AMOEBOMA OR CARCINOMA OF THE HEPATIC COLON.

BY
Lieutenant-Colonel C. M. MARSDÉN,
Royal Army Medical Corps.

Officer in charge of the Surgical Divisions,
Q.A. Military Hospital, Millbank and Horley.

[Received January 1, 1947.]

It is now generally recognized that Entamoeba histolytica can produce a proliferative lesion—an ameboma—which can be very difficult to distinguish from a carcinoma. Much less commonly carcinoma can supervene in a large intestine already affected by the lesions of amebiasis.

That this is a rare complication is shown by the fact that only one reference to this association has been found in the English medical journals (Morgan, 1944).

The following case is reported to show the difficulty of making a clinical diagnosis of carcinoma from ameboma when the past history of amebiasis is strong, even though that infection occurred forty years previously. Further we wish to open the possibility of carcinoma of the colon supervening on old and possibly healed amebic lesions.

A. M. B., aged 65 years.

The patient was always healthy in his youth and never came under medical care until, on entering the Army at the age of 17, he participated in the South African campaign, being wounded in the chest and left leg.

When 22 he proceeded to India, and shortly after arrival began to suffer from chronic ill-health which was only diagnosed after many months as due to amebiasis. By then he was having severe pain in the right chest and he was evacuated home, aged 23, with a large liver abscess. On arrival in U.K. the abscess was drained by open operation and 57 oz. of pus obtained. During the next three years three further liver abscesses were drained. He was then invalided from the Army.

When aged 31 the duodenal ulcer from which he had suffered for some time perforated and was sutured. Three years later he suffered another perforation and on this occasion a gastro-enterostomy was performed as well as closure of the perforation.

After this the patient had several laparotomies for the division of adhesions producing acute intestinal obstruction.

At the age of 50 the patient developed a further attack of acute obstruction, this time due to a strangulated inguinal hernia, and following the operation he had several further attacks of obstruction, some treated by division of bands and some by gastric suction.

The patient remained in fair general health, undertaking a busy administrative post, and yachting at the week-ends, until January, 1945, when he was 64 years old. He was then forced to stop work by a severe dyspnea on slight exertion, which had been growing worse for some six months. This was finally diagnosed as due to pernicious anæmia and the symptoms were greatly relieved by injections of liver extract.

By July, 1945, he was sufficiently well to proceed to Hamburg in the Control Commission Germany. Soon after his arrival he noted that, whereas he had for many years passed two stools each morning, he was now passing three or four looser stools, and suffering from considerable flatulence. Mucus was frequently present in the stools.

These symptoms continued and early in 1946 he noticed loss of weight and a
feeling of great fatigue during the week before he received his monthly injection of liver extract. In April, 1946, he noted a lump in the right upper abdomen. By June the lump had become more prominent and was painful and tender and he was admitted to a military hospital in Germany.

He was then pale and obviously wasted, with a visible and palpable tumour in the right hypochondrium. The blood-count showed Hb. 70 per cent and R.B.C. 4,170,000.

![Fig. 1.—Barium enema to show the complex of the small and large bowel with barium entering into an abscess cavity in the liver substance. This we took to be the fixed filling defect.](image)

The white count was 28,000, with a neutrophil leucocytosis of 25,000. The stools were normal.

Four pints of blood were given with marked symptomatic relief. A barium meal was reported as normal, but the barium enema showed clearly a filling defect of the hepatic flexure which was absolutely constant in shape throughout all films (see fig. 1).

A course of 10 gr. of emetine and one million units of penicillin was given; there was no demonstrable change in the characters of the lump.
The patient was then transferred to this hospital, being admitted on July 16, 1946. On admission, the patient was pale and thin, looking his years but mentally alert and in good spirit. He was edentulous, with a clean moist tongue. The lungs were clinically normal, with a good air entry at the bases and the heart was normal in size with no added sounds; the B.P. was 110/80.

The abdominal wall was heavily scarred and very thin, allowing observation of peristalsis over most of the abdomen. There was no distension and no evidence of free fluid. There was an obvious lump lying on the right side of the abdomen, approximately two inches lateral to the umbilicus. It moved with respiration. Palpation revealed a tender liver to be a hand's breadth below the costal margin; its surface was smooth. The visible lump was attached to the lower margin of the liver. It was not movable independently of the liver. The lump was some three inches in diameter, very hard and moderately tender. No other viscera or lumps were felt in the abdomen. There was no enlargement of any lymph glands and rectal examination was normal.

The blood count on admission showed R.B.C. 3,000,000; Hb. 75 per cent = 12.0 grm. per cent, and W.B.C. 9,800 with 8,000 neutrophils. The E.S.R. = 45 mm. in first hour (Westergren). Examination of the stools revealed no abnormality and a sigmoidoscopy with microscopy of the mucus obtained was also negative.

