Intrathoracic Presentation of Amebic Liver Abscess

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ABSTRACT Amebic infection is endemic in tropical and subtropical countries and still remains a common cause of chronic morbidity in these areas. This is a report of 10 patients with different intrathoracic presentations of amebic liver abscess who were treated surgically after conservative measures had failed. Five of these patients had empyema when first seen, 3 had lung abscess, and 1 had intrathoracic shadow that proved on exploration to be an amebic liver abscess.

All these 9 patients had abscesses on the right side secondary to amebic liver abscess of the right lobe of the liver. The tenth patient had amebic pericarditis secondary to amebic abscess of the left lobe of the liver. Failure of conservative treatment in these patients is attributed to the thick nature of the amebic pus and the severe reaction of the pleura and pericardium to the amebic infection. To avoid the serious complication of pleuropulmonary amebiasis, early operation is advised for large liver abscesses that are unlikely to be controlled by conservative treatment. Transpleural drainage of such abscesses gives direct approach to their sites, which are commonly located in the superior part of the right lobe of the liver. Such drainage has proved to be safe provided that the patient is receiving antiamebic drug treatment.

Amebic infection caused by the protozoan parasite *Entamoeba histolytica* is a widespread endemic infection in many of the tropical and subtropical areas of the world. Despite the common use of antiamebic drugs, it still remains one of the foremost causes of chronic morbidity in the endemic areas [1, 4, 11]. Amebic infection is either intestinal, producing amebic dysentery, or extraintestinal, producing mainly amebic hepatitis. *Entamoeba histolytica* gains access to the colon following ingestion of food or drink contaminated with the cysts of the parasite. In the stomach the wall of the cysts is digested, liberating the parasite, which through its cytolytic enzymes finds its way to the submucosa of the colon, where it produces the characteristic flask-shaped ulcers. At any time during this phase of amebic colitis, *E. histolytica* may erode branches of the portal vein and reach the liver as emboli in the portal blood.

Once established in the liver sinusoids, the amebae produce cytolytic destruction in the liver. This process may be diffuse, giving rise to diffuse amebic hepatitis, or localized, producing an amebic liver abscess, which is commonly found in the right lobe of the liver and less often in the left lobe. Characteristically, the abscess is solitary, containing chocolate brown or anchovy-colored saucelike pus that is sterile on culture. Amebic liver abscess may be secondarily infected; it may rupture in the sub-phrenic area or general peritoneal cavity; it may erode the diaphragmatic opening in the pleura, producing empyema; or it may work its way into the lung, giving rise to lung abscess. Pleuropulmonary complications are more common on the right side, but the left lobe abscess may open into the left side and, rarely, into the pericardium.

Clinical Material and Methods

From 1966 to 1975, 10 patients with different intrathoracic presentations of amebic liver abscess were treated surgically in our Cardio-Thoracic Surgery Unit after conservative management failed. Five of these patients had right-sided empyema, 3 others had right lung abscess, and another had what was diagnosed as a right intrathoracic shadow but proved on exploration to be an uncomplicated abscess in the right lobe of the liver. The tenth patient had amebic pericarditis secondary to left lobe abscess of the liver (Table). All these patients were men; their ages ranged from 17 to 40 years with a mean of 37.5 years. At one stage these patients were diagnosed clinically as having
Types of Intrathoracic Amebiasis and Their Surgical Management

<table>
<thead>
<tr>
<th>Intrathoracic Lesion</th>
<th>No. of Patients</th>
<th>Operation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right empyema</td>
<td>4</td>
<td>Decortication &amp; drainage of liver abscess</td>
</tr>
<tr>
<td>Right empyema with hepatobronchial fistula</td>
<td>1</td>
<td>Decortication &amp; transpleural drainage of liver abscess</td>
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<tr>
<td>Right lung abscess</td>
<td></td>
<td></td>
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<tr>
<td>Middle lobe</td>
<td>1</td>
<td>Middle lobectomy &amp; intrathoracic drainage of liver abscess</td>
</tr>
<tr>
<td>Lower lobe</td>
<td>1</td>
<td>Lower lobectomy &amp; transpleural drainage of liver abscess</td>
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<tr>
<td>Middle and lower lobe</td>
<td>1</td>
<td>Middle &amp; lower lobectomy, then transpleural drainage of liver abscess</td>
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<tr>
<td>Intrathoracic shadow</td>
<td></td>
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<tr>
<td>&quot;liver abscess&quot;</td>
<td>1</td>
<td>Transpleural drainage</td>
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<tr>
<td>Amebic pericarditis</td>
<td>1</td>
<td>Pericardiectomy</td>
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</tbody>
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Fig 1. Right amebic empyema. This patient had hepatobronchial fistula through the middle lobe.

amebic liver abscess and they received full courses of antiamebic treatment in the form of emetine hydrochloride, chloroquine, and tetracyclines. The intrathoracic complications occurred either during or sometime after the course of drug treatment.

