Clinical Surgery

TROPICAL SURGERY

CONSULTANT EDITOR

A. K. BASU

CLINICAL SURGERY—8

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CLINICAL SURGERY

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PREFACE

Practice of surgery in tropical countries necessitates knowledge of numerous interesting and special problems, many of which are not discussed adequately in standard surgical text books. The purpose of this volume is to present to the reader authoritative descriptions of the important surgical lesions that abound in these countries.

Many misconceptions exist about the common diseases which surgeons in the tropics have to deal with, such as amoebiasis, filariasis, leprosy, etc. Further, with the phenomenal increase in global travel and intercommunication, the so-called surgical lesions of the tropics are often found in more temperate climates. It is to be hoped, therefore, that the knowledge gained from the pages of this volume will be of wider interest.

The authors of the different chapters were chosen because of their particular and extensive experiences with the subjects concerned. Emphasis has been laid mainly on the clinical presentations of the diseases, their etiogenesis, pathology, methods of diagnosis and the principles of surgical management. Details of operative technique have on the whole been avoided, except in so far as they relate to the special aspects of tropical surgery.

Obviously it has not been possible to deal exhaustively with all the problems relating to tropical surgery. It is hoped, however, that the important surgical lesions have been covered fully and that the reader will find the different presentations valuable for purposes of reference.

A. K. BASU

June 1965

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CHAPTER 1

AMOEBIASIS

MILROY PAUL

DEFINITION

Amoebiasis is a disease resulting from invasion of body tissues by amoebae. Although invasion is in itself evidence of their pathogenicity (Councilman and Lafleur, 1891), amoebae also produce necrotic and inflammatory reactions wherever they invade the tissues.

EPIDEMIOLOGY

Evidence has been adduced for the view that large abscesses in the liver are acquired only by those who live or have lived in regions where amoebic dysentery is prevalent. Although amoebic dysentery is for the most part a disease of tropical and sub-tropical zones, it is also found in the temperate and cold belts in countries where sanitation is primitive. In this regard, it is of interest to recall that *Entamoeba histolytica* was first demonstrated at St. Petersburg, Russia, in the stools of a patient suffering from recurrent bouts of dysentery (Lösch, 1875).

Although myriads of *E. histolytica* are to be found in the stools of patients suffering from amoebic dysentery, the disease is conveyed from person to person not by these vegetative forms of *E. histolytica*, but by its cysts which are discharged in the stools. The vegetative forms of *E. histolytica* in the stools die out in a few hours at most and even if they were to be ingested they would be destroyed by the acid gastric juices.

The cysts are transmitted by the pollution of food or drink with faeces from carriers of *E. histolytica* and a polluted water supply is the commonest source of such infections. Another common source of infection is the ingestion of uncooked vegetables watered from a polluted water supply or even fertilized with human excreta. It is unfortunate that the chlorination of water supplies, which is so effective against bacterial contaminations, is ineffective against the cysts of *E. histolytica*.

In countries where amoebic dysentery is rife, drinking water should be boiled, and uncooked vegetables should not be eaten.

E. histolytica can persist for years in the colon, multiplying by simple fission in the bowel, and producing cysts which are discharged in the stools. Evidence has been advanced for the view that invasion of the portal venous radicles is an occasional event, sometimes occurring years after infection of the bowel by *E. histolytica*, and occasionally from sub-clinical bowel infections which would have been unnoticed by the patient (Paul, 1960).

Travellers to foreign climes where sanitation is poor, and to countries like

America where sanitation is good but amoebic infections widespread, are liable on occasion to develop liver abscesses years after their return home.

PARASITOLOGY

The only amoeba capable of invading the tissues is *E. histolytica*, which was clearly differentiated from *E. coli* by Schaudinn in 1903. While *E. coli* is a harmless commensal feeding only on the contents of the large intestine, *E. histolytica* is so likely to invade the wall of the bowel that its presence or that of its cysts in the stools is a sufficient warrant for instituting treatment by antiamoebic drugs. There is, however, no evidence to exclude the possibility that *E. histolytica* could live in the large bowel as a harmless commensal, and the factors which determine its invasion of the bowel wall are unknown.

