecac without any operative interference. I can readily believe that if a case could be caught at the incipency of an acute amoebic hepatitis before the formation of pus, that ipecac or

incision at the earliest possible moment after a diagnosis has been made. Temporizing too often means death.

Since the advent of emetin, many physicians in India have given up drainage and use only aspiration plus this drug, and claim better results than under operation alone. Thurston reports 67 cases of hepatic abscess. In 48 of these, aspiration plus emetin was used; 37 were cured and 11 died, or a mortality of 23%. In 7, aspiration plus drainage plus emetin was tried; 4 of these recovered and 3 died; a mortality of 43%. In 12 cases, free drainage plus emetin was used, there were 8 recoveries and 4 deaths, a mortality of 33%. Nott and Sandese both advocate aspiration and emetin rather than drainage.

Personally, I have had no experience with this method of handling such cases, but would hesitate in cases where the needle showed the abscess cavity to be large, to depend upon this method alone. Should the patient, however, be in extremis I would advocate its use as a temporary measure. This plan has been followed in India with some success.

The post-operative handling of hepatic abscess, amoebic in origin, should always include the use of emetin by needle for at least two months, and then I advocate strongly that ipecac be given intermittently for a year afterwards, as we have recurrences both of the bowel condition and further abscess formation.

#### CONCLUSION.

(1) While the amoeba histolytica was found in only 21.5% of this series, I believe that more correct methods of investigation as outlined above, would show it to be the primal cause in at least 80% of the cases.

(2) The most constant symptoms are pain in region of the liver, loss of weight and increased leucocyte count.

(3) The aspirating needle used for exploration should be at least 5 inches in length.

(4) Exploratory laparotomy is justifiable in doubtful cases.

(5) Emetin should be used in all cases of hepatic abscess as a regular post-operative measure.

(6) All recurring diarrhoea in this Southern country should be studied carefully, as I am of the firm belief that many of these cases are amoebic in origin, and it is only by this method that we can reduce the incidence of hepatic abscess.

#### ABSCESS OF LIVER—116 CASES.

Years 1910-1914 Inclusive.

From—		
Charity Hospital of New Orleans.....	91	
Touro Infirmary of New Orleans.....	25	
Number operated upon .....	107	92.2%
Number recovered .....	73	62.9%
Number died .....	40	34.5%
Number moribund on admission..	6	5.1+%

Number not operated upon.....	9	7.7+%
Number rupturing into lung.....	7	6.+
Number giving history of dysentery .....	40	34.9%
Number giving history of chills and fever .....	28	24.1+%
Number giving history of trauma .....	4	3.+
Number in which amoeba were found .....	25	21.5%
Number in which left lobe, only, involved .....	10	8.6+%
Average leucocyte count in 49 cases.....	18,000	
Average neutrophile count in 59 cases....	79.+	%
Average neutrophile count in 16 positive amoebic cases .....	77.2%	
Length of time acutely sick before admission, Charity series .....	11½ wks.	
Length of time acutely sick before admission, Touro series .....	7½ wks.	

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