A chest X-ray showed normal lung fields, with a scarred, peaked, raised and immobile right diaphragm (see fig. 2).

The impression at this time was that clinically the mass was malignant and attached to a very large liver. But there was a strong past history of amebic hepatitis with abscess formation. The liver was very enlarged and its surface smooth.

The diaphragm was immobile, raised and deformed. There was an evening temperature of 99 to 100° with a pulse-rate of 90. He showed at his previous hospital a
leucocytosis with a high percentage of polymorphs. He was haemolysing his red cells. How much of this mass and of this large liver was due to amoebic infection?

We tried a course of emetine again and there was not the slightest change in his physical signs or his temperature chart.

Therefore, on August 9, we proceeded to exploratory laparotomy with a pre-operative diagnosis of carcinoma of the hepatic flexure supervening on chronic inflammation secondary to amoebiasis and with abscess formation.

A slow transfusion of four pints of group B-III. blood was given during the forty-eight hours prior to operation. A Ryle's tube was passed into the stomach immediately prior to operation and suction maintained throughout.

Anaesthetic.—Nembutal 3 gr. was given orally two hours, and atropine 1/100 gr.

Fig. 3.—Photograph of the fixed specimen from the R.A.M. College Museum. Note the glass rod extending through the fistulous opening of the transverse colon into the liver abscess cavity.

H.I. half an hour, before operation. Ephedrine 1½ gr. i.m. immediately prior to the spinal anaesthetic. This consisted of light nupercaine, 1:1,500, 12 c.c. injected in the L3-4 space by Howard Jones technique. Cyclopropane and oxygen were administered by closed circuit throughout the operation, the maximum depth of anaesthesia being the first plane of stage three. One pint of i.v. dextrose saline was given, followed by three pints of whole blood, during the operation. The condition of the patient throughout remained satisfactory, there being no significant change in the blood-pressure during the three and a half hours' operation.

Operation.—Directly over the mass a right upper transverse incision was made, with a vertical mid-line extension downwards. There was no free fluid. The liver was
enlarged a full hand's breadth below the costal margin, and there was adherent to its inferior aspect a large complex made up of terminal ileum, cæcum and ascending and transverse colon. The nature of this mass was slowly elucidated by the division of a vast number of adhesions and it was established that the whole hepatic flexure was adherent to the inferior aspect of the liver and that there was a carcinoma of the ascending colon, spreading directly to the adjacent and adherent transverse colon (see figs. 3 and 4). It was clear that a portion of the liver would have to be excised in order to free the colon.

As the patient's condition was good and no evidence of secondaries could be found in the liver, pelvis or lymph glands draining the area, excision was attempted. The whole complex of terminal ileum, cæcum and colon was mobilized and the adherent portion of liver cut away with diathermy. During this procedure, a sudden faecal leak occurred and it was found that a liver abscess cavity communicating with the malignancy in the hepatic flexure had been entered. The faecal matter was caught in packs and the excision of the specimen was completed. There was brisk haemorrhage from the cut surface of the liver, which was controlled with a pack. A side-to-side ileo-transverse colostomy was performed and the wound closed in layers, with the liver pack left in situ.

The operation specimen consisted of the terminal ileum, cæcum, ascending and transverse colon and a portion of the liver abscess. The specimen is in the museum of the surgical department of the Royal Army Medical College who have kindly supplied the photograph of the fixed specimen (fig. 3). This is difficult to interpret and a line drawing (fig. 4) with the superimposed liver is added. These show a carcinoma of the hepatic flexure extending into the transverse colon. It had necrosed on the contiguous surfaces of the hepatic flexure and the transverse colon to form a fistula between the two. It had extended directly into the liver (D in fig. 4) and that cavity was continuous with the fistula in the hepatic flexure and the transverse colon.
In fig. 3 a glass rod leads from the fistulous opening in the transverse colon into the liver abscess and thence into the carcinoma in the hepatic flexure.

In view of these findings we reviewed the X-rays. What we took to be the constant filling defect was in fact barium in the abscess cavity. This is still seen in the film taken after evacuation (fig. 5).

Sections of the growth in the colon showed it to be a tubulo-adenocarcinoma, which appeared to have penetrated all coats of the bowel. Sections of the wall of the liver abscess showed a metastatic mass of similar growth. This deposit appeared to have directly infiltrated the liver rather than to have spread via the lymphatics or blood-stream. The tumour was classified as Broder Group 2—H. Spencer, Major, R.A.M.C., Demonstrator in Pathology, R.A.M.College.