PATHOLOGY

Routes of Invasion

Amoebiasis is initiated by invasion of the wall of the large intestine by *E. histolytica*. The amoeba secretes a cytolysin which digests a passage through the mucosa of the bowel. The initial lesion is a small superficial pin-point crater in the mucosa which widens into an ulcer. The lesions are multiple and may extend from the ileocaecal valve to the anus. The lesions are most marked in the proximal part of the right colon and the distal part of the left colon. Amoebae which have invaded the wall of the bowel may work their way into the radicles of the mesenteric veins, to be carried from there as emboli in the portal blood stream to lodge in the liver. The suppurative lesions in the liver are the commonest extrabowel lesions of amoebiasis.

Sometimes suppuration in the liver extends through the diaphragm into the base of the right lung, from whence amoebae may very occasionally be carried by the radicles of the pulmonary veins through the left heart into the general circulation to lodge in any part of the body. These lesions are rare, but the least uncommon of them is suppuration in the cerebral hemispheres, probably because the amoebae are swept most often into the common and internal carotid arteries, these arteries being in direct line with the stream of blood in the ascending aorta. Brain abscesses with no liver or lung lesions have been recorded (Swartzwelder and McGill, 1949).

Hepatic Lesions

Post mortem examinations in patients who have died of amoebic abscess of the liver show that there may be more than one abscess, and that some of the lesions are spherical necrotic masses containing no liquid pus (*Figure 1*). Sometimes there are, besides the main lesions, myriads of pin-head abscesses studding the liver.

Examination of livers extensively invaded by amoebae show that all lesions are bordered by normal liver tissue with no separating zone of fibrosis. Amoebae are demonstrable in the pus and in the granulation tissue of the lesions, but there are no amoebae in the liver tissue beyond the lesions. There is, therefore, no evidence of a widespread invasion of the liver by myriads of amoebae causing a diffuse inflammation.

The concept of amoebic hepatitis (Rogers, 1922) has been widely accepted, but the lesion described was never found at a post mortem examination. Does amoebic hepatitis exist? The pain and tenderness over the right intercostal spaces overlying the liver are evidence, not of a generalized

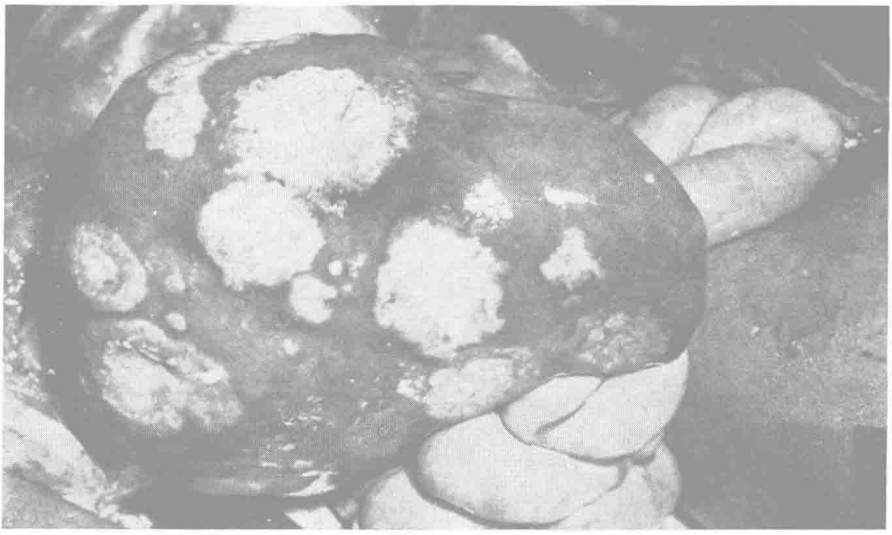

Figure 1.—Multiple, solid, spherical, necrotic amoebic lesions of the liver seen at post mortem examination.

hepatitis, but of an inflammatory lesion in the liver presenting on its right lateral surface. Such a lesion is not amoebic hepatitis. The fact that pus is not found when such a lesion is explored with a needle means that the needle has missed the lesion or that the lesion is a solid necrotic mass.

Nature of hepatic lesions

The large abscess of the liver containing anything from several ounces to several pints of pus is found in populations where amoebic dysentery is rife, and when it is seen very occasionally in other countries it occurs in people who have lived at one time in endemic areas. The only pyogenic infections of the liver occurring in people who have not been exposed to the hazard of amoebic dysentery are suppurative cholangitis and suppurative pylephlebitis and in such cases the pus is mainly confined to the dilated biliary or venous channels. The evidence indicates that large liver abscesses are amoebic.