Pleuropulmonary Amebiasis

All 8 patients with pleuropulmonary amebiasis had it on their right side. Of the 8, 5 had pleural amebiasis in the form of empyema, 1 with hepatobronchial fistula manifested by the coughing up of characteristic amebic pus (Fig 1). The 3 other patients had pulmonary amebiasis in the form of a lung abscess (Fig 2).

The main presenting symptoms that indicated the pleuropulmonary complications were dyspnea together with a dull, aching pain of pleuritic nature that was referred to the right shoulder in 3 patients. Coughing up of amebic pus is a pathognomonic sign of hepatobronchial fistula; this was found in 1 patient with empyema. Mild hemoptysis was found in 2 patients with lung abscess. Mild to moderate temperature elevation ranging from 37.5°C to 38°C was a common finding among these patients. The right lobe of the liver was enlarged both upward and downward, and intercostal tenderness could be elicited in all the patients with pleuropulmonary
amebiasis, mainly in the midaxillary line of the eighth and ninth intercostal spaces. None of the patients were jaundiced, but there was a mild to moderate degree of leukocytosis. Vegetative forms of *E. histolytica* could not be recovered from the stools of these patients, but cysts were found in the stools in 6 of them. Chest roentgenograms showed elevation of the right leaflet of the diaphragm together with opacity of the empyema in 5 patients and consolidation of the middle and lower lobes in the 3 patients with lung abscess.

Conservative treatment was tried first, using emetine hydrochloride, 60 mg intramuscularly for 5 days, and chloroquine tablets, 600 mg/day for 10 days, together with oxytetracycline or ampicillin, 1 gm/day for 5 days. For patients in poor general condition, metronidazole, 1.2 gm/day, was given for 10 to 14 days instead of emetine hydrochloride. Needle aspiration was done for patients with empyema, but it was not easy and the results were unsatisfactory because of the thick, viscid pus. Intercostal drainage was tried in 3 of these patients, 1 of them with hepatobronchial fistula, but the lung did not expand. For patients with lung abscess postural drainage was encouraged, but no roentgenographic improvement was observed. The pleural aspirate was sterile on culture and positive for vegetative forms of *E. histolytica* in all patients with empyema.

In view of the failure of conservative treatment in patients with pleuropulmonary amebiasis, operation was contemplated. Decortication was carried out in 5 patients with empyema. The parietal as well as the visceral pleurae were quite thick in relation to the relatively short period of contamination, which on the average had been three weeks. When the lower part of the lung was dissected from the diaphragm, a rent in the superior part of the diaphragm leading to the abscess in the right lobe of the liver was found in all 5 patients.

In the patient with hepatobronchial fistula the middle lobe adhered to the liver abscess, and on dissection there was some bronchiolar air leak. After dissection the middle lobe expanded completely on inflation, and thus it was preserved. Four months later a bronchogram showed expansion of the middle lobe and no hepatobronchial communication (Fig 3).

In patients with lung abscess the affected part of the lung was consolidated, and on dissection a communication between the liver abscess and lung through an opening in the diaphragm was found. Lobectomy was done for the affected part of the lung using the standard procedure. His-
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Fig 3. (A) Right bronchogram of the same patient as in Figure 1 four months after decortication operation preserving the middle lobe. (B) Right lateral view of the same patient. Note the full expansion of the middle lobe with no trace of the hepatobronchial fistula. The right leaflet of the diaphragm is still high but much less so than before, and there is no evidence of any diaphragmatic herniation.

tological examination of the lung abscess proved positive for *E. histolytica*. Following decortication or lobectomy, intrapleural drainage of the abscess was instituted. The opening in the diaphragm was widened, and the abscess was removed; its contents were thick and had the appearance of a brownish anchovy sauce. In all the patients with pleuropulmonary amebiasis the liver abscess was solitary and situated in the most superior part of the right lobe of the liver; its diameter ranged from $5 \times 2$ to $15 \times 10$ cm. The right leaflet of the diaphragm was found to be adherent to the right lobe of the liver with no subphrenic space. Intercostal drainage was continued for 3 to 5 days after the operation.