Post-operatively the i.v. blood, followed by dextrose-saline, was continued.
for seventy-two hours and gastric suction maintained for forty-eight hours. Four-hourly omnopon 1/3 gr. was given. The patient made a satisfactory recovery from the operation and on August 15, 1946, the liver pack was removed under pentothal, without incident. On August 20, 1946, a fecal fistula developed at the site of exit of the pack, but the general condition remained satisfactory.

The fecal fistula only leaked a small amount of feces, a good evacuation \textit{per rectum} being secured by a daily enema. On September 4, 1946, the patient sat out of bed and from then steady progress was made until, on September 17, he complained of feeling tired and distended. Throughout September 18 the B.P. fell steadily and the pulse grew weaker, the patient sleeping for long periods. He died on September 19, 1946.

The post-mortem showed an inoffensive purulent peritoneal exudate which was presumably due to infection from the liver abscess which opens direct to the general peritoneum; so dense were the adhesions around the site of operation that no satisfactory dissection could be made.

Sections taken from the remaining portion of the wall of the liver abscess showed the presence of carcinomatous tissue. There was no evidence of amebic infection of the liver.

**DISCUSSION.**

A case of carcinoma of the hepatic flexure extending into the liver to form an abscess cavity in that organ which abscess communicated directly with the lumen of the bowel is described. It also extended into transverse colon to form a fistula between the hepatic and transverse colon.

Forty years previously an amebic abscess of the liver had been drained. He presented now not only with a hard lump attached to the liver but with a tender liver enlarged one hand’s breadth, and with its surface smooth, associated with a high immobile deformed diaphragm, an evening pyrexia, a leucocytosis and looseness of the bowels with mucus in the stools. Amebae were not found nor were ulcers of the bowel wall seen with the sigmoidoscope.

Clinically the mass felt malignant. There was no clinical improvement first to emetine and penicillin and later to a further course of emetine. Therefore we made a pre-operative diagnosis of carcinoma of the colon. The persistent pyrexia and the large smooth tender liver prompted us to add that it probably supervened on chronic amebic infection with abscess formation.

We found a carcinoma of the colon but the liver abscess was by virtue of a direct extension of the malignancy into the liver. This accounted for the pyrexia and the large tender liver. The raised and deformed diaphragm was probably the result of the amebic infection forty years previously. The marked anaemia and the diarrhoea are the common features of carcinoma of the right bowel.

Is there any association of carcinoma and amebiasis? A review of the comparatively scanty literature of this subject (see references) shows that there is a period when the symptoms are thought to be due to old amebiasis. Such was the case with the present patient. But the clinical hardness of the mass
even in the presence of an unresolved pyrexia plus the failure of the thera-
peutic test with emetine clinched the diagnosis. It is, however, easy to visualize
a patient in whom there is greater evidence of an amœboma, and it is the pur-
pose of this paper to emphasize that carcinoma and amœbiasis can co-exist
or follow one or the other. For chronic amœbiasis is likely to have long-stand-
ing ulcus or granulomata or polypi which are all precancerous.

SUMMARY.
A carcinoma of the hepatic flexure causing some confusion with amœboma
is described.
Further examples occurring in ex-Service personnel are probable.
My thanks are due to Major Miss K. M. Watson, D.A., R.A.M.C., for an
excellent anaesthetic during a long operation on a very ill man of 65. She also
gave us the line drawing. This patient was under the excellent care of Captain
P. Jones, R.A.M.C., who produced most of these notes and the references.
This article is submitted by permission of Colonel A. R. Oram, O.B.E., M.C.,
Officer Commanding this hospital.

REFERENCES.
CADE and MILHAUD (1939). J. de Medecine de Lyon, 475.
SANTY and MORENAS (1936). Lyon Chirurgical, 452.

HYPOGLYCAEMIC RESPONSE TO GLUCOSE
STIMULATION IN A CASE OF ADDISON’S DISEASE.

BY
Captain A. ERDEI, M.D., M.R.C.P.,
Royal Army Medical Corps,

AND
Lieutenant-Colonel L. R. S. MacFARLANE, O.B.E., M.B., D.P.H.,
Royal Army Medical Corps.

A CASE of Addison's disease came under our observation which offers some
unusual clinical and biochemical features so that it is considered worth record-
ing.

CASE HISTORY.
Pte. J. S., aged 20, was seen by one of us (A. E.) on November 19, 1946, on admission.
He was transferred from a C.R.S. where he was under treatment for pharyngitis, and,
on getting up from his bed, suffered, what the medical officer suspected, a vaso-vagal
syncope. He became giddy, pale, collapsed, the pulse-rate went down to 50 to recover
only slowly. No blood-pressure reading was taken. He admitted that he had suffered
from similar attacks three or four times every year ever since he had cerebrospinal fever in
1941. He also complained of a growing fatigue which started insidiously, and became
noticeable during the last few months.