The pus in a liver abscess may or may not contain amoebae or pyogenic micro-organisms. Direct demonstration of the amoebic origin of a liver abscess is therefore not possible in all cases. The sterile abscess gives evidence that both amoebae and micro-organisms could die out in the pus. The reports of liver abscesses caused by micro-organisms come from countries where amoebic dysentery is rife (Rothenberg and Linder, 1934; Oschner, De Bakey and Murray, 1938; McFadzean, Chang and Wong, 1953). Davidson (1964) reported a solitary pyogenic liver abscess in Great Britain.

Differences between the Lesions in the Bowel and the Lesions in the Liver, Lungs, Brain and other Distant Sites

Amoebae which have invaded the wall of the bowel sometimes remain for years in this site, producing both vegetative forms and cysts, and giving rise to exacerbations of disease from time to time. With the chronic bowel lesions there is sometimes much fibrosis.

The amoebae which enter the portal blood stream, on the other hand, give rise only to acute suppurative lesions, with no fibrosis to indicate that they are of long duration. The extrabowel lesions produce marked constitutional disturbances, remittent fever with chills and rigors, anorexia and profound emaciation. The duration of these symptoms is from a few days to, at the most, a few weeks. In cases coming to necropsy with suppuration in the liver, base of right lung and brain, all lesions are acute suppurations of the same maturity.

The evidence suggests that amoebae do not normally invade the portal blood stream. When they do, amoebae from the bowel wall pass into the blood stream for a short time only, producing acute suppuration in the liver and occasionally at the other sites mentioned. These lesions cause much constitutional disturbance, and the patient either recovers with complete resolution of all these lesions, or he dies from their rapid extension. Although the amoebae at these extrabowel sites proliferate rapidly by binary fission, they do not survive for long and never produce cysts. It is probable that an environment outside the large bowel is unfavourable for *E. histolytica.*

BOWEL LESIONS OF AMOEBIASIS

Pathology

Amoebic dysentery

The ulcers in the mucosa of the large bowel caused by amoebae may extend from the ileocaecal valve to the anus. The small intestine is unaffected except for lesions which have been reported in the last few inches of the ileum in patients with extensive ulceration in the caecum. The ulcers extend down to the muscularis mucosae, and should the amoebae be destroyed healing will occur without scarring. Perforations of the colon occur very occasionally in patients with fulminant amoebic dysentery.

Acute amoebic dysentery is very liable to become chronic, or to develop into recurrent attacks because of the persistence of amoebae in the granulations of the bowel ulcers. Amoebae can be latent in the bowel wall for many years, causing, at a later date, dysentery or abscesses in the liver.

Amoeboma

The amoeboma is a rare lesion which causes considerable diagnostic difficulties. Amoebae persist in a segment of the colon causing thickening of the bowel wall due to fibrosis and granulation. These localized segments are palpable through the abdominal wall and closely resemble a carcinoma of the colon. In the rectum the localized segment causes a sanguineous discharge and the moist proliferative granulations are readily felt and seen from the anal orifice. A biopsy of rectal granulations taken from within the anal canal will

show myriads of amoebae. In the colon the biopsy is usually taken after the mass has been excised by resection of the bowel for carcinoma. This is a matter for regret, as the mass may have regressed on treatment with anti-amoebic drugs.

Management

Patients suffering from dysentery should have their stools examined for *E. histolytica* and its cysts. *E. minuta* is pathogenic and should be dealt with. The earlier treatment with antiamoebic drugs is instituted, the less the chance of the entamoeba entrenching itself and living for years in the bowel wall. Treatment with antiamoebic drugs must be thorough, and should only be given after finding *E. histolytica* or its cysts in the stools, in scrapings of rectal ulcers or in biopsy material from granulomatous masses. The best drug for the control of acute amoebiasis is emetine hydrochloride 40 mg. ($\frac{2}{3}$ gr.)/day by deep subcutaneous injection for 6 days. The patient must be confined to bed as the drug affects the cardiac muscle. A 10-day course of emetine bismuth iodide tablets, 60 mg. (1 gr.) 3 times a day, acts directly on the amoebae in the bowel and as the drug is not absorbed to any appreciable degree by the blood, the risk of cardiac failure is minimal. For the amoebomas, recognized by finding amoebae or its cysts in the stools, treatment with emetine should also be given. Disappearance of the mass will confirm that the mass was, in fact, an amoeboma.