In all the patients, antiamebic treatment was continued in the form of metronidazole, 1.2 gm/day for 10 days, and ampicillin, 1.5 gm/day for 5 days. The postoperative period was uneventful, and early chest roentgenograms showed clear lung fields, expansion of the lungs, but a raised right leaflet of the diaphragm.

Intrathoracic Amebic Shadow

A man aged 25 years complained of pain in the right hypochondrium and had a low-grade fever of 37.5°C. Clinical examination of the chest and heart revealed nothing, but his liver was slightly tender. A chest roentgenogram showed a right oval shadow close to the cardiophrenic angle. Blood analyses were within normal limits. He was diagnosed provisionally as having amebic hepatitis and received a full course of emetine hydrochloride and oxytetracycline, but no improvement was seen on subsequent roentgenograms. Further clinical evaluation and screening raised the possibility of an intrapulmonary lesion or a pericardial cyst (Fig 4), and thus right thoracotomy was advised.

Exploration revealed a normal right lung and normal pericardium, but the anterior part of the diaphragm was raised and fluctuant. Aspiration of the typical anchovy sauce pus confirmed the diagnosis of amebic liver abscess bulging into the chest. Because experience has shown that drainage of amebic liver abscess into the pleura is quite safe as long as the patient is on antiamebic treatment, transpleural drainage of the abscess was performed. The elevated part of the diaphragm was thin and stretched. It was incised to about 5 cm in length, and a huge amount of pus, amounting to nearly half a liter, was suctioned out. The abscess was completely re-
Fig 4. (A) Intrathoracic shadow proved on exploration to be amebic liver abscess in the right lobe of the liver bulging into the chest. The provisional diagnosis was either intrapulmonary lesion or pericardial cyst. Transpleural drainage was done successfully. (B) Right lateral view of the same patient.

moved, and the diaphragm was left open to allow the abscess to drain into the pleural cavity, where the intercostal drain was left for 5 days. The patient had an uneventful recovery. The aspirate was sterile on culture and positive for *E. histolytica*, which was also confirmed by histological examination of the abscess wall. Four weeks later a chest roentgenogram showed clear lung fields, and the right leaflet of the diaphragm was seen to be slightly raised.

**Pericardial Amebiasis**

A 17-year-old boy had a history of amebic dysentery four years before the present illness. He developed amebic liver abscess of the left lobe of the liver and received a full course of emetine hydrochloride and ampicillin with marked regression in the size of the liver abscess. A few days later his general condition deteriorated, with dyspnea, orthopnea, and an increase in cardiac dullness. His temperature was 37.7°C, and he had a paradoxical pulse of 110 per minute, blood pressure of 70/50 mm Hg, and cold extremities, peripheral cyanosis, and engorged neck veins—the picture of cardiac tamponade. The chest roentgenogram showed globular enlargement of the heart shadow and raised left diaphragmatic copula with right pleural effusion (Fig 5A). The total leukocyte count was 11,000 per mm$^3$ with 75% polymorphonuclear leukocytes, and the electrocardiogram showed a low-voltage QRS complex in the limb and chest leads. Repeated pericardial aspirations were done below the xiphoid process, and amounts ranging between 250 and 500 ml of typical amebic pus were withdrawn that proved sterile on ordinary culture and positive for the vegetative forms of *E. histolytica*. Pleural aspiration was done concomitantly twice and yielded 700 and 500 ml, respectively, of straw-colored fluid that was sterile on culture.

About three weeks from the start of the disease the patient's liver returned to normal size, but his neck veins were still engorged and the cardiac size did not show any improvement on roentgenogram. His general condition remained poor. These clinical changes were explained by the development of pericardial constriction, and we decided to perform a pericardiectomy.

Through a left anterolateral thoracotomy incision, the pericardium was found to be tense and full of fibrinous clots. It was excised entirely from the left ventricle and from most of the right side. The pericardium was quite thick in relation to the fairly short duration of the disease, reaching 4 mm in thickness in some parts. The
epicardium was covered by a thin layer of fibrin that interfered with the cardiac action. There was no trace of any communication between the pericardium and the left lobe of the liver or any structure below the diaphragm, which may be explained by the fact that by the time of the operation the liver abscess had healed completely. Histological examination of the pericardium showed chronic, nonspecific inflammation and organization of granulation tissue at various stages of maturation.