The chronic or relapsing amoebic dysenteries of the colon are difficult to cure. Many courses of treatment with changes to other antibiotic drugs will be necessary in such cases. Chloroquine phosphate tablets 150 mg. 4 times a day for 6 days is the best of these alternative drugs.

Complications

Perianal cutaneous amoebiasis

One of the most serious complications of amoebic colitis is perianal cutaneous amoebiasis. The perianal skin ulcerates and the ulceration spreads; there is also increasing toxaemia. The ulcers open into necrotic cavities in the ischiorectal fossae. Biopsy of the granulations will show numerous amoebae.

The perianal skin is the site of these lesions as ischiorectal abscesses and fistulae-in-ano appear in the perianal area. Should such infections occur in a patient suffering from amoebic colitis, amoebae could infiltrate from the rectal wall into the perianal tissues, and once there they could invade the skin and subcutaneous tissues causing an ever-spreading necrosis. The lesion is often unrecognized as a lesion of amoebiasis and death occurs from increasing toxaemia. Recognition is important, for treatment with antiamoebic drugs can determine resolution of the lesions with return to normal.

AMOEBIASIS OF THE LIVER—LIVER ABSCESS

Clinical Picture

The sudden onset of fever with chills or rigors heralds the formation of a liver abscess, and the marked emaciation within the space of a few days or weeks is very characteristic of these infections.

In the classical case, there is pain and tenderness over the right lower intercostal spaces, usually over a circumscribed area. Bulging of the intercostal spaces at the site of the pain and tenderness and even pitting oedema are to be found in many of these classical cases. The confirmation of the diagnosis by the insertion of an exploring needle through the affected intercostal spaces should always be concluded by continued aspiration until no more pus can be obtained. Elevation of the right dome of the diaphragm in an x-ray picture in cases with the classical picture of a liver abscess would indicate a liver lesion of appreciable size bulging both the right lateral surface and the right superior surfaces of the liver.

Diagnosis

A liver abscess becomes clinically evident when it causes general constitutional disturbances. Fever with chills and rigors, anorexia, and profound emaciation cause the patient to seek advice within a few days, or at the most a few weeks of their onset. These constitutional disturbances give no hint that the causative lesion is in the liver, and except in patients in whom there is jaundice, the diagnosis of a liver abscess is made from concomitant symptoms and signs of a local lesion in the liver. These local symptoms and signs are not obtrusive, and a careful clinical examination is needed for their elucidation.

The descriptions given of a liver abscess are those of the common case where the abscess presents under the right lower chest wall causing pain and tenderness over the intercostal spaces. No note is taken of the obvious proposition that an abscess presenting at other surfaces of the liver would not have these localizing signs. The problem is therefore whether an abscess deeply embedded in the liver produces localizing signs or whether there are no localizing signs until it reaches the surface of the liver. Many patients come for advice within a few days of the onset of the general constitutional disturbances, and at this stage there are nearly always localizing symptoms and signs. The early evidence of localizing signs suggests that a liver abscess matures rapidly and reaches the surface of the liver within a few days. The severity of the symptoms of general constitutional disturbance gives evidence that the lesion is an acute suppuration, and the fact that the symptoms are of a few weeks' duration, at the most, suggests that after some weeks the lesions either resolve completely or cause the death of the patient. There is no evidence that a liver abscess persists for as long as a year, nor that it is ever present as a latent lesion [vide Doxiades and his colleagues (1961) for a different view].

Clinical syndromes characterizing abscesses at different sites in the liver

The localizing symptoms and signs of a liver abscess will depend on which surface of the liver the abscess presents and whether or not the abscess has ruptured on this surface. The different clinical syndromes will establish not only that there is a liver abscess, but also at which surface of the liver the abscess has presented.

Right and left lobe abscesses.—A right lobe abscess is more frequent than a left lobe abscess. The right lobe abscess presents under the right lower chest or in the right upper abdomen, while the left lobe abscess presents in the epigastrium. These differences are dependent not on the precise line demarcating liver, formed from, and drained by the right and left hepatic ducts, but

on the configuration and surroundings of the right and left parts of the liver. The line best demarcating abscesses in the right of the liver from those in the left is one encircling the liver along the long axis of the gall-bladder and of that part of the inferior vena cava which is embedded in the liver (*Figure 2a*),

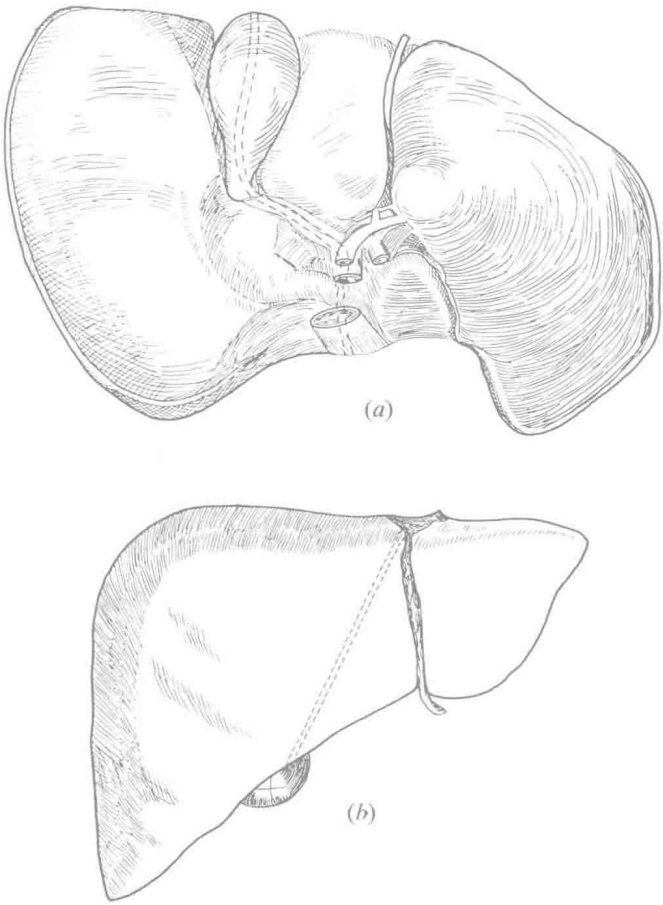

Figure 2.—The line proposed for dividing right lobe from left lobe abscesses of the liver.

and passing over the front of the liver from the posterior end of the falciform ligament to the fundus of the gall-bladder (*Figure 2b*). This line includes in the left lobe a triangular area on the anterior surface between the demarcating line and the falciform ligament, as well as the quadrate and caudate lobes.

Right Lobe Liver Abscess

Right lobe abscess presenting under the right lower chest wall

The liver is separated from the right lower chest wall by the muscular fibres of the right cupola of the diaphragm, and intervening between the

parietal chest wall and the diaphragm is the costophrenic sulcus of the pleura as well as the edge of the right lung. Despite the considerable thickness of tissue intervening between the surface of the liver and the skin overlying it, an abscess in this site commonly presents with pain and tenderness over the intercostal spaces, and sometimes with bulging of these intercostal spaces and pitting oedema as well. The area of pain and tenderness may be circumscribed to an inch over 1 or 2 intercostal spaces or it may extend widely for several inches over 5 or 6 intercostal spaces. The abscess must be beneath the area of maximum tenderness, and an aspirating needle inserted from the skin into the liver at this site will draw pus if this is present.

If an abscess under the right chest wall causes pain before it reaches the surface of the liver, there will be pain without tenderness, for the thickness of tissue separating the skin from the liver will prevent the examining finger from pressing on the abscess if it is deep to the surface of the liver. The records of cases show that pain and tenderness come together, and it is evident that the abscess causes pain only when it reaches the surface of the liver.

An abscess presenting under the right chest wall can rupture on the surface of the liver. When this happens the pus collects between the liver and the diaphragm, and escapes from there into the general peritoneal cavity causing diffuse abdominal pain and rigidity. There is little tendency to form a subphrenic abscess because of the respiratory movements causing the liver to slide up and down past the muscular fibres of the diaphragm. For the same reason, although an abscess presenting under the chest wall can cause bulging and pitting oedema of the intercostal spaces, it would not erode a passage through the diaphragm and chest wall to open on to the skin.

Right lobe abscess presenting under the central tendon of the diaphragm

An abscess presenting beneath the central tendon of the diaphragm will cause inflammation of that structure, and this will become evident by pain referred to the top of the right shoulder. The central tendon of the diaphragm is in apposition to the superior surface of the liver during all phases of the movements of respiration, and an abscess at this site is liable to rupture through the central tendon into the right pleural cavity.