The patient had an uneventful recovery and received metronidazole, 1.2 gm/day, and ampicillin, 1.5 gm/day, for one week. Two months after the operation the patient had returned to normal activities and his chest roentgenogram showed a normal-sized heart and clear lung fields (see Fig 5B).

Follow-up
All the patients in this series were followed from two to eighteen months after operation with no mortality or morbidity. On roentgenograms the lung fields were clear and the diaphragm was found to be raised with no evidence of herniation of the liver through the diaphragmatic defect created for the transpleural drainage of the liver abscess. It takes some time for the diaphragm to regress to its normal level.

Comment
Intrathoracic amebiasis is a serious complication of amebic liver abscess which commonly affects the right lobe of the liver [2, 4, 9] and thus explains the frequent involvement of the right side of the chest. *Entamoeba histolytica* finds its way directly to the thorax through the diaphragm by its lytic action, which erodes the diaphragm. Infection borne through the blood or the lymphatics may explain pulmonary involvement in parts of the lungs which are not directly related to the diaphragm or in patients with no evident hepatic involvement [3, 8]. Diffuse amebic hepatitis or small amebic abscesses can be controlled by antiamebic treatment, but large abscesses with copious amounts of pus cannot be cured by conservative means alone. Such liver abscesses are the source of the pleuropulmonary complications. The development of intrathoracic involvement, either pleural or pulmonary, depends on the pleural cavity and whether it is obliterated by or free from adhesions [9]. If there are adhesions, once the liver abscess bursts through the diaphragm it can find its way into the parts of the lungs related to the diaphragm—ie, the middle lobe or the lower
lobe on the right side—whereas if there are no pleural adhesions, the abscess will open easily into the pleural cavity. In either case, if the pus is under tension, it can find its way through the bronchi, giving rise to hepatobronchial fistula. This is a serious complication, as secondary infection may occur or spill over to the other lung [3, 9, 10].

Once *E. histolytica* reaches the serous membranes of the pleura or the pericardium, it creates a severe reaction, producing thickening of these membranes out of proportion to the duration of contamination, which is a matter of weeks rather than months. This picture is quite different from that presented by nonspecific or other specific inflammatory processes such as tuberculosis. This early thickening of the pleura in patients with empyema prevents lung expansion and enhances the cardiac constriction in amebic pericarditis, which is exacerbated by the viscid nature of the amebic pus. These factors together explain the failure of conservative treatment in patients with intrathoracic amebiasis. We have found that transpleural drainage of amebic liver abscess is safe provided it is done concurrently with adequate antiamebic treatment. The transthoracic approach also gives direct access to the liver abscess.

Amebic pericarditis is a rare complication. Its incidence is 0.2 to 2.8% among patients with amebic liver abscess [6]. Up to January, 1975, 81 cases of amebic pericarditis had been reported [5], most of them secondary to amebic abscess of the left lobe of the liver. There are just a few reports of amebic pericarditis resulting from the extension of abscesses in the right lobe of the liver or from amebic lung abscess [9, 11].

Amebic pericarditis can be seen in two stages: the presuppurative stage and the suppurative stage. The first stage is sometimes termed the reactive or sympathetic stage [5]. All patients in this stage of amebic pericarditis should be viewed as likely to develop advanced suppurative pericarditis. Conservative treatment in the form of antiamebic treatment and proper pericardial aspiration, if needed, yields a recovery rate of 60%, provided it is started early [7]. The average mortality rate from amebic pericarditis is 40% [5]. If the patient does not improve with conservative treatment, one should consider pericardial constriction. Once this complication develops, pericardiectomy is indicated. In advanced cases there will be liver damage because of chronic venous congestion as well as the possibility of myocardial damage caused by the spread of fibrosis.

We concluded that to prevent the serious complication of intrathoracic amebiasis, large amebic liver abscesses that are unlikely to be controlled by conservative treatment can be drained from the chest by opening the right leaflet of the diaphragm, aspirating the contents, and breaking down any septa in the abscess cavity. This allows adequate drainage into the pleura, where an intercostal drain is placed. In our patients there were no complications from incising the right leaflet of the diaphragm and leaving it open for drainage because of the dense adhesions already present between the diaphragm and liver. Intrapleural drainage of the amebic liver abscess did not lead to any complications in the early or late postoperative period as long as adequate antiamebic treatment was given. Early operation is indicated, particularly in patients with hepatobronchial fistula and also in patients with amebic pericarditis once constriction develops.

References

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