It is very necessary, in countries where amoebic abscesses are common, to pay particular heed to the patient who has the disturbances of a pyogenic lesion and complains of pain on top of the right shoulder. These abscesses are usually so large that they also present under the right chest wall causing pain and tenderness there, which gives additional evidence that the abscess is in the liver.

The rupture of an abscess presenting under the central tendon of the diaphragm is, generally, an unheralded event, for the abscess is latent until it reaches the surface of the liver, and even then it will not cause pain on top of the right shoulder until the inflammation extends to the central tendon. The abscess could also rupture into the general peritoneal cavity without warning, causing diffuse abdominal pain and generalized abdominal rigidity. Pain over the top of the right shoulder, even if present, would be unnoticed on account of the intensity of the abdominal pain.

Should the abscess erode a passage through the central tendon of the diaphragm, rupture will occur either into a clean pleural cavity, or into the

right lung after fixation of its base from the inflammation. Rupture into the pleural cavity may cause death from pleural shock, or an empyema with amoebic pus from the liver. Rupture into the right lung causes severe constitutional disturbance till the abscess opens into a bronchus. The coughing up of amoebic pus generally heralds an improvement in the condition of the patient.

Liver abscess presenting in the right hypochondrium

The right hypochondrium demarcated by the vertical lines and the horizontal planes as defined by anatomists is a triangle of so limited an area as to be of little value for defining the position of swellings in the abdomen. A better definition would be that of a swelling in the right upper abdomen lateral to the outer border of the rectus abdominis muscle.

An abscess at this site could still be within a liver which has enlarged below the right costal margin, or it could be a walled-in collection of pus from an abscess presenting on the colic impression of the inferior surface of the right lobe. An abscess within an enlarged liver would give a swelling which would move on respiration, whilst an intraperitoneal collection of pus would not.

Although an abscess presenting on the right lateral or anterior surfaces of the right lobe of the liver does not usually form sub-phrenic abscesses because of the movement of the liver on respiration, if this does occur the abscess presents below the right costal margin as a swelling in the right hypochondrium.

Liver abscess presenting in the right loin

An abscess presenting in the right loin can be a collection of pus in the perinephric cellular tissues, an intraperitoneal abscess, or an abscess still within the liver which has enlarged downwards to occupy the right loin. An abscess which is still within the liver will move on respiration, whilst a perinephric space abscess and an intraperitoneal abscess will not.

An abscess in the perinephric space can have originated from an abscess presenting in the bare area of the liver which has ruptured into the retroperitoneal space. Abscesses presenting on the posterior surface of the right lobe of the liver, or on the renal surface of the liver would usually rupture into the peritoneal cavity, and if walled off would give an intraperitoneal abscess bulging the right loin. The parietal peritoneum is a considerable barrier to the pus from an amoebic abscess, and the rare rupture through the parietal peritoneum in the loin will produce either a perinephric abscess, or the dreaded complication of invasion of the muscles and skin of the abdominal wall.

Liver abscesses presenting in the right loin offer interesting diagnostic problems. Liver abscess pus in the retroperitoneal tissues of the loin will occupy the same site as a perinephric abscess. Pus from a perinephric abscess could have all the characteristics of liver abscess pus, and drainage of a liver abscess in the belief that it was a perinephric abscess could result in the amoebic infiltration of the skin and muscles (*Figure 3*). An intraperitoneal abscess from the liver presenting in the right loin could occupy the same site

as an abscess from a retrocaecal appendix. The pus from an appendix usually has an offensive odour. Drainage of a liver abscess in the belief that it was an appendicular abscess could also cause amoebic invasion of skin and muscles.

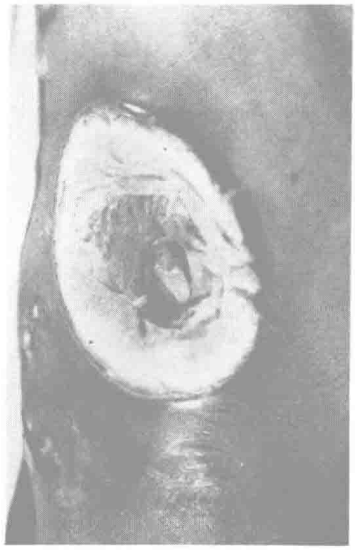

Figure 3.—Photograph of a patient 2 days before death. A liver abscess presenting in the right loin had been incised in the belief that it was a perinephric abscess. The extensive amoebic infiltration of skin and abdominal wall is seen in the picture. The twelfth rib is in the floor of the lesion.

Abscesses presenting in the loin should be aspirated first and the pus examined for amoebae. Due weight should be given to tenderness over intercostal spaces, to a past history of amoebic dysentery, and to the short history of fever with marked emaciation.

Left Lobe Liver Abscess

The left lobe abscess manifests itself by a swelling in the epigastrium. The epigastrium as demarcated by the vertical lines and horizontal planes by the anatomists is too limited to be of value in localizing the epigastric swellings of clinical practice. The natural lower boundary of the epigastrium is the transverse colon with its apron of greater omentum and the boundary passes from the fundus of the gall-bladder in a curve convex downwards through the umbilicus and back to the sub-costal margin on the left, at its lowest level.

Left lobe abscess presenting within the liver in the epigastrium

The normal liver lies under the anterior abdominal wall only in the triangle bounded by the two margins of the costal arches and by a line passing from the tip of the ninth right costal cartilage to the tip of the eighth left costal cartilage. An abscess within the liver at this place causes a swelling in the upper reaches of the epigastrium, and even if the liver enlarges downwards the swelling will still be in the epigastrium. Such a swelling is tender, and moves on respiration. It emerges from beneath the costal arches and has a palpable lower edge (*Figure 4*). In countries where liver abscesses are common, a swelling in the epigastrium with these characteristics could be recognized

as a liver abscess with such certainty as to warrant the insertion of an aspirating needle into the swelling through the abdominal wall.

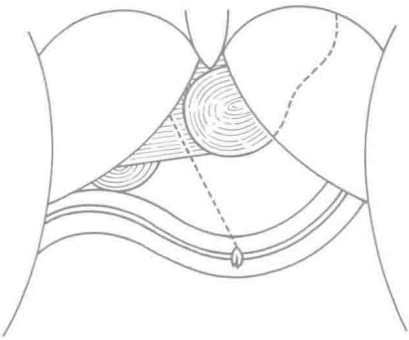

Figure 4.—Left lobe abscess presenting within the liver in the epigastrium.

Intraperitoneal abscess in the epigastrium from rupture of liver abscesses

Epigastric abscess below the liver.—An abscess in the epigastrium below the liver presents with a swelling reaching down to the umbilicus. It is an intraperitoneal collection of pus from the rupture of an abscess on the inferior surface of the left lobe (*Figure 5*). The pus is walled in below by the transverse colon and the great omentum. The swelling does not move on respiration.

Epigastric abscess in front of the liver.—An epigastric abscess in front of the liver presents as a swelling in the epigastrium filling the whole of the triangle between the sub-costal arches down to the umbilicus. The pus is a large intraperitoneal collection extending from the left cupola of the dia-

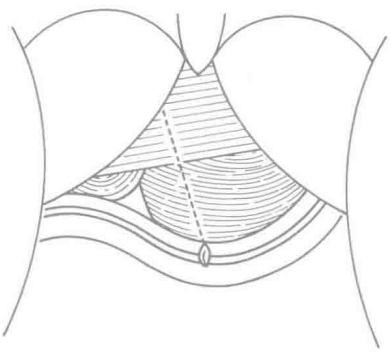

Figure 5.—Intraperitoneal epigastric abscess from rupture of liver abscess on inferior surface of left lobe.

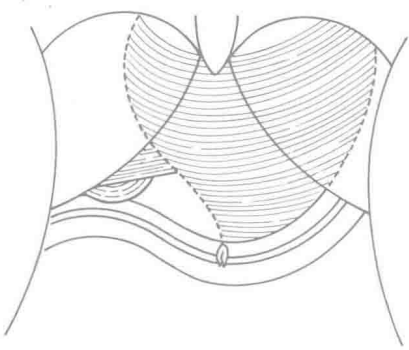

Figure 6.—Intraperitoneal epigastric abscess from rupture of liver abscess on anterior or superior surface of left lobe.

phragm down to the umbilicus where it is walled in by the transverse colon and the great omentum. The falciform ligament walls in the pus in the right (*Figure 6*). The pus comes from the rupture of an abscess on the superior or anterior surface of the left lobe of the liver. The swelling in the epigastrium does not move on respiration.